ISSN: 2349-9141

Available online at http://www.ijirr.com

International Journal of Information Research and Review
Vol. 2, Issue, 04, pp. 628-630, April, 2015

IRR INTERNATIONAL JOURNAL

OPEN ACCESS JOURNAL

Case Study

UNILATERAL ORBITAL EMPHYSEMA FOLLOWING FORCEFUL NOSE BLOWING: A CASE REPORT

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Received 17th April 2015; Accepted 30th April 2015

Abstract

Orbital emphysema is an abnormal condition in which air is present within orbit. It is commonly seen in fractures or bony dehiscence of ethmoid sinus. We present a case of a 45 year old female who developed sudden onset of painless orbital emphysema following forceful nose blowing. There was no vision defect, no previous sinus disease, surgery or facial trauma. CT scan of orbit and PNS showed a deficient area in lamina papyracea with prolapse of orbital fat into the adjacent anterior ethmoidal cells with preseptal emphysema. Patient was managed conservatively with oral antibiotics, nasal decongestants and avoiding nose blowing and straining. The condition resolved after one week.

Keywords: Orbital emphysema, Nose blowing, Lamina Papyracea.

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To cite this paper: *Swayamsidha H. Andhale, Nicola Barreto and Pranav P. Nagarsenkar. 2015. Unilateral orbital emphysema following forceful nose blowing: A Case Report. International Journal of Information Research and Review. Vol. 2, Issue, 04, pp. 628-630.*

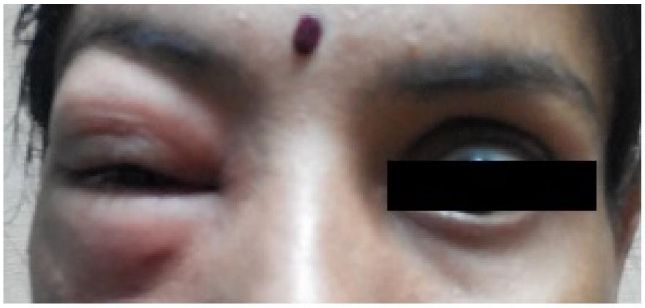
INTRODUCTION

Orbital emphysema is an abnormal condition in which air is present within the orbit (Muhammad and Simpson, 1996). Air in orbital emphysema most commonly enters from fractures or bony dehiscences of the ethmoid sinus and rarely results from isolated maxillary fractures or complex fractures involving the sphenoid and frontal sinuses (Lu *et al.,* 2006; Rosh and Sharma, 2008; Som and Curtin, 2003). This condition is generally sudden in onset and follows a benign course and is treated by supportive measures (Mohan and Singh, 2001). Rarely can this condition potentially cause an acute orbital compartment syndrome with central retinal artery occlusion and ischaemic optic neuropathy (Lee *et al.,* 2006; Dobler *et al.,* 1993). Following is a case report of a patient who developed unilateral orbital emphysema after blowing of nose. The purpose of this article is to remind the readers about the simple and effective treatment of an uncommon condition to prevent potential visual loss.

