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Barotraumatic Orbital Emphysema in Divers: Comprehensive Review with Case Series

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Abstract

Aim. The aim of this paper is to outline the pathophysiological mechanisms underlying orbital emphysema in the context of barotrauma, which may occur in divers, to discuss the associated symptoms, diagnostic evaluation, and management, and to present the clinical cases described in the literature.

Material and methods. The research methods applied included a systematic review of the PubMed, ResearchGate and Cyfrowa Biblioteka Medyczna UJCM databases, as well as other relevant sources, using the keywords: “orbital emphysema”, “diver”, “dive”, “scuba diving”, “eye barotrauma”, “paranasal sinuses anatomy”, “orbit anatomy”.

Summary. Orbital emphysema refers to the presence of air within the orbital cavity, most often resulting from fractures of the orbital walls or barotrauma. Its clinical presentation varies from mild eyelid swelling and crepitus to more severe manifestations such as proptosis, restricted ocular mobility, or elevated intraorbital pressure. Management is conservative in most cases, with spontaneous resolution occurring over several days to weeks; however, urgent decompression may be required if orbital compartment syndrome develops. Among divers, only mild forms of orbital emphysema have been reported to date. Preventive measures include proper pressure equalization during diving, avoidance of forceful Valsalva maneuvers, and refraining from diving during episodes of sinus infection.

Conclusions. Barotrauma-related risk increases when pressure equalization is impaired, the osteomeatal complex is obstructed, or minor facial injuries create pathways for air to enter the orbit during pressure changes. This review outlines the main features of orbital emphysema and its clinical manifestations in divers. Given the limited number of published cases, the condition is insufficiently described and requires further investigation.

Key words: orbital emphysema, diving, barotrauma.

Content

1. Introduction

Orbital emphysema refers to the presence of air within the orbital cavity. This condition most commonly results from a fracture of the orbital wall, creating a pathway between the orbit and the paranasal sinuses. It may also develop as a consequence of barotraumatic injury. Clinically, patients may present with periorbital pain, eyelid swelling, crepitus, diplopia, and restricted extraocular movements. In severe cases, rapid accumulation of intraorbital air can increase orbital compartment pressure, leading to optic nerve compression and potentially irreversible vision loss.

Exposure to environments with fluctuating ambient pressure, such as during scuba diving, contributes to the development of barotrauma. Therefore, adequate preparation for such conditions, awareness of potential complications, and knowledge of appropriate management are crucial to prevent severe risk of vision impairment.

2. Pathophysiology

2.1. Orbital and paranasal sinus anatomy related to pressure regulation

The orbit is a rigid cavity composed of soft tissues and a framework formed by seven bones: the maxilla, and the zygomatic, frontal, ethmoid, lacrimal, sphenoid, and palatine bones. Under physiological conditions, the thin walls separating the orbit from the sinuses remain structurally intact, preventing air movement into the orbital cavity. Of particular importance is the lamina papyracea, which separates the orbit from the ethmoid sinuses and often contains natural defects (dehiscences). It has a very delicate structure, making it susceptible to injury.

Its fragility and frequent anatomical dehiscences allow air to be forced into the orbital space when there is a sudden increase in pressure within the nasal cavity or paranasal sinuses, such as during nose blowing, sneezing, coughing, or performing Valsalva maneuvers.

The anatomy of the orbit and paranasal sinuses is closely interconnected, and the presence of natural air-filled spaces plays a crucial role in pressure dynamics during exposure to fluctuating ambient pressure. The maxillary sinuses border the orbit inferiorly, the frontal sinuses superiorly, the ethmoid cells (sinuses) medially, and the sphenoid sinuses postero-medially. The anterior ethmoid cells, frontal sinus, and maxillary sinus drain through the region of the lateral nasal wall known as the osteomeatal complex (OMC). This structure enables the

paranasal sinuses to maintain continuity with the nasal cavity and, indirectly, with the external environment. Patency of the OMC, together with proper function of the mucociliary apparatus, is essential for maintaining appropriate pressure equilibrium between the paranasal sinuses and the ambient conditions (Bleier et al., 2019, Deniz et al., 2024).

