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A Comparitive Study of Biochemical Profile of Type I and II Diabetes Mellitus

V. Aruna*
Assistant Professor, Department of Biochemistry, Guntur Medical College,Guntur,AP,INDIA

I. Introduction

Diabetes mellitus is a chronic metabolic disorder characterized by inappropriate hyperglycemia secondary to insulin deficiency or insulin resistance and inadequate insulin secretion[1] The clinical syndrome is characterized by impaired metabolism of carbohydrate, protein and fat caused by either lack of insulin secretion or decreased sensitivity of tissues to insulin[2]. To meet the energy requirement fatty acid breakdown increases. Mobilization of lipids from adipose tissue increases. There is resultant hyperlipidemia especially of Non-Esterfied Fatty Acids, Triacylglycerol (TAG) and Choloestrol.

Complications of Diabetes :

Irrespective of type of diabetes and pathophysiology, the complications are proportional to the severity and duration of the disease. The principal complications of DM are retinopathy, neuropathy angiopathy, nephropathy, susceptibility to infections, hyperlipidemia, ketoacidosis and hyperglycemic hyperosmolar nonketotic coma.

Glycemic control[3] :

Glycemic control is defined as maintenance of plasma blood glucoseebetwen 90-130 mg/dl before meals and after an overnight fast and 1 hr post-prandial glucose levels not more than 180 mg/dl and 2 hrs post prandial blood glucose nor higher than 150 mg/dl. Further, glycosylated hemoglobin (HbA_1c) levels higher than 7% indicate poor glycemic control.Formation of glycated hemoglobin is irreversible and blood levels depend on plasma glucose concentration.The HbA1c represents the integrated values of glucose over the preceeding 6-8 weeks and provides an additional criterion for assessing glucose control.

The amount of C-peptide indicates the amount of endogenous insulin secretion. Very low or negligible levels of C-peptide indicates IDDM. Abnormally high levels of C-peptide found in insulinoma, the insulin producing tumor.[4]

In health normal serum C-peptide level is 0.9 - 7.1 ng/ml. In a diabetic patient normal C-peptide levels indicate good response to treatment. It is a hallmark of Type –II Diabetes mellitus. Whereas in Type-I Diabetes mellitus reduced insulin production also reduces C-peptide concentration .[4]

C-peptide estimation differentiates Type-I and Type-II diabetes. Determines degree of insulin resistance.Obesity associated with high C-peptide levels predispose to diagnosis of prostatic carcinoma.

Intracellular Magnesium deficiency may be a consequence of insulin resistance.Hypomagnesemia leads to reduction of Inositol triphosphote (IP_3) diffusion into cells which subsequently leads to hypocalcemia and enhances the development of diabetic complications like retinopathy and hypertension.

II. Material And Methods

From ASRAM Hospital and Medical College 127 subjects belonging to Type I and Type II Diabetic patients in 3 groups 20 - 40 yrs, 41 – 60 yrs and above 60yrs selected for this study. 30 number of controls belonging to the same age and sex selected. Endocrine disorders like cushing's syndrome, Thyroid abnormalities and Gestational Diabetes Mellitus were excluded from the study.

Sample collection & preparation:5ml of Fasting venous blood sample collected under strict aseptic conditions from controls and test group attending ASRAM college & Hospital after obtaining informed & written consent. 1.5ml of sample mixed in EDTA for estimation of Hb A1c.3.5ml sample centrifuged to collect serum for analysis of glucose, Total cholesterol, HDL – C, Sodium , Potassium and Magnesium and C-peptide .

Biochemical analysis of samples :

C –PEPTIDE: Estimated by Direct chemiluminescence - Sandwich format immunoassay.
Glycosylated Hemoglobin(HbA1c) : Estimated by Ion exchange resin method.

Other parameters estimated using standardized kits from "Coral clinical systems". Optical density taken with photoelectric colorimeter.

GLUCOSE**:** Estimated by Glucose Oxidase Peroxidase (GOD-POD) method.

MAGNESIUM**:** Estimated by Calmagite method

CHOLESTEROL**:** Estimated by Cholesterol Oxidase-Peroxidase method.

HDL CHOLESTEROL**:** Estimated by Cholesterol Oxidase-peroxidase method after precipitating chylomicrons, LDL and VLDL.

