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Expert Opinion

Migraine Mimics

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The symptoms of migraine are non-specific and can be present in many other primary and secondary headache disorders, which are reviewed. Even experienced headache specialists may be challenged at times when diagnosing what appears to be first or worst, new type, migraine status, and chronic migraine.

Key words: migraine, migraine mimic, symptomatic migraine, hemicrania continua

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The symptoms of migraine are non-specific and can be present in many other primary and secondary headache disorders.[1,2] Even experienced headache specialists may be challenged at times when diagnosing what appears to be first or worst, new type, migraine status, and chronic migraine. Another diagnosis may be responsible when physicians use the term "atypical migraine."

CASE HISTORIES

Case 1.—This 48-year-old woman was seen for a third opinion with a 20-year history of only menstrual headaches always preceded by a visual aura followed by a generalized throbbing with an intensity of 5–6/10 associated with light and noise sensitivity but no nausea. The headache would last 2–3 days with ibuprofen.

For 3.5 months, she had a daily constant headache, daily since onset, described as a left-sided pressure or throbbing with an intensity ranging from 1 to 10/10 with an average of 6/10 associated with light and noise sensitivity but no nausea, aura, or cranial autonomic symptoms. She had no triggers.

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She had seen 2 headache specialists previously. She had been tried on sumatriptan p.o. and subcutaneously, diclofenac powder, ketorolac oral and intramuscular, dihydroergotamine nasal spray, and had an occipital nerve block without benefit. Gabapentin and pregabalin did not help. She was placed on indomethacin 75 mg sustained release once a day for 8 days without benefit. Prednisone 60 mg daily for 10 days did not help. An intravenous dihydroergotamine regimen for 5 days did not help.

A magnetic resonance imaging (MRI) and magnetic resonance angiogram (MRA) of the brain and cervical spine and magnetic resonance venogram (MRV) of the brain were negative. Blood tests were normal. There was a past medical history of asthma. Neurological examination was normal.

The author placed her on an increasing dose of indomethacin to 75 mg t.i.d. with omeprazole and she became pain free. She has been followed for almost 3 years. The pain resolved for 9 months without medication and then recurred and has been controlled with indomethacin 75 mg daily.

Case 2.—This is a 54-year-old female with a 3-year history of headaches which initially occurred perhaps 2 days per week and then daily and constant for 2 years. She described a left-sided stabbing, pressure, or

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tightness located on left top of the head, left posterior neck, and behind the left eye with an intensity of 6–10 /10 associated with nausea, vomiting (5% of the time), light and noise sensitivity, and intermittent blurry vision. She reported left-sided tearing of the eye, redness, and nasal congestion when the pain was intense.

She had seen 2 neurologists. A MRI of the brain was normal. A erythrocyte sedimentation rate (ESR) was 38 mm per hour (but her body mass index was elevated at 39). She underwent a negative superficial temporal artery biopsy.

Sumatriptan orally and butalbital combination did not help. Intravenous ketorolac and valproic acid daily for 3 days did not help. She was on topiramate 100 mg daily for 2 years and indomethacin 75 mg daily for 5 months without improvement.

There was a past medical history of asthma and hypertension. Neurological examination was normal.

The author titrated up the dose of indomethacin to 75 mg 3 times daily with complete resolution of the headache.

Questions.—What is the cause of these headaches? What are the clinical features and treatment? What are some other primary and secondary migraine mimics?

EXPERT OPINION

Primary Headaches.—*Hemicrania Continua (HC).*—Both cases were misdiagnosed by headache specialists and neurologists, which is easy to do because of the similarity to migraine and also to new daily persistent headache, which can be unilateral in 11% of cases. Case 1 did not have cranial autonomic symptoms absent in about 25% of cases. Case 2 had a mild elevation of the ESR but she was also morbidly obese. The ESR and C-reactive protein can be elevated in obesity.[3] In both cases, indomethacin was tried but underdosed.

In a study of 52 patients with HC, 52% were misdiagnosed with migraine and 40% met migraine criteria during HC exacerbations.[4] The mean time until correct diagnosis was 5 years. The average number of physicians seen before the correct diagnosis was 4.6 with misdiagnoses by a variety of specialists including neurologists and headache specialists. Indomethacin may be underdosed during an indomethacin challenge, which can also lead to misdiagnosis. Other studies have reported a delay until diagnosis of 86.1 months to 12 years[5] and up to 70% meeting migraine criteria during exacerbations.[6] Patients with HC also may undergo unnecessary dental extractions, temporomandibular disorder, or sinus surgery.

