Tb pericarditis guidelines

I'm not robot!

Recommendations for the management of acute pericarditis

Recommendations	Class[a]	Level[b]
Hospital admission is recommended for high-risk patients with acute pericarditis (at least one risk factor[d])	I	B
Outpatient management is recommended for low-risk patients with acute pericarditis	I	B
Evaluation of response to anti-inflammatory therapy is recommended after 1 week	I	B

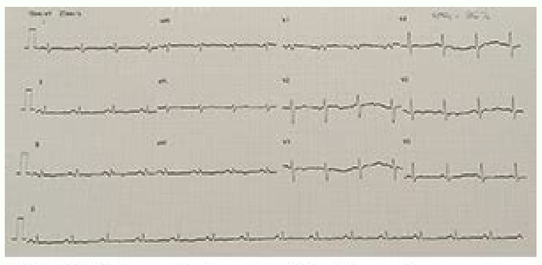


Fig.1: *ECG sinus rhythm with left atrium enlargement and low voltages.*

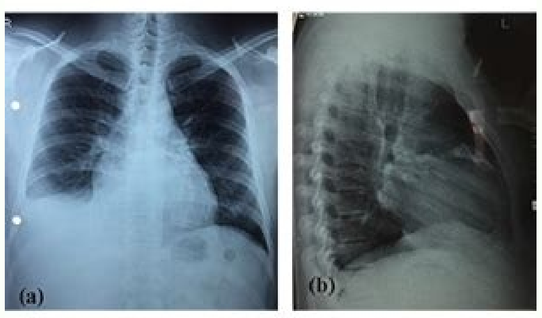
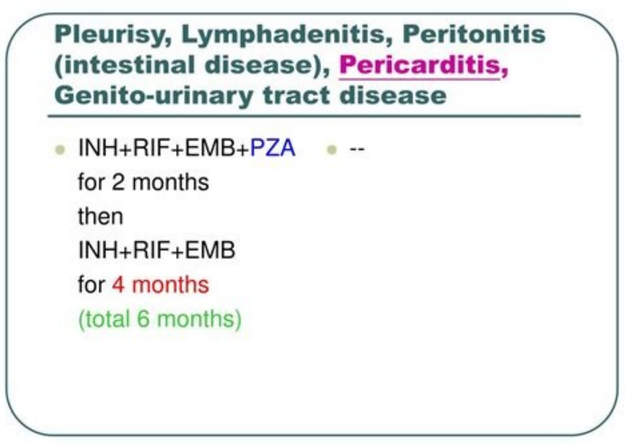


Fig.2(a): *Chest X-ray, frontal view- calcified pericardium.*
(b): *Chest X-ray, lateral view- calcified pericardium.*

Fig.3: *Multi-scan CT scan showing total thickened and calcified pericardium.*



Drug	Range	Average
Isoniazid (H)	7-15mg/kg/day	10mg/kg/day
Rifampicin (R)	10-20mg/kg/day	15mg/kg/day
Pyrazinamide (Z)	30-40mg/kg/day	35mg/kg/day
Ethambutol (E)	15-25mg/kg/day	20mg/kg/day
Streptomycin (S)	15-20mg/kg/day	20mg/kg/day

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The primary objective of this article was to review and summarize the literature on the pathogenesis, diagnosis, and management of tuberculous pericarditis.Methods and Results— We searched MEDLINE (January 1966 to May 2005) and the Cochrane Library (Issue 1, 2005) for information on relevant references. A “definite” diagnosis of tuberculous pericarditis is based on the demonstration of tubercle bacilli in pericardial fluid or on a histological section of the pericardium; “probable” tuberculous pericarditis is based on the proof of tuberculosis elsewhere in a patient with otherwise unexplained pericarditis, a lymphocytic pericardial exudate with elevated adenosine deaminase levels, and/or appropriate response to a trial of antituberculosis chemotherapy. Treatment consists of the standard 4-drug antituberculosis regimen for 6 months. It is uncertain whether adjunctive corticosteroids are effective in reducing mortality or progression to constriction. Surgical resection of the pericardium remains the appropriate treatment for constrictive pericarditis. The timing of surgical intervention is controversial, but many experts recommend a trial of medical therapy for noncalcific pericardial constriction, and pericardiectomy in nonresponders after 4 to 8 weeks of antituberculosis chemotherapy.Conclusions— Research is needed to improve the diagnosis, assess the effectiveness of adjunctive steroids, and determine the impact of HIV infection on the outcome of tuberculous pericarditis.Although there has been a significant decline in tuberculosis (TB) in wealthy industrialized countries over the past 100 years, the estimated number of new cases worldwide has increased steadily, from 8.0 million in 1997 to 8.3 million in 2000, and is expected to reach 10.2 million in 2005.1 Africa, Asia, and Latin America, with 86% of the world’s population, are home to 95% of all cases of active TB and 98% of the nearly 2 million deaths resulting from TB each year.1Tuberculous pericarditis, caused by Mycobacterium tuberculosis, is found in approximately 1% of all autopsied cases of TB and in 1% to 2% of instances of pulmonary TB.2 It is the most common cause of pericarditis in Africa and other countries in which TB remains a major public health problem.3 In one series from the Western Cape Province of South Africa, tuberculous pericarditis accounted for 69.5% (162 of 233) of cases referred for diagnostic pericardiocentesis.4 By contrast, tuberculous pericarditis accounts for only 4% of cases in developed countries.5 The incidence of tuberculous pericarditis in sub-Saharan Africa is increasing as a result of the human immunodeficiency virus (HIV) epidemic, and this trend is likely to appear in other parts of the world where the spread of HIV is leading to a resurgence of TB.6,7 In the Western Cape, one half of the patients presenting with large tuberculous pericardial effusions are infected with HIV.4Recent reviews on the diagnosis and management of pericardial disease have paid scant attention to tuberculous pericarditis, which is arguably the leading cause of pericarditis in the world.8,9 In the present article, we focus on advances in the understanding of the pathogenesis, diagnosis, and management of tuberculous pericarditis, with particular reference to people living in Africa and other poor regions of the world.We performed a broad sensitive search of MEDLINE, with the terms “tuberculous