PHOTO ESSAY

Intraorbital Optic Nerve Signal Hyperintensity on Magnetic Resonance Imaging Sequences in Perioperative Hypotensive Ischemic Optic Neuropathy

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Abstract: A 61-year-old man experienced severe bilateral posterior ischemic optic neuropathy after cardiac bypass surgery. Routine magnetic resonance imaging sequences were normal, but diffusion-weighted and fluid-attenuated inversion recovery (FLAIR) sequences showed abnormal hyperintensity within both intra-orbital optic nerves. This imaging abnormality has not been previously reported in this setting.


A 61-year-old man underwent coronary artery bypass surgery in September 2003, complicated by hemorrhage and prolonged hypotension. On regaining consciousness two days later, he noted profound loss of vision in both eyes. Examination acutely showed normal optic disc appearance. He was treated with high doses of intravenous corticosteroids but experienced no recovery of vision.

Neuro-ophthalmic examination six weeks later revealed no light perception OD and bare light perception OS. Pupils were 6 mm in diameter; the pupil was non-reactive to direct light OD and sluggishly reactive OS with a right relative afferent pupillary defect. Both optic discs were markedly pale.

A fluid-attenuated inversion recovery (FLAIR) magnetic resonance imaging (MRI) sequence performed four days after surgery demonstrated signal hyperintensity within...
FIG. 2. Axial magnetic resonance image performed 4 days after surgery demonstrates an area of hyperintensity in the right basal ganglia on both T2-weighted (A) and diffusion-weighted images (B). This appearance is consistent with subacute infarction.

both optic nerves (Fig. 1A). Restricted diffusion was present within the intra-orbital course of both optic nerves (Fig. 1B). On T2-weighted imaging of the brain, a focus of increased signal was present in the right basal ganglia (Fig. 2A). This region showed restricted diffusion (Fig. 2B).

This case experienced severe bilateral visual loss as a result of retrobulbar optic neuropathy after cardiac surgery. The clinical features in this case are most consistent with perioperative posterior ischemic optic neuropathy (PION), an uncommon but well-described surgical complication (1). Proposed peri-operative risk factors for this event include prolonged systemic hypotension, anemia, and increased orbital venous pressure. Based on post-mortem pathologic examination, the mechanism in postoperative PION involves watershed infarction of the orbital segment of the optic nerves (2,3). This process seems to preferentially affect axial (central) rather than peripheral fibers, affecting the midportion of the nerve most severely (2).

Results of MRI scanning have been normal in most cases of ischemic optic neuropathy, a feature that is sometimes useful for distinguishing between non-arteritic ischemic optic neuropathy (AION) and demyelinating optic neuritis. For example, Rizzo et al (4) found optic nerve enhancement on MRI in 30 (97%) of 31 cases with optic neuritis yet only two (6%) of 32 cases with NAION. Optic nerve enhancement involving the substance of the nerve (5) or occasionally the nerve sheath (16) may be seen in the arteritic form of AION. Radiation optic neuropathy, also thought to represent a form of ischemic insult, is typically associated with optic nerve enhancement (7,8), which may occasionally be seen even before the onset of visual loss (9). A single case of optic nerve enhancement in a case with postoperative PION has recently been reported, but the results of diffusion-weighted imaging (DWI) sequences in that case were not mentioned (10).

In the setting of acute ischemia, cellular energy failure leads to the development of cytotoxic edema, characterized by a shift of water molecules from the extracellular to the intracellular space. The resulting restricted diffusion of these water molecules is displayed as a bright signal on DWI and can be detected within minutes. The imaging findings in our case provide correlation with the previously described pathologic findings in this condition (2). The extent of infarction is well visualized on DWI, and the predilection for the mid-orbital segment with sparing of the immediately retrobulbar portion of the nerve is clearly demonstrated on the FLAIR sequences. We are not aware of similar reports of this imaging abnormality.

REFERENCES


