Case Report

Postoperative posterior ischemic optic neuropathy (PION) following right pterional meningioma surgery

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Abstract

Postoperative visual loss (POVL) is an unpredictable complication of nonocular surgeries. Posterior ischemic optic neuropathy (PION) is particularly feared in spinal surgeries in the prone position. We report a rare case of PION occurring after surgery for a pterional meningioma and discuss the various factors implicated in POVL.

Key Words

PION, Post op blindness, meningioma and PION

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Introduction

Postoperative visual loss (POVL) occurring in nonocular surgeries is a catastrophe with poor prognosis in most cases.[1] The main causes are ischemic optic neuropathies [anterior ischemic optic neuropathy (AION) or posterior ischemic optic neuropathy (PION)], central retinal artery occlusion (CRAO), pituitary apoplexy, and occipital infarction. The risk has been estimated to be around 0.013-0.2% and is higher for spinal surgeries.[2] PION is more common with spinal surgeries or radical neck dissections, whereas AION is more common with cardiac surgeries. We present a case of PION occurring after nonocular surgery (pterional meningioma excision).

Case Report

A 56-year-old man was admitted for elective surgery for an incidental right pterional meningioma. The meningioma was detected when he underwent neuroimaging for anosmia [Figure 1]. On examination, there were no focal deficits and visual acuity and fields were normal. Preoperative routine investigations were within normal limits. He underwent a right pterional incision in the supine position with head tilt to the left side with excision of tumor (Simpsons grade 2 excision) under general anesthesia (GA). Intraoperatively, there was an extraxial lesion seen in the pterional region with attachment to the dura overlying the lateral sphenoid wing, with feeders from the middle meningeal artery. There was no well-defined plane of cleavage from the surrounding brain parenchyma except at some areas in the sylvian fissure. A bone flap was raised and the sphenoid wing was drilled to open the superior orbital fissure. The dura was opened and the tumor was gently decompressed. A small part of the tumor, which was adherent to sylvian vessels was left behind. Hemostasis was attained and the dura was closed with the pericranium and fascia lata. He was extubated within hours of the surgery and started following commands. Overnight, he complained of painless loss of vision in the right eye. On examination, there was a right afferent papillary defect and absence of light perception in the right eye. Emergent computed tomography (CT) of the brain showed only postoperative tumor bed changes. Magnetic resonance image (MRI) showed diffusion restriction

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in the right-sided intraorbital segment of the optic nerve (ON). The intraorbital segment of the right ON appeared hyperintense and slightly edematous. The optic chiasm appeared normal. MRA source images clearly demonstrated a patent right ophthalmic artery till the mid-intraorbital optic nerve. There were postoperative changes with hemosiderin staining and minimal mass effect on the right side with a midline shift of 3 mm [Figure 2]. The MRI findings were consistent with a right-sided PION with optic nerve infarction. Visual evoked potentials showed absent potentials from the right eye. Fundoscopy on day 5 was normal. He was started on intravenous (IV) methylprednisolone 1 g/day over 5 days. At discharge 1 week later, he had not regained light perception in the right eye.

Discussion

POVL with nonocular surgery is a feared and unforeseeable complication [Table 1]. Numerous causes are associated with POVL and practice advisories to offset this catastrophe have been enumerated by societies [Table 2]. PION has been most associated with spine surgery in the prone position; postulated to be due to factors such as increased orbital venous pressure, direct ocular pressure, or increased intraocular pressure. During radical neck dissection, ligation of the internal jugular veins is postulated to lead to distension of ophthalmic veins and compression of the orbital apex with ischemia of the posterior optic nerve (ON).[3]

The peculiarities in the blood supply of the ON result in various clinical presentations. The optic nerve head (ONH) is supplied by the short posterior ciliary arteries (PCAs), which arise from the ophthalmic artery.[4] Moreover, the blood vessels at the ONH anterior to the lamina cribrosa are exposed to the intraocular pressure and posteriorly to the cerebrospinal fluid pressure. The rest of the ON has a dual vascular supply system; a peripheral centripetal system via the pial branches of the ophthalmic artery and a central core centrifugal system via branches of the central retinal artery (CRA) [Figure 3]. These branches sometimes extend 1-4 mm posterior to the site of penetration of the CRA into the ON. Hence, the ON is more vulnerable to ischemia than the ONH as it is supplied through easily compressible centripetal pial vessels.

