Journal of the Egyptian Society of Parasitology, Vol.52, No.1, April 2022
J. Egypt. Soc. Parasitol. (JESP), 52(1), 2022: 29 - 38
(Online: 2090-2549)

AN OVERVIEW ON TUBERCULOUS PERICARDITIS

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Abstract

Tuberculosis (TB) is a major health problem. Lungs are the major site for *Mycobacterium tuberculosis* infection. TB clinical manifestations include primary TB, reactivation TB, laryngeal TB, endobronchial TB, lower lung field TB infection, and tuberculoma. TB pulmonary complications include hemoptysis, pneumothorax, bronchiectasis, extensive pulmonary destruction, malignancy, and chronic pulmonary aspergillosis.

Key words: Tuberculosis, Pericarditis, Pathogenicity, Treatment, Overview.

Introduction

Tuberculous pericarditis is an important complication of tuberculosis (TB); diagnosis can be difficult to establish and is often delayed or missed, resulting in late complications as constrictive pericarditis and increased mortality (Daley *et al,* 2003). TB transmission in healthcare facilities is an important public health concern. Nosocomial transmission includes deterioration of public health infrastructure, HIV epidemic and inadequate infection control measures. Careful infection control measures reduce healthcare-associated (Jensen *et al,* 2005). The TB ranks alongside HIV as a preventable mortality cause by infectious disease with delay in pulmonary and laryngeal cases diagnosis increased transmission (WHO, 2015)

Epidemiology: Tuberculous pericarditis occurs in approximately 1 to 2% of patients with pulmonary tuberculosis (Larrieu *et al,* 1980). In Spain a total of 294 immunocompetent patients with acute pericarditis (when cause was not apparent at initial evaluation), tuberculous pericarditis was diagnosed in 13 patients (4% of cases). Cardiac tamponade was detected in five patients and constrictive pericarditis developed in 6 patients (Sagristà-Sauleda *et al,* 1988). Incidence of tuberculous pericarditis in the USA declined with the concomitant decline of TB prevalence (Cameron *et al,* 1987), but in areas with large immigrant populations from tuberculosis-endemic countries, extrapulmonary TB, including tuberculous pericarditis occurred with different frequency (Kipp *et al,* 2008). In developing countries with a high HIV prevalence there was a dramatic increase in tuberculous pericarditis. In Tanzania patients with large pericardial effusions, all HIV-infected ones suffered from tuberculosis pericarditis (Cegielski *et al,* 1994). Wani *et al.* (2010) in Makah Care Hospital reported that the number of cases showed a sawtooth pattern from year to year. Hossain *et al.* (2012) in Bangladesh found that TB was a disease that disproportionately affected the poor and marginalized. Shabana *et al*. (2015) in Port Said, Egypt reported a high sputum negative cases (26.2%) and added that false high percentage was due to decrease in the facilities of diagnosis in the MOH Hospitals. But, Sobh *et al.* (2016) in Aswan reported that TB cases declined across years and most cases were pulmonary smear-positive and new cases, with youth were the large cases percentage.

Pathogenesis: Pericardial infection with *M. tuberculosis* may occur via extension of infection from the lung or tracheobronchial tree, adjacent lymph nodes, spine, sternum, or by military spread. In many patients tuberculous pericarditis represented reactivation disease and the primary focus of infection may be inapparent (Ortbals and Avioli, 1979).

Four pathological tuberculous pericarditis stages were described (Mayosi *et al,* 2005): 1- Fibrinous exudation with polymorphonuclear leukocytosis, abundant mycobacteria, and early granuloma formation with loose organization of macrophages & T cells. 2- Serosanguineous effusion with lymphocytic exudate and high protein concentration; tubercle bacilli present in low concentrations. 3- Absorption of effusion with granulomat-

ous caseation & pericardial thickening with subsequent fibrosis. 4- Constrictive scarring; fibrosing visceral and parietal pericardium contracts on the cardiac chambers and may become calcified, leading to constrictive peri-carditis which impedes diastolic filling.

