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CASE 5—2015
Early Detection and Treatment of an Air Embolism During Cardiac Surgery

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THE INADVERTENT INTRODUCTION of air during cardiac surgery into the ascending aorta before aortic cross-clamping is a rare but potentially catastrophic complication.[1] Cardiac surgeons and anesthesiologists must be prepared to deal with this emergency, because rapid and decisive action can prevent or attenuate severe neurologic injury that may result from an air embolism. The authors present a case of an air embolism during mitral valve repair surgery that was treated successfully with early detection and management. A continuous quality improvement process following a previous instance of air embolism contributed to the successful outcome.

CASE PRESENTATION*

A 62-year-old woman with a history of severe mitral regurgitation, chronic atrial fibrillation, and mildly dysfunctional left ventricular systolic function presented for mitral valve repair. The patient was monitored with an electrocardiogram, pulse oximetry, a left radial arterial catheter, a right internal jugular pulmonary artery catheter, bispectral index (BIS) monitor (Aspect Medical Systems, Newton, MA), and cerebral oximetry monitoring (FORE-SIGHT, CASMED, Branford, CT). After median sternotomy, the patient was cannulated for cardiopulmonary bypass with an ascending aortic cannula, separate bicaval venous cannulation, an aortic root catheter for antegrade cardioplegia, and a retrograde coronary sinus cardioplegia cannula. Immediately after initiation of cardiopulmonary bypass and before application of the aortic cross-clamp, air was noted in the cardioplegia line. The surgeon instructed the perfusionist to immediately stop flushing through the aortic root catheter. The aorta then was inspected by transesophageal echocardiography, which showed air in the aortic arch and descending aorta. The BIS monitor showed an abrupt reduction from 28 to 0 and then recovered in less than a minute. The cerebral oximetry monitor also showed an abrupt bilateral reduction from 66 to 45 with quick recovery. The patient was placed immediately placed in steep Trendelenburg position, the mean arterial pressure was raised to 120 mmHg, and the patient was cooled to 24°C. One hundred percent inspired fraction of oxygen was used during the resuscitation period to reduce bubble size. Retrograde perfusion via the superior vena cava at a flow of 500 mL/min for 5 minutes was performed with the aortic root vent turned on to aspirate air. Cardiopulmonary bypass then was resumed and the aortic cross-clamp was applied. The patient was maintained at 28°C for the next hour and then slowly rewarmed to 35 degrees. The remainder of the surgery was uneventful, and the patient was weaned from bypass using epinephrine at 50/ng/kg/min.

The patient arrived hypothermic (34.8°C) to the Cardiothoracic Intensive Care Unit. Warming via a bear hugger was commenced, and all sedation was held to assess neurologic status. Two hours later, continuous jerky motions of all 4 extremities were observed. A propofol infusion was started for possible seizure activity, and neurology was consulted. An electroencephalogram (EEG) found mild burst suppression but no seizure activity. The patient reached normothermia 2 hours later. Sedation was held to further assess neurologic status.

During this same time period, the patient was resuscitated from cardiac surgery. Her lactate peaked at 6.5 mmol. She required infusions of norepinephrine at 70 mg/kg/min, epinephrine 50 mg/kg/min, and vasopressin 2.4 u/hour to support her hemodynamics. That evening she suffered a witnessed grand mal seizure and was loaded with diphenylhydantoin and started back on propofol. EEG at this time showed one episode of epileptiform activity from the right hemisphere. A computerized tomography (CT) scan of the head found no hemorrhage, mass, or infarction.

On the morning of postoperative day (POD) 1, the patient was opening her eyes and moving all extremities, but not following commands. There were no metabolic abnormalities at that time. Based on her clinical exam, propofol was discontinued, and she continued to be observed for neurologic recovery. That afternoon the patient suffered another witnessed

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1053-0770/2602-0033$36.00/0
http://dx.doi.org/10.1053/j.jvca.2014.10.007
Key words: cardiac surgery, cardiopulmonary bypass, air embolism

tonic-clonic seizure. Propofol was restarted, and the patient was given a loading dose of levetiracetam (1,500 mg). Additional neuroprotective measures were instituted, including maintaining the mean arterial pressure >80 mmHg for cerebral perfusion, mild hyperventilation with maintenance of arterial partial pressure of carbon dioxide around 35 mmHg, and maintaining the sodium in the range of 140 to 150 mmHg. The patient also became febrile to 38.5°C, and active cooling was commenced. A repeated head CT again found no infarction. Throughout this time period, the patient remained on the same dosages of vasopressor infusions.

On POD 2, the EEG continued to show no signs of seizure activity and was discontinued. The propofol infusion was discontinued, and the patient was observed for clinical signs of seizure. Throughout the day, the patient was noted to be arousable and localizing with both arms but not following commands. She continued to be febrile. On POD 4, the patient woke up and began following commands and moving all extremities purposefully. She was extubated later that evening. She was discharged from the intensive care unit on POD 10 and home on POD 17.

