MEDICINE IN THE TROPICS

Pericarditis

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Considerations of both the high prevalence and the curability of tuberculous disease alert most workers in tropical areas towards a diagnosis of tuberculosis whenever a patient is found to have pericarditis. Indeed, tuberculous pericarditis is common in most tropical areas ; but other forms also occur.

In most analyses of cardiological conditions in African countries, pericarditis accounts for up to 5% of the total. The majority of cases are infective in origin. The classical clinico-pathological descriptions of "dry" and "effusive" pericarditis apply to most forms of acute pericarditis. Chronic pericarditis may take the form of either pericardial effusion or constrictive pericarditis. Malignant pericarditis may also occur in tropical areas.

Acute Pericarditis

The characteristic symptom of acute pericarditis is pain across the precordium. It may vary in intensity, sometimes radiating to the shoulders and arms, and it is made worse by movement of the chest or deep breathing. The patient often obtains comfort by sitting up. Some dyspnoea is usual.

There is often moderate fever and sweating. The characteristic physical sign is a friction rub, which is likened to a to-and-fro-murmur and is of rough rasping quality. It may either be present over a wide area or be localized to a small area of the precordium. It can be differentiated from a pleural friction rub because it continues with the cardiac cycle when the patient is made to hold his breath.

The electrocardiogram in acute pericarditis shows elevation of the S-T segment in all of the leads. The S-T segment preserves its upward concavity.

Effusive Pericarditis

A pericardial friction sound which may have been present earlier in the course of the condition will become no longer audible if a pericardial effusion separates the parietal and visceral pericardial layers.

Symptoms of dyspnoea, dry cough, and a sense of tightness in the chest are related to the size of the effusion. A straight *x*-ray film of the chest will show an enlarged, almost symmetrical, globular heart shadow. This must be differentiated from enlargement due to severe heart failure. Distant heart sounds and the absence of murmurs help to distinguish a pericardial effusion from a case of severe heart failure. In the presence of a pericardial effusion the QRS voltage in the electrocardiogram is usually low and may be less than 5 mm. in height in all of the standard and unipolar limb leads. The T waves are usually flattened.

A large effusion may interfere with cardiac function. Such a situation is called cardiac tamponade. The clinical features of cardiac tamponade are due to restriction in cardiac filling and a consequent reduction in cardiac output. The jugular venous pressure is markedly raised, and the liver is distended and tender. The cardiac output is low, shown by peripheral coldness and cyanosis, a poorly perceptible pulse, and a low systolic blood pressure and pulse-pressure.

The systolic blood pressure shows an exaggerated fall in inspiration and reappears with expiration, and is known as the "pulsus paradoxus." It is best noticed by gentle palpation of the brachial artery with the patient breathing in and out slowly and deeply. Pulsus paradoxus can also be demonstrated with a blood-pressure cuff inflated to just above systolic level ; the cuff pressure is released slowly by deflating the cuff, and auscultation of the first systolic sounds during the phases of respiration will show a drop of up to 15 mm. Hg during inspiration.

The venous pressure may also show a paradoxical rise in inspiration. The heart rate is rapid. A large effusion may cause collapse of the left lower lobe of the lung, which is recognized by diminished percussion note and bronchial breathing (Ewart's sign). The latter features may also be due to a concomitant left-sided pleural effusion.

A rapidly developing pericardial effusion may cause severe shock. The onset of weakness and sweating, marked dyspnoea, tachycardia, and reduction of the blood pressure and pulse volume with a brisk rise in the jugular venous pressure in a patient with known pericarditis will probably signal the onset of cardiac tamponade. Cardiac tamponade is an acute medical emergency and requires immediate relief by pericardiocentesis.

Tapping an Effusion

Pericardiocentesis is not only necessary for relief of cardiac tamponade but it also provides pericardial fluid for examination.

The patient is made to sit up and is supported in the semi-vertical position. The procedure must be explained to him carefully. He may keep his arms by his sides, but it is easier for the attendant if the patient holds his hands together across the top of the head. A nurse can help with this, as it may prove tiring to the patient.

After cleansing of the skin over the precordium, local anaesthetic (procaine or lignocaine 1% to 2%) is injected in the area formed by the angle of the ensiform cartilage and the left costal margin. Care must be taken to raise a weal first before injecting deeply. The plunger is withdrawn repeatedly as more anaesthetic is injected. It is best to be generous with the volume of anaesthetic used, and 3 to 5 ml. is usually necessary. The needle is directed towards the left scapula.

