

Dear Reader,

ICHD-3 thoroughly classifies primary and secondary headaches but such an internationally accepted version for primary and secondary facial pains was until now lacking. A collaborative group consisting of members of the Orofacial and Head Pain Special Interest Group (OFHP SIG) of the International Association for the Study of Pain (IASP), the International Network for Orofacial Pain and Related Disorders Methodology (INFORM), the American Academy of Orofacial Pain (AAOP) and the International Headache Society (IHS) has worked over the past 3 years to present such a classification for facial pain which is herewith attached.

Before publishing this first edition (which will change and adapt over time, just like ICHD) we seek advice and suggestion from researchers and clinicians worldwide. Such advice and suggestions will be considered if backed up by publications (i.e. not just based on a clinical impression).

Please send such comments and a list of references to the following email contact:

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and we will forward these to the respective section heads who are also listed page 3.

We hope you will find the reading inspiring.

International Classification of Orofacial Pain

ICOP

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Preface

We are proud to present the first International Classification of Orofacial Pain (ICOP). While anatomically the face is clearly part of the head we have found far too many cases of misdiagnosis where a clear diagnostic classification may have helped avoid misdiagnosis and resultant mis-directed treatment. Anatomical boundaries, and associated medical specialty boundaries, contribute to the problem. For example, the International Headache Society defines facial pain as “pain below the orbitomeatal line, anterior to the pinnae and above the neck”. Other definitions for facial pain include the forehead as an additional area, while orofacial pain (OFP) would necessarily include all of the structures in the oral cavity.

In clinical practice ‘headaches’ often refer to ‘orofacial’ regions and vice versa. At times ‘headaches’ may be located exclusively around the ‘orofacial’ region and may cause significant diagnostic difficulties. OFPs refer to the head presenting a complex clinical phenotype. In particular, dental pain due to local dental pathology is often blamed for primary orofacial and head pains. There is no comprehensive internationally accepted classification that deals with orofacial pain. These were some of the factors that made it clear to us that a classification for orofacial pains was needed. A fundamental principle in this first classification of orofacial pains is that the disorders, not the location of head vs. face, should guide the new conceptualization and diagnostic criteria.

While creating ICOP we were cognizant that the ICHD-3 thoroughly classifies primary and secondary headaches and we refer the reader to the current version of ICHD-3 for those entities. Moreover, to make ICOP a useful tool for researchers and clinicians accustomed to using ICHD-3 we have adopted the hierarchical design and classification style of ICHD-3. The Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) is a well-tested and established classification that includes regional myalgias and TMJ arthralgia. We have adopted the DC/TMD criteria, only including the painful TMDs and modifying the presentation style of the criteria to that of ICHD-3. Overall, the ICOP is also aligned to the ICD-11/IASP criteria for orofacial pains and headaches.

The aim is therefore to create a tool that will enhance research and clinical management of orofacial pain. Additionally, we are confident that methodology we have adopted will bring

professionals working on head, orofacial, eye, nose, sinus and neck pain closer and encourage active collaboration.

The road to ICOP began in 2016 when we first met at the World Congress of the IASP in Yokohama, Japan. In the one day meeting we discussed the structure and established workgroups. A follow up meeting was held in Rutgers School of Dental Medicine, USA in 2017 where we examined the evidence, or lack of, to establish the individual entities making up ICOP. The result is a classification that is backed in many areas by strong evidence and in others by expert opinion that will encourage and guide research. The members of the classification committee represent the major associations involved in orofacial and head pain and are a true international group, strengthening the future of ICOP.

R. Benoliel, P. Svensson

Using ICOP

Because ICOP is modelled on ICHD-3 the instructions for use of ICOP are similar. Many ICHD-3 users will therefore find ICOP easy to use. Like ICHD-3 the document is intended as a tool to consult particularly for research, but also for the clinical diagnosis and management of OFP.

ICOP will serve as a comprehensive research and diagnostic manual and will be particularly useful when the diagnosis is uncertain, or when the clinician is unaware that such a clinical presentation exists. So, we do recommend that practitioners and researchers read through the classification. Moreover, ICOP joins ICHD-3 and IASP/ICD-11 in establishing clear terminology that will allow communication and data sharing in an unambiguous manner. For research, the classification is indispensable: every patient entered into a research project, be it a drug trial or a study of pathophysiology or biochemistry, must fulfil a set of established diagnostic criteria.

This classification is hierarchical, allowing the user to establish a diagnosis from only the first-digit level or extending to the fifth or even seventh digit. The level of resolution in diagnostic coding clearly depends on the intended use. In general practice, only the first- or second-digit diagnoses are usually applied, while in specialist practice and headache centres a diagnosis including a fourth- or fifth-digit (and even sixth or seventh digit) levels is appropriate.

We deal with primary and secondary orofacial pains (OFP) within the same sections. This is in our opinion a more efficient manner of presenting the various disorders, in part because primary and secondary designations become difficult to differentiate in these overlapping disorders.

1. For most purposes, patients receive a diagnosis according to currently present pain or from within the last year. For genetic research and some other uses, occurrence during the entire lifetime is used.
2. Each distinct type, subtype or subform of OFP and headache that the patient has must be separately diagnosed and coded. For example, a severely affected patient may receive multiple diagnoses and codes: 2.1.2.3.2. Chronic persistent primary myofascial pain with pain referral and 3.1.4.1. Chronic primary TMJ arthralgia without referred pain and possibly 1.1 Migraine without aura from ICHD-3.

3. When a patient receives more than one diagnosis, these should be listed in the order of importance to the patient. i.e. establish which of the diagnoses is causing the most suffering and disability in the patient's view?
4. When it is unclear which type of OFP a particular patient is experiencing, other available information should be used in addition to the diagnostic criteria to decide the more likely diagnosis. This could include the longitudinal pain history (how and when did the pain start?), the family history, the effect of drugs, menstrual relationship, age, gender and a range of other features.
5. To receive a particular OFP diagnosis the patient must, in many cases, experience a minimum number of attacks of or number of days with that pain. This number is often specified in the diagnostic criteria for the OFP type. The OFP diagnoses must fulfil a number of other requirements described within the criteria under separate letter headings: A, B, C *etc.* Some letter headings are monothetic: that is, they express a single requirement. Other letter headings are polythetic, requiring for example any two out of four listed characteristics. This structure has been adopted from ICHD-3.
6. The frequency of OFP disorders varies widely, from occurring only every 1–2 years to daily pain. The severity of attacks also varies. Other than for myofascial and TMJ arthralgia, ICOP does not provide a possibility to code for frequency. None of the diagnostic criteria include routine assessment of severity and frequency, but we recommend that frequency and severity be assessed and specified.
7. *Primary or secondary OFP or both:* When a *new OFP* occurs *for the first time* in close temporal relation to another disorder known to cause OFP (e.g. trauma), the new OFP is coded as secondary and attributed or associated to the causative disorder. This remains true even when the OFP has the characteristics of a primary disorder (myofascial pain, arthralgia). When a *pre-existing* OFP becomes *chronic* in close temporal relation to such a causative disorder, both the primary and the secondary diagnoses should be given. When a *pre-existing* primary OFP is made *significantly worse* (usually meaning a two-fold or greater increase in frequency and/or severity) in close temporal relation to such a causative disorder, both the primary and the secondary diagnoses should be given, provided that there is good evidence that the disorder can cause OFP.

8. The last criterion for almost every headache disorder is “Not better accounted for by another ICOP or ICHD-3 diagnosis”. Consideration of other possible diagnoses (the *differential diagnosis*) is a routine part of the clinical diagnostic process. When an OFP appears to fulfil the criteria for a particular disorder, this last criterion is a reminder always to consider other diagnoses that might better explain the OFP. In particular this applies to assessing whether OFP is secondary or primary. In order to accomplish this goal of differential diagnosis, the clinical diagnostic process may need to consider other disorders outside of the ICOP framework, elaborate on the pain history, and use clinical tests beyond those implied in the ICOP criteria. Referred pain from one structure to the other in the orofacial pain region is extremely common but classifying all of these is beyond the scope of ICOP.
9. Many patients with a diagnosed OFP disorder may report occasional pain episodes that are not typical of that diagnosed specific disorder. This can be due to treatment, inability to recall symptoms exactly, or other factors. The clinician should assist the patient in describing typical, untreated or unsuccessfully-treated attacks and the clinician should determine if there have been enough of these to establish the diagnosis. Any less-typical attacks should also be considered when describing attack frequency.
10. When a patient is suspected of having more than one OFP, it is highly recommended that they complete a diagnostic daily pain diary. It has been shown that such pain diaries improve diagnostic accuracy as well as lead to a more precise judgement of medication consumption. Diaries are typically recommended for a month, during which, for each pain episode, the important characteristics are recorded. Additionally, the diary helps in judging the balance between different OFP types or subtypes. Finally, using the diary is an important tool in explaining to the patient how to distinguish between different OFPs, to be aware of medication consumption, to note triggering factors, and become a more reliable source of follow-up information.

Classification overview

1. Orofacial pain associated with disorders of dentoalveolar and associated structures

1.1 Dental pain

1.1.1 Pulpal pain

1.1.1.1. Pulpal pain attributed to hypersensitivity

1.1.1.1.1 Pulpal pain attributed to a crack in the enamel

1.1.1.1.2 Pulpal pain attributed to exposed dentin

1.1.1.1.2.1 Pulpal pain attributed to tooth wear or abrasion

1.1.1.1.2.2 Pulpal pain attributed to fracture resulting in exposed dentin

1.1.1.1.2.3 Pulpal pain attributed to developmental dental hard tissue defect

1.1.1.1.3 Pulpal pain attributed to hypersensitivity associated with dental procedures

1.1.1.1.3.1 Pulpal pain attributed to recent extensive removal of dentin

1.1.1.1.3.2 Pulpal pain attributed to recent placement of a restoration

1.1.1.1.3.3 Pulpal pain attributed to hypersensitivity due to hyperocclusion or

-articulation following dental restorative procedures

1.1.1.1.4 Pulpal pain attributed to central sensitization

1.1.1.1.5 Pulpal pain attributed to other cause

1.1.1.2 Pulpal pain attributed to pulp exposure due to dental trauma

1.1.1.3 Pulpal pain attributed to pulpitis (pulpal inflammation)

1.1.1.3.1 Pulpal pain attributed to reversible pulpitis due to infection of dentin

1.1.1.3.1.1 Pulpal pain attributed to caries that does not extend to the pulp

1.1.1.3.1.2 Pulpal pain attributed to reversible pulpitis due to fracture of enamel, root cementum, dentin or any combination thereof, resulting in exposure of dentin

1.1.1.3.1.3 Pulpal pain attributed to reversible pulpitis due to a tooth crack without evidence of missing tooth substance

1.1.1.3.2 Pulpal pain attributed to irreversible pulpitis due to infection of dentin

1.1.1.3.2.1 Pulpal pain attributed to caries which may extend to the pulp

1.1.1.3.2.2 Pulpal pain attributed to irreversible pulpitis due to dental hard tissue fracture without pulp exposure

- 1.1.1.3.2.3 Pulpal pain attributed to irreversible pulpitis due to visible crack in the enamel without evidence of missing tooth substance
- 1.1.1.3.3 Pulpal pain attributed to irreversible pulpitis due to infection of the dental pulp
 - 1.1.1.3.3.1 Pulpal pain attributed to caries extending to the pulp
 - 1.1.1.3.3.2 Pulpal pain attributed to irreversible pulpitis due to dental hard tissue fracture without pulp exposure
 - 1.1.1.3.4 Pulpal pain attributed to pulpitis due to external cervical root resorption
 - 1.1.1.3.5 Pulpal pain attributed to pulpitis due to other cause
- 1.1.1.4 Pulpal pain attributed to systemic cause

1.1.2 Periodontal pain

- 1.1.2.1 Periodontal pain attributed to periodontitis (periodontal inflammation of the periodontium)
 - 1.1.2.1.1 Periodontal pain attributed to traumatically induced periodontal inflammation
 - 1.1.2.1.1.1 Periodontal pain attributed to hyperocclusion or -articulation
 - 1.1.2.1.1.2 Postoperative periodontal pain
 - 1.1.2.1.1.3 Periodontal pain attributed to accidental dental trauma
 - 1.1.2.1.1.4 Periodontal pain attributed to other trauma or injury
 - 1.1.2.1.2 Periodontal pain attributed to apical periodontitis due to endodontic disease
 - 1.1.2.1.2.1 Periodontal pain attributed to pulpal inflammation
 - 1.1.2.1.2.2 Periodontal pain attributed to endodontic infection
 - 1.1.2.1.2.2.1 Periodontal pain attributed to intraradicular endodontic infection
 - 1.1.2.1.2.2.2 Periodontal pain attributed to extraradicular endodontic infection
 - 1.1.2.1.3 Periodontal pain attributed to periodontal disease
 - 1.1.2.1.3.1 Periodontal pain attributed to chronic periodontitis
 - 1.1.2.1.3.2 Periodontal pain attributed to aggressive periodontitis
 - 1.1.2.1.3.3 Periodontal pain attributed to periodontitis as a manifestation of systemic disorder known to cause periodontitis

1.1.2.1.3.3.1 Periodontal pain attributed to a hematological disorder

1.1.2.1.3.3.2 Periodontal pain attributed to a genetic disorder

1.1.2.1.3.3.3 Periodontal pain attributed to an unspecified systemic disorder

1.1.2.1.3.4 Periodontal pain associated with necrotizing ulcerative periodontitis (NUP)

1.1.2.1.3.5 Periodontal pain associated with periodontal abscess

1.1.2.1.4 Periodontal pain attributed to apical and marginal periodontitis due to combined endodontic infection and periodontal disease

1.1.2.1.5 Periodontal pain attributed to peri-implantitis due to peri-implant infection

1.1.2.2 Periodontal pain attributed to a local non-inflammatory cause

1.1.3 Gingival pain

1.1.3.1 Gingival pain attributed to gingivitis (gingival inflammation)

1.1.3.1.1 Gingival pain attributed to trauma

1.1.3.1.2 Gingival pain attributed to infection

1.1.3.1.2.1 Gingival pain attributed to bacterial infection

1.1.3.1.2.2 Gingival pain attributed to viral infection

1.1.3.1.2.3 Gingival pain attributed to fungal infection

1.1.3.1.3 Gingival pain attributed to autoimmunity

1.1.3.1.4 Gingival pain attributed to allergic reaction

1.1.3.1.5 Gingival pain attributed to gingival inflammation due to other cause

1.1.3.2 Gingival pain attributed to malignant lesion

1.1.3.3 Gingival pain attributed to neuropathy

1.1.3.4 Idiopathic gingival pain

1.2 Non-dental pain

1.2.1 Oral mucosal pain

1.2.1.1 Oral mucosal pain attributed to oral mucosal inflammation

1.2.1.1.1 Oral mucosal pain associated with trauma

1.2.1.1.1.1 Oral mucosal pain attributed to mechanical, thermal, or chemical injury

1.2.1.1.1.2 Oral mucosal pain attributed to surgical or other iatrogenic injury

- 1.2.1.1.1.3 Oral mucosal pain attributed to radiation or chemotherapy
- 1.2.1.1.2 Oral mucosal pain attributed to infection
 - 1.2.1.1.2.1 Oral mucosal pain attributed to bacterial infection
 - 1.2.1.1.2.2 Oral mucosal pain attributed to viral infection
 - 1.2.1.1.2.3 Oral mucosal pain attributed to fungal infection
- 1.2.1.1.3 Oral mucosal pain attributed to autoimmunity
- 1.2.1.1.4 Oral mucosal pain attributed to allergic reaction
- 1.2.1.1.5 Oral mucosal pain attributed to oral mucosal inflammation due to other cause
- 1.2.1.2 Oral mucosal pain attributed to malignant lesion
- 1.2.1.3 Oral mucosal pain attributed to neuropathy
- 1.2.1.4 Idiopathic oral mucosal pain

1.2.2 Salivary gland pain

- 1.2.2.1 Salivary gland pain attributed to obstructive cause
- 1.2.2.2 Salivary gland pain attributed to infection
 - 1.2.2.2.1 Salivary gland pain attributed to viral infection
 - 1.2.2.2.2 Salivary gland pain attributed to bacterial infection
- 1.2.2.3 Salivary gland pain attributed to non-infectious cause
 - 1.2.2.3.1 Salivary gland pain attributed to recurrent juvenile parotitis
 - 1.2.2.3.2 Salivary gland pain attributed to immunologically mediated disorder
 - 1.2.2.3.3 Salivary gland pain attributed to other cause

1.2.3 Jaw bone pain

- 1.2.3.1 Jaw bone pain attributed to infection
 - 1.2.3.1.1 Jaw bone pain attributed to bacterial infection
 - 1.2.3.1.2 Jaw bone pain attributed to viral infection
 - 1.2.3.1.3 Jaw bone pain attributed to fungal infection
- 1.2.3.2 Therapy related jaw bone pain

- 1.2.3.3 Jaw bone pain attributed to a local benign lesion
- 1.2.3.4 Jaw bone pain attributed to a malignant lesion
 - 1.2.3.4.1 Jaw bone pain attributed to a primary malignant lesion
 - 1.2.3.4.2 Jaw bone pain secondary to a malignant lesion
- 1.2.3.5 Jaw bone pain attributed to systemic disease
- 1.2.3.6 Jaw bone pain attributed to traumatic injury

2. Orofacial pain associated with regional muscles

2.1. Primary myofascial pain

2.1.1. Acute primary myofascial pain

2.1.2. Chronic primary myofascial pain

2.1.2.1. Chronic infrequent primary myofascial pain

2.1.2.2. Chronic frequent primary myofascial pain

2.1.2.2.1. Chronic frequent primary myofascial pain without pain referral

2.1.2.2.2. Chronic frequent primary myofascial pain with pain referral

2.1.2.3. Chronic persistent primary myofascial pain

2.1.2.3.1. Chronic persistent primary myofascial pain without pain referral

2.1.2.3.2. Chronic persistent primary myofascial pain with pain referral

2.2. Secondary myofascial pain

2.2.1. Secondary myofascial pain due to tendonitis

2.2.2. Secondary myofascial pain due to myositis

2.2.3. Secondary myofascial pain due to muscle spasm

3. Orofacial pain associated with disorders of the Temporomandibular Joint (TMJ)

3.1. Primary TMJ arthralgia

3.1.1. Acute primary TMJ arthralgia

3.1.2. Chronic primary TMJ arthralgia

3.1.2.1. Chronic infrequent primary TMJ arthralgia

3.1.2.2. Chronic frequent primary TMJ arthralgia

3.1.2.2.1. Chronic frequent primary TMJ arthralgia without referred pain

3.1.2.2.2. Chronic frequent primary TMJ arthralgia with referred pain

3.1.2.3. Chronic persistent primary TMJ arthralgia

3.1.2.3.1. Chronic persistent primary TMJ arthralgia without referred pain

3.1.2.3.2. Chronic persistent primary TMJ arthralgia with referred pain

3.2. Secondary TMJ arthralgia

3.2.1. TMJ arthralgia attributed to arthritis

3.2.1.1. TMJ arthralgia attributed to arthritis, non-systemic

3.2.1.2. TMJ arthralgia attributed to arthritis, systemic

3.2.2. TMJ arthralgia attributed to disc displacement with reduction

3.2.3. TMJ arthralgia attributed to disc displacement with reduction with intermittent locking

3.2.4. TMJ arthralgia attributed to disc displacement without reduction

3.2.5. TMJ arthralgia attributed to degenerative joint disease

3.2.6. TMJ arthralgia attributed to subluxation

4. Orofacial pain associated with lesion/disorders of the cranial nerves and other regional nerve structures

4.1. Pain caused by a lesion or disease of the trigeminal nerve

4.1.1. Trigeminal neuralgia

4.1.1.1. Classical trigeminal neuralgia

4.1.1.1.1. Classical trigeminal neuralgia, purely paroxysmal

4.1.1.1.2. Classical trigeminal neuralgia with concomitant continuous pain

4.1.1.2. Secondary trigeminal neuralgia

4.1.1.2.1. Trigeminal neuralgia attributed to multiple sclerosis

4.1.1.2.2. Trigeminal neuralgia attributed to space-occupying lesion

4.1.1.2.3. Trigeminal neuralgia attributed to other demonstrable causes

4.1.1.3. Idiopathic trigeminal neuralgia

4.1.1.3.1. Idiopathic trigeminal neuralgia, purely paroxysmal

4.1.1.3.2. Idiopathic trigeminal neuralgia with concomitant continuous
pain

*4.1.2. Trigeminal neuropathic pain other than 4.1.1. Clinically established trigeminal
neuralgia*

4.1.2.1. Trigeminal neuropathic pain attributed to herpes Zoster

4.1.2.2. Trigeminal postherpetic neuralgia

4.1.2.3. Post-traumatic trigeminal neuropathic pain

4.1.2.3.1. Probable post-traumatic trigeminal neuropathic pain

4.1.2.4. Trigeminal neuropathic pain attributed to another disorder

4.1.2.4.1. Probable trigeminal neuropathic pain attributed to another
disorder

4.1.2.5. Idiopathic trigeminal neuropathic pain

4.1.2.5.1. Probable idiopathic trigeminal neuropathic pain

4.2. Pain caused by a lesion or disease of the glossopharyngeal nerve

4.2.1. Clinically established glossopharyngeal neuralgia

4.2.1.1. Classical glossopharyngeal neuralgia

4.2.1.2. Secondary glossopharyngeal neuralgia

4.2.1.3. Idiopathic glossopharyngeal neuralgia

4.2.2. *Glossopharyngeal neuropathic pain*

4.2.2.1. Glossopharyngeal neuropathic pain attributed to a known cause

4.2.2.2. Idiopathic glossopharyngeal neuropathic pain

5. Orofacial pain resembling presentations of primary headaches

5.1. Orofacial migraine

5.1.1. *Orofacial migraine*

5.1.2. *Chronic orofacial migraine*

5.1.3 *Neurovascular Orofacial Pain*

5.1.3.1 Shortlasting Neurovascular Orofacial Pain

5.1.3.2 Longlasting Neurovascular Orofacial Pain

5.2. Tension-type orofacial pain

5.3. Trigeminal autonomic orofacial pain

5.3.1. *Orofacial cluster attacks*

5.3.1.1. Episodic orofacial cluster attacks

5.3.1.2. Chronic orofacial cluster attacks

5.3.2. *Paroxysmal hemifacial pain*

5.3.2.1. Episodic paroxysmal hemifacial pain

5.3.2.2. Chronic paroxysmal hemifacial pain

5.3.3. *Short-lasting unilateral neuralgiform facial pain attacks with autonomic signs (SUNFA)*

5.3.3.1. Episodic SUNFA

5.3.3.2. Chronic SUNFA

5.3.4. *Hemifacial continuous pain with autonomic signs*

5.3.5 *Constant Unilateral Facial Pain with Attacks (CUFPA)*

6. Idiopathic orofacial pain

6.1. Burning mouth syndrome (BMS)

6.1.1. *Burning mouth syndrome associated with somatosensory changes*

6.1.2. *Burning mouth syndrome not associated with somatosensory changes*

6.2. Persistent idiopathic facial pain (PIFP)

6.2.1. *Persistent idiopathic facial pain associated with somatosensory changes*

6.2.2. *Persistent idiopathic facial pain not associated with somatosensory changes*

6.3. Persistent idiopathic dentoalveolar pain

6.3.1. *Persistent idiopathic dentoalveolar pain associated with somatosensory changes*

6.3.2. *Persistent idiopathic dentoalveolar pain not associated with somatosensory changes*

7. Psychosocial Assessment

Classification with diagnostic criteria

1. Orofacial pain due to disorders of dentoalveolar and associated structures

General comments

Pain originating in dentoalveolar and associated structures is the most common reason for the complaint of pain in the orofacial region. The category includes pain caused by diseases, injuries and normal functioning of the tooth pulp, periodontium, gingivae, oral mucosa, salivary glands, and jaw bone tissue. In general, the pain is nociceptive and/or inflammatory in nature, and usually *acute*, meaning that it lasts less than 3 months. When the underlying disorder is adequately treated, the symptom of pain does not remain for a prolonged period of time, but this may not always be the case. The frequency of pain may be continuous, recurrent or occasional. In many cases, the natural history of the underlying disorders allows for fluctuation in all symptoms, including pain, which means this type of pain may be sometimes described as *episodic* (occurring less than 15 days per month, but being present more than 3 months). If the pain is present for more than 3 months, and at least 15 days per month, it is considered as *chronic*.

Since pain associated with dentoalveolar and associated structures is mainly a symptom of an acute disease or disorder, it might also appear relevant to categorize pain in relation to treatment. If the pain-inducing underlying disorder is not treated at all, the acute pain will usually remain and eventually become chronic. The same occurs if the underlying disorder is ineffectively/insufficiently treated, since the disorder (such as a local infection, neoplasm or systemic disorder) and the accompanying pain also might remain for longer than 3 months.

In general, the distinction between acute and chronic pain is important, since chronic pain often requires different management and has a less favorable prognosis. However, it is unknown whether the acute, episodic and chronic forms of dentoalveolar pain (and other types dealt with here) differ in any clinically meaningful aspect except duration. Based on the lack of data supporting that a distinction is relevant from the perspective of treatment or prognosis, the issue of acute/chronic is not reflected in this section of the ICOP. For research aiming to compare *e.g.* dentoalveolar pain of short versus long duration, it is recommended to use the distinction of *acute*, *episodic* or *chronic* pain described above, consistent with IASP/ICD11 and ICHD-3.

If evidence of important differences emerges in the future, the decision not to separate pain conditions based on time in this section must be re-evaluated.

Diagnostic criteria

1.1 Dental pain

Dental pain includes pain attributed to conditions affecting the tooth and its immediately surrounding and supporting structures, i.e. the tooth pulp, periodontium, and gingiva.

1.1.1 Pulpal pain

Description:

Dental pain caused by a disorder involving the tooth pulp

Diagnostic criteria:

- A. Any pain fulfilling criteria B through E
- B. Localized to the dental region of the pulpal disorder or lesion but may refer or radiate to other ipsilateral orofacial locations.
- C. Clinical, laboratory, imaging and/or anamnestic evidence of a lesion, disease, or trauma¹ known to produce pulpal pain
- D. Familiar pain is exacerbated by physical stimulus² applied to the affected tooth.
- E. Not better accounted for by another ICOP diagnosis.

Notes:

¹ as specified per sub-diagnosis

² mechanical, thermal, or chemical; as specified per sub-diagnosis

Comments:

Pulpal pain can be associated with all types of pulpal injury or disease. The pain is predominantly inflammatory and secondary to external or internal events.

1.1.1.1 Pulpal pain attributed to hypersensitivity

Description:

Pulpal pain occurring in association with a clinically normal pulp.

Diagnostic criteria:

- A. The pain fulfills criteria for *1.1.1 Pulpal pain*, and fulfills at least one of B-D and criterion E
- B. Is a sharp, deep sensation evoked by external stimuli (hot, cold and/or sweet)
- C. Subsides within a few seconds
- D. Is poorly localized;
 - a. often only to an approximate area within two or three teeth adjacent to the affected tooth,
 - b. occasionally the patient is unable to distinguish whether the pain originates from the mandible or the maxilla (Sharav et al, 1984; Falace et al 1996)
- E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

1.1.1.1.1 Pulpal pain attributed to a crack in the enamel

Diagnostic criteria:

- A. The pain fulfills criteria for *1.1.1.1 Pulpal pain attributed to hypersensitivity*
- B. The tooth has been diagnosed with a tooth crack/incomplete fracture involving the enamel based on a and at least one of b-d:
 - a. visual identification¹ of crack lines
 - b. sharp pain upon biting
 - c. pain on release of occlusal biting pressure or external application of force
 - d. cold hypersensitivity
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Notes:

¹ if needed, visual identification can be aided by magnification, light enhancement and visualization with dye

1.1.1.1.2 Pulpal pain attributed to exposed dentin

Diagnostic criteria:

- A. The pain fulfills criteria for *1.1.1.1 Pulpal pain attributed to hypersensitivity*
- B. A dentin surface is exposed
- C. The pain may be reproduced by scratching the exposed dentin with a dental explorer or by air blasting

D. Not better accounted for by another ICOP or ICHD-3 diagnosis

1.1.1.1.2.1 Pulpal pain attributed to tooth wear or abrasion

Diagnostic criteria:

- A. The pain fulfills criteria for *1.1.1.1.2 Pulpal pain attributed to exposed dentin*
- B. Clinical evidence of tooth wear or abrasion; smooth, flat surfaces that are not contoured with the natural shape of the anatomic crown of the tooth (von Toil et al, 2002)
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

1.1.1.1.2.2 Pulpal pain attributed to fracture resulting in exposed dentin

Diagnostic criteria:

- A. The pain fulfills criteria for *1.1.1.1.2 Pulpal pain attributed to exposed dentin*
- B. The tooth has been diagnosed with a fracture involving the enamel, root cementum, dentin or any combination thereof based on clinical and/or radiographic observations
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

1.1.1.1.2.3 Pulpal pain attributed to developmental dental hard tissue defect

Diagnostic criteria:

- A. The pain fulfills criteria for *1.1.1.1.2 Pulpal pain attributed to exposed dentin*
- B. The tooth has been diagnosed with a developmental defect involving the enamel, root cementum, and/or dentin

- a. local hypomineralization/hypomaturation of enamel
 - b. amelogenesis imperfecta
 - c. dentinogenesis imperfecta
 - d. other developmental defect
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

1.1.1.1.3 Pulpal pain attributed to hypersensitivity associated with dental procedures

Description:

The pain occurs subsequent to a dental procedure. It originates in dentin with a sharp, deep sensation evoked by external stimuli that subsides within a few seconds. Hot, cold, and sweet are among the external stimuli that may produce pain. Pain is poorly localized, often only to an approximate area within two or three teeth adjacent to the affected tooth. Occasionally, the patient is unable to distinguish whether the pain originates from the mandible or the maxilla. (Sharav et al, 1996; Falace et al, 1996)

Diagnostic criteria:

- A. The pain fulfills criteria for *1.1.1.1 Pulpal pain attributed to hypersensitivity*
- B. The tooth has recently¹ been subject to dental treatment
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ hours to days from dental procedure to pain onset

1.1.1.1.3.1 Pulpal pain attributed to recent extensive removal of dentin

Diagnostic criteria:

A. The pain fulfills criteria for *1.1.1.1.3 Pulpal pain attributed to hypersensitivity associated with dental procedures*

B. Pain onset upon recent¹ removal of dentin fulfilling either or both of a and b

a. deep (i.e. in close proximity to the pulp)

b. wide (i.e. opening up dentinal tubules in a large area)

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ hours to days

1.1.1.1.3.2 Pulpal pain attributed to recent placement of a restoration

Diagnostic criteria:

A. The pain fulfills criteria for *1.1.1.1.3 Pulpal pain attributed to hypersensitivity associated with dental procedures*

B. Pain onset upon recent¹ placement of a direct or indirect dental restoration

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ hours to days

1.1.1.1.3.3 Pulpal pain attributed to hypersensitivity due to hyperocclusion or -articulation following recent dental restorative procedures

Diagnostic criteria:

A. The pain fulfils criteria for *1.1.1.1.3 Pulpal pain attributed to hypersensitivity associated with dental procedures*

B. The tooth is in hyperocclusion and/or hyperarticulation as a result of recent¹ restorative procedures²

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ hours to days

² such as temporization, dental restoration or prosthodontic replacement

1.1.1.1.4 Pulpal pain attributed to central sensitization

Description:

Pulpal pain attributed to central sensitization can present in several teeth simultaneously, often starting in one tooth and then spreading to other teeth. This pain can be continuous or recurrent, and be present for long periods, often it is chronic. Successful treatment of other pain conditions

and associated psychological symptoms often leads to reduced tooth pain. Desensitizing dental treatment may in some cases lead to pain reduction or relief.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.1.1 Pulpal pain attributed to hypersensitivity*
- B. Signs of central sensitization¹ are present
- C. The patient is diagnosed with another orofacial, neck or widespread bodily pain condition, and the tooth pain
 - a. may present spontaneously
 - b. may co-present and co-fluctuate with other pains
- D. Local anesthesia and peripherally acting analgesics do not consistently provide pain relief (Glick et al, 1962)
- E. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ such as pain referral, temporal pain summation and/or allodynia

1.1.1.1.5 Pulpal pain attributed to other cause

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.1.1 Pulpal pain attributed to hypersensitivity*

B. The pain does not fulfil criteria for 1.1.1.1.1 – 1.1.1.1.4

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

1.1.1.2 Pulpal pain attributed to pulp exposure due to dental trauma

Description:

The pain is mild to moderate and exacerbation is typically evoked by air, liquids, or pressure on exposed pulp tissue secondary to dental trauma. When evoked, the pain typically subsides when the stimulus ceases. In the immediate post-trauma period, there is a lack of temperature sensitivity, spontaneous pain, or radiating pain, as these symptoms typically occur later and are associated with inflammation.