DISCUSSION

Cerebral air embolism during cardiac surgery is an exceedingly rare intraoperative event with potentially devastating consequences.[1] Numerous potential sources for air entrainment exist, including air from within the anterograde cardioplegia lines, inadequate air removal from the arterial circuit, nonobservance of cardiopulmonary bypass machine reservoir level, inadvertent reversal of pump heads, spontaneous return of electrical cardiac activity with open cardiotomy, inadequate evacuation of air postcardiotomy, and a defective oxygenator.[2] The ensuing injury is caused by mechanical obstruction of blood flow resulting in inadequate perfusion pressure as well as endothelial damage and inflammation.[3] Air embolism can be detected by visualizing entrainment in the surgical field, hearing the rush of air enter an open vessel, or via intraoperative transesophageal echocardiography.[4]

Other monitoring modalities that may be useful for determining the cerebral effect of the air embolism are cerebral oximetry, BIS monitoring, and transcranial Doppler. Cerebral oximetry, based on near-infrared spectroscopy technology, provides information on the availability of oxygen in brain tissue at the microvascular level. It reflects regional cerebral metabolism and the balance of local cerebral oxygen supply and demand.[5] Noninvasive cerebral oximetry has been used to detect intraoperative stroke.[6] The bispectral index processes a single frontal EEG signal, indirectly measuring voltage fluctuations in neurons, and is quite sensitive to ischemia. Leggat et al[7] reported a case of an acute fall in bispectral index to single digits due to embolic phenomena during cardiopulmonary bypass for aortic valve replacement. Transcranial Doppler has been used to detect microemboli during transcatheter aortic valve implantation and can be used similarly to diagnose cerebral air emboli.[8] However, utilization of Transcranial Doppler is limited by availability of the machine and often poor detection windows in adults.

When cerebral air embolism is suspected, a multidisciplinary strategy is warranted to achieve the best outcome. The patient should be placed in steep Trendelenburg position to assist in preventing further cerebral air entrainment and to exploit the open heart or aorta as a means of air egress.[1] The patient then should be cooled, a higher-than-normal mean arterial pressure should be maintained, and a decision whether or not to use retrograde cerebral perfusion (RCP) needs to be made.[1]

Although the literature on the use of cooling or RCP to provide cerebral protection after air embolism is limited to case reports,[1,9] several authors have used these techniques successfully to prevent further debris and air embolism.[10] The case reports do not provide a standardized time or temperature for cooling or RCP. One report on cerebral air embolism suggested cooling for 1 to 2 minutes at 28°C. The patient in that case ultimately had a full neurologic recovery.[1]

Flushing the cerebral vasculature via RCP and cooling also can be augmented with hyperbaric oxygen therapy (HBOT) and has been shown to be beneficial within 48 hours of the air insult.[1] HBOT works by decreasing the volume of the entrained cerebral air. Gibson et al[11] also showed that HBOT increased oxygenation of ischemic tissues, decreased intracranial pressure and cerebral edema, and truncated the inflammation associated with cerebral air embolism. In their review of 12 patients receiving HBOT after post-cardiac surgery stroke, 10 patients appeared to benefit from this therapy, with either complete resolution of neurologic symptoms or mild residual deficits. However, HBOT is not practical for most patients due to the need to transfer an often unstable postcardiac surgery patient to a facility with an available hyperbaric chamber and only limited monitoring (ECG, cuff blood pressure) is possible in the hyperbaric chamber.

Another consideration in cases of suspected massive cerebral air embolism is whether or not to proceed with the surgical procedure when it is an elective case. Cardiopulmonary bypass (CPB) is known to induce cerebral edema; superimposing CPB on cerebral air embolism may aggravate the neurologic injury. A discussion should be held whether to monitor the patient and return to the operating room a few days later to continue the operation if the patient has recovered.

Critical events in the operating room require attention, swift action, and coordination to minimize patient injury. Among intraoperative crises, massive cerebral air embolism is an exceedingly rare event relative to the more common cardiac arrest and hemorrhage. The manner in which the perioperative team responds to such events may impact patient outcomes. Without routine evaluation and practice of critical skills, practitioners are less able to appropriately manage crises. In reviewing cases in need of advanced cardiac life support, studies have revealed lacking adherence to standard practices as well as knowledge gaps, with skill decay noted as early as three months after certification.[12] In light of this, crisis simulation may prove to be effective in improving the quality of care delivered. Additionally, specific educational programs aimed at management of crises in the cardiac surgery setting have focused on team building during simulation, which has been proven to enhance performance.[13] Studies are needed to assess whether such educational programs will result in beneficial