The introduction of air into subcutaneous tissues during dental procedures can be harmful. However, subcutaneous emphysema after root canal therapy is a rare complication, which is usually restricted to only moderate local swelling on the face and sometimes the neck. This comprehensive review of the literature explains all the possible causative factors of emphysema, as well as symptoms and signs that can be noticed. Finally, it illustrates the preventive measures and the appropriate management of subcutaneous emphysema, which is usually benign and improves within 2 to 3 days.

Keywords: Subcutaneous Emphysema; Root Canal Therapy; Compressed Air; Facial Swelling

Sotiria Liatiri
Thessaloniki, Greece

Introduction

Subcutaneous emphysema, sometimes abbreviated SCE or SE and also called tissue emphysema, occurs when gas or air is present in the subcutaneous layer of the skin. Subcutaneous refers to the tissue beneath the cutis of the skin, and emphysema refers to trapped air. Since the air generally comes from the chest cavity, subcutaneous emphysema usually occurs on the chest, neck and face, where it is able to travel from the chest cavity along the fascia. Subcutaneous emphysema can result from puncture of parts of the respiratory or gastrointestinal systems. Particularly in the chest and neck, air may become trapped as a result of penetrating trauma (e.g., gunshot wounds or stab wounds) or blunt trauma. Infection (e.g., gas gangrene) can also cause gas to be trapped in the subcutaneous tissues. Subcutaneous emphysema can be caused by medical procedures and medical conditions that cause the pressure in the alveoli of the lung to be higher than that in the tissues outside of them. It has been reported in the literature as a result of endodontic, periodontic, oral surgical and operative procedures. It is a possible complication of both nonsurgical and surgical endodontic treatment. When the condition is caused by surgery it is called surgical emphysema. Surgical emphysema may occur during root treatment and reports in the dental literature indicate that the complication occurs most commonly as a result of over-zealous irrigation with hydrogen peroxide, or drying root canals with compressed air blasts. Its progression and severity is limited by the fascial planes and by the amount of air that has been introduced. Sometimes, spread of larger amounts of air into deeper spaces may cause more serious complications, including accumulation of air in the retropharyngeal space, pneumomediastinum, and pneumo-pericardium.

The purpose of this review is to describe all the possible aetiologies of subcutaneous emphysema and present all the clinical symptoms and their importance to diagnosis. It also aims to emphasize the clinical guidelines for prevention and management of subcutaneous emphysema during endodontic procedures.

Etiologic Factors

Physiologically, subcutaneous emphysema can occur as a complication of coughing, sneezing, nose-blowing and vomiting, or other patients' actions that raise intrarotary pressure following a dental extraction. It can also be observed following facial injuries which involve nasal airways and paranasal sinuses. It is believed that in these cases emphysema follows nose sneezing in an attempt to clear the nasal passages or it may be due to oxygen administration with a
It is an infrequent occurrence following general anesthesia, but can occur due to hypopharyngeal, tracheal, or cervical esophageal injury during the intubation procedure. Subcutaneous emphysema occurring during dental procedures while using high-speed air or water drills has been documented mainly in the dental literature. It can be caused by invasion of compressed air into soft tissues through the disrupted intraoral barrier (dento-alveolar membrane or root canal) during tooth extraction (particularly of the third mandibular molars), restorative dentistry, dental implant surgery, and root canal or periodontal treatment. The underlying mechanism in all these procedures is a disruption of the intraoral barrier, allowing air under pressure to tract subcutaneously. A 1957 literature review by Shovelton found 13 reports of air emphysema from endodontic treatment. One of these cited a clarinettist whose playing forced air through an open root canal into his neck.

While opening the access cavity for endodontic treatment, subcutaneous emphysema can be caused by the use of an air-driven high-speed handpiece and compressed air-syringe. Especially the use of compressed air to dry a canal can be very risky for introducing air into tissues. Facial emphysema can also result from a series of technical errors, such as the enlargement of the perforation which increases the chances of injuring the periodontal tissues and entering air into the tissue spaces. Many cases of air entering tissues are complicated by inflammation and infection, perhaps from canal debris and/or microorganisms or perhaps from opportunistic microbes from other sources within the body that find the inflated space. Further risk is found in the fact that typical dental air sources do not provide sterile air. Clinically, in the early phases of endodontic treatment, necrotic tissue and microorganisms are often present, so air pressure within even small diameters can plausibly force such irritants periapically to initiate or worsen an inflammatory reaction.

