REVIEW

Using Diffusion-Weighted Magnetic Resonance Imaging to Confirm a Diagnosis of Posterior Ischaemic Optic Neuropathy: Two Case Reports and Literature Review

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ABSTRACT

Posterior ischaemic optic neuropathy is a rare cause of visual loss believed to be due to infarction in the territory of the pial branches of the ophthalmic artery. The disorder most commonly occurs in the context of prolonged surgery or giant cell arteritis, and the absence of clinical signs in the eye means that the diagnosis is one of exclusion. Here, we present two cases studies of patients who developed posterior ischaemic optic neuropathy confirmed by the observation of secondary changes on diffusion-weighted imaging sequences. In the first case visual loss followed robotic pelvic surgery, and in the second case it was associated with multiorgan dysfunction secondary to severe pancreatitis. Our cases demonstrate that in the right clinical context, diffusion-weighted imaging can provide a positive diagnosis of acute posterior ischaemic optic nerve injury in the acute phase.

Keywords: Diffusion restriction, diffusion-weighted imaging, DWI; PION, posterior ischaemic optic neuropathy

INTRODUCTION

Posterior ischaemic optic neuropathy (PION), first described by Hayreh in 1981,¹ is a rare cause of visual loss² believed to be due to infarction in the territory of the pial branches of the ophthalmic artery that supply the posterior portion of the optic nerve.³ PION can be classified into three main subtypes: arteritic PION (due to giant cell arteritis or other vasculitis), non-arteritic PION, and surgical PION, which is associated with a diverse range of non-ocular surgical procedures,⁴ including spinal surgery^{2,5} and head and neck surgery.^{2,6}

Diffusion-weighted imaging (DWI) is the most reliable magnetic resonance imaging (MRI) sequence capable of mapping ischaemic changes in the central nervous system (CNS)⁷; changes can be identified within minutes of an insult by detecting locally restricted diffusion of water protons.⁸ The optic nerve

is a direct extension of the CNS; thus, the pathophysiology of acute optic nerve ischaemia is analogous to that seen in the brain and spinal cord.⁹ Here, we report two cases that developed PION and secondary changes on DWI in different clinical settings. Previous reports of DWI in PION are reviewed.

CASE REPORTS

Case I

A 74-year-old man was undergoing treatment for high-grade carcinoma in situ of the bladder. His past medical history included diabetes mellitus treated with metformin and gliclazide and hypertension treated with amlodipine and ramipril. He had no significant past ocular history and was independent for all daily activities including driving.

He had previously undergone six treatments with *Bacillus Calmette-Guérin* (BCG) for bladder transitional cell carcinoma (TCC), but subsequently developed recurrent disease. Staging with computed tomography (CT) demonstrated no evidence of metastatic disease. In December 2012 he underwent a robotic cystectomy, intracorporeal ileal conduit, and lymph node dissection under general anaesthesia.

The operative notes documented a routine case aside from an inadvertent injury to the right obturator nerve, which was repaired. The patient was placed in the Trendelenburg position throughout the operation, and there was no specific mention of blood loss and no perioperative blood transfusion. His pre-operative blood pressure was 163/83, which was maintained at 100/60 for 5 hours during the operation. On waking from the general anaesthetic he reported total blindness of his left eye without pain. His mean arterial pressure was 80, heart rate 71, and oxygen saturation was 96%. An immediate ophthalmological consultation found right Snellen visual acuity of 6/12 and left vision of no perception of light with a left afferent pupillary defect. Intraocular pressures were 16 mm Hg on the right and 17 mm Hg on the left. The corneas were clear with deep anterior chambers. Neither fundus showed any abnormality. However, there was considerable facial and periorbital congestion and oedema as is usual following prolonged surgery in the Trendelenburg position.

