



CASE REPORT

REVISED Postoperative visual loss following cerebral arteriovenous malformation surgery: a case report [version 2; peer review: 3 approved]

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Abstract

We report the case of a 46 year-old woman presenting with unilateral postoperative visual loss after right frontal craniotomy for resection of an arteriovenous malformation in the supine position. The intraoperative course was uneventful with maintenance of hemodynamic stability. Blood loss was 300 ml; postoperative hemoglobin was 12.4 g/dl. In the recovery room, the patient reported loss of vision in her right eye. Ophthalmologic examination revealed decreased visual acuity, color vision, and visual field. Assessment of the retina was normal, but the patient showed a relative afferent pupillary defect consistent with the clinical diagnosis of ischemic optic neuropathy. Postoperative computer tomogram showed normal perfusion of ophthalmic artery and vein, no hemorrhage or signs of cerebral ischemia or edema. The patient recovered most of her vision 3 months after surgery. Anesthesiologists should be aware that this condition may follow uncomplicated intracranial surgeries in the supine position, and should obtain prompt ophthalmologic consultation when a patient develops postoperative visual loss.

Open Peer Review

Reviewer Status ✓✓✓

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REVISED			
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version 1 published 27 Jan 2014	✓ report	✓ report	✓ report

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Any reports and responses or comments on the article can be found at the end of the article.

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REVISED Amendments from Version 1

This article update provides more detailed information on the case of postoperative visual loss (POVL) in a neurosurgical patient that we previously reported. We added some important aspects of our patient's personal medical history to emphasize that, apart from mild obesity, risk factors for POVL were absent in this case. We agree with our readers, that the first and most plausible cause for POVL after craniotomy might have been the potential involvement of the arteriovenous malformation (AVM) in the perfusion of the optic nerve, or the direct trauma to the cerebral visual tracts caused by neurosurgery. However, this etiological scenario is uncertain, and we added pre- and postoperative CT angiographic images to provide evidence for regular perfusion of the ophthalmic artery before and after the operation. A diagram of the blood supply of the optic nerve shows the anatomical origin of ischemic optic neuropathy (ION) and outlines some of the underlying pathophysiological mechanisms of POVL. In our opinion, it will remain difficult to definitively determine the cause of ION in this patient. Fortunately, the incidence of POVL is rare in a general surgical population; nevertheless, we would like to draw our reader's attention to the possibility of encountering POVL in non-high-risk cases such as intracranial surgery in the supine position.

See referee reports

Introduction

Postoperative visual loss (POVL) is a known complication of surgery and anesthesia; its incidence varies from 0.03% after spine surgery to 0.086% after cardiac surgery¹. Typically, this involves ischemic optic neuropathy (ION), and is clinically characterized by the acute or subacute loss of visual acuity and/or visual field. Both anterior (diffuse optic disc swelling) and posterior (no optic disc swelling) ION have been reported after general anesthesia in spinal and other non-ocular surgeries¹⁻³. The incidence of POVL in a general surgical population is low (0.0012%)¹; to our knowledge, only one case has previously been reported in intracranial neurovascular surgery, and was included in the POVL registry of the American Society of Anesthesiologists (ASA)⁴.

Case report

A 46-year-old woman initially presented with paresthesia in hands and legs, and was diagnosed with a right frontal, subcortical arteriovenous malformation (AVM) of 1.5 cm in diameter. The AVM was classified as grade I according to Spetzler-Martin criteria⁵ (small size, non-eloquence of adjacent brain, and only superficial venous drainage). Preoperative cerebral angiography showed that arterial blood supply of the AVM was via a frontal cortical branch and a superior frontopolar branch originating from the anterior communicating artery. Venous drainage of the AVM was superficial. Embolization prior to the craniotomy was not performed. **Figure 1** shows the preoperative computer tomography (CT) angiogram.

