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Tension Pneumothorax Associated With A Coughing Episode In A Patient With Hyperosmolar Hyperglycemic Non-Ketotic State: A Case Report

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Abstract
Tension pneumothorax is a life-threatening emergency requiring prompt diagnosis and intervention. Although commonly associated with a trauma or high- pressure mechanical ventilation, it may also occur in patients without pre-existing structural lung disease as a result of complex patient-ventilator interactions. In this case, underscores the importance of recognizing that metabolic derangements and patient- ventilator interactions may act synergistically to precipitate life-threatening pulmonary complications.

Keywords: Tension pneumothorax, hyperosmolar hyperglycemic state, mechanical ventilation, Auto-PEEP, barotrauma, patient-ventilator interaction

Introduction

Tension pneumothorax is a life -threatening medical emergency characterized by the progressive accumulation of air within the pleural space, resulting in increased intrathoracic pressure, lung collapse, impaired venous return, and potentially obstructive shock. In mechanically ventilated patients, tension pneumothorax is most associated with ventilator-induced lung injury and barotrauma, particularly in the presence of elevated airway pressures, excessive tidal volumes, or underlying pulmonary disease. However, this complication may also occur under conventional ventilatory settings when complex interactions between patient-related and ventilator-related factors are present.

Hyperosmolar hyperglycemic state (HHS) is a severe metabolic complication of diabetes mellitus characterized by profound hyperglycemia, hyperosmolarity, and severe dehydration. While pulmonary manifestations of HHS are generally attributed to altered mental status, aspiration risk, infection, or fluid and electrolyte disturbances, experimental evidence suggests that severe hyperglycemia and osmotic stress may adversely affect alveolar-capillary barrier integrity. Hyperglycemia has been associated with increased pulmonary vascular permeability, impaired epithelial repair mechanisms, oxidative stress, and inflammatory activation, potentially increasing susceptibility to pulmonary injury.

The interaction between these metabolic abnormalities and mechanical factors related to invasive ventilation has been rarely described in the literature. In particular, the use of small-caliber endotracheal tubes may increase expiratory airflow resistance, predisposing patients to dynamic air trapping and the development of auto-PEEP during episodes of coughing. This phenomenon may result in transient but substantial elevations in alveolar pressure, ultimately leading to alveolar rupture and barotrauma

even in patients without pre-existing structural lung disease.

We report the case of a patient with hyperosmolar hyperglycemic state who developed tension pneumothorax during the ventilator weaning process following a coughing episode. We hypothesize that the synergistic interaction between hyperosmolar-induced alveolar vulnerability and ventilator-related mechanical stress contributed to the development of this potentially fatal complication. This case highlights the importance of recognizing how metabolic derangements and patient–ventilator interactions may act together to precipitate severe pulmonary complications despite the use of conventional ventilatory settings.

Case Report

A 62- year- old woman with a history of poorly controlled type 2 diabetes mellitus, hypothyroidism and hypertension was admitted with hyperosmolar hyperglycemic non- ketotic state (HHS) associated with altered mental status requiring advanced airway management in the emergency department. Orotracheal intubation was performed using 6 Fr endotracheal tube because of laryngeal oedema, and the patient was subsequently admitted to the Intensive Care Unit under the care of the Internal Medicine.

On admission, physical examination revealed a Richmond Agitation-Sedation Scale (RASS) score of −4 and a Behavioral Pain Scale (BPS) score of 3. The patient was receiving invasive mechanical ventilation in pressure-controlled intermittent mandatory ventilation (PC-IMV) mode with a tidal volume of 370 mL, positive end-expiratory pressure (PEEP) of 6 cmH_2O, and fraction of inspired oxygen (FiO_2) of 40%, resulting in a peak airway pressure of 25 cmH_2O, plateau pressure of 21 cmH_2O, and driving pressure of 15 cmH_2O.

Appropriate treatment for HHS was initiated, leading to significant clinical improvement during the first 48 hours. Consequently, a ventilator weaning protocol was started, including gradual withdrawal of sedation. Following a self-limited coughing episode, the patient developed acute respiratory failure characterized by increasing oxygen requirements, elevated ventilatory pressures (peak airway pressure 40 cmH_2O and plateau pressure 36 cmH_2O), and hemodynamic instability.

