

# Sympathomimetic-Induced Kaleidoscopic Visual Illusion Associated With a Reversible Splenium Lesion

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**Background:** Sympathomimetic-induced metabolic derangements within the central nervous system can result in conspicuous changes in neurological functioning and corresponding radiographic abnormalities that can be reversible.

**Objective:** To describe a patient with a “kaleidoscopic” visual illusion who was found by magnetic resonance imaging to have a transient lesion in the splenium of the corpus callosum.

**Design:** Case report.

**Setting:** The University of Texas Southwestern Medical Center, Dallas.

**Patient:** A 17-year-old adolescent girl who developed an episode of kaleidoscopic vision while using sympathomimetic-containing diet pills that was associated with a reversible lesion of the splenium of the corpus callosum. Her brother has a history of migraine and experienced a similar episode while using illicit stimulant agents.

**Intervention:** Withdrawal of the medication resulted in the cessation of the episodes and normalization of the magnetic resonance image.

**Main Outcome Measures:** Clinical and radiographic improvement.

**Results:** Sympathomimetic-induced metabolic derangements can be associated with reversible lesions within the brain.

**Conclusions:** We hypothesize that the visual fragmentation was a manifestation of a migraine triggered by sympathomimetic-containing diet pills, and that the transient lesion in the corpus callosum was a manifestation of a reversible metabolic derangement. Both the visual fragmentation and the lesion in the corpus callosum resolved once the patient stopped receiving diet pills.

*Arch Neurol.* 2006;63:135-137

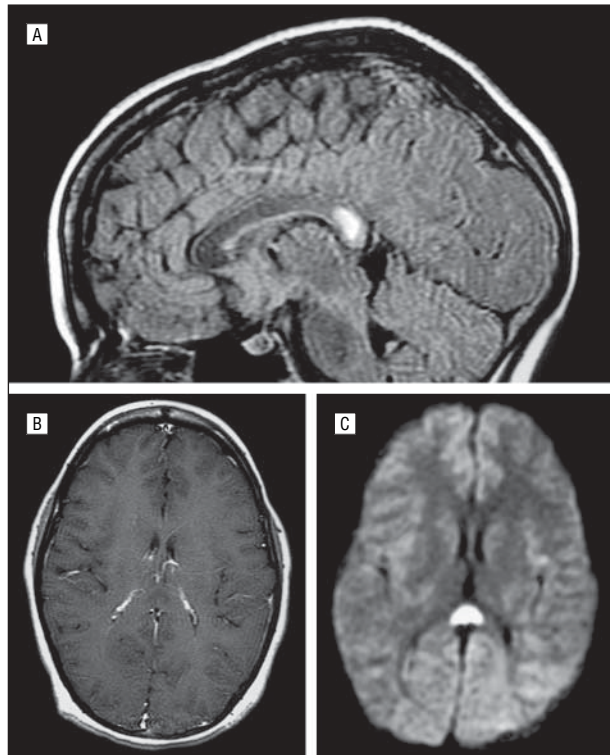
**D**RUGS THAT CONTAIN SYMPATHOMIMETIC agents can induce metabolic derangements within the central nervous system. They thereby result in conspicuous yet reversible alterations in neurological functioning associated with corresponding radiographic abnormalities.

## REPORT OF A CASE

A 17-year-old, right-handed young woman presented with a history of highly unusual yet conspicuous changes in her vision. She described normal vision that was recurrently punctuated by a stereotyped illusion characterized by the perception that she was “looking through a kaleidoscope.” Whole-field visual scenes were fragmented into component parts while the patient was still able to accurately iden-

tify the visual object. This visual fragmentation occurred paroxysmally as stereotyped episodes over a 1-week period, and it involved both eyes. The initial event lasted approximately 60 minutes, and subsequent episodes were substantially shorter yet indistinguishable in character. There were no associated neurological concomitants such as headache, weakness, numbness, or post-ictal fatigue, and there was no significant medical history. She did have a history of a fall from an all-terrain vehicle at age 14 years, but she did not sustain any injuries. Magnetic resonance imaging at an outside institution at the time of initial presentation revealed a solitary, well-circumscribed hyperintensity localized to the splenium of the corpus callosum (CC). The abnormality was identified on T2-weighted, fluid-attenuated inversion recovery, and diffusion-weighted sequences (**Figure 1**). There was no evidence of gadolinium en-

