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THE SILENT THREAT OF WOODEN CHEST SYNDROME: PROMPT MANAGEMENT OF FENTANYL-INDUCED MUSCLE RIGIDITY IN THE INTENSIVE CARE UNIT

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ABSTRACT

Introduction: Wooden chest syndrome (WCS) is a rare, potentially fatal complication of high-dose fentanyl therapy, characterized by generalized muscle rigidity, reduced chest wall compliance, and acute respiratory distress. Frequently underdiagnosed in the intensive care unit (ICU), WCS poses a significant challenge due to its rapid onset and potential to cause ventilatory failure if not addressed promptly. This case report examines WCS in a postoperative setting, emphasizing its recognition and management to improve patient outcomes.

Case Presentation: A 49-year-old female with a history of cervical cancer, hypertension, and recent sigmoidectomy developed WCS 8 hours after starting a fentanyl infusion (50 µg/h, total ~400 µg) following an emergent incision and drainage for a 4 × 3 cm neck abscess. Initially stable on pressure-regulated volume control ventilation (peak airway pressure, Ppeak 20 cmH_2O, tidal volume 450 ml), she presented with sudden chest wall rigidity, respiratory rate of 28 breaths/min, and Ppeak rising to 35 cmH_2O, alongside respiratory acidosis (pH 7.28, $PaCO_2$ 58 mmHg). Fentanyl was stopped, naloxone (2 mg IV) administered, and ventilation shifted to pressure control mode of 25 cmH_2O above positive end-expiratory pressure. Dexmedetomidine (0.5 µg/kg/h) replaced opioids for sedation. Symptoms resolved within 4 hours, enabling a transition to pressure support mode and extubation on postoperative day 2, with discharge to the floor by day 5.

Conclusion: This case highlights WCS as a critical, reversible opioid-related complication in the ICU, necessitating vigilance during prolonged fentanyl use (>8 hours). Early detection via ventilator changes (e.g., Ppeak elevation) and swift intervention with naloxone and non-opioid sedation can prevent adverse outcomes. Increased awareness, staff training, and further research are vital to enhance ICU safety.

KEYWORDS

Wooden chest syndrome, fentanyl, muscle rigidity, intensive care unit, respiratory distress

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LEARNING POINTS

- *Early detection is crucial:* wooden chest syndrome can manifest as sudden muscle rigidity and rising peak airway pressures within 8 hours of fentanyl infusion, requiring prompt recognition to prevent ventilatory failure.
- *Ventilator changes guide diagnosis:* elevated peak pressures with stable plateau pressures signal wooden chest syndrome, distinguishing it from lung pathology, and warrant immediate investigation.
- *Swift management improves outcomes:* stopping fentanyl, administering naloxone, and using dexmedetomidine can reverse wooden chest syndrome rapidly, enabling extubation within 48 hours.

INTRODUCTION/BACKGROUND

Opioids are a cornerstone of analgesia in the intensive care unit (ICU), valued for their efficacy in managing acute pain in critically ill patients. Fentanyl, a synthetic opioid, is particularly favoured due to its rapid onset, short half-life, and minimal hemodynamic effects, making it ideal for procedural sedation and postoperative pain control. However, its potent mu-opioid receptor agonism can lead to rare but severe adverse effects, including wooden chest syndrome (WCS). First documented in forensic reports of fentanyl overdose, WCS manifests as profound skeletal muscle rigidity, predominantly affecting the chest wall and abdomen, resulting in decreased thoracic compliance, impaired ventilation, and acute respiratory distress[1].

The pathophysiology of WCS is complex, involving opioid-induced hyperactivity within the central nervous system (CNS), particularly in regions regulating motor control and respiration. Dysregulation of neurotransmitters such as dopamine and gamma-aminobutyric acid is implicated, though the precise mechanisms remain under investigation[1]. Despite its potentially fatal consequences, WCS is often underrecognized, overshadowed by more common opioid-related complications like respiratory depression or tolerance. In the ICU, where patients are frequently mechanically ventilated and sedated, the insidious onset of WCS can delay diagnosis, increasing the risk of morbidity and mortality. Compounding this challenge is the lack of standardized diagnostic criteria and management protocols, leaving clinicians reliant on clinical suspicion and rapid intervention[2].

