“ERECTILE DYSFUNCTION IN PATIENTS OF DIABETES MELLITUS - A CROSS SECTIONAL STUDY”

REG.NO. BH0111009

Dissertation

Submitted to the
KLE University, Belgaum, Karnataka

In Partial Fulfillment
of the requirements for the degree of

M. S.
in
GENERAL SURGERY

DEPARTMENT OF SURGERY,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
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ENDORSEMENT BY THE HOD/PRINCIPAL/ HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled **"ERECTILE DYSFUNCTION IN PATIENTS OF DIABETES MELLITUS - A CROSS SECTIONAL STUDY"** is a bonafide research work done by **THE CANDIDATE REG. NO. BH0111009**.

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LIST OF ABBREVIATIONS

AD	-	Anno Domini
ADA	-	American Diabetes Association
AGEs	-	Advanced glycation end products
ATP	-	Adenosine triphosphate
B.C.	-	Before Christ
BMI	-	Body mass index
CAD	-	Coronary artery disease
CDC	-	Centre for Disease Control and prevention
DCCT	-	The Diabetes Control and Complications Trial
DKA	-	Diabetic ketoacidosis
dL	-	Decilitre
DM	-	Diabetes mellitus
DNA	-	Deoxyribonucleic acid
ED	-	Erectile dysfunction
ESRD	-	End stage renal disease
ExCEED	-	Exploratory Comprehensive Evaluation of Erectile Dysfunction
HDL	-	High density lipoprotein
HHS	-	Hyperglycemic hyperosmolar state
HNF	-	Hepatocyte nuclear transcription factor
HRQOL	-	Health-related quality of life
ICMR	-	Indian Council of Medical Research
IDDM	-	Insulin dependent diabetes mellitus
IDF	-	International Diabetes Federation

IFG	-	Impaired fasting glucose
IGT	-	Impaired glucose tolerance
IPF	-	Insulin promoter factor
Kg	-	Kilogram
m	-	Meter
mg	-	Milligram
mg/dL	-	Milligram per deciliter
MI	-	Myocardial infarction
mm Hg	-	Millimeter of mercury
mmol	-	Milli mole
MODY	-	Maturity onset diabetes of young
NGT	-	Normal glucose tolerance
NIDDK	-	National Institute of Diabetes and Digestive Kidney Diseases
NIDDM	-	Non insulin dependent diabetes mellitus
NPH	-	Neutral protamine hagedorn
PAD	-	Peripheral arterial disease
PODIS	-	Prevalence of Diabetes in India Study
PZI	-	Potamine insulin
T2D	-	Type 2 diabetes
UKPDS	-	The United Kingdom Prospective Diabetes Study

ABSTRACT

Background and Objectives

One of the independent risk factor for erectile dysfunction is diabetes mellitus. The present study planned to determine the prevalence and factors associated with ED in DM.

Methodology

The present hospital based cross-sectional study was conducted over a period of one year from January 2012 to December 2012 on a total of 208 patients with type 1 or 2 diabetes attending Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. National Institutes of Health (NIH) approved questionnaire for International Index of Erectile Function (IIEF) was used to interview each patient to assess for ED.

Results

In the present study 12.98% of patients had erectile dysfunction score between 13 to 18 suggestive of mild to moderate erectile dysfunction and 9.62% with 19 to 24 scores suggestive of mild degree. The prevalence of erectile dysfunction was 32.21%. The prevalence of erectile dysfunction was significantly high in 71 to 80 years age group (75%) and 61 to 70 years (35.90%) ($p<0.001$). The mean age in patients with erectile dysfunction was significantly high (58.40 ± 10.96 years) compared to those without erectile dysfunction (51.00 ± 11.16 years) ($p<0.001$) Of the 119 patients with duration of diabetes between one to five years 42.02% had erectile dysfunction and of the 3 patients with duration of

more than five years 66.67% had erectile dysfunction ($p<0.001$). Prevalence of erectile dysfunction was higher in patients with HbA1c levels between 7.0 to 8.5 (32.76%) and > 8.5 (37.07%) compared to those who had HbA1c < 7.0 (14.71%). ($p<0.049$). Prevalence of erectile dysfunction was higher in patients with history of smoking (63.64%) ($p<0.001$) and alcohol intake (51.85%) ($p<0.001$). The prevalence of erectile dysfunction was also significantly high in patients with history of hypertension (59.7%) ($p<0.001$).

Conclusion and interpretation

The erectile dysfunction in patients with diabetes mellitus was significantly prevalent with age, diabetic duration of diabetes, history of hypertension, cardiovascular disease, glycemic control and hypertriglyceridemia.

Keywords

Diabetes mellitus; Erectile dysfunction; International Index of Erectile Function;

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Chapter 1

Introduction

INTRODUCTION

The inability to attain or maintain erection sufficient for vaginal penetration is clinically called as erectile dysfunction. The word “impotence” may also be used to describe other problems that interfere with sexual intercourse and reproduction, such as lack of sexual desire and problems with ejaculation or orgasm.[1]

Erectile dysfunction becomes more frequent with age, but is not an inevitable consequence of normal aging. It is usually due to organic factors or diseases, such as pelvic vascular disease, diabetes mellitus, and neurodegenerative disorders, side effects of medication, pelvic surgery, and trauma. Erectile dysfunction (ED) is a major health problem, impairs sexual performance and diminishes self-esteem leading to breech in marital relationship.[2]

Given the trends of increase in life expectancy across the Western world (i.e., the aging of the general population) and the high prevalence of diabetes and cardiovascular disease, the impact on lifestyle and quality of life imposed by ED in men is projected to be substantial. It was estimated that, in 1995, over 152 million men worldwide experienced ED. For 2025, the prevalence of ED is predicted to be approximately 322 million worldwide.[3]

Diabetes Mellitus is a metabolic disorder of multiple etiologies characterized by chronic hyperglycemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action or both.[4]

Diabetes mellitus (DM) is a chronic and potentially disabling disease which is reaching an epidemic proportion in many parts of the world. It is a major and growing threat to global public health. The biggest impact of the disease is on adults of working age; particularly in developing countries. The vast majority of cases of the diabetes fall into two broad categories: those having little or no endogenous insulin secretory capacity (IDDM or type 1 DM) and those who retain endogenous insulin secretory capacity but have a combination of resistance to insulin action and an inadequate compensatory insulin secretory response (NIDDM, or Type 2 DM).[4,5]

Centers for Disease Control and Prevention (CDC) report in 2011 estimated that nearly 26 million Americans have diabetes.[6] Type 2 diabetes mellitus (DM) accounts for more than 90% of the diabetic population world wide. Additionally, an estimated 79 million Americans have prediabetes. Rates of diabetes are increasing worldwide. The International Diabetes Federation predicts that the number of people living with diabetes will to rise from 366 million in 2011 to 552 million by 2030.[7] The top 10 countries in number of people with diabetes are currently India, China, the United States, Indonesia, Japan, Pakistan, Russia, Brazil, Italy, and Bangladesh. The prevalence of diabetes and its adverse health effects have risen more rapidly in South Asia than in any other region of the world.[8]

Thirty years ago, the prevalence of diabetes in India based on the multicentric survey[9] by Indian Council of Medical Research (ICMR) was around two percent in urban India and one percent in rural India. In just three decades, these prevalence rates have shot up to 12% to 16% in urban India and 3% to 8%

in rural India, in adults over 20 years of age. These represents a 600% to 800% increase in prevalence rates of diabetes something which is unparallel in any Western nation. Indeed, India is now referred to as the **"Diabetic Capital"** of the world.

Further, DM is associated with several complications. The complications of diabetes mellitus include retinopathy, nephropathy, and neuropathy (both peripheral and autonomic). The risk for atherosclerotic vascular disease is also increased in persons with DM. The risk for microvascular and neuropathic complications is related to both duration of diabetes and the severity of hyperglycemia; the increased risk for vascular disease actually antedates the onset of hyperglycemia to the degree associated with diabetes mellitus.[4]

People with diabetes have an increased risk of sexual dysfunction. Men with diabetes are three times more likely to experience erectile dysfunction than those without diabetes, according to the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK). In addition, men with diabetes and erectile dysfunction may experience the problem 10 to 15 years earlier in life than non diabetics. It is not uncommon for a man seeking treatment for erectile dysfunction to learn that he has diabetes and that it may be the cause of his sexual difficulties. Diabetic male may experience sexual dysfunction either in form of pure erectile dysfunction or Male hypogonadism. Researchers have found that obesity, diabetes, elevated blood pressure or unhealthy amounts of blood fats (cholesterol and triglycerides) are associated with hypogonadism.[2]

Erectile dysfunction (ED) is a common condition among men with diabetes.[10-15] ED in diabetic men is multi-factorial with neuropathy, atherosclerosis of penile blood vessels and psychological factors being the main underlying contributors.[16]

The prevalence of ED depends on the population studied and the definition and methods used. So far, very few studies have been carried out to establish the incidence and prevalence of this condition in a diabetes mellitus patients. Also, epidemiological data on the prevalence of ED in DM in India is scarce. There are no studies from the state of Karnataka showing the prevalence of ED in DM patients. Hence the present study undertaken to determine the prevalence and factors associated with ED in DM.

Chapter 2

Objectives

OBJECTIVES

The objectives of the present study were;

1. To determine the prevalence of erectile dysfunction in patients with diabetes mellitus attending KLES Dr. Prabhakar Kore Hospital Hospital and Medical Research Centre, Belgaum or Diabetic Inpatients admitted in ward.

2. To determine the factors associated with erectile dysfunction in patients with diabetes mellitus.

Chapter 3

Review of Literature

REVIEW OF LITERATURE

Historical review

Diabetes is perhaps as old as mankind. Cognizance of symptoms related to diabetes and recognition of the disorder was confined to a few geographic and cultural locations in the Ancient Era (up to 600 AD).

The knowledge acquired during this period was lost sight of and progress was tardy and indiscrete during the medieval period (600 to 1500 AD).

With the advent of modern age (1500 to 1758 AD) and its progression to renaissance and industrial revolution (1750 to 1850 AD), certain key features of diabetes were rediscovered and some new information was generated which stand out as landmarks in characterizing diabetes.

During the later decades of the 19th and first half of the 20th century, all round progress was achieved in the knowledge of pathology, predisposing factors, management, course and complications of diabetes mellitus. Growth of knowledge has been very fast in course of the second half of the last century (contemporary period) involving epidemiology, genetics, immunology and molecular biology which has led to accumulation of voluminous information on various aspects of this versatile disorder.[9,17]

Some key developments in scientific and clinical understanding of diabetes may be summarized as follows:

The earliest mention of diabetes like illness characterized by polyuria can be traced to Eqyptian Papyrus dating back to around 1550 B.C.[17]

- The sweet taste of diabetic urine was noted in the 5th and 6th century AD by the Indian physicians and in the 17th century by Thomas Willis. The term 'Diabetes mellitus', an allusion to the honeyed taste of urine, was first used in the late 18th century by John Rollo and others, to distinguish it from other polyuric states in which urine was tasteless.[17]

- In 1776, Matthew Dobson discovered that diabetic serum as well as urine contained sugar, and concluded that diabetes was a systemic condition rather than a disease of kidneys.[17]

- Claude Bernard made numerous discoveries in the field of metabolism and diabetes during the mid to late 19th century, describing the storage of glucose in the liver as glycogen and hyperglycemia in experimental animals.[17]

- In 1889, Oskar Minkowski and Josef Von Mering observed that total pancreatectomy produced diabetes in dogs.[17]

- In 1893, Edovard Laguesse named that pancreatic islets after Paul Langerhans, who had described them in 1869, and suggested that they produced a glucose lowering substance. This then hypothetical hormone was named 'insulin' by Jean de Meyer in 1909, over a decade before its discovery.[17]

- Various workers, including George Zueler (Germany) and Nicolas Paulesco (Romania), isolated active but impure hypoglycemic extracts from the pancreas during the first two decades of the 20th century; but toxic side effects precluded their formal testing in diabetic patients.[17]

- Insulin was discovered at the University of Toronto in 1921, through collaboration between Frederick G. Banting, Charles H. Best, James B. Collip and J.J.R. Macleod. Insulin was extracted from chilled pancreas in an acid / ethanol mixture; the extracts were found to lower blood glucose levels in pancreatectomized dogs and were first tested in a human diabetic in January 1922.[17]

- Major advances in the understanding of diabetes and metabolism have included:

    - The sequencing of insulin in 1955 by Frederick Sanger and elucidation of its three dimensional structure in 1969 by Dorothy Hodgkin.

    - The measurement of insulin concentration using the first radio immunoassay by Solomon Berson and Rosalyn Yalow in 1959.

    - The isolation of proinsulin in 1967 by Donal Steiner's group.

    - Identification of specific insulin receptors by Pierre Freychet and colleagues in 1971, and

    - The sequencing of the insulin receptor in 1985.

Landmarks in insulin discovery and development[9]

Year	Contribution	Discovery, development
1869	Paul Langerhans	Identified Islet cells
1889	Joseph Von Mehring and Oskar Minkowski	Identified pancreas as the origin of fatal diabetes mellitus
1908	George Ludwig Zeuler	Injected 'acomatrol' pancreatic extract into dying patient
1921	Paulesco	Pancreatin (Insulin)
1921	Banting and Best	Work started at the University of Toronto in the month of April
1922	Banting and Best	Insulin Isolation
1923	Nordisk Insulin Laboratory	Started production of Insulin
1926	Abel	Prepared the first crystalline insulin
1934	Svedberg	Molecular weight of insulin was determined
1936	Hagedorn (Novo Nordisk)	Development of the first protamine Insulin (PZI)
1946	Hagedorn (Novo Nordisk)	Development of the first prolonged acting Insulin-Neutral Protamine Hagedorn (NPH) or Isophane insulin
1952	Hallas-Moller and Schlichtkrull	Development of the Lente series of Insulin
1955	Frederik Sanger	Elucidation on the structure of insulin and awarded with Nobel prize
1964	Novo Nordisk	Premixed insulin preparation were made available
1981	Jan Markussen and associates	First commercially available human insulin preparation using DNA technology
1996	Eli Lilly and company	First commercially introduced insulin analog, Lispro
2000	Novo Nordisk	Rapid- acting insulin analog- insulin aspart made available
2000	Aventis Pharmaceuticals	Marketing of long-lasting form of insulin – insulin Glargine
2003	Novo Nordisk	Detemir another long-acting insulin analogue introduced

Diabetes mellitus refer to a group of common metabolic disorder that shares the phenotype of hyperglycemia. Several distinct types of DM exist and are caused by a complex interaction of genetics and environmental factors. Depending on the etiology of the DM, factors contributing to hyperglycemia include reduce insulin secretion, decreased glucose utilization, and increased glucose production.[4,18]

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. In the United States, DM is the leading cause of end-stage renal disease (ESRD), non traumatic lower extremity amputations, and adult blindness. It also predisposes to cardiovascular diseases. With an increasing incidence worldwide, DM will be leading cause of morbidity and mortality for the foreseeable future.[4,9,18-20]

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[9]

- Type 1
- Type 2

Both types of diabetes are preceded by a phase of abnormal glucose homeostasis as the pathogenic processes progresses. 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).[9]

Spectrum of glucose homeostasis and diabetes mellitus[21]

Type of diabetes	Normal glucose tolerance (NGT)	Impaired fasting glucose or impaired glucose tolerance	Hyperglycemia: 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[9]

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

Prevalence

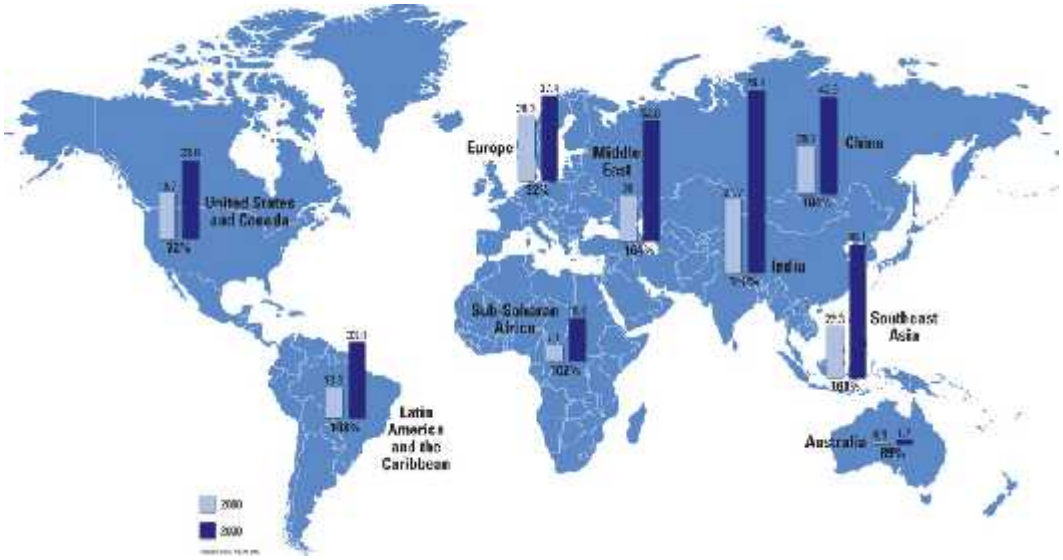


Figure 1. Worldwide prevalence of diabetes - Diabetes is a global problem with devastating human social and economic impact. Today, around 250 million people worldwide are living with diabetes and by 2025 this total is expected to increase to over 380 million

Diabetes is fast becoming the epidemic of the 21st century. Type 2 diabetes, which is more prevalent (more than 90% of all diabetes cases) and the main driver of the diabetes epidemic, now affects 5.9% of the world's adult population with almost 80% of the total in developing countries.[22]

Nowhere is the diabetes epidemic more pronounced than in India as the World Health Organization (WHO) reports show that 32 million people had diabetes in the year 2000.[17] World Health Organization reported that, 346 million people worldwide have diabetes. In 2004, an estimated 3.4 million people died

from consequences of high blood sugar. More than 80% of diabetes deaths occur in low- and middle-income countries. WHO projects that, diabetes deaths will double between 2005 and 2030.[23]

Race

The prevalence of type 2 diabetes mellitus varies widely among various racial and ethnic groups. Type 2 diabetes mellitus is becoming virtually pandemic in some groups of Native Americans and Hispanic people. The risk of retinopathy and nephropathy appears to be greater in blacks, Native Americans, and Hispanics.[24]

Sex

Type 2 DM is slightly more common in older women than men.[24]

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.[24]

Indian scenario

India is in the midst of an ever-increasing epidemic of diabetes mellitus. Data on type 1 diabetes mellitus from our country is scant. Clinic based data from

the mid sixties to the eighties reported the prevalence of childhood diabetes with onset below 15 years of age as being one to four percent of all the diabetic subjects attending clinics in different parts of the country.[9,18]

Figure 2. Prevalence of diabetes in India – India, the world's second most populous contry, now had more people with type 2 diabetes (> 50 million) than any other nation. Diabetes in India has long passes the stage of an epidemic and number have given the country the dubious distinction of "Diabetes Capital" of the world

According to recent study also, almost 95% of childhood diabetes reportedly belongs to Type 1 DM. Early onset type 2 diabetes, MODY, fibrocalculous pancreatic diabetes and diabetes associated with genetic syndromes accounted for the remaining cases.[9]

Type 2 DM accounts for more than 90% of all patients with diabetes in India. According to WHO there were an estimated 19.4 million diabetes individuals in 1995, and this number is projected to increase in 80 million by 2030. The ICMR study (1972 to 1975) was the first systematic nationwide collaborative study on the prevalence of diabetes mellitus.[9,18]

The prevalence of diabetes was found to be 2.8% in rural and five percent in the urban population above the age of 40 years. The prevalence of Diabetes in India Study (PODIS) carried out in 77 centres recently reported a standardized prevalence rate for DM, in the total urban and rural population of 4.3, 5.9 and 2.7% respectively.[9]

Several epidemiological studies in migrant Indians and India itself show that, the population has a high genetic predisposition for diabetes, which is precipitated by environmental factors such as urbanization.[22] The prevalence of diabetes is four to six fold lower in rural areas, which is probably attributed to a conventional lifestyle which has beneficial effect on glucose tolerance (IGT). National Urban Diabetes Survey done in six cities, found age standardized prevalence rates of 12% for diabetes; with a slight male preponderance and 14% for impaired glucose tolerance. Subjects under the age of 40 years, had a prevalence of five percent for DM and 13% prevalence of impaired glucose tolerance.

The International Diabetes Federation (IDF) estimates the total number of diabetic subjects to be around 40.9 million in India and this is further set to rise to 69.9 million by the year 2025.[25] It is clear that in the last two decades, there has

been a marked increase in the prevalence of diabetes among both urban as well as the rural Indians, with a suggestion that Southern India has seen the sharpest increase. Subsequent studies confirmed this high prevalence of diabetes in urban south India. Although in rural India the prevalence of diabetes is much lower than in the urban population, even here the prevalence rates are rapidly rising, though clearly more studies are needed. Variations in the prevalence rates of diabetes in different urban populations of India are expected because of the large variation in the prevalence of cardiovascular risk factors in different regions and states. It is evident that there is a shift in age of onset to younger age groups, which is alarming and this could have adverse effects on the nation's economy. Hence, the early identification of at-risk individuals and appropriate intervention to increase physical activity, bring about changes in dietary habits could to a great extent help to prevent/ delay, the onset of diabetes and thus reduce the burden due to its associated complications in India.[22]

The world wide prevalence of DM has risen dramatically over the past two decades, from an estimated 30 million cases in 1985 to 177 million in 2000. Based on current trends, more than 360 million individuals will have diabetes by the year 2030. Although the prevalence of both type 1 and type 2 DM is increasing world wide, the prevalence of type 2 DM is rising much more rapidly because of increasing obesity and reduced activity levels as countries become more industrialized. This is true in most countries, and 6 of the top 10 countries with the highest rates are in Asia. In the United States, the centre for Disease control and prevention (CDC) estimated that 20.8 million persons, or seven

percent of the population, had diabetes in 2005 (30% of individuals with diabetes were undiagnosed).[9,18]

The prevalence is similar in men and women throughout most age ranges but is slightly greater in men more than 60 years. World wide estimates project that in 2030 the greatest number of individuals with diabetes will be 45 to 64 years of age.[18]

Causes for diabetic pandemic

The type 2 DM epidemic is tightly and consistently linked to that of obesity, both geographically and chronologically. Many factors like, urbanization and mechanization, together with globalized pattern of western pattern of lifestyle, together with poverty, lack of education and low socio-economic status and inner city deprivation are emerging as significant risk factors for DM. Lack of breast feeding, low birth weight is associated with insulin resistance and type 2 DM in adult life (especially in subjects who become obese) due to long term metabolic response during poor fetal nutrition.[26]

Obesity

About 80% of patients are obviously obese at the time of diagnosis, usually with a central fat distribution in and around the abdominal cavity. In addition, many of those who are not traditionally obese, by weight criteria have increased percentage of fat predominantly distributed in the abdominal region. It is the most obvious target to prevent DM.

