**"Deciphering the Culprit: Origins of Non-Cardiogenic Acute Pulmonary Oedema in Opioid Toxicity – Opioid-Induced or Naloxone-Related?"**

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**Introduction:**
Non-cardiogenic Acute Pulmonary Oedema (APO) is a rare but potentially fatal complication of opioid toxicity. Consequently, naloxone is commonly employed to reverse opioid toxicity. Despite its safe nature, one possible complication of naloxone administration is non-cardiogenic APO.

**Case Presentation:**
A 57-year-old gentleman, with underlying sleep disorder on self- buy methadone, was brought to our casualty, after being found unconscious. Patient involved in an altercation and was then observed that patient consumed approximately 100mls of Methadone. Patient started to become drowsy and subsequently unresponsive. At ED, GCS E1V1M1, Central and peripheral Cyanosis, RR=6, Bradypnea, Bilateral Pupil Pin point. Vital Sign BP: 128/98, SPO2: 20% under RA. Auscultation, reduce air entry Bilateral Lower zone with Crepitation. Patient was started on Manual bagging and IV Naloxone 2mg + 2mg given 5 minutes apart. Subsequently, GCS improving E3V4M6 with Respiratory Rate of 16. Later noted crepitation worsening up to midzone with CXR showed congested lung field. Saturation Unable to maintain above 95% while on HFMO2. Bedside Scan noted B-lines over B/L lower till mid-zone with good cardiac contractility. Patient was started with Non-invasive Ventilation (NIV) and IV Furosemide was given. Later In ICU, Infusion of naloxone was commenced for 1 day and the Oxygen supply was being titrate down from NIV. Patient was discharged after 4 days.

**Discussion:**Pulmonary oedema secondary to naloxone administration is because of unrestricted catecholamine surge following opioid reversal. It may also be due to constriction of the pulmonary vasculature due to central neurogenic mechanisms, leading to pulmonary hypertension. A final mechanism is the return of respiratory drive prior to patient control of their own airway, resulting in inspiration against an obstructed glottis, precipitating negative pressure pulmonary oedema.

Finally, the opioid overdose itself can induce pulmonary oedema by inducing histamine release, hypoxia, and acidosis resulting in permeability of the pulmonary vasculature.

**Conclusion:**
Both opioid overdose and the administration of naloxone can trigger acute pulmonary oedema, albeit through distinct pathophysiological mechanisms. Therefore, as physicians, it is imperative for us to anticipate the potential complication of acute pulmonary oedema in patients presenting with severe opioid toxicity.

**Keywords:** Opioid, Naloxone, APO