Orbital Emphysema: Case Reports and Review of the Literature

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- **Objective:** This study was designed to describe the usual clinical findings of orbital emphysema as well as unusual and infrequent but important causes of this condition.
- **Design:** We present seven detailed cases of orbital emphysema and review the pertinent English-language literature published since 1900.
- **Material and Methods:** Characterization of the seven patients with orbital emphysema illustrates the various causes and clinical findings of the disorder. Additionally, we review 78 previously published cases of orbital emphysema to determine the treatment and prognosis of this condition.
- **Results:** Trauma is the most frequent cause of orbital emphysema; however, orbital emphysema also may occur spontaneously or as a complication of pulmonary barotrauma, infection, and operation. In most cases, orbital emphysema resolves spontaneously without compromising ocular function. If excessive amounts of air accumulate within the orbit, however, complications such as occlusion of the central retinal artery or compressive optic neuropathy may lead to loss of vision if not recognized promptly and treated.
- **Conclusion:** In most cases, orbital emphysema is an incidental, benign finding that resolves with time. Careful observation is the only treatment necessary unless an orbital fracture involves an infected sinus, in which case prophylactic orally administered antibiotics may be prescribed.

Orbital emphysema is a well-described yet relatively uncommon clinical occurrence. Accumulation of air within the eyelids or orbit is most commonly associated with trauma and fracture of one of the orbital bones. Other causes include injury from compressed-air hoses, infection, pulmonary barotrauma, and a complication of operation. Although orbital emphysema is generally a benign condition, practitioners must be aware of possible vision-threatening complications and their immediate management. Herein we describe seven representative patients and review previously published reports of orbital emphysema.

**REPORT OF CASES**

**Case 1.**—A 17-year-old healthy boy was brought to the emergency department because of swelling of the left eyelid. This problem developed immediately after a forceful sneeze against a closed glottis and nose, during which the patient "felt something snap" in the left periorbital area. On examination, visual acuity was normal. The left eyelids were swollen shut, and subcutaneous emphysema of the lids was palpable. The intraocular tension was 12 mm Hg in the right eye and 20 mm Hg in the left eye. Results of the rest of the examination were unremarkable. The patient was treated with a 10-day course of orally administered erythromycin and recovered completely.

**Case 2.**—A 35-year-old healthy man sustained blunt trauma to the right eye, and a hematoma subsequently developed in the right upper eyelid. Visual acuity was normal, and the results of the rest of the examination were unremarkable except for mild enophthalmos of the right eye. Orbital radiographs, however, showed air in the superior right orbit, clouding of the right frontal and ethmoidal sinuses, and a fracture of the right lamina papyracea. Recovery was uneventful.

**Case 3.**—A healthy 14-year-old boy fell on ice and struck the left side of his forehead. One hour later, he blew his nose, felt several "pops" in the left upper eyelid, and then noted progressive swelling of the left eyelid. On examination, visual acuity was normal. The left upper lid was considerably swollen (Fig. 1 A), and air was noted under the conjunctiva; however, no facial bruising was evident. Proptosis (5 mm) of the left eye and restriction of upgaze...
were noted (Fig. 1 B). Intraocular pressure was 12 mm Hg in the right eye and 29 mm Hg in the left eye. Both fundi were normal. Facial radiographs showed air in the anterior soft tissues without evidence of orbital fractures (Fig. 2). Tomograms of the left orbit revealed air in the superior left orbit, but no fractures of the orbital walls were detected. The patient was treated with orally administered cephradine and recovered completely (Fig. 3).

Case 4.—A 41-year-old man was struck in the left eye by a high-pressure compressed-air hose. The following day he was referred for ophthalmic examination. Visual acuity was normal. Crepitus was present in the left upper and lower eyelids, and subconjunctival air was seen on slit-lamp examination (Fig. 4). A small conjunctival laceration in the inferior fornix was also noted. The remaining results of the examination were normal. Orbital plain films revealed no orbital or facial fractures. He was treated with orally administered cefadroxil and recovered without complications.

