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Delayed Sudden Blindness From Unilateral Ophthalmic Artery Vasospasm Following Endoscopic Sinus Surgery?

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Endoscopic sinus surgery (ESS) is a generally benign, minimally invasive procedure used for management of paranasal sinus diseases, although complications may occur due to proximity of vital structures such as the brain, orbit and great vessels.[1] The overall ESS major complication rate is 0.5-1%, of which orbital injury accounts for 0.09% due to direct trauma.[2] We report a case of unilateral delayed sudden visual loss without orbital trauma observed intraoperatively or on post-operative imaging studies, following a seemingly routine endoscopic sinus surgery for chronic rhinosinusitis.

CASE REPORT

An 18-year-old lad with no significant medical history underwent ESS for bilateral chronic rhinosinusitis with nasal polyposis. *(Figure 1 A-D)* The surgery and recovery from anesthesia were uneventful. On the 12th hour post-operatively, the patient noted blurring of vision on the left. Ophthalmologic examination revealed hyperemic conjunctiva *(Figure 2A)* with visual acuity of counting fingers at 1 foot while fundoscopy showed retinal hemorrhages. Extraocular eye movements (EOM) and intraocular pressure (IOP) were normal (12mmHg). With an assessment of pre-retinal hemorrhages, 500 mg Tranexamic acid was intravenously infused, and a paranasal sinus (PNS) computed tomography (CT) scan and orbital magnetic resonance imaging (MRI) were requested. A few hours later, he complained of left eye pain with increasing intensity and further deterioration of vision. Repeat visual acuity testing showed light perception. There was now a constricted pupil, non-reactive pupillary light reflex, periorbital swelling and progression of conjunctival chemosis. *(Figure 2B)* The IOP of the left eye had increased to 30mmHg then progressed to 40mmHg with development of total visual loss and a lateral gaze limitation. With an impression of choroidal hemorrhage and retrobulbar hemorrhage, a lateral canthotomy relieved the eye pain.

The contrast PNS CT scan with orbital cuts showed that the lamina papyracea was intact with no definite hemorrhagic collections in the intraconal or extraconal spaces of both orbits. *(Figure 3A, B)* A small hyper density along the lateral inferior margin of the left globe at the intraconal region with slight thickening of the anterior periorbital region represented the lateral canthotomy. The PNS MRI / magnetic resonance angiography (MRA) with orbital cuts showed retinal detachment and periorbital edema in the left eye. *(Figure 4)* A B-Scan ocular ultrasonogram showed retinal detachment and vitreous opacities. The diagnosis was ocular ischemic syndrome secondary to ophthalmic artery vasospasm, and the patient was given sublingual nitroglycerine and intravenous dexamethasone 8mg every 12 hours for 24 hours, with improvement of periorbital swelling. He was discharged after 12 days with no resolution of the unilateral visual loss.

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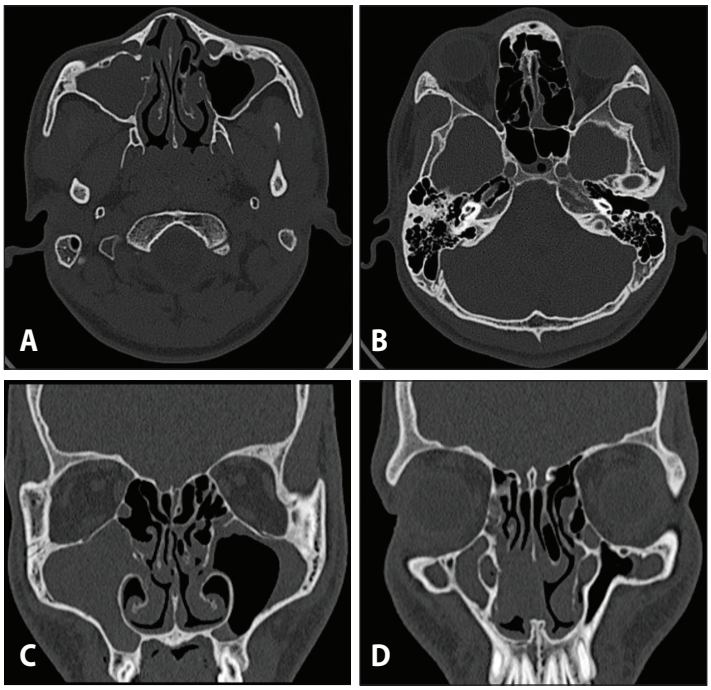

Figure 1. Pre-operative CT scans: **A, B.** axial views; **C, D.** coronal views.

