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Type 2 diabetes mellitus patients in Kashmir valley

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Abstract

The paper is based on the annunciate literature confabulating history, definition, classification, diagnostic criteria and the prevalence of T2DM in J&K. The compendium is based on free text search for various terms epitomizing T2DM which makes us to understand the concept behind it. Besides online database some articles were ascertained from conventional journals and books. After prelustration the relevant articles dealing purely with the subject were classified and presented into five categories:

Keywords: Diabetes, T2DM, prevalence, dietary intake.

Introduction

The history of knowledge of diabetes was given way back in centuries before Christ. A disease similar to that of diabetes associated with the passage of much urine was given by an Egyptian, medical Papyrus (Ebers Papyrus) of herbal knowledge way back in 1500. B.C. Celsus in (30 B.C to 50 A.D.) also observed a similar diseases but it was another Greek Physician Aretaeus of Cappadocia, who after two centuries gave the name Diabetes (a siphon). He described diabetes as "a melting of the flesh and limbs into urine" this was the first complete description of diabetes ever given by any scientist. Similar descriptions about a condition having excessive urination with sweet and sticky urine was also given in 3rd to 6th centuries A.D by several scholars from China, Japan and India. Thus it was known for centuries that diabetic urine tasted sweet but it was Willis in 1674 who added to the previous description, an observation "as if imbued with honey and sugar". It was in this manner that the term diabetes mellitus got established (mellitus = honey). Hundred years after Willis, Dobson actually confirmed that the sweetness in the urine was indeed because of sugar. He actually measured the concentration of glucose in the urine of such patients and found it to be increased. From the period of the earliest recorded history of Diabetes the clarity in the understanding of the disorder came slowly until the middle of the 19th century (MacFarl and, 1991) [1].

Slowly over the centuries the course and complications of diabetes started being recognized. Avicenna, an Arab physician, in about 1000 A.D. recognized and described "Gangrene" as a condition, (Passed with the seed) having two general varieties, one with the Classic Acute Symptoms (Type 1 or IDDM in today's terminology) and the other with "torpor, indolence, and corpulence" (Type 2 or NIDDM). It was in the same century that an association between the disease and the disturbance in the functioning of the beta cells was also established. Brockman as early as 19th century noted the islets in the fish; however Langerhans described them in mammals in 1869. It was on his name that the term "Islets of Langerhans" got coined. Two German Scientists namely Von Mering and Minkowski, studied the dogs and found out that the surgical removal of pancreas in them led to Diabetes. An American scientist, Opie, at the turn of the century, noted the damage of beta cells in the islets of humans dying of the diseases. Finally in 1921, two Canadians viz, Banting and Best, succeeded in preparing an active extract of pancreas, which had a lowering effect on the blood glucose level among diabetic dogs. (Engelhardt, 1989) [2]

Definition

In general Diabetes can be defined as a group of anatomic and chemical problems resulting from different factors, in which there is an absolute or relative deficiency of insulin and its function is present. It predisposes an individual to certain microvascular abnormalities as, retinopathy, nephropathy and neuropathy. Besides, it is also associated with accelerated atherosclerosis, and it also tends to double the risk of stroke. The risk of heart attack is

increased by 2 to 3 fold, and for peripheral vascular problems, particularly in the feet, it is increased by 50 fold. In addition there are various other problems, associated with diabetes, as lessening of resistance to infection, especially if the diabetes is poorly controlled. (Marble *et al.*, 1985) [3]

Classification and types

It was in mid – sixties that a set of diagnostic criteria for diabetes GOT published although there was a wide variation both between and within countries (WHO, 1965) [4]. A variety of descriptive terms were used to classify diabetes since then and before 1978. Some of them were based on the age of onset and the others on the degree of severity of diseases or the stage at which the diseases was. It was also found that there was a substantial difference in the diagnostic criteria used by different diabetic experts worldwide (West, 1975) [5]. As a result of this there was a confusion among different schools of thought which led to hindrance in the assessment of data from studies of the natural history of diabetes and its associated complications. Later, a new classification was given by National Diabetes Data Group (NDDG, USA) which was based on clinical or descriptive observations obtained from various epidemiological studies of large populations. This provided a base for uniform designation and a framework for collecting investigative and epidemiological data on diabetes (NDDG, 1979) [6]. WHO Expert Committee on diabetes mellitus in 1980 (WHO, 1980) [7] devised a similar but more inclusive classification and later modified it in 1985 (WHO, 1985) [8] and received general acceptance worldwide. In their terms diabetes was sub classified as: Type 1, insulin dependent diabetes mellitus (IDDM).

Type 2, Non-insulin dependent diabetes mellitus (NIDDM), Malnutrition related diabetes mellitus (MRDM) and other types of diabetes. Keen *et al.*, 1982 [9, 13] gave a brief description of diabetes. He described IDDM or type 1 diabetes mellitus, as the one occurring primarily in young patients and involving about 15 % of diabetic population. It was however seen to be present at any age. The diseases usually were characterized by an abrupt onset of symptoms. However the present evidence suggests that the development of diseases may involve an antecedent period of slowly progressing auto immune damage to the pancreatic β – cells (Laslie, 1989) [10]. Keen further described NIDDM or type 2 diabetes mellitus as the one usually encountered by adults but also occurring in young patients and happening in 80-85% of the diabetic population. NIDDM is considered to have a strong genetic basis, as evidenced by various studies carried out by Taylor (1989) [11] on identical twins and by familiar transmission of diabetes in an autosomal dominant inheritance pattern.

