

Sudden death after oxygen toxicity seizure during hyperbaric oxygen treatment: Case report

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Abstract

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Acute cerebral oxygen toxicity (ACOT) is a known side effect of hyperbaric oxygen treatment (HBOT), which can cause generalised seizures. Fortunately, it has a low incidence and is rarely harmful. Nevertheless, we present a case of a 37 year-old patient with morbid obesity who died unexpectedly after an oxygen toxicity seizure in the hyperbaric chamber. Considering possible causes, physiologic changes in obesity and obesity hypoventilation syndrome may increase the risk of ACOT. Obesity, especially in extreme cases, may hinder emergency procedures, both in- and outside of a hyperbaric chamber. Physicians in the hyperbaric field should be aware of the possibility of a fatal outcome after ACOT through the described mechanisms and take appropriate preventative measures. Basic airway management skills are strongly advised for involved physicians, especially when specialised personnel and equipment are not immediately available.

Introduction

Acute cerebral oxygen toxicity (ACOT) is a rare side effect of hyperbaric oxygen treatment (HBOT), during which high tissue levels of oxygen in the central nervous system (CNS) exists. Among other symptoms, generalized seizures are observed, which resolve after reducing the inspired fraction of oxygen (FiO₂). The incidence is reported as 1.2 per 10,000 HBOT sessions where air breaks are incorporated in the treatment.¹ No long term effects have been described.² Nevertheless, we present a case of a 37 year-old male, who died unexpectedly after an oxygen toxicity seizure.

Case report

The patient was referred for HBOT to precondition a non-healing venous leg ulcer before skin grafting. He was obese and had hypertension and obstructive sleep apnoea (OSA) syndrome. Although the medical referral mentioned use of a beta blocker for his hypertension, the patient denied this and there was no record of a prescription. Physical examination showed extreme obesity with a height of 185 centimetres and a weight of 180 kilograms (body mass index [BMI] 52.6 kg·m⁻²), and hypertension (blood pressure 150/110 mmHg). The patient was advised to consult his general practitioner on short notice regarding the hypertension, but this was not considered to be a reason to

postpone therapy since hypertension is only considered to be a mild and relative contra-indication for HBOT.

Treatment took place in a HYOT/2200/20/2/RD multiplace chamber (IHC Hytec, Raamsdonksveer, the Netherlands). The chamber was pressurised to 2.4 atmospheres absolute (atm abs; 243 kPa); and then patients breathed 100% oxygen through non-rebreathing masks in four blocks of 20 minutes, with 5-minute air breaks in between.

The first HBOT session the patient received was uneventful. Halfway through the second treatment session the patient exhibited prodromal symptoms of ACOT, immediately followed by a generalised seizure, lasting approximately 1.5 minutes. The attendant, present in the chamber, quickly removed the mask and requested the assistance of a physician. Postictally, the patient slumped down from his chair in a sitting position and had hyperpnoea. Eventually, the patient was positioned supine; the hyperpnoea persisted. Three minutes after the onset of the seizure, a physician was present inside the chamber, who also found tachycardia. Since our centre is not located in a hospital, emergency services were contacted. Due to the hyperpnoea and an absence of snoring, an unobstructed airway was assumed, and decompression was started at the usual rate of 14.9 kPa·min⁻¹ (1.5 meters' seawater per minute equivalent) to minimise the risk of pulmonary barotrauma. During decompression, the patient

Table 1
Stages of obesity hypoventilation syndrome (OHS) (adapted from Randerath et al.⁸)

Stage	Diagnosis	Hypercapnia status
0	At risk	None
1	Obesity-associated sleep hypoventilation	Intermittent during sleep, full recovery during sleep. Serum bicarbonate < 27 mmol·L ⁻¹ awake.
2	Obesity-associated sleep hypoventilation	Intermittent during sleep. Serum bicarbonate ≥ 27 mmol·L ⁻¹ awake.
3	Obesity hypoventilation	Sustained hypercapnia (PCO ₂ > 45 mmHg or 6.4 kPa) awake
4	Obesity hypoventilation	Sustained hypercapnia awake. Cardiometabolic comorbidities.

remained hyperpnoeic and had a nosebleed, possibly from a barotrauma of the ears. In the final minute of decompression, the patient started frothing bloody sputum at the mouth and then stopped breathing. No signs of cyanosis were noted. Cardiopulmonary resuscitation (CPR) was started immediately by the physician. After decompression was completed, emergency services continued CPR inside the hyperbaric chamber. Intubation was difficult but eventually succeeded. After 30 minutes of asystole, CPR was stopped and the patient was declared dead.

Discussion

To our knowledge, this is the first published case of a fatal oxygen convulsion during regular HBOT. Although no absolute contraindications for HBOT were present in this case, several factors that may have caused or contributed to this unfortunate outcome merit discussion.

The patient's medical history contained several conditions that are considered contraindications in diving, but not in HBOT. Since HBOT takes place in a controlled environment, these conditions are far less prohibitive and not considered a contraindication. Hypertension raises the risk of both acute (cerebrovascular incidents) and chronic morbidity (end-organ failure).³ Although HBOT causes a rise in blood pressure, the effect is small.⁴ Obesity is contraindicated in diving because it causes increased nitrogen accumulation in adipose tissue, which increases the risk of decompression sickness (DCS).⁵ Since patients breathe 100% oxygen during HBOT for most of a session, no nitrogen build-up takes place and there is no increased risk of DCS.

