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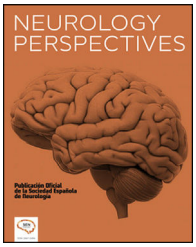

REVIEW

Similarities and differences between migraine and other types of headaches: Migraine mimics

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Abstract Headache impacts the well-being, capacity, and functionality of an individual. Migraine is the most common type of recurrent headache for patients seeking care. It is a complex neurological disorder associated with various symptoms of sensory, autonomic, and cognitive function, suggesting the involvement of multiple neuronal networks. The diagnóstico of migraine warrants both inclusion of specific clinical features and exclusion of other types of headaches. There are many types of headaches that may have "migraineous features". It is essential to recognize and differentiate "migraine mimics" as they may have some common features with migraine but may also be associated with high morbidity and/or mortality. The diagnostic errors often lead to a therapeutic delay or even malpractice. In this review, the most common "migraine mimics" will be discussed.

Introduction

Migraine is a common primary headache, ranked the third-highest cause of disability under the age of 50 years.[1] It is a debilitating, complex neurological disorder, and more than a visceral pain. Migraine patients describe their pain as moderate/severe intensity, throbbing pain or pulsating sensation, usually unilateral but may be on both sides, often accompanied by nausea, vomiting, sensitivity to light (photophobia) and sound (phonophobia), and aggravation by or causing avoidance of routine physical activity. Some migraineurs describe sensitivity to non-noxios stimuli, pain due to a stimulus that does not normally provoke pain (allodynia) or sensitivity to smell/odors (osmophobia). Migraine may present with or without aura. Migraine consists of 5 phases: premonitory, aura, headache, postdrome, and interictal phase.[2] The main features of each migraine phase are represented in Table 1.

Pain impacts the well-being, capacity, and functionality of an individual. The International Association for the Study of Pain (IASP) defines pain as "an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage".[3] Acute pain serves as a protective signal. It elaborates cognitive,

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Table 1 The main features and milestones of each migraine phases.

The main features and milestones of each migraine phases	
1. Premonitory Phase	• The most frequent symptoms described in that phase are *fatigue, drowsiness, mood changes, food craving or loss of appetite, neck discomfort and gastrointestinal disturbances* etc. • These symptoms imply the involvement of the hypothalamus, brainstem, limbic system, and certain cortical areas to the process. • Frequent hypothalamic-related disturbances like *appetite, sleep regulation, fluid imbalance* in migraineurs draw attention to the importance of hypothalamus. • Cranial autonomic symptoms, which are *lacrimation, nasal congestion, and rhinorrhea*, are also suggestive of altered autonomic function • The regulation of locus coeruleus activity by sleep-wakeful orexinergic neurons suggests a link between *sleep and migraine* regulation. • Stress-induced humoral systems may also contribute to stress-induced migraine.
2. Aura Phase	• The most frequent symptom of aura in migraineurs is known as *visual disturbances*; *sensory*, *language/ speech and motor disturbances* and even *disruption of higher cortical functions* are also observed. • CSD can be considered as a neurophysiological reflection corresponding to initiation and progression of the migraine aura. • The increase in the level of extracellular potassium and the 30–50 s depolarization wave leads to the onset of CSD. • The increased potassium intake disrupts the membranous ionic gradient of the cells, leading to elevated sodium, calcium inlet and glutamate release through outer cell membrane.
3. Headache Phase	• The characteristic *throbbing pain* of migraine headache is considered as a result of trigeminovascular pathway activation. • The trigeminovascular pathway transmits the nociceptive to thalamus and to the specified cortical areas, related with *photophobia, phonophobia, cognitive dysfunction, osmophobia, and allodynia*. • The thalamus, which has critical roles in nociceptive, *tactile, visual, olfactory and auditory sensation*, probably represents a key first stage for *multimodal sensory integration* in migraine.
4. Postdrome Phase	• *Fatigue, difficulty in concentrating and neck stiffness* are the typical and commonly reported postdrome symptoms. • As the whole brain structures may contribute to postdrome phase, it is important to draw attention especially to the hypothalamus and prefrontal lobe.
5. Interictal Phase	• The migraine brain is characterized by *increased excitability* in response to a broad spectrum of stimuli including visual stimuli, somatosensory stimuli, auditory stimuli, as well as brainstem reflexes in response to noxious stimuli. • The migraineous brain differs, in terms of microstructural and macrostructural magnitude and the neuronal networks. • Although the main cause of hyperexcitability in migraine is unknown, *some genetic/epigenetic factors* may have an important role in the development of hyperexcitability.

behavioral, autonomic, and emotional response to overcome the threat for the body's integrity and homeostasis. Headaches may also be alarming especially in the "secondary headaches". The "red flag" signs and symptoms (SNNOOP10 list) needs to be clarified in each headache patient, at the time of the consultation.[4]

When it comes to migraine, the unsolved question arises as to whether it has any protective role? The clinical features of migraine may have wide range of symptoms, it may be difficult to diagnose properly at the first visit. Besides, there may be an overlap and similarities in clinical characteristics and associated features in headache patients, which may negatively impact the standard care. A comprehensive and systematic history taking accompanied by physical/ neurological examination and if necessary, targeted diagnostic investigations is undertaken to support or exclude the diagnóstico. The accurate diagnóstico may enable the appropriate headache management. The most common "migraine mimics" listed in the Table 2 and key features of the diagnostic framework listed in **table 3**.

Primary headaches that may have migrainous features

Cluster headache (CH)

Cluster headache (CH) attacks are short-lasting (15–180 min), severe, unilateral, orbital, supra- orbital and/or temporal pain accompanied by at least one autonomic finding (ipsilateral conjunctival injection, lacrimation, nasal congestion, rhinorrhea, forehead and facial sweating, miosis, ptosis, eyelid edema) and/or a sense of restlessness.[5] Migraine subjects may also have unilateral headache. Although migraine subjects describe autonomic symptoms, they are less frequent, usually bilateral, and milder, generally patients report only one symptom.[6]

Table 2 The Most Common Migraine Mimics.

The Most Common Migraine Mimics	
1. Primary Headaches:	Attributed to cranial or cervical vascular disorders:
- Cluster Headache	- Subarachnoid Hemorrhage
- Tension-type Headache	- Arteriovenous Malformations
- New Daily Persistent Headache	- Vascular Malformations
	- Cerebral Venous Thrombosis
2. Secondary Headaches:	- Reversible Cerebral Artery Vasoconstriction Syndrome
	- SMART Syndrome**
Infectious/Inflammatory:	- Cervical Artery Dissection
- Acute and Chronic Sinusitis	- Headaches during Intracranial Endovascular Procedures
- Acute and Chronic Meningitis or Meningoencephalitis	- CADASIL***
- Viral Meningitis	- MELAS****
- HaNDL Syndrome*	- Temporal Arteritis
Attributed to space occupying brain lesions:	Attributed to substance exposure or withdrawal
- Brain Tumors	- Nitric oxide (NO) donor-induced headache
- Pituitary Adenomas	- Medication-overuse headache (MOH)
- Brain Abscess	- Alcohol-induced headache
	- Caffeine-withdrawal headache
Attributed to changes in CSF pressure:	
- Pseudotumor Cerebri Syndrome	Headaches associated with other diseases:
- Hydrocephalus	- Epilepsy
- Spontaneous Intracranial Hypotension	- Acute Glaucoma
	- Arterial Hypertension
Trauma- related:	- Cardiac Cephalalgia
- Post-Traumatic Headache	- Dialysis
	- Sleep apnea

* HaNDL: Headache associated with neurological deficits and cerebrospinal fluid lymphocytosis.
** SMART: Stroke – like migraine attacks after radiation therapy.
*** CADASIL: Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy.
**** MELAS: mitochondrial encephalopathy, lactic acidosis, and stroke-like episodes syndrome.

