

# Asymptomatic pneumopericardium after blunt chest trauma

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## ABSTRACT

A 49-year-old man was admitted to the emergency room after a fall from a height of 4 m. He was conscious with intact neurological status and stable vital function. Blunt chest trauma was presented with left side rib fractures (3-6), ipsilateral lung base contusion, pneumo-liquidothorax, pneumomediastinum and subcutaneous emphysema. A computed tomographic scan also confirmed pneumopericardium. Negative Hamman's sign were presented. Although the patient had no symptoms, the presence of pneumopericardium might be a sign of severe injury and indicates the potential risk of cardiac tamponade and a life-threatening condition. In the presence of pneumopericardium, cardiac tamponade could be triggered secondary by mechanical ventilation support. In our case report, we would like to stress the importance of early diagnostic of asymptomatic pneumopericardium after blunt chest trauma, which may be decisive in choosing the optimal therapeutic procedure.

*Key words: Pneumopericardium; Tension pneumopericardium; Blunt trauma; Pneumomediastinum*

## INTRODUCTION

Pneumopericardium indicates the presence of air in the pericardium. The most common cause of posttraumatic pneumopericardium is penetrating or blunt chest injury. It is a very rare complication following blunt chest trauma that may be present alone or in association with pneumothorax or pneumomediastinum. High-speed motor vehicle accidents (73%) and falls (17%) are the most frequent causes of blunt chest

injuries. (1) In most described events, initially pneumopericardium is nontension, and usually resolves itself spontaneously within a few days. Following some cases where tension pneumopericardium has been reported, the presence of pneumopericardium might be a sign of severe injury and may indicate a potential risk of cardiac tamponade. (2) In this case report we would like to highlight the importance of early diagnostic, surveillance in the intensive care unit (ICU) and the optimisation of respiratory support in patients with pneumopericardium after blunt chest trauma.

## CASE REPORT

A 49-year-old man was admitted to the emergency room five hours after a fall from a height of 4 m. He was fully conscious and oriented (Glasgow coma score 15) with intact neurological status. Intensive left side chest pain was presented along with shallow, mild breathing, with a respiratory rate of 17/min and oxygen saturation (SaO<sub>2</sub>) of 92%. He was haemodynamic, stable with slight tachycardia (heart rate 95/min) and a blood pressure reading of 130/90 mmHg. Auscultation had determined diminished breathing sounds on the left chest side. Heartbeats were clear and regular, no additional noises were presented during the cardiac action in the left lateral decubitus position (negative Hamman's sign). A chest X-ray showed pneumomediastinum, left side rib fractures (3-6) with pneumo-liquidothorax, ipsilateral lung base contusion and subcutaneous emphysema. A computed tomographic (CT) scan also confirmed pneumopericardium with a maximal width of 6 mm (figure 1). Troponin I in serum was less than 0.02 ng/ml and creatine

kinase-MB fraction (CK-MB) was 2.8 ng/ml. ECG and arterial gas analysis were normal. Patient was admitted to the intensive care unit (ICU) with continuous ECG, respiratory and blood pressure monitoring. Oesophageal and/or tracheo-bronchial injury were excluded by transesophageal echocardiogram and bronchoscopy. A chest tube was inserted on the left chest side. Local pleural analgesia (20 ml of 0.25 % levibupivacain was applied every six hours with occluded chest dren for 30 min) in addition to non-opioids analgesics were sufficient for satisfactory analgesia (VAS scale <3). Haemodynamic stability with sufficient spontaneous breathing, good oxygenation and normocapnia were maintained during the entire stay in the ICU. The air in pericardial sac was completely resolved after three days. The chest tube was removed after five days.

## DISCUSSION

Pneumopericardium is a rare complication of blunt chest injury and usually self-limiting under four days to two weeks. (3, 4) Intubation with positive pressure ventilation supports the re-entering of the air into the pericardial space particularly in presence of pneumothorax and disables its output from pericardial sack. Capizzi PJ analysed 32 patients with pneumopericardium after blunt chest trauma. Tension pneumopericardium developed in 12 (37%) patients, and out of them cardiac tamponade occurred in 11 (92%) patients immediately after intubation and start of mechanical ventilation. Zakyntinos E noted that pneumopericardium after blunt chest trauma may progress into tension pneumopericardium with cardiac tamponade after positive pressure ventilation independent

of intubation. (5) In these cases emergency cardiac decompression is indicated. Early diagnosis with non-invasive ventilation selection whenever possible may prevent the induction of unnecessary complications.

In our case, pneumopericardium was secondary to the breakthrough of air through the adjacent structure to the pericardium space after blunt chest trauma. Two united factors are usually responsible in the induction of its pathogenesis: free air vesicles (injured pulmonary interstitial tissue and/or ruptured alveoli, pneumomediastinum, pneumothorax, direct trachobronchal- or/and oesophago-pericardial communication) and pericardial tears (congenital, anatomical or caused by trauma) through which air begins to enter into the pericardial sac repressed by increased pressure or/and volume. In 1939, Macklin CC showed by histologic base that air could track along peribronchial and perivascular sheaths through pulmonary veins to the lung hilum into the pericardium because of discontinuous of pericardial tissue at the reflection of parietal onto visceral pleura

near the ostia of the pulmonary veins. (6) Both the intensity of the force that leads to injury and the extensiveness of the tissue damage are predictors in the induction of pneumopericardium.

Free air may be captured in the pericardium space by a one-way valve mechanism. Appearance of haemodynamic instability depends on the speed input of a certain volume of air. Maurer ER and Stacey S showed that the first clinical signs of pneumopericardium may occur by 60 ml of air if it penetrates rapidly into the pericardial sac, as opposed to 500 ml without haemodynamic changes if it accumulates slowly. (6, 7) In our patient, we supposed that induction of pneumopericardium was caused by air that was released after lung tissue injury and compressed to the pericardial sac by the pressure that arose as a result of the force of the fall. The low volume of accumulated air in the pericardium was the reason for the patient's haemodynamic stability.

Initial, prophylactic pericardial decompression, as the method of potential car-

diac air tamponade prevention, was suggested by some authors if asymptomatic pneumopericardium was presented after blunt chest trauma. (8) Following the clinical reports, Capizzi PJ and Roth TC showed that two-thirds of these patients did not form any serious complications and invasive prophylactic procedures were not justified. (9, 10)

## CONCLUSION

Pneumopericardium is a sign of severe trauma and a potentially life-threatening condition, but in asymptomatic form the primary importance is continuous monitoring and observation of cardio-respiratory function in the intensive care unit. Our case report supports the importance of its early diagnostic and conservative treatment after the exclusion of tracheobronchial tree and/or oesophagus injury.

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