

CASE IN POINT

PEER REVIEWED

Anesthetic Choice in Chronic Systolic Heart Failure and Subsequent Cardiogenic Shock

Authors:

Wilson B. Pfeiffer, MS-IV; Adedeji O. Olusanya, MS-IV

Nova Southeastern University, Fort Lauderdale, Florida

Dinos Smith, DO; Mackenzie Pullee, DO; Diana M. Ledford, MD; and David J. Tunnell, DO

Grandview Medical Center, Dayton, Ohio

Syed A. A. Rizvi, PhD, MS, MBA

Hampton University School of Pharmacy, Hampton, Virginia

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A 68-year-old woman presented to the emergency department with worsening dyspnea and systemic swelling over the past 72 hours.

History. The patient had a medical history of chronic systolic heart failure (HF), coronary artery disease, type 2 diabetes mellitus, hypertension, multiple cerebral vascular accidents, obesity, subdural hematoma, left leg amputation, and diabetic peripheral neuropathy. At presentation, she was accompanied by her daughter, who stated that the woman had had a myocardial infarction (MI) 1 year prior, which had been managed with stent placement as opposed to surgery due to high surgical risk. The daughter further explained that the patient had experienced dyspnea and edema since her MI, and that they had worsened significantly over the past 3 days. The patient denied any history of alcohol use, smoking, or drug use. The patient reported having associated abdominal pain but denied having fever, chills, cough, chest pain, nausea, or vomiting.

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Physical examination. At presentation, the patient was sitting upright, was in no apparent distress, and was awake, alert, and oriented to person, place, and time. Cranial nerves II through XII were intact, speech was normal, and there was no lateralizing weakness. Auscultation of the heart revealed regular rate and rhythm with S₁ and S₂ present and no murmurs. Upon auscultation of the lungs, faint crackles were heard in the lower airways bilaterally, but there were no wheezes or rhonchi. There was tenderness to palpation in all 4 abdominal quadrants, and there was diffuse anasarca present, with 2+ pitting edema present on the right lower extremity and left stump. The skin showed chronic changes consistent with arterial disease.

She had a blood pressure of 80/50 mm Hg, a heart rate of 85 beats/min, a respiratory rate of 23 breaths/min, and an oxygen saturation of 95% on 2 L/min of oxygen via nasal cannula. The patient was admitted to the hospital floor and was started on an infusion of furosemide, 40 mg/h.

Hospital course. On the morning of day 2, the patient was found in a mildly somnolent state and received a diagnosis of type 2 respiratory failure secondary to acute HF exacerbation and pulmonary edema. She was transferred to the intensive care unit.

Her creatinine level was found to be elevated at 4.1 mg/dL (up from a baseline of 2.5 mg/dL), and her blood pressure had decreased to 96/66 mm Hg. A transthoracic echocardiogram showed a left ventricular ejection fraction (LVEF) of 18%, and her N-terminal pro-B-type natriuretic peptide (NT-proBNP) level was extremely elevated at greater than 40,000 pg/mL. The patient was started on a dobutamine infusion (5 µg/kg/min), and bilevel positive airway pressure treatment was initiated.

Over the next 2 days, the patient had increasing somnolence with beginning signs of disorientation. On day 4, the patient was given a one-time dose of metolazone. Her mental status continued to worsen over the next 2 days, with the creatinine level remaining consistently above 4.0 mg/dL despite medical management. After continued failure of medical management, it was determined on day 7 that permanent dialysis was needed, and vascular surgery was consulted for placement of a tunneled dialysis catheter and creation of an arteriovenous fistula.

After extensive discussion with the family and all contributing medical providers about the significant risks involved with surgery, it was decided that the benefits outweighed the risks, and that ultimately the patient needed to undergo placement of a tunneled dialysis catheter.

Preoperative evaluation on day 8 showed severe confusion, with the patient disoriented to person, place, and time. She was ill-appearing with significant pallor. Despite continued administration of dobutamine at 5 µg/kg/min, the patient's blood pressure was 94/69 mm Hg with a respiratory rate of 33 breaths/min and a heart rate of 99 beats/min. There were significant

crackles present in the upper and lower lung fields bilaterally. The abdomen was tense and distended, and there was pitting edema present in the right lower extremity and left stump. There was a grade 2 carotid bruit present on the left side. The patient had full neck range of motion, a thyromental distance of 6 cm, and a modified Mallampati score of 4.

