A STUDY ON PREVALANCE OF PROSTATOMEGALY AND ITS CORRELATION WITH DURATION AND GLYCEMIC CONTROL IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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CERTIFICATE

This is to certify that the dissertation titled "**A STUDY ON PREVALANCE OF PROSTATOMEGALY AND ITS CORRELATION WITH DURATION AND GLYCEMIC CONTROL IN PATIENTS WITH TYPE 2 DIABETES MELLITUS**" is a bonafide work done **by DR.S.PRABHAKARAN** , Post graduate student, Institute of Internal Medicine, Madras Medical College, Chennai -03, in partial fulfillment of the University Rules and Regulations for the award of Degree of MD General Medicine (Branch – I), Internal Medicine, under our guidance and supervision, during the academic year 2014 – 2017.

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DECLARATION

I solemnly declare that the dissertation titled "**A STUDY ON PREVALANCE OF PROSTATOMEGALY AND ITS CORRELATION WITH DURATION AND GLYCEMIC CONTROL IN PATIENTS WITH TYPE 2 DIABETES MELLITUS**" is done by me at Madras Medical College , Chennai – 600 003 during the period April 2016 to September 2016 under the guidance and supervision of **Prof**. **Dr.S.TITO.M.D.,** submitted to the Tamilnadu Dr.M.G.R Medical University towards the partial fulfillment of requirements for the award of M.D. DEGREE IN GENERAL MEDICINE (BRANCH-I).

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INTRODUCTION

INTRODUCTION

Diabetes mellitus considerably contributes to the development and the deterioration of lower urinary tract symptoms. Patients with type 2 Diabetes mellitus are more prone to be diagnosed with benign prostatic hyperplasia and subsequently subjected to prostatectomy than general male population. This fact signifies that the scientific value of the epidemiologic association between benign prostatic hyperplasia and type 2 Diabetes through lower urinary tract symptoms.

Diabetes mellitus affects bladder function producing both obstructive and irritative symptoms, with the classic triad of obstructive symptoms :

- ✓ difficulty initiating voiding,
- ✓ fullness after voiding, and
- ✓ increased post void residual urine volume

characterizing diabetic cystopathy .Similarly, frequency and urgency are associated with both Diabetes-induced detrusor instability and benign prostatic hyperplasia.

From patients with hypertrophy of prostate those with the higher levels of serum glucose (>110mg/dL) had a considerably higher mean prostate volume in comparison with patients with low levels of serum glucose. In patients with lower urinary tract symptoms , a further increase in prostate growth rate with the increasing levels of serum insulin was noticed.

Abnormalities of glucose homeostasis could play a role in the cause of BPH by influencing the proliferation rate of prostate cells. Thus, the study aims at identifying the correlation between duration and glycemic control of Diabetes and prevalence of prostatomegaly.

AIMS AND OBJECTIVES

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PRIMARY AIM:

To study the prevalance of prostatomegaly and morphological charachteristics of prostate in patients with type 2 Diabetes Mellitus in comparison with non diabetic control.

SECONDARY AIM:

1. To study the effect of prostatomegaly and its complications with duration of type 2 Diabetes.
2. To study the effect of prostatomegaly and its complications with glycemic control of type 2 Diabetes.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

INTRODUCTION

Diabetes includes a group of metabolic abnormalities showing phenotype of hyperglycaemia. This metabolic dysregulation leading to Diabetes also leads to secondary pathophysiological changes in multi organ systems, imposing an excessive burden on Patients with Diabetes and also towards health care system predisposing patients to cardiovascular disease.

Factors contributing to hyperglycaemia:

- Reduced insulin secretion
- Decreased utilization of glucose
- Increased production of glucose [1].

Due to an increased incidence worldwide, Diabetes will soon become a leading cause of morbidly and mortality.

CLASSIFICATION OF DIABETES

Diabetes classified based on pathogenic process leading to hyperglycaemia in opposition to criteria such as age of onset, type of therapy which were used earlier.

TABLE 417-1 ETIOLOGIC CLASSIFICATION OF DIABETES MELLITUS

I. Type 1 diabetes (beta cell destruction, usually leading to absolute insulin deficiency)
   A. Immune-mediated
   B. Idiopathic

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

III. Other specific types of diabetes
   A. Genetic defects of beta cell development or function characterized by mutations in:
      1. Hepatocyte nuclear transcription factor (HNF) 4α (MODY 1)
      2. Glucokinase (MODY 2)
      3. HNF-1α (MODY 3)
      4. Insulin promoter factor-1 (IPF-1; MODY 4)
      5. HNF-1β (MODY 5)
      6. NeuroD1 (MODY 6)
      7. Mitochondrial DNA
      8. Subunits of ATP-sensitive potassium channel
      9. Proinsulin or insulin
      10. Other pancreatic islet regulators/proteins such as *KLF11, PAX4, BLK, GATA4, GATA6, SLC2A2* (GLUT2), *RFX6, GLIS3*
   B. Genetic defects in insulin action
      1. Type A insulin resistance
      2. Leprechaunism
      3. Rabson-Mendenhall syndrome
      4. Lipodystrophy syndromes
   C. Diseases of the exocrine pancreas—pancreatitis, pancreatectomy, neoplasia, cystic fibrosis, hemochromatosis, fibrocalculous pancreatopathy, mutations in carboxyl ester lipase
   D. Endocrinopathies—acromegaly, Cushing's syndrome, glucagonoma, pheochromocytoma, hyperthyroidism, somatostatinoma, aldosteronoma
   E. Drug- or chemical-induced—glucocorticoids, vacor (a rodenticide), pentamidine, nicotinic acid, diazoxide, β-adrenergic agonists, thiazides, calcineurin and mTOR inhibitors, hydantoins, asparaginase, α-interferon, protease inhibitors, antipsychotics (atypicals and others), epinephrine
   F. Infections—congenital rubella, cytomegalovirus, coxsackievirus
   G. Uncommon forms of immune-mediated diabetes—"stiff-person" syndrome, anti-insulin receptor antibodies
   H. Other genetic syndromes sometimes associated with diabetes—Wolfram's syndrome, Down's syndrome, Klinefelter's syndrome, Turner's syndrome, Friedreich's ataxia, Huntington's chorea, Laurence-Moon-Biedl syndrome, myotonic dystrophy, porphyria, Prader-Willi syndrome

IV. Gestational diabetes mellitus (GDM)

ETIOLOGICAL CLASSIFICATION OF DIABETES

TYPE 1 – results from a complete or total absence of insulin

TYPE 2 – includes diverse group of diseases involving:

- Various levels of resistance to insulin,
- Insulin secretory impairment,
- Excessive production of glucose.

Abnormal homeostasis of glucose is usually seen prior to developing overt Diabetes.

Even though type 1 Diabetes occurs prior to the age 30,autoimmune destructive process towards beta cell develops at any age.5-10 percent of patients developing Diabetes after age 30 is observed to have type 1 Diabetes Mellitus[2].

Type 2 Diabetes occurs mostly with increased age, in recent times being observed amongst young adults.

MODY AND MONOGENIC DIABETES MELLITUS

Involves autosomal dominant inheritance

Delayed onset of hyperglycaemia (average of 25 years)

GESTATIONAL DIABETES MELLITUS

- Glucose intolerance during pregnancy
- Due to resistance to insulin secondary to metabolic changes of late pregnancy
- Increased requirement of insulin
- Incidence – on an average 7 percent

Most of whom revert to normal in the postpartum period (with a risk of 25- 50 percent for having Diabetes Mellitus in the following 10 years).

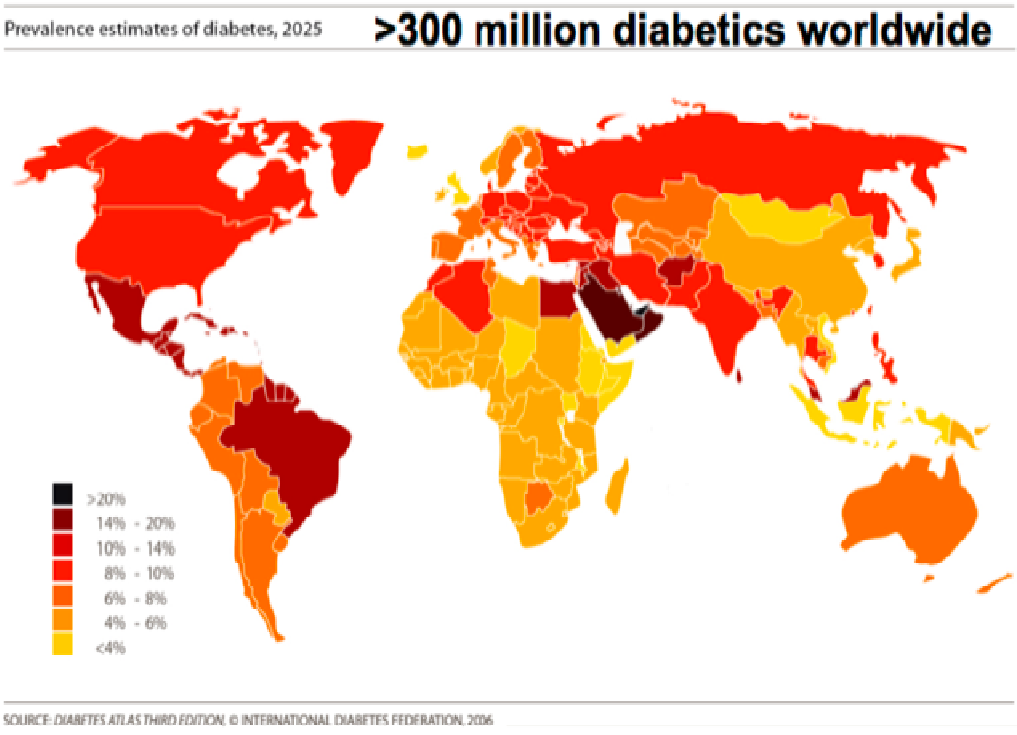
IADP STUDY GROUPS AND ADA suggests once Diabetes is detected at an early prenatal screening can be grouped as overt Diabetes mellitus instead of Gestational Diabetes Mellitus. Due to increasing incidence of obesity, patients with gestational Diabetes mellitus/ overt Diabetes is on rise.

EPIDEMIOLOGY AND GLOBAL CONSIDERATIONS

Diabetic prevalence rose dramatically over the past two decades, from around 32 million patients in 1987 to about 382 million patients in 2014. With the prevailing data, the IDF suggests that 592 million person would have DIABETES by the year 2035. Eventhough

prevalence of both the types of Diabetes are increasing globally, type 2 diabetic patients tend to grow much rapidly due to prevailing obesity, decreased activity levels.

The countries with highest count of patients with DIABETES mellitus in 2014 are India (62.2 million), China (97.3 million), United nations (24.6 million), Brazil (12.1 million), and Russia(11.9 million). In a recent study by Center for Disease Control and Prevention (CDC) showed that around 9.2% of the people has Diabetes (~28% of the patients with Diabetes are not diagnosed; worldwide, it is projected that around 50% of patients might be undiagnosed).



The CDC [3] has shown an estimate of prevalence that has doubled from year 1990–2008, but appeared to have plateaued from year 2008–2012. In 2013, type 2 Diabetes prevalence in United Sates is estimated to be 0.3% in patients aged<25 years and around 12% in individuals aged >20 years. Globally most of the individuals with Diabetes are within the age group of 40 and 59 years.

There are widespread geographic variations among the type 1 and type 2 Diabetes incidence. Scandinavia records the highest number of cases of type1 Diabetes; with the lowest number of cases in the Pacific Rim where it is around 20- to 25-fold lesser. This widespread variability is because of genetic, environmental and behavioural factors.

Prevelance of Diabetes varies amongst populations with different ethnicity inside a given country, with populations indigenous to the area having a greatest prevalence of type 2 Diabetes in comparison with the general population of that country.

Among Asians ,prevalence of Diabetes is on rising trend , and Diabetes phenotype appears to be varied from United states population, with onset at a younger age and a lower body mass index, reduced insulin secretory capacity and a greater visceral adiposity.

RISK FACTORS FOR TYPE 2 DIABETES

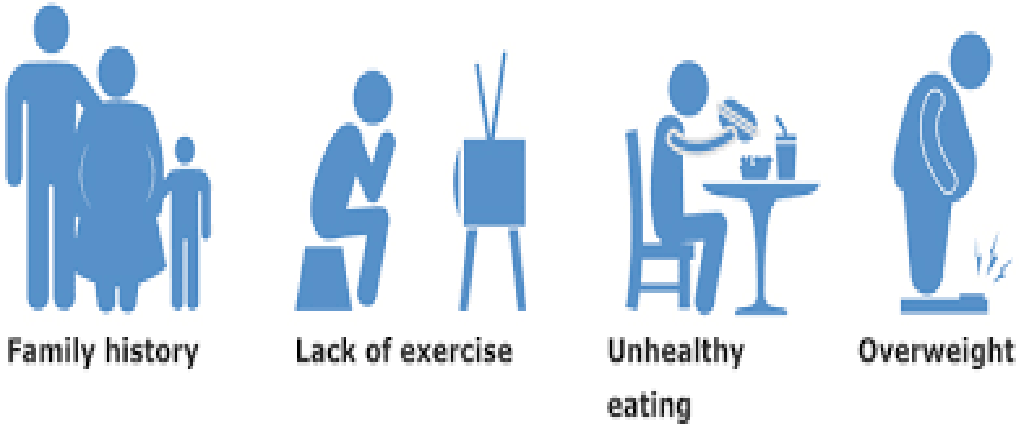
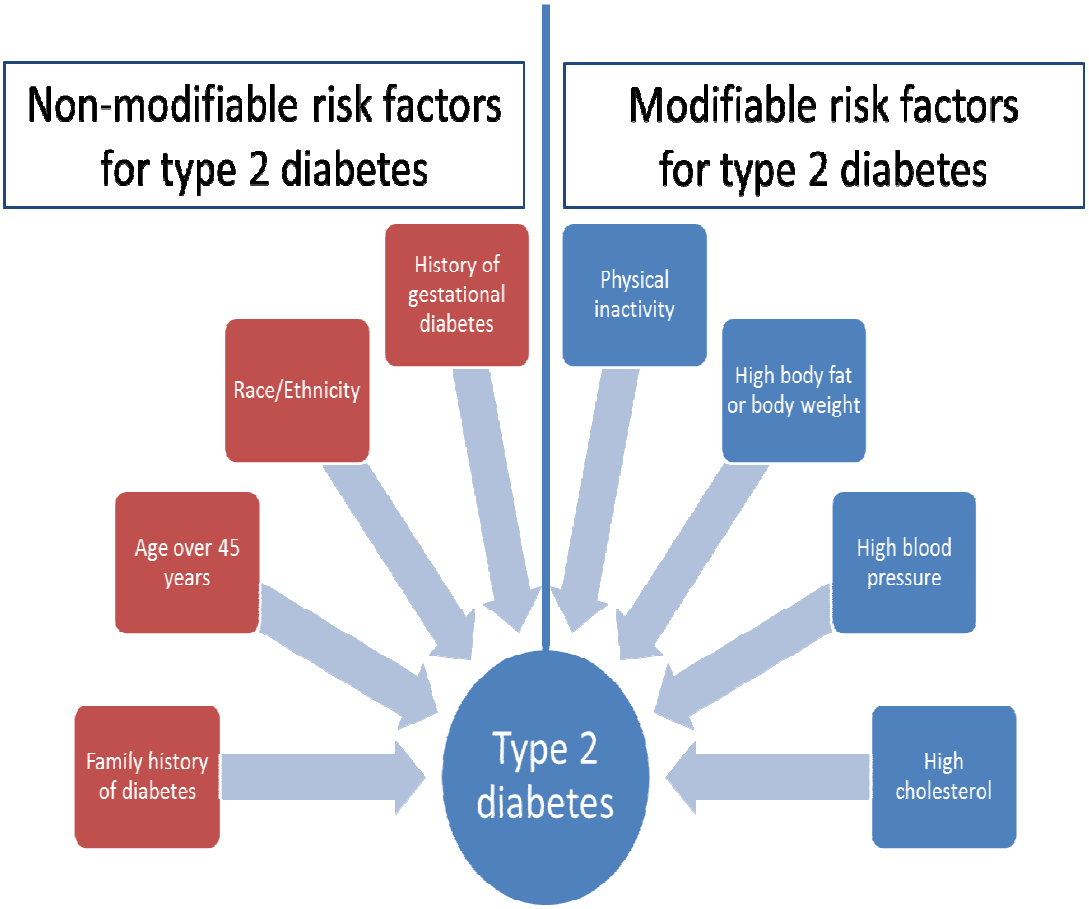


TABLE 417-3 RISK FACTORS FOR TYPE 2 DIABETES MELLITUS

Family history of diabetes (i.e., parent or sibling with type 2 diabetes)
Obesity (BMI ≥25 kg/m^2 or ethnically relevant definition for overweight)
Physical inactivity
Race/ethnicity (e.g., African American, Latino, Native American, Asian American, Pacific Islander)
Previously identified with IFG, IGT, or an hemoglobin A_{1c} of 5.7–6.4%
History of GDM or delivery of baby >4 kg (9 lb)
Hypertension (blood pressure ≥140/90 mmHg)
HDL cholesterol level <35 mg/dL (0.90 mmol/L) and/or a triglyceride level >250 mg/dL (2.82 mmol/L)
Polycystic ovary syndrome or acanthosis nigricans
History of cardiovascular disease



SCREENING

The Use of FPG or HbA1c as a screening test is recommended for type 2 DIABETES globally as:

- a large group of individuals meeting the current criterion for DIABETES are asymptomatic including those not aware that of having the disorder [4] ,
- epidemiologic studies predicts that type 2 Diabetes might be present up to more than a decade before proceeding to diagnosis,

- some of the individuals having type 2 Diabetes may be having more than one Diabetes-induced complications during diagnosis,
- Type 2 Diabetes treatment might alter the history of Diabetes favourably, diagnosis of these pre Diabetes helps in achieving a good prognosis.

ADA recommends screening all individuals who are >45 years once in every 3 years. It also recommends screening individuals at a much earlier age, in case of patients being overweight (BMI >25 kg/m2).In comparison with patients with type 2 Diabetes, a longer asymptomatic period of hyperglycemia is usually rare in patients having type 1 Diabetes prior to diagnosis.

Testing for Type 2 Diabetes and Prediabetes in Asymptomatic Adults

Type 2 diabetes screening should be performed in adults of any age who are overweight or obese, and who have one or more diabetes risk factor (*See Diabetes Risk Factors*)

- Testing should begin at age 45
- If test is normal? Repeat it at least every 3 years (*See Diabetes Risk Factors*):

Screening for prediabetes can be done using A1C, FPG, or 2-hr PG after 75-g OGTT criteria

- CVD risk factors should be identified and treated
- Testing may be considered in children and adolescents who are overweight or obese and have two or more risk factors for diabetes (*See Diabetes Risk Factors*)

TABLE 417-2 CRITERIA FOR THE DIAGNOSIS OF DIABETES MELLITUS

- Symptoms of diabetes plus random blood glucose concentration ≥11.1 mmol/L (200 mg/dL)[a] *or*
- Fasting plasma glucose ≥7.0 mmol/L (126 mg/dL)[b] *or*
- Hemoglobin A_{1c} ≥ 6.5%[c] *or*
- 2-h plasma glucose ≥11.1 mmol/L (200 mg/dL) during an oral glucose tolerance test[d]

REGULATION OF GLUCOSE HOMEOSTASIS

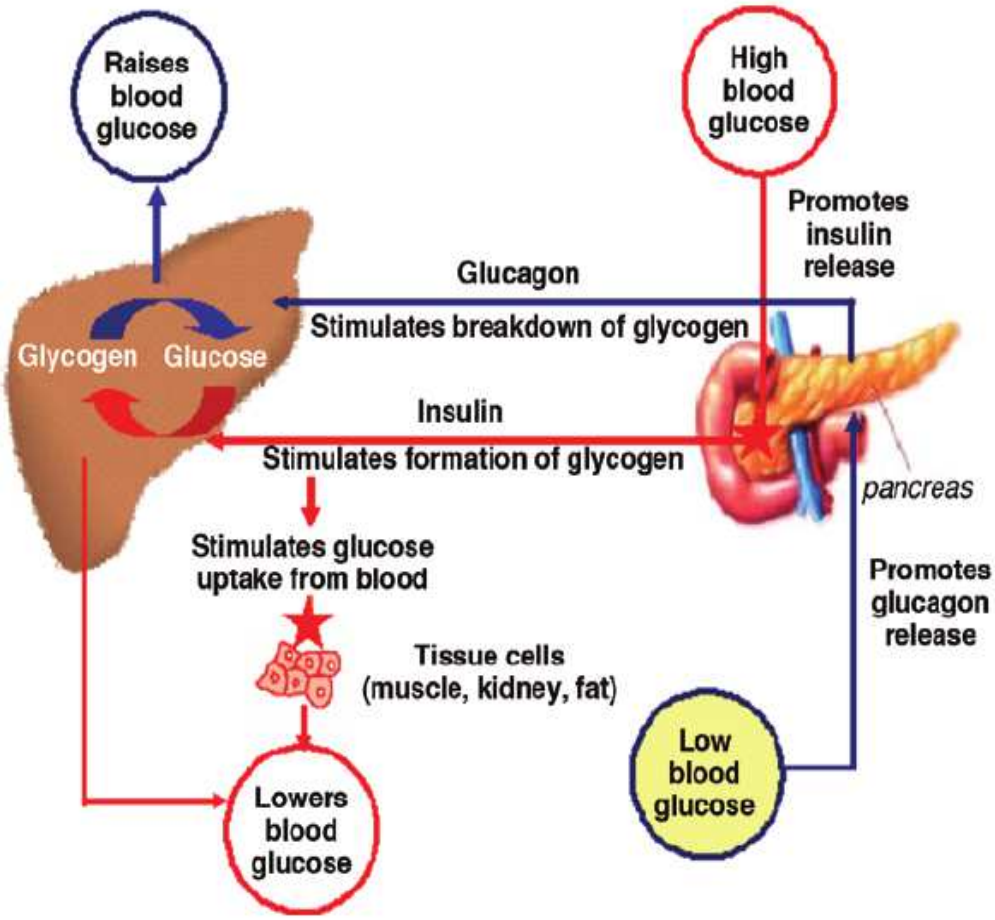
Glucose homeostasis is important in maintaining a balance between the hepatic production of glucose and the peripheral uptake and utilization of glucose. Insulin is an important regulator for this metabolic equilibrium, but metabolic signals, other hormones (glucagon) and neural input and resulting in integrated control of the supply and the utilization of glucose.