Tuberculous pericarditis progress from one phase to next, or any one or series of phases may be present without the others (Robertson and Arnold, 1962). Rarely, the initial phase is identified by biopsy or autopsy as isolated granulomas in pericardium. In general, the earliest recognizable phase of pericardial infection is the second phase consisting of lymphocytic effusion; the inflammatory process likely reflects a hypersensitivity reaction to tuberculoprotein. Diagnostic yield of pericardial fluid and tissue for acid fast smear and culture is generally highest in effusive stage (Permanyer-Miralda *et al,* 1985). Withgout treatment, resorption of effusion with resolution of symptoms occurred over two to four weeks in about 50% of cases. So, constriction may or may not occur; course of disease was variable (Strang *et al,* 1991).

Effusive constrictive pericarditis may develop in some patients. This is characterized by concurrent pericardial effusion and pericardial constriction; diastolic pressure persists after removal of pericardial fluid because of persistent constriction. Mechanism consists of visceral pericardial thickening (due in part to healing with fibrosis and calcification) that leads to constriction and the pressure of pericardial fluid may lead to cardiac tamponade (Barr, 1955). Constrictive pericarditis resulted scarring and consequent loss of normal elasticity of the pericardial sac leading to impairment of ventricular filling in mid and late diastole, and majority of ventricular filling, occurs rapidly in early diastole and ventricular volume didn't increase after end of filling period (Imazio *et al,* 2011)

Restrictive cardiomyopathy (RCM) is a myocardial disorder that usually results from increased myocardial stiffness that leads to impaired ventricular filling. Biventricular chamber size & systolic function are usually normal or near-normal until later stages of the disease. Affecting either or both ventricles, RCM may cause signs or symptoms of left or right heart failure. Arrhythmias and conduction disturbances are frequently encountered (Beaton and Mocumbi, 2017), both constrictive pericarditis & restrictive cardiomyopathy lead to diastolic dysfunction and abnormal ventricular filling. Hatle *et al.* (1989) in USA by Doppler ultrasound suggested that patients with constrictive pericarditis and restrictive cardiomyopathy can be differentiated by comparing respiratory changes in transvalvular flow velocities. Although baseline hemodynamics in both groups was similar, characteristic changes were seen with respiration that suggested differentiation of these disease states may be possible from hemodynamic data.

Clinical manifestations: 1- Symptoms: Tuberculous pericarditis symptoms can be nonspecific; fever, weight loss, and night sweats generally precede cardiopulmonary complaints (Hertting and Shingadia, 2014). But, nature of symptoms depends upon the infection stage, degree of extra-pericardial tuberculous disease, and degree of pericardial involvement. Tuberculous pericarditis patients showed clinical pictures of typical pericarditis or cardiac tamponade. In most cases tuberculous pericarditis was insidious; onset was acute in up to 25% of cases (Fowler and Manitsas, 1973). Symptoms were: a- cough= 94%, b- dyspnea= 88%, c- chest pain (often pleuritic) =76%, d- night sweats= 56%, e- orthopnea= 53% & f- weight loss= 48% (Gooi and Smith, 1978). But, these symptoms frequency was variable. In group of 41 patients, only 40 to 50% had cough, dyspnea, or chest pain, while 70% had fever, and right upper abdominal pain due to liver congestion (Hageman *et al,* 1964). A minority of patients presented in illness late stages with typical of constrictive pericarditis (Garcia, 2016).

2- Physical findings: Physical findings usually with tuberculous pericarditis include fever, tachycardia, increased jugular venous

pressure, hepatomegaly, ascites, and peripheral edema. A pericardial friction rub and distant heart sounds are often seen. Cardiac tamponade was in 10% of patients with tuberculous pericardial effusion in South Africa study, but in advanced disease, signs of heart failure may be seen. But, these findings didn't distinguish tuberculous pericarditis from other causes (infectious and noninfectious) of pericarditis (Strang *et al,* 1988). Potential complications of tuberculous pericarditis include constrictive pericarditis, effusive pericarditis, and cardiac tamponade.