There are many similarities and differences between migraine and cluster type headache.[7] The pain of CH is usually nocturnal, characterized by a circadian and circannual pattern. The CH attacks last shorter and tend to cluster. On the other hand, migraine attacks occur most frequently during the day and a seasonal rhythm has not been established. Also, patients who have more than two headache attacks a day are less likely to have migraine.[6] Unlike migraine, there is a desire to wander instead of sitting still. However, about 24% of CH patients may have at least one migraineous feature.[8] The term "cluster migraine" is used to describe patients with cluster headache and migraine overlap.[9] CH may respond to many medications used in migraine preventive treatment, such as topiramate, verapamil, valproate, steroids, galcanezumab and peripheral nerve blocks.[7,10,11] CH attacks also respond well to acute treatment with triptans (especially subcutaneous sumatriptan) and ergotamine. Compared to migraine, CH attacks responds well to high flow 100% oxygen mask and transnasal sphenopalatine block by 10% lidocaine.[7] However, simple analgesics and non-steroidal anti-inflammatory drugs used in migraine attacks do not relieve pain in CH attacks.[6] CH has striking circadian and circannual periodicity.[12] This rhythmicity and accompanying altered hypothalamic hormonal secretion pattern emphasize the role of the hypothalamus in CH pathophysiology.[13] Hypothalamic involvement usually occurs during the premonitory phase of the migraine attack, the patient experiences mood changes, food craving or loss of appetite, neck discomfort, and gastrointestinal disturbances.[14] Both cluster and migraine demonstrate post-ganglionic parasympathetic outflow from the SPG, cluster to target organs.[15]

CH patients have premonitory and prodromal symptoms.[10,11] The preictal and postictal symptoms were concentration difficulties, restlessness, and mood disturbances, and occurred 20 min before headache in approximately half of the attacks.[15] Postictal concentration difficulties, decreased mood, and fatigue lasted for an hour on average in about a third of the patients.[15]

Tension - type headache (TTH)

TTH is typically bilateral, pressing or tightening in quality, and of mild to moderate intensity, lasting 30 min to days. Routine physical activity does not aggravate pain, and usually TTH is not associated with nausea, although photophobia or phonophobia may be present, but not both.[5] Headache begins at some point during the day and persists in the reminder of the day, with headache aggravation, usually by the afternoon or end of the day.[16] Peripheral mechanisms may play a role in episodic TTH, whereas central pain mechanisms dominate in chronic TTH.

Table 3 The most common migraine mimics and diagnostic frameworks.

Migraine Mimics	Key Features
Cluster Headache	- Short-lasting (15–180 min), unilateral orbital, supraorbital and/or temporal, very severe, excruciating pain. - A sense of restlessness or agitation is reported - Accompanied by autonomic findings ipsilateral to the headache (conjunctival injection and/or lacrimation, nasal congestion and/or rhinorrhoea, eyelid oedema, forehead and facial sweating, miosis and/or ptosis) - Characterized by a circadian or circa-annual pattern. - Occurring with a frequency between 1 every other day and 8 attacks per day
Tension - Type Headache	- Headache is bilateral, pressing or tightening in quality. - Lasting 30 min to 7 days. - Is usually reported by the end of the day and associated with stress. - Routine physical activity does not cause aggravation of headache. - No nausea or vomiting. - Pericranial tenderness may be detected by manual palpation.
New Daily Persistent Headache	- Acute onset of a continuous and unremitting headache, on a clearly remembered day. - May have features of chronic migraine or chronic tension-type headache
Acute and Chronic Isolated Sphenoid Sinusitis	- It may worsen with standing, kneeling, and coughing, and not relieved by sleep. - Fever, nasal congestion/ nasal obstruction may be present. - Compressing to the frontal or maxillary sinuses may evoke pain. - Imaging is recommended
Acute and Chronic Meningitis	- Headache is accompanied by fever, neck stiffness, and other neurological findings. - Neuroimaging and lumbar puncture should be performed.
HaNDL	- Migrainous features - Duration is between 1 h to 1 week - Contrast-enhanced neuroimaging may disclose leptomeningeal enhancement and/or focal hypoperfusion. - Elevated lumbar puncture opening pressure, high protein content (up to 250 mg/dl) and lymphocytic pleocytosis detected on CSF
Brain Tumors	- Accompanied by numbness, seizures, sensory changes, nausea, or vomiting. - Contrast-enhanced neuroimaging is warranted
Brain Abscess	- Headache may be exacerbated by Valsalva maneuver. - Abnormal neurological examination findings may be present - Contrast-enhanced neuroimaging is recommended
Pseudotumor Cerebri Syndrome (Idiopathic Intracranial Hypertension)	- Accompanied by visual changes, pulsating tinnitus and sometimes 6th cranial nerve palsy, - Papilla edema may be present - Patients tend to be overweight or obese - Brain MRI may disclose typical findings - Elevated CSF pressure (>250 mmH_20) on lumbar puncture is essential for the diagnóstico.
Hydrocephalus	- New headache or significant worsening of prexisting headache (implies a two-fold or greater increase in frequency and/or severity) - Evidence of hydrocephalus demonstrated by neuroimaging.
Headache Attributed To Low Cerebrospinal Fluid Pressure	- Headache significantly worsens soon after sitting upright or standing (orthostatic) and/ or improves after lying horizontally - Contrast-enhanced brain MRI and spinal imaging may disclose typical findings
Post-Traumatic Headache	- Headache has a close temporal relation to trauma or injury to the head and/or neck - Advanced neuroimaging modalities may detect structural, functional and metabolic abnormalities in the brain
Subarachnoid Hemorrhage	- A severe and sudden onset (thunderclap) - Worst headache ever - Lumbar puncture should be performed after brain imaging to demonstrate evidence of hemorrhage - Neurovaascular imaging is warranted to detect any vascular abnormality such as aneurysm or AVM.
Arteriovenous Malformations (AVM)	- A typical headache which is always localized to the same side, ipsilateral to the AVM. - Neurovascular imaging is warranted.

