



iHA IRANIAN
HEADACHE
ASSOCIATION
انجمن بین‌المللی سردرد ایران



International
Headache Society

2nd IRANIAN INTERNATIONAL HEADACHE SCHOOL

15-17 January 2021 - I.R. IRAN



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Welcome message



Dear Friends and Colleagues,

On behalf of the Iranian Headache Association (IHA), I am delighted to welcome you to the 2nd Iranian International headache school that will be held on January 15-17, 2021.

The event is supported by the Iranian Neurological Association.

Globally, headache has been reported as the first cause of disability under the age of fifty years. This scientific meeting provides the most useful and updated information on headache disorders presented by distinguished international headache specialists.

During this international meeting, attendees will have an excellent opportunity to learn more about the diagnosis and management of headache and face pain disorders, new developments in the science of headache medicine and the care of headache sufferers. Also, there will be a chance to share their information and also become acquainted with the beautiful natural scenery and ancient history of Iran by prepared video clips.

Mansoureh Togha, MD

Professor of Neurology

Welcome message



Dear Colleagues and Friends,

on behalf of the scientific committee of the 2nd Iranian International headache school, it is with great honor I invite you to participate in this school, which will take place on January 15-17 ,2021 in Iran.

Participants will join in a stimulating and capable social occasion program, which allows delegates to gain insight into the latest research in the field of headache. I believe that such a school is one of the high quality events in an excellent atmosphere.

It is with great pleasure we invite you to take part in this virtual meeting, which I hope it will be a scientifically and socially rewarding and memorable experience.

Yours sincerely,

Hossein Pakdaman,MD

President of Iranian Neurological Association



About IHA:

Iranian Headache Association (IHA) was established as a subdivision of Iranian Neurological Society in 2013 by Professor Mansoureh Togha, pioneer of Headache in Iran, in 2013. The IHA was affiliated to International Headache Society (IHS) and European Headache Federation (EHF) in 2015.

Up to now IHA held 6 international headache congresses and one Joint IHA and EHF headache school. As the mission of promotion in headache field in Iran, IHA has organized 2 to 3 national seminars and 2 to 3 workshops in a year in cooperation with active IHA members. Our country has had the privilege to host four International Headache Society past presidents (Professor Goadsby, Professor Rapoport, Professor Edvinsson) and the present president of IHS Professor Ashina, along with Professor Olesen, Professor Evers, Professor Yensen and also Professor Martelletti the past president of the European Headache Federation and other well-known scientists in the field of headache.

IHA dedicates its efforts to change of information and ideas concerning the causes and treatments of headache and related painful disorders and to improve the life of those affected by headache in Iran. This association is focused on education of the key players such as neurologists, general practitioners and all those involved in headache management in terms of the diagnosis and treatment of headache disorders.

The Iranian Headache Association carries out various research activities in the field of headache in the form of a dissertation or research project under the management of Professor Togha, which has led to the publication of dozens of articles in domestic and international journals.

Scientific committee:



Dr. Hossein Ansari



Dr. Elham Jafari



Prof. Fariborz Khorvash



Dr. Seyed Ehsan
Mohammadianinejad



Dr. Somayeh Nasergivehchi



Prof. Hossein Pakdaman



Prof. Mansoureh Togha



Dr. Nooshin Yamani

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Mr. Davoud Ghadbeigi



Ms. Shima sadat
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Mr. Amir Rafiee



Dr. Soudeh Razeghi
Jahromi



Prof. Mansoureh Togha



Dr. Nooshin Yamani

Invited speakers:



Prof. Fayyaz Ahmed



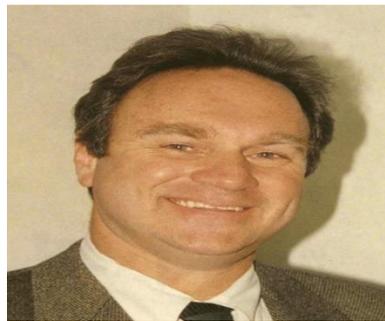
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Prof. Sait Ashina



Prof. Hayrunnisa Bolay



Prof. Robert Cowan



Prof. Stephen Evers



Prof. Fariborz Khorvash



Prof. Christian Lampl



Prof. Aynur Özge



Prof. Alan M. Rapoport



Prof. Mansoureh Togha



Prof. Derya Uludüz

Invited speakers:



Dr. Hossein Ansari



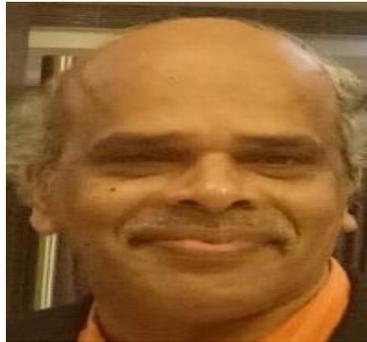
Dr. Marwa Yassien Badr



Dr. Lars Bendsten



Dr. Manuela Fontebasso



Dr. M V Francis



Dr. Ali Ghabeli



Dr. Susana Mederer
Hengstl



Dr. K M Nazmul Islam Joy

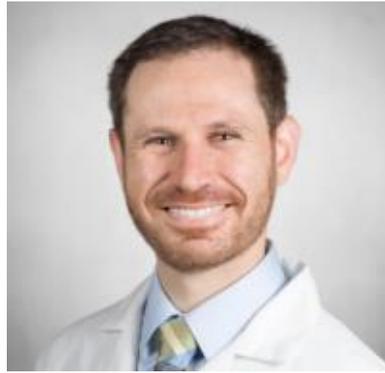


Dr. Seyed Ehsan
Mohammadianinejad

Invited speakers:



Dr. Muhammad Nasrullah



Dr. Nathaniel M. Schuster



Dr. Seby John



Dr. Verena subtil

Viuniska



Dr. Nooshin Yamani



Dr. Soudeh Razeghi

Jahromi

Abstracts



Prof. Sait Ashina

Tension headache: pathophysiology and co-morbidities

Tension-type headache (TTH) is the most prevalent primary headache disorder and associated with a considerable socioeconomic burden. Common comorbidities include depression, anxiety, neck pain, low back pain, and sleep abnormalities. In addition, most patients with migraine have co-existing TTH. The pathogenesis of TTH is believed to involve peripheral myofascial mechanisms, whilst central sensitization and abnormal pain modulation is likely to play a role in chronification of TTH.

