CASE REPORT

Ketamine as a Sedative Agent in Type 2 Respiratory Failure Complicating Severe Bronchial Asthma with Cardiomegaly

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ABSTRACT

Background: When severe asthma progresses to type 2 respiratory failure together with cardiomegaly and circulatory instability, choosing an appropriate sedative becomes particularly difficult. An optimal agent in this scenario needs to maintain tissue perfusion, limit further suppression of breathing, and contribute to lowering airway resistance. These considerations make ketamine attractive, owing to its capacity to dilate the airways, its limited depressant effect on respiration at sedative doses, and its sympathomimetic action that can help sustain arterial pressure when hemodynamics are marginal.

Case: A 66-year-old man was admitted to the intensive care unit (ICU) with type 2 respiratory failure arising from bronchial asthma, together with cardiomegaly and pulmonary congestion. He arrived with a reduced level of consciousness, rapid breathing, wheeze in both lung fields, and unstable circulation. Blood gas sampling showed marked carbon dioxide retention with acidemia (pH 7.2; $PaCO_2$ 118.5 mmHg) and a raised lactate. Imaging and echocardiography pointed to an enlarged heart, a likely pulmonary infection, and diastolic impairment, with the left ventricular ejection fraction measured at 39%. After conventional sedation was replaced, a continuous ketamine infusion (0.5–1 mg/kg/h) was titrated to a Richmond Agitation–Sedation Scale (RASS) target of −2 to −3. Care was multimodal, combining lung-protective ventilation, bronchodilators, systemic steroids, antimicrobials, electrolyte correction, and vasopressor support. The patient improved steadily and was extubated by the fifth day without emergence phenomena or serious adverse events.

Discussion: Ketamine was chosen for its combined bronchodilatory action and sympathomimetic support of the circulation, which suited a patient with coexisting severe bronchospasm and hemodynamic instability. Improvement in ventilatory parameters and successful weaning paralleled the ketamine infusion, although the contribution of ketamine cannot be separated from the comprehensive supportive care provided. The reduced left ventricular function (EF 39%) warranted particular caution, since ketamine's sympathomimetic drive may raise myocardial oxygen demand, underscoring the need for close cardiovascular monitoring and careful, graded dose titration in this high-risk geriatric setting.

Conclusion: Ketamine may be considered as a sedative option in selected mechanically ventilated patients with severe asthma and type 2 respiratory failure, particularly when bronchospasm and hemodynamic instability coexist. Nevertheless, meticulous dose titration and vigilant cardiovascular surveillance are essential, especially where cardiac performance is already compromised.

Keywords: ketamine; ICU sedation; type 2 respiratory failure; bronchial asthma; cardiomegaly

INTRODUCTION

Managing sedation in asthmatic patients who also carry cardiovascular disease and progress to type 2 respiratory failure poses a distinct challenge in the intensive care unit (ICU). Agents commonly used for this purpose, including propofol and the benzodiazepines, can depress cardiac contractility and, in some circumstances, promote airway narrowing.[1,2] By contrast, ketamine acts as an antagonist at the N-methyl-D-aspartate (NMDA) receptor and brings a different set of properties to the bedside: it relaxes the airways, exerts comparatively little depression of ventilation, and supports the circulation through indirect sympathetic stimulation.[3,4]

Evidence indicates that ketamine can enhance pulmonary compliance, lower airway resistance, and help maintain cardiac output among the critically ill.[5,6] Selecting a sedative grows even more complicated in older patients with cardiac dysfunction, in whom drug handling is altered and susceptibility to unwanted effects is greater.[7]

What sets this report apart is the convergence of life-threatening bronchospasm, an unstable circulation, and markedly reduced left ventricular function (ejection fraction 39%) in an elderly individual — a combination that makes the choice of sedative especially precarious. We describe how ketamine was employed as the principal sedative in this high-risk context, with attention to practical dose titration, cardiovascular monitoring, and the resulting clinical course.

CASE

A 66-year-old man weighing 60 kg arrived in the ICU on 21 September 2025 with a declining level of consciousness and severe breathlessness. His asthma had usually been well controlled with only infrequent exacerbations, but on this occasion progressed to respiratory failure that necessitated tracheal intubation at 22:30 in the emergency department.