HC is a rare disorder that may have a prevalence of up to 1% of the population. HC is more common in females than males, 1.6:1. The onset is often during the third decade of life with a range from the first to seventh decade.

The pain is almost always unilateral although occasionally the pain can switch sides and rare bilateral cases have been reported.[7] The pain is throbbing in 69% and exacerbations of pain have the following triggers: stress, 51%; alcohol, 38%; irregular sleep, 38%; bright lights, 36%; exercise, 31%; warm environment, 28%; skipping meal, 23%; strong smell, 15%; weather change, 13%; tiredness, 13%; and period, 10%.[8] Similar to chronic migraine, 75% have exacerbations of severe throbbing or stabbing pain lasting 20" to several days, which can be associated with photophobia (59%), phonophobia, which is often unilateral (59%), nausea (53%), and vomiting (24%). A visual aura can rarely occur.[9]

Exacerbations can last from 20" to several days with pain awakening 1/3 of patients. Cranial autonomic features are present in up to 75% with tearing and then conjunctival injection the most common compared with 56% of migraineurs. A prior history of migraine is common. Primary stabbing headache or jabs and jolts are reported by 41% especially in exacerbations and about 40% of migraineurs.

HC can be labeled chronic when daily and continuous without pain-free periods for a minimum of 1 year and episodic when there are pain-free intervals of at least a day without treatment. In one series, 82% of cases had chronic (unremitting) HC, which was chronic from the onset in 69%.[8] Evolution from the episodic form occurred in 28% after a latency of 7.9 years (range of 2 weeks to 26 years). Some of the patients with the initial episodic form had headaches that were not daily initially and one patient had about 10 headache days per month. Fifteen percent of patients had the episodic form, which was episodic

from the onset in 33% and evolved from the chronic form in 66%.

Indomethacin responsiveness defines HC during an indomethacin trial with complete headache freedom in divided doses from 50 to 300 mg daily, usually 150 mg/day or less. There are rare reports of response at 300 mg daily. Most patients will respond to an indomethacin trial of increasing the dose if not completely headache free as follows: 25 mg tid for 3 days, 50 mg tid for 3 days, 75 mg tid for 3 days,[10] and 100 mg tid for 3 days.[11] Charlson and Robbins recommend titrating up from 75 mg daily to 150 mg daily to 225 mg daily with each dose tried for 5 days. There are rare reports of response at 300 mg daily.[12]

The lowest effective dose of indomethacin should be used because of the risk of side effects including abdominal pain, dizziness, nausea and/or vomiting, diarrhea, ulcer disease, renal impairment, and association with adverse cardiovascular thrombotic events. Some patients may respond to doses as low as 25–50 mg daily. One study found benefit from a median dose of 61 mg daily when the patients were asked to taper the doses down to lowest effective after 6 months of treatment.[13] Because of the risk of gastroduodenal mucosal injury, indomethacin is typically taken with a proton pump inhibitor.

For patients who respond to indomethacin but have tolerability issues or have contraindications to indomethacin, there are other options which, unfortunately, are not nearly as effective. In one series, greater occipital nerve block and intravenous dihydroergotamine were effective as short-term treatments in 35% and 33%, respectively, and topiramate was effective in 41% for prevention,[8] with 100 to 200 mg daily used in different reports. Melatonin 9-15 mg at bedtime, ibuprofen 1600-2400 mg daily, celecoxib 200 mg bid, botulinum toxin A, verapamil 120–480 mg daily, gabapentin 600-3600 mg daily, pregabalin 150 mg daily, intravenous methylprednisolone, and occipital nerve stimulation have been reported as effective in case reports.[11]

Cluster.—Cluster headaches can have migraine features. Although the duration of untreated attacks is 15 to 180 minutes by International Classification of Headache Disorders-III-beta (ICHD-3) diagnostic criteria,[14] one study found an attack duration of 4 hours or more in patients who met all other criteria.[15] In a prospective study of 155 patients with cluster headache, the following migraine features were reported with each attack: nausea/vomiting, 18.1%; bilateral photophobia, 12.3%; phonophobia, 5.2%; osmophobia, 0.6%; and aura (3 visual, 1 somatosensory), 2.6%. Another series found aura symptoms in 14%.[16] In another prospective study of 209 consecutive cluster patients, light and noise sensitivity were reported by 70% and vomiting or nausea in more than 20%. However, unlike migraineurs, 83% were restless during cluster attacks.