Body mass index (BMI)

Three key anthropometric measurements are important to evaluate the degree of obesity – weight, height and waist circumference. The BMI, calculated as weight (kg)/height $(m)^2$, or as weight (lbs)/height$(inches)^2$ x 703, is used to classify weight status and risk of disease. Body mass index, is used since it provides an estimate of body fat and is related to risk of disease. Lower BMI thresholds for overweight and obesity have been proposed for the Asia-Pacific region since this population appears to be at risk at lower body weights for glucose and lipid abnormalities.[4]

Classification of weight status and risk of disease[27]

	BMI (Kg/m^2)	**Obesity Class**	**Risk of Disease**
Underweight	<18.5		
Healthy weight	18.5 – 24.9		
Overweight	25.0 – 29.9		Increased
Obesity	30.0 – 34.9	I	High
Obesity	35.0 – 39.9	II	High
Extreme Obesity	≥ 40	III	Extremely high

Criteria for the diagnosis of diabetes mellitus[17,18]

- 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[4]

Widespread use of the fasting plasma glucose (FPG) as a screening test for type 2 DM is recommended because:

1. A large number of individuals who meet the current criteria for DM are asymptomatic and unaware that they have the disorder.

2. Epidemiologic studies suggest that type 2 DM may be present for up to a decade before diagnosis.

3. As many as 50% of individuals with type 2 DM have one or more diabetes-specific complications at the time of their diagnosis.

4. Treatment of type 2 DM may favorably alter the natural history of DM. The ADA recommends screening all individuals more than 45 years every three years and screening individuals at an earlier age if they are overweight [body mass index (BMI) more than 25 kg/m^2] and have one additional risk factor for diabetes. In contrast to type 2 DM, a long asymptomatic period of hyperglycemia is rare prior to the diagnosis of type 1 DM.[4,9]

Pathogenesis

Type 2 DM

Insulin resistance and abnormal insulin secretion are central to the development of type 2 DM. Although the primary defect is controversial, most studies support the view that insulin resistance precedes an insulin secretory defect but that diabetes develops only when insulin secretion becomes inadequate.

Pathophysiology

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. 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. IGT, 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 may ensue.[4]

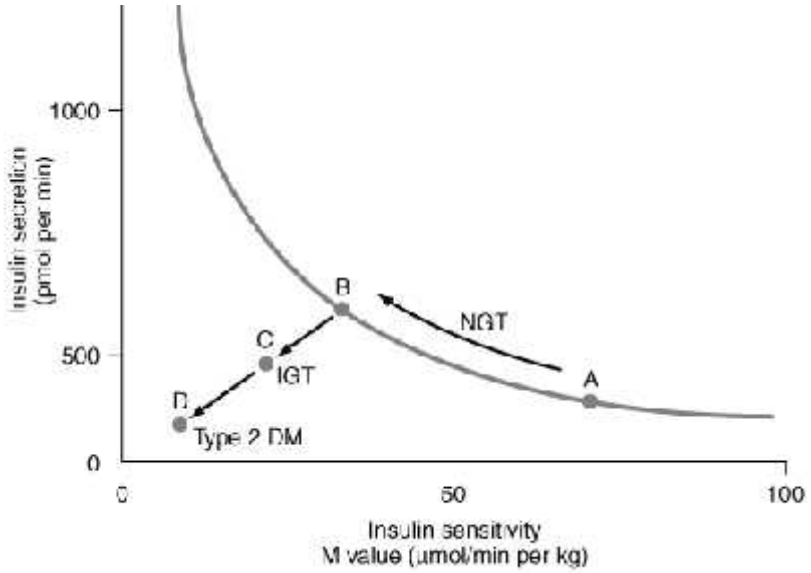


Figure 3. Metabolic changes during the development of type 2 diabetes mellitus[4]

Complications of diabetes mellitus[9,18]

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.[9,18]

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.[18]

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.[20]

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.[28,29]

Glycemic Control and Complications

The Diabetes Control and Complications Trial (DCCT) provided definitive proof that reduction in chronic hyperglycemia can prevent many of the early complications of type 1 DM. This large multicenter clinical trial randomized over 1400 individuals with type 1 DM to either intensive or conventional diabetes management, and prospectively evaluated the development of retinopathy, nephropathy, and neuropathy. Individuals in the intensive diabetes management group received multiple administrations of insulin each day along with extensive educational, psychological, and medical support.[30]

Individuals in the conventional diabetes management group received twice-daily insulin injections and quarterly nutritional, educational, and clinical evaluation. The goal in the former group was normoglycemia; the goal in the

latter group was prevention of symptoms of diabetes. Individuals in the intensive diabetes management group achieved a substantially lower hemoglobin A1C (7.3%) than individuals in the conventional diabetes management group (9.1%).[30]

The DCCT demonstrated that improvement of glycemic control reduced nonproliferative and proliferative retinopathy (47% reduction), microalbuminuria (39% reduction), clinical nephropathy (54% reduction), and neuropathy (60% reduction). Improved glycemic control also slowed the progression of early diabetic complications. There was a nonsignificant trend in reduction of macrovascular events during the trial (most individuals were young and had a low risk of cardiovascular disease). The results of the DCCT predicted that individuals in the intensive diabetes management group would gain 7.7 additional years of vision, 5.8 additional years free from ESRD, and 5.6 years free from lower extremity amputations. If all complications of DM were combined, individuals in the intensive diabetes management group would experience 15.3 more years of life without significant microvascular or neurologic complications of DM, compared to individuals who received standard therapy. This translates into an additional 5.1 years of life expectancy for individuals in the intensive diabetes management group.[30]

The benefit of the improved glycemic control during the DCCT persisted even after the study concluded and glycemic control worsened. For example, individuals in the intensive diabetes management group for a mean of 6.5 years had a 42–57% reduction in cardiovascular events [nonfatal myocardial infarction (MI), stroke, or death from a cardiovascular event] at a mean follow-up of 17

years, even though their subsequent glycemic control was the same as those in the conventional diabetes management group.[30]

The benefits of an improvement in glycemic control occurred over the entire range of A1C values, suggesting that at any A1C level, an improvement in glycemic control is beneficial. The goal of therapy is to achieve an A1C level as close to normal as possible, without subjecting the patient to excessive risk of hypoglycemia.[30]

The United Kingdom Prospective Diabetes Study (UKPDS) demonstrated that each percentage point reduction in A1C was associated with a 35% reduction in microvascular complications. As in the DCCT, there was a continuous relationship between glycemic control and development of complications. Improved glycemic control did not conclusively reduce (nor worsen) cardiovascular mortality but was associated with improvement with lipoprotein risk profiles, such as reduced triglycerides and increased HDL.[31]

One of the major findings of the UKPDS was that strict blood pressure control significantly reduced both macro- and microvascular complications. In fact, the beneficial effects of blood pressure control were greater than the beneficial effects of glycemic control. Lowering blood pressure to moderate goals (144/82 mmHg) reduced the risk of DM-related death, stroke, microvascular end points, retinopathy, and heart failure (risk reductions between 32 and 56%).[31]

Similar reductions in the risks of retinopathy and nephropathy were also seen in a small trial of lean Japanese individuals with type 2 DM randomized to either intensive glycemic control or standard therapy with insulin (Kumamoto

study). These results demonstrate the effectiveness of improved glycemic control in individuals of different ethnicity and presumably, a different etiology of DM (that is phenotypically different from those in the DCCT and UKPDS).[31]

The findings of the DCCT, UKPDS, and Kumamoto study support the idea that chronic hyperglycemia plays a causative role in the pathogenesis of diabetic microvascular complications. These landmark studies prove the value of metabolic control and emphasize the importance of (1) intensive glycemic control in all forms of DM, and (2) early diagnosis and strict blood pressure control in type 2 DM.[31]

Erectile dysfunction in diabetics

The inability to achieve and maintain an erection sufficient for satisfactory sexual intercourse is a distressing and common symptom, affecting up to one-third of adult men.[32] The prevalence of erectile dysfunction increases with age, and it is common in men with systemic disorders such as hypertension, ischemic heart disease, or diabetes mellitus. Diabetes mellitus may lead to disruption of normal sexual function in both men and women via diabetic induced damage to the nerves and blood vessels essential for normal function of the genital organs. Additionally, the presence of poorly controlled diabetes may increase the morbidity associated with the treatment of erectile dysfunction (ED) in men.

Penile anatomy

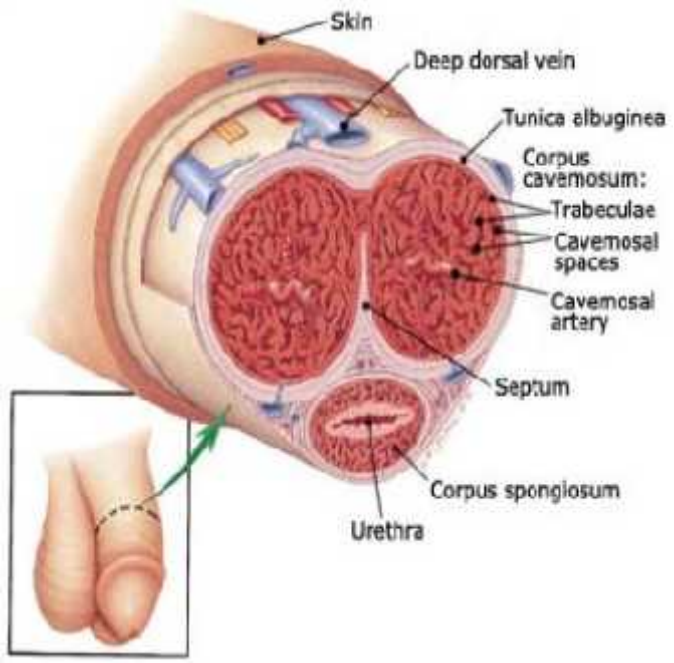


Figure 4. Penile anatomy

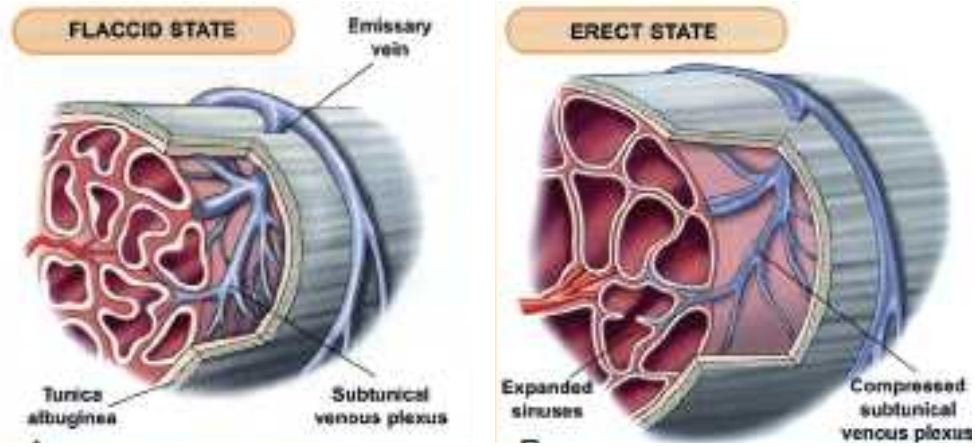


Figure 5. Penile anatomy – Flaccid and erect state

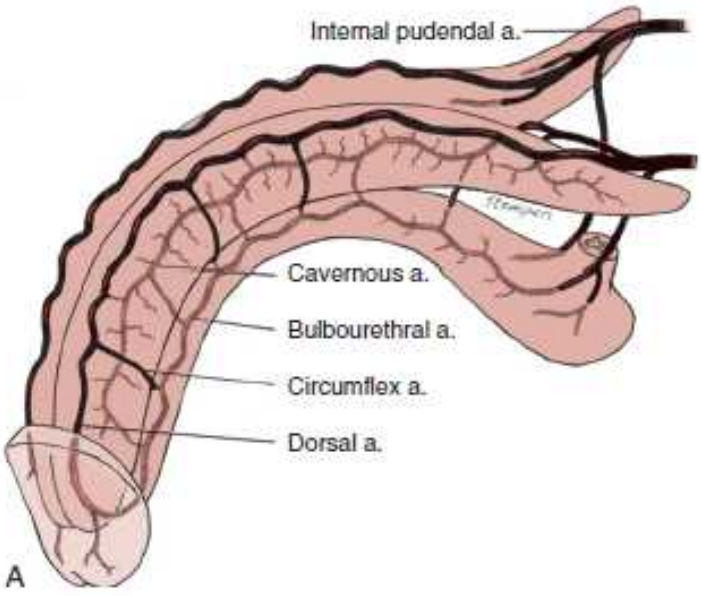


Figure 6. Arterial supply of penis

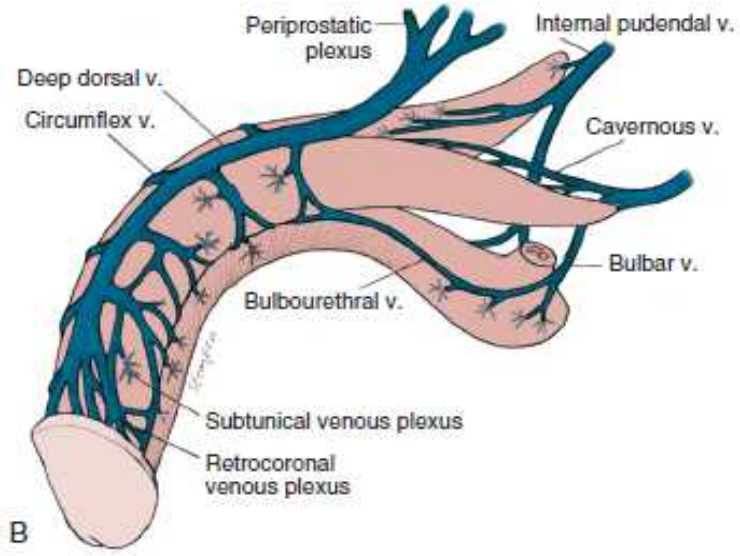


Figure 7. Venous drainage of penis

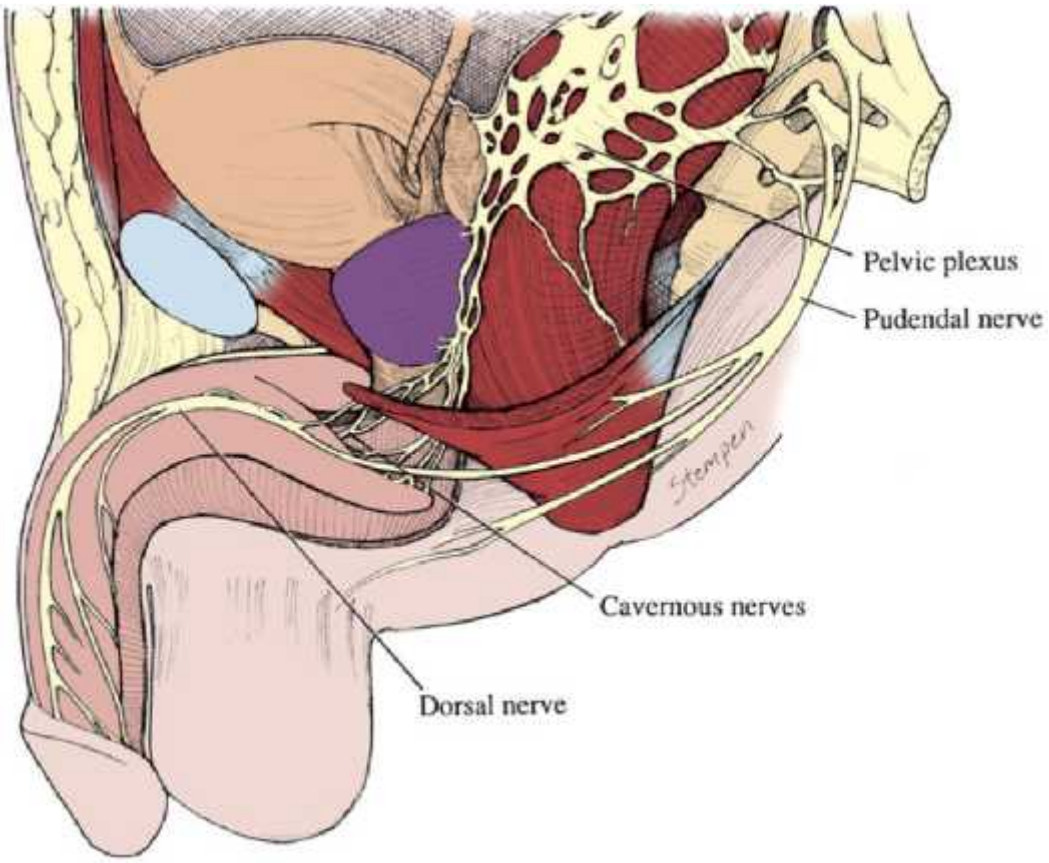


Figure 8. Penile neuroanatomy

Penile components and their functions during penile erection

Corpora cavernosa	Support corpus spngiosum and glans
Tunica albuginea (of corpora cavernosa)	Contain and protects erectile tissue; provides rigidity of the corpora cavernosa; participates in veno-occlusive mechanism;
Smooth muscle	Regulates blood flow into and out of the sinusoids
Ischiocavernosus muscle	Pumps blood distally to hasten erection; provides additional penile rigidity during rigid erection phase
Bulbocavernous muscle	Compresses the bulb to help expel semen
Corpus spongiosum	Pressurizes and constricts the urethral lumen to allow forceful expulsion of semen
Glans	Acts as a cushion to lessen the impact of penis on female organs; provides sensory input to facilitate erection and enhance pleasure; facilitates intromission because of its cone shape

Epidemiology

Cross-sectional and longitudinal epidemiological studies demonstrate that diabetes and several measures of adiposity significantly and independently increase the risk of ED. Multivariate analyses of several population based cohorts show that of all risk factors diabetes imparts the highest risk for ED with an age adjusted relative risk of 1.3 to 3 depending on diabetes type.[33]

In men with diabetes ED begins earlier than in the general population, and is associated with decreased health related quality of life and decreased success of all known ED treatments, including oral pharmacological therapy and penile

implants. An association exists between glycemic control and ED in men with diabetes, in that patients with poor control are at 2 to 5-fold increased risk for ED compared to patients with good control.[34,35]

Other diabetic complications associated with ED in longitudinal and cross-sectional studies were summarized previously, including diabetes duration, limb loss, retinopathy, nephropathy and untreated hypertension.[36]

Body weight and adiposity are significantly associated with ED.[37-39] Convergent data from the Health Professionals Follow-up Study, the National Health and Nutrition Examination Study and MMAS show that compared to men with a BMI of less than 25 kg/m^2 the odds of ED are higher in men with a BMI of 25 to 30 kg/m^2 and even higher in men with a BMI of greater than 30 kg/m^2. The risk is increased 1.5 to 3-fold. Other measures of adiposity, including the waist-to-hip ratio and abdominal circumference, are also independently associated with ED risk.

While low levels of physical activity are often associated with obesity, they impart an additional risk for ED. Independent of BMI, physically active men (greater than 16 MET hours per week of exercise) are at 30% lower risk for ED than sedentary men.[33]

Also, increases in physical activity are independently associated with a lower risk of incident ED. In men without ED at baseline who were followed for 8 years in MMAS the lowest risk of ED was in those who were sedentary at baseline and became physically active (200 kcal or greater per day of activity), whereas the highest risk was in men who were sedentary at each time.[39]

It has become axiomatic that ED shares important risk factors with cardiovascular disease. Results from a longitudinal prostate cancer prevention study showed that incident ED is associated with a higher risk of subsequent cardiovascular events.[40] More recently a similar association was noted in men with T2D and ED.[41] Data from MMAS show that ED predicts subsequent metabolic syndrome in men with BMI less than 25 kg/m^2, a group otherwise considered at low risk for cardiovascular disease (multivariate adjusted RR 2.09; 95% CI 1.09, 4.02).[42] ED also predicts cardiovascular disease events and increased cardiovascular mortality, confirming earlier cited reports.[43]

Hypogonadism may be a link between T2D/metabolic syndrome and ED. Men with ED and T2D have a higher prevalence of hypogonadism, and low testosterone correlates with poor glycemic control and worsening ED. Visceral adiposity and general obesity prevalent in men with T2D and metabolic syndrome can directly impact testosterone. Increased aromatase in adipose tissue can lead to increased androgen conversion from testosterone to estrogens.[33] An association between increased BMI, and waist circumference and hypogonadism has been established in men with T2D.[44-46] Hypogonadism is also associated with other components of metabolic syndrome, such as altered lipid status. Hypogonadal patients with T2D ED have increased triglycerides and lower HDL cholesterol.[47]

A substantial body of literature documents the prevalence of ED in men with diabetes. Unfortunately, the majority of these studies do not distinguish between type 1 and type 2 disease, and, therefore, it is difficult to determine if prevalence rates between the two forms of diabetes differ significantly.