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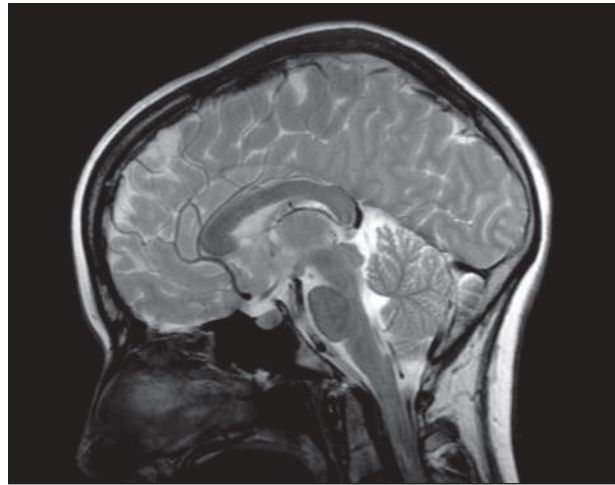


**Figure 1.** Magnetic resonance imaging revealed a conspicuous splenial lesion during the clinical syndrome. A, A sagittal fluid-attenuated inversion recovery magnetic resonance imaging sequence clearly demonstrates a hyperintensity occupying nearly the entire splenium. B, An axial T1-weighted magnetic resonance image with gadolinium infusion fails to show any enhancement. There is, however, a subtle low-density area in the central splenium that is likely suggestive of edema. C, An axial diffusion-weighted magnetic resonance image reveals intense brightness within the splenium.

hancement. A magnetic resonance scan of the cervical and thoracic spinal cord was normal. Cerebrospinal fluid IgG index and synthesis rate were normal, and there was no evidence of oligoclonal bands. Results of a comprehensive metabolic panel including liver functions, electrolyte levels, blood glucose level, complete blood cell count, antiphospholipid antibodies, lupus anticoagulants, protein C and S, antithrombin III, factor V Leiden, VDRL test, cryptococcal antigen, antinuclear antibody, rheumatoid factor, erythrocyte sedimentation rate, Lyme titer, serum protein electrophoresis, and immunofixation were all normal. The differential diagnosis of this lesion included neoplasm, gliosis, traumatic axonal injury, Susac syndrome, a metabolic or infectious derangement, Marchiafava Bignami disease, and demyelination. The patient was referred to our neurological surgery and neurology departments for consultation and further workup.

The patient denied sensory or motor alteration, gait disturbance, and Lhermitte phenomenon. She also denied having any visual disturbances since the episodes that occurred 6 weeks prior to our examination.

Extensive neurological examination demonstrated normal mentation. Visual acuity, confrontational fields, color vision by Ishihara plates, pupillary responses, and fundoscopic examination results were normal. There was no evidence of a relative afferent pupillary defect. The results of the remainder of the examination were normal.



**Figure 2.** A follow-up sagittal fluid-attenuated inversion recovery magnetic resonance image shows nearly complete resolution of the splenial lesion.

Results of laboratory investigations including coagulopathy profile and extensive cerebrospinal fluid analysis with cytology were also normal.

A more extensive history revealed that the patient had been using stimulant-containing diet pills at the time that the visual disturbances occurred. Following discontinuation of this agent, no further episodes of kaleidoscopic visual illusions occurred. A family history of migraine was identified. However, a most intriguing historical point disclosed by our patient's mother was that the patient's brother had previously experienced similar visual disturbances (ie, kaleidoscopic illusions) when he was abusing illicit stimulant agents in the past. With this information, we hypothesized that the visual fragmentation described in our patient was a symptom of a migraine induced by stimulants within the diet pills. Further, we suggested that a complex metabolic derangement was responsible for the lesion within the splenium, and we anticipated that the lesion might resolve spontaneously. A follow-up magnetic resonance image taken at our hospital confirmed our suspicion, demonstrating near-complete resolution of the lesion (**Figure 2**).