Table 3 (*continued*)

Migraine Mimics	Key Features
Cerebral Venous Thrombosis	- Focal neurologic symptoms and /or signs of raised intracranial pressure signs may be present. - Aggravation of headache by straining, lying down, or Valsalva maneuvers - Brain MRI and MR venography should be performed.
RCVS	- Severe thunderclap headache accompanied by migraineous features, frequently visual blurring, scotomas, and transient blindness is reported - Brain MRI should be performed to demonstrate vasospasm.
SMART Syndrome	- Migraine-like headaches accompanied by neurological symptoms and deficits - Seizures are frequent - History of cranial radiation therapy. - Neuroimaging is recommended
Carotid Artery Dissection	- Rapid onset of dull pain - Ipsilateral neck pain or Horner syndrome is alarming. - Vascular imaging should be performed.
CADASIL	- Migraine, cerebrovascular disease and dementia - Neuroimaging is recommended - Genetic testing is diagnostic
MELAS	- Lactic acidosis and recurrent ischemic episodes - Mitochondrial mutation detection by genetic testing - Neuroimaging is recommended.
Temporal Arteritis	- New onset headaches in patients over 50 years - Scalp tenderness, jaw claudication, weight loss, fatigue, and proximal myalgia may present - Elevated ESR, and CRP levels - Temporal artery imaging and biopsy is warranted
Medication Overuse Headache	- Headache is due to an interaction between an overused acute symptomatic headache medications. - Susceptible patient previously been diagnosed with especially migraine or tension-type headache. - The majority of patients with MOH improve after discontinuation of the overused headache medication.
Alcohol-Induced Headache	- Headache is induced within 3 h of alcohol consumption - Headache is usually bilateral and pulsating in quality. - Physical activity may aggravate the headache.
Caffeine-Withdrawal Headache	- Headache is induced within 24 h after the withdrawal of regular caffeine consumption - Headache is typically diffuse, severe, throbbing and is aggravated by exercise or Valsalva maneuver
Epilepsy	- Detailed history of headache and seizure characteristics is warranted - EEG should be performed
Acute Glaucoma	- Severe pain localized to one red eye accompanied by tenderness, blurred vision/vision loss, midriasis - Ophthalmologic evaluation should be performed.
Arterial Hypertension	- Headache is bilateral, pulsating, migraine-like features - Headache associated with an acute increase in systolic blood pressure to $\geq$180 mmHg, diastolic to $\geq$120 mmHg
Cardiac Cephalalgia	- Unilateral or bilateral migraineous headache usually aggravated by exercise, - Evidence of myocardial ischemia is warranted. - Headache is relieved by nitroglycerine.

(Abbrevations: HaNDL: Headache associated with neurological deficits with cerebrospinal fluid lymphocytosis, RCVS: reversible cerebral artery vasoconstriction syndrome, SMART: Stroke-like Migraine Attacks After Radiation Therapy, CADASIL: Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy, MELAS: mitochondrial encephalopathy, lactic acidosis, and stroke-like episodes, CSF: cerebrospinal fluid, MRI: magnetic resonance imaging, EEG: electroencephalography, CRP: C-reactive protein, ESR: erythrocyte sedimentation rate)

Increased peri-cranial tenderness is usually reported in TTH patients associated with stress. Neck pain may be present in about two-thirds of those with migraine, and stress is frequently reported as a migraine trigger, so these clinical symptoms do not help with differentiation.[17] The main distinctive feature of migraine is aggravation of pain by common daily life physical activities, not generally present in TTH. However, precipitating and aggravating factors in

TTH may also overlap with migraine. On the other hand, some chronic migraine patients may have comorbid medication overuse headache and the baseline ongoing headache characteristics may complicate the differential diagnóstico of chronic migraine from TTH.[16]

New daily persistent headache (NDPH)

New Daily Persistent Headache (NDPH) is the abrupt onset of a continuous and unremitting headache on a remembered day. It is characterized by an acute and daily onset of headache that lasts for more than 3 months, never remitting. NDPH may have migraine and/or TTH features. It may be misdiagnosed as chronic migraine (CM).[5] However, patients generally recall accurately the acute onset of continuous headache; if not, a clinician must consider other diagnoses. There may be a preexisting history of migraine in 20% of NDPH patients. In one study with NDPH patients, throbbing pain, photophobia, phonophobia, and increase in the severity of pain by physical activity were reported in about half of the patients. Even visual aura has rarely been reported with NDPH, which has migraineous features and which may sometimes, but rarely, respond to acute and preventive treatments for CM.[5,18,19] Recently, NDPH has been reported following recovery from COVID 19 infection, which is characterized by holocranial, pressure-like pain starting within 2 weeks after recovery of respiratory symptoms.[20]

Secondary headaches that may have migrainous features

Acute and chronic isolated sphenoid sinusitis

The pain is often felt unilaterally in the frontal, temporal, occipital, vertex or retro-orbital locations, and sometimes in more than one area. It may be accompanied by nausea or vomiting, may worsen with standing, kneeling, and coughing, and not relieved by sleep. Fever is reported in half of the patients, while nasal congestion or nasal obstruction is present in approximately 40% of patients. Compressing the frontal or maxillary sinus may evoke the pain.[21,22]

Acute and chronic meningitis or meningoencephalitis

Headache is the predominant symptom accompanied by fever, neck stiffness, and neurological features (altered mental status, focal neurological deficits, or seizures). Headache is due to the irritation/activation of the meningeal nociceptors and thus may resemble migraine. Bilateral throbbing headache and accompanied by nausea, vomiting, and photophobia. Headache is temporary with the resolution of infection. However, meningitis may persist for months, leading to chronic headache, and may rarely persist for more than three months.[5,9] Unlike bacterial meningitis, fever and neck stiffness may not appear in viral meningitis. Fever is also not expected in recurrent benign aseptic meningitis (Mollaret's meningitis), which is frequently associated with herpes virus.[18,23]

Headache associated with neurological deficits and cerebrospinal fluid (CSF) lymphocytosis (HaNDL)

Patients who suffer from HaNDL, may present with bilateral or hemicranial, throbbing, migrainous headache of moderate to severe intensity. Duration is between 1 h–1 week, (mean of 19 h). It may be accompanied by nausea, vomiting, photophobia, phonophobia, and neurological deficits including hemi-paresthesias, hemiparesis, and/or dysphasia. The transient attacks last 5 min–1 week and can repeat several times a week. However, visual symptoms similar to migraine aura are relatively rare.[5] Elevated lumbar puncture opening pressure, high protein content (up to 250 mg/dl), and lymphocytic pleocytosis in the cell count of CSF confirms the diagnóstico. All patients recover completely within 1 day–3 months.[18,24]

Brain tumors and pituitary adenomas (PA)

