Original Article

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Clinical Profile of Patients with Type 2 Diabetes Mellitus at a Tertiary Care Hospital

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Abstract

Background: Diabetes mellitus is described as a group of metabolic disorders of multiple etiology characterized by chronic hyperglycemia associated with disturbances of carbohydrate, fat and protein metabolism due to absolute or relative deficiency of insulin secretion and/or action. Diabetes mellitus is associated with significant long term sequels, particularly damage or dysfunction of various organs especially kidneys, eyes, nerves, heart and blood vessels. **Subjects and Methods:** A pre-structured and pre-tested proforma was used to collect the data. Informed consent was taken from all cases and control subjects. Base line data including age and sex, detailed medical history including conventional risk factors, clinical examinations and relevant investigations were included as part of the methodology. **Results:** The mean serum FBS level among cases was 146.94±54.99 mg % as compared to 86.71±9.71 mg % among controls. There was highly significant difference in serum FBS levels among cases and controls. **Conclusion:** The mean serum PPBS level among cases was 244.26±96.47 mg % as compared to 111.96±8.85 mg % among controls. There was highly significant difference in serum PPBS levels among cases and controls.

Keywords: Diabetes mellitus, FBS, PPBS.

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Introduction

The worldwide prevalence of DM has risen dramatically over the past two decades, from an estimated 30 million cases in 1985 to 382 million in 2013. Based on current trends, the International Diabetes Federation projects that 592 million individuals will have diabetes by the year 2035.[1]

Diabetes mellitus is a complex metabolic disease caused by variable interactions between hereditary and environmental factors. It is the most common endocrine disease.

Diabetes is perhaps as old as mankind. Diabetes was recognized as a disease entity in ancient Indian ―Ayurveda‖ and described as the ―Madhumeha‖ or honey like urine in reference from Charaka (400 BC).[2]

Diabetes mellitus is described as a group of metabolic disorders of multiple etiology characterized by chronic hyperglycemia associated with disturbances of carbohydrate, fat and protein metabolism due to absolute or relative deficiency of insulin secretion and/or action. Diabetes mellitus is associated with significant long term sequels, particularly damage or dysfunction of various organs especially kidneys, eyes, nerves, heart and blood vessels. That imposes a tremendous burden on the individuals with diabetes and on health care system.

Type 2 diabetes mellitus is the most common, accounting for 80-90% of the diabetic population. Insulin resistance along with relative or absolute insulin deficiency are seen in these subjects.[3]

Type 2 DM frequently remains undiagnosed over a long period of time. Because of relative insulin deficiency, hyperglycemia develops gradually and in the initial stages is not severe enough to give rise to the classical symptoms of diabetes.

The exact pathogenesis of type 2 DM is uncertain. Insulin resistance and β-cells dysfunction may exist together in established cases, it is not clear which is the primary one. However hyperglycemia, itself is a known causative factor for insulin resistance and β- cells dysfunction. Genetic and environmental factors play an important role in the pathogenesis of type 2 DM.[4]

Type 2 DM is commonly seen in individuals above 40 years. About 60% of patients are obese. These patients have high plasma insulin levels. Some of them show mutations in the insulin receptor gene.

Another gene that is expressed only in adipose tissue and its protein product Leptin, is vital in regulating body weight. Plasma levels of leptin are increased in some cases ofType2 DM. The maturity onset diabetes of young (MODY) is due to defective glucokinase (GK). The GK gene is on chromosome 7p. This mutation produces relative insulin deficiency by increasing threshold for glucose induced insulin secretion.Hepatic nuclear factors (HNF1α and HNF4α) gene

defects are two other causes of MODY. All these genetic variants of MODY are inherited as autosomal dominant type.[5]

Under activity, over eating with obesity is associated with the development type2 diabetes. Through increasing resistance to the action of insulin, obesity probably acts as a diabetogenic factor.

Regular exercise in early adulthood can significantly reduce subsequent development of type 2 DM. It is proposed that malnutrition in utero and in infancy may damage beta cell development at a crucial period, predisposing to type 2 diabetes later in life.

Certain drugs like beta adrenergic agonists, thiazide diuretics, corticosteroids are known to be diabetogenic in nature.

Insulin is produced by the beta cells of the islets Langerhans in pancreas in response to hyperglycemia. Beta cells have GLUT-2[transporter] through which glucose is absorbed. Inside the cells glucose is phosphorylated by glucokinase so hyperglycemia increases cellular metabolism through glycolysis, citric acid cycle with generation of ATP.

