Introduction

The second meeting in Headache in collaboration with the International Headache Society was held over two days in Tangier on 8-9 May 2015. On the first day, three speakers delivered four speeches. The IHS Visiting Professor, Dr. Gianluca Coppola from Italy, spoke for 30 minutes on Migraine: From Pathophysiology to Treatment. He presented his second 25 minute speech on the topic of Facial Pain. The first speaker from Morocco, Dr Laouina, talked for 20 minutes about Headache in Children and the second Moroccan speaker, Prof. Saadia Aidi, spoke for 20 minutes about Secondary Headache.

On the second day, an interactive session was held. In the morning, each speaker presented a case study for discussion. In the afternoon, we concluded the headache session with an exciting practical activity. Delegates were asked to submit a prescription for the treatment of two cases in headache. These cases were presented on the day before. After lunch, the speakers led a discussion analyzing these prescriptions. Delegates appreciated these practical activities and expressed their hope that the next headache session would include more of these practical activities.

Migraine: From Pathophysiology to Treatment

Dr. Gianluca Coppola

Dr. Coppola started by posing a question about where the headache starts. He said that during migraine without aura, the generator of migraine could be the Brainstem followed by Neurogenic Inflammation. During migraine with aura, the generator of migraine could be Cortical Spreading Depression. He explained the phenomenon of central sensitization during chronic migraine resulting from hyper-cortical excitability producing chronic pain and allodynia. In episodic migraine, he explained that repetitive Transcranial Magnetic Stimulation modifies the cortical response. He said that migraineurs have a defect in cell energy and adding vitamin B2 or Coenzyme Q10 was shown to be effective in reducing migraine attacks. He proposed that we could also introduce a diet like a low-sodium, vegan or ketogenic diet to reduce the frequency of attacks of migraine and to reduce the trigeminal vascular activity. This could avoid the progression from episodic to chronic form. Another example of inhibiting the trigeminal vascular activity is the use of botulinum neurotoxin. Finally, the speaker talked about the recent ongoing studies targeting calcitonin gene-related peptide pathways using monoclonal antibodies for the prevention of migraine.

Headache in Children

Dr. Laaouina, Morocco
The speaker gave a brief introduction on the history of migraine and headache from the Mesopotamian period to the present day.

During the Mesopotamian period, physicians considered headache as a disease rather than a symptom and believed that headache was caused by evil spirits. Nowadays, the interest in headache in children has increased. Many physicians are interested in headache in children, many clinics specialize in this field and migraine in children has its own classification.

The speaker emphasized the 2013 classification of migraine in children in the second part of his talk. He highlighted the two important elements that differentiate migraine in children from migraine in adults. The duration of migraine in children is short, ranging from 2 to 72 hours. The location of the migraine is mostly bilateral. He added that whenever the location is occipital or strictly unilateral, physicians should be aware of the possibility of secondary headache.

In the following section, the speaker said that migraine may be very difficult to diagnose in children when the migraine starts with episodes of vomiting, abdominal pain or paroxysmal vertigo. In these situations, physicians should go through all the investigations to search for organic disease.

The diagnosis of migraine in children is easy but challenging. It requires patience as the examiner is interviewing both the child and his parents. The child has a limited ability to describe characteristics of migraine like the quality, photophobia and phonophobia. These symptoms should be deduced from his behavior. The speaker’s experience suggests that an effective way to elicit an accurate description of the throbbing headache is, after auscultating the child’s heart, to give the stethoscope to the child to discover the throbbing of his own heart. The child is asked to confirm if what he auscultates is the same as what he feels during headache. Patience is also required because parents are worried that the child has a severe disease like a tumor or may doubt the existence of these recurring headaches. Some parents think that the child is making it up in order to avoid going to school. In order to overcome both the challenges related to the child and those related to the parents, the examiner should give sufficient time to the interview, which is the sole basis for the diagnosis of migraine, in order to reassure the parents and the child.

During the interview, the examiner should focus on the characteristics of migraine, the triggers and the cause of migraine.

As we know that migraine in children is frequently associated with tension-type headache, the examiner should first collect data about the characteristics of the most severe headache which resemble the migraine profile. After that, he should look for the characteristics of the mild headache which resemble the tension-type profile.

Concerning the triggers, the examiner should look for family problems, school problems like excessive homework, bullying or school transport and others like excessive video games, excessive physical activity, irregular mealtimes or maltreatment.

Finally, the examiner should look for the cause. This is hereditary, so for this reason, he also needs to interview the parents. During this interview some parents are reminded of their own experience of migraine as a child and start to feel reassured.
The second step is the general examination with the measure of blood pressure, body mass index and cardiac auscultation. The neurological examination should include the palpation of the pericranian muscles and a systematic funduscopry.

Secondary Headache

Prof. Saadia Aidi

Secondary headache is less frequent than primary headache, representing about 40% of all headaches, but it is mandatory to diagnose it because vital and functional prognoses are threatened. With regard to the classification of secondary headache, the most common causes of headache are non-neurological. Neurological causes represent only 5% of these causes. They are dominated by headache attributed to trauma, intracranial vascular disorder (1%) and intracranial non-vascular disorder (0.5%). Extra-neurological causes are the most frequent and are dominated by infection, especially upper-respiratory tract infection like sinusitis, otitis, rhinopharyngitis etc.

