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A Case Report of Right Pneumonectomy with a Focus on the Right Ventricular Function and Hemodynamic Management

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Abstract

Background: In patients undergoing pneumonectomy, intraoperative pulmonary and cardiac complications are the major cause of morbidity and mortality. Protective lung ventilation strategies may decrease the overall lung injury. Right, ventricular dysfunction may occur during the surgery and after the pneumonectomy, in the early postoperative period, with reduced RV ejection fraction and increased RV end-diastolic volume index, caused by increased RV afterload. Case report: We describe the case of a 28-year-old non-smoker female who underwent to a right pneumonectomy. The patient presented intraoperative hemodynamic instability and signs of RV dysfunction, requiring vasoactive amines and nitric oxide. Discussion: This article is intended to provide an overview of the anesthetic management for pneumonectomy including the hemodynamic management and considerations of the causes and management of right ventricular dysfunction.

Keywords

Anesthesia, Lung Resection Surgery, Nitric Acid, Pneumonectomy, Right Ventricular Dysfunction, Thoracic Surgery

1. Introduction

Pneumonectomy, which involves removing the entire lung, is typically done on people who have lung tumors. The anesthesiologist has a special set of difficulties during a pneumonectomy. The total surgical mortality is still high and varies from 5% to 13% [1], notably for the right pneumonectomy, within the first 30 days following the procedure [2]. Each patient should have their estimated respiratory, lung parenchymal, and cardiovascular reserves evaluated prior to sur-

gery [1]. Typically, in patients undergoing pneumonectomy, a preoperative FEV1 of 2L or a %FEV1 of above 80% reduces the occurrence of postoperative respiratory and cardiac problems [3].

This article provides an evidence-based update on anesthetic management for a patient undergoing a pneumonectomy and describes the main causes of ventricular dysfunction after major lung resection.

2. Case Report

A 28-year-old non-smoker female with no significant past medical history presented with persistent cough and fever at term pregnancy. The chest X-ray and computed tomography (CT) revealed a large tumor in the upper/middle right lung with the invasion of the right main bronchus and carina, causing complete atelectasis of the lung and compression of the superior vena cava (**Figure 1**).

The bronchoscopy biopsy showed lung adenocarcinoma. No lymph nodes or distant metastasis were identified. Cardiac function was normal. The echocardiography showed a cardiac ejection fraction of 67% and the estimated pulmonary arterial systolic pressure was 27 mmHg. Labor was induced at 39 weeks gestational age. One month later, she underwent an open right pneumonectomy. She was admitted to the operating theater, eupneic and hemodynamically stable. Before induction of anesthesia, standard monitorization was performed. An 18G thoracic epidural catheter was placed in the T6-7 interspace with an 18G Tuohy needle. Epidural analgesia was performed with a bolus dose (Ropivacaine 0.2%, 12 ml, morphine 2 mg). After preoxygenation by mask, anesthesia was induced intravenously with propofol 2 mg/kg, remifentanil 0.3 mcg/kg/min. Rocuronium 0.6 mg/kg was also administered to facilitate tracheal intubation with a 35F left-sided double lumen Carlens under direct laryngoscopy. The position of the double-lumen tube was verified by fiberoptic bronchoscopy. An arterial catheter was inserted into the left radial artery and connected to the FloTrac/Vigileo system. A central venous catheter was inserted into the left femoral vein. One lung

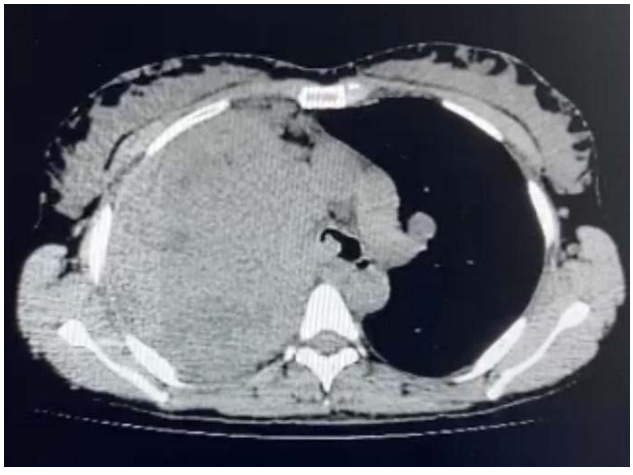

Figure 1. Computed scan showing a large tumor in the right lung invading the right main bronchus causing complete atelectasis of this lung.

