



Acute Pulmonary Edema Following Cardiopulmonary Bypass: A Case Report

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Abstract

Background: Acute pulmonary edema following cardiopulmonary bypass (CPB) is a serious complication, the etiology of which could be classified into two groups: cardiogenic and noncardiogenic.

Case presentation: We present a 42-year-old male with multiple pulmonary bullae scheduled for mitral valve and aortic valve replacement. Acute pulmonary edema occurred approximately 1 hour after weaning from CPB. The diagnosis of non-cardiogenic pulmonary edema was established with changes in pathophysiology and findings on ultrasound. The patient was promptly treated and transferred to the ICU uneventfully.

Conclusions: This case report highlights the changes in pathophysiology combined with ultrasound findings to establish a diagnosis in the operating room. We also emphasize the perioperative management of noncardiogenic pulmonary edema.

Keywords

Acute Pulmonary Edema, Cardiopulmonary Bypass, Case Report

Introduction

Pulmonary complications are common after cardiopulmonary bypass (CPB). Although the majority of pulmonary complications following CPB are of a minor nature, a small but significant number have serious consequences, including acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) [1]. Acute pulmonary edema after CPB is a catastrophic complication that requires prompt diagnosis and treatment.

Clinically, the etiology of pulmonary edema could be classified into two groups: cardiogenic and

noncardiogenic. Cardiogenic pulmonary edema after CPB generally correlates with ventricular dysfunction and increased pulmonary capillary wedge pressures, which can be detected by transesophageal echocardiography (TEE). However, noncardiogenic pulmonary edema is related to increased pulmonary capillary permeability secondary to inflammatory mediators [2,3].

Case Presentation

A 42-year-old male presented for mitral valve and aortic valve replacement. Preoperative chest radiograph and computerized tomography (CT) scan

Case Report

showed cardiomegaly and bilateral emphysema with multiple pulmonary bullae (**Fig-1**). Transthoracic echocardiography suggested severe mitral regurgitation, mild to moderate aortic regurgitation, mild tricuspid regurgitation, severe pulmonary hypertension, and a normal left ventricular ejection fraction of 60%. Anesthesia was induced smoothly, and the airway was secured without difficulty.

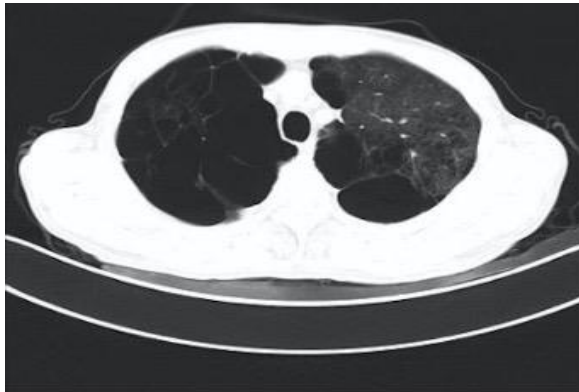


Fig-1: Chest CT scan before surgery showing cardiomegaly and bilateral emphysema with multiple pulmonary bullae

We applied a lung-protective ventilation strategy with a tidal volume of 6 ml/kg, a respiratory rate of 14/min, positive end-expiratory pressure (PEEP) of 6 cmH₂O, and 60% oxygen, with a peak inspiratory pressure (PIP) of 16-18 cmH₂O and an End-tidal CO₂ of 35-37 mmHg. Replacement of the valves was performed successfully, and continuous low-frequency ventilation was applied during the CPB period. The patient was weaned from CPB with a 0.05 µg/kg/min epinephrine infusion after a 215-minute CPB support.

We slowly infused protamine sulfate (3mg/kg) over

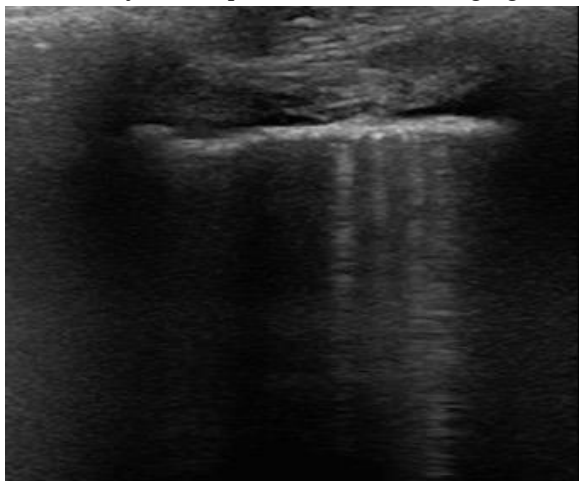


Fig-2: Lung ultrasound showing multiple B-lines

10 minutes to reverse heparin. Approximately 1 hour after the completion of the protamine infusion, PIP increased from 16 to 40 cm H₂O, SpO₂ dropped dramatically from 90% to 70%, and mechanical ventilation became almost impossible. We immediately hand-ventilated the patient with 100% oxygen. A massive outpouring of yellowish and slightly hemorrhagic fluid was aspirated from the endotracheal tube, which was collected for further laboratory examination (**Fig-2**).

