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Fentanyl-induced chest wall rigidity in critically ill patients: A report of two cases

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ABSTRACT:

Introduction: Fentanyl is widely used for analgesia and sedation in critically ill patients. Fentanyl-induced chest wall rigidity, also called wooden chest syndrome, is an uncommon but potentially life-threatening adverse effect that can markedly impair ventilation. Most reports involve pediatric or peri-anesthetic settings, whereas adult intensive care unit (ICU) presentations remain unrecognized.

Case presentation: We describe two adults in whom fentanyl-induced chest wall rigidity was considered during mechanical ventilation. A 76-year-old man with severe chronic obstructive pulmonary disease and end-stage kidney disease developed abrupt patient-ventilator dyssynchrony and markedly reduced delivered tidal volumes after several days of fentanyl infusion, when neuromuscular blockade was withdrawn. No alternative mechanical or pulmonary cause was identified at the bedside. Ventilation improved after fentanyl discontinuation and naloxone administration. A 75-year-old man with myelodysplastic syndrome and cirrhosis developed similar dyssynchrony after cisatracurium discontinuation while receiving fentanyl. Respiratory mechanics improved after fentanyl was stopped and neuromuscular blockade was resumed.

Conclusions: In mechanically ventilated patients receiving fentanyl, otherwise unexplained patient-ventilator dyssynchrony, elevated airway pressure, or a sudden reduction in delivered tidal volume should prompt consideration of fentanyl-induced chest wall rigidity. Immediate bedside assessment should exclude endotracheal tube or circuit problems, mucus plugging, pneumothorax, and bronchospasm. Prompt fentanyl discontinuation, titrated naloxone, and temporary neuromuscular blockade when ventilation remains inadequate may rapidly restore effective ventilation.

Keywords: Fentanyl; Chest wall rigidity; Wooden chest syndrome; Intensive care unit; Mechanical ventilation; Patient-ventilator dyssynchrony

Fentanyl-induced chest wall rigidity in critically ill patients: A report of two cases

Identifying the "Hidden" Red Flags



Sudden Ventilator Dyssynchrony

Look for unexplained drops in tidal volume or sudden spikes in peak airway pressure.



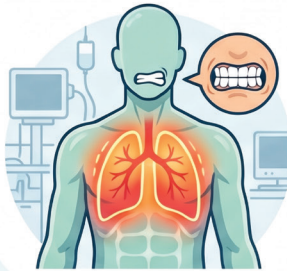
The "Wooden Chest" Effect

Observe for physical rigidity in the thoracic and abdominal muscles during machine-triggered breaths.



The Masking Phenomenon

Rigidity often becomes apparent only after neuromuscular blockade (paralytics) is discontinued or withdrawn.



The 3-Step Bedside Action Plan



Exclude Common Causes

Rule out tube obstruction, mucus plugging, pneumothorax, or ventilator circuit leaks first.



Stop and Reverse

Discontinue fentanyl immediately and administer titrated intravenous naloxone to restore chest compliance.



Restore Stability

Use temporary neuromuscular blockade if ventilation remains inadequate or if rigidity recurs.

Clinical Manifestations & Diagnostic Response

Manifestation	Clinical Observation
Respiratory Mechanics 	Markedly reduced tidal volume (often <100mL)
Physical Signs 	Thoracoabdominal rigidity / Jaw clenching
Diagnostic Response 	Rapid improvement (within 3–5 mins) after Naloxone



Summarized infographic: Sanlee, et al. Clin Crit Care 2026; 34: e260024

INTRODUCTION

Fentanyl is a potent synthetic opioid frequently used for analgesia and sedation during procedures, surgery, and mechanical ventilation. Its recognized adverse effects include respiratory depression, nausea, and hemodynamic instability. A less common but serious complication is fentanyl-induced chest wall rigidity, in which increased thoracic and abdominal muscle tone reduces chest wall compliance and can cause severe ventilatory failure [1-3].

