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Original Research Article

A STUDY ON SERUM ADENOSINE DEAMINASE AS AN INDICATOR OF GLYCAEMIC STATUS IN TYPE 2 DIABETES MELLITUS

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ABSTRACT

Background: Type 2 diabetes mellitus is a common metabolic disorder characterized by high blood glucose levels. Adenosine Deaminase (ADA) is an enzyme which catalyses the irreversible deamination of adenosine to inosine. Adenosine is known to exert potent metabolic effects acting through its receptors. Increased levels of serum ADA has been shown in individuals with type 2 diabetes mellitus. The study aims to understand the relationship between serum ADA and blood glucose levels. The objective is to estimate the value of serum adenosine deaminase (ADA) in patients with uncomplicated cases of type 2 diabetes mellitus and non diabetics control group. To estimate the association between serum ADA values among cases and control based on blood sugars.

Materials and Methods: The study was conducted at Gandhi Medical college, Secunderabad. It involved 50 cases of uncomplicated type 2 diabetes mellitus and 50 age and sex matched healthy controls. Blood and urine samples were collected after obtaining an informed written consent from cases and controls. Serum Adenosine Deaminase levels were estimated colourimetrically, based on the method described by Giusti and Galanti. Serum FBS, PPBS, HbA1c levels, blood urea, serum Creatinine, lipid profile and urinary sugar and proteins were also measured simultaneously using routine laboratory methods.

Results: A clear-cut elevation of serum ADA was found in diabetic subjects as compared to controls. In addition a very large positive correlation was found between serum ADA and blood glucose levels.

Conclusion: This study shows ADA level is significantly related to the glycemic status in type 2 diabetes mellitus. Altered serum ADA level is an indirect expression of the tissue adenosine levels. Adenosine through its multiple metabolic effects is involved in the pathophysiology of diabetes. The present study highlights the importance of this metabolite and the need for further studies.

Keywords: Type 2 diabetes mellitus; Adenosine Deaminase; Adenosine.

INTRODUCTION

The prevalence of type 2 diabetes mellitus and obesity is increasing rapidly. In 2014, 422 million people worldwide were estimated to have diabetes and by 2035 the prevalence of diabetes is estimated to increase to 592 million people (World Health Organization; WHO). Regarding overweight and obesity, 1.6 billion adults were overweight and at least 400 million were obese in 2005, and the WHO projects that approximately 2.3 billion people will be overweight and more than 700 million people obese by 2015. Obesity is associated with increased risk of cardiovascular diseases, type2 diabetes, impaired respiratory and renal function, cancer, osteoarthritis, liver and gallbladder diseases.[1]

Type 2 Diabetes Mellitus has two hallmark features: 1) insulin resistance, defined here as an impaired ability of the hormone to suppress hepatic glucose output and to promote peripheral glucose disposal and 2) compromised function of the pancreatic ß-cell such that insulin secretion is insufficient to match the degree of insulin resistance.[2]

In addition to hyperglycemia, the type 2 diabetic individual almost invariably manifests a serious breakdown in lipid dynamics, often reflected by elevated levels of circulating free fatty acids (FFAs) and triglycerides (TG).Adenosine deaminase (ADA) is an enzyme involved in the metabolism of purine nucleosides, catalyses the irreversible hydrolytic deamination of adenosine (Ado) and 2′-deoxyadenosine (2′-dAdo) to inosine and 2′-deoxyinosine, respectively. Studies have shown that ADA which reduces adenosine levels, increases basal and noradrenaline stimulated lipolysis in adipocytes. Adenosine is an anti-lipolytic factor and lowers free fatty acid levels. Several studies have demonstrated elevated levels of adenosine deaminase in individuals with type 2 diabetes mellitus, but the exact pathogenic role of elevated ADA activity in type 2 DM remains to be elucidated. Insulin administration has been shown to reduce the elevated ADA levels in type 2 diabetics.[3] Adenosine Deaminase exerts its effects predominantly by regulating the concentration of intracellular and extracellular adenosine. Conditions which lead to elevated adenosine formation and release (e.g. hypoxia) have been shown to increase the expression of ADA.[4] Adenosine actions are multiple and it acts through its receptors following release from the cell. The A1 receptor is the only adenosine receptor expressed in the adipose tissue, and acting through this receptor adenosine exerts potent anti-lipolytic effects. In fact, A1 receptor agonists have been shown to decrease free fatty acid levels and increase insulin sensitivity.[1]

Adenosine, acting through its receptors also affects multiple tissue and organ functions including pancreas, liver, kidneys, skeletal muscle, heart, vascular tissue etc. The expression level of adenosine nucleoside transporters and adenosine receptors has been shown to be different in diabetes.