Organs regulating glucose and lipids tend to communicate by various neural and the humoral mechanisms [6] producing myo-kines,adipokines, and metabolites influencing liver function.

During fasting, low insulin levels may increase production of glucose by promoting glycogenolysis and gluconeogenesis with glucose uptake reduction in such insulin-sensitive tissues such as skeletal muscle and the fat , hence promote mobilization of stored precursor form of free fatty acids and amino acids .

Glucagon that is secreted by the alpha cells of pancreas whenever blood glucose levels or levels of insulin are low, stimulating glycogenolysis and gluconeogenesis by renal medulla and liver . Following a meal, glucose load leads to rise in insulin with fall in glucagon which leads to reversal of the above processes.

Insulin, which is an anabolic hormone, promotescarbohydrate storage with synthesis of protein and fat. A larger portion of these postprandial glucose is utilised by skeletal muscle, an insulin-stimulated glucose uptake mechanism. Other organs like brain, use glucose in the otherway, that is by an insulin-independent mechanism. Growth Factors secreted by the myocytes (irisin), fat cells (leptin, resis-tin, adiponectin) and bone may influence homeostasis of glucose too [5].

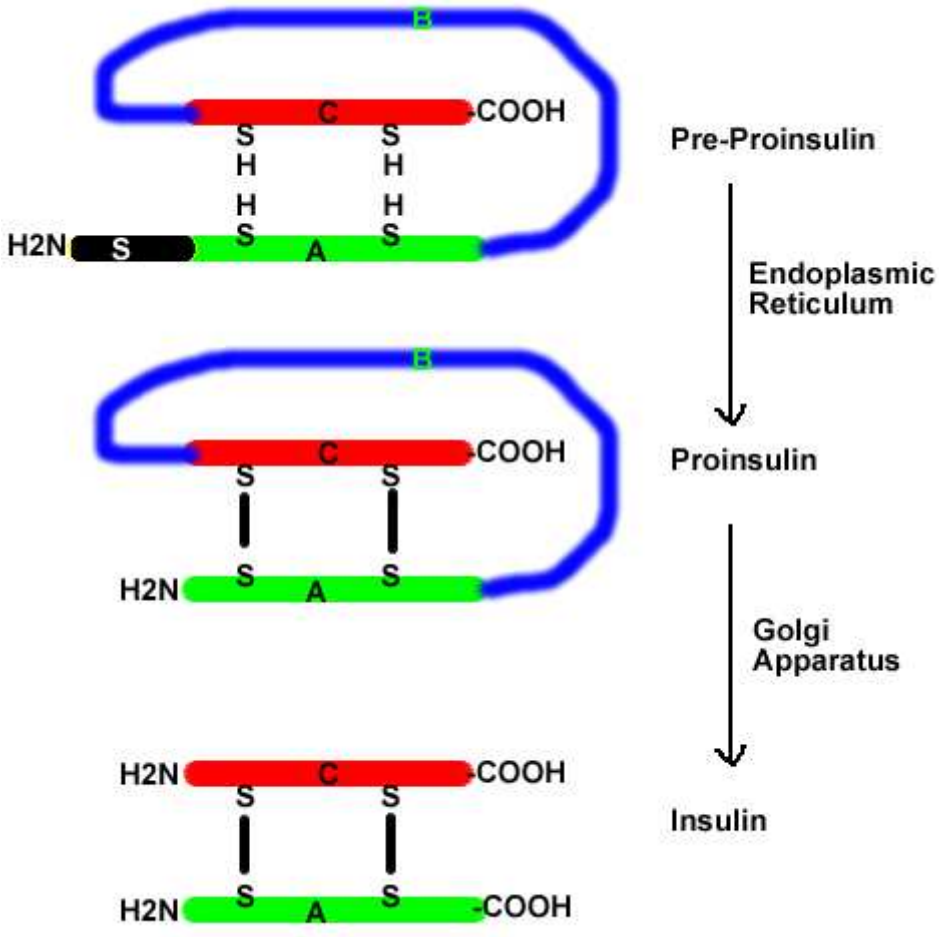


INSULIN BIOSYNTHESIS

Insulin produced by the beta cells in the islets of pancreas. Synthesis initially proceeds as a single-chain of 86-amino-acid precursor polypep-tide, preproinsulin. Further subsequent proteolytic processing aids in removing the amino-terminal signal peptide, thereby giving rise to the proinsulin.

Proinsulin is related morphologically to insulin-like growth factors I and II, that binds to insulin receptor weakly. Proinsulin [7] undergoes cleavage of 31 residue fragment leading to the generation of C peptide with A and B (30 chains of insulin, joined by disulfide bridges.

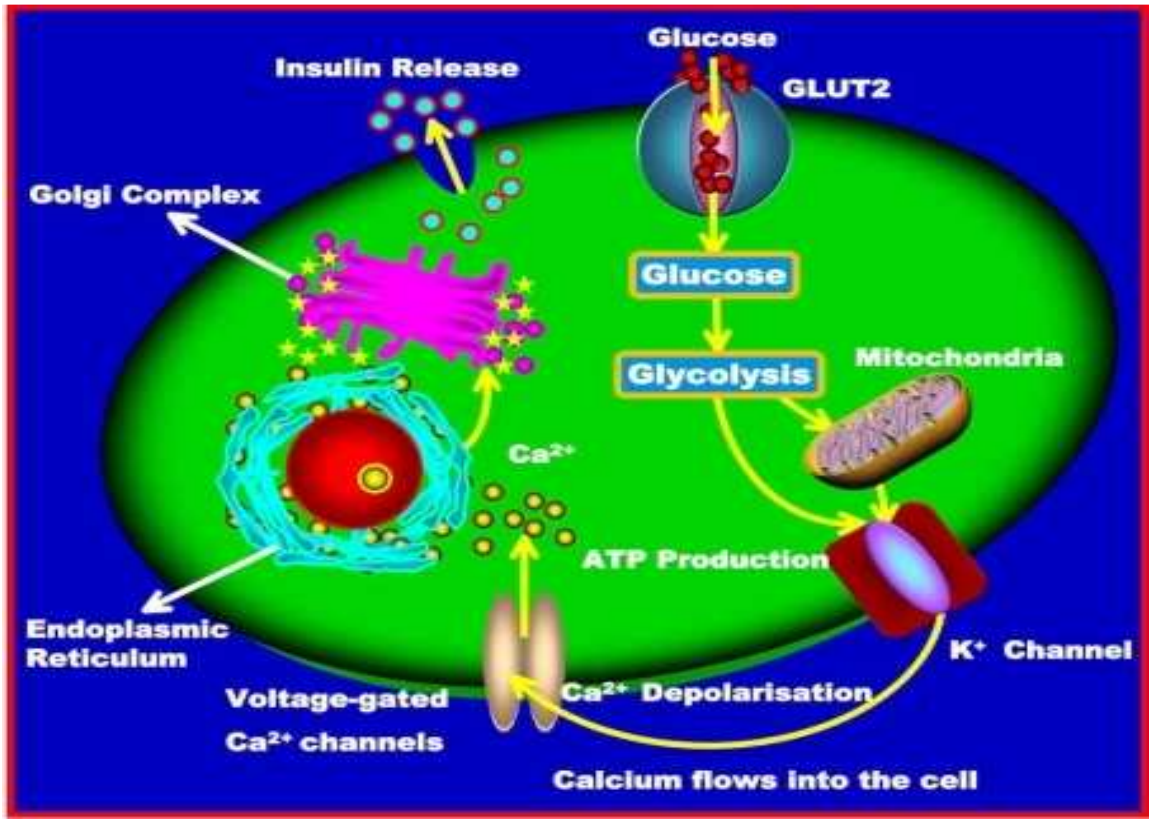
The above said molecules are stored together ,on stimation are secreted from the pancreatic beta cells granules. As C peptide is cleared much slower than insulin, being used as a marker for secretion of insulin. This helps in differentiation between exogenous and endogenous insulin during the hypoglycaemia evaluation.



Beta cells of pancreas also secrete amylin and islet amyloid polypeptide (IAPP) [9] together with insulin. The function of IAPP is incompletely defined yet , but it is considered a prime component of the amyloid fibrils present in pancreatic islets in type 2 DIABETES patients.

INSULIN SECRETION

Glucose is the principle source of secretion of insulinby pancreatic beta cells, even though ketones, amino acids, various other nutrients, gastro-intestinal peptides, key neurotransmitters may also influence insulin secretion.



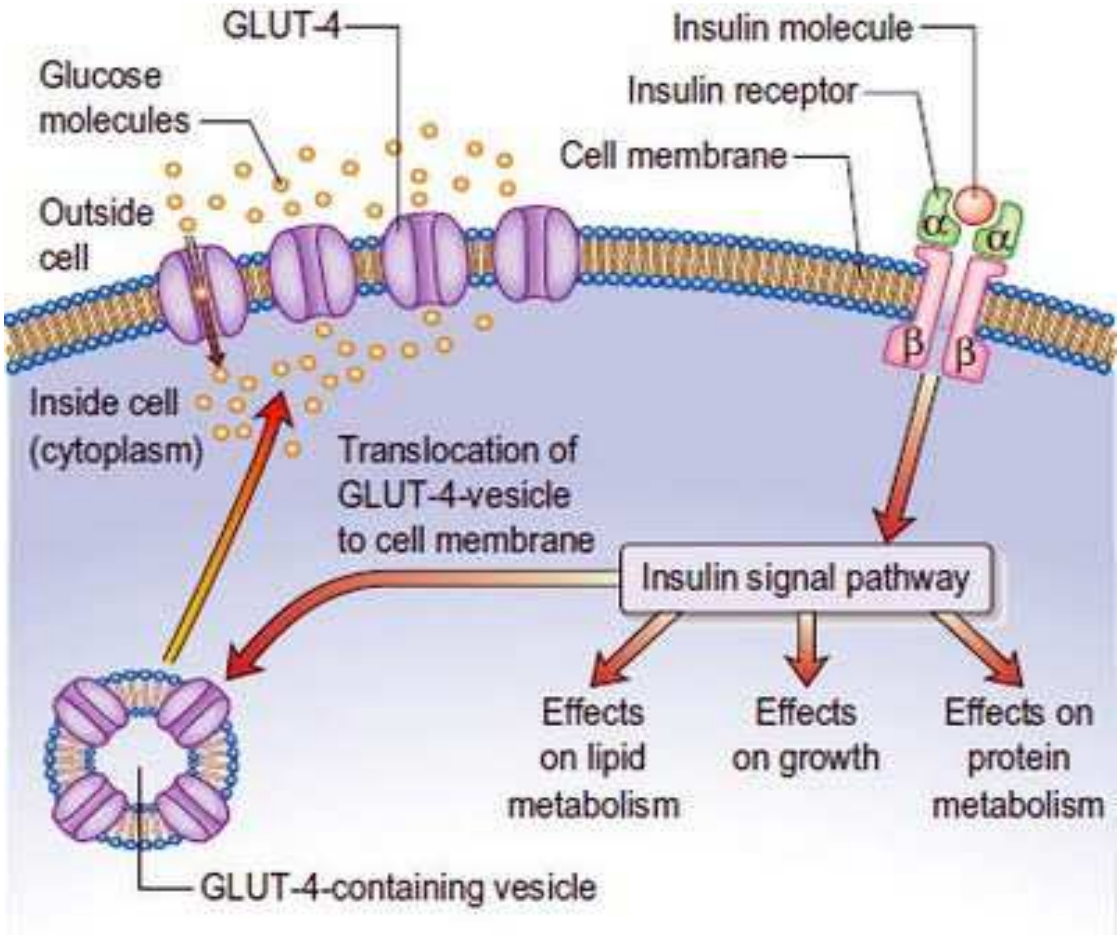
Glucose levels >70 milligram/dL stimulates synthesised insulin, by enhancement of translation of protein and processing. Glycemic stimulation of secretion of insulin is initiated by a facilitative glucose transporter into the beta cell of pancreas.

INSULIN ACTION

Once secretion of insulin occurs in to the portal venous system, nearly 55% is being taken up and thereby destroyed by the liver. Unextracted insulin entering systemic circulation leads to binding of insulin to receptors present in target sites. This process leads to stimulation of intrinsic tyrosine kinase activity, ultimately resulting in autophosphorylation of receptor and intracellular signaling molecule recruitment, like that of insulin receptor substrates (IRS).

Insulin receptor substrates (IRS) initiates a complex series of phosphorylation and dephosphorylation [8] reactions, leading to a enhanced metabolic changes and mitogenic actions of insulin. Phosphatidylinositol-3′-kinase (PI-3-kinase) pathway stimulation causes movement of glucose transporter (e.g., GLUT4) to the cell surface, playing a key role for uptake of glucose by myocytes and adipocytes. Insulin receptor stimulation induces signaling pathways thereby induces

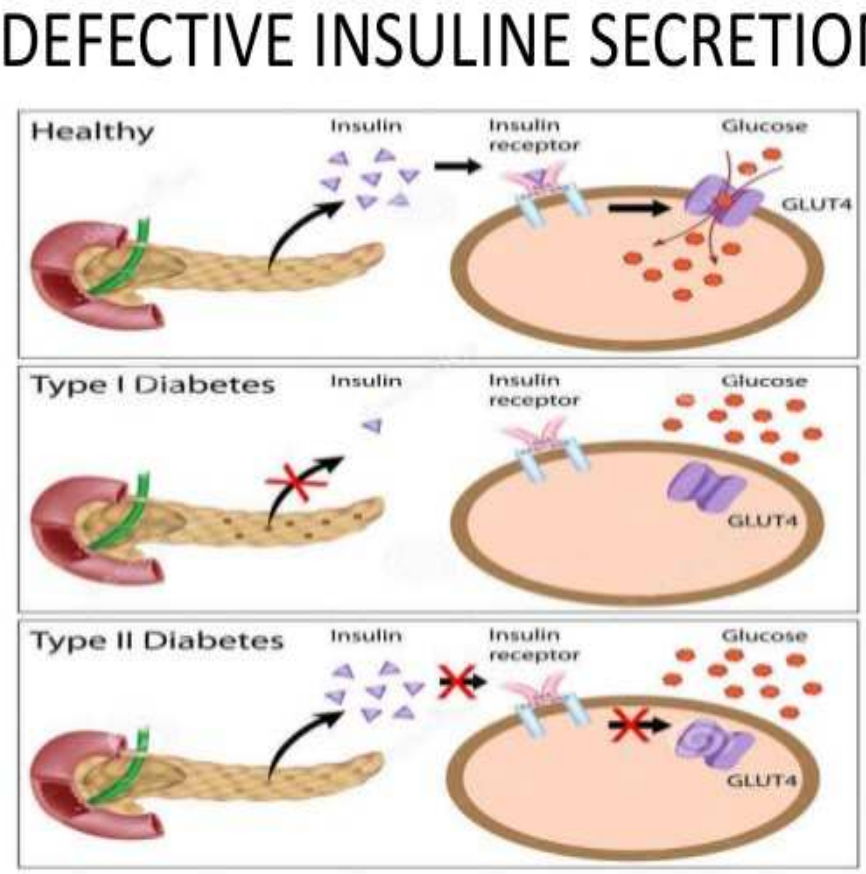
synthesis of glycogen, protein and fat and regulation of many other genes in cells that are responsive to insulin



PATHOGENESIS OF TYPE 2 DIABETES

Resistance to insulin and an reduced secretion of insulin are primary to the pathogenic development of type 2 Diabetes. Resistance to insulin usually precede an secretion of insulindefect but, development of Diabetes occurs only in situations where secretion of insulin reaches to a state of inadequacy. It has become increasingly apparent that Diabetes

has a different pathophysiological process in different ethnic groups (Asian, African, and Latin American).

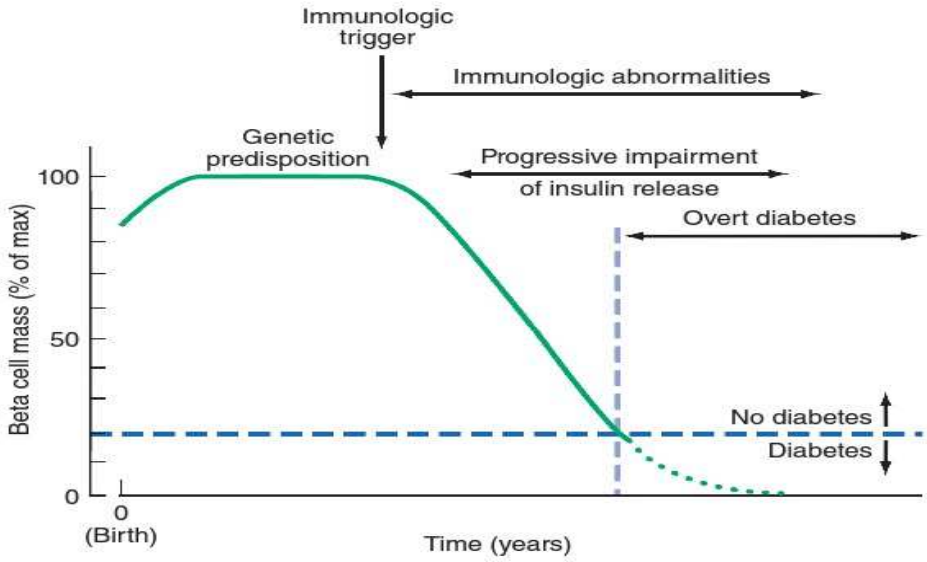


GENETIC CONSIDERATIONS

Type 2 Diabetes have a stronger genetic predisposition. Type 2 Diabetes concordance among identical [12]twins is about 72 to 88%. Individuals with type 2 diabetic parent acquire an much higher tendency to develop Diabetes; in case of type 2 Diabetes in both the parents, the risk reaches upto 38%.

Resistance to insulin, as evidenced by decreased utilization of glucose in peripheral myocytes, is also seen in nondiabetic, first-degree relatives of patients with type 2 Diabetes. The disease is multifactorial and also polygenic , consisting of genetic susceptibility, other environmental factors (such as obesity, dietary habits and physical inactivity) leads to phenotype modulation.

The in utero milieu of the foetus contributes , which can be either an increased or decreased birth weight leading to increase in the risk of type 2 Diabetes in adult life. The genes[11] that are predisposing to type 2 Diabetes are yet not completely defined, but more genome-wide association studies had discovered a much higher count of genes which convey a lesser tendency for type 2 Diabetes.



Major prominent feature is a variant of transcription factor 7–like 2 gene that is found to be linked with type 2 Diabetes in many individuals. The mechanism of genetic predisposition is likely to be alteration in the islet cell development or function, resulting in change of insulin secretion.

PATHOPHYSIOLOGY

Type 2 Diabetes is generally characterized by impairment of insulin secretion, resistance to insulin, increased production of glucose by the liver, and an altered adipose tissue metabolism. Obesity, especially visceral or central (as measured by the hip-waist ratio), is much more prevalent in type 2 Diabetes (≥75% of individuals are obese).

In the earlier stages of disorder, glucose tolerance comes to near normal, even in the presence of insulin resistance, as the pancreatic beta cells tends to compensate by improving the insulin output . As the resistance to insulin and a compensatory raise in insulin progresses, the islets of pancreas are not able to maintain the hyperinsulinemic[13] scenario. Impairment in glucose tolerance, that is characterized by a rise in postmeal glucose, thus develops.

A decline in secretion of insulin and an increment in glucose production by the liver thus leads to overt Diabetes along with fasting rise in glucose. Ultimately leading to failure of beta cells .Although both the resistance to insulin and impairment in secretion contributes to the underlying pathogenesis of type 2 Diabetes, the relative contribution of each of them varies among individuals.

METABOLIC ABNORMALITIES

ABNORMAL MUSCLE AND FAT METABOLISM

Resistance to insulin, leading to the reduced ability of insulin to function effectively on the target cells (especially on myocytes, heptocytes and adipocytes), is thus a prime feature of type 2 DIABETES and resulting from combination of genetic susceptibility and obesity.

Resistance to insulin is however relative, because increased circulating insulin levels will tend to normalize the serum glucose concenteration. Shift of insulin dose response curves to the rightward axis, thereby, indicating [15] decreased sensitivity, and low maximal response, thus leading to an overall decrement in maximual utilization of glucose.

