AN EPIDEMIC OF PLEURISY AMONGST MILITARY RECRUITS

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ABSTRACT

An epidemic amongst recruits who presented with acute viral exudative pleural effusion with lymphocytic pleocytosis is analysed. Histologic and bacteriologic proof of tuberculosis was lacking in majority. Most of them recovered without pleural thickening. Overcrowding, inadequate clothing protection, stress and strain of vigorous recruit training could be important precipitating factors. None reported with parenchymal tuberculosis in two year follow up.

MJAFI 2001; 57 : 19-21

KEY WORDS :Pleural effusion; Viral.

Introduction

Respiratory infections are the leading cause of outpatient illness and a major cause of hospitalisation amongst military personnel. Because of exposure to respiratory pathogens in crowded living conditions and stressful environment, military recruits are a potentially high risk group for respiratory disease epidemics.

Pleural effusion a common manifestations of chest diseases, may turn out to be an enigma even after detailed investigations especially when it occurs in an epidemic. Exudative pleural effusions are distinguished by Light's criteria. No diagnosis is established for approximately 20% of exudative effusions, and these effusions resolve spontaneously with no long-term residua [1]. Viral infections are responsible for a sizable percentage of such undiagnosed exudative pleural effusions. The diagnosis of viral pleural effusion is invariably by exculsion. Adenovirus, influenza virus, cytomegalovirus, herpes simplex virus and Epstein-Barr virus have been incriminated [2].

In Armed Forces tubercular pleural effusion has special importance for recruits as they are boarded out of service despite the fact that the disease is curable. Hence detailed investigation should be done to make an all out effort to reach a firm diagnosis.

This communication deals with a critical analysis of forty such cases of pleural effusion, which were referred to this centre for final disposal.

Material and Methods

40 recruits suffering from exudative pleural effusion, who were transferred from a Military Hospital attached to a training centre

constituted the material for study. 5 of them who had associated pulmonary tuberculosis were excluded. Detailed history and examination was done giving special importance to duration of symptoms and cardinal symptoms of respiratory illness. Old chest skiagrams and pleural fluid results were reviewed. Pleural fluid aspiration was repeated at our center whenever residual fluid was detected on Ultrasound or Chest Radiograph. Pleural fluid analysis, biochemical, cytological, including Adenosine deaminase (ADA), Polymerase Chain Reaction (PCR), smear for acid fast bacilli (AFB), Mycobacterium tuberculosis (MTB) culture and pleural biopsy were done whenever possible. Antitubercular treatment (ATT) was given to only those patients who had positive evidence of tuberculosis on smear, culture or histology. Patients were followed for 20-24 months.

Results

Between Apr 98 to Aug 98, 40 recruits were admitted at a training centre hospital with exudative pleural effusion and lymphocytic pleocytosis. 5 patients who had associated pulmonary tuberculosis were excluded from further analysis. Distribution of remaining 35 cases is shown in Fig 1. All were males between 18-21 years of age. They were undergoing military training for 12-30 weeks prior to their illness.

All had short duration of symptoms ranging from 5-15 days.

Fig. 1 : Epidemiological graph showing monthwise distribution of cases

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Fever, chest pain and cough were the predominant presenting complaints (Table-1). Clinically all had mild to moderate pleural effusion, which was confirmed roentgenographically. Pleural fluid was exudative with lymphocytic pleocytosis. Gram stain and culture of fluid did not grow any organism. All of them received antibiotics for 7-14 days before transfer to our centre. 3 out of 35 had bilateral pleural effusion.

TABLE 1
Showing presenting symptoms

Symptom	Number (n=35)	%
Fever	32	91.43
Cough	28	80.00
Chest pain	22	62.86
Weight loss	7	20.00
Dyspnoea	18	51.43

20 of 35 patients showed positive Mantoux reaction of more than 10 mm induration. Demonstration of AFB in smear and MTB culture confirmed tuberculosis in only 1 patient. Sixty percent of patients had ADA levels of more than 70 IU/l whereas none had positive polymerase chain reaction (Table-2).