The prevalence of headache in patients with intracranial tumors ranges from 32% to 71%. In patients with primary CNS tumors or metastasis, migraine-like headache may be present in 15%.[5,25,26] In a population-based study, compared to unaffected controls, patients with brain tumors had 2.5-fold increased risk of having prior migraine.[27] Worsening of migraine may be considered as a red flag. Migraine-like headaches are reported in patients with craniopharyngioma, colloid cysts, pituitary tumors, brainstem glioma, and cerebral metastases.[28–32] Headaches may occur due to infiltration of bones or meninges, increased intracranial pressure or venous congestion/thrombosis. Regular medical follow-up after migraine diagnóstico can aid in early recognition of key symptoms of brain tumors. Clinicians should be careful about the key symptoms of CNS tumor which are unexpected in migraineurs such as numbness, seizures, sensory changes, nausea or vomiting.[27] The prevalence of headache reported in pituitary adenomas (PAs) ranges from 33% to 72%.[33,34] Multiple pathophysiological mechanisms have been proposed, the mechanical effect of tumor growth and the neuroendocrine effects of the functional pituitary tumors. Mechanical and structural factors are cavernous sinus invasion and infiltration of nociceptive structures.[35] Increase in intrasellar pressure could be another mechanism leading to PA-related headaches. PAs are also associated with the development of secondary short lasting unilateral neuralgiform headaches with conjunctival injection and tearing (SUNCT).[35]

Brain abscess

Brain abscess can mimic migraine status. The headache is typically moderate to severe and may be exacerbated by Valsalva maneuver. Headache was reported to be present in 70%, fever in 50%, and focal neurological deficits in 50% of the patients with brain abscess, 20% patient report all 3 symptoms. Half of the patients reported nausea or vomiting.[36,37] Seizures and cerebral venous thrombophlebitis may be present in these patients.[36]

Pseudotumor cerebri (idiopathic intracranial hypertension, IHH)

Patients with IIH may present with chronic or subacute daily headache. Headache is described predominantly early in the morning or awakening, frontal and bilateral localization, accompanied by vomiting without nausea, visual changes, cranial nerve palsies (especially sixth cranial nerve palsy), and pulsating tinnitus.[9] Neuroimaging findings such as an empty sella, distention of the peri-optic subarachnoid space, flattening of the posterior sclera, and protrusion of the optic nerve (papilledema), support the diagnóstico of IIH, but the key finding is elevated CSF pressure (> 250 mm H_20) on lumbar puncture. Although headache attributed to IIH can mimic CM and CTTH, these disorders commonly coexist with IIH.[38]

Daily headache is not required for the diagnóstico of IIH.[5] Almost 41% of IIH patients are reported to have migraine, while 17% reported migraine with aura.[38] Patients with IIH tend to be overweight or obese.[9] Diagnosis of IIH without papilledema (IIHWOP) requires a constellation of unequivocal MRI findings and/or cranial nerve palsies.[39] Unexpected comorbidity reports of IIH, IIHWOP, and CM noted that these syndromes can be linked with each other.[40] One potential mechanism connecting the three headaches is temporary venous insufficiency due to increased cerebral perfusion, initially described with migraine attacks.[41] Another mechanism is the trigeminovascular firing from dural sinus veins and bridge veins that lead sensitization in central pain pathways.[42] In both IIH and IIHWOP, patients may have dural sinus stenosis. Congestion of the dural sinuses may lead to CGRP - dependent trigeminovascular nociceptive firing, which could result in sensitization of central pain pathways and progression of migraine to its chronic form.[43] There is peptidergic trigeminovascular innervation of the dural sinuses. This feature may lead to function as an intracranial venous pressure sensor, leading to central pain pathway sensitization.[42] Plateau waves consist of a sudden rapid elevation of intracranial pressure (ICP) to 50–100 mmHg for 5 to 20 min. Changes in ICP are thought to produce corresponding changes that include visual obscuration. In this period, patients may experience transient scotomas which last only a few minutes.[44]

Hydrocephalus

New headache or significant worsening of prexisting headache (implies a two-fold or greater increase in frequency and/or severity), accompanid by papiledema may be due to increased intracranial pressure.[5] In hydrocephalus, migraine-like recurrent headache has been reported, such as in slit ventricle syndrome.[18,45] Obstructive hydrocephalus may lead to visual auras as seen in migraine. Idiopathic stenosis of aqueduct may cause episodic severe headaches characterized by temporary scotomas.[46]

Headache attributed to low cerebrospinal fluid (CSF) pressure

Low CSF pressure; either spontaneous or secondary CSF leakage, causes orthostatic headache. Classically, there is a strong relation between headache severity and upright position. Headache significantly worsens soon after sitting upright or standing and/or improves

after lying horizontally.[5] The headache may be throbbing, dull, or pressure-like. The location is usually bilateral, frontal, occipital, fronto-occipital or generalized. Low CSF pressure may lead to a migraine-like headaches with accompanying symptoms such as nausea, vomiting, and sensitivity to light or noise.[18,47] Other symptoms are neck pain, changes in hearing/tinnitus, diplopia, and facial paresthesia.[48] Brain imaging demonstrates brain sagging or pachymeningeal enhancement, or spine imaging (spine MRI, or MRI, CT or digital subtraction myelography) disclose extradural CSF.[5]

Post-traumatic headache (PTH)

Headache is the most common symptom in any acute or chronic phase after traumatic brain injury (TBI). Headaches usually resolve within 6 months. Minority of patients continue to experience headaches, often attributed to PTH. PTH may resemble migraine, tension, or cluster headache.[49]

Due to TBI, microglia are activated. The secretion of CGRP and NO may lead to trigeminovascular system activation and may trigger migraine-like headaches.[50] TBI may induce glutamate release from astrocytes and microglia. Excessive glutamate release may lead to neuronal excitotoxicity, which may trigger CSD and generate central sensitization.[51] CSD has been described in other pathological conditions besides migraine and TBI, such as stroke, epilepsy, and intracranial hemorrhage.[52] Structural brain damage has been described even in minor TBI, and any injury at any stage of pain pathways, including spinothalamic or thalamocortical pathways, may be a source of central pain.[53]

Subarachnoid hemorrhage (SAH)

SAH causes severe "thunderclap" headache.[54] Headache is mostly (55%) reported in occipital location in the SAH group. Headache is described as "stabbing" (35%), meningismus in 80%.[54] Headache may be the only symptom in one third of the SAH patients. Headaches are generally bilateral, and 19% of patients noted gradual increase in severity, as opposed to the thunderclap onset. SAH is usually associated with nausea and vomiting, but 36% of patients do not even have a stiff neck.[55] Twenty five percent of SAH patients may be misdiagnosed as migraine due lack of proper neuroimaging or not performing a lumbar puncture when necessary.[5] SAH may also trigger migraine aura and be relieved transiently by triptans.[55] SAH can mimic so-called "crash migraine" or "migraine with a severe sudden onset", and the "worst migraine ever".[18,56] On the other hand, similar thunderclap headache may be seen in up to 20% of patients with an unruptured cerebral aneurysm. This may result from acute aneurysm expansion in the absence of subarachnoid blood and indicate a higher risk of near future aneurysm rupture.[57,58]

