Tuberculous Pericarditis presenting as Pericardial Tamponade

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SUMMARY: We present one of the last cases from the British Military Hospital in Hong Kong. A 30 year old woman with pericardial tuberculosis and tamponade is described.

Introduction
Over the last few years there has been a world wide resurgence in tuberculosis. Tuberculous pericarditis is a rare manifestation of extrapulmonary tuberculosis and tamponade an unusual feature of this disease process.
This case is especially noteworthy as tamponade developed during pregnancy, possibly due to the changing cardiovascular demands of pregnancy.

Case history
A thirty year-old woman from West Nepal, four months pregnant with her second child, was admitted to hospital with a short history of fatigue and breathlessness associated with fever and central chest pain, worse on inspiration. She was noted to have a markedly raised jugular venous pressure with a low blood pressure and rapid pulse. Chest X-ray revealed a very large heart shadow with clear lungs. A clinical diagnosis of cardiac tamponade was made and pericardial aspiration was attempted. This produced only 50 mls of bloodstained serous fluid.

Because of her deteriorating state a small antero-lateral left thoracotomy was performed, revealing a tense pericardium. Two pericardial windows were made and the removed tissue sent for culture and sensitivity. Granulomata seen on microscopy confirmed the suspicion of tuberculosis and she was placed on triple therapy. The diagnosis was finally proven by positive culture after six weeks.

She responded well and successfully delivered a normal baby. Her husband is still serving in the Royal Gurkha Rifles.

Discussion
The World Health Organisation (WHO) estimates that 1722 million persons are infected with tuberculosis. In most individuals infection is latent or inactive (1). Around 8-10 million new cases of tuberculosis occur each year with about 3 million deaths (2).

There has been a world wide resurgence of tuberculosis, mostly in the developing world but also in industrialised countries. Between 1987 and 1993 tuberculosis rates increased by 35.5% in London compared to 15% in England and Wales as a whole (3).

Tuberculous pericarditis occurs in 1-4% of non respiratory tuberculosis (4,5). Infection may reach the pericardium by lymphatic extension from mediastinal lymph nodes, by direct extension from caseous lung tissue or from haematogenous (miliary) spread. Lymphatic extension from mediastinal nodes is considered to be the commonest mode of infection (6).

The pericardial effusion is thought to be due to a hypersensitivity reaction to tuberculoprotein (5). Untreated; fibrocoseous material accumulates within the pericardial space which interferes with ventricular filling. Still later, calcification and true constrictive pericarditis may occur (7).

Clinical manifestations of pericardial effusions are diverse ranging from the asymptomatic with merely an enlarged heart shadow on X-ray to cardiac tamponade (8). Up to 90% of cases may have features of tamponade, but associated shock is rare (9).

It is known that blood volume in pregnancy is 30% greater than normal by term and that cardiac output is increased by 30-40% by 27 weeks (10). We speculate that the increased cardiovascular demands of pregnancy may have provoked pericardial tamponade in this case.

Diagnosis of tuberculous pericarditis may be difficult. Skin testing may be unreliable in the presence of immunosuppression or anergy (5). Strang et al, however,
found positive Mantoux tests in 239 of 240 patients with tuberculosis pericarditis (11). If treatment is delayed marked thickening of the pericardium may occur and is more likely to result in constrictive pericarditis (6). As in a tuberculous pleural effusion only scanty if any mycobacteria may be detected on ZN staining of pericardial fluid. Confirmation of tuberculous infection is by culture of pericardial aspirate and by culture and histology of pericardium.

Detection of tuberculosis elsewhere in the thorax supports the diagnosis. However active pulmonary tuberculosis may be demonstrated in only 1/3 of cases (11). Indirect methods may be used including detection of adenosine deaminase from pericardial fluid, DNA amplification via the polymerase chain reaction and identification of interferon gamma (6).

Defective immunity may permit recrudescence of previously dormant tuberculosis, while in the past this may have been due to age or alcoholism today the commonest cause is Human Immunodeficiency Virus (HIV) infection (1).

HIV infection is well known to be associated with pericardial disease. AIDS has been identified as the leading diagnosis associated with pericardial effusion in one series in an urban population in the United States (8).

Potential causes of pericardial effusion in AIDS include infection with mycobacteria or cytomegalovirus, or neoplastic processes such as lymphoma, carcinoma and Kaposi's sarcoma (8).

The WHO estimates just over 20 million people are currently infected with HIV and of these million are co-infected with Mycobacterium tuberculosis. People infected with HIV are at increased risk of contracting tuberculosis (3).

Tuberculosis is more common in HIV infection but it is more frequent in its extrapulmonary form (12). Atypical mycobacterial infection is also more common in HIV infection.

Extrapulmonary tuberculosis may occur in greater than 70% of patients with tuberculosis and pre-existing AIDS or AIDS diagnosed soon after the diagnosis of tuberculosis (12), indeed extrapulmonary tuberculosis has been an AIDS defining illness since 1987.

Primary pericardial effusion in the absence of HIV infection may also be idiopathic, or associated with toxoplasma, bacterial pneumonia, systemic lupus erythematosus, rheumatoid arthritis, thyroid disorders or dissecting aortic aneurysm (13).

Treatment of pericardial tuberculosis should be with 6-9 months of standard chemotherapy (5,6). The place of steroids in the treatment of pericardial tuberculosis has become clearer since the publication of large randomised series from South Africa (11,7). Corticosteroids increase the rate of clinical improvement during antituberculosis chemotherapy and probably reduce the risk of death and the need for pericardiectomy.

Multidrug resistant tuberculosis is well recognised, however between 1982 and 1991 only 0.6% of primary isolates were resistant to isoniazid and rifampicin. Provisional figures for 1994 show 2.8% of isolates from the North Thames region and 1.4% of isolates from the South Thames region are multidrug resistant (3).

Pericardiocentesis should be performed for diagnostic purposes and for symptomatic effusions. This can be repeated but for rapidly recurring effusions surgery is indicated. This may be undertaken via a subxiphoid approach, via an antero-lateral thoracotomy or thoracoscopically (14).

Prior to the introduction of antibiotics, tuberculous pericarditis had a mortality rate of 80-90% (4,5). In the modern setting Strang et al reported mortality of 3% in patients treated with steroids and anti-tuberculous drugs and a mortality of 14% in those given anti-tuberculous chemotherapy only (11). Hugo-Hamman and colleagues recently reported a series of 44 cases of tuberculous pericarditis in children from Cape Town, recovery was complete in 43 cases with 1 unrelated death. Only 5 of the children required pericardiectomy (9).

Conclusion
All clinicians need to be aware of tuberculosis and how it may present at various sites. Population movement and the rise in HIV infection have led to an increased incidence of tuberculosis. Pericardial tuberculosis is rare but should be considered in the differential diagnosis of all cases of pericarditis.

We believe that in this case the extra demands placed on the cardiovascular system during pregnancy precipitated pericardial tamponade.

Medical therapy for tuberculous pericarditis is usually successful but surgical approaches allow drainage of fluid, histological diagnosis and obviate the need for repeated pericardiocentesis.

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*J R Army Med Corps* 1998 144: 31-33
doi: 10.1136/jramc-144-01-07

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