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CASE REPORT

Tension pneumopericardium caused by positive pressure ventilation complicating anaerobic pneumonia

C.P. Bleeker-Rovers[1*], F.J.J. van den Elshout[2], T.I.F.M. Bloemen[3], H.A.H. Kaasjager[4]

[1]Department of Internal Medicine 541, University Medical Centre St Radboud, PO Box 9101, 6500 HB Nijmegen, the Netherlands, tel.: +31 (0)24-361 47 63, fax: +31 (0)24-354 17 34, e-mail: C.Bleeker-Rovers@nucmed.umcn.nl, Departments of [2]Pulmonary Disease, [3]Surgery and [4]Internal Medicine, Rijnstate Hospital, PO Box 9555, 6800 TA Arnhem, the Netherlands, * corresponding author

ABSTRACT

A 22-year-old man was admitted with pneumonia. He was immediately intubated and positive pressure ventilation was initiated. Blood and sputum cultures showed *Bacteroides fragilis* and *Corynebacterium* sp., which were treated with metronidazole and clindamycin. Three weeks later his blood pressure suddenly dropped with an elevation of the central venous pressure. Chest X-ray revealed a pneumopericardium. A parasternal mediastinotomy with partial pericardiectomy was immediately performed. On opening the pericardium his blood pressure normalised. The patient gradually recovered and six weeks after admission he was extubated. Two weeks later he was discharged. A pneumopericardium without previous thorax trauma is very rare and early recognition is imperative because a tension pneumopericardium with cardiac tamponade may develop, as happened in this case. A tension pneumopericardium has to be treated with immediate pericardiocentesis followed by partial pericardiectomy to avoid recurrence.

INTRODUCTION

A pneumopericardium is an uncommon disorder that is most often caused by blunt or penetrating chest trauma or iatrogenic trauma related to pericardiocentesis, cardiac surgery or positive pressure ventilation. Spontaneous pneumopericardium is very rare and can be caused by direct extension of infectious or neoplastic processes of the lungs or by pericardial infection with gas-forming bacteria. Anaerobic bacteria are relatively common pulmonary pathogens, most often causing infection after aspiration or in periodontal disease. Lung abscesses and empyema are well-known complications of anaerobic pneumonia, but pneumopericardium is extremely rare in these patients. We describe a patient with a tension pneumopericardium during positive pressure ventilation, complicating pneumonia caused by *Bacteroides fragilis* and *Corynebacterium* sp.

CASE REPORT

Patient A, a 22-year-old man, had been ill for three days with a fever of 40°C, unproductive cough and progressive shortness of breath. He was admitted to another hospital with severe dyspnoea and cyanosis. His previous medical history was remarkable due to a spontaneous right-sided pneumothorax one year before, which was treated conservatively. A tall, thin, very ill young man in respiratory distress was seen with a temperature of 40.2°C, a blood pressure of 82/56 mmHg, a pulse of 106 beats/min and an oxygen saturation of 46%, breathing 10 litres oxygen/min. Heart sounds were normal and bibasilar pulmonary rales were heard. The physical examination was also notable for many carious teeth and gingivitis. A chest X-ray revealed bilateral pulmonary infiltrates in the lower lobes. The patient was immediately intubated and mechanical ventilation was initiated. Initially his pneumonia was treated with amoxicillin-clavulanic acid and gentamycin. One sputum culture and two blood cultures taken in the first two days showed *Bacteroides fragilis* and *Corynebacterium* sp.

Antibiotic therapy was then changed to clindamycin and metronidazole. His pneumonia was complicated by a left-sided pneumothorax on the second day after admission and a right-sided pneumothorax on the fifth day, which were both adequately drained percutaneously. On the seventh day pleural fluid culture still showed *Bacteroides fragilis*. Because of increasing problems with mechanical ventilation caused by the adult respiratory distress syndrome (ARDS) on the sixth day, he was transferred to our hospital. He was ventilated in the prone position for 48 hours and needed high ventilation pressures. Both lungs were ventilated separately through a double-lumen tube for three days.

