

Homicidal Strangulation: Uncommon Cause of Noncardiogenic Pulmonary Oedema

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Abstract

Pulmonary oedema is a common problem seen at emergency treatment unit. Although cardiac causes of pulmonary oedema are so common, noncardiogenic causes are not rare. Sepsis with acute respiratory distress syndrome, fluid overload, expansion pulmonary oedema, hypoalbuminemia and neurogenic are the causes of pulmonary oedema. Here we present a rare homicidal case of pulmonary oedema. We considered presenting this case as many of us may not think or look for it unless we are aware of it.

Keywords: Homicide; Noncardiogenic pulmonary oedema; Cerebral hypoxia

Introduction

Pulmonary oedema is fluid collection within lung parenchyma both within alveoli and interstitial tissues. The cardiogenic, fluid overload, hypoalbuminemia, sepsis, toxins and gas infiltrates are the some causes for pulmonary oedema. Here, we present a homicidal case presenting with noncardiogenic pulmonary oedema.

Case Report

A 22-year-old married man was brought to emergency unit with history of sudden onset of difficulty in breathing, one episode convulsion and loss of consciousness. According to his wife, he was perfectly well till 2 pm on that day. His wife went for a sleep and got up at 5 pm because of hearing noisy breathing of her husband and she witnessed an attack of convulsion at this time. She noticed that he was unresponsiveness and breathlessness. Immediately she called her neighbours and brought him to the hospital.

He was unconsciousness on admission. His glasgow coma scale (GCS) was 4 out of 15. He was cyanosed, tachypnoeic with respiratory rate of 32/min. His pulse rate was 132/min and arterial blood pressure was 130/90 mmHg. His chest was bilaterally clear with adequate air entry on auscultation. Oxygen saturation was 60% on pulse oximetry. The arterial blood gas (ABG) analysis showed PaO₂ 51 mmHg, PaCO₂ 54 mmHg, pH 7.21 and BE 6.9 mEq/L on air.

Immediate intubation was done with 7.5 mm internal diameter sized cuffed endotracheal tube after intravenous midazolam 5 mg and intravenous suxamethanium 100 mg. The endotracheal tube revealed full of secretions which was watery in nature with frothiness. The careful examination revealed the strangulation mark around the neck. His wife denied any incidents related that mark. Computerized tomography of brain and cervical spine showed no evidence of hemorrhage or cervical spine injury. The chest X ray revealed alveolar shadow in favoring of noncardiogenic pulmonary edema. 2D echocardiography showed mild left ventricular dysfunction.

He was managed in intensive care unit on Synchronized Intermittent Mandatory Ventilation (SIMV) with respiratory rate of 12/min, tidal volume 380 mL, positive end expiratory pressure (PEEP) 7 cm of H₂O and FiO₂ 0.8 with midazolam sedation, small doses of furosemide and intravenous antibiotics cover. His GCS was started to improved from 3rd day and FiO₂ requirement started to decline from 0.8 to 0.4. His PEEP requirement also started to decline from 7 cm to 5 cm of H₂O. After 4th day, he was fully conscious. His SpO₂ was 99% on FiO₂ 0.4. Chest X ray detected improved abnormality. On 5th day, endotracheal tube was

removed. His post extubation SpO₂ was 99% on FiO₂ 0.3 by facemask.

History from the patient revealed that he was strangulated by somebody behind him when riding motor bike. He was escaped from that event and went home and developed difficulty in breathing and unconsciousness. However he was unable to recall the event with clear evidence.

Discussion

Pulmonary oedema is one of the common medical emergencies in clinical practice. It is defined as fluid collections within lung parenchyma either due to cardiac or noncardiac causes [1]. The common causes of cardiogenic pulmonary oedema are left ventricular dysfunction due to ischemia, hypertension and valvular heart diseases. Negative pressure pulmonary oedema, neurogenics, high attitude, toxins and acute respiratory distress syndrome are the causes for noncardiogenic pulmonary oedema. Noncardiogenic pulmonary oedema is caused by an increase in the vascular permeability of the lung due to high protein content, resulting in an increased flux of fluid into the lung interstitium [2]. Strangulation is a recognized cause of noncardiogenic pulmonary oedema [3].

Pulmonary edema developed immediately following their rescue from acute airway obstruction caused by ligature applied round the neck. However it may be delayed. Definitive mechanism of noncardiogenic pulmonary edema is unclear. The vasoactive substances like histamine, serotonin and kinins released in blood following cerebral hypoxia have a role in noncardiogenic pulmonary edema. These substances cause pulmonary vasoconstriction, pulmonary hypertension and pulmonary congestion [4]. An increase in the vascular permeability of the lung results in an increased flux of fluid and protein into the lung interstitium and air spaces. The net quantity of accumulated pulmonary edema is determined by the balance between the rate at which fluid is filtered into the lung [5] and the rate at which fluid is removed from the air spaces and lung interstitium. Finally noncardiogenic pulmonary oedema has

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high protein content. Abrupt fall in intrapulmonary pressure suddenly increases the venous return that also leads to pulmonary hyperemia and oedema [6].

As a result of pulmonary edema and cerebral hypoxia, patient usually develops respiratory distress with hypoxia and restlessness, unconsciousness and convulsions respectively [7]. This patient presented with signs of respiratory distress and cerebral hypoxia. The clinico-pathological manifestation varies from hyperemia to frank pulmonary edema through lung congestion [8]. It is characterized clinically from clear lung field to basal crepitation on lungs. This patient showing hypoxia without any added sound in the chest at the time of admission may be due to state of hyperemia in the lungs. Later on, he developed frank pulmonary edema due to continuous process of accumulation of fluid in the lungs.

Adequate oxygenation with tracheal intubation with mechanical ventilation [9] is the crucial role in presence of respiratory distress with pulmonary edema. This patient was managed with mechanical ventilation with high PEEP. Pulmonary edema is reversible in most of the cases, once recognized and treated properly [4]. Even though, this patient had pulmonary and cerebral manifestation, he was managed with aggressive oxygen therapy with mechanical ventilator with high PEEP and recovered successfully [8].

Death can occur within minutes due to vasovagal shock secondary to hypoxia and cerebral ischemia caused by ligature applied round the neck. However patient may be saved by applying specific resuscitative measurements if the victim is rescued within few minutes of incident. He was rescued by his relatives and was brought to emergency unit immediately. He was saved with active resuscitation in emergency unit with adequate oxygenation with tracheal intubation with subsequent mechanical ventilation in intensive care unit.

Conclusion

Strangulation should be considered as cause of noncardiogenic pulmonary oedema in a young patient, as many of us may not think or look for it unless we are aware of it. In addition, we have to provide aggressive oxygen therapy with immediate tracheal intubation with subsequent mechanical ventilation for those suspected victims to save their life, as medical professionals.

Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

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