

Venous Air Embolism due to Hydrogen Peroxide during Anal Fistulectomy under Saddle Block

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ABSTRACT

Hydrogen peroxide (H₂O₂) is used by surgeons due to its oxygen releasing effervescent properties to locate internal openings in fistulous tracts. The oxygen released is known to cause embolism when it enters venous circulation, particularly when it is forced into a closed cavity. The obstruction it causes to the blood flow can cause circulatory and ventilatory failure. It manifests clinically by rapid fall in EtCO₂, hypotension, rise in central venous pressure (CVP), and fall in oxygen saturation. We present a case of a male patient who underwent fistulectomy under a saddle block and later developed air embolism when H₂O₂ was forcefully pushed at the site of surgery.

Keywords: Fistulectomy, Hydrogen peroxide, Oxygen embolism, Venous air embolism.

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INTRODUCTION

Hydrogen peroxide (H₂O₂) is an oxidizing chemical widely used in surgery for the treatment of infected wounds, abscesses, and debridement of necrotic tissue. The effervescent O₂ bubbles released from H₂O₂ have an oxidizing effect on bacteria and viruses, thereby considering it as an effective method for removal of any contaminant. However, O₂ released from H₂O₂ can cause significant morbidity and mortality if it has access to the venous circulation, which occurs mostly when it is pushed forcefully into a closed cavity.¹⁻⁶ We report a case of venous air embolism (VAE) that occurred due to the use of H₂O₂ in a male patient posted for anal fistulectomy in a peripheral hospital with limited resources.

CASE DESCRIPTION

A 42-year-old, 70-kg male patient, diagnosed as a case of fistula-in-ano, was planned to undergo fistulectomy. The patient was a known hypertensive on regular medications and had no previous surgeries. His systemic examination during the preoperative anesthetic evaluation revealed no abnormality, and his vital parameters were within normal limits. His metabolic equivalents (METs) were above 5. Airway examination was normal. He was accepted in American Society of Anaesthesiologists (ASA-II) to undergo anal fistulectomy, and a written informed consent was obtained for a saddle block.

On the day of surgery, an 18-gauge intravenous cannula was secured, and monitoring was established according to the American Society of Anesthesiologists standards. However, capnography monitoring was not available in this operating room. After preloading the patient, Saddle block was given using 1.5 mL of 0.5% bupivacaine heavy at L3-L4 level. The patient was kept in sitting position for 15 minutes, and all monitors were attached for continuous monitoring. After checking level of anesthesia, the patient was placed in lithotomy position.

Per-rectal examination was done by surgeon and an external opening of fistula was seen at 9 o'clock position. Since the surgeon was unable to feel the internal opening, he attempted to locate it using a blunt malleable fistula probe and feeding tube. On failing to do so, the diagnosis was changed from fistula-in-ano to a

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sinus. The surgeon then decided to excise the sinus tract, which was completed successfully. Thereafter, 30 mL of 1.5% hydrogen peroxide was pushed forcefully by using a 20-mL syringe to clean the deep excised wound. H₂O₂ was trapped into a closed cavity which was visualized by the swelling around the wound. Immediately, the patient started coughing and complained of difficulty in breathing. The respiratory rate increased from 14-16 breath/minutes to 30-35 breath/minutes, heart rate increased from 60-70/minutes to 130-140/minutes. Nothing remarkable was heard on lung auscultation; however, on cardiac auscultation, a loud, harsh murmur was heard during systolic and diastolic phases of the cardiac cycle. Unfortunately, end tidal carbon dioxide (EtCO₂) was not available.

At this time, SpO₂ decreased rapidly from 98% on room air to below 90%. His blood pressure decreased from 126/78 mm Hg to 66/40 mm Hg. Operative procedure was stopped, and venous air embolism was suspected. Patient was tilted to 20° toward the left in Trendelenburg position, and 100% oxygen via a face mask connected to the Bain's circuit was instituted. Intravenous fluid bolus of 300 mL normal saline and Inj. Mephentermine in boluses of 6 mg were given. Right internal jugular vein cannulation site was being prepared when hemodynamic parameters started to improve.