Acknowledging this limitation in the literature, prevalence estimates of ED in cross-sectional studies of diabetic populations range from 20 to 71%. Most of these studies did not control for severity of disease, duration of disease, or control of hyperglycemia.[48]

The wide range of prevalence rates noted among the studies can be attributed to a number of factors. First, prevalence rates are affected by the sensitivity and specificity of methods used to assess ED.[49] In addition, a number of these studies used medical record review to identify patients with ED, as opposed to anonymous patient reports. It has been shown in other disease states that patients tend to underreport ED when questioned directly by their providers.[50] Therefore, the use of validated questionnaires that are either self-administered in an anonymous, neutral setting or administered by an objective third-party interviewer are preferred.

Finally, prevalence rates will be affected by whether the study population is accrued from a single hospital/clinic setting or from a more general population of men with diabetes. For example, Siu et al.[51] studied 500 Chinese diabetic men (of which 97% had type 2 disease) seen at a single medical clinic in Hong Kong during 1999 and found the overall prevalence of ED to be 63.6%. Contrast this to Fedele et al.,[52] who studied 9,756 diabetic men accrued from 178 diabetes centers in Italy. Among the 8,373 men with type 2 diabetes, only 37% reported ED, considerably less than in the Chinese study.

This disparity is due not only to the setting in which the patients were accrued, but also to the manner in which they were questioned, because data in

the Italian study were collected by the medical staff during subjects' visits for medical care, which might have also affected reporting rates.

De Berardis et al.[53] used a fairly generalizable cohort of 1,460 Italian men with type 2 diabetes accrued from 114 outpatient clinics and patient lists of 112 general practitioners. However, unlike the other Italian study, they used self-administered, validated questionnaires to assess the prevalence of ED among diabetic men. They found that 34% reported frequent erectile problems, and 24% reported moderate problems, for an overall prevalence of 58%. Depending on how one wishes to define “clinically significant” ED, this is probably a fairly accurate assessment.

Three longitudinal studies have estimated incidence rates of ED in men with diabetes. Unfortunately, none of these studies specifically examined men with type 2 disease. In a cohort of 278 diabetic men with type 1 or type 2 diabetes potent at study entry, the proportion of patients reporting ED at 5-year follow-up was 28%.[54] A follow-up analysis of the Massachusetts Male Aging Study, a community-based cohort of men between 40 and 70 years of age, found that the incidence of ED in the diabetic men was 51/1,000 population-years.[55] This figure was similar to the 68/1,000 person-years crude incidence rate of ED reported in a study of 1,010 men with diabetes.[52]

However, new studies need to be carried out in well-characterized populations of men with diabetes in order to better determine the incidence of ED and potential effects of interventions to reduce complications.

Complications of diabetes that are associated with an increased risk of ED include peripheral or autonomic neuropathy, nephropathy, and retinopathy. Modifiable risk factors include glycemic control, hypertension, BMI, and cigarette smoking. No intervention studies have used ED as a primary outcome variable.[48]

Although ED is a common complication of diabetes, its effect on quality of life is not well understood. Recent work for the Exploratory Comprehensive Evaluation of Erectile Dysfunction (ExCEED) database demonstrates that in the general population of patients presenting to their urologist, ED negatively affects both general and disease-specific health-related quality of life (HRQOL).[58] While this study provides insight into the detrimental affect of ED on quality of life, the cohort is somewhat selected, in that all of the patients were seen in sexual dysfunction clinics and therefore may have been more likely to be bothered by their condition and to report worse quality of life.

However, population-based studies of ED in prostate cancer survivors also document that ED has a negative effect on general health. Penson, et al.[57] studied HRQOL in 2,306 prostate cancer survivors 2 years after their diagnosis. They noted that men with ED (defined as erections that were insufficient for sexual intercourse) had significantly worse general HRQOL when compared to prostate cancer survivors who were potent. Importantly, this association remained in a multivariate analysis that controlled for 31 other potential confounding variables. Finally, this association was noted in both the physical and mental domains of general quality of life, indicating that ED has a much broader effect on quality of life than one might expect.

While these results in prostate cancer survivors are compelling, one wonders if they are generalizable to diabetic men with ED. Numerous studies indicate not only that the findings in prostate cancer survivors are generalizable to all men with ED, but also that they may underestimate the quality of life effect of ED in diabetic men specifically.

Effect of ED on quality of life in men with diabetes

A follow-up study from the ExCEED database compared men with ED and prostate cancer to men with ED without prostate cancer and found that the prostate cancer survivors had worse erectile function but reported better quality of life than those without prostate cancer.[58] The authors hypothesized that the prostate cancer survivors were able to "rationalize" away their sexual dysfunction with the knowledge that they may have been "cured" of their prostate cancer. Clearly, diabetic men could not use the same rationale.

In another study from ExCEED, Penson et al.[59] compared erectile function and disease-specific quality of life of men with ED and diabetes to those of men with ED without diabetes. They found that those with diabetes reported significantly worse erectile function ($P = 0.004$) and intercourse satisfaction ($P = 0.04$) than those without diabetes. Importantly, the diabetic patients also reported that ED had a significantly worse psychological impact on their overall emotional life than did their nondiabetic counterparts ($P = 0.01$). Interestingly, no differences were noted between the two groups in the psychological impact of ED on the sexual experience.

These data indicate that diabetic men are more likely to present with more severe ED than do men in the general population and that ED may have a greater impact on quality of life in diabetic patients.

While these studies document that ED has a unique effect on quality of life in diabetic men, they do not describe the exact effect of ED on general quality of life in diabetic patients. To date, there is a single study that addresses this important issue.

De Berardis et al.[53] assessed general HRQOL in 1,460 men with type 2 diabetes in Italy. Within the cohort, 615 men reported that they never experienced ED, 346 stated that they occasionally had ED, and 449 stated that they frequently had ED. They then compared general HRQOL among these three groups. In the univariate analysis, they found that degree of ED negatively correlated with general HRQOL scores in all eight domains of the Short Form 36 (SF-36) health survey questionnaire. In the multivariate analysis, ED was not independently associated with physical function, bodily pain, or role limitations due to physical problem scores but was independently associated with general HRQOL outcomes in the domains of general health ($P = 0.004$), role limitations due to emotional problems ($P = 0.001$), vitality ($P = 0.001$), social functioning ($P = 0.01$), and overall mental health ($P = 0.002$). Another study examining the effect of ED on quality of life in hemodialysis patients, more than half of whom had diabetes, also noted an independent, negative effect of ED on the emotional domains of general HRQOL.[60]

Diabetes care providers, while becoming more aware of the high prevalence of ED in men with diabetes, may not appreciate the importance of maintaining erectile function to their patients.[48]

A recent study by Rance et al.[61] underscores the fact that diabetic men, regardless of whether they actually have ED, believe that ED has a major impact on quality of life and that it is as important to treat as many other conditions associated with diabetes. In an effort to determine the relative importance of treatment for ED compared to other diabetic complications, they gave 192 consecutive diabetic men and 51 control patients seen at two hospitals a standardized questionnaire that assessed the relative importance of a number of diabetic complications and the patients' willingness to pay per month to avoid a particular complication.

Not surprisingly, they found that diabetic patients rated kidney disease and blindness as the two most important complications of their condition. Diabetic men with ED ranked ED as the third most important complication of diabetes, followed on average in order by foot ulcers, high blood pressure, high cholesterol, migraine headaches, sleeping disorders, and mild indigestion. Diabetic men without ED found ED slightly less important, ranking it behind foot ulcers and high blood pressure, although all three were grouped fairly close together (mean ranks were 4.59, 4.23, and 4.52, respectively). Interestingly, in men both with and without ED, subjects were willing to pay more per month to avoid ED than all other conditions except blindness and kidney disease (mean values for diabetic patients with ED were £50.5, £88.0, and £66.1, respectively).

In summary, erectile function is important to diabetic men, and when ED is present, it has a significant negative effect on quality of life.

Risk factors for erectile dysfunction in general population

Vascular diseases

Vascular diseases account for nearly 50% of all cases of ED in men older than 50 years. These diseases include atherosclerosis, peripheral vascular disease, myocardial infarction (MI), and arterial hypertension.

Vascular damage may result from radiation therapy to the pelvis and prostate in the treatment of prostate cancer.[62] Both the blood vessels and the nerves to the penis may be affected. Radiation damage to the crura of the penis, which are highly susceptible to radiation damage, can induce ED. Data indicate that 50% of men undergoing radiation therapy lose erectile function within 5 years after completing therapy; fortunately, some respond to one of the PDE5 inhibitors.

Trauma

Trauma to the pelvic blood vessels or nerves can also lead result in ED. Bicycle riding for long periods has been implicated as an etiologic factor; direct compression of the perineum by the bicycle seat may cause vascular and nerve injury. On the other hand, bicycling for less than 3 hours per week may be somewhat protective against ED. Some of the newer bicycle seats have been designed to diminish pressure on the perineum.[63,64]

Abnormal cholesterol levels

The Massachusetts Male Aging Study (MMAS) documented an inverse correlation between ED risk and high-density lipoprotein (HDL) cholesterol levels but did not identify any effect from elevated total cholesterol levels.[65] Another study involving male subjects aged 45-54 years found a correlation with abnormal HDL cholesterol levels but also found a correlation with elevated total cholesterol levels. The MMAS included a preponderance of older men.

Respiratory diseases

Men with sleep disorders commonly experience ED.[66] Heruti et al recommended that in adult male patients, ED should be considered when a sleep disorder—especially sleep apnea syndrome—is suspected, and vice versa.[67]

Endocrine disorders

Hypogonadism that results in low testosterone levels adversely affects libido and erectile function. Hypothyroidism is a very rare cause of ED.[68]

Penile conditions

Peyronie disease may result in fibrosis and curvature of the penis. Men with severe Peyronie disease may have enough scar tissue in the corpora to impede blood flow.[68]

Mental health disorders

Mental health disorders, particularly depression, are likely to affect sexual performance. The MMAS data indicate an odds ratio of 1.82 for men with

depression. Other associated factors, both cognitive and behavioral, may contribute. In addition, ED alone can induce depression.[68]

Cosgrove et al reported a higher rate of sexual dysfunction in veterans with posttraumatic stress disorder (PTSD) than in veterans who did not develop this problem.[69] The domains on the International Index of Erectile Function (IIEF) questionnaire that demonstrated the most change included overall sexual satisfaction and erectile function.[70,71] Men with PTSD should be evaluated and treated if they have sexual dysfunction.

Prostate surgery

Prostate surgery for benign prostatic hyperplasia has been documented to be associated with ED in 10-20% of men. This association is thought to be related to nerve damage from cauterization. Newer procedures (eg, microwave, laser, or radiofrequency ablation) have rarely been associated with ED.[68]

Radical prostatectomy for the treatment of prostate cancer poses a significant risk of ED. A number of factors are associated with the chance of preserving erectile function. If both nerves that course on the lateral edges of the prostate can be saved, the chance of maintaining erectile function is reasonable. The odds depend on the age of the patient. Men younger than 60 years have a 75-80% chance of preserving potency, but men older than 70 years have only a 10-15% chance.[68]

The Cancer of the Prostate Strategic Urologic Research Endeavor (CaPSURE) study, designed to determine whether an individual man's sexual

outcomes after most common treatments for early-stage prostate cancer could be accurately predicted on the basis of baseline characteristics and treatment plans, found that 2 years after treatment, 177 (35%) of 511 men who underwent prostatectomy reported the ability to attain functional erections suitable for intercourse.[72]

In comparison, 37% of men who had received external radiotherapy as their primary therapy reported the ability to attain functional erections suitable for intercourse, along with 43% of men who had received brachytherapy as primary treatment. Pretreatment sexual health-related quality of life score, age, serum prostate=specific antigen (PSA) level, race or ethnicity, body mass index, and intended treatment details were associated with functional erections 2 years after treatment.[72]

After surgery, one of the oral PDE5 inhibitors (sildenafil, vardenafil, or tadalafil) is frequently used to assist in the recovery of erectile function. The benefit of penile rehabilitation therapy is under investigation, but results have been mixed.[73]

Medications

ED is an adverse effect of many commonly prescribed medications. For example, some psychotropic drugs and antihypertensive agents are associated with ED.[68]

Inactivity

Exercise and lifestyle modifications may improve erectile function. Weight loss may help by decreasing inflammation, increasing testosterone, and improving self-esteem. Patients should be educated to increase activity, reduce weight, and stop smoking, as these efforts can improve or restore erectile function in men without comorbidities. Precise glycemic control in diabetics and pharmacologic treatment of hypertension may be important in preventing or reducing sexual dysfunction.[74]

Smoking

Cigarette smoking has been shown to be an independent risk factor. In studies evaluating more than 6000 men, the risk of developing ED increased by a factor of 1.5.[68]

Pathophysiology

Hypogonadism, autonomic neuropathy, and arterial insufficiency are all associated with a higher likelihood of ED in cross-sectional and longitudinal studies of men with diabetes.[75] Experimental investigation of these observations has been accomplished with both in vitro and in vivo models using animals or human tissue.

Low testosterone levels have been observed inconsistently in STZ-induced diabetic and BB rats.[76] Androgen deficiency in rats is associated with downregulation of the neuronal isoforms of nitric oxide synthase, suggesting a trophic effect of testosterone on peripheral erectile tissues. In humans, androgens

play a larger role in sexual interest and motivation (libido) than in erectile capacity itself; penile erection is more resistant to androgen withdrawal than is sexual desire. Relaxation of erectile tissue requires nitric oxide from nonadrenergic-noncholinergic neurons and the endothelium.[48]

Penile tissue from diabetic men with ED demonstrates impaired neurogenic and endothelium-mediated relaxation of smooth muscle, increased accumulation of advanced glycation end products (AGEs), and upregulation arginase, a competitor with nitric oxide synthase for its substrate L-arginine. Normal responses to direct smooth muscle relaxants in most of these studies implies that the impairments are due to decreased synthesis, release, or activity of nitric oxide. The fundamental mechanisms mediating these changes are thought to be the same as for other diabetic complications: increased polyol pathway flux, intracellular accumulation of AGEs, activation of protein kinase C, and increased flux through the hexosamine pathway.[48]

Experimental in vivo studies have implicated central and peripheral neuropathy, impaired neurotransmission, and endothelial dysfunction in the pathogenesis of diabetic ED.[48]

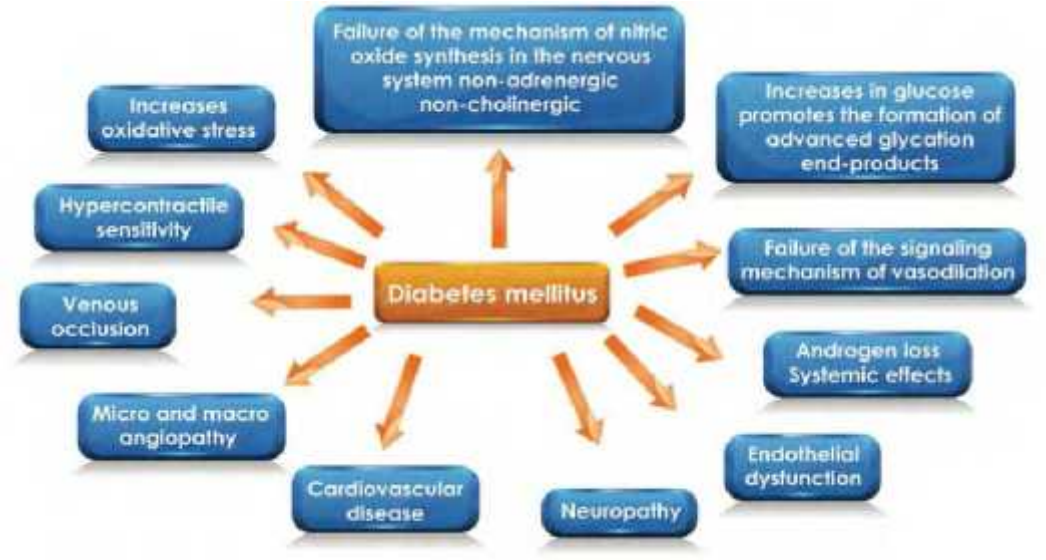


Figure 9. Mechanism of diabetes associated erectile dysfunction[77]

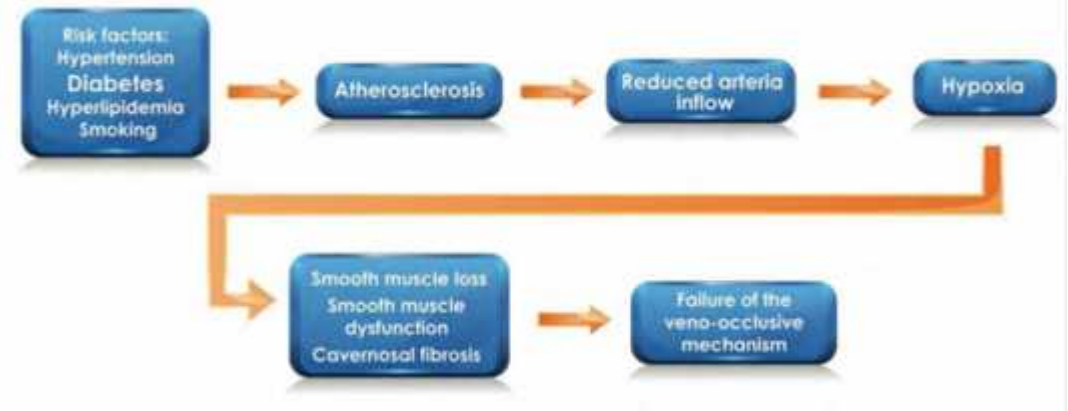


Figure 10. Schematic representation of the relationship between the DM and other risk factors with the development of vascular damages that favour the ED in diabetic patient[77]

Copulatory behavior and penile reflexes are uniformly impaired 4–12 months after the onset of diabetes in the BB rat.[48] McVary et al.[78] found that peripheral neuropathy accounts for only part of the dysfunctional findings, and that spinal sexual reflexes were also severely impaired.

Adequate cavernosal arterial inflow is necessary for penile erection. Arterial morphology, flow, and diameter differ between diabetic and nondiabetic populations with ED. BB and STZ-induced diabetic rats exhibit impairment of endothelium-mediated vascular smooth muscle relaxation, and proposed mechanisms include changes in the expression, activity, or post-translational modification of endothelial NOS.[48]

Experimental hyperglycemia may also affect cavernosal smooth muscle cell contractile responses. In experimental diabetes, penile smooth muscle has augmented force responses to vaconstrictors, possibly mediated by changes in expression of protein kinase C and the RhoA-Rho kinase Ca^{2+}-sensitization pathway. These changes may promote flaccidity and alter the relaxation responses to nitric oxide. End-stage penile dysfunction may occur as a result of diabetes, with progressive loss of normal cavernosal endothelium and smooth muscle cells from the corpus cavernosum. Replacement by fibrotic tissue may lead to complete erectile failure.[48]

Glycemic Control and ED Risk

Although a number of studies show an association between poor glycemic control and an increased risk of ED, so far, no study has been specifically designed to determine whether intensive improvements in glycemic control

would have a beneficial effect on erectile function. The EDIC (Epidemiology of Diabetes Intervention and Complication Study) is a longitudinal cohort followup study for the Diabetes Control and Complication Trial, in which patients with T1D were randomized to conventional or intensive glycemic control. In an ancillary study of urological complications, the Uro-EDIC, the effect of intensive glycemic control on the subsequent risk of ED was assessed.[79] ED was measured using IIEF in a subset of 291 men witha1to 5-year history of diabetes and no microvascular complications (primary prevention), and another group of 280 with a 1 to 15-year history of diabetes with minor complications (secondary intervention). In analyses comparing men initially randomized to intensive vs conventional therapy there was no difference in ED in the primary prevention cohort (OR 1.24; 95% CI 0.68, 2.28). In the secondary intervention cohort ED was significantly less likely in those assigned to intensive control than in those assigned to conventional therapy (OR 0.33; 95% CI 0.18, 0.60).

In men with T2D only limited data have been reported on risk reduction strategies for ED. The 41 men with T2D in a behavioral and pharmacological intervention for cardiac risk reduction experienced significant improvements in hemoglobin A1c, diastolic blood pressure and total cholesterol during 4 weeks of intervention.[80]

Lifestyle Intervention

A number of lines of investigation suggest that weight loss by bariatric surgery or intensive diet and exercise programs improves erectile function in obese men with ED.[33] Lifestyle interventions improve endothelial function and

NO bioavailability, and may have beneficial effects on ED via this mechanism. Weight loss may also improve ED through other mechanisms, including decreased inflammation, increased testosterone, and improved mood and self-esteem.

The strongest evidence supporting the benefit of lifestyle intervention for ED is from the randomized, controlled trial of 110 obese men by Esposito et al in Italy.[81] Men with a mean BMI of 36 kg/m^2 and moderate ED (mean IIEF-EF score 13.7) were randomly assigned to a lifestyle intervention including exercise and weight loss or to an educational control. Notably men with hypertension, diabetes or hyperlipidemia were excluded from study and participants were not seeking help for ED. During 2 years the intervention group lost more weight than controls (15 vs 2 kg) and had greater increases in physical activity (195 vs 84 minutes per week). Erectile function improved in the intervention group (IIEF-EF score 13.9 to 17.0, $p < 0.001$) but did not change in the control group (mean score 13.5 to 13.6, $p = 0.89$). Moreover, 30% of participants in the intervention group recovered normal erectile function (IIEF-EF score 22 or greater) compared to 5% of controls. Improved erectile function correlated significantly with the amount of weight loss and increased activity with each independently explaining about 25% of the variance of change in the IIEF score. Men in the intervention group also had significantly greater improvement in endothelial function (blood pressure and platelet aggregation response to L-arginine), decreases in C-reactive protein and improvement in standard cardiovascular risk

Biomarkers

Numerous markers of systemic inflammation, oxidative stress and endothelial cell injury have been investigated as biomarkers of endothelial dysfunction and many have also been proposed as surrogate markers of ED. In a cross-sectional analysis of a subset of the Health Professionals cohort select biomarkers for endothelial function, thrombosis and dyslipidemia but not for inflammation were associated with the degree of ED.[82]

In an analysis of men with ED and no overt cardiovascular risk factors investigators found that endothelin-1 independently predicted ED.[83]

Preliminary reports from a number of investigators suggest potential targets for further investigation in diabetes associated ED cases, including endothelial microparticles, monocyte activation and various endothelial cell adhesion molecules.[33]

A small randomized, controlled trial in men with T2D and ED showed that short-term continuous sildenafil treatment caused enhanced systemic endothelial function and it remained so after discontinuing sildenafil.[84]

However, there was no associated improvement in long-term erectile function. To our knowledge no large-scale, longitudinal, prospective studies have validated a particular assay for identifying ED or tracking patients at high risk.

