Migrainous Aura Versus Transient Ischemic Attack in an Elderly Migraineur

Case History and Follow-up Submitted by Randolph W. Evans, MD
Expert Opinion by Gretchen E. Tietjen, MD

Key words: aura, ischemia, migraine, elderly
Abbreviations: TIAs transient ischemic attacks
(Headache 2001;41:201-203)

In older patients with migraine, the distinction between a migrainous aura and a transient ischemic episode can be difficult, as this case illustrates.

CLINICAL HISTORY
An 80-year-old woman presented for evaluation of spells. Two and one half weeks prior, she was watching television when her left thumb went numb. Sensory loss followed in each finger, with numbness then progressing up the left arm, the left side of the body, and the entire left lower extremity. She could move the left arm, but it was weak. She could not walk. All of the symptoms came on within 1 minute. There was no headache, facial numbness, visual disturbance, speech or language difficulty, or dizziness. The episode lasted for 5 to 10 minutes. Over the next 2 weeks, she had five additional similar spells with complete recovery.

About 2 years ago, she had approximately 10 episodes within a few weeks of numbness of the left face and left side of the body with a weak feeling lasting a few minutes. There is a history of migraine since she was a teenager which still occur from one to three times per month. An attack lasts about 10 hours without medication and a couple of hours after a sumatriptan tablet. She described a throbbing, always on the left side with light and noise sensitivity, but no nausea or any type of aura. In addition, for the last few months, about twice monthly, she developed visual episodes for the first time of zigzags in both eyes, lasting perhaps 8 minutes, without associated headache or other symptoms.

There is a history of hypertension and chronic atrial fibrillation on warfarin for 5 years. There is no history of diabetes or ischemic heart disease. She does not smoke cigarettes. On examination, the blood pressure was 200/70 mm Hg in the left arm, sitting. There were good carotid pulses without bruits. Neurological examination was normal. She was advised to discontinue sumatriptan.

Questions.—What is the diagnosis? Are these episodes migraine auras (late-life migrainous accompaniments) or transient ischemic attacks (TIAs)? How do you distinguish the two? What testing and treatment would you recommend?

EXPERT COMMENTARY
Regardless of a patient’s age, transient focal neurological episodes (TFNEs) present a frequent conundrum for the clinician. Given the absence of radiological and serological markers for migraine, TIAs, and partial seizures, the diagnosis often hinges on the history and on tests which yield only indirect evidence.

Questions.—What is the diagnosis? Are these episodes migraine auras (late-life migrainous accompaniments) or transient ischemic attacks (TIAs)? How do you distinguish the two? What testing and treatment would you recommend?

EXPERT COMMENTARY

In an elderly patient with multiple stroke risk factors (in this case, hypertension and atrial fibrillation), the foremost concern is for TIAs which may portend
a cerebral infarction. Crescendo TIAs, which are all identical, suggest artery-to-artery embolism or thrombosis of a small vessel, rather than cardioembolism. The onset of the symptoms over less than 1 minute and the overall duration of less than 15 minutes are also characteristic of TIAs. The “march” of sensory symptoms is unusual for TIAs, but has been described in persons with cerebral amyloid angiopathy prior to cerebral hemorrhage. It has also been reported to occur in thalamic ischemia and in polycythemia vera. Transient ischemic attacks are not usually associated with headache, but it has been estimated that approximately 20% of persons with TIAs have accompanying headache, especially with posterior fossa ischemia. The absence of headache in association with the spells does not, however, exclude the diagnosis of migraine.

Late-life migraine accompaniments, a term coined by C. Miller Fisher, refers to migrainous TFNEs which are often not associated with headache. According to the Framingham study, these events are not rare, occurring in 1% to 2% of the older adult population, about half of whom have a history of migraine. Nonvisual accompaniments were reported in 20% of Miller Fisher’s series of 120 persons with this condition. He considered the “march” of numbness to be particularly useful in differentiating the spell from TIA, and found it second in frequency only to scintillations as an accompaniment of migraine. This patient’s march is atypical in its brief period of onset and the overall duration. The associated weakness, although not a common aura symptom, has been described. The occurrence of similar stereotypic transient spells 2 years before is evidence to support the likely benign nature of these episodes. Moreover, this woman has recently had recurrent brief visual episodes with features more typical of migraine than TIA. She also has a history of typical migraine beginning in her teen years and lasting to the present. The left-sided headache with an ipsilateral location of paresthesias and paresis makes a fixed area of ischemia or a structural lesion unlikely. Separation of the localization of aura symptoms and headache does occur in migraine, albeit, in the minority of cases. The fact that sumatriptan worked for the headaches also supports a diagnosis of migraine, although it has also been reported to be effective in headaches of other etiologies, including secondary to subarachnoid hemorrhage and meningitis.

The last consideration would be of a partial sensory seizure. New onset of seizures in elderly patients may be secondary to an ischemic focus or tumor, which in this case would involve the sensory cortex. Although I think that these episodes most likely represent late-life migraine accompaniments, I have enough uncertainty based on the clinical description and the other medical history to recommend some diagnostic studies. Brain magnetic resonance imaging (MRI) would rule out tumor, ischemic infarction, or acute and remote hemorrhage (as might be seen in cerebral amyloid angiopathy). Diffusion-weighted imaging identifies newly infarcted tissue and will be transiently positive in some cases of TIA. An MR angiogram will noninvasively rule out extracranial and intracranial arterial stenosis. I would also perform an electroencephalogram (EEG) to evaluate for epileptiform changes. Given that this patient is already on warfarin because of atrial fibrillation, information from an echocardiogram will likely not alter management, unless a significant valvular abnormality is seen. In my experience, migraine prophylactic medication, such as valproic acid or gabapentin, is often effective in stopping migraine accompaniments and could be started if they do not resolve on their own. I would not prescribe triptans or other vasoconstrictive agents.

REFERENCES

5. Wijman CA, Wolf PA, Kase CS, Kelly-Hayes M,


**FOLLOW-UP**

An MRI scan of the brain, with and without contrast, showed nonspecific white matter abnormalities. An MRA of the brain and neck was normal except for tortuosity of the vertebral arteries and irregularity of the right middle cerebral artery just proximal to the trifurcation. An EEG and erythrocyte sedimentation rate were normal. She was evaluated by her cardiologist with no new findings. A 2D echocardiogram showed no evidence of thrombus. On follow-up visit 2 months later, she reported no further episodes.