The third clinical sub group namely MRDM (Malnutrition related diabetes mellitus) was included in the WHO classification but not in that of NDDG. Since this type of diabetes had distinctive clinical features and was predominantly found to be occurring among young adults in great numbers from certain regions of tropical and developing countries, it led to the creation of this new major class of diabetes. Different clinical studies (Abu – Bakare, 1986) [12] have suggested the existence of at least two sub classes: FCPD (Fibro Calculous Pancreatic Diabetes) and PDPD/PDDM (Protein deficient Pancreatic Diabetes/Protein deficient Diabetes Mellitus).

Other, different types of diabetes mellitus were also found to exist and were generally classified as secondary diabetes. This heterogeneous sub class includes many specific disorders that induced diabetes e.g. pancreatic diseases, hormones, drugs or

chemicals, certain genetic syndromes and insulin receptor abnormalities (Keen *et al.*, 1982) [9, 13]. Individual whose plasma glucose concentration tend to lie between normal and diagnostic values of diabetes, after an oral glucose load are designated of having IGT (Impaired Glucose Tolerance) (Alberti, 1980) [14]. The term IGT excludes the social, economic and psychological disadvantages with such formerly used designations as chemical, latent or sub clinical diabetes. About 2% of pregnancies tend to have Gestational Diabetes Mellitus (GDM). Generally speaking, GDM is a consequence of diabetogenicity of the gravid state in women with a profound marginal insulinogenic capacity. It however is seen to revert to normal, following parturition in majority of the patients. Gestational Diabetes may increase the risk of prenatal morbidity and mortality, if left unrecognized and untreated. (Hadden, 1986) [15].

NDDG (1979) [6] classified individuals of having abnormal glucose tolerance with previous diabetes or IGT who regained normal glucose tolerance in general, this class includes patients formerly suffering from gestational diabetes or acute hyper glycemia, whose metabolic status has gained normalcy after adopting an appropriate treatment regime. However with stress or weight gain, these individuals tend to remain at increased risk for developing diabetes.

Diagnostic Criteria for Diabetes Mellitus

The clinical diagnosis of diabetes is generally prompted by various symptoms like increased thirst (polydipsia), frequent urination (polyurea), excessive hunger (polyphagia), recurrent infections, unexplained weight loss and in severe cases drowsiness and coma. In addition to these, glycosuria (high levels of glucose in urine) is seen most often.

	Glucose concentration, mmol/l-1 (mg dl-1)		
	Whole blood Venous	Whole blood Capillary	Plasma* Venous
Diabetes Mellitus:			
Fasting	≥ 6.1 (≥ 110)	≥ 6.1 (≥ 110)	≥ 7.0 (≥ 126)
Or			
2-h post glucose load	≥ 10.0 (≥ 180)	≥ 11.1 (≥ 200)	≥ 11.1 (≥ 200)
Or both			
Impaired Glucose Tolerance (IGT):			
Fasting	< 6.1 (< 110)	< 6.1 (< 110)	< 7.0 (< 126)
and			
2-h post glucose load	≥ 6.7 (≥ 120) and < 10.0 (< 180)	≥ 7.8 (≥ 140) and < 11.1 (< 200)	≥ 7.8 (≥ 140) and < 11.1 (< 200)
Impaired Fasting Glycaemia (IGT):			
Fasting (if measured)	≥ 5.6 (≥ 100) and < 6.1 (< 110)	≥ 5.6 (≥ 100) and < 6.1 (< 110)	≥ 6.1 (≥ 110) and < 7.0 (< 126)
and			
2-h post glucose load	< 6.7 (< 120)	< 7.8 (< 140)	< 7.8 (< 140)

* Corresponding values for capillary plasma are: for Diabetes Mellitus, fasting ≥ 7.0 (≥ 126), 2-h ≥ 12.2 (≥ 220); for Impaired Glucose Tolerance, fasting < 7.0 (< 126) and 2-h ≥ 8.9 (≥ 160) and < 12.2 (< 220); and for Impaired Fasting Glycaemia ≥ 6.1 (≥ 110) and < 7.0 (< 126) and if measured, 2-h < 8.9 (< 160). (Mbanya Vivian Nchanchou, 2008) [16]

Prevalence in J&K

Kashmir valley is located in the North Indian state of Jammu and Kashmir, with a unique geographical placement. It differs from other areas in terms of its location, climate, and the amount of ultra violet rays received, socio-demographic as well as life style habits. The prevalence of T2DM in Kashmir valley was ~ 6% in subjects aged ≥ 40 years (Zarger *et al.*, 2000) [17]. In the young adults (20-40 years), a prevalence rate of 2.4% was observed (Zarger *et al.*, 2008) [18]. Zarger *et al.*

(2009) ^[19] conducted a hospital based retrospective study in which he studied the trend of death / mortality among diabetic individuals admitted to the tertiary care hospital (Sheri Kashmir Institute of Medical Sciences, Srinagar). This was done on the basis of screening the medical records of the hospital, including diabetic people who expired over the last 9 years. He found that deaths associated with diabetes accounted for 7.11%. Among men the average age at which the diabetic people expired was 60.07 years and in women it was 57.36 years. Owing to the increased susceptibility towards the contributing factors of the diseases and the changing life style, these studies show an increasing trend in the prevalence of diabetes mellitus in Kashmir valley.