Hydrogen peroxide has been implicated, as well as compressed air, in aetiology of subcutaneous emphysema. It has been used as a canal irrigant and disinfectant during routine root canal therapy. When hydrogen peroxide comes into contact with blood or tissue proteins, it very rapidly undergoes effervescence and liberates oxygen. This gaseous expansion may drive debris or simply gas through the apical foramen or into the adjacent bone if an inadvertent perforation of the canal wall were present. Kaufman et al. presented a case of delayed onset of emphysema subsequent to hydrogen peroxide irrigation. Shovelton also presented 4 cases where hydrogen peroxide in the root canal caused oxygen emphysema. Most cases of pneumo-mediastinum and emphysema after endodontic treatment have been related to the root canal that was irrigated with hydrogen peroxide or dried with compressed air.

### Anatomic Pathways of Emphysema Diffusion

The prime area of air entry into anatomical spaces appears to be the root canal space. But it can sometimes pass through the dento-alveolar membrane. Following its introduction into the soft tissues, air remains in the subcutaneous connective tissue and does not spread to deep anatomic spaces in the majority of the cases. However, emphysema can also involve deeper structures as the tissue planes commonly connect.

The potential avenues of travel for compressed air involve the superficial region, the parotid region, the submandibular and sublingual region, the tonsillar and masticator region and the parapharyngeal region (Tab. 1). So, spread of larger amounts of air into these deeper spaces may sometimes cause serious complications. For example, the bases of the first, second and third molars directly communicate with the sublingual and submandibular spaces. These spaces, in turn, communicate with the parapharyngeal and retropharyngeal spaces, where accumulation of air may lead to airway compromise. The retropharyngeal space (“danger space”) is the main route of communication from the mouth to the mediastinum. Once air enters the mediastinum, it can also reach the pleural cavity, the pericardium, and even the retroperitoneum. Consecutive cases of pneumothorax and pneumopericardium may cause cardiac and/or pulmonary failure. Cases of fatal air embolism and optic nerve damage (by access of air to the orbit) have also been described. Generally, air which is introduced into or along fascial planes may present 3 potential sequelae. Initially, it can remain in the space until it is absorbed. This leads to the “ballooning” (emphysema) of tissue and the occurrence of crepitus along the overlying involved tissues, immediately after air entrapment. Secondarily, it can escape along the path of introduction, such as a patent root canal, and be released into room air, causing no damage. Finally, it can enter a blood vessel in a large enough volume to cause obstruction of coronary flow, resulting in cardiac air embolism, or to cause obstruction in cranial flow, resulting in cerebral ischemia (stroke), either of these 2 latter sequelae may result in death. From Rickle’s post-mortem study, it is obvious that one definite risk of air emphysema during endodontic treatment is introduction of air into the cardiovascular system. In large volume, this can cause heart failure.

Respiratory and gastrointestinal tracts are closed systems surrounded by subcutaneous, prevertebral, visceral, and previsceral spaces. Air arising from a breach in mucosal integrity of the respiratory or gastrointestinal tract can enter visceral space and dissect along facial planes into the subcutaneous space, creating subcutaneous emphysema. More distally, air can be introduced into...
the subcutaneous space at the alveolar level in a non-traumatic fashion by alveolar disruption\textsuperscript{46}. 

Table 1. Potential spread of compressed air

<table>
<thead>
<tr>
<th>Region</th>
<th>Area of potential spread</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superficial</td>
<td>cheek, lower lip, intraorbital region</td>
</tr>
<tr>
<td>Parotid</td>
<td>along parotid duct, within parotid gland</td>
</tr>
<tr>
<td>Submandibular</td>
<td>superficial space, sublingual region, parotid gland, masseter region</td>
</tr>
<tr>
<td>Sublingual</td>
<td>submandibular, masseteric, parapharyngeal spaces and ultimately airway</td>
</tr>
<tr>
<td>Tonsillar</td>
<td>submucosa of soft palate, submandibular, sublingual region</td>
</tr>
<tr>
<td>Masticatory</td>
<td>parapharyngeal spaces, parotid, sublingual, submandibular regions, cavernous sinus via foramen ovale, orbit via infraorbital fissure</td>
</tr>
<tr>
<td>Parapharyngeal</td>
<td>speaking, swallowing, with eventual mediastinitis</td>
</tr>
</tbody>
</table>

