Hyperbaric Oxygen & Therapeutic Hypothermia in the Treatment of Arterial Gas Embolisms

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Introduction

Air embolisms is a an uncommon, but potentially catastrophic event that occurs as a consequence of the introduction of gas into the vasculature[1]. Gas embolisms can be further classified by the vasculature involved, organ system, and traumatic or non-traumatic. Cerebral gas embolisms (CAGE) and micro gas embolisms have been reported to occur during open heart surgery [2]. In a recent prospective cohort study conducted in Paris, iatrogenic gas embolism was reported to occur in 2.65 per 100,000 hospitalizations accounting for a crude mortality rate of 21% at one year [3]. Hyperbaric oxygen treatment (HBOT) is considered the only treatment for CAGE [4]. The primary mechanisms involved in treating CAGE is reduction of intravascular bubble size. Added benefits of utilizing HBOT is increasing the oxygen tension, reduction of neutriphils adhesion to the vasculature [5].

Case Report

History
A 23 year-old female with history of Tetralogy of Fallot was admitted to UCLA for pulmonary valve replacement in response to homograft stenosis insufficiency. At two weeks of age in Tehran, Iran she was noted to have a murmur, but was expected to "grow out of this". At one year of life, she had a cyanotic spell which prompted her family's move to the U.S. At 14 months, the patient was found to have Tetralogy of Fallot and anomalous origin of the left anterior descending coronary artery from the right coronary artery. She was then referred for surgical intervention to close her ventricular septal defect and place an aortic homograft in the pulmonary location. At ten years of age, she developed stenosis in her homograft which required replacement of the homograft. At age 18, re-stenosis of her homograft was noted, and required stent placement. When she was 21, in response to increasing gradients across her stent, she underwent angioplasty across the stent.

Intraoperative
The patient was cooled to 32 ºC and placed on cardiopulmonary bypass. Upon intraoperative cannulation of the aorta, transesophageal echocardiogram (TEE) revealed no air in the left side of the heart. However, re-warming and ongoing monitoring of the patient revealed decreasing cerebral oxygen saturation (Table 1[labs?]). After replacement of the homograft with a new 27mm mosaic valve TEE showed a small amount of air in the left ventricle along with right-left PFO shunting. The patient was placed in deep Trendelenburg and a small amount of air was aspirated.

Postoperative Day 0 (Initiation of HBOT and TH)
The patient was transferred to the cardiothoracic ICU intubated and on a ventilator, with vital signs as follows: BP-90/65, HR- 78 bpm, O_2 Sat- 100%. Two hours post-op, the patient still had not started responding despite being off of all sedation. Transthoracic echocardiogram performed 2:19 post-op showed signs of pulmonary hypertension. 2:57 post-op, the patient was given 120 mcg of narcan to rule out overnarcotization, but did not have any significant improvement in mental status. A stat head CT and neurology consult were then completed. The patient began her first hyperbaric oxygen treatment (HBOT) utilizing US NAVY Treatment Table at 5:37 hours post-op and was cooled with ice throughout. During treatment, she was manually bagged and received fentanyl and versed twice each for agitation. Upon completion of HBOT, the patient returned to the cardiothoracic ICU at 8:52 post-op, and therapeutic hypothermia was initiated using Arctic Sun© soon after with a goal core temperature of 33ºC.

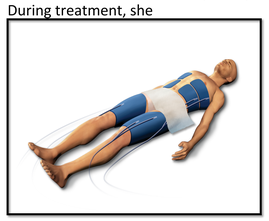

Fig. 1: Arctic Sun™

Case Report

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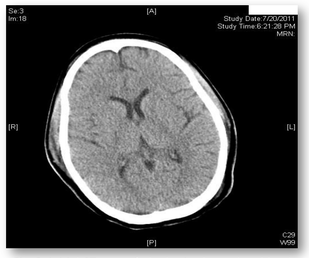

Fig. 2: Initial CT scan of the brain showed no bleed

Postoperative Day 1
Electroencephalogram showed diffuse background slowing suggestive of encephalopathy (anoxic brain injury). The patient received her second HBOT according to US NAVY Treatment Table 6 beginning at 08:56. IV cisatracuium and propofol drips were continued throughout with IV fentanyl boluses for analgesia. Ice packs were applied to the patient in order to continue therapeutic hypothermia, and axillary temperature was monitored throughout. ABGs performed during the procedure showed the patient to by hemodynamically stable. The patient returned to the cardiothoracic ICU upon completion of the treatment at 13:43.

