International Journal Dental and Medical Sciences Research
Volume 5, Issue 5, Sep-Oct 2023 pp 73-76 www.ijdmsrjournal.com ISSN: 2582-6018

Delayed Cerebral edema Leading to Uncal Herniation and Delayed Pulmonary Edema in a Patient with Opioid Toxicity

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Date of Submission: 05-09-2023 Date of Acceptance: 15-09-2023

ABSTRACT
A 24 year old, male patient, who was a poly substance user including opioids presented to the Emergency department in respiratory arrest, altered sensorium and bilateral pinpoint pupils. The patient woke up after 6 mg of Naloxone in aliquots. But his sensorium was not fully recovered and in view of type 2 respiratory failure, patient was intubated and put on mechanical ventilation. The initial CT head showed bilateral globuspallidushypodensities and chest X-ray was normal.However later on patient developed pulmonary edema after about 8 hours. His sensorium further dipped on ventilator with loss of brain stem reflexes after three days. A repeat CT head showeddiffuse cerebral edema with bilateral uncal and tonsillar herniation and patient succumbed on the fifth day.
The plausible mechanism for cerebral edema could have been hypoxic injury due to hypoventilation. Clinicians should have a high index of suspicion for diagnosing and treating opioid toxicity especially when it comes to poly substance users.

I. INTRODUCTION

Globally, drug use accounts for about 0.5 million deaths. Opioids account for more than 70% of these deaths, with more than 30% of those deaths caused by overdose.(1)

According to the World Drug Report 2021, about 5.5 percent of the population aged between 15 and 64 years have used drugs at least once in the preceding year, and 36.3 million people, suffer from drug use disorders. (2) The largest burden of drug use disorders is still attributed to opioids. Heroin is the predominant opioid misused in India.

Opioid-associated out of hospital cardiac arrest is different from cardiac arrest due to other etiologies.Cardiac arrest stemming from opioid overdose is due to asphyxia, respiratory depression, and hypoxemia leading to global ischemia and cessation of cardiac output. Hence it is important for an emergency physician to recognize cardiac arrest due to opioid toxicity promptly, as well as be familiar with the underlying mechanisms, so as to resuscitate successfully. A high degree of clinical suspicion is needed to recognize opioid toxicity.

We report a case in whom history of polysubstance use, initial presentation in respiratory arrest, delayed development of potentially fatal and unusual clinical features, posed a diagnostic and therapeutic challenge.

II. CASE REPORT

A 24-year old, opioid user was brought to the emergency department in an altered sensorium. On initial assessment his airway was threatened, with a respiratory rate of 6 cycles per minute, an unrecordable saturation, heart rate of 108 beats per minute and blood pressure of 89/68 mm Hg.

On examination, patient was cyanosed and pupils bilaterally dilated with sluggish reaction to light. There were multiple hesitation cuts and needle track marks on both forearms. Cardiac and pulmonary system examination at this point was unremarkable. Brain stem reflexes were intact.

As the patient was a chronic opioid user and presented in respiratory arrest, we gave him a cumulative dose of 6 mg naloxone intravenously, starting with 2 mg simultaneously supporting his respiration with bagging and masking. Subsequently, the patient developed spontaneous respiratory efforts and his Glasgow Coma Scale score improved from E1V1M1 to E2V2M5. The arterial blood gas on presentation had a pH of 6.8, pO2 – 30.3, pCO2 – 92.1, HCO3-15.7, lactate-11.6. Complete blood count, metabolic profile, and liver function tests were normal. ECG had sinus tachycardia and cardiac enzymes were undetectable. Urine toxicology test showed positivity for opiates. Patient was intubated and put on mechanical ventilation in view of borderline GCS and type 2 respiratory failure. The Naloxone infusion was continued.

The first computed tomography (CT) of the head showed chronic bilateral globuspallidushypodensities (Panel A). There was no evidence suggestive of any acute intracranial pathology. Ventilatory support was continued along with other symptomatic measures.

Though the patient had a clear chest on auscultation at presentation, a chest x-ray after about 8 hours showed diffuse pulmonary infiltrates suggestive of pulmonary edema (Panel C).

After three days, the patient deteriorated again with loss of all brain stem reflexes. A repeat CT head done after 3 days showed diffuse cerebral edema with bilateral uncal and tonsillar herniation (Panel B). The patient was pronounced brain dead based on brain death criteria after performing all necessary examinations and tests. The patient expired on the fifth day of admission.

III. DISCUSSION

The United States has witnessed ‘ the opioid epidemic’ since the 2000s, secondary to illicit prescription. (3) The diagnosis of acute opioid poisoning is primarily clinical with the triad of miosis, lethargy, and respiratory depression, paired with a positive response to naloxone. The classic sign of pinpoint pupils is not always present, especially in poly substance use. Our patient presented in cardiac arrest. Cerebral hypo-perfusion results in severe anoxic brain injury.

