

## Case Report

# Vacuum-assisted closure therapy as adjunct to treatment of grotesque subcutaneous emphysema after blunt chest trauma: A case report

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## ABSTRACT

Subcutaneous emphysema (SE) is a potentially life-threatening complication derived from a pneumothorax. Treatment can be challenging and on an emergency basis. A 79-year-old patient was admitted with blunt chest trauma after a motor vehicle accident. Computed tomography showed incarceration of lung parenchyma in a fractured rib without pneumothorax. The patient was initially stable, but later on developed several episodes of acute dyspnea with bilateral pneumothoraces and life-threatening SE. Further assessment using chest X-ray was complicated by SE. Treatment consisted of chest tube insertion and additional vacuum-assisted closure (VAC) therapy of the SE using a pectoral incision. Symptoms resolved quickly, and chest tube and VAC-therapy could be discontinued by day 7 and 3, respectively. Immediate chest tube insertion is the first-line treatment for trauma patients with massive SE, even if a pneumothorax may not reliably be diagnosed initially. Supportive VAC-therapy must be considered to accelerate the decline of massive SE.

**Key words:** Chest trauma, Pneumomediastinum, Pneumothorax, Subcutaneous emphysema, Vacuum-assisted closure - therapy

Massive subcutaneous emphysema (SE) originating from the thorax and expanding to head and limbs following blunt chest trauma is a rare condition. Symptoms include crepitus, change in body contour, or impairment of sight due to swollen eyelids. Life-threatening compression of the trachea or the thorax along with critical impairment of respiration has been reported in the literature [1-4]. Normally, SE is associated with intrathoracic or mediastinal pathology, such as pneumothorax or lesions to the bronchi, trachea, or larynx [5,6]. Recalcitrant pneumothorax after pulmonary resection is the most common cause [7]. Most recommendations for the management of SE originate from thoracic surgeons. Chest-tube insertion is the first-line therapy [8]. However, other treatment options such as skin incisions, subcutaneous insertion of angiocatheters, liposuction devices, drains, and subcutaneous vacuum-assisted closure (VAC) therapy have been discussed mainly in case reports [2,5,7-10]. We present a case of SE following blunt chest trauma with a focus on the possible pitfalls in the diagnostic process and our experience with actual management recommendations from a trauma surgeon's perspective.

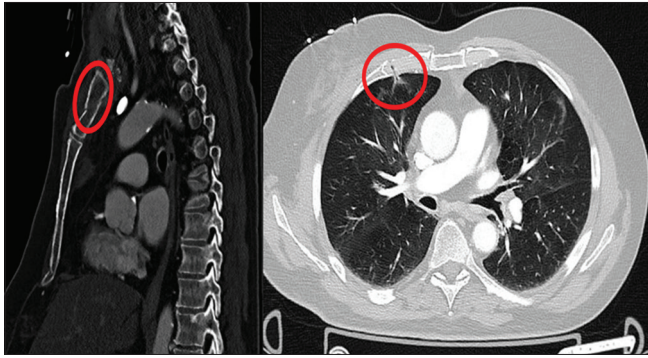
## CASE REPORT

A 79-year-old male sustained a blunt chest trauma when his car was involved in a frontal collision on a motorway. On admission, the patient was hemodynamically stable (blood pressure 148/68 mmHg and heart rate 86 bpm), alert and fully oriented.

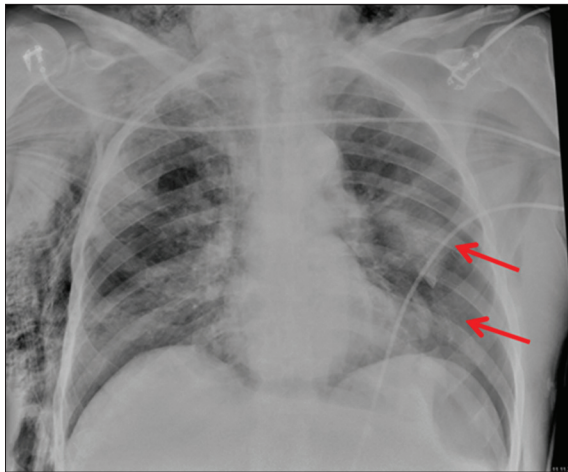
His complaints were soreness in the neck and right ankle. The primary assessment according to the acute trauma and life support algorithm revealed an open airway and a protected cervical spine. Hematoma and crepitus over the sternum were noted along with a local tenderness of the sternum and the right parasternal region. Instability of the thoracic wall or respiratory distress was absent. Breath sounds were normal and symmetrical (respiratory rate 15/min and SaO<sub>2</sub> 96% with 8 L O<sub>2</sub> intranasal).

