

Effect of Adenosine Deaminase in Diabetes Patients With Complications

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Abstract: Diabetes mellitus is a complex syndrome characterized by hyperglycemia, leading to vascular complications such as retinopathy, neuropathy and macro vascular disease like atherosclerosis. Patients with diabetes with complications and without complications are taken up for the study. The parameter serum Adenosine Deaminase, Activity (ADA) is considered which is helpful in understanding the predisposing factors and the assessment of these patients to develop complications. The present study reveals that serum ADA is found to be raised in diabetic patients without complications while it is similar to control group with complications. This indicates that increased susceptibility of diabetes to develop a variety of bacterial and fungal infection may not be due to immune deficiency.

Key Words: Serum Adenosine Deaminase Activity (ADA), Diabetic

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I. Introduction

Adenosine deaminase in lymphoid tissue might efficiently deaminase deoxy adenosine and prevents phosphorylation. Galanti and Giusti Altivita (1968) and Gold Berg and Elis (1976) observed that Human serum ADA increases in acute viral hepatitis and active cirrhosis and only to a much lesser extent in other hepatic disease. Goldblum et al., (1978) demonstrates that increased serum ADA has been found in leukaemic patients and lymphocyte ADA levels can be considered a parameter of immune response Lymphocyte ADA activity is decreased in erythrocytes from patients with severe combined immunodeficiency, while heterozygous carrier of this autosomal recessive defect have half the normal enzyme activity.

Alan Taylor (1986) observed an increase in serum ADA activity in 18 untreated patients with active sarcoidosis and suggested with some reservation that its measurements might be useful for diagnosis of sarcoidosis. Singh et al., (1981) reveals that the estimation of ADA activity will be of value in the diagnosis of tuberculous effusions. The high ADA activity in tuberculous effusions could be attributed to cell mediated immune reactions or to increased demands for energy. This high level may be useful in the pleural differential diagnosis of tuberculous from other pleural effusions. Delias et al., (1987) studied ADA activity in acquired Immunodeficiency syndrome and reveals high ADA activity in these subjects. ADA activity in lymphocytes and erythrocytes as well as in serum, is absent in about 20 -30% of children affected by a severe inherited T cell immune deficiency. Yasuhera and Nakamera (1987) determined the activity of serum ADA in patients who had various types of pneumonia or pulmonary tuberculosis. ADA activity in children with bacterial pneumonia showed a higher value than those of viral and mycoplasma pneumonia but a lower value than that of tuberculosis. The peak ADA activity was found on 5th or 6th disease day in bacterial pneumonia. The number of lymphocytes is predominant over that of neutrophils at this period Serum ADA in tuberculosis showed highest concentration than that of pneumonia. Increased serum ADA in tuberculosis seems to be influenced by activated T lymphocytes.

II. Materials And Methods

The study was carried out in 25 normal adult patients between the age group of 30-55 years from outpatient & inpatient department of OHRC & Princess Esra Hospital, Hyderabad, these patients show no family history of diabetes they did not suffer from any complication.

(1) Estimation of Glucose

Method – Glucose Oxide – Peroxidase Method:

(2) Serum Adenosine Deaminase Activity

Principle

Adenosine deaminase hydrolyses adenosine to ammonia and inosine. The ammonia formed further reacts with a phenol and hypochlorite in an alkaline medium to form blue indophenols complex with sodium nitroprusside acting as a catalyst. Intensity of the blue coloured indophenols complex formed is directly proportional to the amount of ADA present in the sample.

Adenosine+ ADA ammonia + inosine
 Ammonia + Pheno + Hypochlorite
 Alkaline Medium Blue indophenols complex

(3) Estimation of Creatinine
 Jaffe's, method

Table-I Fasting Plasma Glucose (mg/dl)

S. No.	Group-I Normal Control	Group-II NIDDM without complication	Group-III NIDDM with complication
Mean	72.45	159.00	212.64
SD	8.662	43.865	74.938
SE	1.847	9.352	15.977

Plasma Glucose

Source	DF	Sum of square	Mean of square	F-Ration	Significance
Between group	2	127015.23	6250761.50	51.43	P<0.01
Within group	57	70382.00	123477.20		

Table-2 Serum Creatinine

S. No.	Group-I Normal Control	Group-II NIDDM without complication	Group-III NIDDM with complication
Mean	0.753	1.318	2.818
SD	0.1328	0.3661	1.9340
SE	0.0322	0.0888	0.4691

Serum Creatinine

Source	DF	Sum of square	Mean of square	F-Ration	Significance
Between group	2	42.18	21.09	15.74	<0.01
Within group	48	64.62	1.34		

Table-3 Adenosine Deaminase Levels in various study groups

S. No.	Group-I Normal Control	Group-II NIDDM without complication	Group-III NIDDM with complication
Mean	13.800	14.850	13.600
SD	1.5424	2.7198	2.1126
SE	0.3449	0.6082	0.4724

Adenosine Deaminase

Source	DF	Sum of square	Mean of square	F-Ration	Significance
Between group	2	17.73	8.865	2.94	N.S
Within group	57	-171.7	.3.01		
Total	59				

III. Results And Discussion

Results of study indicate that predisposition of diabetes to develop complications such a retinopathy nephropathy and the predisposition to infections in multi factorial.

Fasting blood sugar levels are found to be raised in all patients who have already developed micro vascular complications control (72.45 ± 8.662) NIDDM without complications (159.00±43.865) NIDDM with complications (212.64±74.938) (Table3).

Serum creatinine is raised in chronic diabetic patients who already developed nephropathy (Table 1). In these patients the blood glucose is also raised (Table 3). Different clinical and biochemical studies also show that occurrence of diabetic complications is more in patients with poor glycemic control.

Serum ADA activity is normal in diabetic with retinopathy and nephropathy however the enzyme activity was found to be slightly higher in diabetics without these complications (Table 2). The present study suggest that degree of hyperglycemia related to ADA increased adenosine deaminase level reflecting increased 'T' cell function in diabetics without complications as retinopathy or nephropathy may be due to autoimmune reaction against modification glycated proteins. Serum ADA levels is found to be raised in diabetic patients

without complications while it was similar to control group with above complications this indicate that increased susceptibility of diabetics to develop a variety of bacterial and fungal infection may not be due to immune deficiency (Table 2). It has been conclusively shown that strict control of blood sugar levels reduces the risk of developing complications like neuropathy retinopathy and prevention of CAD.

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