

Estimation of Serum Adenosine Deaminase Enzyme: A New Tool for Diagnosis of Acute Upper Respiratory Tract Infection (URI)

¹Kumar Ashish, ¹K.L. Tiwari, ¹S.K. Jadhav and ²P.K. Patra

¹School of Studies in Biotechnology,
Pt. Ravishankar Shukla University, Raipur, Chhattisgarh 492 001 India

²Department of Medical Biotechnology and Biochemistry Pt. Jawaharlal Nehru
Memorial Medical College, Raipur Chhattisgarh 492 001, India

Abstract: The purpose of this study was to evaluate the usefulness of a new parameter to identify acute upper respiratory tract diseases in different age and sex group. Adenosine deaminase levels were estimated in 150 URI (Upper respiratory tract infection) patients and 50 controls by using Galanti method. The mean serum adenosine deaminase level in control group was 9.93 ± 2.26 U/L in males and 10.38 ± 1.81 U/L in females. The mean serum adenosine deaminase level in male study group was 32.79 ± 10.59 U/L and 32.38 ± 10.18 U/L in females this value was significantly higher than the corresponding value obtained in the control group. Duration of infection was studied and mean serum adenosine deaminase level was 30.48 U/L in patients with less than 15 days and 34.12 U/L found where total duration of illness was more than 15 days. There was no variation in the serum adenosine deaminase level of URI patients belonging to various age groups and either sex.

Key words: Uri • Adenosine deaminase enzyme • Blood • Serum

INTRODUCTION

An upper respiratory tract infection is a worldwide public health problem it affects the people all equally irrespective of age and sex. According to WHO estimates respiratory infections causes about 987000 deaths in India of which 969000 were due to acute lower respiratory infections, 10,000 were due to acute upper respiratory infections (URI). [WHO (2006) world health report 2006 reports of director general WHO]. Adenosine deaminase enzyme (ADA) involves in purine salvage pathway and catalyses the deamination of adenosine to inosine and ammonia. Deficiency of adenosine deaminase leads to the accumulation of deoxyadenosine triphosphate, which is lymphocytotoxic because of its ability to inhibit DNA synthesis via inhibition of ribonucleotide reductase [1]. ADA level was found increased in cases of inflammatory activity only because lesion leads to mucosal damage inflammation leads to reactive oxygen species, which may increase the extent of inflammation during respiratory burst. Adenosine is an important endogenous regulator of neutrophils functioning is

released intracellular and modulates neutrophils activity by interacting with specific adenosine surface receptors where functions are characterized in neutrophils.

Normal steady state of the passage of the enzyme from cells to extra cellular fluid will be altered if there is obstruction to a normal pathway of enzyme secretion or excretion or if there is a change in the cell permeability [2]. An increase in adenosine activity suggests alteration in adenosine control mechanism in respiratory infections upper respiratory tract infections these includes common cold, influenza, sore throat, ferengitis and tonsillitis etc. Any damage to cell, which causes an increase in the permeability of cell membrane even without actual necrosis, will allow enzymes to escape at a greater rate. The serum enzyme concentration represents a balance between leakage of enzyme from the damaged cells and loss of enzyme from the plasma into general extra cellular fluid to catabolism or excretion. Rises in serum enzyme activity generally reflects an increased rate of cell damage rather than the total extent of cell damage [3, 4].

Corresponding Author: Kumar Ashish, School of Studies in Biotechnology, Pt. Ravishankar Shukla University, Raipur, Chhattisgarh, 492 001 India. Tel: +919493865018.

