Thoracic Actinomycosis with Extension of the Infection to the Pericardium and Chest Wall

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Purulent pericarditis is a rare entity in the antibiotic era. It is usually a complication of other thoracic infections, mainly pneumonia. We describe here a case of purulent pericarditis secondary to thoracic actinomycosis that first presented as cutaneous thoracic abscesses and pneumonia. The infection caused constrictive pericarditis and pericardiectomy was performed. After a prolonged course of antibiotic treatment the patient regained full functional capacity. Clinicians should be aware of the ability of infections caused by *Actinomyces israelii* to progress across tissue boundaries.

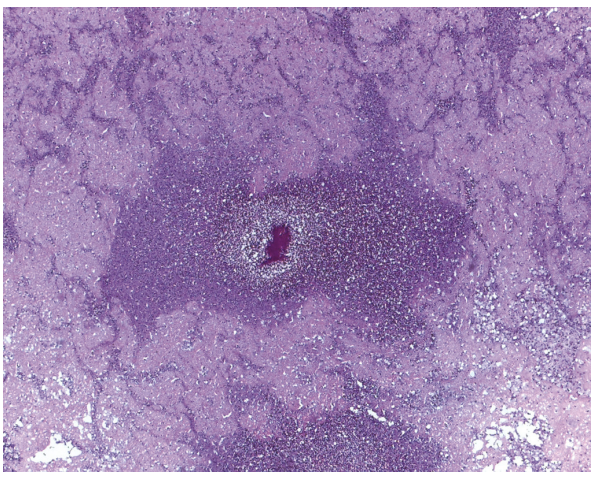
Patient Description

A 16 year old mentally retarded boy suffered from cutaneous abscesses on his right chest and upper abdomen for a month. Local drainage was performed and antibiotics were given. He gradually developed dyspnea, cough, abdominal distension and foot-swelling. He presented to the emergency room, where examination revealed him to be normotensive, tachypneic and tacyhcardic. His temperature was 38˚C and room air oxygen saturation was 88%. Fine crackles in the right lower lobe lung were noted. His heart sounds were normal and he had mild peripheral edema. Very poor oral hygiene was noted. His electrocadiogram demonstrated sinus tachycardia, and chest X-ray showed a right lower lobe infiltrate. White blood cell count was elevated at 18.7/mm^3 with 67% neutrophils, hemoglobin was 9 g/L and platelet count 690,000/mm^3. Alanine aminotransferase and aspartate aminotransferase levels were three times higher than normal.

He was diagnosed with right lower lobe pneumonia and was admitted to hospital for antibiotic treatment with cefuroxime and azythromycin. Five days following admission he developed worsening dyspnea, abdominal distension and foot swelling. On examination he was hypotensive, tachypneic and dyspneic; the jugular venous pressure was elevated and he had anasarca. Laboratory results revealed worsening liver function tests, and albumin level was 32 g/L. Echocardiography demonstrated a constricted heart, poor right and left ventricular function and turbid pericardial fluid. Chest and abdominal computed tomography scans demonstrated infiltrate in the right lower lobe, a thickened pericardium, ascites and an enlarged liver with four cysts consistent with echinococcal cysts. No connection between the cysts and the pericardium was noted. Serological tests for *Echinococcus* were positive. Blood and peritoneal fluid cultures were negative.

The patient was treated with imipenem, albendazole and fluids. Despite treatment anasarca worsened. Repeated echocardiography showed progressive constriction. A culture from his thoracic abscess grew *Actinobacillus actinomycetemcomitans*. Pericardiectomy was performed, which demonstrated a thick constrictive pericardium without pericardial fluid or calcifications. The postoperative course was complicated by severe myocardial dysfunction with severe hypotension and acute renal failure. Treatment with ionotropes and peritoneal dialysis was instituted for 2 weeks. Pericardial pathology demonstrated a fibropurulent infiltrate and severe pericardial fibrosis without granulomas. Sulfur granules were seen [Figure] and a Gram stain revealed Gram-negative bacilli. Cultures of the pericardium were negative, but tissue polymerase chain reaction demonstrated DNA sequence of *Fusobacterium nucleatum* (98% homology). Echocardiography prior to discharge demonstrated normal cardiac function, but a mass 3 x 2 cm was noted in the left ventricle; it did not have the consistency of a thrombus. Reexamination of previous echocardiograms did not show the mass.

We suspected the mass to be part of the infection and continued antibiotic therapy for 8 weeks. The patient improved and regained full functional capacity after 2 months of antibiotic



Tissue biopsy from the pericardium demonstrating fibropurulent effusion with sulfur granule appearance (Hematoxylin and eosin stain).

therapy. Follow-up echocardiography tests demonstrated normal heart function and a gradual decrease in the size of the left ventricular mass until its complete disappearance.

Comment

Purulent pericarditis is a rare disease in the antibiotic era. It is typically an acute illness but may have an indolent course. Pathogens can reach the pericardium by direct spread from an intrathoracic or myocardial focus of infection, by hematogenous spread, as a complication of trauma or surgery, or by extension from a sub-diaphragmatic suppurative focus. Gram-positive bacteria are responsible for 40–45% of cases, among them *Staphylococcus aureus* which accounts for 22–31% [1]. Pure anaerobic and polymicrobial infections are rare. There was only one report of *Fusobacterium nucleatum* pericarditis in the literature [2].

Actinobacillus actinomycetemcomitans was first isolated in 1912 from skin lesions associated with *Actinomyces israelii*. It can mimic most of the clinical syndromes caused by *Actinomyces israelii* and can cause endocarditis. There are two reports of pericarditis caused by this bacterium [3,4]. *Actinomyces israelli* infection is a slow, usually polymicrobial infection. It can cross tissue planes, cause fistulas as well as fibrosis of the involved tissues. A sulfur granule appearance in pathologic specimen is the most typical finding of this infection. Echinococcal infection can involve the pericardium, rarely, and cause constrictive pericarditis by disseminating from the lung pleura or the liver [5].

Our patient had an indolent course of purulent pericarditis caused by unusual bacteria. *Actinomyces israelii* was not isolated directly from the pericardium, probably due to prior antibiotic therapy, but the classic sulfur granule appearance was demonstrated on pathological examination. The presentation and clinical course is compatible with *Actinomyces israelii* thoracic infection combined with *Actinobacillus actinomycetemcomitans*. We assume that the cutaneous abscesses fistulized from the pyogenic pericardial process and were the first presentation of it. Although different bacteria were recovered from the abscesses and the pericardium, both *Actinobacillus actinomycetemcomitans* and *Fusobacterium nucleatum* are part of the normal oral flora and are frequently part of the same infectious process probably due to poor oral hygiene. We believe they had spread through aspiration from the patient's oral cavity to the lungs and from there to the pericardium and skin. The left ventricular mass that developed after pericardiectomy was probably part of the infection and responded to antibiotic therapy. Although the hepatic hydatic cysts seemed initially to be connected to the disease, they were probably a coincidental, and confusing, finding.

The mental retardation of the patient likely contributed to the late diagnosis due to difficulties in verbal communication.

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