

Revisiting opioid overdose induced acute respiratory distress syndrome

Sir,

Opioids are commonly used analgesic drug class with morphine like activity. Although respiratory system is the most affected organ system in opioid overdose; acute lung injury and acute respiratory distress syndrome (ARDS) are infrequently reported.

A 25-year-old male presented after ingesting unknown amounts of hydrocodone. Patient was found to be unresponsive, with agonal respiration. Initial vital signs showed blood pressure 100/60 mmHg, pulse 110 beats/min, respiratory rate 8 breaths/min and oxygen saturation (SpO₂) of 70%. Chest examination revealed decreased air entry bilaterally with bibasal crackles. Initial arterial blood gas (ABG) analysis showed respiratory acidosis with pH 7.22, partial pressure of carbon dioxide (PaCO₂) at 70 mmHg, partial pressure of oxygen (PaO₂) at 42 mmHg and SpO₂ of 72%. An emergent orotracheal intubation was performed due to severe hypoxemia and to maintain a patent airway. Pink frothy secretions were seen in the endotracheal tube immediately after intubation and a chest radiograph showed diffuse bilateral infiltrates [Figure 1]. Mechanical ventilation was started at assist control mode with initial tidal volume of 500 ml, fraction of inspired oxygen (FiO₂) at 100% and positive end expiratory pressure (PEEP) at 5 cm H₂O. Patient remained hypoxemic and repeat ABG analysis showed partial pressure of PaO₂ 42 mm Hg and SpO₂ 80% prompting increase in the PEEP up to 10 cm H₂O, which improved PaO₂ to 85 mm Hg. Based on initial PaO₂/FiO₂ ratio of 52 and characteristic radiographic bilateral opacities, severe ARDS was diagnosed. An echocardiogram showed normal left ventricular function and serum levels of brain natriuretic peptide (BNP) was normal at 88 pg/ml. Lung protective ventilation to achieve adequate oxygenation was continued. Furosemide was given to maintain a negative fluid balance. Patient could be successfully extubated on the 2nd day. Chest radiograph on day 3 showed complete resolution of ARDS [Figure 2].

Opioid overdose has been implicated as a cause of ARDS.^[1] Sporer and Dorn described 27 patients of non-cardiogenic pulmonary edema (NCPE) secondary to heroin overdose.^[2] Overall, clinical presentation of opioid-induced ARDS is generally indistinguishable from other causes of ARDS. Presence of frothy, sometimes blood tinged pulmonary secretions and bilateral diffuse infiltrates on chest radiograph are characteristic. Occasionally, it may be necessary to demonstrate normal cardiac pump function through measurements of serum BNP levels, echocardiogram and rarely right heart catheterization. The mechanisms behind opioid induced NCPE and ARDS remain largely unknown. The role of increased pulmonary capillary permeability due to hypoxemia, potent histamine release and respiratory acidosis caused by depression of medullary respiratory centers have been implicated.^[3,4]

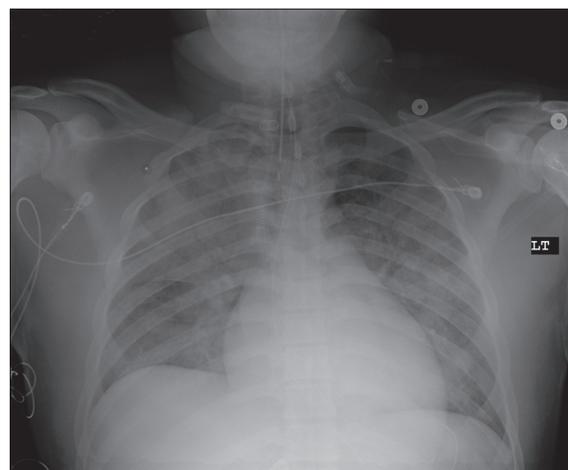


Figure 1: An antero-posterior chest radiograph demonstrating diffuse pulmonary edema in the intubated patient

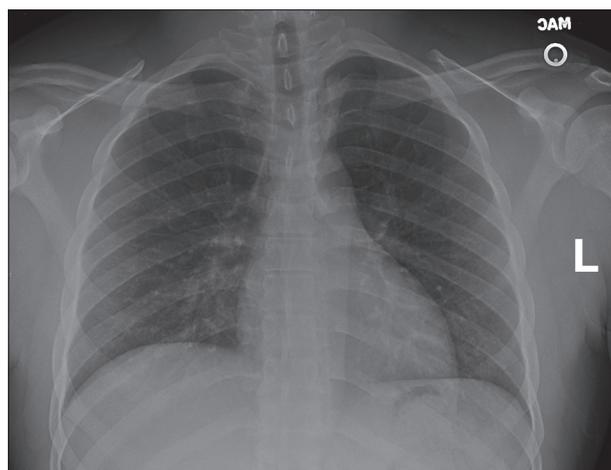


Figure 2: A postero-anterior chest radiograph in the same patient 48 h later demonstrates complete resolution of the pulmonary edema

Interestingly, the use of reversal agents like naloxone itself has also been described as the primary cause of pulmonary edema.^[5]

Patients with opioid-induced NCPE and ARDS can have severe hypoxemia frequently needing invasive mechanical ventilation. An early orotracheal intubation should be considered in obtunded or severely hypoxemic patients. Following intubation, general management strategies used in any form of ARDS comprising of lung protective strategies of ventilation, appropriate PEEP and diuretics are used. Overall prognosis remains good with most patients recovering within 24 h.

In summary, our case demonstrates an infrequently seen respiratory complication in opioid overdose. Timely management decisions about adequate use of reversal agents and considering early mechanical ventilation in severely hypoxemic patients can be lifesaving.

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