"CLINICAL STUDY OF TYPE 2 DIABETES MELLITUS AT PRESENTATION AND ITS RELATION TO GLYCEMIC STATUS"

ABSTRACT

Background and objectives: Type 2 Diabetes is the commonest type of diabetes In India. India accounts for around 50 % of the world's diabetes burden, with approximately 70 million cases in 2017, a figure which is expected to double by 2020. This is the tip of an iceberg suggesting that the submerged portion of the iceberg would be much bigger.

The total sum of people sustaining with diabetes & yet undiagnosed or asymptomatic or untreated is very high. Their presentation modes and the complications with which they present varies considerably. By the time of diagnosis their glycemic level is much higher and are prone to have long-standing hyperglycemia.

Objectives: To be familiar with the prevalence of various patterns of complications and presentations at the time of initial diagnosis of Type 2 diabetes mellitus.

Methods: Based on the World Health Organization criteria a total of 200 patients with Type 2 Diabetes Mellitus are included in the Study. The complications with which they presented was noted in the study. Patients with the history of acute infection, Type 1 DM, pregnant women, MODY, syndromic diabetes, patients on steroids, beta blockers and diuretics were excluded from the study.

Results: Among 100 patients who were enrolled, mean age at presentation with Type 2 D.M was 50.93 ± 10.78. Sex ratio in the study i.e. male: female population

ratio in the study is 1.7:1. Family history was noted in 29% of patients. 24.22 ± 4.36kg/m2 is the mean BMI noted in the study. Waist-hip ratio was 0.936 ± 0.091.

In the study, most common complication with which they presented was retinopathy which constituted 33%, succeeded by infection 12% & nephropathy

constituted 10%. Ischemic heart disease was noted in 6 % of the population. The mean HbA1c was 9.27%. FBS and PPBS in this study were 232.72 ± 92.7 mg/dl and ± 97.58 mg/dl respectively. Low density lipoprotein and Triglycerides abnormalities were found in 45.67% and 25.04% respectively.

Interpretation and conclusion: The likelihood ratio of diabetes steeps after an age of 35 years, along with increased incidence of its complications. This risk of complications increases with late detection of diabetes. Early diagnosis assists in treating the disease promptly and also help in reducing the mortality due to complications. However, in reality, true incidence of the disease burden may be much high as this study represents only the tip of an iceberg.

However, Need of the hour is an effective screening approach and efficient management to decrease the burden of complications once they are detected.

Key words: DM: Diabetes Mellitus, FBS: Fasting Blood Sugar

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INTRODUCTION

From the past 3000 yrs Diabetes - a disease of antiquity is familiar to mankind. The characteristics of Diabetes were first explained by ancient Egypt, though the term “diabetes” was first invented in the 1st century by Aratus of Cappadocia. The word mellitus was added by a British physician by name Thomas wills in 16th century, which means honey sweet after rediscovering that the blood and urine were sweet which was earlier very popular in ancient India. At present, it is the most widespread metabolic & non-communicable disease.

Currently, it"s raising trend, gained epidemic status worldwide. It is a syndromic disease characterized by elevated blood sugar (hyperglycemia) which in turn is associated with disturbances in the metabolism of carbohydrates, protein, fats which in turn correlates with relative or absolute deficiencies in the secretion of insulin and action of insulin. This contributes to acquired insulin resistance among the population. At this rate, India would be the global diabetes capital by the year 2030. As of now the estimated prevalence in India is 8.7% among the age group of 20-70 yrs.

In India the most prevailing form of diabetes is Type2 DM. Patients with Type 2 Diabetes amount for >95% of the diabetic population[1]. Because of its prolonged asymptomatic course, patients prevail undiagnosed for many years. In many individuals who are newly diagnosed or undiagnosed to type 2 Diabetes Mellitus the Microvascular disease gets established.

Incidence of Diabetes has a very strong relationship with hyperglycemic status, that is, reduced fasting glucose and reduced glucose

tolerance with other independent factors such as family history, age, sex, WHR, BMI, blood pressure and serum lipid levels; none taken singly, is as good as measuring blood glucose levels at discriminating who will progress to Diabetes Mellitus.

The main cause for damage to different organs like eyes, renal, nervous and cardiovascular systems in the long term is due to long-standing unrelated hyperglycemia Undiagnosed individuals with diabetes are also significantly at risk for development of Stroke, CAD and Peripheral Arterial Disease than non Diabetic population along with greater Likelihood ratio of having dyslipidemia, hypertension, and obesity which are the outcomes of metabolic syndrome.

Early detection along with timely treatment would reduce the weight of diabetes and also its complications. Thus screening for type 2 diabetes is highly recommended in all health care institutions[2].

The current study was performed to make clear the occurrence of diabetes and its complications in a tertiary hospital in a southern part of India to understand the magnitude of disease burden confronting us. Development of Diabetes Mellitus cannot have a mutual relationship.

However, the probability of developing Diabetes Mellitus would be greater if the number of risk factors increases. Conversely, the probability of developing diabetes in an asymptomatic individual without any risk factors is very minimal.

OBJECTIVES

a. To study various patterns of presentation of type 2 Diabetes Mellitus.

b. To access their glycemic status at presentation.

c. To assess the frequency and severity of complications about age at onset of NIDDM.

d. To determine the prevalence of obesity and a family history of type 2 Diabetes.

e. To compare the complication profile of Type 2 Diabetes Mellitus with previous studies.

REVIEW OF LITERATURE

HISTORY

About 2000 years ago itself diabetes was described. The term "diabetes" was obtained from the Greek language, and it means to "run through a siphon." The term was coined by ARISTAEUS who noted that an extremely large volume of urine ran through the kidneys in this disease. He described it as consisting of a moist and cold wasting of flesh and limbs into urine from a cause similar to dropsy, as the secretion passes in the usual way, by the kidney and the bladder. The patient never ceases making water, but the discharge is incessant as a sluice let off[3].

A disease involving the passage of large amount of "sweet urine" was known to Chinese physicians in the 2nd and 3rd century. Ancient text books of the 5th and 6th centuries referred to a condition involving the passage of "honey urine"[4].

The diagnostic period of the disease began in the 17th century when Thomas first separated diabetes mellitus from diabetes insipidus. A century later Dobson hypothesized that before sugar appeared in urine, its level in the blood rises considerably beyond the normal limits. In 1776 Dobson showed that diabetics excrete sugar in the urine and this threw fresh light on the etiology of diabetes as until then kidney was thought to be the organ affected.

Cawley was the first to report a relation between diabetes and pancreas[5]. Bouchard and Lencereause were the first to identify at least two types of diabetes clinically. The severe type was in young, which responds

poorly to diet regimen and in which autopsy showed pancreatic findings. The other type was in the older obese in whom diet therapy was useful, and who had no evidence of pancreatic involvement at autopsy.

So diabetes of the thin was labelled as „diabetes maigre‟ and of the obese was labelled as “diabetes grass.” Diabetes maigre was thought to be due to pancreatic aetiology[6].

Paul Langerhans a medical student published a paper in 1869 on pancreatic history in which he described an unknown cell type in the pancreas which occur as islands. Langues designated these cells the term “islets of Langerhans‟ 20 years later[7].

Leen de Meyer in 1910 suggested that the pancreatic secretion that was lacking in the diabetics should when focused be called insulin. In the late 19th century and early 20th century research was focused on isolating insulin which could be used therapeutically.

Frederick Banting, an operating surgeon and Charles Best, a medical student with the help of J.Bollip, chemist were the first to produce insulin which could be used successfully in treating diabetes[8,9].

IN 1952, the various purified and modified version of insulin was available. By 1982, recombinant DNA insulin became available.

EPIDEMIOLOGY

Diabetes is world - wide in distribution. Diabetes affects approximately 5% of the United States population [10,11.] Prevalence in Britain is 1-2%. However, almost half of the population with diabetes mellitus remains undetected. Type 2DM is the commonest form of diabetes seen all over the world[12]. `

According to WHO, the world wide frequency of diabetes among all age groups was estimated to be 2.8% in 2000 and is predicted to be 4.4% by 2030[13]. More than 200 million diabetics were predictable within the next ten years. The countries with largest numbers of diabetics are India, China, and US[14]. The occurrence of diabetes is increasing in developing countries. The population, particularly at high risk, are those making the transition from rural to the urban environment and those who are adapting westernized life style and behaviours[15].

Asian Indians constitute for a very high number of type 2 diabetics probably due to the high propensity of insulin resistance[16].

WHO has foretold that India would have the biggest increase, (49% increase in total population and 169% increase in population with >65 years of age). About Over 30 million Indians are affected with diabetes & this is just the tip of an ice-berg[17]. So India can be noted as the diabetes capital of the world.

In urban areas, the occurrences of diabetes are twice to when compared to the rural population. It was predicted that the percentage of people suffering from diabetes in urban areas would escalate from 54% in 1995 to 73% by 2025 [17,18].

When compared to the west, among Indians more number of younger individuals are affected with type 2 DM. The Peak age of DM in India is mostly the productive years of life and, and hence the chances of chronic complications are higher in India[13,14].

Indians have a genetic constitution characterized by low body mass index, however with high upper-body adiposeness, and high level of insulin resistance [20].

Definition:

DM is a syndrome comprising of metabolic, vascular and neuropathic component that are interrelated. The defects in insulin secretion, insulin action or both are the main causes of Hyperglycemia. This leads to alterations in the metabolism of carbohydrate, fat, and protein.

Diabetes can present with features suggestive of hyperglycemia such as polyuria, polydipsia, polyphagia, weight loss, or it may present as one of the acute or chronic complications or it can also be detected incidentally in hospitalized patients. Diabetes can be seen intermittently as in pregnancy.

Classification:

Diabetes is the most common Metabolic and non-communicable disorder. But there is a lot of variation in the manifestations, complications, management, and genetics. This has led the epidemiological agencies to put forth varieties of classifications for this syndrome of hyperglycemia.

In the beginning, The National Diabetes Data Group (NDDG), of United States has given a widely acceptable classification of DM. This formed the basis for WHO classification in 1980 and was later modified in 1985[21].

The WHO classification of diabetes mellitus and allied categories of glucose intolerance[22]. Is as follows :

A) CLINICAL CLASSES

1) DIABETES MELLITUS

a. Insulin dependent diabetes mellitus

b. Non insulin dependent diabetes mellitus

- Non obese
- Obese

c. Malnutrition related diabetes mellitus

- Protein deficient Diabetes mellitus (PDDM)
- Fibro calculus pancreatic diabetes mellitus (FCPD)

d. Other types of diabetes mellitus associated with certain condition and syndrome

- Pancreatic disease
- Diseases of hormonal etiology
- Drug or chemical induced condition
- Abnormalities of insulin or the receptors
- Certain genetic syndromes
- Miscellaneous

2) IMPAIRED GLUCOSE TOLERANCE

3) GESTATIONAL DIABETES MELLITUS

B) STATISTICAL RISK CLASSES (Normal glucose intolerance but substantially increased risk of developing diabetes)

1) Previous abnormality of glucose tolerance

2) Potential abnormality of glucose tolerance

Revised classification suggested by the expert committee on the diagnosis and classification of diabetes constituted by ADA and the WHO is given below[21,23,24,25,26].

ETIOLOGICAL CLASSIFICATION OF DIABETES MELLITUS :

1. Type 1 diabetes (β - cell destruction, usually leading to absolute insulin deficiency).
   a. Immune- mediated
   b. Idiopathic
2. Type 2 diabetes (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly insulin secretory defect with insulin resistance).
3. Other specific types
   a. Genetic defects of β - cell function.
      i. Mitochondrial DNA defect
      ii. Wolfram"s syndrome
      iii. Maturity- onset diabetes of the young (MoDY)
         1. Chromosome 20q, HNF- 4α (MoDY1)

2. Chromosome 7, glucokinase (MoDY2)
3. Chromosome 12q, HNF- 1 α (MoDY3)
4. Insulin promoter factor (IDP)1, (MoDY4)
5. HNF- 1 α (MoDY5)
6. Neuro D1 (MoDY6)

b. Genetic defects in insulin action
   i. Type A insulin resistance
   ii. Leprechaunism
   iii. Rabson – Mendenhall syndrome
   iv. Lipodystrophy syndromes

c. Diseases of the exocrine pancreas
   i. Pancreatitis
   ii. Trauma / Pancreatectomy
   iii. Neoplasia
   iv. Cystic fibrosis
   v. Hemochromatosis
   vi. Fibrocalculus pancreatopathy

d. Endocrinopathies
   i. Acromegaly
   ii. Cushings syndrome
   iii. Glucagonoma
   iv. Pheochromocytoma
   v. Hyperthyroidism
   vi. Somatostatinomas

vii. Aldosteronomas

e. Drug or chemical induced

i. Vacor

ii. Pentamidine

iii. Nicotinic acid

iv. Glucocorticoids

v. Thyroid hormones

vi. Diazoxide

vii. α - adrenergic agonist

viii. Thiazides

ix. Dilantin

x. Alpha interferon

f. Infections

i. Congenital rubella

ii. Cytomegalovirus

iii. Coxsackie- B

g. Uncommon forms of immune-mediated diabetes

i. Stiff man syndrome

ii. Anti-insulin receptor syndrome

h. Other genetic syndromes sometimes associated with diabetes

i. Downs syndrome

ii. Klinefelter syndrome

iii. Turner syndrome

iv. Wolfram syndrome

v. Friedrich"s ataxia

vi. Huntingtons chorea

vii. Laurence moon bill syndrome

viii. Myotonic dystrophy

ix. Porphyria

x. Prader – Willi syndrome

xi. Others

4. Gestational diabetes mellitus (GDM)

Type 2 DM is mostly familial. Genetic propensity for type 2 DM is suggested by a very high rate of concordance (80%) among the monozygotic twins. Inheritance of diabetes is polygenic. Offspring and siblings of type 2 diabetics have a greater risk of inheritance when compared to that of type 1 diabetics. 40% of the siblings with non-insulin dependent diabetes mellitus sufferers might develop diabetes, assuming a maximum expectancy of 80 years[27] even though the recent studies have estimated the concordance to be about 60%[28].

One study[29] has stated that 62% of patients with type 2 diabetes have had a family history, of these 53% had first degree relatives with diabetes. It also reported the incidence of diabetes in children with one diabetic parent is 36% and 73% if both parents are diabetic.

Vishwanathan et al. reported that 50% of progeny from type 2 DM parents have overt diabetes and 30% of diabetic progeny attained the disease before 40 years age[30].

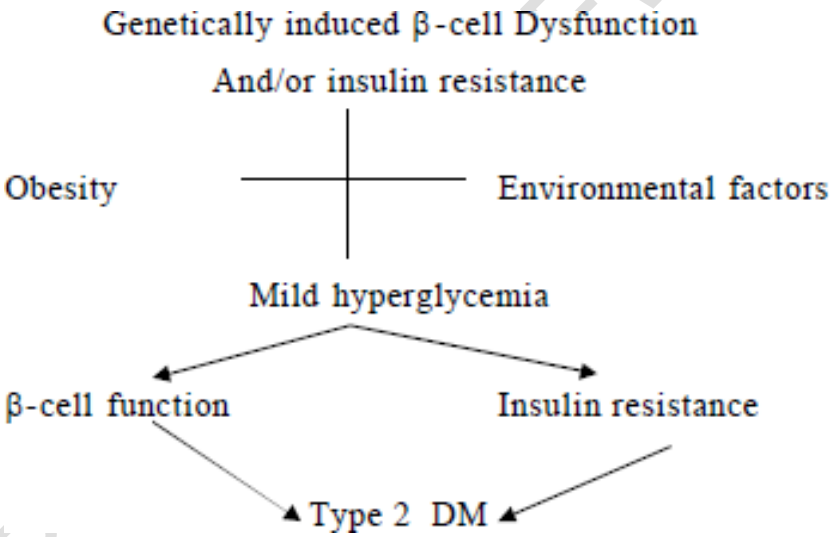
PATHOPHYSIOLOGY OF TYPE 2 DM :

Hyperglycemia is attributable to both defective beta cell function and insulin resistance.[31]

Type two Diabetes is characterized by three pathophysiologic abnormalities

a. impaired insulin production
b. peripheral insulin resistance
c. excessive hepatic glucose production.

Fig 1: Schematic representation of type 2 DM pathophysiology by American Diabetes Association is shown below[32].



Insulin resistance:

The decreased ability of insulin to act efficiently on peripheral target tissues (especially muscle and liver) is the outstanding feature of type two DM which results from a mixture of the genetic composition of an individual and obesity central or visceral. This was proven by the waist - hip ratio.

Adipocytes produce a mixture of biological substances such as leptin, TNF α, free fatty acids and adiponectin. They regulate the secretion of

insulin, action of insulin on peripheral tissues & also the body weight that accounts for the insulin resistance. When the amount of insulin production is not able to make up for the level of insulin resistance, then hyperglycemia will occur[32,33,34].

Impaired insulin secretion:

There is a strong correlation between Insulin sensitivity & insulin production. Insulin production would increase during the initial stages in response to insulin resistance to keep up with glucose tolerance. In the initial stages defect in insulin production is minimal & it is mostly the glucose – triggered insulin secretion.

In due course of time the secretory defect of insulin advances to a state of gross inadequacy. Insulin production increases gradually as the glucose tolerance rises to about 140 mg%, after which there is a further increment in glucose levels which causes a decrease in insulin production. This indicates a noxious effect of hyperglycemia on beta cell[32,34].

Increased hepatic glucose production:

The case of type 2 Diabetes, there is insulin resistance in the liver which leads to the defeat of hyper insulinemia to decrease gluconeogenesis. This results in fasting hyperglycemia and reduced capacity of the liver to store glycogen in the post-prandial condition.

Definition of Overweight:

- Body mass index more the 95 th percentile for respective age and sex.
- Waist- hip ratio higher the 95 th percentile.
- Body Weight higher than 20% of ideal weight for height.

Overweight children when they reach puberty are considered to be at risk for diabetes by the ADA if they satisfy 2 of the below- mentioned criteria[35].

1. Familial History of type 2 DM in first or second-degree relatives.
2. Race or ethnicity - American Indian / Alaska / Black / Hispanic/ Asian or Pacific Islanders.
3. Coexistence of conditions related to insulin resistance like Acanthosis Nigricans, Hypertension, Dyslipidaemia and polycystic ovarian disease.

Asmal et al[36] has reported that 10% of Indians living in South Africa with type 2 diabetes had an age of fewer than 35 years by the time they are diagnosed with diabetes.

Likewise Mohan et al[37] has stated that 18.5% of type 2 diabetics in South Africa had an age of fewer than 35 years by the time they are diagnosed with diabetes.

