Orbital Emphysema

Dear Editor:

Nose-blowing causing orbital emphysema is rare, but has been reported with or without orbital floor fractures and sinusitis. We report the case of a 26-year-old man with a history of an unrepaired right orbital floor fracture without visual sequelae 5 months earlier who blew his nose and developed orbital emphysema with complete loss of vision in the right eye. He also had concomitant right maxillary sinusitis. This case illustrates that orbital emphysema may occur long after orbital fracture and raises the question whether sinusitis in the setting of a previous orbital fracture predisposes one to orbital emphysema.

Our patient presented with right exophthalmos, pain, and vision loss after blowing his nose 16 hours earlier. His history was significant for a "cold" for 1 week and an unrepaired right orbital floor fracture without ophthalmic sequelae 5 months prior. Visual acuity (VA) was no light perception (LP), intraocular pressure (IOP) was 80 mmHg, the pupil was nonreactive, and complete external ophthalmoplegia was noted in addition to exophthalmos (Fig 1 [available at http://aaojournal.org]). Dilated fundus examination was unremarkable. Computed tomography demonstrated right orbital emphysema, exophthalmos, and maxillary and ethmoid sinusitis. The optic nerve was tethered, and the posterior globe angle approached 90° (Fig 2 [available at http://aaojournal.org]). No evidence of orbital cellulitis was noted clinically or radiologically. Lateral canthotomy and cantholysis decreased the IOP to 50 mmHg. Twenty-three–gauge needle orbital decompression performed under topical and local anesthesia aspirated 3 ml of air and 1 ml of fresh blood from the superomedial orbit. Intraocular pressure then decreased and stabilized in the mid-20s. An otolaryngological consultation was obtained, and the patient underwent endoscopic excision of the middle turbinate and uncinate process with maxillary antrostomy 8 hours after admission. There was purulence in the maxillary sinus, and culture revealed coagulase-negative *Staphylococcus*. No gas-producing organisms were isolated. Acid-fast bacilli and fungal cultures were negative. Endoscopic anterior ethmoidectomy also was performed to explore the lamina papyrecea, which was found to be intact. The patient was seen in follow-up over several weeks and showed improvement in exophthalmos, edema, motility, and sinusitis. However, VA remained no LP in the affected eye.

The timing of the orbital emphysema in relation to the fracture and the associated sinusitis are interesting features of this case. Orbital emphysema after nose blowing appeared in the Medline peer-reviewed literature in 1968.[1] Orbital emphysema may cause an orbital compartment syndrome, leading to exophthalmos, increased IOP, and optic nerve stretching; compression; and/or ischemia with irreversible blindness. Treatment includes lateral canthotomy/cantholysis and orbital decompression by needle aspiration, bony decompression, or a combination thereof. The mechanism of orbital emphysema is not fully understood. Shinohara et al proposed that it depends on disruption of the sinus mucosa.[2] Most cases associated with orbital fractures occur in the immediate posttraumatic period, and one should note that orbital floor fracture repair itself does not create an airtight seal, as the mucosa is not directly repaired but rather supported postoperatively while the body heals the defect over time.

Even without surgery, our patient's fracture should have been healed by 5 months, and there was no radiologic evidence of a new fracture. Purulence in the maxillary sinus implied infection before the acute incident, which may have weakened the mucosal lining, an already thin barrier over a preexisting floor fracture site, predisposing it to air conduction. In the absence of trauma, the lamina papyrecea has been considered the most susceptible to injury after nose blowing.[3] The medial location of the air in this case may suggest that the air entered the orbit medially. However, orbital air may redistribute with changes in the patient's position. Furthermore, the lamina papyrecea was intact intraoperatively, suggesting that the medial wall was not the route of air entry. No orbital cellulitis or central retinal artery occlusion was present in this case. Therefore, the visual loss was thought to be the result of optic nerve damage from orbital compartment syndrome causing optic nerve compression, optic nerve stretching, or both.

In summary, we present a patient with orbital emphysema with associated sinusitis occurring late after orbital fracture. It may be prudent to advise patients to avoid vigorous nose blowing and to consider treating sinus disease more aggressively if the patient has a history of ipsilateral orbital fracture.

Scott L. Lee, MD
David M. Mills, MD
Dale R. Meyer, MD
Steven M. Silver, MD
Albany, New York

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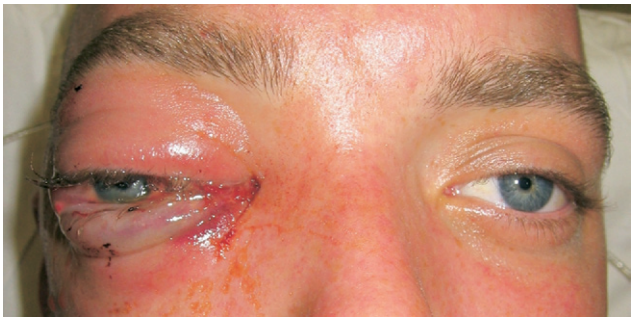

Figure 1. Complete external ophthalmoplegia and exophthalmos.

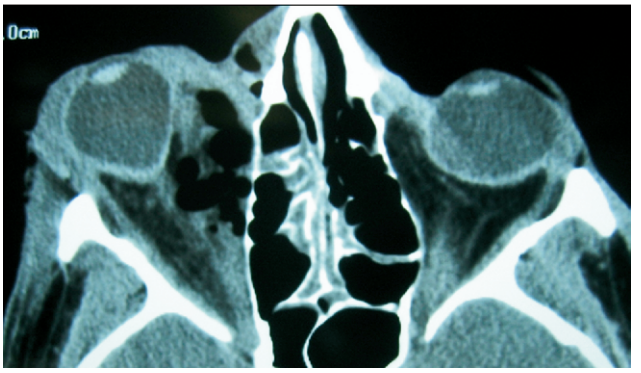

Figure 2. The optic nerve was tethered, and the posterior globe angle approached 90°.