Sleep apnoea is associated with several cardiovascular comorbidities, such as hypertension, arrhythmias and pulmonary hypertension⁶ but is not considered an absolute contraindication in either diving or HBOT. In this case, it is plausible that the diagnosed OSA was accompanied by an obesity hypoventilation syndrome (OHS). OHS is a sleep-disordered respiratory syndrome, defined as sustained

hypercapnia in obese persons without other causes for hypoventilation present.⁷ It is divided in five categories⁸ (see Table 1). The estimated prevalence is 10–38% in patients with OSA,⁹ and at least 14% of OSA patients present with daytime hypercapnia¹⁰ (i.e., stage 3 or 4 OHS). The chronic respiratory changes may lead to pulmonary hypertension, cor pulmonale and peripheral oedema.⁷ Like OSA, so far OHS is not been described as a contraindication for diving or HBOT. However, we hypothesise that the patient was more prone to ACOT due to OHS. While chronic hypercapnia does not influence vasodilation,¹¹ a small case-control study by Hollier et al.¹² showed, that even moderate amounts of supplemental oxygen induces hypoventilation and an acute increase of hypercapnia and acidaemia in OHS patients. This can cause cerebral vasodilation, counteracting the neuroprotective vasoconstriction of hyperoxaemia,¹³ thereby increasing the risk of ACOT during HBOT.

After the seizure, the patient developed hyperpnoea, which is frequently seen postictally.¹⁴ Hyperpnoea was interpreted as a sign of a patent airway. However, it is plausible that the airway was at least partly obstructed by the diminished hypopharyngeal diameter, known to occur in OSA.¹⁵ This relative obstruction combined with hyperpnoea may have resulted in an increased negative intrathoracic pressure, which can cause pulmonary oedema, whose presence was suspected when frothy bloody sputum was observed. This condition is well-known in the field of anaesthesiology.¹⁶

The clinical features are reminiscent of the syndrome known as 'sudden unexpected death in epilepsy' (SUDEP), in which epilepsy patients develop similar cardiorespiratory symptoms after a generalized seizure, which can result in sudden death.¹⁷ In autopsies performed in patients deceased after a seizure, non-cardiac pulmonary oedema is often found and regarded as a significant contributor to mortality and may be provoked by negative lower airway pressure during inspiration.¹⁸ Pulmonary oedema can cause acute loss of the lung reservoir of oxygen. While oxygen reserves should have been adequate in other reservoirs

due to hyperoxygenation, the increased inspiration efforts causing increased respiration workload may additionally have reduced time to desaturation.¹⁹ Moreover, while compression to 2.4 ATA may have caused an adequate tissue oxygen saturation, decompression to 1 ATA diminishes tissue oxygen saturation, similar to ascent hypoxia seen in breath holding diving.²⁰

Because the airway was assumed to be unobstructed at the time of the incident, no airway management devices were used during the decompression phase. Due to the weight of the patient and the confined space of the hyperbaric chamber, attempts to reposition the patient in the recovery position failed, both during and after decompression. Intubation attempts by emergency services were hindered by these factors as well. Since airway patency is compromised in the postictal phase¹⁸ and in overweight patients,¹⁵ desaturation during decompression could have been prevented or ameliorated by using an oropharyngeal airway device. This is especially relevant in hyperbaric centres that do not have immediate access to specialised personnel and equipment, as is available in hospitals.

By law, no autopsy was required as the medical forensic examiner determined the cause of death to be due to a recognised side effect of the therapy. Furthermore, the family did not allow autopsy. As a result, no definitive cause of death could be determined. We can therefore only speculate on possible causes. The factors mentioned here not only increase the risk of oxygen toxicity but also mortality after the seizure has resolved. This has prompted revision of our medical screening protocol. In individuals with a BMI ≥ 35 and/or known OSA/OHS, we now perform arterial blood gas analysis. Hypercapnia ($P_a\text{CO}_2 > 45$ mmHg or > 6.4 kPa) at rest (OHS stage 3–4; see Table 1) is now considered a contraindication for HBOT. During the six months after this case, we screened fourteen patients based on this new criterium and excluded one. Patients weighing > 120 kilograms are counselled on the possibility that emergency procedures may be complicated due to their weight.

In conclusion, a fatal incident following an oxygen seizure during HBOT in a morbidly obese patient is described. Considering the increasing global obesity epidemic and the increasing use of HBOT, it is important to review established safety protocols for these patients. Physicians should be aware of the physiological changes in obesity that may increase the risk of ACOT. The presence of hypercapnia with OSA or OHS is an important risk factor that can easily be detected with arterial blood gas analysis. Furthermore, obesity, especially in extreme cases, may complicate emergency procedures, both in- and outside of the hyperbaric chamber and should be re-evaluated for these patients. Basic airway management skills are strongly advised for physicians working in a hyperbaric centre, especially when specialised personnel and equipment are not immediately available.

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