Although AION can be diagnosed by fundoscopy, the diagnosis of PION is often delayed as fundoscopy is normal. Recent reports have demonstrated the utility of MRI in PION, showing diffusion restriction in the ON with decreased apparent diffusion coefficient (ADC) indicating ischemic injury.[5,6]

At present, preoperative evaluation cannot identify patients at risk of POVL. Nevertheless, it is prudent to inform patients of the small risk of POVL if prolonged surgery or substantial

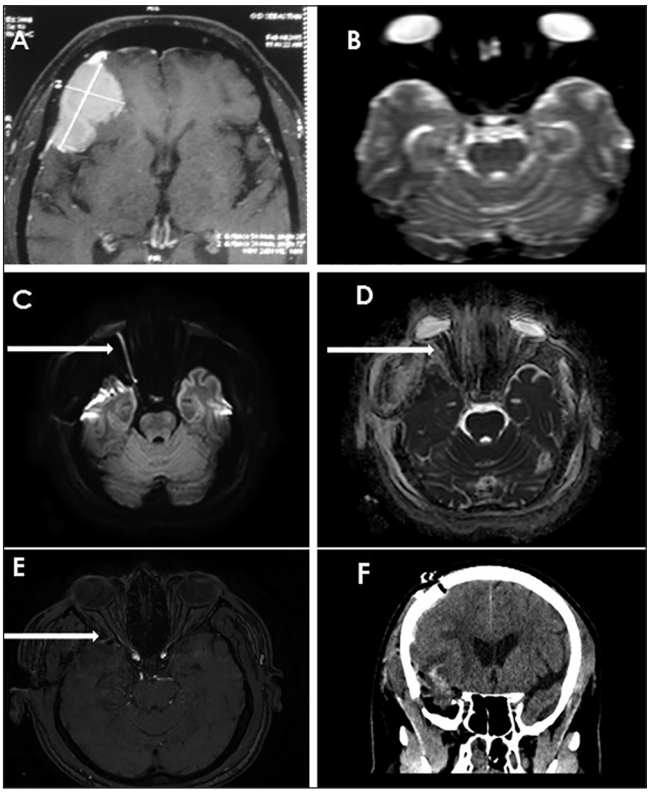

Figure 1: Panel A. Preoperative MRI showing a right pterional meningioma. Panel B shows a preoperative DWMRI with normal optic nerves. Panel C. DWMRI showing diffusion restriction in the right ON. Panel D. ADC images showing reduced ADC in the right ON. Panel E. MRI MIP images showing a patent's right ophthalmic artery. Panel F. Postoperative CT showing minimal postoperative changes

Table 1: Perioperative and intraoperative factors associated with nonocular surgery-related visual loss

Anemia
Carotid artery disease
Hypertension
Smoking
Prolonged prone position surgery
Diabetes mellitus
Perioperative hypotension
Substantial blood loss (>1 L of blood loss)
Artery to artery embolism during carotid endarterectomy
Intraoperative hypoxia
Prolonged surgery (>6.5 h)
Excessive fluid replacement
Direct ocular pressure during prone surgery
Hypothermia during cardiac bypass surgery
Postpump intraocular pressure (IOP) elevation[7]