Fentanyl-induced rigidity has been reported most often in pediatric and anesthesia settings. Although rapid intravenous administration and high cumulative doses are recognized risk factors, the reaction can also occur after analgesic doses and during prolonged infusions [3,4,11]. Proposed mechanisms include central μ -opioid receptor activation and altered dopaminergic and noradrenergic signaling within the brainstem and basal ganglia, leading to increased muscle tone in the chest wall and diaphragm [5,6]. Management includes ventilatory support, fentanyl discontinuation, naloxone, and, when required, short-acting neuromuscular blockade [7,8].

These cases add an ICU-specific perspective by illustrating that fentanyl-induced rigidity may become clinically apparent only after withdrawal of neuromuscular blockade. They also demonstrate how ventilator waveforms and a structured exclusion of common causes of dyssynchrony can support timely diagnosis and management in mechanically ventilated adults.

KEY MESSAGES:

- Fentanyl-induced chest wall rigidity should be considered in otherwise unexplained patient-ventilator dyssynchrony with reduced tidal volume.
- Chest wall rigidity may become apparent after withdrawal of neuromuscular blockade during prolonged fentanyl infusion.
- Prompt fentanyl discontinuation and naloxone or temporary neuromuscular blockade may rapidly restore effective ventilation.
- Careful assessment to exclude common mechanical and pulmonary causes is essential before attributing ventilator abnormalities to fentanyl-induced rigidity.

CASE PRESENTATION

Case 1

A 76-year-old Thai man with chronic obstructive pulmonary disease (COPD; GOLD group E, FEV1/FVC 0.51, and FEV1 29% predicted), end-stage kidney disease treated with continuous ambulatory peritoneal dialysis, and type 2 diabetes mellitus presented with worsening cough, increased whitish sputum, and progressive dyspnea. In the emergency department, he was tachycardic and tachypneic. Influenza

A polymerase chain reaction testing was positive, the sputum culture grew *Escherichia coli* susceptible to piperacillin/tazobactam and resistant to ceftriaxone, and chest radiography showed no new pulmonary infiltrate. The working diagnosis was an acute COPD exacerbation with influenza A infection and *E. coli* tracheobronchitis, complicated by respiratory failure.

The patient was intubated on September 14, 2024. Piperacillin/tazobactam and oseltamivir were administered for 10 days. Severe airflow obstruction and air trapping were managed with pressure-controlled ventilation, inhaled ipratropium/fenoterol and budesonide, and positive end-expiratory pressure titration guided by electrical impedance tomography. Continuous fentanyl, midazolam (2 mg/h), and cisatracurium (6 mg/h) were administered; the fentanyl exposure is summarized in Table 1.

After several days of fentanyl infusion, cisatracurium was discontinued. The patient then developed severe patient-ventilator dyssynchrony, with delivered tidal volumes

varying from 100 to 500 mL. During machine-triggered breaths, delivered tidal volume was minimal, whereas patient-triggered breaths generated larger volumes (Figure 1). Blood pressure was 113/67 mmHg, and oxygen saturation was 96%. Endotracheal tube patency was confirmed by passage of a suction catheter, which yielded no secretions. Bilateral breath sounds were clear. Bedside assessment found no clinical evidence of circuit disconnection or leak, pneumothorax, severe bronchospasm, or inadequate sedation. Abdominal rigidity was observed during machine-triggered breaths. Fentanyl-induced chest wall rigidity was therefore suspected. Fentanyl was discontinued, and naloxone 0.04 mg IV was administered. Delivered tidal volume improved within three minutes, and patient-ventilator synchrony was restored by four minutes. (Figures 2-3). Cisatracurium was resumed temporarily because recurrent rigidity was considered possible after prolonged fentanyl exposure. No further rigidity was observed, and the patient was extubated on September 27, 2024.

Table 1. Chronology of fentanyl administration and clinically relevant events in Case 1.

