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Two cases of highly concentrated hydrogen peroxide poisoning with portal venous gas treated using hyperbaric oxygen therapy

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SUMMARY

Hydrogen peroxide poisoning is a rare but potentially severe poisoning that can cause digestive tract irritation and/or gas embolism when ingested. The clinical presentation varies from asymptomatic patients to severe embolic consequences or even death. There is little evidence on the treatment of such poisoning to guide physicians in caring for these patients. This paper reports on two cases of highly concentrated hydrogen peroxide poisoning after accidental ingestion. Both patients showed evidence of portal venous gas, with one patient exhibiting significant symptoms while the other did not. Both were treated with hyperbaric oxygen therapy (HBOT), with a follow-up CT scan revealing a complete resolution of the portal venous gas. This suggests that HBOT is effective for both symptomatic and asymptomatic patients with portal venous gas and should be considered as an effective treatment option in cases of highly concentrated hydrogen peroxide poisoning.

BACKGROUND

Hydrogen peroxide (H_2O_2) is an odourless, colourless chemical compound, slightly more viscous than water, used in many fields due to its oxidising properties.[1] At low concentrations (3%), it is used as a cosmetic product (hair bleaching solution), surface disinfectant and medical disinfectant.[2 3] In higher concentrations (25% or higher), it has industrial uses, notably paper production, textile manufacturing and in the food industry.[2]

Hydrogen peroxide poisoning is rare[4] but can lead to serious or even lethal consequences. Some patients may require hospitalisation, become permanently disabled or die.[5–7] The lethal risk increases considerably when intoxication occurs at concentrations of more than 10%.[8]

We report the case of two patients who accidentally ingested highly concentrated hydrogen peroxide (approximately 100%). The patients had complications related to the presence of portal venous gas and were treated with hyperbaric oxygen therapy (HBOT), leading to a complete resolution of portal venous gas. This illustrates the variability in clinical presentation: although both patients exhibited digestive symptoms, one developed cardiac complications, while the other experienced neurological symptoms, despite ingesting the same hydrogen peroxide solution at the same time.

We present this report with adherence to CARE (Case Report) case report guidelines.[9] We obtained signed informed consent to publish this case report from the two patients.

CASE PRESENTATION

Two male patients in their late 70s were admitted to the local emergency department after ingesting a sip of an unknown, odourless, viscous liquid that they believed to be white wine. Within 30 min of ingestion, they experienced gastric symptoms (Patient A: nausea, followed by abdominal pain and haematemesis; Patient B: nausea and epigastric discomfort).

Admitted to the emergency department (75 min after ingestion), they were both haemodynamically stable, and the clinical examination, particularly neurological, was unremarkable. The ECG showed a regular sinus rhythm without other abnormalities. For Patient A, all standard biological tests, including blood count, renal function, hepatic function and electrolytes, were within normal limits. In contrast, Patient B had elevated troponin-T level without kinetic change (troponin-T, high sensitivity (normal <14 ng/L)): 33 ng/L (2 hours after poisoning) and 33 ng/L (4 hours after poisoning). Given the absence of both anamnesis and clinical findings indicating excessive alcohol consumption and with both patients and witnesses reporting that they had only taken a single sip from that unknown bottle at that time, blood alcohol concentration was not measured.

Patient A developed somnolence 5 hours post-intoxication and showed objective confusion 6 hours after the incident (GCS 13/15). His wife reported that he began displaying unusual behaviour (difficulty to communicate with) immediately after ingestion. This prompted a cerebral and thoracoabdominopelvic CT, which showed the presence of a significant amount of gas in the venous portal system and a thickening of the oesophageal and gastric walls. There was no evidence of arterial gas embolism on imaging.

Patient B only experienced epigastric discomfort and mild nausea without further symptoms. He underwent gastroscopy, which revealed massive inflammation and erythematous-haemorrhagic mucosa throughout the entire stomach, with no damage to the oesophagus or duodenum (see figure 1).

Given the presence of venous portal gas in Patient A, Patient B also underwent a CT scan, which also revealed venous portal gas. He did not develop any neurological symptoms.