Chronic energy imbalance produces adipocyte hypertrophy and hyperplasia, endoplasmic reticulum stress and mitochondrial dysfuction.[5] The adipocytes produce larger amounts of inflammatory cytokines than normal. Immune cells are already present in the tissue in close proximity of the adipocytes and macrophages easily infiltrate adipose tissue. This inflammation is also associated with insulin resistance and type 2 diabetes. Adenosine has been shown to be a non-redundant endogenous regulator of many different functions in the immune system. Hence, the adenosine receptors can also be of importance as drug targets in the adipose tissue to suppress the underlying inflammation in obesity and thereby increase insulin sensitivity. In addition, the A2B receptor has been reported to mediate effects in the immune system of rodents that can protect against the development of type 1diabetes, which is an autoimmune disease. A2A receptor agonists have been reported to elicit wound healing and anti-inflammatory effects that can be useful for treating diabetic neuropathic foot ulcers.1 The extracellular form of Adenosine Deaminase 1 (the major form of ADA) is also called the large ADA. ADA 1 exists in serum and on extracellular surfaces as a complex: ADA+CP (CP: Complexing protein). Two molecules of ADA associate with one molecule of complexing protein. This complexing protein has been identified as Dipeptidyl Peptidase 4(DPP4) / CD 26. DPP4 has been shown to be involved in the inactivation of incretin hormones (glucose-dependent insulinotropic polypeptide (GIP) and glucagon-like peptide-1 (GLP-1). DPP4 inhibitors (e.g. sitagliptin) are being used in the treatment of diabetes. However, the importance of the interaction of human DPP4 with ADA remains incompletely understood.[6,7]

Aims And Objectives

- To estimate the value of serum adenosine deaminase (ADA) in patients with uncomplicated cases of type 2 diabetes mellitus and non diabetics control group.
- To estimate the association between serum ADA values among cases and control based on blood sugars.

MATERIALS AND METHODS

A case control study was conducted at GANDHI HOSPITAL, Secunderabad, from August 2016 to July 2017. Total sample of n=50 cases of uncomplicated Diabeties and n=50 as control group without diabetes.

Cases

Inclusion Criteria

Individuals with uncomplicated type 2 diabetes mellitus including both male and female, of age group 30-70 years were included in the study.

Exclusion Criteria

Individuals with diabetic complications (neuropathy / retinopathy / nephropathy / vascular complications etc.), hypertension / acute or chronic infection / any medications other than the ones used for diabetic therapy / addictive habits / pregnancy were excluded from the study.

Controls

Healthy individuals in the age group of 30-70 years, confirmed to be free of Type 2 DM / any acute or chronic disease / addictive habits / concurrent or chronic drug usage / pregnancy. The age and sex distribution of the cases and controls was similar. A thorough clinical examination and appropriate investigations were done before selecting the cases and controls for the study.

Data collection: A pre-structured and pre-tested proforma was used to collect the data. Informed consent was taken from all cases and control subjects. A detailed clinical history and physical examination and Relevant investigations (blood urea, serum creatinine, lipid profile, urine protein, ECG, Fundoscopy etc.) were done before selection of subjects for the study. After taking informed consent blood test were conducted, For blood investigations, 5 ml of blood was collected from the median cubital vein under aseptic precautions following an overnight

fast of 10 hours. Serum was separated by subjecting the clotted blood was to centrifugation at 3000 rpm for 10 minutes. 2 mL of blood was transferred to an EDTA tube for estimation of HbA1c. For estimating PPBS, blood was drawn 2 hours after breakfast.Corresponding urine samples were analyzed by dipstick method.

Estimation of serum Adenosine Deaminase [Colourimetric method of Giusti & Galanti]30, The estimations were done using the ADA-MTB kit from Microexpress, a division of Tulip Diagnostics (P) Ltd.

Procedure: The immunoassay method used in this study is: Tina-quant HbA1c Gen.2, on Cobas Integra 400, Roche Diagnostics. Anticoagulated whole blood is lysed and total haemoglobin and HbA1c are estimated in the same sample. The determination of HbA1c is based on the turbidimetric inhibition immunoassay principle. Monoclonal antibodies against the first three N-terminal amino acids and the attached glucose moiety, react with the HbA1c in the sample to form a soluble antigen- antibody complex. A polyhapten reagent containing multiple HbA1c epitopes is then added. This polyhapten reagent reacts with the excess antibodies to form an insoluble complex. The presence of HbA1c in the sample thus inhibits agglutination, resulting in lowering of turbidity. Absorbance is measured. Calibration of this assay is performed using a synthetic polypeptide comprising the N-terminal structure of HbA1c. Measurement of haemoglobin is based on the formation of alkaline haematin which is measured by colorimetric method.

The percentage of HbA1c is calculated from the immunoturbidimetric HbA1c result and the haemoglobin concentration.

Reference range

- Normal (non-diabetic) < 6%
- Near normoglycemic: 6 to 7% (therapeutic goal < 7%)
- In good control: 7 to 8%
- Actions suggested: 8 to 9%

RESULTS

The present study has been undertaken to study the levels of serum Adenosine Deaminase (ADA) in individuals with Type 2 Diabetes Mellitus, and the correlation of serum ADA with blood Glucose parameters. Among study with population n=50 controls non diabetic and n=50 cases of Type 2 diabetes. Of the cases 38% of them had diabetis history for less then 1years, followed 36% had 1-5 years, 12% had 6-10 years and 14% had >10 years of history of diabetes.

