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Cerebral gas embolism after the removal of the jugular vein catheter in a patient with underlying congenitally corrected transposition of the great arteries: A Case Report

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Abstract

Cerebral gas embolism following central venous catheter removal is an uncommon complication. We report a fatal case of cerebral gas embolism occurred in a patient with underlying congenitally corrected transposition of the great arteries after the removal of a jugular vein catheter.

A 57 year- old man with a history of congenitally corrected transposition of the great arteries was admitted to our hospital with cardiac pulmonary oedema. In order to receive renal replacement therapy, a double-lumen 12 French catheter was inserted in the left internal jugular vein. On the discharge day the central venous catheter was removed while the patient was in a sitting position. Immediately after the removal of the catheter, the patient lost his consciousness and no pulse could be detected. Cardiopulmonary resuscitation was performed. Left hemiplegia was diagnosed while an emergency brain computerized tomography scan revealed air bubbles in the brain, suggesting cerebral gas embolism. The patient was intubated and mechanically ventilated. Hyperbaric oxygen therapy was decided after which complete resolution of the air bubbles on the brain computerized tomography scan was shown. The patient remained on mechanical ventilation with no improvement of his condition and two days later a lower respiratory tract infection complicated with severe sepsis and septic shock was diagnosed. The patient deceased one week later.

Cerebral gas embolism is a potentially catastrophic complication of central venous catheter manipulation, which is rarely reported in the literature. Clinicians should be aware of the possibility of this rare entity and always maintain a high index of suspicion, because prompt diagnosis and treatment are the keystones to achieve a favourable outcome.

Keywords

cerebral gas embolism; central venous catheter; congenitally corrected transposition of the great arteries; Hyperbaric oxygen therapy; stroke

Abbreviations

CGE: cerebral gas embolism; CT scan: computerized tomography scan; CVC: central venous catheter; lTGA: levo-Transposition of the great arteries

Introduction

Cerebral gas embolism (CGE) is a potentially catastrophic complication of central venous catheter(CVC) manipulation, which is rarely reported in the literature. Recently, 158 published cases of CGE associated with CVCs were reviewed [1]. The overall mortality was 21.7% and clinical predictors of mortality were increasing age, coma, cardiorespiratory arrest shortly after symptom onset and male sex. The most frequent neurological manifestations were sudden-onset focal neurological sign (67.7%), coma (59.5%), epileptic seizures (24.7%) and encephalopathy (21.5%) [1].

Case Presentation

A 57 year- old man with a history of congenitally corrected transposition of the great arteries (levo-Transposition of the great arteries, l-TGA) was admitted to our hospital with cardiac pulmonary oedema. Due to severe respiratory distress and hypoxia, the patient was transferred to the cardiac intensive care unit, intubated and mechanically ventilated. He was initially treated with diuretics, inotropic agents and antibiotics. Nevertheless, his renal function deteriorated, a double-lumen 12 French catheter was inserted in the left internal jugular vein andrenal replacement therapy initiated. On day 8, the patient was extubated and 3 days later, he was transferred to the ward. On day 16, the patient was scheduled for discharge. The CVC was removed while the patient was in a sitting position. Immediately after the removal of the catheter, the patient lost his consciousness and no pulse could be detected. Cardiopulmonary resuscitation was performed and the patient was readmitted to the cardiac intensive care unit. Left hemiplegia with severe brain injury was diagnosed (Glasgow Coma Scale of 6). An emergency brain computerized tomography(CT) scan revealed air bubbles in the right subarachnoid space as well as the cavernous and superior sagittal sinus, suggesting CGE (Figure 1).

The patient was put in the Trendelenburg and left lateral decubitus position, a manipulation known as Durant's maneuver. Oxygen 100% and intravenous methylprednisolone were administered while blood pressure and $PaCO_2$ were closely monitored. A transthoracic echocardiogram showed air bubbles in the right side of the heart as well as the left atrium (Figure 2).A subsequent transoesophageal echocardiogram did not reveal any atrial or ventricular septal defect.

