

Case Study: Ophthalmic Artery Occlusion after Sinus Surgery

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Background

Endoscopic sinus surgery is a frequently performed procedure in close anatomic proximity to the orbit and globe. Ophthalmic complications of sinus surgery are rare but can result in serious ocular morbidity. We present a case of a patient with painless vision loss after sinus surgery.

Patient Case

Our patient is a 68-year-old male with a past medical history of hypertension and chronic sinusitis presenting with sudden onset vision loss after routine sinus surgery. The patient underwent a routine septoplasty, turbinate reduction, Latera implant, and bilateral maxillary antrostomy at an outside hospital with no apparent intraoperative complications. The patient woke up in the post anesthesia recovery area and noted complete loss of vision in his right eye. The patient had complete darkness from the right eye with minimal discomfort. He denied flashing lights, floaters, eye redness, ocular pain, diplopia, or headache.

The patient was seen in the PACU by an outside ophthalmologist who noted no light perception vision (NLP) and a normal intraocular pressure. Despite the normal intraocular pressure, a canthotomy and cantholysis was performed by the outside ophthalmologist without release of fluid or improvement in vision. The patient was transferred to RUSH University Medical Center for further evaluation and treatment.

Upon arrival to RUMC, the patient was noted to have NLP vision in the right eye and 20/40 vision in the left eye with a dense right afferent pupillary defect. He was also noted to have restricted extraocular movements with -4 adduction and -4 depression, but full elevation and abduction. His intraocular pressures were 10 and 13 mm Hg in the right and left eyes respectively, and the globes were soft to palpation and retropulsion. His external exam was notable for 2+ periorbital ecchymosis of the right lower lid with a lateral canthal incision. The anterior segment exam was unremarkable. His dilated fundus exam was notable for a diffusely pale optic nerve with blunted disc margins. The posterior pole was pale and edematous with a cherry red spot in the macula, and there was deep choroidal whitening nasally. His vessels were diffusely attenuated without focal emboli. The dilated exam of the left eye was unremarkable.

The patient received a CT visual tracking of the sinuses which was notable for early postoperative changes in the sinonasal cavities and thinning of the cribriform plates and lamina papyracea without dehiscence or fracture (Figure 1). An MRI of the orbits and optic nerve was notable for right optic nerve hyperintensity and diffusion restriction likely from acute optic nerve ischemia without optic nerve compression (Figure 2).