(Adapted from Liebgott B. The anatomical basis of dentistry. Toronto: Decker, 1986; 457-463)\textsuperscript{39}

These mechanisms involved in spontaneous mediastinal and subcutaneous emphysema are well described by Macklin and Macklin\textsuperscript{47,48}. They suggested that over-distended alveoli rupture into the pulmonary vascular sheath, resulting in interstitial emphysema\textsuperscript{49}. The basic requirement for rupture of the alveoli is the existence of persistent pressure gradient between the alveolus and the surrounding structures. The pressure within the contiguous alveoli is generally assumed to be equal, and therefore the inter-alveolar walls are expected to remain intact. However, in certain situations, (e.g. decrease in perivascular interstitial pressure or an increased intra-alveolar pressure, or both), a gradient is created\textsuperscript{49}. As illustrated schematically, when a pressure gradient develops, alveoli may rupture at their bases, introducing air into the perivascular adventitia and resulting in interstitial emphysema\textsuperscript{49}. Because mean pressure in the mediastinum is always somewhat lower than that in the peripheral lung parenchyma, air then dissects proximally along the broncho-vascular sheaths to the lung hilum and mediastinal soft tissue and then into the previously described fascial planes\textsuperscript{50}.

Symptoms and Diagnostic Signs

The most prominent clinical feature of the SCE is rapid swelling of the face and sometimes the neck\textsuperscript{8}. However, it is usually restricted to discrete local swelling and tenderness\textsuperscript{51}. The affected area becomes puffy and in almost every case crepitus may be elicited on palpation\textsuperscript{15}. Oedema or lymphadenopathy\textsuperscript{12} may also be observed. Pain is variable and is usually of short duration\textsuperscript{8}. Some patients experience sharp pain as the swelling appears. Other patients complain of fullness due to the enlargement of the area. If the swelling involves the neck area, there is generally some discomfort or difficulty in swallowing, but this very rarely appears to be a respiratory embarrassment\textsuperscript{17} (Tab. 2).

The initial change is soft tissue enlargement from the presence of air in deeper tissues\textsuperscript{52}. Subsequently, the enlargement increases and spreads due to secondary inflammation and oedema. Facial erythema and mild fever may occur. Significant spread into the mediastinum can result in dysphonia, dysphagia or dyspnea\textsuperscript{41} (Tab. 2). Stabbing precordial chest pain, the most common complaint of patients with pneumomediastinum (80-90%), is associated with stretching of interstitial soft tissues caused by dissection of air\textsuperscript{33-35}. Other features suggestive of pneumomediastinum are dyspnea with a brassy voice, chest or back pain, and Hamman sign, which is a crunching and bubbling sound caused by movement or air accompanying cardiac pulsation\textsuperscript{56}. The “mediastinal crunch” described by Hamman is reported in 50% to 80% of patients with pneumomediastinum\textsuperscript{33,55}. The crunching or clicking sound is best heard over the retrosternal area and is occasionally audible without a stethoscope. While it varies with the respiratory cycle and patient position, the timing is synchronous with the heartbeat\textsuperscript{57}.

Table 2. Clinical symptoms and signs

<table>
<thead>
<tr>
<th>immediate</th>
<th>subsequent</th>
</tr>
</thead>
<tbody>
<tr>
<td>rapid swelling</td>
<td>facial erythema</td>
</tr>
<tr>
<td>crepitus</td>
<td>mild fever</td>
</tr>
<tr>
<td>pain (variable)</td>
<td>dyspnea</td>
</tr>
<tr>
<td>local discomfort</td>
<td>oedema</td>
</tr>
<tr>
<td>radiographic findings</td>
<td>difficulty in swallowing</td>
</tr>
</tbody>
</table>

While the SCE should be detectable by physical examination, an alert radiologist often makes the diagnosis\textsuperscript{57}. Radiographic examination consists of a panoramic and an anterior-posterior radiograph\textsuperscript{52}. The standard postero-anterior chest radiograph usually demonstrates a radiolucent line between the left heart border and the mediastinal pleura. Other findings include “highlighting” of the aortic knob and “the contiguous diaphragm” sign\textsuperscript{15}. Postero-anterior chest radiographs can overlook 50% of cases of pneumomediastinum. Considering this fact, lateral chest radiographs, which increase sensitivity to almost 100%, should always be performed. These radiographs can also detect associated
pneumothoraces. Lateral decubitus radiographs can sometimes be useful for distinguishing a pneumothorax from a pneumomediastinum. The SCE can also be seen in CT scans, with the air pockets appearing as dark areas. CT scanning is so sensitive that it commonly makes it possible to find the exact spot from which air is entering soft tissues.