Because of oxygen desaturation, tachypnea (respiratory rate 30 to 35/min) and his poor neurological condition, the patient was intubated and mechanically ventilated. The decision for further hyperbaric oxygen therapy was made as proposed by previous literature. Complete resolution of the air bubbles was shown on the brain CT scan repeated four hours after hyperbaric oxygen therapy, but unfortunately an evolving infarct in the right frontal and parietal area was evidenced too (Figure 3).A repeated transthoracic heart echocardiogram immediately after CT scan, did not detect any air bubbles in the heart chambers.

The patient remained on mechanical ventilation with no dramatic improvement of his neurological condition and two days later a lower respiratory tract infection complicated with severe sepsis and septic shock was diagnosed. The patient deceased one week later.

Discussion/Conclusions

CGE is a potentially catastrophic complication of CVCs manipulation.

From a pathophysiological aspect, the entry of the air into the blood is the result of pressure difference between the venous and atmospheric pressure. This is more frequent during deep inspiration, hypovolemia or having the patient in an upright position at the time of insertion, manipulation or removal of a CVC.

CGE may both involve the venous and arterial circulation.

Air bubbles can travel from the right to the left heart chambers and enter the systemic arterial circulation either via a pulmonary arterial-venous malformation or penetration through the pulmonary capillaries.

In the latter condition, none or minor respiratory symptoms are evident especially when the size of the embolized air bubbles is small since they are usually eliminated in the pulmonary circulation. However, moderate size air bubbles may cause pulmonary vascular damage leading to pulmonary hypertension and pulmonary oedema [2]. Furthermore, larger bubbles may obstruct the right ventricular outflow tract inducing a dramatic increase of the pulmonary artery pressure and may finally pass through the pulmonary circulation and enter the left side of the heart [3].

In the presence of any right-to-left shunt, including atrial (e.g. patent foramen ovale), ventricular septal defect or pulmonary arteriovenous shunt, the air can directly enter the systemic circulation and cause CGE (paradoxical embolism) [4,5].

CGE can also occur retrogradely via the venous system, as demonstrated by experimental data [6,7]. Air bubbles travel upstream as a result of their lower specific weight as compared to blood, and might enter the cerebral circulation, especially when the diameter of the central vein lumen is large [7,8]. Predisposing factors for venous air embolism with CVCs include sitting position, deep inspiration, low central venous pressure and larger size of the catheter.

In our case the transthoracic echocardiogram performed after cardiopulmonary resuscitation showed air bubbles in the right atrium and ventricle as well as in the left atrium with no detection of septal defect which was confirmed in the transesophageal echocardiogram as well. A bubble study was performed during the transesophageal echocardiogram but did not reveal a patent foramen ovale. Unfortunately, the Valsava maneuver could not be performed during the bubble infusion, since the patient was sedated and mechanically ventilated. However, a sustained inspiratory pressure to 25 mmHg for approximately 10 seconds, which can in ventilated patients simulate a Valsava maneuver, was not applied in our patient. Unfortunately, an autopsy which can set the final diagnosis of a patent foramen ovale, was discussed with the patient's family who did not finally give their consent. The presence of air into the left atrium, therefore, can either be attributed to a probe or a temporarily functioning patent foramen ovale ora passive passage from the right to the left heart chambers through the pulmonary circulation. In favor of the latter mechanism is the hemodynamic collapse of our patient, documented shortly after the removal of CVC which can be explained by a sudden obstruction of the right ventricular outflow tract and decreased cardiac output subsequently. However, we cannot completely rule out the possibility of a retrograde venous air embolism.

The diagnosis of CGE is based on medical history, physical examination and clinical suspicion. The clinical presentation of vascular air embolism varies and depends on the affected organ. The cardiac

manifestations are chest pain, bradyarrhythmias or tachyarrhythmias and increased right ventricular filling pressures [9] while the respiratory manifestations include dyspnoea, tachypnoea and pulmonary edema with hypoxaemia and hypercapnia [10].

Principal goals of management include prevention of further air entry, reduction of the volume of the air emboli and hemodynamic support with fluids and inotropic agents [11]. Administration of 100% oxygen improves the patient's oxygenation as well as it reduces air embolus volume by eliminating nitrogen [12]. Previous studies suggested that partial left lateral decubitus position (Durant maneuver) [13], or Trendelenburg position may alleviate air obstruction and excess overloading of the right side of the heart. However, a recent experimental study questions the positive hemodynamic effect of Trendelenburg position after venous air embolism [14]. Rapid initiation of cardiopulmonary resuscitation and chest compression may be particularly efficacious as it improves blood flow and cardiac standstill.

