“DETERMINANTS OF RISK FOR DEVELOPING TYPE 2 DIABETES MELLITUS IN MEDICAL STUDENTS STUDYING IN NORTH KARNATAKA – A CROSS SECTIONAL STUDY”

REG NO. BG0113005

Dissertation

Submitted to the
KLE University, Belgaum, Karnataka

In Partial Fulfillment
of the requirements for the degree of

M. D.
in
GENERAL MEDICINE

DEPARTMENT OF MEDICINE,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
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ENDORSEMENT

This is to certify that the dissertation entitled **"DETERMINANTS OF RISK FOR DEVELOPING TYPE 2 DIABETES MELLITUS IN MEDICAL STUDENTS STUDYING IN NORTH KARNATAKA – A CROSS SECTIONAL STUDY"** is a bonafide research work done by **CANDIDATE REG NO. BG0113005**.

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LIST OF ABBREVIATIONS USED

β	-	Beta
α	-	Alpha
AC	-	Abdominal circumference
ADA	-	American Diabetes Association
ANOVA	-	Analysis of variance
ARIC	-	Atherosclerosis Risk in Communities
ATP	-	Adenosine triphosphate
BMI	-	Body mass index
BP	-	Blood pressure
CAD	-	Coronary artery disease
CDC	-	Centre for Disease Control and Prevention
CHD	-	Coronary heart disease
CI	-	Confidence interval
Cms	-	Centimeters
CRP	-	C-reactive protein
CT	-	Computed tomography
CURES	-	Chennai Urban Rural Epidemiology Study
CVD	-	Cardiovascular disease
DCCT	-	Diabetes Control and Complications Trial
DKA	-	Diabetic ketoacidosis
DM	-	Diabetes mellitus
DNA	-	Deoxyribo nucleic acid
e.g.	-	For example
FBS	-	Fasting blood sugar

FPG	-	Fasting plasma glucose
GDM	-	Gestational diabetes mellitus
GIP	-	Glucose-dependent insulinotropic polypeptide
GIPR	-	Receptor of gastric inhibitory polypeptide
GLP-1	-	Glucagonlike peptide-1
HbA1c	-	Glycosylated haemoglobin
HDL	-	High density lipoprotein
HHS	-	Hyperglycemic hyperosmolar state
HMGA1	-	High mobility group A1
HNF		Hepatocyte nuclear transcription factor
i.e.	-	That is,
IDDM	-	Insulin Dependent Diabetes Mellitus
IDF	-	International Diabetes Federation
IDRS	-	Indian diabetic risk score
IFG	-	Impaired fasting glucose
IGT	-	Impaired glucose tolerance
INSR	-	Insulin receptor gene
kg/m^2	-	Kilograms per meter square
LDL	-	Low density lipoprotein
MDRF	-	Madras Diabetic Research Foundation
mg/dL	-	Milligrams per deciliter
mmol/L	-	Millimole per liter
MODY	-	Maturity onset diabetes of young
n	-	Total number
NCEP	-	National Cholesterol Education Program

NGT	-	Normal glucose tolerance
NHANES III	-	National Health and Nutrition Examination Survey
NIDDM	-	Non Insulin Dependent Diabetes Mellitus
OGTT	-	Oral glucose tolerance test
OR	-	Odds ratio
p	-	Probability
PAD	-	Peripheral arterial disease
PCOS	-	Polycystic ovarian syndrome
R	-	Pearson's correlation coefficient
SD	-	Standard deviation
SNPs	-	Single-nucleotide polymorphisms
T2DM	-	Type 2 diabetes mellitus
UK	-	United Kingdom
US	-	United States of America
vs	-	Versus
WHO	-	World Health Organization
WHR	-	Waist hip ratio

ABSTRACT

Background and objectives

There has been a trend towards shift in the mean age of onset of type 2 diabetes to a much younger age due to western lifestyle, obesity and family history of diabetes. This study was aimed to find out the differences in risk factors which are likely to predict the development of type 2 diabetes mellitus among medical students.

Methodology

This one year cross-sectional study was carried out in the Department of Medicine of a tertiary care centre situated in South India from January 2014 to December 2014 on 200 medical students aged > 18 years. Assessment of diabetes risk was based on obesity status, exercise status, and family history of type 2 DM. Medical students were evaluated for fasting blood sugar and HbA1c.

Results

In this study, 52.50% of the students were males and male to female ratio was 1.10:1. Most of the students were aged 19 years (31%). 41% of the medical students had mild exercise and 33.5% of the students had AC of 90 to 99 cms in males and 80-89 cms in females while family history of diabetes mellitus was present in 26.5% of the students. Higher BMI levels were noted in 4% of the students. The fasting blood sugar levels were between 100 to 126 mg/dL in 24.5% of the students and 16.50% of the students had HbA1c from 5.7 to 6.4. Moderate risk of diabetes was noted in 16.50% based on IDRS. The family

history, central obesity, higher body mass index and sedentary lifestyle were significantly associated with risk of developing diabetes ($p<0.050$).

Conclusion and interpretation

Individuals above 18 years should be screened for the presence of risk factors of diabetes mellitus using IDRS so as to identify the risk of developing DM and initiate preventive measures.

Keywords

Family history of diabetes; Indian Diabetic Risk Score; Sedentary lifestyle; Type 2 Diabetes Mellitus; Waist Circumference;

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INTRODUCTION

Diabetes mellitus (DM), a metabolic disease is characterized by hyperglycemia which results from defects in either insulin secretion or insulin action or both.[1] Patients with little or no endogenous insulin secretory capacity called as Type 1 (previously Insulin Dependent Diabetes Mellitus [IDDM]) and those who retain endogenous insulin secretory capacity but have a combination of resistance to insulin action and an inadequate compensatory insulin secretory response are known as Type 2 (Previously known as Non Insulin Dependent Diabetes Mellitus [NIDDM]).[1,2]

Diabetes mellitus is a chronic and potentially disabling disease which is reaching an epidemic proportion in many parts of the world and a major growing threat to global public health.[1] Diabetes Mellitus has evolved into a global epidemic and India has the second largest population with diabetes. Diabetes and its complications caused 40.9 million deaths in 2014 and every seven second a person dies from diabetes or its complication. Based on the recent statistics of International Diabetes Association it is estimated that worldwide 387 million people have diabetes and by 2035 this will rise to 592 million. The prevalence in India is over 65 million and these figures are expected to increase to over 100 million by 2030.[3,4]

The rise of prevalence has been more alarming in developing countries than in developed countries (69% versus 20%). Unfortunately, more than 50% of the diabetic patients in India remain unaware of their diabetic status, which increases the risk of development of diabetic complications in them.[5] It has also been found that 66% of the Indian diabetics are not diagnosed, as compared to 50% in Europe and

33% in the USA. India currently faces an uncertain future in relation to the potential burden that diabetes may impose upon the country.[6] The rising prevalence of type 2 DM is closely associated with westernization, industrialization and socioeconomic development.[5]

The chronic hyperglycemia of diabetes results in long term damage, dysfunction and failure of various organs, especially the eyes, kidneys, nerves, heart and blood vessels. Hence, early identification of the risk factors associated with diabetes and appropriate interventions aimed at preventing the onset of diabetes and its complications are urgently required.

Previously, DM was a disease of the middle-aged and elderly. Currently there has been a trend towards shift in the mean age of onset of type 2 diabetes to a much younger age especially in high-risk populations.[7,8] This rise in prevalence has been attributed to changes towards a western lifestyle and the rise in prevalence of obesity.[5] The complex interaction between the environment and the genetic makeup also plays a role in the early onset of pathophysiology of diabetes. Classical type 1 and type 2 are considered to be polygenic, however monogenic forms of diabetes have also been discovered.[1]

A recent population based study[9] for diabetes in youth reported that 7695 youth aged <20 years with diabetes were identified. Most young patients with type 2 diabetes remain asymptomatic for a long time and are incidentally detected. This is a disturbing finding as the earlier age of onset combined with increasing prevalence of diabetes could have adverse effects on the nation's health and economy in a

developing country like India. This underscores the need for mass awareness and screening programs to detect pre-diabetes and diabetes at an early stage.

Medical students have a stressfull life, sedentary lifestyle and irregular food habits. Also, at our institution a large number of obese medical students have been observed. This indicates that a percentage of them might be at a higher risk which could pre-dispose them for diabetes or prediabetes at a younger age. Medical students being a very important part of the society and can be easily educated regarding the early identification of impaired glucose tolerance and diabetes to prevent the complications that follow in the later life. Hence, this study was undertaken to find the differences in risk factors among medical students likely versus not likely to develop Type 2 DM.

OBJECTIVES

The objective of the present study was to find out the differences in risk factors among medical students likely versus not likely to develop type 2 diabetes mellitus.

REVIEW OF LITERATURE

DIABETES MELLITUS

Diabetes mellitus is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both..[1]

Diabetes mellitus at present

Several distinct types of DM exist and are caused by a complex interaction between 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.[1,10]

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.[1,11]

CLASSIFICATION OF DIABETES MELLITUS

DM is classified on the basis of the pathogenic process that leads to hyperglycemia, as opposed to earlier criteria such as age of onset or type of therapy. The two broad categories of DM are designated as[11]

- Type 1
- Type 2

Type 1 diabetes is the result of complete or near-total insulin deficiency. Type 2 DM is a heterogeneous group of disorders characterized by variable degrees

of insulin resistance, impaired insulin secretion, and increased glucose production. Distinct genetic and metabolic defects in insulin action and/or secretion give rise to the common phenotype of hyperglycemia in type 2 DM and have important potential therapeutic implications now that pharmacologic agents are available to target specific metabolic derangements. Type 2 DM is preceded by a period of abnormal glucose homeostasis classified as impaired fasting glucose (IFG) or impaired glucose tolerance (IGT).[11]

Spectrum of glucose homeostasis and diabetes mellitus[12]

Type of diabetes	**Normal glucose tolerance (NGT)**	**Hyperglycemia**			
		Impaired fasting glucose or impaired glucose tolerance	**Diabetes mellitus**		
			Not insulin required	**Insulin required for control**	**Insulin required for survival**
Type 1	←	———	———	———	→
Type 2	←	———	———	→	
Other Specific types		———	———	———	- - - →
Gestational diabetes	←	———	———	→	
Time (years)		———	———	———	→
FPG (mg/dl)	< 100	100-125		≥ 126	
2-h plasma glucose (mg/dl)	< 140	140 – 199		≥ 200	

Etiologic classification of diabetes mellitus[11]

I. Type 1 diabetes (-cell destruction, usually leading to absolute insulin deficiency)

A. Immune-mediated

B. Idiopathic

II. Type 2 diabetes (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly insulin secretory defect with insulin resistance)

III. Other specific types of diabetes

A. Genetic defects of β-cell function characterized by mutations in :

1. Hepatocyte nuclear transcription factor (HNF) 4α maturity onset diabetes of young (MODY) 1
2. Glucokinase (MODY 2)
3. HNF – 1α (MODY 3)
4. Insulin promoter factor (IPF) 1 (MODY 4)
5. HNF – 1β (MODY 5)
6. Neuro D1 (MODY 6)
7. Mitochondrial deoxyribo nucleic acid (DNA)
8. Sub units of adenosine triphosphate (ATP) – sensitive potassium channel.
9. Proinsulin or insulin conversion

B. Genetic defects in insulin action.

   1. Type A insulin resistance
   2. Leprechaunism
   3. Rabson-Mendenhall syndrome
   4. Lipodystrophy syndromes.

C. Diseases of the exocrine pancreas – pancreatitis, pancreatectomy, neoplasia, cystic fibrosis, hemochromatosis, fibrocalculouspancreatopathy.

D. Endocrinopathies – acromegaly, Cushing's syndrome, glucagonoma, pheochromocytoma, hyperthyroidism, somatostatinoma, aldosteronoma

E. Drug or chemical induced – Vacor, pentamidine, nicotinic acid, glucocorticoids, thyroid hormone, diazoxide, beta-adrenergic agonists, thiazides, phenytoin, α– interferon, protease inhibitors, clozapine, beta blockers.

F. Infections – congenital rubella, cytomegalovirus, coxsackie.

G. Uncommon forms of immune-mediated diabetes – "stiff-man" syndrome, anti-insulin receptor antibodies.

H. Other genetic syndromes sometimes associated with diabetes – Down's syndrome, Klinefelter's syndrome, Turner's syndrome, Wolfram's syndrome, Friedreich's ataxia, Huntington's chorea, Laurence-Moon-Biedl syndrome, myotonic dystrophy, porphyria, Prader-Willi syndrome.

IV. Gestational diabetes mellitus (GDM)

Epidemiology

Worldwide

The prevalence of diabetes for all age-groups worldwide was estimated to be 2.8% in 2000 and 4.4% in 2030. There are 382 million people living with diabetes worldwide. The worldwide prevalence of diabetes in adults (aged 20-79 years) was 135 million in 1995, 285 million in 2010 and is expected to rise upto 300 million in 2025 and 439 million in 2030. Statistics showed significant increase (150 million) of diabetes in adults from 1995 to 2010. By 2035, 592 million people or 1 in 10 people will have diabetes. Currently 316 million people are at high risk of developing type 2 diabetes(pre-diabetes).[13-19]

Top ten countries with diabetes and number of people with age 20–79 years[20]

Serial number	Country / territory	Number (Million)
1.	China	98.40
2	Indian	65.10
3	United states of America	24.40
4	Brazil	11.90
5	Russian federation	10.90
6	Mexico	8.70
7	Indonesia	8.50
8	Germany	7.60
9	Egypt	7.50
10	Japan	7.20

The above table presents survey of the year 2013 on diabetes affected top 10 countries and their number of diabetic people at age group of 20-79. According to the International Diabetes Federation survey in the year of 2013, nearly 98.4 million people with diabetes (at 20 - 79 years) live in China, is the top most country and India is second in place i.e., nearly 65.1 million.[13-19]

It is estimated that 366 million people had DM in 2011; by 2030 this would have risen to 552 million. The number of people with type 2 DM is increasing in every country with 80% of people with DM living in low- and middle-income countries. DM caused 4.6 million deaths in 2011. It is estimated that 439 million people would have type 2 DM by the year 2030. The incidence of type 2 DM varies substantially from one geographical region to the other as a result of environmental and lifestyle risk factors.[21]

A 2011 Centre for Disease Control and Prevention (CDC) report estimates that DM affects about 25.8 million people in the US (7.8% of the population) in 2010 with 90% to 95% of them being type 2 DM.[22]

Globally, age-standardized prevalence of DM was found to be 9.8% in men and 9.2% in women with observed regional disparity, as a high prevalence of DM was found in South Asia, Latin America, the Caribbean, Central Asia, North Africa, and the Middle East.[23]

It is predicted that the prevalence of DM in adults of which type 2 DM is becoming prominent will increase in the next two decades and much of the increase will occur in developing countries where the majority of patients are aged between 45 and 64 years.[24] It is projected that the latter will equal or even exceed the former

in developing nations, thus culminating in a double burden as a result of the current trend of transition from communicable to non-communicable diseases.[25]

Indian scenario

According to The International Diabetes Federation (IDF) estimation, India will have rise in people living with diabetes up to 87.0 million by 2030 from 50.8 million (2010), making it the 'Diabetes Capital' of the world.[26-28]

This prevalence is increasing not only in urban but also in rural area. According to the World Health Organization (WHO) criteria, the prevalence of known diabetes was 5.6% and 2.7% among urban and rural areas, respectively.[29]

In India, diabetes mellitus is considered to be a disease of grave concern not only because of rapidly increasing prevalence of this disease, but also because various studies have shown rising prevalence of diabetes in young and middle aged people. This is mainly due to the economic transition, rapid urbanization and changing lifestyles, tobacco use, excessive alcohol consumption, and insufficient physical activity which are the major risk factors for diabetes mellitus.[30]

Disparity within country was observed in India as in urban areas the prevalence of DM is from 5.9% to 12.1% (North: 8.6% to 11.6%; South: 13.5% to 19.5%).[31,32] In addition to urban India, rural India also found high prevalence of DM (about 2.0% to 10.0%).[31] A systematic review for DM in tribal population of India observed a ranging prevalence of 0.7% to 10.0%, with a final estimate of 5.9%.[32]

However, more recent studies based on urban populations or rapidly developing regions have reported a higher prevalence of diabetes i.e.10.1%,[33,34]

while other studies from rural Indian populations have demonstrated an even higher prevalence i.e.12.5%–13.2%.[35,36]

Recently, in Karnataka, Rao CR. et al.[37] reported overall prevalence of diabetes as 16%. Increasing age showed two-fold, four-fold, and six-fold higher odds for 40 – 49, 50 – 59, and ≥ 60 years age group, respectively, as compared to the 30 - 39 year age group ($P < 0.001$). In the high socioeconomic strata, 32% of the subjects had diabetes ($P = 0.018$ unadjusted odds ratio 3.29, 95% CI = 1.40 – 7.74).

It had been evident that the Asian Indians are more susceptible to risk factors like age, adiposity (based on BMI), and central obesity (WHR). Despite the low BMI among Asian Indians as compared to other ethnic groups, BMI is strongly associated with glucose tolerance.[38]

Sex

Type 2 DM is slightly more common in older women than men.[26] A nationwide survey across India showed 1.3% prevalence of self-reported DM, which was more in men (1.5%) as compared to women (1.0%).[32]

Age

While type 2 diabetes mellitus traditionally has been thought to affect individuals older than 40 years, it is being recognized increasingly in younger persons, particularly in highly susceptible racial and ethnic groups and the obese. In some areas, more type 2 than type 1 diabetes mellitus is being diagnosed in prepubertal children, teenagers, and young adults. Virtually all cases of diabetes mellitus in older individuals are type 2.[39]

Mortality

In 2012 diabetes and its complications resulted in 1.5 million deaths worldwide making it the 8th leading cause of death and more than 80% of diabetic deaths occurring in low and middle-income countries. More than 21 million live births were affected by diabetes during pregnancy and > 79,000 children developed type 1 diabetes in 2013.[13-19]

CRITERIA FOR THE DIAGNOSIS OF DIABETES MELLITUS[1,2]

- Symptoms of diabetes plus random blood glucose concentration more than 11.1 mmol/L (200 mg/dL)[a] *or*
- Fasting plasma glucose more than 7.0 mmol/L (126 mg/dL)[b] *or*
- Two-hour plasma glucose more than 11.1 mmol/L (200 mg/dL) during an oral glucose tolerance test[c]

Note: In the absence of unequivocal hyperglycemia and acute metabolic decompensation, these criteria should be confirmed by repeat testing on a different day.

[a]Random is defined as without regard to time since the last meal.

[b]Fasting is defined as no caloric intake for at least 8 h.

[c]The test should be performed using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water; not recommended for routine clinical use.

SCREENING[1]

The most widely accepted glucose-based criteria for diagnosis are fasting plasma glucose (FPG) ≥126 mg/dL or a 2-h plasma glucose ≥200 mg/dL during an oral glucose tolerance test (OGTT) on more than one occasion. In a patient with classic symptoms of diabetes, a single random plasma glucose ≥200 mg/dL is considered diagnostic. Before 2010 virtually all diabetes societies recommended blood glucose analysis as the exclusive method to diagnose diabetes.

Notwithstanding these guidelines, over the last few years many physicians have been using hemoglobin A1C to screen for and diagnose diabetes. Although considered the "gold standard" for diagnosis, measurement of glucose in the blood is subject to several limitations, many of which are not widely appreciated. Measurement of A1C for diagnosis is appealing but has some inherent limitations.[40]

These issues have become the focus of considerable attention with the recent publication of the Report of the International Expert Committee that recommended the use of A1C for diagnosis of diabetes,[41] a position that has been endorsed (at the time of writing) by the ADA,[42] the Endocrine Society, and in a more limited fashion by American Association of Clinical Endocrinologists/American College of Endocrinology.[43]

Current criteria for the diagnosis of diabetes[44]

- A1C ≥6.5%. The test should be performed in a laboratory using a method that is National Glycohemoglobin Standardization Program (NGSP)-certified and standardized to the Diabetes Control and Complications Trial (DCCT) assay
- Fasting plasma glucose (FPG) ≥126 mg/dl (7.0 mmol/l). Fasting is defined as no caloric intake for at least 8 h, or
- 2-h plasma glucose ≥200 mg/dl (11.1 mmol/l) during an oral glucose tolerance test (OGTT). The test should be performed as described by the World Health Organization, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water

- In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose ≥200 mg/dl (11.1 mmol/l)
- In the absence of unequivocal hyperglycemia, result should be confirmed by repeat testing.