Non-cardiogenic pulmonary edema (NCPE) is a rare, but fatal complication. It can occur immediately or may be delayed up to 24 hours after an overdose and presents with persistent hypoxia, tachypnea, rales, and bilateral pulmonary infiltrates with a normal cardiac silhouette on the chest radiograph. The incidence of NCPE is 10% in patients with severe toxicity requiring naloxone. The exact pathophysiology of opioid-induced acute lung injury is unclear. The plausible mechanisms could be direct alveolar injury from hypoxia, lung injury from direct opioid exposure, opioid-induced histamine release, or neurogenic injury.

Naloxone-induced NCPE is a rare but reported entity with an incidence of 0.2%- 3.6%. (1) Proposed mechanism is catecholamine surge secondary to naloxone administration. It is more common with higher doses of naloxone. (4)

Our patient developed pulmonary edema 8 hours after presentation.

The neuropathologic findings in heroin users include stroke, vasculitis, recurrent infections, mycoses, seizures, or HIV-1 infection. Other complications include hypoxic-ischemic changes with diffuse cerebral edema, ischemic neuronal damage, and neuronal loss, secondary to hypoxemia or hypercarbia caused by hypoventilation. (5)

The first CT head of our patient revealed bilateral globus pallidus hypo densities and repeat CT head after 3 days had diffuse cerebral edema with bilateral uncal and tonsillar herniation.

Bilateral globus pallidus lesions are typically secondary to global cerebral hypoperfusion and hypoxic changes. (6) Five to ten percent of intravenous heroin addicts might have pallidal infarcts, with or without other features of hypoxic/ischemic brain injury. (7)

The ED physician should promptly recognize opioid poisoning. The priority should be Airway-Breathing-Circulation-naloxone. This can be accomplished with a combination of basic supportive measures and titrated use of antidotal therapy. Patients with acute lung injury may require oxygen and positive-pressure modalities, or mechanical ventilation.

COVID-19 pandemic has brought economic woes that has made fragile rural communities to engage in illicit drug cultivation and the social impact of the pandemic has pushed, the already vulnerable population into drug use.

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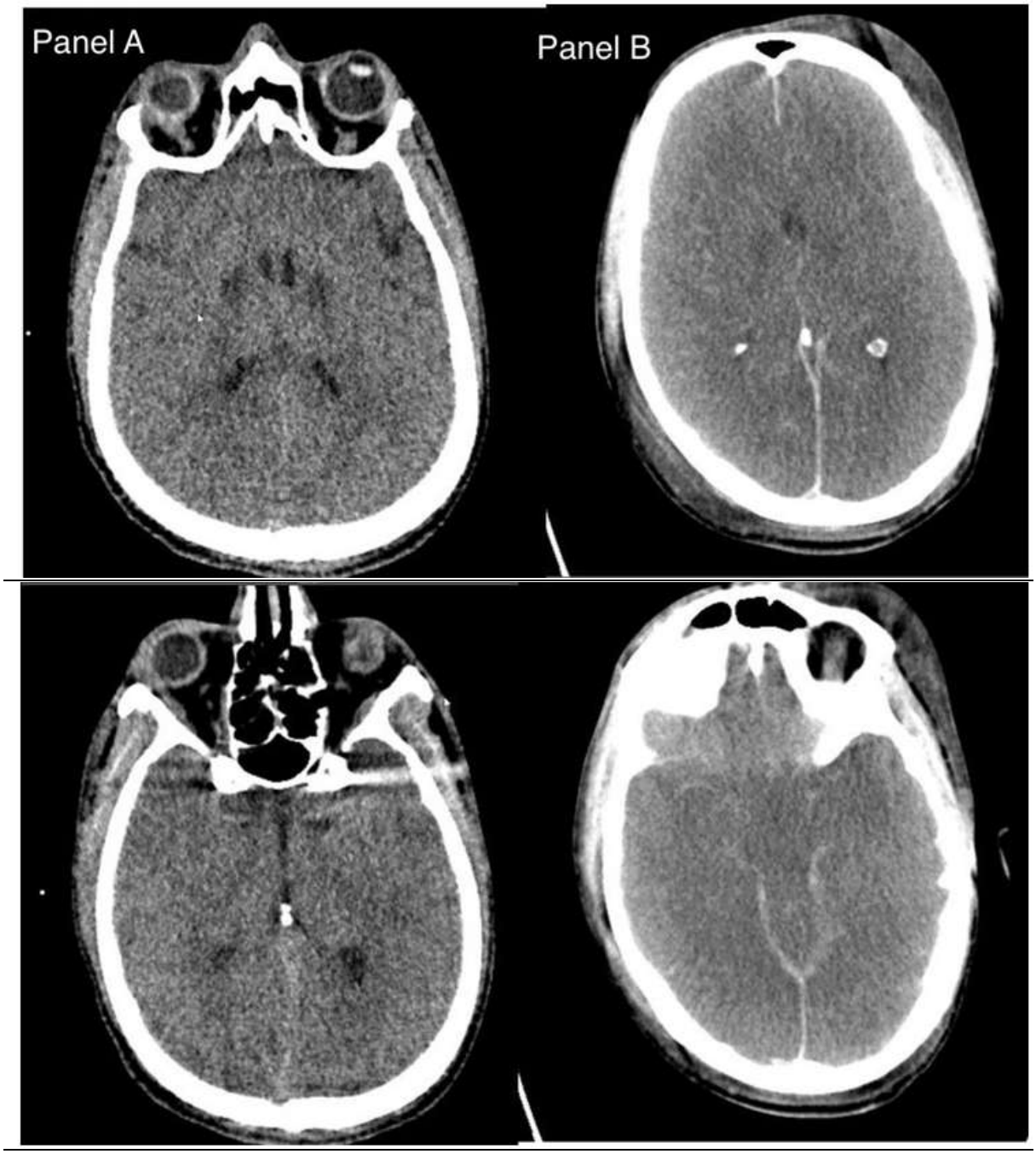
IMAGES



