

Cerebral arterial gas embolism after pre-flight ingestion of hydrogen peroxide

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Abstract

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Cerebral arterial gas embolism (CAGE) is a feared complication of ambient depressurisation and can also be a complication of hydrogen peroxide ingestion. We present an unusual case of CAGE in a 57-year-old woman exposed to both of these risk factors. We describe her subsequent successful treatment with hyperbaric oxygen, despite a 72-hour delay in initial presentation and diagnosis, and discuss the safety of aero-medical transfer following hydrogen peroxide ingestions.

Key words

Hyperbaric oxygen therapy; aviation; barotrauma; barometric pressure; case reports

Introduction

Cerebral arterial gas embolism (CAGE) is a documented but rare complication of hydrogen peroxide (H_2O_2) ingestion.¹⁻⁷ H_2O_2 is a readily available, clear, colourless, oxidizing agent found in different concentrations. Concentrations of approximately 35% are used for bleaching hair. There were 396 H_2O_2 exposures recorded by Australian National Poison Information Centres in 2013. Of these, 92% were recorded as accidental and only 9% where the concentration recorded was greater than 10% (unpublished data, with permission from the New South Wales, Queensland and Western Australia Poison Information Centres). Un-dissociated H_2O_2 undergoes rapid absorption across gastrointestinal mucosa and into the portal circulation. At standard temperature and pressure 1 ml of 35% H_2O_2 reacts with tissue catalases to liberate 100 ml of oxygen.⁸ The volume of the gas emboli will be increased by a further reduction in ambient pressure, such as occurs with flying.

We report an unusual case of a patient who developed CAGE after accidental ingestion of H_2O_2 just prior to boarding an international flight. The patient gave written permission to report her case.

Case report

A 57-year-old retired hairdresser presented to an Australian Metropolitan Emergency Department with a two-day history of nausea and vomiting, abdominal pain, confusion and balance problems following a return flight from Malaysia. She was usually well, apart from well-controlled, late-onset insulin-dependent diabetes mellitus.

Two days prior to her presentation, the patient had been visiting her relatives in Malaysia. Whilst there, she had mixed up some hair perming solution in a plastic water bottle and placed it in her handbag to give to her sister. After celebrating Chinese New Year, she rushed to the airport to return to Australia. On passing through airport security an

official reminded her that she could not take water onto the plane. Forgetting the bottle contained hair perming solution she took a "big gulp". The metallic taste immediately alerted her that she had ingested some of the perming solution; however, keen to get home she discarded the bottle and boarded the aircraft. Once on the plane she began to feel a burning sensation to her oropharynx and epigastrium. As the aircraft took off and gained altitude she began to vomit profusely and later described frothy "bubbles" in the vomit.

Whilst attempting to go to the aeroplane toilet she described feeling generally confused, had difficulty balancing and was unable to walk unaided. She was diagnosed with gastroenteritis and intravenous fluids and an antiemetic were administered during the flight. Interestingly, she described her confusion as improving on descent. On arrival to Australia, she refused a pre-arranged ambulance assuming that her symptoms would continue to improve.

Her symptoms persisted over the next 24 hours; she still felt generally confused and described poor balance. She was unable to eat or drink and developed diarrhoea and right-sided abdominal discomfort. She saw her general practitioner who arranged transfer to the local emergency department. There, she was treated for dehydration and corrosive oesophageal injury, with normal routine blood tests, and admitted under the medical team for blood sugar control and possible endoscopy. A CT scan of her abdomen was performed which did not show a clear cause for her pain but did show "mild ascending colonic thickening of uncertain significance". No portal or other gas emboli were demonstrated at that time. The following day, she had ongoing unexplained neurological symptoms and a toxicology review raised the possibility of CAGE in the setting of H_2O_2 ingestion and she was transferred for consideration of hyperbaric oxygen.

