# Late-life Migraine Accompaniments in Middle Age and the Elderly

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Of all the neurological disorders, migraine is one of the most common and fascinating. In importance, the modern neurobiology of migraine places it among other major neurological conditions of a paroxysmal nature. Migraine occurs at all ages and is not uncommon in middle- and late-life. It can present with aura and no or little headache, and with or without a prior history of migraine in earlier life. Physicians seeing older patients with transient neurological symptoms and headache need to understand the role of migraine in the diagnosis and ensure no other sinister etiology exists or coexists.

Key words: migraine, aura, migraine accompaniment, acephalgic migraine, headache.

#### Introduction

Clinicians frequently see patients with aura symptoms of migraine who have had no headache or very little headache following the aura. These symptoms are recognised as acephalgic migraine or, literally, migraine without headache. This disorder is classified as "migraine aura without headache" by the International Headache Society, and is a migraine variant or equivalent. In the middle-aged and elderly patient, this disorder is what Fisher called late-life migraine accompaniments.

# **Historical Papers**

In 1980, Fisher reported a series of 120 patients with neurological accompaniments of migraine, classified according to symptoms, as follows: visual symptoms only (25); visual symptoms and paresthesias (18); visual symptoms and speech disturbances (7); visual and brainstem symptoms (14); visual symptoms, paresthesias and speech disturbances (7); visual symptoms, paresthesias, speech disturbances and paresis (25); recurrence of old stroke deficit (9) and; miscellaneous symptoms (8).<sup>2</sup> Headache occurred in 50% of cases reported.

In 1986, Fisher subsequently reported a study of 85 cases that were similarly categorised: visual symptoms (21); visual symptoms and paresthesias (6); visual symptoms and speech distur-

bances (2); visual symptoms, paresthesias and speech disturbances (3); visual symptoms, paresthesias, speech disturbances and weakness (20); visual and brainstem symptoms (3) and; no visual symptoms (32). These cases ranged in age from 40–73 years and 40% had some headache associated with the neurological symptoms, with 65% having a history of recurrent headache. He was particularly interested in patients who had normal cerebral angiograms.

In both of Fisher's papers, a significant number of patients had headache; thus, not all were totally acephalgic or with only aura. The essence of his original communication was to explain why patients in the stroke age-bracket (older than 40 years) occasionally have unexplained transient cerebral ischemic attacks in association with normal cerebral angiograms. He emphasized that these occasional patients probably had migraine aura mainly, although those with headache were more clearly suffering migraine.

Fisher was not the only one to note the phenomena of significant aura symptoms without headache. In 1967, Whitty indicated that migraine auras in middleage could occur without a headache following the aura, and that this was more common in people who had prior migraine in the past.<sup>4</sup>

# Transient Migraine Accompaniments (TMAs)

Today, these late-life migraine accompaniments are considered to be transient migraine accompaniments, or TMAs, and are recognised to be generally benign in nature but are of important diagnostic interest. Migraine with aura (formerly called "classic migraine") is distinguished from migraine without aura (formerly "common migraine") by the neurological symptoms of aura rather than the headache symptoms. Furthermore, headache can occur in patients with transient ischemic attack (TIA) or stroke, 1,5 although this is not always recognised or appreciated.

#### Case Presentation

For five years, a man aged 58 had one or two spells each year of combined visual and speech disturbance. For six months, spells occurred two or three times a week. In a spell, the ability to focus was lost and the lateral parts of his visual field were filled with shimmering pinpoints of light. There was a "build-up" for two to three minutes, the symptoms remained at a peak for 30 minutes, and then faded and disappeared in one hour. At the height of the episode, the patient was unable to read. At the same time, enunciation and fluency of speech were impaired. There were no paresthesias. The patient was aware of a slight pressure in the head at the onset but did not call it a headache.

Interestingly, this is Case 12 from Fisher's original series<sup>2</sup> and is representative of the cases seen with this disorder. This patient had the build-up, progression and march from the neural origin of his visual symptoms in his occipital cortex to the speech symptoms arising from his parietal and temporal speech centres. Headache is not seen in all cases, or it may be mild. In mid-life, as in this case, there can be a "flurry" of TMAs.

#### Mechanisms of TMAs

Although the pathophysiology of TMAs is a matter for speculation, it is

probable that the mechanisms of their production are similar to those of migraine, as they are really migraine variants or equivalents. It seems that TMAs are equivalent to migraine auras, which suggests a neurogenic process of cortical spreading depression originating in the posterior pole of the brain, which has been postulated as a possible cause of reduced cerebral blood flow during a migraine attack.<sup>6,7</sup> It may be that any ischemic mechanism could trigger migraine-like phenomena in a patient with a migraine tendency, but this would still suggest that migraine plays a role at some level. A recent case report of a 65-year-old woman suggests that an internal carotid artery stenosis mimicked a TMA in a patient with a history of acephalgic migraine.8

Furthermore, the ability of blood vessels to dilate may be reduced in the elderly, 9 resulting in the absence of headache and the emergence of TMAs in some cases. Other mechanisms, such as down-regulation of serotonin vascular receptors in menopause, may play a role, as many menopausal women have migraine aura without headache.