Biochemical mechanisms of tissue damage:

Most of the impact of long-standing chronic diabetes falls on the microcirculation

↓

Progressive narrowing of vessel lumen followed by eventual occlusion

↓

Impaired perfusion

↓

Tissue dysfunction due to Ischaemia

Earliest damage that occurs is an increase in vascular permeability and extravasation of plasma proteins such as IgG, albumin, and IgM that accumulate PAS - positive deposits on vessel the vessel wall and on numerous cells mainly pericytes, retinal cells, mesangial cells of glomerular capillaries which are the most vulnerable ones.

There occurs Proliferation of cells due to changes in the synthesis of component proteins and glycosaminoglycans leading to the thickening of basement the basement membrane.

- In the retinal capillaries and vasa nervosum
- The mesangial matrix of renal glomerulus.
- Analogous increase in collagen in developing plaques.

Specific tissues are targeted by hyperglycemia and can be largely attributed to the failure of those cells to down regulate their uptake of glucose when there is an elevation of extra cellular glucose concentration.

Mechanism:

1. Increase in the flux of polyol pathway: Sorbitol and galactitol pathway leading to the production of highly active glycated substances.
2. Increase in advanced glycated end products which impair structural, enzymatic and signaling functions of glycated proteins.
3. Increase in protein kinase C levels which favor interaction of advanced glycated end products to cell surface receptors, increase in expression of vascular endothelial growth factors and an increase in basement membrane thickness.

Pathway flux:

1. Increase in hexosamine biosynthesis pathway: Substrates which play a role in obesity and hyperglycemia induced insulin resistance.
2. Increased mitochondrial superoxide production.
3. The possible role of hyperglycemic memory.

Role of hyperglycemia:

As demonstrated by UKPDS (United Kingdom Prospective Diabetes Study) and DCCT (Diabetes Control and Complications Trial)

Microvascular complications are the most prevalent and the most important complications among individuals whose blood glucose levels are poorly regulated over prolonged periods with an exception only among some individuals with relatively mild hyperglycemia..Therefore, the individual's susceptibility to hyperglycemia encouraged that factors genetic diversity & hypertension leads to tissue damage.

Glycosylated hemoglobin (HbA1c) :

It Reflects the metabolic control over the prior 8-12 weeks. It is produced by the non-enzymatic covalent bond of glucose and other sugar component to haemoglobin.

Glycosylated Haemoglobin is a sequence of glycosylated alternatives where various carbohydrates are attached to the N-terminal end of valine in βchain of haemoglobin which is either fructose 1-6 diphosphate (HbA1a1) or glucose six phosphates (HbA1a2) or glucose (HbA1c) or an unknown sugar (Hba1b). Among these, the largest component is HbA1c (60-80%).

The Total glycosylated haemoglobin (Ghb) represents the glucose attached to both N-terminal end of valine and other sites such as the E-amino group of lysine, that won't alter the charge. The span of values for HbA1c is about 4.6%, and that for total glycosylated haemoglobin (GHb) is much higher at about 5-7.5%, however, normal ranges must be established by each laboratory. Glycosylated Hemoglobin reflects the state of body glucose levels over the preceding 8-12 weeks[38].

The sensitivity of HbA1c in detecting a known case of diabetes is only 85%. This shows that a diagnosis of diabetes cannot be ruled out by the presence of normal HbA1c levels. However, an elevated HbA1c is quite accurate to about 91% in identifying the existence of diabetes.

Table: 1 Reference ranges for different glycosylated Hemoglobin method

Method	Reference range %	Hb species measured
Affinity chromatography	4.0-7.7	Total glycated Hemoglobin
Electrophoresis	4.7-7.6	Hemoglobin A1
Immunologic	4.1-5.3	Hemoglobin A1c
Ion exchange	4.2-5.9	Hemoglobin A1c

Variations in HbA1C:

There is an important methodological problem that is, due to various reasons, the mentioned range for glycated hemoglobin and the value of HbA1C for a given blood sample could be interchangeable among laboratories[39]. This arises due to the different species measured, variations in reaction conditions, the lack of suitable standards and varying interference

with non-glucose adducts, particularly carbamylation in uraemia, and also penicillin, aspirin, and metabolites in alcoholism.

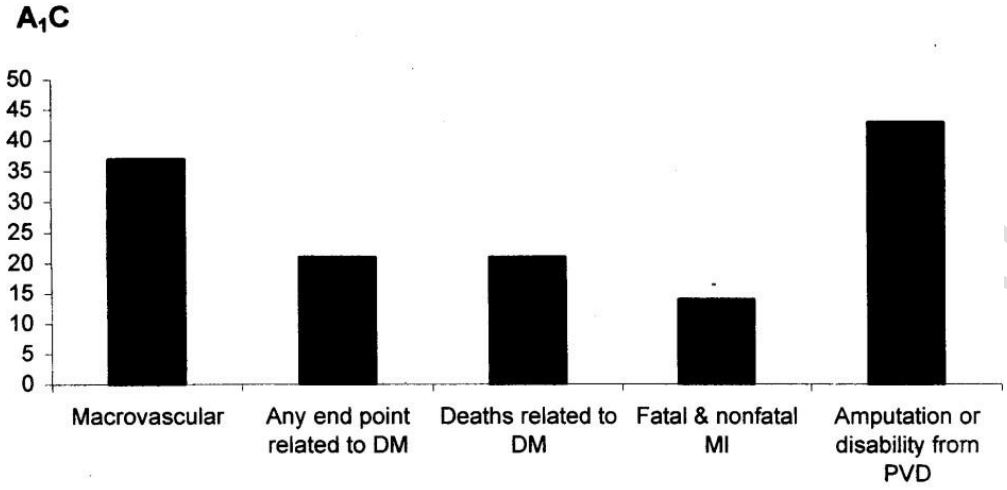
Hemoglobin variant can also cause problems[40]. HbF chromatographs with HbA, in some electrophoresis and ion-exchange methods, show falsely higher values, while HbS and HbC lower results because they coelute with HbA.

Thus, when an elevated occurrence of hemoglobinopathy is expected in a population, a method that is free from such interference should be chosen (i.e., affinity chromatography). There is a decrease in mean red-cell age which falsely lowers the GHb level in the patients who are having chronic blood loss, hemolytic anemia or pregnancy. Vitamin C and vitamin E are also known to lower GHb, perhaps by blocking the glycation[41]. Therefore, conditions such as hypertension & genetic diversity greatly influence the susceptibility of an individual to glucose- induced tissue damage.

Table 2: Good glycemic control reduces incidence of complications [42,43]

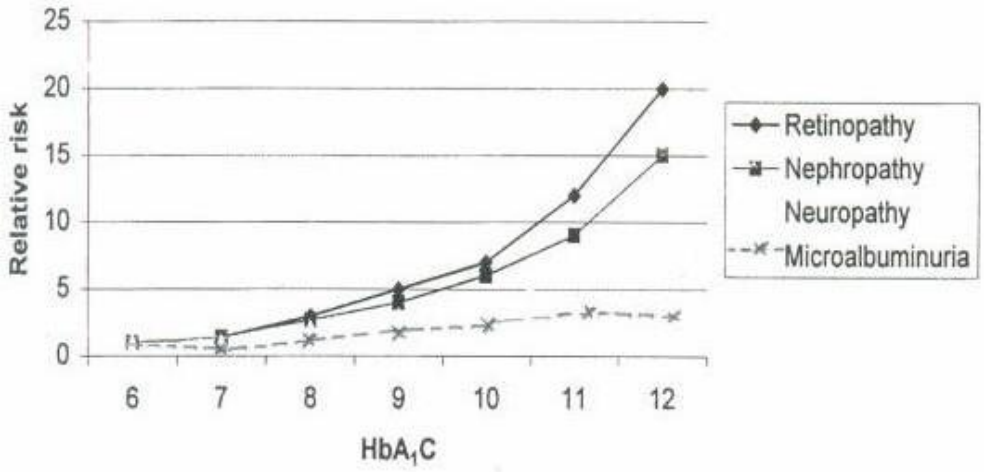
	Risk reduction by percentage decrease in HbA1c values		
	DCCT	Otikubo	UKPDS
Complication	9-7%	9-7%	8-7%
Retinopathy	63%	69%	21%
Nephropathy	54%	70%	34%
Neuroapthy	60%	-	-
Macrovascular disease	16%	-	41%

Fig 2: Risk reduction in complication per each 1% reduction in excess A1C[42,43]



United Kingdom Prospective Diabetic Study (UKPDS) Lancet,1998;352,837-853, BMJ 2000 321, 485.

Fig 3: HbA1C and relative risk of microvascular complications



The chance of acquiring diabetes in high-risk individuals can be better predicted by integrated use of fasting blood glucose levels and glycosylated Haemoglobin.

This has been proved in a study of 2977 Hong Kong Chinese individuals having various risk factors for glucose tolerance done in 1998 in order to assess the rationality of combining fasting blood glucose with HbA1c or fructosamine for detection of diabetes in high-risk individuals using WHO criteria[44].

ROLE OF ADIPOSE TISSUE IN DIABETES MELLITUS

Adipose tissue which was once regarded as irrelevant in various endocrinological disorders, is currently accepted as an important mediator of many physiologic processes like innate immune response, vascular remodelling and energy homeostasis[45]. Adiponectin & resistin which have been discovered in recent times, seem to have apposite effects in the glucose homeostasis of body [46]. Adiponectin increases the sensitivity of insulin by inhibiting the hepatic glucose output[47]. The circulating levels of minimal adiponectin are closely related to the individual's susceptibility to various metabolic disorders like. Obesity, hypertension & diabetes[48].

Hotta et al[49] has shown that a depressed serum adiponectin level in diabetic patients is linked to coronary artery disease. They also showed that serum adiponectin level has a negative relationship with body mass index (BMI), blood glucose, serum insulin & also serum triglycerides.They have also shown that there is a significant rise in circulating serum adiponectin levels from about 40 to 60% in both diabetic and non-diabetic individuals when there is a moderate weight loss, i.e., a fall in BMI by 10%. In addition to this, there is a remarkable decrease in BMI, serum insulin levels & fasting plasma glucose

levels were linked with an increase in serum adiponectin levels to about ~50% the pre-surgical levels and also increase in insulin sensitivity, Yang et al[50].

Decreased serum adiponectin levels result in dysfunctional insulin signalling. In vitro studies have confirmed that free fatty acids play an important role in regulating the hepatic glucose metabolism via increasing the activity of glucose- 6- phosphatase and other multiple glucogenic enzymes.

The risk of coronary artery disease was 3 to 4 times higher in diabetic patients irrespective of the cholesterol level. United Kingdom Prospective Diabetes(UKPDS) Study states that hyperglycemia, hypertension, increased low-density lipoprotein, cholesterol, low levels of high-density lipoprotein and smoking are the main risk factors for coronary artery disease in type 2 diabetes. Thus proposed the trigger of inflammation leading to increased risk of CAD[51].

Table 3: Showing proposed trigger and mediators of inflammation.

Proposed triggers	Significance	Mediators
Obesity	Predisposes to insulin resistance, diabetes and dyslipidemia	Free fatty acids, cytokinin from adipocytes (INF□and IL6).
Post prandial hyperglycemia	Augments proinflammatory cytokine levels and pathways in endothelium.	Advanced glycated end products.
Oxidized lipoproteins	Lipoproteins are more antigenic and trigger immune response	Increased adenosine molecule expression, proinflammatory cytokines and chemokines in macrophages and vascular wall.

LIPID ABNORMALITIES IN DIABETES:

It has been found that about 50% of all type 2 diabetics have Dyslipidemia & the most common pattern being elevated TGL & decreased HDL. Diabetes also produces a pro-coagulant state by increasing the Serum VLDL, LDL, triglycerides. APOE and lipoprotein while reducing the protective HDL. Lipoprotein is more atherogenic than LDL cholesterol & it is an independent risk factor for CDs AK from Pondicherry has shown that with rising levels of blood glucose there is a proportionate increase in total cholesterol and triglycerides[52].

METABOLIC SYNDROME: (Insulin resistance syndrome , syndrome X).

The word “syndrome‟ was obtained from the Greek language which means “to run together‟ of various conditions like hyperglycemia, hyperinsulinemia, dyslipidemia, and hypertension. So It is multifactorial.

Insulin resistance & Central obesity are closely linked to metabolic syndrome. Excess of adipokines & non-esterified fatty acids which are released into the blood leads to the accumulation of atopic fat in the abdominal wall, liver and muscle. This in turn, accounts for the pro-inflammatory state, dyslipidemia, insulin resistance, and pro-thrombotic state. Sedentary life style, hormonal imbalance & increasing age in association with genetic predisposition lead to the development of metabolic syndrome

. The definition of metabolic syndrome was stated by International Diabetes Federation (IDF) in the year 2006:

To define it as metabolic syndrome one should have Central obesity

(waist circumference of 94 cms in Europid men and 80 cms in Europid women) In addition to 2 of the below mentioned four factors:

1) Elevated Triglyceride level : >/= 150 mg/dl or on treatment for this dyslipidaemia .
2) Reduced High-density lipoprotein : to < 40 mg/dl in men and < 50 mg/dl in women or on treatment for this hyperlipidaemia
3) Elevated Blood pressure : systolic BP >/= 130 or diastolic BP >/= 85 mmHg, or treatment of previously detected high blood pressure.
4) Elevated fasting blood glucose : >/=100mg/dl or previously diagnosed type 2 diabetes.

Clinical Identifications of Insulin resistance syndrome:

Problems with NCEP, ATP III definition in Asian Indians

Risk factors	Defining level
Abdominal obesity (waist circumference)	
Men	> 86 cm
Women	> 80 cm
Triglycerides	> 150 mg/dl
High density lipoprotein cholestrol	
Men	< 40 mg/dl
Women	< 50 mg/dl
Blood pressure	> 130/85 mm of Hg
Fasting glucose	> 110 mg/dl

DIAGNOSTIC BASIS FOR DIABETES MELLITUS:

Expert panel under American Diabetes Association has modified the diagnostic basis for Diabetes Mellitus is recommended by NDDG and WHO. As per the report, It was concluded that DM can be confirmed in 3 ways.

1. Random plasma glucose level of >/=200mg/dl associated with symptoms of Diabetes-like polydipsia, polyuria, and unexplained weight loss.
2. Fasting plasma glucose of >/=126 mg/dl (fasting is no caloric intake for minimum 8 hours).
3. 2-hour post prandial glucose >/ = 200mg/dl during an oral glucose tolerance test using a 75 gm of anhydrous glucose in water as advised by WHO.

The expert committee recommends the classification of glucose tolerance as

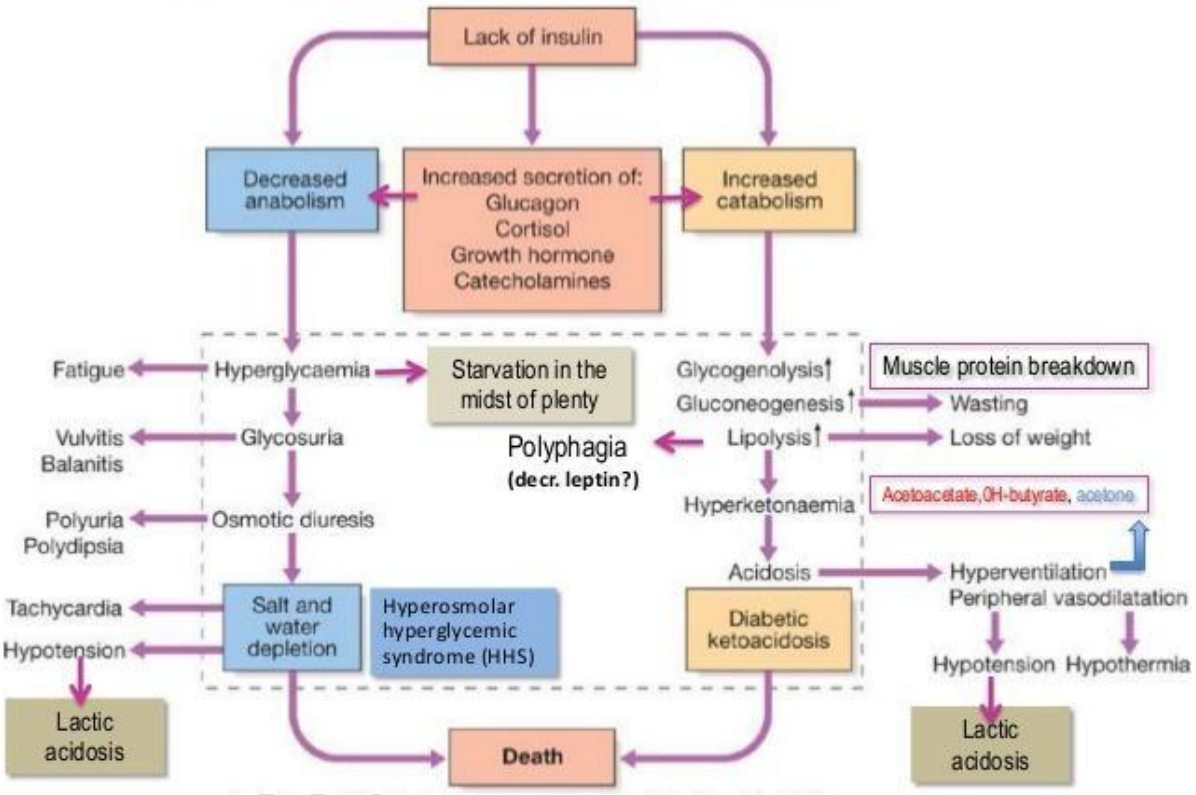
- Fasting plasma glucose less than 100mg/dl is normal fasting blood glucose.
- Fasting plasma glucose 100-125 mg/dl is impaired fasting glucose (IFG).
- Fasting plasma glucose = 126 mg/dl is a provisional diagnosis of diabetes mellitus.

Interpretation of OGTT (Oral Glucose Tolerance Test) :

- If 2 hour Post- Prandial Glucose is less than 140 mg/dl it indicates normal glucose tolerance.
- If 2 hour Post-Prandial Glucose ranges between 140 to 199 mg/dl it indicates impaired glucose tolerance (IGT).

- If 2 hour Post- Prandial Glucose is equal to 200 mg/dl then it is the diagnostic of diabetes.

FIG.4: PATHOPHYSIOLOGICAL BASIS OF SYMPTOMS:



CLINICAL FEATURES OF TYPE 2 Diabetes:

It usually starts in middle age or later. Classical type 2 DM patients are obese and have a long asymptomatic period of hyperglycemia and half of the patients have one or more diabetic complications by the time they are diagnosed[54].