A lung ultrasound examination using the BLUE protocol demonstrated absent pleural sliding and the presence of a barcode sign on M-mode ultrasonography, confirming the diagnosis of tension pneumothorax.

Emergency decompression was performed using a 14-gauge needle, followed by chest tube insertion, resulting in rapid improvement in both hemodynamic status and respiratory parameters.

Discussion

Tension pneumothorax is a medical emergency that requires prompt decompression and chest tube placement. Although uncommon in patients without pre-existing pulmonary disease or a history of trauma, it should be strongly suspected in mechanically ventilated patients who develop acute respiratory failure accompanied by hemodynamic instability and increasing airway pressures.

Our patient had no prior history of pulmonary disease. Hyperosmolar hyperglycemic state (HHS) has been associated with alveolar injury through mechanisms related to severe hyperglycemia and osmotic disturbances, which may increase pulmonary capillary permeability and impair alveolar epithelial repair. These alterations could theoretically increase susceptibility to pulmonary complications and ventilator-induced lung injury.

Following stabilization of the acute metabolic disorder, an investigation into the etiology of the pneumothorax was undertaken. Review of ventilator data revealed a temporal association between coughing episodes and ventilator alarms, with transient increases in peak airway pressure up to 50 cmH_2O and intrinsic positive end-expiratory pressure (auto-PEEP) reaching 16 cmH_2O.

Based on these findings, we

hypothesize that coughing generated a transient increase in expiratory pressures that could not be adequately dissipated because of the increased expiratory resistance imposed by the small-diameter endotracheal tube. This resulted in dynamic air trapping and the development of significant auto-PEEP, leading to increased alveolar stress in a patient who may have already had increased alveolar vulnerability related to HHS. The combination of

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these factors likely promoted alveolar rupture and ultimately resulted in tension pneumothorax.

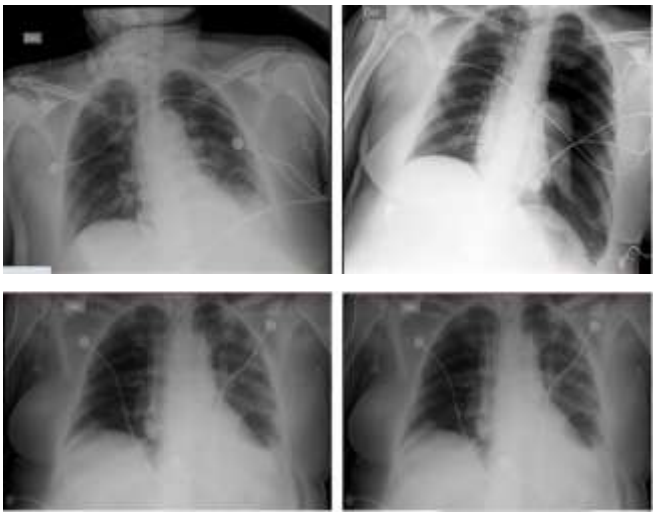

Figure 1. Serial chest radiographs demonstrating the patient´s radiographic pulmonary progression. (On admission) showed preserved pulmonary parenchymal integrity without evidence of acute pulmonary pathology. (Day 3) imaging revealed a massive left-sided pneumothorax involving approximately 50-70% of the lung volume with mild rightward mediastinal shift. (Day 3 left down) A subsequent radiograph obtained after chest tube insertion on the same day demonstrated initial lung re-expansion and correct positioning of the left thoracostomy tube with residual atelectatic changes following re-expansion. (Day 4) Complete re-expansion on the left lung was observed with the left-sided chest tube remaining in place and no evidence of recurrent pneumothorax.

Conclusions

In conclusion, tension pneumothorax may develop even in the absence of aggressive ventilatory settings or trauma. Interactions between patient and ventilator factors, as well as transient physiological events, may trigger traumatic mechanisms such a barotrauma. This case highlights the importance of close monitoring, appropriate endotracheal tube selection, and careful ventilator management.

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