Case report

Pneumocephalus in a patient with multiple stab wounds

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A B S T R A C T

Pneumocephalus is a complication of trauma to the chest and many iatrogenic interventions. It may arise due to systemic air embolism or retrograde cerebral venous air embolism which is an extremely rare complication. We report a 26-years-old female patient who presented to the Emergency Department suffering of multiple stab wounds. She was in a state of shock and after first aid and evaluation she was operated successfully. In the early postoperative period generalized tonic clonic convulsions were observed following cardiopulmonary resuscitation due to sudden cardiovascular collapse. Brain computerized tomography demonstrated free air in intracranial and extracranial venous structures. Pneumocephalus was diagnosed which may be due to a wide spectrum of etiologies including thorax or spinal stab wounds, tube thoracostomy, cardiopulmonary resuscitation or even central venous catheterization. Unfortunately, the patient ended up with brain death despite all effort. In conclusion, we recommend physicians to be aware of this catastrophic complication while taking care of patients with stab wounds.

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1. Introduction

Cerebral air embolism is a complication with a wide variety of etiologies that may cause catastrophic results.1 Air entering the venous system classically embolizes to the right heart, pulmonary artery and at times through a patent foramen ovale or pulmonary arteriovenous shunt to the brain; an entity the so called ‘paradoxical embolism’.2 This is the most common scenario in clinical settings ending up with stroke due to arterial embolism in central nervous system. On the other hand, retrograde cerebral venous air embolism is a rare but fatal complication with few cases reported in literature.3,4

We present a case of pneumocephalus in a patient with multiple stab wounds to the chest and paraspinal region.

2. Case presentation

A 26-year-old woman presented to the Emergency Department (ED) sustaining multiple stab wounds. On arrival, the patient was somnolent. At first evaluation she was hypotensive (66/38 mmHg) and tachycardic (136 beats/min). Other vital signs were: temperature 37 C, respiratory rate 32 breaths/min and oxygen saturation 82% on room air. Physical examination demonstrated a 2 cm-sized stab wound overlying left midclavicular pectoral region, two 2 cm-sized stab wounds on the left posterior hemithorax close to paraspinal region and a 1 cm-sized stab wound on the right posterior hemithorax close to paraspinal region all of which were extending into the subcutaneous tissue. There was no active bleeding but air bubble discharge was observed from the cut wounds on the left posterior hemithorax. Heart sounds were regular and no murmurs detected. Lung auscultation detected decreased breath sounds on the left hemithorax and portable chest x-ray revealed left traumatic hemothorax (Fig. 1). She was electively intubated and controlled mechanical ventilation support started. Emergently a tube thoracostomy was applied on the left side and 800 ml of hemorrhagic fluid and air was drained. The abdomen was not tender. No
additional traumatic injuries were noted. A right subclavian venous catheter was inserted and fluid and erythrocyte transfusion applied. While receiving bolus fluid challenge and mechanical ventilation support she was urgently transferred into the operating room. Intraoperative exploration demonstrated no major vascular or heart laceration. Parenchymal stab wounds at left upper lobe anterior and lower lobe posterior segments and the stab wound at the right hemithorax were primarily sutured. Additionally a right tube thoracostomy was applied prophylactically. Upon intraoperative consultation Neurosurgery consultants recommended primary saturation of the paraspinal and stab wounds. The patient was transferred to the intensive care unit (ICU) and put on mechanical ventilation support after the operation. On admission to ICU the vital signs were: blood pressure 112/58 mmHg (without inotropic or vasopressor support), heart rate 100 beats/min, respiratory rate 16/min (on volume cycled controlled mechanical ventilation), temperature 37.3 C and oxygen saturation 95%. Control chest x-ray in the intensive care unit showed no complication.

Postoperative arterial blood gas, complete blood count and biochemical analysis were found to be in the normal range. She was hemodynamically stable for 3 h but thereafter suddenly hypotension and bradycardia developed and in seconds she hemodynamically collapsed. Cardiopulmonary resuscitation was started immediately. Cardiac rhythm and hemodynamic stability was achieved in 5 min. Soon after, a generalized tonic clonic convulsion was observed lasting for 1 min. Valproic acid therapy and brain computerized tomography (CT) examination was ordered accordingly to the advices of consultant Neurology physician. Bedside echocardiography revealed no bubble, shunt, D-shape or right ventricular dysfunction. Free air was reported in intracranial and extracranial venous structures and in dural venous sinuses most apparent on the posterior side on brain CT by the Radiology consultant (Fig. 2). Thorax CT revealed neither intravascular air nor pulmonary embolism. Carotid sonography displayed no air in the subclavian or jugular veins. Unfortunately we diagnosed pneumocephalus due to cerebral venous air embolism. She clinically deteriorated and despite all effort the case ended up with brain death also documented by cranial CT Angiography.

