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A Case of Pulmonary Edema Induced by Acute Alcoholic Intoxication

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Abstract A 31-year-old male with no medical history, who runs his own nightclub, experienced altered consciousness after heavy alcohol drinking and was transported to our hospital by ambulance. On arrival, he was in a deep coma with tongue base retraction and snoring respiration. Auscultation revealed wet rales in both lung fields, necessitating tracheal intubation. Whole-body computed tomography revealed left-dominant ground-glass opacities and infiltrative shadows in both lungs' dorsal regions. His heart function was normal. The patient was diagnosed with acute alcohol intoxication, bilateral pulmonary edema, and aspiration. His lung function improved without event, and he was discharged on the third day. This case showed rare pulmonary edema induced by acute alcohol intoxication. The mechanism of pulmonary edema might be due to impaired alveolar epithelium and alveolar macrophages, reduction in alveolar fluid clearance, fluid retention in the human body due to the direct effect of alcohol, and/or negative-pressure pulmonary edema caused by airway obstruction due to coma.

Keywords: *alcohol, pulmonary edema, negative pressure*

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1. Introduction

The increased consumption of soft drinks is associated with t Alcohol intoxication is a clinical diagnosis based on the presence of clinically significant problems and behavioral or psychological changes, accompanied by physiological signs of intoxication, including confusing language, incoordination, unstable gait, nystagmus, conjunctival injection, impaired attention or memory, stupor or coma. [1] The main life-threatening consequence of acute intoxication with high blood alcohol concentrations is respiratory depression. In these conditions, intoxication also reduces the sensitivity of the airways and the reflex block of foreign bodies, increasing the risk of aspiration. [1] While pulmonary edema induced by acute alcohol intoxication is rare, we present a case we experienced.

2. Case Report

A 31-year-old male with no medical history, who runs his own nightclub, experienced altered consciousness after consuming two cups of shochu and one 500 ml bottle of tequila in a single sitting, and was transported to our hospital by ambulance. On arrival, the patient's Glasgow Coma Scale was E1V1M1; blood pressure, 110/85 mmHg; heart rate, 90 beats per minute; respiratory rate, 24 breaths per minute; percutaneous saturation, 100%; and body temperature, 35.9°C. He was in a deep coma with tongue base retraction and snoring respiration, and auscultation revealed wet rales in both lung fields, necessitating tracheal intubation. A 12-lead electrocardiogram showed no significant abnormalities, and cardiac ultrasound indicated hyperdynamic wall movement. Whole-body computed tomography revealed left dominant ground-glass opacities and infiltrative shadows in both lungs' dorsal regions (Figure 1). The patient was diagnosed with acute alcohol intoxication, bilateral pulmonary edema, and aspiration, for which sulbactam and mechanical ventilation were administered. Results of arterial blood gas under 6 L per minute of oxygen via mask were pH 7.32, PCO_2 52.3 mmHg, PO_2 112.0 mmHg, HCO_3^- 26.2 mmol/L, base excess -0.6 mmol/L, and lactate 1.9 mmol/L. Calculated ethanol blood concentration based on the osmotic gap was 753 mg/dL. On the second hospital day, the patient regained consciousness and the ratio of arterial oxygen partial pressure to fractional inspired oxygen improved, leading to extubation. On the third hospital day, the patient was discharged as oxygen requirements ceased and the patient was able to walk and eat.

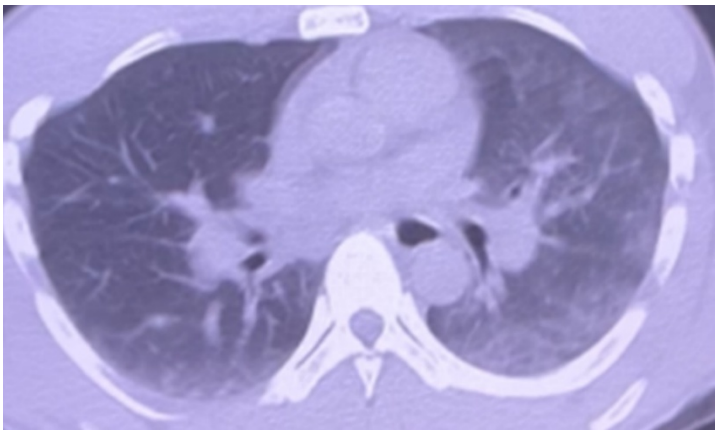

Figure 1. Chest computed tomography (CT) on arrival. The CT revealed left dominant ground-glass opacities and infiltrative shadows in both lungs' dorsal regions

3. Discussion

This is a rare pulmonary edema induced by acute alcoholic intoxication. Previous studies in relevant animal models as well as in human subjects are identifying common mechanisms by which alcohol abuse targets both the alveolar epithelium and the alveolar macrophage, such that the risks for acute lung injury and pulmonary infections are inextricably linked. [2] In addition, both acute and chronic ingestion of alcohol causes an increase in the systemic levels of extracellular adenosine via inhibition of the nucleoside transporter, which impairs the uptake of adenosine.3 Alcohol induces increases in lung adenosine levels, which may be responsible for reduction in alveolar fluid clearance and associated lung injury. [3] Accordingly, acute alcohol intoxication of itself might cause of pulmonary edema.

While, the present case showed initially upper airway obstruction due to deep coma. Negative-pressure pulmonary edema (NPPE) or post-obstructive pulmonary edema is a well-described cause of acute respiratory failure that occurs after intense inspiratory effort against an obstructed airway, usually from upper airway infection, tumor, or laryngospasm. [4] Previously, one report indicated negative pressure pulmonary edema associated with acute ethanol intoxication, similar to our findings. [5]

Alcohol might induce fluid retention in the human body in the early phase of drinking, which could exacerbate pulmonary edema in this case. [6] However, alcohol has a diuretic effect that not only resolves fluid retention but also leads to a dehydrated state in the late phase. [6] Therefore, the resolution of pulmonary edema in the acute phase of this case might be attributed not only to mechanical ventilation but also to the diuretic effect of alcohol.

4. Conclusion

This is a rare pulmonary edema induced by acute alcoholic intoxication. The mechanism of pulmonary edema might be induced by impaired alveolar epithelium and alveolar macrophages, reduction in alveolar fluid clearance, fluid retention in the human body due to the direct effect of alcohol, and/or negative-pressure pulmonary edema caused by airway obstruction due to coma.

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