Diagnostic criteria:

A. The pain fulfills criteria for *1.1.1 Pulpal pain*

B. The tooth has been diagnosed with a recent¹ traumatic injury (a-c) exposing vital pulp tissue

a. fracture involving enamel, dentin and pulp (complicated crown fracture)

b. fracture involving root cementum, dentin and pulp (complicated root fracture)

c. fracture involving enamel, root cementum, dentin and pulp (complicated crown-root fracture)

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ minutes to hours

1.1.1.3 Pulpal pain attributed to pulpitis (pulpal inflammation)

Description:

Pain associated with pulpitis can vary from mild to severe and can be related to the severity of the inflammation (Hargreaves and Seltzer, 2002; Byers and Närhi, 2002). However, severe pulpal inflammation can also be asymptomatic (Hasler and Mitchell, 1970; Tyldesley and Mumford, 1970; Michaelson and Holland, 2012).

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.1 Pulpal pain*
- B. The tooth has been diagnosed with pulpal inflammation¹, i.e. pulpitis
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ due to trauma or infection; as specified by subcategories

1.1.1.3.1 Pulpal pain attributed to reversible pulpitis due to infection of dentin

Description:

Reversible pulpitis pain has been described as typically mild, not spontaneous, and provoked by changes in temperature. When evoked, pain is typically short-lasting and does not outlast the stimulus. (Garfunkel et al 1973). The suggested diagnostic criteria for reversible/irreversible

pulpitis presented below have not been scientifically validated, and the presence and characteristics of symptoms appear poorly related to the condition of the pulp (Mejàre et al 2012). When associated with caries, pulpitis is therefore considered potentially reversible as long as a zone of functionally intact dentin separates the bacterial front from the vital pulp tissue.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.1.3 Pulpal pain attributed to pulpitis*
- B. The dentin is infected¹
- C. The tooth has been diagnosed with reversible pulpitis based on the following:
 - a. clinical and/or radiographic evidence of a zone of intact dentin covering the pulp
 - b. pain may occur spontaneously but is not continuous
 - c. absence of prolonged pain² after stimulation³ of the pulp
 - d. absence of severe pain intensity
 - e. the pain responds to peripheral analgesics (NSAIDS)
- D. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ As evidenced by presence of caries or dentin exposed to the microbiota of the oral cavity for a period of time

² > a few seconds

³ thermal (cold, heat) or mechanical (probing, drilling)

1.1.1.3.1.1 Pulpal pain attributed to caries that does not extend to the pulp

Diagnostic criteria:

A. The pain fulfills criteria for *1.1.1.3.1 Pulpal pain attributed to reversible pulpitis due to infection of dentin*

B. The tooth has been diagnosed with caries that is unlikely to extend to the pulp based on clinical and/or radiographic observations

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

In addition to thermal sensitivity, the pain may be evoked by pressure on the carious dentin. (Sigurdsson, 2008)

1.1.1.3.1.2 Pulpal pain attributed to reversible pulpitis due to fracture of enamel, root cementum, dentin or any combination thereof, resulting in exposure of dentin

Diagnostic criteria:

A. The pain fulfills criteria for *1.1.1.3.1 Pulpal pain attributed to reversible pulpitis due to infection of dentin*

B. The tooth has been diagnosed with a traumatic injury exposing dentin

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

In addition to thermal sensitivity, the pain may be evoked by scratching the surface of the infected dentin. (Smulson and Sieraski, 1996; Abbott and Yu, 2007).

1.1.1.3.1.3 Pulpal pain attributed to reversible pulpitis due to a tooth crack without evidence of missing tooth substance

Diagnostic criteria:

A. The pain fulfills criteria for *1.1.1.3.1 Pulpal pain attributed to reversible pulpitis due to infection of dentin*

B. The tooth has been diagnosed with a tooth crack/incomplete fracture involving the enamel or enamel and dentin based on:

- a. visual identification¹ of crack lines
- b. sharp pain upon biting
- c. pain on release of occlusal biting pressure or external application of force
- d. cold hypersensitivity

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ visual identification can be aided by magnification, light enhancement and visualization with dye

Comments:

The duration of the pain should not outlast the application of the stimulus. Cracked teeth often have deep probing depths associated with the crack (Kang et al, 2016).

1.1.1.3.2 Pulpal pain attributed to irreversible pulpitis due to infection of dentin

Description:

The pain can be exacerbated by changes in temperature. Pain may also be associated with biting or percussion sensitivity. (Abbott and Yu, 2007). When evoked, the pain outlasts the duration of the stimulus. (Sigurdsson, 2008, Berman and Hartwell, 2006). The suggested diagnostic criteria for reversible/irreversible pulpitis presented below have not been scientifically validated and the presence and characteristics of symptoms appear poorly related to the condition of the pulp (Mejàre et al 2012). When associated with caries, pulpitis is therefore considered potentially irreversible when no zone of functionally intact dentin separates the bacterial front from the vital pulp tissue.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.1.3 Pulpal pain attributed to pulpitis*
- B. The dentin is infected¹
- C. The tooth has been diagnosed with irreversible pulpitis based on the following:
 - a. clinical and/or radiographic evidence that no zone of intact dentin covers the pulp
 - b. spontaneous pain that may be continuous
 - c. prolonged pain² after stimulation³ of the pulp

d. severe pain intensity

e. the pain responds poorly to NSAIDS

D. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ As evidenced by presence of caries or dentin exposed to the oral cavity microbiota for a period of time

² > a few seconds

³ thermal (cold, heat) or mechanical (probing, drilling)

1.1.1.3.2.1 Pulpal pain attributed to caries which may extend to the pulp

Diagnostic criteria:

A. The pain fulfills criteria for *1.1.1.3.2 Pulpal pain attributed to irreversible pulpitis due to infection of dentin*

B. The tooth has been diagnosed with deep caries that is likely to extend to the pulp based on clinical and/or radiographic observations

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

The presence of pain is poorly correlated to the status of the pulp. The value of symptoms to determine the condition of the pulp (reversibly/irreversibly inflamed) is debated and controversial, and scientific evidence is scarce (Mejàre et al. 2012). Severe continuous pain that

does not respond to analgesics (NSAID) may indicate irreversible inflammation and need for invasive treatment.

1.1.1.3.2.2 Pulpal pain attributed to irreversible pulpitis due to dental hard tissue fracture without pulp exposure

Diagnostic criteria:

A. The pain fulfills criteria for *1.1.1.3.2 Pulpal pain attributed to irreversible pulpitis due to infection of dentin*

B. Clinical and/or radiographic evidence of a fracture of dental hard tissue (a-c) without exposure of vital pulp tissue

a. fracture involving enamel and dentin (uncomplicated crown fracture)

b. fracture involving root cementum and dentin (uncomplicated root fracture)

c. fracture involving enamel, root cementum and dentin (uncomplicated crown-root fracture)

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments: In addition to thermal sensitivity, the pain may be evoked by scratching the surface of the infected dentin. (Smulson and Sieraski, 1996; Abbott and Yu, 2007)

1.1.1.3.2.3 Pulpal pain attributed to irreversible pulpitis due to a tooth crack without evidence of missing tooth substance

Diagnostic criteria:

A. The pain fulfills criteria for *1.1.1.3.2 Pulpal pain attributed to irreversible pulpitis due to infection of dentin*

B. Clinical evidence of a crack in the enamel or enamel and dentin based on the following:

- a. visual identification¹ of crack lines
- b. sharp pain upon biting
- c. pain on release of occlusal biting pressure or external application of force
- d. cold hypersensitivity

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ visual identification can be aided by magnification, light enhancement and visualization with dye

Comments:

Cracked teeth may result in sharp pain upon biting, unexplained cold sensitivity, pain on release of pressure, or deep probing depths associated with the crack (Kang et al, 2016). The duration of the pain typically outlasts the application of the stimulus.

1.1.1.3.3 Pulpal pain attributed to irreversible pulpitis due to infection of the dental pulp

Description:

The diagnostic criteria for reversible/irreversible pulpitis have not been scientifically validated, and the presence and characteristics of symptoms appear poorly related to the condition of the pulp (Mejàre et al 2012). When the pulp has been directly exposed the oral microbiota for a period of time, it lacks the ability to heal and pulpitis is considered to be irreversible.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.1.3 Pulpal pain attributed to pulpitis*
- B. The pulp is infected¹
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ has been exposed to the microbiota of the oral cavity for a period of time

1.1.1.3.3.1 Pulpal pain attributed to caries extending to the pulp

Diagnostic criteria:

- A. The pain fulfills criteria for *1.1.1.3.3 Pulpal pain attributed to irreversible pulpitis due to infection of the dental pulp*
- B. The tooth has been diagnosed with caries that is likely to extend to the pulp based on clinical and/or radiographic observations
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Histological studies indicate that when the carious lesion (bacterial front) reaches the pulp, inflammation is likely to be irreversible. The assessment is based on clinical and radiographic appearance. If a zone of intact, functional dentin is not seen between the carious dentin and the pulp, it can be concluded that the microflora is in direct contact with and has infected the pulp tissue, resulting in severe inflammation. It should be noted that in many cases, this condition may be symptom free (Michaelson and Holland, 2002).

1.1.1.3.3.2 Pulpal pain attributed to irreversible pulpitis due to dental hard tissue fracture with pulp exposure

Diagnostic criteria:

A. The pain fulfills criteria for *1.1.1.3.3 Pulpal pain attributed to irreversible pulpitis due to infection of the dental pulp.*

B. Clinical and/or radiographic evidence of a fracture of dental hard tissue and exposure of vital pulp tissue

- a. fracture involving enamel, dentin, and pulp (complicated crown fracture)
- b. fracture involving root cementum, dentin, and pulp (complicated root fracture)
- c. fracture involving enamel, root cementum, dentin, and pulp (complicated crown-root fracture)

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

In addition to thermal sensitivity, the pain may be evoked by mechanical stimulation of the exposed pulp or adjacent dentin.

1.1.1.3.4 Pulpal pain attributed to pulpitis due to external cervical root resorption

Description:

Cervical root resorption is a process where dentin is resorbed by osteoclastic activity. The condition is often asymptomatic until the resorptive process reaches the pulp. Secondary infection in the resorbed area stimulates an inflammatory response in the adjacent pulp.

In addition to thermal sensitivity, the pain may be evoked by pressure on the resorptive defect dentin. It should be noted that teeth with external cervical resorptions are usually asymptomatic (Patel et al, 2009).

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.1.3 Pulpal pain attributed to pulpitis*
- B. The tooth has been diagnosed with external cervical root resorption based on clinical and/or radiographic observations
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

1.1.1.3.5 Pulpal pain attributed to pulpitis due to other cause

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.1.3 Pulpal pain attributed to pulpitis*
- B. The pain does not fulfill criteria for 1.1.1.3.1–1.1.1.3.4
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Comments:

As an example, some reports in the literature indicate that pulpitis and pulpal pain may occur secondary to neurovascular events (neurogenic inflammation). Pain symptoms may range from dentinal hypersensitivity to lingering pain, indicative of pulpitis, and are often accompanied by autonomic signs. Pain symptoms may range from that of dentinal pain to pulpitis and often accompanied by autonomic signs (see *5.1 Orofacial migraine*). Pain responds to acute as well as chronic antimigraine medication (Obermann et al 2007, Benoliel et al 2010, Sharav et al 2015, 2017).

1.1.1.4 Pulpal pain attributed to systemic cause

Description:

Pulpal pain can be the result of a systemic disease causing a change in the pulp condition. For example, sickle cell anemia crises might result in dental pain (da Fonseca et al, 2007). Pulpal necrosis, presumed secondary to vaso-occlusive infarcts, has been reported in patients with sickle cell anemia (Costa et al. 2013). The phenomenon of “sickle cell toothache” may occur if sickle cells become trapped in the pulpal vascular supply and impede blood flow to the pulpal tissue. This leads to hypoxia, symptoms of pulpitis, cell death, and ultimately loss of tooth vitality.

Diagnostic criteria:

- A. The pain fulfills the criteria for *1.1.1 Pulpal pain*
- B. The patient has been diagnosed with a systemic disorder or disease known to be able to cause pulpal pain, e.g. sickle cell anemia.
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

1.1.2 Periodontal pain

Description:

Dental pain caused by a disorder involving the periodontium, meaning the periodontal ligament and the adjacent alveolar (periradicular) bone tissue.

Diagnostic criteria:

- A. Any pain fulfilling criteria B through E
- B. Localized to the site of the periodontal lesion, but may also refer or radiate to other ipsilateral orofacial locations
- C. Clinical, laboratory, imaging and/or anamnestic evidence of a lesion, disease or trauma¹ known to be able to cause periodontal pain
- D. Familiar pain is exacerbated by physical stimulus² applied to the affected tooth (horizontally or vertically) or to the tissue overlying the root
- E. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ as specified per sub-diagnosis

² mechanical, thermal, or chemical; as specified per sub-diagnosis

Comments:

Periodontal pain can be associated with all types of periodontal injury or disease. The pain is predominantly inflammatory and secondary to external or internal events.

1.1.2.1 Periodontal pain attributed to periodontitis (periodontal inflammation)

Description:

Periodontal inflammation (marginal as well as apical) is most frequently asymptomatic but can also present with pain and sometimes observable swelling. In such cases, pain is evoked by mechanical stimulation such as biting or chewing and is typically easy for the patient to localize. There may also be spontaneous pain, which is typically ongoing for hours. The intensity may be mild to severe. The pain can be reproduced by percussion or by applying pressure to the tooth.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.2 Periodontal pain*
- B. The tooth has been diagnosed with periodontal inflammation¹
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ due to trauma or infection; as specified by subcategories

Comments:

Periodontal pain attributed to periodontal inflammation is further subcategorized according to cause of inflammation.

In association with this type of pain, gingival pain may also occur.

1.1.2.1.1 Periodontal pain attributed to traumatically induced periodontal inflammation

Description:

Traumatic injury of periodontal tissues causes acute inflammation of the periodontium and can be painful to a varying degree. Causes for this type of pain include accidental dental injuries but also micro-trauma caused by for example changes in occlusion or articulation following dental treatment, and iatrogenic periodontal damage such as periodontal surgery. The condition is further subcategorized according to type of trauma or injury. The pain may be mild to severe and is exacerbated by mechanical provocation of the tooth. Spontaneous pain can occur.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.2.1 Periodontal pain attributed to periodontitis*
- B. The history reveals a recent¹ trauma or injury² involving the periodontal tissues
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ minutes to days

² accidental, self-inflicted or iatrogenic

Comments:

Accidental dental trauma/injury affects approximately 10–30% of the population, and almost exclusively occurs in incisors (maxilla 75–80% and mandible 20–25%). The incidence has been reported to 2–3 injured teeth/100 schoolchildren/year, and the prevalence of traumatized permanent teeth in children and adolescents is reported to 6–34% (Bastone et al. 2000).

Epidemiologic data suggest that while mild trauma is most prevalent, approximately 3% of permanent incisors in a population aged 6–50 years have been afflicted with a traumatic injury severe enough to be painful (Kaste et al. 1996).

1.1.2.1.1.1 Periodontal pain attributed to hyperocclusion or -articulation

Description:

Periodontal pain attributed to occlusal factors involves sensitization of periodontal nociceptors and an inflammatory response due to the excessive loading of the tooth. The history involves recent dental restoration, tooth extraction, or other change in occlusion or articulation. The patient may report that the tooth feels elevated. Clinically, a primary contact in occlusion or articulation is observed. The pain can be reproduced by percussion or by applying pressure to the tooth. The tooth may have increased mobility, and if so, radiographic examination may show widening of the periodontal space.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.2.1.1 Periodontal pain attributed to traumatically induced periodontal inflammation*
- B. The pain has developed in close temporal relation¹ to a change in occlusal conditions involving the painful tooth
- C. Mechanical provocation² reproduces the pain
- D. Hyperocclusion or hyperarticulation on the tooth is identified based on the following:
 - a. primary contact
 - b. hypermobility
- E. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ hours to days

² pressure, percussion

1.1.2.1.1.2 Postoperative periodontal pain

Description:

Postoperative periodontal pain is iatrogenic and caused by surgically induced tissue damage and subsequent inflammation. The pain is typically mild to moderate and may co-occur with clinically observable swelling and occasionally pus formation.

Diagnostic criteria:

A. The pain fulfils criteria for *1.1.2.1.1 Periodontal pain attributed to traumatically induced periodontal inflammation*

B. The pain has developed in close temporal relation¹ to a surgical intervention involving the periodontium

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ hours to days

Comments:

If physiologic (primary) healing occurs normally, the pain duration is typically short (1–2 weeks). Prolonged pain due to secondary healing and/or postoperative infection is occasionally observed but usually does not exceed 3 months.

1.1.2.1.1.3 Periodontal pain due to accidental dental trauma

Description:

Dental trauma frequently causes periodontal pain. The clinical and radiographic presentation, and the characteristics and severity of pain, depends on the nature and severity of the traumatic injury. Below follows a brief description of the trauma diagnoses in used in dental practice (ref The Dental Trauma Guide).

Periodontal pain due to concussion is caused by accidental injury to the periodontium and subsequent inflammation. The tooth displays normal mobility and is not displaced from its alveolar socket. Unless previously root-canal treated, the tooth typically shows evidence of a vital pulp. Imaging shows normal periradicular conditions.

Periodontal pain due to subluxation is caused by accidental injury to the periodontium and subsequent inflammation. The tooth displays increased mobility but is not displaced from its alveolar socket. Clinical findings include bleeding from the gingival sulcus. The tooth responds to pulp vitality testing in about 50% of cases. Radiographic examination may show a widening of the periodontal space.

Periodontal pain due to lateral luxation is caused by accidental injury to the periodontium and subsequent inflammation. The tooth is laterally displaced from its alveolar socket in combination with comminution or fracture of the buccal or lingual/palatal alveolar bone. The periodontal ligament is partially or totally separated and bleeding is seen from the sulcus. The tooth usually displays decreased mobility and may interfere with the occlusion and/or articulation. The tooth usually does not respond to pulp vitality testing. Radiographic examination shows variation in periodontal space width depending on the projection.

Periodontal pain due to intrusion is caused by accidental injury to the periodontium and subsequent inflammation. The tooth is axially displaced into the alveolar bone, and thus appears shorter than the adjacent teeth. The injury is accompanied by comminution or fracture of the alveolus. Other clinical findings may include decreased mobility and high percussion sound. The tooth usually does not respond to pulp vitality testing. Radiographic examination shows absence of (or decreased width of) the periodontal ligament space in all or part of the tooth.

Periodontal pain due to extrusion is caused by accidental injury to the periodontium and subsequent inflammation. The tooth is axially displaced and partially out of its socket, and thus appears elongated. The periodontal ligament is partially or totally separated and there is bleeding from the sulcus, but the alveolar socket bone is intact. The tooth has increased mobility and may interfere with occlusion/articulation. The tooth usually does not respond to pulp vitality testing. Radiographic examination shows increased width of the periodontal ligament space.

Periodontal pain due to avulsion is caused by accidental injury to the periodontium and subsequent inflammation. The tooth is completely displaced out of its socket, which is found empty or filled with a coagulum. The surrounding alveolar bone may be fractured.

Periodontal pain due to root fracture is caused by dislocation or fragments and/or subsequent infection causing periodontal inflammation. The history may or may not reveal an accidental traumatic event. The coronal fragment may be displaced and the tooth may appear longer than the adjacent teeth, may display increased mobility, and may interfere with occlusion/articulation. A local deep periodontal pocket may be present. Imaging shows a vertical or horizontal fracture confined to the root. If not previously root-filled, the tooth may or may not respond to pulp vitality testing.

Diagnostic criteria:

A. The pain fulfils criteria for *1.1.2.1.1 Periodontal pain attributed to traumatically induced periodontal inflammation*

B. The history reveals a recent¹ accidental² trauma affecting the tooth

C. The tooth has been diagnosed with a traumatic injury (a-g) based on clinical and/or radiographic observations

a. concussion

b. subluxation

c. lateral luxation

d. intrusion

e. extrusion

f. avulsion

g. root fracture³

D. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ minutes to days

² or caused by violence (in the case of root fracture, excessive loading of the tooth is also a possible cause)

³ horizontal or vertical

Comments:

Accidental dental trauma/injury affects approximately 10–30% of the population, and almost exclusively occurs in incisors (maxilla 75–80% and mandible 20–25%). The incidence has been

reported to 2–3 injured teeth/100 schoolchildren/year, and the prevalence of traumatized permanent teeth in children and adolescents is reported to 6–34% (Bastone et al. 2000). Epidemiologic data suggest that while mild trauma is most prevalent, approximately 3% of permanent incisors in a population aged 6–50 years have been afflicted with a traumatic injury severe enough to be painful (Kaste et al. 1996).

Concussion, subluxation and extrusion trauma may also include pulpal injury and the periodontal pain may co-occur with pulpal pain, see section 1.1.1 Pulpal pain.

Lateral luxation and intrusion trauma also induce pulpal and alveolar bone injuries and the periodontal pain may co-occur with pulpal pain and jaw bone pain, see sections 1.1.1 Pulpal pain and 1.2.3 Jaw bone pain.

Avulsion trauma may also include alveolar bone injury and the periodontal pain may co-occur with 1.2.3 Jaw bone pain

A root fracture is a hard tissue injury that may or may not reach the pulp space. If the pulp is involved, it is directly exposed to bacterial assault from the oral cavity and quickly becomes inflamed. If the pulp is vital, the pain may coincide with 1.1.3.3.2 Pulpal pain attributed to dental hard tissue fracture with direct pulp exposure. In addition to accidental trauma, other common reasons for root fracture include excessive loading of a root-canal treated tooth, typically with a post-and-core.

1.1.2.1.1.4 Periodontal pain attributed to other trauma or injury

Description:

Minor trauma or injury to the periodontium can also cause pain. By anamnestic, clinical or radiographic or other imaging findings, a trauma known to be able to cause periodontal inflammation can be identified, such as insufficient cooling during dental restorative procedures, interdental foreign body impaction (including food impaction), defective dental restoration, or apically extruded endodontic material. Clinical findings may include clinical signs of acute inflammation (swelling, pus, redness), increased tooth mobility, and local deep periodontal

pocket. Unless root-canal treated, the tooth typically shows evidence of a vital pulp. Imaging may display local marginal bone loss, which may or may not include the periapical region.

Diagnostic criteria:

A. The pain fulfills criteria for *1.1.2.1.1 Periodontal pain attributed to traumatically induced periodontal inflammation*

B. The painful tooth does not fulfill criteria for any of the dental trauma diagnoses included in 1.1.2.1.1.3

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

1.1.2.1.2 Periodontal pain attributed to apical periodontitis due to endodontic disease

Description:

Periodontal pain due to endodontic disease is pain associated with pulpal, periapical, juxtarradicular or periradicular inflammation. Endodontic disease, i.e. pulpal and periapical disease, is frequently associated with pain that may be mild to severe. A broken barrier against the oral cavity, most often caused by caries, and subsequent bacterial invasion of the pulp and root canal system is the main cause for inflammation of the pulp and periapical tissues.

Diagnostic criteria:

A. The pain fulfills criteria for *1.1.2.1 Periodontal pain attributed to periodontitis*

B. The tooth has been diagnosed with endodontic disease

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

This type of pain may also affect the gingivae.

Endodontic disease, including periapical, juxtarradicular or periradicular inflammation, may also be present without any clinical symptoms.

1.1.2.1.2.1 Periodontal pain attributed to pulpal inflammation

Description:

Periodontal pain secondary to pulpal inflammation is associated with symptomatic pulpitis. The periodontal inflammation is centered to the periapical region. The pulp is vital and thus the tooth typically responds to pulp vitality testing. The tooth is often tender to percussion. Clinical findings may include deep caries, deep/defective restoration, or external cervical root resorption. Imaging may or may not show evidence of diffuse local periapical bone resorption or sclerosis.

Diagnostic criteria:

A. The pain fulfils criteria for *1.1.2.1.2 Periodontal pain attributed to apical periodontitis due to endodontic disease*

B. The tooth has been diagnosed with pulpitis¹ based on the following:

- a. vital pulp evidenced by response to pulp vitality testing
- b. evidence of dental disorder known to be able to cause pulpal inflammation

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ reversible pulpitis or symptomatic irreversible pulpitis

Comments:

According to the literature, the association is weak between the actual state of the pulp and the periodontium (histology) and diagnostic findings including present and historical symptoms such as characteristics of tooth pain, clinical observations, and test results (Levin et al. 2009, Gutmann et al. 2009, Mejàre et al. 2012). Current diagnostics are largely based on expert opinion and few studies with quality deficits.

Teeth with periodontal pain secondary to pulpal inflammation frequently also fulfil the criteria for 1.1.1.3 Pulpal pain attributed to pulpitis.

1.1.2.1.2.2 Periodontal pain attributed to endodontic infection

Description:

Periodontal pain due to endodontic infection is associated with non-vital pulp (or previously root filled tooth) and infection of the pulp space. The pulp is totally or partially necrotic (unless the tooth is previously root canal treated), thus the tooth typically does not respond to pulp vitality testing. Although localized, the pain frequently refers to other orofacial sites on the same side, especially if the pain is severe. The pain can be reproduced by percussion or by applying pressure on the tooth and/or the adjacent periapical vestibular region. Imaging typically shows evidence of local periapical bone resorption.

Diagnostic criteria:

A. The pain fulfils criteria for *1.1.2.1.2 Periodontal pain attributed to apical periodontitis due to endodontic disease*

B. The tooth has been diagnosed with partial or total pulp necrosis and endodontic infection based on at least two of the following findings:

a. non-vital pulp evidenced by direct inspection or non-response to pulp vitality testing,
or

b. previously debrided¹ root canal, and

c. clinical² and/or radiographic³ evidence of apical inflammation⁴

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ previously treated or previously initiated therapy

² such as tenderness to percussion/pressure and/or apical palpation

³ apical or juxtaradicular radiolucency or sclerosis

⁴ symptomatic apical periodontitis or acute apical abscess

Comments:

The inflammatory response in the periapical tissues is caused by root canal infection with a mixed flora. An increased incidence of pain and swelling in apical periodontitis is associated with presence of specific anaerobes: Porphyromonas, Peptostreptococcus, and Prevotella species (Yoshida et al. 1987, Gomes et al. 1996). Upon local infection spread, a periapical abscess may form.

1.1.2.1.2.2.1 Periodontal pain attributed to intraradicular endodontic infection

Description:

In periodontal pain due to intra-radicular infection, the infectious agent causing the periodontal inflammation is contained within the root-canal system. Successful infection treatment usually results in pain resolution.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.2.1.2.2 Periodontal pain attributed to endodontic infection*
- B. The tooth has a root canal infection¹
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ bacterial, viral, fungal, or other

Comments:

In most teeth with infected necrotic pulp, the infection is confined to the root canal system.

1.1.2.1.2.2.2 Periodontal pain attributed to extraradicular endodontic infection

Description:

In periodontal pain due to extra-radicular infection, the infectious agent causing the periodontal inflammation resides on the external root surface, apically or in association with accessory canal orifices, or in the periapical tissues. The pain typically does not resolve after successful disinfection of the root canal system. Imaging occasionally reveals signs of external apical root resorption (Laux et al. 2000).

Diagnostic criteria:

A. The pain fulfils criteria for *1.1.2.1.2.2 Periodontal pain attributed to endodontic infection*

B. The tooth has an extraradicular infection¹

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ bacterial, viral, fungal, or other

Comments:

Extraradicular endodontic infection may occur with or without intraradicular infection (Ricucci et al. 2015). In such cases, the microbes colonize the external apical foramen and root surface, forming a biofilm (Ricucci et al. 2010). Anaerobic species such as Actinomyces and Propionibacterium also have the ability to form colonies in the periapical tissues at some distance from the root (Tronstad et al. 1987, Sjögren et al 1988), and this has been associated with remaining symptoms, including pain, after root canal treatment.

1.1.2.1.3 Periodontal pain attributed to periodontal disease

Description:

Periodontal pain due to plaque-induced periodontal disease can be acute or chronic in nature and depending on type the pain intensity ranges from mild to severe.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.2.1 Periodontal pain attributed to periodontitis*
- B. The tooth has been diagnosed with periodontal disease¹
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ as specified per sub-diagnosis

Comments:

The disease can be localized or generalized in the dentition. A number of intrinsic (diabetes, pregnancy, puberty, menopause) and extrinsic (smoking, medications, nutritional deficiencies e.g. avitaminosis-C) factors are considered as disease modifiers (Kinane and Chestnutt, 2000; Armitage 1999). In addition, medications known to be associated with gingival hyperplasia (e.g. phenytoin, ciclosporin, calcium channel blockers, bisphosphonates, and oral contraceptives) may promote periodontal breakdown due to difficulties to maintain proper oral hygiene. (Heasman & Hughes, 2014)

1.1.2.1.3.1 Periodontal pain attributed to chronic periodontitis

Description:

Periodontal pain due to chronic periodontitis may present in association with increased tooth mobility and oral hygiene routines and is typically mild. The pain typically appears only on provocation and does not linger. Most cases of chronic periodontitis are not painful, but may become painful on inflammatory exacerbation (see *1.1.2.1.3.5 Periodontal pain associated with periodontal abscess*).

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.2.1.3 Periodontal pain attributed to periodontal disease*
- B. The tooth has been diagnosed with chronic periodontitis
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

The disease form is characterized by slow progression of attachment loss, sometimes with periods of more rapid progression. The absence or low level of pain in chronic periodontitis has been attributed to the mainly chronic inflammatory cell infiltrates surrounding the infectious source, and functional drainage.

1.1.2.1.3.2 Periodontal pain attributed to aggressive periodontitis

Description:

Periodontal pain due to aggressive periodontitis may present in association with increased tooth mobility and oral hygiene routines and is typically mild to moderate. The pain typically appears only on provocation and does not linger.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.2.1.3 Periodontal pain attributed to periodontal disease*
- B. The tooth has been diagnosed with aggressive periodontitis
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

The disease form is characterized by rapid progression of attachment loss and sometimes early onset.

