

Hyperbaric oxygen-associated seizure leading to stroke.

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Hyperbaric oxygen-associated seizure leading to stroke.

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Introduction

- Oxygen toxicity seizures are a well-known complication of hyperbaric oxygen (HBOT) therapy.
- These seizures are generally regarded as benign.
- Until now, there have not been any reported cases of acute ischemic event (stroke) as the result of an HBO-induced oxygen toxicity seizure.
- This event challenges the view that oxygen toxicity seizures are always benign



Case Report

80 year old male undergoing HBOT for a non-healing arterial insufficiency toe ulcer

Past Medical History included Cerebrovascular Accident without residual deficit, Chronic Obstructive Pulmonary Disease, Coronary Artery Disease, Peripheral Vascular Disease, Stage IV Chronic Kidney Disease

- Hyperbaric therapy was chosen in an attempt to save the patient's leg while avoiding surgery which would likely lead to permanent dialysis.
- First HBO treatment without incident (90' at 2.4 ATA + air breaks) one day prior



Case Report- HBOT

On ascent during his second treatment, he developed expressive aphasia, inability to follow commands on the left, rhythmic lip smacking movements, and right upper extremity spasticity and clumsiness . This was followed by tonic-clonic activity lasting about 90 seconds.

- Noted to be incontinent of urine
- Blood sugar was 89
- No ectopy on cardiac monitor.

Following the seizure, the patient appeared post-ictal.

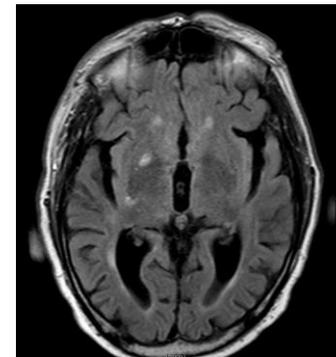
- Responsive to loud voices but otherwise somnolent and did not follow commands
- Speech was mumbling
- Ophthalmic exam demonstrated ocular-clonus
- Physical exam was otherwise unremarkable



Case Report- ED

He was transported rapidly to the Emergency Department where the stroke team was activated.

- Head CT showed age indeterminate lacunar infarcts, but no evidence of hemorrhage or mass effect, with global parenchymal volume loss and sequela of mild to moderate small vessel ischemic disease
- Remainder of ED workup identified no other acute problems
- Rapidly returned to his baseline function and mental status while in the ED and was admitted to the hospital for further work-up



Case Report- Work up

- MRI (5 hour) showed acute/sub-acute infarcts of right putamen and frontoparietal cortex.
- EEG performed was consistent with presence of moderate diffuse cerebral dysfunction; no epileptiform discharges or seizures recorded
- Angiography was not done as this pattern was not suggestive of a single large artery distribution.
- Doppler ultrasound showed mild stenosis of his right and moderate stenosis of his left carotid.
- Chest X-ray without evidence of pneumothorax, blebs or other COPD findings.
- Echocardiography demonstrated no evidence of shunting across the interatrial septum. There was aortic valve sclerosis and mitral calcification but no sign of thrombus or other potential source of emboli.

Case Report- Outcome

Following a full workup, the patient was determined to have had a seizure as a result of CNS oxygen toxicity. **This seizure was thought to have provoked his stroke.**

- Location of the stroke would not produce the symptoms with which the patient presented. Demand ischemia from the oxygen induced seizure in the setting of vascular insufficiency resulted in infarct

Further HBO therapy was deemed to be more risk than benefit to the patient and his treatment was discontinued.



Discussion

- Seizures during HBO treatments are a well known complication and occur with a 0.03% incidence
- Pressure dependent
- Pathophysiology of oxygen toxicity seizures is not well understood
- Known that cerebral blood flow is increasingly reduced at higher ATA
- Thought that the patient had such high metabolic demand during his seizure, coupled with his known vascular insufficiency, that it resulted in an area of ischemia



The patient did not go on to tonic-clonic activity until he was out of the chamber. Was this indeed due to oxygen toxicity?