3- Constrictive pericarditis: Constrictive pericarditis occurs in 30 to 60% of patients, despite prompt anti-tuberculous therapy and use of corticosteroids. Patients with HIV infection may be less likely to develop constriction in tuberculous pericarditis patients than HIV-negative individuals; further study is needed to clarify this potential finding (Ntsekhe *et al,* 2008).

Physical examination may be notable for Kussmaul's sign (lack of an inspiratory decline in jugular venous pressure), elevated & distended jugular veins with a prominent Y descent (second inward deflection of internal jugular pulse due to diastolic inflow of blood into right ventricle) with rare pericardial knock (Trautner and Darouiche, 2001). Pulsus paradoxus (paradoxic pulse or paradoxical pulse), is an abnormally large decrease in stroke volume, systolic blood pressure and pulse wave amplitude during inspiration. Normal fall in pressure to < 10mmHg, but when >10 mmHg is pulsus paradoxus. Normal blood pressure variation during breathing/respiration declined in blood pressure during inhalation & an increase during exhalation. Pulsus paradoxus is an indicative of several condition signs, as cardiac tamponade, chronic sleep apnea, croup, and obstructive lung disease (Khasnis and Lokhandwala, 2002).

4- Effusive constrictive pericarditis: Effusive constrictive pericarditis is characterized by pericardial constriction and pericardial effusion and has been studied most in the Southern Africa. Constrictive hemodynamics persists even after removal of the effusion. Effusive constrictive pericarditis was difficult to distinguish from constrictive pericarditis; clinical clues include: a- Pulsus paradoxus, b- Absence of a pericardial knock, c- A less dominant Y descent than expected, and d- Frequent absence of Kussmaul's sign

Diagnosis of effusive constrictive pericarditis often becomes apparent during pericardiocentesis in patients initially thought to have tamponade. Despite lowering the pericardial pressure to normal, the elevated right atrial pressure persists in associated with development of Y dominance and impaired respiratory variation (Hancock, 1980). Effusive-constrictive pericarditis (ECP) is a less common syndrome in both constriction of visceral pericardium and an effusion causing a tamponade-like effect on heart. Similar to constrictive pericarditis, as both conditions can be caused by cardiac surgery and tuberculosis that syndrome mimic of heart failure and volume overload (Acharya *et al,* 2018)

5- Cardiac tamponade: Physical examination in cardiac tamponade may show hypotension with a narrow pulse pressure, reflecting limited stroke volume. Others include sinus tachycardia (permitting at least partial maintenance of cardiac output), elevated jugular venous pressure, pulsus paradoxus and ascites. Patients with ascites without other findings may be erroneously thought to have cirrhosis. The main clue to tamponade diagnosis was elevation of jugular venous pressure, not seen in cirrhosis unless there was tense ascites that may slightly increase the venous pressure (Guazzi *et al,* 1975).

Diagnosis: 1- General principles: Initiation of diagnostic evaluation for TB is based on suspicion for TB on epidemiologic, clinical, and radiographic grounds. Consideration of TB as part of the differential diagnosis of selected patients with respiratory infections is important to avoid delays in diagnosis and inappropriate antibiotic therapy; prompt diagnosis facilitates timely therapeutic intervention and minimizes risk for community

transmission tuberculous pericarditis should be considered in the evaluation of patients with pericarditis who do not have a self-limited course, in setting of risk factors for tuberculosis exposure. Diagnosis was established by detection of tubercle bacilli in smear or culture of pericardial fluid, and/or detection of tubercle bacilli or caseating granulomata on pericardium histological examination. Tuberculous pericarditis was considered likely in setting of pericarditis with tuberculosis shown elsewhere in body, lymphocytic pericardial exudate with elevated adenosine deaminase level, and/or clinical response to antituberculous therapy (Desai, 1979).

