



Anesthetic Management of an Unusual Case of Severe Pulmonary Hypertensive

Peipei Sun^{1*}

¹Department of Anesthesiology, West China Hospital of Sichuan University, Chengdu, China

Corresponding Author: **Peipei Sun**

Address: Department of Anesthesiology, West China Hospital, Sichuan University, Chengdu, 610041, China;

Email: 121040388@qq.com

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Abstract

The perioperative management of pulmonary hypertension continues to be a great challenge for anesthesiologists. Among the various types of pulmonary hypertension, pulmonary hypertension associated with left heart disease is the most common, while pulmonary hypertensive crisis is one of the causes of perioperative mortality in these patients. We present the case of a 54-year-old female with severe valvular heart disease complicated by severe pulmonary hypertension and massive hemothorax, who underwent emergency exploratory thoracotomy, mitral valve replacement, and tricuspid valvuloplasty. The patient was in critical condition and suspected to have a pulmonary hypertension crisis. The rationale for this case report is to learn about the anesthetic management of patients with severe pulmonary hypertension.

Keywords

Pulmonary Hypertension, Anesthesia, Anesthetic Management, Case Report

Introduction

Pulmonary vascular disease, chronic lung disease, chronic left heart disease, pulmonary embolism, and other causes can lead to pulmonary hypertension (PH). Pulmonary hypertension associated with left heart disease (PH-LHD) is the most prevalent form of PH, accounting for 65-80% of cases [1]. According to the 2022 ESC/ERS Guidelines, PH-LHD is defined by a mean pulmonary arterial pressure (mPAP) >20mmHg and a pulmonary arterial wedge pressure (PAWP) >15mmHg, which is consistent with the definition of PH [2]. The pathophysiological mechanisms of PH-LHD include [2-4]:

1. Increased left ventricular filling pressure due to

left heart disease, transmitted backward to the pulmonary circulation.

2. Pulmonary artery endothelial dysfunction and vasospasm.
3. Pulmonary vascular remodeling.
4. Right ventricular dilatation or dysfunction and functional tricuspid regurgitation.
5. Altered right ventricular-pulmonary artery coupling.

Anesthetic management aims to prevent pulmonary hypertensive crisis and subsequent right ventricular failure.

Case history

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A 54-year-old female was admitted with severe valvular heart disease. She had been experiencing exertional dyspnea for more than 10 years and was previously diagnosed with rheumatic heart disease at a local hospital. Her symptoms had worsened over the past 6 months. She also had a history of diabetes mellitus.

The cardiac ultrasound (**Fig-1**) revealed severe mitral valve stenosis and moderate mitral regurgitation, with a forward blood flow acceleration of 2.2m/s and an effective orifice area of 0.6 cm².

Additionally, there was severe tricuspid regurgitation with a jet velocity of 3.8 m/s and a peak pressure gradient of 58 mmHg, which estimated a pulmonary artery systolic pressure of 73 mmHg.

Left ventricular systolic function, as measured by the ejection fraction, was 61%, and right ventricular systolic function, as measured by the tricuspid annular plane systolic excursion, was within normal limits but at the lower end of the normal range at 16 cm. A twelve-lead electrocardiography showed atrial fibrillation.

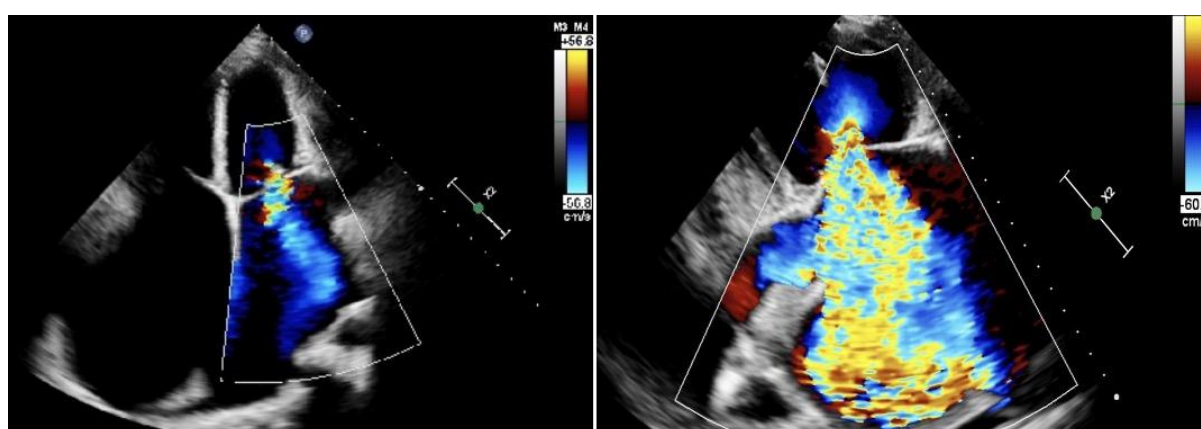


Fig-1:

