

Tuberculous Pericarditis

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ABSTRACT

Tuberculous pericarditis is a serious problem in sub-Saharan Africa with a mortality at six months of about 40% if there is associated HIV infection and 17% without. The key to improved treatment is for the clinician to be alert to the warning features, to conclude the diagnosis promptly and institute treatment as a matter of urgency.

Key words: tuberculous pericarditis, effusion, pericardiocentesis, steroids.

INTRODUCTION

A recent review by Apiu et al in this journal highlighted the importance of recognising extra-pulmonary tuberculosis.^[1] It is now proposed to describe in more detail the various extra-pulmonary sites that may become infected with *Mycobacterium tuberculosis*. This article, based on our “Back to Basics” initiative, focuses on tuberculous pericarditis.

The pericardium is an important structure that surrounds the heart and the great vessels. It comprises two main layers: an outer fibrous layer and a thinner internal serous one. The fibrous pericardium is attached to the central tendon of the diaphragm and is relatively rigid. The serous pericardium has two layers: an outer parietal and an inner visceral one that forms the epicardium. The space between the fibrous and serous pericardial layers normally contains between 20 and 60ml. of fluid. See Figure 1.

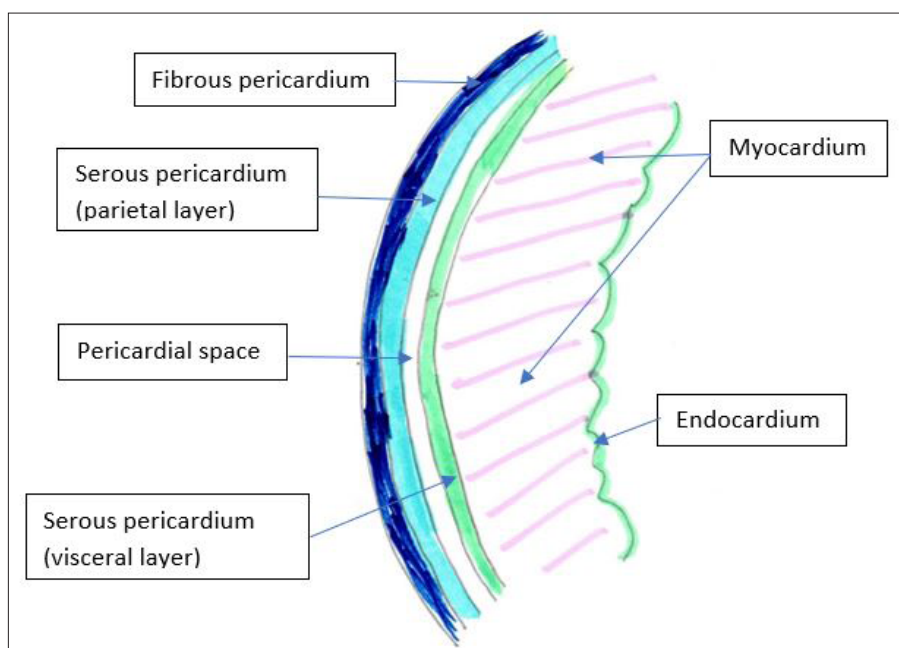


Figure 1. Schematic diagram of pericardial structures

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There are four key functions of the pericardium:

1. Fixation of the heart within the mediastinum.
2. Lubrication of movement of the heart.
3. Limits any overfilling of the heart.
4. Protection against infection from adjacent structures, especially the lungs. However tubercular infection of the pericardium may still result from erosion from adjacent mediastinal lymph nodes.

Inflammation of the pericardium (pericarditis) has numerous causes:

1. Viral including the human immunodeficiency virus and Covid19.
2. Bacterial especially *Staphylococcus*, *Streptococcus* and *Pneumococcus* and *Mycobacterium tuberculosis*.
3. Fungal: e.g. *Candida albicans*.
4. Parasitic e.g. *Taenia solium* (cysticercosis), *Entamoeba histolytica* (amoebiasis), *Echinococcus granulosus*.
5. Dressler's syndrome following myocardial infarction or cardiac surgery.
6. Connective tissue disorders e.g. rheumatoid arthritis and systemic lupus erythematosus.
7. Renal failure.
8. Malignancy especially lung and breast cancer.

Between 1% and 2% of patients with pulmonary tuberculosis (TB) are found to have an associated pericardial infection. The occurrence of this cause of pericarditis has increased with the spread of HIV.^[2] In a series from Tanzania patients with large pericardial effusions and HIV all had TB pericarditis.^[3] TB is the commonest cause of pericarditis in developing and emerging countries.^[4]

WHEN TO SUSPECT TUBERCULOUS PERICARDITIS AND EFFUSION

The clinical history and physical examination are key to indicating the likelihood of tuberculous pericarditis. Known or detection of TB elsewhere in the body should raise suspicions further.

1. Retrosternal chest pain that may be aggravated by deep breathing, coughing and lying supine. It tends not to occur with exercise as with angina pectoris (ischaemic coronary artery disease).
2. Pericardial friction rub heard especially over the lower sternal area. It may be loudest in full expiration, sitting up and leaning forward. It disappears as pericardial fluid accumulates.

