Takotsubo-like myocardial stunning and flash pulmonary edema after ketamine sedation in a prone pain procedure

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BACKGROUND

Ketamine is used for monitored anesthesia care (MAC) during interventional pain procedures, specifically for patients who do not tolerate these procedures well. Its sympathomimetic surge can precipitate acute pulmonary edema and inhibit alveolar fluid clearance. Prone sedation separately risks negative-pressure pulmonary edema (NPPE).

CASE & COURSE

A 53-year-old woman (BMI 35.4, ASA III) with hypertension, non-obstructive coronary artery disease, COPD, and obstructive sleep apnea treated with CPAP presented for a left L4-L5 and L5-S1 transforaminal epidural steroid injection (TFESI). An echocardiogram six months earlier had shown a normal ejection fraction of 58% without diastolic dysfunction.

She was positioned prone and sedated with midazolam, diphenhydramine, glycopyrrolate, and ondansetron, followed by ketamine 50 mg (0.5 mg/kg) and dexmedetomidine.

Five minutes after the ketamine bolus, while the needle was still extraforaminal at L4-L5 and before any contrast or corticosteroid had been injected, she began coughing and abruptly desaturated. She then produced about one litre of pink, frothy secretions, and her blood pressure rose to 239/130 mmHg with a heart rate of 144. The procedure was aborted, and she was turned supine, intubated, and transferred to the ICU. An arterial blood gas on 100% oxygen showed a pH below 7.00 and a $PaCO_2$ above 125 mmHg. She had received only 400 mL of crystalloid.

A same-day echocardiogram showed that her ejection fraction had fallen to 13%, with apical and midventricular akinesis extending beyond a single coronary territory. An NT-proBNP drawn 105 minutes after onset was 109 pg/mL.

METHODS

Single-patient retrospective case report. Competing etiologies were assessed against the documented timeline, hemodynamics, and imaging (Table 1); adverse-drug-reaction probability was scored using the Naranjo algorithm. Ketamine and dexmedetomidine were given intravenously at sedative doses for MAC (off-label use); all other agents were used within labeled indications.

Five minutes after a sedative-dose ketamine bolus, her ejection fraction fell from 58% to 13% — and recovered fully within 72 hours.

The sedation, not the injection, precipitated this event.

EJECTION FRACTION TRAJECTORY



Baseline · 6 months prior

Day 0 · troponin peak 1.06 ng/mL

Day 3 · cath non-obstructive; LVEDP 24, PCWP 15, CI 2.74. Extubated day 4, discharged day 9.

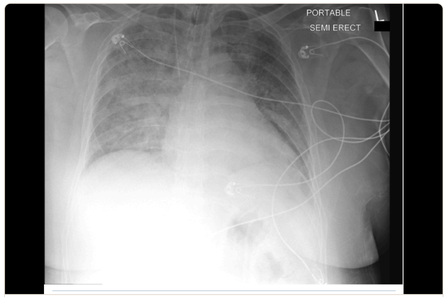


Figure 1. Portable semi-erect chest radiograph shortly after intubation, showing extensive bilateral confluent alveolar opacities with perihilar vascular congestion and a magnified cardiomediastinal silhouette.

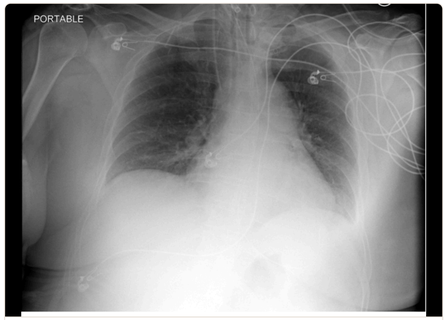


Figure 2. Portable chest radiograph on hospital day four, showing marked interval clearance with only mild residual bilateral infiltrates.

TABLE 1 · DIFFERENTIAL ASSESSMENT

ETIOLOGY AND DISCRIMINATING DATA	ASSESSMENT
Ketamine-triggered takotsubo Apical + midventricular akinesis beyond one vascular territory; GLS −6.0%; troponin 1.06 ng/mL disproportionately low for EF 13%	Leading
Negative-pressure pulmonary edema BMI 35.4, OSA on CPAP, prone deep sedation, cough at onset; does not explain EF 13% or regional akinesis	Possible contributor
Acute myocardial infarction Akinesis crosses coronary territories; angiography with mild luminal irregularities only; complete recovery in 72 h	Excluded
Aspiration NPO > 8 h; secretions pink and frothy, not gastric	Unlikely
Anaphylaxis No rash, bronchospasm, or hypotension; hypertensive throughout	Excluded
Volume overload Only 400 mL crystalloid total; PCWP 15 mmHg; cardiac index 2.74 L/min/m²	Excluded

DISCUSSION & CONCLUSION

Apical-midventricular akinesis crossing coronary territories, non-obstructive CAD, troponin disproportionate to the deficit, and complete recovery satisfy InterTAK criteria for takotsubo syndrome. Onset five minutes after ketamine, with BP 239/130 despite concurrent dexmedetomidine, implicates a catecholamine surge as the trigger.

NPPE from airway obstruction during prone deep sedation in obesity and OSA remains a plausible contributor but does not explain the ventricular findings.

Sedation, not the injection, precipitated this event — vigilance is warranted when ketamine is used for prone interventional pain procedures in patients with obesity and OSA.

As the case report is devoid of patient identifiable information, it is exempt from IRB review per Memorial Healthcare policy. Patient informed consent was obtained for submission of a case report; written informed consent for the planned procedure was obtained preoperatively.

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