The TTE Revealed Severe Mitral Valve Stenosis and Severe Tricuspid Regurgitation

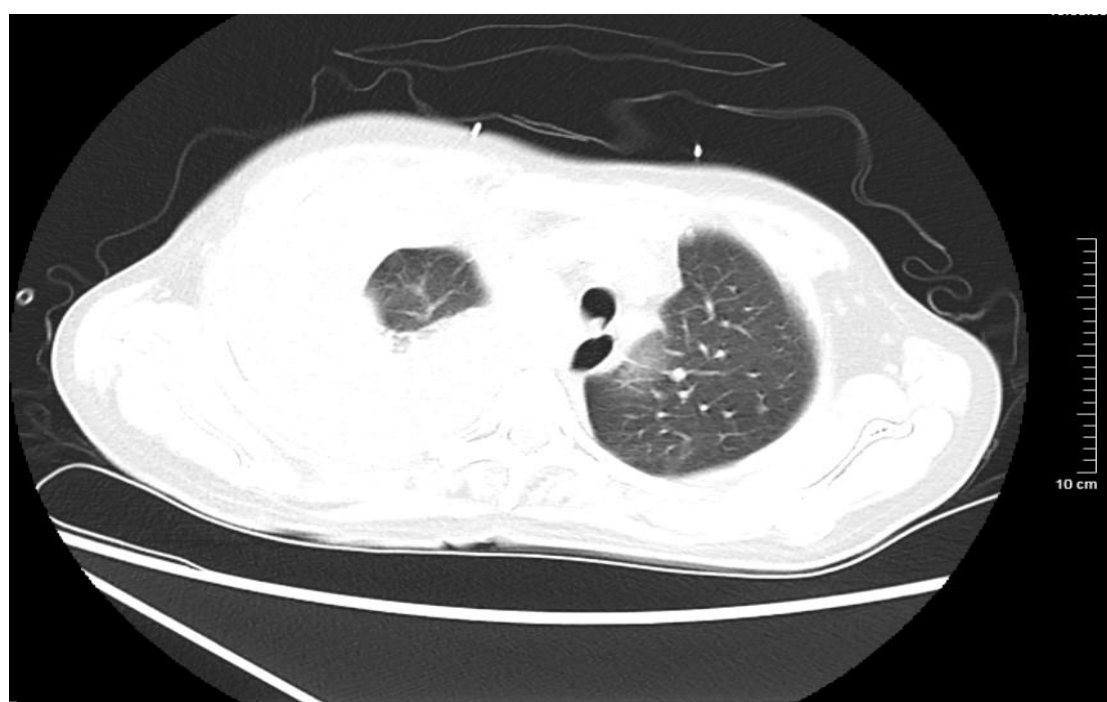


Fig-2:

The Chest CT Scan Showed a Massive Pleural Effusion on the Right Side

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The chest CT scan showed bilateral pleural effusion, and bilateral closed thoracic drainage was performed. The drainage tube was removed three days later. However, she gradually developed dyspnea and experienced progressive worsening over the next two days. A chest CT scan revealed a massive pleural effusion on the right side (**Fig-2**). Thoracentesis indicated hemothorax, and due to the presence of active bleeding, emergency thoracotomy was performed for hemostasis. Mitral valve replacement and tricuspid valvuloplasty were performed simultaneously.

At the time of admission to the operating room, the patient was experiencing orthopnea and was drowsy. The portable ECG monitor showed a blood pressure of 134/71mmHg with a continuous infusion of 0.1ug/kg/min norepinephrine to maintain it, atrial fibrillation rhythm, a heart rate of 134 beats/min, and pulse oxygen saturation of 95% with 100% oxygen. After standard anesthetic monitoring, a right femoral artery puncture tube was placed for invasive blood pressure monitoring. Anesthesia was induced with 0.2ug/kg sufentanil, 0.1mg/kg etomidate, and

0.8mg/kg rocuronium intravenously. An endotracheal tube was inserted for mechanical controlled ventilation. Maintenance of anesthesia was achieved with inhaled sevoflurane and a continuous intravenous infusion of remifentanyl, along with continuous inhalation of nitric oxide through the endotracheal tube. A right internal jugular venous catheter and a transesophageal cardiac ultrasound probe were placed immediately after. The measured central venous pressure was 42 cmH₂O. The transesophageal echocardiography (TEE) showed significant cardiac enlargement, particularly in the left and right atrium, severe mitral stenosis, severe tricuspid regurgitation, and a hypoechoic (dark gray) fluid collection in the pleural space (**Fig-3**). During this period, the patient required higher doses of norepinephrine (0.2-0.3ug/kg/min) to maintain a blood pressure of 60-78/40-51 mmHg. Epinephrine (0.2ug/kg/min) and vasopressin (5ug/h) were simultaneously initiated for cardiac support. The patient's condition was critical, with extremely severe pulmonary hypertension, and it was necessary to establish cardiopulmonary bypass as soon as possible.