3. Malaise.
4. Fever and nocturnal sweating.
5. As the pericardial fluid collects the patient becomes breathless on exercise with the following physical signs:
 - i. Tachycardia and falling blood pressure.
 - ii. Pulsus paradoxus: with respiratory inspiration the arterial pulse becomes weaker and the systolic blood pressure falls.
 - iii. The jugular venous pressure rises and further still on inspiration (Kussmaul's sign) which is the opposite of normal.
 - iv. The heart sounds become less audible with an added third heart sound.
 - v. The cardiac apex beat is less easily palpable.
 - vi. With increasing compression of the heart hepatomegaly and
 - vii. Ascites develop.

If the pericardial effusion collects rapidly there is less time for the pericardium to stretch and the above signs appear more quickly indicating an emergency situation.

CONFIRMATION OF THE DIAGNOSIS

1. An electrocardiogram recording will show a falling voltage of the QRS complex as an effusion collects. In the early stages of the pericarditis there may be an upward curvature of the elevated ST segment.
2. A chest Xray may show evidence of pulmonary TB including a pleural effusion. With the accumulation of pericardial fluid the heart silhouette enlarges and becomes globular. With longer standing disease the pericardium may calcify and be seen on the Xray outlining the heart.
3. An echocardiographic examination confirms the presence of a pericardial effusion. In the absence of this facility an abdominal ultrasound machine may be used directing the probe upwards from the xiphisternum. If the effusion is less than 0.8 cm. thick then there is likely to be up to 200 ml. and if 1.5 to 1.8 cm. the volume may reach 1,000ml.^[5]
4. In 80% the effusion is bloody containing large numbers of lymphocytes and a high protein (>30G/L). In up to two thirds a culture will be positive for TB.
5. In the absence of any other obvious cause (see list above) then TB pericarditis must be seriously considered.

6. In all cases the HIV status of the patient should be established. If positive this will increase TB as the underlying cause.

MANAGEMENT

The rapid onset of cardiac compression (tamponade) is an emergency. Aspiration of the pericardial effusion is required. This is best and most safely done with echocardiographic guidance via the sub-xiphoid route. However, without such guidance the careful insertion of a needle and plastic cannula can be life-saving.

There are many risks with pericardiocentesis.^[6] The key ones are:

1. Puncture of the heart.
2. Damage to epigastric, intercostal and coronary arteries.
3. Pneumothorax.
4. Cardiac dysrhythmias.
5. Infection.

In the absence of an emergency and if no facility or skill is available for diagnostic aspiration of pericardial fluid then a judgement must be made about the likelihood of a tubercular cause. If the latter is concluded then treatment is urgent.

Anti-TB treatment should be along conventional lines including the four drugs isoniazid, rifampicin, pyrazinamide and ethambutol given for four months. Thence another two months of isoniazid and rifampicin.

The benefit of adding steroids to the treatment regimen remains uncertain. There are indications that there is a more rapid recovery, a reduction in re-accumulation of pericardial fluid, less need for pericardectomy later and reduced mortality although the latter has not always reached significance in trials.^[7,8] The schedule of prednisolone cited by Parry et al^[9] is:

- Loading dose of 60 mg,
- 30 mg daily for 5 – 8 weeks reducing to
- 15 mg. daily for 9 – 10 weeks and
- 5 mg. daily for week 11.

With optimal measures being taken to treat, the effusion resolves in about 80% of patients but 20% go on to develop a thick fibrinous pericardial exudate and calcification and

a degree of chronic cardiac constriction with a fifty percent chance of needing a pericardectomy. The mortality remains a challenge: a South African study^[10] revealed at six months a 40% mortality of the group of patients who also had HIV and 17% in those who were HIV free.

References

1. Apiu GK., Sala L, Ianetti R, McIntosh JC. One year's experience of extra-pulmonary TB in a county/regional hospital in South Sudan. *South Sudan Medical Journal* 2022; 15(1):12 – 15.
2. Bongani MM, Burgess LJ, Doubell AF. Tuberculous pericarditis. *Circulation*. 2005;112:3608.
3. Cegielski JP, Lwakatare J, Dukes CS, et al. Tuberculous pericarditis in Tanzanian patients with and without HIV infection. *Tuberc. Lung Dis.* 1994; 75(6): 429.
4. Troughton RW, Asher CR, Klein AL. Pericarditis. *Lancet*. 2004; 363: 717 – 727.
5. Parry E, Godfrey R, Mabey D, Gill G. "Principles of Medicine in Africa". Cambridge University Press. 2004. ISBN 0 521 534380. Part IV, Page 874.
6. Kumar R, Sinha A, Lin MJ, et al. Complications of pericardiocentesis: A clinical synopsis. *International Journal of Critical Injury and Injury Science*. 2015; 5(3): 206 – 212.
7. Hakim JG, Ternouth I, Mushangi E. et al. Double-blind randomized placebo-controlled trial of adjunctive prednisolone in the treatment of effusive tuberculous pericarditis in HIV seropositive patients. *Heart*. 2000; 84:183 – 188.
8. Schutz C, Davis AG, Sossen B. et al. Corticosteroids as an adjunct to tuberculosis therapy. *Expert Review of Respiratory Medicine*. 2018; 12(10): 881 – 891.
9. Parry E, Godfrey R, Mabey D, Gill, G. "Principles of Medicine in Africa". Cambridge University Press. 2004. ISBN 0 521 534380. Part IV, Page 876.
10. Mayosi BM, Wiysonge CS, Ntsekhe M. et al. Mortality in patients treated for tuberculous pericarditis in sub-Saharan Africa. *South African Medical Journal*. 2008; 98(1): 36 – 40.