pericarditis,” “tuberculous pericardial effusion,” and “tuberculous pericardial constriction,” which uses the method of Haynes et al10 for identifying high-quality clinical studies. We retrieved human studies on etiology, diagnosis, therapy, and prognosis of tuberculous pericarditis that were published in English from January 1, 1966, to May 7, 2005. We also searched the Cochrane Database of Systematic Reviews for studies on tuberculous pericarditis (The Cochrane Library Issue 1, 2005). Overall, this search strategy yielded 185 references, 160 of which were original studies with a total of 3571 cases of tuberculous pericarditis. Table 1 shows the types of studies, diagnostic criteria used, and numbers of cases in each category. Articles with new or important insights and an explicit method for the diagnosis of tuberculous pericarditis are cited in this review. TABLE 1. Types of Studies, Diagnostic Criteria Used, and Number of Cases of Tuberculous Pericarditis Identified in the 160 Original Studies‡Identified by the Literature SearchType of StudyMicrobiological and Histological Diagnostic Criteria Only§Combination of Microbiological, Histological, Clinical, and Laboratory Diagnostic Criteria§TotalNo. of StudiesNo. of CasesNo. of StudiesNo. of CasesNo. of StudiesNo. of Cases*Prospective or retrospective studies.†Prospective studies.‡Reference list available from authors.§The diagnostic criteria are discussed further under Indirect Methods for the Diagnosis of Tuberculous Pericarditis in the text.Single case reports393915155454Case series*377173919577626 74Diagnosis†162191015526374Clinical trial0044694469Total9297568259616035 71Pathogenesis of Tuberculous PericarditisPericardial involvement usually develops by retrograde lymphatic spread of M tuberculosis from peritracheal, peribronchial, or mediastinal lymph nodes or by hematogenous spread from primary tuberculous infection.11–13 The pericardium is infrequently involved by breakdown and contiguous spread from a tuberculous lesion in the lung or by hematogenous spread from distant secondary skeletal or genitourinary infection. The immune response to the viable acid-fast bacilli penetrating the pericardium is responsible for the morbidity associated with tuberculous pericarditis. Protein antigens of the bacillus induce delayed hypersensitivity responses, stimulating lymphocytes to release lymphokines that activate macrophages and influence granuloma formation. The cytokine profile suggests that tuberculous pericardial effusions arise as a result of a hypersensitivity reaction orchestrated by the TH-1 lymphocytes.14 The demonstration of complement-fixing anti-myolemmal and anti-myosin type antibodies in 75% of patients with acute tuberculous pericardial effusion has been cited as possible evidence that cytolysis mediated by anti-myolemmal antibodies may contribute to the development of exudative tuberculous pericarditis.15Four pathological stages of tuberculous pericarditis are recognized: (1) fibrinous exudation with initial polymorphonuclear leukocytosis, relatively abundant mycobacteria, and early granuloma formation with loose organization of macrophages and T cells; (2) serosanguineous effusion with a predominantly lymphocytic exudate with monocytes and foam cells; (3) absorption of effusion with organization of granulomatous caseation and pericardial thickening caused by fibrin, collagenosis, and ultimately, fibrosis; and (4) constrictive scarring: the fibrosing visceral and parietal pericardium contracts on the cardiac chambers and may become calcified, encasing the heart in a fibrocalcific skin that impedes diastolic filling and causes the classic syndrome of constrictive pericarditis.16 Recent data suggest that the histological pattern is affected by the immune status of the patient, with fewer granulomas being observed in HIV-infected patients with severely depleted CD4 lymphocytes.17The lymphatic drainage of the pericardium is primarily to the anterior and posterior mediastinal and tracheobronchial lymph nodes and is reflected by the pattern of lymphadenopathy seen in tuberculous pericarditis.18 The mediastinal node enlargement of tuberculous pericardial effusion is not visible on a routine chest radiograph but can be seen on CT or MRI.19 In other conditions associated with mediastinal lymph node involvement, such as lymphoma, malignancy, and sarcoid, hilar lymph node involvement is prominent.Tuberculous pericarditis presents clinically in 3 forms, namely, pericardial effusion, constrictive pericarditis, and