



Venous Air Embolism During Laparoscopic Rectal Surgery: A Case Report of Successful Resuscitation

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Abstract

Venous air embolism (VAE) is a rare but potentially fatal complication of laparoscopic surgery, particularly in procedures requiring Trendelenburg positioning and pneumoperitoneum. This case report describes the successful resuscitation of a 68-year-old male who developed VAE during laparoscopic resection for recurrent rectal cancer. Ninety-four minutes after the start of surgery, the patient developed acute hypotension, bradycardia, hypoxemia, and a marked decrease in end-tidal carbon dioxide (ETCO₂). Bedside transthoracic echocardiography revealed abundant gas echoes within all cardiac chambers, confirming the diagnosis of VAE. Immediate management included repositioning to Durant's position, ventilation with 100% oxygen, intermittent external chest compressions, and vasopressor support. Despite the occurrence of transient ventricular fibrillation, sinus rhythm was restored within minutes, and the patient achieved full recovery without neurological or cardiac sequelae. This case highlights the critical importance of early recognition of VAE through integrated hemodynamic monitoring and echocardiography, and demonstrates the effectiveness of a multimodal resuscitation strategy in achieving favorable clinical outcomes. It also underscores the need for heightened vigilance and well-prepared emergency response protocols during high-risk laparoscopic procedures.

Keywords

Venous Air Embolism, Laparoscopic Surgery, Rectal Cancer, Resuscitation, Echocardiography, Case Report

Introduction

Venous air embolism (VAE) is a rare but potentially fatal intraoperative complication. Although its incidence is low across various surgical procedures, once it occurs it is often associated with extremely high mortality rates [1,2,4,7,8,11]. In laparoscopic surgery, due to the specific characteristics of pneumoperitoneum establishment, patient positioning, and surgical manipulation, the risk of VAE is significantly increased [1,2,9,10,12]. In recent years, with the widespread

adoption of minimally invasive techniques, laparoscopic surgery has been increasingly applied to complex procedures such as rectal tumor resection. However, these procedures frequently require patients to be placed in a head-down Trendelenburg position. This positioning increases the pressure gradient between the venous system and the right heart, thereby facilitating the entry of gas into the circulation [3,4,8,9]. In addition, continuous carbon dioxide insufflation during pneumoperitoneum elevates intra-abdominal pressure,

further increasing the likelihood of gas entering the venous system [1-4,9,10].

This case report aims to provide a detailed analysis of a case of VAE that occurred during laparoscopic radical resection for rectal cancer and was successfully resuscitated. The clinical features, diagnostic clues, and rescue experience are discussed to enhance clinicians' awareness and emergency response capabilities regarding VAE. Although existing literature has described the pathogenesis and clinical manifestations of VAE, its specific presentation during laparoscopic surgery remains insufficiently summarized. Through analysis of this case, we aim to provide reference for the early diagnosis and timely management of similar cases in the future, thereby reducing VAE-related mortality.

It is noteworthy that early diagnosis of VAE is extremely challenging because its clinical manifestations are often nonspecific and may be confused with other intraoperative complications such as allergic reactions, pneumothorax, or pulmonary embolism [2,4,7,9,10]. Therefore, improving awareness of VAE, particularly the identification of high-risk factors, is crucial for the prevention and management of this potentially fatal complication. This case report systematically analyzes the mechanisms of VAE occurrence, diagnostic approaches, and treatment strategies in combination with existing literature, emphasizing the key role of multimodal intervention in successful resuscitation and providing valuable guidance for clinical practice.

Case Presentation

Patient Information:

The patient was a 68-year-old male weighing 58 kg who was scheduled to undergo laparoscopic rectal tumor resection due to postoperative recurrence of rectal cancer. The patient had a significant surgical history, having undergone extended radical resection for rectal cancer with ileostomy 5 years earlier, followed by 14 cycles of chemotherapy and 48 sessions of radiotherapy. Preoperative evaluation revealed chronic obstructive pulmonary disease (COPD) with bullae formation. However, electrocardiography and echocardiography were normal, with a left ventricular

ejection fraction of 60%, indicating acceptable cardiopulmonary reserve. Nevertheless, the patient's complex medical history and prior abdominal surgeries may have increased the risk of intraoperative complications, particularly venous air embolism (VAE).

Surgical Procedure and Acute Event:

The surgery was performed under general anesthesia. Anesthesia induction was achieved using midazolam, propofol, sufentanil, and vecuronium. After successful endotracheal intubation, mechanical ventilation parameters were appropriately set, and anesthesia was maintained using a combined intravenous-inhalational technique.

