

Homonymous Hemianopia From Infarction of the Optic Tract and Lateral Geniculate Nucleus in Deep Cerebral Venous Thrombosis

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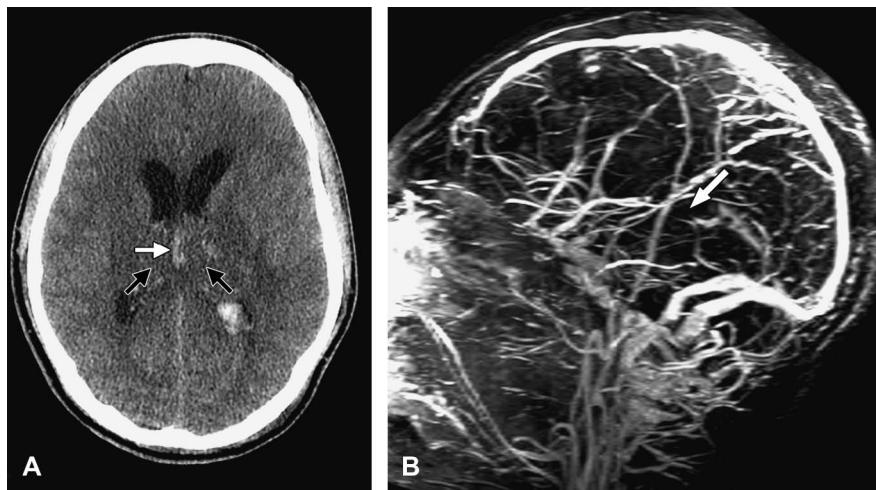


FIG. 1. **A.** Axial precontrast CT performed 5 days after the event demonstrates asymmetric low attenuation of the thalamus (black arrows) with areas of hemorrhage and high attenuation in the region of the internal cerebral veins (white arrow). **B.** Maximum intensity projection image of contrast-enhanced magnetic resonance venogram shows non-opacification of the internal cerebral veins along their expected location (arrow).

Abstract: A 20-year-old man developed right homonymous hemianopia, hemiparesis, and hemisensory loss from deep cerebral venous thrombosis in the setting of high altitude. Approximately 3 months later, brain MRI showed encephalomalacia of the left optic tract and lateral geniculate nucleus, as well as signal abnormalities of the internal capsule and posterolateral thalamus. Homonymous hemianopia has previously been described in 1 case after deep cerebral venous thrombosis but without detailed neuroimaging features.

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CASE REPORT

A 20-year-old man began experiencing intermittent headaches while he was camping in Peru at high altitude of 16,000 feet (4,877 m). After he had descended, the headaches persisted and he felt too ill to eat or drink appropriately. He became progressively confused and somnolent and was found unresponsive. In a local emergency room, hemoglobin was 18.0 g/dL and brain CT showed sagittal sinus thrombosis with bilateral thalamic infarcts.

After transfer to the United States 5 days later, brain CT showed high attenuation in the straight sinus and internal cerebral veins and low attenuation in both thalamus, internal capsules, and the midbrain, signs consistent with thrombosis of the deep cerebral venous system (Fig. 1A). Magnetic resonance venogram at the time showed absent filling of the internal cerebral vein (Fig. 1B). Right lower extremity popliteal deep venous thrombosis was also found on physical examination.

The patient underwent ventriculostomy for hydrocephalus attributed to intraventricular hemorrhage. He was

given heparin as anticoagulation therapy. Within 2 weeks, he began to regain consciousness and was well enough to undergo rehabilitative therapy. He reported being unable to see objects in his right hemifields. All aspects of an ophthalmologic examination were normal, except for confrontation visual fields, which showed a complete right homonymous hemianopia confirmed by automated perimetry (Fig. 2). Neurological examination demonstrated right hemiparesis and right hemisensory deficit. He was found to be heterozygous for the Factor V Leiden mutation and had an elevated Factor VIII level on coagulation workup. Eight weeks later, a right afferent pupillary defect was detected and ophthalmoscopy disclosed bow-tie optic disc pallor in the right eye and temporal optic disc pallor in the left eye. Neurological examination showed dystonic right hemiparesis that was most striking in the hand. He had a right hemisensory loss to pinprick with hyperesthesia on the face and hypesthesia on the right side of his body. Deep tendon reflexes were slightly increased on the right, especially in the upper extremities. He circumducted with the right leg on ambulation.