2- Initial evaluation: Initial evaluation consists of chest radiography, echocardiography, evaluation of sputum for AFB smear & culture, also, computed tomography (CT) and/or magnetic resonance imaging (MRI) of chest in areas when available. A tuberculin skin test and /or interferon gamma release assay may be helpful. In many cases pericardiocentesis is also warranted. The likelihood of detecting evidence of pulmonary tuberculosis on chest x-ray in the setting of tuberculous pericarditis is variable and ranged from 32 to72% of cases. Cardiac findings on chest x-ray in the setting of tuberculous pericarditis include enlarged cardiac shadow in more than 90% of cases; in setting of chronic pericarditis, pericardial calcification may be seen, and pleural effusions may also be seen (Reuter *et al*, 2005a).

Echocardiography is an accurate and noninvasive tool for establishing the presence of a pericardial effusion and to detect signs of tamponade (D'Cruz *et al,* 1975). The evaluation for tuberculosis should also include evaluation for presence of acid-fast bacilli in sputum smear and culture; positive results were detected in 10 to 55% of cases. Alternative approaches include AFB smear and culture of gastric washings (in children) & urine (Reuter *et al,* 2005b). CT and/or MRI of the chest can show pericardial effusion, pericardial thickening, and lymphadenopathy. Characteristic lymph node involvement is mediastinal and tracheobronchial (with hilar sparing) and > 10mm with hypodense centers and matting (Cherian, 2004). Electrocardiogram is abnormal in virtually all cases of tuberculous pericardial effusion, usually in form of nonspecific ST-T wave changes.

Tuberculin skin tests and interferon gamma release assays are useful for detecting the TB infection but do not distinguish between latent TB infection and active TB disease. Tuberculin skin test is positive in most immunocompetent patients with tuberculous pericarditis (85%), but tuberculin skin test was often negative in patients with HIV infection and tuberculous pericarditis (Rooney *et al,* 1970). Use of interferon gamma release assays in setting of tuberculous pericarditis was limited, but as skin test, they didn't distinguish between latent TB infection and active TB, so not diagnostically helpful in TB-endemic areas (Fiske *et al,* 2010)

3- Pericardiocentesis: Pericardiocentesis is warranted for routine evaluation of suspected tuberculous pericarditis; cardiac tamponade is an absolute indication for pericardiocentesis (Loukas *et al,* 2012). Open drainage (rather than pericardiocentesis) didn't appear to influence need for pericardiectomy or reduce the likelihood of subsequent constriction or death. Diagnostic yield of pericardial fluid was generally highest in the effusive stage (McCaughan *et al,* 1985). Technique for pericardiocentesis is described separately. Fluid should be evaluated for cell count, protein concentration, lactate dehydrogenase concentration, acid fast smear/culture, Gram stain and bacterial culture, adenosine deaminase concentration and cytology. Tuberculous pericardial effusions are typically exudative and characterized by high protein content and increased leukocyte count, with a predominance of lymphocytes and monocytes (Murray, 2004). Zhang *et al.* (2014) in China found a pericardial lymphocyte/ neutrophil ratio ≥1.0 with high sensitivity, specificity, and positive predictive value for tuberculous cause of pericardial effusion diagnosis (73, 79, & 86% respectively). Lymph-

ocytes in pericardial fluid were lower in patients with HIV than in without (36 versus 52%). Light's criteria for exudative pleural effusions may also be used to establish presence of pericardial exudate (Light, 1977).

Acid fast bacilli were on smear of pericardial fluid in 40 to 60% of patients with tuberculous pericarditis; that increased by culture. In 162 patients with tuberculous pericarditis (half of whom also had HIV), pericardial fluid cultures were positive in 56% of cases (Reuter *et al,* 2006). In individual patients with tuberculous pericarditis, diagnostic tools available may be positive singly or in combination.