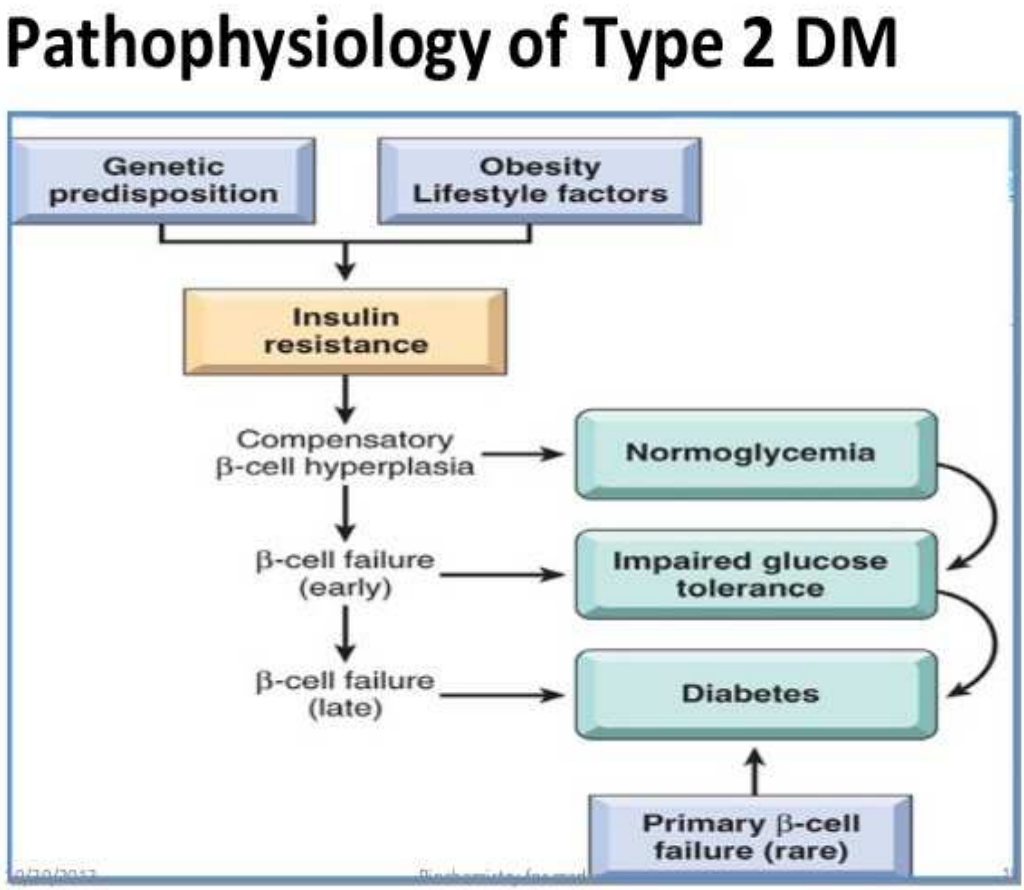
Resistance to insulin leads to impairment of utilization of glucose by the peripheral tissues resulting in a further increase in glucose output by the liver leading to a vicious cycle ; both effects contributing to the resulting hyperglycemia. Increment in glucose output by liver significantly causes raised Fasting plasma glucose levels, whereas reduced l glucose utilisation results in postmeal raised serum glucose.

In myocytes, there is higher impairment in nonoxidative utilisation of glucose than in oxidative glucose metabolism through the mechanism of glycolysis. Metabolism of glucose that occurs in insulin-independent tissues is not changed in type 2 Diabetes.

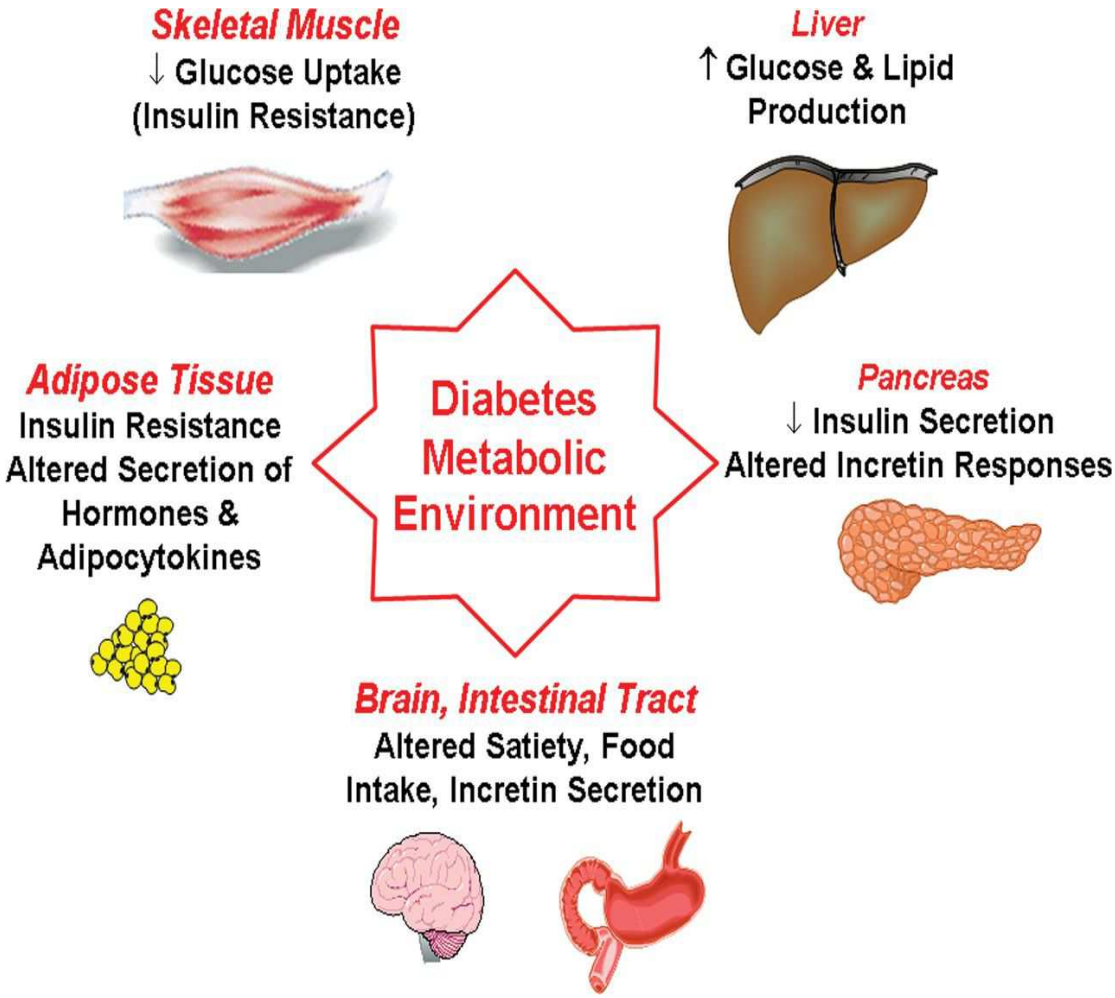
Levels of Insulin receptors and tyrosine kinase activity in myocytes are decreased, but these changes tends to be mostly due to hyperinsulinemia and aren't due to a basic defect. Therefore, these "postreceptor" defects that occurs in insulin dependent phosphorylation /dephosphorylation process appears to play a pivotal role in the resistance to insulin.

Alterations includes the lipid accumulation within myocytes, which might impair the oxidative phosphorylation in mitochondria and reduces insulin dependent mitochondrial production of ATP. Impairment

in oxidation of fat and storage of lipids within muscle also might release free radicles such as lipid peroxides [14].

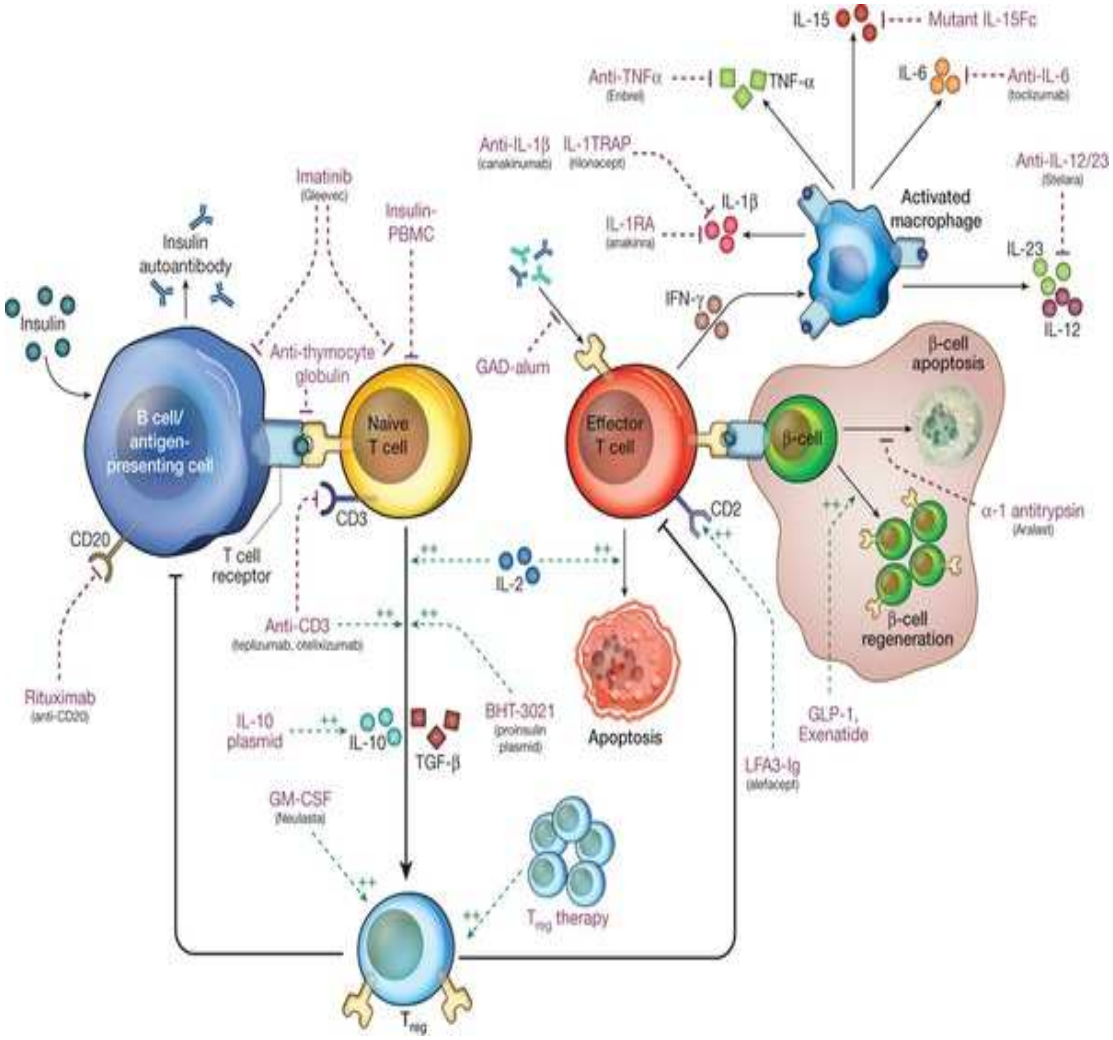


As a result, raised insulin levels can escalate the effects of insulin, thereby stimulating atherosclerotic changes in the vessel wall. Visceral or central obesity, is a part of this pathogenic process . Brown fats have a greater advantage as having thermogenic capcity compared to these white fat depots.



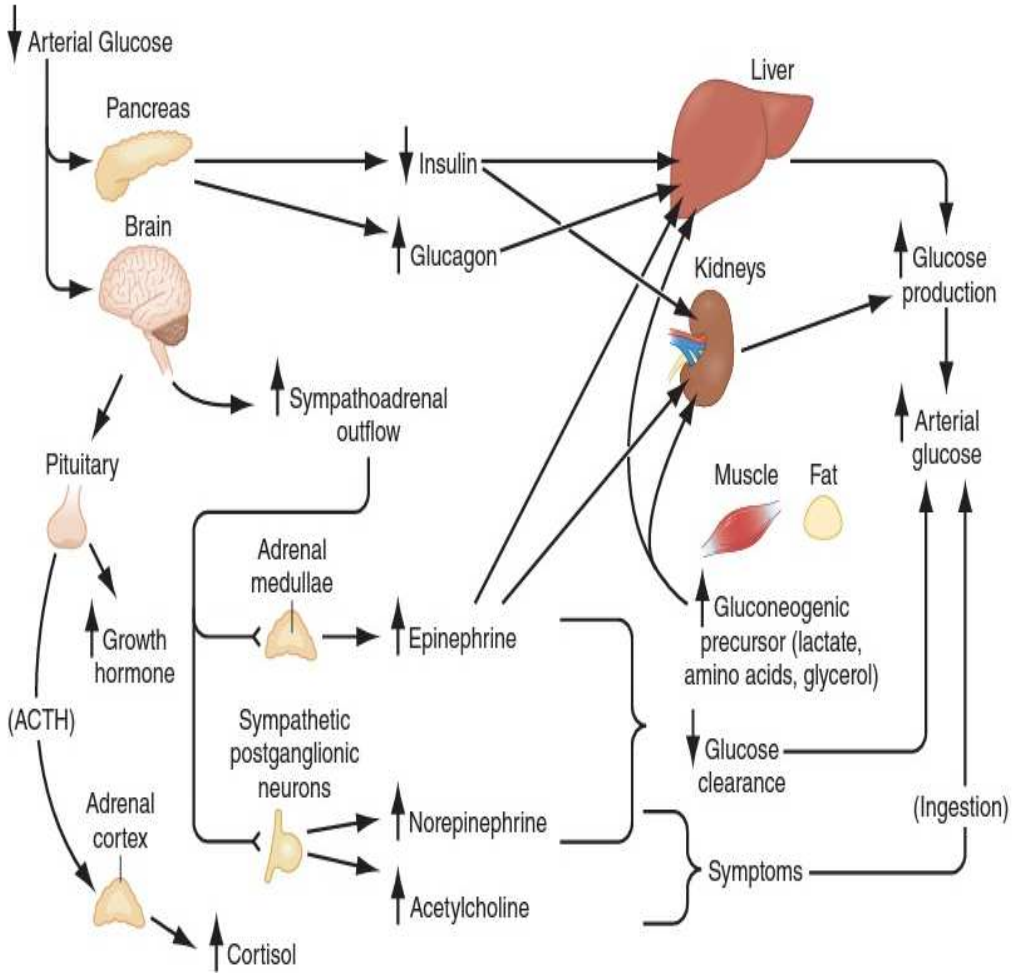
Obesity, in turn stimulates the synthesis and release of many enzymes and hormones (leptin, adipokines, retinol binding protein, Interleukin 6 , resistin and tumour necrosis factor alpha). These factors play a key role in the appetite stimulation , regulation of body weight , expenditure of energy and modulation of insulin sensitivity in the peripheral tissues.however, certain adipokines and free fatty acids leads to insulin resistance in myocytes and hepatocytes.

INFLAMMATORY CASCADE STIMULATED BY INSULIN



Production of excess free fatty acids in patients with obesity not only results in impairment of glucose by the peripheral tissues, but also causes excess production and releases of glucose from the liver with impairing the function of pancreatic beta cells [15]. In contrast to the scenario, releases of adiponection from the adipocytes, which is a insulin sensitizing peptide is much lowered in obese patients thereby, contributing to the elevated insulin resistance.

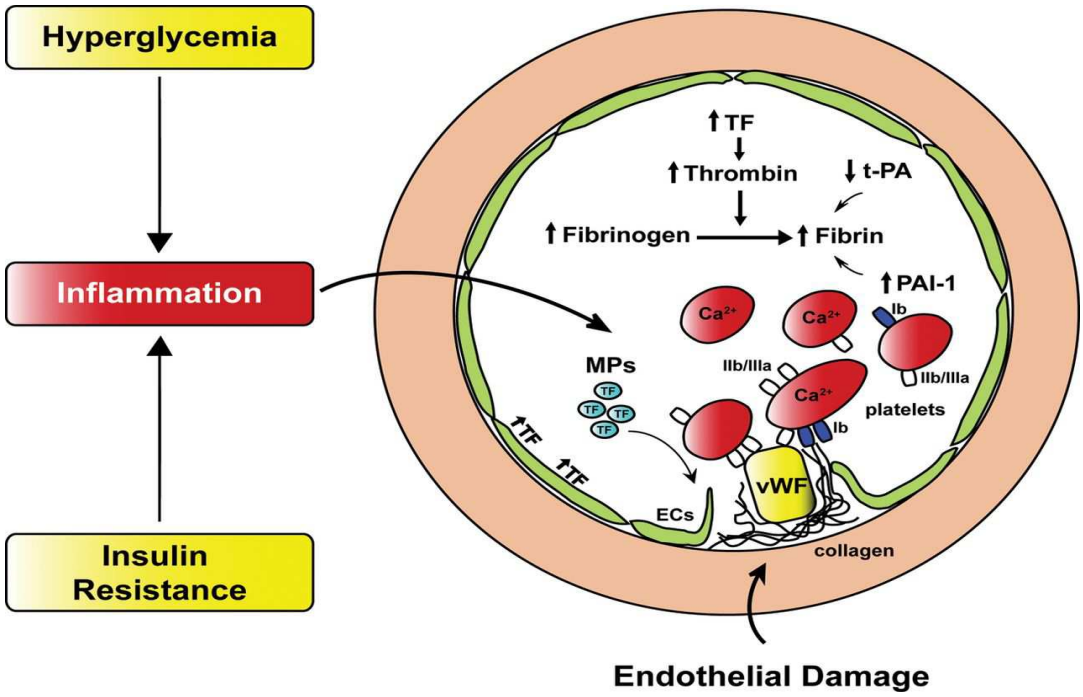
PHYSIOLOGY OF GLUCOSE COUNTER-REGULATION



The products of adipocytes namely, adipokines leads to induction of an inflammatory state and excess production of Interleukin 6 and C-reactive protein with the infiltration of inflammatory cells within the adipose tissue. Experiments in animal and human models has revealed that on inhibiting inflammatory signalling pathways namely nucler factor –kappa B[16] results in reduction of insulin resistance with improvement in hyperglycemia.

Secretion of insulin and its sensitivity in the peripheral tissues are interrelated. In patients with type 2 Diabetes, eventhough, the function of pancreatic beta cells appears to be lowered by 50% ,defect in insulin secretion is only very mild and tends to maintain the normal secretion in response to glucose and other insulin secretogogues like arginine.

Studies indicate that overlapping with the insulin resistance , a second genetic defect results in failure of pancreatic beta cells and reduction of beta cell mass by around 50%. In patients with chronic diabete mellitus, amylin or islet amyloid polypeptide that is secreted along with the insulin gets deposited in the islets forming amyloid fibrillar deposits.



In patients with longstanding Diabetes , chronic hyperglycemia has a negative impact on function of islet cells (glucose toxicity),leading to deterioration of Diabetes. Well controlled Diabetes has a positive effect on islet cell function. Excess dietary fat and free fatty acids (lipotoxicity) also worsens function of islet cells.

GLP 1[17] acts by increasing the secretion of insulin and reduction in glucose output by the liver, decrease in lipid production. Due to resistance of insulin in the adipocytes, lysis of lipids and release of excess of free fatty acids occur with the increment in low density lipoprotein and triglycerides.

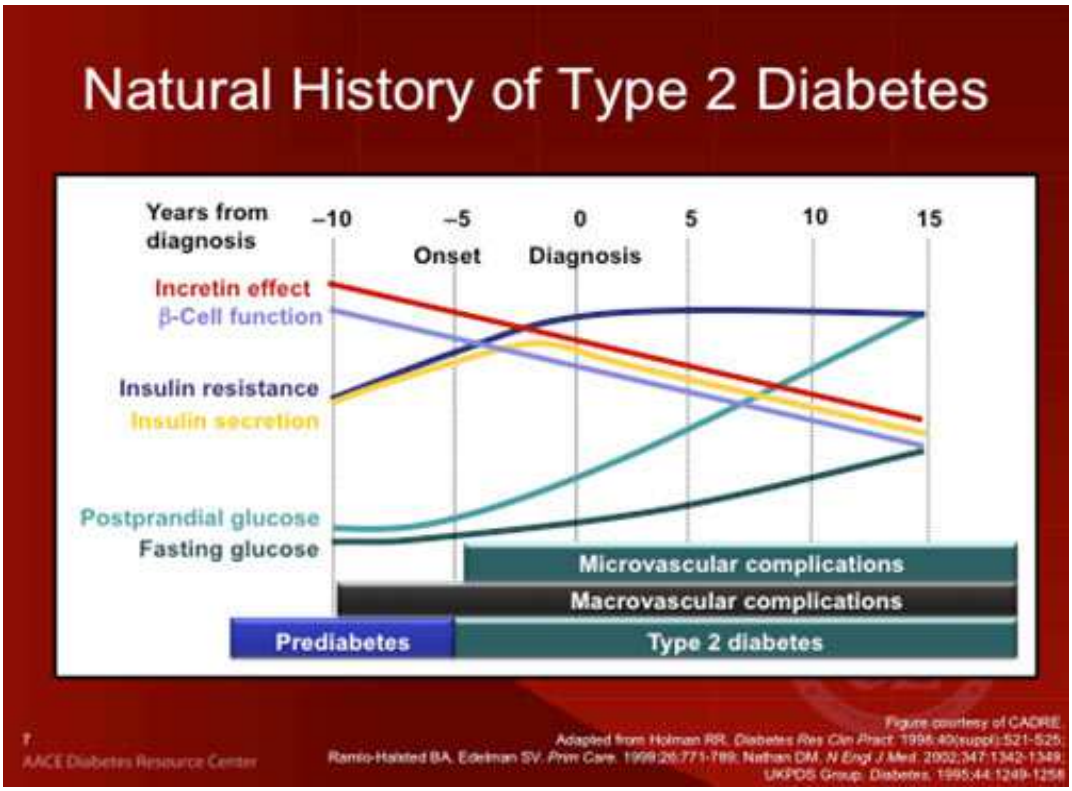
Excess steatosis in the hepatocytes results in development of non-alcoholic steatohepatitis (NASH) with abnormality in liver function tests. The above process results in dyslipidemis with elevation in triglyceride and low density lipoprotein levels with decrement in high density lipoprotein. This insulin resistance results in a group of metabolic derangements such as central or visceral obesity, hypertension , dyslipidemia and an increased risk for cardiovascular events. Acanthosis nigricans and features of hyperandrogenism (acne , hirsutism and oligomenorrhoea)[18], poly cystic ovarian syndrome may predominate.

