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Ischemic Optic Neuropathy Following Craniotomy

Ranjit Kumar Chaudhary,[1] Sanjib Kumar Chaudhary,[2] Elina Gupta,[3] Karthikram Raghuram[1]

[1]Department of Radiology, Neuroradiology Division, University of Texas Medical Branch, Galveston, Texas, USA, [2]Department of Dentistry, BP Koirala Institute of Health Sciences, Dharan, Nepal, [3]Sagarmatha Chaudhary Eye Hospital, Department of Opthalmology, Lahan, Nepal.

ABSTRACT

Ischemic optic neuropathy is one of the major causes of severe impairment of vision often leading to blindness. It has varied etiopathogenesis with limited management options and very often result in poor outcome. Perioperative ischemic optic neuropathy is rare and particularly seen in elderly patients with multiple comorbidities undergoing cardiac or spine surgery. We present a case of young patient who developed ischemic optic neuropathy following craniotomy for recurrent meningioma.

Keywords: Ischemic optic neuropathy; optic nerve vasculature; painless vision loss; perioperative complications; perioperative optic nerve ischemia

INTRODUCTION

Ischemic optic neuropathy (ION) is the most common acute optic nerve disease of adults above 50 years of age.[1] Perioperative blindness are rare. It is seen after spinal, abdominal and cardiac surgeries that are associated with significant blood loss. This mishap is usually anticipated in older patients with multiple cardiovascular comorbidities. The incidence of perioperative ION varies between 0.028 and 1.3%.[2] Perioperative ION following craniotomy in supine position particularly in a young patient has been less frequently described.

CASE

A 25 year old male with prior craniotomy for resection of extensive meningioma had recurrence of frontoparietal meningioma with hyperostosis as well as bone destruction and involvement of superior sagittal sinus on follow up imaging, Patient had no visual symptoms prior to surgery. Surgery was uneventful except for fluctuation in blood pressure and heart rate due to which operation had to be staged for a later date. Intravenous fluid and vasopressor were used to stabilize hemodynamic status. Echocardiogram showed ventricular hypertrophy and hemohydrothorax. Patient developed diplopia, eye pain and blurry vision 12 days after surgery on left side and after 3 weeks on right side. There was notable pain with upwards and lateral gaze, bilaterally. On evaluation, patient was only able to count fingers at two feet. Fundoscopy was unremarkable except for bilateral optic disc edema. Intraocular pressure was normal. Contrast enhanced magnetic resonance imaging (MRI) of brain and orbits were obtained (Figure 1).

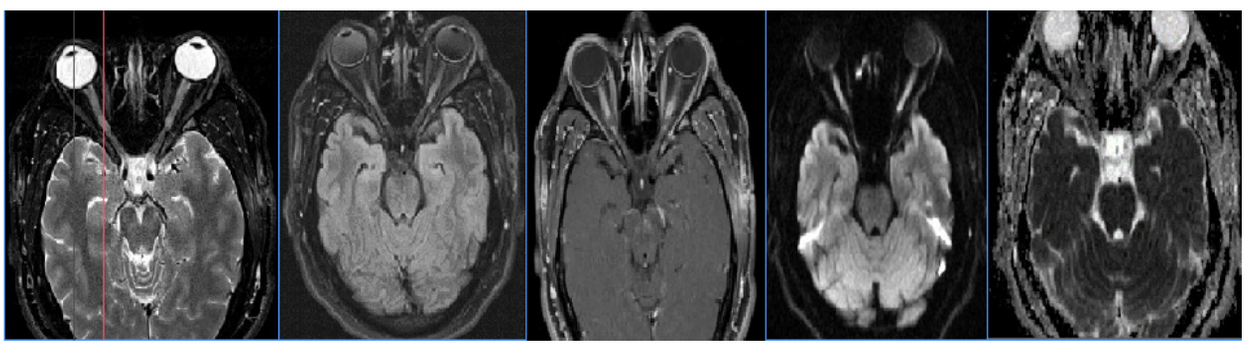

Figure 1. a) T2 axial shows high signal intensity in bilateral optic nerves which also appear slightly swollen b) FLAIR axial image demonstrates hyperintense signal in bilateral optic nerves c) T1 axial post contrast images show enhancement of the optic nerves d & e) DWI and ADC image show diffusion restriction in bilateral optic nerves as well as optic nerve head. (Images previously published in AJNR case of the week on January13, 2022)

Correspondence: Ranjit Kumar Chaudhary, Department of Radiology, University of Texas Medical Branch, 301 University Boulevard, Galveston, Texas, USA. Email: ranjitkchaudhary@hotmail.com, Phone: +14093542814.

