Probable Carbon Dioxide Embolism during Endoscopically Assisted Varicose Vein Stripping

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SUBFASCIAL endoscopic perforator vein surgery is a minimally invasive method of ligating perforating veins in the lower leg and correcting lower-extremity varicosities. It has recently gained popularity in the hope of decreasing patient morbidity associated with the traditional surgical approach to perforator ligation. A similar procedure, endoscopic saphenous vein harvesting, is used to obtain vein grafts in cardiac surgery. Analogous to laparoscopic procedures, carbon dioxide is insufflated to facilitate visualization. Although carbon dioxide embolism is a rare yet well-recognized complication of laparoscopic procedures, it has not been described with endoscopically assisted lower-extremity procedures. We describe a case of probable carbon dioxide embolism during endoscopically assisted lower-extremity vein stripping.

Case Report

A 67-yr-old man with chronic nonhealing venous stasis ulcers was scheduled for bilateral endoscopically assisted stripping of lower-extremity varicose veins. His medical history included severe peripheral vascular disease for which he had undergone several lower-extremity revascularization procedures without any complications. Physical examination was unremarkable.

Anesthesia was induced with propofol and fentanyl, and muscle relaxation was attained with cisatracurium. American Society of Anesthesiologists standard monitors were used, along with a neuromuscular blockade monitor. Intubation was easily accomplished, and proper positioning of the oral endotracheal tube was confirmed by auscultation and the presence of endtidal carbon dioxide. Anesthesia was maintained with sevoflurane (0.85-1.4% end-tidal concentration) in 66% N₂O and 33% O₂. Mechanical ventilation was adjusted to maintain end-tidal carbon dioxide between 30 and 36 mmHg. The patient was in the supine position.

The patient’s hemodynamics and respiratory parameters remained stable for the first 2 h of the surgical procedure, during which classic vein stripping was performed. For the endoscopic part of the operation, a 5-mm port (Stryker Endoscopy, Santa Clara, CA) was inserted through the superficial fascia of the medial aspect of the right calf. Blunt dissection was used to develop a potential space in the superficial posterior compartment of the calf (Spacemaker, General Surgical Innovations, Palo Alto, CA), which was then insufflated with carbon dioxide at 6 l/min (Electronic Endotlator; Karl Storz, Culver City, CA). Insufflation pressure and total amount of carbon dioxide used during the procedure were not documented. A second port was then placed to allow identification and ligation of venous perforators located between the superficial and deep venous systems. Total insufflation time was approximately 90 min.

Approximately 7 min after beginning the endoscopic dissection, spontaneous ventilatory efforts were noted on the capnogram (curare clefts). At this time, the patient was mechanically ventilated, not paralyzed, and end-tidal carbon dioxide was 32 mmHg. Approximately 15 s later, end-tidal carbon dioxide abruptly decreased to 13 mmHg (fig. 1). and severe bradycardia with frequent ventricular escape beats was noted on electrocardiogram. Oxygen saturation by pulse oximetry remained unchanged at 99%. Noninvasive blood pressure measurements were unobtainable, but weak carotid and femoral pulses were palpated. The surgeons were notified, insufflation was stopped immediately, and the patient was placed in a head-down position. Inspired oxygen concentration was increased to 100%, anesthetics were discontinued, and the patient was manually ventilated. Ephedrine (25 mg intravenously) was administered. Anterior chest auscultation confirmed the presence of bilateral, equal breath sounds. With these maneuvers, the blood pressure increased to 160/80 mmHg within 5 min of the initial decrease in end-tidal carbon dioxide, and end-tidal carbon dioxide returned to 30 mmHg.

Anesthesia was continued with sevoflurane in 66% N₂O and 33% O₂. Carbon dioxide insufflation was re-instituted, and surgery was completed endoscopically without further complications. At the end of the 5-h procedure, the patient awoke easily, and the trachea was extubated. Subsequent hospital course and recovery were unremarkable. The patient suffered no neurologic sequelae.