Pathophysiology of type 2 diabetes mellitus

Type 2 DM is characterized by impaired insulin secretion, insulin resistance, excessive hepatic glucose production, and abnormal fat metabolism. Obesity, particularly visceral or central (as evidenced by the hip-waist ratio), is very common in type 2 DM (80% or more are obese). In the early stages of the disorder, glucose tolerance remains near-normal, despite insulin resistance, because the pancreatic beta cells compensate by increasing insulin output. As insulin resistance and compensatory hyperinsulinemia progress, the pancreatic islets in certain individuals are unable to sustain the hyperinsulinemic state. Impaired Glucose Tolerance, characterized by elevations in postprandial glucose, then develops. A further decline in insulin secretion and an increase in hepatic glucose production lead to overt diabetes with fasting hyperglycemia. Ultimately, beta cell failure ensues.[1]

For type 2 diabetes mellitus to occur, both insulin resistance and inadequate insulin secretion must exist. For example, all overweight individuals have insulin resistance, but diabetes develops only in those who cannot increase insulin secretion sufficiently to compensate for their insulin resistance. Their insulin concentrations may be high, yet inappropriately low for the level of glycemia.[39]

A role for excess glucagon cannot be underestimated; indeed, type 2 diabetes is an islet paracrinopathy in which the reciprocal relationship between the glucagon-

secreting alpha cell and the insulin-secreting beta cell is lost, leading to hyperglucagonemia and hence the consequent hyperglycemia.[45]

With prolonged diabetes, atrophy of the pancreas may occur. A study by Philippe et al used computed tomography (CT) scan findings, glucagon stimulation test results, and fecal elastase-1 measurements to confirm reduced pancreatic volume in individuals with a median 15-year history of diabetes mellitus (range, 5-26 years).[46] This may also explain the associated exocrine deficiency seen in prolonged diabetes.

Beta-cell dysfunction

Beta-cell dysfunction is a major factor across the spectrum of prediabetes to diabetes. A study of obese adolescents by Bacha et al confirms what is increasingly being stressed in adults as well: Beta-cell dysfunction develops early in the pathologic process and does not necessarily follow the stage of insulin resistance.[47] Singular focus on insulin resistance as the "be all and end all" is gradually shifting, and hopefully better treatment options that address the beta-cell pathology will emerge for early therapy.

Insulin resistance

In the progression from normal to abnormal glucose tolerance, postprandial blood glucose levels increase first. Eventually, fasting hyperglycemia develops as suppression of hepatic gluconeogenesis fails. During the induction of insulin resistance (such as occurs with a high-calorie diet, steroid administration, or physical inactivity), increased glucagon levels and increased glucose-dependent

insulinotropic polypeptide (GIP) levels accompany glucose intolerance. However, the postprandial glucagonlike peptide-1 (GLP-1) response is unaltered.[48]

Genomic factors

Genome-wide association studies of single-nucleotide polymorphisms (SNPs) have identified a number of genetic variants that are associated with beta-cell function and insulin resistance. Some of these SNPs appear to increase the risk for type 2 diabetes. Over 40 independent loci demonstrating an association with an increased risk for type 2 diabetes have been shown.[49]

A subset of the most potent are shared below:[39,50]

- Decreased beta-cell responsiveness, leading to impaired insulin processing and decreased insulin secretion (*TCF7L2)*
- Lowered early glucose-stimulated insulin release (*MTNR1B, FADS1, DGKB, GCK*)
- Altered metabolism of unsaturated fatty acids (*FSADS1*)
- Dysregulation of fat metabolism (*PPARG*)
- Inhibition of serum glucose release (*KCNJ11*)
- Increased adiposity and insulin resistance (*FTO* and *IGF2BP2*)
- Control of the development of pancreatic structures, including beta-islet cells (*HHEX*)
- Transport of zinc into the beta-islet cells, which influences the production and secretion of insulin (*SLC30A8*)
- Survival and function of beta-islet cells (*WFS1*)

Susceptibility to type 2 diabetes may also be affected by genetic variants involving incretin hormones, which are released from endocrine cells in the gut and stimulate insulin secretion in response to digestion of food. For example, reduced beta-cell function has been associated with a variant in the gene that codes for the receptor of gastric inhibitory polypeptide (*GIPR*).[51]

The high mobility group A1 (HMGA1) protein is a key regulator of the insulin receptor gene (*INSR*).[52] Functional variants of the *HMGA1* gene are associated with an increased risk of diabetes.[39]

Complications of diabetes mellitus

Acute Complications of DM

Diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar state (HHS) are acute complications of diabetes. DKA was formerly considered a hallmark of type 1 DM, but it also occurs in individuals who lack immunologic features of type 1 DM and who can subsequently be treated with oral glucose-lowering agents (these obese individuals with type 2 DM are often of Hispanic or African-American descent). HHS is primarily seen in individuals with type 2 DM. Both disorders are associated with absolute or relative insulin deficiency, volume depletion, and acid-base abnormalities. DKA and HHS exist along a continuum of hyperglycemia, with or without ketosis. Both disorders are associated with potentially serious complications if not promptly diagnosed and treated.[10,11]

Chronic Complications of DM

The chronic complications of DM affect many organ systems and are responsible for the majority of morbidity and mortality associated with the disease. Chronic complications can be divided into vascular and nonvascular complications. The vascular complications of DM are further subdivided into microvascular (retinopathy, neuropathy, nephropathy) and macrovascular complications [coronary artery disease (CAD), peripheral arterial disease (PAD), cerebrovascular disease]. Nonvascular complications include problems such as gastroparesis, infections, and skin changes. Long-standing diabetes may be associated with hearing loss. Whether type 2 DM in elderly individuals is associated with impaired mental function is not clear.[11]

Chronic complications of diabetes mellitus

1. Microvascular
    a. Eye disease
        i. Retinopathy (nonproliferative/proliferative)
        ii. Macular edema
    b. Neuropathy
        i. Sensory and motor (mono- and polyneuropathy)
        ii. Autonomic
    c. Nephropathy
2. Macrovascular
    a. Coronary artery disease
    b. Peripheral arterial disease

c. Cerebrovascular disease

3. Other

    a. Gastrointestinal (gastroparesis, diarrhea)

    b. Genitourinary (uropathy/sexual dysfunction)

    c. Dermatologic

    d. Infectious

    e. Cataracts

    f. Glaucoma

    g. Periodontal disease

The risk of chronic complications increases as a function of the duration of hyperglycemia; they usually become apparent in the second decade of hyperglycemia. Since type 2 DM often has a long asymptomatic period of hyperglycemia, many individuals with type 2 DM have complications at the time of diagnosis.[54]

The Microvascular complications of both type 1 and type 2 DM result from chronic hyperglycemia. Large, randomized clinical trials of individuals with type 1 or type 2 DM have conclusively demonstrated that a reduction in chronic hyperglycemia prevents or delays retinopathy, neuropathy and nephropathy. Other incompletely defined factors may modulate the development of complications.[55,56]

TYPE 2 DIABETES MELLITUS IN YOUNG

Type 2 diabetes was hitherto common after the age of 40 years. However, in recent times individuals aged 25-40 years have been presenting with clinical features of type 2 diabetes.[57] The incidence of type 2 diabetes in children is reported to be increasing in several parts of the world. Among the native North Americans, 30% of the new cases of type 2 diabetes occur in the second decade of life,[58] while among the Japanese school children, type 2 diabetes is 7 times more common than type 1 and a similar trend is reported in the Chinese and Mexican American youth.[58-60]

More recently the prevalence of type 2 diabetes among Japanese subjects aged 10–19 and 20–29 years were ~50% and ~75% respectively.[61] In Finland, between 1992–6 there was a rapid increase in the incidence of type 2 diabetes among young adults of 20–40 years.[62] Early onset type 2 diabetes has also been reported in China, Mexico, India and Australia.[63]

In Pima Indians there is an increase in the incidence in the age group of 10 years and above.[64] Type 2 diabetes presents a decade or two earlier in India than in the West. About 38% of type 2 diabetics are diagnosed below the age of 40 years and in about 4.8% a diagnosis is made below 25 years.[65]

The distribution of patients based on the age of onset, among 4833 consecutive patients registered at Sahay's Diabetic Clinic and Research Centre, Hyderabad, during the period 1999-2002 showed 36.6% of the patients with type 2 diabetes had an onset below the age of 40 years and 7.5% had an onset of diabetes below the age of 30 years.[57]

The younger age of onset of type 2 diabetes is driven by the increasing obesity in the younger age group. Hillier et al.[66] elegantly showed the inverse relationship between obesity and age of diagnosis of type 2 diabetes. Obesity has increased by 70% in adults aged 18–29 years and type 2 diabetes increased by 70% in adults 30–39 years of age making young adults the fastest growing group for both conditions.[67]

Among adolescents (12–19 years), in the USA, the prevalence of obesity has increased from 5% to 17.4% between the late 1970s and 2004.[68] Associated with the rise in obesity is the concomitant increase in metabolic syndrome. Features of metabolic syndrome in childhood in particular, obesity, abnormal glucose metabolism and dyslipidaemia, are predictive of onset of type 2 diabetes in adults below 30 years of age.[69] Of concern is the rise of obesity in childhood and adolescence which has led to the rise in diabetes-associated hospitalisations among young adults. In the UK, hospital admissions in type 2 diabetic patients below 18 years of age rose by approximately 45% between 1996–97 and 2003–04. This parallels the 63% rise in patient admission for obesity in the same period.[70] In the US, diabetes related hospitalisation rate has increased by ~40% between 1993–2004 among those aged 20–29 years.[71]

This rising prevalence of type 2 diabetes in children will expose them to the risk of developing the macrovascular and microvascular complications during the prime of their life – their earning period, which will have an adverse impact on the economy.

The epidemic of type 2 diabetes is a manifestation of globalization. The advent of westernization has resulted in a drastic change in the life style of both adults and children. There is a frightening increase in physical inactivity, unhealthy food habits and obesity in children and adults. When obesity develops in childhood years it generally continues into adulthood and frequently becomes more severe. The health consequences of obesity in adults are well established including greater rates of hyperinsulinemia, hypertension, glucose intolerance and coronary heart disease (CHD) – together also called as syndrome X by Reaven. This is often a precursor of diabetes and antedates the development of diabetes by 7-8 years.[72,73]

Another important factor, which might contribute to the increase in type 2 diabetes, is low birth weight, especially in the developing countries. A number of studies have demonstrated an association between low birth weight and the development of insulin resistance in later life. The thrifty phenotype hypothesis proposes that poor nutrition in fetal and infantile life is detrimental to the development and function of beta cells and insulin sensitive tissue leading to insulin resistance under the stress of obesity. Greater degrees of insulin resistance have been reported in Indians with type 2 diabetes as compared to other populations. Higher levels of insulin after a glucose load have been reported among Asian Indians.[57]

The two common non-immune forms of diabetes encountered in the young are true early onset type 2 diabetes and MODY. Most children with type 2 diabetes are overweight or obese at diagnosis and present with glycosuria without ketonuria, 45-80% of these children have a family history of diabetes, with at least one parent being affected. There may be history of diabetes in first or second degree relatives also.[57]

Acanthosis nigricans and polycystic ovarian syndrome (PCOS) associated with insulin resistance are common among them. Velvety hyperpigmented patches predominantly seen on the nape of the neck, axilla and groin (intertrigenous areas) characterize acanthosis. Presence of acanthosis in a diabetic is a hallmark of type 2 diabetes. Majority of these children (90%) are diagnosed between the ages of 10-18 years.[74]

Puberty appears to play a major role in the development of type 2 diabetes mellitus. The increased growth hormone secretion during this period is responsible for the insulin resistance during puberty. In those with a genetic predisposition for insulin resistance, the environmental factors may tilt the balance towards development of type 2 diabetes during this period. 5-25% of the patients of type 2 diabetes may also have ketonuria at onset.[57]

Risk factors for type 2 diabetes in young

Non modifiable risk factors

Race/ethnicity

The prevalence of type 2 varies considerably among populations of different ethnic origins living in apparently similar environments.[75] For example, in Singapore the frequency of diabetes in 1992 was 8.5–7.7% in Chinese men and women aged 18–69 compared with 13.3 and 12.3%, respectively, among the Asian Indians and Malaysians.[76]

High prevalence rates of diabetes have also been found among Asian Indians compared with the indigenous populations in the United Kingdom, Fiji, South

Africa and in the Caribbean. Considerable differences in the prevalence of diabetes have also been described among the multi-ethnic populations of Hawaii and New Zealand, where the Native Hawaiians and Maori populations, both of Polynesian origin, have higher prevalences than other ethnic groups. While environmental factors undoubtedly account for some of these differences, they are likely also to reflect inherent ethnic differences in susceptibility to the disease.[75]

Familial aggregation

The empirical risk of developing type 2 diabetes is increased 2 to 6-fold if a parent or sibling has the disease.[77] Consequently, a positive family history is a practical, albeit a crude way, of estimating if an individual is likely to have inherited susceptibility to the disease. On the other hand, familial aggregation may occur for non-genetic reasons. Family members often share a similar environment, particularly as children and in adolescence, thus familial aggregation alone is not definitive evidence of genetic determinants. Furthermore, with a disease as frequent as type 2 diabetes two or more family members may well have the disease by chance alone.[75]

Genetic factors

A higher degree of concordance for type 2 diabetes in identical twins than in dizygotic twins provides strong evidence that genetic factors are important in determining susceptibility. However, the fact that not all monozygotic twins are concordant for the disease confirms the importance of environmental factors. Further evidence of the importance of genetic factors as predisposing factors for type 2 diabetes comes from studies of admixed populations. Differences in prevalence among persons of mixed racial background from that in parent populations with

notably different prevalence of the disease are indicative of the importance of genetic determinants. Such relationships have been described among Nauruans and Pima Indians where full-heritage members of these groups have significantly higher rates of diabetes than those of mixed heritage. Similarly, among the Mexican American population of San Antonio, the prevalence of type 2 diabetes is related to the degree of American Indian admixture, with higher rates associated with greater proportions of American Indian genes. Much research activity has centred on attempts to unravel the genes, which confer susceptibility to type 2 diabetes, a number of genes are likely to be involved. At present, it is impossible to quantify the relative contributions of genetic and environmental factors.[75]

Modifiable risk factors

These factors have been shown to have an increased or a decreased risk for the development of type 2 diabetes and can be modified by lifestyle changes.

Insulin resistance in young persons

The development of type 2 diabetes involves a loss of the balance between insulin sensitivity and secretion, as has been reported in adults, where the normal inverse relationship between the two factors leads to a constant glucose disposition index in a given person, with decline in this parameter being associated with the development of IGT and type 2 diabetes. Insulin resistance in the young has been reported in a variety of ethnic groups and is strongly associated with obesity. Furthermore, obese children exhibit glucose intolerance, which is strongly associated with evidence of both insulin resistance and impaired insulin secretion.[78]

There are important ethnic differences in the degree of insulin resistance. In a study comparing 22 black and 22 white nonobese prepubertal children, the former group was found to have a significant decrease in insulin sensitivity with hyperinsulinemia, showing, however, lower glucose disposition indexes, suggesting an increase in ultimate diabetes risk. Circulating levels of the insulin-sensitizing adipocyte secretory product adiponectin were ~60% higher in white children. Important dietary differences were found, with the black children consuming 10% fewer calories from carbohydrates and showing a 36% increase in the dietary fat-to-carbohydrate ratio, which had strong negative correlation with insulin sensitivity. Whether this is causally related to metabolic abnormalities remains to be determined.[78]

One important determinant of obesity may be the relative propensity to retain fat in adipose tissue, with evidence that rates of lipolysis are lower in black than in white boys and girls.[78]

Comparing obese and normal-weight black and white adolescents, insulin sensitivity is decreased with obesity regardless of ethnicity, showing inverse correlation with body fat. A number of studies have shown that black children have higher total fat and cholesterol intake, prefer greater sweetness in liquids, are physically less active, and spend more time watching television. Black girls have higher total energy intake than whites, do not perceive themselves as heavy, and actually express a desire to be on the fat side. Clearly, then, there must be a complex interplay of cultural/environmental and genetic factors explaining the metabolic differences observed between the two ethnic groups.[78]

Of particular importance as a determinant of insulin resistance is central obesity. In a cross-sectional study of 14 adolescents with IGT matched with 14 control subjects of similar age, BMI, body fat, and leptin, the children with IGT were insulin resistant, with increased intramyocellular fat measured by ^{1}H nuclear magnetic resonance spectroscopy showing strong correlation with insulin sensitivity and with 2-h postload plasma glucose. Those with IGT had higher visceral and lower subcutaneous abdominal fat and decreased first-phase insulin secretion and glucose disposition index. Comparing black and white children with obesity and similar insulin sensitivity levels, blacks have lower hepatic glucose output, lower total and LDL cholesterol, and lower triglyceride levels, with considerably lower visceral fat levels. Blacks who do have visceral obesity, however, have a fall in the glucose disposition index, suggesting greater diabetogenic risk of obesity among blacks, but greater atherogenic risk among whites. Important additional risk of diabetes is seen among black children with a positive family history of diabetes, who show an ~20% lowering of insulin sensitivity in the first decade of life.[78]

A major cause of insulin resistance is puberty. Insulin sensitivity decreases by ~30% during puberty with compensatory increase in insulin secretion. Insulin action decreases similarly during puberty in black and white children. The further metabolic derangement of PCOS is associated with decreased glucose disposition, with ~30% of adolescent girls with PCOS having IGT and 4% type 2 diabetes. Adolescents with PCOS who develop IGT have similar degrees of obesity and elevations in circulating testosterone to those with normal glucose tolerance but show blunting of first-phase insulin secretion in response to intravenous glucose with a consequent decrease in the glucose disposition index.[78]

Obesity

Obesity is a frequent concomitant of type 2 diabetes, and in many longitudinal studies has been shown to be a powerful predictor of its development.[79] Obesity has increased rapidly in many populations in recent years because of an interaction between genetic and environmental factors. These include: metabolic characteristics; physical inactivity; habitual energy intake in relation to expenditure; and macronutrient composition of the diet. This increase in obesity has been accompanied by an increasing prevalence of type 2 diabetes. Since obesity is such a strong predictor of diabetes incidence, it appears that the rapid increases in the prevalence of type 2 diabetes seen in many populations in recent decades are almost certainly related to increasing obesity.[75] Data from the Nurses' Health Study suggest that the lowest risk of diabetes occurs in individuals who have a body mass index (BMI) >21, with increasing prevalence seen as obesity levels increase.[80]

There are large differences in age specific incidence rates according to BMI in the Pima Indians. Those with higher BMI have much higher incidence rates of type 2 diabetes at earlier ages than those with lower BMI among whom the incidence rises in the older age groups. In non-obese individuals, the incidence of type 2 diabetes is low even in populations such as the Pima Indians where the overall risk of the disease is very high. The relationship of incidence of type 2 diabetes to obesity also varies with other risk factors. For example, in the Pima Indians the incidence rises much more steeply with BMI in those whose parents have diabetes than in these who do not. This relationship indicates an interaction between risk factors. Several studies indicate that waist circumference or waist-to-hip ratio may be a better indicator of the risk of developing diabetes than BMI. Such

data suggest that the distribution of body fat is an important determinant of risk as these measures reflect abdominal or visceral obesity. In Japanese American men, for example, the intra-abdominal fat, as measured from CAT scans, was the best anthropometric predictor of diabetes incidence. Given the importance of central adiposity as a determinant of diabetes risk it is necessary to consider whether the usually quoted 'normal range' for BMI (18.5–24.9kg/m^2) is appropriate for all populations. It might be appropriate to also suggest an appropriate range for some measure of the distribution of body fat (e.g. waist circumference, waist/hip ratio).[75]

However, for a given BMI, several (perhaps all) populations of Asian descent appear to have an appreciably greater proportion of body fat than that of Europeans. It seems conceivable, therefore, that a lower BMI might be desirable. In the absence of definitive data from prospective studies in these countries, at present it may be appropriate to similarly suggest an optimum level towards the lower end of the normal range. On the other hand, people of Pacific descent (Polynesians) have a relatively high proportion of lean body mass compared with Europeans for any given BMI. Therefore, a higher BMI cut off may be acceptable. However, the particularly high risk of type 2 diabetes and other co-morbidities of obesity in these populations may negate this apparently beneficial anthropometric attribute. Again, in the absence of appropriate prospective studies, it may be wise to suggest that their BMI should not exceed the conventional normal range. Because there are fewer data available concerning waist circumference or waist/hip ratio in different populations, it is appropriate to continue to use the WHO recommended BMI range (18.5–24.9kg/m^2) and population mean of 21kg/m^2.[75]

The prevalence rates of obesity (BMI exceeding the 95th percentile) among U.S. children and adolescents aged 6–11 and 12–19 years, respectively, were 4.2 and 4.6% in 1963–1970, 4.0 and 6.1% in 1971–1974, 6.5 and 5.0% in 1976–1980, 11.3 and 10.5% in 1988–1994, and 15.3 and 15.5% in 1999–2000, an alarming rate of increase. Obesity (weight corrected for height >95th percentile) among U.S. children increased between 1988 and 1999 from 7 to 10% among those aged 2–5 years.[78]

In a cross-sectional survey of children 9–12 years old in Hong Kong, 38% of girls, but 57% of boys, were overweight, with overweight children of both sexes showing higher systolic blood pressure, triglyceride, and insulin and lower HDL cholesterol than the normal-weight group.[81]

In Australia, ~5% of children are currently obese and an additional 16% overweight (BMI 85th to 95th percentile).[82] These prevalences doubled over the past decade after being nearly stable around 10% from 1969 to 1985. There appear to be ethnic differences within countries, with African-American and Hispanic children aged 4–12 years in the U.S. showing an increase to 22% prevalence of overweight between 1986 and 1998, while non-Hispanic whites showed no significant change with a 12% overweight prevalence. It is noteworthy that BMI may underestimate the prevalence of obesity in young people.[78]

Recent analysis of trends in British youth suggest that waist circumference has increased more rapidly than BMI over the past two decades, with 14 and 17% of boys and girls, respectively, exceeding the 98th percentile in this measure in 1997, while 10 and 8% exceed the 98th percentile for BMI; both measures exceeded the 98th percentile only in 2–3% of adolescents between 1977 and 1987.[83]

These considerations suggest that the phenomenon of increasing type 2 diabetes among children and adolescents may be a result of increasing obesity and, particularly, of increasing central obesity. There is a strong relationship between childhood obesity and the development of insulin resistance in early adulthood. Fasting insulin levels show correlation with blood pressure and triglyceride and inverse correlation with HDL cholesterol levels, important components of the IRS.[78]

Physical inactivity

Numerous studies have indicated the importance of physical inactivity in the development of type 2 diabetes.[84-87] Indeed, in most studies its relative importance may be underestimated because of imprecision in measurement. In the Nurses' Health Study, women who reported exercising vigorously had an age-adjusted incidence rate of self-reported clinically diagnosed diabetes that was two-thirds as high as that of women who exercised less frequently.[85] The deleterious effect of low levels of physical activity is seen particularly among those subjects who have other risk factors such as high BMI, hypertension or parental diabetes. Similarly, among male physicians, the incidence of self reported diabetes was negatively related to the frequency of vigorous exercise and the strength of this relationship was greater in those with higher BMI.[85] For equivalent degrees of obesity, more physically active subjects have a lower incidence of the disease. Recommendations with regard to physical activity as a preventative measure for developing type 2 diabetes are still difficult to quantify.