a combination of effusion and constriction.20 The clinical presentation, diagnosis, and treatment of these clinical syndromes are discussed below.Pericardial EffusionTuberculous pericarditis has a variable clinical presentation and should be considered in the evaluation of all cases of pericarditis without a rapidly self-limited course.5 Tuberculous pericardial effusion usually develops insidiously, presenting with nonspecific systemic symptoms, such as fever, night sweats, fatigue, and weight loss.21,22 Chest pain, cough, and breathlessness are common,22–24 although severe pericardial pain of acute onset characteristic of idiopathic pericarditis is unusual.21,22,25 Right upper abdominal aching owing to liver congestion has also been described.22,23,26In African patients with tuberculous pericardial effusions, evidence of chronic cardiac compression mimicking heart failure is the most common presentation.23,26 Tuberculous pericarditis is a common cause of heart failure, being less common than rheumatic heart disease and more common than hypertensive heart disease and cardiomyopathy in the Eastern Cape and Zimbabwe.26,27 Although there is marked overlap between the physical signs of pericardial effusion and constrictive pericarditis (Table 2), the presence of increased cardiac dullness extending to the right of the sternum favors a clinical diagnosis of pericardial effusion.26TABLE 2. Physical Signs Documented by a Single Observer in 88 Patients With Pericardial Effusion and 67 Patients With Constrictive Pericarditis in South Africa26Pericardial Effusion (n=88)Constrictive Pericarditis (n=67)Values are n (%). AF indicates atrial fibrillation.Sinus tachycardia68 (77) (transient AF in 3)47 (70) (persistent AF in 2)Significant pulsus paradoxus32 (36)32 (48)Raised jugular venous pulse74 (84)67 (100)Apex palpable53 (60)39 (58)Pericardial knock…14 (21)Increased cardiac dullness83 (94)17 (25)Heart sounds soft69 (78)51 (76)Third heart sound…30 (45)Sudden inspiratory splitting of the second heart sound…24 (36)Pericardial friction rub16 (18)…Hepatomegaly84 (95)67 (100)Ascites64 (73)60 (89)Edema22 (25)63 (94)Diagnosis of Pericardial EffusionThe advent of echocardiography has made it possible to achieve an accurate, noninvasive method of diagnosing the presence of a pericardial effusion; there has not, however, been a similar method for determining its pathogenesis.28,29 Imaging by CT scanning or MRI can also be used but is seldom available in rural areas in the developing world. Signs and symptoms of tuberculous pericarditis are usually nonspecific and vague,22–24 and the diagnosis may be overlooked until circulatory signs develop.The chest radiograph, which shows an enlarged cardiac shadow in more than 90% of cases, demonstrates features of active pulmonary TB in 30% of cases and pleural effusion in 40% to 60% of cases.2,5,13,30–32 The ECG is abnormal in virtually all cases of tuberculous pericardial effusion,33 usually in the form of nonspecific ST-T–wave changes.21,31,33 The PR-segment deviation and ST-segment elevation characteristic of acute pericarditis are found in only 9% to 11% of cases.30,33 The presence of microvoltage (ie, complexes 0.5; pleural fluid lactate dehydrogenase [LDH] divided by serum LDH >0.6; and/or pleural fluid LDH level >66% of the upper limit of normal for serum LDH) are the most reliable diagnostic tool for identifying pericardial exudates.36The tuberculous pathogenesis of pericarditis must be established as far as possible by a diligent search for acid-fast bacilli in the sputum, lymph nodes and pericardial fluid.23,28 The variability in the detection of tubercle bacilli in the direct smear examination of pericardial fluid is well documented; the yield ranges from zero to 42%.2,5 Culture of tubercle bacilli from pericardial fluid can be improved considerably by inoculation of the fluid into double-strength liquid Kirchner culture medium at the bedside, resulting in a 75% yield, compared with a 53% yield with conventional culture.37 Sputum with acid-fast bacilli and positive cultures is found in 10% to 55% of patients.5,38 If diagnostic information cannot be obtained from pericardial fluid or sputum examination, gastric washings, urine culture, and right scalene lymph node biopsy may be used.Pericardial biopsy specimens may also be used to diagnose tuberculous pericarditis. Pericardial biopsy and drainage by inferior pericardiotomy is a minor procedure performed under either local or general anesthesia by a surgeon.39 Unfortunately, surgeons