

The Use of Ketamine as an Induction Agent for Anesthesia in Pulmonary Thromboendarterectomy Surgery: A Case Series

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ABSTRACT

Pulmonary thromboendarterectomy (PTE) surgery is the treatment of choice for patients with chronic thromboembolic pulmonary hypertension (CTEPH). The induction of anesthesia in patients with severe pulmonary hypertension (PHT) can be challenging, with a risk of cardiovascular collapse. The administration of ketamine in patients with PHT is controversial, with some recommendations contraindicating its use. However, ketamine has been used safely in children with severe PHT. We present a retrospective case series of adult patients with severe PHT presenting for PTE surgery, using intravenous ketamine as a co-induction anesthetic agent.

Keywords: Cardiac anesthesia, chronic thromboembolic pulmonary hypertension, ketamine, pulmonary hypertension, pulmonary thromboendarterectomy

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Submitted: 22-Feb-2021 **Revised:** 08-Jun-2022 **Accepted:** 09-Jun-2022 **Published:** 10-Oct-2022

INTRODUCTION

Pulmonary thromboendarterectomy (PTE) is the treatment of choice for patients with chronic thromboembolic pulmonary hypertension (CTEPH).^[1] The induction of anesthesia in patients with severe pulmonary hypertension (PHT) can be challenging, with a risk of cardiovascular collapse.^[2] The use of ketamine in PHT is controversial with some recommendations advocating the avoidance of ketamine due to its effect of the increasing mean pulmonary artery pressure (mPAP) and pulmonary vascular resistance (PVR).^[3,4] However, ketamine has been used safely in children with PHT.^[5,6] We report a retrospective case series of CTEPH patients with severe PHT, who successfully underwent PTE surgery using intravenous ketamine as a co-induction anesthetic agent.

CASE HISTORIES

The local research and ethics committee's approval was obtained to review the patient records. A retrospective review of the prospectively captured data for the patients undergoing PTE surgery over 2 years was undertaken. Thirty-one patients underwent PTE surgery with preoperative PVR >10 wood units (WU) and received ketamine at the induction of anesthesia. The patient demographics and preoperative data are presented in Table 1.

Anesthetic management

Before the induction of anesthesia, in addition to standard routine monitoring, all the patients had peripheral intravenous access, radial artery invasive blood

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Quick Response Code:	Website: www.annals.in
	DOI: 10.4103/aca.aca_24_21

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How to cite this article: Salaunkey K, Jenkins D, Roscoe A. The use of ketamine as an induction agent for anesthesia in pulmonary thromboendarterectomy surgery: A case series. *Ann Card Anaesth* 2022;25:528-30.

Table 1: Patient demographics and preoperative hemodynamic data

	(n=31)
Age (years)	58.8 (16.4)
Sex	
Female	17 (55%)
Male	14 (45%)
Weight (kg)	81.8 (24.4)
BSA (m ²)	1.86 (0.26)
PVR (WU)	13.8 (3.8)
mPAP (mmHg)	51.3 (8.7)
Cardiac index (L/min/m ²)	1.85 (0.41)
Long-term oxygen therapy	5 (16%)
UCSD disease classification	Type II - 7 (23%) Type III - 22 (71%) Type IV - 2 (6%)
Preoperative RV function parameters	
TAPSE (mm)	15.8 (4.7)
RVFAC (%)	22.8 (10.8)

BSA: body surface area; mPAP: mean pulmonary artery pressure; PVR: pulmonary vascular resistance; RVFAC: right ventricular fractional area change; TAPSE: tricuspid annular plane systolic excursion; UCSD: University of California, San Diego; WU: wood units. Data presented as mean (standard deviation) or number (percentage)

pressure monitoring, and near-infrared spectroscopy cerebral oximetry monitoring. A central venous catheter, pulmonary artery catheter (PAC), and femoral artery invasive blood pressure monitor were inserted after the induction of anesthesia, as per standard institutional practice.

The induction of anesthesia was achieved with intravenous midazolam (0.05 mg/kg), fentanyl (2–4 µg/kg), ketamine (0.75–1 mg/kg), and pancuronium (0.15–0.2 mg/kg).