There was hyperintense signal in fluid attenuated inversion recovery (FLAIR) image with enhancement in post contrast images. Diffusion weight images (DWI) showed high signal in bilateral anterior optic nerves including the optic nerve head (ONH) with low signal in apparent diffusion coefficient (ADC) suggestive of diffusion restriction. Patient was referred to neuroopthalmologist. Combination of high dose methylprednisolone and acetazolamide was tried but without success. There was progressive loss of vision with no perception of light by fourth week. The vision did not improve over follow up period of additional two weeks.

DISCUSSION

Blood supply of the optic nerve is complex (Figure 2). Blood supply to anterior segment of optic nerve is via posterior ciliary arteries (PCA) and central retinal artery (CRA) which are branches of ophthalmic artery. Blood supply to ONH may be from anastomotic arterial circle (the circle of Zinn-Haller) or directly from short posterior ciliary arteries. Pial plexus provides blood supply to both anterior and posterior segment of optic nerve (Table 1).[3] Due to variations in blood supply to the optic nerve, vessels involved determines the different patterns of visual field deficit.

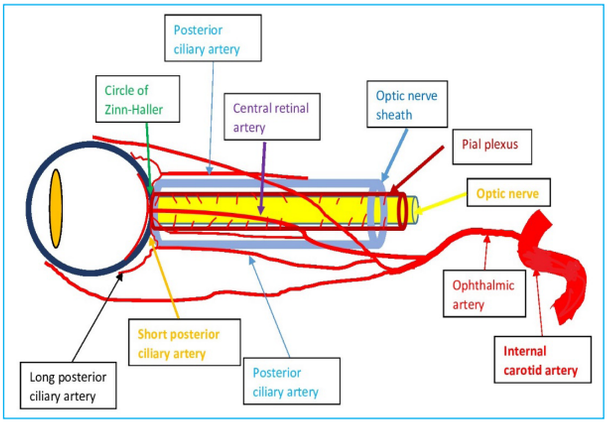

Figure 2. Blood supply of optic nerve.

Table 1. Optic nerve blood supply[3,4]	
Optic nerve part	Blood supply
ONH	Posterior ciliary arteries (PCA) via peripapillary choroid & short PCA (or the circle of Zinn and Haller).
Intra-orbital	anterior segment (between ONH and site of entry CRA into the nerve)	i) pial vascular plexus from peripapillary choroid, circle of Zinn and Haller, CRA ii) intraneural branches of CRA.
	Posterior segment	Small collateral arteries usually directly from ophthalmic artery.
Intracanalicular		Collateral from ophthalmic artery.
Intracranial		Fine branches from intracranial arteries & ophthalmic artery.

ION can be classified as anterior ION (AION) vs posterior (PION) and arteritic vs non-arteritic (not related to vasculitis). AION is more common than PION and non-arteritic AION is the most common type of ION. Hypoperfusion is thought to be the most common etiology but exact etiopathogenesis is still controversial. Half of the non arteritic ION patients have hypertension and a quarter of them have diabetes mellitus.[1,3,4] Acute non-arteritic AION is diagnosed clinically by presence of vision loss, relative afferent pupillary defect and edema of the optic disc. Small cup-to-disc ratio is seen in patients at risk of development non-arteritic AION.[1] Unlike classical non-arteritic AION, incipient type patients have asymptomatic optic disc edema.[5] It is imperative to know the different types of ION as management of each type is different from the other.[5]

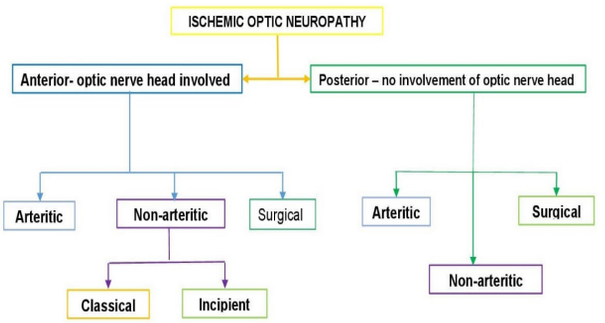

Figure 3. Classification of ischemic optic neuropathy[3-5]

