Oxygen toxicity seizure mimics

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Abstract
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Introduction: Oxygen toxicity seizures (OTS) are a well-recognised complication of hyperbaric oxygen treatment (HBOT). As such, seizure-like activity during HBOT is usually presumed to be a result of central nervous system oxygen toxicity (CNS-OT). Four cases are reported here where causes other than CNS-OT were determined as being the likely cause of the seizure; causes we have labelled 'OTS mimics'. Through review of the current literature, and our hyperbaric medicine unit's experience to date, we aimed to highlight the relevance of these OTS mimics, as the potential for significant morbidity and mortality exists with incorrect diagnoses.
Methods: A retrospective review of the medical records of all patients treated at the Fiona Stanley Hospital and Fremantle Hospital hyperbaric medicine units who had a seizure during HBOT between November 1989 and June 2020. These events were reviewed to determine whether causes for seizures other than oxygen toxicity were evident.
Results: Four OTS mimics were identified: posterior reversible encephalopathy syndrome, pethidine toxicity, previous subarachnoid haemorrhage with resultant epilepsy, and severe hypoglycaemia.
Conclusions: This case series highlights the need for caution when diagnosing an apparent OTS. Multiple conditions may mimic the signs and symptoms of oxygen toxicity. This creates scope for misdiagnosis, with potential for consequent morbidity and mortality. A pragmatic approach is necessary to any patient exhibiting seizure-like activity during HBOT, with suspicion for other underlying pathologies.

Introduction

Oxygen toxicity seizures (OTS) are a rare but well-recognised and feared complication of hyperbaric oxygen treatment (HBOT), with a reported incidence of between 0.01% and 0.06% for 'routine' treatments.[1,2] Although certain prodromal signs and symptoms of oxygen toxicity are now well recognised, there remains a large variation in presentation and onset, often indistinguishable from other diagnoses.[3] There is potential for seizures occurring due to other causes being attributed to central nervous system oxygen toxicity (CNS-OT) if they occur in or around HBOT.

Methods

Approval was obtained for data review and extraction (GEKO Quality Activity 35539).

A retrospective review was undertaken of the medical records of all patients treated at the Fiona Stanley Hospital and Fremantle Hospital hyperbaric medicine units who had a seizure during HBOT between November 1989 and June 2020. These records were reviewed by the author group to determine whether causes for seizures other than oxygen toxicity were evident.

Results

There were 25 OTS in 64,491 patient treatments over the past 30 years; the first 25 years at Fremantle Hospital and the last five years after relocation to Fiona Stanley Hospital. Four cases were found in which patients initially perceived to experience an OTS were, on further assessment, deemed to have suffered a seizure for other reasons. All other seizures that occurred were considered to be OTS. Each case (presented below) highlights the ease of misdiagnosis of oxygen toxicity, while emphasising the need to consider other potentially more sinister pathologies.

CASE REPORTS

Case 1: Posterior reversible encephalopathy syndrome (PRES)

A 22-year-old female was referred for treatment of purpura fulminans, secondary to *Escherichia coli* sepsis,

on a background of systemic lupus erythematosus and immunosuppression. She had developed multiple areas of acral ischaemia of all limbs secondary to sepsis, and was referred for HBOT to maximise limb viability prior to amputation.

HBOT was performed at 203 kPa (two atmospheres absolute [atm abs]) in a monoplace chamber as the patient was unable to hold a mask (required to deliver an air-break if higher treatment pressures were used), and was unable to tolerate a head hood.