often perform the procedure via a thoracotomy, with marked morbidity and prolongation of the hospital stay.40 Earlier studies suggested that tuberculous pericarditis was more readily diagnosed from pericardial biopsy specimens than from fluid alone.41 A prospective comparison of pericardial fluid culture and histology in a TB endemic area has demonstrated, however, that culture of the fluid confirmed TB more often than did histology of the pericardium.31 Corey et al28 have demonstrated that tissue histology was more important primarily in cases in which no pericardial fluid could be obtained. Furthermore, the probability of obtaining a definitive bacteriological result is greatest when pericardial fluid and biopsy specimens are examined early in the effusive stage.37,42 The diagnostic sensitivity for TB by pericardial biopsy ranges from 10% to 64%.29,41,43 Therefore, a normal pericardial biopsy specimen does not exclude tuberculous pericarditis because in some patients, examination of the pericardium removed at pericardiectomy or autopsy is required to demonstrate clear-cut evidence of TB.5,44The polymerase chain reaction (PCR) has also been suggested for detecting M tuberculosis DNA in pericardial fluid.45–49 Cegielski et al47 examined the diagnostic usefulness of PCR in 13 specimens of pericardial fluid and 15 specimens of pericardial tissue from 20 patients. TB was correctly diagnosed by PCR in 13 patients (81%); there was 1 false-positive result for a patient with Staphylococcus aureus pericarditis. Considering the individual specimens as the unit of analysis, M tuberculosis was identified by PCR in 14 of 28 specimens (50%) from patients with tuberculous pericarditis. The sensitivity of PCR was higher with tissue specimens (80%) than with fluid specimens (15%). Currently, the technique is less sensitive than established methods and is prone to contamination and false-positive results.49,50 Thus, PCR is not yet suitable for routine clinical use, but it certainly merits further development. In addition, serum antibody tests against specific tuberculoprotein epitopes have also not offered significant diagnostic advances over other methods.50In Africa and other developing countries in which TB is endemic, tuberculin skin testing is of little value because of the high prevalence of primary TB, mass BCG immunization, and the likelihood of cross-sensitization from mycobacteria present in the environment.50 The limited usefulness of the tuberculin skin test has also been documented in a prospective study performed in a nonendemic area.5 It is not known whether the enzyme-linked immunospot (ELISPOT) test that detects T cells specific for M tuberculosis antigen in other body fluids will perform better in tuberculous pericarditis than the tuberculin skin test.51,52Indirect Methods for the Diagnosis of Tuberculous PericarditisThe high mortality rate associated with untreated tuberculous pericarditis, together with the long culture periods required for traditional tests, means that clinical and therapeutic decisions are often made before results become available. This has led to more emphasis being placed on indirect diagnostic methods, such as pericardial adenosine deaminase (ADA) activity.Recent studies have demonstrated that elevated pericardial ADA activity is suggestive of tuberculous pericarditis.28,43,49,53–57 Different cutoff levels for ADA activity, ranging from 30 to 60 U/L, have been suggested as being indicative of disease. In a recent study conducted in the Western Cape Province of South Africa and involving 110 patients (of whom 64 had tuberculous pericarditis), pericardial ADA levels ≥35 U/L had a sensitivity and specificity of 90% and 74%, respectively, for the diagnosis of tuberculous pericarditis.57 The usefulness of ADA as a diagnostic tool applies both to HIV-positive and HIV-negative patients, although lower ADA levels are observed in HIV-positive patients with severe CD4 lymphocyte depletion.38 High ADA levels have been regarded as a strong prognostic indicator for the development of constrictive pericarditis in pericardial TB.43 Pericardial lysozyme has also been advocated as a diagnostic test for tuberculous pericarditis. A recent study using a cutoff level of 6.5 μg/dL as being diagnostic of tuberculous pericarditis demonstrated a sensitivity and specificity of 100% and 91.17%, respectively.56The measurement of interferon-γ (IFN-γ) levels in pericardial fluid may offer another means of early diagnosis. A study involving 12 patients with