Pulmonary gas embolism after parotid resection

J. P. SOULIOS, Ph. PARIS, M. BRACCIO, N. KUYRKCHYAN and S. DAMMOUS

Abstract: Pulmonary gas embolism can have very variable consequences and may become a real challenge for anaesthesiologists. We hereby report a case of major pulmonary embolism which took place under unusual circumstances and was documented echocardiographically.

Key words: Pulmonary gas embolism; parotid resection; cardiac arrest.

INTRODUCTION

Pulmonary gas embolism is caused by air entering the systemic venous circulation. Air travels to the right ventricle and the pulmonary circulation. The impact of embolism is very variable. Small air emboli are often clinically silent whereas large emboli can cause serious impediment to blood flow and can potentially result in death. The most common predisposing factors to pulmonary air embolism are sitting position surgery and central venous catheterization in hypovolemic states. Many cases occur during catheter manipulation, disconnection, hub fracture or removal, and not only during central venous catheter insertion (15, 16). Definite diagnosis is not always possible.

Our patient (40 y/o, 83 kg, 180 cm, ASA 1) had undergone left parotid resection for benign adenoma. Standard equipment was used to monitor noninvasive blood pressure, SpO2 and EKG. A 20 G catheter was placed in the left hand. Propofol and remifentanil were used for anesthesia and rocuronium was used for curarization. Ventilation was done with 50% O2 and 50% N2O. The surgery went well. Arterial blood pressure had gradually decreased to 70 mm Hg 30 minutes post induction and required 5 mg ephedrine and 500 ml 6% hydroxyethylstarch to normalize. Prior to extubation arterial blood pressure was 115/85 mm Hg, heart rate 48 bpm, sinus rhythm, O2 hemoglobin saturation 98%. The patient was extubated immediately after surgery and developed a stridor immediately following extubation, soon to be followed by cardiovascular collapse and ventricular fibrillation. The patient was reintubated and was given cardiopulmonary resuscitation. During initial CPR exhaled CO2 remained very low (12 mm Hg) despite good resuscitation efforts. Following defibrillation and 1 mg adrenaline × 2, sinus rhythm restarted without exhaled CO2 improvement. CPR was continued. The time lapse between ventricular fibrillation and restored sinus rhythm was 10 minutes. It took 5 additional minutes for exhaled CO2 to suddenly improve and rise to 37 mm Hg. The patient was given 500 ml 6% hydroxyethylstarch × 2 during CPR. When arterial blood pressure normalized copious pink froth was observed in the ET tube.

A cardiologist was called in to perform a transthoracic echocardiogram which showed definite air bubbles in the right ventricle 20 minutes post cardiac arrest. The patient was then placed in Duran position (left lateral decubitus and Trendelenburg) as this will place the right ventricular outflow tract below the right ventricular cavity, and allow air to migrate up and out of the ventricular outflow tract. The operative site was examined and revealed no obvious entry point for air emboli. The patient was intubated for 16 hours. Inotropes were weaned : in 2 hours for norepinephrine and in 24 hours for dobutamine. After extubation the patient regained normal motor function and consciousness. When told of the “incident” a few days later he could not believe what happened. The patient was discharged on day 10 with no complaints or sequelae.

DISCUSSION

When air enters the systemic venous system it travels to the right heart and gets lodged in pulmonary capillaries. Depending on the size and the rate of inflow of air emboli consequences vary from

J. P. SOULIOS; Ph. PARIS; M. BRACCIO; N. KUYRKCHYAN; S. DAMMOUS.
Department of Anesthesia, Saint Joseph’s Hospital, Liège, Belgium.
Correspondence address: J. P. Soullos, Department of Anesthesia, Saint Joseph’s Hospital, 4000 Liège, Belgium.
E-mail: yannissoullos@yahoo.fr

© Acta Anesthesiologica Belgica, 2015, 66, n° 2
minor to catastrophic (1). In the minor cases symptoms are negligible and diagnosis difficult. Air microemboli lodged in pulmonary capillaries cause local vasoconstriction that is often subclinical. Air in excess of pulmonary filtration capacity passes into the arterial circulation (2). An atrial septal defect or open foramen ovale could lead to paradoxical embolism (3). It is estimated that 300 to 500 ml air embolus at 100 ml/sec would be fatal in humans. Such lethal volume is easily reached since a 14-gauge catheter with 5 cm H2O pressure differential promoting air entry would be sufficient for its production (4). When air bubbles are large enough right ventricular output can be severely impeded which may lead to cardiovascular collapse as in our case (5).

The diagnosis of pulmonary gas embolism is not always made with certainty due to air resorption which can occur prior to actual testing, and because many diagnostic signs are indirect: sinus tachycardia, acute cor pulmonale on EKG, pulmonary edema on radiography, hypoxia and hypercapnia on blood gases, drop in exhaled CO2 (1). Demonstration of air embolism can be made by computerized tomography (6), pulmonary angiography (7) or echocardiography, the latter often being the fastest modality (8).

Treatment of major pulmonary embolism consists primarily in the management of cardiovascular collapse: pure O2 mechanical ventilation, vasoconstrictors and IV fluids. Pure O2 has the benefit of lowering blood N2 partial pressure which accelerates air resorption (9, 10). We noted in our patient, after sinus rhythm was restored, very low exhaled CO2 levels, which persisted for about 3 to 5 minutes, and then suddenly normalized. This normalization can be explained by improved cardiac output, but in view of its sudden nature it may also reflect mobilization of flow-obstructing bubbles (11). We did not realize what was happening until transthoracic echocardiogram was performed. The patient was then placed in Durant position which was supposed to direct right ventricle air away from flow into the main pulmonary artery (1, 5, 11). The efficacy of that position has been challenged (17) and because changing body positions during surgical procedures may pose some risk to patients, the potential benefits should be weighed in case the patient requires CPR. Nevertheless, recent guidelines advise the left lateral decubitus position to prevent right ventricular obstruction by airlock (19).

During the stabilization of the patient prior to exploration of the surgical site significant pulmonary edema developed out of proportion to administered IV fluids for resuscitation. Bubbles in the pulmonary circulation are associated with endothelial lesions which can lead to non-cardiogenic edema (12).

No venous line disconnection was found. Examination of the operative site yielded nothing. It is felt that the stridor that developed immediately on extubation probably caused enough negative pressure to explain sizeable air entry through a defect that could not be found later. Semi-recumbent position may have allowed an “a minima” air entry during surgery which resulted in collapse only at the end of the procedure.

Cardiac arrest increases mortality in pulmonary gas embolism. Upon admission to the ICU our patient had no Babinski sign and showed no evidence of renal insufficiency, which are other factors that increase mortality (13). Our patient was not treated with hyperbaric O2 that is known to improve the prognosis (14).

Pulmonary gas embolism is fearsome for two reasons: it can lead to death and most of the time unpredictable.

References