The diagnosis of secondary headache is generally easy although in some cases it can be difficult. The first step is to ask, “Is it a secondary headache?” It is challenging to make the decision that it is a secondary headache because of the consequences of further investigation. Once this possibility has been eliminated we would consider primary headache. The second step is to decide which diagnosis is evoked. There are more than 300 causes of secondary headache. The last step is to decide how to explore it.

The diagnosis of secondary headache starts with the interview. This interview is essential to classify the headache. It helps to know how the headache started, how it evolved and what the characteristics, circumstances and associated symptoms are. The second step is the clinical examination. This involves the general examination with measure of blood pressure and temperature. The local examination includes the palpation of the temporal artery, the sinus and eyes. The neurological examination includes the funduscopry.

The speaker emphasized some red flags:
- The headache is associated with systemic symptoms like fever, myalgia or weight loss or systemic disease like malignancy or acquired immune deficiency.
- Neurological symptoms or signs.
- Sudden onset: thunderclap headache.
- Onset after 50 years of age.
- Changes in the pattern of headache: headache becomes continuous or the type of headache changes.

Prof. Aidi said that when the onset of headache is recent, when the headache gets worse or becomes atypical it is considered as secondary headache. When the headache starts during sexual intercourse or when it becomes worse when changing from a lying position to a standing position it is also considered as secondary headache.

Concerning the neurological causes of headache, Prof Aidi focused first on sudden onset headache. These headaches are severe and have a rapid onset less than one minute. They could be triggered by precipitating factors like cough, physical effort or sexual activity. Their most common causes are
vascular, especially subarachnoid hemorrhage caused mainly by ruptured aneurysm. The second cause of thunderclap headache is artery dissection. She insisted on looking for Claude-Bernard-Horner sign to diagnose this. She explained that sinus thrombosis could rarely have thunderclap headache profile. The diagnosis could be confirmed by MRI to look for sinus thrombosis. She insisted on searching also for cortical vein thrombosis using Echogradient T2* identifying the non-signal of the vein. Some non-vascular causes of thunderclap headache could occur like tumor of the third ventricle or hypotension of the CSF.

Facial Pain

Dr. Gianluca Coppola

Many specialists are concerned with the diagnosis and treatment of facial pain. The diagnosis of facial pain is based on the interview. This interview should specify the localization of the pain, the character, the temporal profile, the signs and symptoms associated with the pain, its triggers and sometimes the therapeutic response.

Dr. Coppola distinguished between non-neurological causes of facial pain and neurological causes. The non-neurological causes include ophthalmic diseases like acute glaucoma, refractive error, heterophoria, disorders of the ear like otitis, causes related to the nose and sinus like sinusitis and those related to temporal mandibular dysfunction and teeth.

The most common neurological causes of facial pain are facial migraine, trigeminal autonomic cephalalgias, trigeminal neuralgia and Tolosa-Hunt Syndrome. Concerning Trigeminal Autonomic Cephalalgia, Dr. Coppola discussed Cluster Headache and Paroxysmal Hemicrania in detail. He spoke about the clinical characteristics of primary cluster headache, its prevalence of 0.2% in Europe, its physiopathology with the hypothalamo-hypophyso hyperactivity and its treatment with the effectiveness of oxygen and sumatriptan during pain. Concerning the preventative treatment of cluster headache, he talked about occipital injection of corticoid with short-term effect and Isoptrisina, Topiramate, Lithium... with long-term effect. He talked about the chronic form of cluster headache which is resistant to these forms of medication and requires surgical treatment with sub-occipital neurostimulation or hypothalamic neurostimulation. He showed an example of secondary cluster headache caused by macroprolactinoma. This form of symptomatic cluster headache is differentiated from episodic headache by persistent pain, the absence of autonomic signs, the absence of circadian rhythm or the presence of neurological deficit. He compared Paroxysmal Hemicrania and Cluster headache highlighting that the former is more predominant in females, the crises are short in duration, from 2–45 minutes, and the crisis should respond to Indomethacin. He gave examples of headaches that respond to Indomethacin like paroxysmal hemicranias, hemicranias continua and hypnic headache.

In the last part of his speech, Dr. Coppola gave the clinical characteristics of primary trigeminal neuralgia comparing it to cluster headache. The former is more predominant in females, the bursts of pain are very short, some less than 1 second, cutaneous triggers are present and the pain responds to Carbamazipin. He emphasized the importance of recognizing Symptomatic Neuralgia of the V. The clinical characteristics are based on continuous pain with long-lasting episodes and the
existence of signs like hypoesthesia. The pain could have neuropathic characteristics when the cause is post-zonal. He added other causes like multiple sclerosis or tumor of the posterior fossa. The treatment of trigeminal neuralgia consists of Carbamazipin and Baclofene. Other treatments could be helpful like Gabapentine, Pregabaline and Lamotrigine. The noninvasive surgical treatment of trigeminal neuralgia consists of Radiofrequency and Gamma knife. The former seems more effective.

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