# Diagnosis of TMAs

Fisher wrote criteria for late-life migraine accompaniments that continue to be valid today.2 His original criteria are listed in the accompanying Table. These criteria are over 20 years old and would be modified today due to neuroimaging and other tests, but are still applicable. These TMAs display a "build-up" and "progression" of symptoms, which are not typical of stroke symptoms. Further, the "march" of symptoms from one area of the body to another—visual to face to arm to hand, for example—is typical of TMAs but not of stroke. The duration of a TMA march (15-25 minutes) is much longer than a seizure march, which is usually a minute or two. TIAs are usually less than 10 minutes in most patients. 10

Other Diagnostic Considerations

Migraine sensory aura also can involve the tongue and oral mucosa, in contrast to a TIA in which such involvement is rare. 3.11 This is an important clinical clue. As well, TMAs can recur for years without sequelae whereas this would be unlikely in atherothrombotic, embolic or cardioembolic stroke, as would full reversibility of any symptoms. Patients with occipital lobe

strokes, for example, usually have persistent or residual visual symptomology, unlike those who have TMAs.

Migrainous visual accompaniments are not rare in late life. Wijman, et al. 12 designed questionnaires to elicit symptoms of TIAs that also may detect late-life transient visual symptoms similar to the visual aura of migraine, often without headache. In their study, 2,110 subjects were questioned twice yearly for 17 years about visual symptoms. Of these, 26 (1.23%) had migrainous visual episodes; the majority (77%) began after 50 years of age and they were stereotypic in 65% of cases. The episodes lasted 15-60 minutes in 50% of subjects, 58% never had headaches accompanying the episodes, and 42% had no headache history. Twelve percent of subjects sustained a stroke after the onset of migrainous visual symptoms. However, of those with TIAs, 33% developed a stroke, two-thirds within six months of TIA onset. Wijman, et al. concluded that late-life onset transient visual phenomena similar to the visual aura of migraine are not rare and often occur in the absence of headache. These symptoms appear not to be associated with an increased risk of stroke, and invasive diagnostic procedures or therapeutic measures are generally not indicated.

There have been reported cases of permanent sequelae following late-life migraine accompaniments, <sup>13,14</sup> but this would be expected since migraine may on occasion lead to migrainous infarction, <sup>1</sup> and stroke is common in the elderly. There are also secondary brain lesions that can present with migraine-like symptoms, such as vascular malformations or tumour, which must be considered in all cases.

Thus, the most important differential diagnosis of TMAs is TIA or stroke. Usually, the criteria noted can make diagnosis relatively straightforward, especially if the patient had similar symptoms earlier in life or many recent repeated stereotypical episodes. The distinctions can be made but must be done carefully, since risk factors for stroke in these patients can be similar. 15,16 Nevertheless, it is wise to recognise that if there are any atypical features, then stroke or another etiology must be considered.

# Diagnostic Criteria for Late-life Migraine Accompaniments<sup>2</sup>

Scintillations or other visual display in the spell.

- Next in order: paresthesias, aphasia, dysarthria and paralysis.

Build-up of scintillations.

- This does not occur in cerebrovascular disease.

"March" of paresthesias.

- This does not occur in cerebrovascular disease.

Progression from one accompaniment to another, often with delay.

The occurrence of two or more spells. This helps to exclude embolism.

Headache in the spell.

Episodes last 15-25 minutes.

Characteristic mid-life flurry of migrainous accompaniments.

A generally benign course.

Normal angiography.

Exclusion of cerebral thrombosis, embolism and dissection, epilepsy, thrombocytopenia, polycythemia and thrombotic thrombocytopenia.

## Investigation of TMAs

In many patients TMAs are benign events and recognised to be so, and do not require investigation. If necessary, magnetic resonance angiography (MRA), echocardiography, doppler ultrasound or digital angiography can be carried out to exclude a source of cerebral embolism, such as atherosclerotic plaques, dissection of cranial arteries and cardioembolic sources, whereas EEG can help exclude seizures. CT and MRA are also useful in detecting the presence of stroke and TIA mimics, such as a tumour or aneurysm. When indicated, blood tests should be done to exclude platelet disorders, polycythemia and hyperviscosity syndromes, as well as the presence of lupus anticoagulant, phospholipid antibodies, other rare coagulopathies and arteritis if there is an index of suspicion that these disorders may be present. Diagnosing temporal arteritis, which can present with visual symptoms as well as with major systemic symptoms, has to be considered in the differential diagnosis of all elderly patients with headache. If suspected, an ESR should be done and, if necessary, a temporal artery biopsy.

#### Treatment of TMAs

There is no evidence-based information on how to treat TMAs, and they may not need any specific intervention. Standard migraine therapy is suggested if there is headache, although vasoconstricting agents such as ergotamines and triptans should probably be avoided in these age groups, especially if there are any significant cardiovascular or cerebrovascular risk factors. Preventive therapeutic suggestions are anecdotal and have not been validated in large trials.

### Conclusions

The mechanisms of TMAs have interested clinicians for many years, as older patients presenting with TMAs are not uncommon. There is no diagnostic test for migraine, and clinicians need to be very diligent in the differential diagnosis of patients with suspected TMAs, as this diagnosis is always one of exclusion. In concluding his original paper, Fisher stated: "The added knowledge concerning the biology of migraine may even provide new clues as to the mysterious mechanism of migraine."2 Fisher recognised the importance of these cases to "students of migraine", and his descriptions have broadened our understanding of this common neurobiological disorder.

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