Currently, guidelines propose moderate physical activity on at least 5 days per week and do not specify heart rate targets However, more recent evidence suggests that vigorous exercise is required to improve insulin sensitivity.[75]

A study by McAuley et al.[88] showed that insulin sensitivity improved in normoglycaemic insulin-resistant adults who undertook vigorous exercise and not in those who complied with current moderate exercise programmes. The vigorous exercise programme required participants to train five times a week for at least 20min per session at an intensity of 80–90% of age-predicted maximum heart rate.

Physical activity and insulin sensitivity.

Children differ from adults in metabolic response to exercise, showing a lesser increase in the intramuscular inorganic phosphate-to-phosphocreatine ratio and a lesser decrease in pH. Obesity and dietary factors may alter the expected metabolic response to exercise. This can be seen with high-fat feeding, which reduces the growth hormone response to exercise. Growth hormone and epinephrine responses to exercise are blunted in obese subjects. Both intra- and extramyocellular triglyceride stores are greater in obese than in lean children. In children, there appears to be a body composition threshold around the 75th percentile of weight for height, above which abnormalities are seen in fitness, as measured by the maximal oxygen consumption, with reductions in insulin sensitivity also seen. Physical activity increaseS insulin sensitivity in children, and this is also seen among obese children undergoing regular exercise who show a fall in fasting insulin that is reversed by a return to a sedentary lifestyle.[78]

Australian aborigines were a population exhibiting high levels of physical fitness in their traditional hunter-gatherer lifestyle, with low BMI, blood pressure, and cholesterol. With westernization, this population changed to one with high levels of unemployment, welfare dependency, poor education, overcrowded living conditions, poor health with heavy infectious disease burden, particularly among children (perhaps causing an inflammatory load), and increased lifestyle-related chronic disease among adults. The change in health and socioeconomic status is associated with central obesity, early-onset type 2 diabetes, and premature CVD with IRS features of dyslipidemia, hypertension, hyperinsulinemia, and microalbuminuria.[78]

Studies in Japan suggest that weight gain is caused by a reduction in energy expenditure among young people, with participation by young people in Japan in exercise and sports showing a consistent decrease in all age-groups.[89] Twenty-seven and 43% of high school boys and girls, respectively, in the U.S. participate in an insufficient amount of physical activity.[90]

Habitual leisure-time physical activity in girls decreases by approximately two-thirds among Caucasian girls and to an even greater extent among African-American girls in the U.S. as age increases from 9 to 18 years.[91]

On average, children's programming includes 12 food advertisements hourly, more than twice that in adult viewing, with the average child in the U.S. seeing >20,000 advertisements per year. Parental influences are also important, with the NHANES III survey[92] showing nearly one-third of mothers of overweight children to believe that the children are at "about the right weight".

Fat: quantity and quality

Both the amount and quality of dietary fat may modify glucose tolerance and insulin sensitivity. A high fat content in the diet may result in deterioration of glucose tolerance by several mechanisms including decreased binding of insulin to its receptors, impaired glucose transport, reduced proportion of glycogen synthase and accumulation of stored triglycerides in skeletal muscle. The fatty acid composition of the diet, in turn, affects tissue phospholipid composition, which may relate to insulin action by altering membrane fluidity and insulin signalling.[75]

Alcohol intake

Several studies have suggested that moderate alcohol intake is associated with a reduced incidence of type 2 diabetes.[93] Among women in the Nurses Health Study, there was a reduced incidence of diabetes in women who consumed alcohol compared with those who did not. There was a strong inverse relation between alcohol consumption and body weight, which could explain much of the apparent protective effect of alcohol consumption.[93] Among 20,000 male physicians, those consuming more than 2–4 drinks per week had a lower incidence of type 2 diabetes in the subsequent 12 years compared with non-drinkers, relationships that persisted after adjustment of BMI and other diabetes risk factors.[94]

These apparent male–female differences were examined among 12,000 45–64 year old participants in the Atherosclerosis Risk in Communities Study (ARIC).[95] After adjustment for other diabetes risk factors men consuming more than 21 drinks per week had a significant increase in the incidence of diabetes, whereas no significant association with alcohol intake was found among the women. The

apparent inconsistencies in the results of these studies preclude clear recommendations regarding alcohol in the prevention of diabetes.[75]

Intrauterine environment

There has been much recent interest in the extent to which intrauterine environment may influence the subsequent risk of developing diabetes and other diseases. Gestational diabetes, which is a strong risk factor for development of type 2 diabetes, is also considered here because of its association with overweight in pregnancy and possible intrauterine factors, which may play a role in the offspring.[75]

Gestational diabetes is more frequent among women from subgroups of the population who have a high risk of type 2 diabetes, e.g. older, overweight or obese women, certain ethnic groups. In some cases, gestational diabetes represents diabetes that was present, but undiagnosed before pregnancy, whereas in others it develops during pregnancy, most frequently towards the end of the second trimester. It is in this latter group, that following delivery glucose tolerance is likely to become normal, but such women carry a high risk for developing diabetes subsequently. The intrauterine environment influences the risk of developing type 2 diabetes. Offspring of diabetic pregnancies are often large and heavy at birth; they tend to develop obesity in childhood and are at high risk of developing type 2 diabetes at an early age. Such individuals have lower insulin secretion than similarly aged offspring of non-diabetic pregnancies.[75]

A substantial part of the excess risk of diabetes in the offspring of diabetic pregnancies appears to be the result of exposure to the diabetic intrauterine environment. Among offspring born to mothers before and after the development of

type 2 diabetes, those born after the mother developed diabetes, have a 3-fold higher risk of developing diabetes than those born before. Thus, the enhanced risk among the offspring from diabetic pregnancies among such women appears to be the result of intrauterine programming that has long-term effects on the offspring in later life. The early appearance of type 2 diabetes in female offspring increases the likelihood that their offspring in turn will be exposed to a diabetic intrauterine environment, leading to an increased prevalence of diabetes in subsequent generations.[75]

Hypertension

Hypertension is known to accompany diabetes in middle age and is a risk factor for diabetic vascular complications. Less well appreciated is how many years before the onset of diabetes blood pressure (BP) begins to rise. Previous studies show that even within the range of normal BP, prediabetic individuals have higher BP 3–16 years before diagnosis compared with individuals who remain nondiabetic.[96]

Many patients with new-onset type 2 diabetes have evidence of complications, such as retinopathy, nephropathy, and cardiovascular disease, at the time of diagnosis. Elevated BP is a risk factor for diabetes complications, and high BP before the onset of diabetes may explain the high prevalence of cardiovascular disease at the time of diabetes diagnosis. Therefore, high BP before the onset of type 2 diabetes is a potential target for the prevention of diabetes complications.[96]

The presence of hypertension may also be an indicator of the pathogenesis of type 2 diabetes. According to the “Common Soil Hypothesis”, elevated BP could be an early sign of underlying insulin resistance, related to central adiposity. An

alternative hypothesis is that elevated BP is a marker of endothelial dysfunction, which is itself a risk factor for the development of insulin resistance, type 2 diabetes, and vascular disease.[96]

Acanthosis nigricans

Acanthosis nigricans is a dermatologic condition associated in some cases with hyperinsulinemia. Children with this condition are 1.6 times to 4.2 times as likely as those without it to have hyperinsulinemia. Acanthosis nigricans is characterized by thickening and darkening of the upper layers of the skin, resulting in a velvety appearance. Typical areas of involvement include the posterior neck, the axilla, the elbows, and the knees; the neck is involved 93% to 99% of the time.[97]

The association of acanthosis nigricans with hyperinsulinemia has led to speculation of a possible further association with type 2 diabetes. The natural history of acanthosis nigricans with respect to type 2 diabetes has not been determined, but evidence suggests the former may be a risk factor for the latter. A readily apparent, rapidly identifiable physical examination marker identifying patients at increased risk for type 2 diabetes could stimulate discussions of lifestyle modifications in the primary care setting.[97]

Other risk factors

Several other risk factors have been related to the development of diabetes. These include several inflammatory markers (e.g. interleukin-6, C-reactive protein, other cytokines and acute phase reactants) and variation in levels of sex hormones (e.g. low levels of sex hormone binding globulin in women, low testosterone levels in men and women with high androgen levels).[75]

Risk profile in India

The important risk factors for the high prevalence of diabetes include High familial aggregation, Obesity especially central obesity, Insulin resistance and metabolic syndrome, Life style changes due to urbanization and Gestational diabetes.[98]

Several studies in India and abroad have shown that nearly 75% of the T2DM patients have first degree family history of diabetes, this indicates a strong familial aggregation in the Indian diabetic patient. Insulin resistance has been demonstrated to be a characteristic feature of Asian Indians.[98]

A comparative study of Asian Indians, Europeans and other ethnic groups have shown that the Asian Indians have higher insulin levels than others, at fasting and in response to glucose. Compensatory increase in insulin secretion bring about a state of chronically increased insulin and glucose levels in the blood (hyperinsulinemia and hyperglycemia) and thus is a predecessor for diabetes.[99]

Central adiposity indicates deposition of large quantities of abdominal fat, which consists of visceral fat and subcutaneous fat. Visceral fat increases the risk of diabetes and hyperlipidaemia by favouring insulin resistance. In several ethnic populations including the relatively non-obese Asians population, the android pattern of body fat, typified by more upper body adiposity measured as waist:hip ratio was found to be a greater risk factor for T2DM than general obesity.[98]

A study concluded that a continuous positive relationship of all markers of obesity (body-mass index, waist size and waist:hip ratio) with major coronary risk

factorshypertension, diabetes and metabolic syndrome while waist hip ratio also correlates with lipid abnormalities.[100]

Indian diabetic risk score (IDRS)

Mohan et al.,[101] from their Chennai Rural Epidemiology Study[5] (CURES) cohort, have developed a single user friendly Indian diabetic risk score (IDRS). Its advantages are its simplicity and low cost and it is easily applicable for mass screening programmes. IDRS is a simple, safe, and inexpensive questionnaire consisting of four simple parameters i.e. age, obesity status, exercise status, and family history of type 2 DM. The validated IDRS has been successfully implemented as a practical screening tool to assess the diabetes risk and to detect undiagnosed type 2 diabetes, it also proved suitable in prediction of metabolic syndrome and cardiovascular disease in the South Indian population which takes into consideration the age, abdominal obesity, physical activity and the family history of the patients. The IDRS has a sensitivity of 72.5% and a specificity of 60.1% and it was derived, based on the large population based studies.[101,102]

The advantage of IDRS are its simplicity, low cost and is easily applicable for mass screening programmes. IDRS should be tested in other population based studies in Indiaboth rural and urban. IDRS may be predictive of metabolic syndrome and cardiovascular disease as three of the factors [age, physical activity and waist circumference] are risk factors for both metabolic syndrome and cardiovascular disease. IDRS uses two modifiable risk factors (waist circumference and physical inactivity) and two non-modifiable risk factors (age and family history of diabetes), providing a clear message that if modifiable risk factors are altered, the risk score

can be considerably reduced. Subjects with high IDRS regardless of their blood sugar status, are ideal candidates for life style modification as these are risk factors for not only diabetes but also for cardiovascular disease. The IDRS score has been tested and approved in the CURES cohort.

METHODOLOGY

The present study was conducted in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2014 to December 2014.

Study design and duration

The study design was a one year cross-sectional study.

Study period and duration

This study was conducted for the of one year from January 2014 to December 2014.

Source of Data

This study was carried out under the Department of Medicine KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. Medical students pursuing MBBS at Jawaharlal Nehru Medical College, Belgaum formed the study sample.

Sample size

A total of 200 medical students were included in the study.

Sampling procedure

The sample size (n) was calculated using the following formula:

$$\mathbf{n = z^2_{1-\alpha/2} * p * q / d^2}$$

Where,

n = Sample size

$z^2_{1-\alpha/2}$ = 1.96 constant at 95% confidence interval

p = Positivity of waist circumference of 90 cms in males (35%)

q = 100 – p = 100 – 35 = 65

d = Absolute error which was considered as 20% of p = 7

Therefore,

$n = 1.96^2 \times 35 \times 65 / 7^2$

n = 200

Hence the sample size of 200 was considered.

Selection criteria

Inclusion

- Medical students pursuing MBBS at Jawaharlal Nehru Medical College, Belgaum.
- Medical students with age >18 years.

Exclusion

- Known diabetic students
- Students on corticosteroids
- Not willing to participate

Ethical clearance

Prior to the commencement, the study was approved by the Ethical and Research Committee of Jawaharlal Nehru Medical College, Belgaum. Also the permission was obtained from Principal, Jawaharlal Nehru Medical College, Belgaum.

Informed consent

Demographic data like gender and age were collected along with relevant history. Medical students were interviewed for family history (cardiovascular, hypertensive and diabetic history), type of regular food intake (vegetarian, non-vegetarian), smoking and alcohol intake. Students were also assessed for the routine physical activity and the responses were recorded on predesigned and pretested proforma.

Examination

Body mass index

A thorough clinical examination was conducted. Height and weight was recorded and body mass index was calculated based on the following formula;

$$\text{Body Mass Index} = \frac{\text{Weight (Kg)}}{\text{Height}^2 \text{ (m)}}$$

Body mass index was classified according to Overweight and obesity by BMI in adult Asians as below.[102]

Classification	BMI (Kg/m^2)
Underweight	< 18.5
Normal range	18.5 to 22.9
Overweight	≥ 23
At risk	23.0 to 24.9
Overweight	25.0 to 29.9
Obese	≥ 30.0

Abdominal obesity

The abdominal obesity was measured by using a measuring tape at the mid-point below the lower rib cage and the highest point of the iliac crest. The measurements were taken with the subjects in minimum clothes and when they were breathing quietly at the end of their expirations.

Indian Diabetic Risk Score[101,102]

IDRS is a simple, safe, and inexpensive questionnaire consisting of four simple parameters i.e. age, obesity status, exercise status, and family history of type 2 DM. The validated IDRS has been successfully implemented as a practical screening tool to assess the diabetes risk and to detect undiagnosed type 2 diabetes, it also proved suitable in prediction of metabolic syndrome and cardiovascular disease in the South Indian population. which takes into consideration the age,

abdominal obesity, physical activity and the family history of the patients. The MDRF-IDRS has a sensitivity of 72.5% and a specificity of 60.1% and it was derived, based on the large population based studies. In this study, all the students were assessed for IDRS. The components of IDRS are as given in Table.

IDRS scoring system to predict the risk of diabetes mellitus

Variable	Sub-groups	Score
Age	< 35	0
	35-49	20
	≥ 50	30
Abdominal Obesity (Cms)	<80 female; < 90 male	0
(Waist Circumference)	80-89 female; 90-99 male	10
	≥ 90 female; ≥100 male	20
Physical activity	Vigorous exercise or strenuous work	0
	Moderate exercise	10
	Mild exercise	20
	Sedentary lifestyle	30
Family history	No family history	0
	Either parent	10
	Both parents	20
Maximum score		100
Risk stratification	Mild risk	< 30
	Moderate risk	30 – 50
	High risk	≥ 60

Physical activity

In the present study physical activity levels were considered as per IDRS.[101] The medical students were asked to answer the following questions to grade the physical activity.

a. How physically demanding is their work

Sedentary (0), Mild (1), Moderate (2) and Heavy (3).

b. Regular exercise in leisure time

Not at all (0), < 3 times a week (1), ≥ 3 times a week (2), Almost daily (3)

c. Physical activity at home

Sedentary (0), Mild (1), Moderate (2), Strenuous (3)

The combined scores of a, b and c were interpreted as below.

≥ 3 – Strenuous i.e., score = 0

2 – Moderate i.e., score = 10

1 – Mild i.e., score = 20

0 – Sedentary i.e., score = 30

Investigations

Under strict aseptic precautions, fasting blood sample was drawn fasting for the estimation of;

1. Fasting blood sugar
2. HbA1c.
3. Lipid profile*
    a. Total cholesterol
    b. Triglycerides

c. High density lipoprotein

d. Low density lipoprotein

**Lipid profile was done in medical students who had moderate to high risk of diabetes mellitus as per the IDRS assessment.*[101]

Fasting blood sugar levels and HbA1c

Based on new ADA criteria 2014[44]

Criteria for the diagnosis of diabetes

- A1C ≥ 6.5%.The test should be performed in a laboratory using a method that is NGSP certified and standardized to the DCCT assay; OR
- FPG ≥ 126 mg/dL. Fasting is defined as no caloric intake for at least 8 h.*; OR

**In the absence of unequivocal hyperglycemia, result was confirmed by repeat testing.*

Categories for the increased risk of diabetes (Prediabetes)

- FPG 100 to 125 mg/dL; OR
- A1C 5.7% to 6.4%.

Fasting lipid profile

Based on NCEP (National Cholesterol Education Program) guidelines[104] normal values of lipid parameters were;

- Low density lipoprotein < 100 mg/dL.
- High density lipoprotein;

  - Female > 50 mg/dL.
  - Males > 40 mg/dL.
- Total Cholesterol < 200 mg/dL.
- Triglycerides < 150 mg/dL.

Outcome variables

The medical students were evaluated for the presence of following risk factors

- Diet pattern
- History of smoking
- History of alcohol consumption
- Physical activity
- Abdominal circumference
- Family history of diabetes
- Body mass index
- Blood pressure
- Indian diabetes risk score

Risk factors

Based on the HbA1c levels, students were divided into two cohorts as below.[44]

- Risk of diabetes mellitus (HbA1c ≥ 5.7)
- No risk of diabetes mellitus (HbA1c <5.7).

Lipid profile

Those medical students who had moderate to high risk according to IDRS[101] were evaluated for lipid abnormalities.[104]

Statistical methods

The data obtained was coded and entered into the Microsoft Excel Spreadsheet (Annexure III). The data was analysed using SPSS version 20. The categorical data was expressed in terms of rates, ratios and percentages. To explore the differences in categorical characteristics Chi square or Fisher's exact test were used. Continuous data was compared using independent sample 't' test. In case of more than two mean the comparison was done using one way ANOVA. The correlation of risk of diabetes based on HbA1c levels and waist circumference, BMI and blood pressure was determine using Pearson's correlation co-efficient. A probability value (p value) of less than or equal to 0.050 at 95% confidence interval was considered as statistically significant.

