

A Case of Pulmonary Oedema Induced by Suffocation

K Nishi, Y Yanagawa, A Hagiwara

ABSTRACT

A 57-year old female civilian was suffocated by an intruder. The victim (experienced severe dyspnoea) but violently resisted the assault. Two hours after this event, on admission to a Trauma Centre, she demonstrated left facial swelling with low percutaneous oxygen saturation. Chest X-ray and computed tomography demonstrated pulmonary oedema. This improved dramatically within a short time and she was discharged on the 5th hospital day.

Pulmonary oedema induced by suffocation has been reported only rarely. The possible mechanisms by which pulmonary oedema might form after the relief of airway obstruction are discussed.

Keywords: Pulmonary oedema, suffocation, catecholamine

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RESUMEN

Una ciudadana de 57 años fue sofocada por un intruso. La víctima experimentó una disnea severa, pero resistió violentamente el ataque. Dos horas después de este acontecimiento, al ser ingresada en un Centro de Traumas, se le observó hinchazón en el lado izquierdo de la cara, con una baja saturación percutánea de oxígeno. La radiografía del pecho y la tomografía computarizada mostraron un edema pulmonar. Este cuadro clínico mejoró dramáticamente en corto tiempo, y fue dada de alta al quinto día de su hospitalización.

El edema pulmonar inducido por sofocación ha sido raramente reportado. Se discuten los posibles mecanismos por los cuales el edema pulmonar podría formarse después de desbloquearse la obstrucción de la vía respiratoria.

Palabras claves. Edema pulmonar, sofocación, catecolamina

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INTRODUCTION

Pulmonary oedema as a complication of suffocation has been reported only rarely (1, 2). Nonetheless, it is important for medical corps to understand this entity which might occur during military combat. We herein report a rare case of pulmonary oedema induced by suffocation in a patient who also demonstrated elevated catecholamine levels on admission.

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CASE REPORT

A 57-year old female civilian was attacked by an intruder while she slept in a supine position. The intruder sat astride her chest and covered her mouth and nose with a futon in order to suffocate her. The victim violently resisted the assault but experienced severe dyspnoea and the intruder eventually abandoned the attempt and escaped. The victim was able to call the police immediately and was interviewed by them. Thereafter, an emergency medical service was called. The attending emergency medical technician noticed that the victim had low percutaneous oxygen saturation (room air; SPO₂ 86%) and she was transferred to a Level 1 Trauma Centre at the National Defense Medical Hospital approximately two hours after the assault. The patient had a history of diabetes mellitus.

On arrival, blood pressure was 120/70 mmHg; pulse rate was 106 beats per minute; tympanic temperature was

38.3 degrees Celsius and respiratory rate was 12 breaths per minute. Left facial swelling was apparent with left subconjunctival haemorrhage and abdominal tenderness. The remaining physiological findings were unremarkable. Results of blood gas analysis on admission (FiO₂ 5L per min) were pH 7.403, pCO₂ 36.2 mmHg, pO₂ 48.9 mmHg, HCO₃⁻ 22.1 mmol/L, and base excess (BE) -1.7 mmol/L. Serum biochemical parameters were adrenaline 47 (< 100) pg/ml, noradrenaline 916 (100–450) pg/ml and dopamine 57 (< 20) pg/ml. Electrocardiography and cardiac ultrasonography were negative. Chest X-rays demonstrated decreased radiolucency throughout both lung fields. Computed tomography (CT) of the head and abdomen was negative; however, chest CT demonstrated pulmonary oedema (Fig. 1a). She was therefore diagnosed with facial and abdominal contusions and pulmonary oedema. After admission, SpO₂ improved dramatically within a short period. By 15 hours after the assault, pulmonary oedema had improved on chest CT (Figure). She was finally discharged on the 5th hospital day after the pain related to the assault had ameliorated.

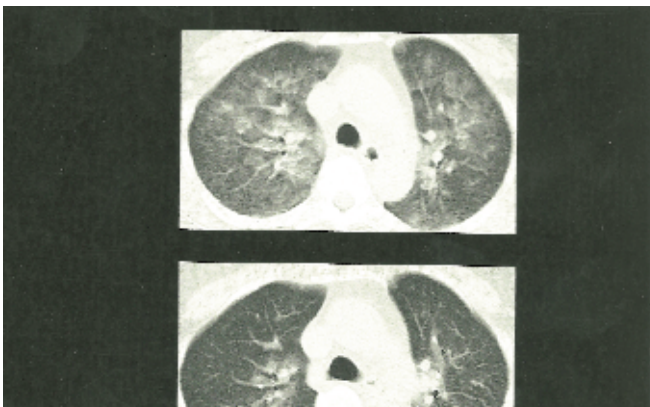


Figure: Chest CT on admission (upper) demonstrating pulmonary oedema. Repeat CT taken 13 hours later (lower) shows dramatic improvement of the oedema.

DISCUSSION

There may have been reports of pulmonary oedema associated with suffocation previously but the new finding is the elevated catecholamine levels.

One possible theory regarding the mechanism causing pulmonary oedema after relief of airway obstruction is the development of a large negative intrathoracic pressure as the victim forcibly tries to inspire against an airway obstruction (2). This favours a large systemic venous return to the right side of the heart and retention of blood within the thoracic cavity (2). Hypoxia leads to pulmonary vasoconstriction and increased permeability of the pulmonary vasculature (3), and both metabolic and respiratory acidosis adversely affect cardiac contractility; these interactions tend to favour the retention of blood in the thoracic cavity, leading to pulmonary oedema (2).

The second theory regarding the mechanism causing pulmonary oedema after the relief of airway obstruction is an elevation in the level of circulating catecholamines. The main mechanism of neurogenic pulmonary oedema is intense pulmonary vasoconstriction which is mainly attributable to the adrenergic response to the cerebral insult, thus resulting in increased pulmonary hydrostatic pressure, followed by an increase in the permeability of the pulmonary capillaries (4). The present patient did not appear to experience any cerebral insult; however, catecholamine levels were elevated on admission, probably in association with the fight-or-flight response. In addition, hypoxia itself also could have led to the release of catecholamines (5). Accordingly, the same mechanism as observed in neurogenic pulmonary oedema, may therefore play a role in the onset of pulmonary oedema after the relief of airway obstruction.

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