Case 5.—A 74-year-old man fell and sustained blunt trauma to the left orbit. His medical history was notable for resection of an orbital tumor from the left lateral orbital wall 11 years previously. On examination, visual acuity was 20/20 in the right eye and 20/30 in the left eye. Proptosis (6 mm), periorbital ecchymosis, total ptosis, and lid crepitus of the left eye were present (Fig. 5). The anterior segments and fundi were normal. A computed tomographic scan of the head revealed the previous postoperative changes in the lateral orbit, proptosis of the left eye, and air in the posterior left orbit (Fig. 6). Additionally, a fracture of the left maxillary antrum was detected. The patient was treated with cefuroxime administered intravenously and erythromycin applied topically to the prolapsed conjunctiva. One month later, recovery was complete, and visual acuity was 20/20-1 in the left eye (Fig. 7).

Case 6.—A 23-year-old man with migraine and sinus headaches came to the Mayo Clinic because of a 3-year history of producing “squishing sounds” when the left upper eyelid was palpated. Additionally, he was occasionally able to induce proptosis of the left eye with a Valsalva maneuver. On examination, visual acuity was normal in both eyes, and crepitus was evident on palpation of the left upper lid. Results of the rest of the examination were unremarkable. A computed tomographic scan of the head revealed an abnormal collection of air in the anterior medial and inferior left orbit.
Fig. 4 (case 4). Slit-lamp examination in 41-year-old man who was struck in left eye by compressed-air hose, showing subconjunctival air (arrows) and small conjunctival laceration in inferior fornix.

Fig. 6 (case 5). Computed tomographic scan of head, showing postoperative changes in left lateral orbit and proptosis and air (arrows) in left orbit.

Fig. 5 (case 5). Periorbital ecchymosis, subcutaneous eyelid emphysema, and subconjunctival emphysema in 74-year-old man who sustained blunt trauma to left orbit. (Completely ptotic upper lid is being held open.)

Fig. 7 (case 5). Complete recovery 1 month after injury. (Mild exotropia of left eye predated orbital trauma.)

orbit, and it seemed to communicate with the left nasopharynx. A diverticulum or fistula was considered present between the left lacrimal sac and the medial inferior orbit. Thus, the patient underwent exploration of the left lacrimal sac and a left dacryocystorhinostomy. Although no definite diverticula or fistulas were detected, 6 months later the patient reported that the symptoms of orbital crepitus had resolved entirely.

Case 7.—A 39-year-old man with a history of asthma and recurrent nasal polyposis underwent bilateral nasal polypectomies and bilateral external total ethmoidectomies. Immediately postoperatively, he had intermittent proptosis of both eyes when he exhaled. Visual acuity and results of the rest of the external ocular examination were normal. The patient was treated with orally administered cephalaxin and recovered without complications.

DISCUSSION
The first detailed account of orbital emphysema was published in 1904 by Heerfordt. His classification system for this entity included (1) palpebral emphysema, (2) true orbital emphysema, and (3) orbitopalpebral emphysema. Isolated palpebral emphysema (subcutaneous air confined solely to the eyelids) is uncommon. Palpebral emphysema may result from a fracture of the lacrimal bone and secondary rupture of the lacrimal sac anterior to the orbital septum. Air from the nose can then traverse the nasolacrimal drainage system and...
enter the eyelid tissues. Palpebral emphysema may also occur simply as a result of facial subcutaneous air spreading into the eyelids along fascial planes. As long as the orbital septum is intact, air should remain confined to the lid.

True orbital emphysema is an abnormal collection of air posterior to an intact orbital septum. Most commonly, this condition occurs as a result of fracture of one or more of the bony orbital walls and laceration of the adjacent sinus mucosa, factors that allow communication of a sinus with the orbit. Frequently, this fracture involves the ethmoid air cells and a break in the fragile lamina papyracea. Less often, fractures involving the frontal, sphenoid, and maxillary sinuses result in orbital emphysema. Notably, the entrance of air into the orbit does not occur spontaneously with the fracture but rather occurs intermittently when the pressure within the upper respiratory passages is increased (for example, with nose blowing or sneezing). Air also can be forced into the subconjunctival space of the orbit through a conjunctival laceration that results from injury involving compressed-air devices.