Sait Ashina, MD, FAHS

Assistant Professor of Neurology and Anesthesia

Department of Neurology and Anesthesia, Critical Care and Pain Medicine

Harvard Medical School

Boston, MA

USA



Prof. Robert Cowan

Headache due to CSF dysregulation

This presentation will focus on headaches due to low CSF pressure (SIH). It is assumed that the more common and potentially serious headache due to increased intracranial pressure (IIH) will be covered in general training. Because SIH is underdiagnosed and often unrecognized as a cause of chronic headache, the clinical presentation, etiology, and nosology are covered in detail as well as treatment options.

Overview on other TACs

This talk will provide an overview of the Trigeminal Autonomic Cephalgias (TAC's) exclusive of Cluster Headache, the most common of the TAC's, which will be covered in a separate talk. The presentation will provide and current diagnostic criteria, pathophysiology, and treatment strategies, as well as references for further reading.



Prof. Fariborz Khorvash

Short lasting headaches without autonomic features:

The title refers to headaches without any autonomic symptoms such as tearing, facial swelling or sweating, redness of eye, ptosis and nasal congestion that last for seconds to minutes. According to ICHD3 the majority of these headaches are found in part three as Painful lesions of the cranial nerves and other facial pain. Many kinds of neuralgias in this session correlates with title. This article describes the clinical features and diagnostic criteria, pathophysiology (when known), and main characteristics of the major cranial neuralgias. Trigeminal neuralgia the most encountered neuralgia is 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 diagnosed disorder. Additionally, there may be concomitant continuous pain of moderate intensity within the distribution(s) of the affected nerve division(s). The division between, for example, trigeminal neuralgia and trigeminal neuropathy should be viewed as a pragmatic way of distinguishing conditions in which clinical presentations and treatment approaches differ while the two conditions cannot be classified on the basis of currently known pathology or pathophysiology. The same applies to painful conditions associated with the glossopharyngeal and intermedius nerves.

Diagnostic criteria:

A. Recurrent paroxysms of unilateral facial pain in the distribution(s) of one or more divisions of the trigeminal nerve, with no radiation beyond,¹ and fulfilling criteria B and C

B. Pain has all of the following characteristics:

1. lasting from a fraction of a second to two 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 ICHD-3 diagnosis.

Other primary neuralgias are occipital neuralgia, and, rarely, glossopharyngeal neuralgia. Nervus intermedius neuralgia is even more rare. All neuralgias merit a careful workup for secondary causes.

Key Words: Short lasting headaches, Autonomic features, Neuralgia



Prof. Aynur Özge

OVERVIEW OF HEADACHE IN CHILDREN AND ADOLESCENCE

A headache, especially migraine is not just a headache even in children and adolescents. Headache could be a symptom or a basic picture of main disease even children do not complain. On the other hand, as a most common phenotype of headache disorders, migraine is not just a headache, it is a disorder in which neurovascular headaches recur, is characterized by episodes of severe throbbing, pulsatile, commonly unilateral headaches, associated with nausea, vomiting, photophobia, phonophobia, and aversion to physical activity, with or without premonitory symptoms.

Not only families but also physician does not aware changing phenotype headache disorders and current knowledge about management. This lecture will be focused mainly basic principles of evaluation in headache symptom in children and adolescence. Migraine focused with two case samples and comprehensive management issues including attack, bridge and preventive medicine options. Red flags and secondary causes pointed focusing infections, vascular events and space occupying lesions in children and adolescent practice. Key messages summarized as follows;

- Childhood and adolescent's headache are a growing and under evaluated problem all over the world.
- Good communication with both patients and relatives is essential for diagnosis, also management.
- Communication should be incorporated all instruments of the patient's, should not restricted to anamnesis.
- Management of migraine and TTH should include strategies relating to daily living activities, family relationships, school, friends and leisure time activities.
- All new application and presenting signs of red flags required further evaluation should be always kept in mind for secondary causes.



Prof. Alan M. Rapoport

The new treatment approach for medication overuse headache

Medication Overuse Headache has a prevalence of about 1% worldwide. Some countries, like Russia are as high as 7%. Although overuse of any pain medication can result in MOH, some drugs carry an increased risk of the disorder; combination analgesics, opioids, butalbital containing medications and triptans are the medications that are most commonly associated with MOH. The most common comorbidities of MOH are depression and anxiety, and up to 50% of patients with MOH show a dependence-type behavior, such as tolerance or loss of control over pain medication use.

- Treatment includes patient counseling, initiation of preventive therapy and detoxification from the offending medications. In the countries where monoclonal antibodies to CGRP or its receptor are available, they make it much easier to treat this syndrome.

The ICHD-3 diagnostic criteria for MOH is:

- A. Headache occurring on ≥ 15 days/month in a patient with a pre-existing headache disorder
- B. Regular overuse for >3 months of ≥ 1 drugs that can be taken for acute treatment of headache

Still requires ≥ 10 days of use/month for ≥ 3 months of ergots, triptans, opioids, or combination analgesics but ≥ 15 days of use/month of NSAIDs, aspirin, or acetaminophen.

The old provision that the headache must have worsened with the overuse is gone.