His immediate preoperative blood results showed haemoglobin (Hb) 10.5 g/dL, creatinine (Cr) 64 µmol/L, and estimated glomerular filtration rate (eGFR) of 106 mL/min/1.73m². The evening after surgery his bloods demonstrated Hb 9.2 g/dL, Cr 161 µmol/L, and eGFR of 37 mL/min/1.73m² and an unchanged urea from the preoperative level. Postoperative lactate was 4 mmol/L. An urgent MRI was obtained, which demonstrated high T2 signal in the posterior part of the left optic nerve on fluid attenuation and inversion recovery (FLAIR) imaging with corresponding restricted diffusion on DWI consistent with acute optic nerve infarction (Figure 1, top line). Although some improvement in his left eye was noted 5 days after the operation with regards to hand movements in the superior fields, 2 months postoperatively visual acuity was 6/12 in the right eye and left eye continued to have no perception of light.

Case 2

A 34-year-old man presented to another hospital with severe pancreatitis and respiratory failure. He was transferred to our hospital for extracorporeal membrane oxygenation. On admission, he had reduced consciousness and a head CT performed on day 1 excluded intracranial haemorrhage and did not reveal

evidence of cerebral infarction. His multiorgan dysfunction progressed, and he suffered a respiratory arrest on day four. After 1 week he improved clinically and was decannulated, but had ongoing difficulties with ventilation and carbon dioxide clearance. Despite being taken off sedation, he showed minimal neurological recovery. On neurological examination, he had an impaired doll's eye reflex and a left relative afferent pupillary defect. The clinical suspicion was one of brainstem and left optic nerve ischaemia, and MRI brain demonstrated bilateral watershed infarcts of the cerebral hemispheres on DWI. The scan also demonstrated high T2 signal intensity in the intraorbital segment of the left optic nerve on FLAIR imaging with corresponding diffusion restriction consistent with optic nerve infarction (Figure 1, bottom line). The patient subsequently died following an 8-day stay in intensive treatment unit (ITU) due to multiorgan failure secondary to severe pancreatitis.

DISCUSSION AND LITERATURE REVIEW

PION classically presents as sudden painless visual loss accompanied by a normal ophthalmological evaluation including the optic disc in the acute phase.⁴ Typically, the visual loss associated with surgical PION is first noticed postoperatively during recovery from general anaesthesia.² Clinically the diagnosis depends on excluding other causes of acute peri-operative visual loss. Unilateral visual loss may be due to central retinal artery occlusion (CRAO), which may be secondary to globe compression in prone surgery, anterior ischaemic optic neuropathy (AION), or acute angle closure glaucoma. Bilateral visual loss may result from any of the above occurring in both eyes but also in bilateral occipital infarction or pituitary apoplexy. These diagnoses should be identifiable by a competent ophthalmic examination, but in PION there are no visible signs other than the absent pupil light reflex.

Ischaemic changes within the CNS results in disruption of normal cellular metabolism and homeostatic mechanisms. Failure of ionic pumps leads to fluid shifts and cytotoxic oedema within the intracellular compartment. As DWI is dependent on the molecular motion of water, intracellular changes result in local restriction of diffusion of water molecules, which can be detected by DWI with high sensitivity and specificity within minutes.^{3,7}

DWI has a central and increasing role in the diagnosis of acute ischaemic lesions within the CNS.¹⁰ A standard brain protocol MRI study (consisting of axial DWI without dedicated thin slices and fat-suppressed T2 or short-tau inversion recovery [STIR]) is commonly used in such situations.

FIGURE 1 Top row showing images from Case 1—coronal T2 FLAIR, DWI, and ADC (apparent diffusion coefficient) maps demonstrate long-segment high T2 signal in the left optic nerve with restricted diffusion (arrows). Bottom row showing images from Case 2—coronal T2-weighted, DWI, and ADC maps demonstrate similar high T2 signal and restricted diffusion in the left optic nerve (arrows).

In some previously reported cases of PION, conventional MRI sequences, such as T1-weighted gadolinium enhanced and spin-echo T2-weighted imaging, have been reported as normal,^{11,12} whereas a number of other case reports in PION^{3,11–14} have shown diffusion restriction on axial DWI with corresponding diffusion restriction seen on apparent diffusion coefficient (ADC) mapping. Both cases presented here displayed high T2 signal within the optic nerves along with the additional finding of restricted diffusion on DWI. The DWI aided in confirming the diagnosis of ischaemia given the particular clinical context.