RESULTS

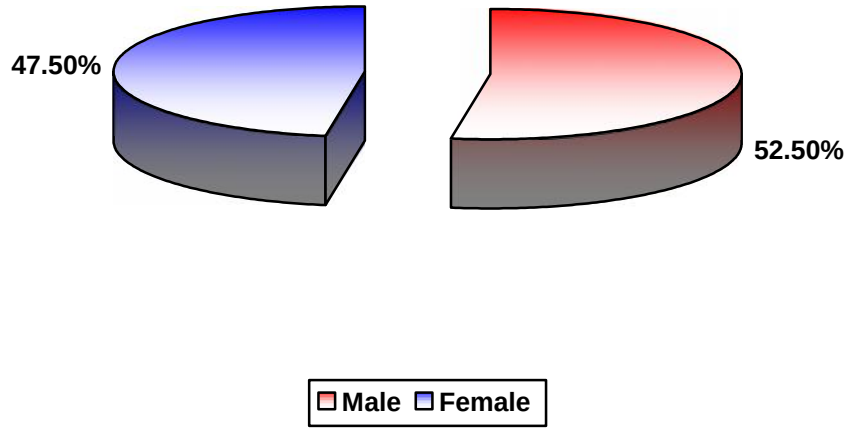
This one year cross-sectional study was performed on a total of 200 medical students under the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2014 to December 2014.

The data obtained was analysed and the final results and observations were tabulated as below.

Table 1. Sex distribution of the study population

Sex distribution	Distribution (n=200)	
	Number	**Percentage**
Male	105	52.50
Female	95	47.50
Total	**200**	**100.00**

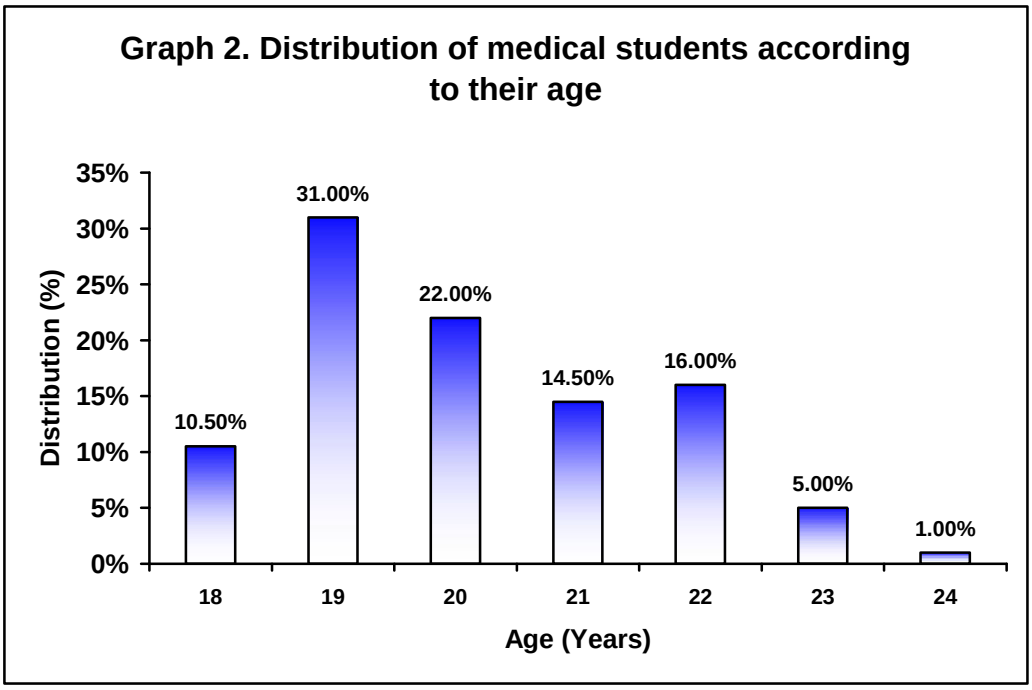
Graph 1. Sex distribution of the study population



In the present study 52.50% of the students were males and 47.50% were females. The male to female ratio was 1.10:1.

Table 2. Distribution of medical students according to their age

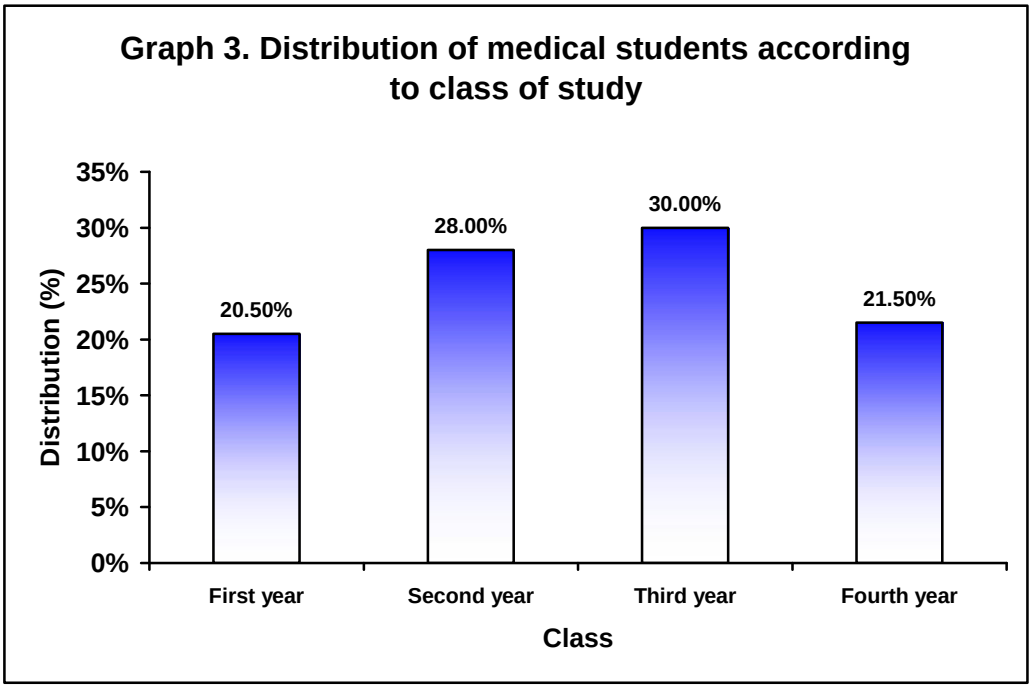
Age (Years)	Distribution (n=200)	
	Number	**Percentage**
18	21	10.50
19	62	31.00
20	44	22.00
21	29	14.50
22	32	16.00
23	10	5.00
24	2	1.00
Total	**200**	**100.00**



In this study most of the students were aged 19 years (31%) followed by 20 years (22%). The age distribution of other students is as shown in table 2 and graph 2.

Table 3. Distribution of medical students according to class of study

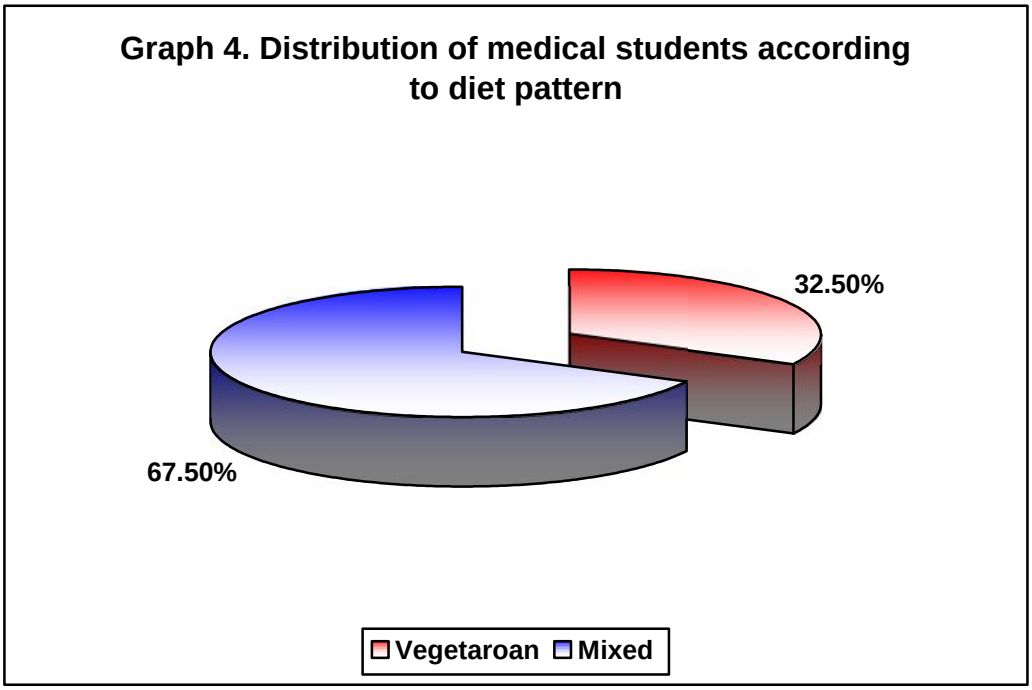
Class	Distribution (n=200)	
	Number	**Percentage**
First year	41	20.50
Second year	56	28.00
Third year	60	30.00
Fourth year	43	21.50
Total	**200**	**100.00**



In the present study 30% of the students were studying in third year MBBS and 28% in second year.

Table 4. Distribution of medical students according to diet pattern

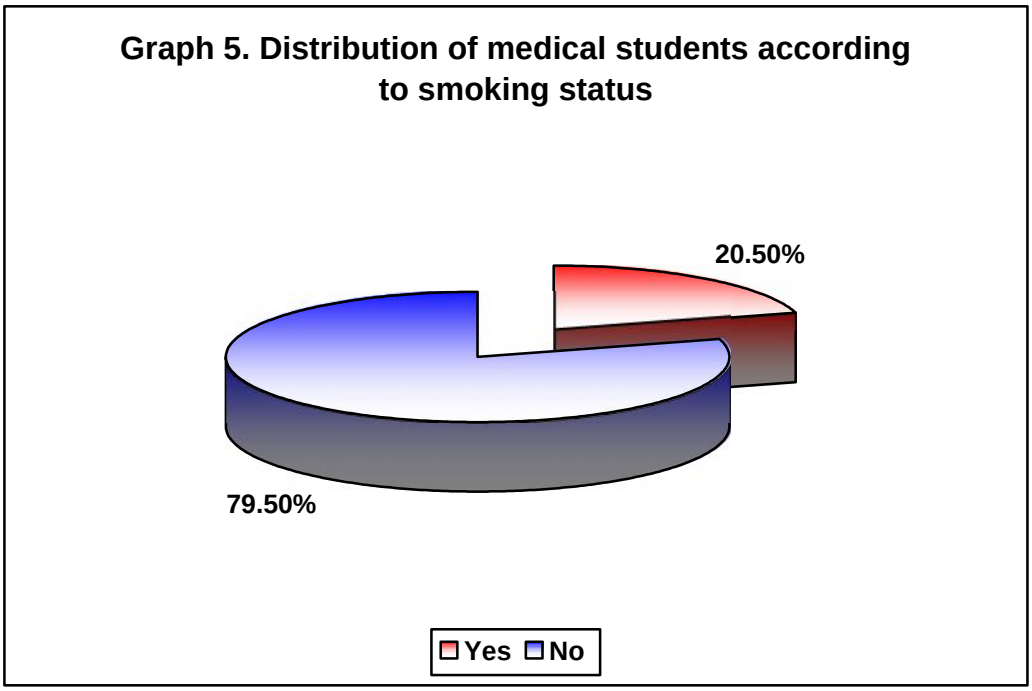
Diet	Distribution (n=200)	
	Number	**Percentage**
Vegetarian	65	32.50
Mixed	135	67.50
Total	**200**	**100.00**



In this study 67.50% of the students had mixed diet while 32.50% of the students reported vegetarian diet.

Table 5. Distribution of medical students according to smoking status

Smoking	Distribution (n=200)	
	Number	Percentage
Yes	41	20.50
No	159	79.50
Total	**200**	**100.00**

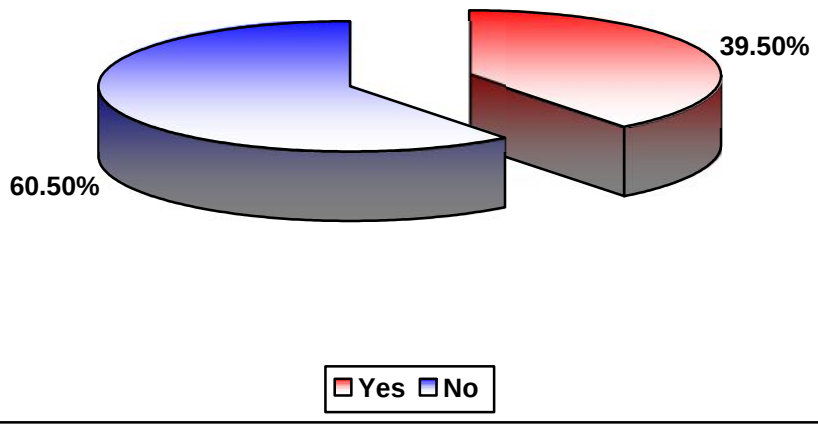


In the present study history of smoking was present in 20.5% of the students.

Table 6. Distribution of medical students according to the history of alcohol consumption

Alcohol consumption	Distribution (n=200)	
	Number	**Percentage**
Yes	79	39.50
No	121	60.50
Total	**200**	**100.00**

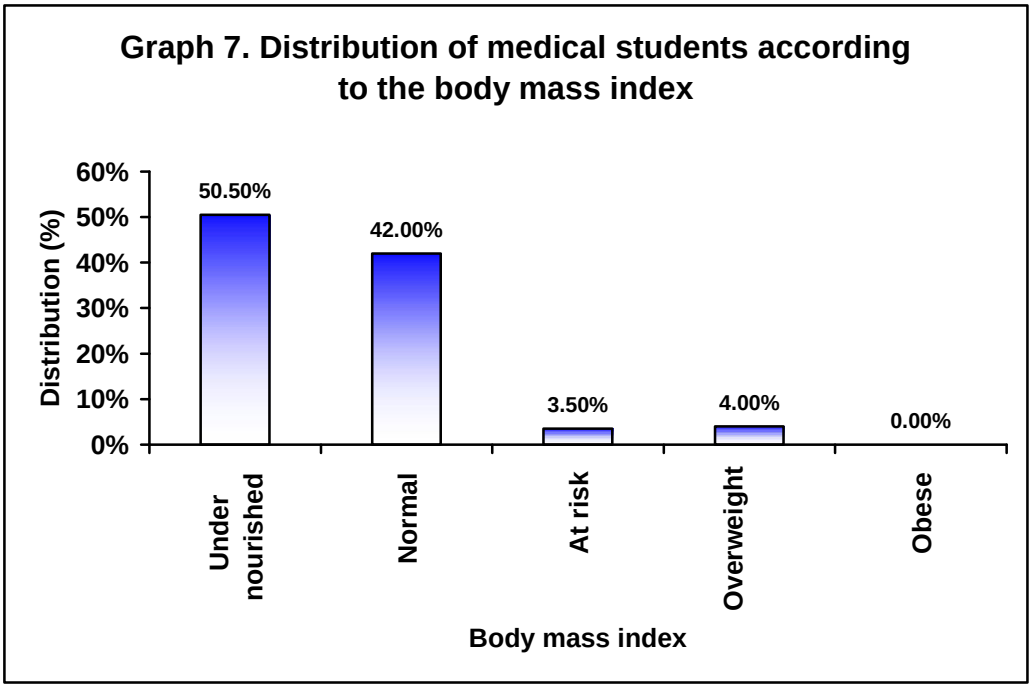
Graph 6. Distribution of medical students according to the history of alcohol consumption



In this study 39.50% of the students reported history of alcohol consumption.

Table 7. Distribution of medical students according to the body mass index

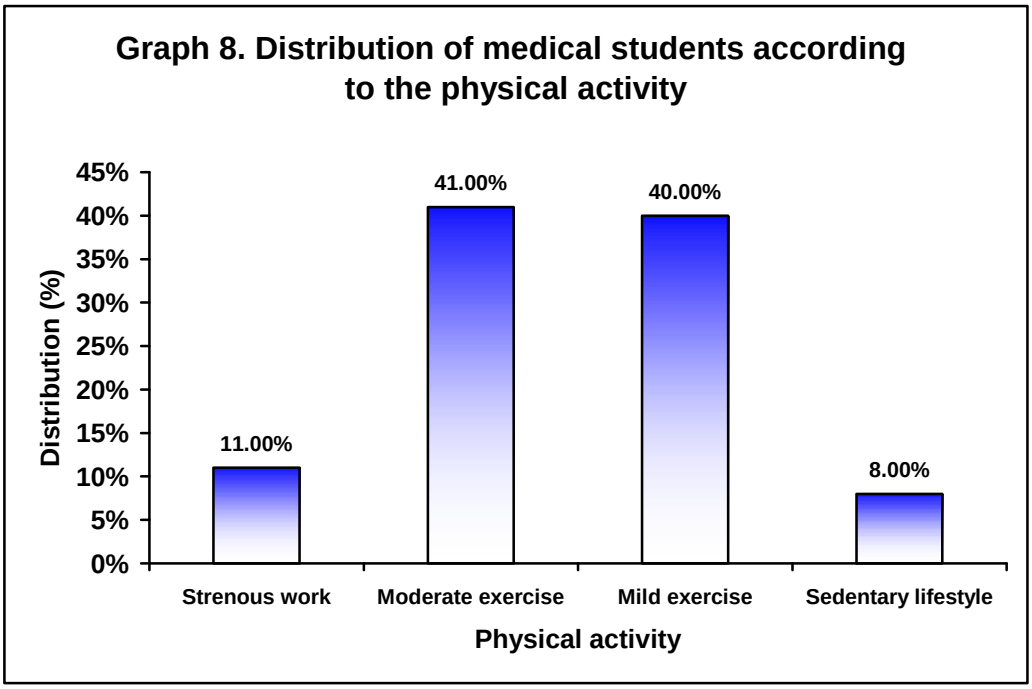
Body mass index (Kg/m^2)	Distribution (n=200)	
	Number	**Percentage**
Undernourished (< 18.5)	101	50.50
Normal (18.5 to 22.99)	84	42.00
At risk (23.00 to 24.99)	7	3.50
Overweight (25.00 to 29.99)	8	4.00
Obese (≥ 30)	0	0.00
Total	**200**	**100.00**



In this study 50.5% of the students were undernourished and normal BMI was noted among 42% of the students. However, 3.5% and 4% of the students were found to have higher BMI levels suggesting at risk and overweight respectively.

Table 8. Distribution of medical students according to the physical activity

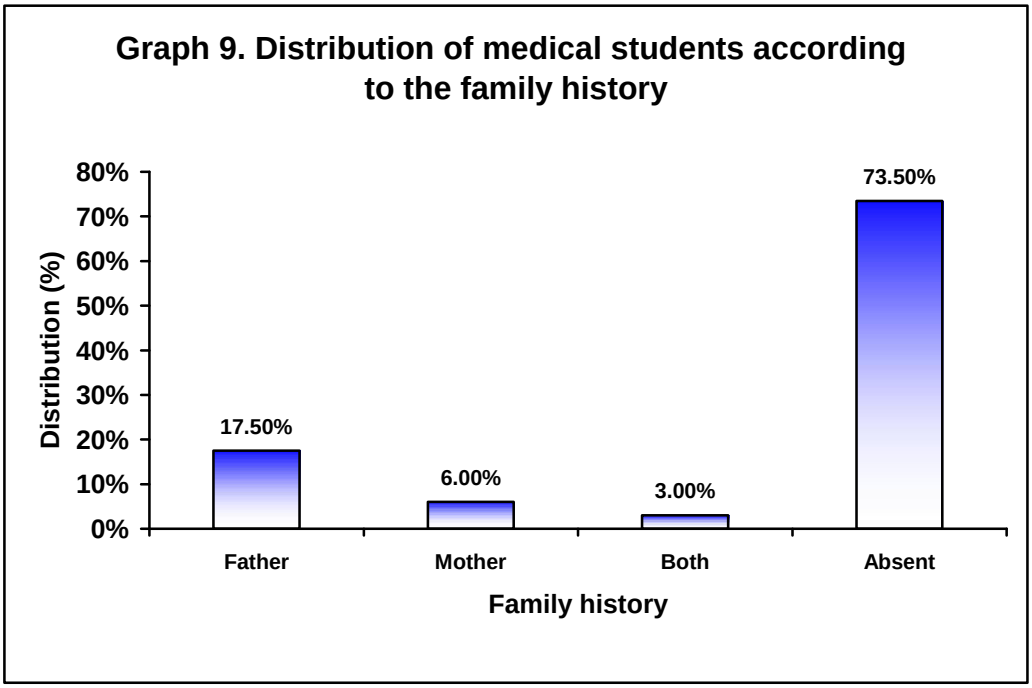
Physical activity	Distribution (n=200)	
	Number	Percentage
Strenous work	22	11.00
Moderate exercise	82	41.00
Mild exercise	80	40.00
Sedentary lifestyle	16	8.00
Total	**200**	**100.00**



In the present study most of the medical students had moderate exercise (41%) and mild exercise (40%). However, strenuous work and sedentary lifestyle were noted among 11% and 8% of the students respectively.