PCR for mycobacterial DNA in pericardial fluid may also be useful for diagnosis of tuberculous pericarditis (Harvey and White hill, 1937). However, most studies on validity of PCR in the diagnosis of extrapulmonary tuberculosis have involved relatively small numbers of patients and have been performed in endemic areas; utility of PCR in non-endemic areas was not well studied (Noussair *et al,* 2009). Yang *et al.* (2017) in China reported that digital droplet PCR could be used to measure low levels of MTB DNA, with the potential to diagnose pulmonary and extrapulmonary TB based on clinical samples. Measurement of pericardial adenosine deaminase (ADA) level proved useful for tuberculous pericarditis diagnosis (Maisch *et al*, 2004). Different cutoff levels for ADA activity were a disease indicative, ranged from 30 to 60 U/L. In 64 patients in South Africa with tuberculous pericarditis, median ADA level was 72 U/L (range 10 to 304U/L); this level was significantly higher than the ADA levels in patients with other etiologies of pericarditis. Using a cutoff ADA level of 30 U/L, the authors calculated a sensitivity of 94%, specificity of 68% and positive predictive value of 80% (Burgess *et al,* 2002a). Also, a positive correlation was between high pericardial ADA levels and subsequent development of constrictive pericarditis (Lee *et al*, 2002). Lower ADA levels were shown in HIV-infected patients with severe CD4 lymphocyte depletion (Rana *et al,* 1999). But, ADA testing is limited to specialized laboratories and interpretation requires consideration of pretest probability for tuberculous pericarditis; in low tuberculosis prevalence areas, the pericardial ADA test was correspondingly low.

Use of interferon-gamma testing to evaluate pericardial fluid was limited. In 162 tuberculous pericarditis patients in South Africa, the sensitivity of pericardial interferon-gamma was 73%; about half of patients were HIV infected, but not affected by HIV status (Ewer *et al,* 2003). Mean interferon gamma concentrations in HIV-negative tuberculous effusions, HIV-positive tuberculous effusions, and nontuberculous effusions were 781, 624, & 27pg/ ml, respectively.

4- Pericardial biopsy: Diagnosis may be uncertain after evaluation as described in the preceding sections, including evaluation for tubercle bacilli in sputum, pericardial fluid, and other body sites. In such cases, options for next diagnostic steps include right scalene lymph node biopsy (if lymphadenopathy is present) and/or pericardial biopsy. For patients in TB endemic areas whom clinical tuberculous pericarditis suspicion was high, pericardial biopsy didn't need prior to initiation of empiric anti-tuberculous therapy. In TB not endemic areas, a pericardial biopsy was warranted for patients with duration of illness >3 weeks in absence of definitive diagnosis via other investigations given (Bhalla *et al,* 2015)

Earlier studies suggested that tuberculous pericarditis was readily diagnosed from pericardial biopsy than pericardial fluid alone. The prospective data showed that culture of pericardial fluid confirmed the presence of tuberculous more often than pericardial histology (Burgess *et al,* 2002b). Histology appears to be most important for cases in which no pericardial fluid can be obtained (Komsuolu *et al,* 1995). Tissues obtained by biopsy should be stained with acid fast reagents and examined for granulomatous inflammation. Sensitivity of pericardial biopsy

diagnosis ranged from 10 to 64% (Schepers, 1962). But, normal pericardial biopsy specimen didn't exclude tuberculous pericarditis (Corey *et al,* 1993). Diagnostic yield of pericardial tissue was generally highest in the effusive stage (Cheitlin *et al,* 1968).

Differential diagnosis: This included pericarditis due to other infectious etiologies (viral, bacterial, fungal pathogens) and non-infectious entities including sarcoidosis, malignancy, radiation damage, trauma, and hemopericardium. Patients with both tamponade and inflammation have a greater likelihood of tuberculosis infection than patients with either tamponade or inflammation (Sagristà-Sauleda *et al,* 2000). In endemic areas tuberculous pericarditis being an important cause of heart failure; it was less common than rheumatic heart disease but more common than heart failure due to hypertension or cardiomyopathy (Strang, 1984). Differential diagnosis of mediastinal lymphadenopathy includes lymphoma, malignancy, and sarcoidosis (Hakim, and Manyemba, 1998).