Some patients who have cluster headaches that meet the ICHD-3 duration criteria will provide an inaccurate duration for their headaches, especially when they have multiple headaches per day and give the duration of 2 or 3 headaches combined such as 6 hours.

Cluster-Migraine.—For occasional patients who present with features of migraine and cluster that do not meet ICHD-3 criteria for either or both diagnoses to be present, the terms "cluster-migraine" or "migraine-cluster" have been used.[17]

New Daily Persistent Headache (NDPH).—Anecdotally, primary NDPH is commonly misdiagnosed by neurologists as chronic migraine. *NDPH* is defined as daily from onset with pain becoming continuous and unremitting within 24 hours present for more than 3 months, unlike chronic migraine, which evolves or transforms from episodic migraine and is not daily from onset.[14] Migraine symptoms are common. In one series, throbbing pain, nausea, photophobia, phonophobia, lightheadedness, and aggravation with physical activity were each present in 41-48% with occasional vomiting and rare visual aura.[18] Twenty percent had a pre-existing history of migraine. NDPH with migraine features may respond to the same acute and preventive medications as chronic migraine.

Secondary Headaches.—*Cerebrovascular.*—ARTERIOVENOUS MALFORMATIONS (AVMs).—The prevalence of AVMs is about 0.5% in postmortem studies.[19] In contrast to saccular aneurysms, up to 50% present with symptoms or signs other than hemorrhage. Headache without distinctive

features (such as frequency, duration, or severity) is the presenting symptom in up to 48% of cases.[20]

Migraine-like headaches with and without visual symptoms can be associated with AVMs, especially those in the occipital lobe, which is the predominant location of about 20% of parenchymal AVMs.[21,22] Although headaches always occurring on the same side (side-locked) are present in 95% of those with AVMs, 17% of those with migraine without aura and 15% of patients with migraine with aura have side-locked headaches.[23]

Migraine due to an AVM is usually atypical and rarely meets the International Headache Society criteria for migraine. In a series of 109 patients with headache and AVMs, Ghossoub et al reported the following features: non-pulsating, 95%; nausea, vomiting, light, or noise sensitivity, 4.1%; unilateral and homolateral to the AVM, 70%; duration less than 3 hours, 77%; 1-2 per month, 82.5%; and usually mild responding to simple analgesics.[24] Bruyn reported the following features in patients with migraine-like symptoms and AVM: unusual associated signs (papilledema, field cut, bruit), 65%; short duration of headache attacks, 20%; brief scintillating scotoma, 10%; absent family history, 15%; atypical sequence of aura, headache, and vomiting, 10%; and seizures, 25%.[25]

VASCULAR MALFORMATIONS.—The following brainstem vascular malformations have been associated with migraine meeting International Headache Society criteria: a hemorrhagic midbrain cavernoma resulting in a contralateral headache;[26] a pontine bleed from a cavernous angioma with initially ipsilateral headache then bilateral with aura;[27] pontine capillary telangiectasia with signs of residual hemorrhage with bilateral headaches initially with aura;[28] and a midbrain/upper pons hemorrhagic AVM/cavernous malformation resulting in a contralateral headache with aura.[29] These cases provide evidence for the involvement of the brainstem in the initiation of migraine.

CERVICAL ARTERY DISSECTION (CAD).—Headache or neck pain is the only symptom of spontaneous CAD in 8% and can mimic migraine with and without aura and migraine status.[30] The headache has a thunderclap onset in about 20% of cases. Headache occurs in 60-95% of those with internal carotid artery dissection (ICAD) preceding other neurological symptoms and/or signs by a mean time of 4 days. The pain of ICAD, which is ipsilateral in 91% of cases, is typically localized to the frontal or temporal area, jaw, ear, and/or orbit and is more often aching than throbbing. A partial Horner syndrome occurs in about 25% of cases with ptosis and miosis. ICAD can have migraine features including nausea and vomiting.

