A strange chest pain after dental surgery

Enzo Porteri · Nicola Rizzardi · Damiano Rizzoni · Andrea Salvi · Carolina De Ciuceis · Davide Farina · Gianluca E. M. Boari · Caterina Platto · Silvia Paiardi · Almajdalawi Raed · Enrico Agabiti Rosei

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Case history

In May 2007, a 29-year-old man was admitted to our ward complaining of chest pain exacerbated by deep breathing but not by change of position. The chest pain episodes were short-lasting (some minutes), but the patient complained of several relapses. Simultaneously, soft-consistency bilateral swelling of the neck appeared in the submandibular and lateral cervical regions.

Ten hours before admission, the patient had undergone dental surgery for removal of the III right inferior wisdom tooth. The procedure was particularly difficult and lasted for more than 90 min.

The family history was negative for cardiovascular disease; no relevant previous disease was present. He was not on any medical treatment; however, he smoked 15 cigarettes a day, and his alcohol intake was moderate.

Physical examination of the patient showed normal blood pressure (120/80 mmHg) and heart rate (65 beats/min). He was eupnoeic, and oxygen saturation of the blood was 97% (no oxygen supply). Body temperature was normal. A physical examination showed bilateral swelling of subcutaneous tissues that were more pronounced on the right side of the neck, chest, and in the submandibular, lateral cervical, supraclavicular, and mammary regions, with modest pain and “crepitation” on digital pressure. The cardiac auscultation produced crackling, bubbling, and rubbing related to systole.

The patient’s white cell count was modestly elevated (11,100/mm³), while the renal and liver functions and urinalysis were normal. No elevation of troponin I or T and of creatinine–phosphokinase was detected. The ECG showed the presence of sinus rhythm and high-voltage T-waves in peripheral leads. The chest X-ray examination showed no abnormal findings (Fig. 1). Based on these findings, a preliminary diagnosis of acute pericarditis was made.

Differential diagnosis

The differential diagnosis in a patient with a combination of head and neck swelling, chest pain, and cardiac crackles includes the following: pneumothorax, oesophageal rupture, expanding haematoma, infection in the fascial planes, subcutaneous emphysema with pneumomediastinum, anaphylaxis, angioneurotic oedema, or other allergic reactions.

Further investigations

The patient underwent a neck/thorax CT scan (Figs. 2, 3) that demonstrated severe subcutaneous emphysema extending from the level of parapharingeal and submandibular spaces to the rest of the neck, to the jugular fossa, to the supraclavicular spaces, and to the retrosternal portion of the mediastinum. No pleural or parenchymal lesions were detected. An echocardiographic evaluation showed a normal ejection fraction and cardiac dimensions, and no signs of pulmonary hypertension or of pericarditis.
were observed. A fibrolaryngoscopic investigation showed normal respiratory space and normal pharyngeal expansion. Serological research for Adenovirus, Coxsackie and Echovirus infection, as well as blood cultures for aerobic and anaerobic bacteria were negative.

In addition, the patient was also evaluated by a maxillofacial surgeon, an otorhinolaryngologist, and a thoracic surgeon. It was agreed that a conservative approach would be followed with clinical observations, fasting, antibiotic therapy, and a repeat CT scan after 36 h.

Management and clinical follow-up

The patient was treated with acetylsalicylic acid 500 mg t.i.d. (plus omeprazole 20 mg o.d.) with rapid resolution of the chest pain and with a progressive reabsorption of the air from the subcutaneous tissue and from the mediastinum. Intravenous antibiotic therapy with ampicillin 2 g + sulbactam 1 g t.i.d. and metronidazole 500 mg q.i.d. were administered for 6 days. Cardiac cracklings disappeared on the fourth day of recovery. The clear improvement of clinical condition ruled out any surgical procedures and the need for any further investigations. A second CT scan was performed on the fifth day showing clear reduction of the subcutaneous air in the neck and in the mediastinum. Some bubbles of air were still observed in the parapharyngeal and submandibular spaces, and a few of them in the mediastinum (retrosternal area) and in the right supraclavicular regions.

Final diagnosis

Based on the patient’s improvement following therapy, our final diagnosis was pneumomediastinum and subcutaneous emphysema after dental extraction. Antibiotic therapy given orally (Amoxicillin/clavulanate 1 g tid) was continued for the next 6 days.