Most often symptoms are due to hyperglycemia manifesting as

polyuria, polydipsia and polyphagia, Weight loss is not a common feature as seen in case of type 1 DM. Only half of patients present with classic symptoms of diabetes and the rest are detected on routine medical check up or during hospital admissions.

Type 2 DM leads to microvascular complications like retinopathy, nephropathy, neuropathy and also macrovascular complications like ischaemic cardiomyopathy or peripheral arterial disease which be presenting features in some cases[55]. Diabetic nephropathy might be there in 2.7% of type 2 DM by the time of diagnosis in 30 to 50 years age group and up to 12.4% in more than 70 years age group as per Rochester study[56]. Pirart study on complications of type 2 DM showed that the incidence of peripheral neuropathy is 8% at the time of diagnosis [57]. In Framingham study the presence of diabetic retinopathy was 5% among patients having diabetes for less than five years & it was 45% among those having diabetes for more than ten years. Majority of retinopathy is non-proliferative type[55].

Hypertension is frequently associated with diabetes mellitus. The incidence of HTN in type 2 DM varies from about 30% to 50% and even higher among females[58]. Hypertension is known to accelerate the microangiopathic complications of DM. Subjects with hypertension have more hyperglycemia than those with normal blood pressure[59].

The incidence of CAD is 2-3 times higher in diabetics. The myocardial infarction can be related through mild symptoms and go unrecognized or may be entirely asymptomatic and therefore silent. In Framingham study, unrecognized infarction accounts for 39% in diabetes.

Atypical symptoms such as confusion, dyspnoea, fatigue, or nausea or vomiting may be the presenting complaint in 32-42% of diabetic patients with myocardial infarcts. However, in contrast to all other complications, the occurrence of IHD is not related to the duration and severity of DM[60].

In Southern India, the prevalence of complications in type 2 DM was studied[61] in 3010 subjects. Retinopathy was observed in 23.7% with background retinopathy as the most common presentation. Persistent proteinuria was seen in 5.5% of patients and coronary artery disease was reported in 11.4%. Peripheral neuropathy was present in 27.5%, and cerebrovascular accidents in 0.9% of cases. Hypertension was present in 38% of cases. Patients with greater HbA1c had an elevated risk of retinopathy, neuropathy and nephropathy. This study highlights the higher incidence of with retinopathy, and neuropathy in Indian type 2 diabetics [62]. It is opined that there is high occurrence of hypertension and IHD in obese type 2 Diabetics whereas the high occurrence of retinopathy and nephropathy is seen in non obese type 2 Diabetics. Neuropathy is common in lean type 2 Diabetics[62]. Erectile dysfunction (ED) occurs in 50% to 76% of diabetic men at an earlier age[63].

COMPLICATIONS OF DIABETES – AN OVERVIEW:

Acute complications:

a) Diabetic ketoacidosis
b) Nonketotic hyperosmolar diabetic coma
c) Hypoglycemia
d) Lactic acidosis (treatment- related/sepsis)

Long- term complications:

1) Macrovascular complications
    - Peripheral vascular disease
    - Ischemic heart disease
    - Cerebrovascular disease
    - Hypertension
2) Infections
    - Genitourinary
    - Dermatologic
    - Respiratory
3) Microvascular complications
    - Retinopathy
    - Neuropathy
    - Nephropathy.

HYPERGLYCEMIC HYPEROSMOLAR STATE :

1. Due to insulin insufficiency and dehydration there occurs hyperglycemic state which causes osmotic diuresis, in turn leading to profound intravascular fluid depletion.
2. Insulin: glucagon ratio will not support ketogenesis.
3. Lower levels of counteracting hormones and free fatty acids have been documented in the hyperglycemic hyperosmolar state.
4. The liver cannot synthesize ketone bodies.

DIABETIC KETOACIDOSIS:

DKA might be the only presentation in about 10% of undetected diabetic individuals [64]. Relative or absolute insulin insufficiency along with an excess of counter regulatory hormones like glucagon, catecholamine, cortisol and growth hormone leads to the occurrence of DKA. Ketosis occurs due to the marked release of free fatty acid from adipose tissue, with a consequential shift towards the ketone body synthesis in the liver. Hyperglycemia in diabetic ketoacidosis alters hepatic glucose metabolism.

HYPOGLYCEMIA:

Physiological defences against the development of hypoglycemia that is a gradual decrease in insulin along with an increase in glucagons and epinephrine are impaired in both type 1 and type 2 diabetes leading to hypoglycemia associated autonomic failure.

CEREBROVASCUCLAR DISEASE:

There is a strong connection of diabetes mellitus with stroke risk, in particular, strokes owing to vascular disease and infarction according to the first publication of diabetes documented in America[65]. Diabetes is a significant risk factor for stroke, particularly in women.

Most ischemic strokes in Diabetics are due to occlusion of small and Paramedical penetrating arteries causing small infarcts in the white substance of the brain. The current progress in the diagnosis of stroke by computerized tomography and magnetic resonance imaging has increased the measured stroke incidence in the population, especially older individuals in the recent

times. So, most of the patients with cerebrovascular accidents can have undetected Diabetes. They all should be diagnosed & treated promptly. The reported occurrence of thrombo-embolic stroke increased by 78% in patients with diabetes.

HEART DISEASE:

It is estimated that up to 50% of newly diagnosed diabetics also have hypertension. With dyslipidemia, hypertension interacts with diabetes to amplify the risk of cardiac mortility[67].

Diabetes causes cardiovascular disease in 3 ways:

1. Atherosclerotic coronary heart disease.
2. Cardiomyopathy
3. Autonomic nervous system dysfunction

CORONARY ARTERY DISEASE (CAD)[68]

Data from UKPDS states that In recently diagnosed type 2 diabetics, the development of coronary artery disease during follow up was significantly associated with increased LDL, decreased HDL, increased HbA1c and increased systolic BP [69].

The clinical manifestations of CAD are stable Angina, Unstable Angina, MI, heart failure and sudden death. Framingham heart study has shown that comparative danger of myocardial infarction is 50% more in diabetic men and 150% more in diabetic women when compared to age- matched non-diabetics. The Study shows that 50% of patients with type 2 DM are positive for myocardial ischemia on the stress test. It can be entirely asymptomatic

and silent. Atypical symptoms like confusion, dyspnoea, fatigue, nausea, vomiting might be the only presenting features in 32-42% of diabetics with myocardial infarcts

Cardiovascular mortality is two times higher in men and four times higher in women with type 2 DM. The relative danger regarding myocardial infarction is 50% more in diabetic men and 150% more in diabetic women [68].

PERIPHERAL VASCULAR DISEASE (PVD) [70,71,72] :

Marinelli et al. in the USA has reported that the prevalence of PVD is 33% in type 2 DM and Walter et al. in the UK reported 23.5% of PVD in type 2 DM. Diabetes causes structural changes in large & small blood vessels leading to ischemia. The occurrence of PVD in south Indian diabetics is 3.9%. The factors triggering PVD are smoking, hypertension, hyperlipidemia & insulin resistance. Smoking enhances the risk of PVD to > 100 times in diabetics.

In diabetics PVD is often bilateral & multi segmental with a preference for vessels below popliteal artery, patients usually present with intermittent claudication, nocturnal pain and rest pain. Failures of intervention at the stage of nocturnal and rest pain results in tissue necrosis. On examination, the feet are cold with absent pulses, blanches on elevation with delayed venous filling. The skin would be shiny with loss of hairs and thickened nails.

INFECTIONS IN DIABETES MELLITUS [73,74]:

Infection has proven association with diabetes especially tuberculosis whose incidence increases by 3 - 16 fold. Bacteremia in female diabetics is four times higher. Malignant otitis externa, emphysematous cystitis, necrotizing cellulitis, emphysematous pyelonephritis, acute papillary necrosis,

mucormycosis, emphysematous cholecystitis and perinephric abscess are common in diabetics.

TUBERCULOSIS [74,75,76]:

Hyperglycemia favors the growth of tubercle bacilli, lowers the resistance by damaging lung vessels, lower the neutralizing antibodies in bronchial secretions & impair phagocytosis leading to higher incidence of TB in diabetes. Diabetics with pulmonary TB will have a paucity of clinical signs, a greater tendency for hemoptysis, multiple lobe affections, more cavitary lesions, less endotracheal lesions and more number of positive sputum cases.

INFECTIONS OF SKIN AND SOFT TISSUE:

10% of diabetics are staphylococcal carriers and gram - negative cellulitis is produced by both aerobic, and anaerobic organisms. Necrotizing fascitis occurs mostly in extremities and perineum.

FUNGAL INFECTIONS:

Mucocutaneous candidiasis & Vulvo – vaginal candidiais is more common in diabetics. Oropharyngeal and oesophageal candidiasis is also common. In the respiratory tract, rhinocerebral mucormycosis and pulmonary mucormycosis are known to occur in diabetics. Fungal meningitis & cavernous sinus thrombosis are common in the central nervous system.

URINARY TRACT INFECTIONS:

Asymptomatic bacteriuria is most common in diabetic women. Emphysematous pyelonephritis & emphysematous cystitis caused by E-coli and other gram- negative has an incidence of about 70% in patients with diabetes.

DIABETIC NEUROPATHY:

Diabetic neuropathy a chronic microvascular complication of diabetes may be clinical or sub-clinical & the later being diagnosed by electrophysiological studies. It is usually asymptomatic a long time in type 2 diabetes. Its Overall incidence in diabetics is 66% & is directly proportional to the duration & severity of diabetes.

Classification of Diabetic Neuropathy by Thomas P.K et al. 1993[77]

1. Hyperglycemic neuropathy
2. Symmetrical polyneuropathy
3. Focal and multifocal neuropathy
    a. Cranial neuropathy
    b. Thoraco abdominal neuropathy
    c. Focal limb neuropathy
    d. Diabetic amyotrophy
4. Mixed forms

The usual form of diabetic neuropathy is a distal sensory polyneuropathy, with or without motor involvement, affecting fibers in a length related pattern, with longer fibers being more vulnerable.

Manifestations of diabetic neuropathy [78,79] :

Initially, there are sensory symptoms like tingling, pricking, burning or numbness in the soles & tips of fingers. Early in the course of the disease, patient may be asymptomatic but careful examination might reveal loss of fine touch, diminished vibration sense and loss of ankle jerk. Thermo anesthesia occurs early while pain sensation is preserved till advanced stage. With

progression of disease, pan-sensory loss spreads over both feet, ankle and knee- jerk are diminished or lost and weakness of dorsiflexion of toes may be present. Classically sensory loss is “stocking and glove” distribution. This proves that neurons with longer axons are more vulnerable.

As neuropathy worsens in a centripetal and symmetrical manner, muscular atrophy, pan sensory loss and areflexia with motor weakness become more evident. This leads to deformity of the foot with clawing of toes, leading to the increased pressure of the tips of toes with a high risk fora foot ulcer. In severe forms of polyneuropathy pain is not perceived frequent micro trauma to one or several joints which are aggravated by weight bearing leading to disorganization of joints in the foot called as charcoal‟s osteoarthropathy.

ACUTE PAINFUL DIABETIC NEUROPATHY is a specific entity of profound and precipitates weight loss associated with severe burning pain distally in lower limbs and cutaneous hyperaesthesia.

DIABETIC MONONEUROPATHY: It is a sudden and completely reversible Involvement of cranial nerves especially third and sixth manifesting as acute unilateral painful ophthalmoplegia with sparing of pupillary reflex.

DIABETIC AMYOTROPHY: consists of acute or subacute, unilateral or asymmetric bilateral proximal limb weakness accompanied by pain. Upper limb involvement is rare.

AUTONOMIC NEUROPATHY is mostly asymptomatic. It leads to postural hypotension, nocturnal sweating, dry feet, erectile dysfunction, gastroparesis, recurrent diarrhoea, cystopathy, gustatory sweating, silent infarction and sudden death.

DIABETIC NEPHROPATHY: Patients who have type 2 DM, the prevalence of nephropathy ranges from 2 - 16%. It is a common life - threatening microvascular complication of DM marked by persistent albuminuria, hypertension and progressive renal insufficiency. The most commonly observed reason for end- stage renal disease (ESRD) is Diabetes. The constant proteinuria in newly diagnosed NIDDM increases with age [80]. Microalbuminuria is a predictive sign of cardiovascular risk. [81]

Clinical Indicators Of Diabetic Nephropathy:

Hypertension is a significant factor predisposing to diabetic nephropathy. Diabetics with nephropathy have more truncal obesity compared to controls. Microalbuminuria is the main risk factor for overt nephropathy and also other micro and macro angiopathic complications in patients who may be either IDDM or NIDDM[82].

The earliest manifestation is intermittent microalbuminuria followed by persistent & established macroalbuminuria as the condition proceeds there occurs edema and hypertension. Later on, progressive renal failure sets in and progresses. About ten years after detection of diabetes, percentage of complications which needed renal replacement treatment would be 2.0, 2.8 and 2.3% for microalbuminuria , macroalbuminuria and an elevated serum creatinine respectively [83].

Type 2 DM can also cause:

1. Type IV renal tubular acidosis.

2. Radio contrast- induced nephropathy.

Angiotensin converting enzyme inhibitors improve diabetic nephropathy.

DIABETIC RETINOPATHY:

Diabetic retinopathy constitutes for a major cause of avoidable blindness all over the world ranking 6th among causes of blindness in India. It is asymptomatic for a long duration, so they might even have advanced retinopathy at the time of diagnosis. Therefore early diagnosis & prompt treatment can control retinopathy in type 2 DM[84]. In the ICMR collaborative study of patients with type 2 diabetes, the prevalence of retinopathy was 16.4% & proliferative retinopathy was only 3%. Up to 3% of diabetics after 30 years can have clinically significant macular edema or high risk proliferative diabetic retinopathy at the time of diagnosis[85].

Diabetic retinopathy can be broadly classified as:

- Nonproliferative retinopathy: There may be generalized venous dilatation, micro aneurysms, hard exudates and hemorrhages (dot or flame- shaped).
- Pre-proliferative retinopathy: There are cotton wool spots, various abnormalities with loops, beading, and reduplication of vessels, arterial abnormalities with segmental narrowing.

Proliferative retinopathy:

Neovascularisation on disc or elsewhere, pre-retinal or vitreous hemorrhage and fibrous tissue proliferation. The main features of diabetic retinopathy are microaneurysms, hard exudates, retinal edema, soft exudates & retinal haemorrhages. Others include Neovascular glaucoma, snow flake cataract, recurrent lid infections, refractive errors, extra ocular muscle palsy, and anterior ischemic optic neuropathy.

Duration of diabetes, glycosylated hemoglobin, type of treatment systolic and diastolic blood pressure and serum creatinine show a positive correlation with retinopathy while BMI showed an inverse association.[86] Better glycemic control by normal islet cell function will decrease the incidence of retinopathy & nephropathy.[87]

