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RESEARCH ARTICLE OPEN ACCESS

ACUTE NON-CARDIOGENIC INDUCED LUNG EDEMA BY DRUG

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ABSTRACT

Objective: To report the case of a patient without comorbidities in whom he performed a orthopedic surgery and evolved with complications of Acute Non-Cardiogenic Lung Edema in the immediate postoperative period, after the use of an antagonist of opiates. **Method:** the information was obtained through review of the medical record, interview with the patient, complementary examinations of images to which the patient was submitted and literature review. **Final considerations:** the reported case and publications raised reveal the discussion of the therapy of a rare and serious situation, which can be avoided and, when diagnosed early, it is treated easily.

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INTRODUCTION

Naloxone works as a competitor for opioid receptors. It is used to reverse the effects generated by opioids. It has adverse reactions such as nausea, vomiting, tremor, tachycardia, non-cardiogenic pulmonary edema and cardiac arrest. Non-cardiogenic pulmonary edema (EPNC) is little reported. It is believed that this happens because it is easily confused with other diagnoses. We report a case of EPNC after aplication of naloxone in a young patient without comorbidity.

OBJECTIVE

Report a case of drug-induced non-cardiogenic acute lung edema.

MATERIALS AND METHODS

The information contained in this work was obtained through review of the medical record, interview the patient, complementary imaging tests to which the patient was submitted, and literature review.

CASE REPORT

Male patient, 22 years old, entered the emergency room after a fall in a football game, reported pain in the left wrist region, without other complaints. The examination reported a left distal radio fracture. Confirmed with X-ray of wrist and anteroposterior forearm (AP) + profile. In the opinion of the orthopedist, needed surgery as the best treatment option. The procedure took place with the help of general anesthesia and lasted 90 minutes. There were no complications. In the immediate postoperative period, the patient remained sleepy, opting for perform naloxone and flumazenil to reverse the clinical picture. There was an improvement in the condition neurological, but evolved with acute respiratory failure requiring oxygen in high flow and non-invasive ventilation to maintain adequate saturation. To the physical examination before the interventions was taquipneic, O2 saturation 79%, presence of diffuse fine stinges, tachycardic (180 bpm) and hypertensive (226x100 mmHg). In the emergency chest X-ray in the bed, was verified infiltrate peripheral diffuse interstitial, ECG with sinus tachycardia. It was transferred to the ICU to continue the oxygen therapy, NIV, use of furosemide and monitoring. In addition, he presented echocardiography on the edge normal bed. After 48 hours in the ICU, the x-ray showed no changes. Image A and Image B.

Lasix 0.5mg/kg and non-invasive mechanical ventilation was performed, obtaining hospital discharge on the 4th day of hospitalization. Thus, the mains diagnosis remained as acute pulmonary edema resulting from drug.

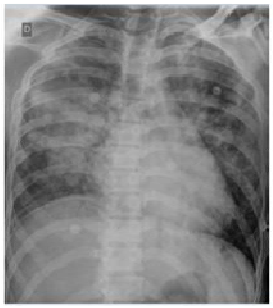

Image A. X-ray of the onset of symptoms

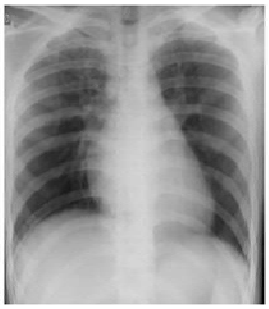

Image B. Post-treatment X-ray

DISCUSSION

The CNS depressant effect was due to the use of the potent sufenta opioid, being naloxane chosen for the reversal of this effect. Therefore, the pulmonary edema was caused by a response adrenergic by an increase in catecholamines mediated centrally after the administration of naloxone. The effect of catecholamines results in hypertension, tachycardia and tachypnea, increasing capillary permeability.

Therefore, it will have a sympathetic release of epinephrine into the plasma, which antagonizes the opioid receptors in the adrenal medulla and raises the sympathetic flow, shifting the volume blood to the pulmonary vasculature, causing pulmonary vasoconstriction and pulmonary hypertension.

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