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Oxygen embolism after hydrogen peroxide irrigation of a vulvar abscess

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We report a case of venous oxygen embolism in a 33-yr-old healthy woman after irrigation of a vulvar abscess with 25 ml of 3% hydrogen peroxide. Venous oxygen embolism was diagnosed by the development of sudden hypoxia associated with a decrease in end-tidal carbon dioxide concentration from 5.3 kPa to 3.2 kPa, and a 'mill-wheel' sound on cardiac auscultation soon after injection of the solution. The patient responded to corrective treatment including the Trendelenburg position and 100% oxygen. She made an uneventful recovery. We discuss the possible causative mechanism of this embolism, the different diagnostic methods, and the controversial aspects of available treatments. We emphasize that hydrogen peroxide is a dangerous and unsuitable agent for routine wound irrigation and debridement.

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Hydrogen peroxide (H_2O_2) is commonly used for the treatment of abrasions, superficial wounds and abscesses. The release of effervescent oxygen bubbles and their oxidizing effects on bacteria and viruses,[1] are normally considered as effective methods for the mechanical removal of any contaminant. However, despite its apparently harmless mechanism of action, H_2O_2 has been implicated in several fatal or near-fatal complications.[2–6] Injected into enclosed body cavities or open wounds, H_2O_2 can provoke venous and arterial oxygen embolism. We describe a case of venous oxygen embolism which occurred during drainage of an infected cyst of the labia majora. This is the first report of an oxygen embolism occurring during a gynaecological surgical procedure.

Case report

A 33-yr-old healthy woman was admitted during the night with a large and painful abscess of the right labia majora. In the morning, after midazolam 7.5 mg (oral premedication), she was taken to the operating theatre. Monitoring included ECG (lead II), non-invasive arterial pressure, pulse oximetry and capnography. Anaesthesia was induced with propofol 150 mg and alfentanil 500 μg and maintained with 1.5–2.5% sevoflurane, 40% oxygen, and nitrous oxide. The patient was breathing spontaneously *via* a face mask. The initial oxygen saturation (Sp_{O_2}) was 100% with an end-tidal carbon dioxide tension ($P\mathrm{E}'_{CO_2}$) of 4.9 to 5.3 kPa. A purulent collection was drained out of the mass and 25 ml of a 3% hydrogen peroxide solution was syringed under pressure into the abscess. Vigorous bubbling was seen at the cavity opening.

Approximately 2 min later, the Sp_{O_2} decreased rapidly to 89% and the $P\mathrm{E}'_{CO_2}$ to 3.2 kPa, without any change in arterial pressure or pulse rate. To ensure adequate ventilation, the patient was given succinylcholine 75 mg, intubated and manually ventilated.

The anaesthetic and breathing system were immediately checked to exclude any malfunction. On auscultation of the lungs, nothing abnormal was detected, but a 'mill-wheel' sound could be heard on auscultation of the heart. The patient was placed in the Trendelenburg position, anaesthetic gases were discontinued and 100% oxygen was administered. Haemodynamic variables remained normal. The operation ended quickly, and after a few minutes the 'mill-wheel' murmur could no longer be detected. Sp_{O_2} and $P\mathrm{E}'_{CO_2}$ returned to their preoperative values and the patient was extubated without incident. Arterial blood gas analysis showed pH 7.32, Pa_{O_2} 12.2 kPa and Pa_{CO_2} 6.7 kPa on a FI_{O_2} of 0.4.

A chest radiograph, an electrocardiogram and a 2D transthoracic echocardiography were performed within 30 min of the incident. They were all normal. A second chest radiograph and a ventilation/perfusion lung scan were performed a few hours later and failed to demonstrate any sign of aspiration or pulmonary thromboembolism.

After close observation in the recovery room for 24 h, the patient showed no signs of any pulmonary complication and was discharged home.

Discussion

H_2O_2 rapidly decomposes to water and oxygen,[1] and is therefore often considered to be a harmless and user-friendly antiseptic and cleansing agent. However, since 1 ml of 3% H_2O_2 produces 9.8 ml oxygen, serious complications can arise if this is liberated in the vascular bed.