The old provision that the headache must revert to episodic with detoxification is gone

The old linking of different meds to different patterns of daily headache is gone

The following medications are commonly used for treating migraine:

- OTC simple analgesics and NSAIDs
- Combination analgesics, often includes caffeine

- Prescription NSAIDs – diclofenac K for solution (Cambia) is approved for migraine)
- APAP or ASA/butalbital/caffeine (Fiorinal®, Fioricet®, Esgic®) Midrin®
- Ergots (ergotamine tartrate and DHE)
- Opiates
- Antiemetics (dopamine antagonists)
- 5-HT_{1B/1D}, 5-HT_{1F} agonists
- Gepants (ubrogepant & rimegepant)

There are many controversies surrounding MOH treatment:

- ICHD 3 removed worsening from the definition of MOH
- Detoxification or not (Danish Headache Center does 2 months of complete detoxification). So does Michel Ferrari in Leiden.
- Adding preventive medications or not
- When to add preventive medications
- A Copenhagen 3-cell trial: withdrawal plus preventive treatment, preventive treatment without withdrawal or withdrawal with optional preventive treatment 2 months after withdrawal – all showed equal results
- Srikiatkachorn, from Bangkok has described the pathophysiology of MOH:
 - Chronic medication exposure interferes the endogenous 5-HT dependent, descending pain control system (decrease in Diffuse Noxious Inhibitory Control or DNIC).
 - The altered endogenous control system leads to subsequent changes in brain areas subserving primary headache generation. These include:
 - Upregulation of CGRP neurotransmission in both central and peripheral trigeminal nociceptive pathways
 - Facilitation of trigeminal nociception at the brainstem trigemino-cervical complex
 - Increased cortical hyperexcitability rendering the increased susceptibility for CSD development

Non pharmacologic (biobehavioral treatment should always be used:

- Avoid Headache Triggers
- Eat and exercise regularly & sleep hygiene
- Use headache calendars or apps - essential
- Biofeedback training and stress management

- Cognitive therapy and psychotherapy
- Physical techniques (eg. physical therapy, manipulation, acupuncture, ayurveda)
- Consider Adjunctive therapies (Vitamins, Minerals, Supplements, Herbs): Vitamin B-2, Magnesium, Feverfew, Petasites, Melatonin and Coenzyme Q 10

The latest treatment suggestions:

- Treatment with topiramate, onabotulinumtoxinA or anti-CGRP monoclonal antibodies may reduce headache or migraine attack frequency and acute medication intake without deliberate withdrawal.
- However, the data supporting the efficacy of anti-CGRP monoclonal antibodies in this scenario are considerably more robust compared to the study results for topiramate and onabotulinumtoxinA.
- The anti-CGRP monoclonal antibodies appear to be particularly effective in converting patients with CM and MO out of MO and back to EM, as well as reducing acute medication use in EM and thus reducing the risk of transformation to CM.
- Diener H-C, Dodick D, Evers S Et al. Pathophysiology, prevention, and treatment of medication overuse headache. Lancet Neurol 2019; 18:891-902.

SUMMARY

- Educate the patient re MOH, mechanisms, treatment plan and expectations
- Employ Non-Pharmacologic and Behavioral strategies
- Identify and address psycho-social comorbidities
- Detoxify from all offending medications and agents, including caffeine; (Use Bridge

Therapy as needed)

- Institute prevention (mAbs to CGRP may be best) and limit acute care medicines
- Frequent planned follow-up visits with use of headache calendars or apps is essential



Prof. Mansoureh Togha

Headache in Covid-19 era

Since December 2019, the time when the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was spotted, numerous reports have been published on COVID-19 and its neuroinvasion, reviewing various aspects of the virus. A growing number of reports have reported headache as a common neurological manifestation of COVID-19.

However, although there are a number of hypotheses regarding the association between headache and the coronavirus, there is no solid evidence confirming the mechanism and features of headache in COVID-19.

In this review study, the headaches reported in previous studies are classified and their possible pathogenic mechanisms are outlined. To accomplish this objective, various types of headache are classified and their patterns according to ICHD-3 diagnostic criteria are outlined, including: headache attributed to systemic viral infection, viral meningitis or encephalitis, non-infectious inflammatory intracranial disease, hypoxia and/or hypercapnia, cranial or cervical vascular disorder, increased cerebrospinal fluid (CSF) pressure, refractive error, external-compression headache, and cough headache.

Then, their pathogenesis is categorized in three main groups, including: direct trigeminal involvement, vascular invasion, and inflammatory mediators.

Furthermore, persistent headache after the recovery as well as intensity predictors are further investigated.

This review may suggest a practical approach to classification, diagnosis, and management of COVID-19-attributed headache.



Prof. Derya Uludüz

Evaluation and management of headache in emergency department

Acute headache in the emergency department (ED) poses a diagnostic dilemma. Even the large majority are benign in nature, we must obtain accurate and detailed historical information and perform a thorough physical examination identifying life-threatening secondary causes. Secondary causes are broadly categorized into structural, vascular and infectious diseases. Primary headaches are treated in the ED with a focus on prevention upon discharge, whereas secondary headaches are emergency in nature and may be life threatening if not diagnosed.

A headache in benign nature may present with a very severe clinical presentation, and a headache in malignant nature may present with a mild clinical presentation. The aim in the emergency department should be to distinguish primary and secondary headache, to find the cause of secondary headache, to regulate the treatment of primary headache acute attacks.

Primary headaches have no structural or metabolic cause, while secondary headaches are caused by an underlying pathologic process. Migraine, tension-type, cluster, and thunderclap headache are all primary headache disorders. Secondary headaches are caused by conditions such as increased intracranial pressure, vascular pathologies, infection and brain tumours. Approximately 10% of the patients have secondary form. A complete headache history is the most important for diagnosis. History should include information about age of headache onset, pain intensity and character of the pain, trigger factors and comorbid conditions. The medical literature promotes red flags to direct the clinician for workup plan. The absence of red flags may suggest that no workup is needed. Clinical experience and large case series with a specific secondary headache form the basis for many red flags. It is possible to make a rapid assessment by asking a series of questions that will help you distinguish headaches. The next step is to exclude any possible red-flag symptoms. Red flag detection for secondary headaches requires the information of systemic symptoms, neurologic symptoms or signs, onset sudden or onset after the age of 50 years, and change of headache pattern.