Differential Diagnosis Conundrum.

- Stroke induced seizure (perhaps from an embolic event)
- Coincidental seizure from prior infarct or disorder
- Presyncope (cerebral hypoperfusion)
- Cerebral gas embolism



Differential Diagnosis Conundrum.

Coincidental seizure from prior infarct or disorder

- Other than prior stroke, the patient had no other seizure history or risk factors. The only MRI evidence of his old stroke was “Scattered foci of T2 hyperintensity throughout the hemispheric white matter, likely sequelae of mild to moderate small vessel ischemic disease.” These lesions are incidental age related changes. It is unlikely that such a lesion could have been an epileptogenic focus as such subcortical structures not thought to be involved in epileptogenesis.
- EEG failed to reveal an underlying epileptiform pattern.



Differential Diagnosis Conundrum.

Presyncope (cerebral hypoperfusion)

- Cardiac monitor- no dysrhythmia was noted.
- Hypertension of 179/84 shortly after the event and 144/50 the day prior. No cause or indication exists for transient hypotension.
- Oral automatisms of rhythmic lip and mouth movements were highly suggestive of temporal lobe or complex partial seizures, before proceeding to a generalized tonic clonic seizure.
- Hypoperfusion or pre syncope can give some shaking movements as in the case of “limb-shaking” carotid TIAs but not complex oral movements or automatisms.
- This mechanism fails to explain the acute/sub-acute infarcts noted on MRI.



Differential Diagnosis Conundrum.

Cerebral gas embolism or cardio embolic stroke

- Onset of symptoms during decompression and COPD, air trapping leading to pulmonary barotrauma and subsequent cerebral arterial gas embolism (CAGE) could be considered.
 - Infarcted areas did not correlate with the early signs prior to tonic clonic activity
 - no evidence of pulmonary barotrauma (or visible COPD changes)
- A cardio embolic stroke is unlikely
 - no evidence of atrial fibrillation or other arrhythmia,
 - no right to left shunt in the heart or embolic source on the echocardiogram.
 - Additionally, seizure activity at onset is not common nor is altered mentation or the other signs noted prior to tonic clonic activity.



Discussion

It is unclear what the effect was of having the patient removed from the hyperbaric chamber.

- It is possible that the drop in plasma oxygen partial pressure occurring with the cessation of HBO₂ combined with ongoing HBO₂ induced vasoconstriction and small vessel atherosclerotic disease provided a situation in which demand ischemia might occur.
- We posit that the stroke was in fact the result of demand ischemia in the face of the seizure.
- The initial symptoms appeared to be due to focal seizure activity which then generalized globally.
- The stroke findings appeared clinically silent.
- The stroke areas are not proximal carotid strokes, but downstream in where the patient's known small vessel ischemic disease would have come into play.

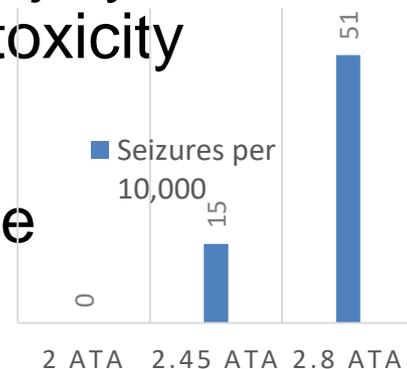
Conclusions

Although brain injury secondary to seizure has been described in the literature, this is the first report of brain injury secondary to hyperbaric oxygen-induced seizure.

Other reports have raised the possibility of hyperbaric oxygen-induced strokes causing injury. This case serves as a reminder that oxygen toxicity seizures may not always be benign.

Hyperbaric oxygen-induced seizures are dose (pressure) related.

Risk factors for cerebrovascular disease should be taken into consideration in determining treatment pressures for hyperbaric oxygen therapy.



Behold! My Hyperbaric Oxygenator



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