This manuscript presents a detailed case of WCS in a postoperative ICU patient, elucidating its clinical presentation, underlying mechanisms, and the pivotal role of prompt management in achieving a favourable outcome. By highlighting this rare entity, we aim to enhance awareness and preparedness among ICU practitioners managing patients on high-dose opioid therapy.

CASE DESCRIPTION

A 49-year-old female with a complex medical background was admitted to the ICU at Monmouth Medical Center following an emergent high-risk incision and drainage (I&D) procedure. Her past medical history included cervical cancer diagnosed four years prior (treated with chemotherapy and pelvic radiation), hypertension managed with lisinopril (10 mg daily), and a sigmoidectomy for complicated diverticulitis performed 6 weeks earlier. She presented to the emergency department with a 4-day history of progressive left-sided facial pain, swelling, and dysphagia, initially attributed to a dental abscess. A computed tomography (CT) scan of the neck revealed a 4 × 3 cm abscess extending from the submandibular space into the deep cervical fascia, with evidence of soft tissue oedema and gas formation, raising concerns for impending airway compromise. Urgent surgical intervention was deemed necessary.

The I&D was performed under general anaesthesia with endotracheal intubation. Intraoperatively, the patient received an intravenous (IV) bolus of fentanyl (100 µg) for analgesia, followed by a continuous fentanyl infusion (50 µg/hour) initiated in the operating room and continued postoperatively to manage severe pain. Additional intraoperative medications included propofol (2 mg/kg IV for induction) and rocuronium (0.6 mg/kg IV for paralysis), with anaesthesia maintained using sevoflurane (1.5% end-tidal concentration). The procedure lasted 90 minutes, during which purulent material was drained, and a surgical drain was placed. Postoperatively, she was transferred to the ICU intubated due to persistent facial swelling, potential airway oedema, and the need for close respiratory monitoring.

On ICU arrival (postoperative hour 0), her vital signs were stable: blood pressure 135/85 mmHg, heart rate 92 beats/min, respiratory rate 16 breaths/min (ventilator-assisted), and oxygen saturation 98% on a fraction of inspired oxygen (FiO_2) of 40%. She was placed on pressure-regulated volume control (PRVC) mode with the following settings: target tidal volume (VT) 450 ml (adjusted to 6 ml/kg ideal body weight), set respiratory rate 14 breaths/min, positive end-expiratory pressure (PEEP) 5 cmH_2O, and inspiratory time 0.8 seconds. Peak airway pressure (Ppeak) was 20 cmH_2O, and plateau pressure (Pplat) was 16 cmH_2O, indicating normal lung and chest wall compliance at baseline. Physical examination revealed significant left-sided facial oedema and erythema, with the surgical drain in situ producing minimal serosanguinous output. No muscle rigidity or respiratory distress was noted at this stage. Sedation was maintained with the fentanyl infusion, and no additional sedatives were administered initially to avoid oversedation.

On postoperative day 1, approximately 8 hours after initiating the fentanyl infusion (total dose administered ~400 µg), the patient exhibited abrupt clinical deterioration.

Her respiratory rate increased to 28 breaths/min despite ventilatory support, oxygen saturation dropped to 88% even after increasing FiO_2 to 60%, and Ppeak escalated to 35 cmH_2O, while Pplat remained relatively stable at 18 cmH_2O. This discrepancy suggested reduced chest wall compliance rather than intrinsic lung pathology. Ventilator waveforms showed a "shark fin" pattern on the expiratory flow curve, indicative of airflow limitation, and patient-ventilator dyssynchrony emerged, with the patient triggering additional breaths against the set rate. Physical examination revealed marked generalized muscle rigidity, most pronounced in the chest wall and abdominal muscles, rendering the thorax stiff and "wooden" to palpation. The rigidity extended mildly to the upper extremities, though the lower limbs remained less affected. Her temperature was 37.8°C, with no hyperthermia, diaphoresis, or clonus suggestive of serotonin syndrome or neuroleptic malignant syndrome (NMS). Neurologic assessment showed no focal deficits, and pupils were 3 mm and reactive bilaterally.

