Case report

Traumatic cardiogenic shock due to massive air embolism. A possible role for cardiopulmonary bypass

R. Rawlins*, A. Momin, D. Platts, A. El-Gamel

Departments of Cardiothoracic Surgery and Cardiology, Kings College Hospital, Camberwell Green, London SE1, UK

Received 7 February 2002; received in revised form 19 May 2002; accepted 17 July 2002

Abstract

Systemic arterial embolism is a potentially lethal complication of bronchopulmonary venous fistula in trauma patients with blunt chest trauma or isolated penetrating lung injury on positive pressure ventilation. A high index of suspicion, early diagnosis and management in specialized centres are keys to a successful outcome. © 2002 Elsevier Science B.V. All rights reserved.

Keywords: Arterial; Air embolism; Bronchopulmonary; Fistula

1. Introduction

Systemic arterial embolism is a recognized complication of blunt or penetrating chest injuries and should be suspected in all trauma patients on positive pressure ventilation who develop unexplained shock, cerebral symptoms or haemoptysis. We present a case report of a 21-year-old man who developed cardiogenic shock due to systemic and coronary arterial air embolism, confirmed by echocardiography and treated with lung resection and the use of cardiopulmonary bypass.

2. Case report

A 21-year-old man was brought to hospital intubated and ventilated following a gunshot wound to the left chest. At the scene of the shooting he was agitated, tachycardic and tachypneic with stable blood pressure. A left anterior needle thoracostomy was performed for suspected pneumothorax but this proved negative. On arrival at hospital he was stable haemodynamically with normal arterial blood gases. Clinical examination revealed an entry wound 6 cm below the angle of the left scapula and 5 cm from the midline. There was no exit wound but the bullet was palpable overlying the seventh intercostal space in the anterior axillary line. A left chest drain was inserted and 300 ml of blood was recovered. Chest X-ray showed a left haemothorax. Two hours after arrival he suddenly became hypotensive with ventricular ectopics and normal filling pressures. Transthoracic echo demonstrated air bubbles within the left ventricular cavity and moderate segmental left ventricular systolic impairment. The inferoposterolateral walls were akinetic; the remaining segments normal with ejection fraction of 35% and no pericardial effusion seen (Fig. 1).

Abdominal ultrasound was unremarkable and computed tomography scan was cancelled due to cardiovascular instability. Emergency surgery was performed via a median sternotomy to allow full inspection of the heart. It also revealed a lacerated and contused left lower lobe secondary to a large blast injury and a left haemothorax. There was significant right ventricle and left ventricle systolic impairment, elevated pulmonary artery pressure and cardiac decompensation. Right atrial to ascending aorta cardiopulmonary bypass (CPB) was instituted on a beating heart at 33 °C without use of cross-clamp as it became essential to provide ventricular assist and circulatory support. Total bypass time was 38 min. Examination revealed no cardiac injury and the cardiac chambers appeared otherwise normal. A stapled wedge resection, (11.5 × 4.5 × 2 cm) of the left lower lobe was performed on CPB which removed the source of air embolism and allowed the heart to recover. De-airing of the heart was performed continuously while beating on CPB using a 16 gauge cannula inserted into the ascending aorta proximal to the cannulation site with the patient in head-down position and by needle aspiration of the apex of the left ventricle prior to termination of CPB with lungs ventilating. A diaphragmatic perforation was also repaired. Ventricular function immediately improved.
and transthoracic echo performed 24 h post-operatively showed normal left ventricular systolic function. Pathologic lung findings revealed congested lung parenchyma with extensive intra-alveolar haemorrhage and adjacent intra-alveolar oedema. The post-operative course was uncomplicated and the patient was discharged from hospital after 8 days. Of note, there was no central nervous system involvement.

3. Discussion

Systemic air embolism (SAE) is a potentially fatal condition [2,6] and mortality rates of up to 66% [3,4] have been reported. It appears to occur more commonly in patients with penetrating lung injury [3] but should be suspected in all trauma patients with blunt or penetrating chest injuries on positive pressure ventilation who develop unexplained shock, cerebral symptoms or haemoptysis [3,7]. A high index of suspicion and early diagnosis is key to a successful outcome. Survival is influenced by the amount of air entering the systemic circulation, organ supplied by the blocked artery and its ability to withstand ischemia [6]. The mechanism involves formation of a bronchopulmonary venous fistula and becomes apparent usually after mechanical ventilation [5–7] due to bronchopulmonary pressure gradient allowing transmission of air through traumatic bronchial to pulmonary vein communication [8]. Diagnosis is made by direct visualization of intracoronary or ventricular air [3,4]. Transthoracic or transoesophageal echo may be diagnostic and should be considered early.

Air in the coronary circulation may be one of the major causes of pump failure and death due to sudden, irreversible and severe cardiovascular collapse has been described [6]. The objectives of treatment are to remove the source of air embolism, support circulation and allow the heart to recover. Conservative measures include expansion of intravascular space, administration of 100% oxygen, decompressing pneumothoraces if present, decreasing airway pressure (release of positive end-expiratory pressure), low tidal volume, use of high frequency jet ventilation if available and selective single lung ventilation in cases of unilateral injury [1,6] is recommended. Hyperbaric oxygen may be useful [6]. Prompt thoracotomy and hilar cross-clamping on the affected side has been recommended for cases unresponsive to conservative measures [3,6]. The use of cardiopulmonary bypass is not documented in the treatment of systemic air embolism but its use in penetrating intracardiac injury of varied aetiology is well recognized [9,10].

Our patient with systemic air embolism needed surgery, but due to haemodynamic instability an operation could not be safely performed without cardiac assist. Cardiopulmonary bypass provided ventricular assist, circulatory support and allowed rapid de-airing. This enabled the heart to recover while the source of air embolism or bronchovenous fistula was treated. It was successful in our patient and in the authors’ opinion CPB may add to our limited treatment options for salvaging this subgroup of patients with cardiogenic shock (due to massive SAE) who require surgical treatment but where haemodynamic instability further increases the risk and outcomes are poor.

References