Ninety-four minutes after the start of surgery (18:57), during the rectal mobilization phase, the patient was in a head-down lithotomy position (Trendelenburg position with a tilt angle greater than 30°), with pneumoperitoneum pressure maintained at 12 mmHg. At this time, the patient suddenly developed acute hemodynamic instability, manifested by a sharp decrease in heart rate from 57 to 39 beats per minute, a drop in blood pressure from 118/62 mmHg to 79/38 mmHg, a reduction in SpO₂ from 95% to 85%, and a decrease in end-tidal carbon dioxide (EtCO₂) from 30 mmHg to 23 mmHg. These abrupt changes indicated severe circulatory disturbance, necessitating immediate intervention.

Initial Management and Clinical Deterioration:

The initial diagnosis was vagal reflex hyperactivity. The surgical procedure was temporarily suspended, and 1 mg of atropine was administered intravenously to increase heart rate, along with a 20 µg bolus of epinephrine to support blood pressure. Ventilation was switched to 100% oxygen. However, the patient's condition did not improve significantly. The heart rate further declined to 21 beats per minute, blood pressure remained persistently low, and oxygen saturation recovered only to 92%. Beginning at 19:02, the patient developed short runs of ventricular premature beats and non-sustained ventricular tachycardia, suggesting myocardial hypoperfusion or increased cardiac strain. Arterial blood gas analysis performed at 19:03 revealed severe hypercapnia (PaCO₂ 79.4 mmHg), while EtCO₂ remained low. The PaCO₂-EtCO₂ gradient exceeding

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50 mmHg indicated severely impaired pulmonary gas exchange, further supporting the suspicion of gas embolism [1,2,4,7-9].

Diagnosis and Emergency Intervention:

Given the rapid deterioration of the patient's condition, venous air embolism (VAE) was highly suspected by the anesthesia team. To confirm the diagnosis, bedside transthoracic echocardiography was performed at 19:22, revealing abundant gas echoes in all cardiac chambers, including both atria and ventricles (**Fig-1**), thereby confirming VAE. A series of emergency

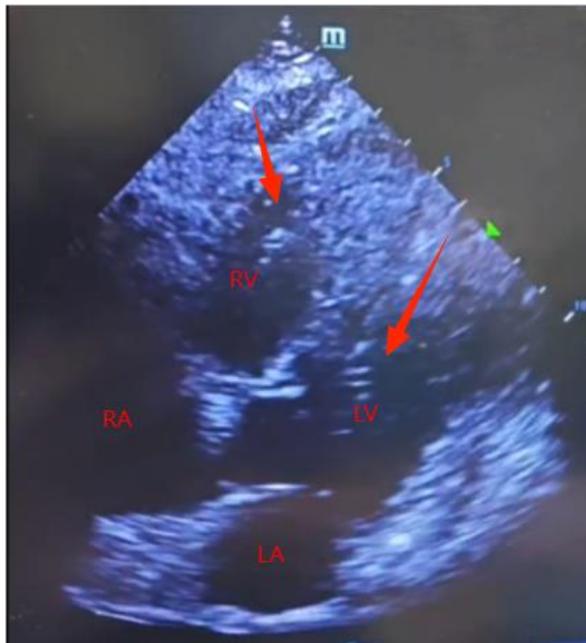


Fig-1:

Bedside transthoracic echocardiogram (apical four-chamber view). Abundant gas echoes (arrows) are seen within the right ventricle (RV) and left ventricle (LV). RA: right atrium; LA: left atrium.

interventions was immediately initiated, including repositioning the patient to the head-down left lateral decubitus position (Durant's position), continuous norepinephrine infusion to maintain blood pressure, and initiation of intermittent external chest compressions to mechanically disrupt intracardiac gas emboli and alleviate pulmonary artery obstruction. Concurrently, an attempt was made to aspirate gas from the right heart via a central venous catheter placed in the right internal jugular vein; however, no visible gas bubbles were obtained. At 19:24, the patient suddenly developed ventricular fibrillation. Immediate external chest compressions were performed, and defibrillation was prepared. Sinus rhythm was restored

approximately one minute later, with blood pressure recovering to 123/56 mmHg, SpO₂ increasing to 99%, and EtCO₂ returning to 29 mmHg, indicating stabilization of hemodynamic status.

