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Review article

Implications, prevention and management of subcutaneous emphysema during endodontic treatment

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Abstract – Subcutaneous emphysema (SCE) is a possible complication of both nonsurgical and surgical endodontic treatment. A review of the literature pertinent to endodontic intervention and SCE is highlighted, while the causes of and recommendations for the prevention of SCE are provided. A review of the pathways whereby compressed air may travel through potential spaces in the head and neck is also illustrated in an attempt to identify the possibility of morbidity and even mortality should operator induced SCE occur in a patient.

Subcutaneous emphysema (SCE) is defined as the abnormal presence of air under pressure, along or between fascial planes (1). Entrapment of compressed air usually occurs rapidly, often causing morbidity and even death. The most common dental etiology of SCE is the introduction of air via the highspeed handpiece during restorative procedures or during the surgical resection of impacted teeth (2). Specific endodontic treatment procedures, however, have also been implicated as etiologic factors in the occurrence of SCE. The purpose of this paper is 1) to provide a contemporary review of the literature pertaining to entrapment of air during endodontic treatment which resulted in SCE; 2) to detail the fascial planes which are apt to be involved in SCE during endodontic procedures; and 3) to provide clinical guidelines, based on sound scientific data, to prevent SCE during endodontic procedures and manage this complication should it occur.

Literature

Tissue emphysema subsequent to extraction was reported to have occurred long before 1900 when Turnbull indicated in 1870 that he extracted the

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tooth of a "bugler" who suffered emphysema while playing his bugle shortly after the extraction. The swelling resolved within days of the extraction when he ceased playing the bugle (2). Since that report, additional cases pertaining to this occurrence with endodontic implications have been identified in the literature (2–11). In all cases, the spread of tissue emphysema was rapid and extensive. The extension of edema often crossed the midline and extended both superiorly and inferiorly from the site of operation. In many of the cases the root canal had been irrigated with a syringe containing hydrogen peroxide.

SCE, in conjunction with endodontic treatment may last several days to a few weeks, usually clearing in facial regions before neck regions (6, 9, 11, 12). In cases where the neck is involved, respiratory difficulty may develop (13). Should this occur, hospitalization usually follows with preparations made for tracheotomy and the potential for mediastinitis (8,13). In most reported cases, antibiotics were prescribed and recovery was usually complete within six weeks. Death was reported in one case, secondary to the use of compressed air in a mandibular anterior tooth (8).

A considerable range of morbidity to mortality due

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to endodontically caused subcutaneous emphysema is evident, even if rarely reported. The accessible literature provides approximately 30 cases which specifically deal with endodontically induced SCE. Clinical features of SCE can be classified as to those occurring immediately or those occurring over various time periods following treatment. Often seen is localized swelling, discomfort, and crepitus, with soft tissue radiographs or CT scans displaying tissue distension. Later sequelae are widespread edema, erythema, pyrexia, and sometimes pain. In serious cases the size of the swelling may increase over one to two hours. The occurrence of trismus is site dependent and not usually a serious problem. Advanced cases may demonstrate stripping of muscle attachments, poor healing of involved soft tissues and chronic pain. The actual occurrence of death has been reported experimentally in dogs (14), in addition to the case mentioned above (8).

Review of the anatomy of the fascial planes

The fascial planes are areas of tissue boundaries, which under nonpathological conditions, are only potential spaces. Liebgott (15) defines seven regions of the head and neck in addition to the mediastinum where fascial planes occur (Table 1). As well as pressurized air, hemorrhage and infection are also possible causes of encroachment of the potential spaces. The prudent clinician will be aware of these potential complications during treatment and avoid causative factors which may effect subcutaneous emphysema during nonsurgical or surgical endodontic procedures.

During nonsurgical endodontic procedures, after coronal access and canal patency have been obtained, visibility is often limited. The temptation to clear the working site using an air syringe with compressed air is great. This action may, however, introduce high pressure air into the periradicular tissues, and in some

Table	1.	Anatomy	of	Fascial	planes –	potential	spread	of	compressed	air
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Region	Area of Potential Spread of Compressed Air
Superficial Parotid Submandibular	Cheek, lower lip, infraorbital region Along parotid duct, within parotid gland Superficial space, sublingual region, parotid gland, masseter region
Sublingual	Submandibular, masseteric, parapharyngeal spaces & ulti- mately airway
Tonsillar Masticator	Submucosa of soft palate, submandibular, sublingual region Parapharyngeal spaces, parotid, sublingual, submandibular regions, cavernous sinus via foramen ovale, orbit via infra- orbital fissure
Parapharyngeal	Carotid sheath & contents; difficulty speaking, swallowing, with evential mediastinitis