#### COMMENT

The prevalence of acquired lesions within the CC has been estimated to be about 3% according to a magnetic resonance imaging study examining 450 patients.<sup>1</sup> The differential diagnostic considerations of CC abnormalities include ischemia, multiple sclerosis, diffuse axonal injury secondary to trauma, hydrocephalus, and tumors.<sup>2</sup> Transient focal lesions of the splenium of the CC also have been described in patients receiving antiepileptic drugs.<sup>3</sup> In contrast to our patient, the described subjects did not show any clinical signs or symptoms related to these lesions. However, similar to our case report, the lesions in these patients resolved after the antiepileptic drug was withdrawn.<sup>3</sup> The transient lesions in the patients with epilepsy were thought to be secondary to vasogenic or cytotoxic edema<sup>3</sup> or to reversible demyelination related to the antiepileptic drugs.<sup>4</sup> The most comprehensive mag-

netic resonance imaging analysis of splenium lesions evaluated 69 patients and found that 28 of them were observed to have complete resolution of the lesion.<sup>5</sup> The most commonly associated symptom was altered mental status. The underlying causes of these lesions were highly diverse, and they spanned all of the major mechanisms of disease.<sup>5</sup> Alterations in commissural cellular fluid mechanics may constitute an injury mechanism germane to many of the causes of splenium lesions. The resolution of the CC lesion in our patient on cessation of stimulant-containing diet pills supports our contention that the lesion was a derivative of a reversible metabolic derangement provoked by a migraine predilection.

Migraines are a common phenomenon and may be associated with a wide array of neurological symptoms.<sup>6</sup> Twenty percent of sufferers have migraine with aura, most typically a visual aura.<sup>7</sup> Most visual auras consist of flickering colored or uncolored lights, zig-zag lines, or patterns. Migraine aura can occur in the absence of headache, in which case it is termed *acephalgic headache*.<sup>7</sup> Migraine triggers are described by 85% of patients. Change in weather, lack of sleep, certain foods, drugs, and trauma are all possible triggers.<sup>8</sup> In our patient, we believe that sympathomimetic drug use may have triggered a migraine aura characterized by a highly complex visual disturbance.

Kaleidoscopic vision fits in the broad spectrum of visual aura that can occur with migraine. The absence of headache does not rule out the diagnosis of migraine. Migraine aura can occur without headache. The patient had a family history of migraine, and the diet pills appeared to be the precipitating factor for producing these complex visual phenomena in a patient with a genetic predilection.

Kaleidoscopic visual illusion and the presence of a transient lesion in the splenium of the CC illustrate an interesting lesson in neuroimaging and migraine. We underscore that transient lesions in the CC can occur secondary to certain drugs, that the lesions can resolve once the drug is discontinued, and that patients with these lesions do not necessarily show any clinical signs or symptoms related to the lesion. The scope of neurological symptoms associated with migraine is broad, and the absence of headache does not rule out the diagnosis (ie, acephal-

gic migraine). What remains unsettled in this patient is the causal relationship between the splenium lesion and the visual disturbance. However, we suggest that the stimulant-containing diet pills triggered an acephalgic migraine with the associated visual changes and the lesion within the CC. Discontinuation of the agent corresponded to resolution of both the visual illusion and the splenium lesion.

**Accepted for Publication:** August 30, 2005.

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**Author Contributions:** *Study concept and design:* Winslow and Frohman. *Acquisition of data:* Winslow, Mickey, and Frohman. *Analysis and interpretation of data:* Mickey and Frohman. *Drafting of the manuscript:* Winslow and Frohman. *Critical revision of the manuscript for important intellectual content:* Mickey and Frohman. *Obtained funding:* Frohman. *Administrative, technical, and material support:* Winslow and Frohman. *Study supervision:* Mickey and Frohman.

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