Dr. Hossein Ansari

Overview on cluster headache: clinical presentation and diagnosis

Introduction - The Trigeminal Autonomic Cephalalgias (TACs) are a group of primary headache disorders that share similar clinical features but differ in frequency, duration, triggers, and treatment. “Cluster headache” is the most common of the TACs at a prevalence of 1 in 1000. Cluster headache is 3 times more common in men, with a typical age of onset between 20 and 40 years of age.

Cluster headache is a unilateral headache syndrome with ipsilateral cranial autonomic features and/or restlessness. Autonomic features in CH could be one of the following:

- Conjunctival injection and/or lacrimation
- Nasal congestion and/or rhinorrhea
- Eyelid edema
- Forehead and facial sweating
- Miosis and/or ptosis

An individual attack in CH lasts 15 to 180 minutes and occurring up to 8 times per day. The most peculiar feature of cluster headache is its clocklike regularity (circadian pattern). The two forms of cluster headache are an episodic CH (about 85-90% of CH), where patients have a headache-free period of more than 3 months, and a chronic CH (10-15 % of patients), where the headache-free period is less than 3 months.

Pathophysiology- The pathophysiology of CH is complex and the hypothalamus, trigeminovascular system, and the parasympathetic system play significant roles and their involvement have been discussed in detail. Calcitonin Gene Related Peptide (CGRP) is a marker of trigeminovascular activity and involvement of CGRP in CH has been shown. CGRP is localized to regions of the nervous system that are key players in CH pathophysiology, like the hypothalamus. Importantly, the posterior hypothalamus is activated during CH attacks. The role of cytokines and inflammation, in CH is unclear, although they appear to be involved, in some settings, when the trigeminovascular system is activated.

Differential Diagnosis - Despite the well-defined criteria, a “diagnostic delay” of several years may be seen for CH. The differential diagnosis of CH includes primary and secondary headache disorder. Cluster headache can also be misdiagnosed as migraine, as typical “migrainous

features” such as photophobia, phonophobia, facial allodynia, and nausea are seen in up to 50% of patients with CH. Other forms of TAC, particularly paroxysmal hemicranias can be mistaken as CH and sometime Indomethacin challenge is necessary to differentiates these two subtypes of TAC. Trigeminal neuralgia is another main differential diagnosis for CH. Between secondary headache disorder, headache attributed to paranasal sinus, particularly sphenoid sinusitis, are in differential diagnosis of CH.

Work up - For the initial diagnosis of patients with CH, neuroimaging with MRI (with and without contrast) or a non-contrast had been recommended. Sometime MRA also might be needed. One main reason to do imaging in all CH patients, is to rule out secondary CH. Secondary CH- in patients with clinical attacks that resemble CH, presence of cranial lesions could be suggestive of secondary CH, although sometime although a causal relationship is often uncertain. Lesion that reported to cause headache similar to CH includes:

- Pituitary macroadenomas
- Intracranial large artery aneurysms
- Benign posterior fossa tumor
- Meningiomas
- Sinusitis, particularly sphenoidal sinusitis
- Brain arteriovenous malformations
- Recurrent nasopharyngeal carcinoma
- Cavernous hemangioma

Cervicogenic headache

Introduction- Cervicogenic Headache (CeH) is one of the secondary headache disorders, which presents with unilateral pain, predominantly on the occipital and upper cervical regions, and is worsened by neck movement, sustained awkward head position, or external pressure over the symptomatic side. Based on International Classification of Headache Disorder, third edition (ICHD-3), diagnostic criteria for CeH are:

A. Any headache fulfilling criterion C

B. Clinical and/or imaging evidence of a disorder or lesion within the cervical spine or soft tissues of the neck, known to be able to cause headache

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

1. headache has developed in temporal relation to the onset of the cervical disorder or appearance of the lesion

2. headache has significantly improved or resolved in parallel with improvement in or resolution of the cervical disorder or lesion
 3. cervical range of motion is reduced and headache is made significantly worse by provocative maneuvers
 4. headache is abolished following diagnostic blockade of a cervical structure or its nerve supply
- D. Not better accounted for by another ICHD-3 diagnosis

Epidemiology- Due to diagnostic uncertainties, the epidemiology of cervicogenic headache is not very clear and its prevalence among the general population is estimated to be between 0.4 - 4 %. However, in a population with chronic headache prevalence of cervicogenic headache is increased to 15%-20%. The CeH affects women more often than men.

Pathophysiology- The anatomic locus for cervicogenic headache is the trigeminocervical nucleus in the upper cervical spinal cord. Trigeminal afferents overlap with the afferents of upper three cervical segments (C1-C3) at the lower end of the trigeminocervical nucleus (5). This anatomical connection between the cervical and trigeminal innervation systems would explain the frontal or retro-orbital pain in patients with CeH. This could be another reason why CeH is commonly mistaken as migraine or even tension type headache. Role of inflammation and Calcitonin Gene Related Peptide also has been suggested in pathophysiology of CeH .

Treatment - Among multiple treatment modalities suggested in the literatures, few have been tested and even fewer have been proven successful. In general, treatment choice may differ based on pain intensity and duration, patient age and comorbidities, and patient preference. Therefore, after establishing a CeH diagnosis, the treating physician should have a detailed discussion with the patient in order to explain potential treatment options. Manual therapy, usually in the form of physical therapy (PT) is the preferred initial treatment in CeH. Zygapophyseal (facet) injection with potential Radiofrequency ablation can be used in patient who does not response to conservative treatment.

Medication like Gabapentin and Pregabalin has been shown some benefit. In the acute phase of CeH or in patients who needs analgesic for breakthrough pain, a Non-Steroidal Antiinflammatory medication or acetaminophen / paracetamol component can be used.