2.2. Pathogenesis of the orbital emphysema

Orbital emphysema is most often caused by a blunt injury to the orbit or face, accounting for about 63% of cases. Other cases include postoperative complications, infection, oesophageal rupture, and barotrauma. There are also instances in which the condition develops as a result of forceful sneezing or nose blowing (Zimmer-Galler et al., 1994).

Orbital emphysema was first thoroughly described in 1904, and categorized into three types: palpebral emphysema, true orbital emphysema, and orbitopalpebral emphysema (Heerfordt, 1904).

Palpebral emphysema is uncommon and refers to air confined to the eyelids. It can occur after a fracture of the lacrimal bone with rupture of the lacrimal sac, allowing air from the nasal cavity to enter through the nasolacrimal system into the eyelids. It may also develop when facial subcutaneous air spreads along fascial planes. As long as the orbital septum is intact, air does not enter the orbit.

True orbital emphysema involves air located posterior to an intact orbital septum. It most often results from fractures of the orbital walls combined with a tear in the adjacent sinus mucosa, creating a direct communication between a sinus and the orbit. This commonly involves the ethmoid air cells and a defect in the lamina papyracea, though the frontal, sphenoid, or maxillary sinuses may also be involved. Air typically enters the orbit not at the moment of injury but when intranasal pressure rises, such as during nose blowing, sneezing or being unable to reduce pressure under changing ambient pressure conditions.

When a pressure gradient arises between the paranasal sinuses and the orbit, the air is forced across anatomical barriers that are capable of withstanding only minimal stress.

In situations involving fluctuating ambient pressure, fractures of the orbital walls, or the presence of natural dehiscences, a pressure gradient between the sinus and the orbit may allow air to enter directly into the orbital tissues. This mechanism becomes particularly relevant in barotrauma associated with diving, where rapid pressure changes can exceed the capacity of normal sinus ventilation, forcing air to traverse the weakest anatomical boundaries.

Obstruction of the osteomeatal complex can significantly contribute to disruption of the osseous barrier, particularly the lamina papyracea. A patent osteomeatal complex allows air to move freely between the nasal cavity and the paranasal sinuses, ensuring pressure equalization without damaging surrounding structures, even under conditions of changing ambient pressure, such as during scuba diving.

When the OMC becomes obstructed, free movement of air is impaired, leading to elevated pressure within the ethmoid sinus and, consequently, to damage or rupture of the lamina papyracea with subsequent passage of air into the orbit.

Orbitopalpebral emphysema can develop when the orbital septum is disrupted. As air builds up inside the orbit, intraorbital pressure rises. If this pressure becomes too high, the septum may tear, allowing air to move freely from the orbit into the eyelid tissues (Zimmer-Galler et al., 1994, Lee et al., 2024, Marfatia et al., 2025, Yilmaz et al., 2015).

2.3. Barotrauma risk associated with descent and ascent

To prevent barotrauma in divers, equalization of pressure between air-filled spaces and the surrounding environment is particularly important during ascent. According to Boyle's law, the volume of a gas is inversely proportional to pressure: during descent, trapped air decreases in volume, whereas during ascent it expands. Both the compression and decompression can result in barotrauma to the surrounding tissues.

Divers commonly refer to the effects of negative pressure developing within closed air spaces during descent as "squeezes". The middle ear, the paranasal sinuses, and the area under the diving mask are the sites most commonly affected, leading respectively to middle-ear squeeze, sinus squeeze, and mask squeeze. The mask squeeze and the sinus squeeze play a crucial role in the development of ocular and orbital injuries.

The mask squeeze primarily affects the soft tissues and, in most cases, is mild and does not require treatment. It most commonly occurs in novice divers. During descent, the diver should exhale gently through the nose into the mask to equalize the low pressure inside it. Failure to perform this manoeuvre results in compression of the air inside the mask and may lead to barotrauma. Wearing swimming goggles can cause a similar type of injury, as they do not cover the nose and therefore prevent proper pressure equalization. Negative pressure inside

the mask pulls the surrounding soft tissues inward, leading to capillary rupture in the periorbital region and petechial haemorrhages of the conjunctiva.