The patient had smoked half to one pack of cigarettes per day since the age of 35. His asthma flared infrequently — most recently in 2023 — and was characteristically triggered by cold weather and by excessive exertion or fatigue; he did not use regular controller therapy and sought hospital care during acute attacks, and there was no previous history of intubation. The present exacerbation was most likely precipitated by exposure to cold weather and overexertion together with a lower respiratory tract infection, as the admission chest radiograph suggested bronchopneumonia and sputum culture subsequently yielded purulent material containing gram-negative and gram-positive cocci. Cardiac comorbidity was confirmed on echocardiography, which showed concentric left ventricular hypertrophy with reduced systolic function (ejection fraction 39%) and a regional wall-motion abnormality.

Day 0 — Admission (21 September 2025): On first ICU evaluation he was already intubated and sedated. Ventilation was delivered in pressure-controlled synchronized intermittent mandatory mode (PC-SIMV) with 5 cmH_2O of positive end-expiratory pressure (PEEP) and an inspired oxygen fraction (FiO_2) of 0.70. He was breathing 20–24 times per minute, the set inspiratory pressure was 15–16 cmH_2O, and exhaled tidal volumes sat around 380–420 mL. Pulse oximetry read 96%, while auscultation disclosed bilateral wheeze without crepitations. The circulation was fragile, with arterial

pressure near 70–75/40 mmHg sustained by norepinephrine at 0.1 μg/kg/min; the heart rate ranged 100–110 beats/min and temperature was 36.9°C.

Blood gas results were consistent with profound respiratory acidosis: pH 7.2, carbon dioxide tension ($PaCO_2$) of 118.5 mmHg, oxygen tension (PaO_2) of 90 mmHg, bicarbonate 45.9 mmol/L, base excess +19, and lactate 5.0 mmol/L. Further laboratory work revealed a slightly low sodium, early ionized hypocalcemia, mildly reduced renal clearance, and a normal glucose. The chest film indicated left-sided cardiac enlargement with congested lung fields and a possible bronchopneumonia.

Sedation had initially been maintained with midazolam plus fentanyl. Because the circulation remained precarious (blood pressure 70/40 mmHg, SpO_2 90%, heart rate 110 beats/min) and bronchospasm continued despite standard measures, the regimen was changed to ketamine. The drug was begun as a continuous infusion of 0.5–1 mg/kg/h, deliberately without a bolus loading dose, aiming for a RASS of −2 to −3. Supporting treatment comprised an aminophylline infusion, nebulized bronchodilators (Combivent), systemic corticosteroid (dexamethasone), levofloxacin 750 mg once daily, stress-ulcer prophylaxis, antipyretics, and calcium gluconate to correct hypocalcemia.

At the time of the switch the patient was still receiving midazolam and fentanyl yet remained clinically unstable: he was hypotensive and tachycardic (70/40 mmHg, 110 beats/min) and vasopressor-dependent, with persistent bronchospasm and severe hypercapnia ($PaCO_2$ 118.5 mmHg) despite maximal bronchodilator therapy, corresponding to an SpO_2/FiO_2 ratio of approximately 129. It was this failure to stabilize — rather than an already-improving trajectory — that prompted the change to ketamine. Over the following 24–72 hours the SpO_2/FiO_2 ratio rose progressively (Figure 3), FiO_2 was reduced from 0.70 to 0.50, inspiratory pressures declined, and norepinephrine was weaned (Figure 2), while sedation was held at the RASS −2 to −3 target.

Days 1–3: Wheeze began to decrease from the second ICU day, although it had not fully cleared, yet oxygen requirements fell from 70% to 50% FiO_2, inspiratory pressures trended downward, and the circulation steadied as norepinephrine was reduced. Sedation was held at RASS −2 to −3, and the patient's awareness improved progressively.

Because severe bronchospasm predisposes to dynamic hyperinflation, intrinsic (auto-) PEEP was monitored by end-expiratory hold. It decreased progressively from 8 cmH_2O on Day 1 to 5 cmH_2O by Day 3 and 4 cmH_2O on Days 4–5 (Figure 2), indicating resolution of air-trapping. This fall paralleled the declining peak inspiratory pressure and inspired oxygen fraction, and lung-protective settings were maintained throughout to limit further hyperinflation while permitting a degree of hypercapnia.

Days 4–5: Support was de-escalated from PC-SIMV to a pressure-support strategy at 40% FiO_2. Ketamine was cut by roughly 25–50% on day 4 and withdrawn on day 5, with ongoing checks of RASS, vital signs, and comfort; an abrupt stop was avoided given the possibility of rebound hypertension. The patient was then extubated and placed on a non-rebreathing mask at 8 L/min.