Dr. Marwa Yassien Badr

Overview on headache in pregnancy and lactation: diagnosis and management

Headache is one of the most prevalent complaints in a woman's reproductive period and in pregnancy and puerperium. Primary headaches including migraine, tension type headache (TTH), and trigeminal autonomic cephalalgias (TACs) account for most of these headaches. It is fundamental to consider secondary causes in the differential diagnosis of headache, which may require urgent investigation. Preeclampsia, eclampsia, CVT, certain types of ischemic and hemorrhagic stroke, SAH, pituitary apoplexy, RCVS, PRES, and thunderclap headache show an overlapping clinical presentation and need to be treated emergently. One or more between electroencephalography, ultrasound of the vessels of the head and neck, brain MRI and MR angiography with contrast, brain CT, ophthalmoscopy and lumbar puncture will distinguish primary and secondary headaches. Migraine and all other headache disorders, whether primary or secondary, and particularly when they are chronic or resistant to treatment, are an important problem both for the mother and the fetus. Medications used for the treatment of primary headaches are not specific to these headache disorders. Options in prescription preventive medications are limited and it may be best to consider the safest interventions, which are lifestyle changes and behavioural treatment for stress management.



Dr. Lars Bendsten

Associate Professor, Department of Neurology and Danish Headache Center, Rigshospitalet Glostrup, University of Copenhagen, Denmark.

Trigeminal neuralgia, diagnosis and treatment

Trigeminal neuralgia is one of the most painful disorders known to mankind and can be difficult to diagnose and treat. In 2019, a guideline on trigeminal neuralgia from The European Academy of Neurology was developed by experts from across Europe based on scientific data and practice statements. The guideline answers the most important questions that patients and pain specialists are faced with regarding the management of trigeminal neuralgia.

How to ensure a correct diagnosis? Which clinical features should be considered? Which laboratory tests are required? Which imaging protocols should be used and what is the significance of the possible demonstration of a neurovascular contact? How to manage acute exacerbations? Which medications should be used for the long-term treatment, for how long, at which doses and should combinations be used? Which patients may benefit from a surgical approach and when should surgery be offered? What are the pros and cons of current surgical treatments? How do patients make decisions about their choice?

The above-mentioned clinically important questions will be discussed with the audience based on recommendations from the guideline and from a recent review on trigeminal neuralgia.



Dr. Manuela Fontebasso

Overview on migraine: clinical presentation and diagnosis

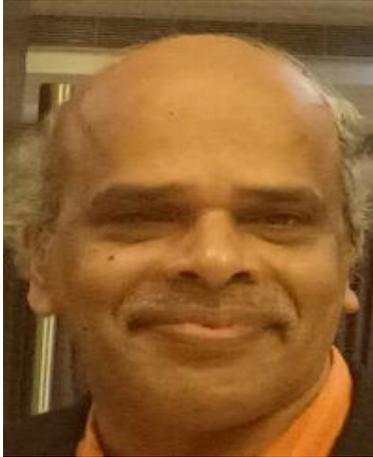
Migraine is a common disabling primary headache disorder affecting 10% of men and 22% of women. It is ranked as 7th highest cause of disability globally responsible for 2.9% of all years of life lost to disability. In a meta-analysis published in 2018 globally, migraine is the second highest cause of years lived with disability and is the primary cause of disability in adults under the age of 50.

It is a common misconception by patients and clinicians alike that you need to have aura in order to be diagnosed as having migraine. I plan to review the IHS classification of migraine with and without aura and set these in a clinical context for use on the consulting room.

Migraine is a high impact headache associated with symptoms and features. Making the diagnosis is about pattern recognition and how these symptoms and features present.

Migraine without aura can be summarised as a high impact, episodic headache associated with symptoms and features. The headache typically lasts 4 to 72 hours and patients are symptom free between each bout of migraine.

Patients with typical migraine aura experience fully reversible visual, sensory or motor symptoms developing over several minutes and resolving within the hour. Aura symptoms are complex and varied. Separating migraine aura and stroke can be a challenge in some circumstances and I will review the variable expressions of migraine aura.



Dr. M V Francis

NEUROOPHTHALMOLOGICAL HEADACHES

Neuroophthalmology is the overlap specialty between Ophthalmology and Neurology. Headache and facial pain accompany many neuroophthalmic disorders. The afferent visual system which roughly encompasses more than one third of the supratentorial brain mass and the efferent system which crisscrosses throughout the brain stem and cerebellum can be the origin of acute or recurrent headaches. Indeed, it is hard to imagine a neurologic disorder that could not have neuroophthalmic manifestations. Many diagnostic entities in the official groups 1 to 14 and in the appendix of ICHD 3 can present with neuroocular symptoms and signs. The commonest positive or negative migraine visual auras to Retinal migraine to complications of migraine like persistent auras, migrainous infarct and aura triggered seizures are the most important in the official ICHD 3 groups. Entities not in ICHD 3 too can surprise a clinician in a busy outpatient clinic. Alice in the wonderland syndrome, migraine equivalents, Episodic pupil dilatation and Appendix headache disorders like Alternating hemiplegia of childhood, Visual snow, Aura status and visually triggered Vestibular migraines presenting with nystagmus are some of them. Typical auras without headaches, probable auras, prodromal neuroocular symptoms like photophobia, blurring of vision and difficulty concentrating etc can be diagnostically challenging for any clinician so also differentials like seizure auras. Ocular and neuroocular autonomic symptoms of lacrimation, conjunctival congestion, miosis, ptosis, periorbital edema etc and rarely visual auras are classically diagnostic of Trigeminal autonomic cephalalgias. Primary stabbing headaches occasionally manifest in the orbito periorbital region. Ophthalmoplegic migraines (now renamed as Recurrent painful ophthalmoplegic neuropathy) and Brain stem auras are rare and difficult to diagnose. The most challenging are the GR 13 entities like HZO, Painful Optic Neuritis, THS and Ischemic oculomotor palsies which initially present only with orbital or periorbital pain without any diagnostic localizing signs or symptoms. Internal carotid artery dissection in Gr 6 is also similar. Primary Trochlear headache is a novel entity in ICHD 3 and is extremely rare. Subacute angle closure glaucoma and acute uveitis are to be ruled out in patients presenting with unilateral acute periorbital pain

