Venous Oxygen Embolism Produced by Injection of Hydrogen Peroxide into an Enterocutaneous Fistula

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We report a venous oxygen embolism that occurred in a 66-yr-old man after 60 mL of 3% hydrogen peroxide was injected into a perianal fistula intraoperatively to locate its internal opening. The diagnosis was made after detecting hypoxemia, decreased end-tidal carbon dioxide tension, systemic hypotension, increased central venous pressure, and a new heart murmur. The patient recovered quickly and had no long-term sequelae. Oxygen embolism is a potentially fatal complication that can develop when hydrogen peroxide is used near venous spaces, and clinicians should be aware of the potential dangers when using this seemingly innocuous chemical.

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Hydrogen peroxide (H₂O₂) is an oxidizing chemical frequently used during surgery for wound irrigation and cleansing. In the presence of the ubiquitous enzyme catalase, H₂O₂ quickly decomposes to water and oxygen in the following manner: 2H₂O₂ → 2H₂O + O₂ (1). At standard temperature and pressure, each mL of 3% H₂O₂ elaborates almost 10 mL of O₂ (2). Surgeons have exploited the effervescent release of O₂ to locate the internal opening of enterocutaneous fistula tracts because when H₂O₂ is injected into the visible external opening of the fistula, O₂ bubbles percolate through the fistula tract and can be observed at the origin of the internal opening (3,4). In recent years, H₂O₂ has been endorsed as a contrast medium for the ultrasonographic assessment of perianal disease including fistula-in-ano and anovaginal fistulae (5,6).

However, the O₂ produced from H₂O₂ can cause significant morbidity and mortality when it has access to the venous circulation (7–13). Therefore, its routine use for fistula identification surgically or ultrasonographically may be unwise. We present the first case of oxygen embolism associated with the intraoperative injection of H₂O₂ to identify the internal opening of a fistula tract.

Case Report

A 66-yr-old, 50-kg man was admitted with a Crohn’s disease exacerbation. He had complex perianal disease that had been surgically treated many times. Magnetic resonance imaging revealed a large gas-containing abscess involving the base of the penis and the corpus cavernosum. The patient was brought to the operating room for total colectomy, end-ileostomy, incision and drainage of his perianal abscesses, and possible fistulotomy.

Monitoring consisted of standard monitors as well as an arterial line, central venous line, and an esophageal temperature probe/stethoscope. Anesthesia was induced with 250 μg fentanyl and 100 mg propofol IV. Tracheal intubation was facilitated with 70 mg rocuronium IV. Anesthesia was maintained with 2.0%–9.0% end-tidal desflurane in air/O₂ (Fio₂ = 0.5). The patient was placed in the lithotomy position, and a baseline blood gas analysis was performed.

The abscess involved the right bulbospongious muscle and corpus cavernosum. Multiple draining fistulae were identified in the perineum. A total of 60 mL 3% H₂O₂ was injected by hand in aliquots from a single syringe over 1 min into several of these fistulae. The foaming was observed in an attempt to identify their internal openings. Within 30 s, there was a precipitous decrease in end-tidal carbon dioxide tension (Petco₂) from 32 to 13 mm Hg. His blood pressure decreased from 140/65 to 80/50 mm Hg, and the oxygen saturation (SpO₂) decreased from 100% to 92%. His central venous pressure (CVP) increased from 8 to 22 mm Hg. A venous gas embolism (VGE) was suspected and the patient was immediately tilted 20° to the left. The Fio₂ was increased to 1.0, and the central venous line was aspirated. No gas was returned from the catheter. Esophageal auscultation revealed a loud, harsh, systolic/diastolic heart murmur and normal breath sounds. His ventilatory variables remained unchanged. No resuscitative drugs were required to maintain hemodynamic stability. Blood gases were sent serially.
for analysis, and showed hypoxemia and hypercapnia (Table 1). These changes resolved fully within 32 min.

His vital signs returned to baseline within 15 min. The new murmur disappeared within two hours. The patient emerged from anesthesia uneventfully, was tracheally extubated, and had no neurologic deficits or further complications.

