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Case series and outcomes of hyperbaric oxygen treatment of acute cerebral gas embolism

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Abstract

Introduction/background: The definitive first line and most beneficial treatment of an arterial gas embolism is hyperbaric oxygen therapy (HBO). Initial management of gas embolism should involve cessation of any operation or procedure to prevent further embolization and cardiopulmonary stabilization until arrangements for HBO treatment can be made. The patient should be started on 100% high-flow oxygen. Intubation may be required for somnolent or comatose patients for airway protection.

Materials and methods: We present eight cases of cerebral arterial gas embolism (CAGE) which we recently treated with HBO.

Results: Four of the eight cases, experienced near to complete restoration of their baseline function. The other four died or were gravely disabled.

Summary/conclusions: HBO therapy treats gas embolism by diminishing the volume of intravascular bubbles via an increase in the ambient pressure and providing a diffusion gradient for nitrogen and other gases to dissipate out of the bubble into the solution. HBO therapy increases the partial pressure of oxygen in the plasma, improving oxygenation of ischemic tissues even with reduced blood flow. It has also been shown to improve cerebral edema by reducing vascular permeability, promoting vasoconstriction, and diminishing adherence of leukocytes to damaged endothelium. It is of the utmost importance to get patients to HBO as quickly as possible when a cerebral air embolism is suspected.

Introduction

Gas embolism is a rare, but serious and potentially life-threatening complication of a variety of medical procedures performed in almost all clinical specialties [1]. Most gas emboli cases are due to iatrogenic causes; however, scuba diving and trauma can be initiating factors as well [2]. A systematic review of gas emboli case reports found that the most common causes of an iatrogenic cerebral gas embolism include central venous catheters, cardiopulmonary bypass, lung biopsies, mechanical ventilation, contrast injection, ERCP, endoscopy, and hemodialysis [3].

The definitive first line and most beneficial treatment of an arterial gas embolism is hyperbaric oxygen therapy. Initial management of gas emboli should involve cessation of any operation or procedure to prevent further embolization and cardiopulmonary stabilization until arrangements for HBO treatment can be made [4,5]. The patient should be started on 100% high-flow oxygen and vital signs should be monitored. Intubation may be required for somnolent or comatose patients to maintain sufficient oxygenation or to protect the airway [1].

We present eight cases of cerebral arterial gas embolism (CAGE) which we recently treated with HBO with outcomes varying from death to complete resolution.

Cases

Case 1 - Elective lung biopsy

A 70-year-old male underwent computed tomography (CT) guided lung needle biopsy for suspected malignant mass. During the procedure, she became unresponsive and apneic requiring intubation. CT of the head showed diffuse bilateral air emboli in the cerebrum. She became bradycardic and hypotensive, so epinephrine and dopamine were administered. The patient was transferred for hyperbaric oxygen (HBO) therapy starting within 4.5 hours of the inciting event. A repeat CT performed the next day after two HBO treatments showed a substantial hypoxic-ischemic insult to his bilateral superior frontoparietal regions, conferring a grim prognosis. The family requested discontinuation of further HBO treatments and the initiation of comfort care. The next day, the patient was compassionately extubated and died within hours.

Case 2 - EGD after food impaction

A 71-year-old male underwent esophagogastroduodenoscopy (EGD) for a piece of meat stuck in his esophagus. The obstruction was advanced into the stomach. While the patient was being prepared for a subsequent dilation, he became bradycardic, hypoxic, and unresponsive. A CT scan showed multiple air emboli within both cerebral hemispheres. The patient was intubated and transferred to our facility for HBO therapy. HBO was initiated within 7.5 hours of symptom onset. A CT scan performed the next day after two HBO

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treatments showed resolution of pneumocephalus, however, the right cerebral hemisphere developed extensive parenchymal swelling. An EEG showed diffuse cerebral dysfunction. The patient remained unresponsive, and after five HBO treatments in the following days with no neurologic improvement, it was determined that further HBO therapy was futile. On the 8th hospital day, the family decided to initiate comfort measures only, with the patient dying shortly afterward.

