Delayed systemic air embolism in a child with severe blunt chest trauma treated with high-frequency oscillatory ventilation

Embolie gazeuse systémique tardive chez un enfant avec un grave traumatisme fermé du thorax et une ventilation oscillatoire à haute fréquence

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Abstract

Purpose Trauma is the leading cause of death in children over one year of age. Even with optimal field care, blunt chest trauma with hemoptysis is a potentially fatal injury due to exsanguination or arterial air embolism. Most often, cardiovascular collapse that is unresponsive to therapy develops shortly after endotracheal intubation and initiation of positive pressure ventilation. We present a case of arterial air embolism after blunt chest trauma that manifested atypically late, i.e., one hour after initiation of positive pressure ventilation.

Clinical features A 13-yr-old Caucasian boy was admitted to the emergency room after he had been run over by a car. While lung protective ventilation, including high frequency oscillatory ventilation, was performed, an alveolar to pulmonary venous fistula developed. Although the complication was diagnosed quickly, involvement of the cerebral and coronary arteries resulted in irreversible cerebral damage and fatal hemodynamic collapse.

Necropsy confirmed severe damage of the right pulmonary lower lobe with involvement of the pulmonary vessels.

Conclusion Patients with blunt chest trauma and hemoptysis present a diagnostic dilemma with limited therapeutic options.

Résumé

Objectif Les traumatismes sont la principale cause de décès chez les enfants âgés de plus d'un an. Même avec des soins optimaux sur le terrain, les traumatismes fermés du thorax accompagnés d'hémoptysie constituent des lésions potentiellement fatales en raison de l'exsanguination ou de l'embolie gazeuse artérielle. La plupart du temps, une défaillance cardiovasculaire ne répondant pas au traitement se manifeste peu après l'intubation endotrachéale et l'amorce de la ventilation en pression positive. Nous présentons un cas d'embolie gazeuse artérielle après un traumatisme fermé du thorax qui s'est développée exceptionnellement tard, soit une heure après l'amorce de la ventilation en pression positive.

Éléments cliniques Un garçon d'origine caucasienne de 13 ans a été admis à l'urgence après avoir été happé par une voiture. Pendant qu'une ventilation protectrice, y compris une ventilation oscillatoire à haute fréquence, était réalisée, une fistule alvéoles-veines pulmonaires est apparue. Bien que cette complication ait été rapidement dépistée, l'atteinte des artères cérébrales et coronaires a provoqué des lésions cérébrales irréversibles et un collapsus hémodynamique fatal. L'autopsie a confirmé la présence de graves lésions au niveau du lobe inférieur du poumon droit et des vaisseaux pulmonaires.

Conclusion Les patients atteints de traumatismes fermés du thorax et d'hémoptysie présentent un dilemme

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diagnostique et les options thérapeutiques pour les soigner sont limitées.

Hemoptysis after blunt chest trauma with lung contusion is a very common symptom and usually self-limiting after several hours of positive pressure ventilation (PPV) with adequate positive end-expiratory pressure (PEEP).¹ Death after blunt chest trauma is usually due to exsanguination or very infrequently due to arterial air embolism.^{2,3} A traumatic fistula between the alveolar space and the pulmonary venous circulation is very unusual but typically presents with hemoptysis as a sole but insensitive clinical sign. We describe the diagnostic and therapeutic dilemma in the emergency room and in the intensive care unit (ICU) involving an unfortunate young child whose mother gave written consent for publication of this report.

Case description

A 13-yr-old Caucasian boy was admitted to the emergency room after he had been run over by a car. On admission, he was conscious (Glasgow Coma Scale 13), but he complained of shortness of breath with hemoptysis and pelvic pain. Pulse oximetry upon arrival showed a saturation of 55% under oxygen insufflation by mask with oxygen at 10 L·min⁻¹. The electrocardiogram (ECG) showed a sinus tachycardia at a rate of 135 beats·min⁻¹; the blood pressure was 65/35 mmHg. Uneventful endotracheal rapid sequence intubation was performed. Reduced right-sided breath sounds led to bilateral chest tube insertion using an atraumatic technique. Ventilation employed a lung protective strategy in the pressure-controlled mode (fraction of inspired oxygen = 1.0; PEEP = 10 cmH₂O; peak inspiratory pressure = 25 cmH₂O; respiratory frequency = 20 breaths·min⁻¹; tidal volume = 250 mL). A triple-lumen high-flow central line was placed in the left subclavian vein. In addition, cannulation of the right radial artery was performed. Volume resuscitation was initiated. Blood gas analysis after initiation of PPV revealed the following results: hemoglobin = 8 mg·dL⁻¹, PaO₂ = 70 mmHg; PaCO₂ = 75 mmHg; pH = 7.19; base excess = -2; SaO₂ = 100 %; SvO₂ = 75%. Transesophageal echocardiography (TEE) was normal except for a hypovolemic left ventricle. In particular, TEE showed no evidence of right ventricular dysfunction. The results of the multislice whole body computed tomography study were available shortly thereafter. There was an occipital fracture of the patient's skull without intracranial pathology and a severe contusion of the right lung with fractures of ribs 1-5 (Fig. 1). The bilateral chest tubes were positioned correctly without evidence of pneumothorax, and a posterior pelvic fracture

