

Cardiac tamponade due to pneumopericardium

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ABSTRACT Pneumopericardium is rare in acute asthma and cardiac tamponade has not been reported. The case is reported of a 20 year old asthmatic patient in whom assisted ventilation and high airway pressures resulted in tension pneumopericardium with clinical signs of cardiac tamponade that were relieved by pericardial aspiration.

Cardiac tamponade is usually caused by the accumulation of blood or other fluid within the pericardial sac. Much rarer is the association of pneumopericardium with cardiac tamponade. We report cardiac tamponade resulting from pneumopericardium in a patient receiving assisted ventilation for severe asthma.

Case report

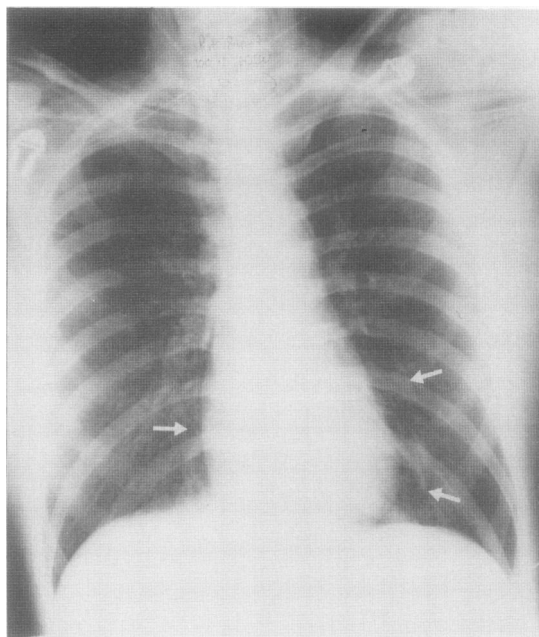
A 20 year old asthmatic woman presented with a week long history of worsening asthma. On admission she was tachypnoeic with severe central cyanosis. Her heart rate was 120/min and blood pressure 130/80 mm Hg with appreciable pulsus paradoxus. Her chest was hyperexpanded, with widespread inspiratory and expiratory wheeze. Chest radiography showed no evidence of pneumothorax. While she was breathing high concentration oxygen her arterial blood gas measurements were: pH 6.8, P_{O_2} 8.8 kPa, P_{CO_2} 22.3 kPa.

Emergency assisted positive pressure ventilation was started. Continuous nebulised salbutamol was combined with intravenous hydrocortisone and an infusion of aminophylline and salbutamol. High ventilation airway pressures (peak airway pressure 52 cm H_2O , mean airway pressure 22 cm H_2O) continued despite ventilation with 6-8% ether and intravenous ketamine. The mechanical ventilator was unable to overcome the high airway pressures and hand ventilation with an Ambubag was required intermittently.

After eight hours cervical subcutaneous emphysema became apparent and this spread to affect most of the chest wall. Chest radiography showed no pneumothorax or mediastinal air.

After 18 hours' ventilation the patient's condition deteriorated suddenly. Her heart rate increased from 90 to 120 beats/min and her blood pressure fell from 120/80 to 50 (systolic) mm Hg. Central venous pressure rose from +15 to +40 cm H_2O . The clinical picture was compatible with cardiac tamponade and chest radiography showed a large pneumopericardium (figure).

A 16 gauge teflon central venous cannula was introduced into the pericardial sac via the subxiphisternal route by an aseptic technique. One litre of air was aspirated and the cannula connected to underwater seal drainage. A rapid response was observed, with restoration of heart rate and blood pressure to their former levels. Two hours later the patient developed a left tension pneumothorax and a chest drain was inserted via the left axilla. Shortly afterwards a right tension pneumothorax appeared, and this too was drained successfully.



Chest radiograph showing a large tension pneumopericardium (pericardial sac arrowed).

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Continuing positive pressure ventilation produced a substantial escape of inspired volume from both chest drains. The patient became deeply cyanosed, with a slow idioventricular cardiac rhythm. Further resuscitation attempts were unsuccessful and the patient died 22 hours after admission. Necropsy was not performed.

Discussion

We are unaware of any previous report of cardiac tamponade caused by pneumopericardium in acute asthma. Assisted ventilation with high airway pressures undoubtedly contributed to its development in the present case. Positive pressure ventilation in neonates may cause pneumopericardium and in these circumstances it is a recognised cause of cardiac tamponade.¹ In acute asthma, pneumopericardium, which occurs rarely, was first reported by Toledo *et al.*² Pneumomediastinum occurs more frequently but usually resolves spontaneously, rarely requiring any specific treatment.

In tension pneumopericardium it is postulated that increased alveolar pressure causes dissection of air along

perivascular and peribronchial sheaths to the hilum of the lung, where it is able to enter the pericardial sac.³ Cardiac tamponade with pneumopericardium is clearly rare, but its presence should be borne in mind in any patient having positive pressure ventilation so that prompt treatment can be given.

We thank Dr K M Shaw for permission to report this unusual case.

References

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- 2 Toledo TM, Moore WL, Nash DA, North RL. Spontaneous pneumopericardium in acute asthma. *Chest* 1972;**62**:118–20.
- 3 Macklin CC. Transport of air along sheaths of pulmonic blood vessels from alveoli to mediastinum. *Arch Intern Med* 1939;**64**:913–26.