A rare complication of a pneumococcal pneumonia

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In this case report we describe the development of a pyopneumopericarditis secondary to pneumonia, a complication that is rarely seen nowadays. (Neth Heart J 2009;17:393-5.)

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A 44-year-old man was admitted to our hospital with dyspnoea, night sweats, loss of weight and a cough of two weeks' duration. He had a possible fever and noticed chest pain when breathing. A few weeks earlier his general practitioner had started a course of amoxicillin/clavulanic acid because of suspected pneumonia. The patient did not respond well and developed progressive dyspnoea.

His medical history revealed cocaine and heroin abuse, for which he was taking methadone: his other medication was mirtazapine. He had never used intravenous drugs. He smoked cigarettes, but did not drink alcohol. His last visit abroad was two years ago, when he visited his native country of Morocco.

On physical examination the patient appeared ill. The body temperature was 36.8°C, with a pulse of 79 beats/min, and a blood pressure of 129/90 mmHg. Examination of the mouth showed an oral candidiasis. Auscultation of the heart revealed normal heart sounds and no murmurs. There was no pulsus paradoxus. Inspiratory crackles were found on both sides of the

lungs. The remainder of the physical examination showed no abnormalities.

The erythrocyte sedimentation rate was 103 mm/h, the haematocrit 38%, the white cell count 11.6×10^9 and the C reactive protein was 295 mg/l. The anti-HIV ELISA was negative. Blood cultures had negative results. Sputum culture showed β -lactamase positive *Haemophilus influenzae*, and the Ziehl-Nielsen stain was negative. The ECG showed a sinus rhythm with ST elevation of 2 mm in almost all leads, with no ST depressions or microvoltages.

Chest radiography showed cardiomegaly and an air/fluid interface in the superior mediastinum, 1 cm beneath the aortic arch. No pulmonary pathology was seen (figures 1A and B).

A computed tomography scan of the thorax showed a large amount of pericardial effusion reaching up to the aortic arch. Within the effusion there was a clear air/fluid interface as well as multiple air bubbles (figure 2).

A transthoracic echocardiogram showed 3.5 cm pericardial effusion with high echodensity behind the left ventricle and apex, and 3 cm behind the right ventricle (figure 3). Furthermore, echocardiography showed right atrial systolic collapse, high respiratory variation in diastolic filling of the left ventricle, and plethora of the inferior vena cava, all physiological changes associated with cardiac tamponade. Pericardiocentesis showed a purulent white fluid (300 cc), which tested positive on the Binax pneumococcal antigen detection test. Cultures and a Ziehl-Nielsen stain were negative.

Discussion

Since the introduction of antibiotics, pyopneumopericarditis is a rarely seen complication of pneumococcal infection. In a 14-year period (1960-1974) the Massachusetts General Hospital found 26 cases of purulent pericarditis, with two cases due to *Streptococcus pneumoniae*.¹ Between 6 and 7.5% of purulent pericarditis is due to a bacterial infection, most commonly *S. aureus*. Nowadays, this clinical problem is mostly seen in immune-compromised patients, after oesophageal interventions, thoracic surgery or thoracic

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Figure 1. Cardio-megaly and a clear air/fluid interface in the superior mediastinum.

Figure 2. Chest radiography showing a large amount of pericardial effusion.

Figure 3. Transthoracic echocardiogram showing 3.5 cm pericardial effusion with high echodensity

trauma.² Spreading mechanisms consist of direct spread from infected tissue into the pericardial space, or indirect haematogenous spread.³

Pneumococcal pericarditis is a serious illness with a high mortality of up to 30%. Cardiac tamponade is usually the cause of death, mainly because of delayed or missed diagnosis.⁴

The diagnosis is made based on purulent pericardial fluid or positive cultures after pericardiocentesis.⁴

We did not find any previous cases in the literature in which the *Streptococcus pneumoniae* antigen test was used for pericardial effusion. This test is a rapid *in vitro* immunochromatographic assay for the detection of *S. pneumoniae* antigen in the urine of patients with pneumonia and in the cerebral spinal fluid (CSF) of patients with meningitis. The sensitivity of the test for urine is 86 and 97% for CSF, with specificities of 94

and 99%, respectively.⁵ We believe that, because the concentration of antigen after lysis of the *Streptococcus pneumoniae* in the pericardial effusion has to be higher than in urine, the sensitivity and specificity of this test is increased in pericardial effusion.

Surgical drainage of the pericardium in conjunction with systemic antibiotic administration results in the best therapeutic results.⁶ Early surgical drainage could prevent the complication of constrictive pericarditis.⁴

We believe, especially because of the positive pneumococcal antigen test, that our patient was suffering from pyopneumopericarditis, secondary to a pneumococcal pneumonia. Based on the results of the sputum culture, *H. influenzae* pericarditis would be the differential diagnosis. *H. influenzae* is not uncommon in the paediatric population, but can also be found in adults.⁷

After pericardiocentesis intravenous treatment was started with antibiotics (first penicillin and, after the positive sputum culture, amoxicillin/clavulanic acid). Over the next few days the patient developed signs of haemodynamic compromise for which he was transferred to a thoracic surgery centre. First, a pericardial window was created by a subxiphoid approach followed by inserting a drain for adequate drainage. At a later stage, pericardiectomy was necessary because of the development of constrictive pericarditis.

In summary, although quite rare nowadays, the diagnosis of pyopneumopericarditis should be considered if a respiratory tract infection is complicated by haemodynamic instability. We would recommend using the *Streptococcus pneumoniae* antigen test to confirm the aetiology. ■

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