Postoperative Day 2
The third and final HBOT was initiated at 09:16. As before, the patient was kept cool with ice and sedation was maintained with IV dopamine, cisatracurium, and propofol throughout with IV fentanyl boluses for analgesia. Temperature post treatment was recorded as 33.2ºC. The patient returned to the cardiothoracic ICU at 11:35, at which point re-warming was initiated at a rate of .5ºC/hour. An MRA performed at 22:30 showed infarctions along the cortical ribbons suggestive of completed infarcts (Fig. 3,4).

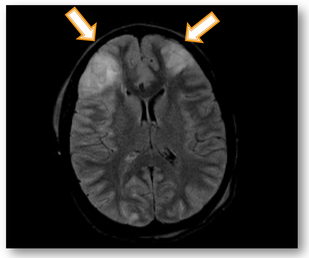

Fig. 3: Flair image showing abnormal signal in the bilateral frontal lobes. There was also restricted diffusion seen on DWI in these areas, which represent completed infarctions in the bilateral frontal lobes in a the watershed MCA-ACA area.

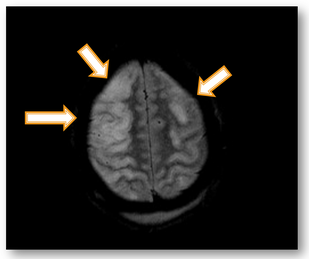

Fig. 4: Flair image showing cortical ribboning in the right greater than left frontal lobes which corresponded to restricted diffusion seen on the DWI image. This abnormal signal represents completed infraction in the right greater than left frontal lobes.

Case Report

Postoperative Day 3
The patient was taken off sedation and muscle relaxation but remained ventilated. Movement of lower extremities and response to basic commands was observed.

Postoperative Day 4
The patient continued to follow commands with increasing consistency (wiggle toes, open eyes). Arctic Sun was restarted in response to her becoming febrile.

Postoperative Day 5
She was extubated, passed a spontaneous breathing trial and was able to speak. Examination revealed 0/5 strength in the left upper extremity, 1/5 in the right upper extremity and 4/5 in both lower extremities.

Postoperative Day 6
Six days post-op the patient was conversant and "making remarkable improvement" according to the neurologist.

Postoperative Day 13 (Discharge)
Patient was able to raise her right arm and feed herself, but remained numb. She was able to lift her left arm from the shoulder but only had weak handgrip. She was discharged to a rehabilitation center for occupational and physical therapy.

Postoperative Day 55
Our unit followed up with the patient via telephone to find her on the bus, conducting her life normally with very few limitations.

Fig. 5: Gonda Center for Wound Healing and Hyperbaric Medicine
David Geffen School of Medicine at UCLA

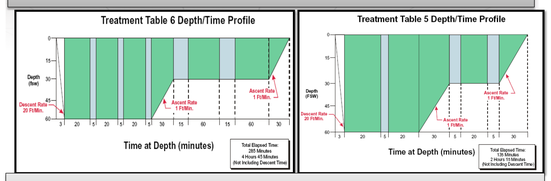

Fig. 6:U.S NAVY Treatment Table 5 and 6
2.81 Atmospheres absolute

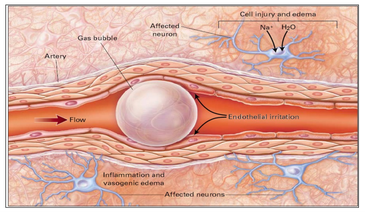

Fig. 7: Bubble Obstructing End-Arterial Flow in a Cerebral Vessel with a Diameter of 30 to 60 µm, Causing Distal Ischemia.The obstruction causes the metabolic processes of neurons to fail. Sodium and water enter the vessel, and cytotoxic edema devel-ops. The surface of the bubble generates a foreign-body response through cellular and humoral immune mechanisms. The bubblealso mechanically irritates the arterial endothelium. Both processes result in vasogenic edema and greater impairment of perfusion.The neuronal injury extends beyond the area of obstruction

Discussion

We presented a case of a young woman treated for arterial gas embolism after undergoing pulmonary valve replacement. This case stands out in the use of hyperbaric oxygen therapy (HBOT) in conjunction with therapeutic hypothermia (TH) to combat anoxic brain injury. There are not many reported cases in which both therapies have been used together. Most of those cases that have been reported utilize the combination of therapies to treat severe head injuries.[8] In our case, the combination of neuroprotective benefits from TH and bubble size reduction from HBOT complement each other in treatment of CAGE.