Patient weight was 91 kg and height 165 cm (body mass index 33). The medical history revealed arterial hypertension, as well as coronary artery disease with previous myocardial infarction (MI) and percutaneous coronary angioplasty. Preoperative transthoracic echocardiography showed a grade II left ventricular function (ejection fraction 50–55%) and mild distal anteroseptal and distal anterior wall hypokinesia. The patient ceased to smoke one year ago with a 30 pack-year history. Apart from distal paresthesia in the

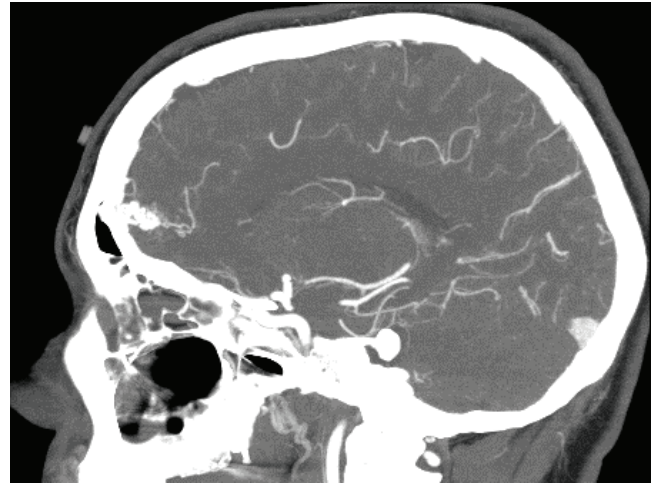


Figure 1. Preoperative computerized tomographic angiography showing the frontal AVM (Spetzler-Martin grade I).

extremities, the patient's neurological status was normal. There was no history of impaired vision or previous eye surgery. Her medications consisted of metoprolol 12.5 mg twice daily, aspirin 81 mg and clopidogrel 75 mg once daily. Aspirin and clopidogrel were stopped 7 days prior to the intervention. Preoperative blood pressure was 130/90 mmHg, and hemoglobin concentration was 14.2 g/dl.

The patient was classified as ASA physical status 3, and underwent craniotomy with surgical excision of the AVM. After induction of general anesthesia with midazolam 2 mg, fentanyl 150 µg, and propofol 150 mg, followed by neuromuscular blockade with rocuronium 50 mg, the patient's trachea was orally intubated. Anesthesia was maintained with sevoflurane and a remifentanyl infusion during the 4-hour surgical procedure. Surgery was performed in the supine position, with the head elevated to 30 degrees and fixation in a head frame. The patient's intraoperative blood pressure was maintained stable around 100 mmHg systolic, and there was minimal blood loss (300 ml). Total crystalloid infusion was 2 l. Emergence from anesthesia was uneventful, and the patient was extubated while awake and obeying commands. There was no sign of external compression of the eyes during and after surgery.

On awakening in the recovery room, the patient complained of blindness in her right eye. Immediate CT angiogram of the brain was inconclusive showing normal perfusion of both ophthalmic artery and vein, no hemorrhage or signs of cerebral ischemia or edema, just evidence of AVM resection (**Figure 2**). Ophthalmology was immediately consulted. Examination of the right eye showed a posterior ION in association with a relative afferent pupillary defect (RAPD), but fundoscopy was normal without optic disc edema or cherry-red spot. Upon diagnosis of ION, the patient immediately received digital massage of the right eye once for a few minutes. Anti-glaucoma treatment (timolol maleate ophthalmic solution 0.5% applied to the affected eye twice daily for 2 days) was given to decrease intraocular pressure (IOP). Mild postoperative pain was treated with small doses of intravenous fentanyl; otherwise, the patient did not have any postoperative complications. On the following day, her vision had somewhat improved; however, the right temporal visual field

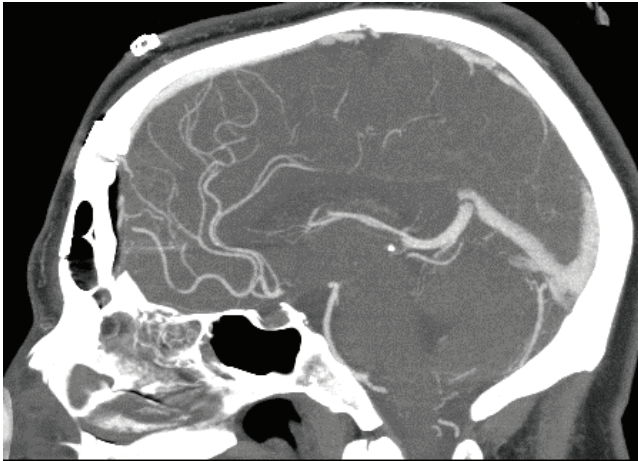


Figure 2. Postoperative computerized tomographic angiography showing normal perfusion of both ophthalmic artery and vein, no hemorrhage or signs of cerebral ischemia or edema, just evidence of AVM resection.

was still missing. Examination of the retina was again normal, and no further treatment was administered. She was discharged from the hospital 2 days after surgery. In a follow-up visit after 3 months, she was neurologically intact, and her vision was better. Her visual field had returned to normal with just some blurring of vision in the right eye. To our knowledge, no formal assessment of visual fields was performed after discharge.