Throughout the ICU course the asthma-directed regimen was continued in parallel with sedation and maintained until the patient was ready for extubation. From Day 3, as bronchospasm improved,

nebulized Combivent was progressively de-escalated from every 4 hours to every 8 hours and then every 12 hours, and the aminophylline infusion was tapered; both were continued while the patient remained ventilated, and the aminophylline infusion was discontinued on Day 5 once the patient was wheeze-free and ready for extubation. The sedation change was therefore made against a background of sustained bronchodilator therapy, so that ventilator weaning and extubation cannot be attributed to the sedative change alone.

Post-extubation: Wheeze settled, pressures normalized, and repeat blood gases showed clear recovery — pH returned to normal, $PaCO_2$ came down, lactate trended lower, and ionized calcium rose. No emergence reactions, whether hallucinations or agitation, occurred at any point during sedation. Sputum testing yielded purulent material containing both gram-negative and gram-positive cocci.

Transthoracic echocardiography on 26 September 2025 demonstrated an enlarged left atrium, concentrically thickened left ventricle, a regional wall-motion defect, and depressed left ventricular systolic performance with an ejection fraction of 39%. There was grade I diastolic dysfunction, a tricuspid annular plane systolic excursion (TAPSE) of 23 mm, mild mitral regurgitation, and moderate tricuspid regurgitation.

Table 1. Summary of the clinical course during the ICU admission. The detailed day-by-day ventilator and physiological parameters are presented in the landscape table that follows.

Day	FiO_2 (%)	PEEP (cmH_2O)	Vent Mode	BP (mmHg)	NE dose	RASS	Key Events
1–3	70→50	5	PC-SIMV	70–90/40–50	0.1→tapered	−2 to −3	Persistent wheeze; ketamine substituted for midazolam/fentanyl; sensorium improving
4–5	40	5	Pressure Support	Stable	Stopped	−1 to 0	Weaning begun; oxygen demand falling; extubation achieved
Post-extub.	NRM 8 L/min	—	Spontaneous	Stable	None	Alert	Wheeze cleared; pH corrected; $PaCO_2$ down; lactate falling

Table 2. Day-by-day ventilator settings, blood gas, hemodynamic, and sedation parameters. PC-SIMV, pressure-controlled synchronized intermittent mandatory ventilation; PS, pressure support; PEEP, positive end-expiratory pressure; FiO_2, fraction of inspired oxygen; NRM, non-rebreathing mask; RASS, Richmond Agitation–Sedation Scale; GCS, Glasgow Coma Scale. Dashes (—) indicate values not recorded on that day.

Parameter	Day 1 (21 September) 2025	Day 2 (22 September) 2025	Day 3 (23 September) 2025	Day 4 (24 September) 2025	Day 5 (25 September) 2025
Ventilation mode	PC-SIMV	PC-SIMV	PC-SIMV	PS 12 → 6	T-piece → extubation, NRM
Pinsp / Psupp (cmH_2O)	Pinsp 15 / Psupp 12	Pinsp 13 / Psupp 12	Pinsp 12 / Psupp 12	PS 12 → 6	—
PEEP (cmH_2O)	5	5	5	5	—
FiO_2 (%)	70	60	50	40	40 (NRM 8 L/min)
Respiratory rate (/min)	16	16	12	—	18–20
Tidal volume (mL)	310–424	380–410	400–420	420	—
SpO_2 (%)	90–95	95–99	96–99	98	100
pH	7.20	—	—	7.438	—
$PaCO_2$ (mmHg)	118.5	—	—	44.5	—
Lactate (mmol/L)	5.0	—	—	2.0	—
Blood pressure (mmHg)	70/40	85/45	115/85	120/85	130/80
Heart rate (/min)	115	98	95	98	95
Norepinephrine (µg/kg/min)	0.1	0.05–0.1	Tapered / off	Off	Off
RASS / GCS	GCS E2M4VET	GCS E3M5VET	GCS E4M6VET	RASS −1 to 0	Alert, E4M6V5
Sedation	Midazolam / Fentanyl	Ketamine 0.5–1 mg/kg/h	Ketamine (full dose)	Ketamine ↓ 25–50%	Ketamine stopped