Adequate sexual expression is essential to many human relationships and provides a sense of physical, psychological and social well-being. Diabetic men have a more than 3-fold increased prevalence of erectile dysfunction (ED)

compared with nondiabetic men. Erectile function is primarily a vascular phenomenon, triggered by neurologic controls and facilitated by appropriate hormonal and psychological components. Recent advances in the understanding of the physiology of penile vasculature and its role in male sexual performance have influenced the clinical approach to ED. Sexual dysfunction may be one of the important chronic complications in men with diabetes, thus deserving further research.[85]

Chapter 4

Methodology

METHODOLOGY

The present study was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum over a period, from January 2012 to December 2012.

Study design

The study design was hospital based cross-sectional study.

Study period and duration

The present study was conducted for one year from January 2012 to December 2013.

Place

The present study was done in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum a teaching hospital attached to KLE University's Jawaharlal Nehru Medical College, Belgaum.

Source of Data

Patients with Type 1 or 2 diabetes mellitus attending KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum were studied.

Sample size

A total of 208 patients with type 1 or 2 diabetes were included in the study.

Sampling procedure

After reviewing the literature and considering prevalence as 58%[1] the sample size was calculated by the following formula.

$n = 4 p q / d^2$

Where, n = Sample size

p = Prevalence of the disease = 58

q = 100 – p = 42

d = Absolute error = 7.5

Therefore,

$n = 4 \times 58 \times 42 / (7.5)^2$

n = 173.22

Considering 20% loss of follow up or not willing

n = 20 x 173.22 / 100 = 34.64

Hence effective size = 173.22 + 34.64

= 207.86

n ≈ 208

Selection criteria

Inclusion

- Patients greater than 30 years of Age diagnosed as type 1 or 2 diabetes mellitus (Self reported diabetes patients who are on treatment)
- Sexually active, stable heterosexual relation for atleast two years (Stable heterosexual relation was defined as one in which man was engaged and maintains sexual relations, regardless of their marital status**)**.[86]

Exclusion

Patients diagnosed as Type 1 or Type 2 Diabetes Mellitus with;

- Other Endocrine disorders
- Sexually inactive
- Hepatic Disorders
- Renal disorders
- Psychiatric disorders
- Pelvic or spinal trauma / surgery
- Men with unfavourable penile anatomy
- Drugs eg-5α reductase inhibitor, antidepressants, antipsychotics,
- Hypotension
- Significant cardiovascular disease within past 3 months.

Ethical clearance

The study was approved from the Ethical and Research Committee, Jawaharlal Nehru Medical College, Belgaum.

Informed Consent

The patients fulfilling selection criteria were informed in detail about the risks and benefits of the procedure and a written informed consent was obtained before enrollment (Annexure I).

Method of collection of data

Demographic data such as age and sex were recorded. Patients were interviewed for the history including diabetic history (duration and treatment), other comorbid conditions, drugs intake, previous surgery, libido, erectile dysfunction and personal history. Further these patients were subjected to thorough clinical examination for anthropometry, waist hip ratio and vitals and genital examination was performed. These findings were recorded on a predesigned and pretested proforma (Annexure II).

Investigations

The patients were subjected to the following routine investigations

- Complete blood count (CBC)
    - Haemoglobin
    - Packed cell volume
    - Total leukocyte count
    - Red blood cell count
- Urine
    - Routine
    - Microscopy
- Blood sugar levels (fasting or post prandial or random)
- Glycocylated haemoglobin
- Fasting lipid profile
    - Cholesterol
    - Triglycerides

  - Low density lipoprotein
  - High density lipoprotein
- Renal function test
  - Urea
  - Serum creatinine
- Liver function test
  - SGOT
  - SGPT

The following investigations were carried out in specific patients after evaluation.

- Serum testosterone
- Serum prolactin
- Thyroid function
  - T3
  - T4
  - TSH

In patients with history of reduced libido and if testosterone levels were subnormal then only prolactin levels were estimated.

Outcome variables

Erectile dysfunction

National Institutes of Health (NIH) approved questionnaire for International Index of Erectile Function (IIEF), printed in different local

languages – Hindi, Kannada, Marathi, was used to interview each patient to assess for ED.[1]

Risk factors

Based on the history and investigations the patients were evaluated for various risk factors.

Statistical analysis

The data obtained was coded and entered in Microsoft Excel Spreadsheet. The categorical data was expressed as rates, ratios and percentages and comparison was done using chi-square test. Continuous data was expressed as mean ± standard deviation. A 'p' value of less than or equal to 0.05 was considered as statistically significant.

Chapter 5

Results

RESULTS

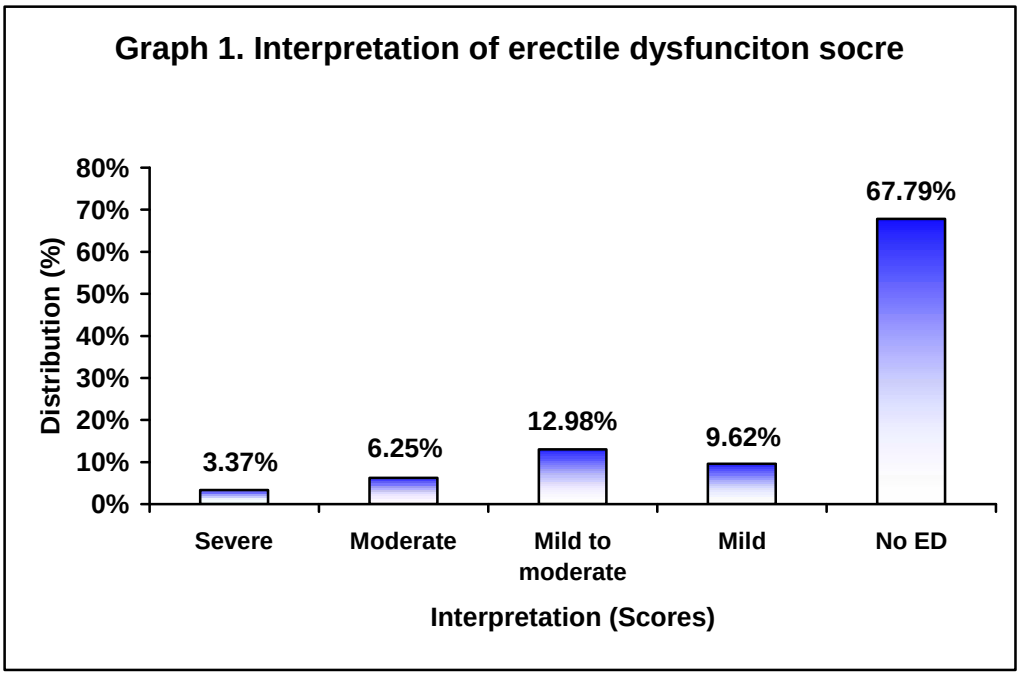
This one year hospital based cross-sectional study was conducted on a total of 208 patients with type 1 or 2 diabetes in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum over a period, from January 2012 to December 2012.

National Institutes of Health (NIH) approved questionnaire for International Index of Erectile Function (IIEF), printed in different local languages – Hindi, Kannada, Marathi and English was used to interview each patient to assess for ED.

The data obtained was coded and entered in Microsoft Excel Spreadsheet. The data was analysed and the observations were interpreted as below.

Table 1. Interpretation of erectile dysfunction score

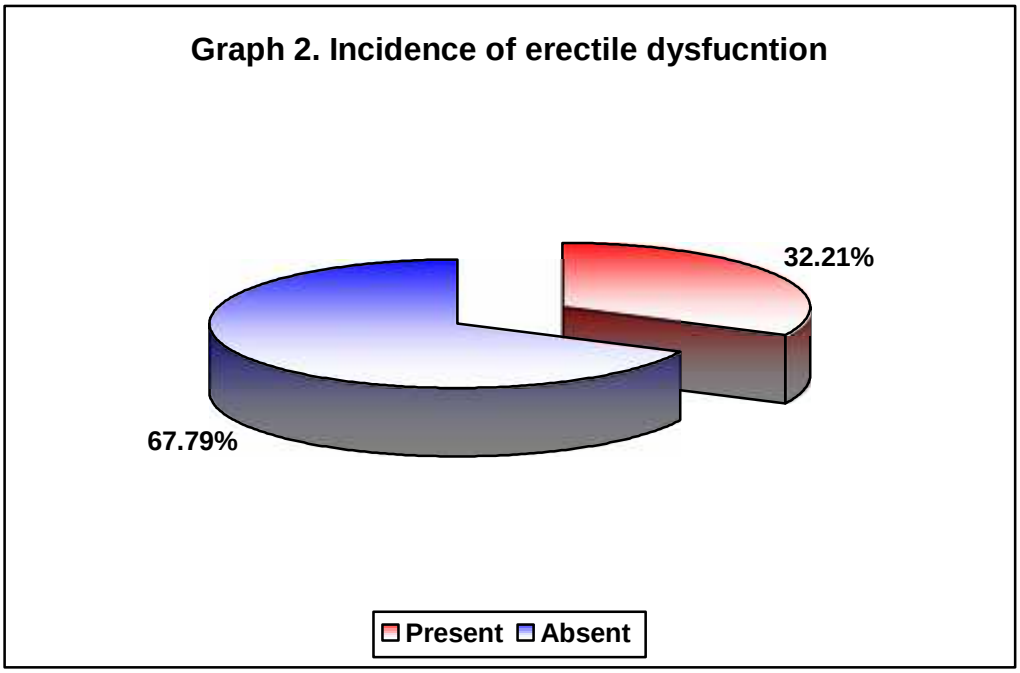
Interpretation (Scores)	Distribution (n=208)	
	Number	**Percentage**
Severe (6 or less)	7	3.37
Moderate (7-12)	13	6.25
Mild to moderate (13-18)	27	12.98
Mild (19-24)	20	9.62
25 (No ED)	141	67.79
Total	**208**	**100.00**



In the present study 12.98% of patients had erectile dysfunction score between 13 to 18 suggestive of mild to moderate erectile dysfunction and 9.62% with 19 to 24 scores suggestive of mild degree. A score of <6 and 7 to 12 was seen in 3.37% and 6.25% of patients. However, 67% of the patients had no erectile dysfunction (score of 25)

Table 2. Incidence of erectile dysfunction

Erectile dysfunction	Distribution (n=208)	
	Number	**Percentage**
Present	67	32.21
Absent	141	67.79
Total	**208**	**100.00**

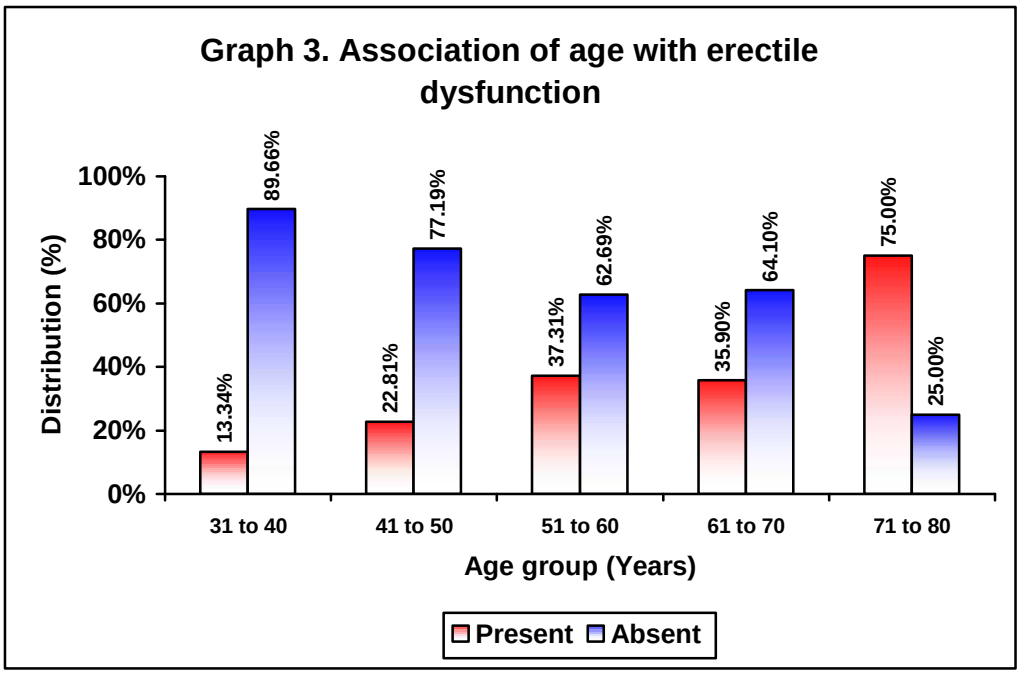


In this study the prevalence of erectile dysfunction was 32.21%.

Table 3. Association of age with erectile dysfunction

Age group (years)	Erectile dysfunction				Total (n=208)	
	Present (n=67)		Absent (n=141)			
	No	%	No	%	No	%
31 to 40	3	10.34	26	89.66	29	100.00
41 to 50	13	22.81	44	77.19	57	100.00
51 to 60	25	37.31	42	62.69	67	100.00
61 to 70	14	35.90	25	64.10	39	100.00
71 to 80	12	75.00	4	25.00	16	100.00
Total	**67**	**32.21**	**141**	**67.79**	**208**	**100.00**

p < 0.001



In the present study the prevalence of erectile dysfunction was significantly high in 71 to 80 years age group (75%) and 61 to 70 years (35.90%) ($p<0.001$).

Table 4. Comparison of mean age in patients with ED and non ED

Erectile dysfunction	Mean age (Years)	
	Mean	**SD**
Present	58.40	10.96
Absent	51.00	11.16

$p < 0.001$

In this study the mean age in patients with erectile dysfunction was significantly high (58.40 ± 10.96 years) compared to those without erectile dysfunction (51.00 ± 11.16 years) ($p<0.001$)

Table 5. Duration of diabetes and erectile dysfunction

Duration (years)	Erectile dysfunction				Total (n=208)	
	Present (n=67)		Absent (n=141)			
	No	%	No	%	No	%
< 1	15	17.44	71	82.56	86	100.00
1 to 5	50	42.02	69	57.98	119	100.00
> 5	2	66.67	1	33.33	3	100.00
Total	**67**	**32.21**	**141**	**67.79**	**208**	**100.00**

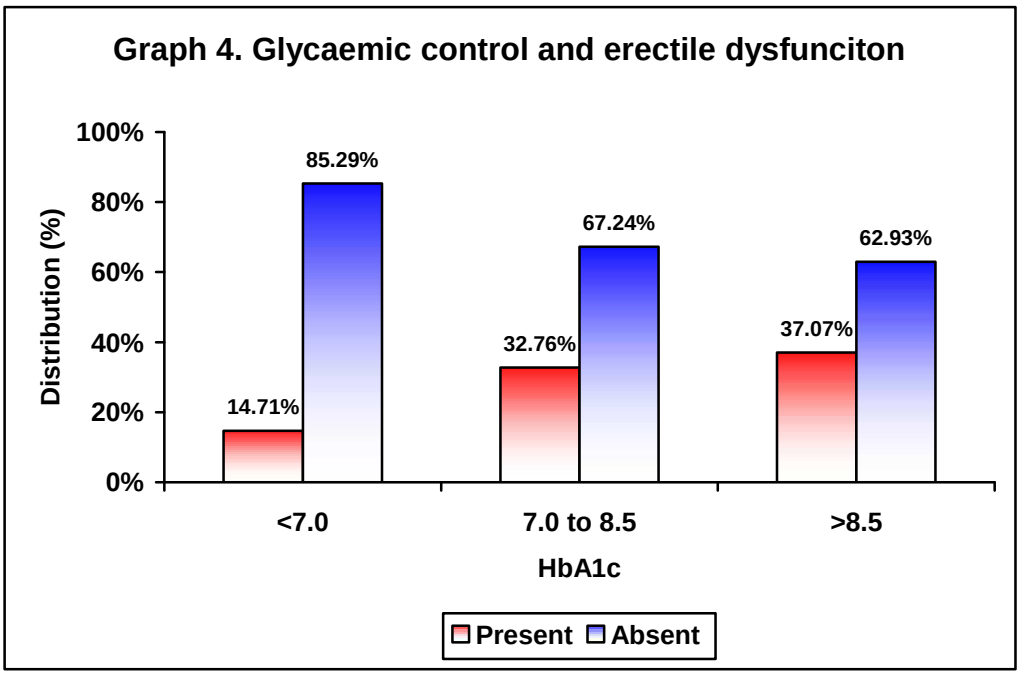
p < 0.001

In this study of the 119 patients with duration of diabetes between one to five years 42.02% had erectile dysfunction and of the 3 patients with duration of more than five years 66.67% had erectile dysfunction ($p<0.001$).

Table 6. Glycaemic control and erectile dysfunction

HbA1c	Erectile dysfunction				Total (n=208)	
	Present (n=67)		Absent (n=141)			
	No	%	No	%	No	%
< 7.0	5	14.71	29	85.29	34	100.00
7.0 to 8.5	19	32.76	39	67.24	58	100.00
> 8.5	43	37.07	73	62.93	116	100.00
Total	**67**	**32.21**	**141**	**67.79**	**208**	**100.00**

p = 0.049



In the present study prevalence of erectile dysfunction was higher in patients with HbA1c levels between 7.0 to 8.5 (32.76%) and > 8.5 (37.07%) compared to those who had HbA1c < 7.0 (14.71%). This difference was statistically significant (p<0.049).

Table 7. Association of smoking with erectile dysfunction

Smoking	Erectile dysfunction				Total (n=208)	
	Present (n=67)		Absent (n=141)			
	No	%	No	%	No	%
Present	21	63.64	12	36.36	33	100.00
Absent	46	26.29	129	73.71	175	100.00
Total	**67**	**32.21**	**141**	**67.79**	**208**	**100.00**

p < 0.001

In the present study prevalence of erectile dysfunction was higher in patients with history of smoking (63.64%). This difference was statistically significant (p<0.001)

Table 8. Association of alcohol intake with erectile dysfunction

Alcohol intake	Erectile dysfunction				Total (n=208)	
	Present (n=67)		Absent (n=141)			
	No	%	No	%	No	%
Present	14	51.85	13	48.15	27	100.00
Absent	53	29.28	128	70.72	181	100.00
Total	**67**	**32.21**	**141**	**67.79**	**208**	**100.00**

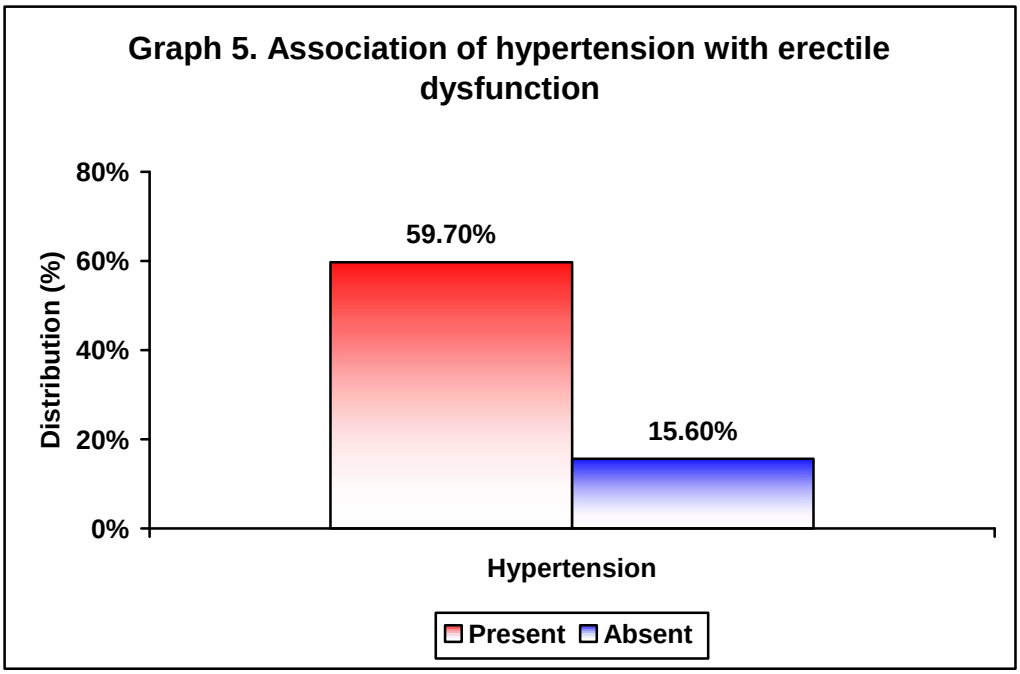
p = 0.019

In the present study 27 patient reported alcohol intake. Among them, 51.85% of patients had erectile dysfunction. This difference was statistically significant (p<0.001)

Table 9. Association of hypertension with erectile dysfunction

Erectile dysfunction	Hypertension	
	Number	**Percentage**
Present	40	59.70
Absent	22	15.60

p < 0.001



In the present study the prevalence of erectile dysfunction was significantly higher in patients with history of hypertension (59.7%) ($p<0.001$)

Table 10. Association of cardiovascular disease with erectile dysfunction

Erectile dysfunction	CVD	
	Number	**Percentage**
Present	22	32.80
Absent	19	13.50

p = 0.011

In this study the prevalence of erectile dysfunction was significantly higher in patients with history of cardiovascular disease (32.80%) (p<0.001)

Table 11. Characteristics of study population with and without erectile dysfunction

Components	Erectile dysfunction				p value
	Present (n=67)		**Absent (n=141)**		
	Mean	**SD**	**Mean**	**SD**	
HbA1c	12.40	14.74	9.80	9.53	0.133
Cholesterol	172.10	44.30	162.80	36.40	0.113
Triglycerieds	160.10	63.88	130.40	47.75	**0.001**
BMI	25.4	4.08	24.4	3.78	0.081
WHR	1.12	1.04	1.01	0.1	0.414
Plasma Glucose	171.8	64.97	165.8	66.47	0.545
Hb	12.09	1.32	13.10	8.18	0.318

In this study, the qualitative analysis of risk factors showed significantly raised mean triglycerides in patients with erectile dysfunction (p=0.001).