After 5-7 minutes of hemodynamic instability, his blood pressure improved to 118/66 mm Hg, saturation started to

improve to 98–100% on facemask, heart rate took 10 minutes to normalize, and after 15 minutes, SpO₂ improved to 98% on room air. Once the patient became hemodynamically stable, a 12-lead electrocardiogram and chest radiograph were performed, and both the investigations were normal. Thereafter, wound dressing was done, and the patient was shifted to intensive care unit for continuous monitoring. Cardiac enzymes, troponins, were checked after 6 hours of incident and were within normal limits as well. The remaining course in the hospital was uneventful, and the patient was discharged after 5 days.

It is pertinent to mention that this incident took place in a peripheral remote hospital with limited resources. Blood gas analysis, lower limb Doppler study, and 2D echocardiography could not be performed immediately. These investigations were performed at a later date and were normal.

DISCUSSION

Hydrogen peroxide (H₂O₂), in the presence of enzyme catalase, decomposes to water and oxygen. Each mL of 3% H₂O₂ releases almost 10 mL of oxygen. Surgeons have utilized the effervescent release of oxygen to locate internal openings of fistulous tracts. 30 mL of H₂O₂ was injected under pressure into a closed cavity, which may have caused a large amount of oxygen to embolize to the right heart and then to the pulmonary circulation. This may have resulted in hypotension and hypoxemia and should have also resulted in decreased EtCO₂ with increased central venous pressure (CVP).

Venous air embolism arises when a pressure gradient allows air to enter the blood stream and subsequently obstruct the blood flow. Iatrogenic procedures are the main causes of VAE, the most common being vascular access-related and certain surgical procedures.⁷ The physiological derangements observed led us to believe that gas embolism occurred due to entrainment of oxygen under pressure in the closed cavity. Spontaneous gas embolism is unlikely, since the site of surgery was below the level of the heart (i.e., the patient was in lithotomy position). Thromboembolism is unlikely as well, since the symptoms subsided in rather quick time. A thrombus large enough to cause hypoxemia and hypotension would have a continued effect on the circulatory system.

Venous air embolism can also result in arterial gas embolism, either by transpulmonary movement of gas bubbles or by patent foramen ovale.⁸ This can cause end-organ ischemia and present as focal neurological deficits, myocardial infarction, or limb ischemia. If these are detected, then paradoxical embolism may be considered.

The standard treatment of gas embolism is applicable to oxygen embolism as well, with attempts made to reduce the air embolus size and prevent its spread into arterial circulation.⁹ Repositioning of the patient by Durant's maneuver, placing the patient in left lateral decubitus and Trendelenburg position, will allow the air to move out of the right ventricular outflow tract (RVOT).^{10,11} The pressure gradient between the surgical site and the right atrium should be removed by repositioning as well. But this was inconsequential, since H₂O₂ was pushed under pressure, so clearing the surgical site by saline should be attempted in such cases. Aspiration of the gas bubble can be attempted via right internal jugular central venous cannulation, which is supported by several case reports.¹²

The rate of elimination of circulating oxygen depends on the partial pressure difference between capillaries and alveoli.

Ventilating the lungs with 100% O₂ might slow this egress from the body, since with 100% O₂, this gradient is decreased. However, in such acute cases, supplementation with 100% O₂ is justifiable, particularly when many patients are reported to have recovered fully with such treatment. Elimination of oxygen can also occur by other mechanisms, such as metabolism and absorption in the peripheral tissues. Patients may also require hemodynamic support with intravenous fluid boluses and vasopressors.

CONCLUSION

Such scenarios do present itself in areas with limited resuscitative resources, and the rapid response with early recognition probably helped in the patients' survival. Caution should be exercised while using H₂O₂ near the venous spaces, and it should be mandatory to have EtCO₂ monitoring available in all operation theaters. Lack of imaging modalities at the peripheral hospitals prevents clinicians from making a definitive diagnosis and may falsely lead to an incorrect conclusion. Also, training in four-chamber cardiac views with basic ultrasonographic techniques may prove helpful in such scenarios.

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