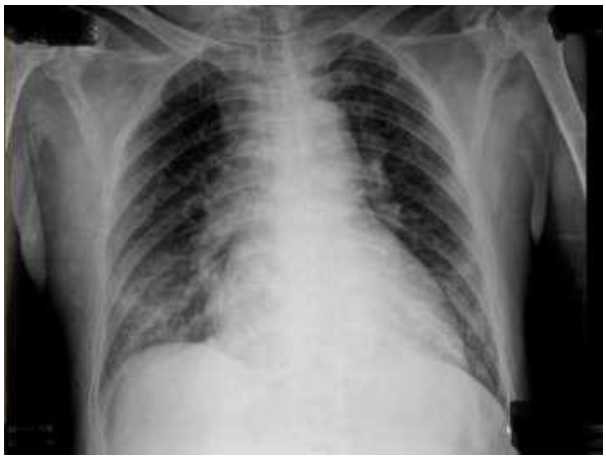


Figure 1. Anteroposterior chest radiograph on admission showing cardiomegaly (left ventricular type) with pulmonary congestion and features suspicious for bronchopneumonia.

Table 3. Transthoracic echocardiography findings (26 September 2025)

Parameter	Finding
Left atrium	Dilated
Left ventricle	Concentric left ventricular hypertrophy (LVH)
LV systolic function	Reduced; ejection fraction (EF) 39%
Regional wall motion	Regional wall motion abnormality (RWMA) present
LV diastolic function	Grade I diastolic dysfunction
Right ventricle	Normal systolic function; TAPSE 23 mm
Valves	Mild mitral regurgitation; moderate tricuspid regurgitation (Vmax 3.1 m/s, max PG 38 mmHg)
Estimated RAP	8 mmHg
Other	No intracardiac thrombus; intact IAS and IVS; no pericardial effusion
Conclusion	Concentric LVH with reduced LV systolic function (EF 39%) and regional wall motion abnormality

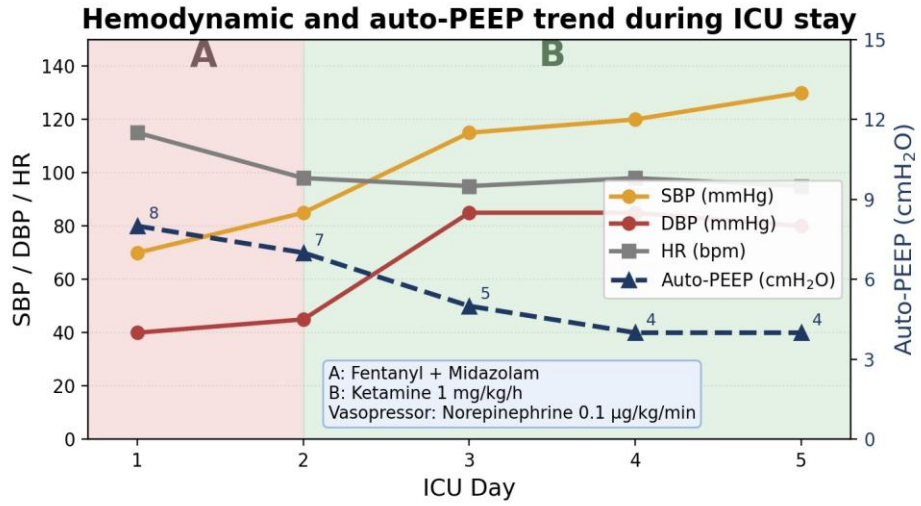


Figure 2. Trend in systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) across the ICU stay. Phase A: midazolam/fentanyl; Phase B: ketamine infusion. Blood pressure rose and heart rate settled after the switch to ketamine while norepinephrine was tapered. Intrinsic (auto-) PEEP is overlaid on the secondary axis and fell from 8 cmH_2O on Day 1 to 4 cmH_2O by Days 4–5, indicating progressive resolution of dynamic hyperinflation.