SODIUM **:** Estimated by Uranyl acetate method.

POTASSIUM**:** Estimated by Tetraphenyl Boron method.

III. Results

Table – I A The Estimated Mean Serum Levels Of Fbs, Hb A_1c And Magnesium in Type II Diabetic Patients (NIDDM)

Parameter	FBS mg/dl	Hb A_1C %	Mg^{+2} mg/dl
20-40 yrs	143.95 ± 42.9	6.66 ±2.98	1.26 ± 0.36
41-60 yrs	147.66± 66.87	7.10 ± 3.04	1.67 ± 0.66
>60 Yrs	135.9 ± 59.94	6.95 ± 2.85	1.25 ±0.49

Table – I B The Estimated Mean Serum Levels Of Fbs, Hb A_1c Andmagnesium In Type I Deabetic Patients (Iddm)

Parameter	FBS (mg/dl)	Hb A_1c (%)	Mg^{+2} (mg/dl)
20-40 yrs	158.37 ± 35.77	8.40 ±1.72	1.34 ± 0.53
41-60 yrs	149 ± 27.81	6.93 ± 3.67	1.13 ± 0.24
>60 Yrs	183 ± 74.96	9.76 ± 1.68	1.15 ± 0.37

Table No II Estimated Mean Serum Levels Of FBS, HbA1c,Magnesium,Sodium,Potassium, C-Peptide And Cholesterol/HDLC Ratio In Controls

	FBS Mg/dl	HbA1c %	Mg++ Mg/dl	Na+ mEq/L	K+ mEq/L	TC/HDLC	C-Peptide ng/ml
Normal range	**70-110**	**4%-7%**	**1.6- 3.0**	**135-145**	**3.5-5**	**<4.5**	**0.9-7.1**
Mean	85.8	4.907	1.82	140.13	4.2	4.47	5.9
S.D.±	7.27	1.023	0.13	3.03	0.3	1.07	0.9

Table – III Anova Of TC/HDL-C Between Controls And Diabetes Patients With Hypomagnesemia

	Controls	Diabetes patients with hypomagnesemia
Mean	4.20	4.21
SD	±0.32	±0.57
SEM	0.06	0.08
P.Vlaue		<0.026

Statistically significant

Student 't' test

Correlation of c-peptide and Glycemic control (HbA1c) in Type II DM patients

Table IV

	Good Glycemic Control	Poor Glycemic Control
N	42	48
Mean ng/ml	3.864	2.254
S.D.	1.428	1.469
SE Mean	0.220	0.212
P-value		**< 0.00001**

N= Number of Subjects

Correlation of HbA1c and Serum Sodium levels in Diabetic patients

Table VI

	Good Glycemic control 4% - 7%	Poor Glycemic control (>8%)
N	42	33
Mean	143	138.36
S.D.	7.358	4.544
SE Mean		1.371
P-value		**< 0.001**