**Differential Diagnosis**

Diagnosis of severe SCE can be misleading, when it occurs after a dental procedure. Differential diagnosis of SCE should be made from an allergic reaction, gas gangrene, infection, hematoma, and angioneurotic oedema. The allergic reaction is far more severe than SCE, with skin manifestations preceding serious cardio-respiratory manifestations. Gas gangrene is also a far more severe condition than emphysema; severe systemic reactions, a very disagreeable odour and marked pain are often reported. It is always associated with deep wound.

Air emphysema during endodontic treatment may also be misdiagnosed as infection because of similar location and size of swelling. In fact, such accidents may develop into an infection from microbes forced into the spaces created by the air blast. As far as hematoma regards, formation is rapid and often without initial discoloration. Although sponginess may be present, crepitus is absent in hematomas. In angioneurotic oedema, circumscribed areas of oedema, sometimes preceded by a burning sensation, may appear on the skin or mucous membrane. The possibility of necrotizing fascitis, in which bacterial gas production is possible, should also be considered.

Other differential diagnoses of acute swelling of the cervicofacial region include acute contact dermatitis and Melkersson-Rosenthal syndrome. Finally, in those cases where general anesthesia was performed, subcutaneous emphysema might be misdiagnosed as a delayed hypersensitivity reaction to general anaesthetic.

**Preventive Measures**

During endodontic treatment, the SCE can be prevented by:
- Using a well-fitted rubber dam;
- Using remote exhaust handpieces or electric motor-driven handpieces;
- Using high-speed aspiration or paper points to dry fluids from the root canal;
- Using loosely placed irrigation needles into the root canal, avoiding wedging the needle syringe or employing excessive pressure during intra-canal injection;
- Avoiding the use of compressed air once the root canal has been opened;
- Avoiding the use of hydrogen peroxide while irrigating canals;
- During surgical endodontic procedures, the SCE can also be prevented by:
  - Using specific surgical high-speed handpieces, which direct the high pressure exhaust away from the surgical site;
  - Using a slow-speed, electrically-driven, or sealed-head air pressurized handpieces to remove bone, cementum and dentin when necessary;
  - Using ultrasonic or sonic instruments for root-end cavity preparations.

Finally, early recognition may be of extreme importance to prevent possible secondary infections and cardiopulmonary complications.

**Management of Subcutaneous Emphysema**

Although the occurrence of the SCE is alarming, the condition is generally not dangerous. There is no specific therapeutic protocol recommended for its treatment. In mild to moderate cases, the treatment consists of observation and reassurance of the patient. In the vast majority of cases, emphysema improves within 2 to 3 days, although residual swelling may be evident for up to 14 days. Cold compresses should be used to minimize swelling and improve circulation to the affected area. If solutions such as H₂O₂ or NaOCl are implicated, it is recommended to irrigate the area gently with water (distilled if available) through the portal of entry. If patient complains for pain, local anaesthetics may be prescribed for discomfort in the appropriate area(s). Broad-spectrum antibiotic coverage is advised in all dental-related cases, since the introduction of air may include microorganisms. If the amount of air is large, the emphysema can interfere with breathing and be uncomfortable. If difficulty in breathing or swallowing occurs, and does not seem to be due to anxiety, prompt medical investigation should be considered.

In severe cases, immediate medical attention is mandatory. Tracheostomy may become necessary in case of retropharyngeal-space emphysema with consecutive airway compromise. It has also been reported that administration of 100% oxygen via a nonrebreather mask can hasten resolution of the emphysema, because oxygen, which replaces the air, is more readily absorbed.
Finally, in cases where the emphysema extends towards the neck or the mediastinum, hospitalization of the patient is necessary for a more complete control and continuous follow up13.

Conclusion

Endodontic treatment is not frequently associated with the presence of the SCE. But it is very important to know how to recognize this situation when occurs, in order to treat the patient appropriately. The appropriate therapy is determined by its aetiology11. Thorough knowledge of the diagnostic clues is important for early recognition and initiation of treatment, which are essential to prevent possible complications12.

References


Correspondence and request for offprints to:
Sotiria Liatiri
Grabias 26
54645, Thessaloniki
Greece
E-mail: ria.dent@gmail.com