MATERIALS AND METHODS

The study groups comprised 150 cases admitted in the Dept. of ENT, Dr. B. R. Ambedkar Memorial Hospital and Pt. Jawaharlal Nehru Memorial Medical College, Raipur C.G., India which were suffering from upper respiratory tract infections, which included 94 males and 56 females. The control group consisted of 50 individuals, which included 27 males and 23 females. All the study and control individuals were divided into age group of < 10 years, 11-20 years, 21-30 years, 31-40 years, 41-50 years and 51-60 years. All possible technique was used to collect the 3 ml blood sample in sterile dry plain vial after which the blood is centrifuged for 15-20 minutes at 5,000 rpm then supernatant (serum) was taken and stored at -20°C for further study. Investigation was performed by method of [3] in parallel four eppendorf tubes for each 25 serum sample i. e. reagent blank, standard and sample blank and sample. Reagent blank contained 1.0 ml (50 mM, pH 6.5) phosphate buffer sol. 0.05 ml distilled water, standard contain only 1.0 ml (75mM) ammonium sulphate standard solution. and 0.05 ml distilled water, sample blank contained only 1.0 ml (21mM, pH 6.5) of buffered adenosine solution, sample contained 1.0 ml (21mM, pH 6.5) buffered adenosine solution. and 0.05 ml serum sample than all the tubes were incubated for 60 minutes in a water bath at 37 °C, further 3-3 ml (106 mM) of phenol/ nitroprusside solution (106mM) and alkaline hypochloride solution (11 mM) was added vigorously, only 0.05 ml serum sample was added in blank tube. Again all four tubes were incubated for 30 minutes in a water bath at 37°C estimation were measured against distilled water and volume activity was performed in following formula.

Volume Activity = (Estimation of sample - Estimation of sample blank)/ (Estimation of standard - Estimation of reagent blank) X 50 (U/L)

RESULTS AND DISCUSSIONS

Age Wise Distributions: Among control group - The cases was distributed in 6 age groups, maximum no. of subjects were in the age group 31 - 40 (32%) followed by age group of 21 - 30 years (20%) and least no. of cases belongs to age group below 10 and 41 - 50 (10%).

Among Study Group: Maximum no. of cases were found in age group of 11-20 years and 31-40 years i.e. 30 cases (20%) and least no. of cases belongs to age group

Table 1: Table showing mean serum ADA levels in study group (sex wise)

S.No.	Sex	No. of Cases	Serum ADA levels		
			Range U/L	Mean U/L	S.D.
1.	Male	94	22.2-42.68	32.79	5.393
2.	Female	56	22.2-42.68	32.38	5.238
3.	Total	150	22.2-42.68	32.58	5.350

< 10 years i.e. 14 cases (9.4%), while in the age group 21-30 year and 41-50 years no. of cases found equal i.e. 29 cases (19.3%).

Sex Wise Distribution: Among control group - Out of 50 patients 27 (54%) males and 23 (46%) females, thus the male and female ratio was 1:09. In our study no. of age variation in male mean serum ADA level was 9.93 ± 2.26 U/L and in female it was 10.38 ± 1.81 U/L. There was significant difference i.e. 0.45 U/L in serum ADA levels in relation to sex, which was insignificant statistically. These findings are similar to those reported by [5-8] in which mean serum ADA level was regularly 8.76 ± 2.90 U/L, 9.7 ± 0.53 U/L, 9.7 ± 0.53 U/L, 10.9 ± 2.99 U/L.

Among Study Group: Out of 150 patient studied male mean serum ADA level was 32.79 ± 10.59 U/L male and in female mean serum ADA level was 32.38 ± 10.18 U/L (Table 1). There was slight increase mean serum ADA level in males, which was insignificant statistically. This was also observed as a study of [8, 9] that mean serum ADA activity, where area of mucous membrane involvement was more i.e. in pulmonary tuberculosis it was high (39.97 ± 2.24 U/L) than in non tubercular localized cases where area of mucous membrane involved was less.

According Effect of Duration of Illness: In our study groups 87 patients (58%) had symptom for less than 15 days, 63 patient (42%) had symptom for more than 15 days and mean ADA level was 30.48 U/L in less than 15 days and 34.12 U/L in more than 15 days. Significantly higher serum ADA level was observed where totals duration of illness prolonged to more than 15 days. This study was same as reported by [10].

CONCLUSION

The mean serum ADA level in study group was 32.58 ± 10.38 U/L these values are significantly higher then the corresponding value $10-15 \pm 2.03$ U/L obtained in the control group. It was observed that there were no

variations in cases belonging to various age groups and either sex. Our study suggested that serum ADA concentration are generally increased in URI patients than those in healthy control due to the stimulation of cell mediated immune response which may be the diagnostic tools to identify acute upper respiratory tract infections.

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