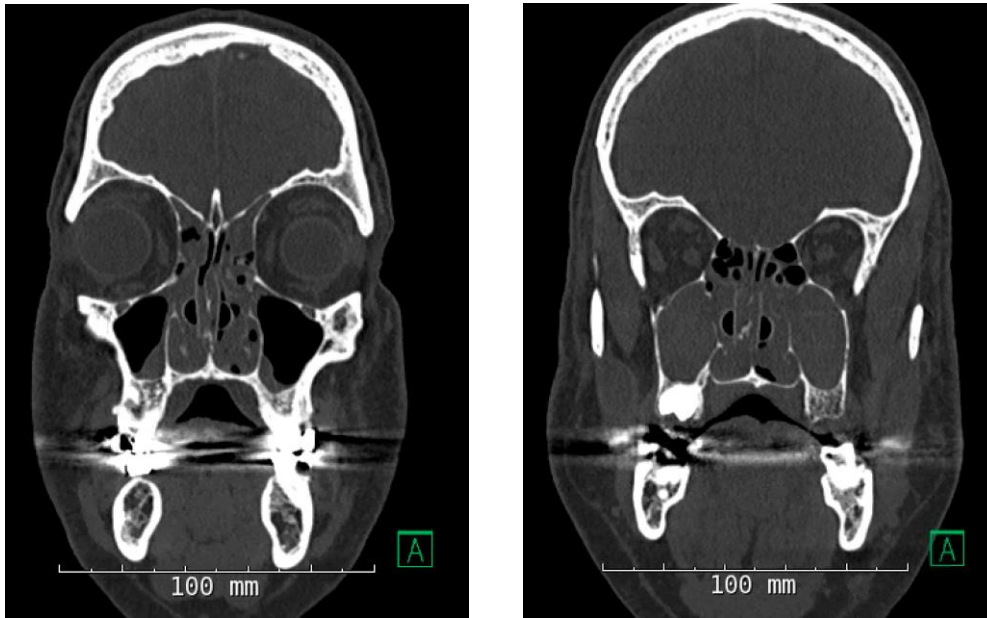


Figure 1: CT orbits. Early postoperative changes in the sinonasal cavities. Fluid levels in bilateral maxillary sinuses and right sphenoid compartment with partial opacification of the ethmoid air cells. Subtle hyperdensity of sinus contents may be from postoperative blood products. Occluded drainage pathways. Thinning of the cribriform plates, fovea ethmoidalis, and lamina papyracea without dehiscence or fracture.

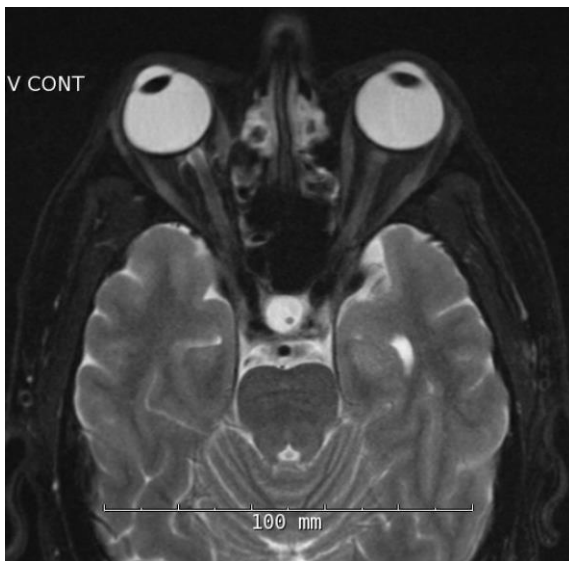


Figure 2: MRI orbits with contrast. The Intraorbital proximal right optic nerve shows subtle T2 hyperintensity and diffusion restriction - likely from acute optic nerve ischemia. Subtle fat stranding and enhancement at the orbital apex - nonspecific and may represent postoperative change secondary to venous congestion.

The patient was brought to the ophthalmology clinic for further testing. An OCT of the retina was performed which showed diffuse thickening and disorganization of the retinal layers (Figure 3). A fluorescence angiography was performed which showed poor choroidal perfusion, significantly delayed arterial transit time, and significant areas of retinal nonperfusion (Figure 4).

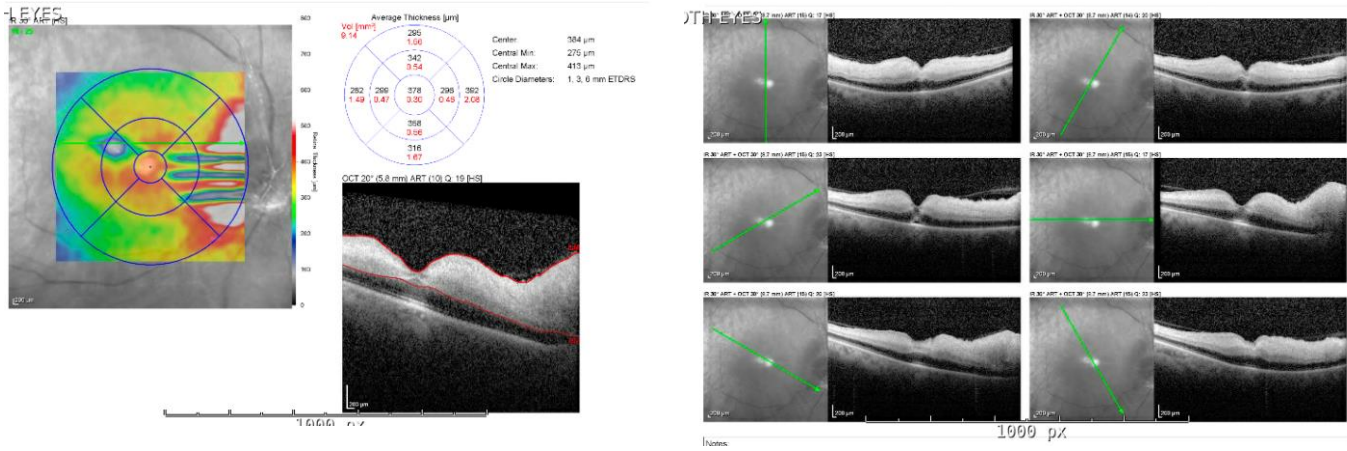


Figure 3: OCT retina of the right eye showing diffuse inner retinal thickening.