Case 3 - CT contrast administration

A 53-year-old female underwent the replacement of a stenotic bicuspid aortic valve without complication. On postoperative day 2, a concerning chest x-ray prompted a CT scan of the thorax. Initial imaging showed gas within the pulmonary arteries consistent with intravenous injection of 50 mL of air, rather than contrast. The patient became hypotensive and hypoxic. She developed right-sided weakness within 20 minutes of the scan as well as a non-reactive and dilated left pupil. A CT scan of the head was performed that was negative for pneumocephalus or ischemic changes but did show some irregularities of her left carotid artery. Neurosurgery was consulted, and CT angiography (CTA) showed a left carotid dissection but no visible air. It was felt that the neurologic deficits and perhaps the dissection were both consequences of arterial embolization although it was somewhat unclear. She regained movement of her right extremities shortly after the CTA, but HBO therapy was still deemed appropriate. The patient underwent two hyperbaric treatments initiated 5 hours after the incident, regaining movement in all extremities and restoration of normal pupil function. The patient was discharged on postoperative day 10 with no further neurologic symptoms or obvious sequelae of gas embolism.

Case 4 - Postpartum s/p C-section

A 31-year-old female had elective Caesarian-section at 39 weeks gestation without complication. The next day she began to feel lightheaded, passed a large blood clot and fluid from her vagina. She was helped to her bed where she became unresponsive. She exhibited seizure-like activity with frothy discharge in her mouth. She became tachypneic, hypotensive, and hypoxic. A nasal airway was placed, and oxygen was administered, restoring her oxygenation to normal. Lorazepam was given for the seizure. She had left upper extremity flaccid paralysis and bilateral vision loss. CT scans revealed gas in her inferior vena cava, right femoral vein, and uterine vasculature. Magnetic resonance angiography was performed showing no cerebral embolism or ischemia. Echocardiography demonstrated a patent foramen ovale. The patient was transferred for HBO therapy for suspected cerebral gas emboli. Upon reaching our facility, all her neurologic symptoms had resolved, without further weakness, paralysis of extremities, or vision changes. Despite the neurologic resolution, the patient was treated with a single prolonged HBO session (US Navy Treatment Table 6) within 6 hours of initial symptom onset. The next day, magnetic resonance imaging (MRI) of her head was performed showing acute to subacute multifocal bilateral watershed infarcts, however, she continued to exhibit no focal neurologic deficits. The patient was discharged in good condition on postoperative day 4 without neurologic sequelae.

Case 5 - IJ central line placement

A 60-year-old female with an extensive past medical history presents to an emergency department (ED) with abdomen and chest pain. She was found to be hypotensive requiring vasopressors. Central venous access was obtained via a right-sided internal jugular (IJ) line. 30 minutes after IJ catheter placement, she developed left-sided hemiparesis. A chest x-ray confirmed line placement in the superior vena cava. A head CT showed air within the vessels between the sulci of the right cerebral hemisphere and within the vessels extending to the right basal ganglia. The patient was transferred for HBO therapy. Upon arrival, she was awake but confused and requiring vasopressors. She continued to have no movement of her left extremities. HBO was initiated 11.5 hours after IJ placement. She had little improvement in movement. A CT scan performed the next day after the completion of two HBO treatments showed resolution of sulcal air, but with loss of gray-white matter differentiation in the right frontoparietal lobe likely representing an acute infarct. The patient completed one more HBO session without improvement, and HBO was stopped due to increasing hypotension, acidosis, and altered mentation. She continued to deteriorate, and her family decided to initiate comfort measures only. Vasopressors were withdrawn and the patient died on hospital day 6.

