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Diabetes mellitus associated mortality: Therapy and awareness

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ABSTRACT

United States studies have estimated nearly 1 million Americans, or 9.3% of the population had diabetes mellitus in the year 2012 and contributed to 234,051 deaths per annum which lead to Diabetes remains the 7th leading cause of death in the United States. This study revealed risk factors associated with various co-morbidities due to increased blood glucose levels such as hypertension, stroke, heart attacks, kidney diseases, lower limb amputations. The report from "American Diabetes Association" survey to examine awareness and pharmacological treatment of uncontrolled diabetes mellitus among age groups with special emphasis on three groups which includes, Persons unaware of diabetes, Persons who are aware but not treated with medication, Persons who are aware and pharmacologically treated with medication but still have uncontrolled. The objective of present work is to create the awareness for diabetes mellitus and it's therapy. The "National Diabetes Statistics" 2014 report of United States reveals in year 2012 about 86 million Americans age 20 and older had pre diabetes. Out of the 29.1 million, 8.1 million were undiagnosed. We assessed in our findings that maximum percentage of patients who are diabetic but not adhering to medicine due to its high cost. we should use different strategies and various awareness programs viz Marathons, flash mobs to significantly reduce the Diabetes associated mortality cases**.** This article represent the application of selected statistical tools such as "SPSS" software for data analysis for assessing the increasing mortality due to Diabetes.

Keywords: Diabetes mellitus, SPSS, SBMG.

INTRODUCTION

Diabetes Mellitus: It is the most common of the endocrine disorders. It is a chronic condition characterised by hyperglycemia and due to impaired insulin secretion with or without insulin resistance. The most common types of diabetes mellitus are Type 1 diabetes and Type 2 diabetes.

Type 1 Diabetes is a disease characterised by the destruction of the insulin producing pancreatic beta cells, the development of which is autoimmune T-cell mediated destruction or idiopathic type.

Type 2 diabetes is a disease which begins with insulin resistance, i.e the cells do not respond to insulin. As the disease progresses, lack of insulin may develop which may require the patients to take extrinsic insulin[1].

Other specific types (Table 3):

Other specific types are currently less common causes of diabetes mellitus, but are those in which the underlying defect or disease process can be identified in a relatively specific manner. They include, for example, fibro calculous pancreatopathy, a form of diabetes which was formerly classified as one type of malnutrition–related diabetes mellitus[5].

Gestational Hyperglycaemia and Diabetes

Gestational diabetes is carbohydrate intolerance resulting in hyperglycaemia of variable severity with onset or first recognition during pregnancy. It does not exclude the possibility that the glucose intolerance may antedate pregnancy but has been previously unrecognized[2]. The definition applies irrespective of whether or not insulin is used for treatment or the condition persists after pregnancy[3]. Table1.

Table 1. A etiological Classification of Disorders of Glycaemia

Type -1	Type-2	Other specific types	Gestational diabetes
beta-cell destruction, usually leading to absolute insulin deficiency) Autoimmune Idiopathic	(may range from predominantly insulin resistance with relative insulin deficiency to a predominantly secretory defect with or without insulin resistance)	Genetic defects of beta-cell function	Includes the former categories of gestational impaired glucose tolerance and gestational diabetes.

	Genetic defects in insulin action Diseases of the exocrine pancreas Endocrinopathies Drug- or chemical-induced Uncommon forms of immune-mediated diabetes. Other genetic syndromes sometimes associated Infections