Hyperbaric oxygenation treats cerebral air embolisms by compression of air bubbles and oxygenation of ischemic tissue with large volumes of plasma-dissolved oxygen. Compression of a vascular bubble in vivo will reduce its size, relieving circulatory obstruction and restoring perfusion. Reduction of bubble size also reduces inflammation caused by interaction between the air bubble and endothelial lumen. High amounts of oxygen dissolve in the plasma when 100% oxygen is inspired since hemoglobin is almost completely saturated at sea level. This oxygen in the plasma reduces cerebral ischemia and facilitates the diffusion of small air bubbles form the pulmonary capillaries to the alveoli for elimination. HBOT may also assist in reducing intracranial pressure in severe cerebral ischemia.[6]

TH uses the controlled reduction of body temperature to protect organs from risk of injury[8]. Proposed functions of TH include reduction of metabolic rate of blood flow in the brain, preservation of high-energy phosphate compounds, prevention of amino acid buildup, and inhibition of innate immune response and associated inflammation[11]. Its protective effects are usually used to treat cardiac arrest patients and those experiencing acute myocardial infarction (MI). Hypothermia may decrease infarct size in these patients who experience acute MI after percutaneous coronary intervention[9].

Open heart surgery comes with a measurable risk of gas emboli. A study by Abu-Oamar et al observed intraoperative microemboli in 45 patients undergoing cardiac surgery. Of these patients, 15 underwent off-pump coronary artery bypass grafting, 15 underwent on-pump coronary artery bypass grafting, and 15 underwent valve replacement with or without coronary artery bypass grafting. Bilateral continuous transcranial Doppler was used to monitor the middle cerebral arteries and formation of microemboli was tracked. A total median of 40 microemboli occurred in the off-pump group with 88% being gaseous, 275 in the on-pump group with 72% gaseous, and 860 in the open procedures group with 78% gaseous. It was found that of the gaseous emboli that occurred, 13% were due to aortic cannulation and crossclamping, 24% were due to the Cardiopulmonary bypass process, 42% were due to removal or aortic crossclamp, sideclamping, and decannulation, and 21% were due to other procedures conducted during surgery (chest opening and closure, harvesting or conduits).[2]

There are numerous other potential causes of AGE occurrence in patients. Venous air emboli may be caused by the manipulation of the central venous catheter. This would include the catheter being unintentionally disconnected, the catheter being improperly placed in the patient, and the catheter being improperly removed from the patient. Venous emboli may also be caused by hemodialysis and neurosurgical procedures. Arterial emboli may occur during extracorporeal circulation or arteriography.[7]

HBOT has been supported as a primary treatment for AGE. A cohort study was performed at Paris' Raymond Poincare´ University-affiliated Hospital in which 119 individuals were admitted to and diagnosed with iatrogenic gas embolisms. A standard treatment of one HBOT session at 4 ATA for 15 minutes followed by two 45 minute plateaus at 2.5 then 2 ATA was administered to all patients. The primary endpoint for the study was 1 year mortality. Crude mortality was 14/119 (12%) at ICU discharge, 19/119 (16%) at hospital discharge, 21/119 (17.6%) at 6 months and 25/119 (21%) at 1-year.[3]

A study by Blanc assessed the relationship between time elapsed before HBOT and clinical outcome of patients with iatrogenic cerebral air embolism. Patients treated with HBOT within 6 hours of embolism introduction had a recovery rate of 68% while patients treated with HBOT after a time lapse of 6 hours had a recovery rate of 40%.[7]

HBOT used in conjunction with TH is rather unique to this case. The combination of treatments in not commonly administered. Very few cases have been recorded using the two treatments, one of which was a patient suffering from cardiac arrest caused by hydrogen sulfide poisoning. 24 year old male was treated with HBOT after collapsing from H_2S poisoning. Due to his experience of cardiac arrest, he was deemed a good candidate for TH, which was administered for 24 hours. The patient made some neurological recovery[10]. HBOT was combined with TH in the case we present to combine the ability to minimize bubble size arteries and the ability to protect and minimize the spread of MI in the brain.

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