1.1.2.1.3.3 Periodontal pain attributed to periodontitis as a manifestation of systemic disorder

Description:

Periodontal pain associated with periodontitis due to a systemic disorder may present in association with increased tooth mobility and oral hygiene routines. The pain is typically mild to moderate, appears only on provocation and does not linger.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.2.1.3 Periodontal pain attributed to periodontal disease*
- B. The tooth has been diagnosed with a systemic disorder¹ known to be able to cause periodontitis
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ Hematological, genetic or other; specified per sub-diagnosis

Comments:

In addition to the more common plaque-induced periodontal disease, a number of systemic disorders manifests as periodontitis. The disorders listed here are considered as causative factors for periodontitis. They may also alter the course of plaque-induced periodontitis from chronic to aggressive. Reports in on to what degree periodontitis as a manifestation of a systemic disorder is associated with pain are essentially lacking the literature.

1.1.2.1.3.3.1 Periodontal pain attributed to a hematological disorder

Description:

Periodontal pain associated with periodontitis due to a hematological disorder may present in association with increased tooth mobility and oral hygiene routines and is typically mild to moderate. The pain typically appears only on provocation and does not linger.

Diagnostic criteria:

A. The pain fulfils criteria for *1.1.2.1.3.3 Periodontal pain attributed to periodontitis as a manifestation of systemic disorder*

B. The patient has been diagnosed with a hematological disorder known to be able to cause periodontitis (Armitage, 1999)

a. acquired neutropenia

b. leukemia

c. other hematological disorder

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

1.1.2.1.3.3.2 Periodontal pain attributed to a genetic disorder

Description:

Periodontal pain associated with periodontitis due to a genetic disorder may present in association with increased tooth mobility and oral hygiene routines and is typically mild to moderate. The pain typically appears only on provocation and does not linger.

Diagnostic criteria:

A. The pain fulfils criteria for *1.1.2.1.3.3 Periodontal pain attributed to periodontitis as a manifestation of systemic disorder*

B. The patient has been diagnosed with a genetic disorder known to be able to cause periodontitis (Armitage, 1999):

- a. familial and cyclic neutropenia
- b. Down syndrome
- c. leukocyte adhesion deficiency syndromes
- d. Papillon-Lefèvre syndrome
- e. Chediak-Higashi syndrome
- f. histiocytosis syndromes
- g. glycogen storage disease
- h. Infantile genetic agranulocytosis

- i. Cohen syndrome
 - j. Ehler-Danlos syndrome (types IV and VIII)
 - k. hypophosphatasia
 - l. other hematological disorder
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

1.1.2.1.3.3.3 Periodontal pain attributed to an unspecified systemic disorder

Description:

Periodontal pain associated with periodontitis due to an unspecified systemic disorder may present in association with increased tooth mobility and oral hygiene routines and is typically mild to moderate. The pain typically appears only on provocation and does not linger.

Diagnostic criteria:

A. The pain fulfils criteria for *1.1.2.1.3.3 Periodontal pain attributed to periodontitis as a manifestation of systemic*

B. The patient has been diagnosed with a systemic disorder (other than hematological or genetic) known to be able to cause periodontitis

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Systemic disorders associated with periodontitis are not currently well described in the literature.

1.1.2.1.3.4 Periodontal pain associated with necrotizing ulcerative periodontitis (NUP)

Description:

Periodontal pain due to necrotizing ulcerative periodontitis is typically severe. Pain is provoked by physical stimuli applied to the affected tooth or surrounding tissue. Pain also occurs spontaneously.

Clinically, necrotic soft tissue lesions and loss of attachment can be observed.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.2.1.3 Periodontal pain attributed to periodontal disease*
- B. The patient has been diagnosed with necrotizing ulcerative periodontitis
- C. The pain has developed in close temporal relation¹ to the onset of the ulcerations.
- D. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ hours to days

Comments:

Necrotizing ulcerative periodontitis is a rare oral infection, a more severe form of Necrotizing (ulcerative) gingivitis which besides soft tissue destruction also includes loss of clinical attachment and alveolar bone. The two conditions are often conflated to Necrotizing periodontal diseases (NPD) and are associated with diminished systemic resistance and immune dysfunction.

The predisposing factors include severe stress, sleep deprivation, alcohol, smoking, and HIV infection (Horning & Cohen, 1995).

1.1.2.1.3.5 Periodontal pain associated with periodontal abscess

Description:

A periodontal abscess is an exacerbation of chronic periodontitis or aggressive periodontitis, and pain due to this acute condition is usually severe. In addition to swelling, other clinical findings include plaque and/or calculus deposit on the root surface, usually with increased tooth mobility and a local deep periodontal pocket. Unless previously root-canal treated, the tooth typically shows evidence of a vital pulp. Although localized, the pain frequently refers to other orofacial sites on the same side, especially if the pain is severe. The pain can be reproduced by percussion or by applying pressure on the tooth and/or the adjacent periapical vestibular region. Imaging shows evidence of marginal and periradicular bone resorption, which may or may not include the periapical region.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.2.1.3 Periodontal pain attributed to periodontal disease*
- B. The patient has been diagnosed with a periodontal abscess based on
 - a. clinical signs of acute inflammation¹ and loss of attachment²
 - b. radiographic evidence of marginal and periradicular bone resorption
- C. The pain has developed in close temporal relation³ to the appearance of the abscess.
- D. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ Swelling, pus, redness, tenderness

² Increased mobility, local deep periodontal pocket

³ Usually hours to days before appearance of the abscess

1.1.2.1.4 Periodontal pain attributed to apical and marginal periodontitis due to combined endodontic infection and periodontal disease

Description:

Periodontal pain due to combined endodontic and periodontal lesion may be symptom free. If present, the pain is typically moderate to severe, and other clinical findings may include clinical signs of acute inflammation (swelling, pus, redness), plaque and/or calculus deposit on the root surface, increased tooth mobility, and deep periodontal pocket(s). If not previously root-canal treated, the tooth shows no or inconclusive evidence of pulp vitality. Although localized, the pain frequently refers to other orofacial sites on the same side, especially if the pain is severe. The pain can be reproduced by percussion or by applying pressure on the tooth and/or the adjacent periapical vestibular region. Imaging shows evidence of marginal and periradicular bone resorption that includes the periapical region.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.2.1.3 Periodontal pain attributed to periodontal disease*
- B. The tooth has been diagnosed with partial or total pulp necrosis, or is previously root canal treated
- C. The tooth has been diagnosed with periodontal disease based on clinical and radiographic observations

D. Not better accounted for by another ICOP or ICHD-3 diagnosis

1.1.2.1.5 Periodontal pain attributed to peri-implantitis due to peri-implant infection

Description:

Periodontal pain due to inflammation surrounding a dental implant is most frequently painless, but if pain occurs it is typically moderate to severe. Other clinical findings may include clinical signs of acute inflammation (swelling, pus, redness), plaque and/or calculus deposit on the implant surface, implant mobility, and local deep pocket. Imaging shows poor bony integration of the implant and evidence of horizontal marginal bone loss or localized peri-implant bone resorption.

Diagnostic criteria:

A. The pain fulfils criteria for *1.1.2.1 Periodontal pain attributed to periodontitis* with the exception that it involves an implant and not a natural tooth

B. Clinical¹ and/or radiographic² evidence of a peri-implant infection

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ signs of acute inflammation (swelling, pus, redness) and/or attachment loss (increased mobility, deep pocket)

² radiolucency partially or totally surrounding the implant

Comments:

Patients diagnosed with periodontal pain attributed to peri-implantitis due to peri-implant infection are also likely to be affected by gingival pain.

1.1.2.2 Periodontal pain attributed to a local non-inflammatory cause

Description:

Periodontal pain due to a local non-inflammatory cause is usually mild to moderate. Periodontal cysts, radicular cysts, and tumors are frequently asymptomatic, but following expansion symptoms such as pain, localized swelling and displacement of one or more teeth may occur. In such cases, pain is occasionally evoked by external mechanical stimulation such as biting or chewing and is typically easy for the patient to localize. There may also be spontaneous pain, which is seldom severe.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.2 Periodontal pain*
- B. The patient has been diagnosed with a non-infectious disorder¹ known to be able to cause periodontal pain based on clinical, imaging, and/or histological examination
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ periodontal cyst, tumor

1.1.3 Gingival pain

Description:

Pain caused by a disorder involving the gingival tissues.

Diagnostic criteria:

- A. Any pain fulfilling criteria B through E
- B. Pain is localized to the site of the gingivae, but may refer to other ipsilateral orofacial locations
- C. Clinical, laboratory, imaging and/or anamnestic evidence of a lesion or disease of the gingival tissues, known to be able to cause pain
- D. Evidence of causation demonstrated by the following:
 - 1. Pain has developed in temporal relation to the onset or appearance of the lesion
 - 2. Familiar pain is exacerbated by manipulation of the affected gingival tissue
- E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

1.1.3.1 Gingival pain attributed to gingivitis (gingival inflammation)

Description:

Pain associated with gingivitis (i.e. inflammation of the gingivae). Inflammation may be caused by infection due to specific or non-specific microbial organisms, trauma (may be physical, thermal, radiation or chemical), autoimmunity, or allergic reaction.

Diagnostic criteria:

- A. The pain fulfills criteria for *1.1.3 Gingival pain*

B. The patient has been diagnosed with gingival inflammation¹ based on the clinical observation of inflammation signs in the gingivae (i.e. tumor, dolor, rubor, and calor)

C. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Notes:

¹ due to trauma, infection or systemic disorder; as specified by subcategories

1.1.3.1.1 Gingival pain associated with trauma

Description:

Traumatic injury of gingival tissues causes acute inflammation and can be painful to a varying degree. Traumatic ulceration of the gingiva may be acute or chronic in nature with the latter diagnostically more challenging due to underlying fibrosis and clinical appearance of neoplastic induration. A thorough clinical history will often alert the clinician to a traumatic aetiology or burns caused by warm food or chemicals. The pain may be mild to severe and is exacerbated by mechanical provocation of the gingivae. Spontaneous pain can occur.

Diagnostic criteria:

A. The pain fulfils criteria for *1.1.3.1 Gingival pain attributed to gingivitis*

B. The history reveals a recent¹ trauma² or injury³ involving the gingival tissues

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ minutes to days

² mechanical, thermal or chemical

³ accidental, self-inflicted or iatrogenic

Comments:

Causes for this type of pain include accidental dental injuries but also micro-trauma caused by for example during eating or drinking overly hot foods or drinks, following dental treatment, trauma due to tooth brushing or flossing or other interdental instruments. Examination may reveal the causative factor, such as a sharp broken tooth or restoration or an ill-fitting denture. Ulceration due to local anaesthetic injection most often occurs in the hard palate, the combined result of pressure and ischemic necrosis. Poorly fitting dentures may cause painful ulcerations. Over-erupted dentition or parafunctional habits may also cause local occlusal gingival trauma with resultant inflammation and pain. Iatrogenic gingival damage occurs during most dental surgery for example dental extraction, gingival or periodontal surgery, or dental restorative therapy. Chemical burns may be related to misuse of anti-inflammatory tablets or occur due to dental treatment. Self-harm may be a rare cause of gingival trauma.

Dental trauma may also cause gingival inflammatory pain, see also *1.1.2.1.1 Periodontal pain attributed to traumatically induced periodontal inflammation.*

1.1.3.1.2 Gingival pain attributed to infection

Description:

Infection of the gingival tissues causes acute inflammation and can be painful to a varying degree. The pain may be mild to severe and is exacerbated by mechanical provocation of the gingivae. Spontaneous pain can occur. The condition is further subcategorized according to category of causative microorganism.

Diagnostic criteria:

A. The pain fulfils criteria for *1.1.3.1 Gingival pain attributed to gingivitis*

B. The patient has been diagnosed with an infection¹ of the gingival tissues based on anamnestic information, clinical observations, and/or microbiological analysis

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ bacterial, viral, or fungal

Comment:

Acquired or congenital immunosuppression may lead to increased risk of gingival infection. Patients on immunosuppressive therapy may develop a variety of opportunistic infections including pseudomembranous candidosis, other fungal and viral infections. TNF- α therapy increases the risk of tuberculosis (TB). Patients on infliximab and adalimumab with combined immunomodulatory therapy may be at increased risk of TB, histoplasmosis, coccidiomy infections. Antirheumatic drugs including methotrexate, abatacept and alefacept have increased the risk of herpes simplex and herpes zoster infections and TB.

1.1.3.1.2.1 Gingival pain attributed to bacterial infection

Diagnostic criteria:

A. The pain fulfills criteria for *1.1.3.1.2 Gingival pain attributed to infection*

B. The patient has been diagnosed with a bacterial infection of the gingival tissues

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Bacterial infections are the most common oral infections and gingival pain may be associated with underlying dental pathology, such as periodontal infection or endodontic infections that may present as swelling, inflammation and pain of the overlying gingivae.

Acute necrotizing ulcerative gingivitis (ANUG) (or necrotising ulcerative gingivitis [NUG], necrotizing ulcerative periodontitis [NUP], or necrotizing ulcerative stomatitis [NUS]) is an opportunistic gingival infection caused by an array of bacteria in malnourished children, young adults and immune deficient patients. NUG is often the initial presentation, proceeding into NUP, NUS and ultimately noma. Necrosis and ulceration of the interdental gingival papilla, excruciating pain, severe halitosis, regional lymphadenopathy, malaise and fever differentiates this form of ulceration from others (Feller et al 2014).

Pericoronitis (inflammation around a tooth crown) causing pain is most often associated with partially erupted third molars. Other dentition, both permanent and deciduous, may have mild pericoronitis during eruption. If the tooth is impacted and unable to fully erupt, continued or recurrent infection may ensue. Pain results from the individual's immune inflammatory response to anaerobic bacteria colonized in biofilm that cannot be shed from third molars partially covered by soft tissue (Kay 1966).

Gingival pain can occur in association with conditions that mainly affects other oral tissues, and is not categorized in this section. For these types of pain, refer to the corresponding sections:

- Gingival pain associated with alveolar osteitis (dry socket), see *1.2.3.2 Therapy related jaw bone pain (c)*
- Gingival pain associated with periodontitis, see *1.1.2.1 Periodontal pain attributed to periodontitis (periodontal inflammation)*
- Gingival pain associated with apical periodontitis, see *1.1.2.1.2 Periodontal pain attributed to apical periodontitis due to endodontic disease*

- Palatal gingival pain associated with acute necrotizing sialadenitis, see *1.2.2.2.2 Salivary gland pain attributed to bacterial infection*

1.1.3.1.2.2 Gingival pain attributed to viral infection

Diagnostic criteria:

- A. The pain fulfills criteria for *1.1.3.1.2 Gingival pain attributed to infection*
- B. The patient has been diagnosed with a viral infection of the gingival tissues
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Viral infections of the gingival tissues include HSV, VZV, HPV, CMV, Coxsackievirus and HIV infection.

The infected gingival tissues may often be ulcerated and painful to touch. Severe local pain is often associated with eating or drinking acidic or hot or cold foods or drinks, which may cause the individual to be unable to eat or drink and become dehydrated.

Herpes simplex (HSV) is the most common virus to affect the oral mucosa. Herpetic gingivostomatitis, the primary HSV-1 infection, mostly affects children and presents either as asymptomatic infection or with mucosal vesicles followed by painful ulceration affecting both keratinised and non-keratinised mucosa and gingivae. Adults with primary infection suffer symptomatic herpetic pharyngotonsillitis initiated as vesicles that rapidly break down into painful shallow ulcerations (Fourie & Boy, 2016)

1.1.3.1.2.3 Gingival pain attributed to fungal infection

Diagnostic criteria:

- A. The pain fulfills criteria for *1.1.3.1.2 Gingival pain attributed to infection*
- B. The patient has been diagnosed with a fungal infection of the gingival tissues
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Gingival pain associated with fungal infection is probably rare, and reports in the literature is essentially lacking. The painful manifestations of oral fungal infection usually affect oral mucosa.

1.1.3.1.3 Gingival pain attributed to autoimmunity

Description:

Inflammation of the gingival tissues causes acute inflammation and can be painful to a varying degree. The condition is further subcategorized according to category of autoimmune disease. The pain may be mild to severe and is exacerbated by mechanical provocation of the gingivae. Spontaneous pain can occur.

Diagnostic criteria:

- A. The pain fulfills criteria for *1.1.3.1 Gingival pain attributed to gingivitis*
- B. The patient has been diagnosed with an autoimmune disease or disorder¹ known to be able to cause gingival pain
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ such as mucous membrane pemphigoid, Sjögren's syndrome, pemphigus, or other

Comments:

Several dermatological immune-mediated vesiculo-ulcerative lesions conditions may present with oral mucosal involvement, either concurrent with the skin pathology, as the initial presentation or sometimes as the only clinical presentation (Bascones-Martinez et al. 2015).

Mucous membrane pemphigoid (MMP) is a common systemic autoimmune blistering disease with preferential involvement of mucosal membranes. The antibodies are directed at the proteins of keratinocyte to connective tissue matrix adhesion or hemi-desmosomes (BP180 and laminin-332) causing the epithelium to split away from its underlying connective tissue bed. The subepithelial nature of the split results in thick roofed vesicles which may still be intact on examination. Rupture of the vesicles leave ulcerative lesions devoid of any epithelium, covered by yellow-white slough. Desquamative gingivitis (erythematous and friable gingiva with epithelial destruction) is a frequent finding (Hasan 2014).

Sjögren's syndrome is a systemic autoimmune disease that frequently presents concomitantly with other systemic connective tissue or organ-specific autoimmune diseases. The association is well described for systemic lupus erythematosus and rheumatoid arthritis. The gingival tissues can become abraded and even cut with dry foods, and sore. The presence of Sjögren's syndrome influences the expression of the other autoimmune disease to some degree, for instance by increasing fatigue and lymphoma risk (Theander & Jacobsson 2008)

Pemphigus, a group of immune mediated subepithelial bullous dermatoses, is mediated by auto-antibodies directed at the proteins of keratinocyte adhesion (desmosomes) causing acantholysis. Pemphigus vulgaris most commonly affects the oral cavity, with autoantibodies mainly directed against desmoglein 1 and 3 (mucocutaneous forms) or only 3 (mucosal forms). Gingival pain due to pemphigus is infrequent, since the disease mostly affects oral mucosa (Harman et al. 2003).

1.1.3.1.4 Gingival pain attributed to allergic reaction

Description:

Inflammation of the gingival tissues causes acute inflammation and can be painful to a varying degree. The condition is further subcategorized according to category of hypersensitive or

allergic condition. The pain may be mild to severe and is exacerbated by mechanical provocation of the gingivae. Spontaneous pain can occur.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.3.1 Gingival pain attributed to gingivitis*
- B. The patient has been diagnosed with hypersensitivity or an allergic reaction in the gingival tissues associated with
 - a. dental material (such as temporary/permanent restorative or impression material)
 - b. oral hygiene product
 - c. topical drug
 - d. systemic drug
 - e. food or additive
 - f. other factor
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Allergic reactions and oral mucosal hypersensitivity reactions are less common than cutaneous ones ascribed to the possible allergen dilution and the continuous rinsing effects of normal saliva flow (Venables et al. 2016). Lesions may present with non-specific tissue oedema, erythema, cracking, ulceration, hyperkeratotic white plaques or mucosal desquamation. Lesions may start long after the introduction of a drug and may remain for months after cessation thereof complicating diagnosis and management.

A hypersensitivity reaction to either a systemic drug or direct contact with an offending agent may result in clinical and histological features reminiscent of lichen planus. The term ‘oral lichenoid drug reaction’ (OLDR) or ‘oral lichenoid contact lesion’ (OLCL) is used respectively, and both may present with significant ulceration, usually with erythema and white striations at

the periphery of the ulceration. A temporal or spatial association with an offending agent can usually be identified. Amalgam is often implicated in OLCL, confirmed by patch testing for mercury or amalgam sensitivity. OLDR is encountered with some frequency in patients treated with angiotensin-converting enzyme (ACE) inhibitors, nonsteroidal anti-inflammatory drugs (NSAIDs) and oral hypoglycaemic drugs (Al-Hashimi et al. 2007).

Potential drug reactions causing oral mucogingival reactions have been well summarised (Yuan & Woo 2015). Fixed drug eruption (FDE) is a form of hypersensitivity remarkable for its fixed anatomical nature and has been described with NSAID's and other oxicam drugs (Andrade et al. 2011), gabapentin (Gupta et al. 2009), fluconazole (Benedix et al. 2008), and systemic antibacterial and antifungal drugs (Savin 2001). FDE should be suspected in cases with a temporal association of drug ingestion, may be confirmed through patch testing or oral provocation tests, and managed through drug avoidance or substitution, while the acute lesions can be treated with topical or systemic steroids.

Allergic contact stomatitis. Although rare, this form of mucositis has been reported in association with dental impression materials (Batchelor & Todd 2010), dental restorative materials (Venables et al. 2016), topical benzocaine application, and more commonly cinnamon in toothpastes, mouth rinses, and chewing gum (Kind et al. 2010). Lesions may appear as mixed red and white patches with ulceration, swelling of the cheeks and desquamation appearing on the lips, cheeks, tongue and gingiva as localised or widely distributed lesions (Calapai et al. 2014).

Drug induced fibrosis epithelial hyperplasia or fibrovascular hyperplasia may occasionally be associated with painful presentation, likely due to underlying periodontal infection due to difficulty with oral hygiene in these conditions.

1.1.3.1.5 Gingival pain attributed to gingival inflammation due to other cause

Description:

Inflammation of the gingival tissues may occur associated with systemic disease, disorder, or condition, or with the treatment of such diseases or disorders, and can be painful to a varying

degree. The pain may be mild to severe, and is exacerbated by mechanical provocation of the gingivae. Spontaneous pain can occur.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.1.3.1 Gingival pain attributed to gingivitis*
- B. The patient has been diagnosed with a systemic disease or condition, or received therapy known to be able to cause oral mucosal pain
 - a. endocrine disorders or alterations
 - b. dietary deficiency
 - c. haematological diseases
 - d. gastrointestinal diseases
 - e. dermatological diseases
 - f. drug induced disorders (not attributable to hypersensitivity or allergy)
 - g. other disease, disorder or treatment
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Alteration of physiological state such as pregnancy and menopause may cause endocrine changes that manifest as gingival discomfort and pain. Systemic disorders that can cause gingivitis include endocrine disease (hypothyroidism, diabetes mellitus); dietary deficiencies (Fe, vitamin B complex, zinc); anaemia; gastrointestinal disorders (gastro-esophageal reflux disease; GERD), and drug induced and genetic disorders.

Epulis is a hyperplastic, non-neoplastic lesion which originates mainly from gingival tissues. Several histologic types occur, of which the prevalent type during pregnancy is the granulomatous type, a form of pyogenic granuloma (Shailesh et al. 2010). The growth is composed mainly of capillary vessels and endothelial proliferation and appears usually on the frontal part of the maxilla during the third trimester, sometimes referred to as "pregnancy tumor". The lesion usually causes no symptoms apart from its very presence, but may become painful because of interference with *e.g.* occlusion or denture wear. Etiologic factors are improper maintenance of oral hygiene which lead to chronic gingivitis and high gingival levels of active progesterone which acts in a yet undefined mechanism.

Antineoplastic therapy-induced mucositis associated with chemotherapy and radiation mainly affects oral mucosa, but can also affect the gingivae and cause gingival pain. See *1.2.1.1.5 Oral mucosal pain attributed to systemic disorder*.

Benign hyperplastic lesions or tumors involving gingivae are usually not directly associated with pain, but may become painful if traumatized and/or infected due to interference with *e.g.* occlusion or dentures. See *1.1.3.1.1 Gingival pain attributed to trauma* and *1.1.3.1.2 Gingival pain attributed to infection*.

1.1.3.2 Gingival pain attributed to malignant lesion

Description:

Gingival pain related to a malignant disease can be painful to a varying degree. The pain may be mild to severe and is exacerbated by mechanical provocation of the gingivae. Spontaneous pain can occur.

Diagnostic criteria:

A. The pain fulfils criteria for *1.1.3 Gingival pain*

B. The patient has been diagnosed with a malignant lesion¹ known to be able to cause gingival pain

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ neoplasia of the gingival tissues

Comments:

The gingiva may be affected by an array of both primary and metastatic malignancies which may all present as non-specific ulcers. Oral squamous cell carcinoma (OSCC) is the most common, frequently presenting as ulceration with clinical induration, fixation to the underlying tissues, rolled exophytic margins, and pain and/or numbness (Warnakulasuriya et al 2007).

1.1.3.3 Gingival pain attributed to neuropathy; see 4.1 Pain attributed to a lesion or disease of the trigeminal nerve

Comments:

Trigger points of trigeminal neuralgia may be located in the gingiva, and light touch will elicit the typical intense paroxysmal pain attacks affecting the whole dermatome corresponding to the affected nerve branch. As a result, patients may find it impossible to wear a denture in the region. For description and diagnostic criteria, see *4.1.1 Clinically established trigeminal neuralgia*.

Gingival pain may also occur as part of the early clinical presentation of trigeminal neuralgia, the diffuse deep pain, “pre-trigeminal neuralgia pain”, that sometimes precedes the onset of characteristic paroxysmal pain.

Peripheral neuropathy may be associated with gingival pain. For description and diagnostic criteria, see *4.1.2 Trigeminal neuropathic pain other than 4.1.1 Clinically established trigeminal neuralgia*.

1.1.3.4 Idiopathic gingival pain; see 6 Idiopathic orofacial pain

Comments:

Burning mouth syndrome (BMS) may also affect the gingivae presenting as localized or more widely distributed gingival pain. For description and diagnostic criteria, refer to *6.1 Burning mouth syndrome (BMS)*.

Persistent idiopathic dentoalveolar pain (PIDAP) is frequently associated with localized pain in the gingivae. For description and diagnostic criteria, refer to *6.3 Persistent idiopathic dentoalveolar pain*.

Consideration must be given to patients presenting with chronic widespread pain or other multiple pain conditions which may be attributable to central sensitization or other mechanisms.

1.2 Non-dental pain

Description:

Non-dental pain includes pain attributed to conditions affecting the non-dental oral tissues, i.e. the oral mucosa, the salivary glands, and the jaw bone tissue. Pain arising from lymph tissue, muscles, joints, skin and sinuses have not been included, and are in part covered elsewhere.

1.2.1 Oral mucosal pain

Description:

Pain involving the oral mucosa which may be attributed to a local or distant cause. Oral mucosal pain is often characterized by a burning, stinging or sore sensation. Various mucosal lesions like ulcers, erosions and vesicles are common causes of oral mucosal pain, and these lesions can occur due to a large variety of local mucosal and systemic diseases. The terms stomatitis and oral mucositis are often used as synonyms, but they do not reflect identical processes. Stomatitis refers to any inflammatory condition of oral mucosa which occurs due to local infections or injuries or underlying systemic diseases. Mucositis occurs due to radiation or chemotherapeutic agents (Scully et al. 2000).

A large variety of local mucosal and systemic diseases are associated with pain due to formation of ulcers or erosions. These lesions differ with regard to extension in the oral mucosa:

- A mucosal ulcer is defined as a loss of surface tissue with disintegration and necrosis of epithelial tissue. It involves damage to both epithelium and lamina propria. It penetrates the epithelial-connective tissue border, and has its base at a deep level in the submucosa, and in some cases even within the muscle or periosteum
- A mucosal erosion is defined as a superficial break on the mucous membrane with loss of the superficial epithelial cells and minor damage to the underlying lamina propria.

Diagnostic criteria:

- A. Any pain fulfilling criteria B through E
- B. Pain is localized to the site of the oral mucosa, but may refer to other ipsilateral orofacial locations
- C. Clinical, laboratory, imaging and/or anamnestic evidence of a lesion or disease of the oral mucosal tissues, known to be able to cause pain
- D. Evidence of causation demonstrated by the following:
 1. Pain has developed in temporal relation to the onset or appearance of the lesion
 2. Familiar pain is exacerbated by manipulation of the affected oral mucosa

E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

1.2.1.1 Oral mucosal pain attributed to oral mucosal inflammation

Diagnostic criteria:

- A. The pain fulfills criteria for *1.2.1 Oral mucosal pain*
- B. The patient has been diagnosed with inflammation of the oral mucosa based on the clinical observation of inflammation signs in the oral mucosa (*i.e.* tumor, dolor, rubor, and calor)
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Comment:

Mucosal pain associated with ulcers or other lesions is often associated with high levels of pain-related unpleasantness. The burning pain is often severe, and oral function (eating, talking), quality of life and sleep are frequently impaired (Abdalla-Aslan et al. 2016).

1.2.1.1.1 Oral mucosal pain associated with trauma

Diagnostic criteria:

- A. The pain fulfills criteria for *1.2.1.1 Oral mucosal pain attributed to oral mucosal inflammation*
- B. The history reveals a recent¹ trauma or injury² involving the oral mucosal tissues
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ minutes to days

²accidental, self-inflicted or iatrogenic

1.2.1.1.1.1 Oral mucosal pain attributed to mechanical, thermal, or chemical damage

Description:

Traumatic injury of the oral mucosa causes acute inflammation and can be painful to a varying degree. Traumatic ulceration of the oral mucosa may be acute or chronic in nature with the latter diagnostically more challenging due to underlying fibrosis and clinical appearance of neoplastic induration. A thorough clinical history will often alert the clinician to a traumatic aetiology or burns caused by warm food or chemicals. The pain may be mild to severe and is exacerbated by mechanical provocation of the oral mucosa. Spontaneous pain can occur.

Diagnostic criteria:

A. The pain fulfils criteria for *1.2.1.1 Oral mucosal pain attributed to oral mucosal inflammation*

B. The history reveals a recent¹ trauma or injury², involving the oral mucosal tissues

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ minutes to days

² accidental, self-inflicted or iatrogenic

Comments:

Causes for this type of pain include accidental dental injuries but also micro-trauma caused by for example during eating or drinking overly hot foods or drinks, following dental treatment, trauma due to tooth brushing or flossing or other interdental instruments. Examination may reveal the causative factor such as an underlying mandibular or maxillary or dentoalveolar fracture, tooth root fracture or solely a soft tissue injury. Ulceration due to local anaesthetic most often occurs in the hard and soft palate, the combined result of pressure and ischemic necrosis. Poorly fitting dentures may cause painful ulcerations. Over-erupted dentition or parafunctional habits may also cause local oral mucosal trauma with resultant inflammation and pain. Chemical burns may be related to misuse of anti-inflammatory tablets or eating or drinking overly hot drinks or food. Self-harm may be a rare cause of oral mucosal trauma. In patients with dystonia or oral neuropathy injury may be recurrent.

1.2.1.1.1.2 Oral mucosal pain attributed to surgical or other iatrogenic injury

Description:

Iatrogenic oral mucosa injury occurs during most dental surgery for example dental extractions, gingival or periodontal surgery.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.2.1.1 Oral mucosa pain attributed to oral mucosal inflammation*
- B. The history reveals a recent¹ surgery involving the oral mucosal tissues
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ minutes to days

1.2.1.1.1.3 Oral mucosal pain attributed to radiation or chemotherapy

Description:

Oral mucositis refers to erythematous and ulcerative lesions of the oral mucosa that may occur in patients who receive anticancer radiotherapy to head and neck cancer involving the oral cavity or chemotherapy. The lesions typically manifest as very painful erythema or ulcerations that compromise nutrition and oral hygiene as well as increased risk for local and systemic infection. The condition may also be accompanied by taste disturbances and xerostomia.