Fig. 1 Multislice computed tomography scan of the chest with contrast agent showing severe right-sided contusion predominantly in the dorsal parts of the lung

with retroperitoneal hematoma was confirmed. Volume resuscitation to this point in time consisted of colloid 1,000 mL and crystalloid 1,000 mL, resulting in an improvement of the arterial pressure to 110/80 mmHg and moderation of the heart rate to 90 beats·min⁻¹. The patient was transferred to the ICU. Forty-five minutes following admission, the patient's hemoglobin was 6.5 mg·dL⁻¹, and 2 U of packed red blood cells were transfused, resulting in partial restoration of hemoglobin to 9.5 mg·dL⁻¹. Since hemoptysis was present at the time of admission, suggesting a connection between the bronchial system and thoracic blood vessels, high frequency oscillatory ventilation (HFOV) was initiated with the following settings: mean airway pressure = 18 cmH₂O; oscillation amplitude = 50 cmH₂O; respiratory frequency = 300 breaths·min⁻¹; F_IO₂ = 1.0. Blood gas analysis ten minutes later showed normocapnia and PaO₂ = 550 mmHg. One hundred fifteen minutes after the boy's admission to hospital, sinus bradycardia (40 beats·min⁻¹) ensued, accompanied by ST-changes in ECG leads II and III and in the ECG augmented limb lead, aVF. In addition, the creatine kinase-MB was elevated significantly (262/369 U·L⁻¹) with a normal troponin I. The patient gradually developed bilateral mydriasis. Anticipating the development of a malignant cerebral edema, 15% mannitol 250 mL was infused. Noradrenaline was increased from 0.2 to 0.9 µg·kg⁻¹·min⁻¹ in order to maintain a mean arterial pressure > 70 mmHg. An emergency computerized tomographic scan of the patient's head and chest at this time revealed arterial air embolism in all cerebral arteries (Fig. 2) as well as in the coronary arteries and in the left atrium and ventricle. There was progressive hemodynamic instability. In response, noradrenaline was increased in a stepwise manner to 150 µg·min⁻¹. A repeated TEE examination showed bilateral ventricular failure with an akinetic free wall of the right ventricle and akinesia of the posterior, lateral, and septal part of the child's left ventricle.

Fig. 2 Cerebral computed tomography scan without contrast agent showing air embolism in the cerebral arteries

Electromechanical dissociation ensued, epinephrine was administered, and mechanical cardiopulmonary resuscitation initiated. Despite best efforts, resuscitation was unsuccessful.

Necropsy findings subsequently revealed a severely squeezed lower lobe of the right lung with multiple tears in branches of the pulmonary vessels, confirming the radiographic findings.

Discussion

This was a case of a fatal systemic air embolism due to severe blunt chest trauma.^{4,5} The patient presented with acute respiratory failure and hemoptysis. Hemoptysis in blunt chest trauma strongly suggests the existence of a communication channel between the pulmonary blood vessels and the airways but can be found in only 22% of patients with systemic air embolism after lung trauma.⁶ Approximately 0.5 to 3 mL of air entering one of the pulmonary veins and being transported to the coronary or cerebral circulation can be fatal. Systemic air embolism is a very rare and often unrecognized complication of blunt chest trauma with a mortality rate of 80%, whereas its occurrence in penetrating chest trauma is more common with fewer deaths (48%).⁶ Typically, refractory circulatory collapse occurs in patients with a traumatic alveolar to pulmonary venous fistula shortly after endotracheal intubation and with the onset of PPV. The resulting diagnostic and therapeutic dilemma in this case results from the fact that blunt chest trauma with hemoptysis is common, whereas the described complication is very rare. Furthermore, there is no

continuous monitoring device to detect arterial air embolism reliably, and finally, potentially beneficial therapeutic measures are both limited and/or complex.