FIG.5: ALGORITHM FOR TREATING TYPE 2 DIABETES

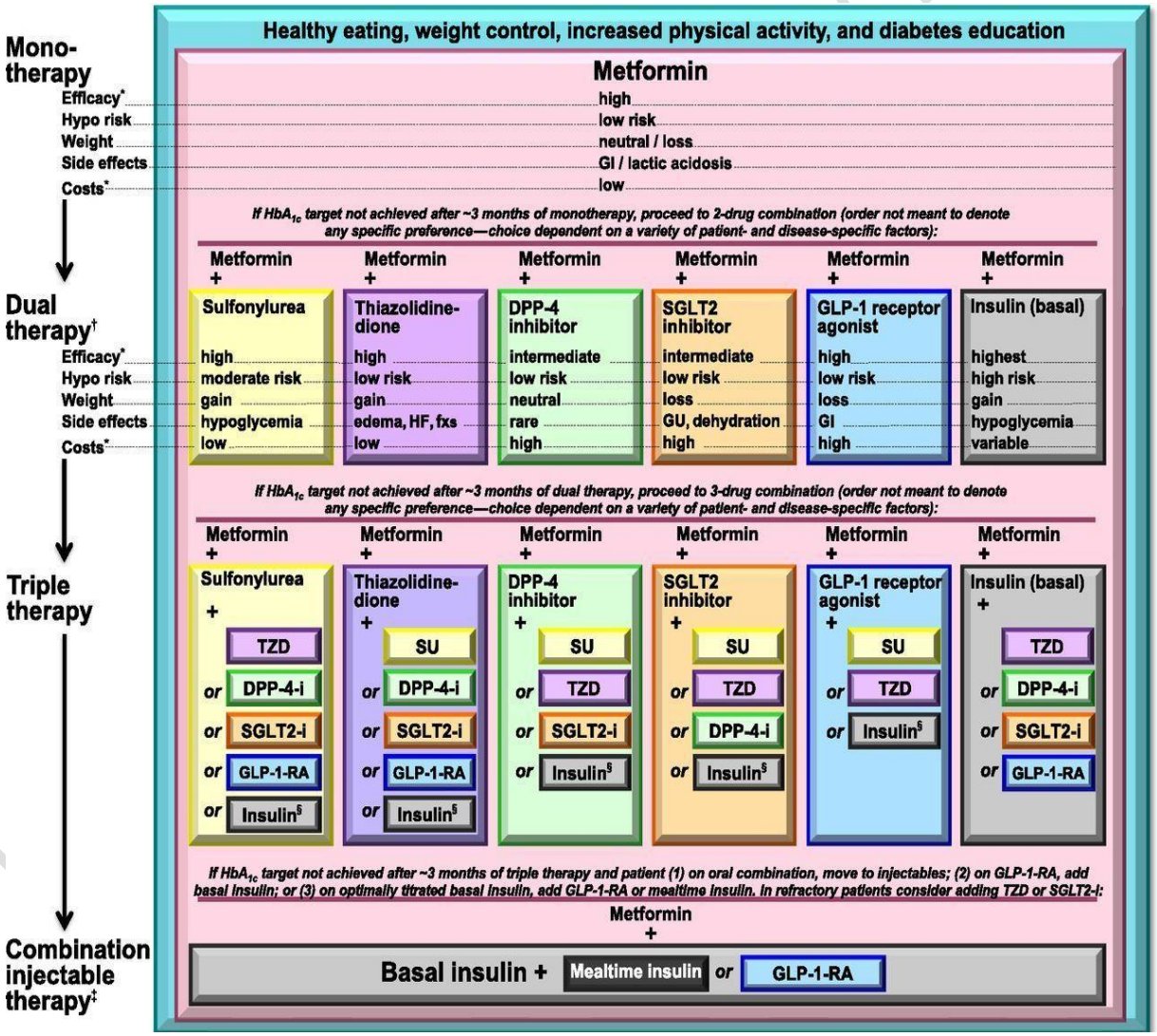
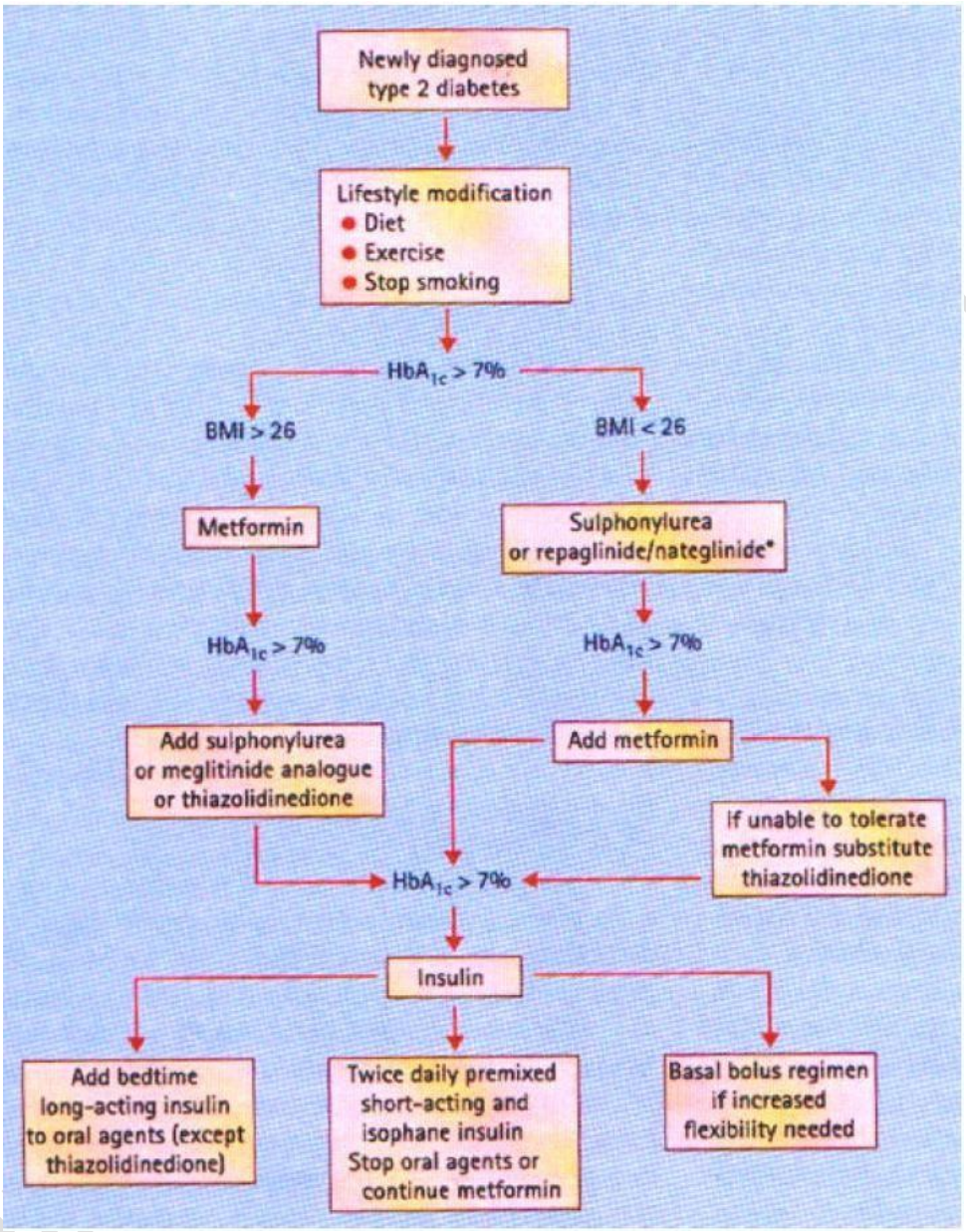


FIG.6 : TREATMENT OF TYPE 2 DIABETES BASED ON BMI:



The American Diabetes Association (ADA):

As per recommendations of ADA all individuals above the age of 45 years should be screened every 3 years and individuals with the below risk factors must be screened at a younger age.

- Family history of diabetes
- Obesity (BMI = 25 kg/m2)
- Habitual physical inactivity
- Race / Ethnicity (e.g., African American, Hispanic American, Asian American)
- Previously identified impaired fasting glucose or impaired glucose tolerance
- History of gestational DM or delivery of baby =4 kg.
- Hypertension
- HDL cholesterol level </=35 mg/dl and / or triglyceride level >/= 250 mg/dl.
- Polycystic ovarian disease or acanthosis nigricans.
- History of vascular disease.

The United States Preventive Services Task Force (USPSTF) has shown that the available screening tests could accurately detect type 2 diabetes at an early asymptomatic phase. It also stated that intensive glycemic control in patients with clinically detected (not screening detected) diabetes can greatly decrease the progression of micro vascuclar disease. But studies could not prove that strict glycemic control can reduce the macrovascular complications like myocardial infarction and stroke.

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METHODOLOGY

The study was conducted at KING GEORGE HOSPITAL, Visakhapatnam Between January 2017 to June 2018 & a total number of 200 cases were included in the study.

Study was done on patients:

a. Patients attending the outpatient department of general medicine KING GEORGE HOSPITAL Visakhapatnam who are suspected of type 2 DM on clinical presentation.
b. Admitted as inpatients for various other presentations and diagnosed as diabetes mellitus on routine screening for the first time.

Inclusion criteria:

a. Both sexes
b. Age (30-70) years
c. Family history
d. Patients previously told as DM but not on any treatment.
e. Previous blood sugar level tests were in the normal range.
f. Proved to have diabetes mellitus by FBS > 126 mg% or PPBS >200 mg% on more than 2 occasions.

Exclusion criteria:

i. Condition like acute infection, sepsis, burns.
ii. Endocrine disorders like acromegaly, thyrotoxicosis.
iii. Patients on steroids, beta blockers, diuretics.
iv. Acanthosis nigricans.
v. Pregnant women
vi. Previous abnormal blood sugar level before 3 months.

- ✓ The study was started after taking consent from the patients.
- ✓ A detailed history was taken which including the details about symptoms, mode of onset, complaints attributable to various complications, diagnosis, and duration of diabetes.
- ✓ Family history was considered to be positive if diabetes was present in first degree relatives of patients.
- ✓ All patients were clinically examined in a detailed manner.

Anthropometric assessment:

$$\text{Body mass index} = \frac{\text{Body weight (kg)}}{\text{Height (m}^2\text{)}}$$

$$\text{Waist -hip ratio} = \frac{\text{Waist circumference (cm)}}{\text{Hip circumference (cm)}}$$

Criteria for hypertension:

Hypertension was defined as blood pressure of more than 140/90 mm of Hg according to JNC VII report[88].

Criteria for neuropathy[89]:

Patients who had sensory or motor symptoms, loss of vibration or other sensory deficit, and who had loss of ankle jerks and who had no other cause to account for these signs were diagnosed to have peripheral neuropathy.

Criteria for retinopathy[90]:

Patients who had evidence of background or proliferative changes in the retina were diagnosed to have retinopathy.

Criteria for nephropathy[91]:

24- hour urinary protein was estimated in most patients as an evidence of renal involvement. Albumin excretion in the range of 30 to 300 mg, for 24 hours considered as microalbuminuria and more than 300mg, was considered as macroalbuminuria, in the absence of urinary tract infection was taken as evidence for nephropathy.

Criteria for ischemic heart disease:

Patients with typical anginal pain and or electrocardiographic changes consistent with ischemic changes were considered to have ischemic heart disease.

Cerebrovascular accident:

CVA was considered in patients with symptoms suggestive of transient ischemic attack or history and examination of stroke proven by CT scan.

Peripheral vascular disease:

Was suspected clinically if the patient presented with symptoms of intermittent claudication, non-healing ulcers, and feeble pulses.

Data collection:

a. Blood sugar will be estimated in all patients, both fasting and 2 hours postprandial venous blood glucose levels were measured at the entry of study. Blood glucose measured by glucose oxidase method.

b. Glycosylated hemoglobin (HbA1C) will be done in biochemistry laboratory via immunoturbidimetric method.

c. Blood urea, serum creatinine, lipid profile and thyroid profile estimated in all patients.
d. Urine analysis was done for physical characters, specific gravity, sugar, proteins, ketone bodies and microscopy.
e. Urine sugar was estimated by Benedict‟s semi-quantitative method. Ketone bodies were detected by Rothera method and urine proteins were detected by the heat coagulation method.
f. A centrifuged sample of urine examined for presence of pus cells, bacteria, casts and crystals.
g. Chest x-ray was done in patients suspected of pulmonary tuberculosis and other respiratory infections.
h. A standard 12 lead ECG was done in all patients.
i. BMI and Waist hip ratio was measured and calculated.
j. Co morbidities at presentation will be noted.

Lipid abnormalities were categorized according the National Cholesterol Education Program (NCEP) Expert Panel, ATP- III.[92]

Table 4: LIPID ABNORMALITIES ACCORDING TO NECP, ATP III

	Total cholesterol (mg%)	**S.triglycerides (mg%)**	**LDL (mg%)**	**HLDL mg%)**
Normal	<200	<150	<100 optimal 100-120 near or above abnormal	>45
Border line high	200- 239	150- 199	130- 159	35 - 45
High	>240	200- 499	160- 189	<35
Very high	-	≥500	≥190	-

Statistical analysis:

Data was calculated on the Microsoft Excel analysis tool pack. Continuous data were presented as mean ± SD and proportion as percentages. Comparing of mean in two groups was done by “unpaired t‟ test.

RESULTS

In the current study, 200 patients with type 2 Diabetes Mellitus diagnosed for the first time based on WHO criteria were included in the study. The age distribution is as follows :

TABLE – 5: AGE WISE DISTRIBUTION

Age in years	No. of patients	Percentage
30- 35	14	7
36- 40	26	13
41- 45	28	14
46- 50	38	19
51- 55	26	13
56- 60	24	12
61- 65	18	9
66- 70	26	13

In the present study which is done in patients of age 30 to 70 years, the calculated mean age is 50.72 years, with a standard deviation of 10.78years.

The mean age of the male population is 49.64years with a standard deviation of 11.19 years; female population is slightly older than males in the current study with mean age of diagnosis is 52.39 years with standard deviation of 9.94 years. Maximum number of patients were between 35-55 years

Graph showing age wise distribution of the study population, 64 % of the study population is between 35 to 55 years.

GRAPH-1 : AGE WISE DISTRIBUTION

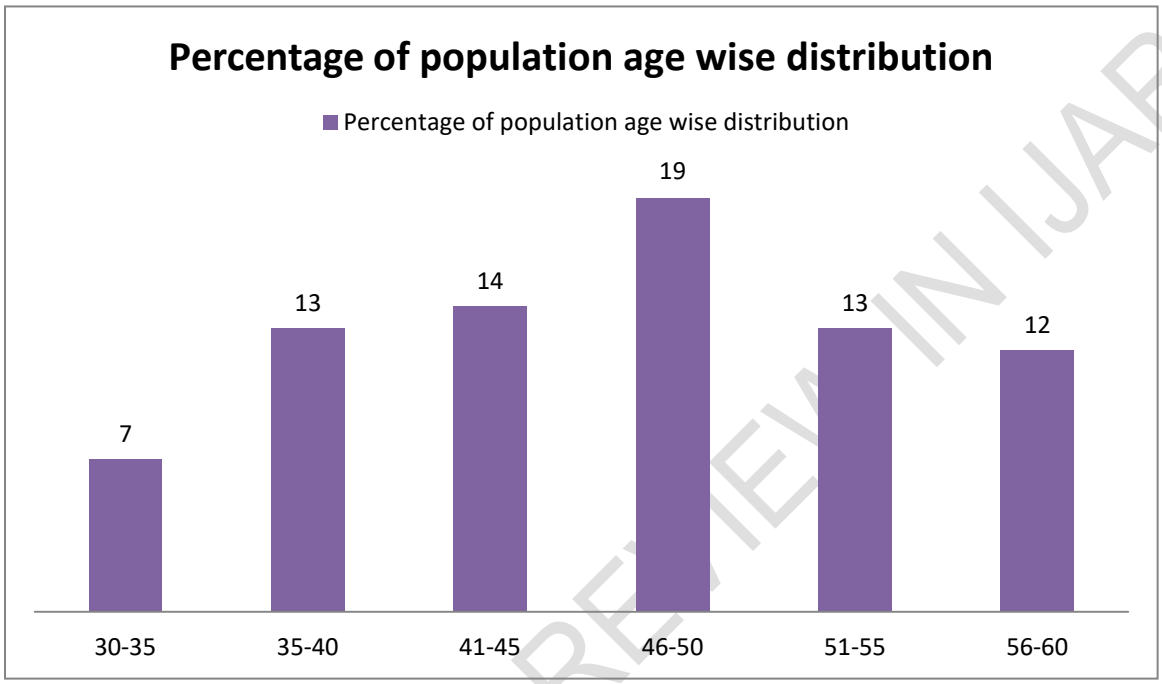


TABLE – 6: SEX DISTRIBUTION

Sex	No.of patients	Percentage
Male	122	61
Female	78	39

In this study, males constitute 61% (122), and females constitute 39% (78) of the study population. In the study population males are 1.56 higher than females. This is shown in graph 2 below.

Positive family history was noted in 31% (62) of the study population. Waist - hip ratio in male subjects was 0.936 ± 0.092 and in

females it was 0.939 ± 0.096, and overall range 0.7- 1.1, from table-11 below. 20 % of male patients had waist-hip ratio of more than 1.0. 30 % (60 patients) of female patients had waist-hip ratio of more than 0.9. These patients were categorized as subjects with central obesity; it is well known that patients with central obesity tend to develop type 2 diabetes mellitus early and more prone to have higher degree of insulin resistance.

GRAPH -2 SEX DISTRIBUTION

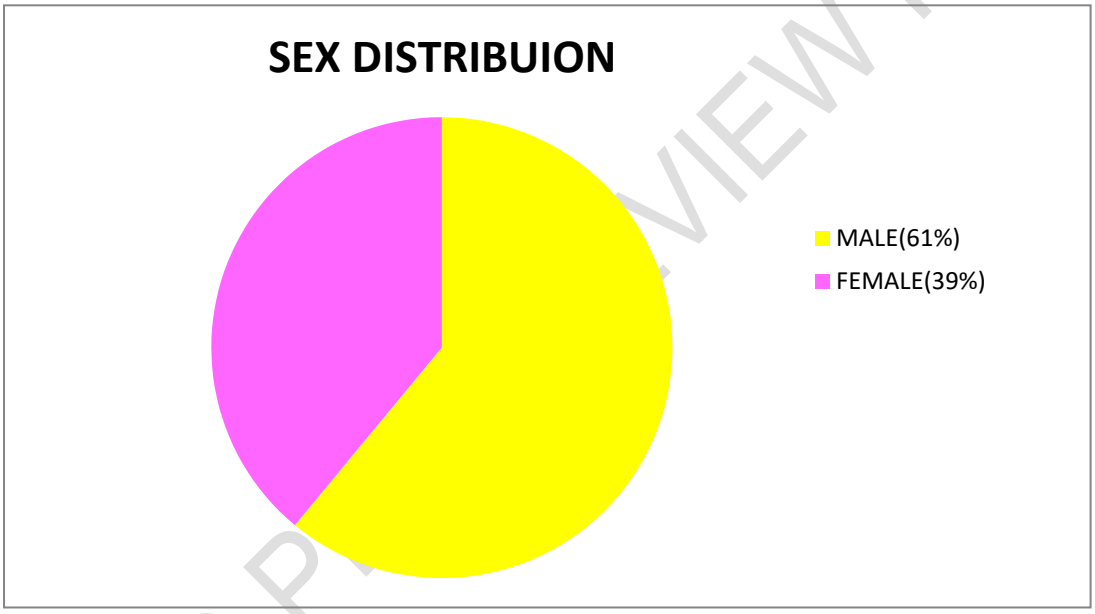


TABLE – 7: BODY MASS INDEX

Total	Mean	SD
Male	23.20	3.61
Female	25.36	5.08

In the current study as shown in table-7 above mean body mass index was 24.04kg/m^2 with a standard deviation(SD) of 4.36kg/m^2 .Body mass index in females is slightly higher (25.36kg/m^2±5.08) than males (23.20kg/m^2±3.61).