Diagnostic criteria:

A. The pain fulfils criteria for *1.2.1.1 Oral mucosal pain attributed to oral mucosal inflammation*

B. The history reveals recent¹ radiation involving the oral mucosal tissues, or recent chemotherapy

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ days to months

Comments:

Oral mucositis describes inflammation of oral mucosa resulting from chemotherapeutic agents or ionizing radiation. The frequency and severity can vary significantly with the type and dose of therapy. The pathogenesis of oral mucositis is multifactorial; a complex five-stage model is proposed in the development of mucositis (Treister & Sonis 2007, Mravak-Stipetić 2010)

Mucositis may be exacerbated by local factors and infections. Infections associated with the oral mucositis lesions can cause life-threatening systemic sepsis during periods of profound immunosuppression. When uncomplicated by infection mucositis heals within 2 to 4 weeks after cessation of cytotoxic chemotherapy. While oral complications primarily are associated with discomfort and interference with oral function, and quality of life (Duncan et al. 2005) in patients who are also immunocompromised or debilitated, these complications can become life threatening. Thus, management of mucositis pain is a primary component of any mucositis management strategy (Sonis et al. 2004, Vera-Llonch et al. 2007).

1.2.1.1.2 Oral mucosal pain attributed to infection

Description:

Infection of the oral mucosal tissues causes acute inflammation and can be painful to a varying degree. The pain may be mild to severe and is exacerbated by mechanical provocation of the oral mucosa. Spontaneous pain can occur. The condition is further subcategorized according to category of causative microorganism.

Diagnostic criteria:

A. The pain fulfils criteria for *1.2.1.1 Oral mucosal pain attributed to oral mucosal inflammation*

B. The patient has been diagnosed with an infection¹ of the oral mucosa based on anamnestic information, clinical observations, and/or microbiological analysis

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ bacterial, viral, or fungal

1.2.1.1.2.1 *Oral mucosal pain attributed to bacterial infection*

Description

Bacterial infection of the oral mucosal tissues causes acute inflammation and can be painful to a varying degree. The pain may be mild to severe and is exacerbated by mechanical provocation of the oral mucosa. Spontaneous pain can occur.

Diagnostic criteria:

- A. The pain fulfills criteria for *1.2.1.1.2 Oral mucosal pain attributed to bacterial infection*
- B. The patient has been diagnosed with a bacterial infection of the oral mucosa
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Bacterial infections are the most common oral infections and oral mucosa pain is often associated with underlying dental pathology with periodontal infection or dental periapical infections may present as swelling, inflammation and pain of the overlying oral mucosae.

Acute necrotizing ulcerative gingivitis (ANUG) or Necrotising ulcerative gingivitis (NUG) / periodontitis (NUP)/ stomatitis (NUS) is an opportunistic Oral mucosa infection caused by an array of bacteria in malnourished children, young adults and immune deficient patients. NUG is often the initial presentation, proceeding into NUP, NUS and ultimately noma. Necrosis and ulceration of the oral mucosa, exquisite pain, severe halitosis, regional lymphadenopathy, malaise and fever differentiate this form of ulceration from others. When the alveolar bone becomes exposed, necrotic bone sequestrae may develop and should be removed with the associated teeth (Feller et al. 2014).

Syphilis is caused by *Treponema pallidum* infection and continues to be widespread, with increasing rates among men who have sex with men. The primary lesion presents at the first site

of mucosal inoculation, frequently the oral mucosa. A highly infective, painless, solitary ulcer with indurated margins and ipsilateral lymphadenopathy is the most common, with healing within three weeks. Non-characteristic mucous patches alerts to the development of secondary syphilis frequently accompanied by a maculo-papular rash of the palmo-plantar surfaces of the hands and feet, and generalised lymphadenopathy (Hertel et al. 2014).

Gonorrhoea lesions may occur in mouth at site of inoculation or secondarily by hematogenous spread from a primary focus elsewhere. Earliest symptoms are burning or itching sensation, dryness or heat in mouth followed by acute pain on eating or speaking. Tonsils and oropharynx are most frequently involved, and oral tissues may be diffusely inflamed or ulcerated. Saliva develops increased viscosity and fetid odor. In severe cases, submaxillary lymphadenopathy with fever occurs.

Tuberculosis (TB). The emergence of multidrug-resistant *Mycobacterium tuberculosis* and the high numbers of HIV-infected individuals in South Africa has resulted in an increase of TB cases urging inclusion in the differential diagnoses of orofacial pathology. Secondary TB in the form of painful, deep irregular ulcers with indurated appearance, undermined edges and thick mucus-like material at the base of any aspect of the tongue are typical. Haematogenous spread from pulmonary TB or secondary inoculation of a traumatic ulcer with infected sputum is the most common pathogenesis. Primary oral TB is distinctly rare, usually associated with *Mycobacterium bovis*. Ulcers resemble chronic traumatic ulceration and even malignancy urging a diagnostic biopsy (von Arx & Husain, 2001; Jain & Jain 2014). Associated symptoms of pain, fever, lymphadenopathy, hoarseness of voice and weight loss frequently accompany the ulcerations.

Acquired or congenital immunosuppression may lead to increased risk of gingival infection. Patients on immunosuppressive therapy may develop a variety of opportunistic infections including pseudomembranous candidosis, other fungal and viral infections. TNF- α therapy increases the risk of tuberculosis (TB). Patients on infliximab and adalimumab with combined immunomodulatory therapy may be at increased risk of TB, histoplasmosis, coccidiomy infections. Antirheumatic drugs including methotrexate, abatacept and alefacept have increased the risk of herpes simplex and herpes zoster infections and TB.

1.2.1.1.1.2 Oral mucosal pain attributed to viral infection

Description:

Viral infection of the oral mucosal tissues causes acute inflammation and can be painful to a varying degree. The pain may be mild to severe and is exacerbated by mechanical provocation of the oral mucosa. Spontaneous pain can occur.

Diagnostic criteria:

- A. The pain fulfills criteria for *1.2.1.1.1 Oral mucosal pain attributed to infection*
- B. The patient has been diagnosed with a viral infection of the oral mucosa as evidenced by
 - 1. A mucosal eruption in the area of pain
 - 2. PCR identification of the virus from swabs taken from the area
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Viral infections of the oral mucosa include HSV, VZV, HPV, CMV, Coxsackie virus and HIV infection. Note that ICHD-3 has a specific set of criteria for Herpes Zoster Virus (Chapter 13, 13.1.2.1 Painful trigeminal neuropathy attributed to acute Herpes zoster). We have modelled criteria to reflect these.

The infected oral mucosa tissues may often be ulcerated and painful to palpation. Severe local pain is often noted, in eating or drinking acidic or hot or cold foods or drinks. Pain is elicited on eating and may be so severe that the individual may be unable to eat or drink and become dehydrated.

Herpes simplex (HSV) is the most common virus to affect the oral mucosa. Herpetic gingivostomatitis, the primary HSV-1 infection, mostly affects children and presents either as

asymptomatic infection or with mucosal vesicles followed by quickly developing painful ulcerations affecting both keratinised and non-keratinised mucosa and gingivae. Fever, malaise, foul odor and cervical lymphadenopathy often accompanies the pain. Adults with primary infection suffer symptomatic herpetic pharyngotonsillitis initiated as vesicles that rapidly break down into painful shallow ulcerations (Fourie & Boy, 2016).

Recurrent manifestations of the virus in the form of herpes labialis are most commonly initiated by various factors including, but not limited to, stress, UV exposure or dental local anaesthetic. Initial prodromal stinging or burning is followed by a cluster of approximately five small fluid-filled vesicles on erythematous mucosa that ruptures to leave painful shallow ulcers which coalesce and crusts.

Herpangina (Hand-Foot-Mouth Disease) caused by *Coxsackie* virus, ECHO virus, and other enteroviruses. It typically affects children below 10 years. Red macules or vesicles are followed by self-limiting ulcerations, approximately 5mm in diameter, on the anterior tonsillar pillars, soft palate, uvula, and/or tonsils. Pyrexia, sore throat and headaches are common. Ulcers heal within 4-6 days.

Varicella zoster virus (VZV or HHV-3) infection is well-known for its pruritic, vesicular skin rash, ulceration and crusting, all occurring concurrently. Crusting is absent in the oral mucosa which instead present as ulcerating papules.

Herpes zoster (shingles) signifies reactivation of dormant VZV infection, mostly affecting old and debilitated patients, and follows the dermatome of the ganglion in which the virus established latency. Severe burning or stinging pain to the affected dermatome is followed by fluid filled vesicles that rupture to leave painful shallow ulcerations that may coalesce to form large denuded areas. Oral manifestations signify involvement of the mandibular or maxillary divisions of the trigeminal nerve with pathognomonic abrupt termination of lesions along the midline. Osteonecrosis with tooth exfoliation has been reported, especially in immune deficient individuals. The infection often involves several locations in the anatomical distribution of the affected nerve branch. See also 4.1.2.1 *Trigeminal neuropathic pain attributed to herpes zoster* and 4.1.2.2 *Trigeminal post-herpetic neuralgia*.

Human papilloma virus may cause single or multiple papillary lesions. These lesions are rarely painful unless traumatized.

Epstein-Barr virus causes mononucleosis, which may involve sore throat and numerous small ulcers that precede lymphadenopathy. Gingival bleeding, petechiae at the border between soft and hard palate are other clinical features.

1.2.1.1.1.3 Oral mucosal pain attributed to fungal infection

Description:

Fungal infection of the oral mucosal tissues causes acute inflammation and can be painful to a varying degree. The pain may be mild to severe and is exacerbated by mechanical provocation of the oral mucosa. Spontaneous pain can occur.

Diagnostic criteria:

- A. The pain fulfills criteria for *1.2.1.1.1 Oral mucosal pain attributed to infection*
- B. The patient has been diagnosed with a fungal infection of the oral mucosa
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

In recent times, the prevalence of oral fungal infections other than candidiasis has been on the rise. Immunodeficiency diseases such as HIV infection and AIDS, immunosuppressive therapy, or prolonged usage of broad-spectrum antibiotics and corticosteroids are some of the notable reasons for disease emergence which occurs when the oral homeostasis is disturbed. Diabetes and salivary gland hyperfunction are other predisposing factors. The most common oral fungal infection is *Candida albicans*. Erythematous candidiasis presents with generalised erythema and pain. Median rhomboid glossitis affects the tongue and has three main types: pseudomembranous type presenting with white patches that are easily wiped off leaving erythematous, bleeding, sore surface; erythematous type with red macular lesions, often with a burning sensation; and angular

cheilitis type which is characterized by sore cracks and redness at angle of mouth. Xerostomia, burning, stinging and itching sensations, and metal taste are accompanying symptoms.

Other mycoses to be considered in the context of gingival pain include mucormycosis, aspergillosis, histoplasmosis, blastomycosis, and paracoccidioidomycosis. *Aspergillus* and *Mucorales* infections, albeit uncommon, are the most commonly encountered and follows the inhalation of the spores from soil, manure, grain, cereal and mouldy flour (Perusquia-Ortiz et al. 2012) Both are superficial and invasive opportunistic fungal infections, encountered in the oral cavity of especially immunocompromised patients.

1.2.1.1.3 Oral mucosal pain attributed to autoimmunity

Description:

Inflammation of the oral mucosa or stomatitis tissues causes pain to a varying degree. The condition is further subcategorized according to category of autoimmune disease. The pain may be mild to severe and is exacerbated by mechanical provocation of the oral mucosa. Both elicited and spontaneous pain may occur.

Diagnostic criteria:

A. The pain fulfils criteria for *1.2.1.1 Oral mucosal pain attributed to oral mucosal inflammation*

B. The patient has been diagnosed with an autoimmune disease known to be able to cause oral mucosal pain

a. pemphigus

b. mucus membrane pemphigoid

c. recurrent aphthous stomatitis

- d. oral lichen planus
 - e. erythema multiforme
 - f. Sjögren's syndrome
 - g. Behçets disease
 - h. graft versus host disease
 - i. lupus erythematosus, systemic or discoid type
 - j. erythema migrans
 - k. Crohn's disease
 - l. ulcerative colitis
 - m. coeliac disease
 - n. other autoimmune disease or disorder
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

The prognosis for the pain depends on the outcome of treatment of the underlying autoimmune disorder.

Several dermatological immune-mediated vesiculo-ulcerative lesions conditions may present with oral mucosal involvement, either concurrent with the skin pathology, as the initial presentation or sometimes as the only clinical presentation (Bascones-Martinez et al. 2015). The prognosis for the pain depends on the outcome of treatment of the underlying autoimmune disorder.

Pemphigus, a group of immune mediated subepithelial bullous dermatoses, is mediated by auto-antibodies directed at the proteins of keratinocyte adhesion (desmosomes) causing acantholysis. PV most commonly affects the oral cavity, it's autoantibodies mainly directed against desmoglein 1 and 3 (mucocutaneous forms) or only 3 (mucosal forms). Patients, typically 40-60 years of age, present with thin-roofed, flaccid intra-epithelial bullae which rupture promptly after development resulting in large irregular areas of painful mucosal ulceration (Harman et al. 2003)

Mucous membrane pemphigoid (MMP) is a common systemic autoimmune blistering disease with preferential involvement of mucosal membranes. The antibodies are directed at the proteins of keratinocyte to connective tissue matrix adhesion or hemi-desmosomes (BP180 and laminin-332) causing the epithelium to split away from its underlying connective tissue bed. The subepithelial nature of the split results in thick roofed vesicles which may still be intact on examination. Rupture of the vesicles leave ulcerative lesions devoid of any epithelium, covered by yellow-white slough (Hasan 2014).

Recurrent aphthous stomatitis (RAS) represents the most common form of oral mucosal ulceration encountered in healthy individuals (Scully 2006; Akintoye & Greenberg 2014). The term should be reserved for recurrent ulcers of the oral mucosa, not associated with any systemic disease and which typically commence in childhood or adolescence. Non-keratinised mucosa of the buccal mucosa, lips and soft palate is most commonly affected. A variety of local and systemic factors including immunologic, allergic, nutritional, microbial organisms, psychosocial stress as well as immunosuppressive drugs, have been proposed as possible etiological factors. Increased prevalence in close family members also indicate a possible genetic background (Slebioda et al. 2013). RAS has an atypical clinical presentation in HIV-infected patients and should always be considered as differential diagnosis of oral mucosal ulceration in them (Shiboski et al. 2009). When RAS starts later in life, additional mucosal surfaces may be affected and a comprehensive physical examination and medical history should be considered to rule out inflammatory gastrointestinal disease such as Crohn's disease, coeliac disease, Behçet's syndrome, Sweet's syndrome, cyclic neutropenia, HIV infection and drug reactions in which case "aphthous-like ulcers" is a more appropriate term. Clinically RAS is subclassified into *RAS minor*; the most common variant which typically presents with 1-5 ulcers, less than 10mm in diameter surrounded by a bright red inflammatory halo, and healing spontaneously within 10-14

days; and *RAS major* (Sutton disease) which present as deeper, larger, persistent ulcerations with irregular borders. The ulcers are larger, usually >10mm in diameter, and typically take weeks or months to heal.

Oral lichen planus (OLP) is a rather common, chronic inflammatory disorder affecting mainly middle-aged females. The pathogenesis remains uncertain but various subsets of T-lymphocytes and mast cells play a role in the basal membrane damage (Firth et al. 2015). The disease may present with a diverse clinical spectrum which includes the atrophic, erosive, ulcerative and less commonly, bullous variants (Gorouhi et al 2014). The lesions typically affect the oral mucosa bilaterally and are fairly symmetric, presenting as either solely an oral mucosal disease or be accompanied by desquamative gingivitis and/or cutaneous manifestations. In the case of the erosive and ulcerative types, painful pseudomembrane covered ulcerations bordered by faint white striae are seen in a multifocal distribution. Recent meta-analyses determined the overall malignant transformation rate of OLP to be around 1%, most commonly affecting the tongue of older females (Fitzpatrick et al. 2014, van der Waal 2014), but the issue remains contentious.

Erythema multiforme (EM) is a T-cell-mediated type IV cytotoxic immune reaction to a variety of antigens (viral, bacterial, pharmacological, or chemical) that result in apoptosis-mediated epithelial cell death. Anti-desmoplakin I and II antibodies were recently demonstrated as a possible instigator of the cytotoxic reaction (Fukiwake et al 2007). EM mostly affects young, otherwise healthy individuals and is often recurrent and temporal with recurrent HSV infections (Carrozzo et al. 1999). Oral lesions may either represent the start of further mucocutaneous involvement or may appear in isolation, classically with swollen, cracked, haemorrhagic and crusted lips with or without mucosal blisters and ulcerations (Farthing et al 2005).

Sjögren's syndrome is a systemic autoimmune disease that frequently presents concomitantly with other systemic connective tissue or organ-specific autoimmune diseases. This association is well described for systemic lupus erythematosus and rheumatoid arthritis. The oral mucosal tissues can become abraded and even cut with dry foods, and sore. The presence of Sjögren's syndrome influences the expression of the other autoimmune disease to some degree, for instance by increasing fatigue and lymphoma risk (Theander & Jacobsson 2008).

Behçet's Disease is an autoimmune multisystem disease of unknown aetiology. It is characterised by oral ulcers, genital ulcers and eye inflammation. There may be dermatologic symptoms along with neurological and vascular involvement. The oral lesions ulcers are painful and characterized by cyclic presentation affecting the lips, buccal mucosa, soft palate and tongue with an appearance resembling aphthous lesions, a few millimeters to centimeters in diameter. The incidence of the disease is higher in Mediterranean and Asian populations, especially in Turkey (Saccucci et al. 2018).

Graft versus host disease is characterized by lichenoid, papular and erythematous lesions, and occasionally ulcerations and desquamation on the buccal and labial mucosa, the palate and dorsal part of the tongue. The oral lesions are often accompanied by fever, malaise, nausea, and xerostomia. The oral findings may be caused by a combination of radiotherapy, chemotherapy, immunosuppressive medications, and secondary infections.

More than half of patients with systemic lupus erythematosus (SLE) may present with oral lesions, most frequently ulceration and pain of the buccal mucosa and lips during the early, active disease phase (Khatibi et al. 2012). Ulcerative lesions and erythematous lesions with or without radiating white striae may also be seen as part of the clinical spectrum of discoid lupus erythematosus (DLE). DLE is considered a potentially malignant disorder of the oral mucosa due to the increased prevalence of oral squamous cell carcinoma among this population, especially involving the lower lip.

Erythema migrans (geographic tongue, benign migratory glossitis) is a common oral inflammatory condition of unknown etiology with an estimated prevalence of 1–3%. About 30 % have oral discomfort, burning and stinging sensation. It usually affects the tongue, although other oral sites may be involved. Presentation may include circular erythematous areas, often sharply defined by elevated, whitish border zones, located at the lateral, dorsal, anterior, and/or ventral parts of the tongue. The erythematous appearance is due to atrophy and loss of filiform papillae lesions. The most commonly suggested associations are atopy and psoriasis. The disorder should not be confused with the characteristic rash of early Lyme disease.

Crohn's disease presents with multifocal, linear, nodular, or diffuse mucosal thickenings in the labial and buccal mucosa and the mucobuccal folds. They may be associated with painful, persistent aphthous-like ulcerations and atrophic glossitis.

Ulcerative colitis presents with scattered, clumped or linearly oriented pustules on an erythematous mucosa at multiple oral sites. Some patients exhibit painful oral aphthous-like lesions in addition to the pustular lesions.

Coeliac disease may present with mucosal pain, commonly associated with aphthous-like ulcers. Malabsorption of iron and vitamin B may lead to burning, stinging sensations in the tongue.

Other rare autoimmune or idiopathic causes of oral mucosal ulceration causing pain and sensitivity include; eosinophilic ulcer, giant cell arteritis hypereosinophilic syndrome, necrotising sialometaplasia, polyarteritis nodosa, reactive arthritis (Reiter's syndrome), acute febrile neutrophilic dermatosis (Sweet syndrome), and Wegener's granulomatosis.

1.2.1.1.4 Oral mucosal pain attributed to allergic reaction

Description:

Inflammation of the oral mucosa or stomatitis tissues related to allergy or hypersensitivity causes pain to a varying degree. The condition is further subcategorized according to category of hypersensitive or allergic condition. The pain may be mild to severe and is exacerbated by mechanical provocation of the oral mucosae. Both elicited and spontaneous pain may occur.

Oral allergy syndrome (OAS) usually occurs in individuals who are allergic to pollen from trees, grasses or weeds. Fresh fruit, raw vegetables and raw nuts are common causes of OAS. The symptoms including itching sensation and/or swelling of all or part of the lips, tongue, mouth or throat, but this can on occasions be severe and also include nausea and vomiting. Dental materials, oral hygiene products and food additives may cause contact allergic reactions in the mouth with varied clinical presentation including stomatitis, lichenoid lesions, erosions, blisters and ulcerations. Elicited and spontaneous pain may occur.

Diagnostic criteria:

- A. The pain fulfils criteria for 1.2.1.1 *Oral mucosal pain attributed to oral mucosal inflammation*
- B. The patient has been diagnosed with hypersensitivity or an allergic reaction in the oral mucosa associated with
 - a. dental material (temporary/permanent restorative or impression material)
 - b. oral hygiene product
 - c. topical drug
 - d. systemic drug
 - e. food or additive
 - f. other factor
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Allergic reactions and oral mucosal hypersensitivity reactions are less common than cutaneous ones ascribed to the possible allergen dilution and the continuous rinsing effects of normal saliva flow (Venables et al. 2016). Lesions may present with non-specific tissue oedema, erythema, cracking, ulceration, hyperkeratotic white plaques or mucosal desquamation. Lesions may start long after the introduction of a drug and may remain for months after cessation thereof complicating diagnosis and management.

Allergic contact stomatitis. Although rare, this form of mucositis has been reported in association with dental impression materials (Batchelor & Todd 2010), dental restorative materials

(Venables et al. 2016), topical benzocaine application, and more commonly cinnamon in toothpastes, mouth rinses, and chewing gum (Kind et al. 2010). Lesions may appear as mixed red and white patches with ulceration, swelling of the cheeks and desquamation appearing on the lips, cheeks, tongue and gingiva as localized or widely distributed lesions (Calapai et al. 2014).

A hypersensitivity reaction to either a systemic drug or direct contact with an offending agent may result in clinical and histological features reminiscent of lichen planus. The term ‘oral lichenoid drug reaction’ (OLDR) or ‘oral lichenoid contact lesion’ (OLCL) is used respectively, and both may present with significant ulceration, usually with erythema and white striations at the periphery of the ulceration. A temporal or spatial association with an offending agent can usually be identified. Amalgam is often implicated in OLCL, confirmed by patch testing for mercury or amalgam sensitivity. OLDR is encountered with some frequency in patients treated with angiotensin-converting enzyme (ACE) inhibitors, nonsteroidal anti-inflammatory drugs (NSAIDs) and oral hypoglycaemic drugs (Al-Hashimi et al. 2007).

Fixed drug eruption (FDE) is a form of hypersensitivity remarkable for its fixed anatomical nature and has been described with NSAID’s and other oximicam drugs (Andrade et al. 2011), gabapentin (Gupta et al. 2009), fluconazole (Benedix et al. 2008), and systemic antibacterial and antifungal drugs (Savin 2001). FDE should be suspected in cases with a temporal association of drug ingestion, may be confirmed through patch testing or oral provocation tests, and managed through drug avoidance or substitution, while the acute lesions can be treated with topical or systemic steroids.

Drug induced fibrosis epithelial hyperplasia or fibrovascular hyperplasia may occasionally be associated with painful presentation, likely due to underlying periodontal infection due to difficulty with oral hygiene in these conditions.

1.2.1.1.5 Oral mucosal pain attributed to oral mucosal inflammation due to other cause

Description:

Inflammation of the oral mucosa may occur associated with systemic disease, disorder, or condition, or with the treatment of such diseases or disorders, and can be painful to a varying degree. The pain may be mild to severe, and is exacerbated by mechanical provocation of the oral mucosa. Spontaneous pain can occur.

Diagnostic criteria:

A. The pain fulfils criteria for *1.2.1.1 Oral mucosal pain attributed to oral mucosal inflammation*

B. The patient has been diagnosed with a systemic disease or condition, or received therapy known to be able to cause oral mucosal pain

a. endocrine disorders or alterations

b. dietary deficiency

c. haematological diseases

d. gastrointestinal diseases

e. dermatological diseases

f. drug induced disorders (not attributable to hypersensitivity or allergy)

g. other disease, disorder or treatment

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Alteration of physiological state such as pregnancy and menopause may cause endocrine changes that manifest as oral mucosal discomfort and pain. Systemic disorders that can cause oral

mucosal inflammation and pain include endocrine disease (hypothyroidism, diabetes mellitus); dietary deficiencies (Fe, vitamin B complex, zinc); gastrointestinal disorders, and drug induced disorders (not attributable to hypersensitivity or allergy).

Iron, vitamin B12 and folate deficiency can cause atrophic glossitis in which the filiform papilla of the dorsum of the tongue undergo atrophy, leaving a smooth, erythematous tongue. Other parts of the oral mucosa may also appear atrophic and red. Aphthous-like ulcers are common in severe cases. Burning, stinging sensation may precede clinically detectable oral lesions. Severe cases of vitamin B12 may also be associated with paresthesia. Patients may have a predisposition to develop angular cheilitis.

Haematological disorders such as anaemia, gammopathies, haematinic deficiencies, leukemia, myelodysplastic syndrome, neutropenia and other white cell dyscrasias may result in friable oral mucosa with resultant ulceration and pain.

Gastrointestinal disorders such as gastroesophageal reflux disorder (GERD) and peptic ulceration may lead to malabsorption and related dietary deficiencies and subsequent related oral mucosal pain.

Dermatological causes of painful mucosal lesions include dermatitis herpetiformis, linear IgA disease, epidermolysis bullosa and chronic ulcerative stomatitis.

Antineoplastic therapy-induced mucositis involves a complex cascade of events which is initiated by reactive oxygen species with extensive inflammation, atrophy, swelling, erythema and ulceration (Raber-Durlacher et al. 2010). This includes chemotherapy induced mucositis as well as radiation lesions (Rosenthal & Trotti 2009). Radiation lesions correspond to the exposed surfaces while chemotherapy induced mucositis affects the entire alimentary tract. The type and dosage of systemic cytotoxic agents, and the dosage and field of radiation will affect the presence and severity of mucositis. Evidence based guidelines for the management of cancer therapy induced oral mucositis was established and should be referred to in all cases of patients receiving these agents.

Benign hyperplastic lesions or tumors involving oral mucosa are usually not directly associated with pain, but may become painful if traumatized and/or infected due to interference with e.g.

occlusion or dentures. See *1.2.1.1.1 Oral mucosal pain associated with trauma* and *1.2.1.1.2 Oral mucosal pain attributed to infection*.

1.2.1.2 Oral mucosal pain attributed to a malignant lesion

Oral mucosal pain related to a malignant disease can be painful to a varying degree. The pain may be mild to severe and is exacerbated by mechanical provocation of the gingivae. Spontaneous pain can occur.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.2.1 Oral mucosal pain*
- B. The patient has been diagnosed with a malignant lesion¹ known to be able to cause oral mucosal pain
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ neoplasia of the oral mucosa

Comments:

The oral mucosa may be affected by an array of both primary and metastatic malignancies which may all present as non-specific ulcers. Oral squamous cell carcinoma (OSCC) is the most common, frequently presenting as ulceration with clinical induration, fixation to the underlying tissues, rolled exophytic margins, pain and/or numbness (Warnakulasuriya et al 2007).

1.2.1.3 *Oral mucosal pain attributed to neuropathy; see 4.1 Pain attributed to a lesion or disease of the trigeminal nerve*

Comments:

Trigger points of trigeminal neuralgia may be located in the oral mucosa, and light touch will elicit the typical intense paroxysmal pain attacks affecting the whole dermatome corresponding to the affected nerve branch. As a result, patients may find it impossible to wear a denture in the region. For description and diagnostic criteria, see *4.1.1 Trigeminal neuralgia*.

Oral mucosal pain may occur as part of the early clinical presentation of trigeminal neuralgia, the diffuse deep pain, "pre-trigeminal neuralgia pain", that sometimes precedes the onset of characteristic paroxysmal pain.

Peripheral neuropathy and may be associated with pain in the oral mucosa. For description and diagnostic criteria, see *4.1.2 Trigeminal neuropathic pain other than 4.1.1 Trigeminal neuralgia*.

1.2.1.4 *Idiopathic oral mucosal pain; see 6 Idiopathic orofacial pain*

Comments:

Burning mouth syndrome (BMS) presents as localized or more widely distributed oral mucosal pain. For description and diagnostic criteria, see *6.1 Burning mouth syndrome (BMS)*.

Persistent idiopathic dentoalveolar pain (PIDAP) is sometimes associated with localized pain in the adjacent oral mucosa. For description and diagnostic criteria, see *6.3 Persistent idiopathic dentoalveolar pain*.

Consideration must be given to patients presenting with chronic widespread pain or other multiple pain conditions which may be attributable to central sensitization or other mechanisms.

1.2.2 Salivary gland pain

Description:

Pain caused by a disorder involving the salivary glands.

Diagnostic criteria:

- A. Any pain fulfilling criteria B through E
- B. Pain is localized to the site of the salivary gland lesion, but may refer to other ipsilateral orofacial locations
- C. Clinical, laboratory, imaging and/or anamnestic evidence of a lesion or disease of the salivary glands¹ known to be able to cause pain
- D. Evidence of causation demonstrated by either or both of the following:
 - 1. Pain has developed in temporal relation to the onset or appearance of the lesion
 - 2. Familiar pain is exacerbated by pressure applied to the affected salivary gland
- E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Notes:

¹ as specified per sub-diagnosis

1.2.2.1 Salivary gland pain attributed to obstructive cause

Diagnostic criteria:

- A. The pain fulfils criteria for *1.2.2 Salivary gland pain*

B. The patient has been diagnosed with a condition causing obstruction of the salivary duct

a. sialolithiasis

b. mucus plug

c. space occupying lesion

d. traumatic or iatrogenic injury of the salivary gland or salivary duct

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Patients with obstruction of the salivary duct most commonly present with history of acute intermittent pain and swelling of the affected major salivary gland. The degree of pain and swelling is dependent on the extent of salivary duct obstruction and the presence of secondary infection.

Iatrogenic causes include therapy-related injury, e.g. I₁₃₁ mediated. Salivary gland function is affected after high-activity radioiodine ablation therapy in patients with differentiated thyroid cancer (Klein Hesselink et al. 2016). Radioactive iodine is actively accumulated in salivary gland tissue, and sialadenitis is a common sequela along with decreased saliva secretion and xerostomia leading to salivary gland infection and pain.

1.2.2.2 Salivary gland pain attributed to infection

Diagnostic criteria:

A. The pain fulfils criteria for *1.2.2 Salivary gland pain*

B. The patient has been diagnosed with an infection of the salivary gland (or glands)

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

1.2.2.2.1 Salivary gland pain attributed to viral infection

Diagnostic criteria:

- A. The pain fulfills criteria for *1.2.2.2 Salivary gland pain attributed to infection*
- B. The patient has been diagnosed with a viral infection of the salivary gland (or glands)
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Viral infections of the salivary glands include mumps, HIV, and CMV infection, which can cause pain in addition to swelling.

Mumps mostly affects the parotid gland, with bilateral sudden enlargement, painful to palpation, but up to 25% may involve unilateral swelling. Severe local pain is often noted, in moving the jaws in talking and chewing, especially if partial duct obstruction occurs. It typically affects children 4–6 years of age.