ION can present with sudden vision loss or maybe progressive over days to weeks after the inciting event.[4] Perioperative ION usually tends to cause bilateral massive visual loss or even complete blindness, which are often permanent and may result in medicolegal cases.[5] Hypotensive drugs have been held culprit in some cases. Surgical or perioperative ION although typically

classified as posterior ION can be either anterior or posterior. Coronary-artery bypass grafting and prolonged spine surgery in prone position are two most commonly associated procedures.[2,5-7] Etiology of perioperative ION is poorly understood but is likely multifactorial.[1] Perioperative ION has been thought to be associated with carotid artery stenosis, smoking and diabetes due to their potential to alter the perfusion of optic nerve. It can also be associated with ocular diseases like hypertensive retinopathy, macular degeneration, glaucoma and cataract.[8] Arterial hypotension, anemia, excessive fluid replacement for massive blood loss leading to hemodilution, decreased oxygenation, pressure on the eyeball and orbit, dependent position of the head and prolonged duration of surgery, all can significantly increase the risk of perioperative ION in addition to preexisting cardiovascular comorbidities.[5-7] One of the predisposing factors for ischemia could be use of vasopressors during hypotensive episode to increase blood pressure may actually decrease the perfusion at capillary level due to vasoconstriction of terminal arterioles.[5,7] Being cognizant of this disease entity, stabilizing cardiovascular status in patients at risk prior to surgery if possible and taking precautions perioperatively may reduce the risk.

Presentation can be early or delayed. Presentation can be delayed due to use of sedation during postoperative recovery period.[8] Classically patient have painless vision loss which can be central or peripheral or altitudinal, superior or inferior, nasal or temporal.[2] Color vision may be decreased or absent. Usually both eyes are involved. However, unilateral cases are also not uncommon. Unilateral or asymmetric cases may show relative afferent pupillary defect.[8] Funduscopy in perioperative ION may be normal or demonstrate optic disc edema depending upon involvement of ONH. Optic disc pallor develops in 4-6 weeks of ONH involvement.[6] Delayed presentation, presence of other confounding factors and paucity of literature may misdirect towards other diagnosis but history of painless bilateral vision loss after surgical procedure and perioperative cardiovascular event are important clinical clues to diagnosis. Perioperative visual loss may be seen due to trauma to optic nerve, retinal artery occlusion, cortical blindness, acute glaucoma and choroidal and vitreous hemorrhage apart from ION.[8] Retinal artery occlusion on fundoscopy may demonstrate cherry spots, retinal opacity in posterior pole, retinal arterial attenuation and optic disc edema within one week of onset. Optic atrophy and retinal arterial attenuation may be seen in later stages.[9] Opthalmoscopy can also help in evaluation of glaucoma as well as choroidal and vitreous hemorrhage. Imaging findings of perioperative ION can mimic, arteritic ION, nonarteritic ION, optic neuritis, toxic, infective or compressive etiologies. Jaw claudication is often seen in arteritic ION while periorbital pain and pain on eye movement are seen with optic neuritis. Computed tomography and MRI may be performed for evaluation and imaging of brain and orbits can also help to rule out mass lesions, stroke or other potential causes of blindness. Although higher DWI and lower ADC values may be seen in ION compared to optic neuritis and other toxic/inflammatory etiologies, usually diffusion restriction in optic nerves are not easily demonstrable in MRI. Additionally, T2/FLAIR increased signal and enhancement may create confusion to diagnosis as entities like optic neuritis may demonstrate similar imaging findings but being cognizant about this entity will help to make the accurate diagnosis of perioperative ION and avoid unnecessary futile treatment.[10] Prophylaxis is the only management available. Avoiding arterial hypotension, use of blood products instead of excessive fluid replacement and hemodilution, judicious use of vasopressors, avoiding pressure on the eyeball and orbit and shortening the duration of surgery to the minimum can decrease the chances of this dreaded complication.[2,5,7]

CONCLUSIONS

Surgical ION is an uncommon but irreversible devastating complication and prophylactic measures should be adopted particularly in patients with cardiovascular comorbidities anticipating surgery for prolonged periods.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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