Chapter 6

Discussion

DISCUSSION

Erectile dysfunction is an extremely distressing and demoralizing disease of male as feeling of lack of man hood sends a shiver of fear down the spine in most of men.[87]

Penile erection is a neurovascular event that depends on the relaxation of smooth muscle in the erectile bodies. The relaxation of smooth muscle involves the release of nitric oxide and other mediators, which stimulate the production of intracellular cyclic nucleotides that cause relaxation. With the relaxation of corporal smooth muscle, rapid arterial filling begins, resulting in engorgement of the sinusoids in the cavernosa and veno-occlusion due to the compression of the sub tunical venules against the tunica albuginea.[87]

One of the independent risk factor for erectile dysfunction is diabetes mellitus. People with diabetes have an increased risk of sexual dysfunction. Men with diabetes are three times more likely to experience erectile dysfunction than those without diabetes, according to the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK).[2]

In addition, men with diabetes and erectile dysfunction may experience the problem 10 to 15 years earlier in life than non diabetics. It is not uncommon for a man seeking treatment for erectile dysfunction to learn that he has diabetes and that it may be the cause of his sexual difficulties.[2]

Researchers have found that obesity, diabetes, elevated blood pressure or unhealthy amounts of blood fats (cholesterol and triglycerides) are associated

with hypogonadism. Recent research suggests that hypogonadism may also be associated with insulin resistance, the progression of type 2 diabetes and possibly heart disease. This very important subject is usually overlooked in diabetic clinics.[2]

However, the prevalence of ED varied based on the population studied, the definition and methods used for the assessment. So far, very few studies have been carried out on the incidence and prevalence of this condition in a diabetes mellitus patients. Also, epidemiological data on the prevalence of ED among DM patients in India is scarce. Hence the present study undertaken to determine the prevalence and factors associated with ED in DM.

The present hospital based cross-sectional study was conducted over a period of one year from January 2012 to December 2012 on a total of 208 patients with type 1 or 2 diabetes attending Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. National Institutes of Health (NIH) approved questionnaire for International Index of Erectile Function (IIEF)[1] was used to interview each patient to assess for ED.

In the present study the prevalence of erectile dysfunction was 32.21% (Score < 25) and 67.79% of the patients had normal erectile dysfunction (score of > 25). Among those with erectile dysfunction, 12.98% of patients mild to moderate erectile dysfunction, 9.62% had mild, 6.25% had moderate and 3.37% had severe erectile dysfunction. ED affects approximately 1 in 10 men worldwide (Benet & Melman, 1995). The prevalence of ED has been reported in various

studies as ranging from 20% to greater than 70%.[16,88] A similar study[16] from Kochi reported 58% (85/147) subjects, who reported to have history of ED. Another study[89] from Rajasthan on 50 type-2 diabetic patients using international index of erectile dysfunction (IIEF-5) reported the prevalence of erectile dysfunction as 78% (mild, moderate and severe ED was present in 6, 36 and 36%, respectively). Schiavi et al,[90] studied 40 diabetic men, free from other illness or drugs that could affect sexual capacity and 40 age-matched healthy control subjects. ED was present in 77% of patients. Sundaram et al,[91] reported that in diabetic patients, the prevalence of ED was 66%. Ledda et al,[92] reported that ED was very common among diabetic patients. They had ED at an earlier age and prevalence was 75%. Sassayam et al,[93] studied 6112 Japanese male patients from 447 outpatient clinics and found that 81% had some degree of ED. Kloner[94] observed that the prevalence of ED in diabetic patients was about 75%. Sasaki et al,[95] reported prevalence of 90% in 1118 male diabetic patients. Prevalence rate was double than that of nondiabetic individuals.

The wide range of prevalence rates noted among the studies can be attributed to a number of factors. Prevalence rates are affected by the sensitivity and specificity of methods used to assess ED. A number of these studies used medical record review to identify patients with ED, as opposed to anonymous patient reports. It has been shown in other disease states that patients tend to underreport ED when questioned directly by their providers. Therefore, the use of validated questionnaires that are either self-administered in an anonymous, neutral setting or administered by an objective third-party interviewer are preferred.[48]

The risk of erectile dysfunction increases with age. As the population continues to grow and age, the prevalence is expected to continue to increase, with an estimate that there will be 322 million men worldwide with ED by the year 2025.[96] Al though awareness of erectile dysfunction has in creased with the advent of oral therapies, a significant number of men remain undiagnosed and untreated. In the present study the prevalence of erectile dysfunction was significantly high in 71 to 80 years age group (75%) and 61 to 70 years (35.90%) ($p<0.001$). The mean age in patients with erectile dysfunction was also significantly high (58.40 ± 10.96 years vs 51.00 ± 11.16 years; $p<0.001$) compared to those without erectile dysfunction. Epidemiologic studies have demonstrated the prevalence of 35% of men aged 40 to 70 years suffer from moderate or severe ED, and an additional 25% have milder forms of ED.[94] A similar study from Kochi[16] reported 58% (85/147) subjects, who reported to have history of ED, were between 30 – 70 years with a mean 52 + 8 years and 89% (76/85) of them were above the age of 45 years. Study also found that, as the age goes over 45 years there is significant increase in the prevalence of ED. A similar study[89] from Rajasthan reported that, prevalence of erectile dysfunction increased with the increase in age. Prevalence increased from 20% in age group of <40 to 100% in age group of >60 years. Similar trends have been shown in earlier studies also.[34] The findings of the present study were consistent with these studies. Most of the earlier studies had also reported significant correlation between ED and age. The effect of age on prevalence and severity of disease might be due to age-related changes occurring in body and also various other complications that may coexist in diabetic patients, but ultimately the accelerated

atherosclerosis is the common denominator for increased prevalence of ED and cardiovascular disease in ageing population.[89]

In this study of the 119 patients with duration of diabetes between one to five years 42.02% had erectile dysfunction and of the 3 patients with duration of more than five years 66.67% had erectile dysfunction ($p<0.001$). These findings suggest that, the erectile dysfunction increased with the duration of diabetes. However, in a study[16] from Kochi, India the duration of diabetes did not show any significant increase in the incidence of ED.

In this study the prevalence of erectile dysfunction was higher in patients with HbA1c levels between 7.0 to 8.5 (32.76%) and > 8.5 (37.07%) compared to those who had HbA1c < 7.0 (14.71%). This difference was statistically significant ($p<0.049$). Similar findings were reported in a study[16] from Kochi, India which stated that, in those patients with ED, the overall glycemic control was significantly worse than in the non-ED group, which reveals the relationship of poor glycemic control to ED. Becon CG et al[38] reported that, for men over age 50 years, increasing duration of diabetes was positively associated with increased risk of ED relative to nondiabetic subjects. This association persisted despite the higher prevalence of other comorbid conditions.[38]

Historically, ED was believed to primarily have a psychogenic origin; however, the majority of individuals are currently identified to have organic ED due to an underlying physiologic cause. The most common cause of ED is atherosclerosis and is associated with disease states such as diabetes mellitus, hypertension, smoking, and dyslipidemia. These risk factors cause oxidative

stress and damage to the endothelial cells.[98] In the present study the prevalence of erectile dysfunction was significantly higher in patients with history of hypertension (59.7%; p<0.001), cerebrovascular disease (32.80%; p<0.001), positive family history for erectile dysfunction (21.05%; p=0.025) and prostate surgery (83.58%; p=0.006). Giuliano et al[99] in 1186 men with both hypertension and diabetes reported, ED in 77%. In contrast, a study[16] from Kochi reported that, comorbidities like hypertension, PVD and dyslipidemia were not statistically significant in those with ED and those without ED.

In the present study prevalence of erectile dysfunction was higher in patients with history of smoking (63.64%; p<0.001) and alcohol consumption (51.85%; p<0.001) Fedele and associates[34] have also shown that smoking habit of the patient acts as a risk factor for ED. In contrast, a study[16] reported that, alcohol and smoking habits in diabetic patients had not shown significant contribution to ED.

Hyperlipidemia characterized by high cholesterol and/or low HDL-cholesterol levels, hypertension and obesity are conditions that often coexist with diabetes; all may be independent risk factors for ED among diabetic men.[100] In this study 23.40% of patients with abnormal liver function had erectile dysfunction (p=0.142). The qualitative analysis showed significantly raised mean triglycerides in patients with erectile dysfunction (p=0.001). It is noteworthy that in two population-based observational studies carried out in China and the United States[101,102] on type 2 diabetic men, there was no significant association between circulating lipid levels (total cholesterol, LDLD-cholesterol, HDL-cholesterol and triglyceride) and the risk of ED, pointing to the apparent conclusion that

dyslipidemia does not have a significant role in the risk of diabetic ED. However, in this study the association was limited to only hypertriglyceraedimia which could be attributed to the smaller sample size.

This study had certain limitation that is, the study included all the age groups. Further studies with large sample size and age standardized selection criteria would determine the exact prevalence and associated risk factors.

Chapter 7

Conclusion

CONCLUSION

The prevalence of erectile dysfunction in patients with diabetes mellitus was 32.21%. Among these, 12.98% of patients mild to moderate erectile dysfunction, 9.62% had mild, 6.25% had moderate and 3.37% had severe erectile dysfunction.

The erectile dysfunction in patients with diabetes mellitus was significantly prevalent with age, diabetic duration of diabetes, history of hypertension, cardiovascular disease, glycemic control and hypertriglyceridemia.

Chapter 8

Summary

SUMMARY

One of the independent risk factor for erectile dysfunction is diabetes mellitus. The present study planned to determine the prevalence and factors associated with ED in DM.

The present hospital based cross-sectional study was conducted over a period of one year from January 2012 to December 2012 on a total of 208 patients with type 1 or 2 diabetes attending Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. National Institutes of Health (NIH) approved questionnaire for International Index of Erectile Function (IIEF)[1] was used to interview each patient to assess for ED.

In the present study 12.98% of patients had erectile dysfunction score between 13 to 18 suggestive of mild to moderate erectile dysfunction and 9.62% with 19 to 24 scores suggestive of mild degree. The prevalence of erectile dysfunction was 32.21%. The prevalence of erectile dysfunction was significantly high in 71 to 80 years age group (75%) and 61 to 70 years (35.90%) ($p<0.001$). The mean age in patients with erectile dysfunction was significantly high (58.40 ± 10.96 years) compared to those without erectile dysfunction (51.00 ± 11.16 years) ($p<0.001$) Of the 119 patients with duration of diabetes between one to five years 42.02% had erectile dysfunction and of the 3 patients with duration of more than five years 66.67% had erectile dysfunction ($p<0.001$). Prevalence of erectile dysfunction was higher in patients with HbA1c levels between 7.0 to 8.5 (32.76%) and > 8.5 (37.07%) compared to those who had HbA1c < 7.0 (14.71%).

This difference was statistically significant ($p<0.049$). Prevalence of erectile dysfunction was higher in patients with history of smoking (63.64%) ($p<0.001$) and alcohol intake (51.85%) ($p<0.001$). The prevalence of erectile dysfunction was also significantly high in patients with history of hypertension (59.7%) ($p<0.001$).

Chapter 9

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Annexures

Annexure I

ANNEXURE I – CONSENT FORM

Title Of Research Study

"ERECTILE DYSFUNCTION IN DIABETES MELLITUS – A CROSS SECTIONAL STUDY"

Principal Investigator

Dr. ** ******
Associate Professor
Department Of Surgery
J.N.Medical College, Belgaum.

Co- Investigators:-

Dr. ****
Associate Professor,
Department Of Urology,
J. N. Medical College, Belgaum.

Dr. *****
Post Graduate,
Department Of Surgery,
J. N. Medical College, Belgaum.

I, Dr. ******, post graduate student of Dept. of Surgery, JNMC, KLE University, Belgaum, am conducting a study for prevalence of erectile dysfunction in diabetic patients in KLE Hospital under the guidance of Dr. **** *******, Professor, Department of Surgery, Dr. **** ***** Associate Professor, Department of Urology, JNMC, KLE University, Belgaum.

Participations of the respondents will be voluntary and informed consent will be obtained. All the patients will be subjected to detailed history and complete physical examination by the investigator and all findings will be documented in pre-designed proforma designed with the help of a diabetologist and a urologist. All patients who do not fulfill inclusion criteria will be excluded in the study. A pretested questionnaire -International Index of Erectile Function (IIEF) will be used to interview each patient with total guarantee of confidentiality and results will be analysed.

You will not be eligible for any kind of monetary benefits or free services by virtue of your participation in the study. You will be benefitted by the health education given during the study regarding appropriate control of diabetes mellitus and erectile dysfunction.

Methods applied to do the study are safe. No risk is involved in the study. By signing this consent form you are not waiving any of your legal rights. The cost of the study will be borne by the researcher. You will not have any costs attached to your participation.

The results of the study may be published for scientific purposes. However your identity will not be revealed. Every effort will be made to protect the confidentiality of the information you provide. All information collected will be coded so that no one other than the investigator will know your identity. You can withdraw from the study at any time if you wish to do so.

Therefore I kindly request you to participate in this research project.

If you have any queries regarding the study, you can contact –

Dr. ******,
Post Graduate,
Department Of Surgery
J.N.Medical College, Belgaum.
Ph. No ***** *******.

Dr. **********
Associate Professor,
Department Of Surgery,
J.N.Medical College, Belgaum
Ph. No ***** *******

Dr. ***** ******
Associate Professor,
Department Of Urology,
J. N. Medical College, Belgaum.
Ph. No ***** ******.

If you have any questions about rights as a research participant you can contact –

Dr ***** ******,
Chairman, Institutional Ethics Committee,
JNMC, Belgaum
Ph.no - ***** *****

CONSENT STATEMENT

I, the undersigned, have been explained in my own vernacular language about the study –

"ERECTILE DYSFUNCTION IN PATIENTS OF DIABETES MELLITUS"

and my participation in the study is voluntary. If I want, I can withdraw at any time. Also I have been given enough time to clear my doubts about the study and my rights as a study patient.

Participants name : _____

Signature/ Left thumb impression of study participant :

Witness name : _____

Signature / Left thumb impression of witness :

Investigator's name: _____

Signature :

Place : _____ **Date : _____**

GUIDE – DR . ** *****

CO GUIDE – DR. *** ******

Annexures

Annexure II

ANNEXURE II – PROFORMA

RECORD NO.	

FOR PURPOSE OF STUDY

ERECTILE DYSFUNCTION IN PATIENTS OF DIABETES MELLITUS

CASE SHEET – DEPARTMENT OF SURGERY & UROLOGY

I) PATIENT IDENTIFICATION DATA :

NAME (in full) :_____

IP/OPD NO.:_____AGE :_____SEX :_____

MARITAL STATUS :_____OCCUPATION :_____

ADDRESS :_____

CONTACT No._____

EDUCATION STATUS -

☐ **BASIC** ☐ **TECHNICAL** ☐ **SECONDARY** ☐ **TERTIARY**

I) PRESENTING COMPLAINS –

II)

III) CARDIOVASCULAR DISEASE –

☐ **ABSENT** ☐ **PRESENT**

(SPECIFY-……)

IV) NEUROLOGICAL/PSYCHIATRIC DISEASE –

☐ **ABSENT** ☐ **PRESENT**

(SPECIFY-……)

V) OTHER ENDOCRINE DISEASE –

☐ **ABSENT** ☐ **PRESENT**

(SPECIFY-……)

VI) DURATION OF D.M. –

☐ **< I YEAR** ☐ **1-5 YEARS** ☐ **>5 YEARS**

VII) FAMILY HISTORY OF D.M

☐ **1°RELATIVES -** FATHER,MOTHER, CHILDREN ,BROTHER,SISTER

☐ **2°RELATIVE –** AUNT,UNCLE,NEPHEWS,NIECES,GRANDPARENTS

☐ **ABSENT**

VIII) DIABETIC TREATMENT :

☐ **NO** ☐ **DIET CONTROL**

☐ **DRUGS** ☐ **INSULIN**

DOSE- …….. **DOSE- ……**

FREQUENCY - …….. **FREQUENCY - ……..**

IX) RISK FACTORS

HISTORY OF SMOKING	☐ **NO** ☐ **YES** **Duration – ……** **No of Cigarettes - ……** **Other types of tobaccos - ……**

HISTORY OF ALCOHOL	☐ **NO** ☐ **YES** **Duration – ……** **Frequency - ……**
KNOWN CASE OF HPERTENSION	☐ **NO** ☐ **YES** **Duration – ……** **h/o treatment - ……**

X) <u>HOSPITAL ADMISSION/ SURGERY/TRAUMA :</u>

☐ **NO** ☐ **PELVIC/SPINAL INJURY**

☐ **PROSTATE SURGERY** ☐ **OTHERS -……**

XI) <u>HISTORY OF REDUCED LIBIDO -</u>

☐ **NO** ☐ **YES**

XII) <u>DO YOU THINK YOU HAVE ERECTILE DYSFUNCTION –</u>

☐ **NO** ☐ **YES** ☐ **NOT SURE**

XIII) <u>HISTORY OF OTHER DRUG INTAKE :</u>

A. 5α reductase inhibitor

☐ **YES** ☐ **NO**

(SPECIFY-……)

B. Antipsychotic/ Antidepressant drugs

☐ **YES** ☐ **NO**

(SPECIFY-……)

PHYSICAL EXAMINATION

GENERAL

- **BODY MASS INDEX (BMI) -**

$$BMI = \frac{\text{WEIGHT (kg)}}{\text{HEIGHT}^2\ (m)^2} =$$

☐ **< 16 (SEVERLY UNDER WEIGHT)**

☐ **16 - 18.5 (UNDER WEIGHT)**

☐ **18.5 - 25 (NORMAL)**

☐ **25 - 30 (OVER WEIGHT)**

☐ **30 -35 (OBESE CLASS I)**

☐ **35 - 40 (OBESE CLASS II)**

☐ **> 40 (OBESE CLASS III)**

- **PULSE -**

☐ **< 60/MIN** ☐ **60-80 /MIN** ☐ **>80/MIN**

- **RESPIRATORY RATE -**

☐ **< 14/MIN** ☐ **14-18/MIN** ☐ **>18/MIN**

- **BLOOD PRESSURE (SUPINE) –**

	CATEGORY	SYSTOLIC (MM HG)	DIASTOLIC (MM HG)
	OPTIMAL	< 120	<80
	NORMAL	<130	<85
	HIGH NORMAL	130 -139	85-89
	STAGE1 (MILD HYPERTENSION)	140 -159	90 -99
	STAGE2 (MODERATE HYPERTENSION)	160 -179	100-109
	STAGE 3 (SEVERE HYPERTENSION)	>=180 or	>= 110

- **WAIST : HIP RATIO -**

WAIST : HIP RATIO (WHR) = WAIST MEASURE / HIP MEASURE

☐ **0.95 OR BELOW (LOW HEALTH RISK)**

☐ **0.96 TO 1.0 (MODERATE HEALTH RISK)**

☐ **1.0 OR GREATER (HIGH HEALTH RISK)**

PERIPHERAL EXAMINATION :

I) LOWER LIMB EXAMINATION :

	LEFT	RIGHT
DORSAL PEDIS ARTERY		
POST. TIBIAL ARTERY		
ANT. TIBIAL ARTERY		
POPLITEAL ARTERY		
FEMORAL ARTERY		

II) PERIPHERAL NEUROPATHY:

VIBRATORY SENSATION	
TOUCH	
PROPRIOCEPTON	
ANAL TONE	
PIN PRICK SENSATION	
TESTICULAR SENSATION	

III) GENITALS EXAMINATION –

PENILE : _____

TESTIS : _____

PHYMOSIS :
_____

IV) SECONDARY SEXUAL CHARACTERSTICS :

INVESTIGATIONS:

I) PLASMA GLUCOSE –

FASTING BLOOD SUGAR OR	
RANDOM BLOOD SUGAR OR	
POST PRANDIAL SUGAR	

II) GLYCOSYLATED Hb (HbA_1C) -……

III) FASTING LIPID PROFILE

CHOLESTEROL	
TRIGLYCERIDE	
LDL	
HDL	

IV) RENAL FUNCTION TEST

UREA	
CREATININE	

V) LIVER FUNCTION TEST

SGOT	
SGPT	

VI) URINE ANALYSIS (Routine)

SPECIFIC GAVITY	**GLUCOSE**
COLOUR	**PROTEINS**
PH	

URINE KETONE BODIES - ……

VII) CBC -

Hb -	**PCV -**
TLC -	**RBC -**

VIII) SERUM TESTOSTERONE–

IX) SERUM PROLACTIN –

X) THYROID FUNCTION TEST -

T3	
T4	
TSH	

RECORD NO.	