Figure 2. A. chemosis of left eye; **B.** progression of chemosis (post-canthotomy)

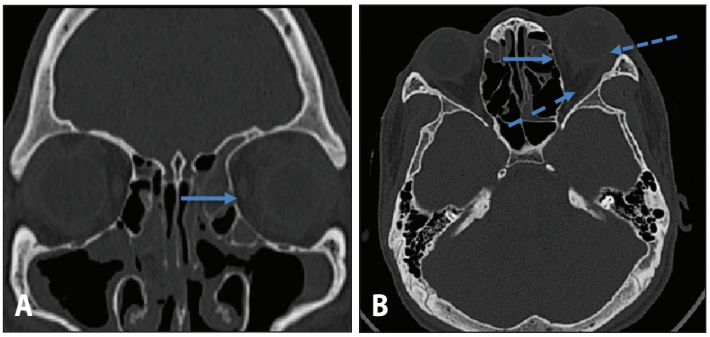

Figure 3. Post-operative CT scans showing intact lamina papyracea (solid arrows); **A.** coronal view; **B.** axial view. No hemorrhagic collection is seen in intraconal and extraconal spaces (dashed arrows).

DISCUSSION

Orbital injury resulting in visual loss is a rare but devastating complication of ESS. The incidence ranges from 0.07-0.09% with iatrogenic injury as the most common cause.[2] Such orbital injury can be attributed to orbital muscle or optic nerve injury and orbital hematoma

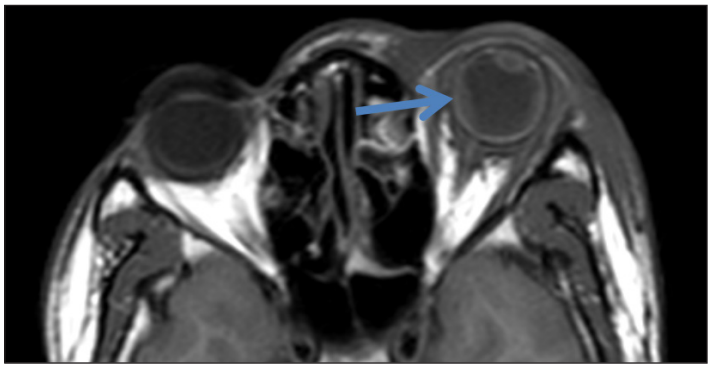

Figure 4. MRI, axial view showing retinal detachment (solid arrow).

which are both easily recognizable intraoperatively and post-operatively.[3] The sequelae of such injuries are dependent on which part of the eye is involved. Eye muscle involvement results in diplopia while optic nerve involvement leads to visual loss.[4] Orbital hematoma presents with rapid unilateral orbital swelling and is easily treated with lateral canthotomy, and rapid recognition of the cause and appropriate management is vital given the disastrous outcome of permanent and irreversible visual loss.[3]

Iatrogenic orbital injury is associated with the extent of ESS. A study of 50,734 patients in Japan showed that the occurrence of total orbital injury was high in patients who had undergone ethmoidectomy, maxillary antrostomy with frontal sinusotomy (<0.2%) compared to zero orbital injuries if only maxillary antrostomy was done.[2] Our patient underwent uncinectomy, partial turbinectomy for the concha bullosa, and antrostomy. The surgeon clearly identified the medial orbital floor, and no further surgery was done beyond the area. This is why orbital injury was highly unlikely in relation to the extent of ESS performed in this case. This was corroborated by the post-operative PNS CT scan, which showed an intact lamina papyracea and no demonstrable periorbital emphysema or retrobulbar hematoma, ruling out direct trauma to the eye. This led the authors to look for other causes of perioperative visual loss especially because this was a probable case of delayed sudden blindness.

Perioperative visual loss (POVL) following a non-ocular surgery has a reported incidence of around 0.056 to 1.3%.[5] The highest POVL rates are associated with cardiac and spine surgery, with an incidence rate of 0.09% and 0.2%, respectively.[6] Although the pathophysiology of POVL is still unknown, proposed mechanisms include increased intraocular pressure, ophthalmic vein congestion, emboli, or direct pressure to the globe, which causes ischemia or vasospasm.[5]

The etiology of POVL can be classified into ischemic optic neuropathy (ION) and retinal vascular occlusion, such as central retinal artery occlusion (CRAO) and branch retinal artery occlusion (BRAO).[5] These were all considered in our case.

