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Choroidal Infarction in Fulminant Idiopathic Intracranial Hypertension

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We report a unique case of idiopathic intracranial hypertension (IIH) in which vision loss likely occurred from a variety of mechanisms, including an acute choroidal infarction.

A 20 year-old white woman complained of severe headaches with nausea, photophobia and pulsatile tinnitus. For the past week, she had noticed rapidly progressive visual loss in both eyes with severe constriction of her peripheral visual field. Review of systems was remarkable only for recent weight gain of 20 pounds over the previous year. On examination, body mass index was 26.6, blood pressure was 110/68 mmHg, and neurologic examination was normal. Visual acuity was 20/20 in the right eye and 20/30 in the left eye. Humphrey and Goldmann visual fields showed bilateral constriction with an inferior nasal defect and enlarged blind spot in the right eye and an inferior altitudinal defect in the left eye. Funduscopic examination (Figure 1) showed bilateral severe optic nerve head edema with peripapillary hemorrhages and white nerve fiber layer infarctions. In the left eye, there was an area of juxtapapillary whitening extending to the macula, which appeared deep in the retina or subretina. A fluorescein angiography (FA) showed normal choroidal, arterial and venous filling 25 seconds after injection of fluorescein and normal retina (no mask effect, no edema), ruling out a branch retinal artery or cilioretinal artery occlusion (Figure 2a). Indocyanine green (ICG) videoangiography (figure 2b) demonstrated a choroidal perfusion defect matching the white patch seen in the left ocular fundus, confirming a choroidal infarction.

We believed the patient's clinical presentation was highly suggestive of raised intracranial pressure (ICP) with severe papilledema complicated by a choroidal infarction in the left eye, and probably also a superimposed anterior ischemic optic neuropathy (AION) in the left eye, given the inferior altitudinal defect. The diagnosis of IIH was confirmed by normal brain magnetic resonance imaging (MRI) and brain magnetic resonance venography (MRV), and elevated CSF opening pressure (560 mm of water) with normal CSF contents¹. Diagnostic lumbar puncture relieved her headaches immediately and she was begun on oral acetazolamide 500mg twice a day. Her visual function, however, remained poor and she

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underwent bilateral sequential optic nerve sheath fenestrations (ONSF) (left eye first) 8 and 21 days later. The headaches did not recur, and her visual function improved over time, especially in the right eye. The disk edema resolved and she developed secondary optic atrophy. The juxtapapillary whitening resolved within two weeks of the ONSF. Her altitudinal defect remained unchanged in the left eye and her visual field improved in the right eye, but remained constricted (Figure 3). Final visual acuity was 20/20 in both eyes.

Comment

This patient developed severe bilateral papilledema in the setting of fulminant IHH² and had visual loss related to visual field constriction from papilledema, to a choroidal infarction, and to possible superimposed AION. Although the anatomy of the short posterior ciliary arteries which vascularize the choroid around the optic disk might logically explain the occurrence of a choroidal infarction in acute and prominent papilledema, we could not find any previous report of such a complication in the setting of severe papilledema. However, five cases with presumed choroidal infarction have been previously reported as a complication of ONSF performed for papilledema.³⁻⁶ Immediate recognition of the mechanism of visual loss in patients with papilledema is essential in making appropriate therapeutic decisions. Choroidal infarction should be added to the list of rare complications of severe papilledema.

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Figure 1. Fundus photography (the right eye is shown on the left and the left eye is shown on the right) showing bilateral optic nerve edema with white exudates. In the left eye, there is an area of whitening extending to the macula and located deeper than the inner retina.

Figure 2.

(a) Fluorescein angiography of the left eye 25 seconds after injection of fluorescein showing normal retinal vascular filling (earlier transit times were not shown and choroidal filling could not be evaluated); (b) Indocyanine angiography of the left eye showing a choroidal filling defect matching the area of whitening seen on fundusoscopic examination.

Figure 3. Humphrey visual fields (24-2 SITA Fast) after ONSF showing persistent constriction and a small paracentral scotoma above fixation in the left eye corresponding to the choroidal infarction.