Hyperbaric oxygen therapy is beneficial resulting in compression of air bubbles, acceleration of their dissolution and finally improvement of the oxygenation of the peripheral organs and tissues. During hyperbaric therapy, patient inspires 100% oxygen at pressure above atmospheric at sea level achieving an arterial partial pressure of oxygen greater than 2000 mmHg [15].

The use of steroids is controversial since they do not have any effect on cytotoxic brain edema [16]. Interestingly, animal studies have showed that anticoagulation therapy with heparin [17]and the use of lidocain [18] may improve neurological impairment and prognosis.

In conclusion, CGE is a rare but serious complication. Strict compliance to simple but necessary procedures before, during or after a routine CVC insertion or removal is required [19,20]. First of all, staff awareness is essential. Deep inspiration should be avoided and active expiration should be encouraged during the procedure. Valsalva maneuver might also confer some advantages. Trendelenburg or lying flat position are generally recommended especially when cannulation of jugular or subclavian vein is attempted. Application of pressure with occlusive dressings after the removal of a catheter is recommended. Moreover, air filters in the lines used for continuous intravenous infusion of fluids, special maneuvers during injection of fluids/medications (i.e. holding the syringe vertically)and continuous aspiration through the syringe before and during the removal of a central catheter are crucial in all patients especially in those with congenital heart defects.

Our case underlines the severity of CGE after CVC removal which might be lethal despite hyperbaric oxygen treatment. The clinician should be aware of this rare entity and always maintain a high index of suspicion, because prompt diagnosis and treatment are the keystones to achieve a favourable outcome.

Figures

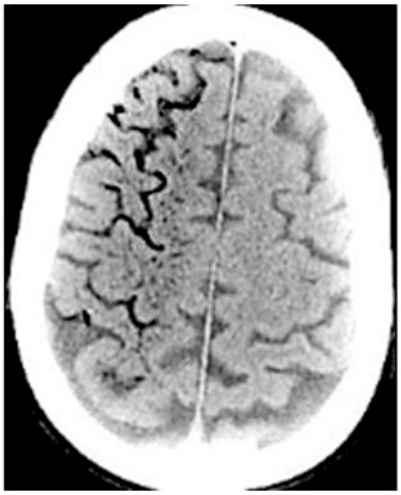

Figure 1: Brain CT showing air bubbles along the sulci in the right frontal and parietal area.

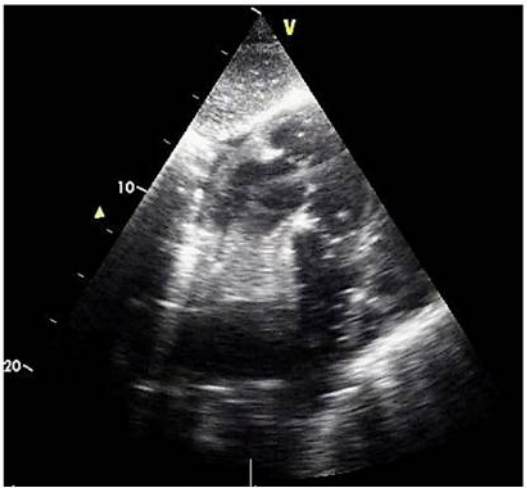

Figure 2: Transthoracic echocardiogram revealed air bubbles in the right heart atrium, right ventricle and left atrium.

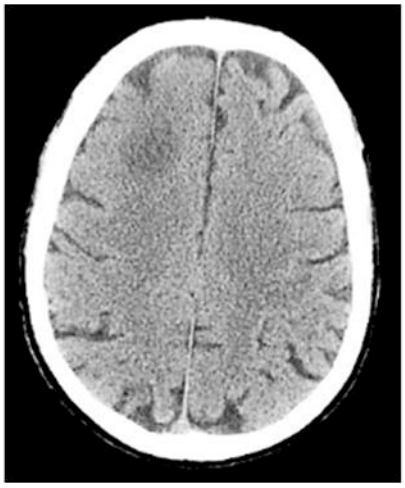

Figure 3: Second brain CT showing resolution of the air and diffuse swelling in the right frontal and parietal area.

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