Arteriovenous malformations (AVMs)

Headache is reported in about half of the AVM patients.[59] Patients report headache localized always in the same side, ipsilateral to the AVM. Twenty percent of parenchymal AVMs are located in the occipital lobe, and migraine-like headaches may be present, regardless of whether they are accompanied by visual symptoms.[60] The aura should be differentiated from focal seizure or blood stealing phenomena.[18] Frequency of migraine-like headaches, vomiting, and atypical auras is 10%, shorter headache attacks in 20%, seizures in 25%, and unusual signs in 65%.[61] Non-pulsatile headache was reported in 95% of patients with AVM, while only 4.7% of them had the trio of nausea, vomiting, and phonophobia. Duration of headache episodes was less than 3 h, with a frequency of 1–2 per month.[62]

Cerebral venous thrombosis

Headache is often the only symptom at onset of cerebral venous thrombosis (CVT).[63] In more than 90% of cases, focal neurologic symptoms and / or signs of raised intracranial pressure signs may be present.[5] Headache features that raise suspicion for CVT even in patients without papilledema or focal neurologic findings are recent onset of persistent headache, thunderclap headache, and worsened headache with straining, lying down, or Valsalva maneuvers.[64] Patients may rarely experience migraine-like visual phenomena.[65] In a case series of patients with CVT, 14% reported headache as the only symptom and migrainous features such as throbbing pain in 76%, severe intensity in 76%, unilateral headache in 76% and nausea, vomiting, and/or phono/photophobia in 59%.[66]

Reversible cerebral artery vasoconstriction syndrome (RCVS)

RCVS is characterized by severe thunderclap headache.[9] It may be indistinguishable from migraine. Severe, bilateral, throbbing thunderclap headaches accompanied by nausea, vomiting, and photophobia are the most common presenting features of cases.[18] About two thirds of cases have an underlying cause such as pregnancy, hypertension, or vasoactive substances or drugs.[9]

It may occur spontaneously or triggered by cough, exertion, or Valsalva is experienced over about a week. Visual blurring, scotomas, and transient blindness are also common in RCVS.[18] Vasospasm may also occur during migraine and lead to infarction, and it is impossible to distinguish from RCVS.[67] When stroke or vasospasm are present in multiple arterial territories, the etiology is most likely RCVS or vasospasm due to subarachnoid hemorrhage (SAH).[67]

Stroke-like migraine attacks after radiation theraphy (smart) syndrome

SMART syndrome describes stroke-like migraine attacks that occur after radiation therapy for intracranial neoplasms. There are cases in the literature reporting migraine-like headaches accompanied by neurological deficits, such as visual loss, confusion, hemiparesis, hemisensory changes, and dysphasia. Seizures are also more frequent in SMART syndrome. The duration of neurological findings in this syndrome may vary from less than 2 h to as long as 3 months.[18]

Artery dissection

Headache or neck pain is the only symptom in 8% of spontaneous carotid artery dissection (CAD) patients[68] and thunderclap headache at onset in 20% of patients.[69] CAD can mimic migraine with or without aura, and it may also mimic migraine status.[68] The rapid onset of dull pain may sometimes help to differentiate from migraine, but the presence of accompanying visual aura may resemble migraine. The ipsilateral neck pain to a headache or Horner syndrome is alarming, as the headache may precede ischemic manifestations.[9] In cases with internal carotid artery dissection (ICAD), headache is usually the first presentation of disease, even 4 days before other neurological features and is more often aching type. In these cases, headache is the clinician's most important clue for diagnóstico, as some patients may present with a stroke several days after dissection. However, patients should be questioned about history of chiropractic maneuvers or mild cervical trauma and newly onset pulsatile tinnitus. The headache is often ipsilateral, and it can be felt in the frontal or temporal zones, extend to ipsilateral ear, chin or orbit. Horner syndrome is reported ¼ of patients. ICAD may have migraineous features such as nausea and vomiting, and aura.[18,69] Headaches occur in 70% of vertebral artery dissection (VAD) cases, on average 14.5 h before other neurological features. Usually severe ipsilateral occipitonuchal throbbing pain or pressure headache is reported. Headache is rarely associated with migraine features such as nausea, vomiting, photophobia, phonophobia, or visual aura.[70] VAD is also associated with neck and arm pain, which may mimic the abrupt onset of pain in Parsonage-Turner Syndrome.[71]

Headaches during intracranial endovascular procedures

During therapeutic embolization, segmental vascular stimulation may cause headache. It is sudden and reaches a maximum intensity at the beginning of the procedure. The headache is focal, unilateral, ipsilateral to the occluded artery, non-pulsatile, and short duration. Some patients may report delayed (24–72 h after procedure) or longer headache.[72]

Cerebral autosomal-dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL)

CADASIL is characterized by middle- age onset cerebrovascular disease, and often progresses to dementia due to mutations in the NOTCH3 gene on chromosome 19. Approximately 30% of patients describe typical migraine as the

initial symptom, and 80–90% of those may have migraine with aura.[18,73]

Mitochondrial encephalopathy, lactic acidosis and stroke-like episodes (MELAS)

MELAS is a mitochondrial disorder, migraine-like attacks, encephalopathy, lactic acidosis and stroke-like episodes. The high frequency of migraine-like attacks as a part of MELAS has led to the hypothesis that mitochondrial mutations may play a role in migraine with aura.[5]

Temporal Arteritis (TA)

Temporal arteritis is usually associated with new onset headaches in patients over 50 years of age.[74] Headache may be throbbing or aching, with an acute or subacute onset, and can be persistent or intermittent.[75] Factors such as scalp tenderness, jaw claudication, weight loss, fatigue, and myalgia, elevated ESR, and CRP levels are important clues for the diagnóstico.[9] Sudden visual loss (unilateral or bilateral) accompanied with proximal polymyalgia symptoms reinforces the suspicion of TA.[76] A monocular scintillating scotoma, usually similar to the migraine aura, has rarely been reported.[18]

Nitric oxide (NO) donor - induced headache

Headache can occur immediately or after a delay following acute exposure to an NO donor. Glyceryl-trinitrate (GTN) induced frontotemporal and pulsating headache resolves spontaneously. Tolerance develops within a week, and headaches disappear in most patients. GTN induces immediate headache in most people, but it may cause delayed headache in people with migraine or other types of primary headaches.[5]

Medication - overuse headache (MOH)

Headache occurring on 15 or more days/month in a patient with a pre-existing primary headache and developing as a consequence of regular overuse (> 3 months) of acute or symptomatic headache medication. Medication overuse headache is due to an interaction between an overused therapeutic agent and a susceptible patient who has previously been diagnosed with migraine or tension-type headache. The majority of patients with MOH improve after discontinuation of the overused medication, as does their responsiveness to preventative treatment.[5]

Alcohol — induced headahe

Headache induced by alcohol is usually bilateral and pulsating in quality. Physical activity may aggravate the headache. The immediate headache develops within 3 h of alcohol ingestion and resolves within 72 h, a delayed alcohol headache may develop within 5–12 h after ingestion and resolve within 72 h.[5] The possibility of the diagnóstico of cluster headache should be kept in mind as alcohol may trigger CH attacks.