Discussion

This patient experienced acute unilateral loss of visual acuity and visual field after AVM surgery in the supine position. Findings were consistent with the diagnosis of POVL due to posterior ION with RAPD. Fundo- and retinoscopy of the affected eye were normal, and there was no sign of external pressure on the ocular globe. Our patient did not have risk factors for POVL, except for mild obesity. Vision loss did not seem to be related to the actual AVM or its surgical removal. Given the characteristics and location of the AVM and the surgical technique used in this case, it seems unlikely that an intraoperative insult to the optic nerve or its blood supply via direct trauma to the nerve or compromise of the blood supply with edema or brain retractors was a factor in the development of POVL. In a review of the literature, we only found one case of unilateral visual loss after intracranial aneurysm surgery (ASA POVL Registry)⁴, suggesting that POVL is a very rare complication of intracranial neurovascular surgery in the supine position. Due to the multitude of perioperative and patient variables, it is difficult to definitively ascertain the etiology of POVL in this case.

Figure 3 shows a diagram of the blood supply of the optic nerve; if any impairment of blood supply occurs, ION may develop. POVL may present as both uni- and bilateral. An association with systemic diseases, such as hypertension, diabetes, hypercholesterolemia, or atherosclerosis, is not well documented. Several potential causes of POVL have been described. Intraoperative corneal trauma may result in irritation, abrasion, or even laceration of the eye. Preventive measures include taping the eyes shut and careful patient

