Tension pneumopericardium in a polytrauma patient

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Introduction. Tension pneumopericardium is a life-threatening condition, manifesting most commonly as hemodynamic instability caused by cardiac tamponade. Reduced cardiac output and blood pressure can lead to difficulties in the detection of arterial bleeding from associated injuries while the increased venous pressure can increase the rate of bleeding.
Case report. This is the case of a patient after a car accident, with bilateral serial fractures, bilateral pulmonary contusion, bilateral pneumothorax, emphysema of the neck and chest, pneumomediastinum and pneumopericardium, and other injuries. During treatment, the patient developed a gradually progressing hemodynamic instability, resulting in pulseless electrical activity. Further progression of the case is detailed in the paper.
Conclusions. Tension pneumopericardium is a rare complication of a high-energy blunt thoracic trauma that manifests through hemodynamic instability. Its treatment requires early diagnosis and immediate decompression of the pericardial cavity, which should, where possible, be performed even before putting the patient on mechanical ventilation as ventilation bears a high risk of worsening the pneumopericardium due to the increased air pressure in the lungs. During diagnosis and treatment of associated injuries, we must bear in mind that the hemodynamic changes caused by pneumopericardium can mask typical signs of such injuries.

Key words: pneumopericardium, trauma, mechanical ventilation, hemodynamic instability

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INTRODUCTION

Pneumopericardium is defined as the presence of air or other gas in the pericardial sac. This rare injury can arise as a result of trauma (blunt or penetrating thoracic injuries) or be of non-traumatic etiology (bronchial asthma, various types of fistulae, iatrogenic in mechanical ventilation[1-3]). A tension pneumopericardium is a life-threatening condition leading to the development of cardiac tamponade, insufficient ventricular filling, and influencing both arterial and venous pressure. Late diagnosis of this injury can lead to increased bleeding from venous plexuses, with potentially fatal consequences. In this paper, we describe the case of a patient bleeding into the peritoneum and retroperitoneum as a result of a car accident who developed a tension pneumopericardium after being put on mechanical ventilation.

CASE REPORT

On 29.6.2020 at 19:45, a 64-year-old man involved in a car accident (driver of a car hit by a truck, ISS 50, AIS thorax 5 – abdomen 4 – extremity 3) was brought to the A&E Department of the University Hospital Ostrava. Immediately after the accident, he lost consciousness temporarily. After regaining consciousness, he complained of dyspnea and chest pain. Once vital functions were under control, the patient was transported by air to the Trauma Center of the University Hospital Ostrava. On admission to the A&E, his blood pressure was 130/80, pulse 80/min, O_2 saturation 86%, breathing frequency 35/min. He showed signs of incipient respiratory distress and bilateral instability of the thoracic wall. Lab tests reported a hemoglobin level of 141 g/L, INR of 1.07, APTt of 32.7 s, lactate of 2.8 mmol/L, and base excess of -1.1 mmol/L. CT with a contrast agent revealed bilateral serial rib fractures (the 1^{st}–10^{th} rib on the right, of which 4^{th} - 9^{th} were double fractured, and 1^{st}–7^{th} rib on the left, of which 4^{th}–7^{th} were double fractured), bilateral pneumothorax and pulmonary contusion, subcutaneous emphysema of the

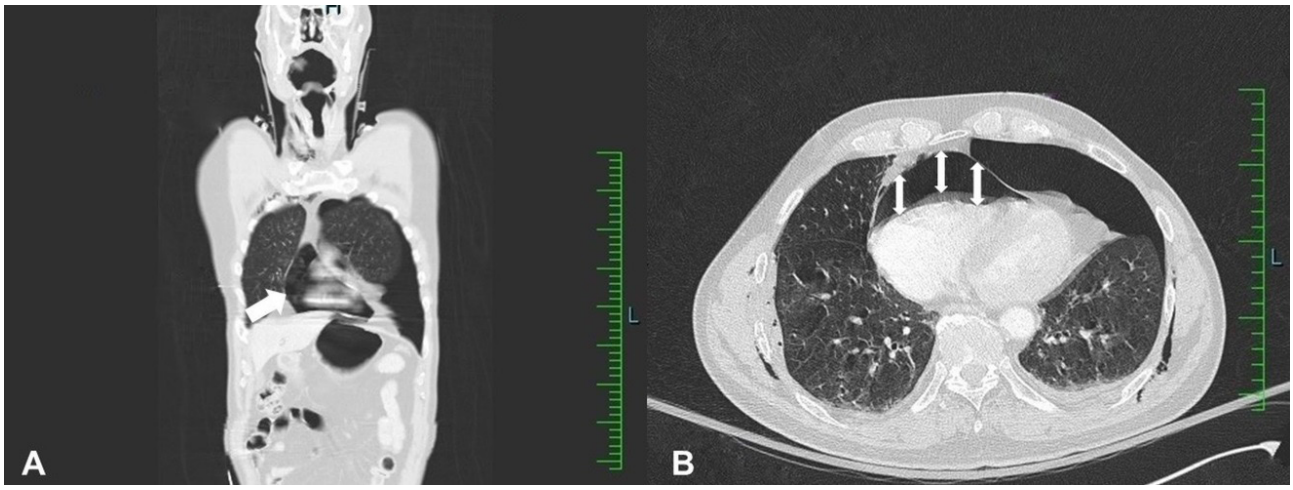

Fig. 1. CT scans showing air collection in the pericardial sac.

neck and chest, pneumomediastinum and pneumopericardium with the heart dorsally compressed (Fig. 1).