We confirmed normal ventricular function, valvular function, and cardiac chamber size on TEE. The left atrial pressure was also measured within the normal range, fluctuating from 10 to 12 mmHg. Multiple Kerley's B lines were found on lung ultrasound, and pleural effusion was detected in the right chest cavity (**Fig-3**). Therefore, the diagnosis of cardiogenic pulmonary edema was excluded, and noncardiogenic pulmonary edema was suspected. We immediately administered 200 mg of hydrocortisone to mitigate the inflammatory response. A remarkable volume of fluid continued to be drained from the endotracheal tube, with a total volume of 360 ml. Goal-directed fluid replacement therapy and vasopressor support were also applied to stabilize blood pressure. Fortunately, the PIP gradually decreased to 23 mmH₂O, and SpO₂ increased to 90%. Finally, the patient was transferred to the ICU uneventfully.



Fig-3: Fluid collected from endotracheal tube

The chest radiography in the ICU showed bilateral infiltration and pleural effusion on the right side (**Fig-4**). The laboratory examination results of the fluid sample from the endotracheal tube showed a protein

Case Report

value of 4.3 g/dL and lactate dehydrogenase (LDH) value of 551 U/L. According to Light's criteria, the sample from the endotracheal tube consisted of exudative fluid [4]. The lung-protective ventilation strategy was continued in the ICU, and ventilatory support was gradually weaned. The patient was extubated on postoperative day 5 and discharged on postoperative day 19.



Fig-4: Chest X-Ray after surgery showing bilateral infiltration

Discussion

Acute pulmonary edema is among the most serious pulmonary complications after CPB. There are two fundamental classifications of pulmonary edema: cardiogenic pulmonary edema and noncardiogenic pulmonary edema. Although it is usually difficult to distinguish the etiology immediately in the operating room, changes in pathophysiology combined with findings on ultrasound could guide us toward a possible diagnosis. It is crucial for anesthesiologists to make the diagnosis and administer treatment promptly and accurately to prevent further deterioration and potentially fatal outcomes.

Knowledge of the underlying pathophysiological changes is the cornerstone for diagnosis. Cardiogenic pulmonary edema results from the rapid increase in hydrostatic pressure in the pulmonary capillaries, leading to transvascular fluid filtration. Non-cardiogenic pulmonary edema is manifested by increased vascular permeability of the pulmonary capillaries, resulting in an increased flux of fluid and protein into the lung interstitium and air spaces [2]. Patients undergoing cardiac surgery with CPB are vulnerable to both types of pulmonary edema.

In this patient, hemoconcentration and diuresis during CPB, as well as normal cardiac function and left atrial pressure after CPB, minimized the likelihood of cardiogenic pulmonary edema. Our diagnosis of non-cardiogenic pulmonary edema was further confirmed by the laboratory result of pulmonary effusion. The etiologies of noncardiogenic pulmonary edema are miscellaneous, including inflammatory response, protamine reaction, transfusion-related acute lung injury (TRALI), and pulmonary emboli. A protamine reaction often occurs rapidly, manifesting as pulmonary hypertension and right ventricular dysfunction [5-9]. However, in this patient, protamine infusion occurred over an hour ago, and TEE did not find significant changes in pulmonary artery pressure or right ventricular function. The possibility of TRALI was also extremely low because only autologous blood was transfused to this patient, although one case of autologous TRALI has been reported [10]. Normal right ventricular function and pulmonary artery pressure made a significant pulmonary embolism less likely. Therefore, given the long duration of CPB, we assume noncardiogenic pulmonary edema in this case most likely resulted from CPB-induced inflammatory response.

CPB is associated with a whole-body inflammatory response, which includes activation of complement, leukocytes, and endothelial cells with secretion of cytokines, proteases, arachidonic acid metabolites, and oxygen free radicals. It is assumed that pulmonary capillaries are more susceptible to this inflammatory response because all active or activating substances and cells generated during CPB transit via the pulmonary circulation. The lung capillaries are smaller in diameter than the average systemic capillaries, resulting in the preferential trapping of aggregates in the lungs, and a considerable pool of neutrophils is present in the lungs [11]. However, only a small part of the mechanisms involved is known, and further investigation is needed.

Although there are various causes of noncardiogenic pulmonary edema, the treatment is relatively established. Aggressive tracheobronchial suction is essential for clearing the high volume of pulmonary edema fluid, and protein analysis should be done on a sample of this fluid to confirm the diagnosis [12]. A

Case Report

lung-protective strategy of ventilation, fluid resuscitation, and other supportive treatments is suggested for noncardiogenic pulmonary edema. Potential protective interventions for noncardiogenic pulmonary edema include off-pump surgery, impregnation of the circuit with less biologically active substances, leukocyte depletion filters and ultrafiltration, and pharmacological agents such as steroids.

Conclusion

In summary, we present a case of acute noncardiogenic pulmonary edema following CPB. This case report highlights the changes in pathophysiology combined with findings on ultrasound to establish a diagnosis in the operating room. We also emphasize the perioperative management of noncardiogenic pulmonary edema.

Conflict of Interest

The authors have read and approved the final version of the manuscript. The authors declare no conflicts of interest.

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