ICAD can mimic migraine with aura with a visual aura only or a march of symptoms (such as visual then sensory then dysphasia) associated with a migraine-like headache.[31,32] In occasional cases where migraine with aura criteria are met in those with a prior history of migraine, the dissection could be incidental or could be a trigger for the migraine with aura episode(s).

Headache occurs in 70% of those with vertebral artery dissection (VAD) with head or neck pain preceding other neurological symptoms and/or signs by a mean time of 14.5 hours. VAD is typically a severe ipsilateral occipitonuchal throbbing or pressure but can be bilateral.[33] The headache is rarely associated with migraine features such as nausea, vomiting, photophobia, or phonophobia, and visual aura.[34,35]

CEREBRAL VENOUS THROMBOSIS.—Headache is the most frequent manifestation of cerebral venous thrombosis (CVT) (80-90% of cases), often the only presenting symptom, and can be the only manifestation at onset and during the course of the disease with a normal neurological examination.[36] In a series of 123 patients with CVT, 14% presented with headache as the only neurological symptom and had the following migraine features: throbbing, 76%; severe, 76%; unilateral, 76%; nausea, vomiting, and/or phono/photophobia, 59%.[37] Rarely, associated visual phenomena similar to migraine aura may be present.[38] Headache features that should raise the suspicion of CVT include the following: recent persistent headache, thunderclap headache or pain worsening with straining, sleep/lying down, or Valsalva maneuvers even in the absence of papilledema or focal signs.[39]

REVERSIBLE CEREBRAL ARTERY VASOCONSTRICTION (RCVS).—About 60% of patients develop RCVS postpartum or after exposure to vasoactive drugs (cannabis, ecstasy, selective serotonin reuptake inhibitor [SSRI], triptans, cocaine, amphetamine, intravenous immune globulin [IVIG]),

occurring more commonly in women (3:1) typically presenting between the ages of 20 and 50 years (range 10-76 years).[40] Multiple severe bilateral throbbing thunderclap headaches (average of 4 although single attacks can occur), which can be associated with nausea and/or vomiting and photophobia, are a presenting feature in 94% of cases over a mean period of 1 week that may occur spontaneously or be triggered by cough, exertion, or Valsalva. Visual blurring, scotomas, and blindness are commonly associated. One of the defining features of RCVS is transient cerebral vasoconstriction, which resolves within 1-3 months.

TEMPORAL ARTERITIS (TA).—TA should always be considered with new onset headaches in patients over the age of 50.[41] As the rare exception, in a Canadian study of 141 consecutive patients presenting to a neuro-ophthalmology practice, there was one patient under the age of 50 (age 47).[42] TA has a female : male ratio of 3:1. The incidence increases with older age, occurring in 29.6/100,000 per year in the 70s. Headache is the most common symptoms reported by 72% and the initial symptom in 33%.

The headache may be throbbing or aching with an acute or subacute onset, can be persistent or intermittent, mild to severe intensity, and in any location.[43] A scintillating scotoma most often monocular similar to a migraine aura is rarely reported. So although rare, consider TA as a possible cause in those age 50 or over with new onset migraine-like headaches as migraine starts over the age of 50 in only 2%.

SUBARACHNOID HEMORRHAGE (SAH).—SAH can mimic so-called crash migraine or migraine with a severe sudden onset, severe migraines upon awakening, and the worst migraine ever.[44,45] Headache may be the only symptom of SAH in about one third of patients and is usually bilateral but can be any location, mild at onset, gradually increasing in intensity in 19%, often associated with nausea and vomiting but with the absence of a stiff neck in 36%. SAH can be a trigger for a migrainous aura.[46] The headache of SAH may improve with treatment with triptans.[47]

CEREBRAL AUTOSOMAL-DOMINANT ARTERIOPATHY WITH SUBCORTICAL INFARCTS AND LEUKOENCEPHALOPATHY (CADASIL).—CADASIL is characterized by the middle-aged onset of cerebrovascular disease demonstrated on MRI scans showing white matter hyperintensities with or without lacunar infarctions and microbleeds that often progresses to dementia.[48] CADASIL is caused by mutations in the NOTCH3 gene located on chromosome 19.

About 30% are affected by migraine attacks often as the first symptom with 80-90% having migraine with aura. Of those with migraine with aura, 56% have uncommon features including aura without headache, hemiplegic aura, migraine with brainstem aura, prolonged aura (including one case with prolonged headache[49]), and acute-onset aura.