During her sixth HBOT session, she suffered a seizure, occurring after 52 minutes of breathing oxygen (O_2) at 203 kPa. Generalised tonic-clonic activity for approximately one minute preceded signs in keeping with an absence seizure lasting a further two minutes. Oxygen delivery to the chamber was replaced with air immediately after seizure onset, and treatment was aborted. The chamber was decompressed over six minutes when seizure activity had ceased and spontaneous respirations had recommenced, during which time the patient was not responding appropriately to questions. On removal from the chamber the patient appeared coherent, but with no recollection of the event. She denied any prodromal symptoms or headache. Her blood pressure was recorded as normal post seizure and other vital signs were within normal limits, with a finger-prick blood glucose level (BSL) of 6.7 $mmol \cdot L^{-1}$ (120.7 $mg \cdot dL^{-1}$). No focal neurological signs were evident on examination.

No previous history of seizures had been documented. On collateral history, the patient's family reported recent episodes of abnormal shaking, while unresponsive to voice. This would occur for two to three minutes at a time before self-resolving. These had not been reported to staff prior to the time that this seizure had occurred. One such episode of abnormal movements had been witnessed by HMU staff following a HBOT session, but was not considered to be a seizure as these movements stopped on demand and there was no post-event confusion. A functional cause/pseudo-seizure was postulated as the aetiology. The patient specifically denied any headache at that time.

Following recovery from her seizure, the treating team was advised to consider the possibility of an organic cause such as encephalitis. A magnetic resonance imaging (MRI) brain scan was performed, showing multiple old infarcts and new areas of hyper-attenuation in the posterior occipital lobes. This finding was in keeping with PRES; a condition characterised by seizures, headaches and altered levels of consciousness. Oral nifedipine was commenced for management of PRES. HBOT was discontinued due to increased risk of seizures during future treatments. The patient had no further seizures during her hospital admission, and was followed up on an outpatient basis by the neurology team.

Case 2: Pre-existing seizure disorder

A 47-year-old male, ex-Navy diver, was referred for emergency HBOT for treatment of presumed cerebral arterial gas embolism (CAGE) with United States Navy Treatment Table 6 (USN TT6). This followed an episode of loss of consciousness following a dive. Initial history was received from the attending ambulance crew, who had received history from a participating dive instructor. The patient was completing a refresher open water dive course at the time of the episode. He had descended to approximately 3 metres depth without issue. He then began staring vacantly and was unresponsive to commands, before floating to the surface. A generalised seizure was witnessed at the surface before the patient was removed from the water and transferred to hospital. Uncertainty regarding speed of ascent or if breath was held prompted a diagnosis of CAGE as the most likely cause for his symptoms.

No focal neurology was noted on arrival to the Emergency Department (ED). Bloods performed were unremarkable. Non-contrast cranial computerised tomography (CT) scanning was performed showing a previously coiled basilar tip aneurysm but no new intracranial pathology.

During the final hour of treatment at a pressure of 190 kPa (1.9 atm abs) on O_2 being delivered by head hood, the patient had a generalised tonic-clonic seizure that lasted approximately two minutes. Blood was noted inside the patient's mouth, and there was loss of urinary continence. He was unresponsive, and making snoring sounds. The BSL was recorded as 6.5 $mmol \cdot L^{-1}$ (117.1 $mg \cdot dL^{-1}$). O_2 was removed immediately upon seizure onset, and the attending doctor entered the chamber to accompany the patient during decompression.

Later a collateral history from the patient's dive instructor was obtained. The patient had slowly risen to the surface, away from the dive group. He was breathing but unresponsive to command. The instructor brought the patient to shore where he proceeded to have a generalised seizure.

The patient was found to have a history of seizures on a background of previous subarachnoid haemorrhage (SAH), with multiple known aneurysms. This evolving history made the diagnosis of CAGE less likely, and more likely to represent a seizure episode underwater. With a further seizure in the hyperbaric chamber at 190 kPa, this was considered not to be a manifestation of oxygen toxicity, but more likely a pre-existing seizure disorder.

The patient was admitted under the neurology team. An electroencephalogram (EEG) was performed showing findings in keeping with disturbance of structure or function over the left hemisphere with epileptogenic potential. MRI brain showed no infarct or acute intracranial findings. He was

commenced on regular leviteracetam. No further HBOT was performed, and the patient was advised to cease scuba diving.