Caffeine withdrawal headache

Caffeine withdrawal headache develops within 24 h after regular caffeine consumption of more than 200 mg/day if consumed for at least two weeks. It recovers spontaneously within 7 days after cessation of caffeine consumption. Headache may also be relieved within 1 h after 100 mg caffeine intake.[5] Headache is typically diffuse, severe, throbbing and is aggravated by exercise or Valsalva maneuver.[77]

Drug — induced headache

Drug- induced migraine type headaches may be due to sex hormones, including intrauterin device with active hormone delivery and oral contraceptives particularly those with higher estrogen concentration, nitroglycerin, phosphodiesterase inhibitors, calcium-channel blockers, dipyridamole, cyclosporin, interferon-beta, ondansetron, tacrolimus, SSRIs (sertraline, fluoxetine), MAO inhibitors, L-dopa, proton pump inhibitors (omeprazole) and nasal decongestants, etc.[78,79]

Headaches associated with other diseases that may have migrainous features

Epilepsy

Comorbidity of migraine with benign occipital epilepsy or benign rolandic epilepsy is reported.[80] The ICHD-3 lists 3 epilepsy-migraine/headache disorders: hemicrania epileptica, post-ictal headache, and migralepsy.[5,81] Although migraine-like headaches may occur before a seizure, there are also three types of headache associated with epilepsy, including preictal, ictal, and a postictal headache.[82] Ictal epileptic headache is a rare disorder in which migraine or tension- like headache is the only manifestation of seizures. Occipital lobe epilepsies can be associated with migraine-like auras such as visual illusions or vision loss, and approximately half of those with occipital lobe seizures experience post-ictal headache.[83] Unlike migraine aura, epileptic visual hallucinations occur within a few seconds, last a few minutes, and often involve colored and circular illusions.[18]

Acute glaucoma

Acute closed-angle glaucoma is an emergency. Severe pain localized to one eye accompanied by tenderness, blurred vision or vision loss, red eye, or haloes around objects confirm the diagnóstico.[9] Patients present with mydriasis during the pain attack. Rarely, subacute angle-closure glaucoma may mimic migraine with or without aura, which can recur over years.[84]

Arterial hypertension

Hypertension is often associated with a bilateral and pulsating headache, and this pain is related to an acute increase in systolic blood pressure to ≥ 180 mmHg, diastolic

to ≥120 mmHg. It is relieved after normalizing the blood pressure.[18] In cases of new migraine-like headaches or worsening migraine, it is essential to check blood pressure.[9]

Cardiac cephalalgia (CC)

Patients with cardiac cephalalgia (CC) experience unilateral or bilateral migraine-like headache which is usually aggravated by exercise, during an episode of myocardial ischemia. Headache is relieved by nitroglycerine.[5] Cardiac cephalalgia can be the only manifestation of angina in 27% of cases. Thirty % of cases may describe migraine-like features accompanying headache, such as photophobia, phonophobia, osmophobia, and nausea.[85]

Dialysis

Dialysis headache develops during a session of hemodialysis and resolves within 72 h. It associated with hypotension and dialysis disequilibrium syndrome. It can present initially with headache and then progresses to obtundation and finally to coma.[5]

Sleep APNEA

Patients with sleep apnea may experience a morning headache. It is usually bilateral and of pressing quality, with a duration of less than 4 h. The apnea - hypopnea index should be more than 5, and the headache should demonstrate temporal relation with sleep apnea.[5]

Alice in wonderland syndrome (AIWS)

AIWS is a rare migraine aura characterized by an altered body image, as enlarged, diminutive, or distorted. Migraine was reported in 11% of patients, and Epstein–Barr virus in 48% of patients.[86]

Conclusions

There are many types of headaches which may have migraineous features. The diagnóstico of migraine includes "not better accounted for by another ICHD-3 diagnosis". SNNOOP10 list needs to be clarified in each headache patient, at the time of the consultation. Due to the diagnostic challenges, these types of headaches may be associated with high morbidity and/or mortality. The diagnostic errors often lead to therapeutic delay or even malpractice. Thus, it is essential to recognize and differentiate "migraine mimics".

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The authors declare that no experiments were performed on humans or animals for this study.

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Confidentiality of data

The authors declare that they have followed the protocols of their work center on the publication of patient data.

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References

1. Safiri S, Pourfathi H, Eagan A, Mansournia MA, Khodayari MT, Sullman MJM, Kaufman J, et al. Global, regional, and national burden of migraine in 204 countries and territories, 1990 to 2019. Pain. 2022;163:e293–309.
2. Goadsby PJ, Holland PR, Martins-Oliveira M, Hoffmann J, Schankin C, Akerman S. Pathophysiology of migraine: a disorder of sensory processing. Physiol Rev. 2017;97:553–622.
3. IASP. cited; Available from: https://www.iasp-pain.org/publications/iasp-news/iasp-announces-revised-definition-of-pain/July 16, 2020.
4. Do TP, Remmers A, Schytz HW, Schankin C, Nelson SE, Obermann M, Hansen JM, et al. Red and orange flags for secondary headaches in clinical practice: SNNOOP10 list. Neurology. 2019;92:134–44.
5. Headache Classification Committee of the International Headache Society (IHS). The international classification of headache disorders, 3rd edition. Cephalalgia. 2018;38:1–211.
6. Vollesen AL, Benemei S, Cortese F, Labastida-Ramírez A, Marchese F, Pellesi L, Romoli M, et al. Migraine and cluster headache - the common link. J Headache Pain. 2018;19:89.
7. Al-Karagholi MA-M, Peng K-P, Petersen AS, De Boer I, Terwindt GM, Ashina M. Debate: Are cluster headache and migraine distinct headache disorders? J Headache Pain. 2022;23:151.
8. Zidverc-Trajkovic J, Podgorac A, Radojicic A, Sternic N. Migraine-like accompanying features in patients with cluster headache. How important are they? Headache. 2013;53:1464–9.