Postoperative Recovery:

The patient was safely transferred to the ICU for continued monitoring postoperatively. On the morning of the first postoperative day, the endotracheal tube was successfully removed. The patient was conscious and alert, with no neurological symptoms such as headache, convulsions, or hemiplegia. Cranial CT showed no signs of air embolism. Cardiac ultrasound also showed no new segmental wall motion abnormalities, and cardiac injury markers were within normal limits. The patient remained hemodynamically stable during observation in the ICU, with no recurrence of arrhythmias or other severe complications. Follow-up after discharge showed good recovery with no long-term neurological or cardiac functional impairment.

Discussion

Analysis of High-Risk Factors:

The occurrence of venous air embolism (VAE) is often closely related to multiple high-risk factors. In this case, surgical positioning, pneumoperitoneum establishment, and tumor recurrence requiring reoperation collectively acted to significantly increase the risk of VAE [1,3,8]. First, the intraoperative use of a head-down lithotomy position (Trendelenburg position) lowers the pressure in the venous system below the level of the heart, creating a pressure gradient favorable for air entry into the right heart. Second, the establishment and maintenance of pneumoperitoneum in laparoscopic surgery further exacerbated this risk. High pneumoperitoneum pressure may compress intra-abdominal veins, promoting gas entry into the bloodstream through open venous defects [1-4]. Furthermore, the characteristics of repeat surgery for recurrent tumor cannot be ignored. Due to tissue adhesions and altered anatomical structure resulting from previous surgery and radiotherapy, blood vessels are more easily injured during tissue dissection, creating channels for gas entry. These factors superimposed, constituting the pathophysiological basis for VAE occurrence in this case.

It is worth noting that similar combinations of high-risk factors have been mentioned in other types of surgeries in the literature. For example, in laparoscopic hepatectomy, injury to hepatic veins is highly likely to lead to carbon dioxide gas embolism [5]. In vitrectomy, the occurrence of ophthalmic venous air embolism (OVAE) is closely related to the anatomical location of the eye and the air-fluid exchange procedure during surgery [6]. Therefore, the analysis of high-risk factors in this case not only helps understand the mechanism of VAE but also provides an important reference for risk assessment in other surgical types.

Diagnostic Challenges and Key Clues:

The early diagnosis of venous air embolism is challenging, primarily due to its lack of specific clinical manifestations, often resembling symptoms of other intraoperative complications such as allergic reactions, pulmonary embolism, or myocardial ischemia. In this case, the patient initially presented with hypotension, bradycardia, and hypoxemia, which could have been misdiagnosed as vagal nerve reflex hyperactivity or inadequate anesthetic depth. However, as the condition progressed, the significant decrease in end-tidal carbon dioxide (EtCO₂) and the dissociation between hypercapnia (elevated PaCO₂) shown by arterial blood gas analysis and low EtCO₂ became important clues suggesting VAE. This dissociation usually indicates severely impaired pulmonary perfusion rather than pure ventilatory dysfunction [1,2,4,7,8,10].

The application of bedside echocardiography played a key role in the diagnostic confirmation process in this case. By real-time observation of abundant gas echoes in the cardiac chambers, it not only confirmed the diagnosis of VAE but also provided a basis for further treatment. The value of this diagnostic tool has been confirmed in multiple studies, especially in emergency situations, where transesophageal echocardiography (TEE) can more clearly display the distribution of gas within the cardiac chambers [1,2,4,7-9,12]. Therefore, for patients with suspected VAE, combining the results of EtCO₂ monitoring, arterial blood gas analysis, and bedside echocardiography can significantly improve early diagnostic accuracy [1,9].

Discussion of Treatment Strategies:

The treatment strategies employed in this case fully demonstrate the importance of multimodal intervention in VAE resuscitation. First, positional adjustment is one of the key steps in emergency management. Placing the patient in a head-down left lateral decubitus position (Durant's position) not only helps reduce the amount of gas entering the right heart but also promotes gas floating upward to the right atrium, thereby reducing the degree of pulmonary artery obstruction [1-2,4,7-8,10]. Second, high-concentration oxygen therapy accelerates gas dissolution by increasing the partial pressure of oxygen in the blood while reducing further tissue damage from gas emboli [1,2,4,7-9]. Furthermore, the application of external chest compressions was particularly prominent in this case. By mechanically disrupting large gas bubbles in the right ventricle, chest compressions effectively improved pulmonary artery obstruction and restored circulatory stability [1-2,4,7-9].