Three weeks after admission, he was slowly recovering and being ventilated with decreasing pressures. Suddenly his blood pressure dropped, his heart rate and central venous pressure increased, and his tidal volumes decreased. On physical examination heart sounds were barely audible and bibasilar rales were heard. Chest X-ray revealed a recurrent partial right-sided pneumothorax and a large amount of gas surrounding the heart, compatible with a pneumopericardium (*figure 1a*). A parasternal mediastinotomy was performed immediately. When the pericardium was opened, a large amount of gas escaped after which his blood pressure, heart rate and tidal volumes normalised almost immediately. A partial pericardiectomy was performed and a new drain was placed in the right hemithorax. A postoperative chest X-ray showed that the pneumopericardium had disappeared (*figure 1b*). After this episode the patient gradually recovered, showing no signs of recurrent pneumopericardium. Six weeks after admission he was extubated, his thorax drains were removed and he was discharged two weeks later. His cellular and humoral immunity proved to be normal and he tested negative for HIV. His carious teeth most probably caused his initial anaerobic pneumonia and he was urgently advised to get dental treatment.

DISCUSSION

A pneumopericardium is a rare disorder most often caused by an abnormal connection between the pericardium and a nearby air-containing structure. In our patient two possible causes for the pneumopericardium were present. In the first place direct extension of the infection with *Bacteroides*, a gas-forming micro-organism, from the pleural space to the pericardium could have occurred, although no fluid or pus was found in the pericardium. Secondly the patient had been ventilated with high positive airway pressures

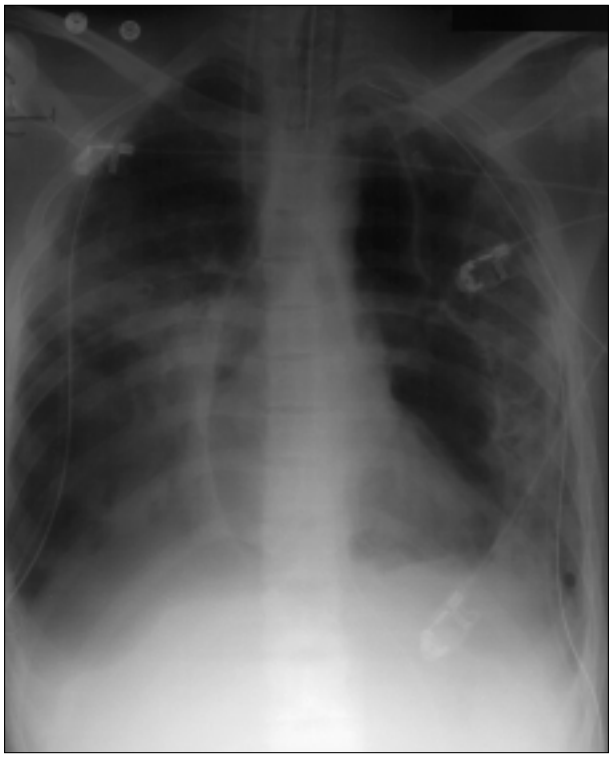

Figure 1a
Chest X-ray showing a partial right-sided pneumothorax, thorax drains on both sides and a large radiolucent rim around the heart and large blood vessels: a pneumopericardium