1.2.2.2.2 Salivary gland pain attributed to bacterial infection

Diagnostic criteria:

- A. The pain fulfills criteria for *1.2.2.2 Salivary gland pain attributed to infection*
- B. The patient has been diagnosed with a bacterial infection of the salivary gland (or glands)
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

The most common bacterial cause is *Staphylococcus* infection.

Bacterial sialadenitis can be either acute or chronic. Decreased saliva flow rate is the primary predisposing factor, and this allows retrograde microbial colonization of the duct, which may result in the development of acute or chronic suppurative infection. Acute sialadenitis is characterized by a painful swelling of a single salivary gland, commonly affecting the parotid gland. A purulent discharge may be expressed from the salivary duct orifice, and the patient may present with redness of the overlying skin or even abscess formation within the inflamed gland tissue, malaise, fever, and cervical lymphadenopathy. Bacterial sialadenitis often occurs in immunocompromised patients and in elderly patients who suffer from salivary gland hypofunction due to systemic diseases, medication intake, or dehydration, or it may be associated with obstruction of the salivary ducts by deposition of calculi, mucus plugs, and tumor growth or by trauma. Chronic sialadenitis may develop following acute sialadenitis if the predisposing factors cannot be eliminated. *Staphylococcus aureus* is the most common pathogen isolated from purulent sialadenitis (Brook 2009).

1.2.2.3 Salivary gland pain attributed to non-infectious cause

Description:

Other causes of salivary gland pain may include pain attributed to allogeneic transplantation with a graft versus host disease (GVHD). Salivary glands are a major target of GVHD and manifest as hyposalivation and xerostomia (Bassim et al. 2015), infection and subsequent pain.

Diagnostic criteria:

A. The pain fulfills criteria for *1.2.2 Salivary gland pain*

B. The pain does not fulfill criteria for 1.2.2.1–1.2.2.2

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

1.2.2.3.1 Salivary gland pain attributed to recurrent juvenile parotitis

Diagnostic criteria:

A. The pain fulfills criteria for *1.2.2.3 Salivary gland pain attributed to non-infectious cause*

B. The patient has been diagnosed with recurrent juvenile parotitis

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Juvenile recurrent parotitis is a common condition of the salivary glands in children, which is characterized by intermittent swelling of the parotid glands on one or both sides, with or without pain, and generally associated with nonobstructive sialectasis of the parotid gland, as well as salivary gland hypofunction (Leerdam et al. 2005). It has a biphasic age distribution, with peaks at 2–5 years and 10 years of age. The most common symptoms are swelling, pain and fever. Symptoms are limited to about 3 days and may be frequent, with about 8 episodes per year. It is diagnosed from the medical history and confirmed by sialography or ultrasonography. The etiology is unclear, but in most patients juvenile parotitis resolves during adulthood.

1.2.2.3.2 Salivary gland pain attributed to immunologically mediated disorder

Diagnostic criteria:

A. The pain fulfills criteria for *1.2.2.3 Salivary gland pain attributed to non-infectious cause*

B. The patient has been diagnosed with an immunologically mediated disorder

a. Sjögren's syndrome

b. Other immunologically mediated disorder

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Immunologically mediated salivary gland pain includes Sjögren's syndrome, an autoimmune disease that results in salivary gland dysfunction. Symptoms include recurrent or persistent swelling of the salivary glands, dryness of the mouth, difficulty of chewing, pain and burning sensation of oral mucosa, chronic sore throat and pain with swallowing.

1.2.2.3.3 Salivary gland pain attributed to other cause

Diagnostic criteria:

A. The pain fulfills criteria for *1.2.2.3 Salivary gland pain attributed to non-infectious cause*

B. The pain does not fulfill criteria for 1.2.2.3.1–1.2.2.3.2

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Infrequent causes for salivary gland pain may include benign and malignant tumors of the salivary glands. These are usually not directly associated with pain (Guzzo et al. 2010), but

secondary pain may occur related to obstruction of the gland or duct (see *1.2.2.1 Salivary gland pain attributed to obstructive cause*) or, for malignant tumors, to nerve impingement (see *4.1.2 Trigeminal neuropathic pain*).

1.2.3 Jaw bone pain

Description:

Pain caused by a disorder involving the jaw bone tissue.

Diagnostic criteria:

- A. Any pain fulfilling criteria B through E
- B. Pain is localized to the site of the jaw bone lesion, but may refer to other ipsilateral orofacial locations
- C. Clinical, laboratory, imaging and/or anamnestic evidence of a lesion or disease of the jaw bone¹ known to be able to cause pain
- D. Evidence of causation demonstrated by either or both of the following:
 - 1. Pain has developed in temporal relation to the onset or appearance of the jaw bone lesion
 - 2. Familiar pain is exacerbated by pressure applied to the jaw bone lesion
- E. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ as specified per sub-diagnosis

1.2.3.1 Jaw bone pain attributed to infection

Diagnostic criteria:

- A. The pain fulfils criteria for *1.2.3 Jaw bone pain*
- B. The patient has been diagnosed with an infection of the jaw bone tissue
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Intra-bony bacterial, viral and fungal infections may cause jaw bone pain. The most common ones are bacterial infections.

Infection can occur secondary to osteo(radio)necrosis of the jaws, which may contribute to the pain associated with osteonecrosis, see *1.2.3.2 Therapy related jaw bone pain*.

1.2.3.1.1 Jaw bone pain attributed to bacterial infection

Diagnostic criteria:

- A. The pain fulfils criteria for *1.2.3.1 Jaw bone pain attributed to infection*
- B. The patient has been diagnosed with a bacterial infection of the jaw bone tissue
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Bacterial infections of the jaw bone tissue include osteomyelitis. Odontogenic infections can spread and cause osteomyelitis of the jaw, but osteomyelitis secondary to odontogenic infection is relatively uncommon. Severe mandibular pain is a common symptom of jaw osteomyelitis and can be accompanied by anesthesia or hypoesthesia on the affected side. In protracted cases, mandibular trismus may develop.

1.2.3.1.2 Jaw bone pain attributed to viral infection

Diagnostic criteria:

- A. The pain fulfils criteria for *1.2.3.1 Jaw bone pain attributed to infection*
- B. The patient has been diagnosed with a viral infection of the jaw bone tissue
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Viral infections of the jaw bone tissue include herpes zoster induced osteonecrosis.

Herpes zoster (HZ) (shingles) results due to reactivation of Varicella zoster virus. Unusual dental complications like osteonecrosis, exfoliation of teeth, periodontitis, and calcified and devitalized pulps, periapical lesions, and resorption of roots as well as developmental anomalies such as irregular short roots and missing teeth may arise secondary to involvement of 2nd or 3rd division of trigeminal nerve by HZ (Gupta et al 2015).

1.2.3.1.3 Jaw bone pain attributed to fungal infection

Diagnostic criteria:

- A. The pain fulfils criteria for *1.2.3.1 Jaw bone pain attributed to infection*

B. The patient has been diagnosed with a fungal infection of the jaw bone tissue

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

The most common fungal infections of the jaw bone tissue are aspergillosis and mucormycosis. Aspergillosis of the oral cavity is an uncommon condition which most frequently occurs in immunocompromised patients, such as those with hematological malignancies. Osteomyelitis caused by *Aspergillus* species is an infection that is often neglected. Invasive oral aspergillosis, though rare, is a potentially lethal disease and it should be considered in immunosuppressed patients with oral lesions (Gabrielli et al. 2014).

Mucormycosis is a rare opportunistic infection invariably affecting immunocompromised patients, but it may affect rarely healthy individuals after tooth extraction (Nilesh and Vande 2018). The organism implicated to cause mucormycosis is a saprophytic fungus, mainly *rhizopus* or *mucor*. It is the most deadly and rapidly progressing form of fungal infection affecting humans.

1.2.3.2 Therapy related jaw bone pain

Diagnostic criteria:

A. The pain fulfils criteria for *1.2.3 Jaw bone pain*

B. The pain has developed in close temporal relation¹ to a therapeutic intervention known to be able to cause jaw bone pain

a. medication related osteonecrosis of the jaws (MRONJ)

b. osteoradionecrosis

c. alveolar osteitis (dry socket)

d. other therapeutic intervention

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ normally hours to weeks; see sub-diagnoses

Comments:

Medication-related osteonecrosis of the jaw is defined by the presence of necrotic bone (that is exposed or can be probed through a sinus tract) for more than 8 weeks in the maxillofacial region of an individual treated with bisphosphonate or other anti-resorptive (e.g. denosumab) or anti-angiogenic (e.g. bevacizumab) medications. MRONJ typically presents as pain, infection, and necrotic bone in the mandible or maxilla in patients receiving these agents. Dentoalveolar surgery is a major risk factor.

Osteoradionecrosis is a complication of radiation therapy (RT) due to vascular obliteration and decreased vascular supply of the irradiated tissues. Symptoms of osteoradionecrosis can include pain, bad breath, dysgeusia, dysesthesia or anesthesia, trismus, difficulty with chewing and swallowing, speech difficulties, fistula formation, pathologic fracture, and infection. The time to onset of osteoradionecrosis is quite variable. In some cases, it may be diagnosed shortly after completion of RT, while in other patients it may not be diagnosed for years after the original cancer treatment. The mandible is the most frequently affected bone while maxillary osteoradionecrosis is rare.

Alveolar osteitis (dry socket) is a complication of dental extractions and occurs more commonly in extractions involving mandibular molar teeth. It is associated with severe pain developing 2 to 3 days postoperatively. A socket that may be partially or totally devoid of blood clot is often found and some patients experience halitosis.

1.2.3.3 *Jaw bone pain attributed to a local benign lesion*

Description:

Benign bone tumors often are asymptomatic and discovered incidentally during evaluation for trauma or another condition. When they are symptomatic, benign bone tumors may present with localized pain, swelling, deformity, or pathologic fracture. Most benign bone tumors have characteristic radiographic features. Advanced imaging techniques (e.g, computed tomography, magnetic resonance imaging) may be necessary to fully characterize bone tumors.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.2.3 Jaw bone pain*
- B. The patient has been diagnosed with a local benign lesion known to be able to cause jaw bone tissue pain
 - a. giant cell tumor
 - b. osteoid osteoma
 - c. osteoblastoma
 - d. other benign lesion
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Giant cell tumor of bone (GCTB) is a relatively rare, benign osteolytic skeletal neoplasm of young adults. The most common presentation of GCTB is pain and swelling. Skull and craniofacial bones are less commonly involved sites.

Patients with osteoid osteoma typically complain of progressively increasing pain that is worse at night and unrelated to activity. The pain is relieved by aspirin or other nonsteroidal anti-inflammatory medications (NSAID), usually within 20 to 25 minutes. Lack of relief by agents should prompt consideration of other diagnoses.

Patients with osteoblastoma typically complain of chronic, continuous pain. The radiographic findings of osteoblastoma are variable, and advanced imaging (e.g., CT or MRI) often is required for identification. Pain is not relieved by aspirin or NSAID.

1.2.3.4 Jaw bone pain attributed to a malignant lesion

Description:

Jaw bone pain attributed to malignant lesions, whether primary or metastatic, may present with localized pain that may increase and wane over a few weeks' or months' duration. Pain may be due to direct mass effect of primary or metastatic tumor, or due to paraneoplastic effect in metastatic cases.

Diagnostic criteria:

- A. The pain fulfills criteria for *1.2.3 Jaw bone pain*
- B. The patient has been diagnosed with a malignant lesion¹
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes:

¹ primary or secondary

1.2.3.4.1 Jaw bone pain attributed to a primary malignant lesion

Diagnostic criteria:

- A. The pain fulfils criteria for *1.2.3.4 Jaw bone pain attributed to a malignant lesion*
- B. The patient has been diagnosed with a primary malignant lesion known to be able to cause jaw bone tissue pain
 - a. osteosarcoma
 - b. Langerhans' cell histiocytosis
 - c. non-Hodgkin lymphoma
 - d. multiple myeloma
 - e. other primary malignant lesion
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Osteosarcoma is an uncommon tumor, but by far the most common primary malignant tumor arising in bone (myeloma excluded). The majority of patients with osteosarcoma present with localized pain, typically of several months' duration. Pain frequently begins after an injury and may wax and wane over of a few weeks' or months' duration. Systemic symptoms such as fever, weight loss, and malaise are generally absent. Osteosarcomas are often considered secondary neoplasms, attributed to sarcomatous transformation of Paget disease of bone, or some other benign bone lesions. The most common sites of involvement are distal femur, proximal tibia, and proximal femur, presence in the jaw bones is rather rare.

In Langerhans cell histiocytosis (LCH), radiologic studies typically demonstrate a lytic, "punched out" appearance, sometimes with an accompanying soft tissue mass. Pain in the jaw and loose teeth may be a presenting symptom. Although bone lesions may be asymptomatic in some areas, those in the mouth are especially troublesome because of tooth loss and a high recurrence rate. Posterior regions of the jawbones are affected more often than anterior regions.

Non-Hodgkin lymphoma is a lymphatic system tumor originating from either B or T lymphocytes and shows a high malignant potential. Non-specific symptoms, such as unclear primary dental pain and unresolved periapical swelling, can make an accurate diagnosis of Non-Hodgkin lymphoma difficult, which frequently lead to delayed diagnosis. A CT or cone beam computed tomography (CBCT) scan of the jaws and immunohistochemical staining of the biopsy specimen is recommended (Zou H et al. 2018). When the lesion affects the bones of the jaws, it is rare in the mandible when compared to the maxilla. In the reported cases, only 0.6% is found in the mandible.

Multiple myeloma is a condition where plasma cells proliferate in the bone marrow and often results in extensive skeletal destruction with osteolytic lesions, osteopenia, and/or pathologic fractures. Bone pain, particularly in the back or chest, and less often in the extremities, is present at the time of diagnosis in approximately 60 percent of patients.

1.2.3.4.2 Jaw bone pain secondary to a malignant lesion

Diagnostic criteria:

- A. The pain fulfils criteria for *1.2.3.4 Jaw bone pain attributed to a malignant lesion*
- B. The patient has been diagnosed with a malignant lesion known to be able to cause secondary jaw bone tissue pain by direct or indirect mechanisms
 - a. direct mass effect of metastatic tumor
 - b. paraneoplastic effect

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Direct mass effects of a metastatic tumor include nerve compression and periosteal stretch.

A paraneoplastic effect is a remote effect with no metastatic spread to the jaws.

1.2.3.5 Jaw bone pain attributed to systemic disease

Description:

Some systemic diseases may present with repeated vaso-occlusive pain episodes, characterized by diffuse bone pain, punctuated by painful crises that often result in osteonecrosis (avascular necrosis).

Diagnostic criteria:

- A. The pain fulfils criteria for *1.2.3 Jaw bone pain*
- B. The patient has been diagnosed with a systemic disease known to be able to cause jaw bone pain
 - a. sickle cell disease
 - b. Gaucher's disease
 - c. Paget's disease
 - d. other systemic disease
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments:

Sickle cell disease (SCD) is characterized by a marked heterogeneity in clinical and hematologic severity, with repeated vaso-occlusive pain episodes as the hallmark of SCD. Pain episodes may occur as often as every week, or individuals with SCD may go with long stretches of time without any pain events. Pain episodes can lead to bone infarcts, necrosis, and, over time, degenerative changes in marrow-containing bone, leading to a chronic state of pain in addition to the more acute painful episodes.

Gaucher disease (GD) is an inborn error of metabolism that affects the recycling of cellular glycolipids, being one of the most common lysosomal storage diseases. Skeletal disease is characterized by diffuse bone pain, punctuated by painful crises that often result in osteonecrosis (avascular necrosis).

Paget disease of bone (PDB) is also known historically as osteitis deformans. PDB is a focal disorder of bone metabolism, characterized by an accelerated rate of bone remodeling, resulting in overgrowth of bone at single (monostotic PDB) or multiple (polyostotic PDB) sites. Commonly affected areas include the skull, spine, pelvis, and long bones of the lower extremity. Similar to osteosarcomas, they may present with localized pain and swelling and typically occur in patients with polyostotic disease.

1.2.3.6 Jaw bone pain attributed to traumatic injury

Description:

Jaw bone pain attributed to traumatic injury includes jaw fracture. Sports, e.g. football, baseball, and hockey, and motor vehicle collisions account for a high percentage of facial injuries among young adults. Chin lacerations in particular are associated with mandibular fractures. A mandible fracture may be present if the patient experiences restricted or abnormal mouth-opening; malocclusion also suggests the presence of a mandibular fracture, as does numbness of the chin that is present immediately following trauma.

Diagnostic criteria:

- A. The pain fulfils criteria for *1.2.3 Jaw bone pain*
- B. The patient has been diagnosed with a traumatic injury of the jaw
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

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(pulpal pain)

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2. Orofacial pain associated with regional muscles

General comments

Temporomandibular disorders (TMD) is a term used to describe a number of painful and non-painful disorders affecting the muscles of mastication, the temporomandibular joint and contiguous structures. The Diagnostic Criteria for Temporomandibular disorders (DC/TMD) published by INfORM are reliable and universally accepted (Schiffman et al., 2014, Peck et al. 2014). Nevertheless, controversy remains regarding terminology of chronic muscle pain. The DC/TMD (1) uses “myalgia” and “myofascial pain” and other suggestions have been e.g. “persistent orofacial muscle pain” (2). We here propose to use the overarching label “orofacial pain associated with regional muscles” and for the specific diagnosis listed below to adhere to the term “myofascial” in recognition of the lack of concrete evidence to link pain to specific structures or tissues in the muscle. The same caution is noted in the overarching label by the use of the word “associated”. Furthermore, the term TMD is being maintained to align with the DC/TMD diagnosis.

Diagnostic criteria

2.1. Primary myofascial pain

2.1.1. Acute primary myofascial pain

Diagnostic criteria

A. Single or repeated days of myofascial pain occurring within the past 3 months and fulfilling criteria B-D

B. Lasting from at least 2 hours daily to days, or unremitting

C. Positive for both of the following⁴

1. Pain in the jaw, temple, in the ear or in front of ear
2. Pain modified with jaw movement, function or parafunction

D. Positive for both of the following⁵

1. Confirmation of pain location(s) in the temporalis or masseter muscle(s)
2. Report of familiar pain in the temporalis or masseter muscle(s) with at least one of the following provocation tests: Palpation of the temporalis or masseter muscle(s) OR maximum unassisted or assisted opening movement(s)

E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

2.1.2. Chronic primary myofascial pain

2.1.2.1. Chronic infrequent primary myofascial pain

Diagnostic criteria

A. At least 10 episodes of myofascial pain occurring on <1 day per month on average (<12 days per year) and fulfilling criteria B-D

B. Lasting from at least 2 hours daily to days, or unremitting

C. Positive for both of the following

1. Pain in the jaw, temple, in the ear or in front of ear AND
2. Pain modified with jaw movement, function or parafunction

D. Positive for both of the following

1. Confirmation of pain location(s) in the temporalis or masseter muscle(s)

2. Report of familiar pain in the temporalis or masseter muscle(s) with at least one of the following provocation tests: Palpation of the temporalis or masseter muscle(s) OR maximum unassisted or assisted opening movement(s)

E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

2.1.2.2. Chronic frequent primary myofascial pain

2.1.2.2.1. Chronic frequent primary myofascial pain without pain referral

Diagnostic criteria

A. Myofascial pain occurring with at least 10 episodes and 1-14 days per month on average for > 3 months (>12 and < 180 days per year) and fulfilling criteria B-D

B. Lasting from at least 2 hours daily to days, or unremitting

C. Positive for both of the following

1. Pain in the jaw, temple, in the ear or in front of ear AND
2. Pain modified with jaw movement, function or parafunction

D. Positive for both of the following

1. Confirmation of pain location(s) in the temporalis or masseter muscle(s) AND
2. Report of familiar pain in the temporalis or masseter muscle(s) with at least one of the following provocation tests: Palpation of the temporalis or masseter muscle(s) OR maximum unassisted or assisted opening movement(s)

E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

2.1.2.2.2. Chronic frequent primary myofascial pain with pain referral

Diagnostic criteria

A. Myofascial pain occurring with at least 10 episodes and 1-14 days per month on average for > 3 months (>12 and < 180 days per year) and fulfilling

criteria B-D

B. Lasting from at least 2 hours daily to days, or unremitting

C. Positive for both of the following

1. Pain in the jaw, temple, in the ear or in front of ear AND
2. Pain modified with jaw movement, function or parafunction

D. Positive for all of the following

1. Confirmation of pain location(s) in the temporalis or masseter muscle(s) AND
2. Report of familiar pain with palpation of the temporalis or masseter muscle(s)
AND
3. Report of pain at a site beyond the boundary of the muscle being palpated

E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

2.1.2.3. Chronic persistent primary myofascial pain

2.1.2.3.1 Chronic persistent primary myofascial pain without pain referral

Diagnostic criteria

A. Myofascial pain occurring on >15 days per month on average for >3 months (>180 days per year), fulfilling criteria B-D

B. Lasting from at least 2 hours daily to days, or unremitting

C. Positive for both of the following

1. Pain in the jaw, temple, in the ear or in front of ear AND
2. Pain modified with jaw movement, function or parafunction

D. Positive for both of the following

1. Confirmation of pain location(s) in the temporalis or masseter muscle(s) AND

2. Report of familiar in the temporalis or masseter muscle(s) with at least one of the following provocation tests: Palpation of the temporalis or masseter muscle(s) OR maximum unassisted or assisted opening movement(s)

E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

2.1.2.3.2. Chronic persistent primary myofascial pain with pain referral

Diagnostic criteria

A. Myofascial pain occurring on >15 days per month on average for >3 months (>180 days per year), fulfilling criteria B-D

B. Lasting from at least 2 hours daily to days, or unremitting

C. Positive for both of the following

1. Pain in the jaw, temple, in the ear or in front of ear AND
2. Pain modified with jaw movement, function or parafunction

D. Positive for all of the following:

1. Confirmation of pain location(s) in the temporalis or masseter muscle(s) AND
2. Report of familiar pain with palpation of the temporalis or masseter muscle(s) AND
3. Report of pain at a site beyond the boundary of the muscle being palpated

E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

2.2. Secondary myofascial pain

Description

Myofascial pain attributed to persistent inflammation (due to e.g. infection, crystal deposition or autoimmune disorders), structural changes (such as osteoarthritis or spondylosis), injury, or diseases of the nervous system.

Diagnostic criteria

- A. Any myofascial pain according to 2.1.1-4 and fulfilling criterion C
- B. The parent disorder meets its respective diagnostic criteria
- C. Evidence of causation demonstrated by at least two of the following:
 - 1. Myofascial pain has developed, or substantially worsened, in temporal relation to the onset of the presumed causative disorder
 - 2. Myofascial pain has significantly worsened in parallel with progression of the presumed causative disorder
 - 3. Myofascial pain has significantly improved or resolved in parallel with improvement in or resolution of the presumed causative disorder
- D. Not better accounted for by another ICOP or ICHD-3 diagnosis.

2.2.1 Secondary myofascial pain due to tendonitis

Diagnostic criteria

- A. Myofascial pain of tendon origin occurring in any masticatory muscle during the last 30 days fulfilling criteria B-D
- B. Evidence of causation demonstrated by at least two of the following:
 - 1. Myofascial pain has developed in temporal relation to onset of tendonitis
 - 2. Myofascial pain has significantly worsened in parallel with progression of tendonitis
 - 3. Myofascial pain has significantly improved or resolved in parallel with improvement in or resolution of of tendonitis
- C. Positive for all of the following
 - 1. Pain in the jaw, temple, in the ear or in front of ear
 - 2. Pain modified with jaw movement, function or parafunction
 - 3. Confirmation of pain location(s) in the affected muscle

4. Report of familiar pain with at least one of the following provocation tests:
Palpation of the affected tendon OR maximum unassisted or assisted opening movement(s)

D. Not better accounted for by another ICOP or ICHD-3 diagnosis.

2.2.2 Secondary myofascial pain due to myositis

Diagnostic criteria

- A. Myofascial pain occurring in any masticatory muscle during the last 30 days fulfilling criteria B-E
- B. Evidence of causation demonstrated by at least two of the following:
 1. Myofascial pain has developed in temporal relation to onset of inflammation, infection or trauma
 2. Myofascial pain has significantly worsened in parallel with progression of inflammation, infection or trauma
 3. Myofascial pain has significantly improved or resolved in parallel with improvement in or resolution of inflammation, infection or trauma
- C. Positive for all of the following
 1. Pain in the jaw, temple, in the ear or in front of ear
 2. Pain modified with jaw movement, function or parafunction
 3. Confirmation of pain location(s) in the affected muscle(s)
 4. Report of familiar pain in the muscle with at least one of the following provocation tests: Palpation of the muscle OR maximum unassisted or assisted opening movement(s)
 5. Presence of edema, erythema, and/or increased temperature over the muscle
- D. Serologic tests may reveal elevated enzyme levels (e.g., creatine kinase), markers of inflammation and the presence of autoimmune diseases.

E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

2.2.3 Secondary myofascial pain due to muscle spasm

Diagnostic criteria

A. Myofascial pain occurring in any masticatory muscle during the last 30 days fulfilling criteria BE

B. Evidence of causation demonstrated by at least two of the following:

1. Myofascial pain has developed in immediate temporal relation to onset of spasm
2. Myofascial pain has significantly worsened in parallel with progression of spasm
3. Myofascial pain has significantly improved or resolved in parallel with improvement in or resolution of spasm

C. Positive for all of the following

1. Pain in the jaw, temple, in the ear or in front of ear
2. Pain modified with jaw movement, function or parafunction
3. Confirmation of pain location(s) in the affected muscle(s)
4. Report of familiar pain in the affected muscle with at least one of the following provocation tests: Palpation of muscle OR maximum unassisted or assisted opening movement(s)
5. Limited range of jaw motion in direction that elongates affected muscle; (i.e. for jaw closing muscles, opening will be limited to <40 mm; for lateral pterygoid muscle, ipsilateral movement will be limited to <7 mm)

D. If diagnosis needs to be confirmed, intramuscular electromyography (EMG) shows elevated activity when compared to contralateral unaffected muscle.

E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Comments

The temporal distinction of the myofascial pains is novel to the DC/TMD but follows the general principles from ICHD-3 for tension-type headaches. The same criteria with regard to episode frequency have been implemented in the current classification. While there may be debates about the clinical significance of infrequent myofascial TMDs there appears to be solid evidence in favor to separate more frequently occurring pain conditions from less frequently occurring pain conditions. Future studies using the proposed temporal distinction between myofascial pains may reveal the therapeutic implications.

Whereas some dentists and many OFP experts may manage cervical muscle pain in addition to jaw muscle pain, it was decided at this stage to not include a classification for cervical muscle pains. However, a possible suggestion for classifying such pains could be to adhere to the same principles laid out in the classification of regional jaw muscle pain, i.e., in terms of frequency, criteria for local examination findings, and with or without referral of pain. The same could be applied to other muscles in the orofacial region, e.g., to the tongue muscles or swallowing muscles. A further comment is that the DC/TMD was not restricted to only the temporalis and masseter; rather, the examination was specific for those muscles due to (a) higher examiner reliability, and (b) nearly all individuals with painful TMD myalgia may have pain in at least the masseter or temporalis, but there is no logical necessity for positive examination findings to not occur in only the other masticatory muscles. In short, the restriction to temporalis and masseter excludes other individuals with highly localized myalgia in the masticatory muscles, and to the clinician this will also appear as a needless restriction. Thus, the proposed classification may also be applicable to other jaw muscles.

Foot notes

1. It is widely recognized and accepted that both acute and chronic types of pain in the jaw muscles can be associated with the clinical phenomenon of pain referral, i.e., pain is perceived at a different site than the origin of the nociceptive or noxious stimulus (3). The pathophysiological significance of this remains unclear as well as do the therapeutic

implications (4); however, from a diagnostic point of view it remains clinically important to distinguish pain referrals from local pains. As a consequence, all myofascial TMD diagnosis can be divided into two categories based on the presence or absence of pain referral during either palpation or during standardized jaw movements. The definition of pain referrals follows the description in the DC/TMD. The DC/TMD also operates with a category of pain spread which in contrast to pain referral remains within the boundary of the anatomical structure. For further research purposes the specific criteria for myofascial pain with pain spread according to the DC/TMD can be applied, if needed.

2. Based on the IASP classification of chronic pain conditions (5), there is a distinction between primary and secondary pain conditions. Primary pain conditions mean that the specific etiology or cause cannot be determined, i.e., they are idiopathic although significant knowledge may exist to their pathophysiological mechanisms. Secondary pain conditions mean that the pain condition is secondary or attributed to a known medical condition or cause. Based on these definitions the proposed category of acute myofascial pain may indeed be a secondary myofascial pain condition due to acute nociceptive and inflammatory processes in the muscle tissue caused by a mechanical, chemical trauma or infection. The infrequent, frequent and chronic persistent myofascial pain conditions (2.2.-2.4.) may fit into the primary pain category.
3. The specific instructions and criteria for a positive finding will follow the DC/TMD description. The importance of standardization of palpation pressure and duration is emphasized, i.e., 1 kg for 2 s to determine pain on palpation and familiar pain and 1 kg for 5 s to determine pain referrals (or spread). It should be noted that while palpation pressure has extensive empirical support with respect to distinguishing cases from non-cases, duration of palpation pressure has only preliminary empirical support. Referral phenomena occur in the expected direction in relation to 2 vs 5 seconds; pressure for 2 seconds has good validity (defined as sensitivity and specificity) but whether 5 seconds is sufficient for the elicitation of reany existing referral phenomena is unknown.
4. For the secondary myofascial pains the Expanded Taxonomy for Temporomandibular Disorders has been used (6). These are suggested criteria for further research and have not yet been validated.

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3. Orofacial pain associated with disorders of the TMJ

General comments

Like the myofascial pains, TMJ arthralgia has been divided into primary and secondary types. For secondary TMJ arthralgia the conditions arthritis, disc displacements, degenerative joint disease and subluxation were included. The reason was that these conditions might require different and specific treatments, because it is of importance to know why there is TMJ

arthralgia. There are other conditions that may very well contribute to TMJ arthralgia, for example generalized pain conditions that sensitize the tissues in and around the TMJ. This must be considered further in future studies.

In general the term “attributed” is preferred instead of “secondary to” for these diagnoses. “Secondary to” implies a strong causality, which may be difficult to establish and the relationships may go both ways.

For the secondary TMJ arthralgia the Expanded DC/TMD Taxonomy definitions for the primary condition were used (Peck et al. 2014).

The definitions of “acute – infrequent – frequent – chronic” arthralgia are in this group used for primary TMJ arthralgia according to the definitions in the ICHD-3 document. It should be pointed out that preferably both the “acute – chronic” distinction as well as the “infrequent – frequent – continuous” scale should be used. The reason being that both acute and chronic pain can be infrequent, frequent and continuous, which may be of importance for treatment planning and prognosis.

Inclusion of sub-diagnoses of arthralgia with or without pain referral was done in order to keep the diagnoses in line with the myofascial pains. These subgroups must be regarded as research topics and, today, may not be considered for clinical use. Future research may hopefully show if there is a point to subdivide arthralgia into with or without referred pain.

Sub-diagnoses with systemic or non-systemic arthritis are included. The reason was that treatment planning and prognosis may differ if the TMJ arthritis is of local or systemic origin.

The working group considered to include the diagnosis “Idiopathic TMJ arthralgia”. However, the overlap with “Primary TMJ arthralgia” is substantial, if not total, and therefore the “Idiopathic TMJ arthralgia” may not contribute to research or clinical work.