Case Report

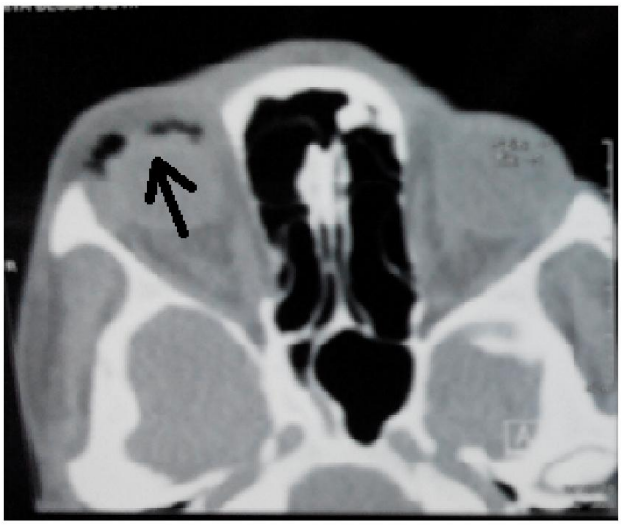
A 45 year old female patient presented with a sudden onset of painless swelling of both the eyelids of right eye after an episode of violent nose blowing (Figure 1). She had difficulty in opening the eyelids. There was no impairment of vision, double-vision, headache, fever or drowsiness. There was no history suggestive of sinus disease or recent or remote facial trauma or surgery. Patient examination revealed crepitus in right upper and lower eyelid. Otorhinolaryngologic evaluation was normal. On ophthalmic examination, the visual acuity was normal for both eyes. Proptosis was not present. There was no palsy of the external ocular muscles. Pupillary reactions were normal. The intraocular pressure was normal and the sclera, cornea, lens, vitreous, retina, and the optic disc were bilaterally normal. Her vitals were within the normal range. A plain computed tomographic (CT) scan of the orbits and paranasal sinuses was performed; 5.0 mm axial images were obtained with 1.0 mm reconstructions in both soft tissue and bone windows. Multiple air pockets were noted in preseptal soft tissues of the right orbit. Few air foci were also noted in extraconal fat above right globe. No intraconal air was evident. The lamina papyraceaof the right orbit was deficient over a segment of 12.0 x 8.0 mm^2. Resultant prolapse of retro orbital fat was noted into the adjacent anterior ethmoid cells. There was no evidence of muscle entraptment. Minimal mucosal thickening was noted in the sphenoid sinus on the right side of intersinus septum. Rest of the paranasal sinuses appeared normal. There was no proptosis. No bone fragment was noted in the region to suggest a fracture. Both optic globes and optic nerves were normal. The orbital walls on the left side were normal (Figures 2 and 3). A final diagnosis of dehiscence of the lamina papyracea with orbital emphysema (right) was made. It is most likely that the high air-pressures produced during violent nose blowing produced mucosal laceration in the dehiscent area with escape of air into the orbital tissues.

The patient was given oral prophylactic antibiotics, nasal decongestants, milking of air towards the nose and advised against blowing the nose or straining. On re-examination after three days, most of the periorbital air had been resorbed (Figure 4). One week later there was no clinical sign of orbital emphysema. The patient was discharged and she remained asymptomatic on follow-up.



Orbital emphysema developed acutely after forceful blowing of nose

Figure 1



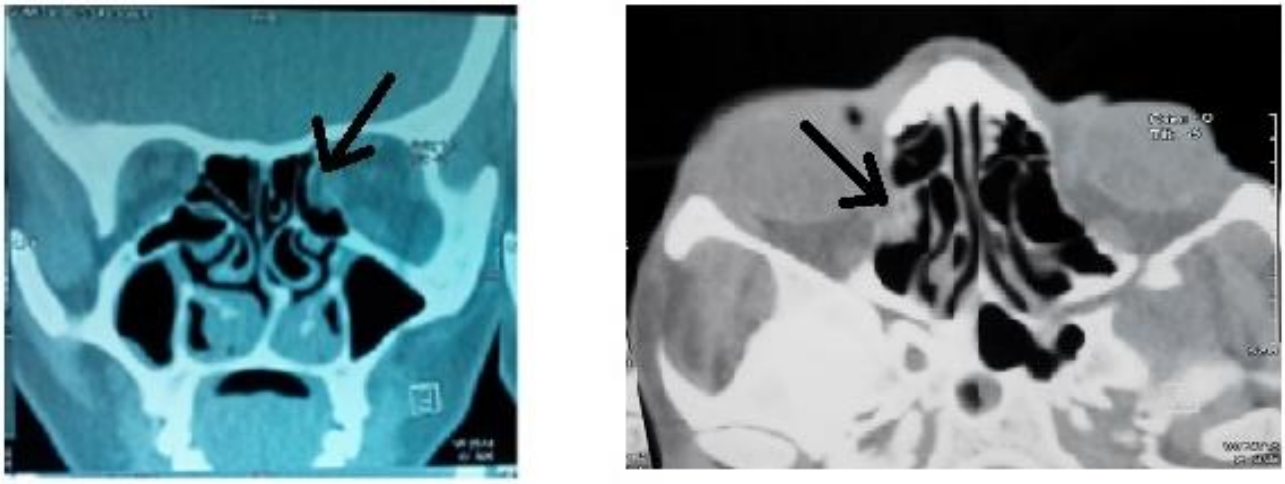
Axial section of orbit showing air trapped in preseptal region of right eye