Among adults, two forms of insulin resistance persists:

(1) type A, affecting young females with features of hyperandrogenism (acne , hirsutism and oligomenorrhoea; and obesity

(2) type B involves middle-aged females that has characteristics of severe hyperinsulinemia, hyperandrogenism features and autoimmune conditions.

Type B syndrome involves production of auto antibodies against insulin receptor, which might block the binding of insulin resulting in hyperglycemia or causes excessive stimulation of receptor leading to intermittent hypoglycaemia.



MANAGEMENT OF TYPR 2 DIABETES

The major components in the treatment of type 2 DIABETESincludes:

-Lifestyle modifications

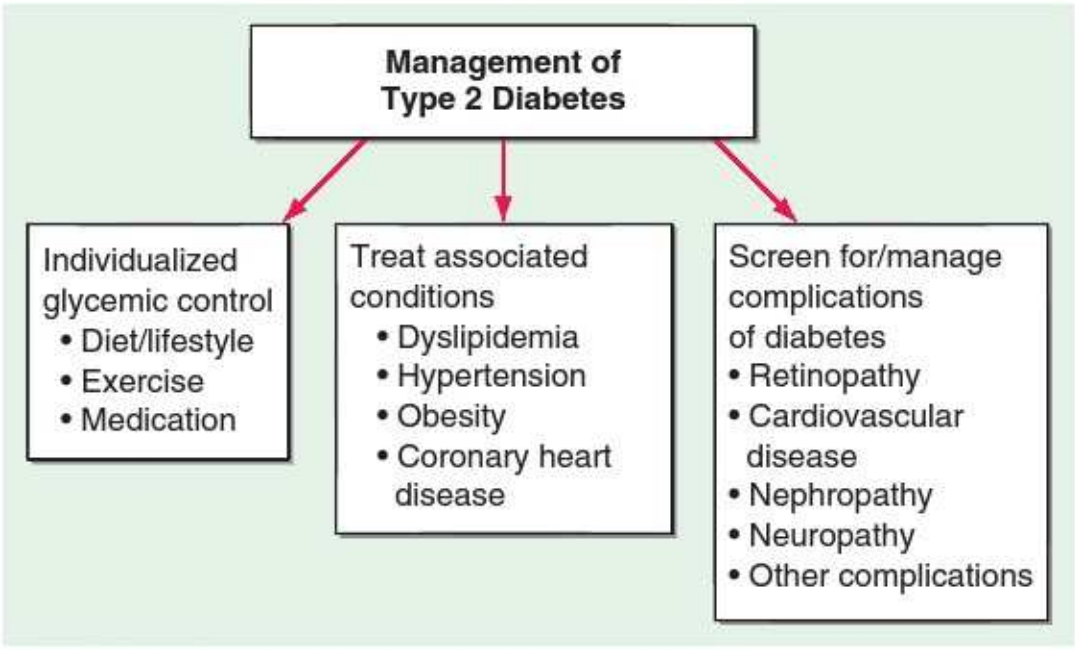
-Pharmacotherapy

- Oral hypoglycaemic agents
- Antihyperglycemic agents
- Injectable agents (Insulin) [1]

-Treatment of complications

-Treatment of associated conditions (hypertension / dyslipidemia)

ESSENTIAL COMPONENTS OF MANAGEMENT OF TYPE 2 DIABETES



LIFESTYLE CHANGES

DIET CONTROL

Dietary approach and meal plan in DIABETES plays an pivotal role in control of hyperglycemia and to overcome the peripheral tissue resistance to insulin, also helps in prevention of further end organ damage and for the control of associated comorbid conditions.

-food containing excess of vegetables should be encouraged

-food containing fat and excess carbohydrate should be avoided

- fruits containing dietary sucrose (mangoes,banana,jackfruit) should be exempted from diet

-vegetables which are cultivated from underground should be avoided (excepting groundnut).

EXERCISE

Daily aerobic exercise (brisk walking, gym, treadmill) lasting for around 20 minutes is essential for atleast 5 days a week. It effectively helps by increasing secretion of insulin with reduction of peripheral resistance.

Table 2: General Guidelines for Diet in Diabetes Mellitus	
Energy (Calories)	25 to 30 calories/kg IBW, reduce calories in obese and increase in underweight individuals
Protein	0.8 g/kg body weight, supplement provided for pregnancy, lactation and growth. A small quota of animal proteins—fish, chicken, milk and yoghurt and appropriate food intake recommended, avoid cattle meat and eggs
Fats	20% to 25% of total calories
Saturated	6% to 7% of total calories
PUFA total	6% to 7% of total calories
N6/N3 ratio	1:1 to 4:1
MUFA	6% to 7% of total calories
Cooking oil	0.5 kg/month/person Total fat intake, with cholesterol 300 mg per day
Carbohydrates	55% to 60% of total calories To encourage complex carbohydrates, i.e. mainly whole grain cereals, pulses, beans, vegetables, and salads. Avoid simple and refined carbohydrates like bakery products or deep fried items
Fruits	Fresh fruits up to 400 g per day. Avoid juices
Dietary fibre	30 to 40 g per day preferably from natural sources; avoid loss from refining and processing. Indian diet is rich in fibre and generally does not require addition of fibre supplements
Common salt	Up to 5 to 6 g per day. Reduced intake to 4 g per day in the presence of hypertension, renal failure and hearing problems
Condiments and spices	Provides anti-oxidants, trace elements, minerals, and n-3 fatty acids
Artificial sweetners	Use of saccharin and aspartame in limited quantity is acceptable. Avoid in pregnancy and lactation
Alcohol	Avoid if possible. If not, drastically restrict. It is utilised as fats. 1 g = 7 calories (2 small drinks/one glass of wine/ pint of beer (preferably avoided)
Tobacco	Avoid smoking or its use in any form

PHARMACOTHERAPY

Treatment of Diabetes with either oral hypoglycaemic agents, antihyperglycemic agents by acting on peripheral tissues to reduce resistance to insulin , acting by reducing gluconeogenesis [20.]

Choice of Pharmacologic Therapy
The choice of pharmacologic therapy should be based on a patient-centered approach with consideration of the following: • Efficacy • Cost • Potential side effects • Effects on weight • Comorbidities • Hypoglycemia risk • Patient preferences
*Metformin is contraindicated in individuals with: • Renal disease or renal dysfunction (e.g., as suggested by serum creatinine levels ≥1.5 mg/dL (males), ≥1.4 mg/dL (females) or abnormal creatinine clearance) which may also result from conditions such as cardiovascular collapse (shock), acute myocardial infarction, and septicemia • Known hypersensitivity to Metformin hydrochloride • Acute or chronic metabolic acidosis, including diabetic ketoacidosis, with or without coma. Diabetic ketoacidosis should be treated with insulin

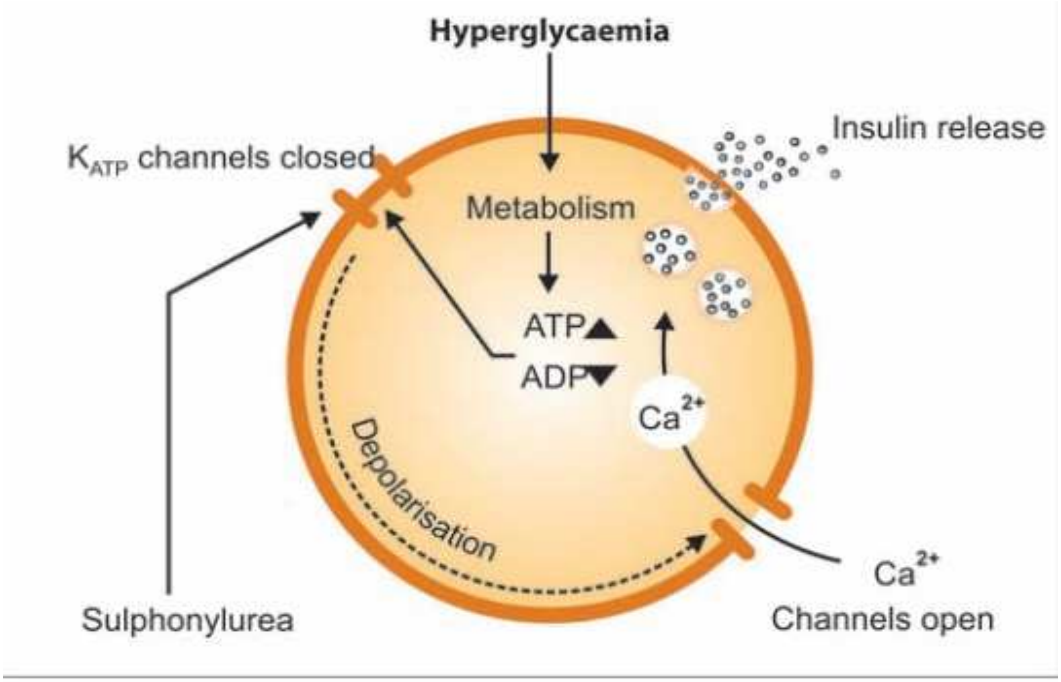
Metformin is the most commonly preferred drug among all the oral hypoglycaemic agents, due to its superior efficacy and potency.

TABLE 418-5 AGENTS USED FOR TREATMENT OF TYPE 1 OR TYPE 2 DIABETES

	Mechanism of Action	Examples[a]	HbA_{1c} Reduction (%)[b]	Agent-Specific Advantages	Agent-Specific Disadvantages	Contraindications
Oral						
Biguanides[c,*]	↓ Hepatic glucose production	Metformin	1–2	Weight neutral, do not cause hypoglycemia, inexpensive, extensive experience, ↓ CV events	Diarrhea, nausea, lactic acidosis	Serum creatinine >1.5 mg/dL (men) >1.4 mg/dL (women) (see text), CHF, radiographic contrast studies, hospitalized patients, acidosis
α-Glucosidase inhibitors[c,**]	↓ GI glucose absorption	Acarbose, miglitol, voglibose	0.5–0.8	Reduce postprandial glycemia	GI flatulence, liver function tests	Renal/liver disease
Dipeptidyl peptidase IV inhibitors[c,***]	Prolong endogenous GLP-1 action	Alogliptin, Anagliptin, Gemigliptin, linagliptin, saxagliptin, sitagliptin, teneligliptin, vildagliptin	0.5–0.8	Well tolerated, do not cause hypoglycemia		Reduced dose with renal disease; one associated with increase heart failure risk; possible association with ACE inhibitor–induced angioedema
Insulin secretagogues: Sulfonylureas[c,*]	↑ Insulin secretion	Glibornuride, gliclazide, glimepiride, glipizide, gliquidone, glyburide, glyclopyramide	1–2	Short onset of action, lower postprandial glucose, inexpensive	Hypoglycemia, weight gain	Renal/liver disease
Insulin secretagogues: Nonsulfonylureas[c,***]	↑ Insulin secretion	Nateglinide, repaglinide, mitiglinide	0.5–1.0	Short onset of action, lower postprandial glucose	Hypoglycemia	Renal/liver disease
Sodium-glucose cotransporter 2 inhibitors[***]	↑ Urinary glucose excretion	Canagliflozin, dapagliflozin, empagliflozin	0.5–1.0	Insulin secretion and action independent	Urinary and vaginal infections, dehydration, exacerbate tendency to hyperkalemia	Limited clinical experience; moderate renal insufficiency
Thiazolidinediones[c,***]	↓ Insulin resistance, ↑ glucose utilization	Rosiglitazone, pioglitazone	0.5–1.4	Lower insulin requirements	Peripheral edema, CHF, weight gain, fractures, macular edema	CHF, liver disease
Parenteral						
Amylin agonists[c,d,***]	Slow gastric emptying, ↓ glucagon	Pramlintide	0.25–0.5	Reduce postprandial glycemia, weight loss	Injection, nausea, ↑ risk of hypoglycemia with insulin	Agents that also slow GI motility
GLP-1 receptor agonists[c,***]	↑ Insulin, ↓ glucagon, slow gastric emptying, satiety	Exenatide, liraglutide, dulaglutide	0.5–1.0	Weight loss, do not cause hypoglycemia	Injection, nausea, ↑ risk of hypoglycemia with insulin secretagogues	Renal disease, agents that also slow GI motility; medullary carcinoma of thyroid
Insulin[c,d,****]	↑ Glucose utilization, ↓ hepatic glucose production, and other anabolic actions	See text and Table 418-4	Not limited	Known safety profile	Injection, weight gain, hypoglycemia	
Medical nutrition therapy and physical activity[c]	↓ Insulin resistance, ↑ insulin secretion	Low-calorie, low-fat diet, exercise	1–3	Other health benefits	Compliance difficult, long-term success low	

SULFONYLUREA

Acts by the mechanism of closure of ATP mediated K channels, which in turn leads to cellular depolarisation causing opening of calcium channels leading to release of Insulin. Thus, it has the disadvantage of producing hypoglycaemia in case the patient takes the drug without the intake of meals.



INJECTABLE INSULIN PREPARATIONS

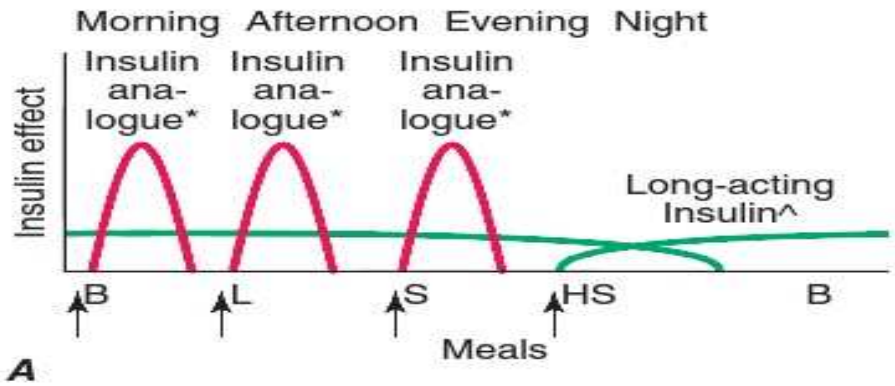
These are being more widely used for those patients who have uncontrolled Diabetes even while on treatment of oral hypoglycaemic agents, microvascular or macrovascular complications.

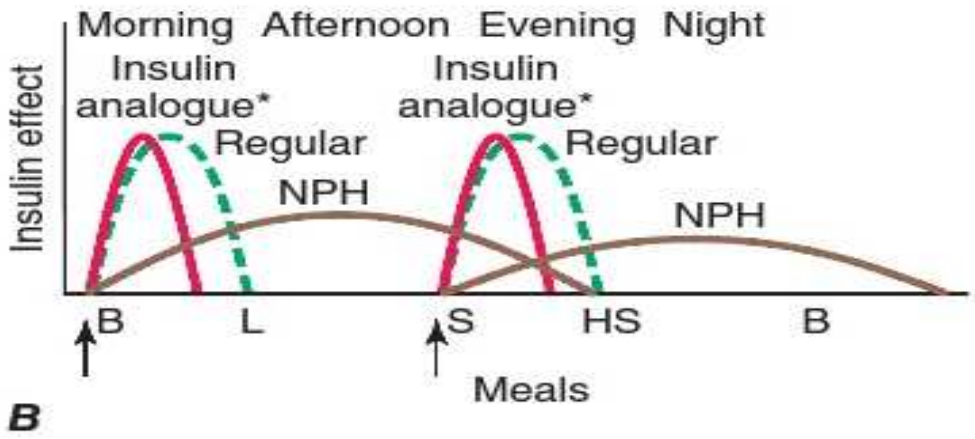
VARIOUS FORMS OF INSULIN PREPARATIONS

TABLE 418-4 PROPERTIES OF INSULIN PREPARATIONS[a]

Preparation	Time of Action		
	Onset, h	Peak, h	Effective Duration, h
Short-acting			
Aspart	<0.25	0.5–1.5	2–4
Glulisine	<0.25	0.5–1.5	2–4
Lispro	<0.25	0.5–1.5	2–4
Regular	0.5–1.0	2–3	3–6
Long-acting			
Detemir	1–4	—[b]	12-24[c]
Glargine	2–4	—[b]	20-24
NPH	2–4	4–10	10–16
Insulin combinations[d]			
75/25–75% protamine lispro, 25% lispro	<0.25	Dual[e]	10–16
70/30–70% protamine aspart, 30% aspart	<0.25	Dual[e]	15-18
50/50–50% protamine lispro, 50% lispro	<0.25	Dual[e]	10–16
70/30–70% NPH, 30% regular	0.5–1	Dual[e]	10–16

TIMING AND TYPE OF INSULIN ADIABETESINISTRATION

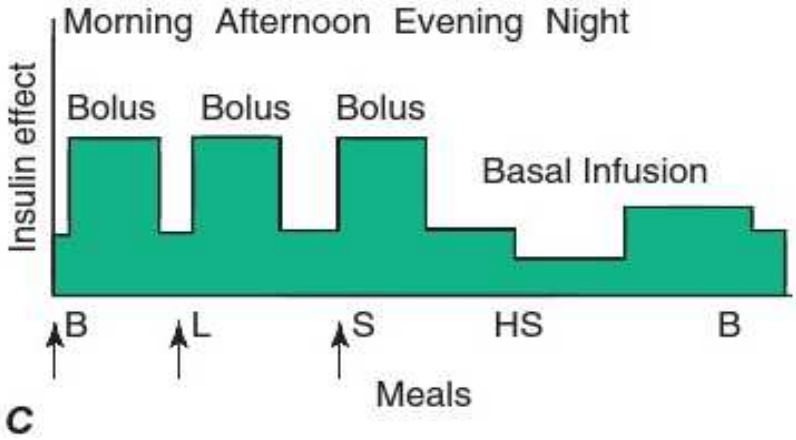




B

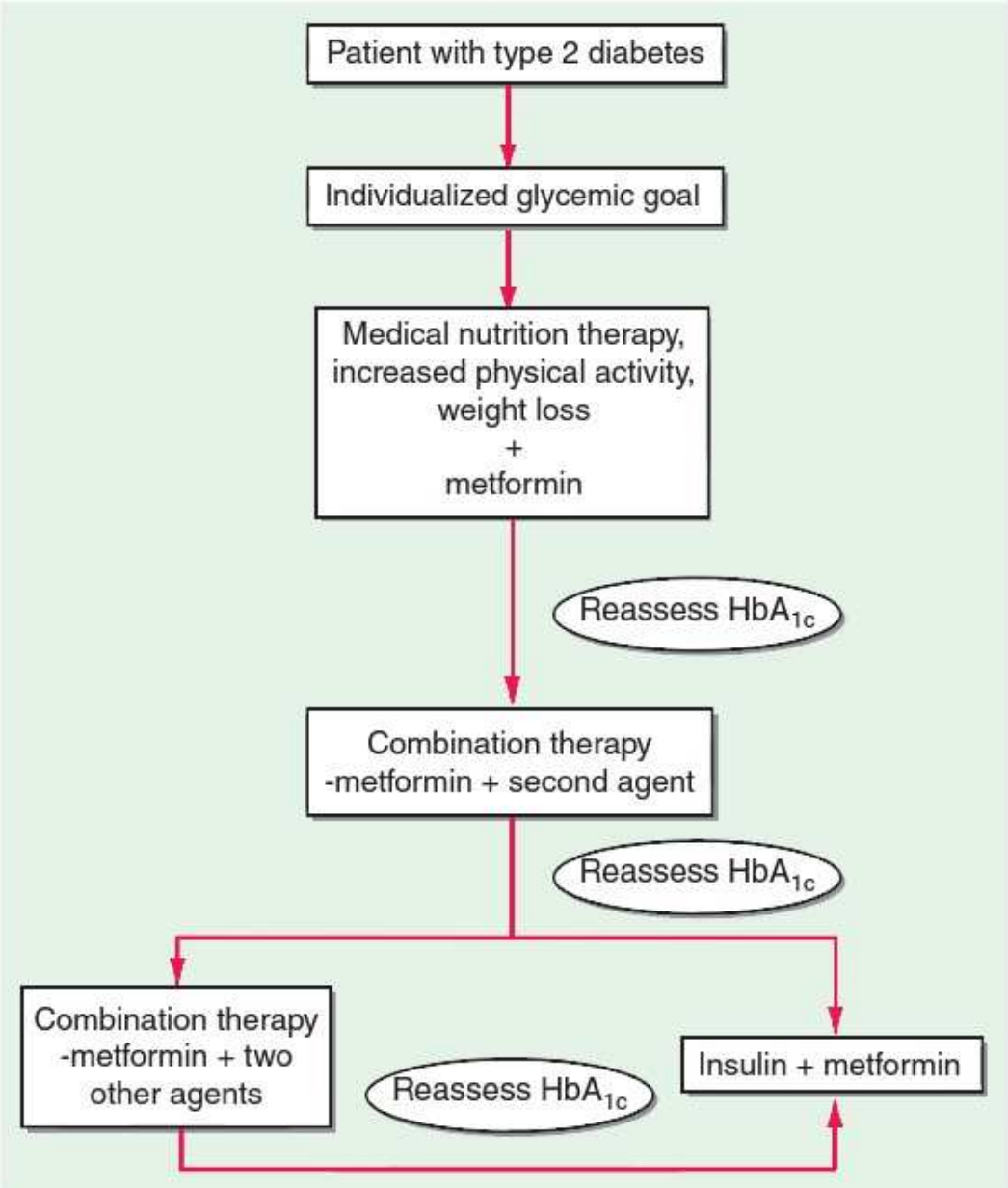
A. Timing of insulin administration varies between each individual and it is an patient tailored approach. In the above diagram, regular / short acting insulin is administered as three doses per day before breakfast, lunch and dinner and a long acting basal insulin once a day.