Discussion

Ten mL of O₂ are released from every mL of 3% H₂O₂, and 60 mL of H₂O₂ was injected; therefore a large amount of O₂ could have embolized to the heart. The likely passage of the O₂ was from the fistula tract to the corpus cavernosum, where it had access to a venous plexus and traveled to the right heart and the pulmonary circulation, resulting in the constellation of decreased PaCO₂, systemic hypotension, increased CVP, and hypoxemia. The new murmur represented the classic "mill-wheel" murmur of VGE (14).

A differential diagnosis for the observed physiologic derangements would include air embolism (from the opened abscess cavity that was exposed to the atmosphere) and thromboembolism. Spontaneous venous air embolism is unlikely in this situation because the operative site was below the level of the patient’s heart, the patients legs were elevated in the lithotomy position after VGE, and that positions other than supine increase is facilitated when right atrial pressure exceeds left atrial pressure. As our patient had an acute infection, the transatrial passage of bubbles was unlikely because the H₂O₂ was forcibly injected, eliminating the need for any pressure gradient. Traditionally, the left lateral, head down position has been recommended to “trap” gas bubbles in the RV and thereby prevent their passage into the RV outflow tract and to direct arterial bubbles away from the cerebral circulation (16). However, in animal studies, both of these tenets have been questioned, as different positions did not affect hemodynamics, time to recovery, cerebral gas embolic events, or survival (16,17). An echocardiographic study showed that, despite the presence of RV air, no RV outflow tract obstruction occurred in any position after VGE, and that positions other than supine could be detrimental (18).

Systemic arterial gas embolism can result from VGE either by transpulmonary movement of gas bubbles (15) or via a patent foramen ovale (PFO), which is present in 20%–34% of the population (19). PFO passage is facilitated when right atrial pressure exceeds left atrial pressure. As our patient had an acute increase in CVP, the transatrial passage of bubbles was a possibility. Transpulmonary movement of gas may be facilitated by volatile anesthetics (20). Arterial gas embolism can cause focal neurologic deficits or diffuse encephalopathic states (14), and if either is detected postoperatively the presumptive diagnosis should be paradoxical gas embolism. These patients should be

<table>
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<th>Time</th>
<th>F₂O₂</th>
<th>pH</th>
<th>PaCO₂ (mm Hg)</th>
<th>Po₂ (mm Hg)</th>
<th>HCO₃⁻ (mmol/L)</th>
<th>CVP (mm Hg)</th>
<th>Pₑ₂CO₂ (mm Hg)</th>
<th>Alveolar dead space fraction*</th>
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<td>11</td>
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*Formula for alveolar dead space fraction: Dead Space/Tidal Volume = (PaCO₂ – PetoCO₂)/PaCO₂.

For (Table 1).

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treated in a hyperbaric facility (21) even if the diagnosis is delayed (22,23).

Because the gas that embolized in our patient was O2, ventilating the lungs with 100% O2 might actually have slowed the rate of egress from the body, as one of the major factors for gas removal is the pulmonary capillary-alveolar gradient. Ventilation with 100% O2 decreases this gradient and retards removal. However, with an acute gas embolism, action must be taken quickly, and switching to 100% O2 is justifiable, especially because other routes of O2 removal, such as plasma dissolution and metabolism, probably work to remove O2 bubbles. In our patient, the gas exchange abnormalities and hemodynamic changes resolved quickly, making it unlikely that ventilating the lungs with 100% O2 impaired recovery to a significant extent.

Conventional transanal ultrasound provides a correct assessment of fistula-in-ano in 62% of patients (5). H2O2-enhanced transanal ultrasound improves the accuracy to 95%, and is associated with a change in surgical management in 50% of cases (5). This diagnostic improvement is remarkable, but magnetic resonance imaging can attain similar accuracy with very minimal risk to the patient (6).

We report the first case of venous oxygen embolism caused by the injection of H2O2 into a fistula to locate its internal origin. Morbidity and mortality produced by H2O2 has also occurred during colonic irrigation (7), lumbar discectomy (8), stereotactic brain biopsy (9), wound packing/irrigation (10–12), and even after accidental ingestion (13). Several authors have recently advocated using H2O2 as a contrast agent for identifying fistula tracts ultrasonographically (5,6), which may increase the number of potentially life-threatening oxygen emboli that occur. Moreover, these emboli may happen in clinical areas without immediate access to resuscitative equipment and personnel, possibly reducing the chance of survival. We advise caution with the routine surgical usage of H2O2, especially near venous spaces.

References