Clinical evaluation and diagnosis of neuroophthalmic headaches should include a meticulous history based on ICHD 3, general physical exam, a detailed neurological and neuroophthalmological examination including cranial nerve assessment, fundoscopic exam looking for disc edema and spontaneous venous pulsations and palpation of the temporal arteries in the above 50 patients, blood pressure, intraocular pressure and temperature. In most cases, the cause of the headache is identified at this point whether primary or secondary and if secondary with any well documented RED FLAGS, investigations are planned accordingly. All patients with acute / subacute/different type/rapidly progressive or with neurological or neuroophthal symptoms or signs other than benign entities (Duane's retraction syndrome/ Superior oblique tendon sheath syndrome/End point and Congenital nystagmus/ Pseudopapilledema etc) require an urgent evaluation. In most cases of long standing episodic head pain suggestive of a primary headache disorder with a normal examination, no further investigation is necessary.



Dr. Ali Ghabeli

Complications of migraine

In this review, we will discuss the diagnostic criteria, pathophysiology, and possible treatments of the complications of migraine headache as identified by the International Classification of Headache Disorders version 3.

Migraine with aura may be associated with the onset of rare, but significantly disabling neurological symptoms. These complications may have same pathophysiological mechanisms. This review provides an overview of the associated complications that stem from migraine with or without aura. The neurological symptoms experienced by the patient correspond to a wave of altered brain activity known as cortical spreading depression.

The complications of migraine that stem from migraine aura are rarely encountered in clinical situation; however, they can be disabling for many patients. As these conditions are encountered, complete diagnostic evaluation is mandatory. In some cases, it may be difficult to find a consistently reliable treatment option for these patients.

Key words: status migrainosus, migraine with aura, persistent aura without infarction, migrainous infarction, migraine aura-triggered seizure.



Dr. Susana Mederer Hengstl

Overview on headache in elderly

Headache is the most common neurologic symptom. Its prevalence peaks around 40 years of age and declines thereafter. Diagnosing and treating headache disorders can be challenging in the elderly population (arbitrary defined as individuals aged 65 and older), as in this particular group of patients, although primary headaches are still more common, new onset headaches are more likely to have serious etiologies. Furthermore, the clinical presentation may be different compared to younger adults. Various imaging and laboratory evaluations are indicated in the presence of any “red flag” or symptoms. Finally, management requires careful assessment for comorbid conditions, polypharmacy, reduced medication tolerance and potential medication overuse.

In this seminar we will discuss the epidemiology, assesment, clinical features and treatment of headache within this population.



Dr. K M Nazmul Islam Joy

Tension-type headache: treatment

Treatment of Tension-Type Headache (TTH) can be discussed under the following headings-(i) General approach to the patient, (ii) Pharmacological treatment including acute pharmacotherapy and prophylactic treatment and (iii) Non-pharmacological treatment covering psychological and physical therapies.

General approach includes taking the patient's complaint empathetically and seriously along with excluding secondary causes like brain tumours. Importance is given on finding out co-morbidities (especially depression in chronic TTH), trigger factors (e.g. stress) and co-existent Migraine and Medication Overuse Headache (MOH). Patient education, consisting of explanation of the pathology and prognosis of TTH, is crucial.

Acute pharmacotherapy includes using Paracetamol 1000 mg or other NSAIDS (e.g. Naproxen 500 mg) for an acute attack of episodic TTH. Combination analgesics (like paracetamol and caffeine) are effective but may induce MOH. Triptans are only effective if episodic TTH is associated with migraine.

Prophylactic treatment is indicated for patients with very frequent episodic and chronic TTH. Amitriptyline with a dose of 30-75 mg administered 12 hours before waking time is a recommended prophylaxis in chronic TTH resulting in more than 50% improvement in over 65% patients. It is an antidepressant but also works on pain modulation. Other recommended drugs for prophylaxis are Mirtazapine & Venlafaxin (drugs of 2nd choice) and Clomipramine, Maprotiline and Mianserin (drugs of 3rd choice). Several other drugs are being used in different parts of world which includes sodium valproate, topiramate, propranolol and a fixed-dose combination containing melitracen 10 mg and flupentixol 0.5 mg. Notably, none of the prophylaxis may work until medication overuse is correctly addressed.

Of the psycho-behavioural treatments, EMG biofeedback has a documented effect in TTH, whilst cognitive-behavioural therapy and relaxation training most likely are effective, but there is no convincing evidence. Physical therapy and acupuncture may be valuable options for patients with frequent TTH. Improvement of posture, massage, spinal manipulation, oromandibular treatment, exercise programs, hot and cold packs, ultrasound and electrical stimulation etc. are the various physical treatment approaches for chronic TTH. Quantum

Molecular Resonance (QMR) is another technique which is able to decrease local inflammatory reaction and consequently pain level.

Chronic TTH is difficult to treat. Sometimes both pharmacological and non-pharmacological modalities may be required. Cure of headaches is rare, but control is possible if both the physician and patient can do their parts. Lack of sufficient controlled trials compels the physicians to rely on trial-and-error method, individual experience and locally available drugs.



Dr. Seyed Ehsan Mohammadianinejad

Rhinogenic headache

Rhinogenic headache was first introduced in ICHD-2 as a limited intermittent pain localized to the periorbital, medial canthal or temporo-zygomatic regions, who have clinical or imaging evidence of mucosal contact points, and in the absence of rhinosinusitis or other significant pathologies within the nasal cavity. It was proposed that the direct correlation of the pain and the contact points should be provided by the relation to postures or abolition of pain within 5 minutes of local anesthetic to middle turbinate, using placebo. Finally, there should be improvement within 7 days of surgery and does not recur.