Brain MRI performed 84 days after the event showed encephalomalacia of the left optic tract (Fig. 3B), lateral geniculate nucleus (Fig. 3C), internal capsule (Fig. 3D), and thalamus (Fig. 3E). Similar, but less marked, changes were noted of the corresponding structures on the right side. This study also demonstrated that the caliber and flow of the

internal cerebral veins was normal, consistent with resolution of the venous thrombosis.

DISCUSSION

We have described a patient who sustained homonymous hemianopia in the setting of deep venous sinus thrombosis at high altitude. This report is distinctive in 2 ways: 1) homonymous hemianopia has only been reported once in deep venous sinus thrombosis (1) and 2) deep venous sinus thrombosis has been rarely reported at high altitude (1).

To our knowledge, the only previously published description of homonymous hemianopia in deep venous sinus thrombosis (2) was part of a small case series in which Case 1 involved a 30-year-old woman with progressive headache and associated emesis who noted "a right visual defect that hindered her walking ability." A "right hemianopia" was found on examination. Subsequent MRI and magnetic resonance venogram demonstrated hemorrhagic infarction of the left thalamus with thrombosis of the deep venous system, including the internal cerebral veins, vein of Galen, and the left transverse and sigmoid sinuses. After 6 months, her "only residual symptoms were fatigue and . . . headache."

Our patient was unconscious for 2 weeks and with recovery had persistent right hemiparesis, hemisensory loss, and a complete right homonymous hemianopia. The visual field defect was accompanied by a right afferent pupil

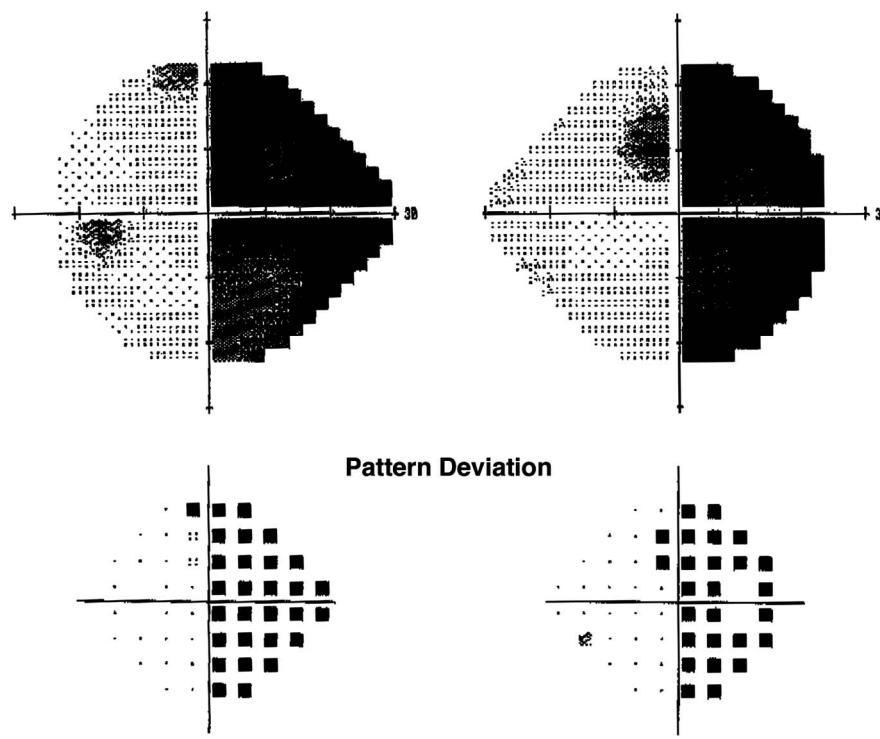


FIG. 2. Automated perimetry reveals a complete right homonymous hemianopia.