Laboratory studies were promptly obtained: creatine kinase was 120 U/l (normal range: 38–174 U/l), ruling out significant muscle breakdown or rhabdomyolysis; white blood cell count was 14,000/µl (consistent with postoperative inflammation); and serum lactate was 1.8 mmol/l (mildly elevated but not indicative of severe hypoxia). Arterial blood gas (ABG) analysis on FiO_2 60% revealed respiratory acidosis: pH 7.28, $PaCO_2$ 58 mmHg, PaO_2 72 mmHg, and bicarbonate 26 mEq/l, confirming hypoventilation secondary to thoracic rigidity rather than intrinsic pulmonary disease. Chest X-ray showed no evidence of pneumothorax, pulmonary oedema, or atelectasis, further supporting an extrapulmonary aetiology. Given the temporal association with fentanyl administration and the clinical trial of rigidity, respiratory distress, and impaired ventilatory mechanics, WCS was suspected. The fentanyl infusion was immediately discontinued, and naloxone (2 mg IV bolus) was administered to reverse opioid effects, with a second dose (1 mg IV) prepared but not required. Concurrently, ventilator settings were adjusted to mitigate dyssynchrony and support respiration: the mode was switched to pressure control ventilation (PCV) with an inspiratory pressure of 25 cmH_2O above PEEP (5 cmH_2O), targeting a VT of 400–450 ml, and the respiratory rate was increased to 18 breaths/min to offload the patient's work of breathing. Dexmedetomidine was initiated at 0.5 µg/kg/h (without a loading dose to avoid hemodynamic instability) for sedation, providing anxiolysis and analgesia without further opioid exposure.

Within 30 minutes of intervention, clinical improvement was evident. Muscle rigidity began to subside, starting with the chest and abdomen, and Ppeak decreased to 22 cmH_2O, aligning more closely with Pplat (17 cmH_2O), indicating restored chest wall compliance. Oxygen saturation improved to 95% on FiO_2 50%, and spontaneous respiratory efforts became more coordinated with the ventilator. Repeat ABG at 1-hour post-intervention showed pH 7.34, $PaCO_2$ 48 mmHg, and PaO_2 85 mmHg, reflecting resolving acidosis. Over the next 4 hours, rigidity fully resolved, ventilatory parameters normalized (Ppeak 20 cmH_2O, oxygen saturation 98% on FiO_2 40%), and the patient became more alert, following commands. The ventilator was transitioned to pressure support (PS) mode with PS set at 10 cmH_2O above PEEP (5 cmH_2O) and a backup rate of 10 breaths/min as a weaning trial. She tolerated this well, maintaining a spontaneous VT of 400–450 ml and a respiratory rate of 14–16 breaths/min. On postoperative day 2 (March 18, 2025), after confirming airway patency (cuff leak test positive) and resolution of facial swelling, she was extubated successfully to nasal cannula (2 l/min). Her subsequent ICU course was uneventful, with pain managed using IV acetaminophen (1 g every 6 hours) and oral tramadol (50 mg as needed). She was discharged to the medical floor on postoperative day (March 21, 2025) for continued recovery.

DISCUSSION

WCS is a rare yet potentially lethal complication of synthetic opioid administration, most notably fentanyl, alfentanil, and sufentanil. Although its precise incidence remains poorly defined—likely due to under recognition and overlap with more familiar opioid-related effects such as respiratory depression—it is increasingly reported in perioperative and critical care settings involving rapid bolus dosing or prolonged infusions[3]. Our case of a 49-year-old female developing WCS approximately 8 hours after initiating a fentanyl infusion (50 µg/hour) following an emergent I&D procedure exemplifies the syndrome's insidious onset and profound impact on ventilatory mechanics. The hallmark of WCS—generalized skeletal muscle rigidity, predominantly affecting the chest wall, diaphragm, and abdominal muscles—leads to decreased thoracic compliance, impaired ventilation, and, if unrecognized, acute respiratory failure. This discussion explores the pathophysiology, clinical presentation, diagnostic challenges, and management strategies illuminated by this case, while highlighting its implications for ICU practice.