Table 9. Distribution of medical students according to the family history

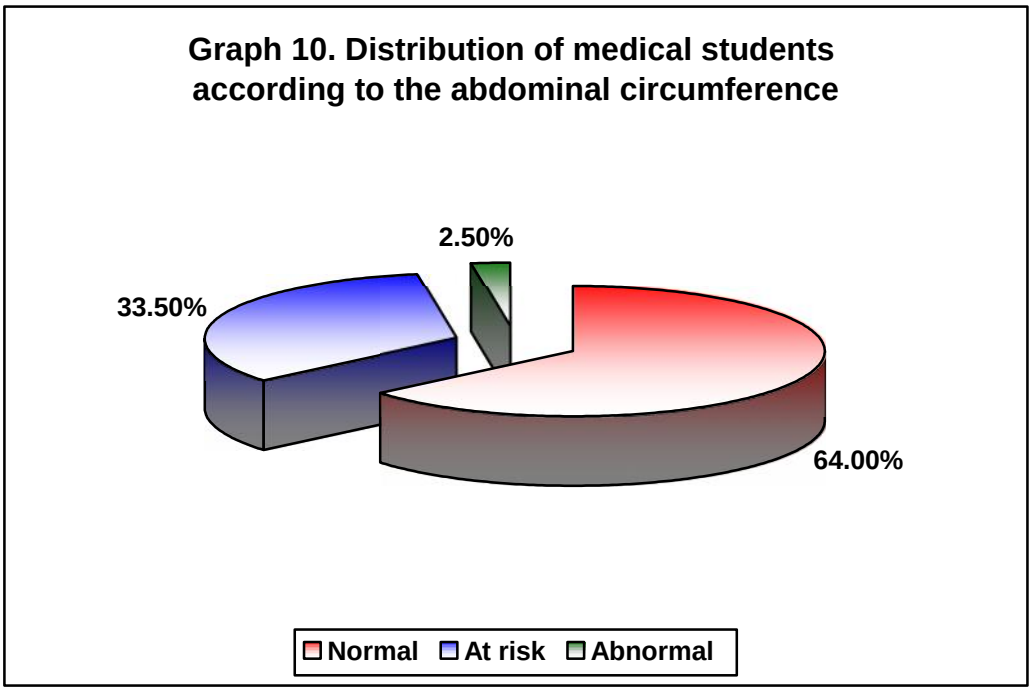
Family history	Distribution (n=200)	
	Number	Percentage
Father	35	17.50
Mother	12	6.00
Both	6	3.00
Absent	147	73.50
Total	**200**	**100.00**



In this study family history of diabetes mellitus was present in 26.5% of the students. The diabetic history in either parent that is father or mother was noted in 23.5% while among 3% of the students the diabetic history was present in both the parents.

Table 10. Distribution of medical students according to the abdominal circumference

Abdominal circumference (Cms)	Distribution (n=200)	
	Number	Percentage
<90 male; <80 female	128	64.00
90 to 99 male; 80 to 89 female	67	33.50
≥ 100 male; ≥ 90 female	5	2.50
Total	**200**	**100.00**



In the present study 64% of the students had normal abdominal circumference (<90 male; <80 female) while 33.5% were at risk (90 to 99 male; 80 to 89 female).

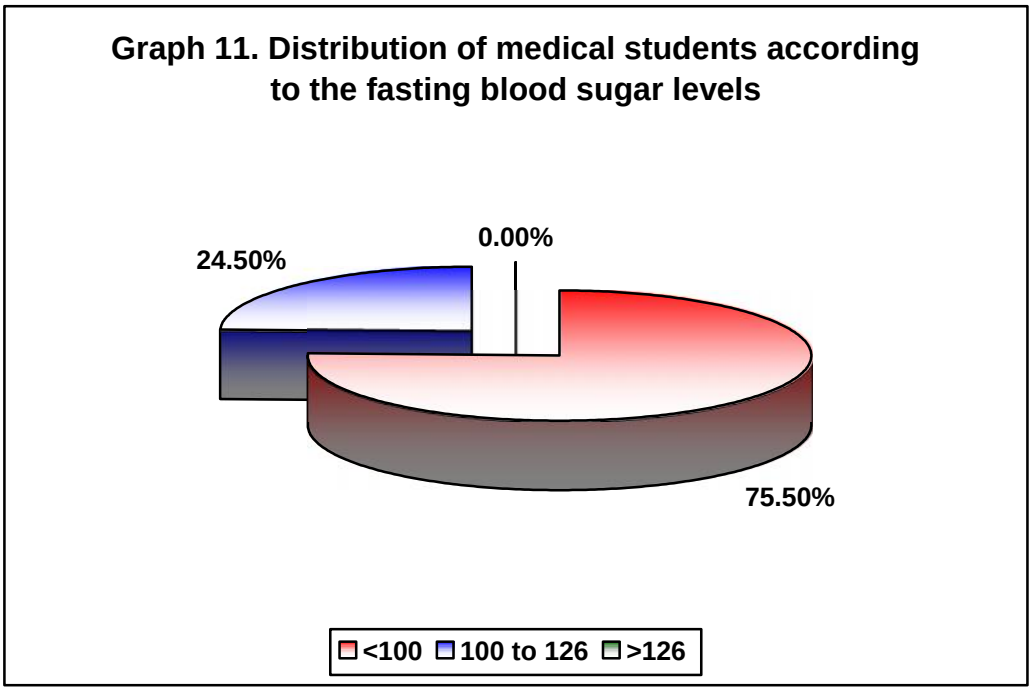
Table 11. Distribution of students according to the IDRS component findings

Components	Risk (IDRS Score)	Distribution (n=200)	
		Number	**Percentage**
Physical activity	Strenous work (0)	22	11.00
	Moderate exercise (10)	82	41.00
	Mild exercise (20)	80	40.00
	Sedentary lifestyle (30)	16	8.00
	Total	**200**	**100.00**
Family history	Absent (0)	147	73.50
	Father (10)	35	17.50
	Mother (10)	12	6.00
	Both (20)	6	3.00
	Total	**200**	**100.00**
Abdominal circumference (Cms)	<90 male; <80 female (0)	128	64.00
	90 to 99 male; 80 to 89 female (10)	67	33.50
	≥ 100 male; ≥ 90 female (20)	5	2.50
	Total	**200**	**100.00**
IDRS score	Low risk (< 30)	165	82.50
	Moderate risk (30 to 50)	33	16.50
	High (≥ 60)	2	1.00
	Total	**200**	**100.00**

On IDRS assessment, maximum students (41%) had moderate exercise (IDRS score 10), family history of diabetes was present among 53 (26.5%) students and maximum students reported family history of diabetes mellitus in father (17.5%). The abdominal circumference was normal in 64% of the students and suggestive of moderate risk in 33.50% and IDRS scores showed moderate risk of diabetes in 16.50% of the students.

Table 12. Distribution of medical students according to the fasting blood sugar levels

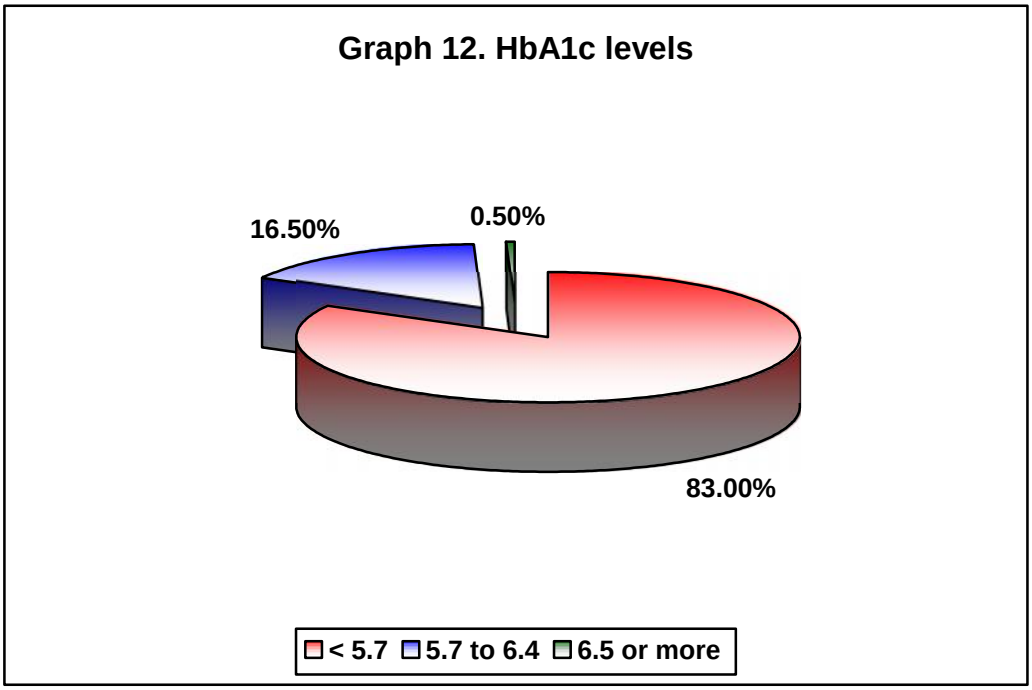
Fasting blood sugar (mg/dL)	Distribution (n=200)	
	Number	Percentage
< 100	151	75.50
100 to 126	49	24.50
> 126	0	0.00
Total	**200**	**100.00**



In the present study fasting blood sugar levels were < 100 mg/dL in 75.5% of the students. In the remaining, (24.5%) the fasting blood sugar levels were between 100 to 126 mg/dL.

Table 13. HbA1c Levels

HbA1c Levels	Distribution (n=200)	
	Number	**Percentage**
<5.7	166	83.00
5.7 to 6.4	33	16.50
6.5 or more	1	0.50
Total	**200**	**100.00**



In this study majority (83%) of the students had HbA1c levels < 5.7. In the remaining, 16.50% had HbA1c 5.7 to 6.4 and 0.5% had same ≥ 6.5.

Table 14. Association between risk of developing diabetes mellitus and physical activity

Physical activity	Risk of DM					
	Absent (HbA1c < 5.7)		Present (HbA1c ≥5.7)		Total	
	No.	%	No.	%	No.	%
Strenous	20	90.91	2	9.09	22	100.00
Moderate	73	89.02	9	10.98	82	100.00
Mild	67	83.75	13	16.25	80	100.00
Sedentary	6	37.50	10	62.50	16	100.00
Total	**166**	**83.00**	**34**	**17.00**	**200**	**100.00**

p<0.001

In the present study frequency of developing diabetes was significantly high (62.5%) among medical students with sedentary life style (p<0.001).

Table 15. Comparison of mean HbA1c levels with physical activity

Physical activity	Total number	Mean	SD
Strenous	22	5.27	0.23
Moderate	82	5.33	0.22
Mild	80	5.35	0.28
Sedentary	16	5.78	0.40
F value		14.908	
'p' value		**<0.001**	

In the present study the mean HbA1c levels showed an increasing trend from mild physical activity to strenuous work. The difference of mean HbA1c among the different physical activities was statistically significant (p<0.001).

Table 16. Association between risk of developing diabetes mellitus and abdominal circumference

Abdominal circumference (Cms)	Risk of DM				Total	
	Absent (HbA1c < 5.7)		Present (HbA1c ≥5.7)			
	No.	%	No.	%	No.	%
<90 M; <80F	116	90.63	12	9.38	128	100.00
90 to 99 M; 80 to 89 F	48	71.64	19	28.36	67	100.00
≥ 100 M; ≥ 90F	2	40.00	3	60.00	5	100.00
Total	**166**	**83.00**	**34**	**17.00**	**200**	**100.00**

p<0.001 (M=Male; F=Female)

In the present study 60% of the students with central obesity were at risk of DM as compared to 28.36% with at risk abdominal circumference and 9.38% with normal AC. This difference was statistically significant (p<0.001).

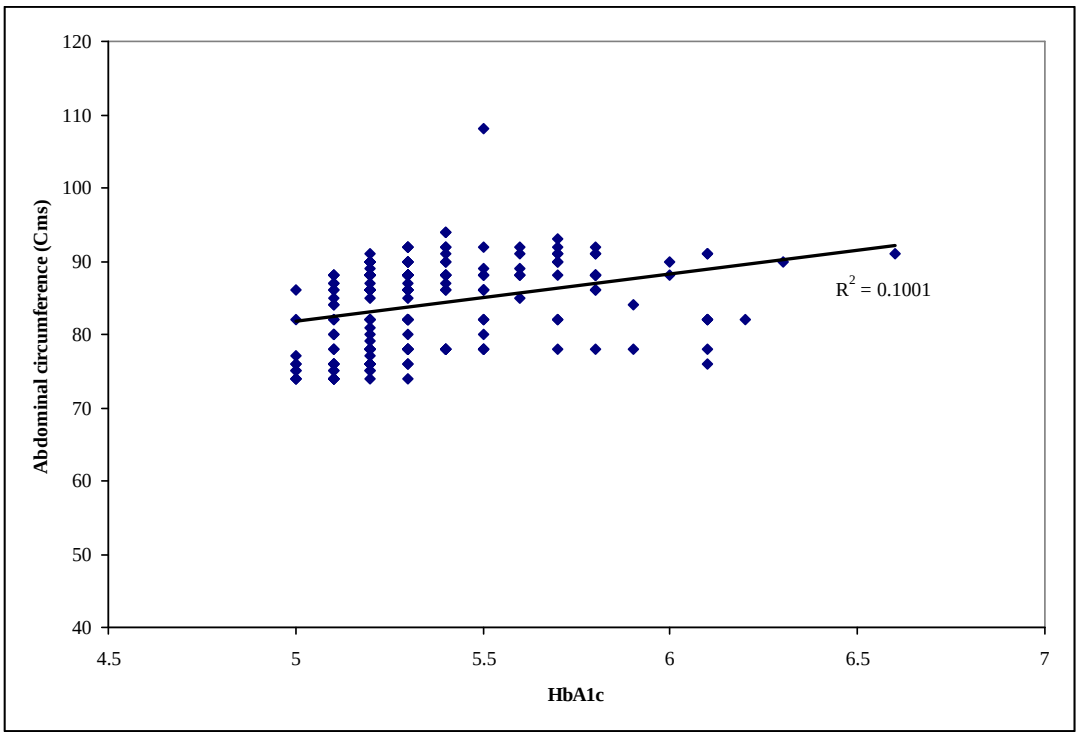
Table 17. Comparison of mean HbA1c levels with abdominal circumference

Abdominal circumference	Total number	Mean	SD
<90 M; <80F	128	5.29	0.23
90 to 99 M; 80 to 89 F	67	5.49	0.34
≥ 100 M; ≥ 90F	5	5.70	0.24
F value		15.422	
'p' value		**<0.001**	

(M=Male; F=Female)

In this study the mean HbA1c levels among the medical students with different grades of central obesity varied significantly (p<0.001).

Graph 13. Correlation of HbA1c with abdominal circumference



In the present study, the correlation of HbA1c with abdominal circumference showed Pearson's correlation coefficient (R) of 0.316 (R^2=0.100; p<0.001) with weak positive correlation.

Table 18. Association between risk of developing diabetes mellitus and family history

Family history	Risk of DM					
	Absent (HbA1c < 5.7)		Present HbA1c ≥5.7)		Total	
	No.	%	No.	%	No.	%
Present	31	58.49	22	41.51	53	100.00
Absent	135	91.84	12	8.16	147	100.00
Total	**166**	**83.00**	**34**	**17.00**	**200**	**100.00**

p<0.001

In this study significantly higher risk of DM was noted among students who reported family history of diabetes mellitus (41.51% p<0.001).

Table 19. Comparison of mean HbA1c levels with family history

Family history of DM	Total number	Mean	SD
Absent	147	5.28	0.21
Either parent	47	5.58	0.36
Both parents	6	5.82	0.19
F value		36.216	
'p' value		**<0.001**	

In this study the mean HbA1c levels among the medical students with family history of diabetes in either or both parents differed significantly when compared to those who did not report the family history of DM (p<0.001).

Table 20. Association between risk of developing diabetes mellitus and IDRS score

IDRS score	Risk of DM				Total	
	Absent (HbA1c < 5.7)		Present (HbA1c ≥5.7)			
	No	%	No	%	No	%
< 30	153	92.73	12	7.27	165	100.00
30 to 50	13	39.39	20	60.61	33	100.00
60 or more	0	0.00	2	100.00	2	100.00
Total	**166**	**83.00**	**34**	**17.00**	**200**	**100.00**

p<0.001

In this study maximum students with IDRS score 30 to 50 (60.61%) and ≥ 60 (100%) had risk of developing DM and this difference was statistically significant (p<0.001).

Table 21. Comparison of mean HbA1c levels with IDRS scores

Risk	Total number	Mean	SD
Mild	165	5.28	0.20063
Moderate	33	5.72	0.34889
Severe	2	6.05	0.07071
F value		57.086	
'p' value		**<0.001**	

In the present study statistically significant difference was noted with regard to HbA1c levels among different categories of risks for DM based on IDRS score (p<0.001).

Table 22. Association between risk of developing diabetes mellitus and sex

Sex	Risk of DM					
	Absent (HbA1c < 5.7)		Present HbA1c ≥5.7)		Total	
	No.	%	No.	%	No.	%
Male	87	82.86	18	17.14	105	100.00
Female	79	83.16	16	16.84	95	100.00
Total	**166**	**83.00**	**34**	**17.00**	**200**	**100.00**

p= 0.955

In the present study the risk of diabetes was comparable among males and females (p=0.955).

Table 23. Association between risk of developing diabetes mellitus and diet

Diet	Risk of DM					
	Absent (HbA1c < 5.7)		Present HbA1c ≥5.7)		Total	
	No.	%	No.	%	No.	%
Mixed	110	81.48	25	18.52	135	100.00
Vegetarian	56	86.15	9	13.85	65	100.00
Total	**166**	**83.00**	**34**	**17.00**	**200**	**100.00**

p= 0.410

In this study no statistically significant difference was observed in the risk of diabetes among mixed and vegetarian diet (p=0.410).

Table 24. Comparison of mean HbA1c levels in vegetarian and mixed diet

Diet pattern	Total number	Mean	SD
Vegetarian	65	5.32	0.25
Mixed	135	5.39	0.31
'p' value		0.081	

In this study the mean HbA1c levels were comparable among the students with vegetarian and mixed diet (p=0.081)

Table 25. Association between risk of developing diabetes mellitus and alcohol consumption

Alcohol consumption	Risk of DM					
	Absent (HbA1c < 5.7)		Present HbA1c ≥5.7)		Total	
	No.	%	No.	%	No.	%
Yes	58	73.42	21	26.58	79	100.00
No	108	89.26	13	10.74	121	100.00
Total	**166**	**83.00**	**34**	**17.00**	**200**	**100.00**

p= 0.004

In this study significantly higher risk of developing diabetes was noted among the students who had history of alcohol consumption (26.58%; p=0.004).

Table 26. Comparison of mean HbA1c levels with consumption of alcohol

Alcohol consumption	Total number	Mean	SD
Present	79	5.46	0.35
Absent	121	5.31	0.22
'p' value		**0.001**	

In this study the mean HbA1c levels were significantly high in medical students with history of alcohol consumption (p=0.001).

Table 27. Association between risk of developing diabetes mellitus and smoking

Smoking	Risk of DM					
	Absent (HbA1c < 5.7)		Present HbA1c ≥5.7)		Total	
	No.	%	No.	%	No.	%
Yes	36	87.80	5	12.20	41	100.00
No	130	81.76	29	18.24	159	100.00
Total	**166**	**83.00**	**34**	**17.00**	**200**	**100.00**

p= 0.358

In the present study the risk of developing diabetes was equal among smokers and non smokers (p=0.358).