Barotrauma in divers leading to orbital emphysema is most often caused by damage to the lamina papyracea as a consequence of sinus squeeze or reverse sinus squeeze. The mechanism of injury is related to obstruction of the osteomeatal complex (OMC), which prevents pressure equalization. Sinus squeeze occurs during descent, when the low pressure inside the sinuses creates a vacuum effect that pulls in and damages the surrounding tissues.

During ascent, the pressure within the air-filled paranasal sinuses rises relative to the surrounding environment ("reverse sinus squeeze"). The resulting increase in intranasal gas pressure and its expansion exert force on the sinus mucosa, which may lead to its compression. In both of these mechanisms, the thin lamina papyracea is particularly vulnerable to the forces involved, which can lead to its damage and subsequently result in orbital emphysema.

It should be noted that in the presence of dehiscences within the lamina papyracea, air is capable of migrating into the orbital compartment despite the absence of fractures. In the context of reverse sinus squeeze, the expansion of air within the paranasal sinuses alone may be sufficient to force air into the orbit (Rudge, 1994, Pennell et al., 2014, Takeuchi et al., 2025).

3. Clinical Presentation

The clinical presentation of orbital emphysema can be highly variable and depends on the mechanism of injury, the size of the fracture and the dynamics of air accumulation within the orbit. In the reported clinical cases, the predominant findings were typical superficial signs such as swelling of the eyelid, restricted ocular motility, crepitus and subcutaneous emphysema. In the majority of cases the visual acuity was preserved and there were no significant abnormalities on external ocular examination. In some instances, additional symptoms included sudden visual deterioration, orbital pain, and diplopia. Elevated intraocular pressure was observed in a subset of patients, suggesting increased intraorbital pressure despite the absence of direct ocular injury.

In more advanced cases, findings included proptosis, marked restriction of upgaze, subconjunctival emphysema, eyelid hematoma, complete ptosis, as well as extensive subcutaneous and subconjunctival emphysema and concurrent maxillary sinus fracture. In several cases, particularly following blunt trauma, air within the orbit was detected despite the absence of fractures on radiologic imaging, confirming that disruption of the wall of the

paranasal sinuses is not required for orbital emphysema to occur (Jawaid, 2015, Oba et al., 2011, Ababneh 2013, Moon et al., 2016, Zimmer-Galler et al., 1994).

The literature describing cases of orbital emphysema demonstrates that this condition may present across a broad clinical spectrum: from nearly asymptomatic forms with minimal swelling and normal visual acuity to severe presentations with proptosis, impaired extraocular motility, and a significant rise in intraocular pressure. The consistent clinical feature is the characteristic presence of crepitus, while the variability in symptom severity depends on the location of the air, the rate at which it accumulates, and any concurrent fractures of the orbital or sinus walls (Moon et al., 2016, Zimmer-Galler et al., 1994).

A clinical study reported by have shown that at least one symptom, such as diplopia, laceration, afferent pupillary defect, hypoesthesia, or proptosis, was present in 47.1% of patients with orbital trauma. Notably, a statistically significant association was identified between orbital floor fractures and the occurrence of diplopia ($p = 0.002$), whereas no similar correlations were observed between other symptoms and radiologic findings (Aslan et al. 2020).

4. Complications

Orbital emphysema rarely results in serious complications and is typically a mild, self-limiting condition that resolves spontaneously within several days to weeks. Nevertheless, isolated reports describe severe outcomes, including permanent vision loss. Two such cases have been documented, both associated with high-pressure compressed air injury, with vision loss resulting from optic neuropathy. It has also been hypothesized that intraorbital air may exert compressive forces on the optic nerve or, in extreme cases, lead to its transection.

Another reported complication of orbital emphysema is a sudden, transient increase in intraocular pressure. As previously noted, in cases of orbital fractures, air may be forced from the sinus into the orbit when pressure within the paranasal sinuses rises. If a one-way valve mechanism develops within the OMC, air can enter the orbit but cannot escape. This results in elevated intraorbital pressure and, in some cases, a secondary rise in intraocular pressure, which may lead to transient loss of vision (Zimmer-Galler et al., 1994).

