Case reports

An uncommon cause of pericardial actinomycosis

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Cardiac actinomycosis is rare; the pericardium is the most frequently involved site, but myocardial, endocardial and valvular involvement have all been documented.

Most cases originate from a thoracopulmonary site of actinomycosis and spread directly to the pericardium. Widespread dissemination from extrathoracic organs is uncommon; in fact actinomycosis is prevented by anatomical barriers and hematogenous diffusion is rare.

We describe an uncommon case of pericardial actinomycosis due to a draining fistula from the liver to the pericardial space across the diaphragm. The massive dissemination through the fistula could explain the peculiar echocardiographic images of macroscopic, echo-reflective, irregular masses, floating in the pericardial space, probably consistent with aggregates of sulfur granules.

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Human actinomycosis is a chronic-suppurative disease caused by any of the five species of the genus of Actinomyces[1]. Cardiac involvement is rare and the pericardium is the most frequently involved site[2]. Dissemination from extrathoracic organs is rare because of the anatomical barriers that surround the thorax and even hematogenous spread is rarely reported[3].

We describe an uncommon case of pericardial actinomycosis due to a draining fistula from the liver to the pericardial space across the diaphragm.

Case report

A 64-year-old man presented with a history of abdominal pain, anorexia, dyspepsia, weight loss, weakness and cachexia over a period of 2 months. The patient's past surgical history included a Wirsung jejunostomy for acute pancreatitis, 2 years before.

On admission to the hospital, the patient's blood pressure and heart rate were normal. Examination of the head and neck revealed no abnormality; the external jugular vein was not distended; no adenopathy was present. Abdominal examination revealed prominent epatomegaly and the spleen tip was also palpable. Abnormal laboratory results included microcytic anemia (hemoglobin concentration 11.1 g/dl), and a white blood cell count of 18 700/mm^3 (70% neutrophils, 12% lymphocytes, and 5% monocytes). Chest radiograph (Fig. 1) and ECG were normal. Echography and computerized tomography of the liver evidenced multiple focal lesions; a needle biopsy of the largest lesion (measured 9 × 8 cm) was performed (Fig. 2), and histologic examination showed microabscesses containing sulfur granules composed of Gram-positive, thin, filamentous branching rods with peripheral clubbing, consistent with the diagnosis of actinomycosis (Fig. 3). He received aggressive medical treatment with parenteral penicillin G.

A few days later, the patient suddenly became agitated, dyspnoic and orthopnoic; his oral temperature was 37.2°C, his pulse rate 105 b/min and arrhythmic, his respiratory rate was 32 breaths/min, and his blood pressure was 80/50 mmHg; jugular venous distension was noted, while no pulsus paradoxus was observed. Cardiac examination revealed a decreased intensity of both the first and the second heart sound but no murmur, gallop or rub. He had soft expiratory wheezing at both pulmonary bases. In the

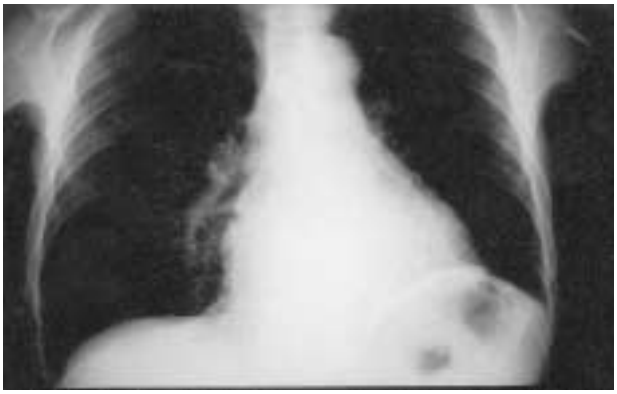

Figure 1. *Chest radiograph demonstrating normal cardiac silhouette (on admission to the hospital).*

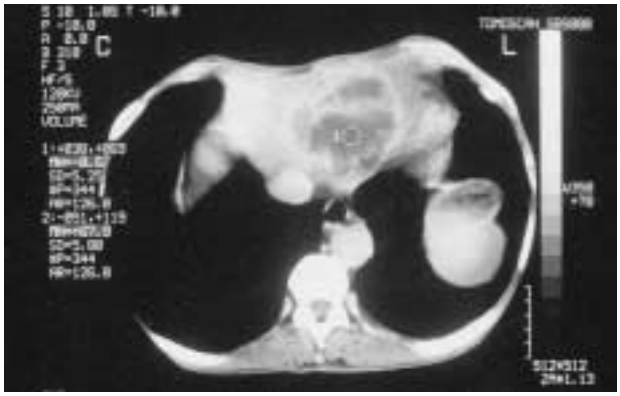

Figure 2. *Computerized tomography demonstrating the largest lesion in the liver (9 × 8 cm).*