Acute retinal ischemia produces permanent visual dysfunction due to the central retinal artery or a retinal artery branch blockage, which

causes retinal hypoperfusion.[5] This leads to rapid and progressive cellular damage then vision loss.[7] Central retinal artery occlusion following ESS has been cited in the literature and usually presents with sudden painless monocular vision loss.[8] Visual acuity may vary from loss of light perception to counting fingers but IOP and EOM are normal.[7,8] Our patient initially had normal IOP and EOM, but when the IOP rapidly increased to 40mmHg and EOM limitations were also noted, a lateral canthotomy was performed. Fundoscopy is the gold standard in the diagnosis of CRAO, with intravenous fluorescein angiography (FA) and/or electroretinography as confirmatory tests.[7] Fundoscopic findings in patients with CRAO show a diffusely pale retina with a cherry-red central spot[7] but these were not evident in our patient. Fluorescein angiography and succeeding fundoscopic examination was not possible because of severe anterior chamber reaction blocking the view to the lens and posterior pole.

Since we did not have any evidence of which vessel was most likely affected, the authors conjectured that it might have been the ophthalmic artery as the main blood supply of the eye. Could it have been a thromboembolic event or vasospastic event leading to ischemia of the eye, with reperfusion injury[5] taking place when the obstruction or vasospasm resolved? Occurrence of ophthalmic artery vasospasm has been reported, and although extremely rare is particularly important to recognize and treat accordingly as permanent, irreversible vision loss is usually seen in 60–90 minutes.[3]

Another pertinent finding in our patient was retinal detachment. This event was associated with clinical findings related to increased orbital compartment pressure such as proptosis, limitations of EOMs, chemosis, and retinal loss. These were all seen in our patient as well as on MRI. Retinal Detachment (RD) has been found to be associated with ocular artery vasospasm in animal studies.[5] One plausible theory that the resistance or obstruction of the venous outflow from the eye can lead to RD may be attributed to ophthalmic artery vasospasm. Obstruction to the venous outflow can rupture fine pupillary and retinal capillaries which may result in significant hemorrhage spreading to the subretinal space, followed by retinal hypoperfusion, eventually leading to retinal detachment.[5] This mechanism may explain the events in this case, resulting in blindness. However, due to unavailability of Optical Coherence Tomography (OCT), we failed to gather additional information that could have explained what happened to his eye vessels. It could have been a good diagnostic tool that would have aided in the diagnosis of the case.[5] A follow-up indocyanine green or fluorescence angiography was not possible because of severe anterior chamber reaction blocking the view to the lens and posterior pole. As for treatment, prompt administration of vasodilating sublingual nitroglycerine has prevented a patient with presumed ophthalmic artery vasospasm from suffering permanent visual loss.[3] In our case, nitroglycerine sublingual tablet and intravenous dexamethasone were given 24 hours post-operatively, which improved the symptoms of orbital swelling but not the patient's vision.

Finally, could the use of the topical nasal vasoconstrictor 1:1000 epinephrine during ESS cause ophthalmic artery vasospasm? Numerous studies have been published on the safety and usefulness of epinephrine in ESS.[9-11] Ophthalmic artery spasm is only highly probable in surgeries extending into the skull base wherein the ethmoidal arteries and the optic nerves can be compromised.[2] In our case, topical epinephrine was bilaterally instilled to optimize the endoscopic field, yet only the left eye was affected.

Direct trauma to the optic nerve or orbital hemorrhage are the most common causes of acute blindness after ESS. In their absence however, the rare possibility of non-iatrogenic delayed sudden perioperative blindness must also be considered. The series of events in our case may plausibly be attributed to ophthalmic artery vasospasm which could have caused permanent blindness. The evidence about its possible mechanisms (such as irregularities in vascular endothelial muscle activity) is still inconclusive and may be conjectural but acknowledging that such conditions have the risk of happening may aid otolaryngologists and ophthalmologists in their early detection and prompt initiation of appropriate management can lead to saving a post-ESS patient's vision.

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