3.1. Primary TMJ arthralgia

3.1.1. Acute primary TMJ arthralgia

Diagnostic criteria

A. Single or repeated days of myofascial pain occurring within the past 3 months and fulfilling criteria B-D

B. Lasting from at least 2 hours¹ daily to days, or unremitting

C. All of the following characteristics:

1. Pain in front of the ear, or in the ear

2. Pain modified with jaw movement, function or parafunction.

3. Examiner confirmation of pain location in the area of the TMJ(s).

4. Report of familiar pain in the TMJ with at least one of the following provocation tests:

a. Palpation of the lateral pole or around the lateral pole

b. Maximum unassisted or assisted opening, right or left lateral movements, or protrusive movements

D. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Comment

¹The pain does not have to be continuous for at least 2 hours. This could be also the temporal sum of episodes that may occur during the day.

3.1.2. Chronic primary TMJ arthralgia

3.1.2.1. Chronic infrequent primary TMJ arthralgia

Diagnostic criteria

A. At least 10 episodes of myofascial TMJ pain occurring on <1 day per month on average (<12 days per year) and fulfilling criteria B-D

B. Lasting from at least 2 hours¹ daily to days, or unremitting

C. All of the following characteristics:

1. Pain in front of the ear, or in the ear

2. Pain modified with jaw movement, function or parafunction.

3. Examiner confirmation of pain location in the area of the TMJ(s).
4. Report of familiar pain in the TMJ with at least one of the following provocation tests:
 - a. Palpation of the lateral pole or around the lateral pole
 - b. Maximum unassisted or assisted opening, right or left lateral movements, or protrusive movements

D. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Comment

¹The pain does not have to be continuous for at least 2 hours. This could be also the temporal sum of episodes that may occur during the day.

3.1.2.2. Chronic frequent primary TMJ arthralgia

3.1.2.2.1. Chronic frequent primary TMJ arthralgia without referred pain

Diagnostic criteria

A. TMJ pain occurring with at least 10 episodes and 1-14 days per month on average for > 3 months (>12 and < 180 days per year) and fulfilling criteria B-D

B. Lasting from at least 2 hours¹ daily to days, or unremitting

C. All of the following characteristics:

1. Pain in front of the ear, or in the ear
2. Pain modified with jaw movement, function or parafunction.
3. Examiner confirmation of pain location in the area of the TMJ(s).
4. Report of familiar pain in the TMJ with at least one of the following provocation tests:
 - a. Palpation of the lateral pole or around the lateral pole
 - b. Maximum unassisted or assisted opening, right or left lateral movements, or protrusive movements

c. pain with TMJ palpation with pain localized to the immediate site of the palpation

D. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Comment

¹The pain does not have to be continuous for at least 2 hours. This could be also the temporal sum of episodes that may occur during the day.

3.1.2.2.2. Chronic frequent primary TMJ arthralgia with referred pain

Diagnostic criteria

A. TMJ pain occurring with at least 10 episodes and 1-14 days per month on average for > 3 months (>12 and < 180 days per year) and fulfilling criteria B-D

B. Lasting from at least 2 hours¹ daily to days, or unremitting

C. All of the following characteristics:

1. Pain in front of the ear, or in the ear

2. Pain modified with jaw movement, function or parafunction.

3. Examiner confirmation of pain location in the area of the TMJ(s).

4. Report of familiar pain in the TMJ with at least one of the following provocation tests:

a. Palpation of the lateral pole or around the lateral pole

b. Maximum unassisted or assisted opening, right or left lateral movements, or protrusive movements

c. pain with TMJ palpation beyond the location of the TMJ

D. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Comment

¹The pain does not have to be continuous for at least 2 hours. This could be also the temporal sum of episodes that may occur during the day.

3.1.2.3. Chronic persistent primary TMJ arthralgia

3.1.2.3.1. Chronic persistent primary TMJ arthralgia without referred pain

Diagnostic criteria

A. TMJ pain occurring on >15 days per month on average for >3 months (>180 days per year), fulfilling criteria B-D

B. Lasting from at least 2 hours¹ daily to days, or unremitting

C. All of the following characteristics:

1. Pain in front of the ear, or in the ear
2. Pain modified with jaw movement, function or parafunction.
3. Examiner confirmation of pain location in the area of the TMJ(s).
4. Report of familiar pain in the TMJ with at least one of the following provocation tests:
 - a. Palpation of the lateral pole or around the lateral pole
 - b. Maximum unassisted or assisted opening, right or left lateral movements, or protrusive movements
 - c. pain with TMJ palpation with pain localized to the immediate site of the palpation

D. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Comment

¹The pain does not have to be continuous for at least 2 hours. This could be also the temporal sum of episodes that may occur during the day.

3.1.2.3.2. Chronic persistent primary TMJ arthralgia with referred pain

Diagnostic criteria

A. TMJ pain occurring on >15 days per month on average for >3 months (>180 days per year), fulfilling criteria B-D

B. Lasting from at least 2 hours¹ daily to days, or unremitting

C. All of the following characteristics:

1. Pain in front of the ear, or in the ear

2. Pain modified with jaw movement, function or parafunction.

3. Examiner confirmation of pain location in the area of the TMJ(s).

4. Report of familiar pain in the TMJ with at least one of the following provocation tests:

a. Palpation of the lateral pole or around the lateral pole

b. Maximum unassisted or assisted opening, right or left lateral movements, or protrusive movements

c. pain with TMJ palpation beyond the location of the TMJ

D. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Comment

¹The pain does not have to be continuous for at least 2 hours. This could be also the temporal sum of episodes that may occur during the day.

3.2. Secondary TMJ arthralgia

Description

TMJ pain attributed to persistent inflammation (due to e.g. trauma, infection, crystal deposition or autoimmune disorders), sensitization of the tissues, structural changes (such as osteoarthritis, disc displacement or subluxation), injury or diseases of the nervous system.

Diagnostic criteria

A. Any TMJ arthralgia according to 3.1.1-2 and fulfilling criteria C-D

B. The parent disorder¹ meets its respective diagnostic criteria²

C. Evidence of causation demonstrated by at least two of the following:

1. TMJ pain has developed in temporal relation to the onset or substantial worsening of the presumed causative disorder
2. TMJ pain has significantly worsened in parallel with progression of the presumed causative disorder
3. TMJ pain has significantly improved or resolved in parallel with improvement in or resolution of the presumed causative disorder

D. Not better accounted for by another ICOP OR ICHD-3 diagnosis.

3.2.1. TMJ arthralgia attributed to arthritis

Diagnostic criteria

A. TMJ pain occurring the last 30 days fulfilling criteria B-E

B. Probable TMJ arthritis diagnosis² by fulfilling both of the following:

1. TMJ pain on maximum mouth opening
2. Contralateral laterotrusion < 8 mm

C. If TMJ arthritis is to be associated with a systemic inflammatory condition,

evidence of association demonstrated by at least two of the following:

1. TMJ pain has developed in close temporal relation to other symptoms and/or clinical or biological signs of onset of the systemic inflammatory condition or has led to the diagnosis of the condition
2. Either or both of the following:
 - a. TMJ pain has significantly worsened in parallel with worsening of the condition
 - b. TMJ pain has significantly improved or resolved with treatment of the condition

D. All of the following characteristics:

1. Pain in the jaw, temple, in front of the ear, or in the ear
 2. Pain modified with jaw movement, function or parafunction
 3. Confirmation of pain location in the area of the TMJ(s)
 4. Report of familiar pain in the TMJ with at least one of the following provocation tests:
 - a. Palpation of the lateral pole or around the lateral pole
 - b. Maximum unassisted or assisted opening, right or left lateral movements, or protrusive movements
- E. Not better accounted for by another ICOP or ICHD-3 diagnosis

Note

²Alstergren et al. 2018

3.2.1.1. TMJ arthralgia attributed to arthritis, non-systemic

Diagnostic criteria

- A. TMJ pain occurring the last 30 days fulfilling criteria B-D
- B. Positive for “3.2.1 TMJ arthralgia attributed to arthritis”
- C. Rheumatologic consultation, when needed, negative for rheumatologic disease
- D. Not better accounted for by another ICOP or ICHD-3 diagnosis

3.2.1.2. TMJ arthralgia attributed to arthritis, systemic

Diagnostic criteria

- A. TMJ pain occurring the last 30 days fulfilling criteria B-D
- B. Positive for “3.2.1 TMJ arthralgia attributed to arthritis”
- C. Rheumatologic diagnosis of a systemic inflammatory joint disease

D. Not better accounted for by another ICOP or ICHD-3 diagnosis

3.2.2. TMJ arthralgia attributed to disc displacement with reduction

Diagnostic criteria

A. TMJ pain occurring the last 30 days fulfilling criteria C-E

B. TMJ disc displacement with reduction has been diagnosed by fulfilling the following:

1. Positive for at least one of the following:

a. In the last 30 days any TMJ noise(s) present with jaw movement
or function

b. Patient report of any noise present during the examination

2. Positive for at least one of the following:

a. Clicking, popping and/or snapping noise detected during both
opening and closing, with palpation during at least 1 of 3
repetitions of jaw opening and closing

OR

b1. Clicking, popping and/or snapping noise detected with
palpation during at least 1 of 3 repetitions of opening or closing

AND

b2. Clicking, popping and/or snapping noise detected with
palpation during at least 1 of 3 repetitions of right or left lateral
movements, or protrusive movements

C. Evidence of association demonstrated by at least two of the following:

1. TMJ pain has developed in close temporal relation to onset of the disc

displacement or has led to the diagnosis of the condition

2. TMJ pain present exactly when the clicking sound occurs

3. Either or both of the following:

a. TMJ pain has significantly worsened in parallel with worsening of the condition

b. TMJ pain has significantly improved or resolved with treatment of the condition

D. All of the following characteristics:

1. Pain in the jaw, temple, in front of the ear, or in the ear

2. Pain modified with jaw movement, function or parafunction

3. Confirmation of pain location in the area of the TMJ(s)

4. Report of familiar pain in the TMJ with at least one of the following provocation tests:

a. Palpation of the lateral pole or around the lateral pole

b. Maximum unassisted or assisted opening, right or left lateral movements, or protrusive movements

E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

3.2.3. TMJ arthralgia attributed to disc displacement with reduction with intermittent locking

Diagnostic criteria

A. TMJ pain occurring the last 30 days fulfilling criteria C-E

B. TMJ disc displacement with reduction with intermittent locking has been diagnosed by fulfilling the following:

1. Positive for at least one of the following:

- a. In the last 30 days any TMJ noise(s) present with jaw movement or function
 - b. Patient report of any noise present during the examination
2. In the last 30 days, jaw locks with limited mouth opening, even for a moment, and then unlocks.
3. Positive for at least one of the following:
- a. Clicking, popping and/or snapping noise detected during both opening and closing, with palpation during at least 1 of 3 repetitions of jaw opening and closing
- OR
- b1. Clicking, popping and/or snapping noise detected with palpation during at least 1 of 3 repetitions of opening or closing
- AND
- b2. Clicking, popping and/or snapping noise detected with palpation during at least 1 of 3 repetitions of right or left lateral movements, or protrusive movements

C. Evidence of association demonstrated by at least two of the following:

- 1. TMJ pain has developed in close temporal relation to onset of the disc displacement or has led to the diagnosis of the condition
- 2. TMJ pain present exactly when the clicking sound occurs
- 3. Either or both of the following:
 - a. TMJ pain has significantly worsened in parallel with worsening of the condition
 - b. TMJ pain has significantly improved or resolved with treatment

of the condition

D. All of the following characteristics:

1. Pain in the jaw, temple, in front of the ear, or in the ear
2. Pain modified with jaw movement, function or parafunction.
3. Confirmation of pain location in the area of the TMJ(s)
4. Report of familiar pain in the TMJ with at least one of the following provocation tests:
 - a. Palpation of the lateral pole or around the lateral pole
 - b. Maximum unassisted or assisted opening, right or left lateral movements, or protrusive movements

E. not better accounted for by another ICOP OR ICHD-3 diagnosis.

3.2.4. TMJ arthralgia attributed to disc displacement without reduction

Diagnostic criteria

- A. TMJ pain occurring the last 30 days fulfilling criteria C-E
- B. TMJ disc displacement without reduction has been diagnosed by fulfilling the following:
 1. Jaw locked or caught so that the mouth would not open all the way
 2. Limitation in jaw opening severe enough to limit jaw opening and interfere with ability to eat.
- C. Evidence of association demonstrated by either or both of the following:
 1. TMJ pain has significantly worsened in parallel with worsening of the condition
 2. TMJ pain has significantly improved or resolved with treatment of the

condition

D. All of the following characteristics:

1. Pain in the jaw, temple, in front of the ear, or in the ear
2. Pain modified with jaw movement, function or parafunction
3. Confirmation of pain location in the area of the TMJ(s)
4. Report of familiar pain in the TMJ with at least one of the following

provocation tests:

- a. Palpation of the lateral pole or around the lateral pole
- b. Maximum unassisted or assisted opening, right or left lateral movements, or protrusive movements

E. Not better accounted for by another ICOP or ICHD-3 diagnosis

3.2.5. TMJ arthralgia attributed to degenerative joint disease

Diagnostic criteria

A. TMJ pain occurring the last 30 days fulfilling criteria C-E

B. TMJ degenerative joint disease has been diagnosed by fulfilling the following:

1. Positive for at least one of the following:

- a. In the last 30 days any TMJ noise(s) present with jaw movement or function
- b. Patient report of any noise present during the examination

2. Crepitus detected with palpation during maximum unassisted opening, maximum assisted opening, lateral, or protrusive movements

C. Evidence of association demonstrated by either or both of the following:

1. TMJ pain has significantly worsened in parallel with worsening of the

condition

2. TMJ pain has significantly improved or resolved with treatment of the condition

D. All of the following characteristics:

1. Pain in the jaw, temple, in front of the ear, or in the ear
2. Pain modified with jaw movement, function or parafunction.
3. Confirmation of pain location in the area of the TMJ(s)
4. Report of familiar pain in the TMJ with at least one of the following provocation tests:

- a. Palpation of the lateral pole or around the lateral pole
- b. Maximum unassisted or assisted opening, right or left lateral movements, or protrusive movements

E. Not better accounted for by another ICOP or ICHD-3 diagnosis

3.2.6. TMJ arthralgia attributed to subluxation

Diagnostic criteria

A. Fulfills criteria for 3.1 TMJ arthralgia as well as criteria C-F

B. Positive for both of the following:

1. In last 30 days, jaw locking or catching in a wide open mouth position, even for a moment, so could not close from the wide-open position
2. Inability to close the mouth without a specific manipulative maneuver

C. Although no exam findings are required, when this disorder is present

clinically, examination is positive for:

1. Inability to return to a normal closed mouth position without the patient

performing a specific manipulative maneuver

D. evidence of association demonstrated by either or both of the following:

1. TMJ arthralgia has significantly worsened in parallel with worsening of the condition
2. TMJ arthralgia has significantly improved or resolved with treatment of the condition

E. All of the following characteristics:

1. Pain in the jaw, temple, in front of the ear, or in the ear
2. Pain modified with jaw movement, function or parafunction.
3. Confirmation of pain location in the area of the TMJ(s)
4. Report of familiar pain in the TMJ with at least one of the following provocation tests:
 - a. Palpation of the lateral pole or around the lateral pole
 - b. Maximum unassisted or assisted opening, right or left lateral movements, or protrusive movements

F. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments

¹Parent disorder can be inflammatory conditions, mechanical impingements, articular tissue sensitization (of peripheral and/or central genesis) that may contribute to TMJ pain diagnosed by adequate diagnostic criteria.

²For diagnostic criteria regarding TMJ arthritis, see Alstergren et al. 2018 (1). For diagnostic criteria regarding disc displacement with reduction, disc displacement with reduction with intermittent locking, disc displacement without reduction with limited mouth opening, disc displacement without reduction without limited mouth opening, degenerative joint disease and subluxation, see the DC/TMD definitions in Schiffman et al. 2014 (2).

The temporal distinction of TMJ arthralgia is novel to the DC/TMD but follows the general principles from ICHD-3 for tension-type headaches. The same criteria with regard to episode frequency have been implemented in the current classification. While there may be debates about the clinical significance of infrequent TMJ arthralgia there appears to be solid evidence in favor to separate more frequently occurring pain conditions from less frequently occurring pain conditions. Future studies using the proposed temporal distinction between TMJ arthralgia may reveal the therapeutic implications.

Foot notes

It is widely recognized and accepted that both acute and chronic types of TMJ pains can be associated with the clinical phenomenon of pain referral, i.e., pain is perceived at a different site than the origin of the nociceptive or noxious stimulus (3). The pathophysiological significance of this remains unclear as well as do the therapeutic implications (4); however, from a diagnostic point of view it remains clinically important to distinguish pain referrals from local pains. As a consequence, TMJ arthralgia diagnoses can be divided into two categories based on the presence or absence of pain referral during palpation. The DC/TMD also operates with a category of pain spread which in contrast to pain referral remains within the boundary of the anatomical structure. For further research purposes the specific criteria for TMJ arthralgia with pain spread according to the DC/TMD can be applied, if needed.

Based on the IASP classification of chronic pain conditions (5), there is a distinction between primary and secondary pain conditions. Primary pain conditions mean that the specific etiology or cause cannot be determined, i.e., they are idiopathic although significant knowledge may exist to their pathophysiological mechanisms. Secondary pain conditions mean that the pain condition is secondary or attributed to a known medical condition or cause. Based on these definitions the proposed category of acute TMJ arthralgia may indeed be a secondary TMJ pain condition due to acute nociceptive and inflammatory processes in the TMJ caused by a mechanical, chemical trauma or infection. The infrequent, frequent and more frequently occurring TMJ arthralgia conditions (3.2. - 3.4.) may fit into the primary pain category.

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4. Orofacial pain associated with lesion/disorders of the cranial nerves and other regional nerve structures

General comments

This section is based in large part on ICHD-3 and IASP/ICD11. Some small changes have been made. Idiopathic conditions, i.e., Persistent Idiopathic Facial Pain and Burning Mouth Syndrome have been placed in the section dealing with idiopathic pain, as there is not yet sufficient evidence to support that these are unequivocally neuropathic pain. In certain disorders we have used the term 'neuropathic pain' in preference to 'painful neuropathy' to comply with the IASP/ICD11 criteria. The ICHD-3 would support the latter but much of the pain literature and the IASP/ICD11 is shifting to the use of 'neuropathic pain'. Those conditions in chapter 13 of

the ICHD-3, where pain is present outside the orofacial region, are only mentioned in the ICOP and the readers are referred to the ICHD-3 for specific criteria.

4.1. Pain attributed to a lesion or disease of the trigeminal nerve

4.1.1. Trigeminal neuralgia

Description

A disorder characterized by recurrent unilateral brief electric shock-like pains, abrupt in onset and termination, limited to the distribution of one or more divisions of the trigeminal nerve and triggered by innocuous stimuli. It may develop without apparent cause or be a result of another disorder. Additionally, there may or may not be concomitant continuous pain of moderate intensity within the affected division(s).

Previously used terms

Tic douloureux, primary trigeminal neuralgia

Diagnostic criteria

A. Recurrent paroxysms of unilateral facial pain in the distribution(s) of one or more division of the trigeminal nerve, with no radiation beyond¹, and fulfilling criteria B and C

B. The pain has all of the following characteristics:

1. Lasting from a fraction of a second to 2 minutes²
2. Severe intensity³
3. Electric shock-like, shooting, stabbing or sharp in quality

C. Precipitated by innocuous stimuli within the affected trigeminal distribution⁴

D. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Notes

¹In a few patients pain may radiate to another division, but remains within the trigeminal dermatomes

2. Duration can change over time, with paroxysms becoming more prolonged. A minority of patients will report attacks predominantly lasting for >2 minutes.

3. Pain may become more severe over time.

4. Some attacks may be, or appear to be, spontaneous, but there must be a history or finding of pain provoked by innocuous stimuli to meet this criterion. Ideally, the examining clinician should attempt to confirm the history by replicating the triggering phenomenon. However, this may not always be possible because of the patient's refusal, awkward anatomical location of the trigger and/or other factors

Comments

1. Other than the triggering phenomenon, most patients with trigeminal neuralgia fail to show sensory abnormalities within the trigeminal territory unless advanced methods are used (e.g., quantitative sensory testing). However, in some patients, clinical neurological examination may show sensory deficits. These should prompt neuroimaging investigations to explore possible cause. Diagnosis of subforms such as 4.1.1.1 *Classical trigeminal neuralgia*, 4.1.1.2. *Secondary trigeminal neuralgia* or 4.1.1.3. *Idiopathic trigeminal neuralgia* is then possible.

When very severe, the pain often evokes contraction of the muscles of the face on the affected side (tic douloureux). Mild autonomic symptoms such as lacrimation and/ or redness of the ipsilateral eye may be present. Following a painful paroxysm there is usually a refractory period during which pain cannot be triggered.

4.1.1.1 Classical trigeminal neuralgia

Description: Trigeminal neuralgia developing without apparent cause other than neurovascular compression.

Diagnostic criteria

A. Recurrent paroxysms of unilateral pain fulfilling the criteria for 4.1.1 *Trigeminal neuralgia*

B. Demonstration on MRI or during surgery of neurovascular compression (not simply contact), with morphological changes¹ in the trigeminal nerve root

Note'

1. Typically atrophy or displacement.

Comments: Nerve root atrophy and/or displacement due to neurovascular compression are independently associated with signs and symptoms of 4.1.1 *Trigeminal neuralgia*. When these anatomic changes are present, the condition is diagnosed as 4.1.1.1. *Classical trigeminal neuralgia*.

The common site of compression is at the root entry zone with compression by an artery more clearly associated with symptoms than compression by a vein. MRI techniques to measure volume and cross-sectional area of the root are available. Atrophic changes may include demyelination, neuronal loss, changes in microvasculature and other morphological changes. While the exact mechanisms of how atrophic changes in the trigeminal nerve contribute to the generation of pain, some evidence suggests that, when present preoperatively, they predict a good outcome following microvascular decompression.

4.1.1.1.1 Classical trigeminal neuralgia, purely paroxysmal

Description: Classical trigeminal neuralgia without persistent background pain.

Diagnostic criteria

A. Recurrent paroxysms of unilateral facial pain fulfilling criteria for 4.1.1.1 *Classical trigeminal neuralgia*

B. Pain-free between attacks in the affected trigeminal distribution.

Comment: 4.1.1.1.1. *Classical trigeminal neuralgia, purely paroxysmal* is usually responsive, at least initially, to pharmacotherapy (especially carbamazepine or oxcarbazepine).

4.1.1.1.2 Classical trigeminal neuralgia with concomitant continuous pain

Previously used terms: Atypical trigeminal neuralgia; trigeminal neuralgia type 2

Description: Classical trigeminal neuralgia with persistent background pain.

Diagnostic criteria

- A. Recurrent paroxysms of unilateral facial pain fulfilling criteria for 4.1.1.1 *Classical trigeminal neuralgia*
- B. Concomitant continuous or near-continuous pain between attacks in the ipsilateral trigeminal distribution.

Comments

Peripheral or central sensitization may account for the continuous pain.

4.1.1.2 Secondary trigeminal neuralgia

Description: Trigeminal neuralgia caused by an underlying disease. Clinical examination shows sensory changes in a significant percentage of these patients.

Diagnostic criteria

- A. Recurrent paroxysms of unilateral pain fulfilling the criteria for 4.1.1. *Trigeminal neuralgia*, either purely paroxysmal or associated with concomitant continuous or near-continuous pain.
- B. An underlying disease has been demonstrated known that it is able to cause, and explaining, the neuralgia¹
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis²

Notes:

1. Recognized causes are tumour in the cerebello-pontine angle, arteriovenous malformation and multiple sclerosis

2. MRI is best equipped to detect an underlying cause for 4.1.1.2 *Secondary trigeminal neuralgia*. Other investigations may include neurophysiological recording of trigeminal reflexes and trigeminal evoked potentials, suitable for patients who cannot undergo MRI.

4.1.1.2.1 Trigeminal neuralgia attributed to multiple sclerosis

Description: Trigeminal neuralgia caused by a multiple sclerosis (MS) plaque or plaques in the pons or trigeminal root entry zone, and associated with other symptoms and/or clinical or laboratory findings of MS.

Diagnostic criteria

- A. Recurrent paroxysms of unilateral facial pain fulfilling the criteria for 4.1.1 *Trigeminal neuralgia*
- B. Both of the following:
 1. Multiple sclerosis (MS) has been diagnosed
 2. An MS plaque at the trigeminal root entry zone or in the pons affecting the intrapontine primary afferents has been demonstrated by MRI, or its presence is suggested by routine electrophysiological studies¹ showing impairment of the trigeminal pathways.
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Note:

1. Blink reflex or trigeminal evoked potentials.

Comments:

4.1.1.2.1 *Trigeminal neuralgia attributed to multiple sclerosis* occurs in 2–5% of patients with multiple sclerosis (MS), sometimes bilaterally. Conversely, MS is detected in only 2–4% of cases of 4.1.1 *Trigeminal neuralgia*. Symptoms of trigeminal neuralgia are rarely a presenting feature of MS. The lesion in the pons affects the intrapontine central terminals of the trigeminal afferents projecting to the trigeminal brainstem nuclei. Pontine lesions affecting the second order neurones of the trigeminothalamic tract usually lead to non-paroxysmal pain and/or

dysaesthesias and should be classified as *Central neuropathic pain attributed to multiple sclerosis* (13.13.1 in the ICHD-3). Some patients with MS are found to have neurovascular compression of the trigeminal root. It is thought that MS increases the susceptibility of the nerve root to

the effects of compression, leading more readily to painful paroxysms. Patients with 4.1.1.2.1 *Trigeminal neuralgia attributed to multiple sclerosis* benefit less from pharmacological and surgical interventions than those with 4.1.1.1 *Classical trigeminal neuralgia*.

4.1.1.2.2 Trigeminal neuralgia attributed to space-occupying lesion

Description: Trigeminal neuralgia caused by contact between the affected trigeminal nerve and a space-occupying lesion.

Diagnostic criteria

- A. Recurrent paroxysms of unilateral facial pain fulfilling the criteria of 4.1.1 *Trigeminal neuralgia*
- B. Both of the following:
 - 1. A space-occupying lesion in contact with the affected trigeminal nerve has been demonstrated by imaging.
 - 2. Pain has developed after identification of the lesion, or led to its discovery.
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments

Patients with 4.1.1.2.2 *trigeminal neuralgia attributed to space-occupying lesion* may or may not have clinically detectable sensory signs, while electrophysiological tests such as trigeminal brainstem reflexes show abnormalities in nearly all cases.

4.1.1.2.3 Trigeminal neuralgia attributed to other demonstrable causes

Description:

Trigeminal neuralgia caused by an underlying disease other than those described above.

Diagnostic criteria

A. Recurrent paroxysms of unilateral facial pain fulfilling the criteria for 4.1.1 *Trigeminal neuralgia either purely paroxysmal or associated with concomitant continuous or near-continuous pain, but not necessarily unilateral*

B. Both of the following:

1. A disorder, other than those described above, but known to be able to cause trigeminal neuralgia, has been diagnosed¹
2. Pain has developed after onset of the disorder, or led to its discovery

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Note:

Recognized causes are skull-base bone deformity, connective tissue disease, arteriovenous malformation, dural arteriovenous fistula and genetic causes of neuropathy or nerve hyperexcitability.

4.1.1.3 Idiopathic trigeminal neuralgia

Description

Trigeminal neuralgia with neither electrophysiological tests nor MRI showing significant abnormalities.

Diagnostic criteria

A. Recurrent paroxysms of unilateral pain fulfilling the criteria for 4.1.1

Trigeminal neuralgia, either purely paroxysmal or associated with concomitant continuous or near-continuous pain.

B. Neither 4.1.1.1 *Classical trigeminal neuralgia* or 4.1.1.2 *Secondary trigeminal neuralgia* has been confirmed by adequate investigations including electrophysiological tests or MRI.

C. Not better accounted for another ICHD-3 or ICOP diagnosis

Note:

1. A contact between a blood vessel and the trigeminal nerve and/or nerve root is a common finding on neuroimaging in healthy subjects. When such a contact is found in the presence of 4.1.1. Trigeminal neuralgia but without evidence of morphological changes (e.g., atrophy or displacement) in the nerve root, the criteria for 4.1.1.1. *Classical trigeminal neuralgia* are not fulfilled and the condition is considered idiopathic.

4.1.1.3.1 Idiopathic trigeminal neuralgia, purely paroxysmal

Diagnostic criteria

- A. Recurrent paroxysms of unilateral facial pain fulfilling the criteria for 4.1.1.3 *Idiopathic trigeminal neuralgia*
- B. Pain-free between attacks in the affected trigeminal distribution.

4.1.1.3.2 Idiopathic trigeminal neuralgia with concomitant continuous pain

Diagnostic criteria

- A. Recurrent paroxysms of unilateral facial pain fulfilling the criteria for 4.1.1.3 *Idiopathic trigeminal neuralgia*
- B. Concomitant continuous or near-continuous pain between attacks in the affected trigeminal distribution.

4.1.2. *Trigeminal neuropathic pain, other than 4.1.1. Trigeminal neuralgia*

Description

Facial pain in the distribution of one or more branches of the trigeminal nerve caused by another disorder and indicative of neural damage. The primary pain is usually continuous or near-continuous, and commonly described as burning, squeezing, aching or likened to pins and needles. Superimposed brief pain paroxysms may occur but these are not the predominant pain type. This combination is distinct from that of 4.1.1 *Trigeminal neuralgia*. There are clinically detectable somatosensory changes within the trigeminal distribution, and mechanical allodynia and cold hyperalgesia/allodynia are common, fulfilling the IASP criteria for neuropathic pain. Allodynic areas may be much larger than the punctate trigger zones present in trigeminal neuralgia.

Comment

See also 1.1.3.2 *Neuropathic gingival pain*

4.1.2.1. Trigeminal neuropathic pain attributed to herpes zoster

Description

Unilateral facial pain of less than 3 months' duration in the distribution of one or more branches of the trigeminal nerve, caused by and associated with other symptoms and/or clinical signs of acute herpes zoster.

Diagnostic criteria

- A. Unilateral facial pain in the distribution(s) of a trigeminal nerve branch or branches, lasting <3 months
- B. One or more of the following:
 - 1. Herpetic eruption has occurred in of the sametrigeminal distribution
 - 2. Varicella Zoster virus (VZV) has been detected in the cerebrospinal fluid (CSF) by polymerase chain reaction (PCR).
 - 3. Direct immunofluorescence assay for VZV antigen or a PCR assay for VZV DNA is positive in cells obtained from the base of lesions

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comment

Herpes zoster affects the trigeminal ganglion in 10–15% of cases, with the ophthalmic division being singled out in some 80% of patients. Rarely, pain is not followed by an eruption or rash (*zoster sine herpate*). The diagnosis in such cases is confirmed by polymerase chain reaction detection of varicella zoster virus DNA in the cerebrospinal fluid. 4.1.2.1. *Trigeminal neuropathic pain attributed to herpes zoster* is usually burning, stabbing/shooting, tingling or aching, and accompanied by cutaneous allodynia. Ophthalmic herpes may be associated with IIIrd, IVth and VIth cranial nerve palsies. Herpes Zoster is common in immunocompromised patients, occurring in about 10% of those with lymphoma and 25% of patients with Hodgkin's disease.