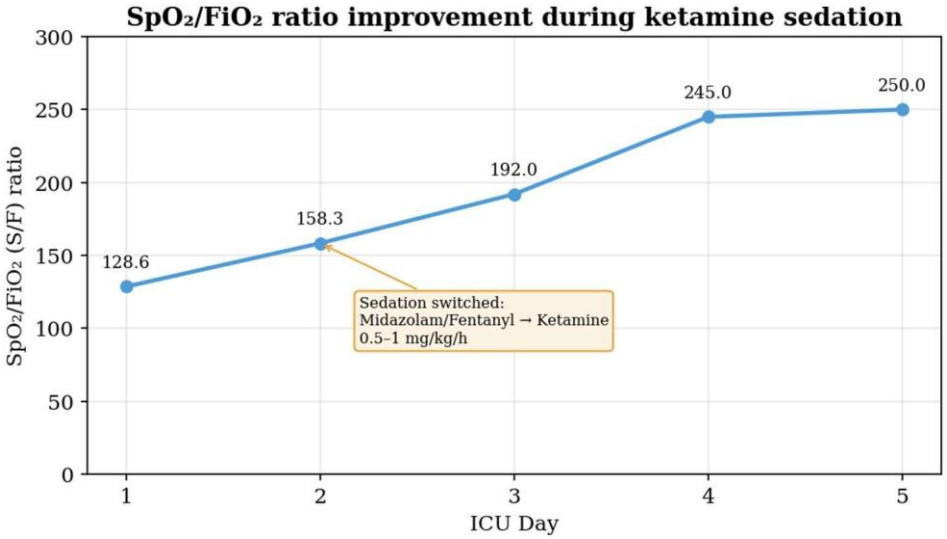


Figure 3. Progressive improvement in the SpO_2/FiO_2 (S/F) ratio during ketamine sedation, reflecting improving oxygenation alongside reductions in FiO_2 requirement.

DISCUSSION

This report illustrates ketamine serving as the main sedative in an elderly, critically unwell patient who simultaneously faced severe bronchospasm, type 2 respiratory failure, an unstable circulation, and depressed left ventricular function (EF 39%). Moving away from midazolam and fentanyl toward ketamine was prompted by the coexistence of treatment-resistant bronchospasm and borderline hemodynamics. It must be stressed, however, that recovery followed a comprehensive package of care — bronchodilators, steroids, antimicrobials, vasopressors, and protective ventilation

— so the specific contribution of ketamine cannot be disentangled from the rest.

Ketamine was favored because its airway-relaxing action arises through several routes: antagonism at the NMDA receptor, blockade of catecholamine reuptake, β_2-adrenergic stimulation, and direct relaxation of bronchial smooth muscle.[1,2,6] One published series recorded a fall in peak airway pressure of about 25–30% among intubated asthmatic patients, which parallels the ventilatory gains seen here.[8] The drug also depresses breathing only minimally at sedative levels and has been described as easing mechanical ventilation and possibly permitting smoother weaning relative to several alternatives.[3–5]

With regard to the heart, the sympathomimetic profile of ketamine can raise cardiac output and help defend blood pressure, which often makes it a circulatory-friendly choice in unstable patients.[3,9,10] A crucial caveat applies in the present case, however: with an ejection fraction of 39% and a failing left ventricle, the same sympathetic drive could increase myocardial oxygen consumption and add to cardiac strain. Moreover, when catecholamine reserves are depleted or autonomic control is severely disturbed, ketamine may unexpectedly lower blood pressure through its intrinsic negative inotropy.[1] For these reasons, continuous hemodynamic observation and cautious titration were indispensable throughout this patient's treatment.

Age was a further consideration in a 66-year-old. The pharmacokinetic and pharmacodynamic shifts that accompany ageing can hasten onset, prolong duration through diminished hepatic elimination, and heighten central nervous system sensitivity, thereby increasing delirium risk.[7] Accordingly, ketamine was given without a loading bolus, kept toward the lower end of the sedative range (0.5–1 mg/kg/h), and titrated gradually. No emergence reactions were encountered, which may reflect both the modest dosing and the continuous-infusion approach.[2]

Weaning followed a structured tapering schedule rather than abrupt cessation (Figure 4). The full dose was maintained for the first three days, reduced by 25–50% on day 4, and further reduced or stopped on day 5, with continuous attention to RASS, vital signs, and comfort. This graded approach was deliberate, since sudden withdrawal of ketamine carries a risk of rebound hypertension. In this patient the transition was uneventful, with no withdrawal syndrome and a successful progression to extubation.[2]