ventilation (OLV) was performed with the following settings: FiO_2 = 60%, Vt = 5 ml/Kg, respiratory rate = 16/min, I:E = 1:2, using pressure control ventilation-volume guaranteed (PCV-VG) mode. Anesthesia was maintained with remifentanil, sevoflurane, and rocuronium when needed. Fluid management was goal-guided through hemodynamic parameters and also by urinary output and blood gas analysis. We administered 50 ml/kg of crystalloid solution and two units of packed red blood cells. Forced-air warming blanket and warm fluids were used to maintain core temperature above 36.0˚C.

The patient was placed in the left lateral decubitus position and right pneumonectomy was performed through a posterolateral incision on the right 5th intercostal space. The surgery constituted right pneumonectomy, hemidiaphragm suture, pericardium repair using a polypropylene mesh, and transarterial stapling of the pulmonary vein. A right chest drain was inserted. During the surgery, vasopressors in bolus (adrenaline and ephedrine) and infusion (vasopressin and noradrenaline) were required to maintain mean arterial pressure above 70 mmHg. Inhaled NO 20 ppm was administered during one-lung ventilation (OLV), after pulmonary artery clamping. Hemodynamic and oxygenation data were collected before and after inhaling NO.

The surgery lasted for about four hours and throughout the procedure, there was no episode of hypercapnia or hypoxia. After completion of the procedure, the double lumen tube was changed into a single lumen tube. She was transferred to the intensive care unit under mechanical ventilation receiving norepinephrine 0.03 mcg/kg/min; vasopressin 0.06 UI/min and NO 20 ppm. On the 3rd postoperative day, she was hemodynamically stable and extubated.

3. Discussion

Anesthetic management is crucial in reducing perioperative complications by understanding the technical aspects and physiological changes associated with pneumonectomy, especially regarding cardiopulmonary alterations [1].

Many factors related to both anesthesia and surgical intervention may cause hypotension. During the induction phase, anesthetic agents and thoracic epidural cause vasodilation. During the dissection phase, blood loss, compression from the tumor, surgical retraction, and also insensible losses may decrease venous return. Tight pericardial patch, acute right ventricle failure, and shifted mediastinum following closure may also occur. During the emergence phase when repositioning from lateral decubitus to the supine position, a cardiac herniation may occur. Many of these factors could be present in this report [4].

The right ventricular function is significantly impacted by pneumonectomy, which also reduces ventilatory performance. Following pneumonectomy, the right ventricle may enlarge and lose some of its function. Because the pulmonary artery is clamped during pneumonectomy and the entire pulmonary circulation volume is sent to the residual lung, the hemodynamics of the patient are complicated [4]. An increase in the right ventricular afterload results in an increase

in pulmonary artery pressure and pulmonary vascular resistance. After significant lung resection, this is one of the main causes of ventricular dysfunction [1].

Thoracic surgery involving one-lung ventilation puts the right ventricle (RV) at risk for damage, dysfunction, and eventual failure due to exposure to abrupt changes in preload, afterload, and contractility. Numerous intraoperative variables, including V/Q mismatch, hypoxemia, and hypercarbia during OLV, might influence the RV, causing pulmonary vasoconstriction and significant increases in RV afterload. The RV damage may also be influenced by the intraoperatively selected mechanical breathing mode. In the presence of underlying RV dysfunction, the severity of the dysfunction may be greater [2] [5] [6].

After the pulmonary artery is clamped to remove the lung, pulmonary hypertension may develop because the remaining lung's pulmonary vascular bed must carry all of the heart's blood. Right ventricular dysfunction and possibly failure could occur from this [2]. According to Okada *et al.*, the main cause of RV dysfunction following pneumonectomy is the rise of RV afterload as a result of rising pulmonary arterial pressure (PAP) and pulmonary vascular resistance (PVR). However, following a pneumonectomy, the pulmonary artery systolic pressure (PSAP) and the right ventricle (RV) enlarge, especially in the case of a right pneumonectomy [7].

