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Case report - Cardiopulmonary bypass

Retrograde cerebral perfusion and delayed hyperbaric oxygen for massive air embolism during cardiac surgery

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Abstract

We report a case of massive air embolism from a ventricular vent line during cardiac surgery successfully treated with emergent retrograde cerebral perfusion and delayed hyperbaric oxygen therapy. The etiologies of this rare but potentially devastating complication are discussed along with prevention and treatment options.

Keywords: Gas embolism; Complications of cardiac surgery

1. Introduction

The occurrence of massive arterial air embolism during cardiac surgery is a rare event but one which surgical teams must be prepared for. Such events may cause severe neurological injury or death by direct occlusion of arteries and perivascular inflammation [1]. The most common causes of massive air embolic events during cardiac surgery include inattention to reservoir level, reversed vent line, ruptured arterial tubing, unexpected heartbeat, air introduction during cardioplegia administration, inadequate air removal from arterial circuit, high flow suction deep in pulmonary artery, oxygenator defect, and a pressurized cardiotomy reservior [2, 3]. Various successful treatment strategies have been described including cooling, retrograde cerebral perfusion (RCP) [4], hyperbaric oxygen (HBO) [5], barbiturate coma, lidocaine, oxygen, volume expansion, and inotropic support [1]. We report successful management of a massive air embolism during aortic valve surgery using RCP and postoperative HBO.

2. Clinical summary

The patient was a 77-year-old diabetic man with symptomatic severe aortic stenosis. Median sternotomy was performed followed by cannulation for cardiopulmonary bypass including a distal ascending aortic cannula, a two-stage venous cannula in the right atrial appendage, an aortic root catheter for antegrade cardioplegia and venting, a retrograde coronary sinus cardioplegia cannula, and a left ventricular vent introduced through the right superior pulmonary vein. Cardiopulmonary bypass was initiated. The left ventricle was moderately distended and the left ventricular vent was turned on. The vent, which had previously been filled with blood, was noted to be full of air. It did not have a one-way valve and was found to be connected backwards in the roller head. The vent was turned off and the aortic arch inspected with transesophageal echo which showed the aorta completely opacified by massive air embolism.

The patient was placed in deep Trendelenburg position, continued on cardiopulmonary bypass and a coronary sinus retrograde cardioplegia cannula was placed emergently into the superior vena cava directed cephalad and the vessel then snared. Approximately 200 cc/min of cold blood from the cardioplegia circuit was directed into this catheter with the cardiopulmonary bypass machine turned off and the ascending aortic vent turned on to aspirate air for one minute. This duration was arbitrary and based on weighing concern for the patient being normothermic without antegrade cerebral blood flow with the need to flush air from the cerebral arterial tree. Cardiopulmonary bypass was resumed and the retrograde cerebral perfusion cannula and superior vena cava snare removed and the patient cooled to a core temperature of 28 °C. The aortic valve replacement was completed expeditiously. The patient weaned easily from bypass. Several hours after arrival in the ICU, the patient awoke and moved all extremities but was lethargic and subsequently had a grand mal seizure. Phenytoin was given and neurology consultation obtained. A head CT-scan was normal.

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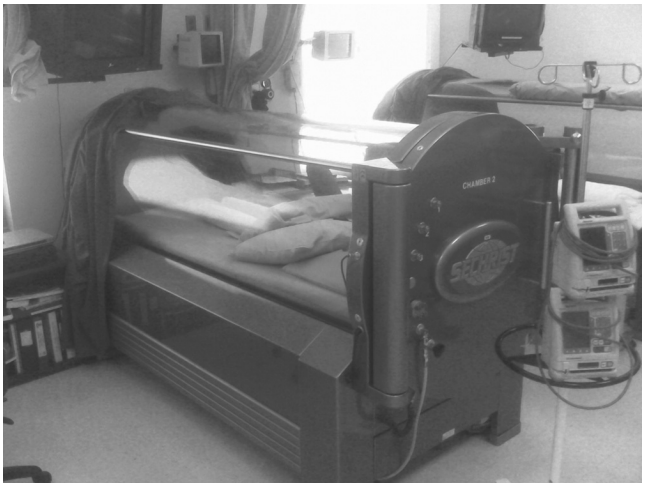

Fig. 1. Hyperbaric oxygen chamber at St Francis Medical Center, San Francisco, CA.

HBO was considered but not done the night of surgery given the need to transfer the patient to another medical facility with a HBO chamber and the associated limitations with monitoring and treating the patient (Fig. 1). In the HBO chamber, only ECG and blood pressure cuff monitors were available. A special ventilator was necessary and arterial or pulmonary artery pressure monitors were not allowed. Special medication infusion pumps are necessary to overcome the increased pressure and these were limited to two. In addition, if the patient had an emergent problem, there would be a significant delay in evacuating him from the chamber.

The morning of postoperative day one the patient had no focal motor deficits but clearly had global cortical depression with severe lethargy. He was taken to a local hospital with a HBO chamber. He was rapidly compressed to 2.8 Atmospheres absolute (ATA) for 30 min, then slowly decompressed to 2.0 ATA for an additional 60 min, and then brought back to surface pressure. The patient was removed from the chamber and subsequently returned to the ICU at our facility. In the ensuing 48 h after this, he slowly regained normal consciousness and was extubated. He had a complete neurological recovery and was discharged.

3. Comment

As a result of this experience, our institution made several changes in our protocols to include testing of all vent lines under saline prior to use, verification of a two-way valve in all vent lines added to the perfusion check-list, and changing the prefabricated pump packs to include two vents with one-way valves already in place. These steps should be considered by all cardiac surgical units and will dramatically reduce the chance of this same event occurring.

The evidence in support of emergent retrograde cerebral perfusion combined with postoperative hyperbaric oxygen is limited to case reports and small series [4]. However, the efficacy of retrograde cerebral perfusion to prevent embolic events in aortic arch surgery is well established [6]. Duration of RCP in this setting is not established but we believe that in a normothermic patient, the duration should be limited to 1–2 min to avoid warm ischemia. Additionally, no specific cooling protocol can be recommended, but we did cool to 28 °C based on the commonly held assertion that this protects the brain from ongoing injury. We would consider this optional.

HBO is the primary treatment for arterial gas embolism. Acutely, HBO can decrease bubble size by increasing ambient pressure. According to Boyle's law, as pressure increases, the volume of a gas decreases proportionally [7]. Elevated arterial oxygen tension produces a sharp gradient that increases the rate of diffusion of nitrogen (or other gas) from the bubble, into solution [8]. Hyperoxia also may be beneficial by inhibiting ischemia-reperfusion injury [1]. Treatment, even delayed by 3–48 h may still benefit the patient by decreasing bubble size, treating hypoxic areas of brain, and impairing WBC adherence to activated endothelial cells [9]. Although successful use of HBO has been most commonly associated with initiation within hours of air embolism, anecdotal successful cases of 20 h and beyond have been reported [5]. We believe HBO should be pursued as soon as feasible but within 48 h of massive air embolism.

The increasing rarity of such catastrophic events can lead to an inadequate response by the surgical team. The solution to this issue may be establishing a standard set of simulated intraoperative crises that the surgical team drills on periodically as in the airline industry.

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