intensive care unit, a Swan-Ganz catheter was inserted for bedside hemodynamic monitoring; right heart catheterization showed equalization and elevated right atrial and mid-diastolic right ventricular pressures. The chest radiogram revealed an enlargement of the cardiac silhouette (Fig. 4). A transthoracic echocardiogram showed evident pericardial effusion with right atrial and ventricular diastolic collapse; significant accumulation of echo-reflective, filamentous or roundish masses free and floating in the pericardial space was evident in all echocardiographic views (Figs. 5 and 6). Pericardiocentesis was performed and 1 liter of purulent material was withdrawn (Fig. 6). The results of cultures of the pericardial fluid were negative, while the histologic examination showed microabscesses containing sulfur granules, in which Actinomyces aggregates were identified (Fig. 7). The suspected pericardial dissemination of purulent material from the liver was confirmed by a laparotomy which revealed a draining fistula from the liver to the pericardial space across the diaphragm. Despite intensive treatment with parenteral penicillin G, he died a few days later in septic shock.

Discussion

Pulmonary actinomycosis accounts for between 15 and 45% of all cases of actinomycosis[4,5], and is second in frequency only to cervico-facial actinomycosis[6]. Cardiac involvement occurs in less than 2% of cases[2]. Pericardial disease is the most common, but myocardial, endocardial and valvular involvement have all been reported[2,3]. Actinomyces usually reach the pericardial space by spreading directly from an adjacent thoracic focus of actinomycosis. This stepwise invasion may account for the greater frequency of pericardial involvement. In a review of 19 cases of actinomycosis of the pericardium, Fife et al.[7] reported a primary thoracic focus of infection in 15 subjects, while 4 had no identifiable contiguous site of infection.

Actinomyces are part of the normal flora in the oral cavity. The major risk factors in the development of an intrathoracic focus of infection is aspiration of oropharyngeal contents; an immunocompromised status, associated bacteria, dental or periodontal disease, and alcohol abuse may play a significant role. In any case, pericardial actinomycosis generally occurs as a result of contiguous spreading from a pulmonary focus. Radi-

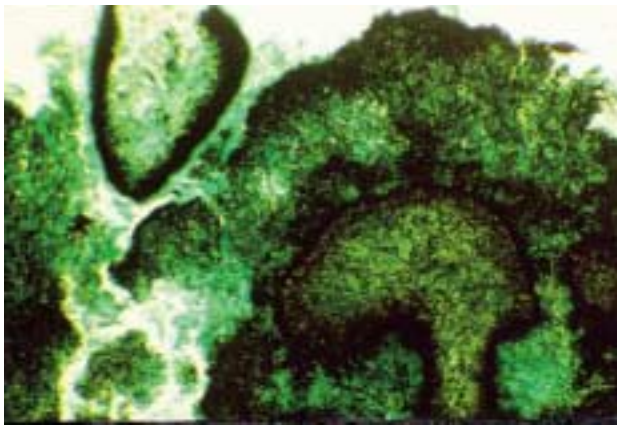

Figure 3. *Photomicrograph. Low power view of a liver microabscess showing sulfur granules (special stain: Grocott's hexamine-silver).*

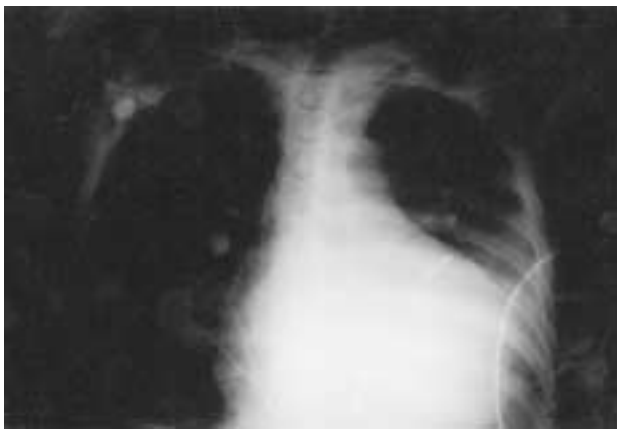

Figure 4. *Chest radiogram showing enlargement of the cardiac silhouette (a few days after admission to the hospital).*

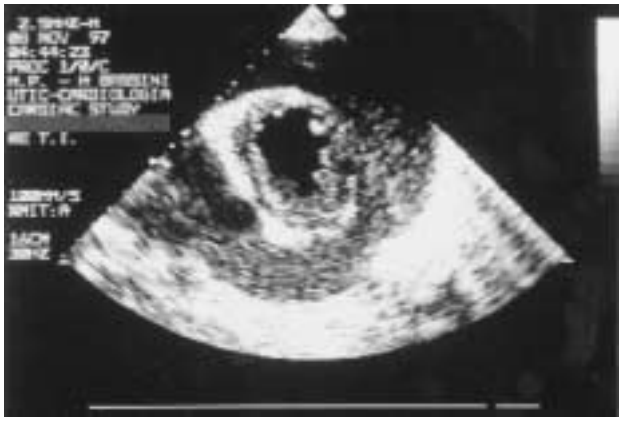

Figure 5. *Transthoracic echocardiogram (parasternal short-axis view) showing pericardial effusion with right atrial and ventricular diastolic collapse, and significant accumulation of echo-reflective, irregular, filamentous or roundish masses free and floating in the pericardial space.*