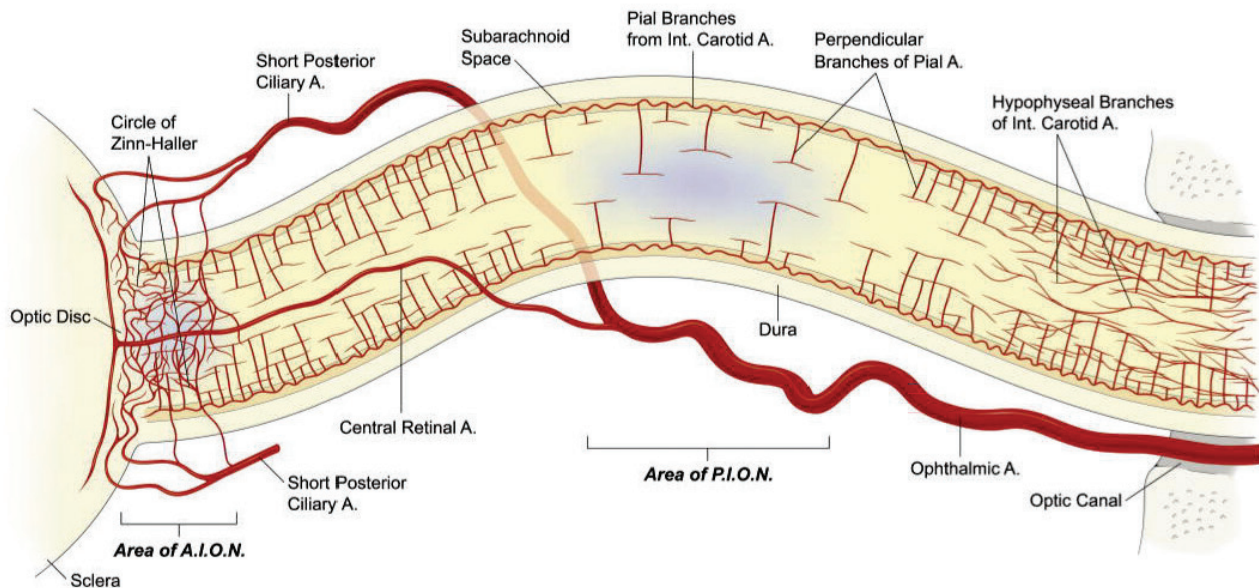


Figure 3. Diagram of the orbital optic nerve with its vascular supply. The areas involved in the ischemic insults are highlighted in purple. The anterior optic nerve is supplied by multiple sources while only the small pial branches supply the mid-orbital optic nerve. It is the mid-orbital region of the optic nerve that is involved in PION. The pial branches are found with variable density and in an unusual perpendicular T-shaped pattern, characteristic of a low pressure system. There is low density of arteriolar and capillary supply to this mid-orbital segment compared with the canalicular or retro-bulbar segments of the optic nerve. A., Artery; AION, Anterior Ischemic Optic Neuropathy; PION, Posterior Ischemic Optic Neuropathy. Image courtesy of Baig M.N. *et al.*, 2007, Vision loss after spine surgery: review of the literature and recommendations, *Neurosurgical Focus*, November, 23 (5): E15⁶. Re-print permission has been obtained from the [Journal of Neurosurgery Publishing Group](#). This figure is not subject to the CC license that the rest of this paper is subject to. Any use of this figure is subject to obtaining the necessary permissions from the [Journal of Neurosurgery Publishing Group](#).

positioning. Intraoperative stroke involving the visual tracts or the visual cortex may lead to hemianopsia and cortical blindness. Cerebral ischemia may be due to prolonged systemic hypotension or thromboembolism. Arteriosclerosis-related embolism also plays an important role in the etiology of central retinal artery occlusion (CRAO)⁶; however, CRAO may be caused by an acute and severe rise of IOP found in trauma or direct external pressure to the ocular globe as well. Clinical findings in CRAO are the unilateral painless loss of vision with signs of external periorbital swelling or ecchymosis, and a pathognomic cherry-red spot at the macula. Recently, posterior reversible encephalopathy syndrome (PRES) has also been proposed as potential cause of POVL⁷.

Risk factors that have been implicated in the development of ION in spine surgery include male sex, obesity, prolonged intraoperative hypotension, long duration of surgery, substantial intraoperative blood loss, and the excessive use of intravenous replacement fluids, or anemia^{8,9}. The management of POVL has been described by an ASA task force³.

The majority of POVL cases are reported after lumbar spine surgery^{1,2,6-10}, suggesting a greater incidence of POVL associated with prone positioning. Increases in IOP during surgery in the supine position were thought to be an important factor for the development of visual loss; the current recommendation of the ASA task force³ is to keep the head elevated higher than the heart and in neutral position whenever possible. Intraoperative blood loss and prolonged arterial hypotension should be avoided. Intravenous administration of colloid solutions and avoidance of excessive crystalloid infusion has been recommended. Excess crystalloid may cause tissue edema, and compromise tissue oxygenation in the orbital cone. If prolonged duration of surgery in the prone position is necessary, the eyes should be examined for external compression or swelling at regular intervals.

Conclusion

POVL involving ION remains a rare, but devastating condition. Unfortunately, our limited knowledge of the pathophysiology restricts the treatment options. Type of surgery, patient-related and intraoperative risk factors have been identified, but were absent in this case, apart from mild obesity. Clinicians should be aware of high-risk cases for POVL such as major spine surgery in the prone position and cardiac surgery. Intraoperative head elevation, use of colloids, avoidance of excessive crystalloid infusion and prolonged arterial hypotension, correction of anemia, and staging of surgery to reduce operating time are preventive measures recommended by the ASA task force on POVL³. Cases of permanent loss of vision have occurred, therefore early diagnosis and treatment are paramount to increase chances of visual recovery in the event of POVL.

Patient consent

Written informed consent for publication of clinical details was obtained from the patient.

Author contributions

Nicolai Goettel and Jayati Ghosh contributed equally to the writing of the manuscript. Michael Tymianski was the neurosurgeon involved in the case. Pirjo H Manninen helped write the manuscript.

All authors have contributed to the revisions of the manuscript and approved the final manuscript.

Competing interests

No relevant competing interests were disclosed.

Grant information

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Sergio Bergese

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After reading "**Postoperative visual loss following cerebral arteriovenous malformation surgery: a case report**" I have some minor additions to propose to the authors:

1. Extend discussions based on patient medical history linked to POVL - neurological, ophthalmological, and concomitant medication.
2. Explain the standardized grading scheme: Spetzler-Martin.

The authors covers a topic of significant scientific interest, with original data and good quality of presentation.

Competing Interests: No competing interests were disclosed.

I have read this submission. I believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard.

Reviewer Report 05 February 2014

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This case report of PION occurring ipsilaterally after a resection of a frontal AVM is very interesting. It is a reminder that PION can occur in many different types of procedures outside spine, cardiac, and head and

neck surgery. During craniotomies, more direct insult to the blood supply of the optic nerve may be an additional etiologic factor via direct trauma to the nerve or blood supply, or compromise of the blood supply with edema or brain retractors. It is difficult to ascertain with the available information the proximity of the AVM to the optic nerve and its blood supply.