The attempt to aspirate gas from the right heart via central venous catheter placement, although unsuccessful in this case, remains a treatment measure worth exploring [1,2,4,7-9]. Literature reports show that some authors even recommend attempting to aspirate gas through a central venous catheter to alleviate the condition. However, the effectiveness of this maneuver may be influenced by the size and location of the gas embolus and individual patient differences. Finally, the choice of vasoactive agents is also crucial. In this case, the continuous infusion of norepinephrine effectively maintained blood pressure, buying time for subsequent treatments [1,9]. Overall, the combined application of these treatment strategies laid the foundation for successful resuscitation in this case.

Lessons and Implications

Through in-depth analysis of this case, several important lessons and implications can be summarized. First, intraoperative monitoring requires multi-dimensional linkage, as an abnormality in a single parameter is often insufficient to indicate VAE. For example, a decrease in EtCO₂ should be combined with arterial blood gas analysis results and other clinical manifestations for comprehensive judgment to avoid delayed diagnosis [1,9]. Second, the importance of

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bedside echocardiography cannot be overlooked. As a rapid, non-invasive diagnostic tool, it can provide direct imaging evidence in emergencies, supporting rescue decision-making [1,2,4,7-9]. Additionally, the rescue process must be time-critical; any delay may lead to irreversible damage [1,2,4,7-9]. In this case, the relatively short interval from initial judgment to final diagnosis and the patient's favorable outcome sufficiently illustrate the importance of rapid response.

The establishment of multidisciplinary collaboration and standardized emergency protocols is a key direction for future clinical work. Close cooperation among anesthesiologists, surgeons, and critical care medicine teams enables the formulation and implementation of effective rescue plans in the shortest possible time [7]. Simultaneously, developing detailed prevention and emergency response plans for high-risk surgeries helps improve overall response capability. In summary, the lessons learned from this case provide valuable reference for handling similar complications in the future and also emphasize the critical role of continuous education and training in enhancing clinicians' emergency response capabilities.

Conclusion

This case report details a patient who developed venous air embolism (VAE) during laparoscopic radical resection for rectal cancer and was successfully resuscitated. This case presented typical clinical features, including acute circulatory failure, hypotension, bradycardia, hypoxemia, and a significant decrease in end-tidal carbon dioxide (EtCO₂). These symptoms were initially misdiagnosed as vagal nerve reflex hyperactivity, but with rapid clinical deterioration, VAE was confirmed through arterial blood gas analysis and bedside echocardiography. This process highlights the insidious and non-specific nature of VAE clinical presentations, with diagnostic difficulty further increased during laparoscopic surgery.

VAE, as a rare but potentially fatal complication, deserves particular attention in laparoscopic surgery. In this case, the patient being in a head-down lithotomy position with pneumoperitoneum maintained at 12 mmHg collectively constituted high-risk conditions for VAE. Furthermore, repeat surgery for tumor recurrence

may lead to altered local anatomy, further increasing the risk of the venous system being exposed to air. Therefore, in similar surgeries, high vigilance regarding these high-risk factors and the adoption of targeted preventive measures, such as optimizing positioning management, controlling pneumoperitoneum pressure, and strengthening intraoperative monitoring, are essential.

Correct diagnosis and timely intervention are key to successful resuscitation. In this case, bedside echocardiography played an important role by directly displaying abundant gas echoes in the cardiac chambers, providing reliable evidence for diagnosis. Simultaneously, the multimodal interventions taken, including head-down left lateral decubitus position (Durant's position), pure oxygen ventilation, intermittent external chest compressions, and attempted central venous catheter placement for aspiration, effectively improved the patient's hemodynamic status. Notably, chest compressions not only help mechanically disrupt gas emboli in the right ventricle but also alleviate pulmonary artery obstruction, thereby restoring cardiac output function.

Based on the experience from this case, the following recommendations are proposed for future clinical work: First, strengthen training on VAE awareness, especially for laparoscopic surgical teams, to improve their sensitivity to early warning signs. Second, promote the application of bedside ultrasound technology in intraoperative monitoring to facilitate rapid identification of potential gas embolism. Finally, establish standardized emergency response protocols, clarify the division of responsibilities among disciplines, and ensure rapid implementation of effective rescue measures in emergencies. In conclusion, through multidisciplinary collaboration and continuous quality improvement, VAE-related mortality can be reduced, ensuring patient safety.

Conflict of Interest

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

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