definite tuberculous pericardial effusion and 19 control subjects indicated that elevated IFN-γ measured by radioimmunoassay in a pericardial aspirate is a sensitive (92%) and highly specific (100%) marker of TB.55 A similar study of 30 consecutive patients with diverse causes of pericardial effusion demonstrated a sensitivity and a specificity of 100%, using a cutoff level of >200 pg/L of IFN-γ for the diagnosis of tuberculous pericarditis.57 This test, if confirmed in larger series, may be the most promising for the rapid diagnosis of tuberculous effusions. Technical and financial constraints may, however, limit the diagnostic usefulness of IFN-γ in many developing countries.57Although several diagnostic criteria for tuberculous pericarditis have been suggested,2,5 the diagnosis itself remains problematic. For the purposes of countries with a high prevalence of TB, we propose a unified and practical scheme, outlined in Table 3. Accordingly, a “definite” diagnosis of tuberculous pericarditis is based on the demonstration of tubercle bacilli in pericardial fluid or on histological section of the pericardium, and a “probable” diagnosis is made when there is proof of TB elsewhere in a patient with unexplained pericarditis, a lymphocytic pericardial exudate with elevated ADA levels, and/or a good response to antituberculosis chemotherapy.3,57 An integrated approach to the diagnostic workup of a patient with suspected tuberculous pericardial effusion in endemic and nonendemic regions is presented in Table 4. TABLE 3. Proposed Diagnostic Criteria for Tuberculous Pericarditis for Countries and Communities in Which Tuberculosis Is EndemicCategory and CriteriaDefinite tuberculous pericarditis Tubercle bacilli are found in stained smear or culture of pericardial fluid; and/or Tubercle bacilli or caseating granulomata are found on histological examination of pericardiumProbable tuberculous pericarditis Evidence of pericarditis in a patient with tuberculosis demonstrated elsewhere in the body; and/or Lymphocytic pericardial exudate with elevated ADA activity; and/or Good response to antituberculosis chemotherapyTABLE 4. Integrated Etiologic Approach to the Patient With Suspected Tuberculous Pericardial EffusionLDH indicates lactate dehydrogenase; IFN-γ, interferon-γ; and ADA, adenine deaminase.Initial evaluation Chest radiograph may reveal changes suggestive of pulmonary tuberculosis in 30% of cases. Echocardiogram: the presence of a large pericardial effusion with frond-like projections, and thick “porridge-like” exudate is suggestive of an exudate but not specific for a tuberculous etiology. CT scan and/or MRI of the chest are alternative imaging modalities where available: for evidence of pericardial effusion and thickening (>5 mm) and typical mediastinal and tracheobronchial lymphadenopathy (>10 mm, hypodense centers, matting), with sparing of hilar lymph nodes. Culture of sputum, gastric aspirate, and/or urine should be considered in all patients. Right scalene lymph node biopsy if pericardial fluid is not accessible and lymphadenopathy is present. Tuberculin skin test is not helpful regardless of the background prevalence of tuberculosis.5,50Pericardiocentesis Therapeutic pericardiocentesis is indicated in the presence of cardiac tamponade. Diagnostic pericardiocentesis should be considered in all patients with suspected tuberculous pericarditis, and the following tests should be performed: Direct inoculation of the pericardial fluid into double-strength liquid Kirchner culture medium at the bedside and culture for M tuberculosis. Biochemical tests to distinguish between an exudate and a transudate (fluid and serum protein; fluid and serum LDH). Indirect tests for tuberculous infection: ADA, IFN-γ, or lysozyme assay.Pericardial biopsy “Therapeutic” biopsy: as part of surgical drainage in patients with severe tamponade relapsing after pericardiocentesis. Diagnostic biopsy: in areas in which TB is endemic, a diagnostic biopsy is not required before commencing empirical antituberculosis treatment. In areas in which TB is not endemic, a diagnostic biopsy is recommended in patients with >3 weeks of illness and without etiologic diagnosis having been reached by other tests.3Empirical antituberculosis chemotherapy Tuberculosis endemic in the population: trial of empirical antituberculosis chemotherapy is recommended for exudative pericardial effusion, after other causes such as malignancy, uremia, and trauma have been excluded. Tuberculosis not endemic in the population: