Study of Serum Adenosine deaminase (ADA) level use as a prognostic and follow up survival tool for Connective tissue diseases (CTD) at M.Y.H. Indore

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ABSTRACT

Aims & Objectives: Connective tissue diseases (CTD) are a group of autoimmune disorders which are characterized by raised presence of Adenosine deaminase enzyme (ADA) in the blood of patients. ADA are Adenosine deaminase is a purine catabolic enzyme that have the ability of binding and destroying certain structures within the nucleus of the cells [1]. ADA involved in the pathogenesis of disease and basis for CTD workup.

ADAs are related to diseases like systemic lupus erythematosus (SLE), rheumatoid arthritis, thyroid disease, antiphospholipid syndrome, discoid lupus, juvenile idiopathic arthritis, psoriatic arthritis, juvenile dermatomyositis, idiopathic thrombocytopenic purpura, some infection even in some cancer. Improve the survival rate of patients. Reduced the morbidity and mortality of, reduced the Connective tissue diseases (CTD) incidence of disease induced acute illness and prevent the complication.

Keywords: NIL

INTRODUCTION

Adenosine deaminase is a purine catabolic enzyme, competent of catalyzing the deamination of adenosine, forming inosine in the result process. It is widely distributed in tissues. 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. The assay of ADA activity in the serum and tissues is very important for a precise prognostic of many pathological situations. In this respect ADA has been shown to increase in several inflammatory conditions such as like systemic lupus erythematosus (SLE), rheumatoid arthritis, thyroid disease, antiphospholipid syndrome, discoid lupus, juvenile idiopathic arthritis, psoriatic arthritis, juvenile dermatomyositis, idiopathic thrombocytopenic purpura, some infection even in some cancer 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.  

**Material & Method**

It was a retrospective and prospective, Observational study, which is conducted in M.Y. Hospital and associated other Indore. In our study, case group included all age group and both sex who were admitted and OPD based both type include in our hospital.

Inclusion criteria for patients were: complain for connective tissue disorder like body rash , myelgia with unknow cause and ana positive. Then samples were centrifuged and serum ADA levels were measured by ADA kit. In the first step, adenosine was affected by ADA and it becomes de-ammonized and shifted to inosine and then ammoniac was released. In the second reaction, because of glometat, released NH had become dehydrogenized and when it got close to allosteric activators, it combined with Nicotinamide adenine dinucleotide phosphate Hydrogen (NADPH) and released Nicotinamide adenine dinucleotide phosphate (NADP).

Consequently, there was a direct relationship between activity (density) of ADA enzyme and speed reduction of radiation absorption in 340 nanometer wavelength (as NADPH changed to NADP+);

**Results**

<table>
<thead>
<tr>
<th></th>
<th>Positive</th>
<th>Negative</th>
<th>Mean IU/L</th>
<th>Range IU/L</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Connective tissue disorder</strong></td>
<td>40</td>
<td>10</td>
<td><strong>21</strong></td>
<td>10- 49</td>
</tr>
<tr>
<td><strong>Inflammatory disorder</strong></td>
<td>36</td>
<td>18</td>
<td><strong>12.3</strong></td>
<td>12 - 51</td>
</tr>
<tr>
<td><strong>Neoplasm</strong></td>
<td>24)</td>
<td>12</td>
<td><strong>17.5</strong></td>
<td>11-49</td>
</tr>
<tr>
<td><strong>Degenerative disorder</strong></td>
<td>03</td>
<td>30</td>
<td><strong>11.2</strong></td>
<td>7 - 15</td>
</tr>
</tbody>
</table>

**Discussion**

In Sharma et al.'s study, sensitivity, specificity, positive predictive value, and negative predictive value were 92.8%, 90%, 92.8%, and 90%, respectively, for prognosis of connective tissue disorder in with an ADA level of more than 49. In Bett et al study, serum ADA level was more than 49 in connective disorder patients and 11.45 in healthy people; in cut-off point of 10.2 U/L, sensitivity and specificity were 82% and 80.6%, respectively. In Lakshmi et al.'s study, average ADA level was 13.3 U/ l. Such differences may be due to CTD 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;[8] it needs further studies.

**Conclusion:** Adenosine deaminase level in raised in serum and tissue in various connective tissue disorder. This tool is used as a prognostic and follow-up survival tool for connective tissue disorders. This is also as a severity indicator of connective tissue disorder. It can be performed with minimum time, cost, and equipments. High ADA value efficiently differentiates connective tissue disorder from non-TB cases. Although for achieving best clinical outcome ADA values should be carefully correlated with the clinical and other biochemical parameters.

**References**


