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Massive Air Embolism at the Initiation of Cardiopulmonary Bypass: Case report

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KARDİYOPULMONER BYPASS ÖNCESİNDE OLUŞAN MASİF HAVA EMBOLİSİ: OLGU SUNUMU

Masif hava embolisi kardiyopulmoner bypass'a (KPB) giren olguların yaklaşık %0.1-0.2'sinde oluşabilir. Bu hastaların yaklaşık yarısında kalıcı nörolojik hasar veya ölüm görülmektedir. Perfüzyon sistemine büyük miktarlarda hava çeşitli yollarla girebilir. Masif hava embolisinin kalıcı hasarlarından korunmak amacıyla derin hipotermi ve serebral koruyucu ajanlar kullanılmaktadır. KPB sırasında oluşan hava embolilerinin tedavisi için superior vena kava yoluyla retrograt serebral perfüzyon kullanımı ve etkinliği bildirilmiştir [1].

Biz bu yazıda KPB öncesinde oluşan masif hava embolisinin başarılı tedavisini sunuyoruz.

Anahtar kelimeler: *Hava embolisi, hipotermik dolaşım arresti, retrograf serebral perfüzyon.*

Embolization of air intraoperatively is a potential cause of cerebral vascular accidents. Air embolization may occur with residual bubbles infused through the arterial inflow line, inadequate deairing of the aorta following removal of side-biting exclusion clamp, venting of the left ventricle, of following combined valve and coronary operations [4].

We report the successful management of an accidental MAE at the initiation of CPB.

CASE REPORT

A 56-year old man who had aortic insufficiency underwent aortic valve replacement. Anesthesia was induced with fentanyl 50 mg/kg, midazolam 0.1 mg/kg, and pancuronium 0.1 mg/kg/min. A pulmonary catheter was inserted via the right internal jugular vein, nasopharyngeal and rectal temperature probes were placed.

After aortic cannulation, two staged single venous cannula was inserted into the right atrium. Vent catheter was inserted into the left atrium through the right superior pulmonary vein. Suddenly the heart was distended and hypotension occured. When we want to initiate CPB a large amount of air was detected in aortic cannula. We disconnected the aortic cannula and realized that the left heart was full of air. When we were trying to purge the air from the heart through the disconnected aortic cannula; the heart fibrillated. At this time bilateral pupillary dilatation occurred, however as we do not use EEG monitoring routinly, no data is available in regard of the electrical activity of the brain. The patient was brought to head-down position at about 20 degree angle and the carotid arteries were compressed. His head and neck were surface cooled. The aorta was clamped and CPB initiated after priming of aortic cannula. At the same time we administered thiopental 5 mg/kg bolus, mannitol 20 %- a- 2mg/kg, methyl prednisolone -2 mg/kg, and then thiopental was infused 1 mg/kg/hour during the CPB and continued for 12 hours. The most probable site for air to enter the heart could be the vent line. Because the clamp on the vent line was loosened, we reclamped the line and at the same time the perfusionist realized that the circuit of the vent was improperly placed to the roller head and instead of creating a suction it caused a positive pressure and MAE.

Immediately after the initiation of CPB, aortic cross-clamp was applied and antegrade/retrograde blood cardioplegia was commenced. The patient was started to cool and during this time we changed from single to two venous cannulas. When nasopharyngeal temperature reached 18 °C, the pump was stopped, the aortic clamp was removed and retrograde cerebral perfusion was initiated at a flow rate of 0.3 l/min, keeping the pressure of superior vena cava below 25 mmHg. Retrograde cerebral perfusion was continued for 15 minutes to purge the embolized air completely from the cerebral artery system. By the mean time carotid arteries were compressed intermittently to purge the air from the vertebrobassilary system. After these manipulations aorta was reclamped and antegrade perfusion recommenced. Aortic valve replacement was accomplished with a Carbomedics 23 A valve (Carbomedics Inc., Austin Texas). Twelve hours after operation he awakened completely having no paralysis or sensory disturbance or convulsion, and he was extubated without any complication.

DISCUSSION

Neurologic and neuropsychological dysfunction are significant and undeniable risks of cardiac surgery [3]. There are several causes for neurologic injury

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during CPB. Accidental massive air embolism frequently leads to a lethal outcome. Stoney and associates[1] estimated the prevalence of air embolism to be 0.11%.

Hypothermic cerebral protection such as perfusion hypothermia with or without selective brain cooling using and antegrade or retrograde approach or circulatory arrest of the brain has been widely used [4]. The safety of hypothermic circulatory arrest (HCA) is largely due to a temperature dependent reduction of metabolic rate. Increased solubility of gas bubbles into solution is clearly another advantage of hypothermia besides reduction of metabolic rate [5]. Neurologic injury is the most feared complication of HCA. The central nevous system is very sensitive to anoxia; traditionally this sensivity has limited the use of HCA to durations less than 60 minutes at 18 to 20 °C. The optimal temperature for cerebral protection during HCA is not known. An experimental study suggested that deeper levels of cerebral hypothermia confer better protection against neurologic injury during prolonged HCA [6]. Currently the most effective means of protecting the brain is hypothermia. Hypothermia reduces cerebral blood flow, metabolism and preserves cellular stores of high-energy phosphates [7].

Retrograde cerebral perfusion through the superior vena cava has been used for the treatment of cerebral air embolism during CPB. Clinically this method using profound hypothermia has also been employed for the protection of the brain during surgical treatment for diseased aortic arches [2]. Watanabe and associates reported successful treatment of MAE by using retrograde cerebral perfusion [8]. In their report, the patient was already cooled when the MAE occurred. However, in our case MAE occurred when the patient was normothermic and before the initiation of CPB. We believe that immediate initiation of deep hypothermia in our case, resulted in increased solubilty of air bubbles into solution and decreased cerebral metabolic rate; thus causing no severe cerebral damage.

Another approach to reduce cerebral injury is pharmacological. Barbiturates represent one of the earliest and most extensively studied agents. Sodium pentobarbital was shown to decrease lactate accumulation and improve maintenance of ATP and phosphocreatin levels in primates when used to induce and maintain anesthesia before normothermic cerebral ischemia. Additional studies have confirmed the protectiv effects of barbiturate administration against normothermic regional ischemic injuries in primates [9]. The use of barbiturates as an adjunct to hypothermia to obtain a grater cerebro protective effect was investigated in a prospective randomized clinical study [10]. This study demonstrated the efficacy of thiopental administered in a sufficient dose before the initiation of hypothermic CPB. We also administered thiopental with continuous infusion in CPB and ICU. W recommend to administer the barbiturates before cooling of brain in all patients undergoing HCA.

We conclude that retrograde cerebral perfusion is convenient with the adjunctive pharmacological therapy for MAE, in all situations of MAE during cardiac surgery.

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