Day of fentanyl infusion	Time	Fentanyl dose	Notes/Events
D1	02:00	50 mcg x 2 doses (purge)	Initial dose after intubation
	02:00	50 mcg/hr x 11 hours	Continuous infusion
	13:10	25 mcg (purge)	Continued respiratory distress
	13:10	25 mcg/hr x 2 hours	
	15:40	25 mcg (purge)	Further deterioration
	15:40	50 mcg/hr x 5 hours	
	20:30	50 mcg x 2 doses (purge)	
	20:30	100 mcg/hr x 4 hours	
D2		100 mcg/hr x 24 hours	
D3		100 mcg/hr x 24 hours	Transferred to ICU
D4		100 mcg/hr x 24 hours	
D5	03:00	75 mcg/hr x 2.5 hours	Fentanyl dose reduction begins
	05:30	50 mcg/hr x 2.5 hours	
	08:00	25 mcg/hr x 7 hours	
	15:00	75 mcg/hr x 2 hours	Fentanyl discontinued at 17:00
	17:00	Off fentanyl	Naloxone administered with improvement

The patient received a continuous fentanyl infusion for 108 hours, with a cumulative infusion dose of 9,087.5 µg and a time-weighted mean infusion rate of 84.1 µg/h. An additional 250 µg was documented as purge or bolus doses.

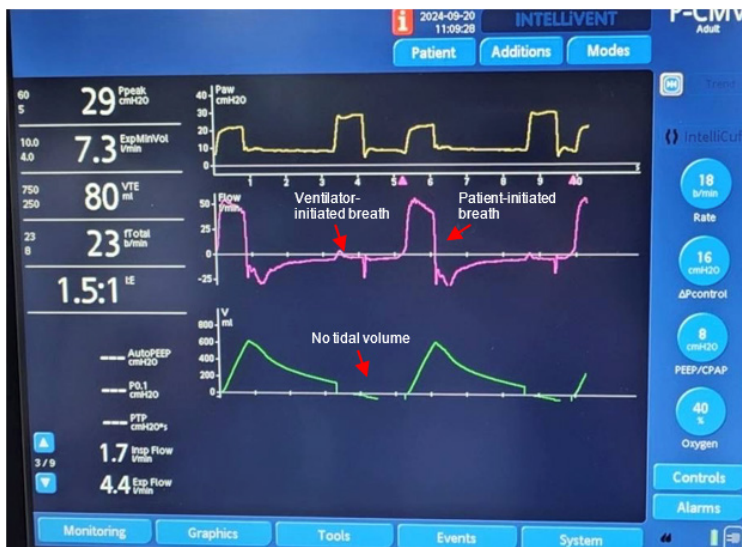


Figure 1. Ventilator waveform before naloxone administration in Case 1, showing severe patient-ventilator dyssynchrony and markedly reduced delivered tidal volume during machine-triggered compared with patient-triggered breaths.

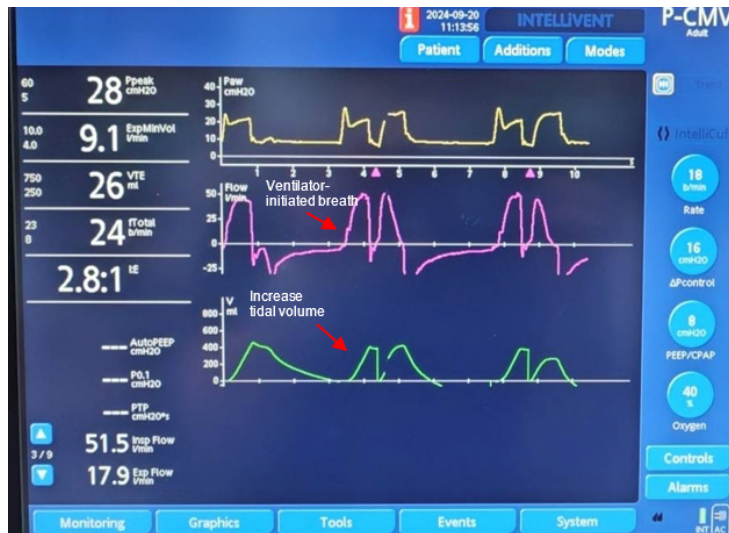


Figure 2. Ventilator waveform three minutes after naloxone administration in Case 1, showing increased delivered tidal volume and partial resolution of patient-ventilator dyssynchrony.



Figure 3. Ventilator waveform four minutes after naloxone administration in Case 1, showing restoration of delivered tidal volume and complete resolution of patient-ventilator dyssynchrony.