5. Diagnostic Evaluation

Orbital emphysema is diagnosed on the basis of patient history and physical examination. A plain radiograph may reveal orbital air, fracture of the orbital wall, as well as the “black

eyebrow sign," a crescent-shaped radiolucency in the superior orbit that indicates vertical globe dystopia. However, this modality may fail to detect subtle abnormalities.

The most accurate imaging modality for assessing orbital emphysema is computed tomography (CT). It allows precise localization of the air, determination of the type and size of the fractured segment of the medial orbital wall, and evaluation of any displacement of the extraocular muscles or the globe. In addition, CT enables the assessment of air within the soft tissues of the upper and lower eyelids, as well as the identification of associated injuries, including orbital or retrobulbar haemorrhage, extraocular muscle swelling, or muscle incarceration at the fracture site (Kang et al., 2024, Sawicki et al., 2011, Moon et al., 2016).

6. Management

A thorough ophthalmologic examination is essential to identify any of the serious complications described above and to guide appropriate management based on the patient's clinical presentation. Vision should be monitored periodically to assess whether the emphysema is progressing. Intraorbital pressure should also be evaluated indirectly by measuring intraocular pressure.

In severe cases, decompression of the orbit may be necessary, either by needle aspiration of the trapped air or by surgical intervention; in emergencies, lateral canthotomy and cantholysis is the procedure of choice.

There are no indications for antibiotic therapy unless sinusitis is present. In such circumstances, prophylactic antibiotics, most commonly second-generation cephalosporins, are recommended.

After the onset of orbital emphysema, patients should avoid activities that may force additional air into the orbit and thereby enlarge the emphysema. Activities resulting in pressure increase in sinuses, such a Valsalva manoeuvre, nose blowing, sneezing or coughing, should be avoided. Patients should also refrain from activities associated with reduced ambient pressure and expansion of intranasal or sinus air. This includes air travel, scuba diving, and high altitude climbing (Marfatia et al., 2025, Zimmer-Galler et al., 1994).

7. Orbital Emphysema in divers: case reports

7.1. Case 1

Five clinical cases of orbital emphysema in divers have been reported in the literature. One involved a 26-year-old diver who, during a scheduled wet training dive, experienced difficulty equalizing pressure using the Valsalva manoeuvre. He developed pain and swelling of the left orbital region, ptosis, and mild periorbital ecchymosis, with palpable crepitus on examination. An anteroposterior X-ray of the head demonstrated air around the left superior orbital margin. Coronal computed tomography revealed air bubbles within the subcutaneous tissue of the left periorbital region. Diagnostic imaging showed no evidence of bone fracture. The patient was managed conservatively, with complete resolution of symptoms after 10 days (Tseng et al., 2017).

7.2. Case 2

Another reported case involved a 23-year-old professional scuba diver who developed pain and swelling around the right eye after surfacing. During the dive, he experienced pain and pressure sensations in the region of the right maxillary sinus and posterior orbit. Approximately six months earlier, he had a similar episode, for which maxillary sinus barotrauma had been suspected, though no additional diagnostic studies were performed at that time. Computed tomography demonstrated gas bubbles in the extraconal anterior region of the right orbit, hypoplastic frontal sinuses, mild nasal septal deviation with no visible fracture. The patient was treated with high-flow oxygen therapy and maintained in an upright position overnight. He was discharged the following day with complete resolution of symptoms and advised to use intranasal decongestants (Pennell et al., 2014).

7.3. Case 3

A 26-year-old breath-hold diver experienced difficulty equalizing pressure in his right ear and developed tinnitus. The symptoms occurred during approximately the thirtieth descent while spearfishing with a mask covering both of his eyes and nose. To relieve these symptoms, he repeatedly performed the Valsalva manoeuvre. After surfacing, swelling and turgidity of the upper eyelid were noted. Physical examination revealed crepitus and ptosis of the right upper eyelid. Visual acuity in both eyes was normal, and no subconjunctival emphysema was observed.