Case 3: Pethidine toxicity

A 28-year-old female underwent emergency HBOT for traumatic compartment syndrome of her right arm. Referral for HBOT was made post emergency fasciotomy.

Treatment at 284 kPa (2.8 atm abs) was initially planned, until the patient was found to be 22 weeks pregnant. The decision was changed at this point to treat at 203 kPa (2.0 atm abs). The rationale for this choice of treatment pressure was not documented.

The patient was initially quite anxious, with some difficulty equalising her ears during compression. Intravenous (IV) pethidine titrated to 130 mg was administered for severe pain in the affected limb. Seventeen minutes into the first O_2 period the patient suffered a generalised tonic-clonic seizure, lasting approximately five minutes before self-resolving. O_2 via head hood was removed immediately after onset and the attending doctor entered the chamber. Diazepam 2.5 mg IV was administered in the post-ictal phase and the hyperbaric treatment was aborted. Observations were within normal limits. The patient was rousable but irritable and no focal neurological deficits were demonstrated. Subsequently it was noted the patient had received 1,300 mg of pethidine in the preceding 28 hours. Pethidine's major metabolite (norpethidine) has well documented epileptogenic effects, and a relatively long half-life of 14–21 hours. HBOT was discontinued after this. It is possible that oxygen toxicity may have contributed to the onset of this seizure, although seizures may occur spontaneously with pethidine toxicity, and 'pure' oxygen toxicity seizures are uncommon at 203 kPa (2.0 atm abs).[2] Had the information regarding the patient's recent extremely high dosing of pethidine been known, it may have been that the risk benefit decision would have been not to proceed with HBOT.

Case 4: Hypoglycaemia

A 38-year-old female was referred for HBOT for a non-healing wound on her right forefoot, secondary to poor diabetic control. Thirty HBOT sessions were originally planned at a pressure of 243 kPa (2.4 atm abs) in a multiplace chamber, with O_2 delivered by head hood. During her fifth treatment, she became confused towards the end of the second O_2 period. She was noted to be staring vacantly, and unresponsive to voice at which point her head hood was removed. She then began twitching, and briefly lost consciousness, presumed secondary to oxygen toxicity. At this point the attending doctor entered the chamber and the hyperbaric treatment was aborted. BSL was checked, and had decreased to 1.4 mmol·L^{-1} (25.2 mg·dL^{-1}). This was successfully treated with oral glucose. Fifteen minutely BSLs were performed, with slow recovery. After monitoring in the HMU and after provision of a meal, the patient was discharged home, with alterations made to her insulin regime.

That day the patient had reported a BSL of 3.1 mmol·L^{-1} (55.9 mg·dL^{-1}) on waking, prior to breakfast and administration of 6 units of Novorapid® insulin. Her next BSL was 10.1 mmol·L^{-1} (182.0 mg·dL^{-1}) immediately prior to commencing HBOT, at approximately 0900 hours. For subsequent treatments, her BSLs were checked half hourly while in the chamber, with no further issues noted. Nineteen treatments were completed in total, discontinuing due to lack of clinical response.

Discussion

The symptoms and signs of CNS-OT, as described by Donald in 1947, have been observed in many patients since his original research.[2–5] Certain predisposing factors to CNS-OT have since been identified, with higher OTS rates documented with higher pressure treatment tables and for emergency indications.[2,6–9] When treating dysbarism, the rates of OTS have been reported from 0.28–1.11% of treatments.[2,6,10,11] An increased rate of OTS occurrence has also been reported with the first HBOT for carbon monoxide poisoning.[2,7,9]

The signs and symptoms of OTS have been described as notoriously "*unpredictable*", with "*large variation*".[3] Patients receiving elective HBOT will often be elderly, suffer from chronic pain, and have multiple comorbidities including diabetes mellitus, cardiac disease, or an active infection. These factors may lead to diagnostic uncertainty in the event of loss of consciousness, or a seizure during HBOT.

