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Unilateral blindness following nasal septoplasty

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Abstract. *Unilateral blindness following nasal septoplasty. Problems/objectives*: Septoplasty is a frequently performed ENT surgical procedure and anatomical reference points are well established. Nevertheless, it can be associated with serious complications.
Methodology: We report on a 27-year-old female patient, who was admitted to our hospital 16 days after septoplasty in another institution. She complained of vision loss in her left eye and yellowish discharge from the nose. The MSCT scan revealed fracture of the upper and lateral walls of the left sphenoid sinus with the bone fragment penetrating the left optic canal and signs of left maxillary, ethmoid, and sphenoid sinusitis. During functional endoscopic sinus surgery, a lesion of the left optic canal was clearly seen.
Results: Despite many treatments, the patient was released from our hospital without any residual sight in her left eye.
Conclusion: Unilateral blindness is a rare, but devastating complication following septoplasty.

Introduction

Unilateral blindness is one of the most serious complications of septoplasty; although, it is exceptionally uncommon. Only a few cases of this complication have been published, with only one case involving a direct lesion of the optic nerve after septoplasty, which was verified with computed tomography (CT).[1] We report the case of a 27 year old patient with loss of vision in one eye, which she noted 3 days after septoplasty. To the best of our knowledge, this is the first report with endoscopic substantiation of the injury.

Case report

A 27-year-old female patient was admitted 16 days after septoplasty performed in another institution. The patient's history and available medical records revealed that before septoplasty local findings in the nose corresponded to deviation of the nasal septum to the right, and the surgery under general anesthesia was uneventful. Immediately after the surgery, she complained of nausea and headache, and on postoperative day 3 she noted vision loss in her left eye. A CT scan showed pneumocephalus on the left parietal side. Magnetic resonance imaging (MRI) showed a marked presence of liquid with inflammatory features in all paranasal sinuses and signs of edema and inflammation along the left optic nerve from the orbital apex towards the chiasm, without any pathological findings on the chiasm itself. An ophthalmologist and neurosurgeon were consulted and the patient was treated with mannitol, acetazolamide, pentoxifylline, ceftriaxone, and amikacin.

Ophthalmologic findings on postoperative day 3 suggested papillitis of the left optic nerve and ipsilateral maculopathy. The following day, there was edema of the optic discs in both eyes, the interpapillomacular bundle, and the macula of the left eye. On postoperative day 7, a CT scan showed no signs of pneumocephalus, and oral corticosteroid therapy (prednisone, 40 mg/day) was started. The next day ophthalmologic findings suggested bilateral withdrawal of the optic disc edema but the optic nerve was paler on the left eye.

According to available medical records, a sphenoidectomy was then performed. During that operation, the inferior-medial part of the anterior wall of the left sphenoid sinus was fractured and a thick mucosal hemorrhagic suffusion was seen in the sphenoid sinuses. After the surgery, the corticosteroid (prednisone) dose was increased to 120 mg/day and the patient was transferred to our hospital. All documentation of the radiology findings (CT scan, MRI) and the treatments that followed in the other hospital were provided in

written form since they were done in another country. To our knowledge, surgery was performed by an ENT specialist with several years of experience in nose surgery.

On admission to our institution, the patient was conscious, aware, and without fever. She complained of visual loss in the left eye and thick bloody-yellowish discharge from the nose. The blood tests were within the normal values, except for the leukocytes (11×10^9/L), neutrophils (86.3%), lymphocytes (10.8%), erythrocytes (3.96×10^{12}/L), hemoglobin (110 g/L), and hematocrit (0.329). The nasal swab sample was sterile. Clinical examination revealed a blood clot in the middle nasal meatus and posterior parts of the common nasal meatus on the left side, while the epipharynx contained mucopurrulent discharge without any signs of cerebrospinal fluid (CSF) leakage. Ophthalmologic examination showed symmetrical, circular pupils, while the left pupil responded consensually, but not directly to light. Visual evoked potentials (VEP) showed prolonged latencies (162 ms) and reduced amplitude (1.913 μV) on the left side. Neurological examination was normal. Ceftriaxone and fluconazole were introduced along with vasoconstriction nasal drops.

A multislice CT (MSCT) scan showed liquid content in the left maxillary, ipsilateral ethmoid, and sphenoid sinuses. In addition, the left sphenoid sinus was wide open on the anterior wall with visible fracture of the upper and lateral walls and a bone fragment penetrating the left optic canal (Figure 1). On the posterior part of the left orbital roof, a fracture was seen with a bone fragment separated and located in the area of the upper part of the optic canal, and the left optic nerve was edematous immediately in front of the bone fragment (Figure 2).

Endoscopic examination of the nose and paranasal sinuses was performed under general anesthesia. Foamy content with coagulated blood was found in the maxillary sinus and posterior ethmoid cells. In the posterior part of the septum, a defect (1×1 cm) was found next to the mobile sphenoid rostrum. Hemorrhagic content was evacuated from the sphenoid lumen together with bone fragments. Endoscopically, a lesion of the left optic nerve was clearly seen in front of the optic chiasm.

After the functional endoscopic sinus surgery, the patient received antibiotics (imipenem, cilastatin) and anti-edematous therapy (oral prednisone 120 mg/day, acetazolamide). Since no signs of infection ensued, the therapy was discontinued on day 4, and the nasal package was removed. Ophthalmologic examination before discharge from our hospital showed a pale optic disc on the left eye with no light perception. The follow-up VEP were only insignificantly different from the first VEP (latency 158 ms, amplitude 2.012 μV). On day 7, the patient was discharged without light perception in her left eye.

