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A study on clinical profile of patients with the diabetic complications: descriptive study

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Abstract

Type 2 diabetes, is caused by insulin resistance which is characterized by a decreased effectiveness of insulin. In contrast to type 1 diabetes in which the pancreatic islets are destroyed and no insulin can be synthesized anymore, in type 2 diabetes insulin secretion is normal, elevated or reduced. Unlike in patients with type 1 diabetes, symptoms do not appear abruptly, but set on gradually so that the disease often remains undiagnosed for a long time. The present study included 150 patients of type 2 diabetes mellitus. Patients with Type 2 Diabetes mellitus in outpatient and inpatient departments were study subjects. The present study had diabetic patients ranging from 41 to 80 years of age. Majority of cases were in the 61 to 70 age group. Male cases were 58.7% and female cases were 41.3%. 60% of cases of Type 2 DM were associated with complications of which 66.7% were microvascular and 33.3% were macrovascular complications.

Keywords: Type 2 diabetes, macrovascular complications, clinical profile

Introduction

Diabetes mellitus (DM) refers to a group of common metabolic disorders that share the phenotype of hyperglycemia. Several distinct types of DM are caused by a complex interaction of genetics and environmental factors. Depending on the etiology of the DM, factors contributing to hyperglycemia include reduced insulin secretion, decreased glucose utilization, and increased glucose production. The metabolic dysregulation associated with DM causes secondary pathophysiologic changes in multiple organ systems that impose a tremendous burden on the individual with diabetes and on the health care system [1, 2].

Type 2 diabetes, is caused by insulin resistance which is characterized by a decreased effectiveness of insulin. In contrast to type 1 diabetes in which the pancreatic islets are destroyed and no insulin can be synthesized anymore, in type 2 diabetes insulin secretion is normal, elevated or reduced. Unlike in patients with type 1 diabetes, symptoms do not appear abruptly, but set on gradually so that the disease often remains undiagnosed for a long time [3].

The global burden due to diabetes is mostly contributed by type 2 diabetes which constitutes 80% to 95% of the total diabetic population. Diabetes Mellitus is a major public health

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concern of the twenty-first century. The explosive increase in the prevalence of diabetes seen in the last three decades pose huge clinical and economic burden in many countries. The estimates by the International Diabetes Federation (IDF) show that285 million adults (20 years to 79 years) were affected by the disorder in 2010. Epidemiological trends indicate that without proper control and prevention, its prevalence increase further to 438 million in 2030 (33.4%). There is little gender difference in the distribution in the number affected with diabetes. The largest numbers with diabetes are in the 40 to 59 age groups (132 million, in 2010) which are expected to rise further. By 2030, there will be more diabetic people in the 60 to 79 age groups (169 millions) [4].

Magnesium is a ubiquitous element in nature and forms an estimated 2.1% of the earth's crust. Mg plays an essential role in a wide range of fundamental biologic reactions. It is the second most abundant intracellular cation in the human body (second only to potassium) and the fourth most abundant total cation. Mg serves as cofactor in more than 300 enzymatic reactions involving energy metabolism and protein and nucleic acid synthesis. 31 free ionized Mg is the physiologically active form of the element. The intracellular level of free Mg2+ serves to regulate intermediary metabolism through activation of such rate-limiting enzymes as hexokinase, pyruvate dehydrogenase, enolase or creatine phosphokinase [5, 6].

Methodology

Study subjects

Patients with Type 2 Diabetes mellitus in outpatient and inpatient departments.

- **Sample size:** 150
- **Type of study:** Cross sectional

150 patients with Type 2 DM underwent the following tests:

1. Detailed history
2. Clinical Examination
3. Glycemic Status (FBS, PPBS, HbA1C)
4. Fasting Serum Magnesium Levels
   Method: Colorimetric method using calmagite dye:
   Reference range: 1.8-2.5 mg/dL.

Inclusion criteria

- Age > 18 yrs
- Diagnosed cases of Type 2 Diabetes Mellitus based on FBS, PPBS

Exclusion criteria

- Alcohol abuse.
- Patients on loop or thiazide diuretics.
- Patients on antibiotics like Aminoglycosides.
- Pregnancy.
- Congestive heart failure.
- Long term parenteral nutrition.

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Results

Table 1: Age Distribution

Age in years	No. of cases of Type 2 DM	Percent
41-50	30	20%
51-60	40	26.7%
61-70	65	43.3%
71-80	15	10%
Total	150	100%

The mean age of the group being 59.58 years and the standard deviation being 9.68 years (Mean +/- SD → 59.58+/- 9.68).

Table 2: Sex Distribution

Sex	No. of cases	Percent
Male	88	58.7%
Female	62	41.3%
Total	150	100%

Out of 150 cases of type 2 DM, male subjects were 88 i.e 58.7% and female subjects were 62 i.e 41.3%.

Table 3: Type 2 DM and Complications

No. of cases with T2 DM	With complications	Without complications
150	90	60
	60%	40%

Out of 150 cases of Type 2 DM, 90 subjects were detected to have complications i.e 60%.