N= Number of Subjects

Figure :1Comparison of C- Peptide in Type I & Type II Diabetes mellitus

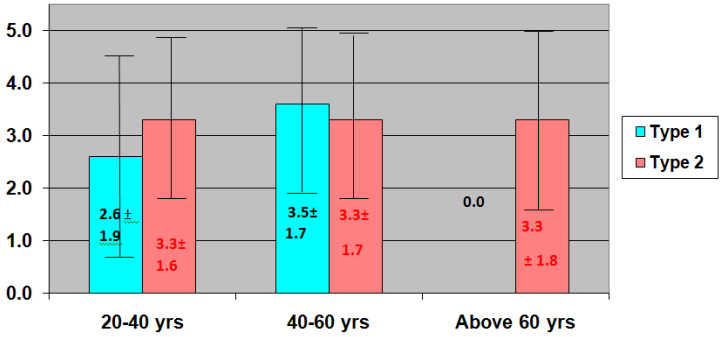


Figure : 2

Comparison of Mg^{++} in Type I & Type II Diabetes mellitus

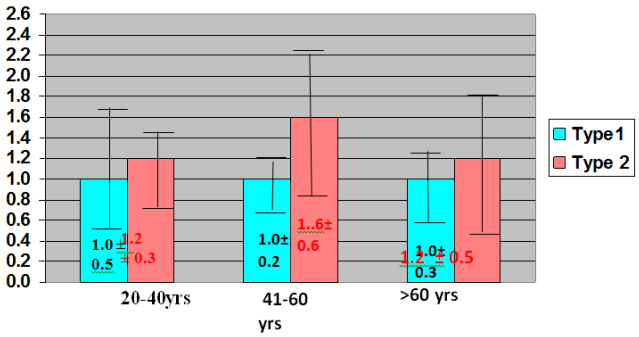


Figure : 3

Comparison of HbA_1c in Type I & Type II Diabetes mellitus

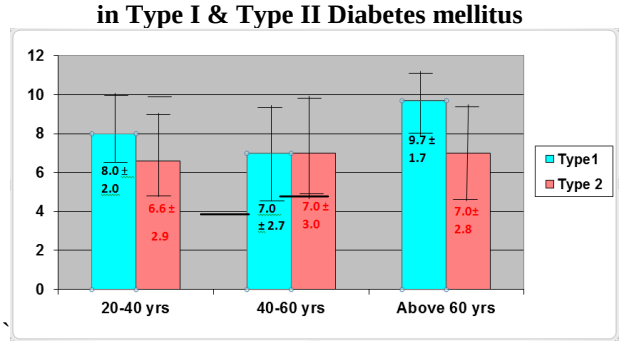
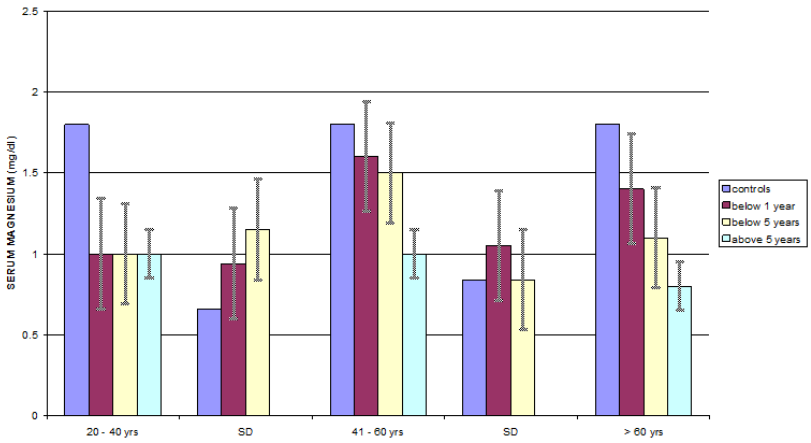


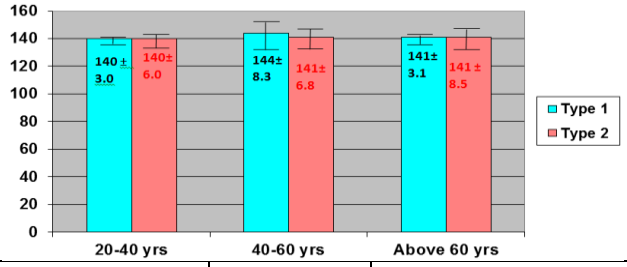
Figure 4

CORRELATION OF CHRONICITY OF TYPE – II DIABETES AND SERUM MAGNESIUM LEVELS



	20 – 40 yrs	41 - 60 yrs	> 60 yrs
controls	1.8	1.8	1.8
below 1 year	1 ± 0.35	1.6 ± 0.5	1.4 ± 0.3
below 5 years	1 ± 0.25	1.5 ± 0.3	1.1 ± 0.4
above 5 years	1 ± 0.15	1 ± 0.1	0.8 ± 0.15

Figure 5 :
Comparison of Na^+ in Type I & Type II Diabetes mellitus



Na^+	20-40yrs	41-60yrs	>60yrs
IDDM Mean± S.D.	140±3.0	144±8.3	141±3.1
NIDDM Mean ±S.D.	140±6.0	141±6.8	141±8.5
K^+			
IDDM Mean± S.D.	4.0±0.5	4.0±0.4	4.0±0.5
NIDDM Mean ± S.D.	4.3±0.4	4.6±1.0	4.3±0.6