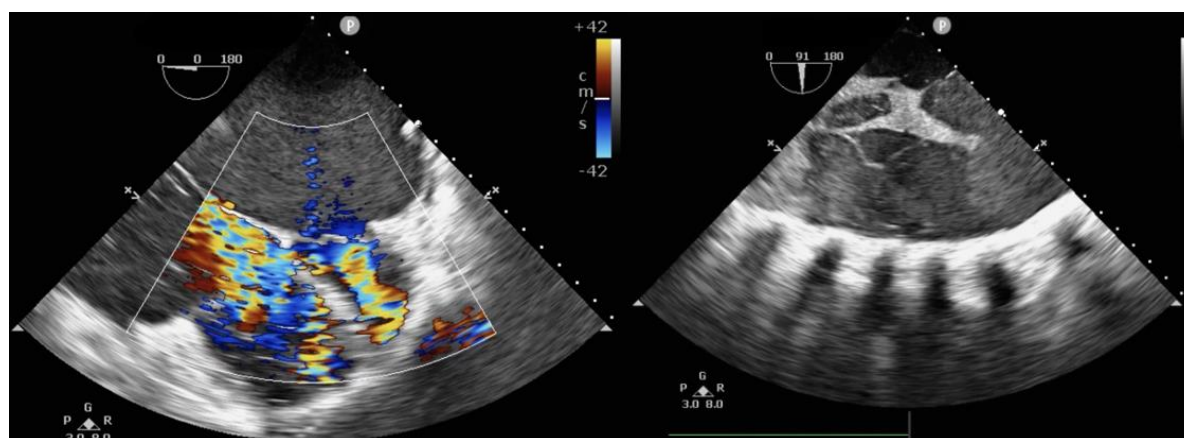


Fig-3:

The TEE Showed Severe Mitral Stenosis, Severe Tricuspid Regurgitation, and a Hypoechoic Fluid Collection in the Pleural Space

After positioning the patient, the anesthesia was deepened, and the surgery was initiated. Following the initiation of cardiopulmonary bypass, blood and thrombus were removed from the right side of the chest cavity (**Fig-4**), and bleeding from the 7th intercostal artery in the posterior chest wall was addressed. Mitral valve replacement and tricuspid valvuloplasty were subsequently performed. The

duration of the operation lasted 8 hours and 22 minutes, with a cross-clamp time of 45 minutes and a cardiopulmonary bypass time of 217 minutes. The patient was successfully weaned off bypass with high positive inotropic and vasopressor support and then transferred to the intensive care unit. The positive inotropic drugs and vasopressors were gradually reduced, and the patient was extubated on the fifth

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postoperative day.

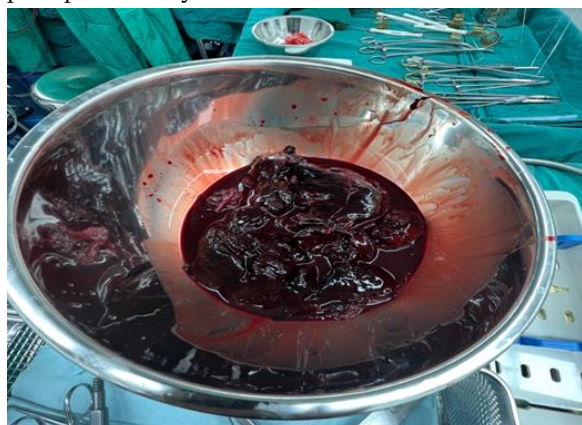


Fig-4:

Thrombus Removed from the Right Side of the Chest Cavity

Discussion

General anesthesia with endotracheal intubation is preferred for patients with pulmonary hypertension undergoing cardiac surgery. Upon entering the operating room, patients should routinely inhale pure oxygen to alleviate hypoxic pulmonary vasoconstriction. In addition to standard monitoring, continuous invasive arterial monitoring, central venous pressure monitoring, and temperature monitoring are required. Monitoring of pulmonary artery pressure is also essential. While Swan-Ganz catheters can provide direct and accurate measurements of pulmonary artery pressures, their routine use has decreased due to potential complications such as infection, thrombosis, and pulmonary artery rupture [5]. Alternative methods, such as non-invasive transesophageal echocardiography (TEE), are considered for monitoring and assessing pulmonary artery pressures. Although TEE cannot directly measure pulmonary artery pressure, it can indirectly determine the level of pulmonary artery pressure using parameters such as pulmonary artery flow velocity and right ventricular ejection velocity. TEE can also provide information about cardiac output estimation, myocardial contractility, volume, valve function, and cardiac structure, making it a valuable tool for guiding anesthesia management and a standard monitoring technique for cardiac surgeries.