Table 1: showing descriptive data of the study population.

S.no	Based Age group in Years	Cases		Controls	
		No. of Patie NTS	Percentage (%)	No. of Patients	Percentage (%)
1	30-40	03	06%	03	06%
2	41-50	24	48%	26	52%
3	51-60	14	28%	13	26%
4	61-70	09	18%	08	16%
Total		50	100%	50	100%
Mean+ SD		51.10 + 09.21		50.54 + 08.80	
S.no	**Gender**	**Cases**		**Controls**	
1	Male	27	54%	28	56%
2	Female	23	46%	22	44%
Total		50	100%	50	100%
S.no	**BMI Distribution (Kg/M2)**	**Cases**		**Control**	
1	18.5-24.9	25	50%	26	52%
2	25 – 29.9	16	32%	17	34%
3	More than 30	9	18%	7	14%
Total		50	100%	50	100%
S. no	**ADA Done**	**Cases**		**Control**	
1	Fasting	21	42%	22	44%
2	Postprandial	29	58%	28	56%
3	Total	50	100%	50	100%

Table 2: variables showing association between cases and control among the study population.

S.NO	Variables	Cases	Controls	P-Value
1	FBS (mg/dl)	172.24 + 76.47	82.34 + 10.77	<.0001**
2	PPBS (mg/dl)	222.14 + 96.46	110.72 + 10.56	< .0001**
3	ADA	40.60 + 8.65	19.75 + 2.65	< . 0001**
4	BMI	35.415+4.96	24.765 +3.66	0.793

The above table infers that there statistical significant association of between sugar level among cases and control. Cases had raised ADA compared to the comparison group with statistical significant association.

Table 3: Pearson Correlation between ADA and Hb1A/C.

Sl. No	Variable	Mean + SD	Correlation/P-Value
1	ADA	30.17+12.26	R=0.88
2	Hb1A/C	7.94 + 2.92	P< 0.0001**

**Statistically significant.

DISCUSSION

The present study found an elevated serum Adenosine Deaminase (ADA) activity in cases of Type 2 Diabetes Mellitus when compared to the control group. The present study showed that strongly statistical association between the long term index of glycemic control viz. Glycated haemoglobin (HbA1c) and serum ADA. This finding is also agreement with some previous studies which show a significant correlation of serum ADA with HbA1c.

the mean age of study group is 51.10±09.21 and in the control group is 50.54±08.80. Mohan boro et al,[8] mean age of study group was 53.25±8.39 and control group was 51.5±6.86. Vineeth kumar et al mean age of study group was 54.52±10.66 and control group was 52.42±5.79. The Mean age of present study is correlating with these two studies.

FBS in the present study group is 172.24±76.47 and in the control group is 82.34±10.77. Vineeth kumar et al the mean FBS of study group was 167.4±60.3 and in the control group was 80.9±8.55, which is similar to the present study. Mohanboro et al,[8] the mean FBS of study group was(149.65±12.78) Amandeep kour et al the mean FBS of study group was(146.8±16.2) which is less compared to the present study.

In the present study the mean BMI in study group is 23.66±3.33 and in the control group is 24.41±3.61. Amandeep kour et al9 the mean BMI in study group was 23.68±2.9 and control group was 24.53±3.2. Vineeth kumar et al the mean BMI in study group was 22.47±1.79 and control group was 24.15±1.47, which are similar to the present group.

In the present study the mean HbA1c of study group is 7.94±2.92 and in the control group is 4.92±0.23. Vineeth kumar et al the mean HbA1c of study group was 7.88±1.08(<0.001) and in the control group was 4.82±0.43. Mohan boro et al,[8] the mean HbA1c of study group was10.47±1.48<0.0001) and in the control group was 5.12±0.39. Bhavitha et al the mean HbA1c of study group was 8.21±1.12 (<0.001)and in the control group was4.98±0.56. which are correlating with the present study.

In the present study the mean ADA in study group is 40.60±8.65 and in the control group is 19.75±2.65. Bhavitha et al the mean ADA in study group was 4o.79±18.12 (<0.01) and in the control group was19.94±7.89. Mohan boro et al8 the mean ADA in study group was 32.77±7.36 (<0.001) and in the control group was 15.06±1.21. Amandeep kour et al 9the mean ADA in study group was 37.2±5.0 (<0.001) and in the control group was 18.2±5.6. present study also show raised ADA level among diabetic compared to other studies with statistical significant association.

CONCLUSION

Serum Adenosine Deaminase (ADA) levels were evaluated in Type 2 Diabetes mellitus cases, through a Cross sectional study using age and sex matched controls.

Individuals who had developed complications of diabetes were not included in the study.

- Serum levels of ADA were found to be significantly higher in type 2 diabetics when compared to controls.
- A very large correlation was found to exist between serum ADA and blood glucose values.
- Statistical association was found between serum ADA and the long tem index of glycemic control, HbA1c.

These findings clearly project that serum Adenosine Deaminase reflects the current glycemic status in the diabetic individual, indicating a possible involvement of the enzyme substrate Adenosine, in the diabetic process.

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