Comparison of K^+ in Type I and Type II Diabetes Mellitus **Figure 6**

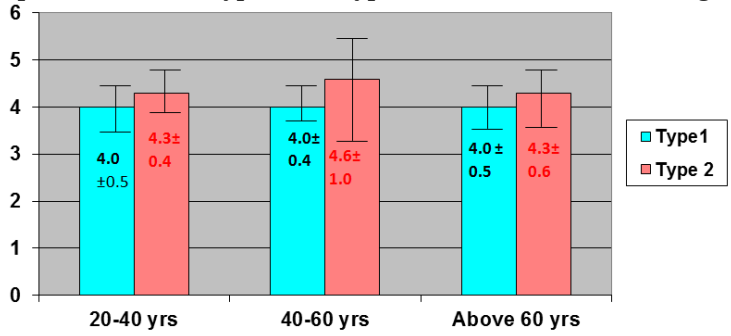
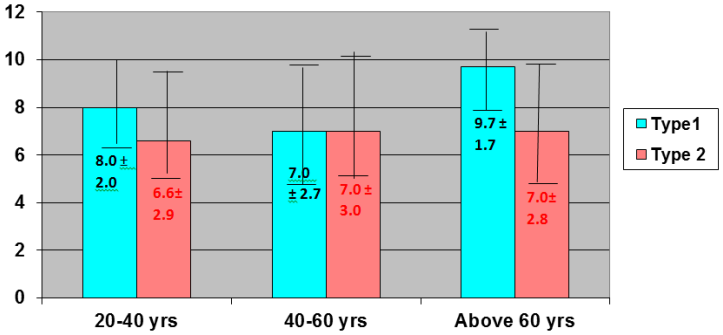


Figure 7 :
Comparison of HbA_1c
in Type I & Type II Diabetes mellitus



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HbA1c %	20-40yrs	41-60yrs	>60yrs
IDDM Mean ±S.D.	8.0 ± 2.0	7.0 ± 2.7	9.7 ± 1.7
NIDDM Mean ±S.D	6.6 ±2.9	7.0± 3.0	7.0± 2.8

The estimated mean serum Magnesium levels were reduced in Type I compared to Type II. in all age groups .This study correlated with Chetan P.,Hans R. Sialy and Devi D. Bansal .[5]**(Figure 1).** The serum C-Peptide levels showed greater variance in diabetic patients with good glycemic control - HbA1c>8% compared to diabetic patients with poor glycemic contro l-HbA1c<4% (P <0.00001)**(Table IV).** correlated with study of Sjoberg et al.[6] In this study good glycemic control related to hyponatremia. P-value <0.001.(**Table V**) TC/HDLC ratio of above 4.5 certainly correlated to hypomagnesemia p-value < 0.026 (Table III) when compared to controls.

In our study the serum Sodium levels were within normal range in both Type I and in Type II diabetic patients in all three age groups. There is no significant influence of age or Type of DM on mean serum Sodium levels**.(Figure 5)** Hypokalemia observed in all age groups in both Type I and Type II diabetes which correlated with the findings of Ryan M.P.[8]**(Figure 6)**. In the present study hyponatremia showed correlation with poor glycemic control (8%) P-value <0.001,(**Table VI)** in accordance with that of Grafton G and Baxter M. A. et al.[7] A definite hypomagnesemia worsening with increasing duration of illness found. Strong correlation observed between serum Mg++ levels and chronicity of illness in elderly diabetic patients(>60yrs) The observations are in accordance with the study of Shills M.E., Garland H.O.[9] and that of Sialy R., Chetan[5] P. et al.(**Figure 4)**

The study concluded by the principal finding that hypomagnesemia , reduced C-Peptide levels and glycemic control play pivotal role in the pathogenesis of metabolic disturbances in Type I & II DM. that chronicity of illness, glycemic control, presence of associated complications and reduced C-Peptiide levels influenced hypomagnesemia. Deleterious effects of dyslipidemia of Diabetes Mellitus per se were exacerbated by hypomagnesemia and reduced C-Peptide levels.

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Abbreviations :

>	-	More than
<	-	Less than
IDDM	-	Insulin Dependent Diabetes Mellitus
NIDDM	-	Non-Insulin Dependent Diabetes Mellitus
C-Peptide	-	Connecting peptide

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