UNDERLYING SEIZURE DISORDERS

To date, patients with epilepsy have not been found to be at an increased risk of OTS.[4,7]

The recommended management of epileptic patients undergoing HBOT includes confirmation of therapeutic levels of any anti-epileptic medication. However, seizures during treatment due to underlying disorders can still occur, and pose a diagnostic dilemma. The question remains as to whether such seizures would have occurred regardless of HBOT, or were provoked by a synergistic effect from hyperoxia.

Case 1 involved a female with PRES; characterised by white matter vasogenic oedema of the posterior occipital and parietal lobes of the brain, leading to headaches, seizures, altered mental status and visual loss. It is often associated with acute hypertension, which if treated, will usually resolve the syndrome within a week.[12] There seem to be many

possible triggers, including abrupt arterial hypertension, impaired renal function, pregnancy, immunosuppressive therapies and various inflammatory conditions. It is becoming an increasingly recognised disorder with the advent of neuroimaging.[13] In this instance, suspicion of alternate diagnoses prompted an MRI brain, revealing pathognomonic findings of PRES. While approximately 75 percent of patients have moderate to severe hypertension at presentation, PRES may occur in normotensive patients, and is more common in patients with systemic lupus erythematosus; both seen in the patient presented. Treatment with an antihypertensive is still recommended and was given in this case. A combination of seizures, visual disturbance and / or headache, should lead to an early MRI brain.

Case 2 involved a male with previously documented seizures with a known cause that had not been divulged. Although previously working as a Navy diver, to the best of our knowledge no dive medical had been performed since suffering a SAH in 2014. Collateral history for this patient revealed likely seizure activity soon before treatment, initially presumed to be the result of CAGE. This was followed by a further seizure in the chamber, mistakenly attributed to oxygen toxicity. The identification of the underlying pathology facilitated improved patient disposition, with further unnecessary HBOT avoided. This highlights the importance of repeat dive medicals following significant morbidity, and the need to closely investigate anyone with a history of intracranial pathology.

EPILEPTOGENIC MEDICATIONS

Emergency HBOT indications such as necrotising infections, severe decompression illness (DCI) or crush injury may warrant large doses of analgesics such as pethidine, fentanyl, or tramadol. Those with coinciding acute or chronic infections may be receiving high doses of penicillins, cephalosporins or antifungals. Each of these medications has the potential to lower the seizure threshold in a susceptible individual, or in some cases, directly induce a seizure through neurotoxic effects.[14]

Case 3, originally reported in 1998, involves the effects of pethidine toxicity, which through its active metabolite, norpethidine, has the direct potential to induce seizures.[14–16] Currently many patients will receive HBOT despite co-administration of such medications. Difficulty may arise in distinguishing between the provocation of an OTS, and the unmasking of an underlying seizure disorder. A retrospective analysis from 2004 examined the rate of OTS from 107,264 HBOT sessions performed in 30 hyperbaric centres in Germany.[17] Two cases were excluded from this series, with seizures in the chamber instead being deemed due to high dose cefazolin treatment. To date, minimal literature exists surrounding seizure-provoking medications during HBOT, focussing more on cases of true oxygen toxicity. A case series from several hyperbaric units in Milwaukee, Wisconsin, USA reviewed seven seizures among five patients undergoing HBOT.[18] Each case highlighted other potential causes for seizures, including high doses of ceftriaxone, tramadol, selective serotonin reuptake inhibitors, and tricyclic antidepressants concurrently used among these patients. Other relevant drugs include high dose penicillins and cephalosporins, narcotics, pethidine, corticosteroids, and acetazolamide. Other factors referenced included narcotic withdrawal, alcohol withdrawal, and carbon dioxide retention in patients undergoing HBOT.

Additional medication choices during HBOT remain an important factor before and during treatment. If possible, seizure-provoking medications are best avoided, but each case requires a consideration of risk versus benefit.