TABLE -8 WAIST- HIP RATIO ACCORDING TO NO.OF PATIENTS

	No.of patients	**Percentage**
Male		
<1.0	82	41
> 1.0	40	20
Female		
<0.9	18	9
>0.9	60	30

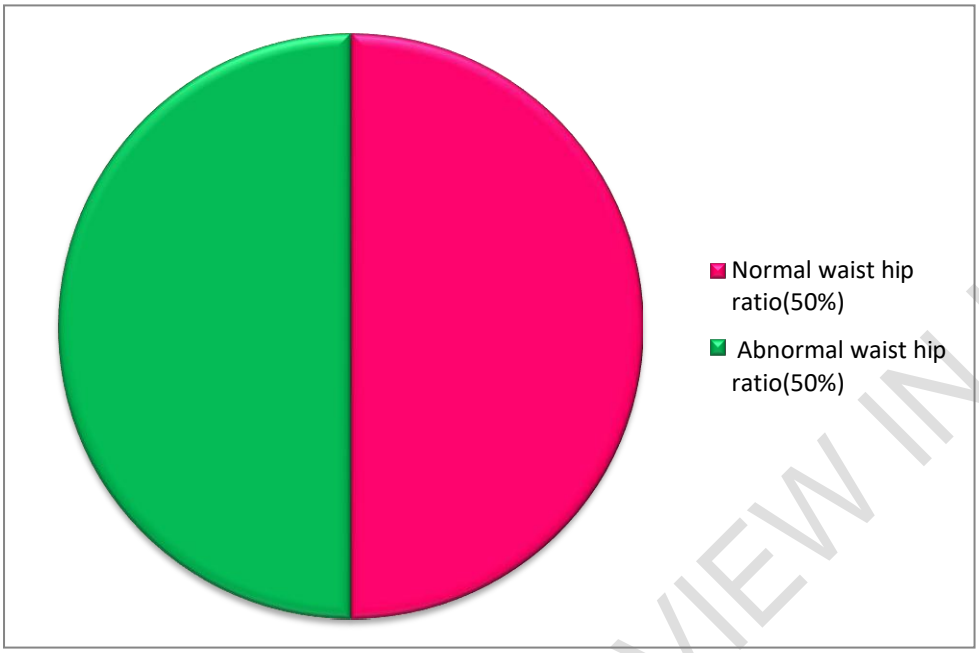
Total Waist Hip ratio Abnormal - 50%

Normal - 50%

TABLE – 9: MEAN WHR

	WHR	SD
Total	0.936	0.091
Male	0.933	0.092
Female	0.939	0.096

GRAPH-3: WAIST HIP RATIO



GRAPH-4: WAIST HIP RATIO

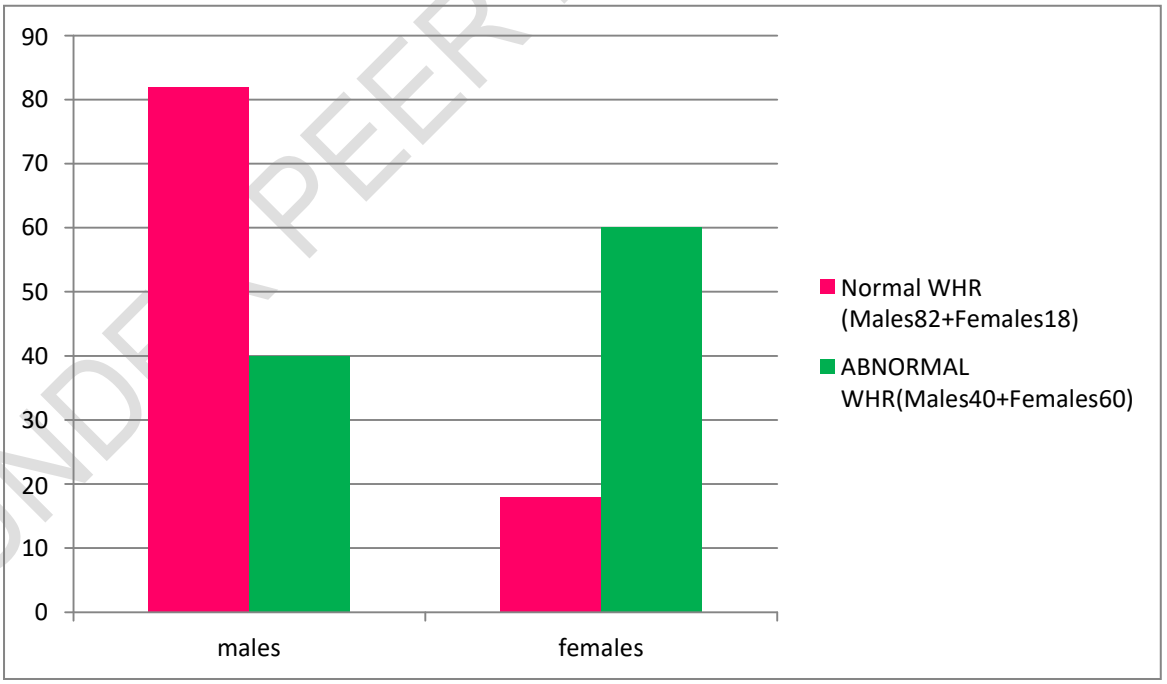


TABLE – 10: FASTING BLOOD SUGAR

Fasting blood sugar (mg/dl)	No.of patients	Percentage
126- 150	20	10
151- 200	62	31
201- 250	62	31
251- 300	38	18
301- 350	8	4
>350	12	6

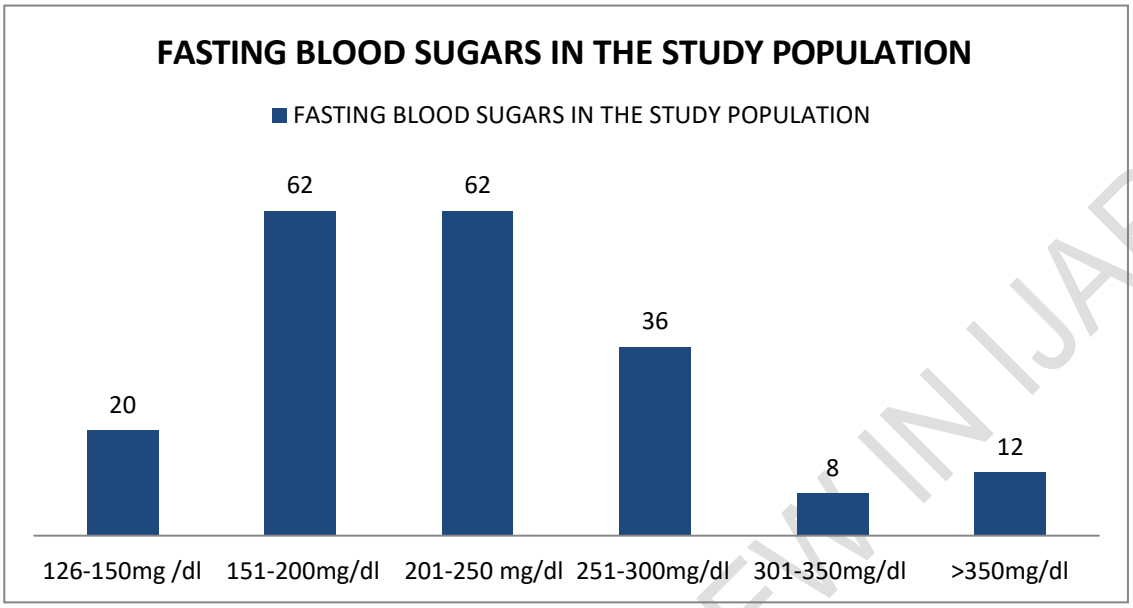
Mean FBS 231.33±88.24 mg/dl, Fasting blood sugar (FBS) in the study group varied from 139 mg/dl to 600mg/dl ,In males mean FBS is 228.27 ± 88.81mg/dl in females it was slightly high 236.10 ± 88.29 mg/dl as shown in Table -10 above

TABLE – 11: POST PRANDIAL BLOOD SUGAR

Postprandial Blood Sugar	No. of patients	Percentage
200- 250	40	20
251- 300	64	32
301- 350	28	14
351- 400	30	15
>400	28	14

Mean post prandial blood sugars in the current study was 294.49 mg/dl ± 97.58 , it varied from 209mg/dl to 510mg/dl, as shown in table-11 above.

GRAPH NO-5 SHOWING FASTING BLOOD SUGARS IN STUDY POPULATION



GRAPH-6: POST PRANDIAL BLOOD SUGAR

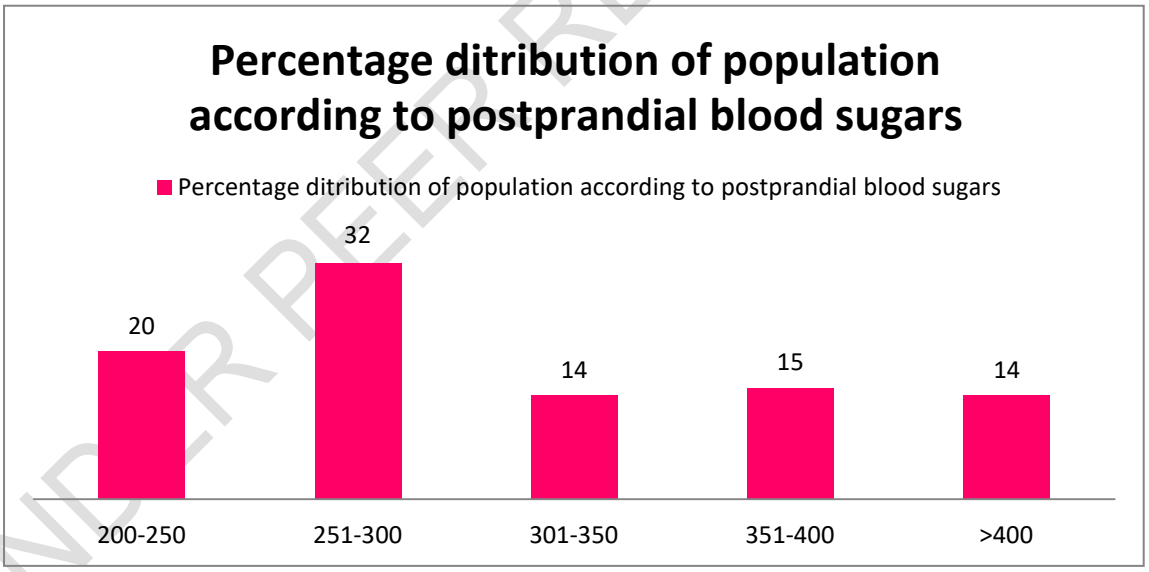


TABLE – 12: GLYCOSYLATED HEMOGLOBIN

HbA1C (%)	No. of patients	Percentage
6- 8	54	27
>8- 10	92	46
>10- 12	46	23
>12	8	4

Mean Glycosylated hemoglobin (HbA1C) in the current study was 9.09 ±1.55% with a range from 6.6%to12.8%. In males meanHbA1C, is 9.07±1.41% in females it is 9.13±1.75% as shown in Table -12 above.

TABLE – 13: FASTING LIPID PROFILE

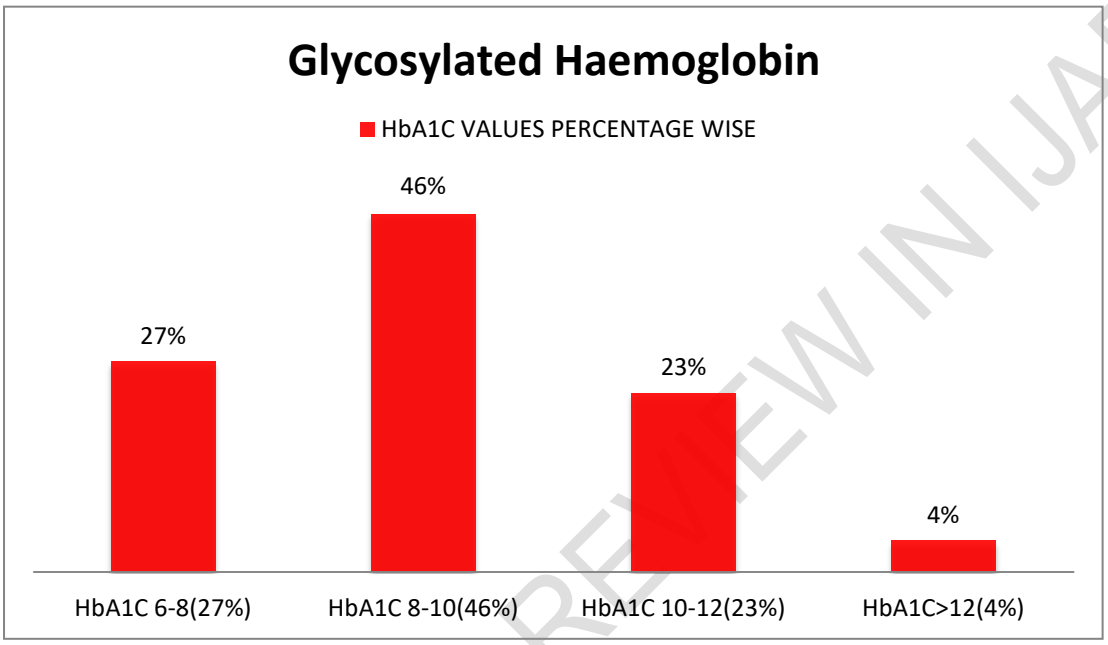
Parameter	No. of patients	Percentage
TG > 150 mg%	23	25.5
LDL > 100 mg%	43	45.7
HDL < 40 mg%	10	10.4
Cholesterol > 220 mg%	16	17.02

TABLE –14: LIPID ABNORMALITY

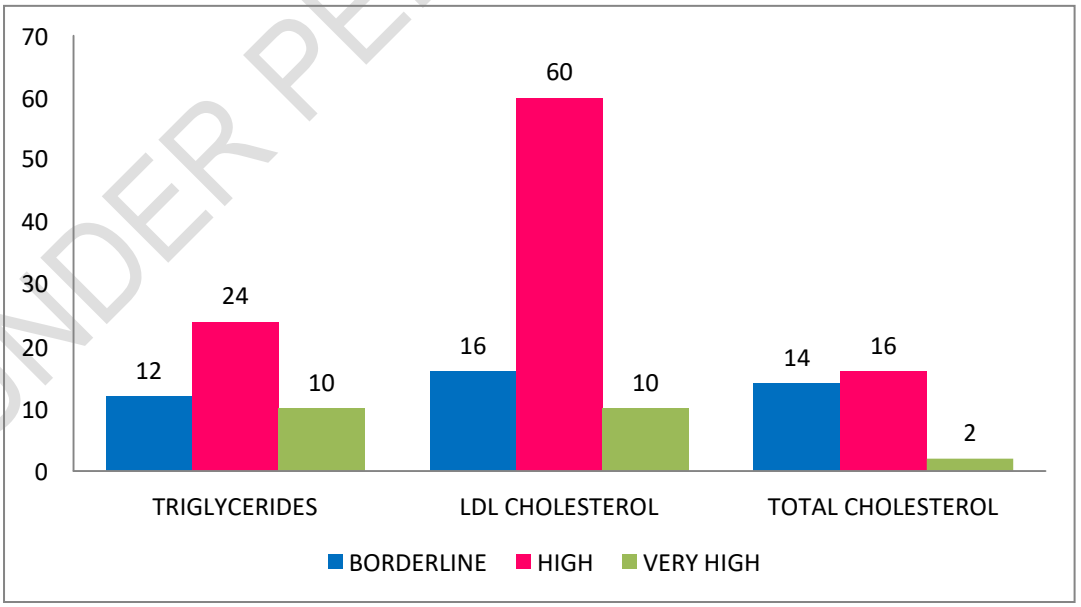
	TG mg/dl	**LDL mg/dl**	**HDL mg/dl**	**TC mg/dl**
Border line high	6 (150- 199)	8 (139- 159)	7 (35- 45)	7 (200- 239)
High	12 (200- 499)	30 (160- 189)	3 (<35)	9 >240
Very high	5 (> 500)	5 (> 190)	-	1

According to the National cholesterol education program (NCEP) expert panel, ATP (III) [92]. At least 45.7% had an LDL value above the recommended values for type 2 diabetics, requiring intervention.

GRAPH-7: GLYCOSYLATED HAEMOGLOBIN



GRAPH-8 : LIPID ABNORMALITY



COMPLICATIONS:

Complications of type 2 diabetes mellitus Identified at the time of diagnosis in the present study shown in Table -15 arranged in decreasing order of frequency.

TABLE NO -15 SHOWING COMPLIACTIONS IN STUDY POPULATION

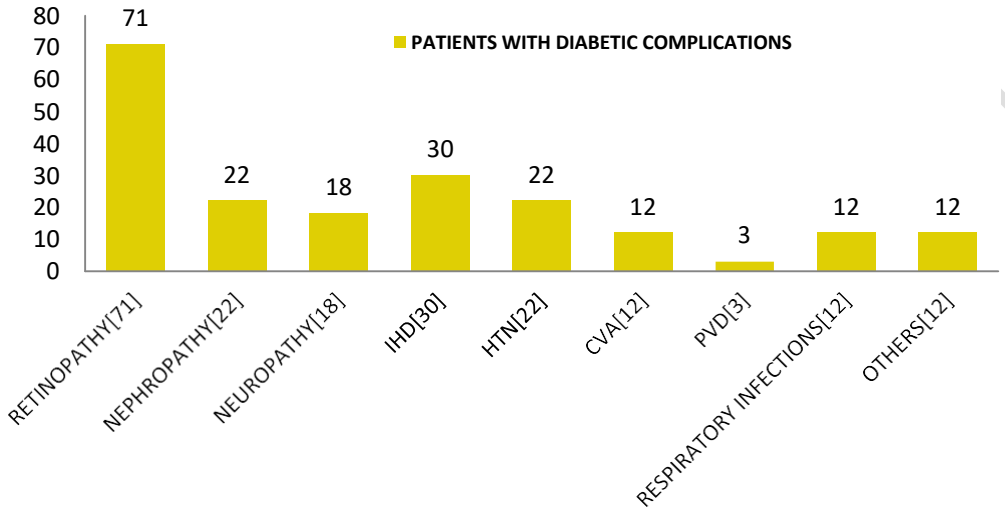
Complications	No of Patients	Percentage
Retinopathy	71	35.5
Nephropathy	22	11
Neuropathy	18	9
HTN	22	11
IHD	30	15
CVA	12	6
PVD	3	1.5
Infection Respiratory pulmonary TB Others(soft tissue) Skin UTI	8 4 8 4	4 2 4 2
Acute hyperglycemic complication DKA HHS	10 6 4	5 3 2
Other	12	6

In the current study – retinopathy was noted in 35.5% of the patients. Majority had non- proliferative type of retinopathy. 64 patients had non proliferative diabetic retinopathy, 7 patients had proliferative diabetic retinopathy .11% (22) patients had cataract at presentation.

- 11% of patients of the patients had diabetic nephropathy
- Peripheral neuropathy was present in 9% of patients.
- Hypertension was noted in 11% (22 patients)of the study population
- 15% of patients had IHD at presentation, which included unstable angina in 12 patients, myocardial infarction in 15 patients, bundle branch block in 3 patients.
- 6% of patients had cerebrovasular accidents as confirmed by CT brain imaging at the hospital, which included 8 patients with ischemic stroke and 4 patients with hemorrhagic stroke
- Peripheral vascular disease was noted in 3 (1.5%) patients – presented with non healing ulcer over great toe.
- 12 (6%) patients presented with respiratory infection out of which 6 patients had pulmonary tuberculosis, 4 patients had bronchitis and 2 patient had tubercular pleural effusion.
- 12 patients presented with other infections out of which 4 patients had urinary tract infection. 8 patients had skin infections like cellulitis, folliculitis and dermatitis.
- Acute hyperglycemic complications were noted in 10 patients, out of which 6 patients had diabetic ketoacidosis and 4 patients had hyperosmolar coma.
- 6 patients presented to other departments for other complications like road traffic accident, hernial repair and abscess.
- In the current study on routine examination type 2 diabetes mellitus was detected in 16 patients.

- In the current study 14% of patients presented with multiple complication (>2) at the time of diagnosis of type 2 DM.

GRAPH 9 SHOWING DIABETIC COMPLICATIONS



DISCUSSION

Diabetes mellitus is a disorder of body metabolism characterized by a state of chronic hyperglycemia which tends to cause multiorgan dysfunction over time. Patients usually present late with complications at the time of diagnosis of Type 2 diabetes mellitus. At the time of first presentation to hospital often many patients develop subtle complications, usually asymptomatic detected by focused clinical examination.[93]

To determine the prevalence of complications of DM in patients presenting to a tertiary teaching hospital in south India to enable us to have a picture of the general pattern present in the society, this would help us determine the high risk groups and evolve screening strategies aimed at early diagnosis, thus reducing the morbidity due to the disease.