B. The diagram depicts the insulin Administration as a dual rapidly acting dosage, dual intermediately acting insulin.



C

C. The diagram shows that insulin is given as a single basal formulation with multiple bolus injection before each meal[21].



COMPLICATIONS OF DIABETES

TABLE 419-1 DIABETES-RELATED COMPLICATIONS

- Microvascular
  - Eye disease
    - Retinopathy (nonproliferative/proliferative)
    - Macular edema
  - Neuropathy
    - Sensory and motor (mono- and polyneuropathy)
    - Autonomic
  - Nephropathy (albuminuria and declining renal function)
- Macrovascular
  - Coronary heart disease
  - Peripheral arterial disease
  - Cerebrovascular disease
- Other
  - Gastrointestinal (gastroparesis, diarrhea)
  - Genitourinary (uropathy/sexual dysfunction)
  - Dermatologic
  - Infectious
  - Cataracts
  - Glaucoma
  - Cheiroarthropathy[a]
  - Periodontal disease
  - Hearing loss

Other comorbid conditions associated with diabetes (relationship to hyperglycemia is uncertain): depression, obstructive sleep apnea, fatty liver disease, hip fracture, osteoporosis (in type 1 diabetes), cognitive impairment or dementia, low testosterone in men

Since progression of DIABETES (uncontrolled / chronicity) is associated with multiple microvascular and macrovascular complications , resulting in early morbidity and mortality.

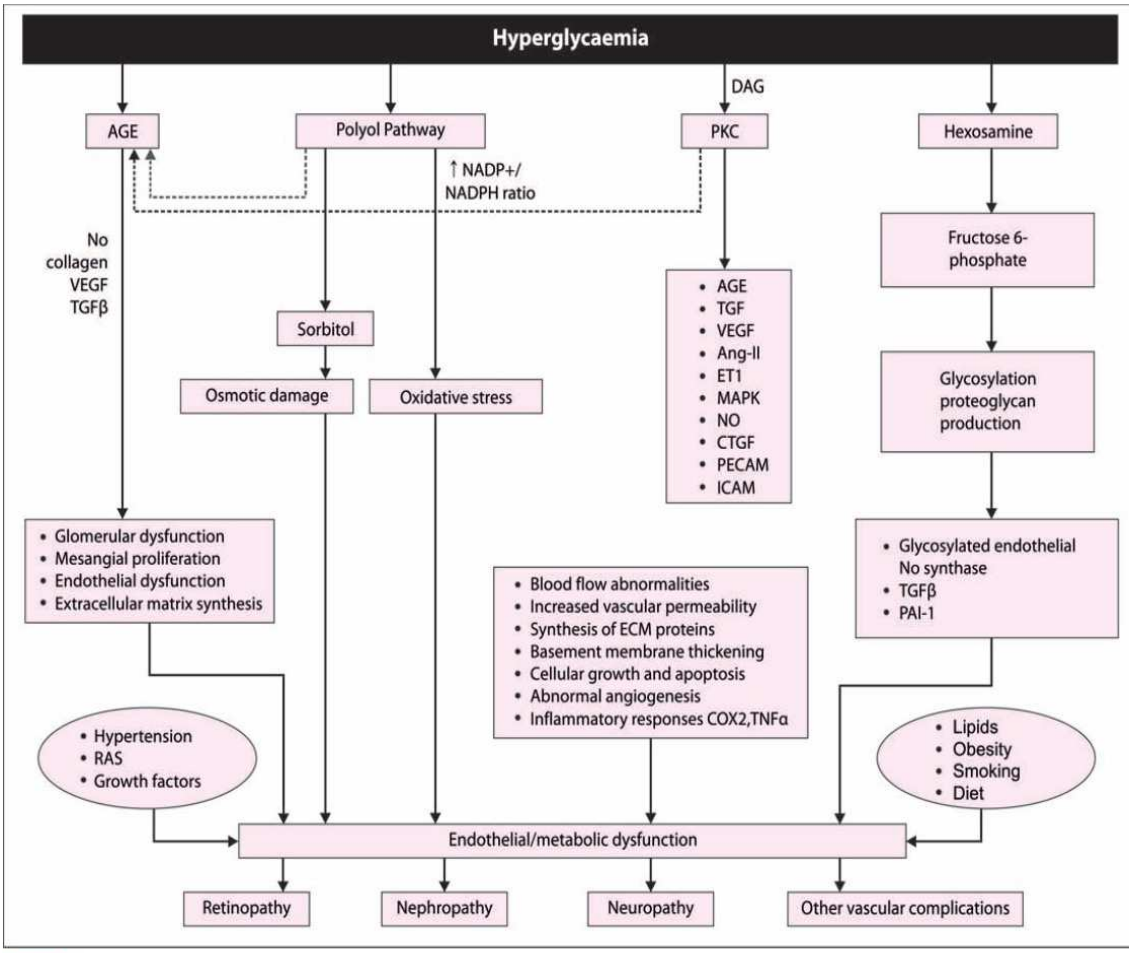


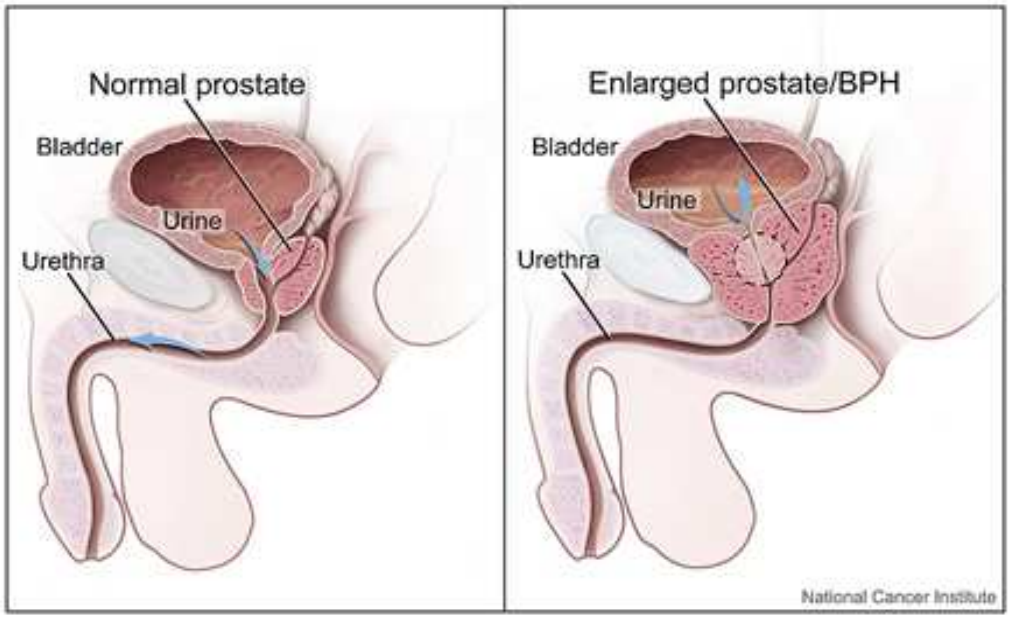
Figure 1: Pathways leading to microvascular diseases in diabetes mellitus.

The mainstay of primary treatment aims at prevention of vascular complications, in case of occurrence of complications secondary treatment should be aimed at treating the disease and preventing it from further progression , thereby decreasing the mortality and morbidity.

BENIGN PROSTATIC HYPERPLASIA (BPH)

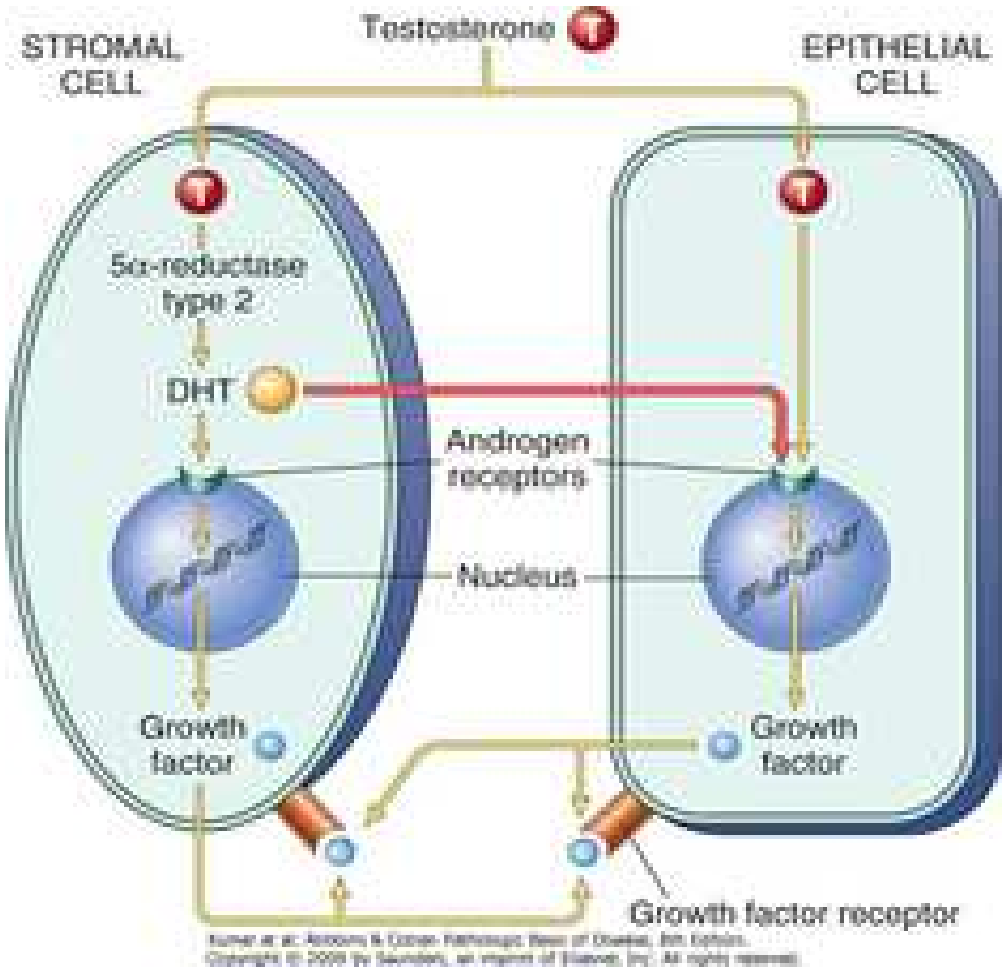
INTRODUCTION

Benign prostatic hyperplasia (BPH), charachterised by an increase in size and number of cellular elements of prostatic tissues is an diagnosis made histologically.



Longstanding bladder outlet obstruction (BOO) due to Benign prostatic hyperplasia might result in retention of urine, recurrent urinary tract infections, renal insufficiency and stone in bladder[16].

Benign prostatic hyperplasia primarily involving the epithelial and stromal components of prostate that is present in the periurethral and transition zone leads on to restriction of urinary flow . The basic underlying pathogenesis would be excess proliferation of stromal and epithelial cells or impairment of programmed cell death (apoptosis).



Since, benign prostatic hyperplasia is primarily dependent on testosterone and dihydrotestosterone, it is projected as a part of aging process. Approximately, about 53% of males evidence histopathologic Benign prostatic hyperplasia by the age of 60 years. This figure further increases to about 90% by the age of 85 years.

The voiding dysfunction that is resulting from bladder outlet obstruction (BOO) is named as lower urinary tract symptoms (LUTS) or as prostatism . These entities tends to overlap; that not all males with LUTS have BPH and that not all the males with BPH have LUTS[22].

Approximately around 50% of males being detected with histopathologic evidence of benign prostatic hyperplasia are found to exhibit moderate-to-severe LUTS.

FUNCTIONS OF PROSTATE GLAND

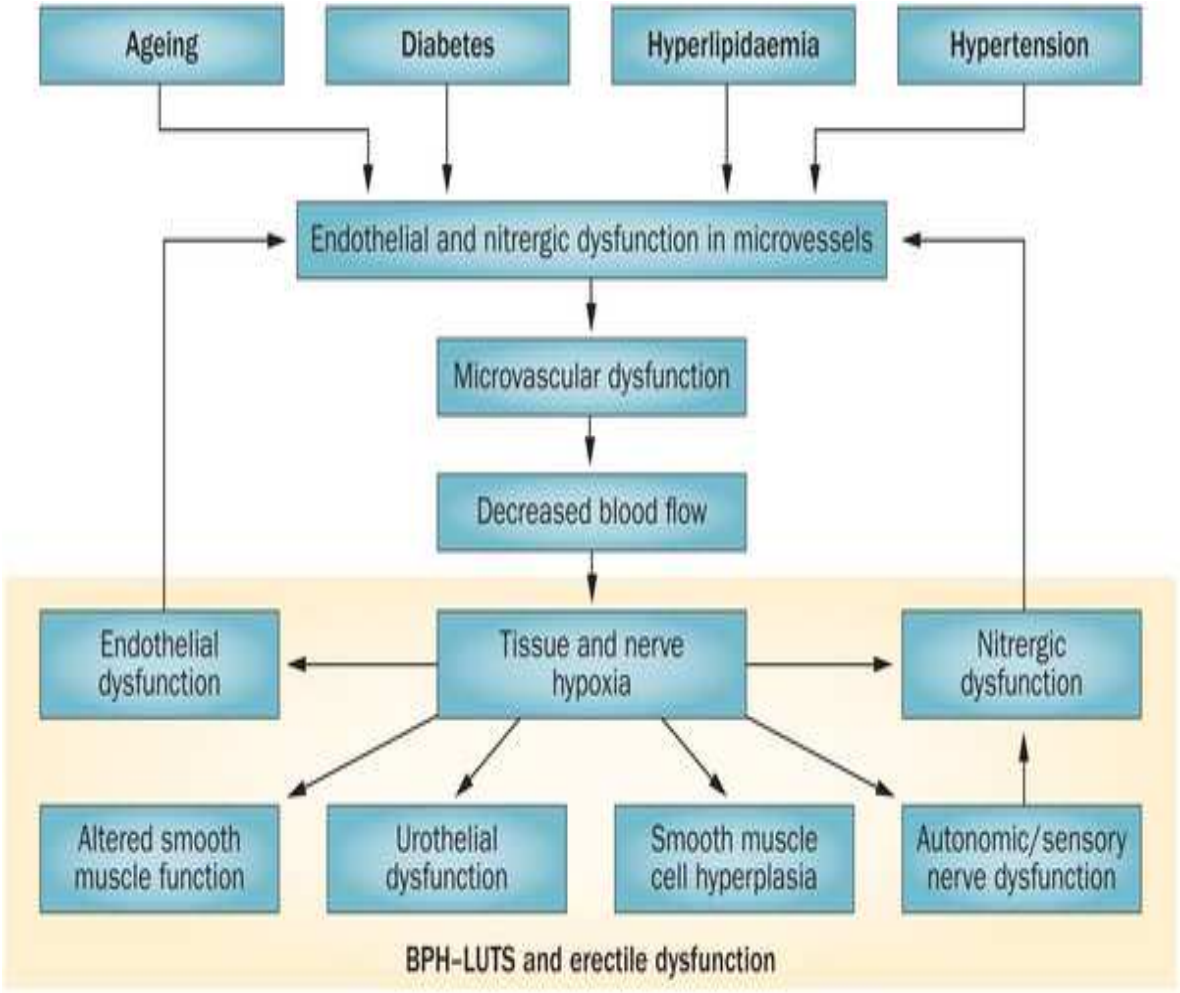
The major role of prostate gland is to synthesize and secrete an alkaline fluid that includes constituting around 70% of the volume of semen. They aid in lubrication and nourishment for the sperm , helps liquefaction of seminal plug and in neutralizing the acidic nature of vaginal environment.

The prostatic urethra acts as a pathway for passage of semen and controls retrograde by blocking the bldder neck at the time of sexual climax. Ejaculation tend to involve a coordinated action of various tissue components, including vasa deferentia, smooth muscles of seminal vesicles, the ischiocavernosus and bulbocavernosus muscles and the ejaculatory ducts.

PATHOPHYSIOLOGY OF BPH

Enlargement of prostate primarily depend on the most potent androgen dihydrotestosterone (DHT). In the prostate gland, type II 5-alpha-reductase tends to metabolize circulating testosterone into its

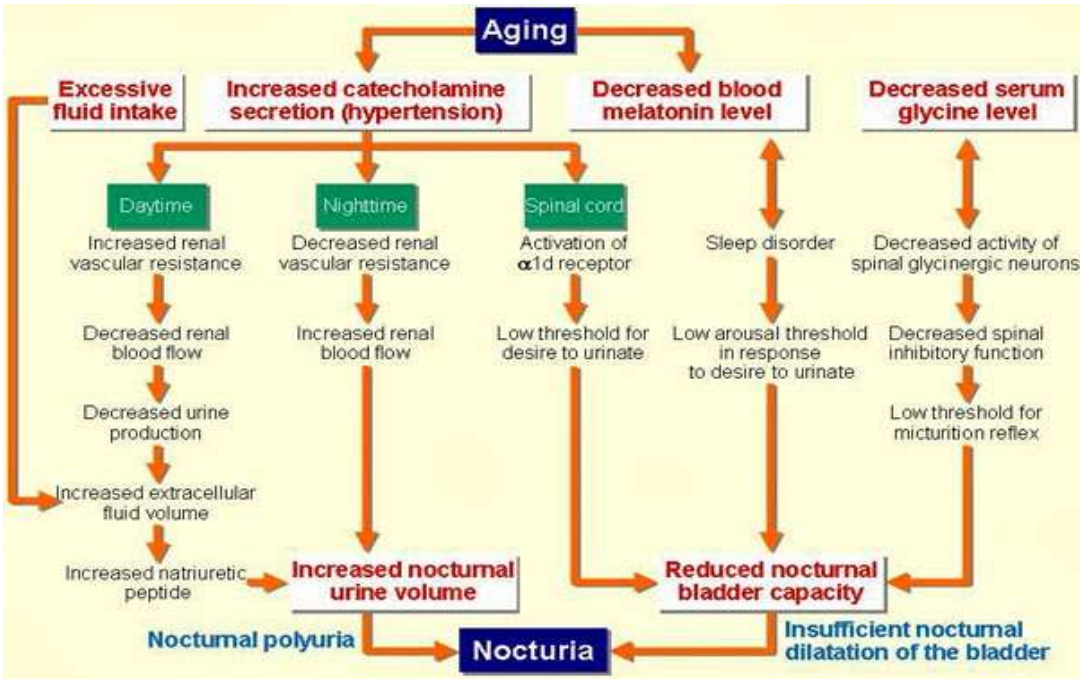
metabolically active product DHT, which work over local tissues, not systemically. DHT act by binding to androgen receptors in the cellular nucleus, lading on to benign prostatic hyperplasia.



Numerous studies had projected that huge number of alpha-1-adrenergic receptors are present in stromal and capsular smooth muscles of the prostate, and in the neck of bladder. Stimulation of these receptors cause an increment in tone of smooth-muscles , that tend to worsen lower urinary tract symptoms[15]. Conversely, receptor blockade tends to loosen the muscles, with subsequent LUT symptom relief.

Microscopically, BPH is characterized by a hyperplastic process. The hyperplasia thus results in enlargement of the prostate gland that might restrict the urinary flow from the bladder, resulting in clinical symptoms of BPH. The prostate gland enlarges with age in a hormonally dependent manner. Thus, castrated males (who are not able to make testosterone) do not develop BPH.

The knowledge behind Benign prostatic hyperplasia is that, as the gland enlarges in size, the overlying capsule tends to prevent it from expanding radially, leading on to urethral compression and LUTS. However,bladder dysfunction due to obstruction contribute predominantly to LUTS. The bladder wall become much trabeculated, thickened, and irritable on forcing it to hypertrophy and increases its own contractile power.



Urinary frequency and symptoms of lower urinary tract are secondary to increase in sensitivity (detrusor overactivity) of the myocytes. Bladder might weaken gradually and loses its ability to completely empty, leading to higher amount of residual urine and, possibly, to bacterial infection, acute or chronic urinary retention.

