Study of Serum Adenosine deaminase (ADA) level use as a prognostic and diagnostic tool for various Body Fluids analysis at M.Y.H. Indore

Authors
Prof. Dr C.V. Kulkarni (Prof. & Head), Dr Khushboo Likhar (Senior Resident)*
Dept. of Pathology, M.G.M. Medical College, Indore, MP, India
*Corresponding Author
Dr Khushboo Likhar
Email: drkhushboo12@gmail.com

Abstract
Aim: Improve the survival rate of patients. Reduced the morbidity and mortality of tuberculosis, Reduced the incidence of tuberculosis induced acute illness and prevent the complication.
Materials and Methods: The present study was done at M.Y. Hospital, Indore. In our study, case group included 71 cases attending OPD.
Results: In our study, ADA level is positive in 31 cases in pulmonary tuberculosis, in 14 cases in lung cancer and in 26 cases in pneumonia.
Conclusion: Adenosine deaminase level in body fluids is a good indicator of TB. It can be performed with minimum time, cost and equipments.
Keywords: Adenosine Deaminase, body fluid.

Introduction
Adenosine deaminase is a purine catabolic enzyme, competent of catalyzing the deamination of adenosine, forming inosine in the result process.¹ It is widely distributed in tissues and body fluids. The most important biological activity of ADA is related to lymphoid tissue and is necessary for proliferation and differentiation of T lymphocytes as well as for the maturation and function of blood monocytes and macrophages. The activity of ADA is ten times greater in lymphocytic cells than in erythrocytes and, in relation, ADA level is greater in T lymphocytes than in B lymphocytes and varies during T-cell differentiation, with significant increases of its level in immature or undifferentiated states.² The assay of ADA activity in the serum and other biologic fluids is very important for a precise diagnosis of many pathological situations. In this respect ADA has been shown to increase in several inflammatory conditions such as rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), pancreatic disorders, acute appendicitis, celiac disease (CD) and tuberculosis.³
Adenosine deaminase, ADA, is an enzyme tithe purine salvage pathway which catalyzes the irreversible deamination of adenosine into inosine. Its main biological role is related to proliferation and differentiation of lymphocytes. Specific
activity of this enzyme is higher in T-lymphocytes than in B-lymphocytes, being inversely correlated to the degree of T-cell differentiation. In recent years clinical interest in this enzyme has been focused on immunodeficiencies, a hereditary deficiency of ADA being associated with defective cellular and humoral immunity. Furthermore, increased serum ADA catalytic concentrations have been found in diseases where a cell mediated immune-response is involved.  

**Aims & Objectives**

Improve the survival rate of patients. Reduced the morbidity and mortality of tuberculosis. Reduced the incidence of tuberculosis induced acute illness and prevent the complication..

**Material & Method**

The present study was done at M.Y. Hospital, Indore. In our study, case group included 71 cases attending OPD.

<table>
<thead>
<tr>
<th></th>
<th>15.5 IU/L</th>
<th>Mean IU/L</th>
<th>Range IU/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td>31 (77.5)</td>
<td>24</td>
<td>9 - 59</td>
</tr>
<tr>
<td>Negative</td>
<td>9 (22.5)</td>
<td>14.9</td>
<td>5 - 56</td>
</tr>
</tbody>
</table>

In our study, ADA level is positive in 31 cases in pulmonary tuberculosis, in 14 cases in lung cancer and in 26 cases in pneumonia.

**Discussion**

In Rasolinejad’s study,\(^5\) serum ADA level was 21.51 in pulmonary TB patients and 11.47 in healthy people; in cut-off point of 14.5 U/L, sensitivity and specificity were 82% and 80.6%, respectively. In Lakshmi *et al.*’s study,\(^6\) average ADA level was 13.3 U/L in negative smear and negative tuberculin patients, 33.52 (±15.22) in smear-positive and purified protein derivative (PPD) positive patients, and 16.5 (±3.18) in volunteer healthy people. Such differences may be due to TB severity, age groups, genetic differences, and dissimilarities in control groups. Therefore, further studies for identifying normal ADA levels in different societies may be useful. Fortunately, in some autoimmune patients like rheumatoid arthritis, synovial ADA level is normal and it is similar to control group. Thus, in autoimmune diseases that involve lung, ADA level could be used for TB differentiation. In some studies, ADA2 was also considered a useful tool for diagnosis; it needs further studies.

**Results**

In Rasolinejad’s study,\(^5\) serum ADA level was 21.51 in pulmonary TB patients and 11.47 in healthy people; in cut-off point of 14.5 U/L, sensitivity and specificity were 82% and 80.6%, respectively. In Lakshmi *et al.*’s study,\(^6\) average ADA level was 13.3 U/L in negative smear and negative tuberculin patients, 33.52 (±15.22) in smear-positive and purified protein derivative (PPD) positive patients, and 16.5 (±3.18) in volunteer healthy people. Such differences may be due to TB severity, age groups, genetic differences, and dissimilarities in control groups. Therefore, further studies for identifying normal ADA levels in different societies may be useful. Fortunately, in some autoimmune patients like rheumatoid arthritis, synovial ADA level is normal and it is similar to control group. Thus, in autoimmune diseases that involve lung, ADA level could be used for TB differentiation. In some studies, ADA2 was also considered a useful tool for diagnosis; it needs further studies.

**Conclusion**

Adenosine deaminase level in body fluids is a good indicator of TB. It can be performed with minimum time, cost and equipments. High ADA value efficiently differentiates TB from non-TB cases. Although for achieving best clinical outcome ADA values should be carefully correlated with the clinical and other biochemical parameters.

**References**

