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Tension Subcutaneous Emphysema

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The incidence of barotrauma during controlled ventilation of critically ill patients ranges from less than 1% to more than 40%.[1–3] Pulmonary barotrauma may present as pulmonary interstitial emphysema, pneumothorax, pneumomediastinum, pneumopericardium, retroperitoneal air dissection, pneumoperitoneum, systemic air embolism, and subcutaneous emphysema. While tension pneumothorax, tension pneumomediastinum, and tension pericardium are recognized causes of hemodynamic and pulmonary deterioration, the presence of subcutaneous emphysema in amounts sufficient to cause cardiopulmonary embarrassment is not commonly recognized. We report a patient who suffered severe restriction of ventilation by tense subcutaneous emphysema.

REPORT OF A CASE

A 25-year-old woman suffered a severe closed head injury (Glasgow coma score-4), pneumothorax, and subcutaneous emphysema in a motor vehicle accident. Her trachea had been intubated at the scene of the accident. She was mechanically hyperventilated to decrease intracranial pressure (ICP).

The initial radiologic examination of the chest revealed fractured ribs, a left pneumothorax, and subcutaneous emphysema of the left chest wall. Several hours after admission, a repeat chest roentgenogram showed the development of pneumomediastinum and a decrease in the amount of subcutaneous emphysema, after which the chest tube was repositioned on the left side. Consolidation developed at both lung bases. The left chest tube was removed on the second day, and two subsequent roentgenograms demonstrated the absence of pneumothorax and no change in pneumomediastinum, subcutaneous emphysema, or infiltrates. On the third to sixth day, pneumothorax of varying degrees recurred, despite repeated chest tube insertions. Suction at 25 cmH_2O was applied to all chest tubes at all times.

On the sixth day, tension pneumothorax again became evident. Despite replacement and repeated manipulation of both chest tubes on the left side, the tension pneumothorax persisted. Since air was leaking through chest tube insertion sites, purse string sutures were placed, occluding the leak. The pneumomediastinum began to increase. Subcutaneous emphysema on the right side was noted for the first time and became progressively more severe.

The patient was ventilated with a volume preset ventilator (Emerson) at 12 breaths/min, positive end-expiratory pressure of 7.5 cmH_2O, and a peak inspiratory pressure (PIP), 60 ± 2 cmH_2O. Exhaled tidal volume (Vt), measured at the connection between the endotracheal tube and ventilator circuit, remained constant at 750 ml/breath.

ICP increased from less than 10 to greater than 20 mmHg. Concurrently, jugular venous pressure rose from 20 to 36 mmHg. Pa_{CO_2} increased from 36 mmHg to 48 mmHg; Pa_{O_2} fell from 111 mmHg on an FI_{O_2} 0.80, to 60 mmHg on FI_{O_2} 1.00; ICP rose further to 48 mmHg. The heart rate increased from 105 to 140 bpm, but mean systemic arterial pressure remained stable at 77–83 mmHg.

At this time, the subcutaneous emphysema was found to be extremely tense, with tympany and crepitus extending from the patient's ankles to the mandible and extending down both arms to the wrists. The skin of the chest wall, breasts, and abdominal wall was extremely tense, wrinkle-free, and shiny. The sutures around the chest tubes were removed in order to express some of the subcutaneous air in the hope that chest wall compliance would be improved sufficiently so that ventilation would improve. Removal of the sutures was accompanied by the release of air under obvious pressure with an immediate fall in ICP and jugular venous pressure. PIP fell from 60 to 50 cmH_2O. Pa_{CO_2} decreased from 48 to 40 mmHg. A chest roentgenogram taken immediately after clinical improvement revealed that the pneumothorax had, in fact, increased in size, and a lateral chest film showed that one of the two chest tubes was not within the pleural space. Fiberoptic bronchoscopy revealed copious, thick, white secretions without evidence of tracheobronchial trauma.

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The chest tubes were inserted again. Ventilator PIP fell further, from 50 to 40 cmH_2O. A roentgenogram taken shortly afterward demonstrated complete reexpansion of the lung on the left side. No further difficulties with subcutaneous emphysema or pneumothorax were encountered. The patient subsequently died from her head injury.

