Pneumothorax, Pneumomediastinum, Pneumoperitoneum and Surgical Emphysema in Mechanically Ventilated Patients

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Abstract: A 29 year old male patient of Indian ancestry was admitted to an outside hospital with rapid deterioration of his level of consciousness. The patient required mechanical ventilation and transfer to MICU at Hamad Medical Corporation. The patient remained hypoxic. Chest X-ray, CT of chest, abdomen, pelvis and proximal areas of both lower limbs were performed. Pneumomediastinum, pneumoperitoneum, and extensive surgical emphysema were the diagnoses.

Key words: Pneumomediastinum, pneumoperitoneum, surgical emphysema, barotrauma, mechanical ventilation.

Introduction
Laennec, in 1827, was the first to define pneumomediastinum as the presence of air in mediastinum [1]. Pulmonary barotrauma in mechanically ventilated patients is a result of alveolar rupture and characterized by development of extra-alveolar air. Perivascular sheaths may be dissected by interstitial emphysema into the mediastinum [2]. If the mediastinal parietal pleura subsequently rupture, a pneumothorax results [3]. Dissection of air elsewhere along fascial planes can result in pneumomediastinum, pneumoperitonium or subcutaneous emphysema [4].

Case report
An Indian male patient, age 29, with no history of chronic illness, presented to the Emergency Department in an outlying hospital with complaints of fever, headache, and transient nausea and vomiting. The patient was admitted to the medical floor and a lumbar puncture was performed. Blood pressure initially was 128/72, heart rate 89/ regular, respiratory rate 21, temperature 39.1 C. Glasgow Coma Scale was 13. Weight was 55 kg. Initial laboratory tests were normal (CBC, chemistries). CSF examination revealed a mild elevation of protein with increased leukocytes, primarily lymphocytes. Chest X-ray and ECG were normal on admission. The diagnosis was viral encephalitis, and acyclovir was initiated. There was a rapid decrease in level of consciousness with Glasgow Coma Scale to 6. The patient was intubated, mechanically ventilated, and transferred to MICU at Hamad Medical Corporation.

Chest X-ray post intubation was normal with endotracheal tube in proper position with no evidence of pneumothorax. During transfer the patient was connected to a small portable ventilator with tidal volume of 700 ml, PEEP of 8, rate of 16 breaths per minute, and PIP of 45 cm H2O. No central line insertions were attempted in the initial admitting hospital or during transfer. The transfer time between the hospitals was about 50 minutes by ambulance. Upon arrival, the patient began to desaturate. He became hypotensive. Chest X-ray revealed a right side pneumothorax. A chest tube was inserted with improvement of saturation and restoration of normal blood pressure. The patient was mechanically ventilated with tidal volume 400, rate 14/minute, peep of 5, and PIP of 30 cm H2O. Acyclovir was continued with gradual steady improvement in the level of consciousness.

On the second day, physical examination was unremarkable. Suddenly the patient desaturated to 82%, with clinical evidence of surgical emphysema over the anterior chest wall and upper abdomen. Chest X-ray, CT chest, abdomen and proximal areas of both lower limbs was performed. Diagnosis of pneumomediastinum, extensive surgical emphysema extending from neck down to knees, left sided pneumothorax and pneumoperitonium was diagnosed. Insertion of left sided chest tube (Chest X-ray 1) was then performed, with conservative treatment of pneumoperitonium and surgical emphysema. Patient improved clinically, and was able to breathe on his own after five days with improved level of consciousness (Glasgow Coma Scale 15). Patient was transferred to the medical floor in improved condition. There was gradual resolution of pneumothorax, pneumomediastinum, surgical emphysema and pneumoperitonium until it resolved after two weeks, and the patient was discharged in good condition.

Discussion
Incidences of pulmonary barotraumas in association with mechanical ventilation are about 3%. Asthma, chronic interstitial lung disease and acute respiratory distress syndrome (ARDS) are independent risk factors for barotrauma. The development of barotrauma is associated with an increased ICU length of stay and an increased mortality rate [5].

High ventilation pressures and global or regional over distension of the lungs are responsible for most cases of barotrauma [6]. Elevation in peak airway pressure during volume-cycled mechanical ventilation often precedes the development of barotrauma [7]. In one report, the incidence of barotrauma was low in patients whose peak airway pressures were less than 50 cm H2O, suggesting this may be a useful therapeutic target [8]. But it deserves mentioning that the relation between peak pressure and barotrauma has been challenged by many other studies[9].
It can be difficult to diagnose pneumomediastinum because it may have many intrathoracic and extrathoracic causes, and radiological findings may simulate other conditions [10].

The mediastinum communicates with several anatomic structures including the submandibular space [11], retropharyngeal space, and vascular sheaths in the neck [12]. A tissue plane extends anteriorly from the mediastinum to the retroperitoneal space through the sternocostal attachment of the diaphragm [13]. This space is continuous with the flank and extends to the pelvis [14]. The mediastinum also communicates directly with the retroperitoneum by way of the periaortic and perivesophageal fascial planes [15-16].

The pneumomediastinum and pneumoperitoneum usually resolve spontaneously without any intervention. Radiological and clinical signs of the condition usually disappear within 14 days [17]. Pneumoperitoneum, if not due to visceral perforation in the setting of mechanical ventilation, can usually be managed conservatively [17].

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