During the procedure, the anesthesiologist must maintain blood pressure, manage fluids, ensure proper oxygenation, and maintain acid-base balance [6].

The key principle of intraoperative management and maintenance of anesthesia is to avoid any factors that could increase pulmonary hypertension and decrease right ventricular contractility.

During the induction of anesthesia, systemic vasodilation induced by anesthetic agents can lead to a significant drop in mean arterial pressure, which has the potential to reduce coronary perfusion pressure and severely affect right ventricular contractility [7]. Therefore, it is extremely important to avoid a decrease in arterial blood pressure during this phase. It is also important to aware that intubation before the full onset of anesthetic drugs can cause intense sympathetic stimulation and aggravate pulmonary hypertension. Intravenous induction drugs such as midazolam, propofol, etomidate, can be safely used in combination with opioids, but etomidate is better for circulatory stabilization than propofol. Histamine-releasing relaxants (atracurium, mivacurium) should be avoided for patients with pulmonary hypertension, as they may further increase pulmonary resistance [7].

After tracheal intubation, the use of both volatile anesthetic agents and total intravenous anesthesia remain safe [8]. However, nitrous oxide should be avoided as it increases pulmonary vascular resistance. Treatments to decrease right ventricular afterload include inhalational agents (prostaglandin, nitric oxide) or continuous low dose intravenous infusions (prostaglandin), but prostaglandin particularly with intravenous should avoid being used in PH-LHD, due to the risk of systemic hypotension and profound pulmonary vascular dilation, resulting in pulmonary oedema [8]. Intraoperative treatment to avoid an increase of pulmonary hypertension include ventilator parameter settings, temperature management, acid-base and fluid management. Ventilator parameter settings include using low tidal volume ventilation to avoid alveolar overinflation (target: 6-8 ml/kg ideal body weight), keeping peak airway pressure below 30 mmHg, keeping higher inspiratory FiO_2 (titrate to 60%-100%) and hyperventilating to maintain exhaled partial pressure of carbon dioxide between 30 and 35 mmHg [9,10]. Hypothermia and shivering can significantly increase pulmonary pressure and should be strictly avoided, so does metabolic acidosis, should maintain $\text{pH} > 7.4$. Fluid balance should be carefully

optimized based on TEE monitoring; right ventricular preload is essential to maintain cardiac output. However, to maintain cardiac output, in addition to appropriate preload, inotropes (adrenaline, dobutamine), vasopressors (noradrenaline, vasopressin) and cardiotoxic vasodilators (milrinone, enoximone) may be required, avoiding negative inotropic agents, such as beta-blockers.

In the case of this patient, who already had severe pulmonary hypertension due to mitral valve disease, the large amount of blood in the right thoracic cavity after thoracentesis compressed the lung tissue, thus limiting the respiratory motion of the lung, resulting in inadequate alveolar ventilation as well as imbalance of the V/Q ratio. At the same time, the accumulation of blood led to compression of the pulmonary vessels, which increased pulmonary vascular resistance, thus made matters worse for the preexisting condition. In this condition, the patient presented with unconsciousness, dyspnea and decreased blood pressure, and pulmonary hypertensive crisis (PHC) should be highly suspected. PHC is a clinical crisis caused by a rapid increase in pulmonary vascular resistance induced by various causes, with pulmonary artery pressure exceeding aortic pressure, leading to acute right heart failure. PHC is a significant challenge for anesthesiologists because of its high mortality rate, which is the most dangerous intraoperative complication of exacerbation of pulmonary hypertension. Risk factors associated with perioperative PHC include hypercapnia, hypoxia, fluid overload, painful, acidosis, and tracheal intubation, and requires immediate management to reduce pulmonary vascular resistance through pulmonary vasodilators, positive inotropic drug support, and identification of the cause [11]. The most immediate cause of exacerbation of pulmonary hypertension in this patient was progressive hemothorax, and therefore she may not be effective with drugs to lower pulmonary artery pressure, should use positive inotropic drugs and vasopressor to maintain circulatory stability while establishing cardiopulmonary bypass as soon as possible to remove the intra-thoracic clot and treat the underlying valve disease at the same time.

Conclusion

A patient with severe pulmonary hypertension present challenge to anesthesiologist because pulmonary hypertension is at high risk of developing a pulmonary hypertensive crisis during surgery. The general principle is to maintain circulatory stability, appropriate right heart preload, enhance right heart function, and avoid triggering factors for pulmonary hypertension.

Conflict of Interest

The author has read and approved the final version of the manuscript. The author has no conflicts of interest to declare.

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