Late-Life Migraine Accompaniments: A Case Presentation and Literature Review

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Migraine headaches that occur in the 15- to 30-year-old age group are well documented. In patients in the stroke age bracket, however, who present with a history of neurologic deficit, transient ischemic attacks can be confused with migraine accompaniments. The typical patient is 50 years old, is without a past history of migraines, and complains of scintillating visual disturbances (20 percent), marching paresthesis (22 percent), or a myriad of neurologic deficits. In one series of 70 neurology patients aged over 55 years, 16 percent reported that they experience the new onset of scintillations. Once fully evaluated, the cause of unexplained marching paresthesias, dysphagia, or hemiplegia, once reserved for thrombotic or embolic phenomena, may be attributed to migraine accompaniments. In the face of a normal evaluation, neurologic deficit in the stroke age bracket may be attributed to migraine accompaniments.

A case of a 47-year-old woman with sudden onset of left-sided paresthesia, dysarthria, and confusion is presented. The discussion includes a description of migraine pathophysiology and a review of concepts regarding accompaniments.

The percentage of family practice patients seen for vascular headaches has not been elucidated; however, vascular headaches are a common problem. The estimated prevalence of migraines in the general population is 10 to 15 percent,[1] and in the United States 1.7 percent of all visits to the family physician's office are for headaches.[2] Among migraineurs, 10 percent will experience a neurologic deficit, or accompaniment. *Migraine accompagneé,* a French term for accompaniment, are neurologic deficits that may occur before, during, after, or without headaches and are often referred to as an "aura."[3] The migrainous nature of the aura is well accepted in the young. Patients aged over 40 years, however, who present with similar phenomena often present a diagnostic puzzle. This patient population commonly presents with symptoms consistent with transient ischemic attacks (TIAs). The family physician must maintain a high level of suspicion for cerebral vascular disease, but migrainous accompaniments, as an explanation of neurologic deficit, should be considered in the differential diagnosis of TIA. Furthermore, the lack of pathologic confirmation and the necessity to rely on clinical features complicate the diagnosis of accompaniments. For this reason, it is important to be aware of the pathophysiology and neuroanatomy of cerebrovascular events so as to direct the investigation and differential diagnosis.

A case of a 47-year-old woman with sudden onset of left-hand paresthesia, dysarthria, and confusion is presented, with emphasis on the pathogenesis and anatomy of *migraine accompagneé.* Fisher[3] has offered a list of 11 diagnostic criteria that may be useful in the evaluation of these patients. With a normal and complete evaluation, the cause of these symptoms, once reserved for cerebrovascular disease, may be attributed to migraine accompaniment.

CASE REPORT

A 47-year-old woman with a past history of hypertension presented to the emergency room after an episode of paresthesia of her left hand and face, dysarthria, and confusion. This episode lasted one-half hour and was associated with a slight, bitemporal headache. Her symptoms resolved spontaneously. She described a similar episode during her menstrual period the previous month but did not seek medical advice.

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The patient was hospitalized and begun on heparin. An angiogram was obtained and was unremarkable. During the angiogram she complained of paresthesias of the left hand and tongue. After she was returned to her room, she became more dysarthric, disoriented, and finally apneic. She was transferred to the Intensive Care Unit, intubated, and monitored. Laboratory evaluation, which included a computerized axial tomography (CT) scan and clotting studies, was unremarkable, and an electroencephalogram revealed diffuse slowing in the right temporal area. The patient returned to normal without any neurologic deficit. After discharge from the Intensive Care Unit, she was observed to become dysarthric, which progressed to disorientation. She was given one sublingual ergotamine tartrate tablet (Ergomar) and she returned to normal. She was subsequently discharged to home with propranolol (Inderal) 20 mg three times daily. Since her discharge she has had similar episodes that were aborted after one sublingual ergotamine tartrate tablet.

PATHOGENESIS AND ANATOMY

The brain is devoid of sensory innervation; however, the meninges and proximal portions of the cerebral vessels are supplied with sensory fibers.[3,4] The accepted mechanism of the migraine is constriction of the cerebral vessels followed by vasodilation. The cycle begins with contraction of smooth muscle in the walls of blood vessels. This ischemic phase is responsible for the aura. Second, a vasodilatory phase, primarily of the external carotid branches, triggers the sensory receptors and is responsible for the headaches.

Studies of regional cerebral hemodynamics conclude that the vasodilatory phase consists of cerebral hyperperfusion that is due to postischemic reactive hyperemia.[4] The cerebral hemodynamics are altered by two mechanisms, exogenous and endogenous factors, which alter monoamine oxidase (MAO) type B in platelets and contribute to vasoconstriction and subsequent dilation. Also, a neurovascular instability may influence the hemodynamics.

