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ORIGINAL ARTICLE

The diagnostic value of adenosine deaminase activity in pulmonary tuberculosis: Comparison between sputum and serum

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KEYWORDS

Pulmonary tuberculosis;
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Abstract *Background:* Tuberculosis is a chronic specific bacterial infection caused by bacteria of the Mycobacterium tuberculosis. It remains one of the deadliest diseases in the world. It is the second leading infectious cause of death after HIV infection. Adenosine deaminase (ADA) activity is increased in various conditions such as liver disease, tuberculosis, typhoid, infective mononucleosis and certain malignancies, especially those of haematopoietic origin.

Objective: The aim of the work was to assess the diagnostic value of ADA in pulmonary tuberculosis by comparing levels in sputum and serum.

Subjects and methods: The present study included 15 patients with active pulmonary tuberculosis, 15 patients with pneumonia and 15 patients diagnosed as lung cancer. All patients were subjected to: Full history taking, complete clinical examination, laboratory investigations, and plain X-ray chest. Measurement of sputum and serum level of ADA of all patients using specific immunoassay method.

Results: There was a significant increase in sputum ADA in tuberculous group in relation to the other groups (mean sputum ADA was 159.76 ± 16.95, 84.34 ± 6.87 and 67.30 ± 7.47 U/L, respectively). The mean serum ADA was 31.99 ± 8.85, 24.15 ± 4.22and 14.84 ± 2.43 U/L in tuberculous group, pneumonia group and bronchogenic carcinoma group, respectively. There was a statistically significant increase in serum ADA in the tuberculous group in relation to the other groups (F 10.65, P 0.001*). There was a statistically significant positive correlation between sputum and serum ADA ($r = 0.75$, $P = 0.0001$).

Conclusion: The results support the feasibility of using sputum ADA for diagnosis of pulmonary tuberculosis.

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Introduction

Tuberculosis (TB) is a chronic specific bacterial infection caused by bacteria of the Mycobacterium tuberculosis [1].

TB remains one of the deadliest diseases in the world. It is the second leading infectious cause of death after Human Immunodeficiency Virus (HIV) infection [2]. It is an ancient disease with the evidence of the organism being present in skeletons over 4000 years ago [3]. The earliest records that are consistent with tuberculosis are the Egyptian wall paintings that described the typical hunchback deformities and correlate with the findings of spinal tuberculosis in mummies [4,5].

Adenosine deaminase (ADA) is an enzyme involved in purine catabolism, the enzyme catalyzes the hydrolytic and irreversible deamination of adenosine to inosine and deoxyadenosine to deoxyinosine [5].

ADA has been extensively used in the diagnosis of tuberculous pleural effusion, two isoenzymes, ADA1 and ADA2, have been described [6].

In humans two different isozymes are encoded by different genes, ADA1 is a single-chain Zn-binding protein and almost all activities are attributed to this protein. ADA2 is believed to be produced by monocytes and is found in negligible quantities. Mutations in the ADA1 gene, where expression is blocked, cause immunodeficiency, whereas mutations that cause overexpression cause hemolytic anemia [5].

Few studies have investigated the use of ADA activity in material other than pleural fluid. There are conflicting data regarding the use of ADA activity in bronchoalveolar lavage (BAL) as a diagnostic tool in pulmonary TB. Moreover, patients may have relative contraindications that make bronchoscopy difficult to carry out, whereas sputum is easily obtainable. However, sputum may be AFB-negative even in the presence of the disease [6].

Subjects and methods

The study included 45 patients divided into three groups:

Group I: 15 patients diagnosed as active pulmonary tuberculosis. All patients had symptoms and signs of active pulmonary tuberculosis, positive sputum smear for acid fast bacilli, with X-ray findings consistent with active pulmonary tuberculosis.

Group II: 15 patients diagnosed as pneumonia. All patients had symptoms and signs of pneumonia, with X-ray findings consistent with pneumonia and consolidation.