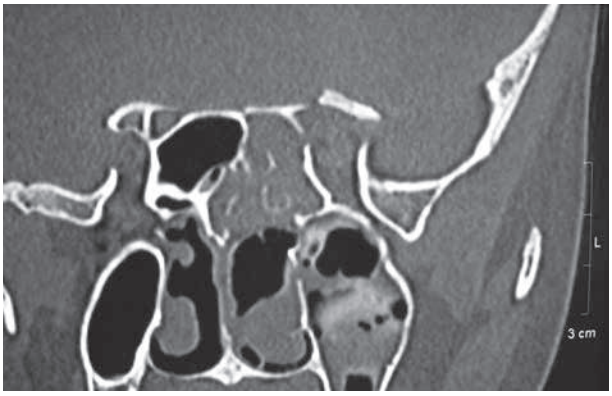

Figure 1
MSCT scan: Fracture of the upper and lateral walls of the left sphenoid sinus with the bone fragment penetrating the left optic canal.

Discussion

Although septoplasty is preformed quite frequently in ENT surgery and anatomical reference points have been well established, it is associated with a series of complications.[2-3] Even though septoplasty is one of the first surgical procedures performed by ENT residents, it is not simple. Surgical manipulations in the nose and paranasal sinuses require particular caution because of the surrounding nervous and vascular structures (cerebral structures of the anterior cranial fossa, optic nerve, internal carotid artery, cavernous sinus) and anatomical variations are to be expected. Orbital complications can be transient or cause permanent visual loss.[4]

To the best of our knowledge, there are only a few reported cases of visual loss after septoplasty. There were no anatomical causes of the visual loss identified; therefore, in most cases the event was attributed to intra-arterial injection of the local anesthetic and adrenalin in the region of the septal mucosa or nasal conchae, which due to the

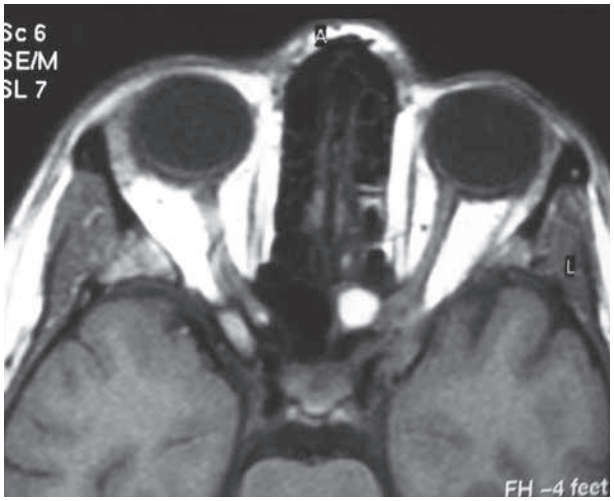

Figure 2
MSCT scan: Edematous left optic nerve in front of the bone fragment on the posterior part of the left orbital roof.

retrograde flow leads to the occlusion and spasm of the ophthalmic artery branches.[5-9] Another reported case occurred after re-septoplasty due to an old nasal fracture, and unilateral proptosis ensued with ophthalmoplegia and loss of vision in that eye with a dilated pupil and no response to light. [10] In this case, the postoperative CT scan showed an intact orbital wall, and there were no signs of massive bleeding after orbitotomy. The MRI findings suggested inflammation of the retro bulbar tissue, which was likely due to aberrant blood vessels between the nasal mucosa and orbit resulting from an old trauma, previous surgery, or a reaction of the tissue to local anesthetic. An unusual case of unilateral visual loss and 3rd cranial nerve palsy after a nasal septoplasty was reported due to nasal packing extending from the right nasal cavity into the right frontal lobe passing through the right orbit.[11]

In our case the presence of pneumocephalus indicated an injury of the skull base and dura, which can be a devastating complication of septoplasty.[12-13] Injury of the skull base didn't cause any significant CSF leakage and the nasal microbiology test was sterile, presumably due to previous antibiotic therapy. The MSCT scan showed the presence of bone fractures on the upper and lateral walls of the left sphenoid sinus and at the orbital roof. On admission to our institution, prolonged latency of VEP was demonstrated for the left eye, suggesting hindered propagation of impulses along the sight pathways. On discharge from our hospital, the pale optic disc on the left eye suggested atrophy of the optic nerve, which was substantiated by the VEP at discharge and a year later.

On the basis of radiological and endoscopic findings, we concluded that this complication of septoplasty was a surgical mistake and that the chisel slipped upwards, which caused a fracture of the left sphenoid sinus and directly injured the optic nerve in the region of the optic canal. The probable cause could have been an inadequate position of the head during the operation. Our patient noted visual loss on postoperative day 3. In this particular case, it is also possible that the loss of optic nerve function was gradual – due to a combined effect of direct trauma and subsequent inflammation.

We opted for an endoscopic surgical approach because of persisting sinusitis, which was resistant to antibiotic treatment. After removal of the bone fragments from the left sphenoid sinus, the site of the lesion of the left optic canal was clearly seen immediately in front of the chiasm and there was subsequent protrusion of the optic nerve towards the chiasm. Decompression of the optic nerve was not considered since the proposed time for recovery was exceeded.

Conclusion

Unilateral loss of vision is a rare, but extremely serious complication of septoplasty. This surgical procedure has to be performed with utmost caution since vital and functionally important structures are situated around the surgical site with possible anatomical variations. This is particularly relevant for the surrounding nerves and blood vessels. Surgeons must be aware of the possible complications and surgical mistakes, proper head positioning of the patient during the operation, and must avoid trauma of the surrounding tissue. Management of this complication includes MSCT of the correct quality with meticulous exploration of the orbit and optic canal wall followed by optic nerve decompression with steroids or surgical intervention. Accordingly, patients have to be informed about all possible risks and complications associated with this operation for underlying legal reasons.

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