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Wooden Chest Syndrome: A Case Report of Fentanyl-Induced Chest Wall Rigidity

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Abstract

Wooden chest syndrome (WCS) describes a finding of fentanyl-induced skeletal muscle rigidity causing ventilatory failure. Known primarily to anesthesiology, pulmonary, and critical care fields, WCS is a rare complication that may affect patients of all ages if exposed to intravenous fentanyl, characterized by a patient's inability to properly ventilate. Given the rise of synthetic opioid deaths across the United States in the past decade, an understanding of all of fentanyl's effects on the body is necessary. In this article, we present a case of WCS in a patient with acute respiratory distress syndrome in a 61-year-old female.

Keywords

fentanyl, respiratory failure, wooden chest syndrome, fentanyl-induced chest wall rigidity, opioids

Introduction

Fentanyl-induced chest wall rigidity, otherwise known as wooden chest syndrome (WCS), is a rare complication of intravenous fentanyl. Fentanyl is an opiate medication that is often used in critical care and procedural settings as an analgesic paired with sedatives for intubated patients. We describe a case of a patient intubated for acute respiratory distress syndrome who developed a complication of WCS in a medical intensive care unit.

Case Report

A 61-year-old female with a history of prior episodes of pancreatitis presented with a chief complaint of left-sided, sharp, abdominal pain associated with nausea. Physical examination demonstrated epigastric tenderness without rebound or guarding. Chest examination demonstrated strong inspiratory effort and was negative for rales, rhonchi, or accessory muscle use. Vital signs demonstrated no abnormalities and she had oxygen saturations >92% on room air. Laboratory findings revealed an elevated lipase of 1086 U/L (reference range: 10-140 U/L). A diagnosis of pancreatitis was made. The patient was given intravenous lactated Ringer's solution. The patient developed respiratory distress, and chest X-ray findings revealed pulmonary edema consistent with acute respiratory distress syndrome, requiring endotracheal intubation. She was sedated initially with an intravenous fentanyl infusion at 50 μg/h. An intravenous midazolam infusion at 1 mg/h was added to achieve a Richmond Agitation-Sedation Scale goal of −4. The fentanyl infusion was incrementally increased, roughly 50 to 75 μg/h per day, and ultimately maximized to 300 μg/h on mechanical ventilation day 5. The dose of intravenous midazolam infusion reached 6 mg/h. On maximization of the intravenous fentanyl infusion at 300 μg/h, the patient began experiencing periods of hypoxia. The patient had received roughly 11 000 μg (11.1 mg) of intravenous fentanyl when these periods of hypoxia began. Physical examination at this time was significant for a tense abdomen, facial cyanosis, and episodes of what appeared to be breath-holding spells. Ventilator readings revealed drastically elevated airway pressures. The patient required bag valve mask ventilation, which was met by strong resistance. Passage of the suction catheter revealed no obstruction or mucus. An emergent bedside bronchoscopy confirmed these findings. A repeat chest X-ray revealed no pneumothorax. This presentation raised concern for WCS. The intravenous fentanyl infusion was titrated down

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rapidly over a 3-hour period and she was transitioned to an intravenous dexmedetomidine infusion at 0.2 mg/h in addition to the intravenous midazolam infusion. The patient exhibited no further episodes was successfully extubated 3 days later.

Discussion

Opioid-induced chest wall rigidity was first coined by Hamilton and Cullen in 1953 when studying opioid effects on respiratory depression.[1] Since then, reports of patients exhibiting similar reactions to opioids have been noted but are quite limited. The term "wooden chest syndrome" has since been adopted to describe these findings.[1] Review of literature suggests that a cohort of neonatal and pediatric population is affected; however, no specific pattern of adult patients has been observed.

The proposed mechanism of WCS is activation of μ-opioid receptors in the central nervous system (CNS) via a dopaminergic pathway resulting in skeletal muscle rigidity.[2] Lipophilicity is an important property in opioid delivery to the CNS. Fentanyl is a highly lipophilic molecule, allowing for it to readily cross the blood-brain barrier and interact with the CNS. In addition, a study by Henthorn et al posits that an active transporter may contribute to increased fentanyl levels within the CNS.[3] These findings may suggest why skeletal chest wall rigidity is observed more so with fentanyl as compared with equivalent doses of other commonly used opioids. Dose dependency, however, is debated. Some case reports have demonstrated clinical findings of WCS with low doses of fentanyl and rapid administration, while others report very high doses in the form of infusions, as in our patient.[2,4]

Common physical examination findings in WCS are episodic "breath-holding spells," tense abdominal muscles, a firmly locked jaw, and stiff extremities. Hypoxia and hypertension are also common during these episodes, but no other patterns of changes in vital signs are reported. One case reported a fever to 103 °F; however, a concurrent diagnosis of serotonin syndrome was made as well.[5]

These breath-holding spells are a manifestation of prolonged chest wall skeletal muscle contraction. The subsequent decreased chest wall compliance results in ineffective assisted and spontaneous ventilation, translating to elevated pressures within the ventilator circuit. Bag valve mask ventilation is exercised to combat hypoxia but is often met by a high resistance.

When managing a ventilated patient for respiratory failure and increased peak pressures, early consideration of WCS is helpful, yet ruling out other life-threatening causes of hypoxia and elevated ventilator peak pressures is paramount. Spontaneous pneumothorax, mucus plugging, and/or defects in the endotracheal tube and ventilator circuit should be ruled out in a timely manner. A chest X-ray, passage of suction catheter, and, in some cases, an emergent bedside bronchoscopy should be performed. When WCS is suspected, there are 3 reported options for management. The first option is administration of intravenous naloxone, an opioid antagonist and reversal agent for opioid overdose. This is the most common treatment and can result in rapid reversal of chest wall rigidity.[6] In patients requiring high doses of fentanyl, the decision to administer naloxone should be carefully considered as withdrawal symptoms may rapidly follow. The second route of management is the administration of neuromuscular blocking agents such as rocuronium and cisatracurium. These agents competitively block transmission of neuronal signaling at the level of nicotinic receptors, resulting in decreased skeletal muscle tone. They are useful with ventilated patients early on in their medical intensive care unit course and allow for the continued use of fentanyl. The last management route is cessation of the offending agent. This decision can be made if the patient is able to tolerate the time required for clearance of fentanyl. In our case, fentanyl was rapidly titrated down and resulted in cessation of ventilator desynchrony. In order to maintain adequate sedation, other agents should be administered; in our case, dexmedetomidine and hydromorphone were chosen.

Conclusion

Prolonged chest wall skeletal muscle contraction is a result of fentanyl-bound μ-opioid receptors on the CNS and activation of a dopaminergic pathway. The subsequent decreased chest wall compliance results in ineffective assisted and spontaneous ventilation, translating to elevated pressures within the ventilator circuit. Management of this syndrome is with the opioid receptor antagonist Naloxone, neuromuscular blocking agents such as rocuronium, or cessation of fentanyl infusion with supportive care. In light of the coronavirus disease 2019 pandemic and surge in intensive care unit admissions, analgesic fentanyl use has risen. Therefore, an understanding of this complication is necessary.

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Ethics Approval

Our institution does not require ethical approval for reporting individual cases or cases series.

Informed Consent

Verbal informed consent was obtained from the patient for their anonymized information to be published in this article.

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