The release of serotonin, stored in platelets, enhances capillary dilatation and concomitant constriction of extracranial arteries. The serotonin is metabolized in the tissues, which accounts for the rapid fall in concentration during migraine episodes. The sudden drop causes a rebound vasodilation of the arteries. During this phase, the external carotid may be palpable; however, the blood flow is actually decreased because of edema of the arterial wall. Both the distension and resultant inflammation contribute to the headache.[5,6] It is believed that monoamine oxidase type B in platelets causes serotonin and other stored substances to be released, which contribute to the inflammatory biochemical millieu of substances that aggravate the neurovascular instability.[7] The instability is apparent with the high frequency (20 percent) of electroencephalogram abnormalities and disturbances in the autonomic system.[8] The electroencephalogram characteristically has 3- to 6-Hz slow wave abnormalities and may persist as long as three weeks after a migraine. Excessive slowing with hyperventilation and photoconvulsivity has also been documented, but is less prevalent.

TABLE 1. MIGRAINOUS ACCOMPANIMENTS

Aphasia	Dysarthria
Blindness	Hemianopia
Blurring of vision	Mydriasis
Brain stem symptoms	Oculosympathetic palsy
Chorea	Ophthalmoplegia
Confusion and stupor	Paresthesias
Cyclical vomiting	Recurrence of old stroke deficit
Deafness	Scintillating scotoma
Diplopia	Seizures

From Fisher[3]

SIGNS AND SYMPTOMS

The symptoms of sensory disturbance found in migraine accompaniments depend on the organization of the motor and sensory system; therefore, the cerebral region involved dictates the clinical presentation. For example, 78 percent of patients in a series by Bruyn[9] described a "cheiro-oral" or "digito-lingual" syndrome. The feelings of paresthesias or dysesthesias begin in the fingers and hands, most commonly skip the upper arm and seem to become manifest again around the corner of the mouth, the ipsilateral half of the lips, or the tongue. In fact, sterotaxic procedures with electrostimulation of the ventroposterior nuclear group of the thalamus elicit paresthesias in a cheiro-oral distribution. It is postulated that selective vasospasm of the artery may produce these symptoms. It is conceivable, therefore, that a myriad of neurologic deficits occur depending on the specific artery involved. Vasomotor symptoms, such as rhinorrhea, pallor, and diaphoresis, are well-known autonomic system manifestations during migraine and are manifestations of the neurovascular instability.

Many of the accompaniments have been classified into various groups by Crowel,[8] Bruyn,[9] and Catino.[10] Fisher,[3] also, describes 20 recognized accompaniments to be considered in the evaluation of neurologic symptoms (Table 1).

DIAGNOSIS

Clinical criteria for diagnosis of migraine are well established. A complete evaluation of the differential diagnosis

TABLE 2. DIFFERENTIAL DIAGNOSIS OF MIGRAINE ACCOMPANIMENTS

Diagnosis	Symptoms
Thromboembolic phenomena	Transient ischemic attacks, hyperviscosity, erythrocytosis
Cerebral ischemia	Stroke, vertebral-basilar insufficiency
Intracranial mass	Tumor, arterial-venous malformation, aneurysm, space-occupying lesion
Seizure disorder	
Trauma	
Psychiatric	
Drug intoxication	

TABLE 3. MAIN CRITERIA FOR THE DIAGNOSIS OF LATE-LIFE MIGRAINOUS ACCOMPANIMENTS

Scintillations or other visual display in the spell: next in order, paresthesias, aphasia, dysarthria and paralysis
Building of scintillations (does not occur in cerebrovascular disease)
March of paresthesias (does not occur in cerebrovascular disease)
Progression from one accompaniment to another often with a delay
The occurrence of two or more similar spells (helps to exclude embolism)
Headache in the spell
Episodes last 15 to 25 minutes
Characteristic midlife flurry of migrainous accompaniments
A generally benign course
Normal angiography (excludes thrombosis)
Exclusion of cerebral thrombosis, embolism and dissection, epilepsy, thrombocythemia, polycythemia, and thrombotic thrombocytopenia

From Fisher[3]

and characteristics of recurrent accompaniments must be considered; however, the diagnosis is recognizable if the patient is young and has symptoms that fit into a classical pattern of migraine. In older patients who present without a history of migraine headache, there is a greater possibility of other disease.

The differential diagnosis of *migraine accompagneé* is extensive (Table 2). Primarily accompaniments can masquerade as transient ischemic attacks, and angiography is the cornerstone of the evaluation. To establish a migrainous nature of the episodes, angiography must demonstrate normal cerebral vessels. Although infrequent, results of postmortem studies of patients with complicated migraines are usually normal. A study by Dorfman et al,[11] however, revealed three of 14 patients with *accompagneé* had demonstrated cerebral artery lesions. Unfortunately migraine patients may also have atherosclerotic vascular disease, which complicates the evaluation. The associated atherosclerosis is nevertheless rare, and angiogram remains a valuable tool for diagnosis. In addition, other causes of thromboemobolism, such as hyperviscosity and polycythemia, must be sought.

Finally, seizure disorders, postictal periods, space-occupying lesions, and the post-trauma syndrome can mimic certain migraine accompaniments. The history, CT scan, and electroencephalogram are useful in the evaluation and, in conjunction with other criteria, make it possible to discriminate among disease processes. Eleven such criteria have been useful in the explanation of new-onset neurologic symptoms in the stroke age bracket (Table 3).