Al-Zubidi *et al.* point out that susceptibility artefact from adjacent structures and partial volume averaging effect from routine axial DWI acquisition makes discovery of ischaemic changes along the optic nerve difficult to recognise³ and detection may be improved by imaging in the coronal plane using 3-mm slices.^{3,15} The principle behind this theory is that coronal imaging takes a cross-section of the nerve, enhancing spatial resolution for infarct detection, whereas with axial imaging the entire nerve may not be visible on one slice due to slice angulation: there may be partial volume averaging artefacts due to slice thickness

in axial slices. Although coronal slices theoretically give a higher likelihood of detection of ischaemia, there is as yet no strong empirical evidence to confirm this.^{12,14,16}

Restricted diffusion on DWI within the optic nerves has also been shown to be associated with acute inflammatory disorders such as lymphomatous optic neuropathy¹⁷ and optic neuritis.¹⁸ The frequency of restricted diffusion in optic neuritis is not clear; however, one retrospective study found that 8% of patients with clinical optic neuritis showed restricted diffusion on DWI.^{19,20} Furthermore, changes within the optic nerve are more commonly seen with gadolinium enhanced imaging,²⁰ a feature not usually associated with acute ischaemic lesions.

Case 1 reports DWI changes in the optic nerve in the context of PION following robotic cystectomy and demonstrates that in the right clinical context it is possible to positively confirm an ischaemic aetiology; previous cases were diagnosed on clinical grounds rather than with radiological imaging.^{16,21} PION confirmed by restricted diffusion on DWI has previously been reported,^{3,11–14} most commonly following cardiac surgery. Although the exact cause of the

development of PION is not clear, a combination of perioperative risk factors such as profound anaemia, hypotension, increased orbital venous pressure, prolonged operative time, and the steep Trendelenburg position in robotic pelvic surgery are postulated to contribute in susceptible individuals with cardiovascular risk factors.^{5,22}

Case 2 reports PION in the context of multiorgan dysfunction and peri-cardiac arrest secondary to severe pancreatitis. Significant hypotension tests the brain's tolerance to global ischaemia.²³ Hypotension can lead to a decrease in oxygen carrying capacity and in arterial perfusion² and in concert with the delicate blood supply of the posterior portion of the optic nerve, which also lacks an autoregulatory mechanism,²⁴ is the likely underlying pathophysiology explaining the optic nerve infarction in this patient. It has also been suggested that PION can develop through hypoxic oedema, secondary to systemic hypotension, causing compression of pial vessels of the orbital optic nerve.²⁵

Only one optic nerve was affected in this patient, but cases of bilateral PION have been more commonly reported. Similar stress on both optic nerves have been published post-operatively following cardiac transplantation²⁰ and lumbar surgery.²⁶ A number of cases of unilateral PION in association with systemic conditions have previously been reported.^{27–29} Although the definitive explanation for this phenomenon is not clear, it has been postulated that certain individual optic nerves are more or less susceptible to hypotension depending on the individual vascular border zone.^{25,30}

There is currently no effective treatment for surgical PION. Although there is a suggestion that systemic corticosteroids in non-arteritic AION might improve vision,³¹ this is a different process to surgical PION. Suggested treatments have involved high-dose corticosteroid treatment and mannitol and other medications to reduce intraorbital pressure; however, there is no evidence to support any of these interventions.²⁷ It is therefore imperative that appropriate preoperative measures are taken to reduce the risk of the development of PION. These include avoiding peri-operative pressure on the eyeball and orbit, dependant positioning and avoiding arterial hypotension, markedly prolonged operative times, and excessive fluid replacement.⁴

Our cases demonstrate that in the right clinical context, DWI can provide a positive diagnosis of acute posterior ischaemic optic nerve injury that has previously been a diagnosis of exclusion.¹ Furthermore, in cases of high-risk surgery such as spine surgery and robotic pelvic surgery, awareness of PION as a rare but devastating entity is important and the threshold for including DWI sequences as part of neuroimaging in suspected PION should be low. An objective positive confirmation of the diagnosis can be very helpful, particularly in the difficult post-operative

situation where loss of vision has occurred following a surgical procedure with no direct relationship to vision.

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