Discussion

This case is instructive from several aspects. First, the subcutaneous emphysema in this case was sufficiently tense to cause an elevation in central venous pressure and a restriction of ventilation that caused a 33% increase in Pa_{CO_2}. The elevated Pa_{CO_2} may have resulted from an increase in zone 1 (dead space) ventilation secondary to the rise in airway pressure combined with a reduction in cardiac output. The significant rise in heart rate is consistent with a compensatory response to a low cardiac output. Since V_t was measured at the endotracheal tube, reduction in tidal volume due to compression volume was probably minimal. Perhaps air loss via the bronchopleural fistula increased as well. Total volume delivered by the ventilator before and after the period of hypercarbia was unchanged without readjusting the machine, and the pop-off valve was set well above the peak inspiratory pressures observed. Since V_t was not measured during the acute elevation of Pa_{CO_2}, venous pressure likely led to the increase in ICP. The subcutaneous air in the neck also may have caused extrinsic jugular venous compression.

While subcutaneous emphysema commonly is seen in association with other evidence of barotrauma, it is rarely more important than a temporary cosmetic disfigurement. We believe that hypoventilation and elevated central venous pressure were due to the subcutaneous emphysema under tension. In support of this belief, decompression of the skin resulted in an immediate decrease in venous, intracranial and airway pressures, and Pa_{CO_2}. The chest roentgenogram following clinical improvement showed a worsening of the pneumothorax and pneumomediastinum. At no time was pneumoperitoneum, which may cause respiratory embarrassment by encroaching upon diaphragmatic descent,[4-7] detected. In addition, the subsequent evacuation of the pneumothorax was not associated with any further change in venous or intracranial pressure.

The development of subcutaneous emphysema initially occurred prior to the arrival of this patient at our facility. This may have been due to pulmonary laceration by ribs fractured at the time of the accident or the insertion of the first chest tube. The careful occlusion of the skin wounds around the chest tubes was performed on the sixth day because air was noted to be leaking around the tubes while the pneumothorax persisted. This was interpreted as a potential reason for the persistence of the pneumothorax. However, it ultimately prevented the egress of subcutaneous air and led to the clinical deterioration. In retrospect, the transthoracic air leak was an indication of inadequate evacuation of the pleural space, not the source of the intrapleural air.

The development of tension subcutaneous emphysema probably resulted from a series of interrelated problems. We hypothesize the following sequence. Air entered the pleural space via a pulmonary laceration. The chest tubes were not capable of evacuating the air as rapidly as it accumulated. As intrapleural pressure increased, air began escaping around the chest tube through the intercostal muscle and skin. Once the cutaneous insertion site was occluded, air dissected widely in the subcutaneous tissue planes, ultimately distending this space to its elastic limit. Air could not reenter the chest because the intercostal insertion site acted as a one-way flap valve. The chest wall and diaphragm were splinted pneumatically. Theoretically, the subcutaneous pressure could equal peak inspiratory pressure if the flap valve mechanism remained competent. In any event, subcutaneous pressure might well exceed intrapleural pressure.

While the course of this patient is not entirely typical for major airway disruption, a diagnostic bronchoscopy probably was indicated. Reexpansion of the left lung did not occur until after bronchoscopy, at which time a large amount of thick secretions was removed from almost all segments of the left lung. Perhaps the pneumothorax under tension with the lack of collapse of the upper lobe resulted from a ball valve mechanism in the upper lobe, which contributed to the persistent air leak. Such an event has been reported in association with intraluminal blood clot.[8] Alternatively, obstruction of the lower lobe may have prevented its reexpansion. Once the pleural surfaces were opposed, the air leak diminished significantly.

The malpositioned chest tube probably was not the cause of the persistent pneumothorax, since a second tube was in proper position. In cases where pneumothorax persists despite the insertion of a chest tube, a lateral roentgenogram of the chest should be obtained to confirm proper intrapleural positioning.

In summary, subcutaneous emphysema can cause restriction of the chest wall with subsequent respiratory and, potentially, hemodynamic embarrassment. When a pneumothorax persists despite the presence of a chest tube, its position should be confirmed with a lateral

roentgenogram of the chest. Bronchoscopy should be performed in all cases of persistent pneumothorax in association with trauma to evaluate the presence of major tracheobronchial rupture and also to remove partially obstructing secretions. When air is found to be leaking around a chest tube, the underlying cause of the leak should be corrected before occluding the leak.

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