On arrival at the State Hyperbaric Unit, examination revealed a listless patient who had a flat affect, slow speech and was unable to stand unaided for more than a few seconds. She

had some subjectively altered sensation of her right upper limb and weakened hip flexion bilaterally. She scored 29/30 on a mini mental state examination but slow answers and difficulty writing were noted. It was felt her symptoms and signs were consistent with a diagnosis of CAGE.

She was treated with hyperbaric oxygen (HBO) to a pressure of 284 kPa on a US Navy Treatment Table 5 (USN T5). At 284 kPa, her symptoms improved and within 15 minutes the patient stated that her vision had focussed, her mind had sharpened and her voice was clearer. At the conclusion of her initial treatment, she walked from the chamber unaided and was able to perform a sharpened Romberg test for 30 seconds. After her second treatment the following day, at 243 kPa for ninety minutes with a five-minute air break, her abdominal symptoms had resolved and she was bright, talkative and able to perform a sharpened Romberg for more than 60 seconds. She had one final session, again at 243 kPa and then was discharged feeling well and symptom free. The medical team chose not to perform any imaging of the brain as she had made a full recovery. On phone follow up one month later, she had no recurrence of her symptoms.

Discussion

CAGE resulting from pulmonary barotrauma is a well-recognised phenomenon and cause of mortality and morbidity after compressed gas diving. It is also seen post open cardiac surgery,⁹ and is rarely associated with other surgical and gynaecological procedures,¹⁰ complications of central vascular access,¹¹ and inhalation of pressurised helium.¹² CAGE from hydrogen peroxide ingestion is a rare but recognised occurrence with several cases reported in the literature.¹⁻⁷

The patient in this case ingested approximately 30 ml of high concentration H₂O₂ which would liberate up to 3 litres of oxygen into the portal circulation.⁸ Any shunt between venous and arterial circulation will allow direct transfer of these gas emboli. In animal models it has been shown that a rate of infusion of venous gas of 0.3 ml·kg⁻¹·min⁻¹ will overcome the pulmonary filter and produce arterial gas embolism.¹³

During commercial flights, the ambient pressure decreases during ascent. A recent appraisal of cabin pressures measured an average ambient pressure of 0.78 bar (79 kPa).¹⁴ Using Boyle's law this would result in an approximate 28% increase in gas volume and exacerbate the effects of gas emboli, as seen in this case.

Regarding this patient's abdominal pain and CT findings of colonic thickening, two case reports exist of ischaemic colitis due to arterial gas embolism.^{15,16} H₂O₂ peroxide can also cause toxicity by corrosive effects on the gastrointestinal tract (GI) system but two recent reviews found no reports of significant GI morbidity.^{17,18}

HBO is the accepted standard treatment of CAGE from any cause. It has been shown that without treatment neurological manifestations may persist or be fatal.^{1,4} Recent literature recommends treatment for H₂O₂ ingestion with a US Navy Treatment Table 6.¹ However, delayed treatment has also been shown to be effective with shorter tables.² In the case presented, the patient's neurological and abdominal symptoms improved after the first treatment with a USN T5 and had resolved entirely after the second treatment, despite 72 hours elapsing between poisoning and initiation of HBO.

This case highlights the risks of flying post H₂O₂ ingestion. There are no current guidelines on when it is safe to fly following ingestion, but clearly from this case there are inherent risks in transferring a patient via air. In compressed air diving, current guidelines suggest a minimum safe pre-flight surface interval of at least 12 hours after a single, no-decompression dive.¹⁹ Given the lack of evidence that H₂O₂ causes significant GI morbidity, the authors recommend following these guidelines. Aeromedical transfer should be delayed by at least 12 hours unless the patient has already developed neurological symptoms suggestive of CAGE or other urgent need for transfer. In these CAGE cases, we suggest treating these patients as we treat divers with decompression illness and flying without reduction in ambient cabin pressure.

Conclusions

This unusual case of CAGE caused by H₂O₂ ingestion and flying reminds physicians to be alert for such presentations and to be aware that delayed hyperbaric treatment may still be effective. We must also be aware of the risks of aeromedical transfer of patients with a history of H₂O₂ ingestion.

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