(Adapted from Liebgott B. *The anatomical basis of dentistry*. Toronto: Decker, 1986; 457–63)

cases along fascial planes (4–7). Likewise, the forced removal of debris with oxygenated irrigants, such as hydrogen peroxide which may pass beyond the apical foramen, can also create the abnormal presence of air trapped in the tissues. Subsequent air entrapment can cause minor tissue swelling, which is usually self-limiting (7). However, it can also be absorbed into the circulatory system and may cause embolism formation in various parts of the body, including the coronary and cerebral circulation. The consequences of this may be tragic (8).

Mechanism for SCE morbidity

The potential avenues of travel for compressed air are shown in Figs. 1 & 2. Air which is introduced into or along fascial planes presents with three potential sequelae. Initially, it can remain in the space until it is resorbed. This leads to the "ballooning" (emphysema) of tissue and the occurrence of crepitus along the overlying involved tissues, immediately after air entrapment (1). Secondarily, it can escape along the path of introduction, such as a patent root canal, and be released into room air, causing no damage (2). 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)



Fig. 1. Sagittal view indicating possible routes of movement of compressed air in the anterior region; \mathbf{n} =nasal cavity; \mathbf{p} =palate; \mathbf{s} = superficial fascial space; \mathbf{sl} =sublingual space; \mathbf{sme} =submental space; \mathbf{v} =vestibule. (By permission from CV Mosby-Year Book: Liebgott B. *The anatomical basis of dentistry*. Toronto: Decker, 1986; 460)

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Fig. 2. Coronal section through the molar region to demonstrate possible routes of movement of compressed air in the posterior region; ms=maxillary sinus; p=palate; s=superficial fascial space; sl=sublingual space; smd=submandibular space; v=vestibule. (By permission from CV Mosby-Year Book: Liebgott B. *The anatomical basis of dentistry*. Toronto: Decker, 1986; 461)

(15). Either of these two latter sequelae may result in death.

Hydrogen peroxide has been implicated as well as compressed air in the etiology of SCE (2,5,6,9,10). It has been used as a canal irrigant and disinfectant during routine root canal therapy (16). However, since oxygen is liberated from hydrogen peroxide on contact with blood and tissue proteins (17,18), this gaseous expansion may drive debris or simply gas through the apical foramen (17) or into the adjacent bone if an inadvertent perforation of the canal wall were present.

Bhat (10), reported a case in which hydrogen peroxide was injected into loose infraorbital tissue as a result of faulty access and subsequent lateral perforation of a maxillary central incisor. In this case the swelling was non-infective and resolved in about a week. Kaufman (9) reported on a maxillary first premolar which was instrumented beyond the apical constriction to a #40 K-file and then irrigated with 3% hydrogen peroxide. The onset of emphysema was sudden and painful, extending from the lower eye lid to the lower lip, and laterally to the nose. In both these cases the apical foramen was compromised, an incorrect length was determined, and binding of the irrigation needle occurred, such that hydrogen peroxide was forcefully extruded beyond the apex. A similar report, highlighting the binding of the irrigation needle followed by a forceful injection of hydrogen peroxide, was presented by Walker (5).

Kaufman et al. (12) presented a case of delayed onset of emphysema subsequent to hydrogen peroxide irrigation. The patient had undergone root canal instrumentation and within two hours returned in excruciating pain. The incision of a large intraoral vestibular swelling with crepitus, resulted in the release of a bloody, foaming liquid. There was an immediate cessation of pain.

Hirschmann & Walker (6) cited a case of endodontic treatment on a grossly carious maxillary right canine, where compressed air was used often during the caries removal procedure. The patient experienced an immediate swelling on the right side of the face which rapidly spread to the left side. The patient was given penicillin and three days later the swelling had disappeared. In a second case report, the same authors reported the loss of a fractured reamer out the apex subsequent to forceful hydrogen peroxide irrigation which had been used in an attempt to dislodge the broken instrument. This patient experienced tissue emphysema which subsided over five days. Medication included a course of oral ampicillin. Subsequent extraction of the tooth showed the reamer had perforated the buccal plate.

A similar problem was reported by Falamo in 1984 (7), when compressed air was used to dry a root canal of a maxillary right central incisor. Within minutes, the left eyelid, cheek and upper lip were swollen. Antibiotics were prescribed and resolution occurred in six days. Interestingly, the right side was not involved, pointing out the possible movement of air along fascial planes, crossing the midline.