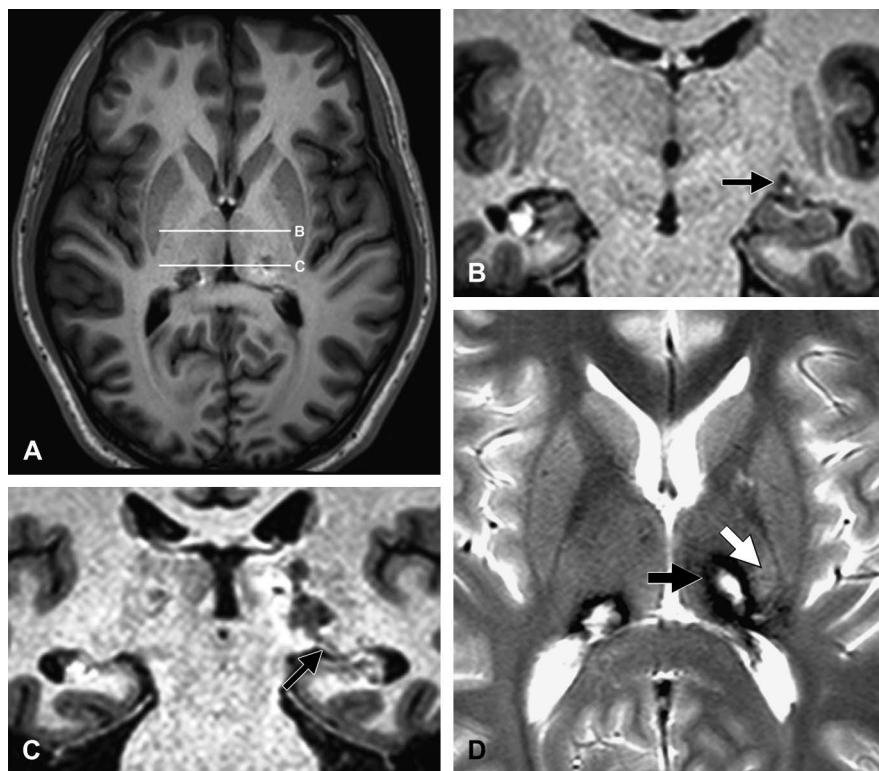


FIG. 3. MRI performed 84 days after the event. **A.** Spoiled gradient T1 axial MRI shows the planes of the coronal images corresponding to **(B)** and **(C)**. **B.** The left optic tract (arrow) has low signal in comparison to the normal right optic tract. **C.** The region of left lateral geniculate nucleus (arrow) has low signal compared to the normal right lateral geniculate nucleus. The low-signal area on the left extends dorsally to encompass the inferolateral thalamus. **D.** T2 axial MRI demonstrates hemosiderin of old hemorrhagic infarction in the thalamus bilaterally, greater on the left side (black arrow) than the right side. A high-signal area is present in the region of the left internal capsule (white arrow).

defect, bow-tie pallor of the right optic disc, and temporal pallor of the left optic disc, findings indicative of left optic tract axonal injury.

The spoiled gradient MRI sequence, which provided high-resolution detail, showed encephalomalacia in the region of the left optic tract and lateral geniculate nucleus (Fig. 3B, C), as well as the posterolateral thalamus, responsible for the hemisensory loss, and the medial internal capsule, responsible for the hemiparesis (Fig. 3D). We believe that such clinical-anatomical correlation has not been previously demonstrated in this setting.

The structures affected in our patient are all drained by the deep cerebral venous system. Specifically, the thalamostriate (terminal) vein drains the internal capsule and is usually the largest tributary of the internal cerebral vein (3). The inferior and posterior thalamic veins drain the posterolateral thalamus and lateral geniculate nucleus and empty into the basal vein of Rosenthal (3). The internal cerebral veins then join the basal veins of Rosenthal to become the major contributors to the great cerebral vein of Galen (3).

Our case is also unusual in that it occurred at high altitude. Venous thrombosis at high altitude has

traditionally involved the *superficial* cerebral venous system (4,5). Fujimaki et al (4) reported a 27-year-old man who developed thrombosis of the vein of Labb   due to high-altitude polycythemia. Song et al (5) described 3 mountain climbers in the third and fourth decades of life who developed thrombosis at various sites: subarachnoid veins, superior sagittal sinus, and transverse sinus.

We were only able to find 1 documented case of *deep* cerebral venous system thrombosis at high altitude (1). It involved a 37-year-old woman who developed dizziness, headache, and vomiting on the fourth day of a mountain-climbing expedition in Nepal reaching a maximum altitude of approximately 4,000 m. Brain CT performed 10 days later demonstrated symmetrical hypodensities of the deep gray nuclei with relative hyperdensity of the internal cerebral veins and straight sinus. She had a right gaze preference but no visual field information was provided.

The contribution of hemoconcentration to cerebral venous thrombosis at high altitude is uncertain. Gradual ascent to an altitude of about 4,500 m does not appear to alter platelet activation, plasma coagulation, or fibrinolysis (6). Hemoconcentration alone is not thought to cause thrombosis. The

slowing of cerebral blood flow secondary to cerebral edema is speculated to play a major pathogenetic role (5).

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