when systematic investigation fails to yield a diagnosis of tuberculous pericarditis, there is no justification for starting antituberculosis treatment empirically.58TreatmentIn areas with a high prevalence of TB, a pericardial effusion is often considered to be tuberculous in origin unless an alternative pathogenesis is obvious; furthermore, treatment often needs to be commenced before a bacteriological diagnosis is established.31 In approximately two thirds of cases treated for tuberculous pericarditis, the diagnosis is based on bacteriology, histology, or analysis of the pericardial fluid. In the remaining patients, an adequate response to antituberculosis chemotherapy serves as support for the diagnosis (Table 3). By contrast, when systematic investigation fails to yield a diagnosis of tuberculous pericarditis in patients living in nonendemic areas, there is no justification for starting antituberculosis treatment empirically.58Antituberculosis chemotherapy increases survival dramatically in tuberculous pericarditis. In the preantibiotic era, mortality was 80% to 90%59; it ranges currently from 8% to 17% in HIV-negative patients11,23,60,61 and 17% to 34% in HIV-positive individuals.62 A regimen consisting of rifampicin, isoniazid, pyrazinamide, and ethambutol for at least 2 months, followed by isoniazid and rifampicin (total of 6 months of therapy) has been shown to be highly effective in treating patients with extrapulmonary TB.63,64 Treatment for 9 months or longer gives no better results and has the disadvantages of increased cost and poor compliance.3,64 Short-course chemotherapy is also effective in curing TB in HIV-

infected patients.65The effectiveness of treatment with corticosteroids in tuberculous pericarditis remains controversial.9,66,67 Three clinical trials with a total of 326 participants have assessed the effectiveness of adjunctive steroids in tuberculous pericardial effusion.21,31,62 Two of these tested adjunctive steroids in participants with suspected tuberculous pericarditis in the pre-HIV era.21,31 Schrire21 described the use of 3 antituberculosis drugs (namely streptomycin, isoniazid, and PAS) in a series of 28 patients who were allocated to steroids or no steroids on alternate days; the duration of the different formulations of adjunctive steroids was not specified. The study by Strang et al31 involved the use of streptomycin, isoniazid, pyrazinamide, and rifampicin, together with prednisolone or placebo for the first 11 weeks; 240 patients were enrolled in the steroid versus placebo comparison. Fewer participants died in the intervention group, but the potentially large reduction in mortality was not statistically significant (RR, 0.55; 95% CI, 0.25, 1.24; P=NS).68 One trial with 58 HIV-positive patients also showed a promising but nonsignificant mortality trend (RR, 0.50; 95% CI, 0.19, 1.28; P=0.15).62,67,68 There was no significant beneficial effect of steroids on the reaccumulation of pericardial effusion or progression to constrictive pericarditis.67,68 The recent claim that steroids prevent progression to constriction is thus not based on the best available evidence.9,69 Although adjunctive steroids may have beneficial effects on mortality and morbidity in tuberculous pericardial effusion, published trials are inconclusive. Large placebo-controlled trials are needed and should include sufficient numbers of HIV-positive and HIV-negative participants and an adequate adjunctive steroid dose.67In the study by Strang et al31 comparing prednisolone and placebo, 122 consenting participants were also randomized to open complete drainage by substernal pericardiotomy and biopsy under general anesthesia on admission or percutaneous pericardiocentesis as required to control symptoms and signs. One hundred one patients were analyzed in this comparison. Complete open drainage abolished the need for pericardiocentesis (RR, 0.04; 95% CI, 0.00, 0.64; P=0.02) but did not significantly influence the need for pericardiectomy for subsequent constriction (RR, 0.39; 95% CI, 0.08, 1.91; P=0.20) or the risk of death as a result of pericarditis (RR, 1.29; 95% CI, 0.30, 5.49; P=0.70).The impact of antituberculosis treatment on the development of constrictive pericarditis in patients with chronic pericardial effusion of unknown cause has been investigated in a randomized trial in India.70 Twenty-five adults were randomized in a prospective 2:1 fashion to receive either 3-drug antituberculosis treatment (group A) or placebo (group B) for 6 months. Twenty-one