As soon as the pericardium is touched, a sensation of resistance is noted and further injection and withdrawal will result in aspiration of the effusion. At this stage it is advisable to change to a larger bore needle attached to a two-way tap and a large capacity syringe—e.g., 20 or 50 ml.

The needle is steadied in one position throughout aspiration. Specimens are collected in sterile containers for cytology, culture, and chemistry. To avoid clotting of protein-rich fluid, it is advisable to heparinize the containers (0.5 ml. heparin 1,000 units/ml. in each container). Aspiration is continued until no more fluid is obtainable.

Up to two litres of fluid may be removed at a time. The time involved in switching the two-way tap is sufficient to avoid a rapid change in intrapericardial pressure. The procedure is tiring for the operator and he may need to change the syringe if the plunger gets too sticky from the protein-rich fluid.

As soon as the pericardium is dry a coarse grating caused by friction of the needle tip on the pericardium may be heard. The fluid aspirated may be clear and serous, purulent, or haemorrhagic. Unless the fluid is turbid, suggesting a pyogenic infection, there is no need to instil a chemotherapeutic agent

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into the pericardial cavity. In the presence of turbid fluid 1,000,000 units of benzyl penicillin in 10 ml. saline should be instilled.

It is sometimes helpful to visualize pericardial thickening by placing in the pericardial cavity up to 200 ml. of air via the aspirating needle after the last aspiration. A straight film taken immediately afterwards in the erect position will demonstrate the size of the heart and the thickened or normal appearance of the pericardium contrasted against air in the pericardial sac.

Care must be taken to avoid accidental perforation of the heart and the needle should not be advanced if there is resistance or if cardiac pulsation is readily transmitted to it. In case of accidental perforation of the myocardium harm is unusual, but a haemopericardium may develop rapidly with aggravation of the cardiac tamponade.

Pericardial aspiration may be repeated for relief of effusion as often as necessary. In case of tamponade the immediate relief of distress with improvement in dyspnoea and pulse rate and rise in blood pressure and reduction of jugular venous pressure can be quite dramatic.

Clinical evidence of dullness to the right sternal border, signs of restricted cardiac filling and output, and straight radiological follow-up will provide clues for further management.

Clinical Types of Acute Pericarditis

The causes of acute pericarditis may be classified as follows:

(1) Rheumatic.
(2) Pyogenic.
(3) Amoebic pericarditis.
(4) Acute "benign" pericarditis.
(5) "Incidental" pericarditis.

Rheumatic pericarditis occurs as part of the picture of rheumatic pancarditis. A past history of sore throat, flitting articular rheumatism, rheumatic nodules, erythema marginatum, or chorea is unlikely to be obtained unless the patient has been under continuous observation. Because of the lack of easy accessibility to hospital facilities in most African countries patients tend to be admitted only with the severe illness of rheumatic carditis.

The association of fever, tachycardia, and weakness with cardiac enlargement and murmurs, and possibly signs of cardiac failure, indicates rheumatic carditis. The presence of pericarditis is confirmed by the finding of a pericardial friction sound. It may be localized to one area, present minimally as a systolic rasping sound, or may be more extensive in duration throughout the cardiac cycle. It may be transitory or persistent.

A typical case is exemplified by a patient with established aortic incompetence and mitral stenosis who is admitted in cardiac failure. Observation in hospital reveals a transitory pericardial friction rub at the left parasternum and erythema marginatum over the anterior abdominal wall. In such a patient pericarditis indicates rheumatic reactivity against the known background of rheumatic valvular disease. A chest *x*-ray film will show only a cardiac configuration consistent with the valvular lesions present.

Treatment of rheumatic pericarditis is the same as that of rheumatic carditis. A throat swab is taken to detect continuing haemolytic streptococcal infection. Penicillin 500,000 units b.d. is advisable during the course of active rheumatic carditis, which can be judged by the presence of a polymorphonuclear leucocytosis, anaemia, and a raised sedimentation rate. Aspirin (up to 60 gr. (3.6 g.) daily in the young adult), in divided doses given with meals or alkali to minimize gastric irritation, should be administered for as long as there is reason to believe that the rheumatic carditis is active. Where facilities are available a raised serum antistreptolysin O titre—i.e., above 200 Todd units—is evidence of rheumatic activity.

