

Massive subcutaneous emphysema after domestic fall

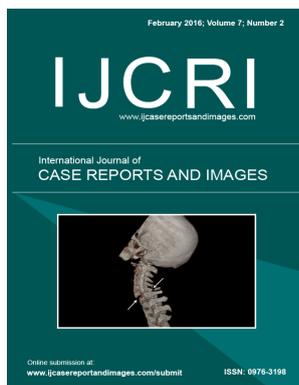
Ida Carine Bø, Erik Waage Nielsen

ABSTRACT

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CASE REPORT

A 72-year-old male was admitted to the hospital with shortness of breath and massive swelling of the face after a domestic fall the previous day. Upon admission the patient complained of nasal voice, difficulties with opening his eyes due to swelling and pain in the left hemithorax.

The patient recounted the previous day having stumbled on his right foot and hit the coffee table, with his left hemithorax taking most of the impact. In hospital, the patient was received by the trauma team. The examining surgeon noted nasal voice, and upon palpation midline trachea and massive subcutaneous emphysema of the face, neck, thorax and upper extremities (Figure 1). Auscultation of the lungs revealed bilateral breath sounds, described as weakened. Blood pressure was 150/95, saturation was 90% in room air and 94% with 3 L oxygen on a nasal cannula. Respiration rate 24 per min. There was a bruise on the left thorax, but no skin penetrating wound. Chest X-ray showed a small, left sided pneumothorax, but this was difficult to assess due to massive subcutaneous emphysema (Figure 2).

Arterial blood gas on admission showed a slight respiratory acidosis and hypoxemia with pH 7.33 (7.37–



Figure 1: Photo taken 21 hours after admission (20 hours after bilateral thorax drainage).



Figure 2: Chest X-ray taken in the emergency room upon admission. Massive subcutaneous emphysema.

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7.45), pCO₂ 6.1 (4.7–6.0) kPa, pO₂ 8.4 (> 9.3) kPa and BE -2 (-2 - +3) mmol/L.

Computed tomography (CT) scan of the abdomen revealed subcutaneous emphysema involving erector spinae and deltoid muscles and bilateral upper extremities, in addition to thorax, neck and face. There was also pneumomediastinum and a relatively small pneumothorax bilaterally, but no lung collapse or dislocation of the trachea. The patient had a left sided flail chest with multiple fractures of costa 6–9. There was no visible injury to the trachea or bronchus (Figure 3 A–B). As the CT scan showed bilateral pneumothorax and the origin of the subcutaneous emphysema was uncertain the patient was immediately taken to the operating room and bilateral thorax drainage was inserted. The procedure was done in local anesthesia and slight sedation. Postoperatively, his respiration and circulation was normal.

The patient refused thoracic epidural as pain relief. Subsequently, he required large doses of opioids, in addition to NSAIDs and paracetamol. Over the following days the subcutaneous emphysema improved. Bilateral chest drainage was continued. However, the patient needed increasing doses of opioids, which resulted in inadequate respiration. Four days after admission arterial blood gas showed increasing respiratory acidosis and hypoxemia with pH 7.20 (7.37–7.45), pCO₂ 10.9 kPa (4.7–6.0), pO₂ 8.7 kPa (>9.3) and BE 4 mmol/L (-2-+3). The trachea was intubated and the patient ventilated with pressure control ventilation. Inspiratory pressure was set at 18 cm H₂O, PEEP 8 cm H₂O and FiO₂ 0.35. Sedation with fentanyl (0.10–0.40 mg/h) and midazolam (2.5–6 mg/h) was initiated. We expected a prolonged need for respiratory support in combination with high opioid requirements, and a percutaneous tracheostomy was performed uneventfully on day-5 after admission. This also allowed for sedation to be tapered, but he still required large doses of fentanyl intravenous. Nerve blocks of the intercostal nerves T6–9 with bupivacaine were successfully performed on day-7. This was repeated on day-9 and day-10. After the intercostal block, opioids were gradually tapered and the patient recovered rapidly. The chest drains were removed on day-7. On day-8 he

was decanulated. He was severely reduced and therefore kept for another three days in ICU. Eleven days after admission he was transferred to a surgical ward for continued rehabilitation and physiotherapy. When transferred to the surgical ward his pain was treated with a fentanyl patch of 25 µg/h, oral diclofenac 75 mg x 3 and paracetamol 1 g x 4. Sixteen days after admission the patient was discharged home from the hospital.

DISCUSSION

After blunt trauma our patient displayed the triad of subcutaneous emphysema, pneumomediastinum and pneumothorax. These air collections may come from various origins. As described by Wintermark et al. [1] pneumomediastinum may arise from tracheobronchial or esophageal ruptures, which create a direct air leak into the mediastinum. The CT scan of our patient showed no signs of tracheo-esophageal injury. Another mechanism for pneumomediastinum may be spread of subcutaneous emphysema e.g., caused by rib fractures. This may be in combination with pneumothorax and a spread along fascial sheaths to the mediastinum [1]. The patient had fractures in ribs 6–9, but there was no visible skin perforation. Conversely, a primary pneumomediastinum may also spread to create subcutaneous emphysema [1].

The most common cause of pneumomediastinum is alveolar rupture [2]. Elevated alveolar pressure caused by e.g. airway obstruction, coughing or blunt trauma leads to rupture of the alveoli and air leakage. This could be the most likely explanation for the condition of our patient.

Pneumomediastinum, pneumothorax and subcutaneous emphysema are most commonly seen after severe trauma. In our patient, the mechanism of injury was modest, but caused extensive clinical findings. Probably, the delay from injury to hospitalization contributed. The exact mechanism for development of air collections within the thoracic cavity remains uncertain. As the patient responded well to treatment and was rapidly discharged from ICU further diagnostic examination was considered unnecessary.

CONCLUSION

Even modest blunt trauma such as domestic falls may occasionally lead to considerable subcutaneous emphysema. In our patient the subcutaneous emphysema was most likely from a pneumomediastinum. Air in the mediastinum is most often provided by alveolar rupture, and this could be the cause in our patient as well.

Keywords: Pneumomediastinum, Pneumothorax, Subcutaneous emphysema.

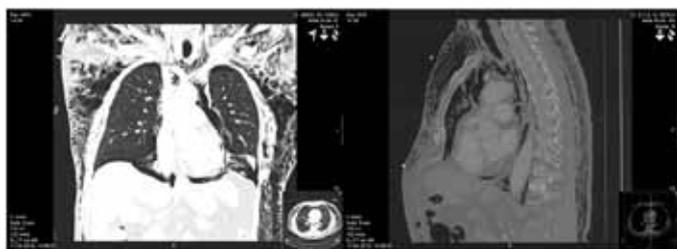


Figure 3: Chest computed tomography scan with intravenous contrast. (A) Marked bilateral apical pneumothorax and basal pneumothorax on the left side, in addition to pneumomediastinum spreading along arcus aortae, (B) Subcutaneous emphysema involving dorsal muscles.

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Author Contributions

Ida Carine Bø – Substantial contributions to conception and design, Drafting the article, Final approval of the version to be published

Erik Waage Nielsen – Substantial contributions to conception and design, Revising it critically for important intellectual content, Final approval of the version to be published

Guarantor

The corresponding author is the guarantor of submission.

Conflict of Interest

Authors declare no conflict of interest.

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