Pneumonectomy, according to Sueshiro *et al.*, results in an abrupt decrease in the ventilation volume of the remaining lung as well as the pulmonary vascular bed after surgery, which causes right-sided heart failure due to acute pulmonary hypertension intraoperatively or postoperatively. Based on these results, the authors hypothesize that the development of postoperative heart failure is significantly correlated with fluid infusion volume, fluid balance volume, intraoperative total balance, blood loss volume, and blood transfusion [8].

During pneumonectomy, vasoactive substances can be utilized to improve RV performance. Norepinephrine has been demonstrated to enhance RV functionality and cardiac output. High doses, however, may raise pulmonary vascular resistance. Vasopressin's pulmonary vasodilating capabilities, which stimulate endothelial nitric oxide in the pulmonary vascular tree at low dosages (0.01 - 0.03 U/min), may make it superior to norepinephrine. At higher doses, this effect is reduced, resulting in a decrease in RV stroke volume and coronary vasoconstriction. Additionally, epinephrine, dobutamine, milrinone, and levosimendan can also improve RV contractility [5].

Right ventricular afterload reduction is the intended treatment in this situation, thus vasodilator therapy utilizing NO may be a viable choice [7]. Because of this, we have decided to employ the NO in this instance. Nitric oxide improves V/Q mismatch, oxygenation, RV systolic function, and mixed venous oxygen saturation by reducing PVR only in parts of the lung that are well ventilated [9].

Pneumonectomy frequently results in hypotension, which can be brought on by a variety of different causes. By far the most frequent mechanism resulting from mechanical pressure applied to the mediastinum during the dissection or

twisting of major arteries is a decreased venous return. If accessible, transesophageal echocardiography (TEE) can assess the function of the right and left ventricles during operatio [10]. Another factor that is thought to increase the risk of postoperative pulmonary problems is general anesthesia. Atelectasis, ventilation-perfusion mismatch, and hypoxemia can result from prolonged anesthetic exposure due to decreased surfactant production, increased alveolar-capillary permeability, diminished macrophage activity, and sluggish mucociliary clearance [11].

Fluid delivery is still a problem following pneumonectomy. According to studies, giving thoracic surgery patients more than 3 liters of intravenous fluid in the first 24 hours increases the chance of an acute lung injury. Right now, a moderately cautious approach should be taken into consideration because it can prevent fluid overload and reduce the danger of organ hypoperfusion and acute renal injury [12] [13]. Some pneumonectomies now employ goal-directed fluid therapy (GDFT), which uses dynamic hemodynamic parameters to target fluid administration. Most perioperative techniques to enhance patient recovery while reducing postoperative complications are thought to be built around GDFT [14]. It has been proposed that regulating the volume of fluid infusion and avoiding both fluid overload and hypovolemia with organ hypoperfusion, utilizing clinical measurement of cardiac preload to direct fluid therapy may lower the risk of pulmonary injury following surgery [13]. The GDFT strategy, as determined by pulse control analysis (Flotrac-Vigileo System), makes use of hemodynamic characteristics that forecast fluid responsiveness, which, in theory, allows for the optimization of cardiac performance and prevents the administration of excessive fluids [14]. Jiang Zhang *et al.* demonstrated that fluid therapy in thoracic surgery had been successfully guided by SVV. At the conclusion of the procedure, the PO_2/FiO_2 ratios were higher and the extubation time was shorter in the goal-directed therapy group who received a fluid bolus guided by SVV utilizing the Flotrac-Vigileo system [13].

Due to the loss of more than half of the lymphatic drainage in the right lung following a pneumonectomy, the risk of pulmonary edema in the left lung is greatly increased. Additionally, lymphatic drainage may be further hampered by RV dysfunction since increased central venous pressure decreases the lymphatic system's capacity for drainage [13].

After a pneumonectomy, particularly the right pneumonectomy, it is possible to have an abrupt herniation of the heart along with twisting of the major vessels and circulatory arrest. The issue arises right after the procedure, when the patient resumes the supine position [10].

4. Conclusion

Right pneumonectomy in particular is a difficult procedure with a high rate of morbidity and mortality. To improve patient outcomes, numerous management factors must be taken into consideration when managing anesthesia. Simple ac-

tions like reducing breathing pressures and treating acidosis, hypoxia, hypercarbia, and hypothermia can have a positive impact on outcomes.

Conflicts of Interest

The authors declare no conflicts of interest.

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