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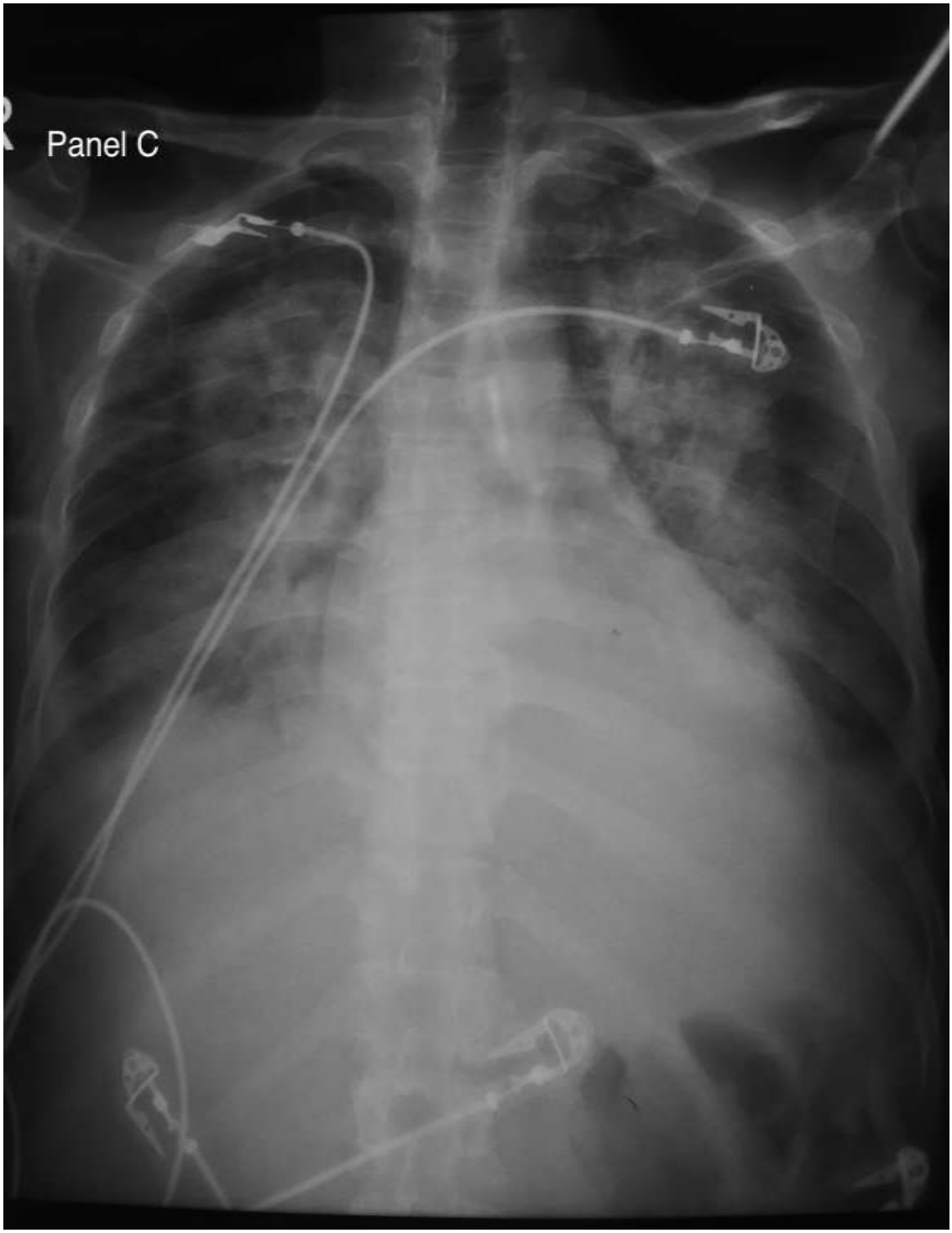
Volume 5, Issue 5, Sep-Oct 2023 pp 73-76 www.ijdmsrjournal.com ISSN: 2582-6018

LEGENDS OF FIGURES



Panel A-Computed tomography (CT) of the head showed chronic bilateral globuspallidushypodensities
Panel B- Diffuse cerebral edema with bilateral uncal and tonsillar herniation
Panel C- Chest X-ray showing diffuse pulmonary infiltrates suggestive of pulmonary edema