Discussion

Endoscopic subfascial ligation of perforating veins offers many advantages over traditional surgical approach to venous ligation. It allows more precise localization of perforating veins and the use of smaller incisions placed...
CASE REPORTS

Carbon dioxide embolism is a potentially lethal complication of laparoscopic procedures, although its incidence is low (15 of 113,253 gynecologic laparoscopies). Diagnostic criteria for gas emboli vary considerably in the literature, with the only absolute criterion being visualization of gas bubbles in the vasculature. Most cases are diagnosed based on clinical signs. The volume of gas and its rate of entry into the blood determines the clinical presentation of a gas embolism. Cardiovascular collapse, hypotension, and dysrhythmias have all been reported to occur with carbon dioxide embolism. The median lethal dose of carbon dioxide required to change end-tidal carbon dioxide is approximately 0.66 ml/kg. Because carbon dioxide is highly soluble in blood, it is rapidly absorbed from the bloodstream, and if embolization does occur, it is less likely to be fatal than if air or oxygen is used for insufflation.

In our case, the initial presentation of presumed carbon dioxide embolism was spontaneous ventilatory efforts in a nonparalyzed mechanically ventilated patient. This most likely occurred because of an increase in arterial carbon dioxide above the apneic threshold as a result of absorption of carbon dioxide into the bloodstream. A sudden decrease in end-tidal carbon dioxide and profound hypotension were then noted. Massive carbon dioxide embolism can obstruct pulmonary outflow, dramatically decreasing cardiac output and possibly resulting in right heart failure. The abrupt decrease in end-tidal carbon dioxide was likely a result of decreased cardiac output and increased dead space.

The diagnosis of carbon dioxide embolism under these clinical circumstances is a diagnosis by exclusion. Unless gas is aspirated from the right side of the heart, detected by precordial Doppler or transesophageal echocardiography, the diagnosis of venous carbon dioxide embolism can only be presumptive. Transesophageal echocardiography is the most sensitive method of detection of carbon dioxide embolism, whereas changes in end-tidal carbon dioxide, pulmonary artery pressure, and precordial Doppler are less sensitive.

Two previous reports of carbon dioxide embolism describe an initial increase in end-tidal carbon dioxide followed by an acute decrease. In this case, we did not observe this pattern. However, the sudden appearance of spontaneous ventilatory efforts suggests that arterial carbon dioxide was increased before the decrease in end-tidal carbon dioxide. The absence of increased end-tidal carbon dioxide may represent the inability of capnography to reflect accurately arterial carbon dioxide in the face of rapid, massive changes in arterial carbon dioxide.

Other causes of hemodynamic collapse that need to be considered included hemorrhage, tension pneumothorax, pulmonary thromboembolism, and anaphylactic shock. In this patient, the entry of carbon dioxide into the circulation was presumably through the perforating veins into the deep venous system. Rapid recovery of systemic circulation after minimal drug therapy without other manipulations helped to refute the other differential diagnoses. The solubility of carbon dioxide contributed to the rapid reversal of the clinical signs with treatment.

In conclusion, we present a patient with probable carbon dioxide venous embolism who developed profound hypotension and dysrhythmias with carbon dioxide insufflation during endoscopically assisted lower-extremity vein stripping. He was successfully treated with intravenous ephedrine with complete resolution of symptoms and excellent outcome.

Because endoscopically assisted lower-extremity procedures may gain popularity in the future, anesthesiologists should be aware of this possible complication. To identify potential factors that contribute to carbon dioxide embolism, further study is needed.
TRANSMISSION-related acute lung injury (TRALI) is the cause of 15% of all fatal complications of blood transfusion. The pathophysiologic mechanism is a specific antigen-antibody reaction involving donor antibodies specific for leukocyte antigens of the recipient in most cases. Neutrophils are activated and aggregation in small pulmonary vessels occurs, initiating the complement and cytokine cascade that leads to capillary leakage. Clinically, TRALI is characterized by hypoxia, respiratory failure, and a noncardiogenic pulmonary edema occurring during or shortly after transfusion. Because TRALI is clinically indistinguishable from acute respiratory distress syndrome, we present a case report in which a severe leukopenic reaction after the infusion of fresh frozen plasma (FFP) could be documented.

**Case Report**

A 58-yr-old man with carcinoma of the stomach was admitted to our hospital for gastrectomy. The patient had a 20-yr history of heavy smoking with chronic obstructive pulmonary disease, emphysema, and mild hypertension. Long-term medication consisted of β-sympathomimetics and nifedipine. Preoperative laboratory values, including white blood cell (WBC) count, were within the normal range.

**References**