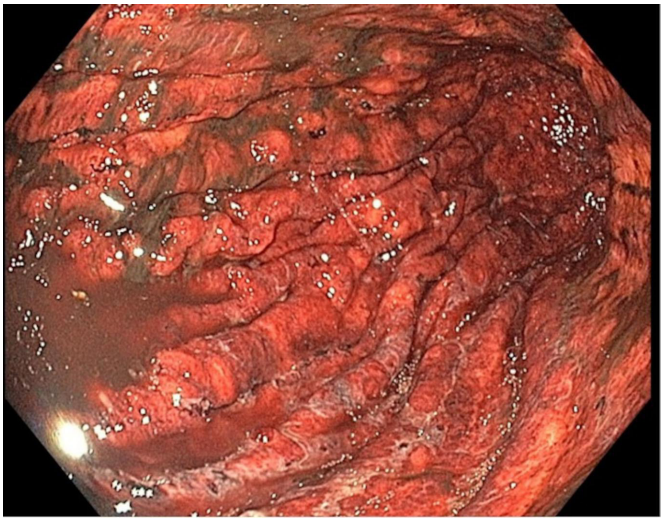

Figure 1 Gastroscopy was performed on Patient B, revealing signs of diffuse haemorrhagic gastritis. The abdominal CT scans of Patients A and B before and after hyperbaric therapy are presented. Images A and B show portal gas embolism discovered during initial CT. Images A′ and B′ show radiological resolution of the embolism following a single hyperbaric therapy (26 hours after poisoning, 9 hours after hyperbaric therapy).

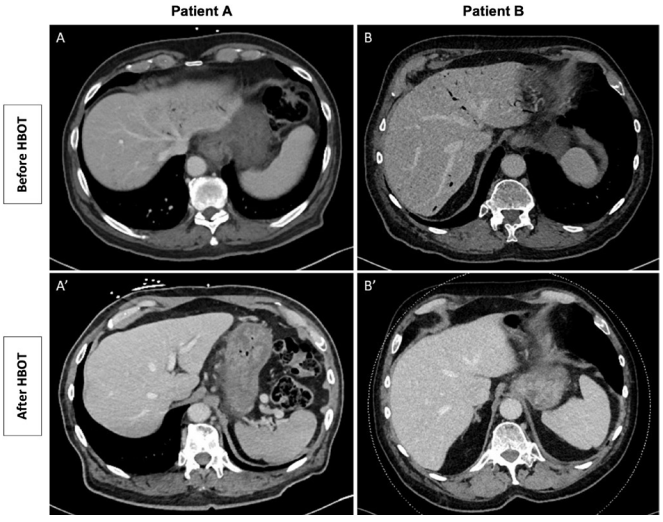

Figure 2 CT scanner of the two patients, before and after hyperbaric therapy. The abdominal CT scans of Patients A and B before and after hyperbaric therapy are presented. Images A and B show portal gas embolism discovered during initial CT. Images A′ and B′ show radiological resolution of the embolism following a single hyperbaric therapy (26 hours after poisoning, 9 hours after hyperbaric therapy). HBOT, hyperbaric oxygen therapy.

Meanwhile, the bottle with the unknown liquid was taken to the toxicology laboratory for analysis, and almost pure hydrogen peroxide was identified (ie, >90%).

Given the presence of portal venous gas on imaging in both patients, along with clinical symptoms in Patient A indicative of a possible cerebral arterial gas embolism, both patients were promptly transferred to the nearest hyperbaric medicine centre.

TREATMENT

Both patients were transferred by ground ambulance to the hyperbaric medicine centre approximately 7 hours after ingestion. Both patients underwent HBOT approximately 13 hours after poisoning. Based on our internal protocols, we used a long Comex 18 table which is the equivalent of the classical US Navy Table 6 (see figure 2).

Patient A, who was still confused at the time of HBOT, complained of otalgia and became agitated, requiring deep sedation throughout the session, with a propofol infusion (between 1 and 3 mg/kg/hour) and non-invasive ventilation. Patient B did not complain of any discomfort during the session. For both patients, the session was complicated by bilateral grade III ear barotrauma.