Computed tomography revealed extensive soft-tissue oedema in the right orbital region with notable subcutaneous emphysema. Air was also observed within the orbital cavity, particularly along the roof and lateral wall. No fractures of the orbital bones or paranasal sinuses were identified; however, a defect of the lamina papyracea near the anterior ethmoid air cells was present. The orbital wall adjacent to the defect appeared thinner than in its superior portion, and no bone was visible at the site of dehiscence. It was presumed that the osseous defect preceded the incident and that repeated forceful Valsalva manoeuvres forced air into the intraorbital space, causing rupture at a structurally weakened area.

Treatment consisted of prophylactic oral antibiotics and intranasal decongestants. Follow-up examinations on the third and seventh day showed substantial resorption of the peri-orbital air. After two weeks, the external emphysema had fully resolved, although a small amount of intraorbital air likely remained for several additional days (Bolognini et al., 2008).

7.4. Case 4

Another reported case involved a diver who developed facial and left orbital swelling, as well as eyelid closure while using a surface-supplied air helmet. Two hours earlier, the diver had struck the left side of his face against the water surface, causing a short episode of eye pain prior to the incident. The symptoms developed after surfacing from a shallow-water training. Crepitus was detected in the periorbital region and upper left maxilla, and examination of the left eye was impossible due to swelling of the eyelid. Computed tomography revealed extensive orbital and facial emphysema on the left side, along with the presence of air within the lamina papyracea without evidence of a fracture. The clinical report did not describe the treatment administered in this case (Hall, 2013).

7.5. Case 5

In 1969, a case was reported involving a Royal Navy diver who developed symptoms during an air dive in a compression chamber. Four days before the dive, the patient had experienced nasal congestion, and the day prior he presented with a minor injury to the left circumorbital region. During the dive, as the ambient pressure decreased, he presented with acute pain accompanied by progressive swelling of the left cheek and eyelids, along with palpable crepitus. During ascent, air was aspirated with a needle, yet the swelling continued to increase until it involved the entire left side of the face, from the neck to the temple. A small

episode of epistaxis occurred later on the same day. Two sets of radiographs did not reveal any fracture. Over the next five days, the swelling gradually resolved, leaving only ecchymosis related to the initial trauma (Leitch, 1969).

8. Discussion

In all described cases, symptoms appeared during ascent or immediately afterwards. Two patients directly associated their symptoms with an inability to equalize pressure, while in two others orbital emphysema developed as a result of prior facial trauma. In four of the five patients, no orbital fractures were identified despite the presence of substantial amounts of air within the tissues. The remaining case involved a likely pre-existing lamina papyracea defect that was further disrupted by forceful Valsalva manoeuvres.

Symptoms common to all patients included periorbital swelling and pain involving the orbit or face. Frequently reported findings were ptosis and palpable crepitus. Additional symptoms included epistaxis and periorbital ecchymosis. Ophthalmologic examination was documented in three cases, none of which demonstrated abnormalities; the remaining two patients reported no visual disturbances.

Regardless of the management approach, all patients experienced complete resolution of symptoms within two days to two weeks. They were advised to refrain from diving for several weeks, to avoid hazardous conditions during descent, and to abstain from diving during episodes of an upper respiratory tract infection.

9. Conclusions

Orbital emphysema most commonly results from trauma to the orbital region. In divers, this condition is exceedingly rare, yet, as demonstrated by the cases described above, clearly possible. In environments where barotrauma may occur, increased caution and proper preparation are essential. Prevention of orbital emphysema requires maintaining patency of the osteomeatal complex, avoidance of forceful Valsalva manoeuvres, and refraining from diving during episodes of sinus infection. Even minor peri-orbital injuries may create a pathway between the sinus and orbital cavities, allowing air to enter the orbit under changing pressure conditions.

This review summarizes the key aspects of orbital emphysema and illustrates its clinical presentation in the reported cases. Given the limited number of documented episodes in divers, the condition remains insufficiently investigated and requires additional research.

Disclosure

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The authors deny any conflict of interest.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this article, ChatGPT by OpenAI was used to improve language quality, as well as to verify grammatical accuracy. After using the tool, the text was reviewed and corrected by the authors, and the final version was evaluated and approved by them. The authors accept full responsibility for the substantive content of the publication.

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