ATP opens calcium channels. Increased intracellular calcium activates adenylcyclase, which produces cyclic AMP. This cyclic AMP along with calcium causes insulin secretion. Insulin secretion is enhanced by GTP, secretin, gastrin, proteins amino acids, fatty acids, ketone bodies and sulfonylurea drugs. Inhibition of secretion occurs following α-adrenergic stimulation and vagotomy.[6]

Subjects and Methods

A pre-structured and pre-tested proforma was used to collect the data. Informed consent was taken from all cases and control subjects. Base line data including age and sex, detailed medical history including conventional risk factors, clinical examinations and relevant investigations were included as part of the methodology.

For blood investigations, 5ml of blood was collected under aseptic precautions from selected subjects on overnight fasting of 12 hours.

Hundred patients of type 2 diabetes mellitus between age group of 30-70 years attending General Medicine OPD, Medical College and Hospital, were included in the study. Also hundred healthy volunteers in the age group 30-70 years, sex matched during the same period were included in the study under the control group.

Inclusion Criteria

Hundred subjects of type 2 diabetes mellitus between age group of 30-70 years attending general medicine OPD at Medical College and Hospital.

Exclusion Criteria

- Type 1 diabetes mellitus
- Other states associated with altered serum ferritin levels like-o Hemochromatosis
- Chronic alcoholics
- Chronic inflammatory condition like SLE, rheumatoid arthritis o Hepatitis, pancreatitis
- Patients with repeated blood transfusions o Iron deficiency anemia
- Hypothyroidism
- History of Iron Supplementation, pregnancy.

Results

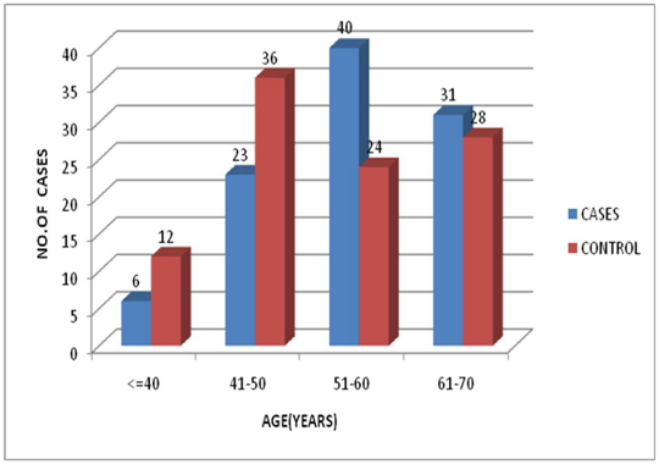

Figure 1: Age Distribution in study group

The mean age was 55.93±7.89 years in cases (type 2 diabetes mellitus) in controls (healthy individuals) it was 53.36±9.56 years. The maximum number of patients were in the age group 41-60 years.

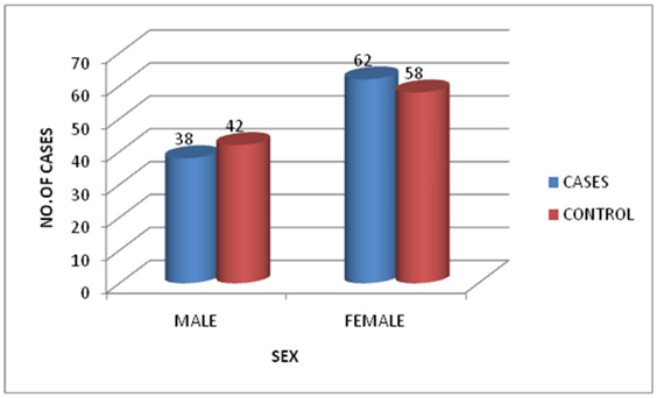

Figure 2: Sex Distribution in study group

Among cases 38% were males and 62% were females and among controls 42% males and 58% females was the sex distribution in this study.

Table 1(a): Comparison of Glucose parameters in study group

FBS		Cases		Controls		Total
		No	%	No	%	
FBS	<110	25	25	100	100	125
	110-200	57	57	0	0	57
	>200	18	18	0	0	18
Total		100	100	100	100	200

Table 1(b): Comparison of Glucose parameters in study group

PPBS		Cases		Controls		Total
		No	%	No	%	
PPBS	<126	5	5	100	100	105
	126-200	38	38	0	0	38
	>200	57	57	0	0	57
Total		100	100	100	100	200

The mean serum FBS level among cases was 146.94±54.99 mg % as compared to 86.71±9.71 mg % among controls.

There was highly significant difference in serum FBS levels among cases and controls.

The mean serum PPBS level among cases was 244.26±96.47 mg % as compared to 111.96±8.85 mg % among controls. There was highly significant difference in serum PPBS levels among cases and controls.