After endotracheal intubation, the patients were ventilated with oxygen/air mixture with a fraction of inspired oxygen 0.6–0.8, tidal volume 6–7 mL/kg, positive end-expiratory pressure 5 cmH₂O, and respiratory rate 12–18 per min, adjusted to target arterial pCO₂ 35–40 mmHg. The maintenance of anesthesia was achieved using an intravenous propofol infusion at 60–75 µg/kg/min. A second dose of fentanyl (2–4 µg/kg) was administered immediately prior to the start of the surgery.

No patient suffered a cardiovascular collapse after the induction of anesthesia. All the patients were hemodynamically stable and no patients required inotropic or vasopressor support through to the initiation of cardiopulmonary bypass (CPB).

The PTE surgery was conducted as previously described with the patients having predominantly type II and type III disease resected.^[11] After rewarming, the patients were weaned from CPB. The first-line inotropic support administered was dopamine (5 µg/kg/min).

A central venous pressure of 50% of the baseline was targeted to achieve optimal volemic status. Additional vasoactive agents were administered depending upon the thermodilution cardiac output studies performed using the PAC. In addition to dopamine, four patients required noradrenaline support. One patient required vasopressin in addition to both dopamine and noradrenaline. One patient suffered a surgical tear of the pulmonary artery and was initially supported with central veno-arterial extracorporeal membrane oxygenation after discontinuation of CPB. At the end of the surgery, all the patients were transferred to the intensive care unit and followed a standard hemodynamic and ventilatory postoperative weaning protocol. The postoperative hemodynamic data and patient outcomes are presented in Table 2.

Statistics

Data are presented as mean (± standard deviation) for normally the distributed data, as median (interquartile range [IQR]) for the ordinal data, and number (percentage) for the categorical data.

DISCUSSION

The induction of anesthesia in patients with severe PHT can result in right ventricular (RV) failure, cardiovascular collapse, and cardiac arrest.^[2] The basic hemodynamic goals include avoiding RV ischemia by maintaining systemic blood pressure and RV perfusion pressure; preserving RV contractility by avoiding negative inotropic agents; optimizing preload and preventing RV overdistension; and reducing RV afterload by implementing measures to decrease PVR: avoidance of hypoxemia, hypercarbia, acidemia, and hypothermia.^[7]

The use of ketamine in patients with PHT is contentious.^[8] The earlier studies demonstrating an increase in mPAP and PVR on the administration of intravenous ketamine were performed on spontaneously breathing patients with normal baseline PVR, receiving doses of 2 mg/kg or greater.^[9,10] The later studies, with controlled ventilation in children with baseline PHT, showed no effect of ketamine on PVR.^[6,11] The co-induction of anesthesia using a combination of ketamine with a benzodiazepine and opioid has been shown to be hemodynamically stable, with no significant increase in PVR, in adult patients undergoing coronary artery bypass graft surgery.^[12] The preservation of a sympathetic tone by ketamine at the induction of anesthesia is advantageous in maintaining the Systemic Vascular Resistance (SVR) and RV perfusion pressure, and some authors recommend it as the agent of choice in severe PHT.^[2]

Table 2: Intraoperative data, postoperative hemodynamic data, and patient outcomes

	(n=31)
CPB time (min)	278.9 (46.0)
AXC time (min)	58.2 (14.6)
DHCA time (min)	35.3 (11.1)
Postoperative PVR (WU)	3.6 (2.9)
Postoperative mPAP (mmHg)	24.7 (8.7)
Postoperative Cardiac Index (L/min/m ²)	2.45 (0.46)
ICU length of stay (d)	4.3 (IQR 3-6.4)
Hospital length of stay (d)	13.2 (IQR 9-22.5)
In-hospital/30-day mortality	0 (0%)
1-year mortality	2 (6.4%)

AXC: aortic cross clamp; CPB: cardiopulmonary bypass; DHCA: deep hypothermic circulatory arrest; ICU: intensive care unit; mPAP: mean pulmonary artery pressure; PVR: pulmonary vascular resistance; WU: wood units. Data presented as mean (standard deviation), median (interquartile range [IQR]), or number (percentage)

Although our cases series is not able to directly show the precise effect of ketamine on PVR in severe PHT, the hemodynamic stability of the patients after the induction of anesthesia suggests that ketamine was not detrimental to patient outcome. We have shown that ketamine in a dose less than 1 mg/kg can be used safely as a co-induction agent in adult patients with severe PHT presenting for PTE surgery, and the presence of severe PHT should not preclude its use.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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