On the basis of WHO criteria patients who were detected to have Type 2 Diabetes mellitus for the first time were included, a total of 200 patients were included in the study.

Age of the patient:

Table No -16 : study population compared to other Indian studies

Mean age ± SD years	Chennai study[94]	Rakesh[95]	Present study
	52.97 ±9.7	55.981±1.26	50.721±0.78

Compared to other Indian studies, the mean age of presentation was lesser 50.721±0.78 years, in the current study, indicating early onset of Diabetes and most of the study population belong to 35-50 year age group, which constitutes the mid productive years of life.

With the increased prevalence of more stressful life style, and increased incidence of diabetes at a much younger age, emphasizes the need to screen younger age group to detect DM at an early stage which helps to prevent or decrease the evolution of complications during this latent phase of hyperglycemia.

Type 2 DM Incidence in the current study has shown male preponderance with Male to Female ratio of 1.56:1.

A study from South Africa conducted by Siemens DZ et al. found that family history had a significant association with diabetes mellitus.[93]

In the current study family history is present in 31% of the patients also found to be a major predictor of diabetes. Whether this finding emphasizes the need to submit younger age group people with a family history of DM for diabetes screening tests from younger age and continue periodically is debatable. This association

not only highlights the importance of shared genes and environment in diabetes but also opens the possibility of formally adding family history to public health strategies aimed at detecting and preventing the disease.

Obesity rates are increasing world-wide concordant to surge in Diabetes prevalence. Shlgikaret al. earlier in 1991, showed that central obesity as determined by BMI and waist to hip ratio is correlated with hyperglycemia more in Asian Indian subjects than generalized obesity.[96]

In this study it has been found 50% were lean diabetics out of which males were 41% and females 9% which shows lean diabetes is more common in males with male BMI < 25% and WHR < 1 and females BMI.25 in females and WHR <0.9. These findings are consistent with the findings of Chennai study[94] and Rakesh et al[95] study.

In the current study out of 200 patients it was found (85%) had allowed their symptoms(diabetes- related or unrelated) to progress without evaluation and presented to hospital, asymptomatic patients were only 22% (44)patients. This emphasizes that majority of patients when type 2 diabetes is detected for the first time, organ dysfunction exists at a latent stage making them unrecognizable unless focused examination is performed.

Analysis of complication:

The detailed examination was done in search of micro and macro vascular complications.

Estacio et al. in previous studies in Hispanic population showed that One of the earliest organ systems to be involved is the eye and the Complication was non proliferative retinopathy which in itself indicates that the presence of the disease is more than 5 years duration. Neuropathy is found to be closely related with retinopathy[97] .

DIABETIC RETINOPATHY

Table No-17 DIABETIC RETINOPATHY INCIDENCE COMPARED WITH OTHER STUDIES

	Chennai Study [94]	Rakesh [95]	Nakagami[84]	Ramchandra[61]	Present Study
Diabetic Retinopathy	23.7%	28%	17.2%	23.7%	35.5%

In a study conducted by Abraham et al in Indian subcontinent, the prevalence of Diabetic retinopathy in type 2 DM varies widely from 4.34% among diabetic clinic patients in India.[98] In South India Ramachandra et al study had a prevalence of retinopathy in 23.7% of patients, while Mohan et al from the same place had a prevalence of 34.1% in type 2 DM.[99] In the current study 35.5%(71 patients) had retinopathy out of which 32%(64 patients) had non proliferative diabetic retinopathy and 11%(22 patients) had others like cataract, glaucoma. The incidence of retinopathy in the present study

correlates with the study of Rakesh et al[95] , Ramchandra and Chennai study. This huge incidence of retinopathy at the diagnosis of Type 2 diabetes emphasizes the need for routine ophthalmologic screening in type 2 Diabetes Mellitus patients at the initial diagnosis.

DIABETIC NEPHROPATHY:

Table 18: Comparing incidence of Diabetic nephropathy with other studies

Diabetic Nephropathy	Chennai study94	Rakesh[95]	Rochester study [56]	Pirart study [57]	Present study
	5.55%	47%	2.7%	8%	11%

Diabetic end-stage renal disease is a devastating condition that can be avoided in some cases and substantially delayed in many. The detection of microalbuminuria identifies a subgroup of patients with a high risk of cardiovascular morbidity and mortality as well as diabetic renal disease and aggressive management of these patients can greatly improve their outlook. At the time of diagnosis of type 2 Diabetes mellitus in the current study the incidence of diabetic nephropathy is 11% which is almost similar to the incidence of diabetic nephropathy in Chennai study and Pirart study and less than Rakesh et al[95] as the age group in present study is less at the time of diagnosis of type 2 Diabetes mellitus.

Proteinuria is present in 55% of type 2 DM at presentation in Ramachandran et al[61], In present study it is 22% out of which 11% has Massive proteinuria and remaining 12% has mild to moderate

proteinuria. This confirms to institute early screening methods to detect nephropathy at an earlier stage. By doing so we gain a lead period to institute methods to reverse the ongoing renal damage.

DIABETIC NEUROPATHY:

Table 19: Comparing the incidence of Diabetic neuropathy with other studies.

Diabetic Neuropathy	**Chennai study[94]**	**Rakesh[95]**	**Ramchandra et al[61]**	**Present study**
	27.5%	43.7%	27.5%	9%

The incidence of peripheral neuropathy in the study conducted by Ramchandra et al. study peripheral neuropathy 27.5%.while the prevalence of diabetic neuropathy in Vijay et al is 10%

In the present study, neuropathy incidence is less than other India studies on early presentation of type 2 DM are younger age group. This also could be partially explained by the fact that objective nerve conduction was not performed in the current study and the assessment of neuropathy in the current study is only subjective.

The commonest type of neuropathy in all above studies is Distal symmetrical sensory motor neuropathy.

HTN :

In type 2 DM the incidence of hypertension varies from 30- 50%. Ramchandran et al[6 1] study on complications type 2 DM has shown an incidence of hypertension in type 2 DM as 36%. In the current study the incidence of hypertension is (11%), which was less compared to other Indian studies. This lower incidence of Hypertension in the current study is explained by the fact that this included naive Diabetic patients into the study. The incidence of hypertension increases and reaches the same proportion as in Ramachandran et al study possibly, if the study is continued on the subjects of the present study for few more years.

IHD : In the current study the incidence of ischemic heart disease is 15%, this is almost similar to the results of a study conducted by Ramachandra et al[61] which showed an incidence of almost 11.5%.

DYSLIPIDAEMIA: In the current study low- density lipoprotein abnormality was found in a significant proportion of the population,

in 43% (86) patients had elevated LDL levels in accordance to the current guidelines, out of which a value above > 160 mg/dl was noted in 70 patients who need definite intervention. Triglyceride levels were high > 150 mg/dl in 46 patients. This finding of dyslipidemia to an extent of needing pharmacological intervention obviates the need for a periodic screening of diabetes for dyslipidemias

CVA:

Table 20: Comparing incidence of CVA with other studies.

	Ramchandra et al[61]	Chennai study[94]	Present study
CVA	0.9%	3%	6%

The incidence of the cerebrovascular accident is 6%(12 patients) in the present study, in Chennai urban study it is 3% and Ramachandran it is 0.9%. The marginally higher incidence of IHD and CVA could probably be addressed if the other risk factors for IHD and CVA are accounted for.

PVD :

Table 21: Comparing the incidence of PVD with other studies.

	Mohan et al[71]	Chennai study [94]	Present study
PVD	3.9%	4.5%	1.5%

There is limited data available on peripheral vascular disease (PVD) from the subcontinent of India eventhough there have been reports that PVD is less common among migrant Indians. This is of particular importance in view of increased incidence of coronary artery diseases in Indian subcontinent population .Majority of studies from india were based on clinical profile rather than Doppler parameters.In the current study, peripheral vascular disease incidence is 1.5%, this was noted to be 3.9% by Mohan et al[71] in which

4591 patients were studied in the year 1995 and 4.5% has noted by Chennai study. The incidence was similar in studies conducted from srilanka by Desilva et.al. European studies have much higher incidence of peripheral vascular disease incidence among diabetics.Thus the current study is in line with the other subcontinental studies which showed a low incidence of peripheral vascular disease.

Infection:

Skin Infection like dermatitis, folliculitis, cellulitis and UTI is 6% which is more than Chennai study and Ramachandra A Pulmonary tuberculosis was present in 4% of patients in the current study this incidence was almost twice high compared to incidence of tuberculosis in nondiabetic individuals. In general, infectious diseases are more frequent and/or serious in patients with diabetes mellitus, which potentially increases their morbidity & mortality. The greater frequency of infections in diabetic patients is caused by the hyperglycemic environment that favors immune dysfunction. Majority of patients with Diabetes develop MDR-TB more commonly compared to normal individuals.

Acute Hyperglycemic complications:

The prevalence of DKA as presenting feature is about 10% of patients with undiagnosed diabetes as noted by V.Sheshaiah[64] . In the present study, it is 3% of patients had diabetic ketoacidosis, 2% had hyperosmolar nonketotic coma. So early diagnosis can reduce the incidence of this life-threatening complication.

Glycosylated Hemoglobin :

Table 22: Comparing levels of glycosylated Hb with other studies.

Mean HbA1c	Rakesh et al[95]	Present study
	8.64±1.19	9.091±.55

High HbA1c is present in the present study in patients diagnosed as Type 2 DM as compared to Rakesh et al. study[95] indicating much more chronicity in this group of study population compared to other studies.

In present study, 12% of patients presented with multiple complications (>2) at the time of diagnosis, and 90% of them had retinopathy with nephropathy, which shows the close relation between each other as shown in other studies.[97]

Followed in incidence by retinopathy and neuropathy. These multiple complications are frequent in patients whom fasting blood glucose and HbA1c values were higher.

In this study, there was a significant correlation between fasting blood sugar and serum HbA1c. This was proved by Mayor B, Davidson et al. in 1999.[100] The paired values of fasting plasma glucose and HbA1c or fasting plasma glucose and fructosamine values together predict the likelihood of having diabetes in high- risk subjects; and could replace the time consuming and laborious 2 hour oral glucose tolerance test in the diagnosis of diabetes. In fact- American Diabetes Association has recently recommended moving away from O.G.T.T. to using fasting plasma glucose as a diagnostic procedure.

LIMITATIONS OF THE STUDY:

- Due to financial constraints, patients could not be subjected to Nerve conduction studies or other objective evidence of neuropathy and the examination had to be limited to only clinical methods.
- Younger age groups could not be subjected to islet cell antibody testing to eliminate the possibility of type 1 diabetes.
- Limited sample size.

CONCLUSION

The present study was an endeavor to estimate the prevalence of complications in new diabetics at diagnosis and to compare the changing trends, if any, at presentation.

- Males were more often affected than females. Family history was positive in 31% of the patients.
- 44% of the patients were less than 50 years of age. The blood sugar estimation may need to be done in younger age groups so as to effectively detect diabetes early.
- The percentage of asymptomatic patients i.e., those being incidentally detected was only 16%.
- Nephropathy was found to be common with a proportion of patients in the microalbuminuric phase, which is reversible if detected early.
- Incidental diabetes on evaluation during a hospital admission was much more common than those presenting for symptoms of hyperglycemia.
- There is a positive correlation between fasting blood sugar and serum glycosylated hemoglobin.
- As hyperglycemic status becomes chronic or long-standing (increased blood sugar levels and HbA1C level) the complication particularly microvascular complication increases and a significant number of patients had multiple complications (>2) in 12% patients.

SUMMARY

This cross-sectional study entitled “Clinical study of type 2 DM at presentation and its relation to glycemic status” was conducted at King George hospital , attached to Andhra Medical College, between the period of January 2017– June 2018 A total of 200 patients were studied on diagnosis of type 2 DM based on WHO criteria.

The study population had 122 males and 78 females. The mean age of onset of DM in present study was 50.72±10.78 years. The family history of diabetes was present in 31% of patients. 78% of patients present with symptoms suggestive of complication of DM at the first presentation, which necessitates the need for routine screening for Type 2 DM. Positive family history in 31% of the study population warrants more rigorous screening in this set of population, which could reduce the disease burden in later life.

Retinopathy was the commonest presentation followed by ischaemic heart disease, nephropathy. Patients also presented on routine examination (22%) and to other departments (6%) followed by infections. The mean BMI in the present study is 24.04 ± 4.36 central obesity was present in 48% of patients, the incidence of peripheral vascular disease, cerebrovascular disease was low.

The patients in the study group had significant hyperglycemia with Mean fasting and post prandial blood sugar which were 231.33 ± and 294.49 ± 97.58. Fasting lipid profile is abnormal in 45% had raised LDL and 25% had raised triglycerides.

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ANNEXURES

ANNEXURE – I

PROFORMA

Address :

Name : IP/OP No. :

Age :

Sex : D.O.A. :

Occupation : D.O.D. :

Life style : Sedentary/Moderate/Heavy

PRESENTING SYMPTOMS DURATION:

Polyuria	:	Present/Absent
Polydipsia	:	Present/Absent
Polyphagia	:	Present/Absent
Weight loss	:	Present/Absent
Generalized weakness	:	Present/Absent

CNS :

Giddiness	:	Present/Absent
Headache	:	Present/Absent
Vomiting	:	Present/Absent
Loss of consciousness	:	Present/Absent
Convulsion	:	Present/Absent
Tingling	:	Present/Absent
Numbness	:	Present/Absent
Parasthesia	:	Present/Absent

Polyneuropathy:

Syncope	:	Present/Absent
Weakness of limb	:	Present/Absent
Proximal	:	Present/Absent
Distal	:	Present/Absent

Mononeuropathy:

Wrist drop	:	Present/Absent
Foot drop	:	Present/Absent

ANS:

Dysphagia	:	Present/Absent
Vomiting	:	Present/Absent
Nocturnal diarrhea	:	Present/Absent
Urinary incontinence	:	Present/Absent
Impotence	:	Present/Absent

Amyotrophy:

Thinning of Proximal muscle	:	Present/Absent
Weakness of Proximal muscle	:	Present/Absent

CVS:

Chest Pain	:	Present/Absent
Palpitation	:	Present/Absent
Dyspnea	:	Present/Absent
Orthopnea	:	Present/Absent
PND	:	Present/Absent
Cough	:	Present/Absent

Productive	:	Present/Absent
Hemoptysis	:	Present/Absent
Fever	:	Present/Absent
Evening rise of temp.	:	Present/Absent

GIT:

Nausea	:	Present/Absent
Heart burn	:	Present/Absent
Constipation	:	Present/Absent
Diarrhea	:	Present/Absent

RENAL:

Swelling of feet	:	Present/Absent
Puffiness of face	:	Present/Absent
Distension of abdomen	:	Present/Absent

OTHERS:

Depression	:	Present/Absent
PAST HISTORY	:	
HTN IHD TB CVA MI		
FAMILY HISTORY OF DM	:	F/M/B/S Others
DRUG HISTORY	:	H/O Usage of steroid, Beta Blockers, Diuretics

GPE:

Conscious	:	Present/Absent
Oriented	:	Present/Absent
Built	:	Lean/ Normal/ Obese
Height	:	

Weight :

BMI:

Waist :

Hip :

Waist/Hip :

Radial pulse:

Rate Rhythm Condition of Vessel Wall

Volume Other peripheral pulses:

BP:

Single : JVP:

Standing : Skull & Spine

Anaemia : Present/Absent

Icterus : Present/Absent

Cyanosis : Present/Absent

Lymphadenopathy : Present/Absent

Pedal oedama : Present/Absent

Puffiness of face : Present/Absent

Skin Changes:

Colour Changes : Present/Absent

Eruptions : Present/Absent

Swellings : Present/Absent

Carbuncles : Present/Absent

Fungal infections : Present/Absent

Oral Cavity: :

SYSTEMIC EXAMINATION:

Cardiovascular System:

Respiratory System:

Per Abdomen:

CNS:

HMF:

Cranial nerves:

Eye:
Cataract: Present/Absent
Fundus Examination

Motor:

Nutrition

Tone

Power

Involuntary movements

Reflexes - Superficial

-Corneal

-Abdominal

-Cremasteric

DTR:	BJ	TJ	KJ	AJ
Rt				
Lt				

Plantars

SENSORY:

Pain:

Temperature:

Touch:

Joint Position:

Vibration:

CEREBELLAR:

INVESTIGATIONS:

FBS: PPBS:

Glycosalated Hemoglobin

Blood Urea

Serum Creatinine

Lipid Profile

S. Total Cholesterol

S. Trigluycerides

S. HDL Cholesterol

S. LDL Cholesterol

Urine: Albumin: Sugar: Ketone bodies:
Microscopy:

ECG:

Chest X-Ray PA View:

DIAGNOSIS:

WRITTEN INFORMED CONSENT

I, …… hereby give my consent in writing, for including myself in this study titled "CLINICAL STUDY ON TYPE 2 DIABETES MELLITUS

AT PRESENTATION AND ITS RELATION TO

GLYCEMIC STATUS". The investigators explained me in detail about the study and I understood the following information.

a) The investigator will take my demographic details like age, gender, occupation, education, area of residence, life style, personal, family & past medical history.
b) Investigator will do A detailed clinical examination & Anthropometric assessment .
c) Investigator will collect 8 ml of blood is collected from me to perform laboratory tests related to blood glucose, renal profile, lipid profile & thyroid profile. Also tests like urinary protein, chest x ray & ECG will be done .
d) Personal details will not be revealed to any person without my written consent.
e) I am giving this consent without undue pressure.
f) Any complication during study period will be taken care off by the investigator.

