Migraine aura without headache can be easy to diagnose with an appropriate migraine history. However, the diagnosis is more difficult without an established migraine history, especially during pregnancy.

**CLINICAL HISTORY**

A 30-year-old woman, gravida 1, was seen at 37 weeks’ gestation for evaluation of an episode of visual loss and paresthesias. The evening prior, the patient had a moderate, bifrontal, aching, and throbbing headache without associated symptoms. She took acetaminophen, went to bed, and the headache was gone in the morning. Later that morning, she felt stressed. She developed blurred vision in the left visual field with a scotoma lasting about 30 minutes. One hour later, she had tingling of the left side of the lips for 10 minutes, followed by numbness of the left thumb and index finger spreading to all the fingers which also resolved within 10 minutes. She had no further headaches and felt fine. There was a history of a severe headache with light sensitivity 6 years previously lasting about 1 day after her father died. Otherwise, she had occasional mild headaches without associated symptoms. Past medical history was otherwise unremarkable. She does not smoke. Blood pressure was 100/70 mm Hg. Neurologic examination was normal.

Urine was negative for protein. An MRI of the brain with magnetic resonance angiography (MRA) of the brain and neck and intracranial magnetic resonance venography (MRV) were normal. An ECG and 2D echocardiogram were normal. The following blood studies were normal: complete blood count with platelets, prothrombin time, partial thromboplastin time, chemistry profile, antinuclear antibodies, rheumatoid factor, lupus anticoagulant, anticardiolipin antibodies, protein C, and protein S. An erythrocyte sedimentation rate was 55 mm/hr (frequently elevated during pregnancy).

**Questions.**—Was this episode due to migraine? Can you reliably distinguish between the presentation of a migraine aura and a transient ischemic attack (TIA)? Was the diagnostic testing indicated?

**EXPERT COMMENTARY**

Migrainous sensory aura is at once the boon and bane of the headache specialist. While practically everyone has heard the words *scintillating scotoma* at one time or another, many nonneurologists are ignorant of sensory aura. We consequently can reassure patients referred by worried primary caregivers who mistake sensory aura for a TIA, partial seizure, or something else equally ominous . . . especially when the patient relates to us a well-established antecedent history of migraine, a slow “march” of the sensory symptoms, “tingling” (a “positive” symptom) as well as “numbness” (“negative” symptomatology) and, just for confirmation, temporally associated visual symptoms which precede the sensory symptoms, with the whole
complex followed by a typical migrainous headache. This is migraine, and the diagnosis is made with a simple history and physical examination; carotid duplex, MRI, or MRA/MRV is usually not needed.

But, it is not always that easy. What about the patient who lacks an established history of migraine; who tells you her numbness appeared all at once, with no march and no tingling; who complains of limb heaviness as well as sensory disturbance; whose examination persistently is notable for a subtle downward arm drift, despite normal MRI? These and others are the stories we so often hear, histories which do not fall so neatly into our migraine/sensory aura basket and which necessarily must arouse us to suspect and exclude a nonmigrainous source for the symptomatology expressed. In the case described here, though, I would have been content to make a diagnosis of migraine (with sensory and visual aura) on the basis of the history and physical alone. True, the patient had not experienced five or more attacks of IHS-diagnosed migraine. True, consequent to her pregnancy, this patient’s risk of cerebral venous or arterial thrombosis is increased, and ischemia can induce spreading depression which itself may generate aura and, possibly, migrainous headache. Even so, I find compelling the typical pregnancy-associated eruption of migrainous symptomatology, the sequence and character of the visual and sensory symptoms themselves, and, finally, the normal examination.

If this patient’s headache and aura symptoms persist after pregnancy, should she be prescribed a triptan for acute treatment? Not infrequently at lectures and conferences, primary care physicians have posed questions to me which, by their nature, imply that those physicians are reluctant to prescribe triptans to patients who report typical migrainous visual or sensory aura. This is puzzling and should be addressed directly. Aside from the special circumstance of hemiplegic or basilar migraine, in none of the large-scale clinical trials involving a triptan has the presence of aura represented an exclusion to subject participation. And from none of those trials have we derived information suggesting that patients with aura disproportionately may suffer untoward vasospastic side effects.

We do know that migraine itself conveys roughly a three-fold increase in stroke risk for both men and women between 15 and 45 years of age, and it appears that aura (along with smoking and oral contraceptive use) may further increase that risk. While alarming anecdotal reports of catastrophic triptan-related vascular events occasionally emerge, there is no convincing evidence available to indicate that triptan use adds measurably to the risk of migraine-associated stroke. To the contrary, one large study evaluating the long-term safety and efficacy of repetitive treatment with subcutaneously administered sumatriptan recorded a stroke incidence no greater than would be expected in the migraine population at large. Following pregnancy, and in the event of continued migraine attacks, triptan therapy would be appropriate for the patient reported here.

REFERENCES