Cardiac tamponade is unusual in rheumatic pericarditis as the pericardial reaction is of the "bread and butter" type. There is no excess fluid accumulation.

Pyogenic pericarditis is more often noticed in childhood than in adult life. It is invariably secondary to a respiratory tract infection, usually pneumonia, by direct extension of the infection. The organisms responsible may be pneumococci, streptococci, or staphylococci. When the pericarditis occurs at the height of the respiratory infection the features of the latter will dominate.

Pyogenic pericarditis is usually in the form of a pericardial collection of pus. The patient is extremely ill with a constitutional reaction in the form of high fever and marked malaise. A plain *x*-ray film of the chest will reveal a large globular heart shadow. There may be residual pneumonic signs in the lung fields. Tamponade may occur.

Diagnosis is confirmed by exploratory pericardiocentesis. Treatment consists of repeated pericardial aspiration, and systemic penicillin (1,000,000 units benzyl penicillin six-hourly) or oral tetracycline (1 g. to start followed by 250 mg. six-hourly) for a minimum of 10 days or until the effusion dries. Appropriate bacteriological sensitivity tests if available will provide guidance as to the choice of chemotherapeutic agents.

A straight Gram stain will show the infecting organism. If previous chemotherapy has been given the pus is likely to be sterile on culture.

Recovery in the early case is prompt and complete. There are no sequelae unless the pyogenic pericarditis has been the result of extension of an empyema thoracis.

Amoebic pericarditis.—In areas where amoebiasis is endemic the complication of pericarditis due to amoebic abscess must be borne in mind. Amoebiasis of the heart arises from direct extension of infection to the visceral pericardium from the left lobe of the liver. Sudden pericardial effusion may cause tamponade.

After a radiological check to exclude associated amoebic empyema thoracis, aspiration of the effusion reveals typical "anchovy sauce" pus in the pericardial cavity. Direct examination may be positive for vegetative forms of *Entamoeba histolytica*.

The treatment consists of repeated aspiration until the pericardium is dry: emetine hydrochloride 60 mg. daily subcutaneously for 10 days, and chloroquine phosphate tabs. 1 g. stat. followed by 1 g. daily in divided doses for five days. Chloroquine is necessary because emetine usually has no effect on hepatic amoebiasis. Care must be taken to ensure complete bed rest during and after emetine therapy to avoid the known risks of myocardial toxicity.

Acute "benign" pericarditis is presumed to be viral in origin (Coxsackie type). Pericardial effusion is common and may be serous or haemorrhagic. The effusion may recur several times.

Cytological examination of the fluid may show lymphocytes and polymorphonuclear cells. The culture is sterile. There is no specific remedy. Because of the need for early treatment in tuberculous pericarditis, the latter must always be excluded (see below).

"*Incidental pericarditis*" is recognized when a friction rub occurs in chronic uraemia, as a terminal event, or following myocardial infarction. The latter condition is rare in African communities.

In both cases the primary disorder dominates the clinical picture. In the case of pericarditis following myocardial infarction mild pain of pericardial origin may be present. Typically the pericarditis of myocardial infarction occurs about the fourth to the eighth day after infarction.

Chronic Pericardial Effusion

In middle African countries the following causes have to be borne in mind:

(1) Tuberculous pericardial effusion.
(2) Endomyocardial fibrosis.
(3) Chronic idiopathic pericardial effusion.
(4) Malignant pericarditis.

Tuberculous pericardial effusion presents in a similar way to acute benign pericarditis and its main differential diagnosis. The pericardial fluid may be serous or haemorrhagic.

Air contrast studies of the pericardium may reveal thickening of the pericardium.

Suggestive evidence of tuberculous aetiology is shown by a positive Heaf test and historical or radiological evidence of past tuberculosis.

Direct examination of the fluid will show a high cell-count, consisting mostly of lymphocytes, and a raised protein. Direct examination of the fluid deposit by Ziehl-Neelsen staining sometimes shows acid-fast bacilli. Culture and biological tests usually entail a wait for several weeks for confirmation of the diagnosis.

The treatment programme is one of repeated aspiration to relieve tamponade and antituberculous therapy (streptomycin 1 g. and thiacetazone 100 mg. with isoniazid 300 mg. in divided doses daily) preferably under clinic conditions for a minimum of three months.