Table 28. Comparison of mean HbA1c levels with smoking status

Smoking	Total number	Mean	SD
Present	41	5.40	0.32
Absent	159	5.36	0.28
'p' value		0.477	

In the present study among the students with and without smoking, the mean HbA1c levels were comparable (p=0.477)

Table 29. Association between risk of developing diabetes mellitus and body mass index

Body mass index (Kg/m^2)	Risk of DM				Total	
	Absent (HbA1c < 5.7)		Present HbA1c ≥5.7)			
	No.	%	No.	%	No.	%
< 18.5	99	98.02	2	1.98	101	100.00
18.50 to 22.99	63	75.00	21	25.00	84	100.00
23.00 to 24.99	2	28.57	5	71.43	7	100.00
25.00 to 29.99	2	25.00	6	75.00	8	100.00
Total	**166**	**83.00**	**34**	**17.00**	**200**	**100.00**

p<0.001

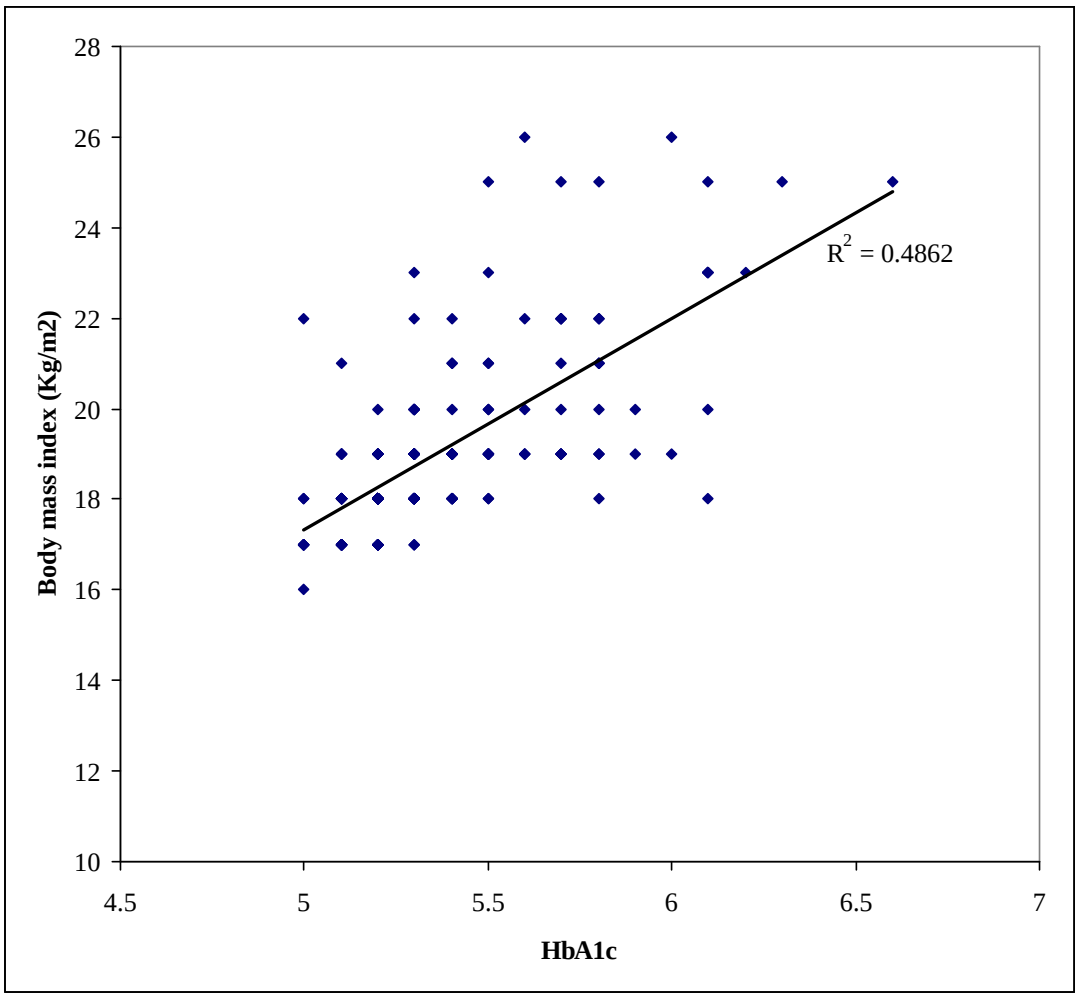
In this study significantly higher number of medical students with at risk (71.43%) and overweight (75%) grades of obesity had chances of developing DM (p<0.001).

Table 30. Comparison of mean HbA1c levels with body mass index

BMI	Total number	Mean	SD
< 18.5	101	5.21	0.15
18.50 to 22.99	84	5.45	0.24
23.00 to 24.99	7	5.91	0.36
25.00 to 29.99	8	5.95	0.37
F value		59.286	
'p' value		**<0.001**	

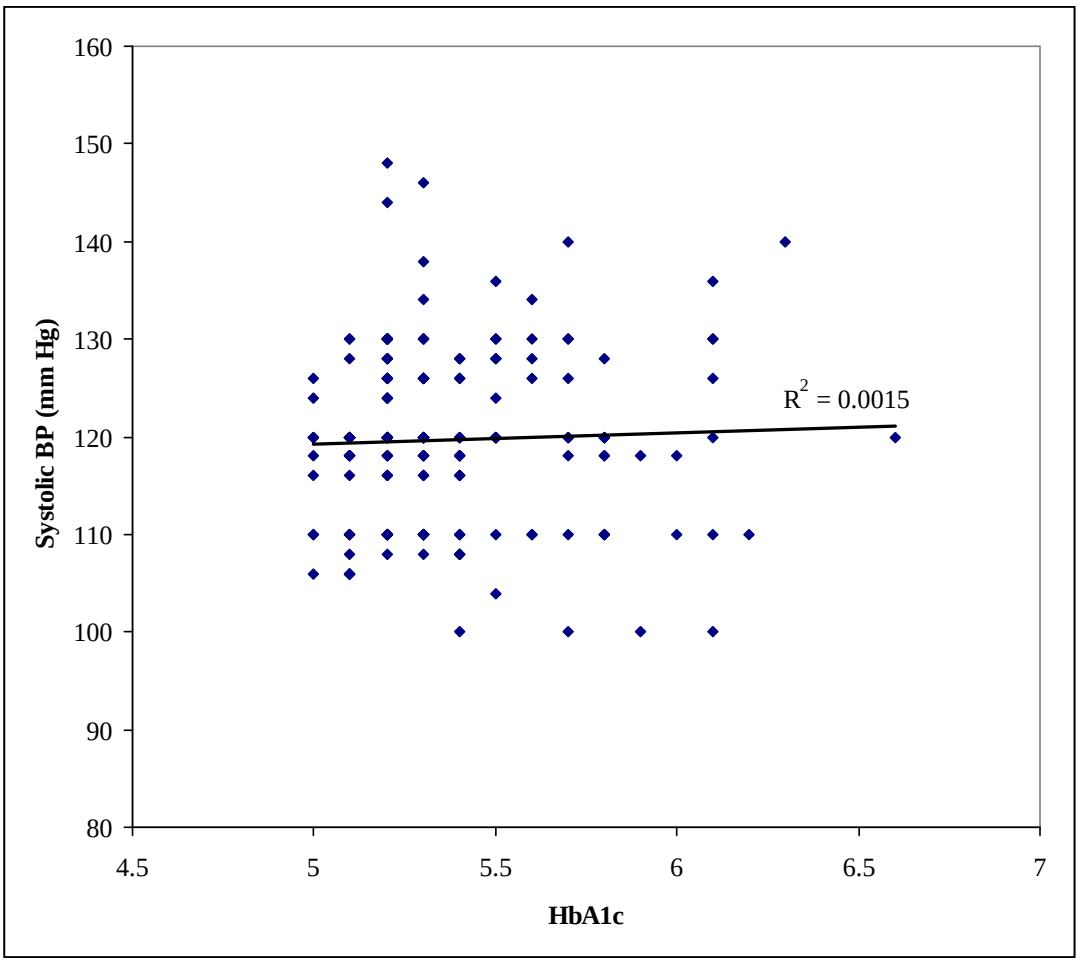
In this study the comparison of mean HbA1c levels among different grades of obesity revealed statistically significant difference in mean HbA1c levels ($p<0.001$)

Graph 14. Correlation of HbA1c with body mass index



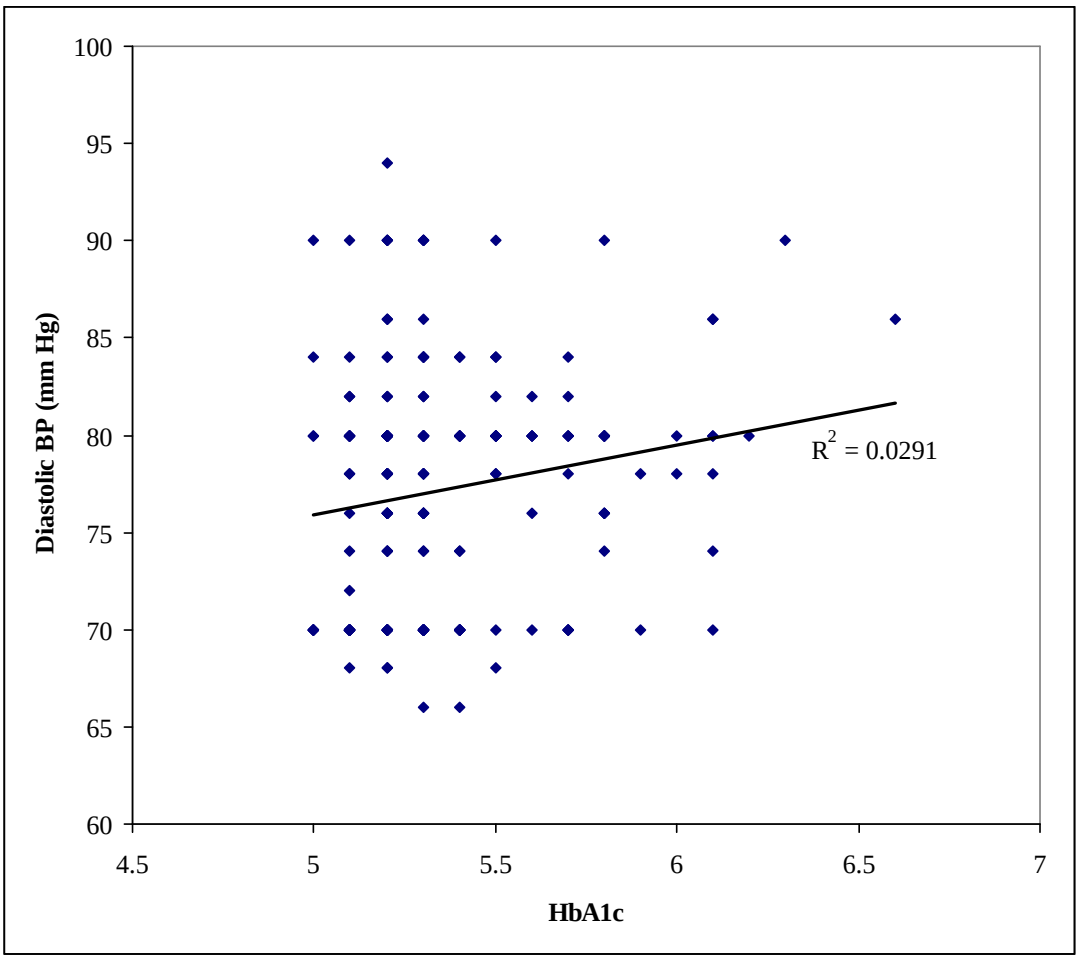
In this study, the correlation of HbA1c with body mass index revealed Pearson's correlation coefficient (R) of 0.697 (R^2=0.486; p<0.001) with moderate tendency for higher body mass index levels with higher HbA1c levels.

Graph 15. Correlation of HbA1c with systolic blood pressure



In the present study, the correlation of HbA1c with systolic blood pressure showed Pearson's correlation coefficient (R) of 0.038 (R^2=0.001; p=0.584) with weak positive correlation.

Graph 16. Correlation of HbA1c with diastolic blood pressure



In this study, the correlation of HbA1c with diastolic blood pressure showed Pearson's correlation coefficient (R) of 0.170 (R^2=0.029; p=0.015) with weak positive correlation.

Table 31. Distribution of students according to lipid profile

Variables	Sub-groups	Distribution (n=35)	
		Number	**Percentage**
Total	< 200	32	91.43
Cholesterol	200 or more	3	8.57
(mg/dL)	**Total**	**35**	**100.00**
	Mean ± SD	**152.89**	**27.83**
LDL	< 100	24	68.57
(mg/dL)	100 or more	11	31.43
	Total	**35**	**100.00**
	Mean ± SD	**94.17**	**23.87**
HDL	< 40	24	68.57
(mg/dL)	40 or more	11	31.43
	Total	**35**	**100.00**
	Mean ± SD	**37.83**	**7.93**
Triglycerides	< 150	31	88.57
(mg/dL)	150 or more	4	11.43
	Total	**35**	**100.00**
	Mean ± SD	**101.71**	**34.91**

Among the students with moderate and high risk of diabetes mellitus based on IDRS, lipid profile was abnormal in considerable subset of students that is 31.43% of the students had raised LDL, 68.57% had abnormal HDL while triglycerides and total cholesterol were raised (≥150 mg/dL and ≥200 mg/dL respectively) in 11.43% and 8.57% respectively

DISCUSSION

Diabetes is fast gaining the status of a potential epidemic in India with more than 62 million diabetic individuals currently diagnosed with the disease.[6] India currently faces an uncertain future in relation to the potential burden that diabetes may impose upon the country. Many factors affect the prevalence of the disease and identification of those is necessary.[6]

Chennai Urban Rural Epidemiology Study (CURES)[5] has reported rising prevalence of diabetes in our country. This study reported that within a span of 14 years, the prevalence of diabetes increased significantly by 72.3%. They also reported a temporal shift in the age at diagnosis to a younger group when compared to the previous studies. A study[105] from Delhi also reports a high prevalence of insulin resistance in post pubertal children which was associated with excess body fat and abdominal adiposity. These finding are disturbing as the earlier age of onset of diabetes and the increasing prevalence can have adverse effects on nation's health and economy.

The shift from pediatric age to adulthood is a critical period. Those adolescents with risk factors for diabetes are more prone for deterioration in glycemic control. Those individuals diagnosed with diabetes at this age are at increased risk of developing acute complications and psychosocial, emotional, and behavioral issues.[106]

Based on the recently published projections from Centers for Disease Control and Prevention for type 2 diabetes, assuming a 2.3% annual increase, the

prevalence of type 2 diabetes in those under 20 years of age will quadruple in 40 years. The incidence of type 2 diabetes in children is reported to be increasing in several parts of the world. Among the native North Americans, 30% of the new cases of type 2 diabetes occur in the second decade of life,[58] while among the Japanese school children, type 2 diabetes is 7 times more common than type 1[61] and a similar trend is reported in the Chinese and Mexican American youth.[58-60] In Pima Indians there is an increase in the incidence in the age group of 10 years and above.[64] Type 2 diabetes presents a decade or two earlier in India than in the West.[65] About 38% of type 2 diabetics are diagnosed below the age of 40 years and in about 4.8% a diagnosis is made below 25 years.[65]

Significant comorbidities may already be present at the time of diagnosis of type 2 diabetes mellitus like polycystic ovary disease, pediatric obesity, sleep apnea, hepatic steatosis, and psychosocial problems.[106]

Hence, the current ADA guidelines (2015) recommend screening of type 2 diabetes in children and adolescents who are overweight or obese and who have two or more additional risk factors for diabetes. Further, screening of type 2 diabetes in asymptomatic people should be considered in adults of any age who are overweight or obese (BMI $\geq$ 25 kg/m^2 or $\geq$ 23 kg/m^2 in Asian Americans) and who have one or more additional risk factors for diabetes.[106] This prompted us to study the differences in risk factors in medical students likely vs not likely to develop Type 2 Diabetes Mellitus in our institute. Also medical students can be educated regarding risk for developing type 2 diabetes mellitus.

This one year cross-sectional study was done in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2014 to December 2014. A total of 200 medical students studying in Jawaharlal Nehru Medical College, Belgaum were included in the study. Students were evaluated for the presence of risk factors based on IDRS as mentioned earlier and subjected to fasting plasma glucose and HbA1c. Lipid profile was done in those students whose IDRS scores were moderate to high. Further, these medical students were divided into two cohorts based on the HbA1c levels that is, HbA1c ≥ 5.7 (Medical students with risk of developing diabetes mellitus) and < 5.7 (No risk of diabetes mellitus).

In this study, the demographic assessment revealed equal number of males (52.50%) and females (47.50%) with male to female ratio being 1.10:1.

In this study, on estimation of FBS, 24.5% of the students revealed FBS levels between 100 to 125 mg/dL suggesting prediabetes. In 16.5% of the students HbA1c levels were between 5.7 to 6.4 suggesting prediabetes and one student had HbA1c between 6.5 to 7.0 confirming the diagnosis of type 2 DM. These findings suggest that impaired glucose tolerance is highly prevalent in young adults to the extent one in six presented with FBS levels between 100 to 125 mg/dL. Similar findings were reported in a study by Al-Farai HH. et al.,[107] in Oman where the prevalence of insulin resistance was 16% in healthy young Omanis. Another study[57] done among 4833 patients registered at Sahay's Diabetic Clinic and Research Centre, Hyderabad, during the period 1999-2002 reported 7.5% of diabetic patients below the age of 30 years.

In the present study, assessment of abdominal circumference showed 33.5% of the students with abdominal circumference between 90 to 99 Cms in males; 80 to 89 Cms in females while 2.5% of the students had abnormal abdominal circumference ≥ 100 Cms in males and ≥ 90 Cms in females. Further, the risk of developing diabetes was significantly high in students who had abdominal circumference between 90 to 99 Cms in males and 80 to 89 Cms in females (28.36%) and in those medical students who had abdominal circumference >100 Cms in males and >90 Cms in females (60%) ($p<0.001$). Also, HbA1c levels were significantly high in these patients (5.49 ± 0.34 and 5.70 ± 0.24 respectively; $p<0.001$). These findings suggest that, central obesity is a strong risk factor for the development of diabetes mellitus and it prompts assessment of glycaemic status at regular intervals.

A study by Lima et al.,[108] also indicated that the most prevalent risk factor for the development of diabetes in young adults was central obesity. In a cross-sectional study of 14 adolescents with Impaired Glucose Tolerance matched with 14 control subjects of similar age, BMI, body fat, and leptin, the children with Impaired Glucose Tolerance were insulin resistant, with increased intramyocellular fat measured by 1H nuclear magnetic resonance spectroscopy showing strong correlation with insulin sensitivity and with 2-h postload plasma glucose. Those with IGT had higher visceral and lower subcutaneous abdominal fat and decreased first-phase insulin secretion and glucose disposition index.[109]

Also in this study, 7.5% of the students were found to have higher BMI levels out of which 4% were overweight and 3.5% were at risk of obesity. In our study, significantly higher number of medical students with risk of obesity (71.43%)

and who were overweight (75%) had HbA1c levels ≥ 5.7 ($p<0.001$) and the mean HbA1c levels were significantly high (5.91 ± 0.36 and 5.95 ± 0.37 respectively; $p<0.001$). The correlation of HbA1c with body mass index revealed Pearson's correlation coefficient (R) of 0.6973 ($R2=0.4862$; $p<0.0001$) which indicates moderate association between high body mass index levels and high HbA1c levels. These findings predict higher risk of diabetes mellitus among the younger individuals with obesity which is consistent with previous studies. A study by Al-Farai HH. et al.,[107] found a high prevalence (26%) of obese and overweight Omani students who had insulin resistance. Another study done in 202 Omani students concluded that, either 28% students were overweight or obese.

Family history plays a crucial role, with more than two-thirds of children with type 2 diabetes having at least one parent with type 2 diabetes.[75] In this study more than one fourth of the study population reported family history of diabetes mellitus (26.5%). Further diabetic history in either parent (father or mother) was present in 23.5% and 3% in both the parents. Significantly higher number of student with risk of DM (Prediabetes) was noted among students who reported family history of diabetes mellitus (41.51% $p<0.001$). In this study the mean HbA1c levels among the medical students with family history diabetes in either or both parents differed significantly when compared to those who did not report the family history of DM ($p<0.001$). Similar to the present study Sahay BK et al.,[57] in their study reported that, majority of the children with type 2 diabetes have a family history of diabetes, with at least one parent being affected. There may be history of diabetes in first or second degree relatives also. Another study[110] from Pune demonstrated family history of DM in a first degree relative was present in up to 70% of patients.

