and was hypotensive for 5-6 min. After emergency aid, she regained consciousness and complained that she could not see. On neurologic examination, both pupils were isocoric and equally reactive to light and accomodation. There was perception of hand motion. Fundoscopic examination was normal. Cranial magnetic resonance imaging (MRI) revealed hypodense areas involving the occipital lobes bilaterally [Figs. 1, 2a and b]. D-dimer test was positive. Deep vein thrombosis in the right popliteal vein was detected ultrasonographically. In spiral computerized tomography (CT) of the thorax scan, there were patchy consolidation areas. Her electrocardiogram showed sinusal tachycardia with an incomplete right bundle-branch block. There were moderate tricuspid insufficiency and mild pulmonary hypertension echocardiographically. She was diagnosed to have massive pulmonary embolism and cortical blindness, and was admitted to the intensive care unit. Treatment was started with continuous intravenous heparin infusion. The patient's vision gradually began to improve within 24 h. Radiological cure with normal thoracal CT was seen on the seventh day.

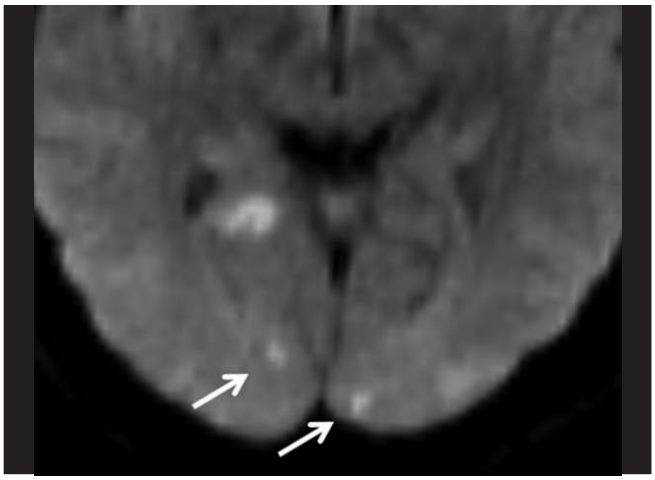

Figure 1: Diffusion-weighted image showing diffusion restriction compatible with the acute infarct involving the striate and extrastriate cortex (arrows)

Transient cortical blindness after spinal surgery as initial presenting sign of hereditary thrombophilia

Sir,

Cortical blindness is a rare complication of spine surgery,[1] and is followed by a period of recovery due to resolution of inflammation and edema around the lesion and to the re-activation of partially damaged perilesional tissue.[2,3] Bilateral occipital abnormalities caused by hypotension, ischemia and infarction are associated with poor prognosis. Herein, we report complete recovery of a case with cortical blindness, despite having an ischemic infarct.

A 33-year-old female patient who was otherwise healthy underwent uneventful simple discectomy operation. In the immediate postoperative period, she had a syncopal attack

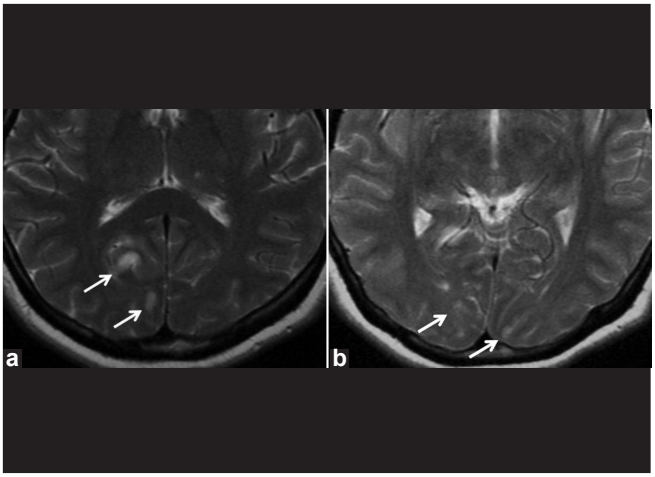

Figure 2: (a and b) Axial T2-weighted images showing hyperintense areas compatible with the acute infarct involving the striate and extrastriate cortex (arrows)

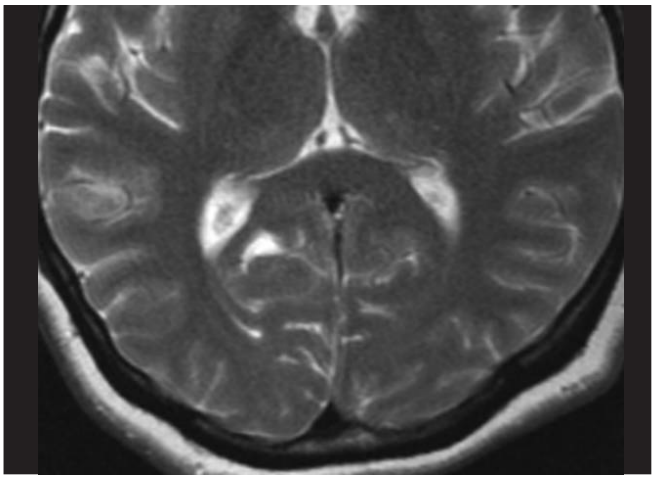

Figure 3: Axial T2-weighted control image showing near-complete regression. There is an encephalomalasic area in the anterior part of the right occipital lobe

Her vision restored to its preoperative level on the 25th day. After 2 months, there were relative deficits in the right lower temporal quadrant and in the left lower hemifield in visual field examination. Contrast sensitivity evaluation showed losses at high frequencies in the right eye and at all spatial frequencies in the left eye. Color vision assessment revealed low pattern discrimination. For excluding any prothrombotic state, a haemotologic evaluation revealed heterozigousity for factor V Leiden R506Q, protrombin G20210A and MTHFRC677T. Anticoagulant therapy was initiated. In the 3rd year follow-up of the patient, near-complete regression of the infarct area was found in the MRI and improvement of visual field defects was observed [Fig. 3]. Color vision was near-perfect in both eyes. Contrast sensitivity was slightly improved.

Visual impairment in cortical blindness is highly variable, and deficits between the hemifields may be different.[4] In our patient, the infarct area was mainly in the cuneus, and corresponding visual field defects were in the lower hemifields. Besides, infarct area comprised both striate and extra-striate visual cortex initially and corresponding abnormalities in color vision and contrast sensitivity recovered with regression of the infarct area.

Occipital lobe infarcts were reported to be frequently associated with a prothrombotic state, and were seen more frequently in younger patients and in patients of the female sex.[5] In young patients with cortical blindness, screening for thrombophilia should be made. Because visual loss may be reversible with initiation of immediate anticoagulation therapy, awareness, evaluation and prompt management of this rare complication is critical.

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