Other injuries included bleeding in the choroid plexus of the left lateral ventricle and post-contusion parietal brain changes, non-dislocated bilateral fractures of both branches of the pubic bone, and a left-sided fracture of the lateral mass of the sacrum without dislocation detectable on CT. No signs of hepatic injury, hemoperitoneum, or bleeding in the retroperitoneum were apparent.

After CT, the patient was sedated, intubated, and put on mechanical ventilation. A central venous catheter and bilateral chest drainage were inserted; the chest drainage produced 200 mL of sanguinolent fluid. Shortly after putting the patient on ventilator, the patient developed circulatory instability necessitating vasopressor support. Two red blood cell transfusions and fluid resuscitation (colloids – 1500 mL, crystalloids – 3000 mL) were administered at the A&E. Lab results showed a drop in hemoglobin levels to 62 g/L and an increase in lactate to 9 mmol/L. The circulatory instability progressed to pulseless cardiac electrical activity. With resuscitation ongoing, pericardial drainage was performed at A&E using the subxiphoid approach (Fig. 2). During the drainage, hemoperitoneum was detected. The patient was urgently transferred to the operating theatre where extended laparotomy was performed and 700 mL of hemoperitoneum from the vicinity of the liver was evacuated. This was combined with perihepatic packing due to two superficial 5 cm long fissures in segment VI. In view of the right-sided retroperitoneal hematoma, the retroperitoneum was also explored; no kidney injury was found. Further worsening of the patient's condition necessitated open cardiac massage, which was, however, unsuccessful due to insufficient filling of the heart compartments and the patient died at 22:27.

The subsequent autopsy confirmed the fractures of the chest and pelvis, contusion of both lungs, and the pericardial sac in the region of the right ventricle. Minor contusions were also found in the pericardial adipose tissue (inside the epicardium). The left lung and the liver in the region of the right hepatic lobe were fissured, which caused bleeding into both the peritoneal and thoracic cavities. The autopsy did not reveal any bleeding from major arteries in the peritoneal or thoracic cavities. Hypovolemic shock was established as the immediate cause of death, which was made worse by the development of the tension pneumopericardium. Toxicology examination did not detect any toxicologically significant substances.

DISCUSSION

Where blunt thoracic trauma is concerned, pneumopericardium is mostly accompanied by pneumothorax and pleuropericardial ruptures, through which the air from the pleural cavity penetrates into the pericardium. Another mechanism of developing pneumopericardium is an injury to the alveoli, from which the air can leak into peribronchial and perivascular adipose tissues and along vessels, into the pericardial sac[2]. This latter mechanism is known as the Macklin effect[4-6]. A direct apposition of tracheobronchial and pericardial fissures is another possible mechanism[6-9]. Literature also reports cases where the air can leak into the pericardial cavity through the venous subadventitia in the case of an open injury of veins of the throat. In such cases, the injury to the skin may not be apparent. Besides, pneumopericardium can also arise during venous cannulation and catheterization[10].

In penetrating trauma, pneumopericardium most commonly occurs as a result of open stab wounds. An injury to the pericardium (and heart) by a bone fragment during penetrating rib injuries directed into the thoracic cavity is another, although less common, mechanism of developing pneumopericardium[11]. If a lung and pleura adjacent to the pericardium are both injured, the air from the injured lung can leak into the pericardium.

The pneumopericardium may or may not affect the hemodynamics; the so-called simple pneumopericardium with no influence on hemodynamics usually resolves spontaneously within just a few days[12-14]. In some cases, however, pneumopericardium can rapidly progress and cause circulatory instability or even arrest. In such cases, the term the "tension pneumopericardium" is used.