4.1.2.2. Trigeminal post-herpetic neuralgia

Previously used term: Post-herpetic trigeminal neuropathy

Description

Unilateral facial pain persisting or recurring for at least 3 months in the distribution(s) of one or more branches of the trigeminal nerve, with variable sensory changes, caused by herpes zoster.

Diagnostic criteria

- A. Unilateral facial pain in the distribution(s) of a trigeminal nerve branch or nerve branches, persisting or recurring for >3 months and fulfilling criterion C
- B. Herpes zoster has affected the same trigeminal nerve branch or branches
- C. Pain developed in temporal relation to the acute herpes zoster infection¹
- D. Not better accounted for by another ICOP or ICHD-3 diagnosis

Note:

1. Usually, pain will have developed while the rash was still active, but on occasion later, after rash has healed. In such cases, pale or light purple scars may be present as sequelae of the herpetic eruption.

Comment

Despite its long-preferred name, *post-herpetic neuralgia* is actually a neuropathy or neuronopathy: significant pathoanatomical changes have been shown in the nerve, ganglion and nerve root. In 4.1.2.2. *Trigeminal post-herpetic neuralgia*, there is also evidence of the inflammation extending into the trigeminal brainstem complex.

Following acute herpes zoster, post-herpetic neuralgia is more prevalent in the elderly.

The first division of the trigeminal nerve is most commonly affected in 4.1.2.2. *Trigeminal post-herpetic neuralgia*, but the second and third divisions can be involved also.

Typically, the pain of post-herpetic neuralgia is burning and itching – the latter sometimes very prominent and extremely bothersome. Also, typically, patients with postherpetic neuralgia show a clear sensory deficit and brush-evoked mechanical allodynia in the territory involved. Many patients, however, show little sensory loss and instead demonstrate heightened responses to thermal and/or punctate stimuli.

4.1.2.3 Post-traumatic trigeminal neuropathic pain

Previously used terms

Anaesthesia dolorosa, Painful post-traumatic trigeminal neuropathy

Description

Unilateral or bilateral facial or oral pain following and caused by trauma to the trigeminal nerve(s), with other symptoms and/or clinical signs of trigeminal nerve dysfunction.

For the diagnosis of post-traumatic trigeminal neuropathic pain (PTNP), pain must persist or recur for ≥ 3 months and fulfill all criteria below.

Diagnostic criteria

- A. The pain is characterized by all of the following:
1. History of a mechanical, thermal, radiation or chemical injury with possible peripheral trigeminal nerve involvement¹
 2. Pain onset in close temporal relation to the injury²
 3. Pain distribution neuroanatomically plausible
- B. Pain is associated with somatosensory signs in the same neuroanatomically plausible distribution
- C. Diagnostic test confirming the lesion of a peripheral trigeminal nerve (or nerves) explaining the pain
- D. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes

¹The severity of nerve injuries may range from mild to severe. These include external trauma and iatrogenic injuries from dental treatments such as local anesthetic injections, root canal therapies, extractions, oral surgery, dental implants, orthognathic surgery and other invasive procedures.

²Pain appears no later than 6 months after nerve injury

Comments

The structure of the diagnostic criteria deviates from the ICHD-3 for this particular diagnosis in order to comply with IASP criteria.

Pain duration ranges widely from paroxysmal to constant, and may be mixed. Specifically following radiation-induced postganglionic injury, neuropathic pain may appear after more than 3 months.

Somatosensory signs may be negative (hypoesthesia and/ or hypoalgesia) and/or positive (hyperalgesia and/or allodynia). Note that positive somatosensory signs are not specific to neuropathy. Negative or positive somatosensory signs consistent with the distribution of the pain may be sufficient to indicate the presence of a lesion of the trigeminal nerve. The clinical examination is supplemented by laboratory tests, e.g., quantitative sensory testing.

Tests that reveal a relevant lesion or disease affecting the trigeminal nerve may, e.g., consist of surgical or radiological confirmation of nerve compression or lesion, nerve conduction study, laser-evoked potentials, blink reflex, or skin biopsy confirmation of reduced nerve fiber terminals. Positive findings in these investigations may provide important diagnostic hints at the source of pain. However, all clinical and diagnostic aspects of the pain need to be considered.

There may seem to be a partial overlap to “persistent idiopathic dentoalveolar pain associated with somatosensory changes” 6.3.1. but in this condition there may be no clear temporal relationship and the somatosensory changes may not be limited to a neuroanatomically confined area in contrast to the criteria for “post-traumatic trigeminal neuropathic pain”.

Neuroablative procedures for trigeminal neuralgia, aimed at the trigeminal ganglion or nerve root, may result in neuropathic pain involving one or more trigeminal divisions and should be coded as *4.1.2.3 Post-traumatic trigeminal neuropathic pain*. Such pain may in some cases coexist with trigeminal neuralgia, e.g. when the latter recurs following remission.

4.1.2.3 Post-traumatic trigeminal neuropathic pain rarely, if ever, crosses the midline. Over time, *4.1.2.3 Post-traumatic trigeminal neuropathic pain* may in some cases become more diffusely distributed.

4.1.2.3.1 Probable post-traumatic trigeminal neuropathic pain

Diagnostic criteria

A. Pain fulfilling all but criterion C for *4.1.2.3 post-traumatic trigeminal neuropathic pain*.

4.1.2.4 Trigeminal neuropathic pain attributed to another disorder

Description

Unilateral or bilateral facial or oral pain in the distribution(s) of one or more branches of the trigeminal nerve, caused by a disorder other than those described above, with other symptoms and/or clinical signs of nerve dysfunction.

Diagnostic criteria

For the diagnosis of trigeminal neuropathic pain attributed to another disorder, pain must persist or recur for ≥ 3 months and fulfill all criteria below.

A. The pain is characterized by all of the following:

1. Pain has developed after onset of a disorder known to be capable of causing trigeminal neuropathic pain, or it has led to its discovery
2. Pain distribution neuroanatomically plausible

B. Pain is associated with somatosensory signs in the same neuroanatomically plausible distribution

C. Diagnostic test confirming the diagnosis of the disorder or a lesion of the somatosensory system explaining the pain

D. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Comment

Trigeminal neuropathic pain may develop secondary to multiple sclerosis, space-occupying lesion or systemic disease, with only the clinical characteristics (quality of spontaneous pain, evoked pain and presence of sensory deficits) distinguishing between 4.1.1.2. *Secondary trigeminal neuralgia* and 4.1.2. *Trigeminal neuropathic pain, other than 4.1.1. Trigeminal neuralgia*.

4.1.2. *Trigeminal neuropathic pain, other than 4.1.1. Trigeminal neuralgia* caused by a connective tissue disease or hereditary disorders is usually bilateral but may begin asymmetrically and occasionally present with paroxysmal pain superimposed on the background pain. Patients will eventually develop bilateral sensory deficits and continuous pain, which clarifies the diagnosis. MRI is normal, but trigeminal reflexes are invariably delayed or absent.

4.1.2.4.1 Probable trigeminal neuropathic pain attributed to another disorder

Diagnostic criteria

A. Pain fulfilling all but C for 4.1.2.4 *trigeminal neuropathic pain attributed to another disorder*.

4.1.2.5 Idiopathic trigeminal neuropathic pain

Description

Unilateral or bilateral facial pain in the distribution(s) of one or more branches of the trigeminal nerve indicative of neural damage but of unknown etiology

Diagnostic criteria

For the diagnosis of idiopathic trigeminal neuropathic pain, pain must persist or recur for ≥ 3 months and fulfill all criteria below.

A. The pain is characterized by all of the following:

1. No history of trauma or disorder with possible peripheral trigeminal nerve involvement
2. Pain distribution neuroanatomically plausible

B. Pain is associated with somatosensory signs in the same neuroanatomically plausible distribution

C. Diagnostic test confirming the lesion of a peripheral trigeminal nerve (or nerves) explaining the pain

D. Not better accounted for by another ICOP or ICHD-3 diagnosis.

4.2. Pain caused by a lesion or disease of the glossopharyngeal nerve

4.2.1 Glossopharyngeal neuralgia

Previously used term

Vagoglossopharyngeal neuralgia

Description

A disorder characterised by unilateral brief stabbing pain, abrupt in onset and termination, in the distributions not only of the glossopharyngeal nerve but also of the auricular and pharyngeal branches of the vagus nerve. Pain is experienced in the ear, base of the tongue, tonsillar fossa and/or beneath the angle of the jaw. It is commonly provoked by swallowing, talking or coughing and may remit and relapse in the fashion of trigeminal neuralgia.

Diagnostic criteria

- A. Recurring paroxysmal attacks of unilateral pain in the distribution of the glossopharyngeal nerve¹ and fulfilling criterion B
- B. Pain has all of the following characteristics:
 - 1. Lasting from a few seconds to 2 minutes.
 - 2. Severe intensity
 - 3. Electric shock-like, shooting, stabbing or sharp in quality
 - 4. Pain is precipitated by swallowing, coughing, talking or yawning
- C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Note

- 1. Within the posterior part of the tongue, tonsillar fossa, pharynx or angle of the lower jaw and/or in the ear.

Comments

4.2.1. Glossopharyngeal neuralgia can occur together with *4.1.1. Trigeminal neuralgia*.

The superior laryngeal nerve is a branch of the vagus. Neuralgia of the superior laryngeal nerve presents similarly to *4.2.1. Glossopharyngeal neuralgia* in its location and clinically can be difficult to distinguish from it.

Imaging may show neurovascular compression of the glossopharyngeal nerve.

Prior to development of *4.2.1. Glossopharyngeal neuralgia*, unpleasant sensations may be felt in affected areas for weeks to several months.

The pain in 4.2.1. *Glossopharyngeal neuralgia* may radiate to involve the eye, nose, chin or shoulder. It can be severe enough for patients to lose weight. In rare cases, attacks of pain are associated with vagal symptoms such as cough, hoarseness or syncope and/or bradycardia. Some authors propose distinguishing between a pharyngeal, otalgic and a vagal subforms of neuralgia, and have suggested the term “vagoglossopharyngeal” neuralgia, when pain is accompanied by asystole, convulsions and syncope.

Clinical examination usually fails to show sensory changes in the nerve distribution but if mild sensory deficits are encountered, they do not invalidate the diagnosis. Major changes or a reduced/missing gag reflex should prompt aetiological investigations.

4.2.1. *Glossopharyngeal neuralgia* is usually responsive, at least initially, to pharmacotherapy (especially carbamazepine or oxcarbazepine). It has been suggested that application of local anaesthetic to the tonsil and pharyngeal wall can prevent attacks for a few hours.

4.2.1.1 Classical glossopharyngeal neuralgia

Diagnostic criteria

A. Recurrent paroxysms of unilateral pain fulfilling the criteria for 4.2.1

glossopharyngeal neuralgia

B. Demonstration on MRI or during surgery of neurovascular compression of the glossopharyngeal nerve root

4.2.1.2 Secondary glossopharyngeal neuralgia

Description

Glossopharyngeal neuralgia caused by an underlying disease.

Diagnostic criteria

A. Recurrent paroxysms of unilateral pain fulfilling the criteria for 4.2.1

Glossopharyngeal neuralgia

B. An underlying disease has been demonstrated known to be able to cause, and explaining the neuralgia.¹

Note

1. There are single reports of 4.2.1.2. *secondary glossopharyngeal neuralgia* caused by neck trauma, multiple sclerosis, tonsillar or regional tumours, cerebello-pontine angle tumours, and Arnold-Chiari malformation.

4.2.1.3 Idiopathic glossopharyngeal neuralgia

Diagnostic criteria

A. Recurrent attacks of unilateral pain fulfilling the criteria for 4.2.1

Glossopharyngeal neuralgia

B. Criteria for 4.2.1.1 Classical glossopharyngeal neuralgia or 4.2.1.2

Secondary glossopharyngeal neuralgia are not fulfilled

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

4.2.2. *Glossopharyngeal neuropathic pain*

Description

Pain within the distribution of the glossopharyngeal nerve, (posterior part of the tongue, tonsillar fossa, pharynx or beneath the angle of the lower jaw). In addition, pain is commonly perceived in the ipsilateral ear. The primary pain is usually continuous or near-continuous, and commonly described as burning, squeezing, or likened to pins and needles. Brief paroxysms may happen superimposed but they are not the predominant pain type. This combination distinguishes 4.2.2. *Glossopharyngeal neuropathic pain* from that of 4.2.1 *Glossopharyngeal neuralgia*. Sensory deficits may be present in the ipsilateral posterior part of the tongue and tonsillar fossa, and the gag reflex may be weak or missing.

4.2.2.1. Glossopharyngeal neuropathic pain attributed to a known cause

Description

Unilateral continuous or near-continuous pain, with or without superimposed brief paroxysms, in the distribution of the glossopharyngeal nerve and caused by another identified disorder.

Diagnostic criteria

A. Unilateral continuous or near-continuous pain¹ in the distribution of the glossopharyngeal nerve and fulfilling criterion C

B. A disorder known to be able to cause glossopharyngeal neuropathic pain, has been diagnosed²

C. Evidence of causation demonstrated by both of the following:

1. Pain is ipsilateral to the glossopharyngeal nerve affected by the disorder

2. Pain has developed after onset of the disorder or led to its discovery.

D. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes

1. Brief paroxysms may be superimposed but they are not the predominant pain type
2. Tumours of the cerebellopontine angle and iatrogenic injury during procedures have been reported to cause glossopharyngeal neuropathic pain

4.2.2.2 Idiopathic glossopharyngeal neuropathic pain

Description

Unilateral continuous or near-continuous pain, with or without superimposed brief paroxysms, in the distribution(s) of the glossopharyngeal nerve and of unknown aetiology.

A. Unilateral continuous or near-continuous pain¹ in the distribution of the glossopharyngeal nerve

B. No cause has been identified

C. Not better accounted for by another ICOP or ICHD-3 diagnosis

Note

1. Brief paroxysms may be superimposed but are not the predominant pain type.

For the following, less common clinical entities, where pain mainly presents outside the orofacial region, the reader is referred to the current version of ICHD-3.

4.3. Pain caused by a lesion or disease of nervus intermedius

4.4. Occipital neuralgia

4.5. Neck-tongue syndrome

4.6. Painful optic neuritis

4.7. Headache attributed to ischaemic ocular motor nerve palsy

4.8. Tolosa-Hunt syndrome

4.8. Paratrigeminal oculosympathetic (Raeder's) syndrome

4.9. Recurrent painful ophthalmoplegic neuropathy

4.10. Central neuropathic pain

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5. Orofacial pain resembling presentations of primary headaches

Introduction

In clinical practice we often see three types of patients that seem to typify the crossroads between headache and orofacial pain.

Type 1: Headache patients who report additional facial pain during and usually ipsilateral to the headache attacks.

Type 2: Headache patients whose headache attacks have stopped and have been replaced by facial pain attacks of the same quality, length and severity including the occurrence of associated symptoms of the former headache.

Type 3: Headache naïve patients who develop de-novo facial pain attacks which resemble one of the primary headache types in pain character, duration and severity with or without associated symptoms of such headache types.

This section in the new classification is for patients in the 3rd category. Pain exclusively occurring in the facial region resembling primary headaches but with NO head pain. All others should be coded as the primary headache as per ICHD-3.

5.1. Orofacial migraine

5.1.1 Orofacial migraine

Description

Recurrent orofacial pain attacks lasting 4–72 hours. Typical characteristics of the pain are unilateral location, pulsating quality, moderate or severe intensity, aggravation by routine physical activity and association with nausea and/or photophobia and phonophobia.

Diagnostic criteria

- A. At least five attacks fulfilling criteria B–D
- B. Facial and/or oral pain attacks lasting 4-72 hours (untreated or unsuccessfully treated)
- C. Facial and/or oral pain has at least two of the following four characteristics:
 - 1. Unilateral location
 - 2. Pulsating quality
 - 3. Moderate or severe pain intensity
 - 4. Aggravation by, or causing avoidance of routine physical activity (e.g. walking or climbing stairs)
- D. During facial and/or oral pain at least one of the following:
 - 1. Nausea and/or vomiting
 - 2. Photophobia and phonophobia

E. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comments

Bilateral orofacial migraine has until today not been described. Exclusive orofacial migraine (Type 3 above) seems to be very rare. A group of patients with attacks of intraoral pain of varying length with atypical migraine-like features have been described and may be unrelated to migraine which is why they are described below under neurovascular orofacial pain.

Orofacial migraine with aura has not been extensively described and has been excluded until data accumulates.

5.1.2 Chronic orofacial migraine

Description

Facial and/or oral pain occurring on 15 or more days per month for more than 3 months, which has the features of migraine headache on at least 8 days per month.

Diagnostic criteria

A. Facial and/or oral migraine-like pain on ≥ 15 days per month for >3 months² and fulfilling criteria B and C

B. Occurring in a patient who has had at least five lifetime attacks fulfilling criteria B-D for 1.1 Orofacial Migraine

C. On ≥ 8 days per month for >3 months, fulfilling any of the following³:

1. Criteria C and D for 1.1 Orofacial Migraine

2. Believed by the patient to be migraine at onset and relieved by a triptan or ergot derivative, or beta blocker

D. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Comments

Characterization of frequently recurring orofacial pain generally requires a pain diary to record information on pain and associated symptoms day-by-day for at least 1 month.

References

[1-12]

5.1.3 Neurovascular Orofacial Pain

Various studies have suggested that a specific entity is recognizable that is similar in phenotype to the migraines and to TACs [13-15] . In spite of these similarities it seems to be a separate entity that deserves investigation.

Description

The essential features are attacks of various length of severe intraoral pain, often accompanied by 'toothache' like symptoms, with mild autonomic and/or migrainous symptomatology. Within this group there are patients with relatively short attacks (1-4 hours) and those with longer attacks (> 4 hours).

5.1.3.1 Shortlasting Neurovascular Orofacial Pain

Diagnostic Criteria

- A. At least 5 attacks of facial pain fulfilling criteria B-E
- B. Moderate to Severe, intraoral pain¹
- C. At least one of the following characteristics:
 - 1. Toothache with no local pathology²
 - 2. Throbbing pain
- D. Episodic pain lasting 1 to 4 hours
- E. Clinical and radiographic examination are normal and no local cause may explain the pain
- F. Accompanied by at least one of the following:
 - 1. ipsilateral lacrimation and/or conjunctival injection
 - 2. ipsilateral rhinorrhea and/or nasal congestion
 - 3. ipsilateral cheek swelling

4. photo and/or phonophobia

5. nausea and/or vomiting

G. Not better accounted^d for by another ICOP or ICHD-3 diagnosis

Comments:

Although existing in the literature since 1997 this entity needs thorough and prospective examination. Although essentially intraoral pain there may be referral to adjacent sites particularly when pain is severe. This phenomenon needs to be carefully followed and documented. There are reports of abnormal sensitivity to cold both interictally and during attacks. This finding needs to be investigated thoroughly as it would be a useful test and may link the entity to migraine where mechanical allodynia occurs during attacks. To obtain all the needed parameters above the use of pain diaries is essential.

5.1.3.2 Longlasting Neurovascular Orofacial Pain

Diagnostic Criteria

A. At least 5 attacks of facial pain fulfilling criteria B-E

B. Moderate to Severe, intraoral pain¹

C. At least one of the following characteristics:

1. Toothache with no local pathology²

2. Throbbing pain

D. Clinical and radiographic examination are normal and no local cause may explain the pain

E. Episodic pain lasting > 4 hours

F. Accompanied by at least one of the following:

1. ipsilateral lacrimation and/or conjunctival injection

2. ipsilateral rhinorrhea and/or nasal congestion

3. ipsilateral cheek swelling

4. photo and/or phonophobia

5. nausea and/or vomiting

G. Not better accounted for by another ICOP or ICHD-3 diagnosis

[13-15]

Comments:

Although existing in the literature since 1997 this entity needs thorough and prospective examination. Although essentially an intraoral pain there may be referral to adjacent sites particularly when pain is severe. This phenomenon needs to be carefully followed and documented. There are reports of abnormal sensitivity to cold both interictally and during attacks. This finding needs to be investigated thoroughly as it would be a useful test and may link the entity to migraine where mechanical allodynia occurs during attacks. To obtain all the needed parameters above the use of pain diaries is essential.

5.2 Tension-type orofacial pain

Comments

There are great similarities in signs, symptoms epidemiology and treatment response between TTH here and myofascial pain in section 2 of ICOP. At this point, there is insufficient evidence to establish any type of relationship between them

There may exist a facial pain that is unrelated to TMD and is described as “facial muscle tension” only occurring during rest, which resolves with voluntary muscle activity, e.g. mastication. At this time there is insufficient evidence that such symptoms form a separate group.

5.3 Trigeminal autonomic orofacial pain

5.3.1 Orofacial cluster attacks

Description

Attacks of severe, strictly unilateral facial and/or oral pain, lasting 15–180 minutes and occurring from once every other day to eight times a day. The pain is associated with ipsilateral conjunctival injection, lacrimation, nasal congestion, rhinorrhoea, forehead and facial sweating, miosis, ptosis and/or eyelid oedema, and/or with restlessness or agitation. To reiterate the general approach of this classification: The pain attacks are exclusively in the facial area and not in the head. If headache occurs, these syndromes should be coded “cluster headache” with facial component).

Diagnostic criteria

- A. At least five attacks fulfilling criteria B–D
- B. Severe or very severe unilateral facial and/or oral pain lasting 15–180 minutes (when untreated)
- C. Either or both of the following:
 - 1. At least one of the following symptoms or signs, ipsilateral to the headache:
 - a. Conjunctival injection and/or lacrimation
 - b. Nasal congestion and/or rhinorrhoea
 - c. Eyelid oedema
 - d. Forehead and facial sweating
 - e. Forehead and facial flushing
 - f. Sensation of fullness in the ear
 - g. Miosis and/or ptosis
 - 2. A sense of restlessness or agitation
- D. Attacks have a frequency between one every other day and eight per day for more than half of the time when the disorder is active
- E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Comment

Autonomic symptoms in facial cluster headach might be alleviated or different from autonomic symptoms associated with headache. At this time there is insufficient evidence that this is indeed the case and further research is needed. A group of patients with facial and/or oral pain with atypical cluster-like features have been described. At this time there is insufficient evidence that these form a separate group.

5.3.1.1 Episodic orofacial cluster attacks

Description

Orofacial Cluster attacks occurring in periods lasting from 7 days to 1 year, separated by pain-free periods lasting at least 3 month.

Diagnostic criteria

- A. Attacks fulfilling criteria for 5.3.1 Orofacial cluster attacks and occurring in bouts (cluster periods)
- B. At least two cluster periods lasting from 7 days to 1 year (when untreated) and separated by pain-free remission periods of 3 month.

5.3.1.2 Chronic orofacial cluster attacks

Description

Unilateral facial and/or oral pain occurring for more than 1 year without remission, or with remission periods lasting less than 3 month.

Diagnostic criteria

- A. Attacks fulfilling criteria for 5.3.1.1 Orofacial cluster attacks, and criterion B below
- B. Occurring without a remission period, or with remissions lasting <1 month, for at least 1 year.

Comment

5.3.1.2 Chronic orofacial cluster attacks may arise de novo (previously referred to as primary chronic cluster headache), or evolve from 5.3.1.1 Episodic orofacial cluster attack (previously secondary chronic cluster headache). In some patients change occurs from 5.3.1.2 Chronic orofacial cluster attacks to 5.3.1.1 Episodic orofacial cluster attack.

References

[13, 14, 16-25]

5.3.2 Paroxysmal hemifacial pain

Description

Attacks of severe, strictly hemifacial pain which, lasting 2–30 minutes and occurring several or many times a day. The attacks may be associated with ipsilateral conjunctival injection, lacrimation, nasal congestion, rhinorrhoea, forehead and facial sweating, miosis, ptosis and/or eyelid oedema.

Diagnostic criteria

- A. At least 20 attacks fulfilling criteria B-E
- B. Severe unilateral facial and/or oral pain lasting 2–30 minutes
- C. Either or both of the following:
 - 1. At least one of the following symptoms or signs, ipsilateral to the pain:
 - a. Conjunctival injection and/or lacrimation
 - b. Nasal congestion and/or rhinorrhoea
 - c. Eyelid oedema
 - d. Forehead and facial sweating
 - e. Forehead and facial flushing
 - f. Sensation of fullness in the ear
 - g. Miosis and/or ptosis

2. Attacks are prevented absolutely by therapeutic doses of indomethacin.

D. Attacks have a frequency above five per day for more than half of the time.

E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Comments

There are reports of paroxysmal hemifacial pain without prominent autonomic signs. In general, the number and quality of autonomic symptoms may differ between 1st and 2nd or 3rd trigeminal pain [26, 27]. Further data is needed to determine if these are a separate group.

The ‘absolute’ response to indomethacin in paroxysmal hemifacial pain is as yet unestablished.

5.3.2.1 Episodic paroxysmal hemifacial pain

Description

Attacks of paroxysmal hemifacial pain occurring in periods lasting from 7 days to 1 year, separated by pain-free periods lasting at least 3 months.

Diagnostic criteria

A. Attacks fulfilling criteria for 5.3.2 Paroxysmal hemifacial pain and occurring in bouts

B. At least two bouts lasting from 7 days to 1 year (when untreated) and separated by pain-free remission periods of at least 3 months.

5.3.2.2 Chronic paroxysmal hemifacial pain

Description

Attacks of paroxysmal hemifacial pain occurring for more than 1 year without remission, or with remission periods lasting less than 3 months.

Diagnostic criteria

A. Attacks fulfilling criteria for 5.3.2 Paroxysmal hemifacial pain, and criterion B below

B. Occurring without a remission period, or with remissions lasting <3 month, for at least 1 year.

References

[13, 14, 16-24, 28-32]

5.3.3 Short-lasting unilateral neuralgiform facial pain attacks with autonomic signs (SUNFA)

Description

Attacks of moderate or severe, strictly unilateral oral and/or facial pain lasting seconds to minutes, occurring at least once a day and usually associated with prominent lacrimation and redness of the ipsilateral eye or with other local autonomic signs as below.

Diagnostic criteria

- A. At least 20 attacks fulfilling criteria B–E
- B. Moderate or severe unilateral facial and/or oral pain, lasting for 1–600 seconds and occurring as single stabs, series of stabs or in a saw tooth pattern
- C. At least one of the following cranial autonomic symptoms or signs, ipsilateral to the pain:
 - 1. Conjunctival injection and/or lacrimation
 - 2. Nasal congestion and/or rhinorrhoea
 - 3. Eyelid oedema
 - 4. Forehead and facial sweating
 - 5. Forehead and facial flushing
 - 6. Sensation of fullness in the ear
 - 7. Miosis and/or ptosis
- D. Attacks have a frequency of at least one a day for more than half of the time when the disorder is active.
- E. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comment

The occurrence and distribution of autonomic signs in SUNFA is unclear and needs to be studied. In SUNCT pain and accompanying autonomic signs may occur throughout the trigeminal region and the location of autonomic symptoms may indeed be related to pain location [33].

5.3.3.1 Episodic SUNFA

Description

Attacks of SUNFA occurring in periods lasting from 7 days to 1 year, separated by pain-free periods lasting at least 3 months.

Diagnostic criteria

- A. Attacks fulfilling criteria for 5.3.3 Short-lasting unilateral neuralgiform facial pain attacks with autonomic signs and occurring in bouts
- B. At least two bouts lasting from 7 days to 1 year and separated by pain-free remission periods of 3 months.

5.3.3.2. Chronic SUNFA

Description

Attacks of SUNFA occurring for more than 1 year without remission, or with remission periods lasting less than 3 months.

Diagnostic criteria

- A. Attacks fulfilling criteria for 5.3.3 Short-lasting unilateral neuralgiform facial pain attacks with autonomic signs, and criterion B below
- B. Occurring without a remission period, or with remissions lasting <3 months, for at least 1 year

References

[33-36]

5.3.4 Hemifacial continuous pain with autonomic signs

Exacerbations of pain in hemicrania continua into the face have been described (Hryvenko et al, in press). As yet, no isolated facial equivalent of hemicrania continua has been clearly established. Hemicrania continua has been described with a paucity of autonomic signs and may have a representative within the group of PIFP syndromes. Pain referral to oral and/or facial structures may cause diagnostic difficulties.

References

[37-42]

5.3.5 *Constant Unilateral Facial Pain with additional Attacks (CUFPA)*

Description:

Constant dull unilateral pain of the maxilla and/or mandible of mild to moderate intensity, accompanied by distinct attacks of moderate to severe pain in the same location lasting 10 to 30 minutes. There are no typical autonomic and/or migrainoid features accompanying neither with constant pain nor with pain attacks.

Diagnostic criteria

- A. Facial pain fulfilling criteria B and C
- B. Strictly unilateral pain of mild to moderate intensity recurring daily for >2 hours per day for >3 months
- C. ≥ 2 additional distinct attacks per day of moderate to severe pain exacerbations in the same location lasting 10 to 30 minutes
- D. Clinical and radiographic examination are normal and no local cause may explain the pain
- E. Not better accounted for by another ICOP or ICHD-3 diagnosis

Comment

Autonomic symptoms should be absent, but do not exclude CUFGA. A response to Indomethacin should rather lead to the diagnosis of 5.3.2 Paroxysmal hemifacial pain. At the moment there are not enough data available to discuss episodic and chronic types.

The additional attacks have to be clearly distinct from the constant pain and patients should describe the pain consisting of two components, otherwise the diagnosis of 5.3.2 *Paroxysmal hemifacial pain* or 6.2 *Persistent idiopathic facial pain* (PIFP) should be considered.

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6. Idiopathic orofacial pain

6.1 Burning mouth syndrome (BMS)

Previously used terms

Stomatodynia, or glossodynia when confined to the tongue. Primary burning mouth syndrome.

Description

An intraoral burning or dysaesthetic sensation, recurring daily for more than 2 hours per day over more than 3 months, without evident causative lesions on clinical examination and investigation.

Diagnostic criteria

- A. Oral pain fulfilling criteria B and C
- B. Recurring daily for >2 hours per day for >3 months¹
- C. Pain has both of the following characteristics:
 - 1. Burning quality
 - 2. Felt superficially in the oral mucosa
- D. Oral mucosa is of normal appearance and no local or systemic causes may explain the pain.²
- E. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes

¹It is possible to diagnose burning mouth syndrome prior to 3 months if all exclusions for secondary burning mouth symptoms have been made.

²Quantitative sensory testing is often abnormal, whereas clinical sensory examination very rarely reveals slight sensory deficits.

6.1.1 Burning mouth syndrome associated with somatosensory changes

Diagnostic criteria

- A-C. Oral pain fulfilling criteria for 6.1 for burning mouth syndrome

D. Both of the following

1. Oral mucosa is of normal appearance and no local or systemic causes may explain the pain
2. Somatosensory changes are present on qualitative or quantitative somatosensory testing¹.

Notes

¹Negative or positive sensory signs.

6.1.2 Burning mouth syndrome not associated with somatosensory changes

Diagnostic criteria

A-C. Oral pain fulfilling criteria for 6.1 for burning mouth syndrome

D. Both of the following

1. Oral mucosa is of normal appearance and no local or systemic causes may explain the pain
2. Somatosensory changes are not present on qualitative or quantitative somatosensory testing.

Comments

The pain of 6.1 burning mouth syndrome (BMS) is usually bilateral, but can on rare occasion be unilateral, and its intensity fluctuates. The most common site is the tip of the tongue. Subjective xerostomia (~2/3 of cohort), dysaesthesia and altered taste (~2/3 of cohort) are often present.