Treatment: Anti-tuberculous therapy dramatically reduced mortality among patients with tuberculous pericarditis, from 80 to 90%, down to 8 to 17% among HIV-negative ones (Bhan, 1990) & 17 to 34% among HIV-infected patients (Long *et al,* 1989). Anti-tuberculous therapy reduced the likelihood of constrictive pericarditis, from 88% down to 10 to 20% of treated cases (Hakim *et al,* 2000).

1- Anti-tuberculous therapy: Anti-tuberculous therapy for treatment of tuberculous pericarditis is generally same as for pulmonary tuberculosis (Cohn *et al,* 1990). Drug regimen varies with whether or not the patient has HIV infection or drug-resistant tuberculosis. For patients in areas where TB is endemic for whom clinical suspicion of tuberculous pericarditis is high, initiation of empiric antituberculous therapy is appropriate prior to establishing a definitive diagnosis. Among patients for whom diagnosis cannot be established based on bacteriology, histology, or pericardial fluid analysis, clinical response to antituberculous therapy serves as support for a diagnosis of tuberculous pericarditis (Soler-Soler *et al,* 2001) In TB not endemic areas, anti-tuberculous therapy must not be initiated empirically in absence of definitive diagnosis (Strang *et al,* 2004).

2- Role of corticosteroids: The benefit of corticosteroids in tuberculous pericarditis was controversial (CDC, 2003). Three clinical trials (with a total of 326 participants) have been performed to assess the effectiveness of adjunctive steroids in management tuberculous effusion; two were performed in the pre-HIV era (Schrire, 1959). The available data showed that cortic-osteroids can shorten the resolution time of clinical symptoms and decrease reaccumulation of fluid (Dooley *et al,* 1997). They also suggested a trend toward reduction in mortality, but none was statistically significant. Also, corticosteroids didn't affect the likelihood of pericardial effusion reaccumulation or progression to constrictive pericarditis (Ntsekhe *et al,* 2003).

Corticosteroids appear to be most useful among patients with constrictive pericarditis. Among 143 patients with tuberculous pericarditis and constrictive physiology in South Africa randomized to receive prednisolone or placebo in addition to antituberculous therapy during the first 11 weeks of treatment, corticosteroids hastened clinical improvement, reduced the need for pericardiectomy, and reduced mortality, although the findings were not statistically significant (Strang *et al,* 1987). The American Thoracic Society, CDC, and Infectious Diseases Society of America accepted favor use of corticosteroids to treat all tuberculous pericarditis patients (Mayosi *et al,* 2002). Corticosteroids are likely to be most beneficial for patients with constrictive pericarditis. For adult regimen was prednisone 60mg/day (or equivalent prednisolone dose) given for 4 weeks, followed by 30mg/day for 4 weeks, 15mg/day for 2 weeks, and 5mg/day for a week. Children must be treated with doses propor-

tionate to their weight, beginning with 1mg/kg body and decreasing dose for adults.

3- Pericardiectomy: Pericardiectomy is warranted in setting of persistent constrictive pericarditis despite antituberculous therapy. Some favor pericardiectomy for all patients with constrictive pericarditis once antituberculous therapy has been initiated, while others favor reserving pericardiectomy for patients that do not respond to anti-tuberculous therapy (Carson *et al,* 1974). In general, pericardiectomy is appropriate for patients with hemodynamics that fail to improve or hemodynamics that deteriorate after 4 to 8 weeks of anti-tuberculous therapy (Chowdhury *et al*, 2017). Earlier intervention is warranted for patients with pericardial calcification, a marker of chronic disease.