Orbitopalpebral emphysema may occur if a break is present in the orbital septum. As air accumulates within the orbit, the intraorbital pressure increases. Ultimately, the orbital septum may rupture, and air is allowed to traverse freely from the orbit into the eyelids. By experimentally injecting air into cadavers, Heerfordt\(^1\) found that a mean intraorbital pressure of 40 to 50 mm Hg was necessary to rupture the septum. The cadavers of older patients required much lower mean pressures to rupture the orbital septum, which becomes more attenuated with increasing age, than did the cadavers of young patients.

Since 1900, 78 cases of orbital emphysema have been published in the English-language literature. The underlying causes are listed in Table 1. Most of the cases (63%) were due to blunt trauma to the orbit, face, or head.\(^2\)\(^,\)\(^18\) Approximately half had radiographic evidence of a bony fracture into a sinus; the most common was a violation of the ethmoid air cells. Other cases associated with fractures involved the frontal or maxillary sinuses. Radiographic diagnosis of small fracture lines involving the sinuses is frequently difficult. Although not radiographically obvious, orbital wall fractures or defects presumably were the cause in the remaining cases. Additionally, in most of these cases, orbital emphysema was not apparent until after pressure inside the respiratory passages was artificially increased by sneezing, nose blowing, coughing, or emesis. Such maneuvers readily force air from the nasal sinuses through the fracture site into the orbital tissues. In one instance, recompression from a 300-foot dive in a compression chamber 1 day after peri-orbital trauma forced air through a presumed small orbital wall fracture and caused striking palpebral and hemifacial subcutaneous emphysema.\(^7\)

<table>
<thead>
<tr>
<th>Cause</th>
<th>No. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blunt trauma</td>
<td>49</td>
</tr>
<tr>
<td>Compressed-air injury</td>
<td>7</td>
</tr>
<tr>
<td>Postoperative complication</td>
<td>7</td>
</tr>
<tr>
<td>Pulmonary barotrauma</td>
<td>6</td>
</tr>
<tr>
<td>Infection</td>
<td>5</td>
</tr>
<tr>
<td>Spontaneous</td>
<td>2</td>
</tr>
<tr>
<td>Factitious</td>
<td>1</td>
</tr>
<tr>
<td>Esophageal rupture</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>78</td>
</tr>
</tbody>
</table>

Orbital emphysema has been reported to occur spontaneously after a musical wind instrument has been played\(^19\) or the nose has been blown violently,\(^20\) similar to the cause in one of our patients (case 1). No antecedent trauma or disease was noted in these instances, and a small congenital or acquired breach in one of the orbital walls was suspected.

In more recent years, an increasingly common cause of orbital emphysema has been injury involving high-pressure compressed-air guns or hoses. Approximately 10% of patients with orbital emphysema have sustained such compressed-air injuries.\(^21\)\(^,\)\(^27\) These cases are not associated with sinus fractures; air does not enter into the orbit from the respiratory passages but is most likely injected into the orbit by the pneumatic instrument. In several of these cases, the compressed-air blast was sufficient to rupture the orbital septum.\(^21\)\(^,\)\(^22\)\(^,\)\(^27\) These high-pressure systems may force air into the eyelids also, and then air may dissect along fascial planes to the face and neck or to the opposite eyelids. In two cases, such an air-blast injury was reported to cause not only orbital emphysema but also accumulation of air within the mediastinum or within the intracranial cavity.\(^23\)\(^,\)\(^24\) Air may have initially dissected along the lining of the optic nerve to intracranial spaces and to the carotid artery sheath and then into the mediastinum. Alternatively, air simply may have dissected along the cervical neck planes into the mediastinum.