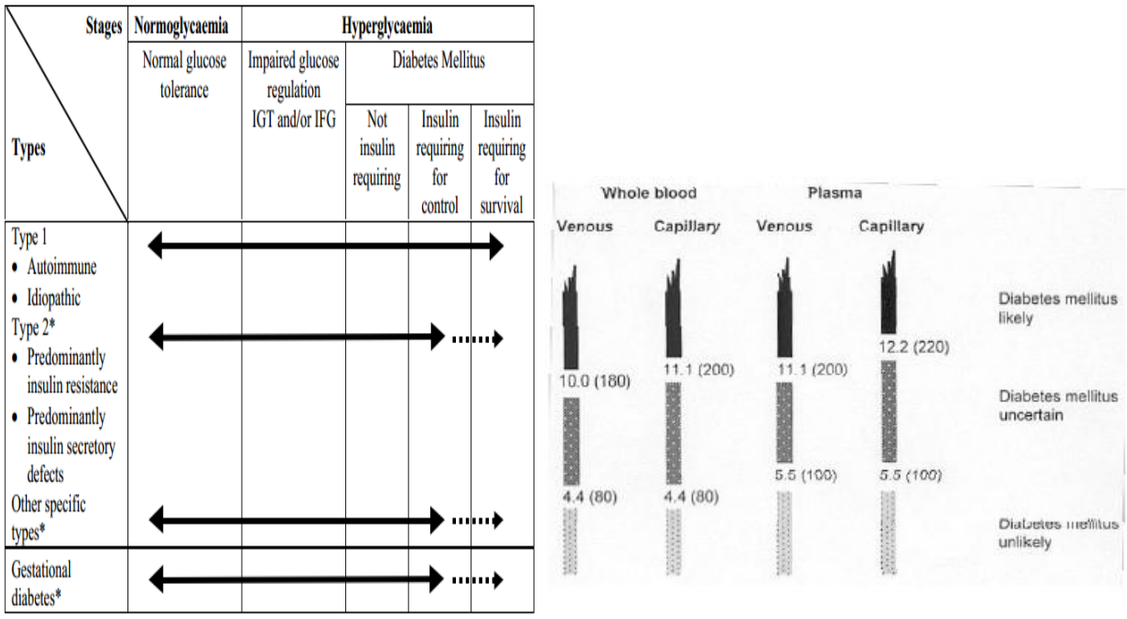

Figure 1: Unstandardized (casual, random) blood glucose values in the diagnosis of diabetes in mmol l-1 (mg dl-1). Taken from the 1985 WHO Study Group Report

The effects of diabetes mellitus include long-term damage, dysfunction and failure of various organs. Diabetes mellitus may present with characteristic symptoms such as thirst, polyuria, blurring of vision, and weight loss. In its most severe forms, ketoacidosis or a non-ketotic hyperosmolar state may develop and lead to stupor, coma and, in absence of effective treatment, death [4].

Insulin

Chemistry

The b (or B) cells of pancreatic islets synthesize insulin from a single-chain precursor of 110 amino acids termed pre proinsulin. After translocation through the membrane of the rough endoplasmic reticulum, the 24-amino-acid N-terminal signal peptide of pre proinsulin is cleaved rapidly to form proinsulin (Figure 60-1). Thereafter, proinsulin folds, and the disulfide bonds form. During conversion of human proinsulin to insulin, four basic amino acids and the remaining connector or C peptide are removed by proteolysis. This gives rise to the A and B peptide chains of the insulin molecule, which contains one intra subunit and two inter subunit disulfide bonds. The A chain usually is composed of 21 amino acid residues, and the B chain has 30; the molecular mass is thus about 5734 daltons. (Figure 2.)

Insulin Production

The islet of Langerhans is composed of four types of cells, each of which synthesizes and secretes a distinct polypeptide hormone: insulin in the b (B) cell, glucagon in the (A) cell, somatostatin in the d (D) cell, and pancreatic polypeptide in the PP or F cell.

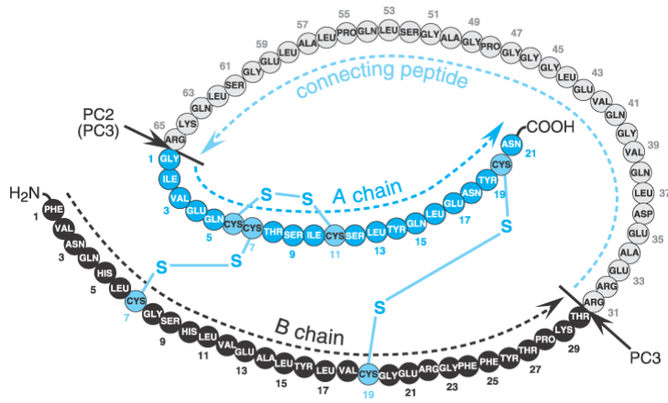

Figure 3: Human proinsulin and its conversion to insulin.

The amino acid sequence of human proinsulin is shown. By proteolytic cleavage, four basic amino acids (residues 31, 32, 64, and 65) and the connecting peptide are removed, converting proinsulin to insulin. The sites of action of the endopeptidases PC2 and PC3 are shown.

Recent Statistics for Prevalence of diabetes mellitus In India

Table 2. Recent studies (2000-2012) on prevalence of diabetes mellitus in urban and rural Indian population

First author	Year	Place	Age (yr)	Sample Size	Prevalence (%)
Urban Population					
Anand MP	2000	Mumbai	30-60	1662	34.0
Gupta PC	2004	Mumbai	≥ 35	88653	47.9
Prabhakaran D	2005	Delhi	20-59	2935	30.0
Reddy KS	2006	National	20-69	19973	27.2
Mohan V	2007	Chennai	≥ 20	2350	20.0
Kaur P	2007	Chennai	18-69	2262	27.2
Yadav S	2008	Lucknow	≥ 30	1746	32.2
Rural Populations					
Hazarika	2004	Assam	>30	3180	33.3
Thankappan	2006	Kerala	>30	2159	36
Krishnan A	2008	Harayana	15-64	2828	9.3
Todkar SS	2009	Maharashtra	≥ 20	1297	7.2
Vijaykumar G	2009	Kerala	≥18	1990	36.1
Bhardwaj R	2010	Himachal	≥ 18	1092	35.9
Kinra S	2010	National	20-69	1983	20.0

Table 3: Statistical data

Type of Diabetes mellitus	**2012**	**2013**	**2014**	**2015**	**Total**
Not aware of Diabetes	30	45	35	10	120
Aware of Diabetes but no treatment is taken	50	65	60	35	210
Treated but no results	25	45	15	10	95
Total	105	155	110	55	425

After applying the CHI-SQUARE (X2) technique level of significance of 5% and degree of freedom 12 and then observed and expected reading are calculated are the results are in the below table.