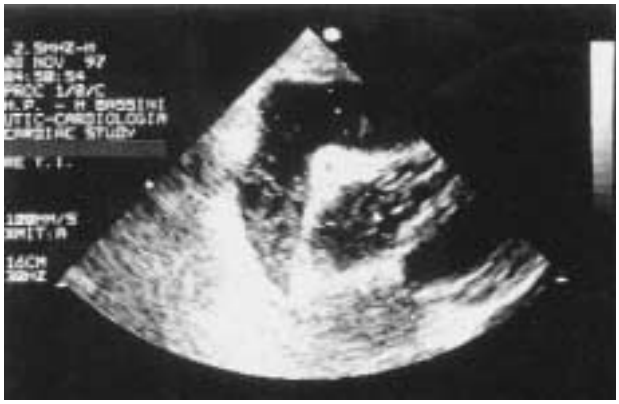

Figure 6. *Transthoracic echocardiogram (subcostal 4-chamber view) after partial pericardiocentesis.*

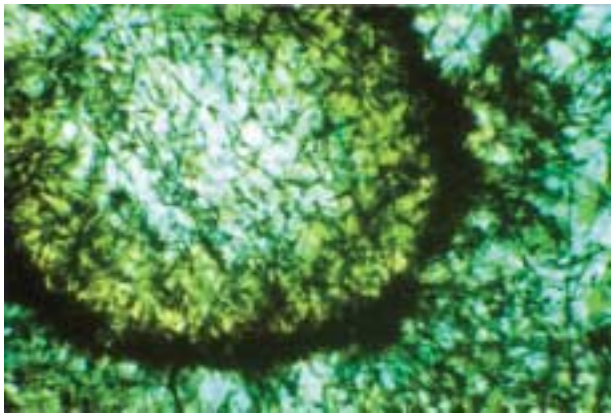

Figure 7. *High power view of a sulfur granule from a specimen of pericardial effusion, showing Gram-positive, thin, filamentous branching rods with peripheral clubbing consistent with Actinomyces.*

ograph of the chest is helpful in detecting a thoracic source of infection. When no primary pulmonary or thoracic source of infection can be seen on a radiograph of the chest, computerized tomography may help detect a pleuropulmonary site of infection[8].

Hepatic actinomycosis is reported in 15% of patients with abdominal infection. Involvement of the liver is usually due to spreading via the portal vein from an intestinal source, and may also occur by direct extension or via the hepatic artery during disseminated infection. In most previously reported cases of hepatic actinomycosis, the patient has had a history of abdominal surgical procedures[9]. In this case report, we can hypothesize that actinomycosis of the liver developed at the time of the previous surgery of Wirsung jejunostomy, performed 2 years earlier. The involved liver tissue became indurated and developed irregular masses which were shown by echography and computerized tomography; histologic examination confirmed the presence of abscesses composed of sulfur granules, strongly suggestive of the diagnosis of actinomycosis. Hematogenous spreading from the abdomen to the thorax is rarely seen[3]. To our knowledge, this is the first case in which a draining fistula from the liver across the diaphragm caused a widespread dissemination of purulent material in the pericardial space.

This rare way of dissemination was confirmed both by a computerized tomography, which demonstrated a partial resolution of the largest lesion of the liver, and by a laparotomy which confirmed the draining fistula across the diaphragm.

A review of the literature of pericardial actinomycosis reported only one case of macroscopically evident sulfur granules, and that was during an open thoracotomy[10]. The remaining cases all had microscopic evidence of sulfur granules. In this report, the massive dissemination through the fistula could explain the echocardiographic evidence of macroscopic, echo-reflective, irregular masses, floating in the pericardial space, probably consistent with aggregates of sulfur granules.

Purulent pericarditis is uncommonly encountered in clinical practice. When bacterial infection of the pericardial space does occur, the causative organism is usually the *Staphylococcus* or *Streptococcus* species. In these patients overt symptoms of pericarditis are usually detected within 1 week. In cases of pericardial actinomycosis, the mean interval between symptom onset and the time of diagnosis is 25 weeks. Only one case in which the patient had a presumed 20-year history of chronic actinomycosis has been described[11]. In our patient, the fulminant course could be related to the massive dissemination from the liver to the pericardial space through the fistula.

In conclusion, we can find three distinctive features in this report: 1) the fulminant course of the pericardial infection, 2) the rare way of dissemination to the pericardial space through a draining fistula from the liver to the pericardial space across the diaphragm, and 3) the peculiar echocardiographic images of macroscopic, echo-reflective, irregular masses, floating in the pericardial space, probably consistent with aggregates of sulfur granules.

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