Both patients were put on high-dose gastric protectors (esomeprazole 80 mg intravenous bolus, then 40 mg intravenously two times per day) and hospitalised in a monitored care unit.

OUTCOME AND FOLLOW-UP

Patient A had a persistent altered mental status (GCS 13/15) with hypoactive delirium without focal neurological deficit that lasted until the next day. A follow-up cerebral CT performed 26 hours after poisoning (9 hours after the end of HBOT) showed no cerebral abnormalities.

Patient B remained asymptomatic throughout his hospital stay. As part of the protocol followed by his local care unit, serial troponin measurements showed significant kinetic changes (troponin-T, high sensitivity (normal <14 ng/L)): 65 ng/L (18 hours after poisoning, immediately after the end of HBOT), 68 ng/L (21 hours after poisoning, 3 hours after the end of HBOT) and 61 ng/L (32 hours after poisoning, 24 hours after the end of HBOT). Additionally, routine monitoring 5 hours after the end of HBOT revealed an ECG showing <1 mm ST-segment depression in II and aVF, which resolved within 24 hours. Based on these findings, along with prior troponin measurements, a diagnosis of type 2 non-ST-elevation myocardial infarction, possibly secondary to cardiac arterial gas embolism, was made. Given that Patient A was hospitalised in another care unit following different protocols and exhibited no ECG anomalies, no troponin measurements were conducted.

Both patients underwent a follow-up abdominal CT (26 hours after poisoning, 9 hours after HBOT) which revealed no remaining gas in the portal system (see figure 3).

Both were discharged from the hospital after 48 hours of observation with gastric protective treatment, a soft diet and a follow-up appointment with gastroenterology.

DISCUSSION

Clinical manifestations of hydrogen peroxide poisoning depend on the volume, concentration and type of exposure: ocular exposure causes irritation, blurred vision and subepithelial and conjunctival bullae at concentrations of 3%,[10] ulceration[11] and even perforation of the cornea in concentrations above 10%.[3] Chemical colitis in the case of hydrogen peroxide enema[12] and respiratory tract damage in the case of inhalation have been described.[13]

The toxic effects of hydrogen peroxide poisoning are caused by three main mechanisms: direct irritation of tissues by the formation of corrosive lesions, lipid peroxidation of cellular membranes and the formation of oxygen.[14] This latter effect is due to a reaction ($2H_2O_2 \rightarrow 2H_2O + O_2$) mediated by an endogenous catalase, found in mucous membranes, liver, kidneys, erythrocytes and bone marrow.[3] Therefore, hydrogen peroxide creates digestive tract irritation immediately after ingestion. Then, after absorption in the portal system, hydrogen peroxide produces an important volume of oxygen that can lead to the

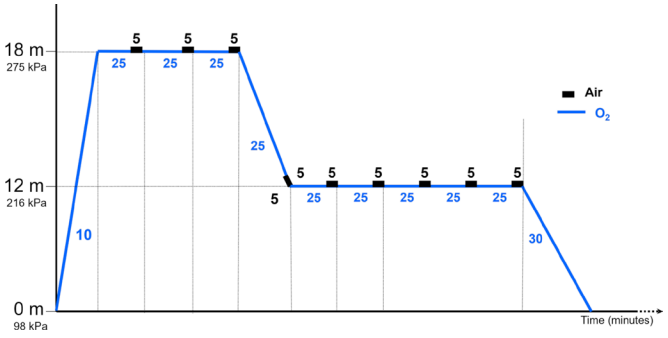

Figure 3 Hyperbaric oxygen treatment: long Comex 18 table.

presence of a large amount of gas in the portal vein and potentially in the general circulation. It has been shown that 30 mL of 35% hydrogen peroxide can form up to 3.5 L of oxygen.[15]

Most exposures occur through ingestion. This causes caustic lesions of the digestive mucosa, as well as the formation of gas bubbles responsible for pneumatosis of the digestive walls,[7] portal venous gas,[6] stroke[15] and ischaemic coronary events.[6] Most clinically significant systemic embolic phenomena occur within 10 hours but may also be delayed.[16]

