Figure 1 MRI, sagittal section

cyst. Repeat ultrasound three months later showed no reaccumulation of fluid or recurrence.

COMMENT

The pericardial cyst in this patient was atypical only in its presentation. It was so centrally placed that, instead of going off to the right, it caused a bulge in the epigastrium. These cysts are focal outpouchings of the parietal pericardium, which may or may not communicate with the actual pericardial space. They are lined by a single layer of mesothelial cells; calcification is rare. On aspiration or removal, the contents are clear and colourless; to a thoracic surgeon, the words 'spring water' immediately indicate the diagnosis. Multiple internal loculations occur in approximately 20% of cases.¹

Pericardial cysts are likely to result from congenital persistence of the parietal pericardial recess, due to failure of complete fusion of the mesenchymal coelomic lacunae. Focal weakness of the parietal pericardium with herniation and diverticulum formation, with or without preserved communication with the pericardial space, is another proposed aetiology.²

Most of these are found to arise in the right anterior cardiophrenic angle although they may occur anywhere in the pericardium, posterior cardiophrenic angle or superior retrocardiac pericardial recess.

Plain radiographs may reveal a rounded mass, generally (70%) adjacent to the right heart border. The appearance is non-specific, reflecting the gross anatomic morphology of any unilocular cystic mass. On CT, the walls of the cyst are smooth and thin (or imperceptible) and the content has attenuation equal to that of water. There is no enhancement with intravenous contrast. Owing to their fluid content,

pericardial cysts will be low-intensity on T-1 weighted images MR images and high-intensity on T-2 weighting. Radiologically, differential considerations include bronchogenic cysts, foregut cysts, lymphangiomas and cystic hydatid disease.

Symptomatic pericardial cysts are rare; however, chest pain (22%) and dyspnoea (10%) can result from rapid increase in size, haemorrhage or torsion. In the past, symptomatic cysts were often removed surgically, but image-guided aspiration is now popular.³ When they do not recur after aspiration, a possible reason is that the lesion in the cyst wall remains open, allowing the fluid to recirculate. Percutaneous ethanol induced sclerotherapy has been successful in limited applications.

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***Penicillium marneffei*: a pathogen on our doorstep?**

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Penicillium marneffei, an invasive fungus, has emerged as the third most common opportunistic pathogen among HIV-positive individuals in Southeast Asia. With intercontinental travel commonplace, it needs to be borne in mind by healthcare workers elsewhere.

CASE HISTORY

A man aged 46 was admitted to hospital in the UK after three months of intermittent fever, general malaise, night sweats and chills, chronic productive cough, and weight loss (about 14 kg). He was HIV-positive but not on anti-retroviral drugs. Other medical history included typhoid

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and dengue fever, hepatitis A and shingles. He had been resident in Thailand and regularly visited bat caves, though without directly contacting the animals. His temperature was 40°C. Apart from a few crackles in the left lung base and mild right upper abdominal tenderness, nothing of note was found on physical examination. He was pancytopenic with haemoglobin 8 g/dL, white cell count $1.9 \times 10^9/L$ (normal differential) and platelet count $136 \times 10^9/L$. The CD4+ count was 60/ μ L and the CD8+ count 400/ μ L. Biochemical values were within normal limits except for slightly raised alkaline phosphatase (110 iu/L), gamma-glutamyl transpeptidase (170 iu/L) and alanine aminotransferase (83 iu/L). Chest and sinus X-rays and an abdominal ultrasound were normal. A full sepsis screen proved negative. He improved clinically with no firm diagnosis and was discharged home on no medication.

A fortnight later he was readmitted with recurrent pyrexias and diarrhoea and was found to have groin and axillary lymphadenopathy. Serial blood tests showed progressive worsening of liver function and CT of the abdomen revealed hepatomegaly with hypodense areas. Stool and repeat blood cultures were negative. A liver biopsy showed granulomatous hepatitis with multiple small rounded organisms which were considered to be fungi, possibly *Histoplasma capsulatum*. Intravenous amphotericin (1 mg/kg per day) was started. Four days later, *P. marneffei* was isolated from the liver biopsy specimen. A papular rash appeared on his forehead; no biopsy was taken and it faded over the subsequent week. A week later he was allowed home, to return thrice weekly for amphotericin infusions. After four weeks of amphotericin, he began maintenance therapy with alternating six-week cycles of oral fluconazole and itraconazole. Two years later he relapsed, after not taking his prophylactic drugs. Because of renal impairment he was then treated with liposomal amphotericin, to which he responded. After an eight-week course of this agent he was started on long-term prophylactic itraconazole and was well when last seen 4 years later.

COMMENT

P. marneffei is a dimorphic fungus (i.e. exists in hyphae and yeast forms) first isolated from captive bamboo rats in Vietnam in 1956.¹ The first naturally acquired human infection was also Vietnamese, in a patient with Hodgkin's disease.¹ Sporadic cases followed until the dramatic increase associated with the advent of HIV infection. Penicilliosis is now the third most common opportunistic infection (after extrapulmonary tuberculosis and cryptococcosis) in HIV-positive individuals within the endemic area of Southeast Asia. Some 20% of affected patients are apparently immunocompetent.¹ The major risk factor for acquisition seems to be exposure to soil rather than rats.² Our patient

was regularly exposed to soil in the bat caves. Airborne infection is probably the main route of acquisition. *P. marneffei* infection appears late in the course of HIV disease, the average CD4+ cell count being less than 50/ μ L in one study.³ The clinical manifestations of disseminated *P. marneffei* infection are non-specific and include fever (99%), anaemia (78%), severe weight loss (76%), generalized lymphadenopathy (58%) and hepatomegaly (51%).¹ Cutaneous manifestations (75%), when present, may provide a clue to the diagnosis—typically, umbilicated papules with or without central necrosis (molluscum-contagiosum-like) distributed on the upper trunk, upper extremities, face and scalp.² Pulmonary symptoms are noted in one-third of cases, with variable radiological appearances. Less common features include diarrhoea, pericarditis, arthritis, osteolytic lesions and splenomegaly. Unlike cryptococcal infection, *P. marneffei* infection does not seem to involve the central nervous system.²

The diagnosis of penicilliosis depends largely on histological identification of the characteristic fungus with Wright, Giemsa, and periodic acid/Schiff stains. The presence of intracellular and extracellular basophilic, pleomorphic, yeast-like organisms that divide by fission and therefore display clear central septation is characteristic⁴ and aids differentiation from *H. capsulatum* and other fungi that divide by budding. Routine mycological media are usually sufficient for culture, typically producing a red diffusible pigment in the mould phase.⁴ The highest rate of fungus recovery is achieved from bone marrow aspirates (100%) and lymph node biopsy specimens (100%), followed by touch smears of a skin biopsy specimen (90%). Blood cultures have a yield of 76%.¹ Despite advances in serology, a commercial widely available method for early detection of the fungus is still awaited.

Untreated, disseminated *P. marneffei* infection is almost invariably fatal. The current recommended treatment is intravenous amphotericin B 0.6 mg/kg per day for two weeks followed by oral itraconazole 400 mg daily for a further ten weeks. This regimen has a response rate of up to 95% and is well tolerated.⁵ Thereafter, lifelong prophylaxis with oral itraconazole 200 mg daily is recommended.⁶ In our patient the relapse was probably due to a combination of non-adherence to prescribed medication and the poor efficacy of fluconazole against this organism.⁷ In patients with advanced HIV infection in endemic areas, primary prophylaxis with itraconazole significantly reduced the rate of infection, though without an apparent survival advantage.⁸

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