The mechanisms underlying WCS are multifactorial and not fully elucidated, reflecting the complexity of opioid interactions within the CNS. Fentanyl, a highly potent mu-opioid receptor agonist, is implicated due to its rapid CNS penetration—driven by its lipophilicity—and its ability to induce hyperactivity in motor and respiratory regulatory regions, such as the basal ganglia and brainstem[4]. Preclinical studies suggest that excessive mu-receptor stimulation disrupts the balance between excitatory and inhibitory neurotransmitters, notably dopamine and gamma-aminobutyric acid, resulting in extrapyramidal rigidity akin to that seen in Parkinsonism or NMS[1]. In our patient, the continuous fentanyl infusion likely led to drug accumulation, with a total dose of approximately 400 mcg over 8 hours exceeding the threshold for WCS onset. This may have been exacerbated by her postoperative inflammatory state, which could alter opioid pharmacokinetics, though no hepatic or renal dysfunction was evident to suggest impaired clearance.

Emerging evidence also points to a potential role for serotonin dysregulation, given anecdotal overlaps with serotonin syndrome in some WCS cases[2]. However, our patient lacked hyperthermia, clonus, or autonomic instability, distinguishing WCS from these entities. The rapid resolution of symptoms following naloxone administration further supports a direct opioid-mediated effect, likely via reversal of mu-receptor overstimulation. The exact contribution of fentanyl's metabolites (e.g., norfentanyl) remains unclear, though their minimal activity at mu-receptors suggests the parent compound is the primary driver[4]. Future research, including neuroimaging and neurotransmitter profiling, is needed to clarify these mechanisms and identify predisposing factors, such as genetic polymorphisms in opioid receptors or concurrent medications.

WCS typically manifests acutely with profound muscle stiffness, respiratory distress, and reduced chest wall compliance, often within hours of opioid initiation, as seen in our case at the 8-hour mark. The rigidity predominantly affects thoracic and abdominal muscles, though it may extend to the extremities, compromising ventilatory mechanics more than diaphragmatic function alone. In our patient, initial stability on PRVC mode (Ppeak 20 cmH_2O, target VT 450 ml) gave way to a sudden increase in Ppeak to 35 cmH_2O, with a stable Pplat (18 cmH_2O), reflecting extrapulmonary restriction rather than parenchymal lung disease. Ventilator waveforms displaying a "shark fin" expiratory pattern and patient-ventilator dyssynchrony further corroborated the diagnosis, highlighting the mechanical burden imposed by WCS.

Differentiating WCS from other conditions is critical yet challenging in the ICU. Unlike opioid-induced respiratory depression, which primarily stems from medullary suppression and responds to ventilatory support, WCS involves a peripheral musculoskeletal component that resists standard ventilation strategies[5]. NMS features hyperthermia, autonomic instability, and elevated creatine kinase—absent in our case (120 U/l)—while malignant hyperthermia includes hypermetabolism and a clear anaesthetic trigger (e.g., succinylcholine), not applicable here[6]. Laryngospasm or tetanus were excluded by the absence of stridor or neurological prodrome, respectively. The temporal link to fentanyl, the triad of rigidity, hypoventilation, and respiratory acidosis (pH 7.28, $PaCO_2$ 58 mmHg), and the rapid response to naloxone solidified WCS as the diagnosis, underscoring the value of clinical suspicion in the absence of standardized criteria.

Effective management of WCS hinges on swift recognition and a multifaceted approach. Immediate cessation of the causative opioid is paramount, as continued administration exacerbates rigidity and respiratory compromise. In our case, discontinuing the fentanyl infusion halted further progression, while naloxone (2 mg IV) competitively antagonized mu-receptors, initiating symptom reversal within 30 minutes. Titration is key to avoid precipitating withdrawal, though our patient required only a single dose, reflecting the moderate total fentanyl exposure (400 µg). Switching to PVC with an inspiratory pressure of 25 cmH_2O above PEEP (5 cmH_2O) addressed the acute ventilatory challenge by ensuring adequate tidal volumes (400–450 ml) despite elevated airway pressures, bridging the gap until rigidity subsided.