Both the Undersea and Hyperbaric Medical Society in the USA and the European Committee for Hyperbaric Medicine have approved HBOT for gas embolism. HBOT consists of breathing 100% oxygen at a higher pressure than sea level. Due to the Boyle-Mariotte law, increased pressure results in decreased size of enclosed gas spaces, including bubbles in gas embolism situations, therefore restoring downstream blood flow. HBOT has been shown to reduce the risk of sequelae and mortality, particularly in the case of neurological or cardiac symptoms, or the presence of peripheral embolisation.[6 16 17] Cases presenting with the presence of venous gas (eg, in portal vein) alone have also been successfully treated conservatively.[18 19]

Both our patients underwent HBOT, resulting in the rapid and complete resolution of the radiological signs of portal venous gas. Patient A's neurological symptoms improved gradually, which may indicate a persistent ischaemic effect of cerebral arterial gas emboli, despite the absence of radiological signs on the initial cerebral scan.

In the case of Patient B, who initially had the presence of portal venous gas with no other symptoms, one may wonder whether HBOT was superfluous or whether it reduced the risk of secondary systemic embolism. We opted for hyperbaric oxygen for the patient due to the early onset of poisoning symptoms, the high concentration of the ingested hydrogen peroxide and the observation that Patient A, who consumed the same concentration and dose at the same time, exhibited symptoms consistent with cerebral gas embolism. The troponin-T peak, recorded 21 hours after poisoning, suggests the occurrence of a cardiac embolism as a possible cause. However, it remains unclear whether HBOT influenced cardiac function, as the timing of ECG and biomarker changes does not suggest a direct causal relationship with the therapy.

Whether the presence of portal venous gas alone after hydrogen peroxide poisoning is an indication for HBOT remains unclear. In the two cases reported here, there was a discrepancy between the laboratory results, which remained within normal ranges, and the presence of portal venous gas on imaging. This underscores the limitation of laboratory tests (eg, liver function test) in distinguishing patients who may remain asymptomatic from those who may become symptomatic and experience clinical deterioration in cases of hydrogen peroxide poisoning. Published evidence suggests that higher concentration and volume of hydrogen peroxide may be associated with increased complications.[7] Also, evidence shows that 'over 90% of embolic symptoms occurred within 10 hours of ingestion'.[16] Based on this evidence and on the cases reported here, we suggest that for asymptomatic patients, assessed within a few hours after highly concentrated and/or high-volume hydrogen peroxide ingestion, an immediate CT scan may be useful to assess the presence of portal gas. If imaging shows portal gas in these cases, it may be reasonable to promptly transfer the patients to a facility equipped with HBOT, should the patient become symptomatic or display signs of gas embolism.

Finally, most of the cases described in the literature develop symptoms within the first hour, but the time taken to treat them varies. Early HBOT appears to reduce the risk of complications or death.[6] Therefore, it is important to refer the patient to the nearest hyperbaric medicine centre if there is radiological or clinical evidence of gas embolism.

Learning points

- There is an inter-individual variability in patients' symptomatology, despite the same concentration and volume of hydrogen peroxide ingested.
- Hydrogen peroxide poisoning can result in significant portal venous gas, which may remain asymptomatic or progress to symptomatic arterial gas embolism. Even in asymptomatic patients (ie, presence of portal venous gas without embolism), imaging (considered alongside the concentration and volume of hydrogen peroxide and the timing of ingestion) may help clinicians in deciding whether to initiate hyperbaric oxygen therapy (HBOT).
- HBOT is an effective treatment for gas in the portal system or arterial gas embolism caused by hydrogen peroxide poisoning and should be considered as soon as possible in severe cases.

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Contributors The following authors were responsible for drafting of the text, sourcing and editing of clinical images, investigation results, drawing original diagrams and algorithms and critical revision for important intellectual content: MV, AJ, MS, CS and SB. MV, AJ, MS, CS and SB were involved in patients care and met the following criteria: conception and design, acquisition of data or analysis and interpretation of data, drafting the article or revising it critically for important intellectual content, final approval of the version published and agreement to be accountable for the article and to ensure that all questions regarding the accuracy or integrity of the article are investigated and resolved. Guarantor is SB.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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