Wright and others (19) reported facial emphysema in a two year old during general anesthesia, while undergoing a pulpectomy of a primary central incisor. Due to continual seepage from the canal, air from the triplex syringe was used and ballooning of the lip was immediately noticed. The tooth was subsequently ex-

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tracted due to the inability 'to stop seepage'. Although the child was discharged the same day with antibiotics coverage, the patient was readmitted three days later with facial swelling. Intravenous cephlorexin was administered and again the patient was discharged. After 28 days, the patient was readmitted with fever, swelling on the affected side with persistent nasal discharge. The diagnosis of abscess of the infraorbital area anterior to the right maxilla was made. Under general anesthesia, examination showed chronic ulceration and necrotic mucosa of the nasal vestibule, consistent with chemical burn. Culture and sensitivity showed *Staphloccocus aureus*. It should be noted that irrigation of the anterior tooth had been performed with sodium hypochlorite and formocresol, prior to the use of compressed air, in an attempt to control the bleeding from the canal. The child eventually healed without adverse sequelae.

A severe sequelae, pneumomediastinum, was reported by Lloyd (11) and Nahieli and Neder (8) subsequent to the use of compressed air and hydrogen peroxide, respectively. In the latter case, the patient had undergone pulp extirpation of the mandibular right third molar, using 3% hydrogen peroxide as irrigant. Subcutaneous emphysema was noted within minutes and the patient thereafter complained of shortness of breath and pressure in the chest. Chest radiography showed air in the mediastinum. Treatment consisted of ampicillin for ten days and the emphysema subsided in two weeks.

Most recently, Pynn et al. (20) reported a sudden swelling subsequent to the perforation of a maxillary left lateral incisor during endodontic treatment. The swelling extended from the left orbit to the submandibular and subclavicular areas and crossed the midline in the submental area. Again, marked crepitus was noted. Complete resolution occurred subsequent to antibiotic treatment and the passage of six weeks time.

A review of the literature has failed to disclose any reported cases of SCE during surgical endodontic procedures. However, there are reports in the oral surgical literature regarding SCE in which the high speed handpiece was used to section teeth prior to extraction (21, 22), following trauma (23), or subsequent to temporomandibular joint surgery (24). It follows that caution should be exercised when exposing tissue during surgical procedures in order to avoid the introduction of compressed air along fascial planes.

Alternatives to the standard air driven highspeed handpiece exist for endodontic surgical entry. Bellizi & Loushine (25) recommend the high-torque surgical drill that relies on an electric motor driven system instead of compressed air. This apparatus avoids the exhaustion of air into the surgical field, thereby negating the possibility for SCE. The use of "high-speed" reduction sealed-head hand pieces pressurized by air or nitrogen have also been recommended to prevent SCE, as long as they are used with copious water lavage (26).

Another alternative to minimize or negate SCE during surgical procedures would be to use ultrasonic preparation as an alternative to handpiece preparation of teeth during root-end preparation. This approach has been recommended as a viable alternative to preparation with a compressed air hand piece. Problems of visualizing the surgical site in the absence of compressed air from the hand held syringe can be eliminated with the judicious use of vasoconstrictors prior to the procedure and copious rinsing with saline during the surgical entry (27,28).

Discussion

Clinical guidelines for prevention, identification and management

During endodontic treatment the actions which may contribute to the occurrence of SCE vary considerably. The prime area of air entry into anatomical spaces appears to be the root canal space. However air movement through soft tissue lacerations, such as from the rubber dam clamp, or during surgical procedures cannot be overlooked, although the latter has not been reported.

The most prominent clinical feature of SCE is rapid swelling of the face and sometimes the neck. The affected area becomes puffy and in almost every case crepitus may be elicited on palpation (2). Pain is variable and is usually of short duration. Sometimes only a slight discomfort or sensation of fullness is felt. If the neck is involved there is generally some discomfort with difficulty in swallowing.

Differential diagnosis of SCE should be made from an allergic reaction, hematoma, and angioneurotic edema. The former is far more severe than SCE, with the skin manifestations preceding serious cardiorespiratory manifestations. Hematoma formation is rapid and often without initial discoloration. Although sponginess may be present, crepitus is absent. In angioneurotic edema, circumscribed areas of edema, sometimes preceded by a burning sensation, may appear on the skin or mucous membrane. The possibility of necrotizing facilitis, in which bacterial gas production is possible, should also be considered (23).