The most important criterion is the presence of luminous phenomena. The visual attacks are typically transient, lasting 15 to 25 minutes, and can occur with or without a headache. The presence of homonymous, scintillating phenomena, however, is strong evidence for migraine, as 90 percent of all patients with migraine complain of visual disturbances.[3] The scintillations described by Crowel[8] are perceived as fortification spectra, which begin centrally and enlarge for approximately 15 to 30 minutes. This buildup is not characteristic of calcarine region ischemia that results from thromboembolism. Also, scintillations do not occur in anterior and middle cerebral circulation. To document the scintillations, it is helpful to describe the symptoms as to position, form, motion, color, brightness, expansion, and migration, because many patients will describe the scintillations as atypical such as "foggy vision" or a "kaleidoscope effect."

Also suggestive of migraine is the "march of the paresthesia," described in the cheiro-oral syndrome. The gradual spread is rare in thrombotic disease and is more rapid than the jacksonian march in the generalization of a seizure. The pure sensory stroke of the thalamus may also present with a rapid march, but it is not transitory.

The appearance of a headache is evidence of migraine, especially if it is a throbbing headache with associated photophobia, nausea, and autonomic disturbance. Fisher[3] noted, however, that 50 percent of a series of patients with late-life accompaniments denied the presence of headache. Although it is difficult to consider a migraine without the presence of a headache, a localized vasoconstriction of a small artery supplying the thalamus will result in a large sensory deficit but not necessarily global rebound dilatation, hyperemia, and headache.

The appearance of a midlife flurry of migraine symptoms, especially after a long period of quiescence from childhood migraine, is another clue to the diagnosis. In fact, the occurrence of two or more episodes, particularly if they closely resemble one another, favors migraine over thrombotic vascular disease. The diagnosis can be made even with one episode when the evolution of the accompaniment shows classical buildup, march, and progression, or when the symptoms cross to the opposite side of the body.

Permanent sequelae are rare. Although there are a few cases of stroke associated with prolonged migraine, the

study by Dorfman et al[11] concluded that the incidence is low. In the majority of *migraine accompagneé* the course is usually benign.[11]

TREATMENT

The management of *migraine accompagneé* is aimed at either the vasodilation or vasoconstrictive phase. As previously mentioned, the vasodilation phase is complicated by arterial wall inflammation and edema; therefore, the prophylaxis of *migraine accompagneé* is aimed at the vasoconstrictive phase. The medications available alter serotonin effects as well as inhibit vasodilation. Propranolol hydrochloride (Inderal), the mainstay of prophylaxis, prevents vasodilation and also blocks serotonin receptors. Diamond and Medina[5] found propranolol helpful in the prevention of migraines in 50 percent of their patients. The initial dose is 20 mg three times daily with a maximum of 240 mg/d.

Other antihistamine and antiserotonin medications, such as cyproheptadine (Periactin) 4 to 12 mg daily, amitriptyline (Elavil) 50 to 300 mg daily, and chlorpromazine (Thorazine) 50 to 1,000 mg daily, have been found useful in prophylaxis and are well reviewed in the literature.[4,5,8]

If prevention fails, a trial to abort the aura may be helpful. Abortion is accomplished both with vasodilators and ergot preparations. Vasodilators such as carbon dioxide and amyl nitrate have been used with little effect. Inhalation of 1 percent nebulized isoproterenol may reduce the duration and severity of the accompaniment.[5] Neither have shown to be very effective. The ergot preparations, on the other hand, are effective.[12]

The exact mechanism of ergotamine action is not well understood. Recent studies, however, demonstrate marked regional differences in pharmacologic effects of ergotamine tartrate on cerebral vasculature. Success with prevention and abortion may[1] be due to varied vasoconstriction and dilation of blood flow in the regions of the brain.

SUMMARY

Patients in the stroke age bracket who present with neurologic symptoms are often diagnosed as having transient ichemic attacks. If an angiogram and evaluation are unremarkable, the patient may be discharged without a definitive diagnosis and sometimes placed on anticoagulation therapy. The applications of criteria, presented in this report, permit atypical cases to be attributed to migraine accompaniments. Furthermore, if the pathogenesis and anatomy are understood, evaluation of neurologic deficit is simplified. Many physicians may be hesitant about discharging a patient with new onset of transient neurologic symptoms with a presumptive diagnosis of migraine. With a clinical tool to assist in the diagnosis, a high level of suspicion, and a complete evaluation consisting of angiography, CT scan, electroencephalogram, and blood chemistries, transient deficits can be attributed to migraine accompaniments.

The recognition of new-onset, late-life migraine accompaniments is important for both treatment and diagnosis. Propranolol and antiserotoninergic medications are effective in the prevention of migraines. Ergot preparations are important in the abortion of a migraine. As there is no specific test, a guideline is useful in the diagnosis. Working closely with the neurologist, complicated cases of transient neurologic deficit may be recognized as *migraine accompagneé* and appropriate therapy instituted.

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