9. Angus-Leppan H. Migraine: mimics, borderlands and chameleons. Pract Neurol. 2013;13:308–18.
10. Blau JN, Engel HO. Premonitory and prodromal symptoms in cluster headache. Cephalalgia. 1998;18 91–3; discussion 71–2.
11. Torelli P, Manzoni GC. Pain and behaviour in cluster headache. A prospective study and review of the literature. Funct Neurol. 2003;18:205–10.
12. Barloese M, Lund N, Petersen A, Rasmussen M, Jennum P, Jensen R. Sleep and chronobiology in cluster headache. Cephalalgia. 2015;35:969–78.
13. May A, Bahra A, Buchel C, Frackowiak RS, Goadsby PJ. Hypothalamic activation in cluster headache attacks. Lancet. 1998;352:275–8.
14. Maniyar FH, Sprenger T, Monteith T, Schankin CJ, Goadsby PJ. The premonitory phase of migraine–what can we learn from it? Headache. 2015;55:609–20.
15. Snoer A, Lund N, Beske R, Hagedorn A, Jensen RH, Barloese M. Cluster headache beyond the pain phase: A prospective study of 500 attacks. Neurology. 2018;91:e822–31.
16. Kahriman A, Zhu S. Migraine and Tension-Type Headache. Semin Neurol. 2018;38:608–18.
17. Ashina S, Bendtsen L, Lyngberg AC, Lipton RB, Hajiyeva N, Jensen R. Prevalence of neck pain in migraine and tension-type headache: a population study. Cephalalgia. 2015;35:211–9.
18. Evans RW. Migraine mimics. Headache. 2015;55:313–22.
19. Robbins MS, Grosberg BM, Napchan U, Crystal SC, Lipton RB. Clinical and prognostic subforms of new daily-persistent headache. Neurology. 2010;74:1358–64.
20. Liu J, de Luca RD, Mello Neto HO, Barcellos I. Post-COVID-19 Syndrome? New daily persistent headache in the aftermath of COVID-19. Arq Neuropsiquiatr. 2020;78:753–4.
21. Marmura MJ, Silberstein SD. Headaches caused by nasal and paranasal sinus disease. Neurol Clin. 2014;32:507–23.
22. Ng YT, Butler IJ. Sphenoid sinusitis masquerading as migraine headaches in children. J Child Neurol. 2001;16:882–4.
23. Lamonte M, Silberstein SD, Marcelis JF. Headache associated with aseptic meningitis. headache. J Head Face Pain. 1995;35:520–6.
24. Finke C, Mengel A, Pruss H, Stocker W, Meisel A, Ruprecht K. Anti-NMDAR encephalitis mimicking HaNDL syndrome. Cephalalgia. 2014;34:1012–4.
25. Forsyth PA, Posner JB. Headaches in patients with brain tumors: a study of 111 patients. Neurology. 1993;43:1678–83.
26. Schankin CJ, Ferrari U, Reinisch VM, Birnbaum T, Goldbrunner R, Straube A. Characteristics of brain tumour-associated headache. Cephalalgia. 2007;27:904–11.
27. Chen CH, Sheu JJ, Lin YC, Lin HC. Association of migraines with brain tumors: a nationwide population-based study. J Headache Pain. 2018;19:111.
28. Khan RB, Merchant TE, Boop FA, Sanford RA, Ledet D, Onar-Thomas A, Kun LE. Headaches in children with craniopharyngioma. J Child Neurol. 2013;28:1622–5.
29. Levy MJ, Jager HR, Powell M, Matharu MS, Meeran K, Goadsby PJ. Pituitary volume and headache: size is not everything. Arch Neurol. 2004;61:721–5.
30. Gabrielli M, Gasbarrini A, Fiore G, Santarelli L, Padalino C, De Martini D, Giacovazzo M, et al. Resolution of migraine with aura after successful treatment of a pituitary microadenoma. Cephalalgia. 2002;22:149–50.
31. Lim EC, Wilder-Smith EP, Chong JL, Wong MC. Seeing the light: brainstem glioma causing visual auras and migraine. Cephalalgia. 2005;25:154–6.
32. Porta-Etessam J, Berbel A, Martinez A, Nunez-Lopez R. Cerebral metastasis presenting as Valsalva-induced migraine with aura. Headache. 1998;38:801.
33. Schankin CJ, Reifferscheid AK, Krumbholz M, Linn J, Rachinger W, Langer S, Sostak P, et al. Headache in patients with pituitary adenoma: clinical and paraclinical findings. Cephalalgia. 2012;32:1198–207.
34. Yu B, Ji N, Ma Y, Yang B, Kang P, Luo F. Clinical characteristics and risk factors for headache associated with non-functioning pituitary adenomas. Cephalalgia. 2017;37:348–55.
35. Suri H, Dougherty C. Clinical presentation and management of headache in pituitary tumors. Curr Pain Headache Rep. 2018;22:55.
36. Brouwer MC, Coutinho JM, van de Beek D. Clinical characteristics and outcome of brain abscess: systematic review and meta-analysis. Neurology. 2014;82:806–13.
37. Bruera OC, de Lourdes Figuerola M, Gandolfo C, Saggese J, Giglio JA. Status migrainosus: an unusual presentation of a brain abscess. Headache. 1999;39:55–7.
38. Wall M, Kupersmith MJ, Kieburtz KD, Corbett JJ, Feldon SE, Friedman DI, Katz DM, et al. The idiopathic intracranial hypertension treatment trial: clinical profile at baseline. JAMA Neurol. 2014;71:693–701.
39. Friedman DI, Liu GT, Digre KB. Revised diagnostic criteria for the pseudotumor cerebri syndrome in adults and children. Neurology. 2013;81:1159–65.
40. Vieira DS, Masruha MR, Goncalves AL, Zukerman E, Senne Soares CA, Naffah-Mazzacoratti Mda G, Peres MF. Idiopathic intracranial hypertension with and without papilloedema in a consecutive series of patients with chronic migraine. Cephalalgia. 2008;28:609–13.
41. Bateman GA, Stevens SA, Stimpson J. A mathematical model of idiopathic intracranial hypertension incorporating increased arterial inflow and variable venous outflow collapsibility. J Neurosurg. 2009;110:446–56.
42. De Simone R, Ranieri A, Sansone M, Marano E, Russo CV, Sacca F, Bonavita V. Dural sinus collapsibility, idiopathic intracranial hypertension, and the pathogenesis of chronic migraine. Neurol Sci. 2019;40:59–70.
43. De Simone R, Ranieri A, Cardillo G, Bonavita V. High prevalence of bilateral transverse sinus stenosis-associated IIHWOP in unresponsive chronic headache sufferers: pathogenetic implications in primary headache progression. Cephalalgia. 2011;31:763–5.
44. Daley ML, Leffler CW, Czosnyka M, Pickard JD. Plateau waves: changes of cerebrovascular pressure transmission. Acta Neurochir Suppl. 2005;95:327–32.
45. Agarwal N, Vernier E, Ravenscroft S, Schwartz L, Oleske J, Ming X. Slit ventricle syndrome: a case report of intermittent intracranial hypertension. J Child Neurol. 2013;28:784–6.
46. Mucchiut M, Valentinis L, Tuniz F, Zanotti B, Skrap M, Bergonzi P, Zanchin G. Adult aqueductal stenosis presenting as a thunderclap headache: a case report. Cephalalgia. 2007;27:1171–3.
47. Schievink WI, Deline CR. Headache secondary to intracranial hypotension. Curr Pain Headache Rep. 2014;18:457.
48. Kranz PG, Gray L, Malinzak MD, Amrhein TJ. Spontaneous Intracranial Hypotension: Pathogenesis, Diagnosis, and Treatment. Neuroimaging Clin N Am. 2019;29:581–94.
49. Mares C, Dagher JH, Harissi-Dagher M. Narrative review of the pathophysiology of headaches and photosensitivity in mild traumatic brain injury and concussion. Can J Neurol Sci. 2019;46:14–22.
50. Benromano T, Defrin R, Ahn AH, Zhao J, Pick CG, Levy D. Mild closed head injury promotes a selective trigeminal hypernociception: implications for the acute emergence of post-traumatic headache. Eur J Pain. 2015;19:621–8.
51. Maroon JC, Lepere DB, Blaylock RL, Bost JW. Postconcussion syndrome: a review of pathophysiology and potential nonpharmacological approaches to treatment. Phys Sportsmed. 2012;40:73–87.
52. Torrente D, Cabezas R, Avila MF, Garcia-Segura LM, Barreto GE, Guedes RC. Cortical spreading depression in traumatic brain injuries: is there a role for astrocytes? Neurosci Lett. 2014;565:2–6.