How such oxygen bubbles enter the capillaries remains unclear. In one study,[7] it was suggested that the high heat of decomposition of H_2O_2 favoured tissue disruption and enabled oxygen bubbles to penetrate into the vascular bed. But in another study,[8] the authors noted that subcutaneous injection of two or three times the volume of gaseous oxygen produced by a lethal dose of H_2O_2 was possible without killing laboratory rats. The authors concluded that embolization was caused by oxygen bubbles being released within the vascular bed by absorbed H_2O_2, rather than by penetration of the capillaries by gaseous oxygen. This may explain why many reports underline the danger of injecting H_2O_2 under pressure in closed or semi-closed cavities.[3 5 6] It may force H_2O_2 into blood vessels, rapidly producing critical amounts of gaseous oxygen. In our case, 25 ml of 3% H_2O_2 was probably sufficient to trigger a venous gaseous embolism, since it was injected under pressure into a semi-closed space which has extensive venous drainage.[9]

Once produced, the venous oxygen bubbles are carried to the right heart where they can impede cardiac output, or are trapped in the lungs, with a spatial distribution depending on their buoyancy and the flow dynamics within the pulmonary circulation.[10] If the lungs are severely overloaded with gas, bubbles may also pass through the pulmonary filter and increase the likelihood of coronary or cerebral vascular obstruction.[11]

Early diagnosis of venous gas emboli can be made by precordial Doppler, transthoracic and transoesophageal echocardiography, the latter detecting as little as 0.02 ml

kg^{-1} of air.[12] Less sensitive are the presence of a decreased $P_{E'CO_2}$, an increased mean pulmonary arterial pressure, a 'mill-wheel' murmur, hypoxaemia, hypercapnia and hypotension.[12 13] In our case, the diagnosis was made by the presence of sudden and reversible hypoxaemia associated with a decreased $P_{E'CO_2}$ and a transitory mill-wheel sound on cardiac auscultation, soon after application of H_2O_2.

Most cases[14] of gas embolism involve air (neurosurgical procedures, central venous lines, diving, extracorporeal circulation), or carbon dioxide (laparoscopic procedures). The usual preventive measures and treatments for air embolism are also recommended for oxygen embolism. They include flooding the wound with saline, increasing right atrial pressure by military antishock trousers, or positive end-expiratory pressure ventilation and avoiding volume depletion.[4] If an embolism is suspected or detected, it is recommended that the patient is placed in the Trendelenburg position to entrap bubbles in the right ventricle and reduce the rate of embolization.[2] However, in the presence of a patent foramen ovale which is present in 25–30% of the population, increased right atrial pressure can cause arterial embolization. Further treatment includes aspiration of gas from the right side of the heart through a central venous line, and discontinuation of nitrous oxide (N_2O).[2 3] In the event of cardiovascular collapse, cardiopulmonary resuscitation should be initiated. Finally, if neurological damage following arterial gas embolism is diagnosed, hyperbaric oxygen therapy is suggested.[15]

However, if we consider specifically embolism due to oxygen and H_2O_2, some of these measures may be regarded as controversial. For instance, since H_2O_2 might be partially forced into the vascular bed at the time of injection and before it vaporizes,[8] flooding the wound with saline will not prevent the risk of gas entrapment within the capillaries.

As N_2O is more soluble in plasma than oxygen, stopping the N_2O and ventilating the lungs with 100% oxygen is also recommended to prevent any oxygen bubble expansion. But one of the most likely mechanisms of gas elimination is molecular diffusion directly across the arteriolar wall into the alveolar spaces, which depends on the existence of a partial pressure difference between the capillaries and the alveoli.[16 17] In the case of oxygen bubbles within the pulmonary circulation, if 100% oxygen is applied to the alveolar space, the partial pressure difference theoretically falls to zero, preventing any excretion. As most patients treated with 100% oxygen have, however, recovered without long-term complications after oxygen emboli, there are probably other mechanisms of elimination such as metabolism and absorption, mainly in peripheral tissues. Ventilating the lungs with 100% oxygen can still be considered necessary, since the cause of the hypoxia and fall in $P_{E'CO_2}$ may not always be clear initially.

We conclude that hydrogen peroxide is a potentially dangerous substance, particularly when applied to vascular closed cavities. The risk of oxygen embolization together with its tendency to cause oxidative tissue damage makes it an unsuitable agent for routine wound debridement.

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