Discussion

The present study included 150 patients of type 2 diabetes mellitus. The present study had diabetic patients ranging from 41 to 80 years of age. Majority of cases were in the 61 to 70 age group. Male cases were 58.7% and female cases were 41.3%. 60% of cases of Type 2 DM were associated with complications of which 66.7% were microvascular and 33.3% were macrovascular complications.

Relative insulin deficiency and inadequate fluid intake are the underlying causes of HHS. Insulin deficiency increases hepatic glucose production (through glycogenolysis and gluconeogenesis) and impairs glucose utilization in skeletal muscle. Hyperglycemia induces an osmotic diuresis that leads to intravascular volume depletion, which is exacerbated by inadequate fluid replacement. The absence of ketosis in HHS is not understood. Presumably, the insulin deficiency is only relative and less severe than in DKA. Lower levels of counter regulatory hormones and free fatty acids have been found in HHS than in DKA in some studies. It is also possible that the liver is less capable of ketone body synthesis or that the insulin/glucagon ratio does not favor ketogenesis [7].

The prototypical patient with HHS is an elderly individual with type 2 DM, with a several-week history of polyuria, weight loss, and diminished oral intake that culminates in mental confusion, lethargy, or coma. The physical examination reflects profound dehydration and hyperosmolality and reveals hypotension, tachycardia and altered mental status. Notably

absent are symptoms of nausea, vomiting and abdominal pain and the Kussmaul respirations characteristic of DKA. HHS is often precipitated by a serious, concurrent illness such as myocardial infarction or stroke. Sepsis, pneumonia, and other serious infections are frequent precipitants and should be sought [8]. In addition, a debilitating condition (prior stroke or dementia) or social situation that compromises water intake usually contributes to the development of the disorder.

The microvascular complications in diabetes include retinopathy, nephropathy and neuropathy. The causes are not fully understood, but there are a few hypotheses. The 'glucose hypothesis' postulates that chronic hyperglycemia causes these complications, and that correction of hyperglycemia will prevent them. Studies have documented associations between the duration and degree of hyperglycemia and the severity of microvascular and neuropathic complications. Pirart *et al*. for example, followed 4,400 diabetic patients for up to 25 years and showed that poor glycemic control was clearly related to a higher prevalence of retinopathy, neuropathy and nephropathy. However, not all patients 22 with hyperglycemia develop complications. Other factors, including age, sex, race, ethnicity, socioeconomic status, hyperlipidemia, and smoking have been implicated. In type 1 diabetics, intensive therapy has shown to delay the onset and slow the progression of retinopathy, nephropathy and neuropathy by 35-70%. For type 2 diabetes, up to now, there are no convincing data from prospective studies to support a beneficial effect of intensive therapy on microvascular complications [9, 10].

Hyperglycemia appears to damage tissues by causing both acute, reversible changes in cellular metabolism and cumulative, irreversible alterations in stable macromolecules such as extracellular matrix components and nucleic acids. The reversible abnormalities include abnormal polyol metabolism and the formation of early glycation products on matrix, cellular and plasma proteins. The polyol pathway is based on a family of enzymes, which can utilize as substrates a wide variety of sugar-derived compounds and reduce these to their respective sugar-alcohols (polyols); for example glucose is converted to sorbitol.

Conclusion

- Male cases were 58.7% and female cases were 41.3%.
- 60% of cases of Type 2 DM were associated with complications.

References

1. McNair P, Christensen MS, Christiansen C, Madsbad S & Transbol I. Renal hypomagnesaemia in human diabetes mellitus: its relation to glucose homeostasis. Eur J Clin Invest. 1982;12:81-85.
2. Paolisso G, Scheen A, D' Onfrio F, Lefebvre P. Magnesium and glucose. Diabetologia. 1990;33:511-514.
3. Schmidt LE, Arfken CL, Heins JM. Evaluation of nutrient intake in subjects with non-insulin dependent diabetes mellitus. J Am Diet Assoc. 1994;94:773-774.
4. Resnick LM, Gupta RK, Bhargava KK, Gruenspan H, Alderman MH & Laragh JH. Cellular ions in hypertension, diabetes, and obesity. A nuclear magnetic resonance spectroscopic study. Hypertension. 1991;17:951-957.
5. Kolterman OG, Gray RS, GriYn J, Burstein P, Insel J, Scarlett JA, *et al*. Clin. Invest. 1981;68:957-969.
6. Tosiello L. Hypomagnesemia and Diabetes Mellitus. Arch Intern Med. 1996;156:1143-1148.
7. Corsonello A, Ientile R, Buemi M, Cucinotta D, Mauro VN, Macaione S, *et al*. Serum ionized magnesium levels in type 2 diabetic patients with micro albuminuria or clinical

proteinuria. Am J Nephrol. 2000;20(3):187-192.
8. Paolisso G, Passariello N, Pizza G, *et al*. Dietary magnesium replacements improve B-cell response to glucose and arginine in elderly non-insulin dependent diabetic subjects. Acta endocrinol. 1989;121:16-20.
9. Standards of medical care in diabetes. Diabetic Care. 2012;35:511-563.
10. Pirart J. Diabetes mellitus and its degenerative complications: a prospective study of 4,400 patients observed between 1947 and 1973. Diabete Metab. 1977;3:245-256.