53. Defrin R, Riabinin M, Feingold Y, Schreiber S, Pick CG. Deficient pain modulatory systems in patients with mild traumatic brain and chronic post-traumatic headache: implications for its mechanism. J Neurotrauma. 2015;32:28–37.
54. Mac Grory B, Vu L, Cutting S, Marcolini E, Gottschalk C, Greer D. Distinguishing characteristics of headache in nontraumatic subarachnoid hemorrhage. Headache. 2018;58:364–70.
55. Dreier JP, Sakowitz OW, Unterberg AW, Benndorf G, Einhaupl KM, Valdueza JM. Migrainous aura starting several minutes after the onset of subarachnoid hemorrhage. Neurology. 2001;57: 1344–5.
56. Hankey GJ, Nelson MR. Easily missed? Subarachnoid haemorrhage. Bmj. 2009;339, b2874.
57. Day JW, Raskin NH. Thunderclap headache: symptom of unruptured cerebral aneurysm. Lancet. 1986;2:1247–8.
58. Schwedt TJ, Matharu MS, Dodick DW. Thunderclap headache. Lancet Neurol. 2006;5:621–31.
59. Arteriovenous malformations of the brain in adults. N Engl J Med. 1999;340:1812–8.
60. Kupersmith MJ, Vargas ME, Yashar A, Madrid M, Nelson K, Seton A, Berenstein A. Occipital arteriovenous malformations: visual disturbances and presentation. Neurology. 1996;46:953–7.
61. Bruyn GW. Intracranial Arteriovenous Malformation and Migraine. Cephalalgia. 1984;4:191–207.
62. Ghossoub M, Nataf F, Merienne L, Devaux B, Turak B, Roux FX. Characteristics of headache associated with cerebral arteriovenous malformations. Neurochirurgie. 2001;47:177–83.
63. Agostoni E. Headache in cerebral venous thrombosis. Neurol Sci. 2004;25(Suppl 3):S206–10.
64. Timoteo A, Inacio N, Machado S, Pinto AA, Parreira E. Headache as the sole presentation of cerebral venous thrombosis: a prospective study. J Headache Pain. 2012;13:487–90.
65. Newman DS, Levine SR, Curtis VL, Welch KM. Migraine-like visual phenomena associated with cerebral venous thrombosis. Headache. 1989;29:82–5.
66. Cumurciuc R, Crassard I, Sarov M, Valade D, Bousser MG. Headache as the only neurological sign of cerebral venous thrombosis: a series of 17 cases. J Neurol Neurosurg Psychiatry. 2005;76:1084–7.
67. Fisher CM. Late-life migraine accompaniments–further experience. Stroke. 1986;17:1033–42.
68. Arnold M, Cumurciuc R, Stapf C, Favrole P, Berthet K, Bousser MG. Pain as the only symptom of cervical artery dissection. J Neurol Neurosurg Psychiatry. 2006;77:1021–4.
69. Silverman IE, Wityk RJ. Transient migraine-like symptoms with internal carotid artery dissection. Clin Neurol Neurosurg. 1998;100:116–20.
70. Kim JG, Choi JY, Kim SU, Jung JM, Kwon DY, Park MH, Oh K. Headache characteristics of uncomplicated intracranial vertebral artery dissection and validation of ICHD-3 beta diagnostic criteria for headache attributed to intracranial artery dissection. Cephalalgia. 2015;35:516–26.
71. Smith CC, Bevelaqua AC. Challenging pain syndromes: Parsonage-Turner syndrome. Phys Med Rehabil Clin N Am. 2014;25:265–77.
72. Martins IP, Baeta E, Paiva T, Campos J, Gomes L. Headaches during intracranial endovascular procedures: a possible model of vascular headache. Headache. 1993;33:227–33.
73. Liem MK, Oberstein SA, van der Grond J, Ferrari MD, Haan J. CADASIL and migraine: a narrative review. Cephalalgia. 2010;30:1284–9.
74. Smith JH, Swanson JW. Giant cell arteritis. Headache. 2014;54: 1273–89.
75. Ward TN, Levin M, Wong RL. Headache caused by giant cell arteritis. Curr Treat Options Neurol. 2004;6:499–505.
76. Ling ML, Yosar J, Lee BW, Shah SA, Jiang IW, Finniss A, Allende A, et al. The diagnosis and management of temporal arteritis. Clin Exp Optom. 2020;103:572–82.
77. Alstadhaug KB, Andreou AP. Caffeine and primary (migraine) headaches-friend or foe? Front Neurol. 2019;10:1275.
78. Charles A. The pathophysiology of migraine: implications for clinical management. Lancet Neurol. 2018;17:174–82.
79. Ferrari A, Spaccapelo L, Gallesi D, Sternieri E. Focus on headache as an adverse reaction to drugs. J Headache Pain. 2009;10:235–9.
80. Wirrell EC, Hamiwka LD. Do children with benign rolandic epilepsy have a higher prevalence of migraine than those with other partial epilepsies or nonepilepsy controls? Epilepsia. 2006;47:1674–81.
81. Mantegazza M, Cestele S. Pathophysiological mechanisms of migraine and epilepsy: Similarities and differences. Neurosci Lett. 2018;667:92–102.
82. Bauer PR, Carpay JA, Terwindt GM, Sander JW, Thijs RJ, Haan J, Visser GH. Headache and epilepsy. Curr Pain Headache Rep. 2013;17:351.
83. Adcock JE, Panayiotopoulos CP. Occipital lobe seizures and epilepsies. J Clin Neurophysiol. 2012;29:397–407.
84. Molyneux PD, Jordan K. Migraine, an open and shut case? Pract Neurol. 2010;10:172–5.
85. Wei JH, Wang HF. Cardiac cephalalgia: case reports and review. Cephalalgia. 2008;28:892–6.
86. Lanska JR, Lanska DJ. Alice in Wonderland Syndrome: somesthetic vs visual perceptual disturbance. Neurology. 2013;80:1262–4.