The computed tomography (CT) trauma scan showed a minimally dislocated fracture of the manubrium sterni with accompanying retrosternal hematoma, serial right-sided rib fractures at the costochondral junction (ribs 2–8) with the incarceration of lung parenchyma in the fracture zone of the third rib (Fig. 1). Hemopneumothorax was excluded. The patient was admitted to the intermediate care (IMC) unit for further monitoring after a soft tissue injury on the right ankle was cleaned and the wound dressed. 6 hours after the injury, a sudden onset of dyspnea with an SE on the right side of the chest was observed. Chest X-ray revealed a left-sided pneumothorax and a vast SE on the contralateral side (Fig. 2). Insertion of a chest-tube into the left pleural space resulted in a quick improvement of the patient.

18 hours later, followed a second-look of the right ankle under general anesthesia to exclude a penetrating wound to the ankle joint. Surgical exploration showed a superficial wound and delayed closure was performed. After uneventful surgery and extubation, the patient developed another episode of agitation with dyspnea.



**Figure 1:** Computed tomography performed on the patient's admission shows fracture to manubrium sterni (red circle and left) and incarceration of lung parenchyma at the fractured costochondral junction of the third rib (red circle and right)



**Figure 2:** Chest X-ray performed after the first episode of dyspnea shows right-sided emphysema and left-sided pneumothorax (arrows)

Chest X-ray was repeated immediately and revealed progressive SE, now bilateral. Since right-sided pneumothorax could not be excluded as being the potential origin of the progressive SE, another chest tube was inserted into the right-sided pleural space. After the intervention, the patient recovered quickly. Observation for another 12 hours in the IMC unit was uneventful and the patient transferred to the trauma ward. Chest tubes were removed 1 (left) and 2 days later (right), with air leaks absent and SE reabsorbing.

After 1 day, the patient again complained of acute dyspnea with rapidly progressing SE including the whole torso and face. The massive swelling of the eyelids caused an inability to open the eyes leaving the patient temporarily blind. An urgent chest X-ray was inconclusive regarding pneumothorax due to the massive superimposition of the SE which made proper assessment impossible (Fig. 3). Therefore, bilateral chest tubes were inserted again. CT revealed no new bronchial or parenchymal lesions leaving the known lung tissue incarceration at the fracture site the only obvious source for the recurrent dyspnea and SE. Additional bronchoscopy also showed pathologies. To support reabsorption of the SE, a 5cm incision was applied over a large subcutaneous bulla in the right pectoral region under local anesthesia (Fig. 4). A VAC device was installed, and VAC therapy with the continuous negative pressure of 75 mmHg was initiated as described previously [1,11,12]. SE

rapidly improved (Fig. 5). VAC therapy was discontinued after 3 days. 2 and 3 days after that, the chest tubes were removed as well. The patient was finally discharged from the hospital 18 days after trauma with full recovery of SE. The later course was uneventful.