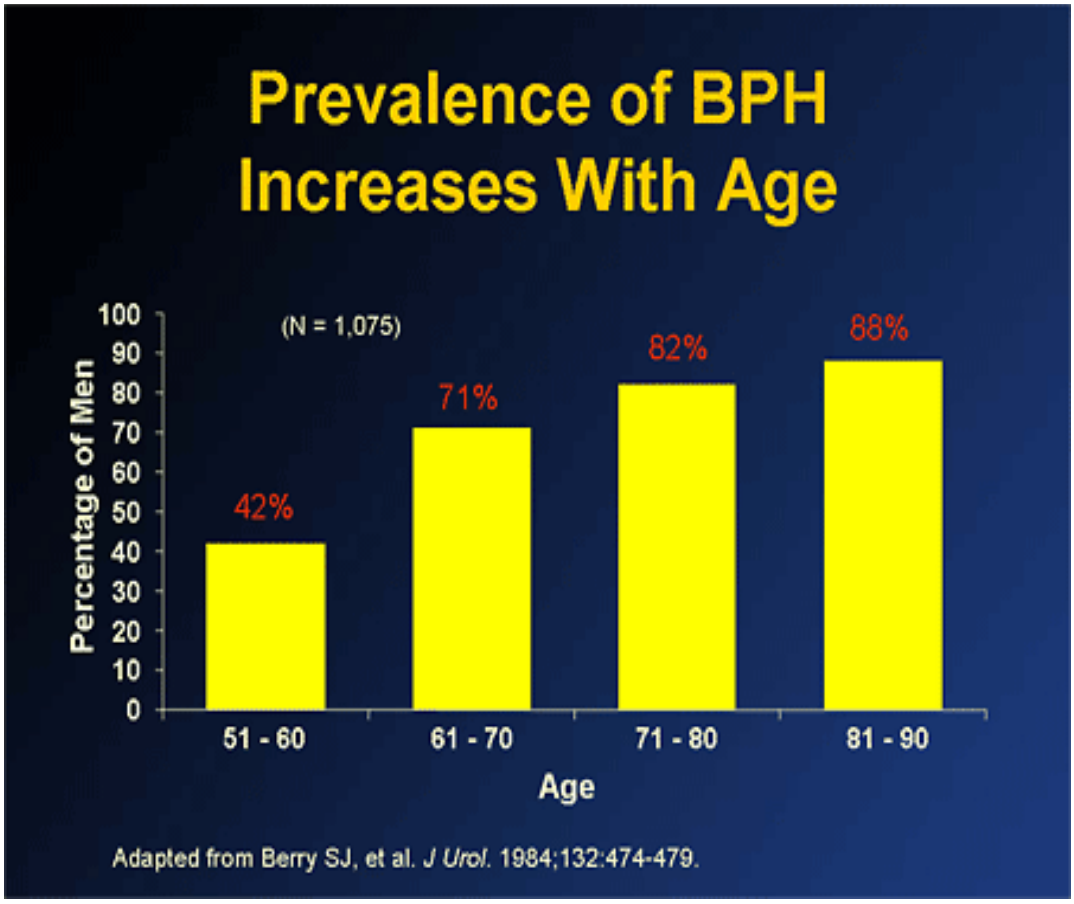
This tends to become a vicious cycle with obstruction leading on to hypertrophied smooth muscles. Specimens of trabeculated bladder demonstrates evidence of few myocytes with an increment in collagen content. The collagen fibres limits its compliance, leading to higher amount of bladder pressure upon filling.

EPIDEMIOLOGY

Benign prostatic hyperplasia is a widespread condition that affects the quality of life significantly in approximately 30% of males above 50 years. Benign prostatic hyperplasia is found to be histologically evident in around 88% of men by age 80 years. Globally, around 28 million males are likely to have symptoms secondary to Benign prostatic hyperplasia

The prevalence of Benign prostatic hyperplasia in whites and American-african males are the same[14] . However, BPH has a severe and a progressive course in American-african males, due to the higher levels of testosterone, 5-alpha-reductase activity, expression of androgen

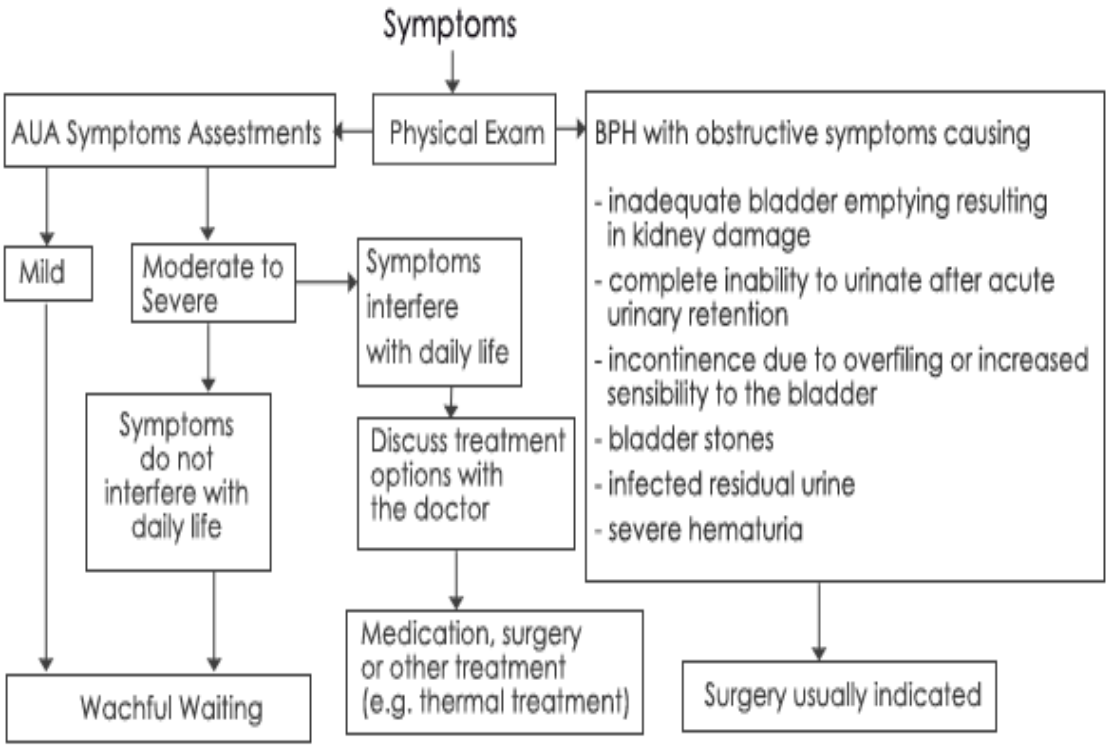
receptors, and activity of growth factor in this group of population. This increment in activity leads to an further higher rate of hyperplastic proatate and its complicating sequelae.

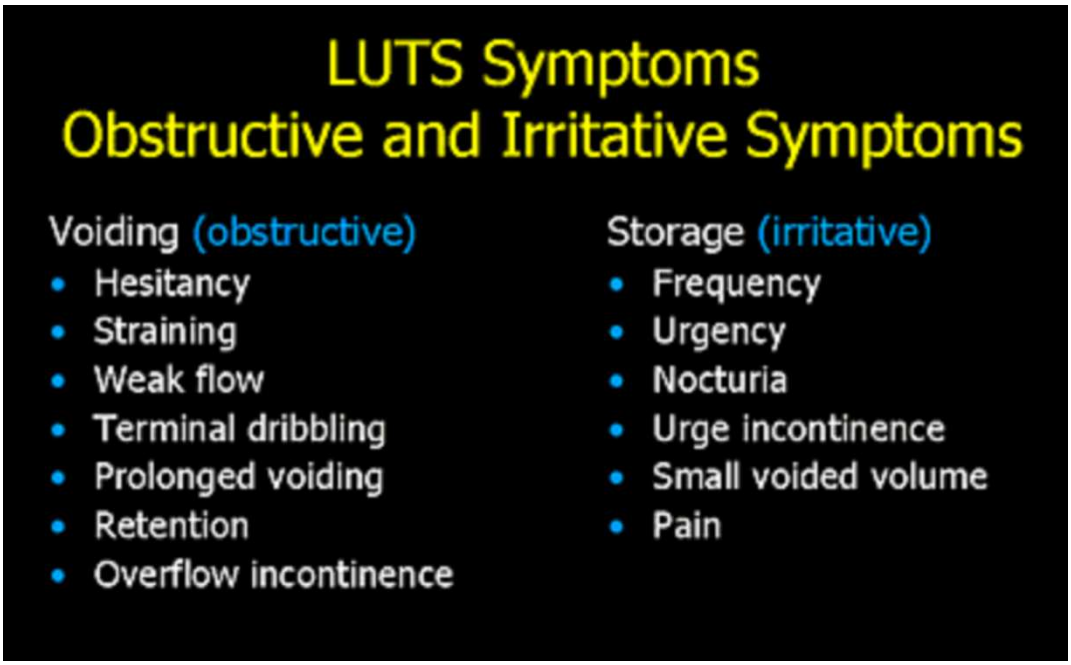


CLINICAL MANIFESTATIONS

Signs and symptoms

With enlargement of prostate, it tends to constrict the urinary flow. Nervous system present inside the prostate and bladder might also be a factor involved in causing the symptoms.





The symptoms of benign prostatic hyperplasia includes difficulty in initiation of urinary stream along with difficulty in maintaining the stream, frequent passage of urine at night secondary to retention of urine

(post void residual urine), dribbling of urine , secondary bacterial infections and loss of libido.

DIAGNOSIS

DIGITAL PER-RECTAL EXAMINATION

It the major test aiding in evaluation of patients with BPH, helps in assessing the contour, size, consistency , texture of the prostate , evaluation of nodules if present, where areas of malignancy can be detected.

LABORATORY STUDIES

URINE ANALYSIS

Examination of centrifuged sediment urine with the help of dipstick methods aids in identification of presence of blood , bacteria , protein , leukocytes or glucose.

Urine culture

In case the urine analysis predicts an abnormality, infectious cause of irritative voiding needs to be excluded.

PROSTATE-SPECIFIC ANTIGEN

Benign prostatic hyperplasia is not routinely associated with malignancy, but however they tend to carry a higher risk compared to a general population. Thus, screening routinely is a prerequisite.

Electrolytes, Blood Urea Nitrogen (Bun), And Creatinine

Helpful in patients with chronic renal disease who had large amounts of postvoid residual (PVR) urine volume.

ULTRASONOGRAPHY

Ultrasonography is a helpful tool in order to assess bladder and prostate size and presence of hydronephrosis (if present) in males with retention of urine and symptoms or signs of kidney disease.

Generally in the evaluation of uncomplicated LUTS, ultrasonography is not routinely indicated. Done in the radiology department by a senior radiologist using 3.5 MW Mechanical Probe USG machine for various radiological diagnostic conditions causing prostatic hypertrophy (such as BPH, prostatic abscess, prostatitis) with special importance aimed at radiological grading of prostatomegaly, pyelonephritis [presence of particulate matter in the collecting system, appearance of decreased vascularity in cortex , presence of air bubbles (emphysematous pyelonephritis), abnormal renal parenchymal echogenecity, cystitis [thickened bladder wall as a result of wall edema] and estimating the amount of residual urine[12].

LOWER URINARY TRACT ENDOSCOPY

Cystoscopy might be useful in males who are posted for invasive treatment or in patients to whom we are suspecting the presence of foreign body or malignancy. Endoscopymis useful in patients with a

prior sexually transmitted disease (eg, gonococcal urethritis) history , history of prolonged urinary catheterization, or presence of trauma.

IPSS/AUA-SI

The grade of BPH is assessed using INTERNATIONAL PROSTATE SYMPTOM SCORE (IPSS)/AMERICAN UROLOGICAL ASSOCIATION SYMPTOM INDEX (AUA-SI) with a disease-specific quality of life (QOL) question.

Symptoms / Score	Not at all	Less than 1 time in 5	Less than half the times	Around half the times	More than half the times	Almost always
Do you have a sensation of not emptying your bladder completely after you finish urinating?	0	1	2	3	4	5
Do you have to urinate again less than 2 hours after you finish urinating?	0	1	2	3	4	5
Do you stop and start several times when you urinate?	0	1	2	3	4	5
How often is it difficult to postpone urination?	0	1	2	3	4	5
Do you have a weak urinary stream?	0	1	2	3	4	5
Do you often have to push or strain to begin urination?	0	1	2	3	4	5
	Never	1 Time	2 Times	3 Times	4 Times	5 Times
How many times do you get up to urinate from the time you go to bed at night until you get up in the morning?	0	1	2	3	4	5

UROFLOWMETRY

Flow rate

Turns out to be helpful to assess the response to treatment.

PVR urine volume

Useful in gauging the bladder decompensation severity and also be obtained invasively with catheter or sometimes, noninvasively with a transpelvic ultrasonic scanner[18].

Pressure flow studies

Results might be useful in evaluation of outlet obstruction of bladder.

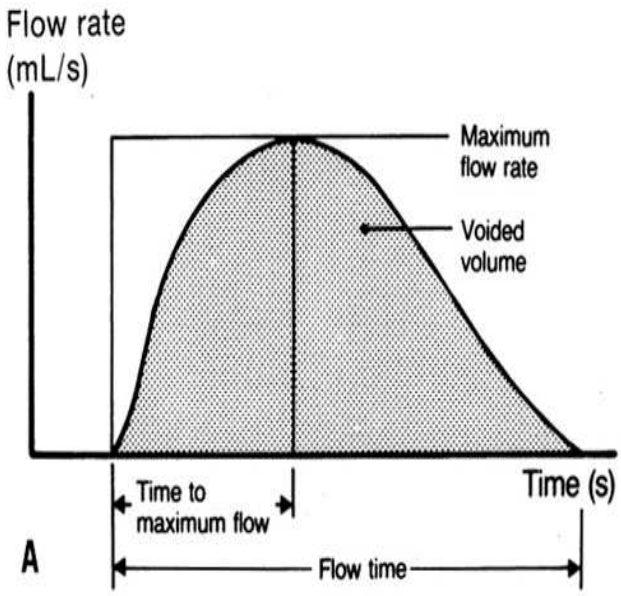
Urodynamic flow studies

Helps in distinguishing poorer bladder contractility (decreased detrusor activity) from outlet obstruction of bladder.

CYTOLOGIC EXAMINATION OF THE URINE

Used in those patients with irrittive symptoms.

Uroflowmetry A



MANAGEMENT

Pharmacotherapy

Agents that are used in the treatment of BPH includes the following:

- Alpha-adrenergic receptor blockers
- Alpha-1–receptor blockers
- Phosphodiesterase-5 enzyme inhibitors
- 5-alpha reductase inhibitors
- Anticholinergic agents

SURGERY

Transurethral resection of the prostate (TURP)

The standard procedure to relieve bladder outlet obstruction that occurs due to BPH.

Open prostatectomy

Usually reserved for:

- males with a prostates size of >75 g.,
- males with associated bladder stones or diverticulum of bladder
- males who cannot tolerate or position for transurethral surgery

Minimally invasive treatment

1.Transurethral incision of the prostate (TUIP)

2.Laser treatment – helps in cutting or destroying the prostatic tissues.

3.Transurethral microwave therapy (TUMT)

Acts by producing heat that destroys the prostatic cells , resulting in shrinkage and reduction in prostatic volume.

4.Transurethral needle ablation of the prostate (TUNA)

5.High-intensity ultrasonographic energy therapy

6.Prostatic stents

These are Flexible devices that tends to dilate once placed in position to improve the .urinary flow rate.

7.Laparoscopic procedures (prostatectomy).

MATERIALS AND METHODS

MATERIALS AND METHODS

Study Centre

Department of Diabetology and Institute of Internal medicine,

Madras medical college,

Rajiv Gandhi government general hospital,

Chennai-3.

Duration of Study

6 months

Study Design

Observational Study (prospective and retrospective)

Sample Size

100 patients

Inclusion Criteria

Patients with Type 2 Dibetes mellitus aged 40- 60 years.

Exclusion Criteria

1.Known case of :

-BENIGN PROSTATIC HYPERPLSIA

-CARCINOMA PROSTATE

2.Age > 60 years

Data Collection and Methods

Type 2 diabetic patients attending Diabetology OP of RGGGH and inpatients of Institute of Internal Medicine are subjected to detailed history taking , clinical examination and required investigations.

Materials and Methods

- From Type 2 Diabetes patients getting Admitted in Medicine ward/ attending the Diabetology department OPD, selected for clinical study as per inclusion/exclusion criteria the following data are collected:

  -Demographic data

  -Medical history

- Patients are subjected to:

  -Blood sugar estimation (fasting /postprandial)

  -HbA1c

  -Serum Prostate Specific Antigen(PSA)

  {for selected patients}

  -Ultasound pelvis.

Analysis Plan

SPSS, Epi INFO softwares

Sponsorship

No

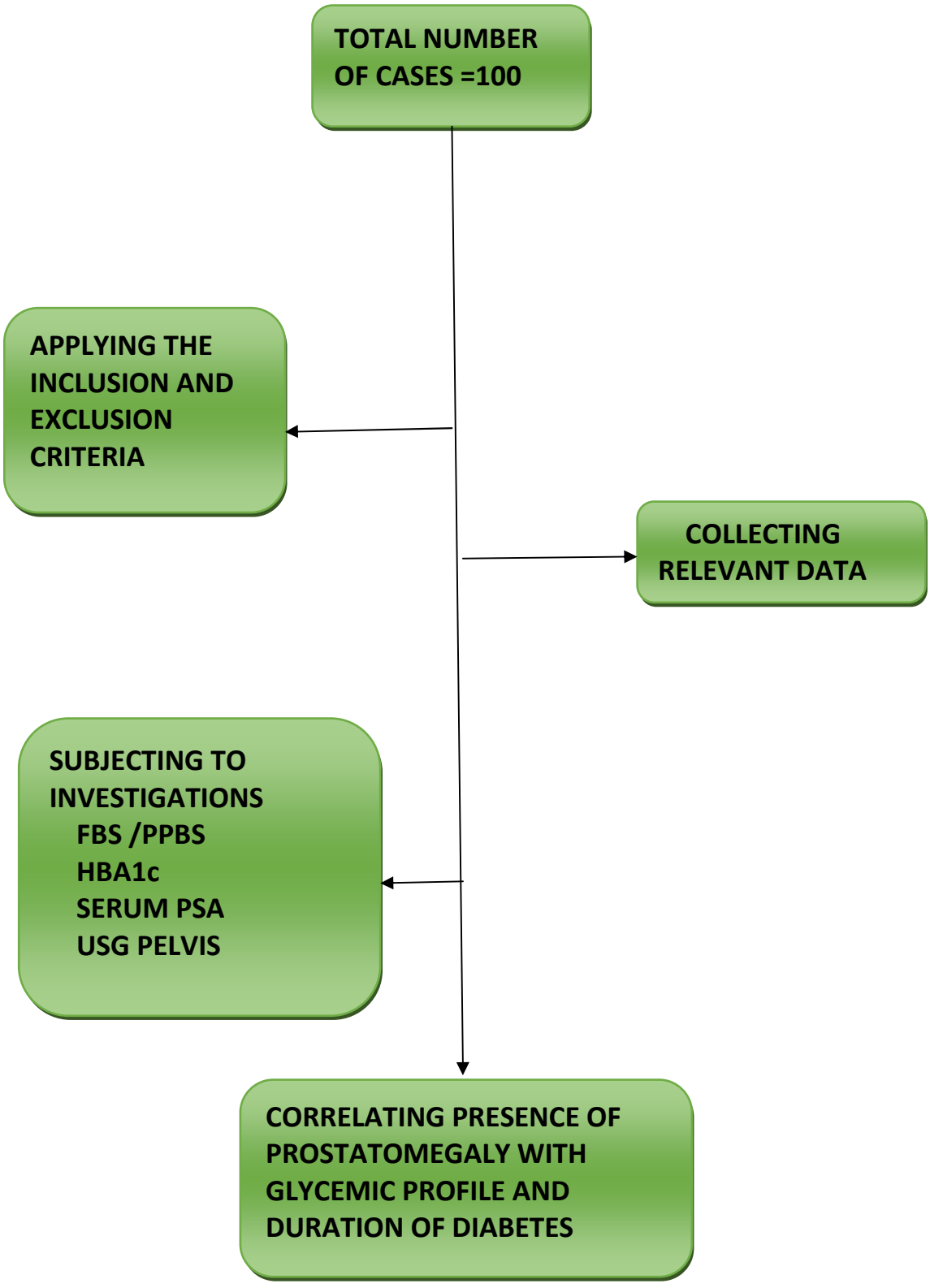
Conflict of interest

None

OBSERVATIONS AND RESULTS

OBSERVATION AND RESULTS

FLOW CHART OF THE METHODOLOGY

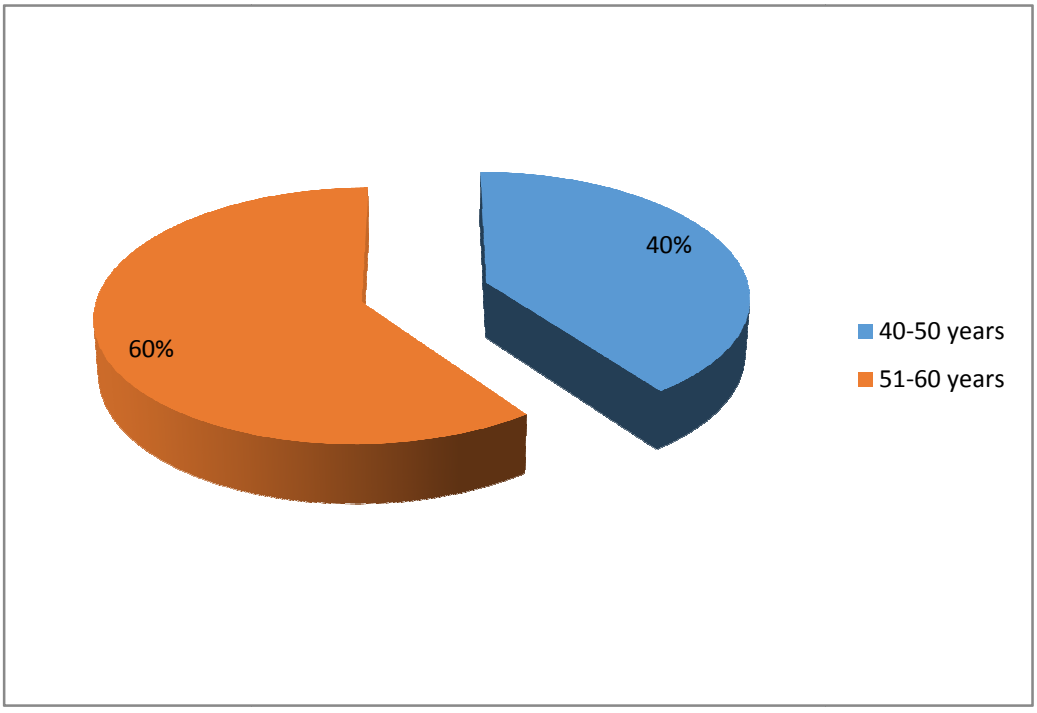


FREQUENCY OF AGE WISE DISTRIBUTION IN OUR STUDY GROUP

AGE_GROUP	FREQUENCY	PERCENT
40-50 years	40	40.0
51-60 years	60	60.0
Total	100	100.0

From the above table,it is evident that around 40% patients fall within 40-50 years of age and about 60% patients fall within 50-60 years of age.