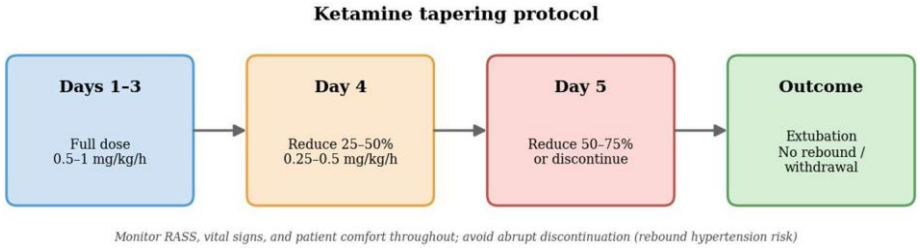


Figure 4. Stepwise ketamine tapering protocol applied in this case, from full-dose maintenance through graded reduction to discontinuation and extubation.

Ketamine may also form part of a multimodal analgesic plan; by antagonizing the NMDA receptor it blunts central sensitization and may lessen opioid demand in the critically ill.[1]

When weighed against alternative sedatives in this clinical context (Table 3), ketamine was distinctive in offering a favorable combination of airway relaxation, hemodynamic preservation, and minimal respiratory depression. Propofol and benzodiazepines carried a greater risk of circulatory instability in this already-unstable patient, opioids posed respiratory depression and potential histamine-mediated bronchospasm, and dexmedetomidine — although a reasonable adjunct — raised concern for bradycardia in the setting of cardiomegaly.

A recognized drawback of ketamine is its tendency to increase salivary and tracheobronchial secretions (hypersalivation/bronchorrhea) through cholinergic stimulation, which can add to secretion burden and, rarely, provoke laryngospasm — a concern chiefly at induction or in the non-intubated airway. In a patient already intubated and undergoing regular endotracheal suctioning this risk is largely mitigated, and an antisialagogue (e.g., glycopyrrolate or atropine) may be used if secretions become troublesome. In the present patient, hypersalivation was observed during the ketamine infusion; however, it was controlled with routine endotracheal suctioning and did not lead to mucus plugging or endotracheal tube obstruction, nor did it impede ventilator weaning or extubation.

Table 4. Comparison of sedative agents in asthma and airway disease

Sedative Agent	Bronchodilation	Hemodynamic Effect	Respiratory Depression
Ketamine	+++	Stable; ↑ sympathetic tone	Minimal
Propofol	Mild bronchoconstriction	↓ BP, ↓ SVR, ↓ contractility	Dose-dependent depression
Midazolam / benzodiazepine	—	Minimal	CNS depression; accumulation in renal impairment
Dexmedetomidine	—	Bradycardia	Mild (bradycardia, mild hypotension)
Opioid (fentanyl, morphine)	± histamine release	Largely neutral	Marked depression; bronchospasm risk

Existing data support ketamine as an adjunct in near-fatal asthma that resists conventional therapy.[4,5] Pediatric reports have noted improvements in the Clinical Asthma Score and a shorter time on the ventilator without a meaningful rise in complications.[5]

Systematic reviews and meta-analyses of ketamine in mechanically ventilated and broader critically ill populations likewise suggest a sedative-sparing effect with an acceptable safety profile, while emphasizing that the certainty of evidence remains limited.[10,11,12,13,15]

Lower-dose ketamine has additionally been associated with reduced delirium in ventilated ICU patients.[14] In line with these observations, practice guidelines list ketamine as an alternative sedative for patients with bronchospasm or circulatory instability, though robust randomized evidence in adults is still lacking.[3]

CONCLUSION

Ketamine may be considered as a sedative option in selected mechanically ventilated patients with severe asthma and type 2 respiratory failure, particularly where bronchospasm and circulatory instability occur together. In this instance, a continuous intravenous infusion of 0.5–1 mg/kg/h over five to six days accompanied a favorable course, marked by steadier hemodynamics, easing bronchospasm, and successful liberation from the ventilator, all without emergence phenomena. Even so, in a heart with impaired left ventricular function the risk that ketamine raises myocardial oxygen demand must be weighed carefully, and close cardiovascular monitoring is mandatory. Among older patients, dose moderation and unhurried titration are advisable.

Causal claims should remain restrained, since improvement rested on an integrated treatment approach, and further well-conducted studies are required before this strategy can be generalized.

ETHICAL STATEMENT

Written informed consent for publication of this case was obtained from the patient's family. In keeping with institutional policy, formal ethics committee approval was not required for a single anonymized case report.

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The authors report no conflict of interest.

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