Discussion

The first case of subcutaneous emphysema associated with a dental procedure was reported in 1900 by Turnbull [1], who referred to a case in which a bugle player, shortly after undergoing a tooth extraction, developed facial subcutaneous emphysema as a result of the Valsalva’s manoeuvre while playing his instrument [2, 3].

Subcutaneous emphysema is a rare complication of routine dental extraction, and is usually due to the use of high-speed dental drills. These air-driven drills direct a steam of air towards the burr as a coolant, and the air exhaust from the turbine is discharged in the area of the surgical field. Under these conditions, it is highly possible for pressurized air to be driven into the subcutaneous layer through the surgical wound inducing subcutaneous emphysema. This situation may lead to error in treatment, because almost 10% of cases are misdiagnosed as “allergic reaction” in the emergency department [4].

The roots of the first, second, and third molars are directly aligned with the sublingual and submandibular spaces. The sublingual space is also aligned with the pterygomandibular, parapharyngeal, and retropharyngeal spaces. The pressurized air may enter the gums beneath the periostium of the mandible and dissect through the cervical facial planes [5–8]. If a large amount of air is injected, it may dissect into deeper structures to more distant sites, such as mediastinum or pericardium [9] and even cause a pneumothorax. Air passing into the mediastinum is most commonly associated with extraction of the third molar [10].

Other complications of air passing through the potential spaces of neck have shown to cause otalgia, Eustachian tube dysfunction and temporary hearing loss, dysphagya and dysphonia, pneumoperitoneum, pneumoparotid, and air emboli dissemination.

Hamman’s crunch is the typical sign found when air reaches deep spaces in the thorax. This was first described...
by Louis Hamman in 1937 as a crunching, rasping sound, synchronous with heartbeat, heard over the precordium, and sometimes at a distance from the chest in spontaneous mediastinum emphysema. It is caused by the cardiac filling–emptying, cyclic anterior–posterior cardiac motion, or a combination of both channelling pleural air into the lung fissure and then forcing it out creating the noise. Most pleural rubs will increase with inspiration, whereas pericardial rubs are not related to the respiratory cycle. The mechanism of the expiratory increase appears to be related to the change in pleural pressure with the respiratory cycle. If the crunch is caused by intrapleural air or mediastinal air, expiration will cause an increase in pressure in the mediastinal and the intrapleural spaces, which will force air to move around within the spaces. If the air is next to the pericardium, it will also move synchronous with cardiac filling or contraction. Summation of the two forces will cause a sound that varies with heartbeat and increases with expiration [11].

The antibiotic therapy is based on broader spectrum antibiotics, because of the frequent finding of beta-lactamase producing bacteria, especially the oral anaerobes (e.g., pigmented Prevotella spp. and Fusobacterium spp.). Ampicillin–sulbactam (2 g i.v. every 4 h) provides extended coverage against oral anaerobes, including those that produce beta-lactamases, and it is therefore the treatment of choice. Penicillin G (2–4 MU i.v. every 4–6 h) in combination with metronidazole (500 mg i.v. every 6 h), an agent active against anaerobic Gram-negative bacilli, is an alternative regimen. Penicillin-allergic patients should be treated with clindamycin (600 mg i.v. every 6 h) [12].

Oxygen supplementation has been shown to hasten the resolution of the emphysema; it replaces nitrogen in the potential spaces, and is more readily reabsorbed into the surrounding tissues [13].
Complications of cervical emphysema and pneumomediastinum after oral surgery may include cardiac tamponade, airway obstruction, simple and tension pneumothorax and with pneumoperitoneum and secondary infective mediastinitis (in particular Pseudomonas Aeruginosa, Legionella Pneumophilia, Anaerobes, and Acanthamoeba) [14, 15]. Ludwig’s angina is a particular serious infectious complication that arises from bacterial spread through these fascial planes [16].

In conclusion, tooth extraction and dental surgery are routine procedures that are frequently performed. Recently, improvement of surgical techniques has shown a reduction of many incidences of complications from dental surgery. However, despite the apparent slight invasiveness of the procedure, serious clinical complications may still arise. The time course of events, and namely, the time relation with surgical dental procedures may aid in establishing a correct and timely diagnosis [17].

References