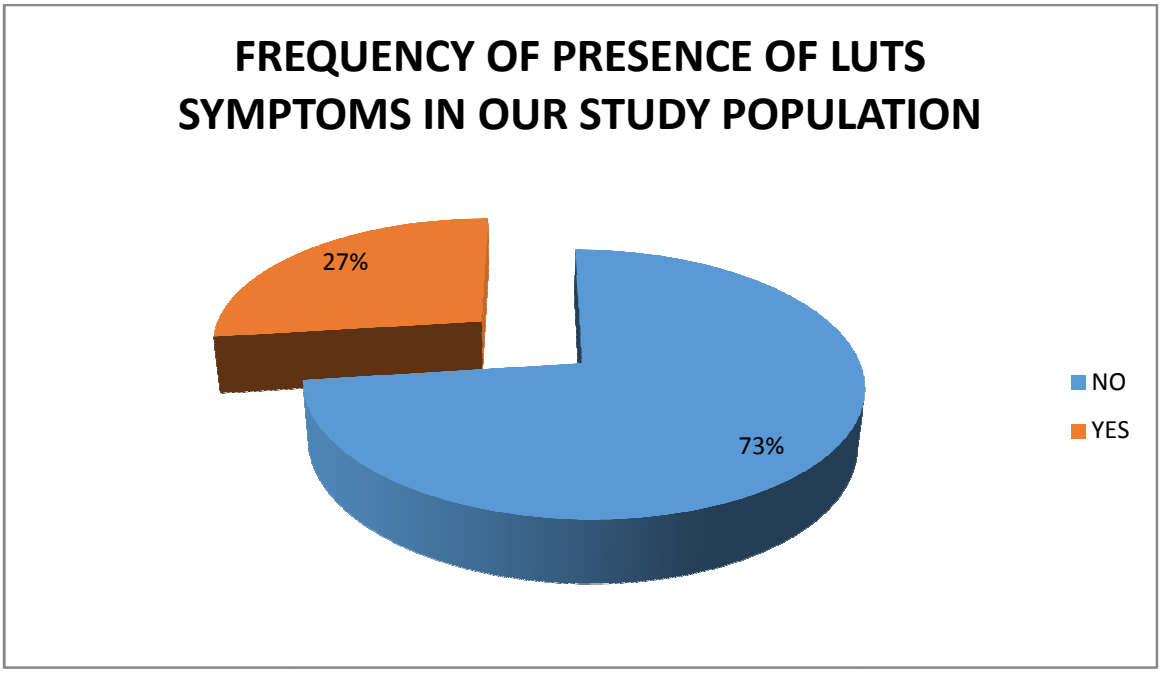
PIE CHART SHOWING AGEWISE DISTRIBUTION IN OUR STUDY GROUP



FREQUENCY OF PRESENCE OF LUTS SYMPTOMS IN OUR STUDY POPULATION

LUTS SYMPTOMS	FREQUENCY	PERCENT
NO	73	73.0
YES	27	27.0
Total	100	100.0

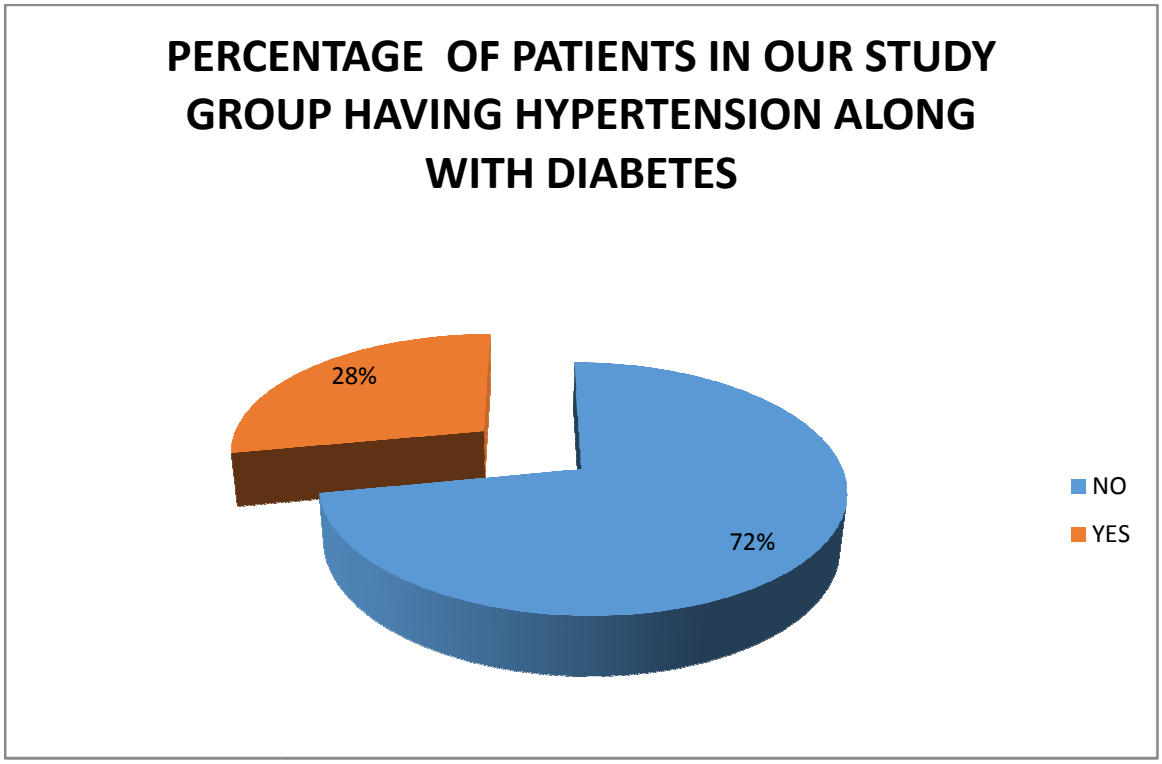
In our study group, out of the total population with prostatomegaly around 27% of patients had symptoms that are attributable to lower urinary tract.



PERCENTAGE OF PATIENTS IN OUR STUDY GROUP HAVING HYPERTENSION ALONG WITH DIABETES

HYPERTENSION	FREQUENCY	PERCENT
NO	72	72.0
YES	28	28.0
Total	100	100.0

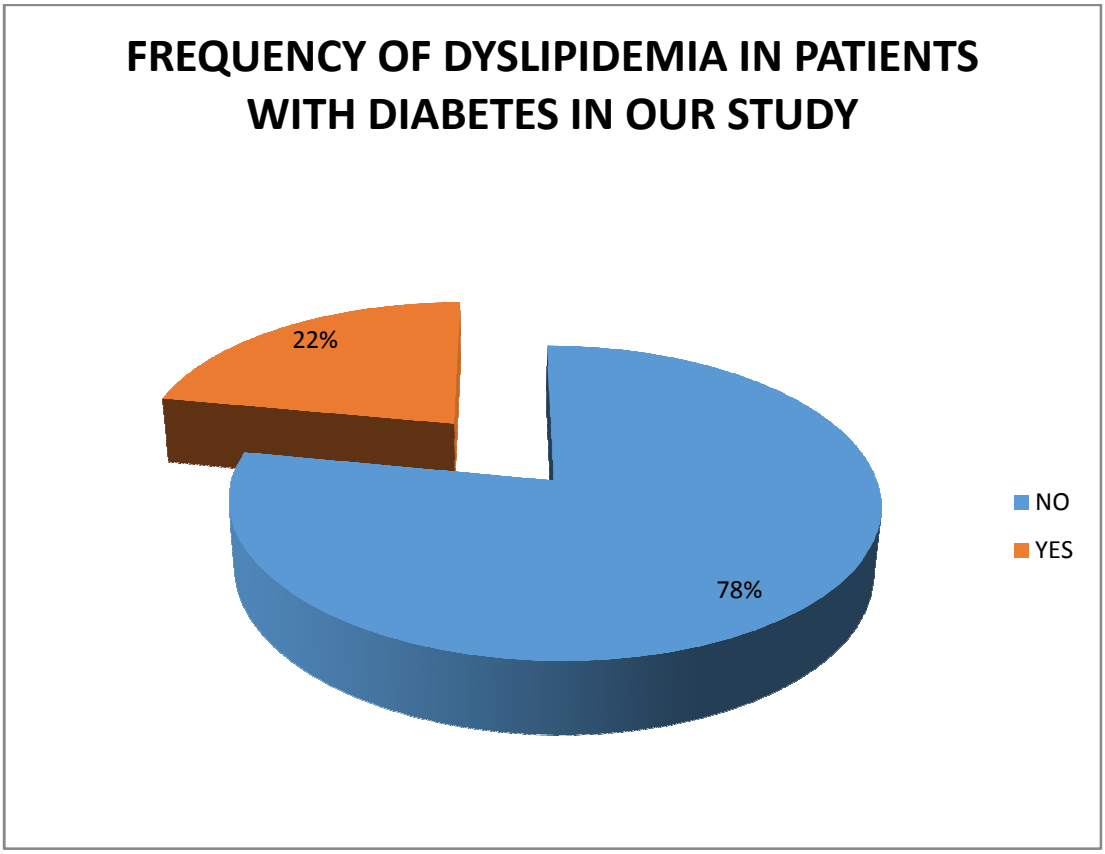
The above table depicts that among 100 patients with type 2 Diabetes, around 28% patients had coexistent hypertension.



FREQUENCY OF DYSLIPIDEMIA IN PATIENTS WITH DIABETES IN OUR STUDY

DYSLIPIDEMIA	FREQUENCY	PERCENT
NO	78	78.0
YES	22	22.0
Total	100	100.0

In our study group, among 100 patients with type 2 Diabetes about 22% patients had dyslipidemia.



PERCENTAGE OF PATIENTS ON OHA / INSULIN FOR THE DIABETES MANAGEMENT IN OUR STUDY POPULATION

TREATMENT	FREQUENCY	PERCENT
INSULIN	31	31.0
OHA	69	69.0
Total	100	100.0

It is evident from the above table that around 70 % patients are on OHA and 30% patients on Insulin.From the other studies,it was predicted that more the number of patients on insulin for uncontrollable hyperglycaemia, more the incidence of prostatomegaly.

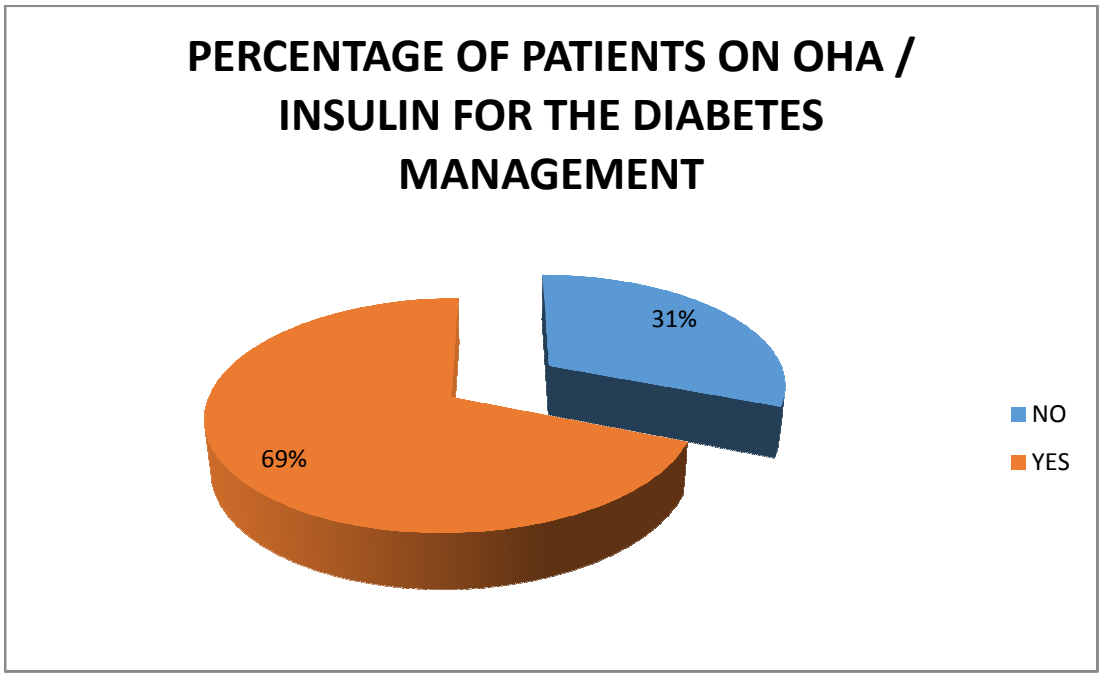
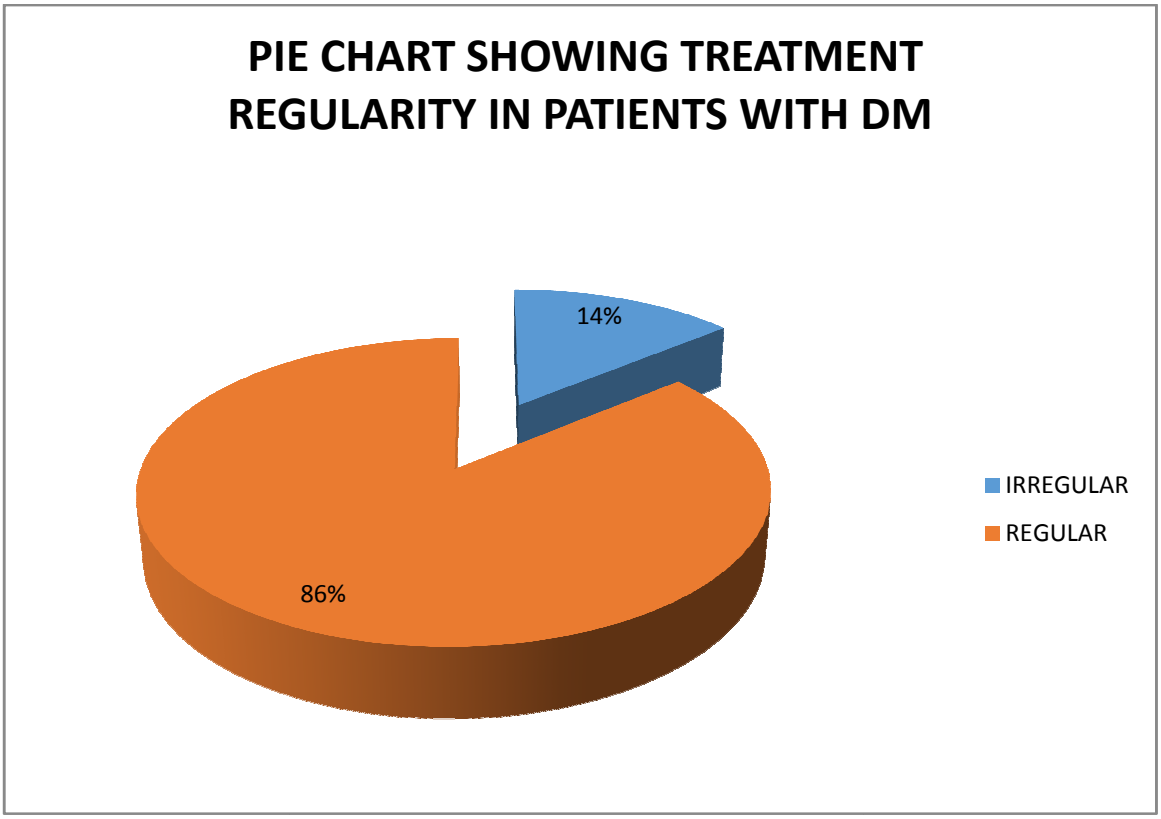


TABLE SHOWING TREATMENT REGULARITY IN PATIENTS WITH DIABETES

TREATMENT REGULARITY	FREQUENCY	PERCENT
IRREGULAR	14	14.0
REGULAR	86	86.0
Total	100	100.0

Around 86 % patients are on regular treatment, remaining 14% on irregular treatment for their Diabetes.



FREQUENCY OF PATIENTS IDENTIFIED ON PER RECTAL EXAMINATION TO HAVE PROSTATOMEGALY

IDENTIFICATION OF PROSTATOMEGALY ON PR	FREQUENCY	PERCENT
NO	87	87.0
YES	13	13.0
Total	100	100.0

From the given table, it would be suggested that around 13 % patients could be identified to have prostatomegaly on per rectal examination.

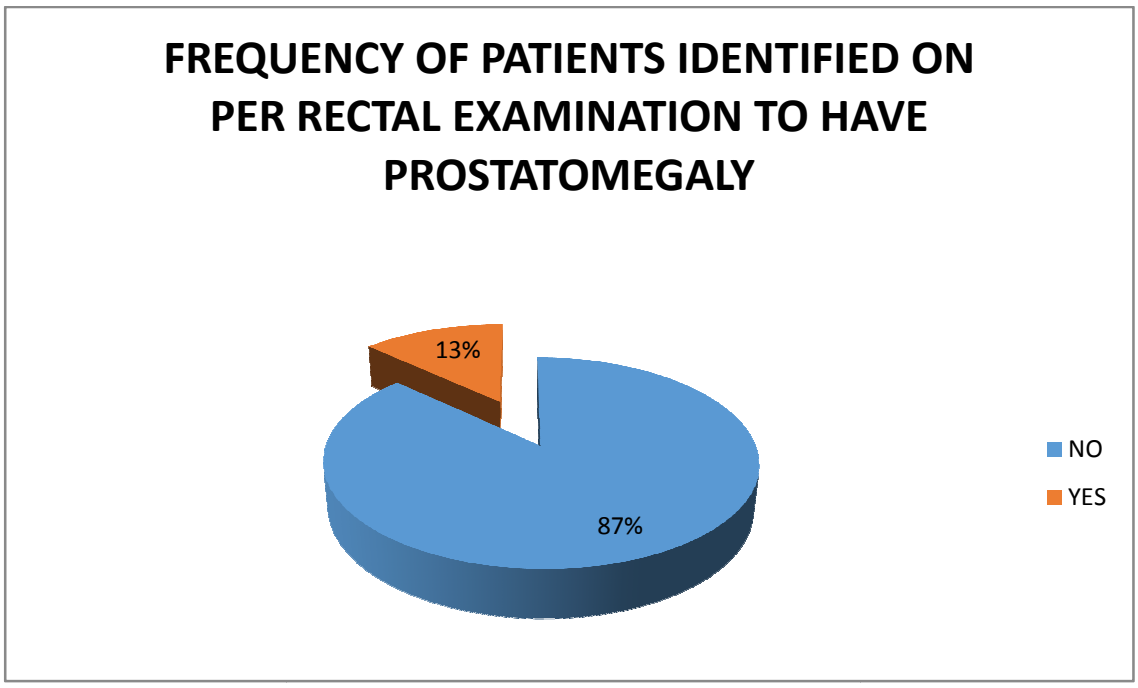


TABLE DEPICTING NATURE OF GLYCEMIC PROFILE IN OUR STUDY

GLYCEMIC PROFILE	FREQUENCY	PERCENT
POOR CONTROL	52	52.0
GOOD CONTROL	48	48.0
Total	100	100.0

It is obvious from the above table that around 52% patients had poor glycemic control.