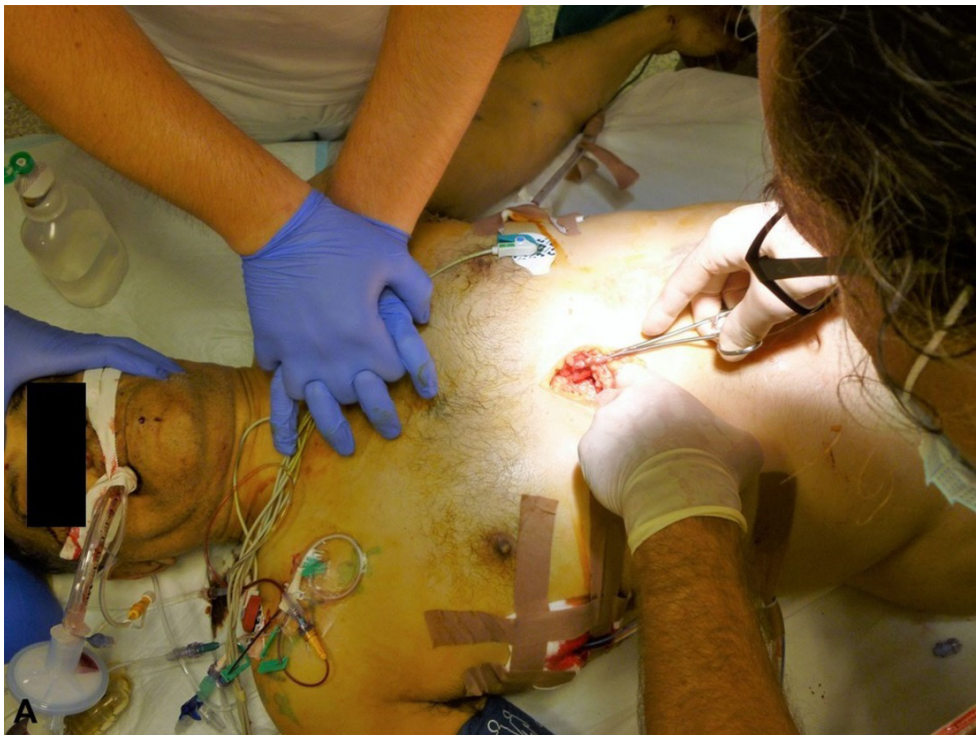

Fig. 2. Drainage of the pericardium via subxiphoid approach.

It most commonly develops in patients on mechanical ventilation[1,8]. Capizzi et al. described 32 cases of pneumopericardium after blunt thoracic injury, of which 12 patients developed tension pneumopericardium. A correlation between mechanical ventilation and cardiac tamponade was observed in their study – 83% of patients became unstable after positive pressure ventilation was initiated[8]. Although there are several hypotheses about the pathogenesis of pneumopericardium in patients on mechanical ventilation, the reasons and conditions leading to the development of the tension pneumopericardium and gaseous cardiac tamponade are not known with sufficient certainty[12]. According to the literature, most tension pneumopericardium cases result from the air leak from adjacent structures into the pericardial space, where it accumulates as the pericardium acts as a one-way valve[15].

Clinical symptoms of tension pneumopericardium include progressive respiratory distress, increased central venous pressure, hypotension, and muffled heart sounds. The diagnosis of pneumopericardium is established using imaging methods; thoracic CT examination is the method of choice as it is capable of displaying the presence of air in the pericardial cavity[1,7,16].

Our patient also developed tension pneumopericardium causing hemodynamic instability only after being put on mechanical ventilation. The increasing venous pressure accompanied by the increasing pressure in the pericardium led to increased bleeding from presacral venous plexuses, which are usually injured in sacral fractures. The pressure of the surrounding tissues was not sufficient for developing a tamponade and stoppage of bleeding from the injured veins. The increased venous pressure might have also increased the pressure in the liver parenchyma and accentuated bleeding from hepatic fissures that were only detected during the surgery as they were not apparent on the CT scans. The change in the hemodynamic parameters in the tension pneumopericardium also altered the distribution of the contrast agent during CT and, in effect, led to the unreliable diagnosis of the injuries to the liver and retroperitoneum.

In view of the above-stated facts, we propose that in the case of a patient with tension pneumopericardium, pericardiocentesis or percutaneous epicardial catheter insertion should be performed (if possible) before putting the patient on mechanical ventilation. These urgent procedures are, of course, only a temporary solution and it is subsequently necessary to insert soft drainage surgically through the subxiphoid approach, open thoracotomy, or thoracoscopy[8,17]. In our case, we opted for the subxiphoid approach, which was further extended to a revision of the peritoneal cavity. Unfortunately, this intervention came too late – only after medical ventilation significantly worsened the pneumopericardium.

CONCLUSIONS

Tension pneumopericardium is a rare, life-threatening condition that can develop as a result of a high-energy blunt thoracic trauma. It must be thought of in the differential diagnosis of patients with signs of hemodynamic instability, especially patients who are not improving despite treatment. Tension pneumopericardium is usually apparent on thoracic CT; if this finding is present in a polytrauma patient, it is necessary to remember the effect of pneumopericardium on the blood pressure in the arteries and veins, which can mask bleeding from concomitant injuries. Moreover, we propose that if possible, pericar-

diocentesis should ideally be performed in such patients before intubation and putting the patient on mechanical ventilation to mitigate the effects of pneumopericardium on hemodynamic instability.

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