Pulmonary barotrauma is another infrequent but important cause of orbital emphysema; it occurred in 8% of reported cases. Pulmonary barotrauma may cause subcutaneous emphysema, pneumomediastinum, and pneumothorax and usually results from mechanical ventilation. These complications are particularly known to occur in association with the use of high positive end-expiratory pressure. Two cases of subconjunctival emphysema as complications of positive end-expiratory pressure have been described.\(^28\)\(^,\)\(^29\) Pulmonary alveoli distend and may rupture because of high-pressure ventilation. This event can lead to escape of air initially into fascial tissues of the mediastinum and then, possibly along the vascular sheaths, into the subcutaneous tissue spaces of the chest wall, neck, and face. The appearance of subconjunctival emphysema indicates continuity between the sub-
conjunctival and subcutaneous spaces, but the mechanism of
dissection of air between these spaces is unclear. Two
patients with palpebral emphysema resulting from spontane-
ous pneumothorax have been described. These patients
suffered the same sequelae of pneumothorax, pneumo-
mediastinum, and subcutaneous emphysema. In these cases,
however, facial subcutaneous air dissected into the eyelids
and caused only palpebral emphysema. Two other cases of
pulmonary barotrauma with associated pneumomediastinum
have been described. In both instances, facial subcutane-
ous air was associated with tense orbital emphysema under
enough pressure to cause proptosis. This unusual route of air
dissection into the orbit is not yet well defined, but investiga-
tors have postulated that air may have initially tracked along
vascular sheaths to the base of the skull and then through the
inferior orbital fissures into the orbit.31

Orbital emphysema may occur as a complication of vari-
ous surgical procedures. Six such cases (8% of all reported
cases) have been described. In one instance, a patient under-
going bilateral endoscopic ethmoidectomies and creation of
nasal anastral windows. Approximately 30 minutes postop-
eratively, bilateral orbital emphysema and palpebral crepitus
were noted. The mechanism of orbital emphysema in this
case was the same as in the cases of blunt trauma except that
the break in the orbital wall was surgically produced. In a
recently reported case, subcutaneous eyelid emphysema de-
veloped after dacryocystorhinostomy. In this instance, manual
high-pressure ventilatory assistance was necessary postopera-
tively to treat laryngospasm. Eyelid swelling and palpable subcutaneous eyelid emphysema were noted there-
after. The investigators concluded that air was forced from
the lacrimal sac into the subcutaneous tissues of the eye-
lids. Intraorbital air has also been reported as a complica-
tion after a turbinectomy in one patient. In this patient, the
procedure had been performed 2 years before onset of ipsi-
lateral proptosis, which occurred with nose blowing. With
computed tomography and at subsequent operation, a locu-
culated air-containing sac communicating with the maxillary
sinus was found within the posterior orbit. When increased
pressure in the upper nasal passages forced air into this sac, it
became enlarged and caused proptosis. Because the air was
not free and nonencapsulated within the orbit, the investiga-
tors termed this an “intraorbital aerocele.”

Another unusual case of orbital emphysema occurred as a
complication of an intracranial-extracranial craniofacial sur-
gical procedure. As a part of this operation, the brain is
decompressed. Postoperatively, the brain reexpands and
fills the enlarged bony cranial vault. In this case, air was
pushed out the enlarged anterior fossa space into the orbit,
and massive orbital emphysema resulted.

Two cases of orbitalpalpebral emphysema occurred after a
dental procedure. In one case, the dental canal and the upper
roof of the maxillary sinus were perforated during drilling on
the root of an upper bicuspid. This situation allowed air to
spread into facial and palpebral subcutaneous tissues. In the
other case, a high-speed, pneumatically cooled rotary drill
was used to extract a mandibular second molar. During the
procedure, sudden hemifacial swelling was noted. This
complication was determined to be subcutaneous emphy-
sema involving the preseptal region. Investigators postu-
lated that the air coolant was forced through the gingival
wound into the fascial planes of the face, with dissection
superiorly into the eyelids. Finally, a case of subcutaneous
eyelid and subconjunctival emphysema was reported to oc-
cur after closure of a persistent tracheoectaneous fistula.