In considering ventilation strategies for this type of clinical presentation, the pressure gradient between the airways and the pulmonary venous circulation should be maintained as low as possible. This can be achieved either by carefully titrating volume resuscitation in order to optimize filling pressures, or by further reducing tidal volumes, either by application of HFOV or by avoidance of PPV altogether.

An alternative therapeutic approach is to direct therapy towards “separation of the lungs and the air from the blood”^{7,8} by means of selective lung ventilation strategies using double-lumen tubes in patients with known unilateral lung lesions, emergency thoracotomy with hilar clamping, or even early use of cardiopulmonary bypass. However, it is known that thoracotomy in cases of blunt chest trauma may be associated with very poor outcomes.^{2,9,10} A third therapeutic option is to minimize secondary damages in the affected organs, e.g., by means of hyperbaric oxygen therapy.¹¹

The most important differential diagnoses in this case include venous air embolism combined with a persistent foramen ovale cordis, pulmonary arteriovenous fistulas, and anomalous origins of coronary arteries.¹² Undoubtedly, unsupported spontaneous ventilation was not an option in this patient. Retrospectively, one might suggest that the applied airway pressures should have been even lower. We did ventilate the patient's lungs with a lung protective strategy, including further minimization of the applied tidal volume using HFOV. Nevertheless, the resulting PaO₂ of 550 mmHg shortly after initiation of HFOV and a repeat computed tomography scan of the chest suggest that the extent of pulmonary recruitment may have been excessive. In this particular case, the effects of coronary and cerebral arterial air embolism became obvious one hour after starting PPV. It is plausible that successful recruitment of the injured lung (Fig. 3) did indeed restore pulmonary gas exchange, but it also may have contributed significantly to the fatal transfer of air into the pulmonary venous circulation via conduits of previously closed traumatic fistulae.

The patient in this case reached the hospital with severe hypoxemia requiring immediate respiratory assistance. Considering the initial blood gas analysis, one-lung ventilation was not initiated in this case, since the risk of hypoxemia was estimated to be too high, and the initial TEE assessment shortly after admission did not demonstrate air embolism. Using TEE, Saada *et al.* described modifications of ventilation patterns that could minimize air embolism.¹³ In our case, delayed air embolism ensued despite maintenance of low mean airway pressure and tidal volumes.

Fig. 3 Multislice computed tomography scan of the chest with contrast agent showing nearly complete recruitment of the right lung

Since minimal amounts of air in the systemic circulation have an enormous risk of causing death or significant disability, preventive measures might be considered, especially since polytraumatized patients are often young and healthy. Pumpless arteriovenous extracorporeal lung assist (pECLA) is used increasingly in trauma patients.^{14,15} The highly effective CO₂ elimination via pECLA allows separation of ventilation from oxygenation. The required mean airway pressure as the main determinant for oxygenation can be maintained with a respiratory pressure amplitude that is significantly reduced, especially if pECLA is combined with HFOV. Subsequently, the pressure gradient maintaining air entry into the pulmonary venous circulation is decreased.^{16,17} Full heparin coating of the artificial lung and the whole extracorporeal circuit eliminates the need for complete anticoagulation.¹⁸ In the very near future, a miniaturized pump-driven extracorporeal lung-assist system will be available that works with a single venous cannulation technique and can be inserted very quickly using local anesthesia.¹⁹

In conclusion, this case describes the diagnostic and therapeutic dilemma in patients with an alveolar to pulmonary venous fistula after blunt chest trauma: There is no adequate monitoring system to test continuously for the presence of systemic air embolism. Even if such a device were to exist, if the cerebral circulation is involved, the time lag between detection of air entry into the systemic circulation and initiation of a potentially successful therapy would be too long in order to prevent the patient from serious harm. Sound clinical judgment with ongoing evaluation and rapid and aggressive interventions are essential. Therefore, even if the complication described in this case report is very rare, it might be worth considering

extracorporeal lung assistance as a first-line therapeutic option in patients with severe chest trauma who reach the hospital breathing spontaneously and present in the emergency department with severe hemoptysis.²⁰ In any case, recruitment strategies in blunt chest trauma with hemoptysis bear the risk of systemic air embolism and should therefore be avoided.²¹

Competing interests None declared.

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