Group III: 15 patients diagnosed as bronchogenic carcinoma. All patients had symptoms and signs of bronchogenic carcinoma, with X-ray, CT and histopathological findings consistent with bronchogenic carcinoma.

After taking an informed consent, all subjects were subjected to detailed history taking including symptoms, complete clinical examination including general and local chest examinations, anthropometric measurements, routine laboratory investigations: including complete Blood count (CBC), liver and renal function tests, erythrocyte sedimentation rate (ESR) first and second hours and chest X-ray (standard postero-anterior (PA) chest radiographs).

Sputum smears for acid-fast bacilli: Early morning expectorated sputum specimens obtained after a deep, productive cough were collected on three days from each patient in a clean tightly closed plastic disposable container properly labeled and stained with Ziehl–Neelsen stain, quantitation scale for acid fast bacillus smears was done [7].

Measurement of serum and sputum ADA in all patients [8]: Early morning sputum samples and 5 cc venous blood samples were obtained from all patients.

Blood samples were centrifuged at 3500 rpm for 10 min to separate the sera. Sputum samples were homogenized with 70 milli-mol phosphate buffer (pH: 6.0) containing 0.5 mol NaCl (1 ml sputum + 5 ml buffer). They were centrifuged at 5000 rpm for 30 min and ADA activity of the supernatant was measured by Gusti method. Results were corrected by multiplying with the dilution coefficient.

We used Human Adenosine Deaminase ELISA kit with Catalog Number: MBS733123.

All reagents stored at 2–8 °C with valid period: six months for samples like: Cell culture fluid & body fluid & tissue homogenate Serum or blood plasma and for 96 tests.

Reagents handling: ADA reagent came in a liquid two reagent system ready to use for both manual and automated analyzer method. ADA controls and calibrator are in lyophilized form, and need to be reconstituted with 1.0 of DI water before use.

Assay procedure: ADA kit was used on automated clinical chemistry analyzers.

Calibration: 0.9% saline and ADA calibrator were needed for calibration. *Results:* The ADA results were printed out in U/L.

Results

The demographic data of the three studied groups as regards age and sex were shown in Table 1.

In tuberculous patients sputum ADA ranged between 125.4 and 180.3 U/L with a mean value of 159.76 ± 16.95 U/L, in pneumonia patients ranged between 72.5 and 95.3 U/L with a mean value of 84.34 ± 6.87 U/L and in bronchogenic carcinoma patients ranged between 51.8 and 83.2 U/L with a mean value of 67.30 ± 7.47 U/L. There was statistically significant difference between the three studied groups regarding sputum ADA ($F = 25.65$, $P = 0.0001$).

There was a significant increase in sputum ADA in group I than group II and group III and there was significant increase in sputum ADA in group II than group III ($F = 25.65$, $P1 = 0.0001$, $P2 = 0.0001$, $P3 = 0.0001$ respectively) Table 2.

Serum ADA in group I ranged between 17.7 and 54.1 U/L with a mean value of 31.99 ± 8.85 IU/L, in group II ranged between 18.3 and 29.8 U/L with a mean value of 24.15 ± 4.22 U/L and in group III ranged between 11.3 and 19.2 U/L with a mean value of 14.84 ± 2.43 U/L. There was a statistically significant difference between the three studied groups regarding serum ADA ($F = 10.65$, $P = 0.001$).

There was a significant increase in serum ADA in group I than group II ($P < 0.01$), also there was a significant increase in serum ADA in group I than group III ($P < 0.001$), Group II serum ADA was significantly increased than group III ($F = 10.65$, $P1 = 0.0022$, $P2 = 0.0001$, $P3 = 0.0001$, respectively) Table 2.

ADA sputum/serum ratio, in group I ranged between 3.30 and 9.25 with mean value of 5.31 ± 1.47, in group II ranged between 2.99 and 4.84 with mean value of 3.59 ± 0.67 and

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