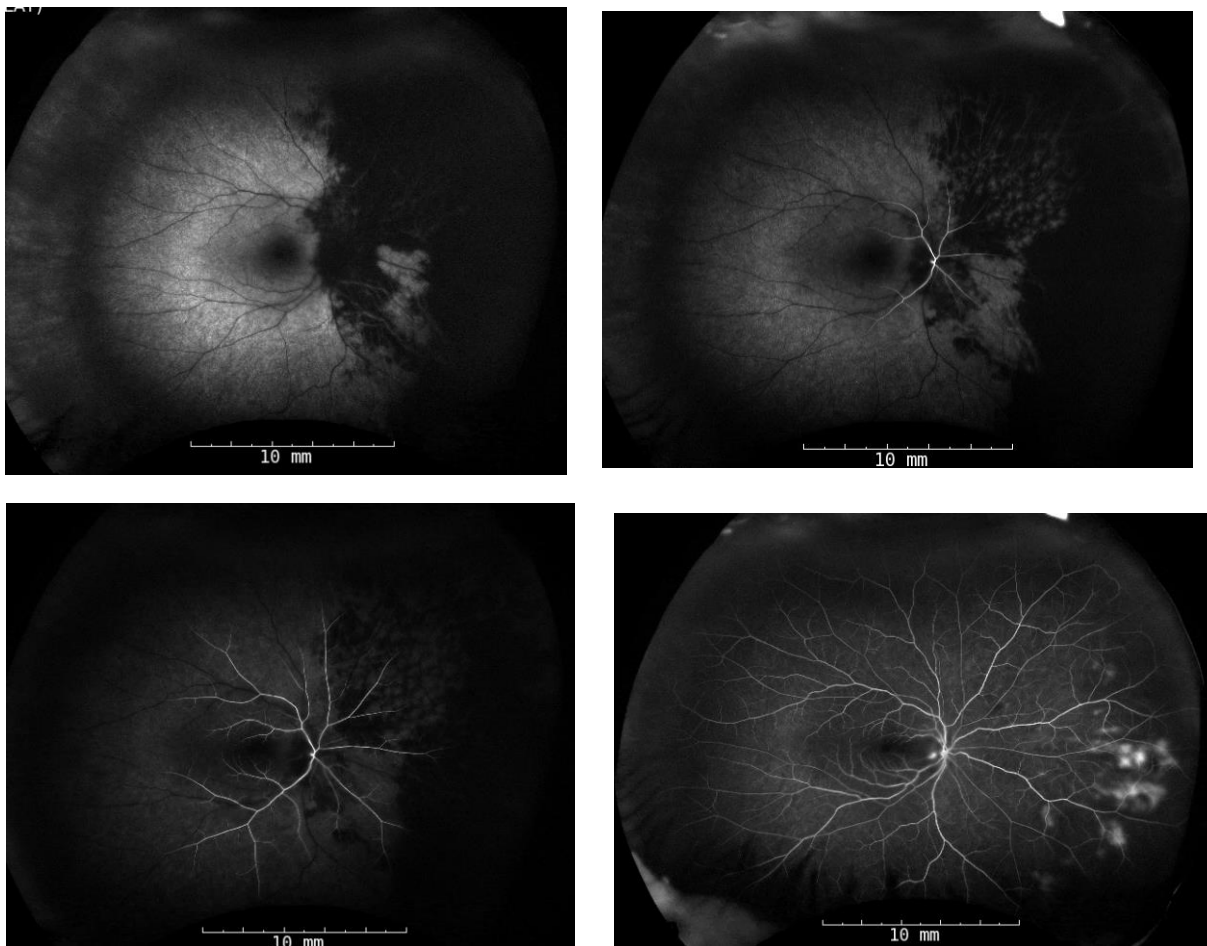


Figure 4: Fluorescein angiography of the right eye showing early choroidal nonperfusion, significantly delayed arterial transit time, and late retinal nonperfusion.