Because the patient was already struggling to sustain adequate perfusion despite treatment with dobutamine, it was determined that she would not be able to tolerate any form of myocardial or respiratory depression caused by administration of anesthetics. Furthermore, it was clear that the patient would not be able to tolerate any medication that would cause even a slight decrease in blood pressure. The anesthetic plan included monitored anesthesia care with a focus on limited fluid administration and preservation of end-organ perfusion; sedation and analgesia would be accomplished with a single bolus dose of ketamine (25 mg) and continuance of both the dobutamine and furosemide infusions at their previous rates. To further prevent fluid buildup in the cardiopulmonary system, the surgical bed was placed in reverse Trendelenburg position for the duration of the procedure.

After an uneventful and successful surgical procedure, the patient was found to be awake and hemodynamically stable in the recovery room, with spontaneous breathing and a patent airway. Blood pressure was 125/65 mm Hg, heart rate was 101 beats/min, respiratory rate was 33 breaths/min, oxygen saturation was 97% on 3 L/min oxygen via nasal cannula, and temperature was 36.1°C. After adequate stabilization was achieved in phase 2, the patient was promptly taken for hemodialysis via her newly inserted tunneled catheter.

Discussion. Choosing the correct anesthetic medication regimen for surgical procedures is critical to the success of surgery and for the well-being of every patient who undergoes a surgical procedure.¹ When evaluating a patient for surgery, it is critical to consider every organ system and any pathology that may exist.² Every anesthetic agent that can be administered for surgery has an adverse-effect profile that must be considered.³

While many pathological states pose significant risks for anesthesia, one of the biggest risks is underlying HF.^{4,5} Not only did our patient have chronic systolic HF with an LVEF of 18%, but also she had been struggling to maintain adequate blood pressure despite dobutamine treatment. The status of our patient's cardiothoracic system necessitated the avoidance of most anesthetic agents due to their adverse effect of dose-dependent myocardial and respiratory depression.⁶ Not only do patients with a significantly depressed LVEF have problems with end-organ perfusion, but also they have the propensity to develop pulmonary edema, causing inadequate oxygenation of hemoglobin at baseline. Administering a drug that will cause myocardial depression, respiratory depression, or both will exacerbate the patient's underlying pathology, potentially leading to death.^{7,8}

With the exception of ketamine, most intravenous anesthetic agents have a dose-dependent respiratory depression.⁹ Propofol, a commonly used GABA_A receptor agonist, not only causes a dose-dependent respiratory depression but also causes direct myocardial depression and a significant decrease in systemic vascular resistance. Opioids such as fentanyl cause respiratory depression, decreased cardiac output, decreased stroke volume, and decreased heart rate. Benzodiazepines such as midazolam cause myocardial and respiratory depression. Thiopental, a barbiturate that had been used previously in the United States, can cause direct myocardial and respiratory depression as well as decreased systemic vascular resistance. Dexmedetomidine, a less commonly used α_2 agonist, causes heart block, myocardial depression, and hypotension.¹⁰

In a patient with severe HF such as ours, this essentially leaves 2 intravenous anesthetic agents to choose from. While etomidate (a selective GABA_A receptor modulator) is relatively unique in that it is able to preserve cardiac output and stroke volume, it still has a minimal (but nonetheless present) decrease in mean arterial pressure along with a proven adverse effect of adrenal suppression when used extensively.¹¹ In comparison, ketamine (an NMDA receptor antagonist with profound analgesic effects) is useful in patients with HF for a multitude of reasons. It does not cause a dose-dependent respiratory depression as do other intravenous agents. In addition, it is beneficial in patients with HF due to its sympathomimetic properties, which cause increased cardiac output, increased mean arterial pressure, and increased heart rate, allowing for reasonable preservation of the circulatory system during surgical procedures.¹²

Additionally, ketamine has been documented to cause direct myocardial depression in patients who have a severe baseline catecholamine-depleted state.¹² However, this occurrence is extremely rare, and in a patient with underlying severe HF who requires surgical treatment, ketamine is the most reasonable anesthetic agent in which the benefits of administration outweigh the risks.

Our patient's case illustrates the ability of patients with severe systolic HF to undergo necessary surgical treatment under the administration of a ketamine-only anesthetic regimen. While it is not an otherwise ideal anesthetic regimen, the sympathomimetic activity of ketamine makes it a reasonable and preferred choice of anesthesia in patients requiring surgery with severe HF, due to the drug's maintenance of adequate myocardial contractility, systemic perfusion, and oxygenation.

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