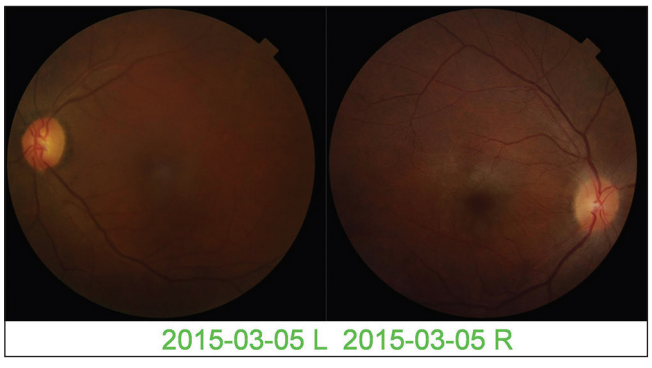

Figure 2: Fundoscopy showing normal findings on both sides

Table 2: Salient features of various causes of POVL

Etiology	Visual loss	Fundus findings	Other features	Cause	Neuroimaging
AION	Painless visual loss	Diffuse or segmental OD edema at onset Splinter hemorrhages at disc margin OD atrophy after 4-8 weeks	Sectoral, altitudinal (usually inferior), central scotoma, or other OD-related field defects FFA shows filling defects in the optic disc, peripapillary choroid, and/or choroidal watershed zones or absence of choroidal and OD filling in PCA distribution	Occlusion or hypoperfusion of the ONH by the PCA	MRI is normal
PION	Painless visual loss	Normal fundus exam at onset OD atrophy after 2-3 months	ON-related field defects with or without central visual acuity loss Normal appearance on FFA	Infarction of the intraorbital ON supplied by pial vessels	MRI shows gadolinium enhancement and/or restricted diffusion of ON[8]
CRAO	Painless visual loss	Macular cherry-red spot, white ground-glass retinal appearance, attenuated arterioles	FFA shows occlusion or stasis of affected branches of CRA	Occlusion of CRA or branches	MRI is normal
Pituitary apoplexy	Painful visual loss	Fundus may be normal	Acute onset of headache, vomiting, unilateral or bilateral vision loss, ophthalmoplegia, stupor	Acute hemorrhage or infarction of pituitary adenoma causing chiasmal compression	CT and MRI show definitive appearances
Cortical blindness	Painless visual loss	Normal fundus; Pupil reactions intact	Visual perceptual disturbances, abnormal ocular pursuit movements, occipital cortical signs	Watershed or embolic occipital infarction	CT and MRI demonstrate infarcts

OD = Optic disc, ONH = Optic nerve head, ON = Optic nerve, FFA = Fundus fluoroscein angiography, CRA = Central retinal artery, PCA = Posterior ciliary arteries, CT = Computed tomography, MRI = Magnetic resonance imaging

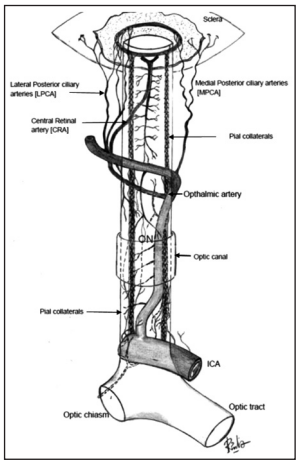

Figure 3: Optic nerve blood supply

blood loss is anticipated. Postoperative visual assessment should be performed in all high-risk patients. If visual loss is identified, urgent ophthalmologic evaluation can help to determine the etiology [Table 2]. Currently, there is no role for the use of antiplatelets, steroids, or intraocular pressure-lowering agents in POVL although it is beneficial to optimize hemoglobin values, hemodynamic status, and arterial oxygenation parameters. MRI is helpful in establishing the etiology of POVL.

Conclusion

In conclusion, our patient had PION based on the normal fundoscopic exam and MRI appearance of diffusion restriction in the ON. Our patient did not have any perioperative risk factors; hence, our postulation is that a sudden decrease in intracranial pressure during dural opening could have led to altered perfusion of the ON and PION. POVL and PION can occur postoperatively even in the absence of established risk factors.

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Conflicts of interest
There are no conflicts of interest.

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