Observed (O)	**Expected (E)**	**(O-E)**	**$(O-E)^2$**	**$(O-E)^2/E$**
30	29.6	0.4	0.16	5.40
50	51.8	1.8	3.24	0.0625
25	23.4	1.6	2.56	0.109
45	43.7	1.3	1.69	0.0386
65	76.5	-11.5	132.25	1.728
45	34.6	10.4	108.1	3.124
35	31	4	16	0.516
60	54.3	5.7	32.49	0.598
15	24.5	-9.5	90.25	3.683
10	15.5	-5.5	30.25	1.951
35	27.1	7.9	62.41	2.302
10	12.2	-2.2	4.84	0.396
				Total= 19.90

Under the level of significance of the 5% table value the test have been passed.

Treatment strategies

To control the disease oral hypoglycemic drugs which are the conventional treatment is given. The various drugs used as conventional therapy[10] are,

- Sulphonylureas: Tolbutamide, chlorpropamide, Glibenclamide, Glipizide, Glimepiride.
- Biguanides: Metformin
- Meglitinides: Repaglinide, Nateglinide
- Thiazolidinediones: Pioglitazone
- Dipeptidyl peptidase-4 inhibitors: Sitagliptin, Vildagliptin
- Incretin mimetics.

For patients who do not achieve effective glycemic control, they are given insulin along with the conventional therapy of oral hypoglycemic drugs. The conventional insulin therapy involves taking injections at meal times. These injections are taken subcutaneously or intravenously[6,7].

Insulin therapy

Insulin lowers the concentration of glucose in blood by inhibiting hepatic glucose production and by stimulating the uptake and metabolism of glucose by muscle and adipose tissue (Table 4). These two important effects occur at different concentrations of insulin[11]. Production of glucose is inhibited half maximally by an insulin concentration of about 20 munits/ml, whereas glucose utilization is stimulated half maximally at about 50 munits/ml. In both types of diabetes, glucagon (the levels of which are elevated in untreated patients) opposes the hepatic effects of insulin by stimulating glycogenolysis and gluconeogenesis, but it has relatively little effect on peripheral utilization of glucose. Thus, in the diabetic patient with insulin deficiency or insulin resistance and hyper glucagonemia, there is an increase in hepatic glucose production, a decrease in peripheral glucose uptake, and a decrease in the conversion of glucose to glycogen in the liver[9].

Table 4. Hypoglycemic Actions of Insulin

LIVER	MUSCLE	ADIPOSE TISSUE
Inhibits hepatic glucose production (decreases gluconeogenesis and glycogenolysis)	Stimulates glucose uptake	Stimulates glucose uptake (amount is small compared to muscle)
Stimulates hepatic glucose uptake	Inhibits flow of gluconeogenic precursors to the liver (*e.g.*, alanine, lactate, and pyruvate)	Inhibits flow of gluconeogenic precursor to liver (glycerol) and reduces energy substrate for hepatic gluconeogenesis (non esterfied fatty acids)

Pathophysiology:

Figure 5 &6 Diabetes mellitus Type 1 &2[8]

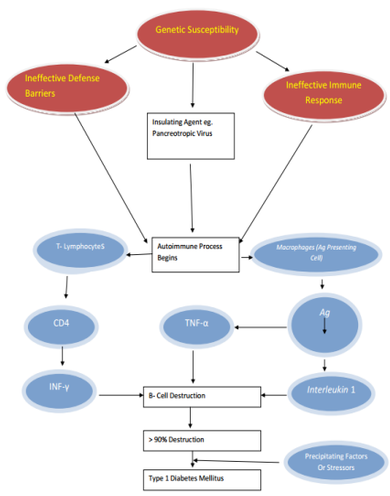

Figure 5

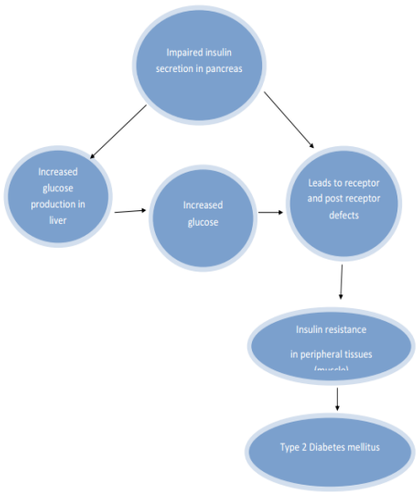

Figure 6

CONCLUSION

This article reflects increasing Mortality rate due to diabetes mellitus and Need to spread awareness among people about the disease. Many people step back for the treatment for the costly medications which leads to severe complications. Currently, only invasive procedures such as SMBG and continuous glucose monitoring (CGM) are able to provide

accurate information on the daily profile of blood glucose levels. There is a need of optimization of anti-diabetic therapy in addition to suggestions concerning its use to introduce necessary changes of daily lifestyle habits. It can also provide information about treatment adherence.

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