Case 2

A 75-year-old Thai man with myelodysplastic syndrome with refractory cytopenia and multilineage dysplasia, alcoholic cirrhosis, and type 2 diabetes mellitus presented on September 24, 2024, with symptoms of an upper respiratory tract infection, one day of small-volume hemoptysis, and worsening dyspnea. On arrival in the emergency department, he was unresponsive and pulseless. The initial electrocardiographic rhythm was pulseless electrical activity. Return of spontaneous circulation was achieved after five minutes of cardiopulmonary resuscitation. After resuscitation, he opened his eyes but did not follow commands. Chest radiography showed multiple alveolar consolidations in the right middle and lower lobes. Computed tomographic angiography of the thoracic aorta showed no dilated bronchial arteries or pulmonary pseudoaneurysm. Tracheal aspirate culture grew *Haemophilus influenzae*. Ceftriaxone and levofloxacin were administered for 14 days. Norepinephrine was required from September 24 to 30, with a maximum dose of 0.22 µg/kg/min. Sedation and paralysis included fentanyl 75 µg/h, cisatracurium 8 mg/h, and midazolam 4 mg/h; intravenous hydrocortisone was also administered.

On September 27, cisatracurium was discontinued. The patient subsequently developed marked patient-ventilator dyssynchrony and variable delivered tidal volumes. Endotracheal tube position and patency were confirmed, no obstructing airway secretions were found, and dyssynchrony persisted despite sedation. The ventilator waveform showed markedly reduced tidal volume during machine-triggered breaths (Figure 4). Fentanyl was discontinued, cisatracurium was resumed, and propofol was added. Patient-ventilator synchrony was restored, and the PaO₂/FiO₂ ratio improved to 220. The patient was extubated on October 9, 2024.

The temporal association with continuous fentanyl exposure and the emergence of dyssynchrony after cisatracurium withdrawal raised strong suspicion for fentanyl-induced chest wall rigidity. However, fentanyl discontinuation, re-initiation of cisatracurium, and addition of propofol occurred concurrently, and naloxone was not administered. The diagnosis in Case 2 was therefore considered probable rather than definitively confirmed.

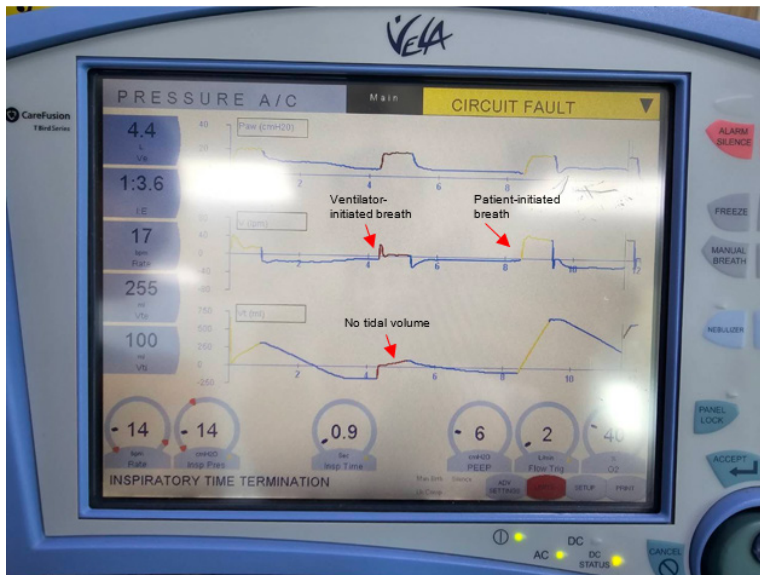


Figure 4. Ventilator waveform in Case 2, demonstrating patient-ventilator dyssynchrony with markedly reduced tidal volume during machine-triggered breaths. The abnormality became clinically apparent after withdrawal of neuromuscular blockade and was compatible with probable fentanyl-induced chest wall rigidity.

Table 2. Suggested bedside approach to suspected fentanyl-induced chest wall rigidity.

