We report a case of a suspected oxygen embolus immediately following hydrogen peroxide irrigation during orthopaedic surgery. A 69-yr-old female suffered reversible cardiovascular collapse immediately following the introduction of hydrogen peroxide to the femoral shaft. We highlight a potential adverse effect of this commonly used chemical, potential risk factors and patient management.

Introduction

Hydrogen peroxide is commonly used during orthopaedic procedures to clean bone surfaces and improve adhesion of cement and prostheses in major joint surgery. Its use is also well established in 'open' procedures for tumour resection and irrigation of infected cases.

Report

A 69-yr-old female was admitted following a fall at home during which she sustained a fractured neck of femur and fractured distal radius. She was scheduled to have a total hip replacement and open reduction and internal fixation of her wrist the day after the accident. A fascia iliaca catheter was inserted and 0.125% bupivicaine infusion commenced at 8 ml.hr⁻¹ for pre-operative analgesia.

She was in general good health and had been independent, being a full time carer for her husband. Her medications included aspirin, amitryptiline, simvastatin, tramadol, solofenacin and terbinafine. Initial investigations were unremarkable with haemoglobin (Hb) 112 g.l⁻¹ and ECG showing sinus rhythm.

The fascia iliaca catheter was topped up with 30 ml 0.25% plain levo-bupicaine with the patient awake. General anaesthesia was induced with fentanyl 100 mcg, propofol 50 mg, atracurium 35 mg, the trachea was intubated and anaesthesia maintained with isoflurane in a 50:50 oxygen:air mixture. Metaraminol 1 mg was given immediately following anaesthetic induction because the blood pressure decreased from 160/92 mmHg to 95/56 mmHg but thereafter, cardiovascular stability was maintained. She received 1l Hartmann's solution, tranexamic acid 1 g, teicoplanin 400 mg and morphine 10 mg during the first 90 minutes of surgery.

After 1 hr 45 min the acetabulum was washed with hydrogen peroxide and there was a drop in systolic blood pressure from 120 to 90 mmHg, with no change in heart rate or oxygen saturation. This responded to metaraminol (3 x 0.5 mg) boluses, and remained stable during introduction of cement into the acetabulum. Hb reading on a haemocue reading was 96 g.l⁻¹.
Thirty minutes later, hydrogen peroxide was introduced to the femoral shaft whereupon the oxygen saturation trace disappeared but the ECG remained at 80 beats per minute. Non-invasive blood pressure was unrecordable (although a carotid pulse was palpable), and end-tidal carbon dioxide decreased from 4.3 kPa to 3.7 kPa. Airway pressures remained unchanged and ventilation continued as before.

Surgery was paused, the cement which had been prepared but not introduced, was discarded, while supportive management was commenced. This included 100% oxygen, continued positive pressure ventilation, ephedrine 30 mg, metaraminol 2.5 mg, Hartmanns solution 1l, and the commencement of a phenylephrine infusion. Haemocue reading taken at this time showed that the Hb had decreased to 78 g.l\(^{-1}\) and two units of red cells were administered. An arterial catheter was also inserted.

Within 15 min, cardiovascular stability had returned at which point the decision was made to continue surgery. The cement was introduced to the femoral canal without incident and the total hip replacement was completed with no further problems. The decision was made to postpone fixation of the fractured wrist.

At the time of the collapse, no rash was present, her chest was clear, airway pressures were normal, there were no ECG changes and an initial arterial blood gas showed pH 7.37, PaCO\(_2\) 5.28 kPa, PaO\(_2\) 42.3 kPa, bicarbonate of 22.5 mEq.l\(^{-1}\), with a base excess of -2.4 mmol.l\(^{-1}\). Post operatively the 12 lead ECG was unchanged and there was no troponin rise at 12 hr. The remainder of her postoperative course was otherwise unremarkable.

Two days later, she returned to theatre for open reduction and internal fixation of her wrist fracture, following which she had an episode of shortness of breath. A computed tomography pulmonary angiogram was performed, which showed bilateral basal atelectasis but no evidence of a pulmonary embolus.

**Discussion**

We postulate that the introduction of hydrogen peroxide into the bony cavity caused an oxygen gas embolus resulting in cardiovascular collapse. This is the first example we could find of this reaction occurring during orthopaedic surgery. Hydrogen peroxide is known to effervesce and release oxygen at a volume of 10 ml oxygen for every 1 ml of 3% hydrogen peroxide used \([1,2]\). Release of a large volume of oxygen into a highly vascular enclosed space would explain the rapid onset of this clinical syndrome and swift resolution.

Bone cement implantation syndrome in orthopaedic surgery \([3,4]\) has a presentation similar to that mentioned here, but it is our belief that this was not a contributing factor on this occasion. The timing of the initial deterioration occurred immediately following the first irrigation with hyrdogen peroxide and bore no temporal relationship with use of the bone cement. The peri-arrest episode occurred immediately after washing the femur with hydrogen peroxide (at which point the cement had not been used). The highly vascular region and enclosed space of the femur, as compared with the acetabulum, would explain the more severe deterioration during the second irrigation. Finally, there was no adverse response to cementing of the femur for insertion of the prosthesis, which, if bone cement implantation syndrome was the cause, we would have expected to have caused a clinically significant deterioration.

The speed of patient recovery would also support our diagnosis of oxygen embolism. Although bone cement implantation syndrome is poorly understood, there are reports demonstrating that increases in pulmonary artery pressure, caused by emboli, can persist for up to 24 hr. Regarding case reports relating to bone cement implantation syndrome, none mention such a rapid recovery as occurred in our patient. The few case reports that cite hyrdogen peroxide as a problem, describe a rapid resolution of symptoms \([2,5,6]\).

On discussion with the orthopaedic team, we have agreed that, in the future, if there is any patient instability with hydrogen peroxide use in the acetabulum, the need to use it in the femoral canal
should be decided on a case-by-case basis. Following this episode we have changed our practice to pre-emptively administer vasopressors when hydrogen peroxide is used in the femoral canal, if the patient exhibits any instability.

This case report highlights the potential pitfalls associated with hydrogen peroxide use in routine orthopaedic practice, specifically in regard to enclosed cavities, and the role of pre-emptive use of appropriate vasopressors to prevent cardiovascular instability or collapse in susceptible patients.

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Competing Interests

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References