During this procedure, air was probably initially blown
through the tracheal wound into the subcutaneous tissues of
the neck and then spread into the face.

Infection as a cause of orbital emphysema has been infre-
quently described. All five reported cases were associ-
ated with paranasal sinusitis. Communication with the in-
fected sinus led to orbital cellulitis and orbital emphysema.
In one instance, air even traversed the orbital septum, and
subcutaneous facial and palpebral air resulted. In two pa-
tients, investigators postulated that the air within the orbit
originated in the nasal passages and passed through an or-
bital wall dehiscence caused by osteomyelitis. In two other
patients, the orbital air was thought to have been pro-
duced by a gas-forming organism within the orbit, although
results of culture did not prove this theory.

Unilateral orbital emphysema and proptosis have been
described as the initial features of Boerhaave’s syndrome
(spontaneous rupture of the esophagus). One patient had
pneumomediastinum, subcutaneous emphysema of the
chest, and other typical clinical findings of a ruptured
esophagus. The investigators postulated that the force of
esophageal air was great enough to push air along fascial
planes and into orbital tissues along a route similar to that in
the previously described cases of pulmonary barotrauma.

Finally, a bizarre case of factitious orbital emphysema in
a patient with Munchausen syndrome was reported in two
publications. This patient underwent an extensive work-
up for severe, debilitating orbital emphysema after exten-
teration of the other eye for similar symptoms. The orbital
emphysema was eventually determined to be self-induced by
repeated injection of air into the conjunctival sac.

Most of the usual clinical findings of orbital emphysema
were mentioned in the cases we describe herein. In cases of
palpebral or orbitalpalpebral emphysema, swelling of the
eyelids is usually obvious. Subconjunctival air may be
visible if orbital or orbitalpalpebral emphysema is present. In
most cases, crepitus is elicited by palpating the lids. If only
retroseptal air is present, crepitus may be elicited only indi-
rectly by applying pressure to the globe. Proptosis occurs
only in cases of true orbital emphysema with an intact orbital septum. If the orbital septum has defects, air will escape into the lids, and minimal or no proptosis occurs. In cases of severe proptosis and an increased intraorbital pressure, ocular motility may be limited. Depending on the cause of the orbital emphysema, concomitant findings may include subcutaneous air in the chest, neck, and face or signs of periorbital trauma.

Usually, orbital emphysema is a benign, self-limited condition that resolves spontaneously without complications within several days to weeks, as illustrated by a favorable outcome in each of the cases we describe. Indeed, serious complications related to orbital emphysema are uncommon, but when they occur they can be devastating. Of the 77 previously described patients, only 2 (3%) had permanent loss of vision directly attributable to orbital emphysema. Both of these patients had injuries sustained from high-pressure compressed air.21,23 The loss of vision (in one patient to the level of no light perception and in the other to the level of counting fingers) was due to optic neuropathy. In the first patient, intracranial air near the optic chiasm was demonstrated radiographically. In the second patient, air bubbles were noted along the intraorbital section of the optic nerve on computed tomography, and penetration of the orbital septum was evident during surgical exploration of the wound. The investigators postulated that the air forcibly injected into the orbit surrounded the optic nerve sheath and separated the nerve from its vascular supply (the surrounding pial plexus), an outcome that resulted in ischemia and optic atrophy.21 Another suggestion is that intraorbital air may cause optic nerve compression (as described in more detail subsequently) or even optic nerve transection.23 Two additional patients with orbital cellulitis and orbital emphysema became blind, but that loss of vision was most likely a result of the infection rather than a direct effect from orbital air.40,43