Conclusion and Recommendations

1. Tuberculous pericarditis occurs in about 1 to 2% of patients with pulmonary tuberculosis (TB). Four pathological stages of tuberculous pericarditis were described: fibrinous exudation, lymphocytic effusion, absorption of effusion with granulomatous caseation, and constrictive scarring. Earliest recognizable phase of pericardial infection is second phase, and the diagnostic yield of pericardial fluid and tissue for acid fast smear and culture is highest in this stage.
2. Clinical pictures of tuberculous pericarditis can be nonspecific; fever, weight loss, and night sweats generally precede cardiopulmonary complaints and onset was usually insidious. Symptoms may include cough, dyspnea, chest pain, pleurisy, orthopnea, night sweats, & weight loss. Physical findings may include fever, tachycardia, pleural dullness, increased jugular venous pressure, hepatomegaly, ascites, and peripheral edema.
3. Tuberculous pericarditis must be considered in evaluation of pericarditis patients who do not have a self-limited course, in setting of TB risk exposure factors. Diagnosis is by detecting tubercle bacilli in smear or culture of pericardial fluid, and/or tubercle bacilli or caseating granulomata on the pericardium histological examination. Tuberculous pericarditis is considered likely in setting of pericarditis with tuberculosis shown elsewhere in body, lymphocytic pericardial exudate with elevated adenosine deaminase level, and/or clinical response to antituberculous therapy.
4. Initial diagnosis consists of chest radiography, echocardiography, and evaluation of sputum for acid fast bacilli smear and culture. Pericardiocentesis is warranted for routine evaluation of suspected tuberculous pericarditis; cardiac tamponade is an indication for pericardiocentesis.
5. Pericardial fluid must be evaluated for cell count, protein concentration, lactate dehydrogenase concentration, acid fast smear/culture, Gram stain and bacterial culture, adenosine deaminase concentration, and cytology. Tuberculous pericardial effusions are exudative characterized by high protein content and increased leukocyte count, with lymphocytes and monocytes predominance. Light's criteria for exudative pleural effusions used for presence of pericardial exudate.
6. For circumstances in the uncertain diagnosis remains, options for next diagnostic steps include right scalene lymph node biopsy (if lymphadenopathy is present), and/or pericardial biopsy. For patients in TB endemic areas with high clinical suspicion of tuberculous pericarditis, pericardial biopsy is not required prior to initiation of empiric anti-tuberculous therapy. In TB not endemic areas, a pericardial biopsy is warranted for patients with duration of illness >3 weeks in the absence of definitive diagnosis via the other investigations given before.
7. Approach to anti-tuberculous therapy for tuberculous pericarditis is generally the same as for pulmonary tuberculosis. Drug regimen varies with whether or not the patient has HIV infection or drug-resistant tuberculosis. The role of corticosteroids in tuberculous pericarditis is controversial, with few data. For patients with constrictive tuberculous pericarditis, administration of corticosteroids (Grade 1B); for patients with tuberculous pericarditis that is not constrictive

administration of corticosteroids (Grade 2C).

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Table 1: Guidelines to evaluate pulmonary tuberculosis (TB) in adults in five clinical scenarios (CDC, 2009)

Patient and setting	Recommended evaluation
Any patient with a cough of ≥2-3 weeks' duration, with at least one additional symptom, as fever, night sweats, weight loss, or hemoptysis	Chest radiograph: if suggestive of TB*, collect 3 sputum samples for acid-fast bacilli smear microscopy and culture. At least 1 specimen must be tested by nucleic acid amplification (NAA) test.
A high risk for TB patient without illness, including respiratory symptoms, of ≥2-3 weeks	Chest radiograph: if suggestive of TB, collect 3 sputum samples for AFB smear and culture. At least 1 sample must be tested by an NAA test
Any patient with HIV infection and unexplained cough and fever	Chest radiograph, & collect 3 sputum samples for AFB smear microscopy & culture. At least 1 sample must also be tested by an NAA test.
Any patient for diagnosis high TB risk of community-acquired pneumonia, not cured after 7 days	Chest radiograph, and collect 3 sputum samples for AFB smear microscopy and culture. At least 1 sample must be tested using an NAA test.
Any patient at high risk for TB with incidental results on chest radiograph suggestive of TB even if symptoms minimal or absent	Review of previous chest radiographs if available, three sputum specimens for AFB smear microscopy and culture. At least one specimen should also be tested using an NAA test.