Case 6 - Rapid infusion of fluids s/p epidural placement and C-section

During a routine epidural placement for a term vaginal delivery, the 26-year-old female patient became hypotensive. Fluid resuscitation was performed with a pressure bag, at which time air was noted to be in the IV line. The patient reported chest pain, shortness of breath, and lightheadedness. It was estimated that 70-100 mL of air was administered into the line. She was stabilized with vasopressor support, and taken for emergent C-section, without further complication. She was then transferred for HBO for possible cerebral air embolism. Upon arrival, the patient was feeling better, with symptoms resolved. She was still tachycardic at 124 with mild hypotension and normal oxygen saturation. Echocardiography showed normal cardiac activity, with no heart strain or air bubbles. CT scans were negative for pulmonary and cerebral gas emboli. She was placed in the HBO chamber 14 hours after delivery for a single treatment. She exhibited no further neurologic or respiratory symptoms and was discharged the next day.

Case 7 - Esophageal stricture dilations

A 58-year-old male underwent esophageal dilation for strictures from esophageal cancer. He was recovering in post-op when a new left facial droop and left extremity weakness were noted. CT revealed air in his right cerebral hemispheric sulci. The patient was transferred for HBO, arriving intubated and unresponsive. He began HBO within 14.5 hours of the EGD procedure. After two treatments, MRI showed extensive ischemic infarction of his right cerebral hemisphere. He exhibited some mild improvement with continued treatments and was extubated. After six HBO treatments, the patient communicated that he no longer wished for further treatments or life-prolonging therapies. He was discharged home on hospice with left hemiparesis.

Case 8 - Manipulation of the stent during ERCP

A 75-year-old male presents with fever, chills, and tachycardia after a biopsy of a liver mass by interventional radiology (IR). A few weeks earlier, the patient had jaundice and an abdominal CT scan showed a new liver mass suspicious for cholangiocarcinoma. The patient was evaluated by gastroenterology and had a stent placed during an endoscopic retrograde cholangiopancreatography (ERCP). After the IR biopsy, the patient was found to have gram-negative bacteremia, concerning for septic cholangitis. The patient underwent a second ERCP, and during the procedure, while the previous stent was being manipulated, the patient became hypotensive and hypoxic. The patient subsequently went into sustained pulseless ventricular tachycardia. The patient was stabilized and intubated, and echocardiography was

performed showing air in the left ventricle and aorta. Head CT scan showed no evidence of pneumocephalus. HBO therapy was initiated within 4.5 hours of symptom onset. By the next day, after two HBO treatments, the patient was extubated, alert, and responsive, exhibiting no further symptoms or residual effects of the arterial gas embolism.

Discussion

The introduction of gas into the vasculature can have a myriad of clinical consequences. The types of injuries incurred from air emboli differ depending on whether they are venous or arterial and where the gas bubbles travel to in the vasculature [6]. A venous gas embolism enters the vasculature in the venous system, where it can then travel to the lungs. Although the lungs can dissipate small amounts of intravascular gas by diffusion across the alveolar membrane [7], larger air bubbles can cause damage by direct occlusion of blood flow in the pulmonary vasculature resulting in reflexive vasoconstriction. This leads to an increase in pulmonary venous pressure causing right heart strain and elevating central venous pressure [1]. Air bubbles also cause mechanical endothelial damage to the pulmonary microcirculation triggering an inflammatory reaction, resulting in cytokine release and neutrophil activation [8]. As illustrated in our case involving the rapid infusion of fluids after epidural placement, these effects create a mismatch between ventilation and perfusion creating a clinical picture nearly identical to that of pulmonary thromboembolism including potential cardiogenic shock and arrest.