Participant signature

Investigator signature

సమ్మతి పత్రము

..... అను నేను మెడిసిన్ డిపార్ట్మెంట్ , కింగ్ జార్జ్ ఆసుపత్రి , విశాఖపట్నం వారి ఆధ్వర్యంలో నిర్వహిస్తున్న " టైప్ 2 డయాబెటిస్ వ్యాధి యొక్క తొలిదశ లక్షణాలు మరియు దానియొక్క గ్లైసెమిక్ స్థాయిల కి గల సంబంధము "అనే అధ్యయనంలో పాల్గొనుటకు వ్రాతపూర్వకముగా నా సమ్మతి తెలుపుచున్నాను.
పరిశోధకురాలు నాకు ఈ క్రింది విషయములు వివరముగా చెప్పినారు.

a) నా వయస్సు , లింగం , వృత్తి , విద్య , నివాస ప్రదేశం , జీవిత శైలి , వ్యక్తిగత , కుటుంబం మరియు గత వైద్య చరిత్ర వంటి వివరములు తెలియజేయవలసి ఉంటుంది.
b) పరిశోధకురాలు ఎత్తు మరియు నడుము చుట్టుకొలత యొక్క కొలతలు కలిగి ఉన్న పూర్తిస్థాయి భౌతిక పరీక్ష చేసెదరు.
c) పరిశోధకురాలు గ్లైసెమిక్ నియంత్రణ , మూత్రపిండ ప్రొఫైల్ , లిపిడ్ ప్రొఫైల్ మరియు థైరాయిడ్ ప్రొఫైల్కు సంబంధించి ప్రయోగశాల పరీక్షలు నిర్వహించడానికి 8 మిల్లిలిటర్ల రక్తం నా నుండి సేకరించెదరు.

d) నా వ్యక్తిగత విషయములు నా వ్రాతపూర్వక సమ్మతి లేకుండా ఎవరికి తెలుపబడవు .

e) నేను ఈసమ్మతి ఎటువంటి ఒత్తిడి లేకుండా ఇస్తున్నాను .

f) అధ్యయన వ్యవధిలో ఏదైన ఆరోగ్య పరమయినసంక్లిష్టత ఏర్పాడిన యెడలపరిశోధకురాలు జాగ్రత్త
తీసుకోనును .

అధ్యయనంలో పాల్గొనే వ్యక్తి సంతకం

పరిశోధకురాలి సంతకం

INSTITUTIONAL ETHICS COMMITTEE
AMC-KGH VISAKHAPATNAM

REG NO: ECR/197/Inst/KGH/2013/RR-16

LETTER OF APPROVAL

SERIAL NO: 45/IEC AMC-KGH/NOV/2018

This letter of approval is hereby accorded to **Dr. GRACE MADHURI JOHN** (Department of General Medicine) for conducting the research work entitled **"Clinical Study of type2 diabetes mellitus at presentation and its relation to glycemic status"** after the necessary scientific evaluation and ethical review of the above cited research protocol, by the **INSTITUTIONAL ETHICS COMMITTEE AMC-KGH VISAKHAPATNAM.**

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KEY TO MASTER SHEET

IP	Inpatient Number
OP	Outpatient number
FH	Family History
BMI	Body Mass index
WHR	Waist Hip Ratio
HTN	Hypertension
IHD	Ischaemic Heart disease
CVA	Cerebro vascular accident
PVD	Peripheral vascular disease
FBS	Fasting Blood sugar
PPBS	Post prandial blood sugar
HbA1c	Glycosalated Hemoglobin
HDL	High Density lipo protein
LDL	Low density lipo protein
TG	Triglyceride
TC	Total cholesterol

Sl. No.	Name	IP Number	Sex	Age (yrs)	FH	BMI	WHR	Retinopathy	P.neuropathy	Nephropathy	Acute Hyperglycemi	HTN	IHD	CVA	PVD	Respiratory Infections	Other infe. (Skin/UTI)	Asymptomatic	Others	FBS (mg/dl)	PPBS (mg/dl)	HbA1C (%)	HDL(mg/dl)	LDL (mg/dl)	TC (mg/dl)	TG (mg/dl)	Urinary protein
1	sumithra devi	10589	F	60	-	21.4	0.9	+	+	-		-	-	-	-	-	-			228	347	8.8	-	R	R	-	-
2	santhamma	10843	F	70	-	18.6	0.8	+	+	+		+	-	-	-	-	-			216	209	7.5	-	-	-	-	-
3	anandamma	10945	F	66	+	16.6	0.82	+	-	-		-	-	-	-	-	-			290	338	10.2	-	R	-	-	-
4	sardar kaur	11951	M	52	-	19.2	0.74	-	-	-		-	-	-	-	-	-			133	266	7.2	-	-	-	-	+
5	paramesh	12314	M	48	-	25.8	0.93	+	-	+		-	-	-	-	-	-			209	239	9.7	-	-	-	-	2+
6	Laxamanna	12831	M	52	-	21.68	0.98	+	-	-		-	-	-	-	-	-			141	217	8.2	-	-	-	-	-
7	basha	12831	M	33	+	22.9	1.02	-	-	-		-	-	+	-	-	-			168	263	9.8	-	R	R	R	-
8	visaladevi	13517	F	50	-	27.8	0.95	-	+	-		-	-	-	-	-	-			188	309	8	-	-	-	-	-
9	Ibrahimsab	14364	M	46	-	18.5	0.86	-	-	-		-	-	-	-	-	-			140	254	6.8	-	-	-	-	-
10	chandra shekhar	14721	M	56	-	24.55	0.8	-	-	-		+	-	+	-	-	-			243	378	10.3	-	R	R	R	-
11	Razia	14816	F	35	+	22.13	0.92	-	-	-		-	-	-	-	-	-		+	214	314	9.8	-	-	-	-	-
12	venkatesh	14911	M	36	-	22.67	1	-	+	-		-	-	-	-	-	-			259	401	10.1	-	-	-	-	+
13	chandravathi	15260	F	52	-	20.88	0.87	+	-	-	HHS +	+	-	-	-	-	-			610	0	13.2	L	R	R	R	-
14	tata babu	16324	M	60	+	16.4	0.9	-	-	-		-	-	-	-	-	-			139	246	7.5	-	-	-	-	-
15	Padmavathi	16411	F	46	+	23.2	0.9	-	-	-		+	-	-	-	-	-	+		156	236	7.5	-	-	-	-	-
16	rajeswari	16413	F	53	+	25.72	0.9	-	-	-		-	-	-	-	-	-	+		183	255	10	-	-	-	-	-
17	danayya	17004	M	70	+	25.21	0.9	+	-	-		+	+	-	-	-	-			164	274	8.5	-	-	-	-	-
18	someswara rao	17214	M	70	-	23.6	0.96	-	-	-	DKA +	-	-	-	-	-	-			528	0	11.5	-	R	-	R	-
19	himaja devi	17468	F	36	+	29	0.92	-	-	-		-	-	-	-	-	-	+		187	206	7.4	-	-	R	-	-
20	subba rao	18344	M	36	+	20.96	0.76	+	-	-		-	-	-	-	-	-			352	468	10.9	L	R	-	-	-

Sl. No.	Name	IP Number	Sex	Age (yrs)	FH	BMI	WHR	Retinopathy	P.neuropathy	Nephropathy	Acute Hyperglycemia	HTN	IHD	CVA	PVD	Respiratory Infections	Other infe. (Skin/UTI)	Asymptomatic	Others	FBS (mg/dl)	PPBS (mg/dl)	HbA1C (%)	HDL(mg/dl)	LDL (mg/dl)	TC (mg/dl)	TG (mg/dl)	Urinary protein
21	Lalithamma	18383	F	70	-	25.06	0.82	-	-	-		-	+	-	-	-	-			252	376	10.4	-	R	-	-	-
22	fathima	18414	F	43	+	27.63	0.94	-	-	-		-	-	-	-	-	-		sur +	234	306	8.2	L	R	-	-	-
23	Dyanakumari	18481	F	50	+	29.14	0.9	-	-	-		-	-	-	-	+	-			152	218	6.9	-	-	-	-	-
24	diwakar	18492	M	50	-	16.6	0.87	-	-	-		-	-	-	-	+	-			214	280	8.9	-	-	-	-	-
25	sagar babu	18739	**M**	47	-	22.4	0.8	-	-	-		-	-	-	-	-	-	+		282	374	9.4	-	-	-	-	-
26	prameela	18831	F	56	-	23.7	1.1	+	-	-		-	-	-	-	-	-			140	222	7.4	-	-	-	-	-
27	sri ramulu	18914	M	50	-	21.2	1	+	-	-		-	-	-	-	-	-			194	281	7	-	R	-	-	-
28	rangamma	19374	F	63	-	24.1	0.9	-	-	+		-	-	-	-	-	-			280	421	8.7	-	-	-	-	3+
29	sujatha	19472	F	40	+	28.8	0.97	-	-	-	DKA +	-	-	-	-	-	-			597	0	11.4	-	-	-	-	-
30	jyothi	19494	F	56	-	23.95	1.1	-	-	-		-	+	-	-	-	-			148	287	10.2	-	-	-	-	-
31	Anwarsab	20985	M	45	+	24.17	1	+	-	-		-	-	-	-	-	-	+		214	254	8.2	-	-	-	-	-
32	pavithra	21054	F	60	-	21.4	0.9	-	+	-		-	-	-	-	-	-			228	347	8.8	-	R	R	-	-
33	jagan	21231	M	70	-	23.6	0.96	-	-	-	DKA +	-	-	-	-	-	-			528	0	10.5	-	R	-	R	-
34	nagmabe	21643	F	37	+	30.44	0.96	-	-	-		-	-	-	-	-	-			162	218	6.6	-	R	R	R	-
35	surya rao	21895	M	41	+	23.3	0.94	-	-	-		-	-	-	-	-	-			213	510	9.2	-	R	-	-	-
36	sadaanand	21903	M	42	-	23.5	0.97	-	-	-		-	-	-	-	-	-			214	382	8.4	-	-	-	-	-
37	veer reddy	21940	M	70	-	23.8	0.9	+	-	+		-	-	-	-	-	-			253	413	10.2	-	-	-	-	3+
38	Siddaram	23014	M	53	-	21.7	0.92	+	+	-		-	-	-	-	-	+	skin +		256	411	9.8	-	-	-	-	-
39	pentayya	23152	M	70	+	25.21	0.9	-	-	-		-	+	-	-	-	-			164	274	8.5	-	-	-	-	-
40	Kamalabai	23583	F	58	-	18.3	0.92	-	-	-	DKA +	-	-	-	-	-	-			488	0	12.9	-	R	-	-	2+

Sl. No.	Name	IP Number	Sex	Age (yrs)	FH	BMI	WHR	Retinopathy	P.neuropathy	Nephropathy	Acute Hyperglycemia	HTN	IHD	CVA	PVD	Respiratory Infections	Other infe. (Skin/UTI)	Asymptomatic	Others	FBS (mg/dl)	PPBS (mg/dl)	HbA1C (%)	HDL(mg/dl)	LDL (mg/dl)	TC (mg/dl)	TG (mg/dl)	Urinary protein
41	annapurna	23648	F	46	-	31.2	0.9	-	-	+		-	-	-	-	-	-			144	206	9.7	-	-	-	-	3+
42	grandhi ram	23913	M	37	-	20.7	0.88	-	-	-		-	-	-	-	-	-	+		240	256	7.4	-	-	-	-	-
43	hari babu	23944	M	34	+	24.9	1.02	-	-	-		-	-	+	-	-	-			168	263	9.8	-	R	R	R	-
44	Mahabalesh	24019	M	42	+	24.65	0.96	-	-	-		-	-	-	-	-	-		RTA +	156	212	8.8	-	-	-	-	-
45	kameswari	24236	F	46	-	18.7	0.8	-	-	-		-	-	-	-	-	+			244	288	7.5	-	-	-	-	-
46	shiva	24732	M	64	-	21.52	0.9	+	-	-		-	-	-	-	-	-			175	255	8.2	-	-	-	-	-
47	bhagya	25382	F	66	-	33.2	1.1	-	-	-		-	+	-	-	-	-		+ ort	178	252	7.2	-	-	-	-	-
48	gunamma	26825	F	67	-	25.1	0.89	-	-	-		-	-	+	-	-	-			181	229	8.2	-	-	R	R	-
49	jamuna	27264	F	38	+	27	0.92	-	-	-		-	-	-	-	-	-	+		187	206	7.4	-	-	R	-	-
50	Durgamma	27437	F	54	-	20.36	1	-	-	-		-	+	-	-	-	-			161	385	10.4	-	R	-	-	-
51	savithri	28197	F	54	+	26.72	0.9	-	-	-		-	-	-	-	-	-	+		183	255	10	-	-	-	-	-
52	yashoda	28490	F	52	-	30.13	1	-	-	-		+	+	-	-	-	-			160	299	8.7	L	R	R	R	-
53	girija	28598	F	56	-	20.5	0.94	+	-	-		-	-	-	-	-	-			262	409	8.8	-	-	-	-	-
54	nataraj	28742	M	65	-	20.7	0.98	+	-	-		-	-	-	-	-	-			185	355	9.4	-	-	-	-	+
55	nagoor bash	28781	M	67	-	21.3	0.9	-	-	-		-	+	-	-	-	-	+		207	330	8.3	-	R	R	R	-
56	nagaraju	29023	M	60	-	23.2	0.91	+	-	-		-	-	-	-	-	-			146	243	7	-	-	-	-	-
57	karunakaram	29302	M	44	-	22.49	0.93	-	-	+		-	-	-	-	-	-	+		233	324	7.1	-	-	-	-	-
58	durga rao	29479	M	45	-	28.76	0.92	-	-	-		-	-	-	-	-	-	+		165	234	8.3	-	-	-	-	-
59	Nagaraj	29752	M	62	-	25.34	1.04	+	-	-		-	-	-	-	-	-			231	251	6.6	-	-	-	-	+
60	Girijamma	29752	F	58	-	20.5	0.94	+	-	-		-	-	-	-	-	-			262	409	8.8	-	-	-	-	-

Sl. No.	Name	IP Number	Sex	Age (yrs)	FH	BMI	WHR	Retinopathy	P.neuropathy	Nephropathy	Acute Hyperglycemia	HTN	IHD	CVA	PVD	Respiratory Infections	(Skin/UTI)	Asymptomatic	Others	FBS (mg/dl)	PPBS (mg/dl)	HbA1C (%)	HDL(mg/dl)	LDL (mg/dl)	TC (mg/dl)	TG (mg/dl)	Urinary protein
61	madhusudan	29984	M	56	+	17	0.73	-	-	+		-	-	-	-	-	-			259	356	11.6	-	R	R	R	2+
62	mohan rao	30691	M	36	-	22.09	0.84	-	-	-		-	-	-	-	-	+		Skin +	199	275	9.8	-	R	R	-	-
63	hari	32172	M	45	+	24.05	1.1	+	+	-		-	-	-	-	-	-			176	238	10.4	L	R	-	-	+
64	basheer	32231	M	63	+	26.52	1.1	+	-	-		-	-	-	-	-	-			239	343	9.8	-	-	-	-	+
65	ramesh	32372	M	42	-	27.4	1.1	-	-	-		-	-	+	-	-	-			227	279	8	L	R	-	R	-
66	geetha	32394	F	56	-	26.42	0.8	-	-	-		-	+	-	-	-	-			309	300	11.2	-	-	-	-	+
67	Thungabhdram	32483	F	67	-	40.7	1.1	-	-	-		-	-	-	-	-	-		+ surg	198	303	11.7	-	-	-	-	-
68	naga babu	32579	M	43	+	32.8	0.98	-	-	-		-	+	-	-	-	-			188	297	9.2	L	R	R	R	-
69	Shankar rao	33847	M	50	-	21.2	1	+	-	-		-	-	-	-	-	-			194	281	7	-	R	-	-	-
70	thowdamma	34039	F	50	+	23.4	1.1	+	+	-		-	-	-	-	-	-			286	475	9	-	R	-	-	-
71	maya devi	34042	F	51	-	24.6	1	-	-	-		-	-	-	+	-	-			161	327	8	-		-	-	+
72	lakshmi	34101	F	44	-	30.04	0.9	-	-	-		-	+	-	-	-	-			222	278	7.8	-	R	-	R	+
73	ramayya	34217	M	53	-	21.7	0.92	-	-	-		-	-	-	-	-	+	skin +		256	411	9.8	-	-	-	-	-
74	bharath babu	34294	M	43	-	22.7	0.8	-	-	-		-	-	-	-	+	-			237	328	8.9	-	-	-	-	-
75	Rudrakar	34621	M	52	-	22.8	0.9	-	-	-		-	-	-	-	-	+			190	202	8	-	-	-	-	+
76	Iqbal Beig	34763	M	50	-	25.03	0.92	+	-	-		-	-	-	-	-	-			178	268	9.6	-	-	-	R	+
77	swamy	34874	M	35	+	25.7	0.92	-	+	-		-	-	-	-	-	-			312	376	10.3	-	R	-	-	-
78	himakar rao	35651	M	42	-	27.4	1.1	-	-	-		-	-	+	-	-	-			227	279	8	L	R	-	R	-
79	appa rao	36248	M	62	-	26.4	1.1	+	-	+		-	-	-	-	-	-			209	279	11.1	L	R	R	R	2+
80	surappa	36478	M	39	-	20.3	1.01	-	-	-		-	-	+	-	-	-			296	434	11.8	-	R	R	R	-

Sl. No.	Name	IP Number	Sex	Age (yrs)	FH	BMI	WHR	Retinopathy	P.neuropathy	Nephropathy	Acute Hyperglycemia	HTN	IHD	CVA	PVD	Respiratory Infections	(Skin/UTI)	Asymptomatic	Others	FBS (mg/dl)	PPBS (mg/dl)	HbA1C (%)	HDL(mg/dl)	LDL (mg/dl)	TC (mg/dl)	TG (mg/dl)	Urinary protein
81	vara prasad	37328	M	38	+	19.9	0.9	-	-	-		+	-	-	-	+	-			237	279	9.6	-	R	-	-	-
82	atchim naidu	38371	M	70	-	23.8	0.9	+	-	+		+	-	-	-	-	-			253	413	10.2	-	-	-	-	3+
83	Bibijan	38728	F	45	-	21.06	0.82	+	-	-		-	-	-	-	-	-			212	268	10.8	-	-	-	-	-
84	gangadhar	38911	M	62	-	13.7	0.7	+	-	-		-	+	-	-	-	-			268	437	11.5	-	-	-	-	-
85	swamy naidu	38985	M	35	+	25.7	0.92	-	+	-		-	-	-	-	-	-			312	376	10.6	-	R	-	-	-
86	Saikumar	39034	M	44	-	21.49	0.93	-	-	-		-	-	-	-	-	-	+		233	324	7.1	-	-	-	-	-
87	suresh babu	39154	M	44	+	23.3	0.94	+	-	-		-	-	-	-	-	-			213	510	9.2	-	R	-	-	-
88	sarveswara	39310	M	46	+	25.06	1	-	-	-		+	+	-	-	-	-			281	298	8.3	-	R	-	R	+
89	satyanarayana	39822	M	63	+	26.52	1.1	+	-	-		-	-	-	-	-	-			239	343	9.8	-	-	-	-	+
90	chandra paul	39992	M	52	-	19.2	0.74	-	-	-		-	-	-	-	-	-			133	266	7.2	-	-	-	-	+
91	Srinivas.A	40992	M	51	-	30.34	1	-	-	-		+	+	-	-	-	-			223	374	11.2	-	R	-	R	-
92	Shobha	41126	F	52	-	20.88	0.87	+	-	-	HHS +	-	-	-	-	-	-			510	0	13.2	L	R	R	R	-
93	ananda babu	41363	M	45	+	24.17	1	+	-	-		-	-	-	-	-	-	+		214	254	8.2	-	-	-	-	-
94	rajeshwar rao	41768	M	57	-	28.68	1.08	-	-	-		-	-	-	-	-	-	+		154	279	7.5	-	R	-	R	-
95	chandra	43578	F	46	-	18.7	0.8	-	-	-		-	-	-	-	-	+			244	288	7.5	-	-	-	-	-
96	Pavan Kumar	43864	M	37	+	22.7	0.9	-	-	-		-	-	-	-	-	-	+		152	242	7.2	-	-	-	-	-
97	laxman kumar	43892	M	52	-	21.68	0.98	+	-	-		-	-	-	-	-	-			141	217	8.7	-	-	-	-	-
98	kanthamma	45032	F	56	-	18.3	0.92	-	-	-	DKA +	-	-	-	-	-	-			588	0	12.9	-	R	-	-	2+
99	dayamani	45115	F	41	+	32.8	0.98	-	-	-		-	+	-	-	-	-			188	297	9.2	L	R	R	R	-
100	vijay prasad	45632	M	50	-	16.6	0.87	-	-	-		-	-	-	-	-	-			214	280	8.9	-	-	-	-	-