Non-opioid sedation is a critical adjunct to maintain patient comfort without risking recurrence. Dexmedetomidine (0.5 µg/kg/h), an alpha-2 agonist with minimal respiratory depression, proved effective in our patient, facilitating a smooth transition to PS mode (PS 10 cmH_2O above PEEP) for weaning[7]. The rapid normalization of ventilatory parameters (Ppeak 20 cmH_2O, oxygen saturation 98% on FiO_2 40%) and successful extubation on postoperative day 2 highlight the efficacy of this strategy. In contrast, benzodiazepines or propofol might have prolonged ventilator dependence, while additional opioids would have been contraindicated. Mechanical ventilation, while necessary in severe cases, should be tailored to the patient's improving mechanics, as demonstrated by our shift from PCV to PS before extubation.

Fentanyl's widespread use in the ICU for analgesia and sedation—owing to its rapid onset and hemodynamic stability—necessitates heightened vigilance for WCS, particularly in patients receiving infusions exceeding 8 hours or those with inflammatory states that may enhance opioid sensitivity. Our case underscores early warning signs: rising Ppeak, unexplained hypoventilation, and subtle rigidity on examination. Ventilator data, such as the Ppeak-Pplat gradient and waveform abnormalities, offer objective clues that should prompt immediate evaluation, especially in patients on PRVC or similar modes where compliance changes are readily apparent.

Preventive strategies include minimizing bolus doses (e.g., avoiding >100 µg IV as in our case), titrating infusions conservatively, and integrating multimodal analgesia—such as ketamine, acetaminophen, or regional blocks—to reduce opioid reliance[8]. Regular reassessment of sedation and analgesia needs, guided by tools like the RAAS or pain scores, can pre-empt accumulation. Education of ICU staff on WCS recognition—emphasizing its distinction from respiratory depression or NMS—is equally vital to reduce diagnostic delays. Simulation-based training or case discussions could enhance preparedness, given the syndrome's rarity.

This case also raises questions for future investigation: Are certain patient populations (e.g., those with prior opioid exposure or CNS comorbidities) at higher risk? Can ventilator analytics predict WCS onset? Standardized protocols for diagnosis (e.g., rigidity scoring) and management (e.g., naloxone dosing algorithms) could streamline care and improve outcomes, warranting multicentre studies to address these gaps.

CONCLUSION

WCS remains an underrecognized yet formidable complication of fentanyl therapy in the ICU, as illustrated by this case of a 49-year-old female who developed profound

muscle rigidity and respiratory compromise 8 hours after initiating a fentanyl infusion. This rare entity, characterized by decreased chest wall compliance and acute ventilatory failure, poses a silent threat to critically ill patients reliant on opioids for pain control, particularly in the postoperative period. Our patient's rapid deterioration—evidenced by escalating Ppeaks on PRVC mode and respiratory acidosis—was swiftly reversed through prompt discontinuation of fentanyl, administration of naloxone, and transition to dexmedetomidine, culminating in successful extubation on postoperative day 2 following a PS weaning trial. This outcome underscores the critical interplay of clinician awareness, early diagnosis, and decisive intervention in averting life-threatening consequences.

The case highlights the need for heightened vigilance during opioid administration, especially with continuous infusions exceeding 8 hours or in patients with inflammatory states that may amplify fentanyl's effects. Ventilator parameters, such as rising Ppeak and waveform abnormalities, serve as early harbingers of WCS, necessitating their routine monitoring in sedated patients. Alternatives to high-dose opioids—multimodal analgesia and non-opioid sedatives like dexmedetomidine—offer viable strategies to mitigate risk, while staff education can bridge the gap in recognition and response. However, the lack of standardized diagnostic criteria and management protocols remains a barrier to consistent care, calling for further research to define WCS's epidemiology, refine its pathophysiology, and establish evidence-based guidelines.

Ultimately, enhancing ICU preparedness for WCS can improve patient safety and reduce the burden of opioid-related complications in critical care. This case serves as both a cautionary tale and a roadmap for navigating this rare but reversible syndrome, urging clinicians to balance the benefits of fentanyl with its potential perils and to act swiftly when the wooden chest emerges.

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