FOR PURPOSE OF STUDY ONLY

THE INTERNATIONAL INDEX OF ERECTILE FUNCTION QUESTIONNAIRE (IIEF -5)

(TICK YOUR ANSWERS IN BOX AND ADD UP THE TOTAL).

1. **Over the last month, how often were you able to get an erection during sexual activity?**

0 No sexual activity ☐

5 Almost always or always ☐

4 Most times (much more than half the time) ☐

3 Sometimes (about half the time) ☐

2 A few times (much less than half the time) ☐

1 Almost never or never ☐

2. Over the last month, when you had erections with sexual stimulation, how often were your erections hard enough for penetration?

0 No sexual activity ☐

5 Almost always or always ☐

4 Most times (much more than half the time) ☐

3 Sometimes (about half the time) ☐

2 A few times (much less than half the time) ☐

1 Almost never or never ☐

Q3. Over the last month, when you attempted intercourse, how often were you able to penetrate your partner?

0 No sexual activity ☐

5 Almost always or always ☐

4 Most times (much more than half the time) ☐

3 Sometimes (about half the time) ☐

2 A few times (much less than half the time) ☐

1 Almost never or never ☐

Q4. Over the last month, during sexual intercourse, how often were you able to maintain your erection after you had pentrated your partner?

0 No sexual activity ☐

5 Almost always or always ☐

4 Most times (much more than half the time) ☐

3 Sometimes (about half the time) ☐

2 A few times (much less than half the time) ☐

1 Almost never or never ☐

Q5. Over the last month, during sexual intercourse, how difficult was it to maintain your erection to completion of intercourse?

0 No sexual activity ☐

1 Extremely difficult ☐

2 Very difficult ☐

3 Difficult ☐

4 Slightly difficult ☐

☐

5 Not difficult

NAME (IN FULL) :_____

IP/OPD NO.:_____AGE :_____SEX :_____

	SCORE RANGE	MAXIMUM SCORE	YOUR SCORE
QUESTION 1	**0 - 5**	**5**	
QUESTION 2	**0 - 5**	**5**	
QUESTION 3	**0 - 5**	**5**	
QUESTION 4	**0 - 5**	**5**	
QUESTION 5	**0 - 5**	**5**	

TOTAL SCORE -

[CLINICAL INTERPRETETION]

ERECTILE FUNCTION TOTAL SCORES CAN BE INTERPRETED AS FOLLOWS:

0-6 Severe dysfunction

7-12 Moderate dysfunction

13-18 Mild to moderate dysfunction

19-24 Mild dysfunction

25 No dysfunction

ANNEXURE III - MASTER CHART

Serial Number	In patient number	Demo-graphy			DM history		History of other illnesses					Surgical history				Drug history					Risk factors		General physical examination						Genital examination				Investigations															Questionnaire						
		Age (Years)	Marital status	Educational status	Duration (Years)	Treatment	Cardiovascular disease	Neurological/psychiatric	Other endocrine disease	Hypertension (Years)	Family history of DM	History	Pelvic/spinal	Prostate	Other	5α reductase inhibitor	Antipsychotic/ Antidepressant drugs	History of reduced libido	Erectile dysfunction	Circumcision	Smoking (Years)	Alcohol consumption (Years)	Height (Cms)	Weight (Kgs)	Body mass index (Kg/m2)	Waist hip Ratio	Blood pressure	Peripheral pulses	Penile	Testis	Phimosis	Secondary sexual characteristics	Plasma glucose (FBS/RBS/PPBS)	HbA1c	Cholesterol	Triglycerides	Low density lopoprotein	High density lipoprotein	Urea	Creatinine	SGOT	SGPT	Urine routine	Urine microscopy	Haemoglobin	Thyroid function T3	Thyroid function T4	Thyroid function TSH	Q1	Q2	Q3	Q4	Q5	Total score
1	2E+06	45	Y	1	1	2	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	165	95.0	34.9	1.00	4	+	-	-	-	-	140	11.5	225	207	181	35	22.00	1.00	38	69	-	-	12.5	-	-	-	0	0	0	0	0	0
2	1E+06	44	Y	1	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	170	75.0	26.0	9.50	2	+	-	-	-	-	135	9.3	182	251	93	39	23.00	0.90	35	60	-	-	11.2	7.00	13.20	0.80	3	4	3	2	4	16
3	484333	65	Y	1	1	1	+	-	-	25	-	+	-	+	-	-	-	+	+	-	-	-	180	70.0	21.6	1.06	1	+	-	-	-	-	80	9.8	206	136	98	32	25.00	1.40	18	26	-	-	11.4	-	-	-	4	3	3	3	4	15
4	1E+06	46	Y	1	2	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	170	89.0	30.8	1.00	4	+	-	-	-	-	193	9.8	234	182	160	38	15.00	0.90	17	42	-	+	11.8	-	-	-	1	1	1	1	5	25
5	827698	50	Y	1	1	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	170	65.0	22.5	0.94	2	+	-	-	-	-	264	16.1	233	129	172	35	18.00	0.80	19	37	-	-	12.2	-	-	-	1	1	1	1	5	25
6	2E+06	70	Y	1	2	0	+	-	-	10	-	+	-	+	-	-	-	+	+	-	-	-	165	50.0	18.4	0.94	3	+	-	-	-	-	132	12.8	236	172	108	36	22.00	0.90	13	24	-	-	12.2	7.00	23.26	0.86	3	2	3	3	4	14
7	1E+06	60	Y	1	1	1	+	-	-	3	-	+	-	+	-	-	-	+	+	-	40	-	157	52.0	21.1	1.05	2	+	-	-	-	-	216	9.9	108	110	56	30	15.00	0.60	18	22	-	-	12.4	-	-	-	1	1	1	1	5	25
8	933256	54	Y	1	1	1	-	-	-	10	-	-	-	-	-	-	-	-	-	-	30	-	166	90.0	32.7	0.90	4	+	-	-	-	-	188	15.9	150	227	70	70	26.00	0.90	19	35	-	-	11.8	-	20.18	2.36	4	3	3	3	4	15
9	121898	68	Y	1	2	2	+	-	-	2	-	+	-	+	-	-	-	+	+	-	-	-	166	69.5	25.2	0.89	3	+	-	-	-	-	118	8.0	136	171	96	36	22.00	0.80	19	43	-	-	10.8	-	10.26	1.04	0	0	0	0	0	0
10	767649	42	Y	1	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	172	98.0	33.1	1.08	3	+	-	-	-	-	248	8.3	170	219	82	37	18.00	2.00	17	37	-	-	13.6	-	6.80	1.05	1	1	1	1	5	25
11	626766	60	Y	1	2	2	-	-	-	-	2	+	-	+	-	-	-	+	+	-	-	-	160	70.0	27.3	1.08	3	+	-	-	-	-	148	8.4	170	209	90	38	29.00	0.90	19	34	-	-	14.4	-	-	-	1	1	1	1	5	25
12	2E+06	70	Y	1	3	3	+	-	-	-	-	+	-	+	-	-	-	+	+	-	-	30	170	75.0	26.0	0.97	3	+	-	-	-	-	204	9.8	167	111	100	45	20.00	1.20	20	30	-	-	13.2	-	17.32	2.10	3	2	2	2	4	20
13	1E+06	56	Y	1	2	2	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	174	63.0	20.8	0.94	1	+	-	-	-	-	278	9.8	155	189	99	36	28.00	0.90	52	86	-	-	15.0	-	-	-	1	1	1	1	5	25
14	584219	57	Y	1	2	3	-	-	-	10	-	-	-	-	-	-	-	-	-	-	-	-	173	69.0	23.1	0.95	2	+	-	-	-	-	172	9.3	179	140	110	42	18.00	0.20	20	43	-	-	12.8	-	-	-	1	1	1	1	5	25
15	1E+06	52	Y	1	2	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	164	67.0	24.9	1.00	2	+	-	-	-	-	135	9.7	198	284	100	41	20.00	0.70	34	42	A	A	14.6	-	-	-	1	1	1	1	5	25
16	50822	76	Y	1	2	2	-	-	-	11	-	-	-	-	-	-	-	-	-	-	-	-	175	73.0	23.8	0.94	2	+	-	-	-	-	104	7.1	205	222	120	41	18.00	0.70	23	46	-	-	13.4	-	-	-	1	1	1	1	5	25
17	2E+06	50	Y	1	1	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	30	167	69.0	24.7	1.00	2	+	-	-	-	-	153	8.2	163	111	104	37	18.00	0.90	29	57	A	A	11.8	-	-	-	1	1	1	1	5	25
18	62092	54	Y	3	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	171	59.0	20.2	0.98	2	+	-	-	-	-	255	10.3	180	140	98	52	23.00	0.90	25	42	-	-	13.6	-	-	-	1	1	1	1	5	25
19	2E+06	61	Y	3	1	1	-	-	-	6	-	-	-	-	-	-	-	-	-	-	-	-	163	77.6	29.2	1.05	3	+	-	-	-	-	90	8.2	110	138	50	32	12.00	0.50	29	45	-	-	14.8	-	-	-	1	1	1	1	5	25
20	2E+06	58	Y	2	2	2	+	-	-	11	-	-	-	-	-	-	-	-	-	-	-	-	165	77.0	28.3	1.05	3	+	-	-	-	-	200	10.6	137	127	61	49	21.00	1.10	30	47	-	-	10.8	-	-	-	1	1	1	1	5	25
21	2E+06	62	Y	1	2	1	-	-	-	-	2	-	-	-	-	-	-	-	-	+	-	-	168	72.0	25.5	1.08	3	+	-	-	-	-	143	8.6	180	112	96	72	22.00	0.80	23	39	A	A	11.8	-	-	-	1	1	1	1	5	25
22	2E+06	58	Y	2	2	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	160	72.0	28.1	0.95	3	+	-	-	-	-	199	13.1	187	101	122	45	22.00	1.10	15	38	-	-	14.1	-	10.10	2.05	1	1	1	1	5	25
23	1E+06	55	Y	0	1	2	-	-	-	-	2	-	-	-	-	-	-	-	-	+	30	30	173	50.0	16.7	1.06	2	+	-	-	-	-	163	13.8	228	127	165	38	20.00	0.90	19	43	A	A	14.9	-	-	-	1	1	1	1	5	25
24	2E+06	52	Y	2	2	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	161	79.0	30.5	1.00	2	+	-	-	-	-	127	9.1	147	85	102	28	18.00	0.80	30	50	-	-	14.1	-	8.60	8.67	1	1	1	1	5	25
25	1E+06	58	Y	1	2	1	-	-	-	1	-	-	-	-	-	-	-	-	-	-	30	25	174	70.0	23.1	0.98	3	+	-	-	-	-	168	9.3	149	152	78	41	24.00	0.90	27	41	-	-	11.8	-	-	-	3	3	3	4	4	16
26	135674	61	Y	3	2	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	157	58.0	23.5	1.30	2	+	-	-	-	-	360	11.8	180	110	70	42	18.00	0.60	22	36	A	A	11.8	-	-	-	1	1	1	1	5	25
27	926956	80	Y	1	2	3	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	168	66.0	23.4	0.90	2	+	-	-	-	-	235	8.0	119	70	65	40	12.00	1.30	21	34	-	-	12.1	-	-	-	1	1	1	1	5	25
28	2E+06	71	Y	1	2	2	-	-	-	22	2	-	-	-	-	-	-	-	-	-	40	-	163	84.0	31.6	1.00	4	+	-	-	-	-	159	8.2	140	130	81	33	18.00	0.90	21	30	-	-	11.8	-	10.80	2.08	3	3	3	3	4	16
29	2E+06	62	Y	1	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	20	-	172	75.0	25.4	1.00	3	+	-	-	-	-	203	10.0	274	394	149	46	28.00	0.90	16	41	-	-	13.2	-	2.90	1.77	2	2	2	1	5	22
30	2E+06	52	Y	1	3	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	30	-	167	98.0	35.1	1.00	5	+	-	-	-	-	127	8.5	174	116	116	35	23.00	0.80	19	43	-	-	12.6	-	10.20	1.21	1	1	1	1	5	25
31	1E+06	55	Y	1	2	1	+	-	-	10	-	+	-	+	-	-	-	+	+	+	-	-	174	84.0	27.7	1.03	3	+	-	-	-	-	233	11.3	122	96	68	35	19.00	0.90	27	55	-	-	13.8	4.76	11.86	0.56	2	2	2	2	5	21
32	238979	52	Y	1	2	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	163	65.0	24.5	1.05	2	+	-	-	-	-	103	7.0	160	194	91	30	18.00	0.80	40	65	-	-	14.6	-	-	-	1	1	1	1	5	25
33	2E+06	53	Y	1	1	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	162	64.0	24.4	0.98	4	+	-	-	-	-	118	13.1	187	101	122	45	22.00	1.10	15	38	-	-	14.1	-	10.10	2.05	1	1	1	1	5	25
34	1E+06	55	Y	0	2	3	-	-	-	6	-	-	-	-	-	-	-	-	-	-	-	-	172	65.0	22.0	0.94	2	+	-	-	-	-	185	8.0	135	67	74	63	7.44	0.90	28	18	-	-	13.3	82.00	10.40	0.97	3	3	2	2	4	18
35	1E+06	43	Y	0	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	172	76.0	25.7	0.87	3	+	-	-	-	-	83	7.6	176	112	109	44	22.00	0.80	23	42	-	-	12.2	-	-	-	1	1	1	1	5	25
36	1E+06	44	Y	1	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	166	79.0	28.7	0.96	3	+	-	-	-	-	121	7.8	228	137	162	39	32.00	1.20	38	66	-	-	11.2	-	-	-	1	1	1	1	5	25

ANNEXURE III - MASTER CHART

Serial Number	In patient number	Demo-graphy			DM history		History of other illnesses					Surgical history				Drug history					Risk factors		General physical examination						Genital examination				Investigations																Questionnaire						
		Age (Years)	Marital status	Educational status	Duration (Years)	Treatment	Cardiovascular disease	Neurological/psychiatric	Other endocrine disease	Hypertension (Years)	Family history of DM	History	Pelvic/spinal	Prostate	Other	5α reductase inhibitor	Antipsychotic/ Antidepressant drugs	History of reduced libido	Erectile dysfunction	Circumcision	Smoking (Years)	Alcohol consumption (Years)	Height (Cms)	Weight (Kgs)	Body mass index (Kg/m2)	Waist hip Ratio	Blood pressure	Peripheral pulses	Penile	Testis	Phimosis	Secondary sexual characteristics	Plasma glucose (FBS/RBS/PPBS)	HbA1c	Cholesterol	Triglycerides	Low density lopoprotein	High density lipoprotein	Urea	Creatinine	SGOT	SGPT	Urine routine	Urine microscopy	Haemoglobin	Thyroid function T3	Thyroid function T4	Thyroid function TSH	Q1	Q2	Q3	Q4	Q5	Total score
37	5E+06	51	Y	0	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	20	-	165	69.0	25.3	0.97	3	+	-	-	-	-	132	8.4	276	170	160	23	32.00	1.10	23	48	-	-	11.8	-	22.80	2.08	4	4	4	4	2	10
38	377133	75	Y	0	2	3	-	-	-	12	-	-	-	-	-	-	-	-	-	-	-	-	175	71.0	23.2	0.94	2	+	-	-	-	-	133	84.0	205	222	120	41	13.00	0.90	18	45	-	-	13.6	-	-	-	2	2	3	3	5	19
39	509610	54	Y	0	2	0	-	-	-	-	-	-	-	-	-	-	-	-	-	-	28	20	163	58.0	21.8	0.98	2	+	-	-	-	-	124	5.8	132	76	76	40	21.00	0.80	36	42	-	-	11.8	-	-	-	1	1	1	1	5	25
40	509922	32	Y	1	1	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	177	65.0	20.7	0.94	2	+	-	-	-	-	118	5.6	162	111	109	82	18.00	0.70	18	14	-	-	9.3	-	-	-	1	1	1	1	5	25
41	509781	30	Y	1	1	0	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	173	86.0	28.7	0.96	2	+	-	-	-	-	116	6.8	112	80	82	72	28.00	1.20	28	32	+	-	10.8	-	-	-	1	1	1	1	5	25
42	509384	38	Y	1	1	3	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	174	63.0	20.8	0.94	2	+	-	-	-	-	129	7.6	142	78	96	44	21.00	0.80	38	44	-	-	13.6	-	-	-	1	1	1	1	5	25
43	509418	50	Y	1	1	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	170	65.0	22.5	0.94	2	+	-	-	-	-	130	11.8	180	129	108	42	18.00	0.80	19	37	-	-	12.8	-	-	-	1	1	1	1	5	25
44	508215	53	Y	1	2	1	-	-	-	10	2	-	-	-	-	-	-	-	-	-	-	-	165	69.0	25.3	0.97	3	+	-	-	-	-	142	9.8	286	172	168	32	32.00	1.40	32	28	-	-	11.8	-	-	-	4	4	4	4	2	10
45	509603	35	Y	3	1	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	5	8	157	52.0	21.1	1.05	2	+	-	-	-	-	150	5.8	108	98	56	38	17.00	0.80	31	24	-	-	13.8	-	-	-	1	1	1	1	5	25
46	503914	56	Y	1	2	3	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	158	52.0	20.8	1.03	2	+	-	-	-	-	176	12.8	192	206	100	51	24.00	0.80	16	39	T	-	12.1	-	-	-	4	1	3	3	4	16
47	509327	47	Y	1	2	2	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	157	67.0	27.2	0.94	3	+	-	-	-	-	92	8.2	144	138	86	38	32.00	0.80	18	32	-	-	10.8	-	-	-	2	2	2	2	5	21
48	509318	53	Y	1	1	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	177	65.0	20.7	0.92	2	+	-	-	-	-	101	5.6	134	111	72	54	16.00	1.10	20	41	-	-	11.8	-	-	-	1	1	1	1	5	25
49	509418	50	Y	1	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	177	65.0	20.7	0.90	2	+	-	-	-	-	160	6.6	126	108	120	50	28.00	1.10	26	41	-	-	14.2	-	-	-	1	1	1	1	5	25
50	509970	55	Y	1	1	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	167	61.0	21.9	0.94	2	+	-	-	-	-	145	5.2	172	82	78	54	29.00	0.70	32	46	-	-	11.8	-	-	-	1	1	1	1	5	25
51	509789	50	Y	0	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	168	65.0	23.0	0.98	2	+	+	+	-	-	152	8.8	136	140	132	52	21.00	6.00	42	32	-	-	11.8	-	-	-	1	1	1	1	5	25
52	509384	38	Y	2	1	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	158	48.0	19.2	0.93	2	+	-	-	-	-	156	7.2	116	94	88	76	28.00	1.30	18	32	-	-	11.1	-	-	-	1	1	1	1	5	25
53	508215	53	Y	1	2	2	-	-	-	5	-	-	-	-	-	-	-	-	-	-	-	-	177	65.0	20.7	0.86	2	+	-	-	-	-	196	12.0	176	76	130	39	22.00	0.90	23	31	-	-	12.1	-	-	-	4	4	4	4	3	11
54	507672	53	Y	3	2	3	+	-	-	-	-	-	-	-	-	-	-	-	-	-	4	6	165	67.0	24.6	0.93	2	+	-	-	-	-	81	8.0	132	162	54	30	32.00	0.80	32	47	-	-	11.8	-	-	-	2	2	1	2	4	21
55	507896	42	Y	0	2	2	-	-	-	10	-	-	-	-	-	-	-	-	-	-	-	40	161	63.0	24.3	0.96	4	+	-	-	-	-	102	5.8	108	98	56	38	17.00	0.80	31	24	-	-	13.8	-	-	-	0	0	0	0	0	0
56	509197	50	Y	0	1	1	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	160	70.0	27.3	1.08	3	+	-	-	-	-	112	5.8	170	80	92	62	28.00	1.10	72	32	-	-	13.2	-	-	-	1	1	1	1	5	9
57	509139	32	Y	0	1	0	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	170	89.0	30.8	1.00	4	+	-	-	-	-	142	6.8	134	132	128	42	30.00	0.90	27	19	A	A	13.8	-	-	-	1	1	1	1	5	25
58	509709	55	Y	0	1	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	157	52.0	21.1	1.05	2	+	-	-	-	-	123	6.8	108	110	56	48	15.00	0.60	18	22	-	-	12.8	-	-	-	1	1	1	1	5	25
59	509121	37	Y	1	1	1	-	-	-	5	2	-	-	-	-	-	-	-	-	-	5	-	159	56.0	22.2	0.94	2	+	-	-	-	-	326	7.3	160	116	112	33	14.00	0.70	28	46	A	A	11.2	-	8.80	1.19	1	1	1	1	5	25
60	509138	51	Y	1	1	1	+	-	-	10	-	-	-	-	-	-	-	-	-	-	20	-	172	75.0	25.4	1.00	3	+	-	-	-	-	182	10.4	262	300	150	52	20.00	0.80	21	34	-	-	14.6	-	-	-	2	2	2	1	5	22
61	509213	45	Y	0	1	0	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	157	52.0	21.1	1.05	2	+	-	-	-	-	174	6.8	107	109	56	52	31.00	0.90	32	43	-	-	11.8	-	-	-	1	1	1	1	5	25
62	508947	34	Y	1	1	0	-	-	-	5	2	-	-	-	-	-	-	-	-	-	-	-	163	58.0	21.8	0.98	2	+	+	+	-	-	160	4.4	132	78	77	52	20.00	0.70	21	32	A	A	12.7	-	-	-	1	1	1	1	5	25
63	508427	65	Y	3	2	1	-	-	-	-	2	+	-	+	-	-	-	+	+	-	-	-	167	84.0	30.1	1.08	3	+	-	-	-	-	138	7.9	145	118	83	36	45.00	1.20	35	24	-	-	10.4	-	-	-	1	1	1	1	5	25
64	508010	42	Y	1	1	1	+	-	-	-	-	+	-	+	-	-	-	+	+	-	-	-	177	65.0	20.7	0.92	2	+	-	-	-	-	112	5.6	135	112	88	52	16.00	0.80	20	28	-	-	12.7	-	-	-	1	1	1	1	5	25
65	508522	30	Y	1	1	1	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	177	65.0	20.7	0.94	2	+	-	-	-	-	118	5.6	162	111	100	52	18.00	0.70	18	14	-	-	9.3	-	-	-	1	1	1	1	5	25
66	508044	38	Y	1	1	1	+	-	-	-	-	+	-	+	-	-	-	+	+	-	-	-	177	65.0	20.7	0.90	2	+	-	-	-	-	118	5.6	126	108	120	52	18.00	10.00	26	41	-	-	15.5	-	-	-	1	1	1	1	5	25
67	504815	70	Y	1	2	2	+	-	-	15	2	-	-	-	-	-	-	-	-	-	-	-	163	84.0	31.6	1.00	4	+	-	-	-	-	149	8.2	140	130	82	33	25.00	1.40	21	30	-	-	10.3	-	-	-	3	3	3	3	2	14
68	508048	55	Y	3	2	2	+	-	-	11	-	+	-	+	-	-	-	+	+	-	-	25	170	75.0	26.0	1.00	3	+	-	-	-	-	112	5.6	167	141	108	32	18.00	1.10	22	34	-	-	11.8	-	-	-	1	2	2	2	4	20
69	507929	35	Y	1	1	0	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	167	61.0	21.9	0.97	2	+	-	-	-	-	112	5.2	162	74	80	52	28.00	0.80	20	39	-	-	12.6	-	-	-	1	1	1	1	5	25
70	508368	65	Y	2	2	2	+	-	-	-	2	+	-	+	-	-	-	+	+	-	-	-	158	48.0	19.2	0.93	2	+	-	-	-	-	126	6.2	118	92	98	76	18.00	0.70	15	28	-	-	13.6	-	-	-	1	1	1	1	5	25
71	813419	35	Y	3	1	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	160	70.0	27.3	1.08	3	+	-	-	-	-	112	5.2	170	80	90	52	28.00	1.20	34	28	-	-	14.2	-	-	-	1	1	1	1	5	25
72	508627	42	Y	1	2	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	174	63.0	20.8	0.94	2	+	-	-	-	-	142	7.6	152	88	98	42	28.00	3.00	32	42	-	-	13.8	-	-	-	1	1	1	1	5	25