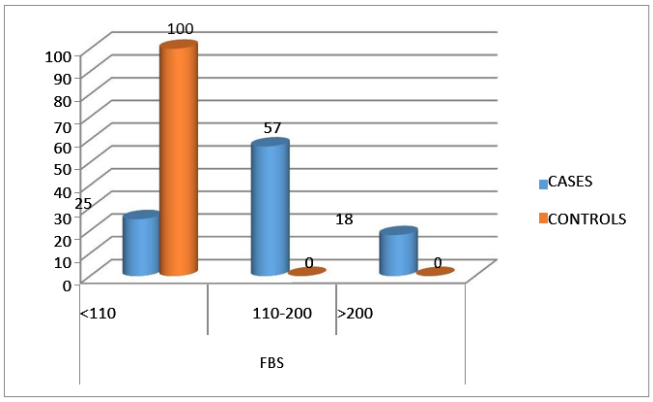

Figure 3: Sex Distribution in study group

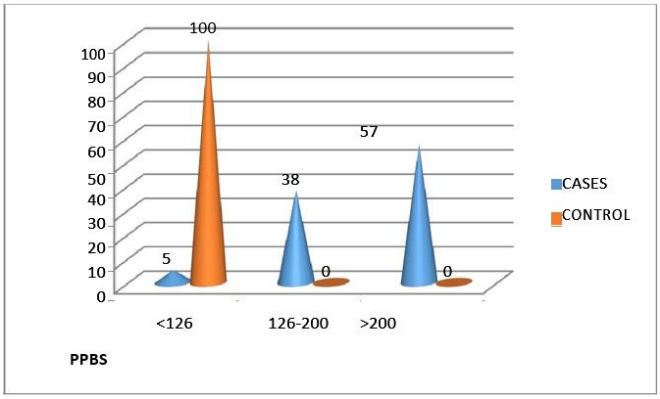

Figure 4: Comparison of Glucose Parameters In Study Group (PPBS) (%)

Discussion

Numerous longitudinal and cross sectional studies have provided evidences that hyperglycemia antedates the development of type2 diabetes mellitus. The insulin resistance can occur in various tissues, liver, muscle, etc. In muscles, the defects in actions are

- Impaired insulin receptor tyro kinase activity,
- Diminished glucose transporters
- Diminished glycogen synthatase and pyruvate dehydrogenase.[7]

Thus causes disturbances in major intracellular pathways of glucose disposal, namely glycogen synthesis and glucose oxidation.

In type 2 DM, both receptor and post receptor defects contribute to insulin resistance.

In diabetic subjects with moderate to severe hyperglycemia, post binding defects in insulin action are responsible for the insulin resistance.

In subjects with impaired glucose tolerance, the defect may be at insulin binding to its receptor.

First phase

Plasma glucose remains normal despite demonstrable insulin resistancebecause the insulin levels are increased.

Second phase

Insulin resistance tends to worsen so that postprandial hyperglycemiadevelop despite elevated insulin concentration.

Third phase

Insulin resistance does not change but declining insulin secretion causesfasting hyperglycemia.

Glycosuria when plasma glucose concentration exceeds the renal threshold. The severity of the classical osmotic symptoms of polyuria and polydipsia is related to Glucosuria.

Non enzymatic glycosylation results from interaction of glucose derived dicarbonyl precursor like glyoxal, methyl glyoxal etc with the amino group of both intracellular and extra cellular proteins.[8]

Once glycation of proteins occur, it is completely irreversible. AGEs cause cross linking between extracellular proteins like collagen and trapping LDL particles.

Circulatory particles modified by addition of AGEs bind to endothelial cells, mesangial cells and macrophages leading to release of growth factors, increased endothelial permeability, increased pro coagulant activity and enhanced fibroblast and smooth muscle cell proliferation, resulting in atherosclerosis andmicroangiopathy.

HbA_{1c}reflects average plasma glucose over the previous 8 to 12 weeks. It can be performed at any time of the day and does not require any special preparation such as fasting.

More recently there has been substantial interest in using it as a diagnostic test for diabetes and as a screening test for persons at high risk of diabetes.[9]HbA_1C is normally about 4 to 6 % of total HbA_1 in healthy individuals .Hyperglycemia causes intracellular de novo synthesis of diacyl glycerol from glycolytic intermediates and hence causes activation of PKC. The downstream effect of PKC are alteration of transcription of genes for fibronectin, type4 collagen, contractile proteins and extracellular matrix protein in endothelial cells, neurons and increased production of VEGF, TGF-β, PDGF, EGF, IGF-1 leading to diabetic vascular episodes.[10]

Conclusion

- The mean age of diabetes were 55.93±7.89 years and controls were 53.36±9.56 years. Peak age incidence was seen in the age group of 41 – 60 years.
- Among cases 38% males and 62% females, among controls 42% males and 58% females was the sex distribution in this study.
- The mean FBS was 146.94±54.99 mg/dl in diabetes and 86.71±9.71 mg/dl in controls($p<0.001$**).
- The mean PPBS was 244.26±96.47 mg/dl in diabetes and 111.96±8.85 in controls($p<0.001$**).

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