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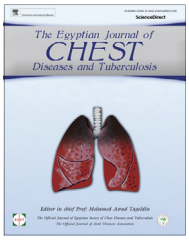

ORIGINAL ARTICLE

Role of thoracentesis in the management of tuberculous pleural effusion

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KEYWORDS

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Abstract *Background:* Tuberculous pleural effusion (TPE) is the second most common form of extrapulmonary tuberculosis (EPTB). Up to 50% after treatment complicated with pleural thickening. Pleural biopsy has been considered the gold standard in diagnosis of TPE but it is invasive, so that pleural fluid markers of TPE have been extensively evaluated as an alternative to pleural biopsy. Thoracentesis for measuring these fluid markers is important.

Aim: Assessing the value of diagnostic thoracentesis (by measuring pleural adenosine deaminase levels) and role of therapeutic thoracentesis in preventing pleural thickening.

Subjects and methods: 10 cases with transudative pleural effusion and 45 cases with already diagnosed exudative effusion (30 cases of TPE, and 15 cases of Malignant PE) were included. 50 ml pleural fluid samples were aspirated and sent for measuring ADA levels. The 30 cases of TPE were classified into 2 equal groups the 1st group started 6 months anti tuberculous therapy plus repeated thoracentesis while the 2nd started anti tuberculous therapy only. Chest CT scan was done after 2 and 6 months for assessment of pleural effusion and pleural thickening.

Results: Patients with tuberculous pleural effusion had higher pleural effusion ADA levels (mean ± SD 68.51 ± 24.06) than those with malignant pleural effusion (mean ± SD 25.47 ± 12.09) or transudative pleural effusion (mean ± SD 16.58 ± 2.93) and these levels had highly a significant difference (P-value <0.001). Also, there was a significant difference (P-value <0.05) between levels of ADA in malignant and transudative pleural effusion. Using a cut-off point of the pleural fluid ADA (30.49 IU/L) with AUC of 96.7 (sensitivity 96.7%, specificity 84%, NPV 88%, PPV 95% and accuracy 91%) discrimination between tuberculous and other causes of pleural effusion occurred. Regarding the pleural thickening, after 2 months of ttt, in group I, 3 cases developed pleural thickening, while in group II, 9 cases developed thickening. After 6 months, there was one case of pleural thickening in group I, while in group II, 5 cases developed pleural thickening. And there was a significant difference (P value <0.05) between both groups, after 2 and 6 months of treatment.

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Conclusions: Thoracentesis is very important in the diagnosis of TPE either through diagnostic thoracentesis by measuring fluid markers such as ADA or therapeutic thoracentesis which is not only important for relieving dyspnea but also in preventing occurrence of pleural thickening that complicated cases of TPE.

Introduction

Tuberculosis (TB) is a major public health problem in developing countries [1]. It is important to consider the possibility of tuberculous pleuritis in all patients with an undiagnosed pleural effusion. TB pleural effusion is the second most common form of EPTB, only less frequent than lymph node TB [2]. In contrast to pulmonary TB, most TB pleural effusions manifest as an acute illness, with approximately one third of patients being symptomatic for less than 1 week and two thirds for less than 1 month. The most common presenting symptoms are pleuritic chest pain (75%) and nonproductive cough (70%). TB pleural effusion is being increasingly recognized, even in developed nations, as the incidence of EPTB has more than doubled following the HIV pandemic [3]. Between 3% and 25% of patients with tuberculosis will have tuberculous pleuritis [3]. Pleural fluid cultures are positive for *Mycobacterium tuberculosis* in less than 40% and smears are virtually always negative. A pleural biopsy has been considered the gold standard in diagnosis of TPE but it is invasive [4]. The diagnosis cannot be established in 10–20% of the patients with these methods even in the best conditions [4]. Pleural fluid markers of TPE have been extensively evaluated as an attractive alternative to pleural biopsy.

Polymerase chain reaction (PCR) is an expensive diagnostic test. Hence many markers that may be helpful in the differential diagnosis were studied in the pleural fluid. Two of these, ADA and interferon gamma are the most widely used and currently the most accepted tests [5]. Especially ADA has been more commonly preferred for the diagnostic algorithms in the countries with a moderate to high incidence of tuberculosis because it is a more inexpensive method that can be accessed more quickly [6]. ADA is an enzyme catalyzing the conversion of adenosine and deoxyadenosine to inosine and deoxyinosine in the purine degradation pathway. Its quantity increases in the immature and non-differentiated T-lymphocytes following mitogenic and antigenic stimulation [7]. It has been estimated that up to 50% of tuberculous pleural effusions develop pleural thickening and such development can result in restrictive lung movement [8].

Aim of the work

The aim of this work was to study the role of diagnostic thoracentesis assessed by measuring pleural ADA levels in diagnosing tuberculous pleural effusion and the value of using therapeutic thoracentesis beside anti-tuberculous therapy in preventing pleural thickening associated with tuberculous pleural effusion.

Subjects and methods

During the period from March 2013 to June 2014, 55 patients with already diagnosed pleural effusion admitted to the Chest Department in Menoufiya University Hospital, Egypt, were included in the study. After the diagnosis of pleural effusion had been confirmed, thoracentesis was done. Using Light's original criteria (ratio of pleural fluid/serum protein > 0.5, ratio of pleural/serum LDH > 0.6 or pleural fluid LDH more than two-thirds of the upper limit of normal serum value), 10 patients with transudative pleural effusions were diagnosed. Of the 45 patients with exudative pleural effusion, 30 cases were tuberculous (diagnosed with Abram's needle biopsies), and 15 cases were malignant pleural effusion (diagnosed either with cytology or histopathology).

Exclusion criteria

- Tuberculous pleural effusion diagnosed with VATS.
- TB pleuritis in patients with hepatic or renal impairments.
- Presence of pleural thickening before starting anti-tuberculous treatment.

Ethical research approval from our hospital's ethics committee and informed consent from the patients were obtained.

Step 1: thoracentesis was done for the already diagnosed 45 exudative pleural effusion and 10 transudative cases and sent for measuring adenosine deaminase (ADA).

Specimen collection [9]

For each subject, at least 50 mL of pleural fluid was collected in a syringe during thoracentesis. ADA activity was measured by an auto analyzer using commercially available kits. The laboratory clinical pathologist was blinded to the diagnosis of each patient.

Step 2: Thirty tuberculous cases diagnosed with Abram's needle were classified into 2 groups; group I included 15 cases that started anti-tuberculous therapy associated with repeated therapeutic thoracentesis and group II included 15 cases that started anti TB therapy alone for 6 months. Follow up chest computed tomography (CT) scans were done following 2 and 6 months of treatment.

Radiologically: Using plain chest X rays (CXR), pleural effusions were classified into; mild (border of effusion rising till lower border of the anterior end of the 5th rib; moderate (border of effusion rising till lower border of the anterior end of the 3rd rib; and massive (border of effusion rising above the 3rd rib) [10]. Chest computed scans were done 3 times; 1st