PHOTO ESSAY

Optic Nerve Enhancement and Restricted Diffusion in Postoperative Visual Loss

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ABSTRACT

Postoperative visual loss is rare and most often due to posterior ischemic optic neuropathy. We describe optic nerve MR imaging of a 37-year-old man with postoperative visual loss due to posterior ischemic optic neuropathy after complicated aortic aneurysm surgery. MR demonstrated restricted diffusion and focal enhancement of both optic nerves. Combined restricted diffusion and focal enhancement is a unique MR imaging feature with postoperative vision loss.

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Optic nerve; posterior ischemic optic neuropathy; watershed; MRI, enhanced; MRI, diffusion; postoperative vision loss

Introduction

Postoperative visual loss (POVL) is a rare complication that effects 0.013% of all surgeries.¹ Usually following cardiac and spine surgery but also associated with a broader spectrum of procedures associated with hypotension and vascular risk factors.^{1,2} The most common cause of severe POVL is posterior ischemic optic neuropathy (PION) that most often is bilateral¹ and is associated with a poor prognosis. MR imaging abnormalities of the optic nerve with POVL are limited to case reports and include restricted diffusion³ and a sole case of optic nerve enhancement.⁴ Our patient uniquely manifest combined restricted diffusion and focal enhancement of the optic nerves.

Case history

A 37-year-old man with a history of hypertension and large vessel vasculitis was transferred to our hospital for an enlarging thoracoabdominal aortic aneurysm concerning for acute aortitis. His vasculitis diagnosis was based on symptoms and elevated inflammatory markers and treated with 1000 mg of pulsed IV methylprednisolone then 1 mg/kg per day. His inflammatory markers returned to normal and treatment continued with Prednisone 10 mg daily. The patient underwent an open repair of the aneurysm and intraoperatively he developed acute hypoxic respiratory

failure complicated by an abdominal compartment syndrome requiring decompressive laparotomy. Postoperatively he required continued sedation and repeat abdominal washouts and extracorporeal membrane oxygenation therapy for 5 days. Upon awakening on postoperative day 8 he was disoriented and complained of not being able to see. The resected aneurysm as well as a subsequent temporal artery biopsy showed no evidence of inflammation or vasculitis.

Examination showed the patient was awake and alert but disoriented to time and place. Visual testing showed no light perception, pupils were 6 mm bilaterally and non-reactive to light. Proptosis was present bilaterally and there was complete ophthalmoplegia of both eyes, vestibulo-ocular reflex was not tested. Remainder of the neurologic examination including fundoscopic exam was unremarkable. On the ninth postoperative day routine brain MR imaging, with 5 mm axial cuts, showed restrictive diffusion of both optic nerves (**Figure 1(a,b)**) and bilateral proptosis and prominent retrobulbar fat was seen on other sequences. Repeat MR on postoperative day 13 showed a focus of increased restricted diffusion in both optic nerves (**Figure 1(c)**). MR on postoperative day 28 with gadolinium showed focal enhancement of both optic nerves, right more than left (**Figure 1(d-f)**).

Figure 1. Top row: (A) diffusion-weighted image (DWI) and (B) apparent diffusion coefficient (ADC) sequences 9 days postoperative showing restricted diffusion along length of optic nerves (arrow) and (C) DWI 13 days postoperative showing persistent focus and increased restricted diffusion (arrow). Bottom row: T1 weighted gadolinium enhanced MR 28 days postoperative (A) axial, (B) sagittal, and (C) coronal images showing focal optic nerve enhancement (arrow).

After an initial encephalopathic period, mental status returned to normal after three weeks. Extraocular movements improved modestly in all directions by the time of discharge at 5 weeks, however, vision remained unchanged.

Discussion

MR defined optic nerve lesions in POVl from PION are rare and include DWI abnormalities³ and focal enhancement.⁴ Ophthalmoplegia is also rare and when present is unilateral.^{5,6} Bilateral ophthalmoplegia as seen in our patient is novel and considered a consequence of the underlying orbitopathy. MR imaging of the orbit

showed prominent retroorbital fat without focal abnormality. Its hypothesized ischemia and its sequelae compromised orbital structures to result in proptosis, ophthalmoplegia and compression of the optic nerve pial vessels.² Histological studies localize PION to involve the vascular supply of the optic nerve in a vulnerable watershed area⁷ (Figure 2). Considering the locus of MR enhancement in our patient and the prior reported case⁴ we suggest a possible correlation of the imaging findings with the watershed area in PION. Combined optic nerve restricted diffusion and focal enhancement seen in our patient has not to our knowledge previously been described with POVl.

Figure 2. Diagram of orbital optic nerve vascular watershed area in posterior ischemic optic neuropathy (arrow). Published with permission.⁷

Declaration of interest

Dr. Finelli has no conflict of interest.

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