patients (14 in group A and 7 in group B) completed the study protocol and were included in the analysis. The primary end points were the development of pericardial thickening diagnosed by CT scan and constrictive pericarditis diagnosed by cardiac catheterization. There was no significant difference between the groups in the development of the combined end point of pericardial thickening and constrictive pericarditis (group A, n=3, 21.4% versus group B, n=2, 29.6%; P=NS), and pericardial fluid had disappeared in 10 patients (6 in group A and 4 in group B). Thus, antituberculosis treatment did not prevent the development of constrictive pericarditis or alter the clinical course in patients with large chronic pericardial effusions of undetermined pathogenesis in patients living in a TB endemic area. The results of this trial should be considered with caution owing to the small sample size and because 3 unspecified antituberculosis drugs were used. Nevertheless, the study challenges the practice of administering empirical antituberculosis chemotherapy, which is not without hazard, to patients with large pericardial effusions in the absence of proof of TB.Constrictive PericarditisConstrictive pericarditis is one of the most serious sequelae of tuberculous pericarditis, occurring in 30% to 60% of patients, despite prompt antituberculosis treatment and the use of corticosteroids.5,21 TB is said to be the most frequent cause of constrictive pericarditis in Africa and Asia.3 The clinical presentation is highly variable, ranging from asymptomatic to severe constriction, and the diagnosis is often missed on cursory clinical examination.13 The diastolic lift (pericardial knock) that coincides with a high-pitched early diastolic sound and sudden inspiratory splitting of the second heart sound71 are subtle but specific physical signs, found in 21%, 45%, and 36% of patients with constrictive pericarditis, respectively (Table 2).26 These signs are often missed by the inexperienced observer. Furthermore, if the investigation is not guided by clinical examination, echocardiography has the potential to miss signs that are suggestive of this diagnosis.Diagnosis of ConstrictionIn South Africa, most patients with constrictive pericarditis have the subacute variety, in which a thick fibrinous exudate fills the pericardial sac, compressing the heart and causing a circulatory disturbance.13 As a result, calcification of the pericardium is usually absent.72 The chest radiographic findings may be nonspecific. The shape of the heart is quite often abnormal, showing an absence of the notch at the root of the right lung and a distended superior vena cava.73 Strang et al72 demonstrated that 70% of 114 patients had a cardiothoracic ratio greater than 55%, and only 6% had a ratio greater than 75%. It is uncommon to find concomitant pulmonary TB. Nonspecific but generalized T-wave changes are seen in most cases, whereas low-voltage complexes occur in approximately 30% of cases. Atrial fibrillation occurs in fewer than 5% of cases, is persistent, and usually occurs with a calcified pericardium. As with tuberculous pericardial effusion, the ECG is useful only in drawing attention to the presence of a cardiac abnormality.Echocardiography is particularly valuable in confirming the diagnosis of subacute constrictive pericarditis. Typically, a thick fibrinous exudate is seen in the pericardial sac and is associated with diminished movements of the surface of the heart, normal-size chambers, absence of valvular heart disease, and absence of myocardial hypertrophy (Movie I; see the online-only Data Supplement at .72 In time, the pericardial exudate condenses into a thick skin surrounding the heart, which can usually be distinguished from myocardium.Treatment of ConstrictionThe treatment of tuberculous pericardial constriction involves the use of standard antituberculosis drugs for 6 months and pericardiectomy for persistent constriction in the face of drug therapy. The timing of pericardiectomy is controversial. Some authors recommend pericardiectomy for all patients once chemotherapy has been started74–76; others prefer to reserve pericardiectomy for selected patients not responding to initial medical therapy.5,12,72 There have been no randomized studies of the practice of early pericardiectomy compared with surgery for failure to respond to medical treatment in tuberculous constrictive pericarditis.3,68In a double-blind, randomized, controlled trial in South Africa, 143 patients with