Vascular.—HYPERTENSION.—An often bilateral and pulsating headache may be associated with an acute rise in a systolic blood pressure to ≥180 mmHg and/or diastolic to ≥120 mmHg, which remits after normalization of blood pressure. Mild or moderate chronic hypertension does not appear to cause headache.

CARDIAC CEPHALALGIA OR ANGINAL HEADACHE.—A unilateral or bilateral headache in any part of the head brought on by exercise and relieved by rest due to cardiac ischemia.[50,51] Pain may occur at rest in cases of unstable angina. Chest pain, pain in the left arm which can radiate to the mandible, or epigastric pain are present in 50% of cases. Cardiac cephalalgia is the only manifestation of angina in 27% of cases. Thirty percent have associated symptoms such as photophobia, phonophobia, osmophobia, and nausea. Thirty-six cases have been reported, 58.3% males, usually over the age of 50 years, although 22% were younger than 50 with the youngest age 35.

Neoplasms.—Migraine or migraine-like headaches with and without aura have been reported in patients with colloid cysts of the third ventricle,[52,53] craniopharyngioma,[54] cerebral metastasis,[55] brainstem glioma,[56] and pituitary tumors.[57,58] Pituitary hemorrhage can produce a migraine-like acute headache with a normal neurologic examination. (Pituitary hemorrhage, even in a macroadenoma, can be overlooked and underimaged on a routine computerized tomography (CT) scan of the head for acute headache using 10-mm and even 5-mm slices. An MRI scan even without pituitary views will routinely identify the pathology.) Pituitary infarction, with severe headache,

photophobia, and cerebrospinal fluid (CSF) pleocytosis, can initially be quite similar to aseptic meningitis or meningoencephalitis.[59] Migraine-like headaches occur in up to 15% of patients with primary and metastatic tumors[60-62] and can present with nonacute migrainous headaches with a normal neurological examination without seizures.[63]

Stroke-Like Migraine Attacks After Radiation Therapy (SMART) Syndrome.—There are about 40 cases reported of migraine-like headaches associated with neurological deficits including dysphasia, visual loss, confusion, hemiparesis, hemisensory change, and/or seizures occurring in children and adults 1-35 years after radiation treatment for intracranial neoplasms lasting less than 2 hours to 2.5 months with full recovery in most, but some have an incomplete recovery.[64,65] MRI shows thick unilateral gyriform cortical enhancement in previously irradiated areas developing after 2-7 days and typically resolving in 14-35 days but can last between 11 and 84 days. The pathophysiology is not known.

Infections.—ACUTE AND CHRONIC ISOLATED SPHENOID SINUSITIS.—Usually causes a frontal, occipital, temporal, vertex, or retro-orbital pain in single locations or a combination of these locations, which is often associated with nausea and vomiting worse with standing, walking, bending, or coughing and not relieved with sleep.[66-68] Fever is present in more than half while purulent nasal discharge or nasal obstruction is present in 40%.

VIRAL MENINGITIS.—Viral meningitis can present with a severe bilateral throbbing headache associated with nausea, vomiting, and photophobia without fever or stiff neck.[69] Recurrent benign meningitis or Mollaret's meningitis, which has been attributed to many different causes, most commonly herpes virus, can occur without fever.[70]

HEADACHE ASSOCIATED WITH NEUROLOGICAL DEFICITS AND CEREBROSPINAL FLUID LYMPHOCYTOSIS (HaNDL or Pseudomigraine With Temporary Neurological Symptoms).— May be caused by activation of the immune system by a viral infection . A viral prodrome is reported in up to 40% of cases.[71] Some 100 cases have been reported ranging in age from 7 to 50 years. The headache is typically throbbing, moderate to severe, and bilateral or hemicranial, which may be associated with nausea, vomiting, photophobia, or phonophobia with a duration of 1 hour to 1 week with a mean of 19 hours. Eighty percent have transient neurological deficits restricted to one hemisphere (75% left hemisphere) and 6% to the basilar artery territory lasting between 5 minutes and 1 week and may recur several times over days to week. The most frequent focal deficits are sensory symptoms, language disorders, and hemiparesis. The lumbar puncture opening pressure is elevated in 60-70%. The cerebrospinal fluid shows a lymphocytic pleocytosis (10-760 cells/mL) and protein is elevated in up to 96% (up to 250 mg/dL). All patients recover completely within 1 to 84 days. Patients with suspected HaNDL syndrome associated with confusion and agitation, which are not typical features, should be tested for neuronal antibodies as anti-N-methyl-D-aspartate receptor encephalitis can mimic HaNDL.[72]