Figure 2

DISCUSSION

The medial orbital wall is formed partly by the frontal process of the maxilla, the lacrimal bone, lateral wall of the ethmoid bone and the lamina of papyracea. Dehiscence of the lamina papyracea is an uncommon cause of orbital emphysema. Orbital emphysema in this condition may occur after any condition that increases nasopharyngeal pressure like after forceful nose-blowing, sneezing, coughing or vomiting, and scuba diving. Chronic mucosal disease may also weaken the lamina papyracea and lead to dehiscence secondary to elevated intranasal pressures generated from nose blowing. In this case, the orbital emphysema most likely occurred due to mucosal laceration at the dehiscent lamina papyracea with escape of air into the orbital tissues. This is the most common site of bony defect in this area and point of air entry into the orbit. The posterior limit of the dehiscence is always the basal lamella (bony attachment of the middle turbinate which separates the anterior and posterior ethmoidal air cells). The anterior limit is variable (Som *et al.,* 2003). The diagnosis of orbital emphysema is usually made by history alone, supported by results of external examination and confirmed with orbital CT. Orbital emphysema can be extraconal (periorbital) or intraconal (intraorbital). Extraconal (periorbital) usually presents as sudden onset crepitantperiorbital swelling. Subconjunctival ecchymosis, pain and tenderness and proptosis can also be present. Clinical characteristics of intraorbital emphysema may be similar to those seen for a post-traumatic retrobulbarhaemorrhage.

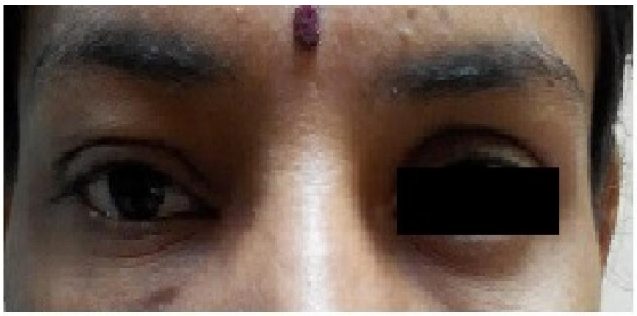
There may be diplopia, ophthalmoplegia, and vision loss. Any visual acuity abnormality in the setting of orbital emphysema should be considered an ophthalmologic emergency. The management depends on the emphysema extent and the severity of the symptoms. In all cases instructions must be given to the patient against nose blowing, sneezing, diving and flying and to refrain from performing a Valsalvamanoeuvre for 7–10 days. Nasal mucosal congestion is prevented by administering topical decongestants. Treatment options include observation as it is usually benign and spontaneous resolution occurs in two to three weeks (Wearne *et al.,* 1998). However, it can cause ischemic optic neuritis (Fleishman *et al.,* 1984) and central retinal artery occlusion and may lead to visual loss. Hence when orbital emphysema shows signs of pressure effect like restricted ocular motility, sluggish pupillary reaction



Coronal and Axial section of orbit and PNS showing prolapse of periorbital fat into ethmoid sinus on right side through dehiscent lamina papyracea

Figure 3

disc edema or decreased visual acuity then drainage of trapped air in the subcutaneous tissue should be considered (Dobler *et al.,* 1993). It can be done effectively by simple underwater drainage of air by 24-gauge needle (Benharbit *et al.,* 2003) or lateral canthotomy and cantholysis or bony decompression.



Orbital emphysema settled after 3 days of conservative treatment

Figure. 4

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