3. Discussion

Systemic air embolism most commonly arises from traumatic penetrating or blunt disruption of lung or iatrogenic procedures.5,6 Lung trauma lacerating lung parenchyma, blood vessels or air passages may cause entrance of air into the pulmonary venous system. The incidence of systemic air embolism due to severe lung trauma is reported to be 4–14%.7 Cerebral venous air embolism is extremely rare and may be easily missed especially in an anesthetized or comatous patient. It was reported that air bubbles can cause cerebral venous embolism by retrograde flow in patients with central venous catheters depending on bubble volume, blood flow velocity, superior vena cava diameter and cardiac output.8 Absence or insufficiency of valves in the jugular vein were presumed to be the cause for retrograde embolism.9 External cardiac resuscitation was speculated to cause retrograde cerebral venous air embolism due to increased intrathoracic pressure.10 Heckmann et al reported forced inspiration and hypovolemia as predisposing factors for air embolism in patients with central venous catheters.11 Pneumocephalus usually presents with symptoms including hypotension, bradycardia, coma, hemiparesis and generalized tonic clonic seizures.12 Upon suspicion, the diagnosis can be easily confirmed by brain CT. Further examination of any intracardiac and intravascular air can be performed by echocardiography and doppler ultrasonography.13 Penetrating lung injury or cardiothoracic surgery as in our case are very well known causes of air embolism, but in these cases air entering pulmonary venous system causes systemic air embolism. In our case, according to the report of brain CT by Radiology consultant cerebral venous air embolism in absence of systemic air embolism was remarkable.

As a matter of fact it is not easy to differentiate arterial vs venous embolism in a brain CT but we have to rely on the report of the Radiology Unit. Pneumocephalus was reported to develop due to various etiologies such as thoracic stab wounds, tube thoracostomy, cardiopulmonary resuscitation, injury of the spinal area and neck, central venous catheterization or even in the absence of any air entrance point.14–19 Pneumocephalus in our case can not be attributed to a vague cause but could have happened due to several reasons. Unfortunately, a brain CT evaluation could not be done in the ED because of the hemodynamic status of the patient and emergent indication for operation. Therefore according to one scenario; pneumocephalus could have been already present when she arrived to the ED but sedative and neuromuscular blockage agents may have masked the seizures. Postoperatively the gradual increase of embolus size and weaning of the sedatives may have caused clinical symptoms in the patient. Our patient had almost all of the risk factors for the development of pneumocephalus as reported in the literature. The central catheter was in place when the
tonic clonic seizures started which were attributed to pneumocephalus and all the lines were closed and not in touch with air. If central venous catheterization is the cause, the only left mechanism is that air entrance into the central venous system may have occurred during the placement of the catheter. In such a severe trauma where venous access line is of life threatening importance and peripheral venous puncture was impossible due to shock state there seems to be no other alternative than a central venous line but as a selfcriticism in such a case with multiple risk factors for pneumocephalus femoral vein instead of subclavian vein could have been preferred. Nevertheless when subclavian or internal jugular vein is preferred, lying the patient in Trendelenburg position during central venous catheter placement and removal and firm compression for a considerable time period after removal is strictly recommended to avoid this life threatening complication.11,12

In case of a documented cerebral venous embolism hyperbaric oxygen is the mode of treatment strictly recommended to accelerate the dissolution of air bubbles in the circulation and limit the surface of venous infarction.20 Hyperbaric oxygen treatment could not be applied to our patient because this treatment was not applicable in our institute and the patient was hemodynamically not stable for transport.

Pneumocephalus is not a rare complication in patients suffering from especially thoracic and spinal stab wounds and may have catastrophic results. Physicians must be aware of this potential hazardous complication and various etiologies while taking care of patients with multitrauma.

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**Conflicts of interest**

None declared.

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