Arterial gas emboli have a distinct pattern of clinical manifestations. An arterial gas embolism can occur by direct inoculation of air into the arterial vasculature, the passage of air through the pulmonary capillary bed to the pulmonary veins, or by paradoxical embolization through a right to left shunt such as a patent foramen ovale, arterial-venous malformation, or pulmonary shunt [6]. Once a gas bubble is introduced into the arterial circulation, it can cause end-artery obstruction resulting in ischemia. Gas emboli can migrate to any organ in the body, however, if they travel to the brain or coronary arteries, they can cause significant cardiovascular and neurologic sequelae due to these systems' high vulnerability to hypoxia [6]. The damage produced by these gas emboli is primarily caused by a reduction in perfusion distal to the obstruction and an inflammatory endothelial reaction to the bubble [1]. An arterial gas bubble can embolize to a coronary artery, causing myocardial ischemia and arrhythmias resulting in hypotension, cardiac failure, and shock [4]. Arterial gas emboli similarly travel to the cerebral vasculature, resulting in neuronal cell death. The ischemic injury can quickly progress to infarction, leading to diffuse brain edema and increased intracranial pressure [6], causing a host of neurologic sequelae, as seen in several of our cases. Microbubbles bypassing larger arteries to become trapped in capillaries cause substantial endothelial damage. This results in a disruption of the blood-brain barrier and resultant perivascular edema further reducing blood flow. The endothelial damage also incites leukocyte activation, ensuing cytokine, cytotoxic protease, and free radical release causing additional cellular damage [9].

Treatment with HBO involves placing the patient in a chamber where they breathe 100 percent oxygen at pressures between 2 and 3 atmospheres absolute [10]. HBO therapy treats gas emboli by diminishing the volume of intravascular bubbles via an increase in the ambient pressure and providing a diffusion gradient for nitrogen and other gases to dissipate out of the bubble into solution [11,12]. HBO also increases the partial pressure of oxygen in the plasma, improving oxygenation of ischemic tissues despite reduced blood flow [4,6]. It has been shown to improve cerebral edema by inducing vasoconstriction, reducing vascular permeability, and diminishing leukocyte adherence to damaged endothelium [1]. Furthermore, HBO reduces platelet aggregation, inhibits activation of the coagulation cascade, and reduces free radical production [8].

Table 1. Time to treatment with Hyperbaric Oxygen Therapy and subsequent outcome.

Case	Source	Approximate time to treatment (hours)	Outcome
CT-guided lung biopsy	Biopsy needle	(4.5)	Death
EGD after food impaction	EGD	7.5	Death
CT contrast administration	IV line	(5)	Full recovery/no residual sequelae
Post-partum s/p C-section	Unknown	6	Full recovery/no residual sequelae
IJ central line placement	Central line	11.5	Death
Fluid infusion s/p epidural	IV line	14	Full recovery/no residual sequelae
Esophageal stricture dilation	EGD	14.5	Left hemiparesis on hospice care
Hepatobiliary stent manipulation	ERCP	4.5	Full recovery/no residual sequelae

An important factor in the determination of outcomes for patients with a gas embolism is the time to HBO treatment. In a retrospective study that assessed the relationship between time to HBO therapy and clinical outcome in 86 cases of cerebral air embolisms, it was shown that patients treated within 6 hours of insult had a better outcome [13]. Another study of 36 patients similarly found that patients treated within 6 hours had better outcomes [14]. A study of 16 cases of cerebral air emboli reported half with complete recovery after HBO treatment while 5 more had partial resolution [15].

Although several of our cases had CT scans demonstrating gas emboli, neuroimaging has poor sensitivity and cannot exclude the diagnosis [16]. High suspicion of cerebral gas embolism should avoid delays for imaging in favor of prompt HBO.

Conclusion

In the case reports we've presented, there is no obvious relationship between time to treatment and outcome, but our sample size is small (Table 1). It is likely that the clinical outcome in an arterial gas embolism is as dependent on the circumstances of the initial insult, such as the size of the embolus and its distribution within the vasculature, as on the immediacy of treatment. Nonetheless, the role of HBO therapy in the treatment of air emboli should not be diminished and any delay in treatment should be avoided.

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