HYPOGLYCAEMIA

A large cohort of patients receiving HBOT suffer from diabetes mellitus, notably those with consequent peripheral vascular disease and non-healing lower limb wounds. Some evidence suggests that HBOT does not cause a clinically significant decrease in BSL among diabetics.[19] However a study from 2013 demonstrated that finger-prick capillary sampling may not be an accurate reflection of venous glucose during HBOT.[20] Wilkinson et al reported increased peripheral insulin sensitivity following HBOT, maintained for at least 30 minutes after exiting the hyperbaric chamber. This was initially demonstrated in healthy individuals, and later in obese males, both with and without type 2 diabetes mellitus.[21–23] Irrespective of potential direct effects of hyperbaric oxygen, these patients will undergo periods of fasting in the chamber, while isolated if treated in a monoplace environment. With this comes the potential for adverse events, particularly in those with labile BSLs, and those who remain asymptomatic until severe hypoglycaemia occurs.

Common symptoms of hypoglycaemia such as twitching, agitation, confusion, nausea, visual changes, peri-oral paraesthesia and eventually seizures, all overlap with those of CNS-OT. Case 4 demonstrates their homogeneous presentation, as symptoms began with twitching and confusion, followed by loss of consciousness. The BSL just prior to commencement of that day's HBOT was 10.1 $mmol \cdot L^{-1}$ (182.0 $mg \cdot dL^{-1}$), resulting in the attending medical staff initially identifying the episode as a result of oxygen toxicity. As per the unit's emergency procedures for OTS, BSL was checked immediately as standard, and the patient was promptly treated for severe hypoglycaemia. Failure to identify the true cause of symptoms in this case could have led to significant deleterious effects for this patient.

It has become routine in our unit since 2003 to document finger-prick glucose in diabetic patients immediately before

and after each HBOT. This is also performed during HBOT if the patient develops symptoms of hypoglycaemia or oxygen toxicity. Diabetic patients treated in the monoplace chambers are supplied with a prophylactic syringe of oral glucose, to be consumed if symptoms of hypoglycaemia develop.

Many additional seizure mimics are now well recognised within the emergency setting. A clinical review published in 2016 lists these seizure differentials, along with salient signs and symptoms to aid in differentiation.[24] That study reported 20% of presumed epileptic patients were misdiagnosed in emergency departments, being later identified as suffering most commonly from syncope or psychogenic non-epileptic seizures. Patients receiving HBOT not uncommonly suffer from some form of cardiac disease. A forceful Valsalva manoeuvre combined with a degree of autonomic failure and the 'dive reflex' may be enough to induce a syncopal episode.[25] Other causes include stroke, hyponatraemia, sleep disorders such as narcolepsy with catoplexy, movement disorders, and migraine.[26] From this we can extrapolate the possibility of confusion between seizures and seizure-like presentations.

OTS, in general, occur at higher pressures, with a significantly lower incidence at treatment pressures of 203 kPa (2 atm abs) or less.[2] A review of 62,614 HBOT sessions from a single unit in Israel reported no OTS from 12,303 treatments performed at pressures below 203 kPa.[27] However most (> 51%) treatments included in this cohort were at a maximum pressure of 151 kPa (1.5 atm abs). This is below the minimum therapeutic pressure used across Australasia. Reports of three OTS occurring at 190 kPa (1.9 atm abs) have been documented.[2] The seizure in each of these cases occurred during decompression from earlier treatment pressure exposure to 243 or 284 kPa. This highlights the difficulty in diagnosing or excluding OTS based solely on treatment pressure.

Conclusion

This case series highlights the need for caution when labelling an OTS. Multiple conditions may mimic the signs and symptoms of oxygen toxicity. This creates scope for misdiagnosis, with potential for morbidity and mortality. A pragmatic approach is necessary to any patient exhibiting seizure-like activity in the hyperbaric chamber, with suspicion for other underlying pathologies.

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