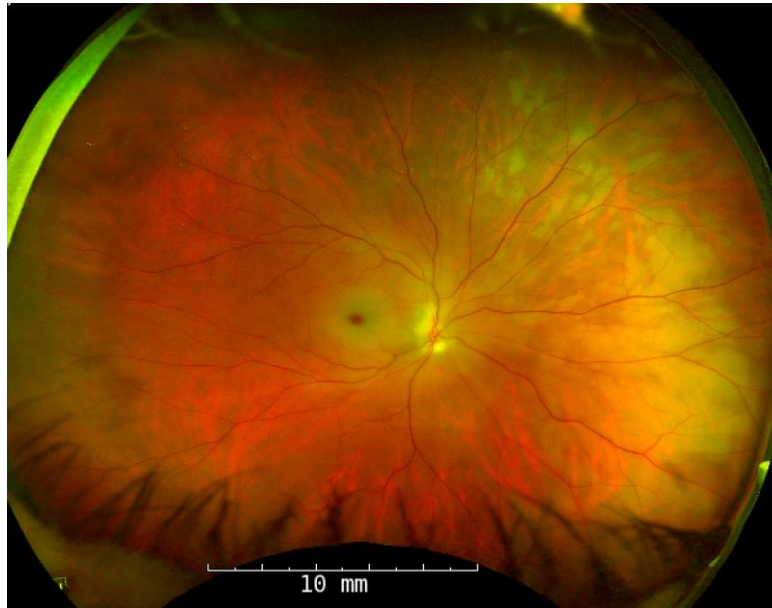


Figure 5: Optos fundus photo of the right eye showing choroidal whitening nasally, optic disc and retinal edema, and a cherry red spot.

The patient was diagnosed with an ophthalmic artery occlusion causing ischemia to his optic nerve, choroid, retina, and the medial and inferior rectus. There was no structural compression or damage to the optic nerve or orbital bones. A proposed mechanism of injury is inadvertent intravascular injection of lidocaine with epinephrine into tributaries of the ophthalmic artery during the transpalatal or middle turbinate injections performed during surgery. There can then be retrograde flow of the epinephrine causing vasospasm and ischemia of the ophthalmic artery and its branches. The patient was given IV solumdrol 500 mg twice daily and discharged with an oral prednisone taper. He remained with NLP vision on outpatient follow up.

Discussion

Ophthalmic complications of endoscopic sinus surgery are rare but can be devastating. There is an estimated 0.12% incidence of major orbital complications, including orbital hemorrhage, optic nerve damage, diplopia, enophthalmos, epiphora, and blindness.¹ The most common of these is a retrobulbar hemorrhage, which is most often caused by inadvertent entry of the orbit from the paranasal sinuses through the lamina papyracea.²

Optic nerve injury can be from direct mechanical damage or indirect optic neuropathy from a compressive orbital hemorrhage or interruption of the vascular supply.^{2,3} Injury to the extraocular muscles can be caused by direct injury, most commonly to the medial rectus from a break in the lamina papyracea. Indirect injury to the muscles can be caused by muscle entrapment or damage to the neural or vascular supply.⁴

There are case reports in the literature of more rare causes of vision loss after sinus surgery. One case reports light perception vision and central retinal artery occlusion with yellow colored fat emboli in the retinal vasculature from inadvertently damaged bones.⁵ There are several case reports of ophthalmic artery occlusion and vision loss after facial injection of lidocaine with epinephrine. The proposed mechanism is inadvertent injection of epinephrine into arterioles with retrograde flow and epinephrine-induced vasospasm of the ophthalmic artery leading to ischemia, similar to our case.^{6,7,8} Unfortunately, the prognosis of iatrogenic ophthalmic artery occlusion is poor with limited treatment options and often no visual recovery.⁹

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