Sl. No.	Name	IP Number	Sex	Age (yrs)	FH	BMI	WHR	Retinopathy	P.neuropathy	Nephropathy	Acute Hyperglycemia	HTN	IHD	CVA	PVD	Respiratory Infections	(Skin/UTI)	Asymptomatic	Others	FBS (mg/dl)	PPBS (mg/dl)	HbA1C (%)	HDL(mg/dl)	LDL (mg/dl)	TC (mg/dl)	TG (mg/dl)	Urinary protein
101	Nazeer Sab	45736	M	67	-	22.86	0.9	+	-	-		-	-	-	-	-	-	+		207	330	8.3	-	R	R	R	-
102	aamani	45922	F	46	-	31.2	0.9	-	-	+		-	-	-	-	-	-			144	206	9.2	-	-	-	-	3+
103	aravindu	47592	M	35	-	20.7	0.84	-	-	-		-	-	+	-	-	-			168	297	9.7	-	-	-	-	-
104	ahmad	47661	M	52	-	21.63	1	-	-	+		-	-	-	-	-	-			222	366	8.8	-	-	-	-	2+
105	kumari	48102	F	51	-	22.6	1	-	-	-		-	-	-	+	-	-			161	327	8	-		-	-	+
106	satyavathi	48221	F	50	-	30.49	1	+	-	-		-	-	-	-	-	-			281	453	10.3	-	R	-	-	-
107	rama rao	48382	M	47	-	19.05	1	+	-	-		-	+	-	-	-	-			249	402	9.8	L	R	-	R	-
108	b srinivas	49192	M	52	-	30.34	1	+	-	-		+	+	-	-	-	-			223	374	11.2	-	R	-	R	-
109	mallesh rao	49200	M	45	-	26.73	1.1	-	+	-	HHS +	-	-	-	-	-	-			561	0	9.7	-	R	-	R	+
110	rajendra	49892	M	43	-	34.07	1	-	-	-		-	+	-	-	-	-	+		149	269	8.1	-	R	-	R	Traces
111	janaki devi	50135	F	66	+	16.6	0.82	-	-	-		-	-	-	-	+	-			290	338	10.2	-	R	-	-	-
112	Mansoor	50136	M	67	-	23.02	0.9	-	-	-		+	-	-	-	-	+	skin +		311	409	11	-	R	-	R	-
113	girish	50348	M	37	-	20.7	0.88	-	-	-		-	-	-	-	-	-	+		240	256	7.4	-	-	-	-	-
114	siva prasad	51069	M	46	+	25.06	1	-	-	-		+	+	-	-	-	-			281	298	9.3	-	R	-	R	+
115	vasantha	51201	F	44	-	30.04	0.9	-	-	-		-	+	-	-	-	-			222	278	7.8	-	R	-	R	+
116	uma maheswara rao	51318	M	45	-	28.76	0.92	-	-	-		-	-	-	-	-	-	+		165	234	8.3	-	-	-	-	-
117	ganga devi	52314	F	70	-	26	1.1	+	-	+		+	-	-	-	-	-			212	311	9.2	-	-	-	-	+
118	begum	52495	F	62	+	22.1	0.9	-	-	+		-	-	-	-	-	-			280	421	8.7	-	-	-	-	3+
119	ganga	52834	F	45	-	23.06	0.82	+	-	-		-	-	-	-	-	-			212	268	10.8	-	-	-	-	-
120	visweswara rao	52914	M	65	-	25.23	1	+	-	-		-	-	-	-	-	-			209	315	9.6	-	-	-	-	-

Sl. No.	Name	IP Number	Sex	Age (yrs)	FH	BMI	WHR	Retinopathy	P.neuropathy	Nephropathy	Acute Hyperglycemia	HTN	IHD	CVA	PVD	Respiratory Infections	(Skin/UTI)	Asymptomatic	Others	FBS (mg/dl)	PPBS (mg/dl)	HbA1C (%)	HDL(mg/dl)	LDL (mg/dl)	TC (mg/dl)	TG (mg/dl)	Urinary protein
121	Narayan	52961	M	65	-	20.7	0.98	+	-	-		-	-	-	-	-	-			185	355	9.4	-	-	-	-	+
122	bhimeswar	52984	M	46	-	19.98	0.9	-	-	-		-	-	-	-	-	-			249	397	12.1	-	R	-		+
123	Gangamma	53095	F	50	+	21.4	1.1	+	+	-		-	-	-	-	-	-			286	475	9	-	R	-	-	-
124	srinivas setty	53292	M	43	-	34.07	1	-	-	-		-	+	-	-	-	-	+		149	269	8.1	-	R	-	R	Traces
125	ratnamma	54291	F	70	-	18.6	0.8	-	-	-		-	-	-	-	-	-			216	209	7.2	-	-	-	-	-
126	venku naidu	54327	M	36	-	22.09	0.84	-	-	-		-	-	-	-	-	+		Skin +	199	275	9.8	-	R	R	-	-
127	ganapathi	54906	M	61	+	25.72	0.9	+	+	-		-	+	-	-	-	-	+		251	374	9.2	-	R	-	-	-
128	jagadeeswara rao	55329	M	50	-	25.39	1	+	-	+		-	-	-	-	-	-			293	318	10.6	-	R	-	R	3+
129	bhaskar	56023	M	46	-	19.71	0.9	+	-	-		-	-	-	-	-	-			249	397	12.2	-	R	-	-	+
130	madhavi	56523	F	38	-	23.42	0.82	-	-	-		-	-	-	-	-	+		UTI	235	265	9.7	-	-	-	-	-
131	kannayya	58342	M	66	-	23.52	0.9	+	-	-		-	-	-	-	-	-			175	255	8.2	-	-	-	-	-
132	polamma	58398	F	59	-	23.7	1.1	+	-	-		-	-	-	-	-	-			140	222	7.4	-	-	-	-	-
133	mahendra	60041	M	45	-	29.72	1.1	-	+	-	HHS +	-	-	-	-	-	-			561	0	9.7	-	R	-	R	+
134	raja rao	60321	M	63	+	25.72	0.9	-	+	-		-	-	-	-	-	-	+		251	374	9.2	-	R	-	-	-
135	Mohan	62109	M	56	+	17	0.73	-	-	+		-	-	-	-	-	-			259	356	11.6	-	R	R	R	2+
136	sarath kumar	62458	M	34	+	28.73	0.94	-	+	-		-	-	-	-	-	-			165	234	7	-	-	-	-	-
137	chaamundeswari	63293	F	60	-	23.2	0.91	+	-	-		-	-	-	-	-	-			146	243	7	-	-	-	-	-
138	Subbash	63427	M	36	+	20.96	0.76	+	-	-		-	-	-	-	-	-			352	468	10.9	L	R	-	-	-
139	durga devi	63743	F	54	-	20.36	1	-	-	-		-	+	-	-	-	-			161	385	10.4	-	R	-	-	-
140	Padmavathi	66471	F	46	+	23.2	0.9	-	-	-		+	-	-	-	-	-	+		156	236	7.5	-	-	-	-	-

Sl. No.	Name	IP Number	Sex	Age (yrs)	FH	BMI	WHR	Retinopathy	P.neuropathy	Nephropathy	Acute Hyperglycemia	HTN	IHD	CVA	PVD	Respiratory Infections	(Skin/UTI)	Asymptomatic	Others	FBS (mg/dl)	PPBS (mg/dl)	HbA1C (%)	HDL(mg/dl)	LDL (mg/dl)	TC (mg/dl)	TG (mg/dl)	Urinary protein
141	appalraju	67523	M	67	-	23.02	0.9	+	-	-		+	-	-	-	-	+	skin +		311	409	11	-	R	-	R	-
142	omkar	68641	M	30	-	25.7	0.87	-	-	-		-	-	-	-	-	-		+	195	231	7	-	-	-	-	-
143	suseela	69545	F	40	+	26.6	0.97	-	-	-	DKA +	-	-	-	-	-	-			519	0	11.4	-	-	-	-	-
144	chinnaswamy	71323	M	36	-	21.67	1	-	+	-		-	-	-	-	-	-			259	401	10.1	-	-	-	-	+
145	shivani	71641	F	34	+	28.73	0.94	-	+	-		-	-	-	-	-	-			165	234	7	-	-	-	-	-
146	vijay kumar	73014	M	54	-	22.8	0.9	-	-	-		-	-	-	-	-	+			190	202	8	-	-	-	-	+
147	jogendra	73243	M	46	-	18.5	0.86	-	-	-		-	-	-	-	-	-			140	254	6.8	-	-	-	-	-
148	Basavaraj	73441	M	70	-	19.5	0.9	+	-	-		-	-	-	-	-	-			189	241	8.2	-	-	-	-	-
149	rama devi	73846	F	47	+	22.37	1	-	-	+		-	-	-	-	-	-			215	269	6.9	-	-	-	-	3+
150	balakrishna	74275	M	43	-	22.7	0.8	-	-	-		-	-	-	-	+	-			237	328	8.9	-	-	-	-	-
151	chidambaram	74331	M	62	-	26.4	1.1	+	-	+		-	-	-	-	-	-			209	279	11.1	L	R	R	R	2+
152	sudheer babu	74578	M	30	-	25.7	0.87	-	-	-		-	-	-	-	-	-		+	195	231	7	-	-	-	-	-
153	govinda rao	74929	M	62	-	13.7	0.7	+	-	-		-	+	-	-	-	-			268	437	11.5	-	-	-	-	-
154	Prahalad	76369	M	36	+	19.9	0.9	-	-	-		+	-	-	-	-	-			237	279	8.6	-	R	-	-	-
155	mallika devi	77418	F	53	-	34.4	1	+	-	-		+	+	-	-	-	-			325	400	12.8	L	-	R	-	-
156	radha krishna	77459	M	45	+	24.05	1.1	+	+	-		-	-	-	-	-	-			176	238	10.4	L	R	-	-	+
157	Kanaka mahalakshmi	78324	F	52	+	18.9	0.9	-	-	-		-	-	-	-	-	-			197	355	8.4	-	-	-	-	-
158	rajesh kumar	78328	M	47	-	19.05	1	+	-	-		-	+	-	-	-	-			249	402	9.8	L	R	-	R	-
159	kanna rao	78346	M	60	+	16.4	0.9	-	-	-		-	-	-	-	+	-			139	246	7.2	-	-	-	-	-
160	Hanumavva	78348	F	66	-	25.1	0.89	-	-	-		-	-	+	-	-	-			181	229	8.2	-	-	R	R	-

Sl. No.	Name	IP Number	Sex	Age (yrs)	FH	BMI	WHR	Retinopathy	P.neuropathy	Nephropathy	Acute Hyperglycemia	HTN	IHD	CVA	PVD	Respiratory Infections	(Skin/UTI)	Asymptomatic	Others	FBS (mg/dl)	PPBS (mg/dl)	HbA1C (%)	HDL(mg/dl)	LDL (mg/dl)	TC (mg/dl)	TG (mg/dl)	protein
161	Suresh	78487	M	47	-	25.8	0.93	+	-	+		-	-	-	-	-	-			209	239	8.7	-	-	-	-	2+
162	vimala	78781	F	50	-	27.8	0.95	-	+	-		-	-	-	-	-	-			188	309	8	-	-	-	-	-
163	manjari	81318	F	48	-	27.06	0.98	-	-	-		-	-	-	-	-	-	+		179	259	8.4	-	-	-	-	-
164	sri raam	81942	M	47	-	21.4	0.8	-	-	-		-	-	-	-	-	-	+		282	374	9.4	-	-	-	-	-
165	yerra babu	82314	M	36	+	21.79	0.86	-	-	-		-	-	-	-	-	-	+		269	429	9.3	-	-	-	-	-
166	srinu	82428	M	42	-	23.5	0.97	+	-	-		-	-	-	-	-	-			214	382	8.4	-	-	-	-	-
167	jagadamba	82718	F	52	+	18.9	0.9	-	-	-		-	-	-	-	-	-			197	355	8.4	-	-	-	-	-
168	deva raj	83252	M	30	+	21.3	1	+	-	-		-	-	-	-	-	-			238	264	9	-	R	-	-	-
169	bhagavanulu	83281	M	70	-	19.5	0.9	+	-	-		-	-	-	-	-	-			189	241	8.2	-	-	-	-	-
170	radhaa devi	83742	F	70	-	25.06	0.82	-	+	-		-	+	-	+	-	-			252	376	10.1	-	R	-	-	-
171	upendra	83811	M	56	-	22.49	0.9	+	-	-		-	-	-	-	-	-			288	355	7.1	-	R	-	-	-
172	rama murthy	84389	M	50	-	25.03	0.92	+	-	-		-	-	-	-	-	-			178	268	9.6	-	-	-	R	+
173	devamani	84622	F	50	+	29.14	0.9	-	-	-		-	-	-	-	+	-			152	218	6.9	-	-	-	-	-
174	yasoda devi	84832	F	52	-	30.13	1	-	-	-		+	+	-	-	-	-			160	299	8.1	L	R	R	R	-
175	Ashabee	85622	F	36	-	23.42	0.82	+	-	-		-	-	-	-	-	+		UTI	235	265	9.7	-	-	-	-	-
176	matthew	86348	M	48	-	27.06	0.98	-	-	-		-	-	-	-	-	-	+		179	259	8.4	-	-	-	-	-
177	prasad rao	86484	M	39	-	20.3	1.01	-	-	-		-	-	+	-	-	-			296	434	11.8	-	R	R	R	-
178	Savitha	86813	F	50	-	30.49	1	+	-	-		-	-	-	-	-	-			281	453	10.3	-	R	-	-	-
179	ramani	87124	F	53	-	34.4	1	+	-	-		+	+	-	-	-	-			325	400	12.8	L	-	R	-	-
180	eswara rao	87189	M	36	+	21.79	0.86	+	-	-		-	-	-	-	-	-	+		269	429	9.3	-	-	-	-	-

Sl. No.	Name	IP Number	Sex	Age (yrs)	FH	BMI	WHR	Retinopathy	P.neuropathy	Nephropathy	Acute Hyperglycemia	HTN	IHD	CVA	PVD	Respiratory Infections	(Skin/UTI)	Asymptomatic	Others	FBS (mg/dl)	PPBS (mg/dl)	HbA1C (%)	HDL(mg/dl)	LDL (mg/dl)	TC (mg/dl)	TG (mg/dl)	protein
181	Putamma	87214	F	56	-	26.42	0.8	-	-	-		-	+	-	-	-	-			309	300	11.2	-	-	-	-	+
182	gowramma	87248	F	70	-	26	1.1	+	-	+		+	-	-	-	-	-			212	311	9.2	-	-	-	-	+
183	jagapathi raju	87384	M	65	-	25.23	1	+	-	-		-	-	-	-	-	-			209	315	9.6	-	-	-	-	-
184	Pavan Kumar	87493	M	39	+	22.7	0.9	-	-	-		-	-	-	-	-	-	+		152	242	7.2	-	-	-	-	-
185	raja babu	87582	M	30	+	23.3	1	-	-	-		-	-	-	-	-	-			238	264	9	-	R	-	-	-
186	raghavendra	87989	M	57	-	24.55	0.8	-	-	-		+	-	+	-	-	-			243	378	10.3	-	R	R	R	-
187	Anjaneeya	89126	M	52	-	21.63	1	-	-	+		-	-	-	-	-	-			222	366	8.8	-	-	-	-	2+
188	devi	89127	F	43	+	26.24	0.94	-	-	-		-	-	-	-	-	-		sur +	234	306	9.2	L	R	-	-	-
189	simhachalam	89520	M	42	+	24.65	0.96	-	-	-		-	-	-	-	-	-		RTA +	156	212	8.8	-	-	-	-	-
190	khan	89841	M	35	-	20.7	0.84	-	-	-		-	-	+	-	-	-			168	297	8.7	-	-	-	-	-
191	shanthi	90274	F	37	+	30.44	0.96	-	-	-		-	-	-	-	-	-			162	218	6.6	-	R	R	R	-
192	rani	90537	F	35	+	22.13	0.92	-	-	-		-	-	-	-	-	-		+	214	314	8.8	-	-	-	-	-
193	Thungabhdram	92950	F	66	-	40.7	1.1	-	-	-		-	-	-	-	-	-		+ surg	198	303	11.7	-	-	-	-	-
194	maheswara rao	93215	M	56	-	22.49	0.9	+	-	-		-	-	-	-	-	-			288	355	7.1	-	R	-	-	-
195	Nagaraj	94522	M	64	-	25.34	1.04	+	-	-		-	-	-	-	-	-			231	251	6.6	-	-	-	-	+
196	kiran kumar	97342	M	50	-	25.39	1	+	-	+		-	-	-	-	-	-			293	318	10.6	-	R	-	R	3+
197	parvathi	97659	F	47	+	22.37	1	-	-	+		-	-	-	-	-	-			215	269	6.5	-	-	-	-	3+
198	Bagyamma	98313	F	67	-	33.2	1.1	-	-	-		-	+	-	-	-	-		+ ort	178	252	7.2	-	-	-	-	-
199	mamatha	98343	F	56	-	23.95	1.1	-	-	-		-	+	-	-	-	-			148	287	10.2	-	-	-	-	-
200	ganesh	10328	M	56	-	28.68	1.08	-	-	-		-	-	-	-	-	-	+		154	279	6.5	-	R	-	R	-