A 28-year-old woman, nine days postpartum, was admitted to hospital after experiencing bilateral transient visual obscurations every few minutes. These visual disturbances were associated with blurring of vision and scintillating scotomata lasting 5-60 seconds followed by complete resolution of symptoms. She experienced hundreds of episodes per day and she had no other neurological symptoms. She had experienced a normal pregnancy with no obvious hypertension or signs of preeclampsia, and delivered a healthy male infant via an uncomplicated elective C-section for a breech presentation.

Neurologic and ophthalmologic clinical examination were unremarkable, with normal fundi and no evidence of papilledema. Her visual acuity was 20/25 OS, and 20/30 OD. Blood pressure was 123/81 mmHg. She was afebrile. Magnetic resonance (MR) imaging revealed a small 10 mm lesion in the splenium of the corpus callosum, which exhibited restricted diffusion, was correspondingly bright on diffusion imaging, and showed a subtle hyperintensity on T2-weighted and fluid-attenuated inversion recovery (FLAIR) imaging (Figure A). The initial diagnosis was a postpartum ischemic stroke and she was admitted to hospital and anti-platelet therapy was begun. Stroke work-up showed no abnormalities of the extra- or intra-cranial vasculature on MR angiography and venography, and no abnormalities of the heart structure or rhythm. Routine blood tests were within normal limits, complete blood count was normal with no signs of the HELLP syndrome; ESR was 16 mm/h.

Magnetic resonance imaging had been done the same day as presentation. The restricted diffusion was quite intense (ADC = 355 x 10^-6 mm^2/sec), without the expected T2-weighted hyperintensity typically seen at this time point after ischemic stroke. The patient was observed and managed expectantly. Blood pressure values collected during her time in hospital ranged from 112 to 120 mmHg systolic. Her visual symptoms gradually decreased in frequency and then abated over several days. Follow-up MR six weeks later revealed resolution of the splenial lesion (Figure B).

**DISCUSSION**

Transient lesions of the splenium of the corpus callosum have been termed ‘Reversible Splenial Lesion Syndrome (RESLES)’, and are reported to be associated with a wide array of syndromes, diseases, and metabolic disturbances. Patients with hypoglycemia, seizures, anti-epileptic drug (AED) withdrawal or toxicity, vitamin B12 deficiency, and migraine with aura have all been described showing similar transient ovoid lesions in the splenium of the corpus callosum. Despite the large number of reports describing the same findings on MRI, the exact pathogenesis of these reversible splenial lesions is not well understood. The restricted diffusion focused in the splenium may be explained by constrained water flow due to edema between the myelin sheath and the axon, and not due to cytotoxic edema. Diffusion is restricted due to the anatomic directionality of the white matter fibres. Axon integrity is maintained and therefore the restricted diffusion is reversible and associated with symptom resolution.

Preeclampsia and other conditions associated with uncontrolled hypertension tend to affect the posterior white matter most commonly. A similar patient was found to have a reversible splenial lesion in the setting of late postpartum preeclampsia; however in contrast to our case this patient was found to be suffering from marked hypertension. There is an increasing awareness of a more subtle presentation of late postpartum preeclampsia in the literature, made unusual due to a lack of classic signs such as edema, proteinuria, and hypertension. Our patient may have experienced an uncommon variant of postpartum preeclampsia with no detectable typical signs, but with a subtle relative increase in blood pressure.

Although not well documented, our patient’s visual disturbances are a common symptom associated with splenial lesions. Others have reported transient lesions in the splenium of the corpus callosum associated with intermittent visual blurriness, and impaired visual memory. This is consistent with the known role of the splenium in processing and integration of visual information.
Clinicians should recognize that lesions of this type in the splenium may not represent stroke but instead a benign and reversible condition.

REFERENCES


Figure: (Panel A). Initial brain MRI. Diffusion weighted image (DWI), ADC map, FLAIR sequences showing restricted diffusion in the splenium of the corpus callosum with only a faint T2-hyperintensity within the same region. (Panel B). Follow-up MRI six weeks later showing lesion resolution.