Step	Action
1. Recognize	Suspect the condition when a patient receiving fentanyl develops otherwise unexplained dyssynchrony, reduced delivered tidal volume, elevated airway pressure, or thoracoabdominal rigidity.
2. Exclude common causes	Check endotracheal tube position and patency, ventilator circuit integrity, secretions or mucus plugging, pneumothorax, severe bronchospasm, ventilator settings, and depth of sedation.
3. Stop the trigger	Discontinue fentanyl and provide immediate ventilatory support.
4. Reverse opioid effect	Administer titrated intravenous naloxone when clinically appropriate and reassess respiratory mechanics promptly.
5. Restore ventilation	Use temporary neuromuscular blockade if adequate ventilation cannot be restored or recurrent rigidity is anticipated after prolonged fentanyl exposure.

DISCUSSION

Fentanyl-induced chest wall rigidity is a rare but potentially life-threatening adverse effect characterized by increased thoracic and abdominal muscle tone, reduced chest wall compliance, and impaired ventilation [1-4]. Adult ICU cases are particularly difficult to recognize because ventilator dyssynchrony has many more common causes and because deep sedation or neuromuscular blockade may obscure the physical findings.

Experimental data suggest that central μ -opioid receptor activation in the brainstem and basal ganglia, including the nucleus raphe pontis and caudate nucleus, contributes to opioid-induced skeletal muscle rigidity [5,6]. Fentanyl rapidly crosses the blood-brain barrier because of its high lipophilicity. The resulting increase in thoracoabdominal muscle tone can limit inspiratory expansion, reduce delivered tidal volume, and raise airway pressure [6-8]. Rapid administration and higher cumulative exposure increase risk, but reported cases after very low doses indicate that the reaction is not strictly dose dependent [4,11,12].

In Case 1, rigidity became apparent after cumulative fentanyl exposure from repeated documented bolus or purge doses and continuous infusion. Machine-triggered breaths produced minimal delivered tidal volume, whereas patient-triggered breaths generated larger volumes. The

rapid improvement after low-dose naloxone provided strong pharmacologic support for the diagnosis. In Case 2, the same ventilator pattern emerged after cisatracurium withdrawal, suggesting that neuromuscular blockade had masked the clinical manifestation. This ICU-specific phenomenon may delay recognition until the paralytic effect is removed.

In mechanically ventilated patients, fentanyl-induced chest wall rigidity typically presents as a sudden spike in peak airway pressure, premature termination of breaths, markedly reduced tidal volume, or abdominal rigidity, leading to severe patient-ventilator dyssynchrony despite adequate sedation [4,10-12]. Before attributing these findings to fentanyl, clinicians must systematically exclude common alternative causes, including endotracheal tube obstruction, circuit leaks, mucus plugging, pneumothorax, and severe bronchospasm. A practical bedside approach to its recognition and management is summarized in Table 2.

Compared with previously reported adult ICU cases, both patients in this report received prolonged fentanyl infusions rather than a single procedural bolus. Similar to the cases reported by Ming et al. and Rosal et al., the principal manifestations were reduced delivered tidal volume and patient-ventilator dyssynchrony [9,10]. Previously reported adult cases and case series are summarized in Table 3, demonstrating that fentanyl-induced chest wall

Table 3. Previously reported adult cases and case series of fentanyl-induced chest wall rigidity.