ANNEXURE III - MASTER CHART

Serial Number	In patient number	Demo-graphy			DM history		History of other illnesses					Surgical history				Drug history					Risk factors		General physical examination						Genital examination				Investigations																Questionnaire						
		Age (Years)	Marital status	Educational status	Duration (Years)	Treatment	Cardiovascular disease	Neurological/psychiatric	Other endocrine disease	Hypertension (Years)	Family history of DM	History	Pelvic/spinal	Prostate	Other	5α reductase inhibitor	Antipsychotic/ Antidepressant drugs	History of reduced libido	Erectile dysfunction	Circumcision	Smoking (Years)	Alcohol consumption (Years)	Height (Cms)	Weight (Kgs)	Body mass index (Kg/m2)	Waist hip Ratio	Blood pressure	Peripheral pulses	Penile	Testis	Phimosis	Secondary sexual characteristics	Plasma glucose (FBS/RBS/PPBS)	HbA1c	Cholesterol	Triglycerides	Low density lopoprotein	High density lipoprotein	Urea	Creatinine	SGOT	SGPT	Urine routine	Urine microscopy	Haemoglobin	Thyroid function T3	Thyroid function T4	Thyroid function TSH	Q1	Q2	Q3	Q4	Q5	Total score
73	507902	48	Y	1	2	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	173	69.0	23.1	0.95	2	+	-	-	-	-	118	9.8	132	98	76	52	31.00	1.20	21	16	-	-	14.6	-	-	-	1	1	1	1	5	25
74	507922	45	Y	3	1	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	172	48.0	16.2	0.98	4	+	-	-	-	-	158	5.6	170	98	80	37	20.00	0.80	38	24	-	-	10.9	-	-	-	1	1	1	1	5	25
75	507890	50	Y	0	2	2	+	-	-	10	-	-	-	-	-	-	-	-	-	-	-	-	174	87.0	28.7	1.03	3	+	-	-	-	-	243	12.8	122	96	58	45	15.00	0.80	40	24	-	-	14.6	-	-	-	2	2	2	2	5	21
76	507952	68	Y	1	2	2	+	-	-	10	-	-	-	-	-	-	-	-	-	-	-	-	174	70.0	23.1	0.98	2	+	-	-	-	-	152	8.3	139	142	72	40	29.00	1.50	36	35	-	-	14.2	-	-	-	1	1	1	1	5	25
77	508974	42	Y	3	1	1	+	-	-	8	2	-	-	-	-	-	-	-	-	-	-	-	173	81.0	27.1	1.40	3	+	-	-	-	-	129	6.2	128	136	58	29	16.00	0.80	10	14	-	-	14.2	-	-	-	1	1	1	1	5	25
78	508973	38	Y	1	2	3	-	-	-	8	2	-	-	-	-	-	-	-	-	-	-	-	173	73.0	24.4	1.00	3	+	-	-	-	-	281	8.9	116	76	68	33	16.00	0.80	24	26	-	-	13.5	-	-	-	1	1	1	1	5	25
79	508252	31	Y	1	2	3	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	168	66.0	23.4	0.90	2	+	-	-	-	-	118	8.0	116	68	60	42	20.00	0.70	22	21	-	-	12.7	-	-	-	1	1	1	1	5	25
80	506945	46	Y	0	2	3	-	-	-	4	1	-	-	-	-	-	-	-	-	-	15	15	161	79.0	30.5	0.99	4	+	-	-	-	-	137	10.4	167	86	112	38	28.00	0.90	30	42	-	-	12.8	-	8.60	8.67	3	3	3	3	4	16
81	507962	50	Y	1	2	1	+	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	157	58.0	23.5	1.30	2	+	-	-	-	-	180	9.8	132	98	68	42	42.00	1.00	29	35	-	-	12.3	-	-	-	1	1	1	1	5	25
82	509826	38	Y	1	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	160	72.0	28.1	0.95	3	+	-	-	-	-	132	9.1	137	101	122	35	22.00	1.10	15	38	-	-	13.8	-	-	-	1	1	1	1	5	25
83	506579	58	Y	1	1	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	172	76.0	25.7	0.87	3	+	-	-	-	-	94	7.2	172	108	107	43	21.00	0.90	22	38	-	-	12.2	-	-	-	1	1	1	1	5	25
84	506570	72	Y	1	2	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	30	30	167	98.0	35.1	1.00	5	+	-	-	-	-	132	6.8	162	114	115	40	20.00	0.90	39	22	-	-	12.6	-	-	-	1	1	1	1	5	13
85	506580	40	Y	1	1	2	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	166	81.0	29.4	0.93	2	+	-	-	-	-	162	10.1	132	176	62	36	39.00	1.00	34	46	-	-	15.2	-	-	-	1	1	1	1	5	9
86	506762	30	Y	1	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	164	67.0	24.9	1.00	2	+	-	-	-	-	128	8.8	172	182	98	44	32.00	0.80	28	32	-	-	12.6	-	-	-	1	1	1	1	5	25
87	506261	48	Y	1	1	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	167	69.0	24.7	1.00	2	+	-	-	-	-	148	9.8	152	98	103	38	18.00	0.90	29	28	A	A	108.0	-	-	-	1	1	1	1	5	25
88	506762	30	Y	1	1	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	165	76.0	27.9	1.05	2	+	-	-	-	-	136	6.8	120	118	56	48	21.00	0.80	30	47	-	-	10.8	-	-	-	1	1	1	1	5	25
89	506571	65	Y	1	1	1	-	-	-	6	2	-	-	-	-	-	-	-	-	-	-	-	163	77.6	29.2	0.94	3	+	-	-	-	-	118	8.2	98	136	50	48	31.00	1.70	46	31	-	-	14.3	-	-	-	1	1	1	1	5	25
90	507934	60	Y	3	2	0	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	171	59.0	20.2	0.98	2	+	-	-	-	-	180	9.8	160	138	98	56	16.00	0.80	45	18	-	-	12.0	-	-	-	1	1	1	1	5	25
91	509281	65	Y	3	2	2	-	-	-	15	2	-	-	-	-	-	-	-	-	-	-	-	170	72.0	24.9	1.22	2	+	-	-	-	-	109	10.6	160	136	130	41	28.00	1.00	21	20	-	-	11.0	-	-	-	2	2	2	2	4	20
92	509330	68	Y	3	2	2	-	-	-	11	-	-	-	-	-	-	-	-	-	-	-	-	175	73.0	23.8	0.94	2	+	-	-	-	-	100	7.8	118	98	94	52	18.00	1.20	42	15	-	-	10.7	-	-	-	1	1	1	1	5	25
93	506340	60	Y	3	2	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	168	72.0	25.5	1.08	3	+	-	-	-	-	133	8.6	136	112	86	68	22.00	0.80	23	28	-	-	11.8	-	-	-	1	1	1	1	5	25
94	506314	49	Y	1	2	2	+	-	-	8	-	-	-	-	-	-	-	-	-	-	15	-	166	90.0	32.7	0.90	4	+	-	-	-	-	188	15.9	138	227	68	72	26.00	0.90	19	35	-	-	11.8	-	-	-	4	3	3	3	4	15
95	505325	59	Y	1	1	3	+	-	-	-	-	-	-	-	-	-	-	-	-	-	10	-	166	55.0	20.0	0.97	2	+	-	-	-	-	214	7.6	167	123	117	25	32.00	0.80	11	26	-	-	10.2	-	-	-	4	2	2	3	5	18
96	506389	46	Y	1	1	3	+	-	-	-	2	-	-	-	-	-	-	-	-	-	15	18	173	50.0	16.7	1.06	2	+	-	-	-	-	180	11.8	198	126	155	48	26.00	1.20	38	60	-	-	14.2	-	-	-	1	1	1	1	5	25
97	505477	55	Y	3	1	0	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	163	60.0	22.6	0.92	2	+	-	-	-	-	212	12.5	157	176	91	30	39.00	1.20	30	32	-	-	14.1	-	-	-	1	1	1	1	5	25
98	506552	30	Y	1	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	166	79.0	28.7	0.96	2	+	-	-	-	-	121	7.8	228	137	162	39	32.00	1.20	38	66	-	-	11.2	-	-	-	1	1	1	1	5	25
99	505245	46	Y	3	2	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	160	57.0	22.3	0.95	2	+	-	-	-	-	109	68.0	210	170	116	36	28.00	0.80	26	37	-	-	11.2	-	-	-	1	1	1	1	5	25
100	506311	55	Y	1	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	178	68.0	21.5	1.26	2	+	-	-	-	-	119	7.6	214	159	149	33	25.00	1.10	32	36	-	-	11.1	-	-	1.05	1	1	1	1	5	25
101	506026	32	Y	1	2	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	170	75.5	26.1	0.94	3	+	-	-	-	-	120	9.3	210	251	39	99	32.00	0.90	14	28	-	-	10.8	-	-	-	1	1	1	1	5	25
102	505386	72	Y	1	2	2	-	-	-	8	-	-	-	-	-	-	-	-	-	-	-	-	172	65.0	22.0	0.94	2	+	-	-	-	-	185	8.0	135	67	74	63	27.00	0.90	28	18	-	-	13.3	-	-	-	3	3	2	2	4	18
103	506808	32	Y	1	1	1	-	-	-	-	2	-	-	-	-	-	-	-	-	+	-	-	168	68.0	24.1	1.03	2	+	-	-	-	-	112	7.9	145	118	82	36	30.00	0.44	34	30	-	-	15.4	-	-	-	1	1	1	1	5	25
104	506603	51	Y	1	2	3	-	-	-	13	-	-	-	-	-	-	-	-	-	-	10	-	166	90.0	32.7	0.90	4	+	-	-	-	-	176	14.8	140	127	68	72	26.00	0.90	29	30	-	-	11.8	-	-	-	4	3	3	3	4	15
105	506603	40	Y	1	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	172	62.0	21.0	0.84	2	+	-	-	-	-	161	7.6	187	160	78	58	35.00	1.00	29	49	-	-	11.8	-	-	-	1	1	1	1	5	25
106	505949	64	Y	1	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	167	66.0	23.7	1.08	2	+	-	-	-	-	157	7.1	194	222	117	38	24.00	0.70	18	28	-	-	11.1	-	-	-	4	4	4	4	4	12
107	505228	68	Y	1	1	3	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	160	70.0	27.3	0.96	3	+	-	-	-	-	162	7.8	136	90	90	48	18.00	0.70	32	28	-	-	11.8	-	-	-	1	1	1	1	5	25
108	506392	50	Y	1	1	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	167	66.0	23.7	1.06	2	+	-	-	-	-	142	6.7	180	130	76	70	31.00	0.80	18	20	-	-	10.2	-	-	-	1	1	1	1	5	25

ANNEXURE III - MASTER CHART

Serial Number	In patient number	Demo-graphy			DM history		History of other illnesses					Surgical history				Drug history						Risk factors		General physical examination						Genital examination				Investigations																Questionnaire						
		Age (Years)	Marital status	Educational status	Duration (Years)	Treatment	Cardiovascular disease	Neurological/psychiatric	Other endocrine disease	Hypertension (Years)	Family history of DM	History	Pelvic/spinal	Prostate	Other	5α reductase inhibitor	Antipsychotic/ Antidepressant drugs	History of reduced libido	Erectile dysfunction	Circumcision	Smoking (Years)	Alcohol consumption (Years)	Height (Cms)	Weight (Kgs)	Body mass index (Kg/m2)	Waist hip Ratio	Blood pressure	Peripheral pulses	Penile	Testis	Phimosis	Secondary sexual characteristics	Plasma glucose (FBS/RBS/PPBS)	HbA1c	Cholesterol	Triglycerides	Low density lopoprotein	High density lipoprotein	Urea	Creatinine	SGOT	SGPT	Urine routine	Urine microscopy	Haemoglobin	Thyroid function T3	Thyroid function T4	Thyroid function TSH	Q1	Q2	Q3	Q4	Q5	Total score
109	502869	42	Y	1	1	1	-	-	-	-	2	-	-	-	-	-	-	-	-	+	-	-	167	66.0	23.7	1.06	2	+	-	-	-	-	110	6.8	180	70	79	80	39.00	1.30	30	30	-	-	11.6	-	-	-	1	1	1	1	5	25
110	506421	67	Y	1	1	2	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	165	95.0	34.9	1.00	4	+	-	-	-	-	140	11.5	225	200	170	38	19.00	0.70	29	28	-	-	14.2	-	-	-	1	1	1	1	5	25
111	502903	43	Y	1	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	163	74.0	27.9	1.05	3	+	-	-	-	-	150	8.1	212	167	46	42	25.00	0.80	34	63	-	-	11.1	-	14.20	2.80	1	1	1	1	5	25
112	503460	56	Y	1	2	3	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	154	58.0	24.5	1.04	2	+	-	-	-	-	137	8.6	206	90	140	30	37.00	1.40	28	42	-	-	10.9	-	10.50	2.70	1	1	1	1	5	9
113	560237	38	Y	1	2	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	175	75.0	24.5	1.08	2	+	-	-	-	-	282	15.7	204	183	126	41	38.00	0.90	21	24	-	-	14.5	-	12.70	1.80	1	1	1	1	5	25
114	560224	55	Y	3	1	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	167	66.0	23.7	0.94	2	+	-	-	-	-	285	7.7	170	130	117	38	19.00	0.90	20	38	-	-	10.8	4.86	12.30	1.76	1	1	1	1	5	25
115	501611	62	Y	2	2	3	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	152	65.0	28.1	1.08	3	+	-	-	-	-	135	6.8	92	80	40	30	22.00	1.00	25	56	-	-	12.8	5.40	18.42	1.76	1	1	1	1	5	25
116	503056	60	Y	3	2	2	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	12	178	68.0	21.5	0.94	2	+	-	-	-	-	93	8.6	155	100	99	36	34.00	1.00	41	33	-	-	9.4	-	-	-	1	1	1	1	5	25
117	503561	49	Y	3	2	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	172	98.0	33.1	1.08	4	+	-	-	-	-	248	8.3	170	219	82	37	18.00	10.80	17	37	-	-	11.8	-	4.80	1.02	1	1	1	1	5	25
118	504520	42	Y	3	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	164	66.0	24.5	1.12	2	+	-	-	-	-	164	8.8	172	182	98	44	32.00	0.80	28	32	-	-	12.6	-	-	-	1	1	1	1	5	25
119	505502	42	Y	1	2	3	-	-	-	3	-	-	-	-	-	-	-	-	-	-	-	-	180	70.0	21.6	1.06	2	+	-	-	-	-	255	13.8	216	142	100	33	48.00	1.40	20	24	-	-	10.5	-	-	-	4	3	3	3	4	15
120	502772	52	Y	1	2	3	-	-	-	8	-	-	-	-	-	-	-	-	-	-	30	-	167	66.0	23.7	0.94	2	+	-	-	-	-	355	10.8	218	182	120	52	29.00	0.90	18	20	-	-	17.4	-	-	-	4	4	4	0	0	6
121	505406	66	Y	1	1	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	5	15	168	58.0	20.5	0.94	2	+	-	-	-	-	336	14.8	168	116	108	33	21.00	1.00	32	28	-	-	11.8	-	8.80	1.19	1	1	1	1	5	25
122	505475	65	Y	1	2	3	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	178	82.1	25.9	0.95	3	+	-	-	-	-	187	11.8	182	88	135	29	38.00	1.00	19	33	-	-	11.4	-	-	-	1	1	1	1	5	25
123	506274	45	Y	1	1	0	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	167	66.0	23.7	1.06	2	+	-	-	-	-	220	8.8	150	120	92	52	20.00	1.60	36	28	-	-	10.4	-	-	-	1	1	1	1	5	25
124	502429	45	Y	1	1	2	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	165	45.0	16.5	1.00	1	+	-	-	-	-	172	9.0	214	182	76	32	12.00	0.80	28	46	-	-	14.3	-	-	-	1	1	1	1	5	25
125	506697	75	Y	1	2	3	-	-	-	3	-	-	-	-	-	-	-	-	-	-	-	-	175	71.0	23.2	0.94	2	+	-	-	-	-	236	10.8	208	228	140	31	34.00	1.00	18	32	-	-	12.8	-	-	-	2	2	3	3	4	18
126	505494	72	Y	1	2	1	-	-	-	20	-	-	-	-	-	-	-	-	-	-	-	-	178	68.0	21.4	1.06	2	+	-	-	-	-	203	8.6	220	180	120	40	14.00	1.10	18	36	-	-	11.8	-	-	-	3	2	2	2	4	19
127	506234	50	Y	1	1	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	165	69.0	25.3	1.25	3	+	-	-	-	-	107	8.9	182	106	98	26	27.00	0.80	31	30	-	-	9.9	-	-	-	1	1	1	1	5	25
128	505812	49	Y	1	1	2	+	-	-	5	1	-	-	-	-	-	-	-	-	-	14	-	157	52.0	21.1	1.05	2	+	-	-	-	-	216	9.9	108	110	56	30	32.00	0.80	18	22	-	-	11.2	-	-	-	1	1	1	1	5	25
129	506542	30	Y	1	1	2	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	170	65.0	22.5	0.94	2	+	-	-	-	-	254	16.1	233	129	172	35	20.00	1.60	32	28	-	-	9.8	-	-	-	1	1	1	1	5	25
130	506340	60	Y	1	2	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	170	80.0	27.7	1.00	3	+	-	-	-	-	193	9.8	234	182	160	38	14.00	0.80	36	28	-	-	10.4	-	-	-	1	1	1	1	5	25
131	506347	60	Y	3	2	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	168	65.0	23.0	0.98	2	+	-	-	-	-	242	10.1	236	181	156	44	33.00	1.50	16	19	-	-	11.4	-	6.70	2.46	1	1	1	1	5	25
132	506261	48	Y	3	2	1	+	-	-	-	2	-	-	-	-	-	-	-	-	-	-	20	168	83.2	29.5	0.96	3	+	-	-	-	-	123	8.4	177	163	111	33	21.50	0.58	38	34	-	-	11.2	6.80	12.30	2.80	1	1	1	1	5	25
133	506083	49	Y	1	1	1	+	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	171	69.0	23.6	1.10	2	+	-	-	-	-	111	4.8	113	80	70	86	48.00	1.89	32	38	A	A	13.7	-	-	-	1	1	1	1	5	25
134	506450	54	Y	1	2	3	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	172	63.0	21.3	0.91	2	+	-	-	-	-	173	8.9	168	144	109	30	31.00	0.90	29	35	-	-	13.4	-	-	-	1	1	1	1	5	25
135	505370	35	Y	1	2	3	-	-	-	4	2	-	-	-	-	-	-	-	-	-	-	-	167	66.0	23.7	1.06	1	+	-	-	-	-	98	5.6	158	128	80	72	31.00	0.90	30	40	-	-	13.4	3.60	15.00	0.60	1	1	1	1	5	25
136	506852	60	Y	1	2	2	+	-	-	3	-	-	-	-	-	-	-	-	-	-	-	-	177	65.0	20.7	0.86	2	+	-	-	-	-	196	11.6	172	72	130	29	30.00	1.80	23	31	A	A	10.5	17.20	2.08	0.42	4	4	4	4	2	10
137	526312	43	Y	3	2	3	+	-	-	5	2	-	-	-	-	-	-	-	-	-	-	-	174	69.0	22.8	1.06	2	+	-	-	-	-	168	16.1	233	129	172	35	20.00	1.60	32	28	-	-	9.8	-	-	-	1	1	1	1	5	25
138	518420	52	Y	1	2	0	+	-	-	5	-	-	-	-	-	-	-	-	-	-	-	-	172	67.0	22.6	0.98	2	+	-	-	-	-	153	9.3	185	331	88	31	32.00	0.90	18	34	-	-	13.2	-	-	-	1	1	1	1	5	9
139	482722	72	Y	1	1	1	-	-	-	10	-	-	-	-	-	-	-	-	-	-	-	-	178	68.0	21.5	1.06	2	+	-	-	-	-	65	7.2	220	180	120	40	14.00	0.90	18	48	-	-	11.8	-	20.26	0.82	3	2	2	2	4	19
140	485104	80	Y	3	2	3	-	-	-	5	2	+	-	+	-	-	-	+	+	-	40	-	167	66.0	23.7	0.94	2	+	-	-	-	-	120	8.8	230	180	120	42	18.00	1.00	20	46	A	A	13.0	-	-	-	0	0	0	0	0	0
141	471242	45	Y	1	1	3	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	167	66.0	23.7	1.06	2	+	-	-	-	-	98	5.6	160	130	80	72	20.00	0.70	14	32	-	-	10.5	3.60	15.00	0.60	1	1	1	1	5	25
142	472909	49	Y	1	1	3	-	-	-	-	2	-	-	-	-	-	-	-	-	-	20	20	172	70.0	23.7	0.94	2	+	-	-	-	-	320	14.2	115	119	61	30	18.00	0.70	44	45	A	+	10.9	-	-	-	3	2	3	3	4	18
143	487321	38	Y	1	2	3	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	172	63.0	21.3	0.91	2	+	-	-	-	-	173	8.9	168	144	109	30	32.00	1.10	32	23	A	A	11.8	-	-	-	1	1	1	1	5	25
144	492013	51	Y	2	2	1	+	-	-	-	2	-	-	-	-	-	-	-	-	-	-	20	168	83.2	29.5	0.96	3	+	-	-	-	-	123	8.4	177	163	111	33	21.50	0.54	40	36	A	A	11.2	6.80	12.30	3.80	1	1	1	1	5	25