There is a high preponderance in menopausal women, and some studies show psychosocial comorbidities similar to other persistent pain conditions. Recent data point to varying levels of changes in somatosensory function in BMS patients. These findings encourage further research into BMS as a possible neuropathic pain condition.

Burning mouth **symptoms** may occur as a secondary phenomenon attributed to a local condition such as candidiasis, lichen planus, hyposalivation and contact mucosal reactivity. It has also been attributed to systemic disorders such as medication induced, anaemia, deficiencies of vitamin B12 or folic acid, Sjögren's syndrome, diabetes. Oral burning related to these conditions have previously been known as "secondary burning mouth syndrome", but burning mouth syndrome (6.1) here only refers to burning symptoms that have had all local and systemic causes excluded (previously "primary burning mouth syndrome"). The diagnosis secondary BMS falls within the domain of oral mucosal pain with a known local or systematic cause covered in section 1.2.

6.2 Persistent idiopathic facial pain (PIFP)

Previously used term

Atypical facial pain.

Description

Persistent facial pain, with varying presentations but recurring daily for more than 2 hours per day, over more than 3 months, without neurological deficits in clinical examination or close temporal preceding event.

Diagnostic criteria

- A. Facial pain fulfilling criteria B and C
- B. Recurring daily for >2 hours per day for >3 months
- C. Pain has both of the following characteristics:
 - 1. Poorly localized, and not following the distribution of a peripheral nerve¹
 - 2. Dull, aching or nagging quality²
- D. Clinical and radiographic examination are normal and no local cause may explain the pain
- E. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes

¹Pain may be described as either deep or superficial and may radiate from face to mouth or vice versa. With time, it may spread to a wider area of the craniocervical region.

²A wide variety of words are used to describe the character and the pain can have exacerbations, and be aggravated by stress.

³Clinical neurological somatosensory assessment with pin prick or light touch perception may very rarely reveal slight somatosensory changes. Nociceptive pain reflecting altered processing in the somatosensory system may be present and related to alteration in the modulatory pain inhibitory system.

6.2.1 Persistent idiopathic facial pain associated with somatosensory changes

Diagnostic criteria

A-C. Facial pain fulfilling criteria for 6.2 for persistent idiopathic facial pain

D. Both of the following:

1. Clinical and radiographic examinations are normal and no local and/or distant cause may explain the pain.
2. Somatosensory changes are present on qualitative or quantitative somatosensory testing¹.

Notes

¹Negative or positive sensory signs.

6.2.2 Persistent idiopathic facial pain not associated with somatosensory changes

Diagnostic criteria

A-C. Oral pain fulfilling criteria for 6.2 for persistent idiopathic facial pain

D. Both of the following:

1. Clinical and radiographic examinations are normal and no local and/or distant cause may explain the pain.
2. Somatosensory changes are not present on qualitative or quantitative somatosensory testing.

Comments

In the previous version of ICHD-3 two disorders were treated as one entity: Persistent idiopathic facial pain and atypical odontalgia. These new criteria give an overarching categorization of persistent idiopathic facial pain and define two distinct entities: Persistent idiopathic facial pain and persistent idiopathic dentoalveolar pain. These two conditions cause either facial or dentoalveolar pain of a fairly constant nature that may be prone to exacerbations.

Patients with *Persistent idiopathic facial pain* may report a minor operation or injury to the face, maxillae, teeth or gums but upon clinical and radiographic examination there is no demonstrable local cause. *Persistent idiopathic facial pain* may be comorbid with other pain conditions such as chronic widespread pain and irritable bowel syndrome. In addition, it can present with psychosocial comorbidities similar to other persistent pain conditions.

6.3 Persistent idiopathic dentoalveolar pain

Previously used term

Atypical odontalgia, Primary PDAP, Phantom tooth pain

Description

Unilateral, or rarely multiple sites of intra-oral dentoalveolar pain with varying presentations but recurring daily for more than 2 hours per day, over more than 3 months without close temporal preceding event.

Diagnostic criteria

- A. Unilateral, or rarely multiple sites of intra-oral dentoalveolar pain fulfilling criterion B and C
- B. Recurring daily for >2 hours per day for >3 months

C. Pain has both of the following characteristics:

1. Localized to dentoalveolar site or sites (tooth or alveolar bone)¹
2. Deep, dull, pressure-like quality²

D. Clinical and radiographic examination are normal and no local cause may explain the pain³.

E. Not better accounted for by another ICOP or ICHD-3 diagnosis

Notes

¹Pain may be described as either deep or superficial. With time, it may spread to a wider area of the craniocervical region.

²A wide variety of words are used to describe the character and the pain can have exacerbations, and be aggravated by stress. Adjunctive symptom description may also be used.

³Clinical neurological somatosensory assessment with pin prick or light touch perception only very rarely reveals sensory abnormalities. Nociceptive pain reflecting altered processing in the somatosensory system may be present and related to alteration in the modulatory pain inhibitory system.

Comment

See also *1.1.3.3 Idiopathic gingival pain*

6.3.1 Persistent idiopathic dentoalveolar pain associated with somatosensory changes

Diagnostic criteria

A-C. Oral pain fulfilling criteria for 6.3 for persistent idiopathic dentoalveolar pain

D. Both of the following:

1. Clinical and radiographic examinations are normal and no local and/or distant cause may explain the pain.

2. Somatosensory changes are present on qualitative or quantitative somatosensory testing¹.

Notes

¹Negative or positive sensory signs present but not spatially confined to a neuroanatomical relevant area in contrast to post-traumatic trigeminal neuropathic pain 4.1.2.3.

6.3.2 Persistent idiopathic dentoalveolar pain not associated with somatosensory changes

Diagnostic criteria

A-C. Oral pain fulfilling criteria for 6.3 for persistent idiopathic dentoalveolar pain

D. Both of the following:

1. Clinical and radiographic examinations are normal and no local and/or distant cause may explain the pain.
2. Somatosensory changes are not present on qualitative or quantitative somatosensory testing.

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7. Psychosocial Assessment¹

Introduction

The biopsychosocial model incorporates psychological and social factors in order to more comprehensively understand and manage both disease (as related to the traditional medical factors) and illness across time and circumstance. Major psychological factors associated with pain disorders include anxiety, catastrophizing, depression, physical symptom reporting, and fear-avoidance; and major social factors include access to medical care, stigma, and support from family and friends. Each of these factors has extensive empirical support for their association with pain disorders, and evidence clearly supports the significance of the biopsychosocial model as critical for understanding the complexity of pain processing in general [4] as well as related to orofacial pain disorders.[6; 21; 22; 31; 39] Notably, the implementation of the biopsychosocial model into both research and clinical pain medicine remains variable; further detail is available,[10; 35; 36] and new taxonomies for chronic pain of all types clearly highlight the central importance of both physical criteria for the disorders as well as assessment of psychosocial factors.[16]

For present purposes, recommendations for best research practices in support of the intent of the ICOP taxonomy are presented for the orofacial pain field broadly and follow previously established recommendations for the RDC/TMD [14] and the DC/TMD [47; 48] which specify appropriate constructs and instruments for the assessment of musculoskeletal pain (e.g., painful TMDs). While these recommendations emerge from substantial research on the TMDs, a subset of orofacial pains, no evidence exists at this time to suggest that pain from the non-TMD orofacial pain conditions is any different from the pain associated with the TMDs in terms of

pain processing models. Consequently, research at this stage of development of ICOP should include equivalent attention to the full biopsychosocial model and thereby include assessment of recommended psychosocial constructs. Subsequent structured and systematic evidence will permit a more empirically supported assessment model for the non-TMD OFPs and lead to revision of these initial recommendations.

Levels of psychosocial assessment

Two levels of psychosocial assessment are defined by the DC/TMD [48] and one more was developed in response to specific clinical request. See Table 1 for summary. The brief screening version is intended for research (and clinical) settings where only the briefest biopsychosocial

Instrument	# items	Comprehensive assessment	Standard screening	Brief screening
Pain drawing	1	4	4	4
GCPS v2.0	8	4	4	4
JFLS (long form)	20	4		
JFLS (short form)	8		4	
PHQ-4	5*		4	4
PHQ-9	10*	4		
GAD-7	8*	4		
PHQ-15	15	4		
OBC	20	4	4	

assessment using the fewest number of questions can be incorporated.[42] Interestingly, the same components of this brief screening have been informally described by other colleagues, suggesting a convergence into a core minimal set of psychosocial assessment domains. The

standard screening version incorporates two more instruments. Both forms of screening should be recognized as very limited.

Table 1. Different levels of psychosocial assessment. See text for details

*Table Comments: * Item count includes reflective question regarding functional impact of any reported symptoms.*

GCPS: Graded Chronic Pain Scale; JFLS: Jaw Functional Limitation Scale; PHQ: Patient Health Questionnaire; GAD: Generalized Anxiety Disorder; OBC: Oral Behaviors Checklist

The comprehensive assessment is intended specifically for clinical researchers so that they can more reliably measure all constructs of interest, thereby permitting full stratification of their samples based on a psychosocial profile. All of these instruments are freely available with interpretation guides for the scoring at the following website: www.rdc-tmdinternational.org. In addition, a few other instruments are recommended below for consideration.

Pain- and function-related constructs and instruments for OFP

Extent of pain

The pain drawing (also known as a “body manikin”) provides ready identification of all pain location(s), known to be one major risk determinant for pain chronicity.[37] All pain disorders, regardless of putative nociceptive mechanism, appear to be similarly affected in terms of the extent of pain.

Pain intensity and pain-related disability

The Graded Chronic Pain Scale (GCPS, v 2.0) is a widely used and validated instrument examining pain persistence, pain intensity and pain-related disability also called graded chronic pain status, which has utility to stratify patients for levels of care.[12; 13; 15] Graded chronic pain status is also an indicator of prognosis in that higher graded chronic pain status predicts greater chance of pain chronicity.[55]

Functional limitation

The person's experience of impaired functional ability is known as functional limitation.[33] The Jaw Functional Limitation Scale (JFLS) has 2 versions, an 8-item version which yields a global score; and a 20-item version which measures three domains: limitation in chewing, jaw opening, and verbal and emotional expression.[38; 40] Both versions are equally reliable, valid, and sensitive to change. While functional limitation is central to musculoskeletal pain (and thereby self-evident for relevance to TMD), functional consequences are assumed to occur in response to the non-TMD OFPs; the nature of those consequences is suspected [40] and warrants further investigation in order to be understand the full dimensionality of pain in the orofacial region.

Over-use behaviours

The Oral Behaviors Checklist (OBC) contains a list of 21 oral region activities individuals may engage in, such as clenching the teeth, bracing the mandible, or talking. Psychometric properties are strong [24; 29; 41] and OBC values are associated with TMD.[5; 18-20; 34; 37] Whether these behaviours are specifically associated with the OFPs remains unknown; however, guarding behaviours are known to affect non-musculoskeletal back pain,[32] suggesting applicability for non-TMD OFPs.

Psychosocial constructs and instruments for OFP

Depression and anxiety

The PRIME-MD project (**PRIM**ary care **E**valuation of **M**ental **D**isorders) [50] was anchored initially in psychiatric disorders and aimed to develop psychosocial instruments for the assessment of five of the most common mental health problems presenting to primary care: anxiety, depression, somatoform, alcohol, and eating disorders.[27] Particularly relevant for pain disorders are the 9-item Patient Health Questionnaire (PHQ-9) for depression and 7-item Generalized Anxiety Disorder scale (GAD-7) for anxiety; each of these instruments will permit reliable and valid measurement of the respective core constructs. Each instrument contributes 2 questions to create the brief PHQ-4 depression and anxiety screening instrument, often considered to assess "distress" and which is widely used across North America and Europe. Depression (measured with PHQ-9, PHQ-4) is a mood state known to be affected by the

presence of persistent pain and known to affect pain processing, and it appears to be highly relevant to OFP.[11] Anxiety (measured with GAD-7, PHQ-4) in a medical context often manifests as worry and general sympathetic nervous system activation, and it is associated with pain perception [46] and hypervigilance.[7] Anxiety pervades medical settings, and it appears to be highly relevant to OFP.[1]

Somatoform disorders

The PRIME-MD project also yielded the Patient Health Questionnaire-15 (PHQ-15) for somatic symptom severity. Physical symptoms not accompanied by appropriate signs supportive of a disease diagnosis remain a considerable challenge across all medical domains; such findings are appropriately termed somatic symptom disorder, functional disorders, medically unexplained symptoms, and symptoms without medical utility,[25; 45] yet none of these terms are fully satisfactory for what appears to be a more complex construct than previously assumed. While there are several proposed mechanisms underlying physical symptom reporting,[8; 9; 45] all are consistent with the various symptoms that accompany the persistent OFPs.[1; 43] An extension of this phenomenon involves occlusal dysesthesia,[30] which has potential relevance to at least a few of the OFPs within the ICOP.

Catastrophizing

Catastrophizing about pain is “characterized by the tendency to magnify the threat value of pain stimulus and to feel helpless in the context of pain, and by a relative inability to inhibit pain-related thoughts in anticipation of, during or following a painful encounter” [44]. Higher levels of catastrophizing are linked to increased utilization of healthcare, increased expression of pain, and poorer treatment outcomes. [3; 28; 52] Catastrophizing is not included as a standard measure within the DC/TMD framework because the evidence for the relevance of catastrophizing to TMD was not sufficiently strong when the DC/TMD Axis II recommendations were formulated. The situation has since changed and, moreover, its relevance to any pain disorder is now appropriate for this construct to be included as a recommended domain within the ICOP. Appropriate validated measures include the Pain Catastrophizing Scale [51] and the Coping Strategies Questionnaire.[23]

Fear avoidance

The fear-avoidance model emerged from operant models pertaining to low back pain,[17] specifically, behavioural observations that individuals reported pain incongruent with physical findings. The fear-avoidance model has since had extensive supporting research.[54] In the model, having no fear of injury-related new onset pain leads to engaging in the appropriate behaviours that will result in recovery from the injury. In contrast, having fear of that pain leads to catastrophizing about that pain, avoidance of circumstances that may cause pain, and consequent disuse, depression, and disability. Disability then feeds forward to further pain experience, avoidant behaviour, and the absence of recovery; as such this model is clearly relevant to motor behaviour, and support of this construct for TMD is slowly emerging. The model, however, is a person-level model with regard to the effects of behaviour and beliefs on the central nervous system and thereby assumed to be linked to pain processing. Consequently, the clearly plausible hypotheses relating fear of movement to recovery amongst those with injury to the masticatory system and the probable emergence of chronic pain among some of those individuals warrant investigation,[56] and present data suggest that this perspective is applicable to OFP as well.

Fear of pain is measured via several instruments, of which the Tampa Scale for Kinesiophobia (TSK) [26] is the best known and has very strong utility for back pain.[2] The TSK was adapted for the masticatory system, the TSK-TMD,[53] and it appears to capture both the somatic experience as well as avoidant behaviours that OFP may precipitate.

Conclusions and Future Directions

Further research of the biopsychosocial model and its clinical as well as research relevance to OFP is clearly needed. As the criteria for the disorders within the ICOP are better developed and refined, a similar progression should occur regarding our understanding of the persons with these pain disorders. Consequently, as taxonomy and diagnosis improve, improved understanding of pain mechanisms as well as sound treatment recommendations should emerge. Multi-modal approaches are clearly needed to augment the potential therapeutic yield of standard biomedical therapies such as pharmacological or surgical approaches.[49] For the present time, consistent use of a standardized format for psychosocial assessment of the individual with orofacial pain is

strongly recommended in parallel with use of the stated criteria (and their extensions for research purposes) of the disorders within the ICOP.

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Definitions of terms

Attributed to: This term in ICOP is in accordance with ICHD-3 and describes the relationship between a secondary pain and the disorder believed to cause it. It requires fulfilment of criteria establishing an accepted level of evidence of causation.

Chronic: In pain terminology, chronic signifies long-lasting, specifically over a period exceeding 3 months. In headache terminology, For primary headache disorders that are more usually episodic (qv), chronic is used whenever attacks of headache (qv) occur on more days than not over a period longer than 3 months. The trigeminal autonomic cephalalgias are the exception: in these disorders, chronic is not used until the disorder has been unremitting for more than one year with less than 3 months attack free. Results from research based on ICOP will allow us to establish how applicable these criteria are to orofacial pain (see also Benoliel et al 2010)

Duration of attack: Time from onset until termination of an attack of headache (or pain) (qv) meeting criteria for a particular headache type or subtype. After migraine or cluster headache, a low-grade non-pulsating headache without accompanying symptoms may persist, but this is not part of the attack and is not included in duration. If the patient falls asleep during an attack and wakes up relieved, duration is until time of awakening. If an attack of migraine is successfully relieved by medication but symptoms recur within 48 hours, these may represent a relapse of the same attack or a new attack. Judgement is required to make the distinction (see also Frequency of attacks).

Episodic: Recurring and remitting in a regular or irregular pattern of attacks of headache (or pain) (qv) of constant or variable duration. Through long usage the term has acquired special meaning in the context of episodic cluster headache, referring to the occurrence of cluster periods (qv) separated by cluster remission periods (qv) rather than to attacks. Similar usage has been adopted for paroxysmal hemicrania and short-lasting unilateral neuralgiform headache attacks.

Facial pain: Pain below the orbitomeatal line, anterior to the pinnae and above the neck.

Focal neurological symptoms: Either negative signs of one or more cranial nerves, or central symptoms of focal brain (usually cerebral) disturbance such as occur in migraine aura (qv).

Frequency of attacks: The rate of occurrence of attacks of headache (or pain) (qv) per time period (commonly one month). Successful relief of a migraine attack with medication may be followed by relapse within 48 hours. The IHS Guidelines for Controlled Trials of Drugs in

Migraine, 3rd edition, recommend as a practical solution, especially in differentiating attacks recorded as diary entries over the previous month, to count as distinct attacks only those that are separated by at least 48 hours headache-free.

Headache: Pain (qv) located in the head, above the orbitomeatal line and/or nuchal ridge.

Headache days: Number of days during an observed period of time (commonly one month) affected by headache for any part or the whole of the day.

Intensity of pain: Level of pain may be scored on a four-point numerical rating scale (0-3) equivalent to no, mild, moderate and severe pain, or on a visual analogue scale (commonly 10 cm). It may also be scored on a verbal rating scale expressed either on a scale from 0-10 or in terms of its functional consequence: 0, no pain; 1, mild pain, does not interfere with usual activities; 2, moderate pain, inhibits but does not wholly prevent usual activities; 3, severe pain, prevents all activities.

Persistent: This term, used in the context of certain secondary headaches, describes headache, initially acute and caused by another disorder, that fails to remit within a specified time interval (usually 3 months) after that disorder has resolved. In many such cases, the headache is recognized as a distinct subtype or subform, with evidence of causation depending upon earlier fulfilment of the criteria for diagnosis of the acute type, and persistence of the same headache.

Phonophobia: Hypersensitivity to sound, even at normal levels, usually causing avoidance.

Photophobia: Hypersensitivity to light, even at normal levels, usually causing avoidance.

Primary pain (disorder): Pain (OFP or headache) not caused by or attributed to another disorder. It is distinguished from secondary OFP and headache disorders.

Refractory period: The time following resolution of an attack of pain (qv) during which a further attack cannot be triggered.

Sidelocked: Unilateral occurrence of pain that never changes sides.

Unilateral: On either the right or the left side, not crossing the mid line. Unilateral headache does not necessarily involve all of the right or left side of the head, but may be frontal, temporal or occipital only. When used for sensory or motor disturbances of migraine aura, the term includes complete or partial hemidistribution.

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Pain Terminology

Pain

An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.

Note: The inability to communicate verbally does not negate the possibility that an individual is experiencing pain and is in need of appropriate pain-relieving treatment. Pain is always subjective. Each individual learns the application of the word through experiences related to injury in early life. Biologists recognize that those stimuli which cause pain are liable to damage tissue. Accordingly, pain is that experience we associate with actual or potential tissue damage. It is unquestionably a sensation in a part or parts of the body, but it is also always unpleasant and therefore also an emotional experience. Experiences which resemble pain but are not unpleasant, e.g., pricking, should not be called pain. Unpleasant abnormal experiences (dysesthesias) may also be pain but are not necessarily so because, subjectively, they may not have the usual sensory qualities of pain. Many people report pain in the absence of tissue damage or any likely pathophysiological cause; usually this happens for psychological reasons. There is usually no way to distinguish their experience from that due to tissue damage if we take the subjective report. If they regard their experience as pain, and if they report it in the same ways as pain caused by tissue damage, it should be accepted as pain. This definition avoids tying pain to the stimulus. Activity induced in the nociceptor and nociceptive pathways by a noxious stimulus is not pain, which is always a psychological state, even though we may well appreciate that pain most often has a proximate physical cause.

Allodynia

Pain due to a stimulus that does not normally provoke pain.

Note: The stimulus leads to an unexpectedly painful response. This is a clinical term that does not imply a mechanism. Allodynia may be seen after different types of somatosensory stimuli applied to many different tissues.

The term *allodynia* was originally introduced to separate from hyperalgesia and hyperesthesia, the conditions seen in patients with lesions of the nervous system where touch, light pressure, or moderate cold or warmth evoke pain when applied to apparently normal skin. *Allo* means "other" in Greek and is a common prefix for medical conditions that diverge from the expected. *Odynia* is derived from the Greek word "odune" or "odyne," which is used in "pleurodynia" and "coccydynia" and is similar in meaning to the root from which we derive words with *-algia* or *-algesia* in

them. *Allodynia* was suggested following discussions with Professor Paul Potter of the Department of the History of Medicine and Science at The University of Western Ontario.

The words "to normal skin" were used in the original definition but later were omitted in order to remove any suggestion that allodynia applied only to referred pain. Originally, also, the pain-provoking stimulus was described as "non-noxious." However, a stimulus may be noxious at some times and not at others, for example, with intact skin and sunburned skin, and also, the boundaries of noxious stimulation may be hard to delimit. Since the Committee aimed at providing terms for clinical use, it did not wish to define them by reference to the specific physical characteristics of the stimulation, e.g., pressure in kilopascals per square centimeter. Moreover, even in intact skin there is little evidence one way or the other that a strong painful pinch to a normal person does or does not damage tissue. Accordingly, it was considered to be preferable to define allodynia in terms of the response to clinical stimuli and to point out that the normal response to the stimulus could almost always be tested elsewhere in the body, usually in a corresponding part. Further, allodynia is taken to apply to conditions which may give rise to sensitization of the skin, e.g., sunburn, inflammation, or trauma.

It is important to recognize that allodynia involves a change in the quality of a sensation, whether tactile, thermal, or of any other sort. The original modality is normally nonpainful, but the response is painful. There is thus a loss of specificity of a sensory modality. By contrast, hyperalgesia (q.v.) represents an augmented response in a specific mode, viz., pain. With other cutaneous modalities, *hyperesthesia* is the term which corresponds to hyperalgesia, and as with hyperalgesia, the quality is not altered. In allodynia, the stimulus mode and the response mode differ, unlike the situation with hyperalgesia. This distinction should not be confused by the fact that allodynia and hyperalgesia can be plotted with overlap along the same continuum of physical intensity in certain circumstances, for example, with pressure or temperature.

See also the notes on hyperalgesia and hyperpathia.

Analgesia

Absence of pain in response to stimulation which would normally be painful.

Note: As with allodynia (q.v.), the stimulus is defined by its usual subjective effects.

Dysesthesia

An unpleasant abnormal sensation, whether spontaneous or evoked.

Note: Compare with pain and with paresthesia. Special cases of dysesthesia include hyperalgesia and allodynia. A dysesthesia should always be unpleasant and a paresthesia should not be unpleasant, although it is recognized that the borderline may present some difficulties when it comes to deciding as to whether a sensation is pleasant or unpleasant. It should always be specified whether the sensations are spontaneous or evoked.

Hyperalgesia*

Increased pain from a stimulus that normally provokes pain.

Note: Hyperalgesia reflects increased pain on suprathreshold stimulation. This is a clinical term that does not imply a mechanism. For pain evoked by stimuli that usually are not painful, the term *allodynia* is preferred, while *hyperalgesia* is more appropriately used for cases with an increased response at a normal threshold, or at an increased threshold, e.g., in patients with neuropathy. It should also be recognized that with allodynia the stimulus and the response are in different modes, whereas with hyperalgesia they are in the same mode. Current evidence suggests that hyperalgesia is a consequence of perturbation of the nociceptive system with peripheral or central sensitization, or both, but it is important to distinguish between the clinical phenomena, which this definition emphasizes, and the interpretation, which may well change as knowledge advances. Hyperalgesia may be seen after different types of somatosensory stimulation applied to different tissues.

Hyperesthesia

Increased sensitivity to stimulation, excluding the special senses.

Note: The stimulus and locus should be specified. *Hyperesthesia* may refer to various modes of cutaneous sensibility including touch and thermal sensation without pain, as well as to pain. The word is used to indicate both diminished threshold to any stimulus and an increased response to stimuli that are normally recognized.

Allodynia is suggested for pain after stimulation which is not normally painful. *Hyperesthesia* includes both allodynia and hyperalgesia, but the more specific terms should be used wherever they are applicable.

Hyperpathia

A painful syndrome characterized by an abnormally painful reaction to a stimulus, especially a repetitive stimulus, as well as an increased threshold.

Note: It may occur with allodynia, hyperesthesia, hyperalgesia, or dysesthesia. Faulty identification and localization of the stimulus, delay, radiating sensation, and aftersensation may be present, and the pain is often explosive in character.

Hypoalgesia

Diminished pain in response to a normally painful stimulus.

Note: Hypoalgesia was formerly defined as diminished sensitivity to noxious stimulation, making it a particular case of hypoesthesia (q.v.). However, it now refers only to the occurrence of relatively less pain in response to stimulation that produces pain. Hypoesthesia covers the case of diminished sensitivity to stimulation that is normally painful.

Hypoesthesia

Decreased sensitivity to stimulation, excluding the special senses.

Note: Stimulation and locus to be specified.

Neuralgia

Pain in the distribution of a nerve or nerves.

Note: Common usage, especially in Europe, often implies a paroxysmal quality, but neuralgia should not be reserved for paroxysmal pains.

Neuritis

Inflammation of a nerve or nerves.

Note: Not to be used unless inflammation is thought to be present.

Neuropathic pain*

Pain caused by a lesion or disease of the somatosensory nervous system.

Note: Neuropathic pain is a clinical description (and not a diagnosis) which requires a demonstrable lesion or a disease that satisfies established neurological diagnostic criteria. The term *lesion* is commonly used when diagnostic investigations (e.g. imaging, neurophysiology, biopsies, lab tests) reveal an abnormality or when there was obvious trauma. The term *disease* is commonly used when the underlying cause of the lesion is known (e.g. stroke, vasculitis, diabetes mellitus, genetic abnormality). *Somatosensory* refers to information about the body per se including visceral organs, rather than information about the external world (e.g., vision, hearing, or olfaction). The presence of symptoms or signs (e.g., touch-evoked pain) alone does not justify the use of the term *neuropathic*.

Some disease entities, such as trigeminal neuralgia, are currently defined by their clinical presentation rather than by objective diagnostic testing. Other diagnoses such as postherpetic neuralgia are normally based upon the history. It is common when investigating neuropathic pain that diagnostic testing may yield inconclusive or even inconsistent data. In such instances, clinical judgment is required to reduce the totality of findings in a patient into one putative diagnosis or concise group of diagnoses.

Central neuropathic pain*

Pain caused by a lesion or disease of the central somatosensory nervous system. *See neuropathic pain note.*

Peripheral neuropathic pain*

Pain caused by a lesion or disease of the peripheral somatosensory nervous system. *See neuropathic pain note.*

Neuropathy*

A disturbance of function or pathological change in a nerve: in one nerve, mononeuropathy; in several nerves, mononeuropathy multiplex; if diffuse and bilateral, polyneuropathy.

Note: Neuritis (q.v.) is a special case of neuropathy and is now reserved for inflammatory processes affecting nerves.

Nociception*

The neural process of encoding noxious stimuli.

Note: Consequences of encoding may be autonomic (e. g. elevated blood pressure) or behavioral (motor withdrawal reflex or more complex nocifensive behavior). Pain sensation is not necessarily implied.

Nociceptive pain*

Pain that arises from actual or threatened damage to non-neural tissue and is due to the activation of nociceptors.

Note: This term is designed to contrast with neuropathic pain. The term is used to describe pain occurring with a normally functioning somatosensory nervous system to contrast with the abnormal function seen in neuropathic pain.

Nociceptive stimulus*

An actually or potentially tissue-damaging event transduced and encoded by nociceptors.

Nociceptor*

A high-threshold sensory receptor of the peripheral somatosensory nervous system that is capable of transducing and encoding noxious stimuli.

Nociplastic pain*

Pain that arises from altered nociception despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors or evidence for disease or lesion of the somatosensory system causing the pain.

Note: Patients can have a combination of nociceptive and nociplastic pain

Noxious stimulus*

A stimulus that is damaging or threatens damage to normal tissues.

Pain threshold*

The minimum intensity of a stimulus that is perceived as painful.

Note: Traditionally the threshold has often been defined, as we defined it formerly, as the least stimulus intensity at which a subject perceives pain. Properly defined, the threshold is really the experience of the patient, whereas the intensity measured is an external event. It has been common usage for most pain research workers to define the threshold in terms of the stimulus, and that should be avoided. However, the threshold stimulus can be recognized as such and measured. In psychophysics, thresholds are defined as the level at which 50% of stimuli are recognized. In that case, the pain threshold would be the level at which 50% of stimuli would be recognized as painful. The stimulus is not pain (q.v.) and cannot be a measure of pain.

Pain tolerance level*

The maximum intensity of a pain-producing stimulus that a subject is willing to accept in a given situation.

Note: As with pain threshold, the pain tolerance level is the subjective experience of the individual. The stimuli which are normally measured in relation to its production are the pain tolerance level stimuli and not the level itself. Thus, the same argument applies to pain tolerance level as to pain threshold, and it is not defined in terms of the external stimulation as such.

Paresthesia

An abnormal sensation, whether spontaneous or evoked.

Note: Compare with dysesthesia. After much discussion, it has been agreed to recommend that *paresthesia* be used to describe an abnormal sensation that is not unpleasant while *dysesthesia* be used preferentially for an abnormal sensation that is considered to be unpleasant. The use of one term (*paresthesia*) to indicate spontaneous sensations and the other to refer to evoked sensations is not favored. There is a sense in which, since paresthesia refers to abnormal sensations in general, it might include dysesthesia, but the reverse is not true. Dysesthesia does not include all abnormal sensations, but only those that are unpleasant.

Sensitization*

Increased responsiveness of nociceptive neurons to their normal input, and/or recruitment of a response to normally subthreshold inputs.

Note: Sensitization can include a drop in threshold and an increase in suprathreshold response. Spontaneous discharges and increases in receptive field size may also occur. This is a neurophysiological term that can only be applied when both input and output of the neural system under study are known, e.g., by controlling the stimulus and measuring the neural event. Clinically, sensitization may only be inferred indirectly from phenomena such as hyperalgesia or allodynia.

Central sensitization*

Increased responsiveness of nociceptive neurons in the central nervous system to their normal or subthreshold afferent input.

Note: See note for sensitization and nociceptive neuron above. This may include increased responsiveness due to dysfunction of endogenous pain control systems. Peripheral neurons are functioning normally; changes in function occur in central neurons only.

Peripheral sensitization*

Increased responsiveness and reduced threshold of nociceptive neurons in the periphery to the stimulation of their receptive fields.

Note: See note for sensitization above.

References

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