Author/Year	Clinical setting	Fentanyl exposure	Onset	Key features	Management	Outcome
Çoruh et al., 2013 [3]	76-year-old man; bronchoscopy sedation	Total 3.6 µg/kg IV; rapid 1.4 µg/kg bolus before event	2 min after bolus	Jaw clenching, chest wall rigidity, desaturation	Bag-mask ventilation; naloxone 0.2 mg IV	Rapid recovery
Ham et al., 2016 [7]	76-year-old woman; acute COPD exacerbation with respiratory failure.	Fentanyl 50 µg IV bolus	Immediate	Elevated airway pressure, reduced chest wall movement, desaturation	Neuromuscular blocker	Recovery
McGreevy et al., 2019 [8]	46-year-old man; ICU exposure with serotonin syndrome	Continuous IV fentanyl infusion	During ICU treatment	Chest wall rigidity and respiratory compromise	Fentanyl discontinued; supportive therapy	Clinical improvement
Ming et al., 2019 [9]	80-year-old woman; ICU respiratory failure	Infusion up to 180 µg/h; previous tolerance of 50 µg/h	During prolonged infusion	Minimal tidal volume and very high airway pressure	Fentanyl withdrawn; neuromuscular blocker	Respiratory mechanics improved
Rosal et al., 2021 [10]	61-year-old woman; ARDS on mechanical ventilation	Continuous infusion up to 300 µg/h; cumulative exposure approximately 11 mg	During ICU treatment	Hypoxemia, rigid abdomen/chest, ventilator dyssynchrony	Fentanyl discontinued; sedation changed	Ventilation improved
Tammen et al., 2023 [11]	42 critically ill adults; median age 71 years	Rate: 100 µg/h (IQR, 100–150) Cumulative dose: 3119 (1354–8653) µg Dose/kg: 29.6 (13–84) µg/kg	Median fentanyl exposure 34.38 h (IQR 11.2–53.3)	Dyssynchrony, thoracic/abdominal rigidity, high airway pressure	Naloxone and/or cisatracurium ; fentanyl discontinued	Ventilator compliance improved in most patients
Zoorob et al., 2023 [12]	80-year-old patient; procedural sedation	Fentanyl 25 µg IV	Immediately	Inability to ventilate due to rigidity	Naloxone and ventilation	Rapid recovery
McElrath et al., 2024 [13]	52-year-old woman; outpatient cervical facet procedure	Fentanyl 50 µg IV × 2	2 min after second bolus	Apnea, jaw clenching, limb rigidity, desaturation	Naloxone 0.4 mg IV	Rapid recovery
Devarashetty et al., 2025 [14]	49-year-old woman; post-operative ICU ventilation	Fentanyl infusion 50 µg/h; approximately 400 µg total	Shortly after infusion began	Chest wall rigidity, tachypnea, increased peak airway pressure, respiratory acidosis	Naloxone 2 mg IV; non-opioid sedation; ventilatory support	Recovery

Abbreviations: ARDS: Acute respiratory distress syndrome; COPD: Chronic obstructive pulmonary disease; ICU: Intensive care unit; IV: Intravenous

rigidity can occur across a wide range of clinical settings and fentanyl doses. A distinguishing feature was the emergence of the abnormal ventilator pattern after neuromuscular blockade withdrawal. Case 1 improved rapidly after naloxone, whereas Case 2 was managed with fentanyl discontinuation and temporary re-initiation of neuromuscular blockade.

Naloxone is commonly used to reverse opioid-induced rigidity, while neuromuscular blocking agents can rapidly restore ventilation when naloxone is insufficient, contraindicated, or not immediately effective [3,7,11]. After prolonged fentanyl infusion, recurrent rigidity may occur if the opioid effect outlasts naloxone. Temporary neuromuscular blockade can therefore serve as a bridge until the clinical opioid effect has resolved. In Case 1, cisatracurium was resumed for approximately 24 hours; in Case 2, it was resumed until ventilation remained stable after fentanyl discontinuation.

Prevention is based on cautious fentanyl titration, avoidance of unnecessary rapid boluses, and close monitoring of respiratory mechanics during prolonged infusions. Clinicians should remain alert when ventilator dyssynchrony becomes apparent after neuromuscular blockade is reduced or stopped.

CONCLUSION

Fentanyl-induced chest wall rigidity should be considered when a mechanically ventilated patient receiving fentanyl develops otherwise unexplained patient-ventilator dyssynchrony, elevated airway pressure, or a sudden reduction in delivered tidal volume. Immediate bedside assessment should exclude endotracheal tube or ventilator circuit problems, mucus plugging, pneumothorax, and bronchospasm. Prompt fentanyl discontinuation, naloxone, and, when ventilation remains inadequate or recurrent rigidity is anticipated, temporary neuromuscular blockade may restore effective ventilation. Slow fentanyl titration and avoidance of unnecessary rapid boluses may reduce the risk of this complication.

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Informed Consent Statement: Written informed consent was obtained from both patients for the publication of their clinical data and ventilator images.

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