An inappropriate dietary intake is widely recognized as a major etiological factor for most of the long term non communicable diseases. The problem of not taking the healthy diet has now become a societal problem rather than being an individual problem (Somatunga LT, 2004) ^[20]. However, because of the variability in food preference and availability, socio economic factor, cultural concern and educational level, it is difficult to access the dietary habit of free living individuals (Stamler J, 1994) ^[21]. The national dietary survey has several important functions and provides valuable information on dietary habits and nutritional status. Moreover it becomes important for any country serious about promoting the health and well-being of its population, to conduct nutritional monitoring for implementation of programmes related to food, nutrition and health promotion (Lee RD *et al.*, 2002) ^[22].

References

1. MacFarlane I, Bliss M. The history of diabetes (1). In: PicKup J, Williams G. Textbook of diabetes (eds). Blackwell Scientific publications, 1991.
2. Engelhardt D. Von (ed). Diabetes-Its medical and cultural history. Outlines, texts, Bibliography. 1st edn. Berlin, Heidelberg, Spring-Verlag, 1989.
3. Marble A, Krall L, Bradley R. (eds). Joslin's Diabetes Mellitus, 12th ed. Philadelphia, USA: Lea and Febiger 1985; 260:261-333.
4. WHO Expert Committee on Diabetes Mellitus. First Report. Technical Report Series No 310. Geneva: World Health Org, 1965.
5. West K. Substantial differences in the diagnostic criteria used by diabetes experts. Diabetes, 1975; 24:641.
6. National Diabetes Data Group. Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. Diabetes 1979; 28:1039-1057.
7. WHO Expert Committee on Diabetes Mellitus. Second Report. Technical Report Series No 646. Geneva: World Health Organization, 1980.
8. WHO Study Group on Diabetes Mellitus. Third Report. Technical Report Series No 727. Geneva: World Health Organization, 1985.
9. Keen H, Ng Thng Fui S. The definition and classification of diabetes mellitus. Clin Endocrinol Metab. 1982; 11:279.
10. Laslie R, Lazarus N, Vergani D. Aetioloogy of insulin-dependent diabetes. British Medical Bulletin, 1989; 45:58.
11. Taylor R. Aetiology of non-insulin dependent diabetes mellitus. British Medical Bulletin, 1989; 45:73.
12. Abu-bakare A, Gill G, Taylor R. Tropical or malnutrition-related diabetes: a real syndrome. Lancet, 1986; I:1136.
13. Keen H, Jarrett R, McCartney P. The ten-year follow up of the Bedford survey (1962-1972): Glucose tolerance and diabetes. Diabetologia 1982; 22:73.

14. Alberti KGMM. Impaired glucose tolerance (Editorial). Lancet, 1980; II:211.
15. Hadden D. Diabetes in pregnancy. Diabetologia, Finer, 1986; 29:1.
16. Mbanya Vivian Nchanchou. Type 2 Diabetes and Its Association with Lifestyle Factors, 2008.
17. Zargar AH, Khan AK, Masoodi SR, Laway BA, Wani AI, Dar FA. Prevalence of Type 2 Diabetes Mellitus and impaired glucose tolerance in the Kashmir valley of Indian subcontinent. Diabetes Res Clin Prac, 2000; 47:35-46.
18. Zargar AH, Wani AA, Laway BA, Masoodi SR, Wani AI. Prevalence of Diabetes mellitus and other abnormalities of glucose tolerance in young adults aged 20-40 years in North India (Kashmir Valley). Diabetes Res Clin Pract, 2008; 82(2):276-281.
19. Zargar AH, Wani AI, Masoodi SR, Bashir MI, Laway BA, Gupta VK *et al.* Mortality trends among people of Kashmir valley with diabetes admitted to the Tertiary care hospital (Sher-i-Kashmir Institute of Medical Sciences, Soura, Srinagar). Postgrad Med J. 2009; 85:227-32.
20. Somatunga LC. NCD Risk factor survey in Sri Lanka (STEP Survey). Geneva: World Health Organisation, 2004. www.who.int/chp/steps/SriLankaSTEPSReport2003.
21. Stamler J. Assessing diets to improve world health: nutritional research on disease causation in populations. Am J Clin Nutr. 1994; 59(1):146S.
22. Lee RD, Nieman DC. Nutritional Assessments. 3rd edition. McGraw-Hill: Science Engineering, 2002.