Iranian Journal of Clinical Infectious Diseases
2006;1(4):195-197
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ORIGINAL ARTICLE

Frequency of tuberculous pericarditis in patients with the diagnosis of pericarditis

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ABSTRACT

Background: During the past decades the prevalence of tuberculosis (TB) has declined and its prognosis has changed dramatically due to chemotherapy; however, cases of tuberculous pericarditis are still reported worldwide.

Materials and methods: The study population included 19 men and 11women aged 12-76 years admitted to the hospital for acute pericardial disease. The diagnosis of tuberculous pericarditis was verified based on the adenosin deaminase activity (ADA), culture on Lowenstein-Jensen media, Ziehl Neelsen staining, biopsy of pericardial tissue and evaluation of tuberculin test.

Results: Of 30 patients, 13 (43.3%) had tuberculous pericarditis. All of whom had clinical TB manifestations. The culture of pericardial fluid in 6 patients (46%), staining in 3 (23%), and the culture of pericardial tissue in 6 patients (46%) were positive. The caseating granuloma w identified in 46% of cases. Ten case (77%) had positive PPD reaction. All tuberculous pericarditis patients had ADA level of >45U/l.

Conclusion: Results have revealed that measurement of ADA level may prove a good screening test for early diagnosis of tuberculous pericarditis.

Keywords: *Pericarditis, Tuberculosis, Adenosine deaminase activity.*

(Iranian Journal of Clinical Infectious Diseases 2006;1(4):195-197).

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INTRODUCTION

Pericarditis, inflammatory of the pericardium, may be caused by virtually any infectious agent and by a wide variety of noninfectious processes including neoplastic disease. Acute serofibrinous pericarditis, the most common form, is associated with enteroviral infections, myocardial infarction, or systemic inflammatory disease such as rheumatoid artheritis; however, the course is usually benign (1,2). Acute purulent pericarditis is usually bacterial in origin and may be rapidly fatal because of cardiac tamponade. Chronic pericarditis is classically caused by mycobacterium tuberculousis. Hence, the symptoms actually reflect the tuberculosis infection (fever, night sweats, weight loss, fatigue), however cardiac tamponade causes progressive circulatory failure (slowly progressive dyspnea, ascites, and edema) (1).

The diagnosis of tuberculous pericarditis is verified based on any of the following criteria: identification of tubercle bacilli in the pericardial

Received: 4 October 2005 *Accepted*: 6 September 2006
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fluid or tissue; identification of tubercle bacilli elsewhere in the body; and identification of caseating granulomas in the pericardium or elsewhere.

Prior investigators showed that the combination of sensitive tests such as adenosine deaminase, chest X-ray and clinical features has a higher diagnostic efficiency than pericardial biopsy in diagnosing tuberculous pericarditis (3), however, other studies demonstrated that in developing countries where TB is quite prevalent, chest X-ray plays an important role in identification of large pericardial effusions. Although sonography will still be required for a definite diagnosis, the results of this study show that chest X-ray is a useful screening tool (4). Aggeli et al showed that the measurement of pericardial ADA needs to be taken into account when attempting the early diagnosis of tuberculous pericarditis (5).

The present study was conducted to determine the frequency of tuberculous pericarditis in patients admitted to the hospital for acute pericardial disease.

PATIENTS and METHODS

Study population included 19 men and 11 women aged 12 to 76 years, admitted to Modarres General Hospital in Tehran, between July 2003 and July 2004, to rule out the etiology of their pericardial disease. All of whom had clinical manifestations lasting for at east 2 weeks. They presented with the following signs and symptoms: chest pain, dyspnea, cough, weight loss, fever, pericardial rub, pericardial effusion, cardiac tamponade and pleural effusion.

Tuberculous pericarditis was diagnosed based on the following standard methods: staining of the samples (pericardial fluid and biopsy) with Ziehl-Neelsen method and culture in solid egg medium (Lowenstein-Jensen), the measurement of adenosine deaminase activity (ADA), and the evaluation of tuberculin test (2). Specimens were obtained through aseptic pericardiocentesis by needle and syringe aspiration. The syringe was immediately sealed and transported to the laboratory. ADA level was determined by a commercial kinetic, enzymatic ultraviolet test that employed reduced nicotinamid adenine dinucleotide as substrate, measuring its clearance at 340 nm (normal values <45U/liter).

RESULTS

Of 30 patients, 13 had tuberculous pericarditis. All of whom had clinical TB manifestations.

The tubercle bacilli were identified by culture in pericardial fluid (46.2%), and pericardial biopsy (46.2%), as well as staining the pericardial fluid (23.1%). The caseating granuloma was detected in 6 cases (46.2%). Ten cases (76.9%) had positive tuberculin reaction.

All patients with tuberculous pericarditis were revealed to have elevated ADA level (>45U/L). Our results suggest that ADA level could be used in addition to other clinical and laboratory parameters for early diagnosis of tuberculous pericarditis.

DISCUSSION

We observed that the diagnosis of tuberculous pericarditis can be made through many different diagnostic studies. In some of the patients, however, the direct Ziehl-Neelsen stain was negative. Culture of pericardial effusion provided positive results in 6 of the 13 patients. In prior studies, the diagnostic yield of pericardiocentesis in tuberculous pericarditis has ranged from 30% to 76% (6). This has been explained by the low concentration of tubercle bacilli in the pericardial exudates (7). Ziehl-Neelsen stain of pericardial fluid has not identified tubercle bacilli, but culture in Lowenstein-Jensen medium or guinea pig inoculation revealed to be necessary, although it

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generally causes considerable delay in the diagnosis.

Our results suggest that ADA level could be used in addition to other clinical and laboratory parameters for early diagnosis of tuberculous pericarditis. Recently, the usefulness of measuring adenosine deaminase activity in the diagnosis of pleural tuberculosis has been reported (8-10); indeed, an activity value of >45U/liter in pleural fluid has a reported sensitivity of 100% and a specificity of 97% for the diagnosis of pleural tuberculosis (11). These observations are of great interest in view of the simplicity, low cost and speed of adenosine deaminase activity measurement and the frequent association of pleural effusion with pericarditis in general and specifically with tuberculosis pericarditis. In the other hand, the measurement of ADA in the pleural fluid of patients with pericarditis may prove a good screening test for tuberculous pericarditis. False positive results may be observed in neoplastic disease; a fact that might reduce its specificity.

In summary, tuberculous pericarditis should be kept in mind in patients presenting with pericardial manifestations in endemic region. ADA level could be served as a good screening test for the aforementioned subjects.

ACKNOWLEDGEMENT

This study was supported by Shahla Zabihian, and staff of the central laboratory of Shaheed Modarres and Massih Daneshvari hospitals.

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