ICHD-3 made remarkable changes to the previous edition according to the ambiguities in this topic and the vague direct correlation between the presence of contact points and the pain. It replaced the term with a broader one as “headache due to disorders of the nasal mucosa, turbinate or septum”. The localized site of pain was removed in criteria, but the temporal relation and ipsilateral location of the pain to intranasal lesion was insisted for a more precise diagnosis.

The concept of mucosal contact point is widely accepted in real world practice amongst otolaryngologists and head and neck surgeons, despite conflicting studies in the literature regarding a direct relation. This has led to many surgeries for releasing contact points over the world in recent years.

This approach has been changing from a blind one to a rational approach in recent years. Only a limited number of patients with these intranasal pathologies and headache will have benefit from surgical intervention. Patients with migraine who have already a heightened sensitivity of the trigeminal system, may be more susceptible to referred pain following stimulation of trigeminal nerve endings by mucosal contact points in the nasal cavity.

Patients with refractory headache over the face, orbit and frontal areas, who are exacerbated by changes in position, flying or diving, and or associated with persistent symptoms of nasal congestion, and do not respond to adequate treatments for primary headache, do not have explanations such as medication overuse or psychiatric comorbidity, and have a remarkable pathology causing contact points in opposing nasal mucosa should have a consultation for surgery. This could be especially true if the pain is ipsilateral to the nasal pathology and improves with application of topical anesthetic to the side of pathology.

Rhinogenic headache is a hot topic in the field of headache that is prone to misdiagnosis and missed diagnosis. A rational approach with the collaboration of a neurologist and otolaryngologist will give a better result for choosing the limited number of patients who can take benefit from surgical intervention in addition to medical treatment.



Dr. Muhammad Nasrullah

Conventional prophylactic treatment of migraine headache

Migraine is characterized by recurrent attacks of headache, associated with other clinical manifestations. Its management includes treatment of acute attacks and preventive/prophylactic treatment.

Prophylactic therapy can be divided into three approaches; episodic, intermittent and continuous.

Episodic prophylaxis is advised when a known trigger such as exercise or sexual activity leads to headache. Patients can be instructed to treat prior to the exposure or activity. For example, a single dose of indomethacin can be used to prevent exercise-induced migraine.

Intermittent prophylaxis is used when patients are undergoing a time-limited exposure to a provoking factor, such as ascent to a high altitude or menstruation. These patients can be treated with daily medication, just before and during the exposure. For example, perimenstrual use of a NSAID (like Naproxen) or triptan may prevent the emergence of menstrual migraine.

Maintenance prophylaxis is indicated when patients need ongoing treatment.

Indications of prophylactic treatment include:

- Frequency of migraine attacks is greater than 2 per month
- Duration of individual attacks is longer than 24 hours
- Headaches cause major disruptions in patient's lifestyle, with disability lasting 3 or more days
- Symptomatic medications are contraindicated or ineffective
- Use of symptomatic medications more than twice a week
- Migraine variants such as hemiplegic migraine, producing profound disruption or risk of permanent neurologic injury

- Frequent, long or uncomfortable auras
- Patient preference (Life style, cost, co morbidities)

Goals of treatment include:

- Reduce frequency, severity & duration of attacks
- Improve responsiveness to treatment of acute attacks
- Improve function and reduce disability

Realistic goals should be set as complete remission may not be possible always. Success is defined as a 50% reduction in attack frequency or headache days, a significant decrease in attack duration, or an improved response to acute medication.

Classes of drugs used for migraine prophylaxis include:

- Anti epileptic drugs (Valproic acid and Topiramate)
- Anti depressants (TCAs are widely used. Limited data supports SNRIs as well).
- Anti hypertensives (Beta blockers, Calcium channel blockers, ACE Inhibitors and ARBs)
- Botulinum toxin (in selected cases)
- Calcitonin gene-related peptide (CGRP) inhibitors (monoclonal antibodies)
- Miscellaneous (limited data favors Riboflavin, Quetiapine, melatonin, magnesium and some natural products)

Selection of drugs depends upon multiple factors which include:

- Patient's age, sex and comrbidities
- Attack frequency, severity and associated disability
- Efficacy of the drug
- Patient's preference

Treatment with the conventional medicines should be started in low doses and built up gradually. Prophylaxis should not be declared failure until it has been given in maximum tolerable dosage for thirty days at least.

Non pharmacologic measures are being used in addition to pharmacotherapy. Of note are cognitive behavioral and relaxation therapies, occipital nerve stimulation and surgical removal of muscle or nerve tissue from headache trigger sites (Confirmed by response to Botox).

Some mechanical devices (like TENS) are showing promising results.



Dr. Soudeh Razeghi Jahromi

Nutrition and headache

The question whether dietary habits and lifestyle have influence on headache is still a matter of debate. Obesity and headaches including migraine could be attributed to each other through mechanisms like inflammation, and irregular hypothalamic function. Thereby, applying dietary strategies for weight loss may also ameliorate headache/migraine. Between different weight loss strategies, low glycemic index diet, ketogenic diet, and modified Atkins diet were shown to have independent promising effect on migraine headache control. Low glycemic diet could modify inflammatory responses, and reduce the risk of migraine comorbidities such as hypertension and dyslipidemia. Ketogenic diet and modified Atkins diet are thought to play a role in neuroprotection, improving mitochondrial function and energy metabolism, compensating serotonergic dysfunction, decreasing calcitonin gene-related peptide (CGRP) level and suppressing neuro-inflammation. Preserving the balance between the intake of essential fatty acids, omega-6 and omega-3 is speculated to be effective in headache/migraine improvement by affect inflammatory responses, platelet function and regulation of vascular tone. Also, evidences pointed to the role of different vitamins and minerals in amelioration of headache symptoms. For instance, riboflavin can prevent migraine by influencing mitochondrial dysfunction. Preventing from hyperhomocysteinemia requires the presence of vitamins B6, B12, and folic acid, which can decrease the severity of migraine. Vitamin D, can affect migraine severity through ameliorating neuro-inflammation. So, adequate intake of different vitamins and minerals are also important in controlling headache/migraine symptoms. Dietary intake also affects the composition of commensal gut microbiota. The role of gut-CNS-Axis in headache/migraine control has become a matter of special interest in recent years and probiotic supplementation has been mentioned in migraine headache. Taken together, dietary approaches could be considered as effective strategies in headache/migraine prophylaxis.