For the readers of this case report, it might be helpful to discuss the blood supply of the optic nerve with a diagram, including both the superior hypophyseal artery and the ophthalmic artery and branches. The patient's preoperative angiogram and MRI/CT of the head, showing the proximity of the AVM and venous drainage to the optic nerve and its blood supply would be helpful information for the clinician.

Other useful information would include a statement re: any other postoperative complications; any signs of edema/ischemia on later CT/MRI of the head; later ophthalmologic findings such as optic nerve pallor on funduscopy (confirmation that it was PION); and if formal assessment of visual fields was performed after discharge.

It will remain difficult to definitively ascertain the etiology of PION in this case, even if all the requested information is provided. Fortunately, perioperative PION is relatively rare; however, its low incidence, high severity of injury, and the multitude of perioperative and patient variables make it exceedingly unlikely that studies with a high level of evidence will be conducted in the future. Animal models of PION simulating perioperative conditions may provide important insights into etiology, prevention and treatment of this problem. Unlike most patients with perioperative PION, this patient seems to have had good recovery of vision.

(N.B. The lack of reports of craniotomies and POVL in the ASA POVL Registry is more likely a result of exclusion of cases in that Registry where direct trauma to the optic nerve or its blood supply was possible.)

Competing Interests: I provide medicolegal review and testimony regarding cases of POVL.

I have read this submission. I believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard.

Reviewer Report 29 January 2014

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The authors report a case of partially reversible right unilateral visual loss following elective resection of a right frontal arteriovenous malformation. Immediate ophthalmologic assessment in recovery is compatible with ischemic optic neuropathy (ION), a condition not commonly reported following neurosurgical surgery in supine position. The pathophysiology of ION remains unclear; it is most likely multifactorial.

Abstract

Although clearly specified in the case description, I would clarify the abstract by adding a couple of details about the procedure: right frontal craniotomy for resection of an AVM in supine position, under general anesthesia. This is just too properly set the table before digging in, so to speak... Also, I would mention that the patient partially recovered vision 3 months after surgery, for the sake of completeness.

Introduction

I would leave the specifics of ION (presentation, assessment, causes & mechanisms) for the discussion altogether. POVL prevalence reports (0.0012% general surgery; 0.03% spine surgery; 0.086% cardiac surgery) and general classes of POVL (corneal, retinal including CRAO, optic, chiasma and tracts, cortical) are all that is needed for the introduction here.

Case description

Since obesity is a known risk factor for ION, I would mention the patient's height & weight. Were there any visual issues at baseline? I assume there weren't any, but I would mention it anyway. Were any of the AVM feeders embolized prior to craniotomy? Were there any other abnormalities noticed on the cerebral angiogram? In particular, since the AVM was in proximity to the right optic area; were there any arterial branches coming off the feeders? If so, could arterial exclusion have caused optic ischemia? Was the circle of Willis normal on preoperative control? We typically perform an immediate postoperative angiogram following AVM resection to confirm complete resection of the AVM, as well as to assess perfusion of normal vessels. Did you perform such an exam before emergence and extubation? If so, I would mention it with a brief description of results.

The discussion about potential mechanisms of ION is interesting. It appears to me that ION is a multifactorial process combining the focal effects of arterial hypotension, venous hypertension, and decreased oxygen capacity. Arterial blood pressure and oxygen capacity seem to have been managed properly in this case. The combination of elevated intra-ocular pressures and borderline low arterial pressures puts the optic nerve at risk of ischemia. You mention anti-glaucoma therapy having potentially helped to improve perfusion of the optic nerve; was this patient known for glaucoma? Venous hypertension remains a possibility; how was the head positioned during the case? Did mechanical ventilation require high peak pressures restricting cerebral venous drainage?

In any case, I believe this case report deserves an 'Approved' status but needs minor additions imposed by the specific location of the AVM relative to the visual loss. Mainly:

1. minor details about patient at baseline (height & weight, visual status)
2. preoperative angiographic assessment (feeders, Willis) and management (embolization)
3. postoperative angiographic assessment, if any.

The general discussion about ION is interesting and most relevant to this case. I found it clear and well referenced.

Competing Interests: No competing interests were disclosed.

I have read this submission. I believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard.

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