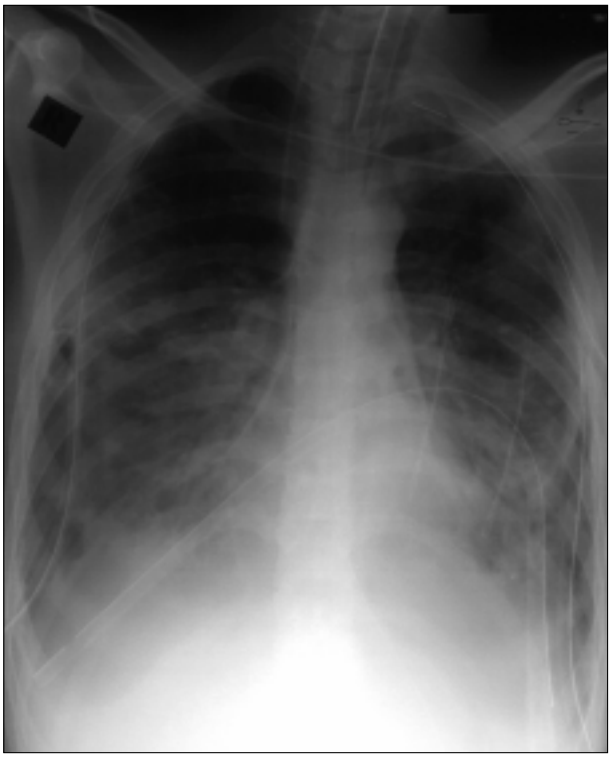

Figure 1b
Chest X-ray after pericardiocentesis, partial pericardiectomy and insertion of a new thorax drain on the right side: the pneumopericardium has completely disappeared

for several weeks, but these pressures were considerably diminished when the pneumopericardium appeared. We speculate that in this case the pneumopericardium was most probably caused by long-term positive pressure ventilation, because there were no signs of infection in the pericardium at the time of pericardiectomy. Also, the apparently adequate antibiotic treatment leading to the gradual recovery made infection less likely as the major cause of this complication. The extensive infection of the lungs and pleural space with *Bacteroides fragilis* and *Corynebacterium* sp. could, of course, have been a contributing factor. Patients with a pneumopericardium caused by *Bacteroides fragilis*, massive *Aspergillosis, Staphylococci, Klebsiella* and *Escherichia coli* have been described before.[1-5] Barotrauma after mechanical ventilation, especially when large tidal volumes or high end-expiratory pressures were used, has been described as a cause of pneumopericardium, especially in neonates.[3]

Clinically, pneumopericardium typically presents with dyspnoea and precordial chest pain.[3] On physical examination heart sounds are usually 'distant' and precordial tympany may be elicited.[1,3] The cardiac examination classically shows a typical auscultatory finding called Hamman's sign. This is a loud gurgling or splashing metallic sound in the precordial area, synchronous with the heartbeat. This sound does not disappear when breathing is stopped and is often heard by the patient. Hamman's sign is not only noticed in pneumopericardium, but also in some cases of pneumomediastinum and very rarely in left-sided pneumothorax.[6] On the chest X-ray pneumopericardium appears as a continuous radiolucent rim of air around the heart and the large blood vessels, and is outlined by a fine line representing the pericardial sac. The air surrounding the heart gives an appearance referred to as the 'halo' sign.[3] Radiologically a pneumopericardium can be reliably distinguished from the more common pneumomediastinum. Pneumomediastinum usually manifests as multiple streaks of air that do not completely surround the heart, and which usually extend into the superior mediastinum and neck while pneumopericardium virtually always consists of a single continuous band of air extending from the hemidiaphragms to the ascending aorta and pulmonary arteries.[6] Air in the pericardial space shifts to the non-dependent side, whereas air in the mediastinum stays fixed.[7] It has been suggested that a decreasing cardiac size on serial chest X-rays, in the presence of a pneumopericardium, strongly supports the diagnosis of tension pneumopericardium.[8] Electrocardiography shows low voltages in patients with pneumopericardium.[3]

CONCLUSION

A tension pneumopericardium is rarely seen, usually occurs after blunt chest trauma or in positive pressure ventilation, and has a very high mortality rate without early recognition and acute intervention.[9,10] Tension pneumopericardium clinically presents with cardiac tamponade, leading to decreased cardiac output, hypotension, increased central venous pressure, tachycardia and pulsus paradoxus, as was seen in our patient. Immediate percutaneous or surgical pericardiocentesis to relieve the tamponade is essential and is usually lifesaving.[1,3] In most cases a (partial) pericardiectomy is necessary to avoid recurrence and prevent pericardial constriction from occurring later.[1] In the absence of tamponade, pneumopericardium can probably be safely observed while treating the patient's primary condition.[11]

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