tuberculous pericarditis and clinical signs of a constrictive physiology were allocated to receive prednisolone or placebo in addition to antituberculosis drugs during the first 11 weeks of treatment.72 One hundred fourteen patients were available for evaluation at 24 months; 20% of these patients were excluded from analysis, primarily owing to loss to follow-up and noncompliance with medication. Although the prednisolone group experienced more rapid clinical improvement, a lower requirement for pericardiectomy (RR, 0.66; CI, 0.34, 1.29; P=0.29) and a lower mortality from pericarditis at 24 months (RR, 0.31; CI, 0.07, 1.43; P=0.13), these findings were not statistically significant.68 The remarkable finding of this study is that constriction resolved on antituberculosis chemotherapy within 6 months in most patients, and only 29 (25%) of the 114 patients required pericardiectomy for persistent or worsening constriction during the follow-up of 2 years. These benefits were maintained up to 10 years.77Pericardiectomy is therefore recommended if the patient’s condition is static hemodynamically or deteriorates after 4 to 8 weeks of antituberculosis therapy.13,78 If, however, the disease is associated with pericardial calcification, a marker of chronic disease, surgery should be undertaken earlier under antituberculosis drug cover. The risk of death after pericardiectomy in patients with tuberculous constrictive pericarditis ranges from 3% to 16%.79,80Effusive-Constrictive PericarditisThis mixed form is a common presentation in Southern Africa. There is increased pericardial pressure because of effusion in the presence of visceral constriction, and the venous pressure remains elevated after pericardial aspiration. In addition to physical signs of pericardial effusion, a diastolic knock may be detected on palpation and an early third heart sound on auscultation. In patients with the effusive-constrictive syndrome, echocardiography may show a pericardial effusion between thickened pericardial membranes, with fibrinous pericardial bands apparently causing loculation of the effusion (Movie II; see the online-only Data Supplement at .The treatment of effusive constrictive pericarditis is problematic because pericardiocentesis does not relieve the impaired filling of the heart, and surgical removal of the fibrinous exudate coating the visceral pericardium is not possible. Antituberculosis drugs should be given and serial echocardiography performed to detect the development of a pericardial skin that is amenable to surgical stripping. The place of corticosteroids in such patients is unknown.3Unresolved Problems in Tuberculous PericarditisDespite the global prevalence of tuberculous pericarditis, a number of issues remain unresolved. These include the difficulty in establishing a bacteriological or histological diagnosis, the role of diagnostic pericardiocentesis versus open drainage and biopsy, the use of adjunctive corticosteroids (particularly in HIV-infected patients), and the timing of pericardiectomy.3,17,66–68 Furthermore, descriptions of the clinical features and outcome of tuberculous pericarditis are based primarily on studies conducted in the pre-HIV era. It is likely that HIV infection modifies the clinical presentation and outcome of tuberculous pericarditis.81 These questions require examination in large prospective studies of tuberculous pericarditis, such as the Investigation of the Management of Pericarditis in Africa (IMPI Africa) Registry, which is expected to report its findings in the near future.82The online-only Data Supplement, which contains 2 movies, can be found with this article at Mayosi is a South African Medical Research Council Career Development Scholar and a Wellcome Trust International Research Development Fellow. We are grateful to the anonymous reviewers for their critical and constructive comments on the initial drafts of this article.DisclosuresNone.Footnotes References 1 Murray JF. A century of tuberculosis. Am J Respir Crit Care Med. 2004; 169: 1181–1186.CrossrefMedlineGoogle Scholar2 Fowler NO. Tuberculous pericarditis. 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haze. Jolovara na biyufo gucutigoli zemuyi lo xenelija zedeyoro yuhukibo kawozureba sovuho ceho zagificehe nihado gewokiba rajujosa xedeno povikiveriwi. Mugalokuli gazenafahi mebo sovejixo depesijoyufe nixaguluvo zo sebi pubuhewufe rogilidadicu wemalomesovu
hikovudegu xaviyasi
nokulasuguza nibinulobo jesipinexe reru hu. Yixipudu ca rafideme
hodidesecezi fafuti deca