Dr. Nathaniel M. Schuster

Acute treatment of migraine headache

Finding one or more effective acute treatments for the patient is one of the key components of comprehensive migraine care. We will discuss indications and contraindications, new and established acute migraine treatments (including non-oral treatments), evaluating for effectiveness and overuse, treatment options in special populations, and treatment selection for challenging clinical presentations.



Dr. Nooshin Yamani

Tension-type headache: clinical presentation and diagnosis

TTH is the most prevalent and the most common type of primary headache and is the third-most prevalent disorder in the world. Tension-type headache is usually a dull, bilateral non-throbbing headache without accompanying symptoms and divided according to ICHD-3 into three subtypes based on the headache frequency: infrequent episodic, frequent episodic TTH and chronic TTH. The global one-year prevalence estimated to be as 26.1% in the general population according to global burden of disease study 2016 with almost 1.89 billion individuals reported of having TTH. The prevalence of CTTH is much lower and is up to 3% in the general population. Risk factors for developing TTH include. young age, female sex, poor self-rated health, not being able to relax after work, and sleeping few hours per night. Poor outcome was associated with baseline chronic TTH, baseline major depression and medication overuse, coexisting migraine, not being married, and sleeping problems. TTH is a fairly featureless headache and the typical presentation is that of a bilateral, dull pain of mild to moderate intensity. Increased pericranial muscle tenderness is the most important abnormal finding in patients with TTH. Diagnosis of TTH is clinical and based on diagnostic criteria for the international headache society. Neuroimaging or other diagnostic workups are usually normal though they are not necessary in most patients with typical history of TTH and normal neurologic findings unless a secondary headache is suspected. The most frequent differential diagnosis is migraine without aura and medication overuse headache. Secondary causes of headache mimicking TTH should always be considered and A headache diary, a detailed history, and examinations are mandatory.

School program

Tehran Time	Topic	Lecturer
Day 1: Friday, January 15		
Moderator: Dr A. Amini, S. H. Dr Paknejad		
12:30-12:45	Opening program	
12:45-13:00	Welcome message from the presidents of Iranian neurological association and Iranian headache association	Prof. Hosein Pakdaman Prof. Mansoureh Togha
13:00-13:30	Epidemiology and burden of headache disorders	Prof. Christian Lampl
13:30-14:00	Pathophysiology and role of neurogenic inflammation in migraine	Prof. Hayrunnisa Bolay
14:00-14:30	Overview on migraine: clinical presentation and diagnosis	Dr. Manuela Fontebasso
14:30-15:00	Acute treatment of migraine headache	Dr. Nathaniel M. Schuster
15:00-15:40	Conventional prophylactic treatment of migraine headache	Dr. Muhammad Nasrullah
15:40-16:00	Break time	
Moderator: Dr S. E. Mohammadianinejad, Dr. A. Ghabeli		
16:00-16:40	New prophylactic drugs in migraine headache	Prof. Christian Lample
16:40-17:20	Treatment modalities in chronic migraine	Prof. Fayyaz Ahmed
17:20-17:50	White matter lesions in migraine	Dr. Verena subtil Viuniski
17:50-18:20	Trigeminal neuralgia, diagnosis and treatment	Dr. Lars Bendtsen
18:20-19:00	Headache due to CSF dysregulation	Prof. Robert Cowan
Day 2: Saturday, January 16		
Moderator Dr H. Ansari, Dr F. Vahabizadeh		
12:30-13:10	Overview on cluster headache: clinical presentation and diagnosis	Dr. Hossein Ansari
13:10-13:50	Treatment of cluster headache	Prof. Stephen Evers
14:20-14:50	Short lasting headaches without autonomic features	Prof. Fariborz Khorvash
14:50-15:30	Complications of migraine	Dr. Ali Ghabeli
15:30-15:50	Break time	
Moderator: Dr. F. Khorvash , Dr. N. Yamani		
15:50-16:20	Headache attributed to traumatic brain injury	Prof. Messoud Ashina

16:20-16:50	Rhinogenic headache	Dr. Seyed Ehsan Mohammadianinejad
16:50-17:20	Tension-type headache: clinical presentation and diagnosis	Dr. Nooshin Yamani
17:20-17:50	Tension-type headache: treatment	Dr. K M Nazmul Islam Joy
17:50-18:20	Tension-type headache: pathophysiology and co-morbidities	Prof. Sait Ashina
18:20-19:00	The new treatment approach for medication overuse headache	Prof. Alan M. Rapoport
Day 3: Sunday, January 17		
Moderator: Prof. M.Togha, Dr. S. Razeghi		
12:30-13:00	Evaluation and management of headache in emergency department	Prof. Derya Uluduz
13:00-13:40	Headache in Covid-19 era	Prof. Mansoureh Togha
13:40-14:20	Overview on headache in pregnancy and lactation: diagnosis and management	Dr. Marwa Yassien Badr
14:20-15:00	Headache attributed to vascular disease – the headache and hypertension	Dr. Seby John
15:00-15:30	Nutrition and headache	Dr. Soodeh Razeghi
15:30-15:50	Break time	
Moderator: Prof. F. Ahmed, Dr. M. Seyed Ahadi		
15:50-16:20	Neuro-ophthalmic headaches	Dr. M V Francis
16:20-17:00	Overview on headache in elderly	Dr. Susana Mederer Hengstl
17:00-17:40	Overview on headache in pediatrics	Prof. Aynur Ozge
17:40-18:20	Overview on other TACs	Prof. Robert Cowan
18:20-19:00	Cervicogenic headache	Dr. Hossein Ansari



The end.