Another reported complication of orbital emphysema is a dramatic, transient increase in intraocular pressure as high as 100 mm Hg (normal, 8 to 22).17,26,27,47 This outcome results from an increase in intraorbital pressure as air accumulates posterior to an intact orbital septum. As mentioned previously, in cases of orbital fractures, air is forced from the sinus into the orbit when pressure in the upper respiratory passages is artificially increased, such as when sneezing, coughing, vomiting, or blowing the nose. In most instances, air can escape through the fracture site as readily as it entered, and a high intraorbital pressure is not sustained; however, if a one-way valve mechanism due to a bone fragment or a lobule of orbital fat is present at the fracture site, air may enter and remain trapped in the orbit. The result is an increase in intraorbital pressure and, in some cases, a consequent increase in intraocular pressure. Proptosis of the orbital contents or rupture of the orbital septum or Tenon's capsule and escape of air into the eyelids or subconjunctival space are protective measures that may lower intraorbital pressure. Intraorbital pressure may be monitored indirectly by measuring the intracocular pressure.18

Five patients with transient loss of vision caused by a substantial increase in intraorbital pressure associated with traumatic orbital emphysema have been described.3,6,8,47 Each patient had an abrupt decrease in visual acuity (20/400 to counting fingers) after emesis, coughing, or nose blowing. In three patients, occlusion of the central retinal artery was confirmed by ophthalmoscopy, whereas in the other two patients, the central retinal artery was patent at the time of the examination. Four patients underwent a decompression procedure in an attempt to salvage vision. The orbit was decompressed by direct aspiration of air with a needle, an orbitotomy, or a lateral canthotomy and cantholysis. After the procedure, the retinal circulation was normal, and vision was restored to baseline in all patients. Increased intraorbital pressure may obstruct not only the retinal circulation but also (as mentioned previously) the optic nerve pial and arachnoidal blood supply; optic nerve ischemia or retinal ischemia (or both) results.

The literature has no clear consensus on the management of patients with orbital emphysema. In all cases (except perhaps after injury from a compressed-air hose), patients should be advised to avoid any Valsalva maneuvers, such as sneezing, coughing, or nose blowing, which may force additional air from the nasal passages into the orbit. If retroseptal air is evident, the patient's vision should be periodically monitored. The intraorbital pressure may be indirectly monitored by measuring the intracocular pressure. If loss or threatened loss of vision is noted, air in the orbit must be immediately released by careful, direct aspiration with a needle. Infrequently, surgical orbital decompression may be needed. In urgent cases, the easiest surgical decompression is lateral canthotomy and cantholysis. Additionally, all patients with evidence of orbital emphysema should be advised to avoid flying or climbing to high altitudes, activities that may allow expansion of air within the orbital soft tissues.

For orbital fractures into a nasal sinus but no orbital emphysema, the patient should be instructed to watch for signs of orbital emphysema that may develop several hours later. In recent years, the tendency has been to treat orbital emphysema with orally administered antibiotics for 7 to 10 days to prevent orbital cellulitis. This approach is of unproven benefit, however. The use of antibiotic prophylaxis in orbital wall fractures remains controversial. Westfall and Shore48 stated that because normal sinuses are sterile, a simple fracture into them may be considered a clean wound. If sinusitis is present, a fracture into the sinus is considered a nonsterile wound. Prophylactic antibiotics seem prudent in such cases. A second-generation cephalosporin antibiotic...
should provide adequate coverage against most common bacteria that cause sinusitis. Any associated trauma, such as an orbital wall fracture, should be managed in the appropriate standard manner.

CONCLUSION

Orbital emphysema is an uncommon clinical occurrence. It is most often caused by trauma to the orbit, but it may also occur spontaneously or be caused by pulmonary barotrauma, operation, or infection. In most instances, orbital emphysema is an incidental, benign finding that resolves with time. Careful observation is the only treatment necessary unless an orbital wall fracture involves an infected sinus, in which case prophylactic orally administered antibiotics may be prescribed. Rarely, intraorbital pressure may increase as air accumulates within the orbit and lead to transient or permanent loss of vision due to occlusion of the central retinal artery or optic neuropathy. In such cases, the orbit may need to be decompressed.

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