While cases reported of endodontically-induced subcutaneous emphysema resolved, the morbidity was remarkable. In an experimental canine model, Rickles & Joshi (13) reported the death of 4 of 7 dogs subsequent to the administration of compressed air into patent root canals. Autopsy revealed air in the right ventricle, large thoracic vessels and coronary vessels. One case of human mortality with endodonticallyinduced SCE has been noted (8). Table 2. Prevention of subcutaneous emphysema during endodontic procedures (1-3, 5-7, 9, 10, 25)

1. Always use a rubber dam

- 2. Loosely place irrigation needles into the root canal
- 3. Deliver contents of the irritating syringe gently
- 4. Avoid the use of hydrogen peroxide while irrigating root with open apices
- 5. Avoid the use of hydrogen peroxide in highly hemorrhagic pulp canals

6. Use high-speed aspiration or paper points to dry fluids from the root canal

7. Avoid directing compressed air into the endodontic access opening during treatment

8. Consider using "vented" high speed handpieces or motorized surgical handpieces during surgical osseous entry and root-end resection

9. Use sonic or ultrasonic devices for surgical root-end preparations

Table 3. Management of subcuteaneous emphysema (1, 6, 7, 19, 23, 27, 29)

1. Discontinue root canal treatment

- 2. Reassure the patient
- 3. Attempt to ascertain the cause of the accident; eg., perforation of the apex or root wall, induction of compressed air into the tissues from the highspeed handpiece during surgery, introduction of H_2O_2 and so forth.
- 4. If solutions such as H_2O_2 or NaOCI are implicated, gently irrigate the area with water (distilled if available) through the portal of entry
- 5. If the patient reports pain, administer local anesthetics in the appropriate area(s)
- 6. If the swelling appears unrelated to SCE, consider an allergic reaction or angioedema and treat accordingly
- 7. Consider prescribing antibiotics, such as penicillin for 5 days, since the introduction of air may include microorganisms
- 8. Consider prescribing analgesics since pain due to distention will occur and that may take several days to subside
- If difficulty breathing or swallowing occurs and does not seem to be due to anxiety (ie., not quickly resolved), consider prompt medical investigation

One of the significant findings when comparing the reports of SCE in the literature is the unpredictability of the morbidity magnitude. Most practitioners at one time or another may find they have used small amounts of compressed air at the orifice of a tooth with a patent canal without inducing SCE. Therefore, it would appear that the occurrence of SCE is due to the combination of several factors; 1) procedural accidents causing perforation of the apex or root of a tooth allowing passage of air to the potential spaces; 2) inadvertent irrigation of subcutaneous tissues with oxygen producing irrigants $(H_2 0_2)$ under pressure; 3) use of highspeed handpieces without exhaust protection to prevent compressed air from being delivered to the surgical site; and 4) prolonged or excessive use of hand-held air syringes for clearing surgical sites for improved visibility.

The most effective treatment, however, is the prevention of SCE during nonsurgical root canal treatment by; 1) avoiding the use of compressed air once the root canal has been opened; 2) using paper points to dry canals; and, 3) ensuring, if hydrogen peroxide is used, that it is retained within the canals (Table 2).

During surgical endodontic procedures SCE is prevented by; 1) avoiding the use of the compressed air

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syringe during irrigation; 2) using specific surgical highspeed handpieces, which direct the high pressure exhaust away from the surgical site; or 3) using a slow speed, electrically-driven, or sealed-head air pressurized handpieces to remove bone, cementum and dentin when necessary. Additionally, the use of ultrasonic or sonic instruments for root-end cavity preparation may also decrease the likelihood of inducing SCE (Table 2).

Although the occurrence of SCE is alarming, the condition is generally not dangerous, and the air is absorbed in the course of three or four days without active treatment. Should SCE occur, there are some reported treatment options, however, none have been scientifically tested (Table 3). In most cases antibiotics, such as penicillin, were prescribed, presumably on the assumption that if air has traveled into tissue spaces then microorganisms may follow suit. This complication and the need for antibiotics have been challenged as to its validity in some of the earlier reports (2), while other authors claim an incidence of infection and mediastinitis, and the need for prophylactic antibiotic coverage (8,19,26). Likewise, the reported use of penicillin or erythromycin in these cases may be based on empirical speculation, as the nature of the organisms present and their position in the tissues may warrant the use of antibiotics such as metronidazole or clindamycin.

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