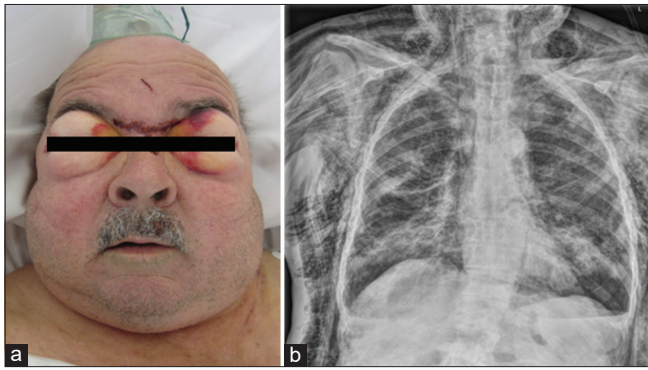
## DISCUSSION

Thoracic SE starts with a lesion of the pulmonary parenchyma. In atraumatic situations with an intact chest wall, for example, spontaneous pneumothorax, there is leakage of air into the mediastinal space, and a pneumomediastinum may develop. This is observed in 70–90% of SE [13]. Further leakage will result in air being trapped and distributed along the fascial layers of the neck, face, torso, and finally the limbs. A life-threatening situation occurs when pneumothorax evolves into tension-pneumothorax. However, massive SE itself may be life-threatening when it causes compression of the cervical viscera or thoracic outlet obstruction [4,6,13]. This has been observed in cases of SE following tracheal injuries caused by a dog bite or kite string injuries [6,14]. In trauma or after thoracotomy with lesions of the parietal pleura, SE may occur directly without preceding pneumomediastinum. In our case, lung tissue was incarcerated between displaced rib fragments resulting in a delayed pneumothorax and SE.

Theoretically, SE could have developed without initial concomitant pneumothorax if the incarcerated lung tissue sealed the defect of the chest wall like a plug leaving only a direct connection between lung parenchyma and chest wall with the release of only small amounts of air into the soft tissues of the chest wall. One could speculate that the seal broke in the later course spontaneously or triggered by the mechanical ventilation leading to symptomatic pneumothorax and progressive SE. The development of SE from an initial parenchymal lesion to its clinical presentation can range from several hours to a few days [4,6]. Especially in the context of blunt chest trauma without an apparent wound or hematoma, SE can remain undiagnosed or misdiagnosed (e.g., as an anaphylactic shock) [8,11,12]. The occurrence of SE in a patient with chest trauma needs always a high suspicion of a concomitant pneumothorax. Therefore, establishing a safe airway and immediate chest tube insertion are crucial for the successful management of unstable patients with SE.

Establishing the diagnosis of pneumothorax in a patient with SE only by a clinical examination and chest X-rays can be challenging as demonstrated in our case. SE makes auscultation difficult, and superimposition of subcutaneous air may jeopardize interpretation of chest X-rays. CT is recommended in these cases for further evaluation if permitted by the condition of the patient. Mechanical ventilation with positive end-expiratory pressure (PEEP) in the presence of an air leak despite the insertion of a chest tube may also be a reason for progression of SE. We want to emphasize that in this situation continued PEEP ventilation without sufficient drainage of SE will finally lead to fatal compression of the airway or thoracic outlet obstruction.

Depending on the symptoms and dimension of SE, the physiological reabsorption of SE may be accelerated and supported by additional procedures. Different options have been



**Figure 3:** (a) Clinical picture of the massive subcutaneous emphysema (SE) impairing sight (left). (b) Chest X-ray inconclusive for pneumothorax due to the superimposition of the SE (right)



**Figure 4:** Computed tomography showing a large subcutaneous bulla pectoral on the right side, next to the fractured third rib. Insertion site of the sponge for the vacuum-assisted closure-therapy



**Figure 5:** Clinical pictures of the decline of the subcutaneous emphysema under vacuum-assisted closure therapy (photos taken from day 1 to 3 and on day 7, starting left)

described. Increasing negative pressure on an existing chest tube or a second chest tube may also drain subcutaneous air [8]. Infraclavicular skin incisions (so-called blow-holes), optionally along with repeated massaging of the trapped air, subcutaneous insertion of large bore and specially prepared angiocatheters, liposuction devices, or Jackson-Pratt drains with suction have been described in various case presentations [3,7,9,10,15]. More recently, VAC-therapy applied in an infraclavicular incision has been proposed to deal with post-thoracotomy SE as described in the thoracic surgery literature [1,11,12].

Subcutaneous VAC-therapy is widely available. It is used in a vast number of different clinical settings and can be installed easily even under local anesthesia. The wound is sealed, and continuous negative pressure is applied to drain the subcutaneous air for several days without changing the dressing. Most of the authors

propose the application of one VAC-drain as described in our case. However, bilateral placement has also been reported [11]. In most of the cases, a maximum of 4 days of VAC-therapy yielded a satisfying decline of SE. So far no complications of VAC-therapy for this specific indication such as occlusion of the drainage or bruising around the insertion site as seen with the other techniques have been observed [1,7,10-12].

## CONCLUSION

Immediate chest tube insertion is the first-line treatment for trauma patients with massive SE, even if a pneumothorax may not reliably be diagnosed initially. Supportive VAC-therapy should always be considered to accelerate the decline of massive SE. However, this recommendation is based on a low level of evidence, since larger case series and studies with higher level of evidence are still lacking.

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