ANNEXURE III - MASTER CHART

Serial Number	In patient number	Demo-graphy			DM history		History of other illnesses					Surgical history				Drug history					Risk factors		General physical examination						Genital examination				Investigations																Questionnaire						
		Age (Years)	Marital status	Educational status	Duration (Years)	Treatment	Cardiovascular disease	Neurological/psychiatric	Other endocrine disease	Hypertension (Years)	Family history of DM	History	Pelvic/spinal	Prostate	Other	5α reductase inhibitor	Antipsychotic/ Antidepressant drugs	History of reduced libido	Erectile dysfunction	Circumcision	Smoking (Years)	Alcohol consumption (Years)	Height (Cms)	Weight (Kgs)	Body mass index (Kg/m2)	Waist hip Ratio	Blood pressure	Peripheral pulses	Penile	Testis	Phimosis	Secondary sexual characteristics	Plasma glucose (FBS/RBS/PPBS)	HbA1c	Cholesterol	Triglycerides	Low density lopoprotein	High density lipoprotein	Urea	Creatinine	SGOT	SGPT	Urine routine	Urine microscopy	Haemoglobin	Thyroid function T3	Thyroid function T4	Thyroid function TSH	Q1	Q2	Q3	Q4	Q5	Total score
145	501342	35	Y	0	1	1	-	-	-	3	2	-	-	-	-	-	-	-	-	-	-	15	153	60.0	25.6	0.94	2	+	-	-	-	-	136	9.3	185	331	88	31	32.00	0.90	18	34	-	-	13.2	-	-	-	1	1	1	1	5	25
146	1E+06	45	Y	1	1	1	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	163	74.0	27.9	1.05	3	+	-	-	-	-	115	8.0	212	167	47	41	25.00	0.70	34	63	-	-	11.1	-	14.20	2.80	1	1	1	1	5	25
147	742312	45	Y	1	2	2	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	167	66.0	23.7	1.08	2	+	-	-	-	-	157	7.1	194	222	117	38	24.00	0.70	18	28	-	-	11.1	3.60	11.86	0.42	4	4	4	4	4	12
148	750138	62	Y	2	2	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	170	72.0	24.9	1.22	3	+	-	-	-	-	99	7.4	160	36	131	41	22.00	1.30	14	20	A	A	12.2	-	-	-	1	1	1	1	5	25
149	1E+06	52	Y	1	1	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	165	69.0	25.3	1.25	3	+	-	-	-	-	107	8.9	182	106	98	26	32.00	0.90	14	26	A	A	13.6	-	-	-	1	1	1	1	5	25
150	163452	44	Y	1	2	3	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	170	75.5	26.1	0.94	2	+	-	-	-	-	120	9.3	210	251	39	99	32.00	0.90	14	28	-	-	10.8	-	-	-	1	1	1	1	4	25
151	2E+06	59	Y	1	2	3	+	-	-	9	1	-	-	-	-	-	-	-	-	-	-	-	157	71.0	28.8	1.11	3	+	-	-	-	-	99	6.4	190	140	128	34	22.00	0.80	18	32	-	-	11.8	4.20	13.26	0.40	2	2	2	2	5	21
152	169802	65	Y	1	2	3	+	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	152	65.0	28.1	1.08	3	+	-	-	-	-	135	6.8	92	81	42	33	22.00	1.00	25	56	-	-	12.8	5.40	18.42	1.76	1	1	1	1	5	25
153	906618	48	Y	1	2	2	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	165	45.0	16.5	1.00	1	+	-	-	-	-	247	9.0	214	182	76	32	12.00	0.80	28	46	-	T	14.3	-	-	-	1	1	1	1	5	25
154	614489	71	Y	0	2	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	158	52.0	20.8	1.03	2	+	-	-	-	-	176	10.9	192	206	100	51	24.00	0.80	16	39	-	T	12.1	-	-	-	4	2	3	3	4	16
155	658298	49	Y	0	1	1	-	-	-	-	2	-	-	-	-	-	-	-	-	+	-	-	162	58.0	22.1	1.01	2	+	-	-	-	-	182	8.9	116	76	68	33	22.00	0.80	23	34	+	a	11.8	-	-	-	1	1	1	1	5	25
156	825469	58	Y	1	2	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	25	165	67.0	24.6	0.93	2	+	-	-	-	-	81	8.0	132	162	54	30	32.00	0.80	32	47	A	T	11.8	-	10.80	2.50	2	2	2	2	5	22
157	2E+06	37	Y	1	1	0	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	167	66.0	23.7	1.06	2	+	-	-	-	-	261	10.3	215	199	139	36	22.00	0.90	35	40	++	A	12.8	-	-	-	1	1	1	1	5	9
158	2E+06	68	Y	1	2	2	-	-	-	-	2	-	-	-	-	-	-	-	-	-	10	3	166	55.0	20.0	0.97	2	+	-	-	-	-	274	7.6	167	123	117	25	32.00	0.80	11	26	-	-	9.8	3.60	11.08	2.16	4	2	2	3	5	18
159	2E+06	59	Y	0	2	2	+	-	-	6	-	+	-	+	-	-	-	+	+	+	-	-	163	81.0	30.5	0.95	4	+	-	-	-	-	309	12.4	74	152	25	19	26.00	1.60	14	33	A	A	11.6	5.50	3.85	0.32	2	2	2	2	4	20
160	2E+06	62	Y	0	2	3	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	178	68.0	21.5	1.26	2	+	-	-	-	-	119	9.8	152	77	122	45	23.00	1.05	22	23	A	A	11.1	-	-	1.05	1	1	1	1	5	25
161	215715	65	Y	1	2	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	30	165	62.0	22.8	0.95	2	+	-	-	-	-	139	6.7	185	87	132	37	20.00	0.80	20	32	A	A	12.1	-	6.50	1.13	1	1	1	1	5	25
162	2E+06	56	Y	1	2	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	160	59.0	23.0	0.95	2	+	-	-	-	-	104	7.1	185	169	116	35	32.00	1.30	28	39	A	A	10.1	-	-	-	1	1	1	1	5	25
163	768152	63	Y	1	1	3	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	163	60.0	22.6	0.92	2	+	-	-	-	-	321	12.5	157	176	91	30	20.00	0.90	20	35	A	T	13.7	-	-	-	1	1	1	1	5	25
164	873388	67	Y	1	2	3	-	-	-	9	2	-	-	-	-	-	-	-	-	-	-	-	173	73.0	24.4	1.00	2	+	-	-	-	-	281	8.9	116	76	68	33	22.00	0.80	23	34	+	a	11.8	-	-	-	1	1	1	1	5	25
165	2E+06	75	Y	1	2	3	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	154	58.0	24.5	1.04	2	+	-	-	-	-	137	9.8	207	93	158	30	23.00	0.90	18	44	A	A	12.8	-	10.50	2.70	1	1	1	1	5	25
166	2E+06	42	Y	1	2	3	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	175	75.0	24.5	1.08	2	+	-	-	-	-	282	15.7	204	183	126	41	32.00	1.10	38	23	A	A	13.2	-	12.70	1.80	1	1	1	1	5	25
167	2E+06	77	Y	1	2	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	10	-	160	50.0	19.5	0.91	2	+	-	-	-	-	135	8.8	136	78	47	73	32.00	1.00	35	40	A	A	12.8	-	9.80	2.37	3	2	3	3	4	17
168	2E+06	52	Y	1	1	0	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	168	65.0	23.0	0.98	2	+	-	-	-	-	171	101.1	236	181	156	44	23.00	0.90	23	47	A	A	11.8	-	6.70	2.46	1	1	1	1	5	25
169	793450	55	Y	0	1	2	-	-	-	-	2	-	-	-	-	-	-	-	-	-	15	15	169	63.0	22.1	1.03	2	+	-	-	-	-	180	8.0	155	100	99	36	18.00	0.80	32	23	-	-	13.2	-	-	-	1	1	1	1	5	25
170	788469	63	Y	1	1	1	-	-	-	10	2	-	-	-	-	-	-	-	-	-	-	-	173	81.0	27.1	1.40	3	+	-	-	-	-	529	12.7	127	184	59	31	64.00	1.90	32	58	++	-	15.9	-	-	-	1	1	1	1	5	25
171	2E+06	50	Y	1	2	1	-	-	-	8	2	-	-	-	-	-	-	-	-	-	-	-	167	61.0	21.9	0.97	2	+	-	-	-	-	142	11.3	160	74	111	34	8.00	22.00	20	39	++	A	12.6	-	10.00	2.03	1	1	1	1	5	25
172	775438	58	Y	0	2	1	-	-	-	11	-	-	-	-	-	-	-	-	-	-	15	-	168	95.0	33.7	0.90	3	+	-	-	-	-	176	8.8	136	112	156	44	20.00	0.70	37	58	-	-	13.8	-	-	-	1	1	1	1	5	25
173	2E+06	71	Y	0	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	162	95.0	36.2	0.94	5	+	-	-	-	-	181	8.5	167	111	100	45	22.00	0.90	35	49	-	-	14.8	-	-	-	1	1	1	1	5	25
174	2E+06	50	Y	2	1	1	-	-	-	-	2	-	-	-	-	-	-	-	-	+	-	-	162	68.0	25.9	0.92	2	+	-	-	-	-	132	9.8	155	189	99	36	28.00	0.70	52	86	-	-	13.2	-	-	-	1	1	1	1	5	25
175	2E+06	52	Y	0	1	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	166	81.0	29.4	0.93	3	+	-	-	-	-	222	9.8	132	176	62	36	32.00	0.60	30	46	-	-	11.8	-	-	-	1	1	1	1	5	25
176	2E+06	55	Y	2	2	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	178	68.0	21.5	0.94	2	+	-	-	-	-	133	8.0	155	100	99	36	18.00	0.80	32	23	-	-	13.2	-	-	-	1	1	1	1	5	25
177	1E+06	62	Y	1	2	3	-	-	-	5	2	-	-	-	-	-	-	-	-	-	-	-	159	65.0	25.7	1.27	3	+	-	-	-	-	94	7.4	130	60	79	39	23.00	0.80	12	23	A	A	11.8	-	23.20	0.86	3	3	4	4	4	14
178	1E+06	42	Y	1	2	3	-	-	-	1	2	-	-	-	-	-	-	-	-	-	-	-	177	65.0	20.7	0.86	2	+	-	-	-	-	196	11.9	176	76	130	29	22.00	0.90	23	31	A	A	12.1	18.20	3.08	0.38	4	4	4	4	3	11
179	2E+06	48	Y	1	2	2	+	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	178	82.0	25.9	0.95	3	+	-	-	-	-	193	11.8	182	88	135	29	23.00	0.90	14	22	A	A	14.8	-	-	-	1	1	1	1	5	25
180	1E+06	54	Y	1	2	3	-	-	-	3	-	-	-	-	-	-	-	-	-	-	-	-	157	67.0	27.2	0.94	3	+	-	-	-	-	100	7.7	154	143	88	37	12.00	0.90	18	32	A	A	10.8	-	11.80	2.76	2	2	2	2	5	20

ANNEXURE III - MASTER CHART

Serial Number	In patient number	Demo-graphy			DM history		History of other illnesses					Surgical history				Drug history						Risk factors		General physical examination						Genital examination				Investigations																		Questionnaire						
		Age (Years)	Marital status	Educational status	Duration (Years)	Treatment	Cardiovascular disease	Neurological/psychiatric	Other endocrine disease	Hypertension (Years)	Family history of DM	History	Pelvic/spinal	Prostate	Other	5α reductase inhibitor	Antipsychotic/ Antidepressant drugs	History of reduced libido	Erectile dysfunction	Circumcision	Smoking (Years)	Alcohol consumption (Years)	Height (Cms)	Weight (Kgs)	Body mass index (Kg/m2)	Waist hip Ratio	Blood pressure	Peripheral pulses	Penile	Testis	Phimosis	Secondary sexual characteristics	Plasma glucose (FBS/RBS/PPBS)	HbA1c	Cholesterol	Triglycerides	Low density lopoprotein	High density lipoprotein	Urea	Creatinine	SGOT	SGPT	Urine routine	Urine microscopy	Haemoglobin	Thyroid function T3	Thyroid function T4	Thyroid function TSH	Q1	Q2	Q3	Q4	Q5	Total score
181	721881	80	Y	1	2	1	-	-	-	16	1	-	-	-	-	-	-	-	-	-	12	15	169	79.0	27.7	1.08	3	+	-	-	-	-	265	10.8	142	138	83	31	32.00	1.80	12	26	A	A	10.2	-	-	-	0	0	0	0	0	0
182	789561	58	Y	1	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	172	65.0	22.0	1.30	2	+	-	-	-	-	160	8.8	150	120	92	52	20.00	1.60	36	28	-	-	10.4	-	-	-	1	1	1	1	5	25
183	810951	46	Y	2	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	159	68.0	26.9	1.18	2	+	-	-	-	-	142	9.0	214	182	76	32	12.00	0.80	28	46	-	-	14.3	-	-	-	1	1	1	1	5	25
184	1E+06	52	Y	1	2	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	162	64.0	24.4	1.20	3	+	-	-	-	-	156	10.8	208	228	140	31	34.00	1.00	18	32	-	-	12.8	-	-	-	3	2	3	2	4	18
185	1E+06	50	Y	1	1	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	164	70.0	26.0	1.01	2	+	-	-	-	-	169	8.6	220	180	120	40	14.00	1.10	18	36	-	-	11.8	-	-	-	1	1	1	1	5	25
186	1E+06	53	Y	1	2	3	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	170	66.0	22.8	1.09	2	+	-	-	-	-	220	8.9	182	106	98	26	27.00	0.80	31	30	-	-	9.9	-	-	-	1	1	1	1	5	25
187	1E+06	54	Y	2	1	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	12	-	173	69.0	23.1	0.98	3	+	-	-	-	-	190	9.9	108	110	56	30	32.00	0.80	18	22	-	-	11.2	-	-	-	1	1	1	1	5	25
188	484395	54	Y	1	1	1	-	-	-	10	-	-	-	-	-	-	-	-	-	-	30	-	166	90.0	32.7	0.90	4	+	-	-	-	-	188	15.9	150	227	70	70	26.00	0.90	19	35	-	-	11.8	-	20.18	2.36	4	3	3	3	4	15
189	498747	68	Y	1	2	2	+	-	-	2	-	+	-	+	-	-	-	+	+	-	-	-	166	69.5	25.2	0.89	3	+	-	-	-	-	118	8.0	136	171	96	36	22.00	0.80	19	43	-	-	10.8	-	10.26	1.04	0	0	0	0	0	0
190	500059	42	Y	1	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	172	98.0	33.1	1.08	3	+	-	-	-	-	248	8.3	170	219	82	37	18.00	2.00	17	37	-	-	13.6	-	6.80	1.05	1	1	1	1	5	25
191	497566	60	Y	1	2	2	-	-	-	-	2	+	-	+	-	-	-	+	+	-	-	-	160	70.0	27.3	1.08	3	+	-	-	-	-	148	8.4	170	209	90	38	29.00	0.90	19	34	-	-	14.4	-	-	-	1	1	1	1	5	25
192	456588	70	Y	1	3	3	+	-	-	-	-	+	-	+	-	-	-	+	+	-	-	30	170	75.0	26.0	0.97	3	+	-	-	-	-	204	9.8	167	111	100	45	20.00	1.20	20	30	-	-	13.2	-	17.32	2.10	3	2	2	2	4	20
193	483449	71	Y	0	2	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	158	52.0	20.8	1.03	2	+	-	-	-	-	176	10.9	192	206	100	51	24.00	0.80	16	39	-	T	12.1	-	-	-	4	2	3	3	4	16
194	467425	49	Y	0	1	1	-	-	-	-	2	-	-	-	-	-	-	-	-	+	-	-	162	58.0	22.1	1.01	2	+	-	-	-	-	182	8.9	116	76	68	33	22.00	0.80	23	34	+	a	11.8	-	-	-	1	1	1	1	5	25
195	471519	58	Y	1	2	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	25	165	67.0	24.6	0.93	2	+	-	-	-	-	81	8.0	132	162	54	30	32.00	0.80	32	47	A	T	11.8	-	10.80	2.50	2	2	2	2	5	22
196	477659	37	Y	1	1	0	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	167	66.0	23.7	1.06	2	+	-	-	-	-	261	10.3	215	199	139	36	22.00	0.90	35	40	++	A	12.8	-	-	-	1	1	1	1	5	9
197	463304	68	Y	1	2	2	-	-	-	-	2	-	-	-	-	-	-	-	-	-	10	3	166	55.0	20.0	0.97	2	+	-	-	-	-	274	7.6	167	123	117	25	32.00	0.80	11	26	-	-	9.8	3.60	11.08	2.16	4	2	2	3	5	18
198	461940	59	Y	0	2	2	+	-	-	6	-	+	-	+	-	-	-	+	+	+	-	-	163	81.0	30.5	0.95	4	+	-	-	-	-	309	12.4	74	152	25	19	26.00	1.60	14	33	A	A	11.6	5.50	3.85	0.32	2	2	2	2	4	20
199	479521	62	Y	0	2	3	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	178	68.0	21.5	1.26	2	+	-	-	-	-	119	9.8	152	77	122	45	23.00	1.05	22	23	A	A	11.1	-	-	1.05	1	1	1	1	5	25
200	478429	65	Y	1	2	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	30	165	62.0	22.8	0.95	2	+	-	-	-	-	139	6.7	185	87	132	37	20.00	0.80	20	32	A	A	12.1	-	6.50	1.13	1	1	1	1	5	25
201	478024	56	Y	1	2	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	160	59.0	23.0	0.95	2	+	-	-	-	-	104	7.1	185	169	116	35	32.00	1.30	28	39	A	A	10.1	-	-	-	1	1	1	1	5	25
202	480914	63	Y	1	1	3	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	163	60.0	22.6	0.92	2	+	-	-	-	-	321	12.5	157	176	91	30	20.00	0.90	20	35	A	T	13.7	-	-	-	1	1	1	1	5	25
203	483715	67	Y	1	2	3	-	-	-	9	2	-	-	-	-	-	-	-	-	-	-	-	173	73.0	24.4	1.00	2	+	-	-	-	-	281	8.9	116	76	68	33	22.00	0.80	23	34	+	a	11.8	-	-	-	1	1	1	1	5	25
204	481669	60	Y	3	2	0	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	171	59.0	20.2	0.98	2	+	-	-	-	-	180	9.8	160	138	98	56	16.00	0.80	45	18	-	-	12.0	-	-	-	1	1	1	1	5	25
205	484035	65	Y	3	2	2	-	-	-	15	2	-	-	-	-	-	-	-	-	-	-	-	170	72.0	24.9	1.22	2	+	-	-	-	-	109	10.6	160	136	130	41	28.00	1.00	21	20	-	-	11.0	-	-	-	2	2	2	2	4	20
206	476722	68	Y	3	2	2	-	-	-	11	-	-	-	-	-	-	-	-	-	-	-	-	175	73.0	23.8	0.94	2	+	-	-	-	-	100	7.8	118	98	94	52	18.00	1.20	42	15	-	-	10.7	-	-	-	1	1	1	1	5	25
207	479429	60	Y	3	2	1	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	168	72.0	25.5	1.08	3	+	-	-	-	-	133	8.6	136	112	86	68	22.00	0.80	23	28	-	-	11.8	-	-	-	1	1	1	1	5	25
208	477429	49	Y	1	2	2	+	-	-	8	-	-	-	-	-	-	-	-	-	-	15	-	166	90.0	32.7	0.90	4	+	-	-	-	-	188	15.9	138	227	68	72	26.00	0.90	19	35	-	-	11.8	-	-	-	4	3	3	3	4	15

Annexures

Annexure III

ANNEXURE III – KEY TO MASTER CHART

-	-	Absent
+	-	Present
A	-	Absent
Cms	-	Centimeters
DM	-	Diabetes mellitus
HbA1c	-	Glycated haemoglobin
Kgs	-	Kilograms
N	-	No
Q	-	Question
SGOT	-	Serum glutamic oxaloacetic transaminase
SGPT	-	Serum glutamic-pyruvic transaminase
T	-	Traces
TSH	-	Thyroid stimulating hormone
Y	-	Yes