Among children with type 2 diabetes in Japan, a study showed familial clustering, with siblings having a 175- to 250-fold increase in diabetes compared to the general population and children with diabetic parents having a 48–60% likelihood of having type 2 diabetes.[78]

In the present study, with regard to physical activity, 8% of the medical student had sedentary lifestyle, while 41% of the students performed moderate physical activity, 40% mild physical activity and 11% performed strenuous physical activity. There was positive association between sedentary lifestyle and risk of developing diabetes mellitus that is, maximum medical students (62.5%) with sedentary lifestyle had higher HbA1c levels (≥ 5.7) ($p<0.001$). The findings suggest direct relationship between sedentary lifestyle and risk of developing diabetes.

Alcohol causes insulin resistance and pancreatic β-cell dysfunction that may be a prerequisite for the development of diabetes.[111] In the present study history of alcohol consumption was reported by 39.5% of the student and they had higher risk of developing diabetes mellitus (26.58%; $p=0.004$) as the mean HbA1c levels were significantly high (5.46 ± 0.35 vs 5.31 ± 0.22; $p=0.001$). Alcohol consumption in diabetes has been controversial and more detailed information on the diabetogenic impact of alcohol seems warranted. Diabetes, especially T2DM, causes dysregulation of various metabolic processes, which includes a defect in the insulin-mediated glucose function of adipocytes, and an impaired insulin action in the liver. In addition, neurobiological profiles of alcoholism are linked to the effects of a disruption of glucose homeostasis and of insulin resistance, which are affected by altered appetite that regulates the peptides and neurotrophic factors. Though the present study, found positive association between risk of developing diabetes and

alcohol consumption it cannot be generalized, as the history of alcohol intake was of short duration.

In the present study, the overall risk of diabetes among the medical students was stratified based on IDRS.[101] Assessment of IDRS components showed maximum risk of lack of physical activity (79.5%) followed by central obesity as measured by abnormal circumference (36%) and family history (27.5%). With regard to physical activity, 30% of the students had mild physical activity, 28% had moderate physical activity and 21.5% had sedentary lifestyle. The family history of diabetes mellitus was reported by 23.5% of the students in either father or mother and 3% of the students reported same among both the parents. Based on these statistics the IDRS score showed lower risk of developing diabetes in 82.5% of the students while moderate and high risk was found in 16.5% and 1% of the students respectively. Further, positive association was noted between risk of diabetes based on IDRS with fasting plasma glucose levels ($p<0.001$). Also the mean IDRS score in medical students with risk of developing diabetes were significantly high (34.41 ± 14.18 vs 18.61 ± 9.40; $p<0.001$). The mean HbA1c levels in medical students with high risk were significantly high (6.05 ± 0.07) compared to those students who had moderate (5.72 ± 0.34) and mild risk (5.28 ± 0.20) ($p<0.001$). These findings show that, IDRS is a good screening tool to identify young adults with risk of diabetes. Similar findings were reported by Mohan V. et al.,[112] who showed an increase in the IDRS was associated with a worsening of glucose tolerance.

A similar study[110] conducted in Pune on 261 medical students in 2011, reported 4%, 76%, and 20% in high, moderate and low risk group, respectively as per IDRS for developing type 2 DM. Also major contribution to risk score was

sedentary lifestyle in 62% students followed by abdominal obesity in 38% of students. Another study[113] from India in 2011 on 150 medical students reported 101 students with an IDRS of <30, 42 students with a moderate IDRS (30-50) and 7 who had a high IDRS of ≥60 resulting in nearly one third of the young medical students had moderate to high risk of diabetes. Although only 5% were in the high risk category, about 28% were in the moderate risk category. The increased risk scores were mainly due to a decreased physical activity (in 22% of the students), a family history of diabetes in about 13% students and an increased abdominal circumference in about 8% of the students.

Overall, the present study confirms that family history, central obesity, obesity as determined by body mass index and sedentary lifestyle are the risk factors for the development of diabetes mellitus in younger individuals. However, the present study lacked the relationship between other risk factors including gender (p=0.955), diet (p=0.410), history of smoking (p=0.358), and systolic and diastolic blood pressure.

A study by V. Mohan et al.,[112] showed that high IDRS score was associated with hypertriglyceridaemia and hypercholesterolemia. In the present study, 35 students with moderate and high risk as per IDRS were investigated further for fasting lipid profile in which more than two third of the students (68.57%) had low HDL levels and nearly one third (31.43%) had higher LDL levels. The mean HDL and LDL levels were 37.83 ± 9.93 mg/dL and 94.17 ± 23.87 mg/dL respectively. The above finding suggest that even though IDRS does not include all the risk factors like BMI, history of hypertension, type of diet and history of smoking or alcohol consumption it could still be an indirect predictor of dyslipidemia.

IDRS is a very useful tool that can be used for predicting the risk of developing diabetes. In a resource limited setting like ours, it can also be used as a tool for predicting dyslipidaemia. Since the major cause of high risk score among the young students was physical inactivity, health education and life style modifications could be suggested based on the IDRS score.[75,78,101] Hence, unnecessary investigations for identification of population at risk of developing type 2 DM can be avoided, reducing the economic burden of the nation.

There are several limitations in this study. The population studied is limited to medical students of a single medical college and hence larger studies are required to generalize these finding among all medical students. Also, the screened population consists of only 200 students and fasting lipid profile was not measured in all the students due time and cost constraints. Another major limitation is that Insulin Resistance was not measured in the high risk population.

CONCLUSION

The results of the present study on medical students highlight the important risk factors associated with development of diabetes mellitus. The study showed that, family history of diabetes mellitus among parents, central obesity, sedentary lifestyle and high body mass index are the risk factors in medical students which pose high risk for development of diabetes mellitus. Hence, young individuals having these risk factors should undergo screening for the diabetes mellitus so as to diagnose and treat the condition early and avoid further complications.

Further, there are a significant number of medical students at risk of developing type 2 diabetes mellitus (Prediabetes). The major concern here being, that medical students are well aware of the burden of diabetes mellitus and its complications. Also these medical students will be the health care providers in the near future and will be educating the general population regarding health issues. Its about time we understand that the same medical education applies to the medical health providers also and and the lifestyle modifications should be brought in their life as well.

To conclude, in resource limited settings, where a large population has to be screened, IDRS is a simple screening tool which aids in selecting high risk people for further investigations thereby saving time and resources. This will not only help to predict the risk of developing diabetes but will also help in prevention of future risk of diabetes at an early stage.

SUMMARY

Earlier diabetes mellitus (DM) was thought to be the disease of elderly but at present there has been a trend towards shift in the mean age of onset of type 2 diabetes to a much younger age. This rise in prevalence has been attributed to changes towards a western lifestyle, obesity and family history. This study was aimed to find out the differences in risk factors which are likely to predict the development of type 2 diabetes mellitus among medical students.

This one year cross-sectional study was carried out in the Department of Medicine of a tertiary care centre situated in South India from January 2014 to December 2014 on 200 medical students aged > 18 years. Assessment of diabetes risk was based on obesity status, exercise status, and family history of type 2 DM.

In this study, 52.50% of the students were males and male to female ratio was 1.10:1. Most of the students were aged 19 years (31%). Diet pattern revealed 67.50% of the students with mixed diet. With regard to personal history, smoking and alcohol consumption were noted in 20.5% and 39.5% of the students respectively. Most of the medical students were in mild exercise (41%) group and 33.5% of the students had abdominal circumference between 90 to 99 Cms in males and 80 to 89 Cms in females while family history of diabetes mellitus was present in 26.5% of the students. The assessment of BMI revealed higher BMI levels in 4% of the students. The fasting blood sugar levels were between 100 to 126 mg/dL in 24.5% of the students and 16.50% of the students had HbA1c from 5.7 to 6.4 and 0.5% with > 6.5. On IDRS assessment moderate risk of diabetes was noted in 16.50% of the students. The family history, central obesity, higher body mass index

and sedentary lifestyle were significantly associated with risk of developing diabetes ($p<0.050$) while gender, diet, history of smoking, systolic and diastolic blood pressure showed no relationship with risk of diabetes.

Medical students should be screened for the presence of risk factors including physical activity, obesity and family history of diabetes mellitus using IDRS which will not only help to predict the risk of developing DM but also helps in prevention.

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ANNEXURE I – CONSENT FORM

TITLE OF RESEARCH STUDY: DETERMINANTS OF RISK FOR DEVELOPING TYPE 2 DIABETES MELLITUS IN MEDICAL STUDENTS STUDYING IN NORTH KARNATAKA-A CROSS SECTIONAL STUDY.

Principal Investigator

Dr. **** *******
Post Graduate Student,
Department of General Medicine,
Jawaharlal Nehru Medical College,
Belgaum-590 010

GUIDE

Dr. **** *******
Prof. and Head of Unit
Department of General Medicine,
Jawaharlal Nehru Medical College,
Belgaum-590 010

Introduction and Purpose

The rising prevalence of Diabetes mellitus and with more than 50% percent of diabetic patients being unaware of their diabetic status in India holds the need for a mass screening simple, practical and user friendly tool for an early detection of diabetes and prevention of its complications.

Medical students having a sedentary lifestyle, holds the need to identify them at early stages and Indian Diabetes Risk Score (IDRS) is a simple and effective mass screening tool to identify risk of diabetes. The high risk and medium risk students will be subjected to investigations.

Procedure

If you agree to be part of the research study, you will be asked the relevant history and will be subjected to relevant clinical examination and investigations. You will also have to give blood samples for the necessary investigations.

Risk and Benefits

The only risk and possible discomfort you might get is while taking blood from your arm for the investigations. It may cause swelling and pain at the site from where the blood is drawn.

The benefit will be that you will know if you are in the high or moderate risk for the development of type 2 diabetes mellitus and what steps can you take to get in the lower risk group. You will not be charged for any of the investigations as they will be covered under health scheme.

Alternatives

Taking part in this study is voluntary. You may choose not to take part in this study, or if you decide to take part in the study, you can withdraw from the study at any time if they wish to. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsor may stop your participation in this study at any time. If you choose not to take part in the study, you will still receive the standard treatment.

Privacy and Confidentiality

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy

Does not apply to this research

Financial incentives for participation

You will not be paid / offered any gifts /incentives for participating in the study.

Authorization to publish the results

The results of the study would be forwarded to the KLE University, Belgaum as part of requirement towards the completion of MD degree, review and publishing. In case of the queries during study or in future you may contact following persons.

Dr. **** *******,**
Investigator,
Post Graduate in General Medicine,
Jawaharlal Nehru Medical College,
Belgaum-590 010
Phone No - **********.

Dr. ****** *******,**
Professor and Head of Unit,
Department of General Medicine,
Jawaharlal Nehru Medical College,
Belgaum-590 010
Phone Number - **********

Dr. **** **********
Chairman,
Jawaharlal Nehru Medical College,
Ethical Committee for Human Research,
Phone Number -***** ******

Consent Statement

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read, or it has been read to me, this entire consent form, and have had all my questions answered.

Name of the Participant: _____

Signature / Thumb print: _____

Name of the Witness: _____

Signature/ Thumb print: _____

Investigator Name: _____

Signature: _____

Date:

Place:

ANNEXURE II – PROFORMA

TITLE OF RESEARCH STUDY: DETERMINANTS OF RISK FOR DEVELOPING TYPE 2 DIABETES MELLITUS IN MEDICAL STUDENTS STUDYING IN NORTH KARNATAKA-A CROSS SECTIONAL STUDY.

SERIAL NUMBER :

NAME :

ADDRESS :

TELEPHONE NUMBER :

INDIAN DIABETES RISK SCORE

SCORE

[1] AGE

[2] PHYSICAL ACTIVITY

STRENOUS

MODERATE

MILD

SEDENTARY

[3] FAMILY HISTORY OF DIABETES

NON-DIABETIC PARENTS

EITHER PARENT

BOTH PARENTS

[4] ABDOMINAL CIRCUMFERENCE

FEMALE

MALE

TOTAL SCORE =

HABITS

1) SMOKING

2) ALCOHOL INTAKE

DIETARY HABITS

INVESTIGATIONS

[1] FASTING BLOOD SUGAR

[2] HBA1c

[3] FASTING LIPID PROFILE

ANNEXURE III – KEY TO MASTER CHART

-	-	Absent
+	-	Present
AC	-	Abdominal circumference
b	-	Both
Cms	-	Centimeters
f	-	Father
F	-	Female
HbA1c	-	Glycosylated haemoglobin
HDL	-	High density lipoprotein
IDRS	-	Indian Diabetic Risk Score
Kg/m^2	-	Kilograms per meter square
LDL	-	Low density lipoprotein
M	-	Male
m	-	Mother
mg/dL	-	Milligrams per deciliter
mm Hg	-	Millimeters of mercury
N	-	No
VEG	-	Vegetarian
Y	-	Yes

ANNEXURE III - MASTER CHART

Serial Number	Class	Sex	Age (Years)	Diet	Smoking	History of Alcohol consumption	Physical activity	Family history	Relative	Abdominal circumference (Cms)	Body mass index (Kg/m^2)	IDRS Score	Systolic blood pressure (mm Hg)	Diastolic blood pressure (mm Hg	Fasting blood sugar (mg/dL)	HbA1c	Total Cholesterol (mg/dL)	LDL (mg/dL)	HDL (mg/dL)	Triglycerides (mg/dL)
1	4	F	22	VEG	N	Y	SEDENTARY	+	f	82	22	50	120	90	105	5.3	161	88	65	40
2	1	F	17	MIXED	N	Y	MILD	+	f	82	23	40	110	80	116	6.2	166	82	37	72
3	2	F	19	MIXED	N	Y	SEDENTARY	-	-	82	23	40	110	80	98	6.1	157	94	42	105
4	2	F	18	MIXED	N	Y	SEDENTARY	+	f	82	23	50	130	80	111	6.1	121	69	37	75
5	2	F	19	MIXED	N	N	SEDENTARY	+	f	76	20	40	120	86	118	6.1	180	106	36	124
6	4	F	23	MIXED	Y	Y	MILD	+	f	86	23	40	120	78	55	5.5	177	126	34	89
7	3	F	21	MIXED	N	N	MILD	+	f	85	21	40	130	84	99	5.1	162	95	45	111
8	2	F	19	MIXED	N	N	SEDENTARY	+	m	87	21	50	110	70	102	5.4	114	56	46	61
9	3	F	22	MIXED	Y	Y	MILD	+	f	86	21	40	118	84	88	5.4	154	103	32	98
10	3	F	21	MIXED	N	Y	MILD	+	b	90	26	60	110	80	105	6	164	105	46	68
11	4	F	23	MIXED	Y	Y	MILD	+	f	88	23	40	126	80	90	5.3	158	99	39	99
12	3	M	22	VEG	N	N	SEDENTARY	+	f	91	26	50	130	70	116	5.6	203	144	41	90
13	3	M	22	VEG	Y	Y	SEDENTARY	+	f	82	22	40	120	90	88	5	127	74	39	70
14	1	M	18	MIXED	N	N	MILD	-	-	108	25	40	130	90	92	5.5	124	76	36	47
15	1	M	19	VEG	N	N	MILD	+	b	82	22	40	120	84	104	5.7	126	88	35	83
16	2	M	20	VEG	N	Y	SEDENTARY	+	m	82	22	40	140	80	102	5.7	140	92	30	89
17	4	M	22	MIXED	N	N	MILD	+	m	91	25	40	130	70	124	5.7	182	153	16	64
18	4	M	23	MIXED	Y	Y	SEDENTARY	+	f	91	25	50	120	86	91	6.6	204	129	38	188
19	3	M	20	MIXED	N	Y	MILD	+	f	91	22	40	118	70	106	5.7	164	105	35	70
20	4	M	22	MIXED	Y	Y	SEDENTARY	+	f	80	21	40	120	80	86	5.5	130	92	45	80
21	4	M	24	MIXED	Y	Y	SEDENTARY	+	f	91	23	50	130	86	96	6.1	210	140	28	160
22	4	M	23	MIXED	Y	Y	MILD	+	m	88	22	30	134	80	90	5.6	150	98	38	100
23	3	M	22	MIXED	N	Y	MILD	+	f	90	21	40	120	78	90	5.7	126	86	42	86
24	3	M	22	MIXED	Y	Y	MILD	+	m	90	25	40	140	90	121	6.3	142	90	39	108
25	3	M	21	VEG	Y	Y	MILD	+	f	88	20	30	120	80	89	5.8	138	82	37	114
26	2	M	21	VEG	N	Y	SEDENTARY	-	-	91	22	40	118	80	96	5.8	154	102	32	146
27	1	F	18	MIXED	N	N	SEDENTARY	-	-	91	25	50	110	76	108	5.8	171	88	32	130
28	1	F	19	VEG	N	Y	MILD	+	f	92	21	50	120	74	88	5.8	105	52	28	126
29	4	F	23	MIXED	N	N	SEDENTARY	-	-	86	21	40	110	80	94	5.8	163	94	33	152
30	4	F	24	MIXED	N	Y	MILD	+	f	88	21	40	120	90	108	5.8	171	92	38	113
31	4	F	21	MIXED	Y	N	MILD	+	f	90	22	50	120	70	78	5.4	116	54	48	69
32	3	M	20	MIXED	N	Y	SEDENTARY	+	b	91	23	60	136	74	102	6.1	184	105	33	161
33	3	M	20	MIXED	N	Y	MILD	+	b	88	22	40	128	80	92	5.8	129	93	44	142
34	3	F	20	MIXED	N	Y	SEDENTARY	+	m	82	21	50	120	80	82	5.5	105	53	34	92
35	3	F	21	MIXED	N	Y	MILD	+	f	82	25	40	126	78	102	6.1	173	91	44	138
36	1	F	19	VEG	N	N	STRENOUS	-	-	74	17	0	110	70	82	5	-	-	-	-
37	1	F	19	MIXED	N	Y	MILD	-	-	76	18	20	120	82	85	5.1	-	-	-	-
38	1	M	18	VEG	N	N	STRENOUS	+	f	88	18	10	120	80	91	5.3	-	-	-	-
39	1	M	18	VEG	N	Y	MILD	-	-	84	19	20	120	80	88	5.1	-	-	-	-