BRAIN ABSCESS.—In a meta-analysis of 9484 patients with brain abscess with a mean age of 33.6 years, headache was present in 69%, fever in 53%, and focal neurological deficits in 48% but all 3 in 20% with a mean duration of symptoms of 8.3 days.[73] Nausea and vomiting was present in 47%. The headache is typically moderate to severe and aggravated by straining or other Valsalva maneuver. Brain abscess can mimic status migrainosus.[74]

High and Low Cerebrospinal Fluid Pressure Disorders.—PSEUDOTUMOR CEREBRI SYNDROME.—In one prospective study of pseudotumor cerebri syndrome, headache was present daily in 73% with an increasing pulsatile headache different and more severe than prior headaches associated with nausea.[75] Seventy percent had headaches with focal unilateral pain (retro-orbital, frontal, temporal, occipital, or vertex pain) but often had a generalized headache as well. The headache has a typically frontal and bilateral location with an intensity that may range from mild to severe.[76] The headache cannot be distinguished from migraine or tension type based solely upon headache features.

In the Idiopathic Intracranial Hypertension Treatment Trial, 41% of patients reported a premorbid history of migraines and 17% had migraine with aura.[77] In another series, there were no features

that distinguished chronic daily headaches due to idiopathic intracranial hypertension (IIH) without papilledema from chronic migraine.[78]

HYDROCEPHALUS.—Obstructive hydrocephalus can cause a migraine-like visual aura [lane] and idiopathic aqueductal stenosis may cause episodic severe headaches with transient visual scotomas.[79] Recurrent migraine-like headaches have been reported in slit ventricle syndrome.[80]

SPONTANEOUS INTRACRANIAL HYPOTENSION.—Can cause a migraine-like headache that can be associated with nausea ± vomiting, light and noise sensitivity.[81,82] Although an orthostatic headache is the most common clinical manifestation, the headache can gradually evolve into a nonorthostatic chronic daily headache or be a nonorthostatic chronic daily headache from onset or be intermittent. The headache may be dull, throbbing or pressure, mild to severe, usually but not always bilateral and can be frontal, fronto-occipital, generalized, or occipital.

Epilepsy.—Migraine-like headaches can precede a seizure, preictal headaches (occur in 5-15% of patients with epilepsy); ictal headache (reported by 3–5% of patients with epilepsy); and can occur immediately after a seizure, a postictal headache (10–50% of patients with epilepsy).[83]

Ictal epileptic headache is a rare disorder where a migrainous or tension-type headache is the sole manifestation of a seizure. Ictal epileptic headache has been reported in patients with focal seizures arising predominantly from the occipital lobes, with nonconvulsive status epilepticus, and in generalized idiopathic epilepsy.

Occipital lobe seizures can resemble a migraine aura with visual hallucinations, illusions, and reduced vision with about 50% having migraine-like post-ictal headaches.[84] However, epileptic visual hallucinations usually occur within a few seconds, last a few minutes, and are usually colored and circular. In contrast, migraine visual aura develops over minutes and typically lasts 5-60 minutes and is often uncolored and linear. In cases difficult to distinguish clinically, an ictal electroencephalogram may be diagnostic.

Glaucoma.—Rarely, with a normal scan of the brain and a white eye, subacute angle closure glaucoma can mimic migraine with and without aura, which can even recur over a period of years.[85]

Alice in Wonderland Syndrome (AIWS).—AIWS syndrome is a rare migraine aura usually where patients experience distortion in body image characterized by enlargement, diminution, or distortion of part of or the whole body, which they know is not real. The syndrome can occur at any age but is more common in children. The cause may be migrainous ischemia of the nondominant posterior parietal lobule.

In a review of 81 cases, migraine was the cause in 11% and Epstein–Barr virus in 48%.[86] There were a number of other causes including other infections, toxic encephalopathy, major depression, epileptic seizures, and medications. Topiramate, used for migraine prevention, is a reversible medication cause of AIWS reported in a 31-year-old female with chronic migraine[87] and a 17-year-old female with episodic migraine.[88]

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