TABLE 2
Showing results of pleural fluid analysis (n=20)

Test/Procedure	Number +ve
Exudative with lymphocytic pleocytosis	20
AFB smear and MTB culture	1
ADA	
< 40	0
40-70	8
> 70	12
PCR (10)	0
Pleural biopsy (Positive)	
Closed (20)	1
Open (2)	1

8 recruits out of 35 received ATT and were invalided out of service subsequently. Of these 1 had hemoptysis, 1 had hepatosplenomegaly, 1 had associated tubercular lymphadenitis proved on fine needle aspiration cytology (FNAC), 2 patients showed tubercular granuloma on histology of pleural biopsy, 3 patients had significant pleural thickening with moderately restrictive ventilatory defect on spirometry. Remaining 27 recruits were retained in service, none of whom received ATT. 24 of them had normal chest skiagram on recovery, while 3 had blunted costophrenic angle. All 27 patients showed normal lung functions on spirometry. All patients were HIV negative.

Discussion

Although usually considered a chronic illness, one third of patients with tuberculous pleuritis have an acute illness of less than 1 week duration, and two-third seek medical attention within 1 month of the time of onset of symptoms [3]. All 35 patients of this series presented acutely but histologic or bacteriologic confirmation of tuberculosis was lacking in most of them,

thus suggesting a non-tubercular aetiology.

In tuberculous pleural effusion, a smear of the fluid is usually negative for tubercle bacilli, but cultures are positive in more than half the cases. High rate of diagnosis, around 80%, can be achieved by combining histologic examination and culture of pleural biopsy with study of fluid [4]. In present series only 2 patients showed positive histological lesion suggestive of tuberculosis of whom only 1 was confirmed by culture. Rest of the biopsies revealed non-specific chronic inflammatory reaction.

A pleural effusion ADA greater than 70 IU/l has shown a sensitivity of 98% and specificity of 96% for the diagnosis of pleural tuberculosis [5]. Increased levels of ADA have been found in non-tubercular effusions, and histologic or bacteriologic confirmation of tuberculosis remains a necessity. All the patients in present series had ADA more than 40 IU/l. 12 of 20 had levels above 70 IU/l. ADA alone can not confirm a diagnosis of tuberculous pleural effusion.

Polymerase chain reaction to tuberculosis although rapid, sensitive and specific diagnostic test is at present only a valuable adjunct to diagnosis in pleural effusion [6]. None of the 10 samples tested positive for PCR. All 10 patients did not show any evidence of tuberculosis and were thus retained in service.

65% of patients with tuberculous pleuritis in whom the effusion resolves spontaneously, symptomatic parenchymal disease usually occurs within 12 months [7]. None of our patients reported with any manifestation of tuberculosis during 2 year follow up.

Viral infections probably account for a larger percentage of pleural effusions than realised. The diagnosis depends on isolation of virus and demonstration of significant increase in antibody titre especially paired sera. Such viral studies could not be done due to lack of facilities. A similar interesting epidemic of viral pleural effusion at a military base in Turkey has been reported in 1955 affecting 559 individuals. The disease was self-limiting and almost all recovered within 90 days [8]. The present series involved fewer patients but was of similar background related to Armed Forces.

Few cases of pulmonary tuberculosis and pleural effusion amongst recruits from different training centres occur sporadically. Vigorous recruit training is likely to precipitate development of pleural effusion in recruits with old primary tuberculous focus acquired before training. These 35 cases were more than normal occurrence from a single training centre and that too over a short period of four months. Also such

high incidence has not been observed from the same centre subsequently. This prompted us to think of viral (non-tuberculous) etiology. An epidemic amongst 35 recruits who presented with acute self limiting exudative pleural effusion of infective origin is analysed. Although ADA levels were significantly high, histologic and bacteriologic proof for tuberculosis was lacking. Few of them were suffering from tuberculosis, but majority had viral etiology, as most of them recovered without significant pleural thickening. Overcrowding, inadequate clothing protection and vigorous exercise in cold climate could be important precipitating factors. Also none reported with parenchymal tuberculosis in two years follow up. Military personnel because of crowding and unique stress are subject to respiratory disease epidemics.

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