The patient should be reviewed clinically and radiologically for development of pericardial fibrosis and calcification. Signs suggestive of pericardial constriction are those of restricted cardiac filling: persistent jugular venous engorgement, hepatic distension and ascites, and a low cardiac output state—low blood pressure, pulsus paradoxus, a third sound, and sometimes atrial fibrillation.

Surgical relief by pericardiectomy is indicated in the presence of constrictive pericarditis.

Endomyocardial fibrosis.—Reference has been made to this cardiac disorder in an earlier article in this series.[1]

In Uganda endomyocardial fibrosis is associated with a 33% incidence of chronic pericardial effusion. The age group affected is childhood and adolescence. The clinical features are those of pericardial effusion. Confirmation is by pericardiocentesis.

As with tuberculous pericardial effusion, the fluid is protein-rich and clots readily on removal. It is serous, and examination shows the presence of lymphocytes and polymorphonuclear cells. The culture is negative. In the natural course of the illness the pericardial effusion may persist for up to five years. With time it tends to dry, leaving the classical features of endomyocardial fibrosis, which can be differentiated only with difficulty from constrictive pericarditis following "tuberculous" pericardial effusion in such instances.

The absolute diagnosis of endomyocardial fibrosis is obtainable by cineangiocardiography (right or left sided), which will demonstrate flattening of the ventricular apex, and atrioventricular incompetence.

There is no known treatment for endomyocardial fibrosis apart from relief of tamponade as necessary. The disease is usually progressive. Heart failure is intractable and there is only a limited response to digitalis and diuretic agents.

Chronic idiopathic pericardial effusion is known in both temperate and tropical environments. The cause is not clear and may have to do with abnormalities of venous drainage of the pericardium. Patients are known to survive for many years and it is known for female patients to survive pregnancy and labour without ill-effects. The radiological and electrocardiographic signs are those of pericardial effusion. The fluid is serous and may contain large amounts of protein. It is possible that chronic idiopathic pericardial effusion in tropical Africa is a variant of endomyocardial fibrosis.

Malignant pericarditis secondary to a primary carcinoma in the breast, kidney, or thyroid is known to occur in tropical countries. It is usually in the form of a haemorrhagic effusion. Air contrast studies will show thickened parietal pericardium, and special staining malignant cells in the effusion deposit.

In childhood especially, Burkitt's tumour may arise in the pericardium and produce signs of a pericardial effusion or pericardial constriction.

Summary

Pericarditis is an important cardiac disorder in the tropics. It is frequently infective in origin; tuberculous and pyogenic causes are the usual ones, and they can be readily diagnosed and cured. Other specifically tropical causes of pericarditis include endomyocardial fibrosis and amoebic pericarditis.

Features relating to pericarditis as seen under tropical conditions are discussed with regard to diagnosis and management.

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REFERENCE

[1] Parry, E. H. O., *Brit. med. J.*, 1966, 2, 1119.

TODAY'S DRUGS

With the help of expert contributors we publish below notes on a selection of drugs in current use.

Suppository Treatment of Haemorrhoids

There are many factors which make it difficult to determine the value of suppositories in the treatment of haemorrhoids. Firstly, spontaneous remission is common. Secondly, haemorrhoids can be demonstrated in most people if the clinician tries hard enough. Thirdly, there is no objective evidence that suppositories can effect a cure.

Diagnosis and Treatment

As precise a diagnosis as possible must be made and other rectal disease should be excluded by sigmoidoscopy. The symptoms which the patient may attribute to haemorrhoids may in fact be due to other conditions such as fissure, fistula, perianal haematoma, redundant skin tags, or hypertrophied anal papillae. When an acute fissure lies concealed between haemorrhoids it is often not diagnosed. It may heal spontaneously, but local analgesics are helpful in reducing the pain on defaecation and promoting a remission. A perianal haematoma has been described as a "painful five-day self-curing lesion" and is best left alone, unless the lesion is very large and painful, when surgical evacuation is justified. Fistulae, redundant skin tags, and anal papillae need surgical treatment. If the complaint is of bleeding or mucous discharge due to first or second degree haemorrhoids, most surgeons would advise injection of the piles with phenol in oil. If the lesion is third degree then injection or operation would be advised. Patients who refuse both suggestions may reduce or even stop the bleeding if they soften the stool by altering the diet or by