

Naloxone Causing Acute Pulmonary Oedema in a Previously Healthy Patient

Sir,

We like to report an unusual complication of acute pulmonary oedema following naloxone administration in a previously healthy young individual. This is the first reported incidence in Malaysia. A 23-year-old male, non smoker presented for septoplasty operation. A similar operation in 1988 was uneventful. Premedication included pethidine 50 mg and phenergen 25 mg given 3 hours prior to anaesthesia. He was induced with thiopentone 250 mg, vecuronium 6 mg and intubated orally. Anaesthesia maintained with oxygen/nitrous oxide/isoflurane. Vital signs remained stable throughout. Fifteen minutes before the end of operation 3 mg morphine was given to supplement post operative pain relief. On conclusion of operation, throat pack removed and direct laryngoscopic suction showed a good gag reflex. Neuromuscular block reversed using neostigmine and atropine. The patient showed signs of arousal by lifting his head and reacted strongly to the endotracheal tube. He was extubated and 100% oxygen given via a face mask. However the patient noted to have shallow breathing, not obeying commands and had pin-point pupils. He required reintubation as he began to desaturate. Naloxone 0.4 mg was given slowly to reverse a suspected opiate induced respiratory depression. Sixty seconds later large copious pink frothy fluids poured out of the endotracheal tube together with bilateral fine crepitations on auscultation of the lung suggestive of acute pulmonary oedema (APO). Eighty mg of Frusemide given and patient then admitted to ICU. Chest x-ray showed definite changes consistent with APO. Symptoms and signs of APO subsided 3 hours after admission. Repeat CXR was normal. Possible differential diagnoses that had

been considered were : Firstly, APO secondary to acute airway obstruction. However this is more common in children. In our case there was no laryngospasm or upper airway obstruction. Secondly, fluid overload that can be ruled out as only 350 mls of Hartman's solution was transfused and patient was an ASA 1. In all reported cases¹⁻³, only ten patients suffered some cardiac complications following naloxone administration and three were fatal cases. The exact mechanism not known. One postulation is that APO occurs after profound CNS stimulation. Postoperatively CSF endorphins levels are lower than controls. Naloxone further inhibits the endogenous pain suppression pathway and permits unopposed noradrenergic transmission from medullary centres which result in outpouring of catecholamines causing pulmonary vasoconstriction, increased pulmonary vascular permeability and hypertension. Other effects include centrally mediated increase in sympathetic tone causing shift of blood volume from systemic to pulmonary vascular bed as well as inhibition of parasympathetic outputs. Although APO following naloxone is rare, a high index of suspicion should be exercised during usage of naloxone.

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