ANNEXURE III - MASTER CHART

Serial Number	Class	Sex	Age (Years)	Diet	Smoking	History of Alcohol consumption	Physical activity	Family history	Relative	Abdominal circumference (Cms)	Body mass index (Kg/m^2)	IDRS Score	Systolic blood pressure (mm Hg)	Diastolic blood pressure (mm Hg	Fasting blood sugar (mg/dL)	HbA1c	Total Cholesterol (mg/dL)	LDL (mg/dL)	HDL (mg/dL)	Triglycerides (mg/dL)
40	1	M	18	MIXED	N	Y	STRENOUS	-	-	86	17	0	130	84	84	5.2	-	-	-	-
41	1	M	18	MIXED	N	N	MODERATE	-	-	92	19	20	126	70	101	5.3	-	-	-	-
42	1	F	19	MIXED	N	N	MODERATE	-	-	82	18	20	110	70	82	5.1	-	-	-	-
43	1	F	17	VEG	N	N	MODERATE	-	-	77	17	10	120	80	83	5.2	-	-	-	-
44	1	F	18	MIXED	N	Y	MODERATE	-	-	80	17	20	116	74	78	5.1	-	-	-	-
45	1	F	17	VEG	N	N	STRENOUS	+	f	81	17	20	120	90	81	5.2	-	-	-	-
46	1	F	18	VEG	N	N	STRENOUS	-	-	75	16	0	126	70	76	5	-	-	-	-
47	1	F	19	VEG	Y	Y	MODERATE	+	m	78	18	20	124	70	93	5.5	-	-	-	-
48	1	M	18	VEG	N	N	MILD	-	-	85	18	20	120	84	101	5.3	-	-	-	-
49	1	M	20	MIXED	Y	Y	MODERATE	-	-	92	19	20	130	80	113	5.7	-	-	-	-
50	1	M	19	MIXED	N	N	MILD	-	-	88	18	20	120	76	98	5.3	-	-	-	-
51	1	M	19	MIXED	N	N	MILD	-	-	86	17	20	110	68	87	5.1	-	-	-	-
52	1	M	19	VEG	N	N	MODERATE	+	f	88	17	20	110	66	94	5.3	-	-	-	-
53	1	M	19	MIXED	N	N	STRENOUS	-	-	87	18	0	120	76	84	5.2	-	-	-	-
54	1	M	19	VEG	N	N	MODERATE	-	-	91	18	20	118	70	105	5.4	-	-	-	-
55	1	M	19	MIXED	N	Y	MILD	-	-	86	17	20	126	86	83	5.2	-	-	-	-
56	1	M	18	MIXED	N	N	STRENOUS	-	-	88	18	0	110	80	85	5.2	-	-	-	-
57	1	M	19	MIXED	N	Y	MILD	-	-	88	17	20	130	82	86	5.2	-	-	-	-
58	1	F	19	MIXED	N	N	STRENOUS	+	m	82	18	20	120	78	92	5.2	-	-	-	-
59	1	F	19	MIXED	N	Y	MODERATE	-	-	75	17	10	120	70	87	5.1	-	-	-	-
60	1	F	19	VEG	N	N	MILD	-	-	77	18	20	118	70	86	5	-	-	-	-
61	1	F	19	VEG	N	N	MODERATE	-	-	82	18	20	110	70	96	5.2	-	-	-	-
62	1	M	19	VEG	N	Y	MODERATE	-	-	90	18	20	148	94	90	5.2	-	-	-	-
63	1	M	19	MIXED	N	Y	MILD	-	-	89	18	20	120	82	98	5.2	-	-	-	-
64	1	F	19	MIXED	N	N	MILD	-	-	74	17	20	120	76	88	5.1	-	-	-	-
65	1	F	18	MIXED	N	Y	MODERATE	-	-	76	17	10	118	70	107	5.3	-	-	-	-
66	1	M	18	MIXED	N	Y	MODERATE	+	f	86	19	20	136	82	112	5.5	-	-	-	-
67	1	F	18	VEG	N	N	MILD	-	-	79	18	20	120	80	86	5.2	-	-	-	-
68	1	M	19	MIXED	N	N	MODERATE	-	-	87	19	10	128	80	90	5.1	-	-	-	-
69	1	F	18	VEG	N	N	MODERATE	-	-	82	18	20	120	78	96	5.2	-	-	-	-
70	1	M	18	VEG	N	N	MODERATE	-	-	94	19	20	128	80	101	5.4	-	-	-	-
71	1	M	18	MIXED	N	N	STRENOUS	-	-	88	18	0	120	78	90	5.1	-	-	-	-
72	2	F	19	MIXED	N	N	MODERATE	-	-	86	19	20	120	70	93	5.3	-	-	-	-
73	2	F	19	MIXED	N	Y	MILD	-	-	76	18	20	124	74	92	5.2	-	-	-	-
74	2	F	19	MIXED	N	N	MILD	-	-	75	18	20	118	70	88	5.1	-	-	-	-
75	2	F	20	MIXED	N	N	MODERATE	-	-	82	18	20	130	80	96	5.3	-	-	-	-
76	2	M	19	MIXED	N	Y	MODERATE	-	-	92	20	20	134	78	108	5.3	-	-	-	-
77	2	M	20	VEG	N	N	MODERATE	+	f	88	19	20	120	80	113	5.5	-	-	-	-
78	2	M	19	VEG	N	N	MILD	-	-	86	18	20	120	76	89	5.2	-	-	-	-

ANNEXURE III - MASTER CHART

Serial Number	Class	Sex	Age (Years)	Diet	Smoking	History of Alcohol consumption	Physical activity	Family history	Relative	Abdominal circumference (Cms)	Body mass index (Kg/m^2)	IDRS Score	Systolic blood pressure (mm Hg)	Diastolic blood pressure (mm Hg	Fasting blood sugar (mg/dL)	HbA1c	Total Cholesterol (mg/dL)	LDL (mg/dL)	HDL (mg/dL)	Triglycerides (mg/dL)
79	2	M	22	MIXED	Y	N	MODERATE	-	-	90	18	20	128	74	95	5.2	-	-	-	-
80	2	M	19	VEG	N	N	MODERATE	-	-	87	18	10	110	72	97	5.1	-	-	-	-
81	2	M	19	VEG	N	N	MODERATE	-	-	91	18	20	118	68	107	5.2	-	-	-	-
82	2	F	19	MIXED	N	Y	MILD	-	-	78	18	20	108	66	99	5.4	-	-	-	-
83	2	F	19	VEG	N	Y	STRENOUS	-	-	76	17	0	106	70	83	5	-	-	-	-
84	2	F	19	MIXED	N	N	STRENOUS	-	-	78	18	0	128	86	88	5.2	-	-	-	-
85	2	M	19	MIXED	N	Y	MILD	-	-	86	18	20	118	82	92	5.1	-	-	-	-
86	2	F	19	MIXED	N	N	MODERATE	+	f	78	19	20	126	78	97	5.3	-	-	-	-
87	2	F	19	VEG	N	N	MODERATE	-	-	80	19	20	120	80	103	5.2	-	-	-	-
88	2	F	19	MIXED	N	N	MODERATE	-	-	74	17	10	124	84	95	5	-	-	-	-
89	2	M	20	MIXED	N	Y	MODERATE	+	m	89	19	20	128	82	110	5.6	-	-	-	-
90	2	F	20	MIXED	Y	Y	MODERATE	-	-	74	18	10	126	76	92	5.3	-	-	-	-
91	2	M	19	MIXED	N	N	MODERATE	-	-	92	19	20	120	74	102	5.4	-	-	-	-
92	2	F	19	VEG	N	N	MILD	-	-	76	18	20	120	70	88	5.1	-	-	-	-
93	2	F	19	MIXED	N	N	MODERATE	-	-	82	19	20	118	70	105	5.3	-	-	-	-
94	2	M	20	MIXED	N	N	STRENOUS	+	f	88	18	10	120	80	95	5.1	-	-	-	-
95	2	M	19	VEG	N	N	MODERATE	-	-	90	18	20	120	80	109	5.3	-	-	-	-
96	2	M	19	VEG	N	Y	MILD	-	-	88	19	20	126	80	101	5.2	-	-	-	-
97	2	M	21	VEG	N	N	MILD	-	-	89	19	20	130	84	105	5.5	-	-	-	-
98	2	F	19	MIXED	N	N	MODERATE	-	-	86	18	20	124	80	98	5.2	-	-	-	-
99	2	M	19	MIXED	N	Y	MODERATE	-	-	92	20	20	120	70	94	5.3	-	-	-	-
100	2	F	19	MIXED	N	N	STRENOUS	+	f	85	20	20	126	80	107	5.6	-	-	-	-
101	2	M	19	VEG	N	Y	MILD	-	-	88	19	20	110	70	101	5.3	-	-	-	-
102	2	M	19	MIXED	N	N	MILD	-	-	86	19	20	118	80	96	5.3	-	-	-	-
103	2	M	19	MIXED	N	N	MILD	-	-	88	18	20	116	74	94	5.4	-	-	-	-
104	2	F	19	VEG	N	N	MILD	-	-	78	20	20	110	70	102	5.3	-	-	-	-
105	2	F	19	MIXED	N	Y	MODERATE	-	-	82	19	20	120	80	90	5.1	-	-	-	-
106	2	F	21	VEG	Y	N	MODERATE	-	-	80	19	20	106	70	88	5.1	-	-	-	-
107	2	F	20	MIXED	Y	N	MODERATE	-	-	76	18	10	108	70	93	5.1	-	-	-	-
108	2	M	19	MIXED	N	Y	MODERATE	-	-	88	19	10	130	70	96	5.2	-	-	-	-
109	2	M	18	MIXED	N	Y	MODERATE	+	m	86	20	20	128	84	98	5.5	-	-	-	-
110	2	M	19	MIXED	N	Y	MODERATE	-	-	94	19	20	120	80	103	5.4	-	-	-	-
111	2	M	19	MIXED	N	N	MILD	-	-	88	19	20	110	70	98	5.4	-	-	-	-
112	2	F	19	MIXED	N	Y	MILD	-	-	78	19	20	110	76	84	5.3	-	-	-	-
113	2	M	19	MIXED	N	N	MILD	-	-	86	19	20	116	82	88	5.3	-	-	-	-
114	2	M	19	VEG	N	N	MODERATE	-	-	90	19	20	110	70	85	5.2	-	-	-	-
115	2	M	19	VEG	N	N	MODERATE	-	-	88	20	10	110	80	94	5.3	-	-	-	-
116	2	M	20	MIXED	N	N	MODERATE	-	-	90	19	20	120	82	101	5.3	-	-	-	-
117	2	M	19	MIXED	Y	N	MILD	-	-	87	19	20	130	84	96	5.3	-	-	-	-

ANNEXURE III - MASTER CHART

Serial Number	Class	Sex	Age (Years)	Diet	Smoking	History of Alcohol consumption	Physical activity	Family history	Relative	Abdominal circumference (Cms)	Body mass index (Kg/m^2)	IDRS Score	Systolic blood pressure (mm Hg)	Diastolic blood pressure (mm Hg	Fasting blood sugar (mg/dL)	HbA1c	Total Cholesterol (mg/dL)	LDL (mg/dL)	HDL (mg/dL)	Triglycerides (mg/dL)
118	2	M	19	MIXED	N	N	STRENOUS	+	f	92	19	20	128	80	106	5.5	-	-	-	-
119	2	M	19	MIXED	N	Y	MODERATE	-	-	88	20	10	130	76	87	5.2	-	-	-	-
120	2	F	19	MIXED	N	N	MILD	-	-	76	18	20	118	70	88	5.2	-	-	-	-
121	2	F	21	MIXED	Y	Y	MILD	-	-	78	20	20	104	68	98	5.5	-	-	-	-
122	3	M	20	MIXED	N	N	MODERATE	-	-	92	20	20	108	70	101	5.4	-	-	-	-
123	3	M	20	MIXED	Y	N	MODERATE	-	-	90	19	20	116	70	94	5.3	-	-	-	-
124	3	M	20	VEG	N	N	STRENOUS	+	f	90	20	20	110	70	103	5.7	-	-	-	-
125	3	M	21	VEG	Y	Y	MODERATE	-	-	88	19	10	126	84	95	5.4	-	-	-	-
126	3	M	20	VEG	N	Y	MILD	-	-	88	19	20	146	90	94	5.3	-	-	-	-
127	3	M	20	VEG	N	N	MILD	-	-	88	19	20	138	84	97	5.3	-	-	-	-
128	3	M	20	MIXED	Y	N	MILD	-	-	86	18	20	130	80	82	5.2	-	-	-	-
129	3	M	21	MIXED	Y	N	MILD	-	-	88	19	20	128	80	93	5.4	-	-	-	-
130	3	F	20	MIXED	N	N	MODERATE	-	-	86	19	20	120	78	86	5.3	-	-	-	-
131	3	F	20	MIXED	Y	N	MODERATE	-	-	82	18	20	126	76	97	5.3	-	-	-	-
132	3	F	20	MIXED	N	Y	MILD	-	-	78	18	20	120	70	91	5.1	-	-	-	-
133	3	F	20	MIXED	N	N	MILD	-	-	76	17	20	130	90	84	5.1	-	-	-	-
134	3	F	22	MIXED	Y	N	MILD	-	-	78	18	20	108	70	102	5.4	-	-	-	-
135	3	F	20	VEG	N	Y	MODERATE	-	-	86	19	20	118	76	97	5.8	-	-	-	-
136	3	F	20	MIXED	N	N	MODERATE	+	f	76	18	20	120	80	85	5.2	-	-	-	-
137	3	F	20	VEG	N	N	MILD	-	-	74	17	20	118	78	83	5.1	-	-	-	-
138	3	F	21	MIXED	N	N	MILD	-	-	78	19	20	110	70	93	5.3	-	-	-	-
139	3	M	20	VEG	N	N	MILD	-	-	86	18	20	120	80	95	5.2	-	-	-	-
140	3	F	20	VEG	N	N	MODERATE	-	-	80	18	20	110	76	92	5.3	-	-	-	-
141	3	F	20	MIXED	N	N	MILD	-	-	75	17	20	116	70	84	5	-	-	-	-
142	3	M	21	MIXED	N	Y	MODERATE	-	-	88	19	10	120	80	98	5.8	-	-	-	-
143	3	M	22	MIXED	N	N	MODERATE	-	-	93	19	20	120	80	96	5.7	-	-	-	-
144	3	M	21	MIXED	N	Y	MODERATE	-	-	88	18	10	110	76	92	5.2	-	-	-	-
145	3	M	21	MIXED	N	N	MODERATE	-	-	90	19	20	120	70	88	5.2	-	-	-	-
146	3	F	20	MIXED	N	N	MODERATE	+	f	76	17	20	120	80	85	5	-	-	-	-
147	3	F	20	MIXED	Y	N	MILD	-	-	78	18	20	118	70	96	5.3	-	-	-	-
148	3	F	20	VEG	Y	N	MODERATE	-	-	82	19	20	108	74	102	5.3	-	-	-	-
149	3	F	20	VEG	N	N	MILD	-	-	78	18	20	110	70	91	5.2	-	-	-	-
150	3	F	21	VEG	Y	N	MILD	-	-	78	19	20	116	78	95	5.2	-	-	-	-
151	3	F	20	VEG	N	N	MILD	-	-	74	17	20	106	70	84	5.1	-	-	-	-
152	3	M	21	VEG	N	N	MODERATE	-	-	86	18	10	126	80	95	5.4	-	-	-	-
153	3	M	20	VEG	N	N	MODERATE	-	-	88	18	10	130	86	102	5.3	-	-	-	-
154	3	M	22	MIXED	N	Y	MODERATE	-	-	88	18	10	126	80	93	5.2	-	-	-	-
155	3	F	20	MIXED	N	N	STRENOUS	+	f	82	19	20	120	78	97	5.5	-	-	-	-
156	3	F	20	MIXED	N	N	MILD	-	-	78	18	20	120	80	84	5.1	-	-	-	-

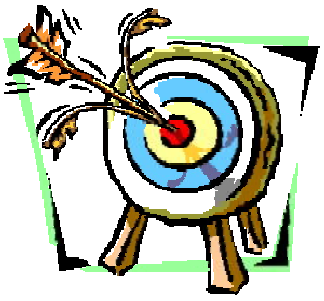
ANNEXURE III - MASTER CHART

Serial Number	Class	Sex	Age (Years)	Diet	Smoking	History of Alcohol consumption	Physical activity	Family history	Relative	Abdominal circumference (Cms)	Body mass index (Kg/m^2)	IDRS Score	Systolic blood pressure (mm Hg)	Diastolic blood pressure (mm Hg	Fasting blood sugar (mg/dL)	HbA1c	Total Cholesterol (mg/dL)	LDL (mg/dL)	HDL (mg/dL)	Triglycerides (mg/dL)
157	3	F	20	MIXED	N	N	MODERATE	-	-	78	18	10	130	90	85	5.2	-	-	-	-
158	3	M	20	MIXED	Y	N	STRENOUS	+	m	86	18	10	118	76	93	5.2	-	-	-	-
159	3	M	20	MIXED	N	Y	MILD	-	-	88	18	20	126	78	97	5.3	-	-	-	-
160	3	M	21	MIXED	N	Y	MODERATE	-	-	90	18	20	144	90	88	5.2	-	-	-	-
161	3	F	20	MIXED	N	N	MODERATE	-	-	84	20	20	118	78	96	5.9	-	-	-	-
162	3	F	20	VEG	Y	N	MILD	-	-	74	17	20	120	70	81	5.1	-	-	-	-
163	3	F	21	MIXED	Y	Y	MODERATE	-	-	78	18	10	110	80	100	5.5	-	-	-	-
164	3	F	20	MIXED	N	N	MODERATE	-	-	76	18	10	126	78	85	5.2	-	-	-	-
165	3	F	21	MIXED	Y	N	MILD	-	-	74	18	20	124	80	84	5.2	-	-	-	-
166	3	M	21	MIXED	N	N	STRENOUS	-	-	86	18	0	110	70	88	5.2	-	-	-	-
167	3	M	21	MIXED	N	Y	MILD	-	-	88	18	20	120	80	87	5.1	-	-	-	-
168	3	F	20	VEG	N	N	STRENOUS	-	-	74	17	0	120	80	84	5	-	-	-	-
169	4	F	21	VEG	N	N	MODERATE	-	-	78	19	10	110	76	93	5.2	-	-	-	-
170	4	F	21	MIXED	Y	Y	MODERATE	-	-	76	18	10	110	70	87	5.2	-	-	-	-
171	4	F	21	MIXED	N	N	MILD	-	-	78	19	20	100	70	94	5.4	-	-	-	-
172	4	M	22	MIXED	N	N	STRENOUS	+	b	88	19	20	126	82	96	5.7	-	-	-	-
173	4	M	22	MIXED	N	N	MODERATE	-	-	92	19	20	110	76	102	5.6	-	-	-	-
174	4	M	21	VEG	N	N	MODERATE	-	-	88	19	10	118	78	96	6	-	-	-	-
175	4	M	23	VEG	Y	Y	MILD	-	-	86	18	20	110	70	94	5.3	-	-	-	-
176	4	M	22	MIXED	Y	N	MODERATE	-	-	90	18	20	110	74	88	5.3	-	-	-	-
177	4	M	22	MIXED	Y	N	MILD	-	-	88	19	20	120	80	103	5.5	-	-	-	-
178	4	F	21	MIXED	N	N	MILD	-	-	78	19	20	100	70	94	5.9	-	-	-	-
179	4	M	22	MIXED	Y	N	MODERATE	-	-	86	17	10	130	84	85	5.2	-	-	-	-
180	4	F	22	VEG	N	N	MODERATE	-	-	74	17	10	106	70	78	5.1	-	-	-	-
181	4	F	21	VEG	N	Y	MODERATE	+	f	78	18	20	100	70	93	6.1	-	-	-	-
182	4	F	22	MIXED	N	Y	MODERATE	+	f	78	18	20	116	70	104	5.4	-	-	-	-
183	4	M	22	VEG	N	Y	MILD	-	-	88	18	20	126	80	89	5.3	-	-	-	-
184	4	M	22	VEG	N	N	MILD	-	-	86	18	20	128	80	84	5.2	-	-	-	-
185	4	M	22	MIXED	N	N	STRENOUS	-	-	86	18	0	110	78	85	5.2	-	-	-	-
186	4	M	22	MIXED	Y	Y	MILD	-	-	85	17	20	120	80	87	5.2	-	-	-	-
187	4	M	22	MIXED	Y	Y	MODERATE	-	-	88	18	10	120	84	92	5.3	-	-	-	-
188	4	M	23	MIXED	N	Y	MILD	-	-	88	18	20	130	78	90	5.2	-	-	-	-
189	4	M	22	MIXED	N	Y	MODERATE	-	-	90	19	20	118	80	95	5.4	-	-	-	-
190	4	F	21	MIXED	N	N	MODERATE	+	m	78	18	20	110	76	96	5.8	-	-	-	-
191	4	F	22	VEG	N	N	MODERATE	-	-	76	18	10	110	70	89	5.3	-	-	-	-
192	4	M	22	MIXED	N	N	MILD	-	-	84	17	20	118	70	82	5.1	-	-	-	-
193	4	M	22	VEG	N	Y	MODERATE	-	-	86	18	10	108	74	86	5.2	-	-	-	-
194	4	M	22	VEG	N	N	STRENOUS	+	b	88	19	20	110	80	106	5.6	-	-	-	-
195	4	M	22	MIXED	N	Y	MILD	-	-	88	20	20	120	90	87	5.3	-	-	-	-

ANNEXURE III - MASTER CHART

Serial Number	Class	Sex	Age (Years)	Diet	Smoking	History of Alcohol consumption	Physical activity	Family history	Relative	Abdominal circumference (Cms)	Body mass index (Kg/m^2)	IDRS Score	Systolic blood pressure (mm Hg)	Diastolic blood pressure (mm Hg	Fasting blood sugar (mg/dL)	HbA1c	Total Cholesterol (mg/dL)	LDL (mg/dL)	HDL (mg/dL)	Triglycerides (mg/dL)
196	4	F	22	MIXED	Y	N	MILD	-	-	74	18	20	106	70	77	5.1	-	-	-	-
197	4	F	23	MIXED	N	N	MODERATE	-	-	78	19	10	100	70	97	5.7	-	-	-	-
198	4	F	23	MIXED	N	N	MODERATE	-	-	75	17	10	120	68	83	5.2	-	-	-	-
199	4	M	22	MIXED	N	Y	MILD	-	-	86	18	20	110	70	86	5	-	-	-	-
200	4	F	23	MIXED	Y	Y	MODERATE	-	-	75	17	10	116	70	83	5.2	-	-	-	-

Introduction



Objectives

Review of Literature

Methodology

Results

Discussion

Conclusion

Summary

Bibliography

Annexure-I

Annexure-II

Annexure-III