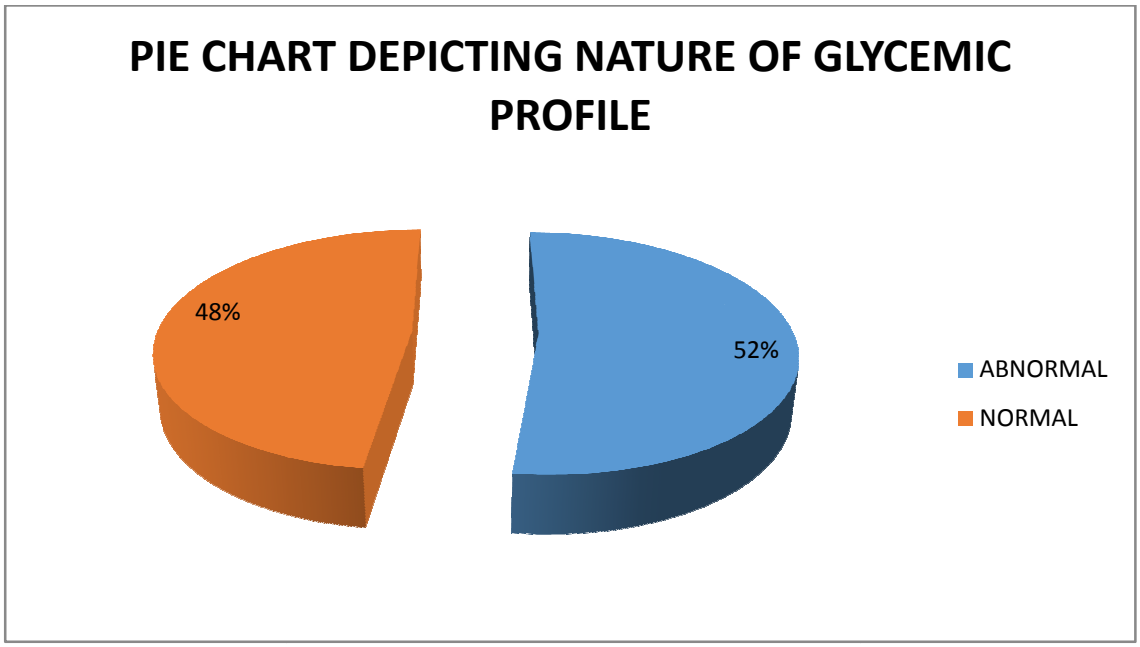
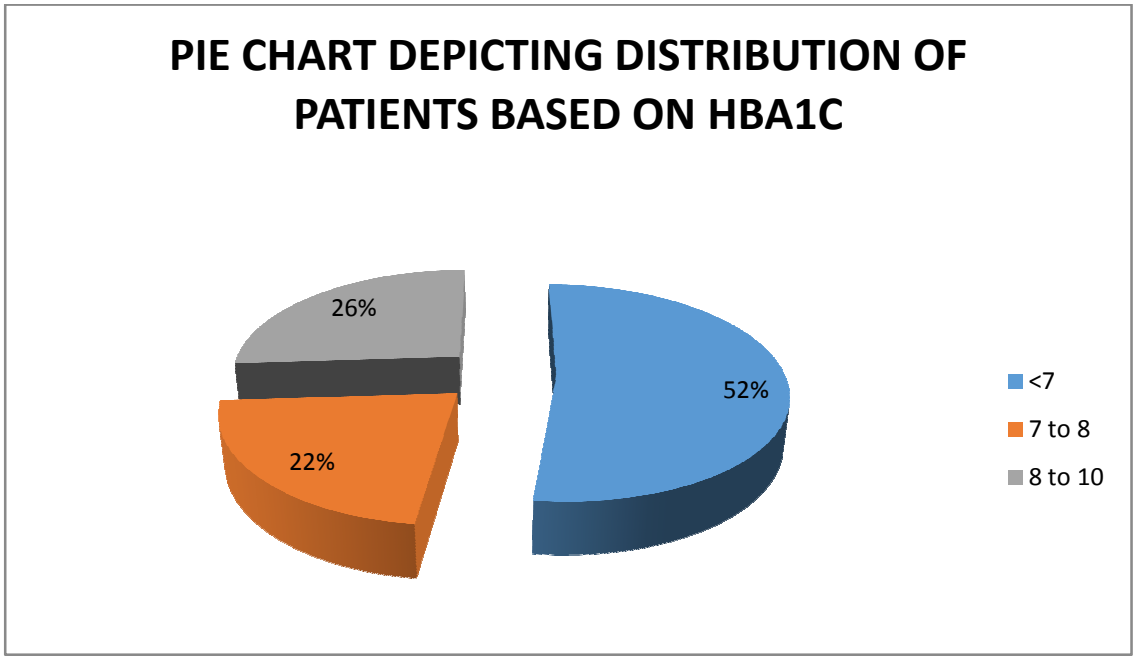


TABLE DEPICTING DISTRIBUTION OF PATIENTS BASED ON HBA1C

HBA1C	FREQUENCY	PERCENT
<7	52	52.0
7-8	22	22.0
8-10	26	26.0
Total	100	100.0

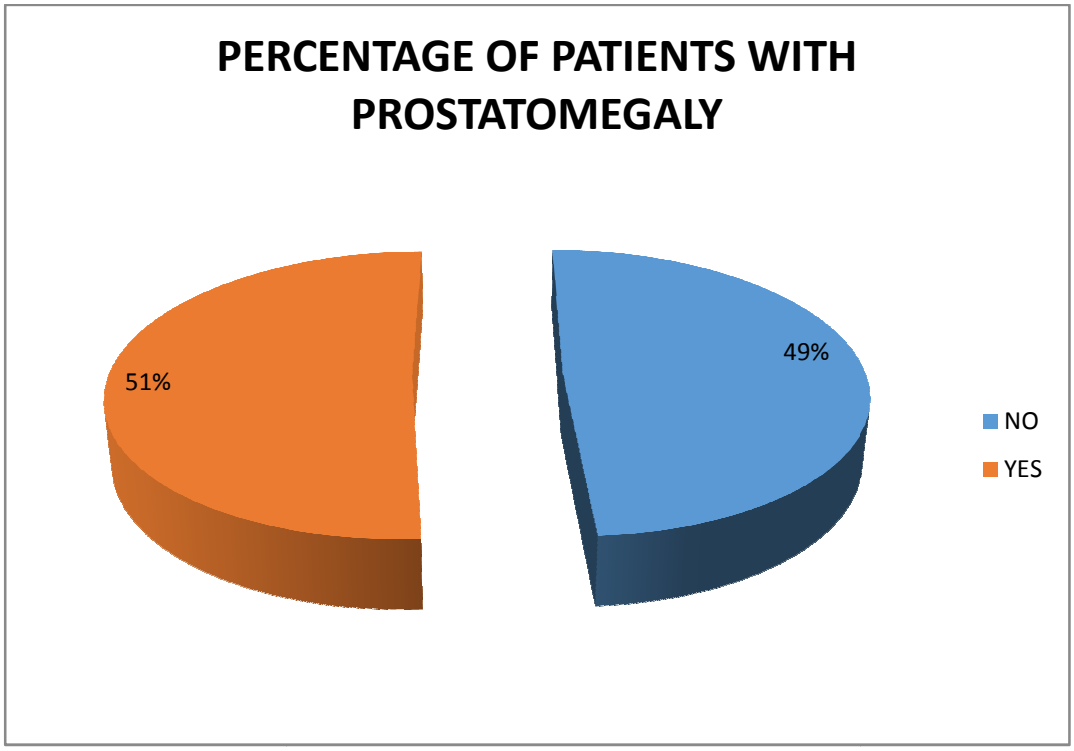
From the above table, it is evident clearly that around 50% patients have well controlled Diabetes, in comparison to 48% patients who have a poorly controlled Diabetes over a period of 3 months duration.



PERCENTAGE OF PATIENTS WITH PROSTATOMEGALY IN OUR STUDY

PROSTATOMEGLY	FREQUENCY	PERCENT
NO	49	49.0
YES	51	51.0
Total	100	100.0

Above table gives an idea that around 50% patients in our study group had prostatomegaly.



FREQUENCY OF PATIENTS FOR WHOM SERUM PROSTRATE SPECIFIC ANTIGEN WAS TESTED

SERUM PSA	FREQUENCY	PERCENT
	49	49.0
<4ng/ml	51	51.0
Total	100	100.0

In our study, out of 100 patients serum prostrate specific antigen was only tested to those patients who for whom prostatomegaly was present and it came to around 51% .For all the 51 patients, level of serum prostrate specific antigen was <4 ng/ml.

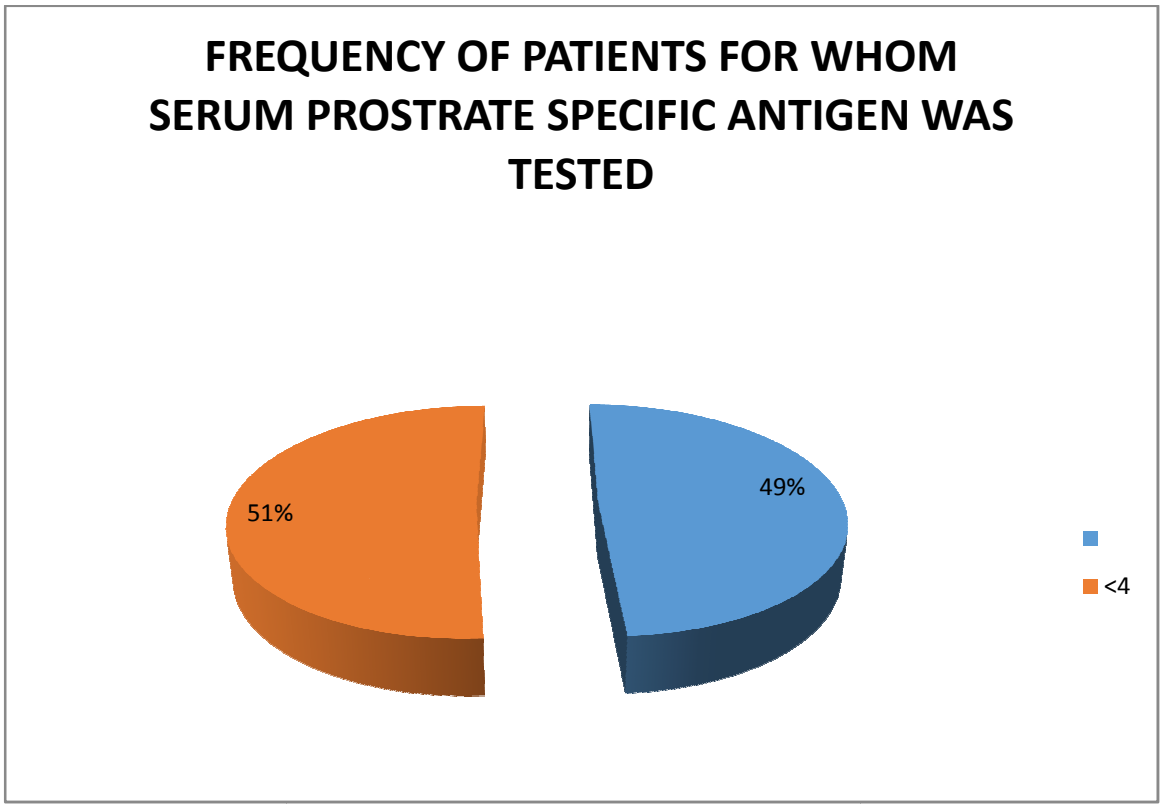


TABLE SHOWING DISTRIBUTION OF PATIENTS BASED ON DURATION OF DIABETES

DURATION OF DIABETES	FREQUENCY	PERCENT
<5 years	48	48.0
5 -10 years	15	15.0
10-20years	37	37.0
Total	100	100.0

From the given table,it is depicted that 48% of our patients had Diabetes for a period of <5 years,15 % patients had Diabetes for about 5-10 years, with the remaining population having Diabetes for a duration of 10- 20 years.

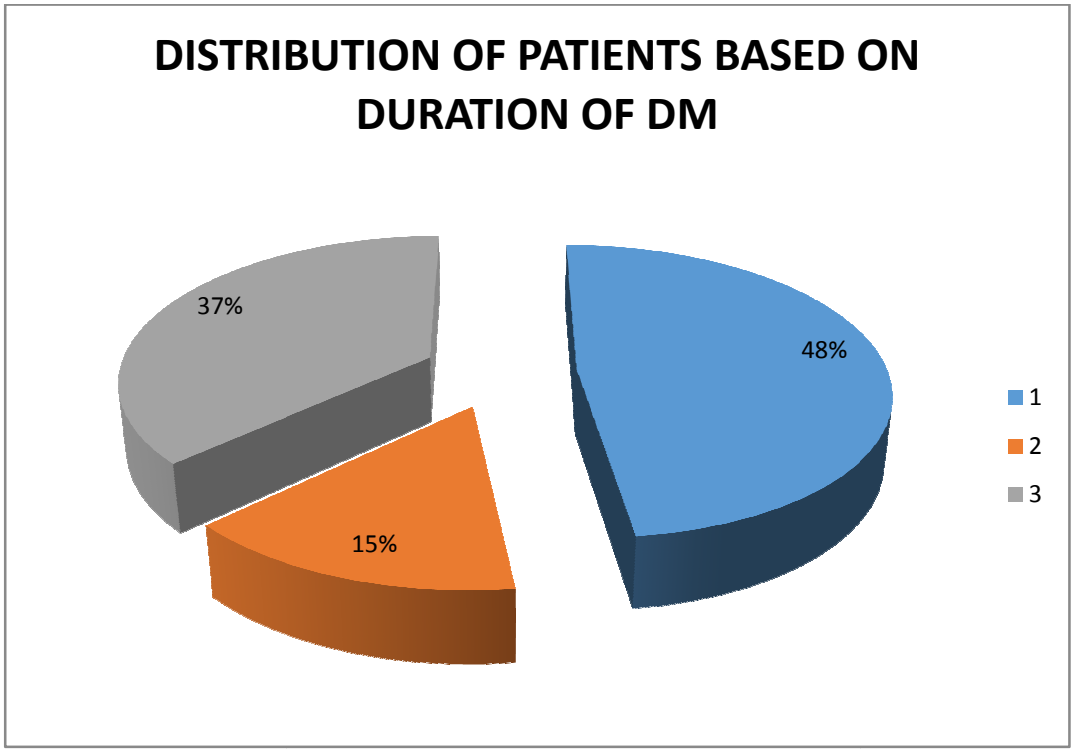


TABLE DEPICTING RELATIONSHIP BETWEEN DURATION OF DIABETES AND PRESENCE OF PROSTATOMEGALY

CROSSTAB			DURATION OF DIABETES			Total
			1-5 years	6-10 years	above 10 years	
Prostatomegly	NO	Count	38	6	5	49
		% within PROSTATOMEGLY	77.6%	12.2%	10.2%	100.0%
	YES	Count	10	9	32	51
		% within PROSTATOMEGLY	19.6%	17.6%	62.7%	100.0%
Total		Count	48	15	37	100
		% within PROSTATOMEGLY	48.0%	15.0%	37.0%	100.0%

Chi-Square=36.611**P<0.001

From the given cross tabulation ,it is evident that as the duration of Diabetes increases, size of prostrate gland also increases.

There is a strong correlation between duration of Diabetes mellitus and prostatomegaly, as the p value is <0.001 which is considered as significant.

BAR DIAGRAM SHOWING CORRELATION BETWEEN DURATION OF DIABETES AND PROSTATOMEGALY

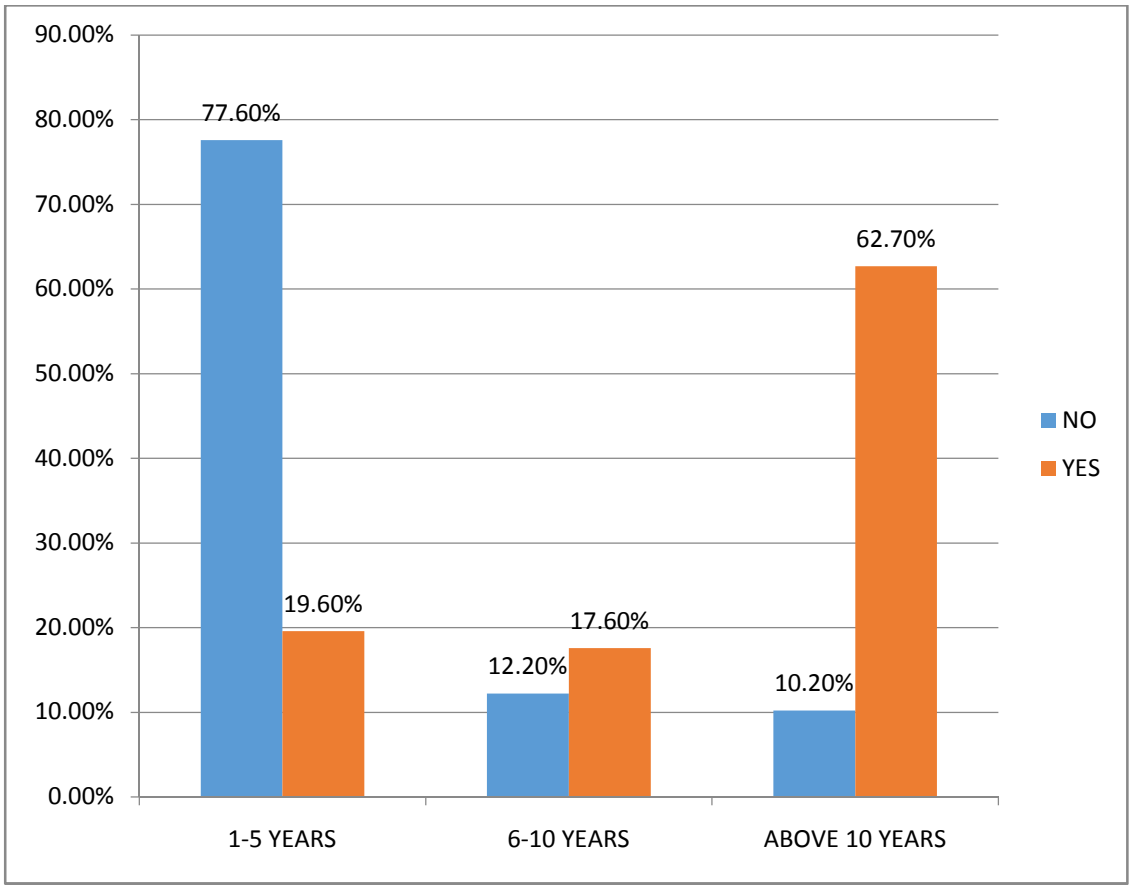


TABLE DEPICTING RELATIONSHIP BETWEEN CONTROL OF DIABETES AND PRESENCE OF PROSTATOMEGALY

CROSSTAB			HBA1C			Total
			<7	7-8	8-10	
Prostatomegly	NO	Count	35	8	6	49
		% within PROSTATOMEGLY	71.4%	16.3%	12.2%	100.0%
	YES	Count	17	14	20	51
		% within PROSTATOMEGLY	33.3%	27.5%	39.2%	100.0%
Total		Count	52	22	26	100
		% within PROSTATOMEGLY	52.0%	22.0%	26.0%	100.0%

Chi-Square=15.372**P<0.001

In our study,it is evidenced that those patients who had DIABETES of poorer control over a period of time has increasing number of prostatomegaly . Similarly, there is very less number of patients with prostatomegaly in those who have strict control of DIABETES.

It is found to be correlating significantly as the p value is < 0.001.

BAR DIAGRAM DEPICTING RELATIONSHIP BETWEEN CONTROL OF DIABETES AND PRESENCE OF PROSTATOMEGALY

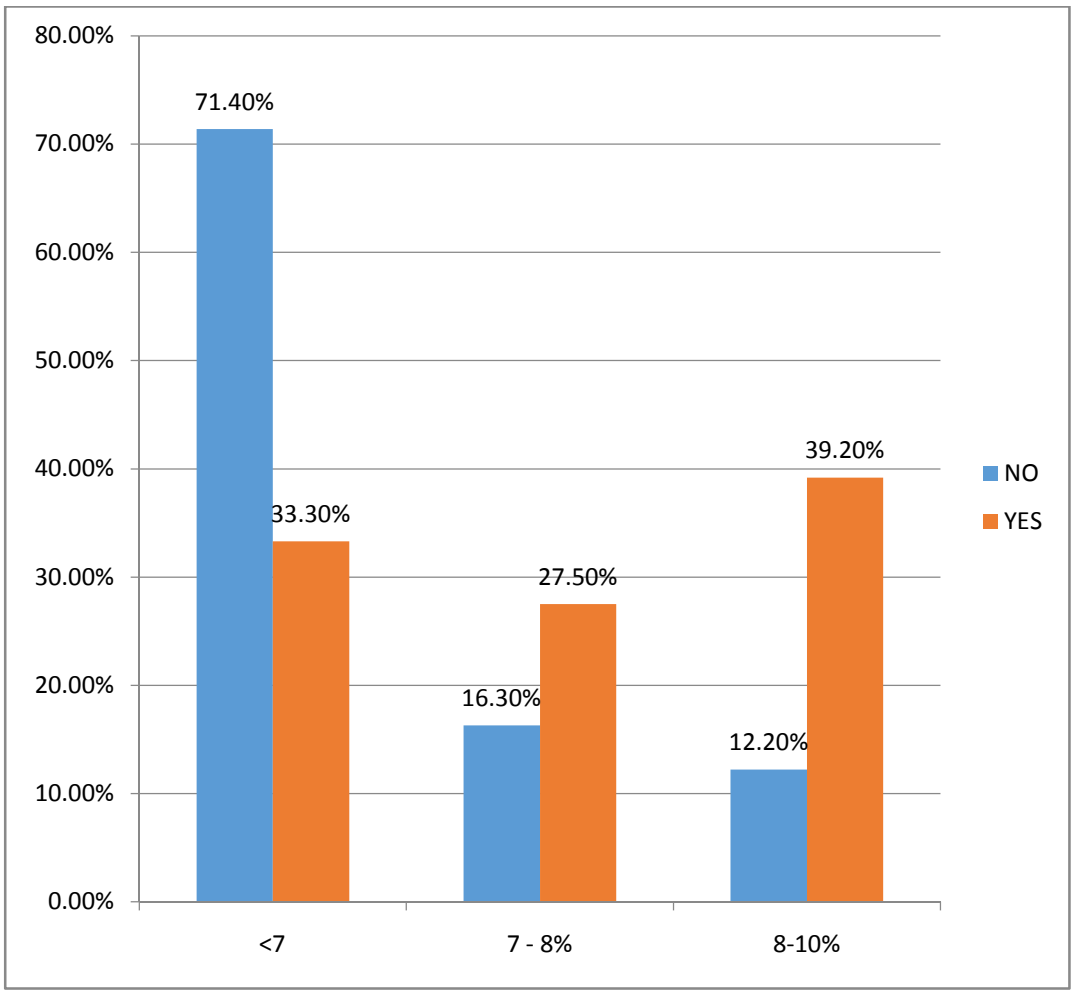


TABLE SHOWING PERCENTAGE OF PATIENT DISTRIBUTION BASED ON THE TREATMENT FOR DIABETES (OHA /INSULIN)

CROSSTAB			TREATMENT		Total
			INSULIN	OHA	
Prostatomegaly	NO	Count	0	49	49
		% within PROSTATOMEGALY	0.0%	100.0%	100.0%
	YES	Count	31	20	51
		% within PROSTATOMEGALY	60.8%	39.2%	100.0%
Total		Count	31	69	100
		% within PROSTATOMEGALY	31.0%	69.0%	100.0%

Chi-Square=43.166**P<0.001

The above table clearly depicts that those patients on Insulin has a higher incidence of prostatomegaly,in comparison to patients who were on oral hypoglycaemic agents. It is found to be correlating significantly as the p value is < 0.001.

BAR DIAGRAM SHOWING PERCENTAGE OF PATIENT DISTRIBUTION BASED ON THE TREATMENT FOR DIABETES (OHA /INSULIN)

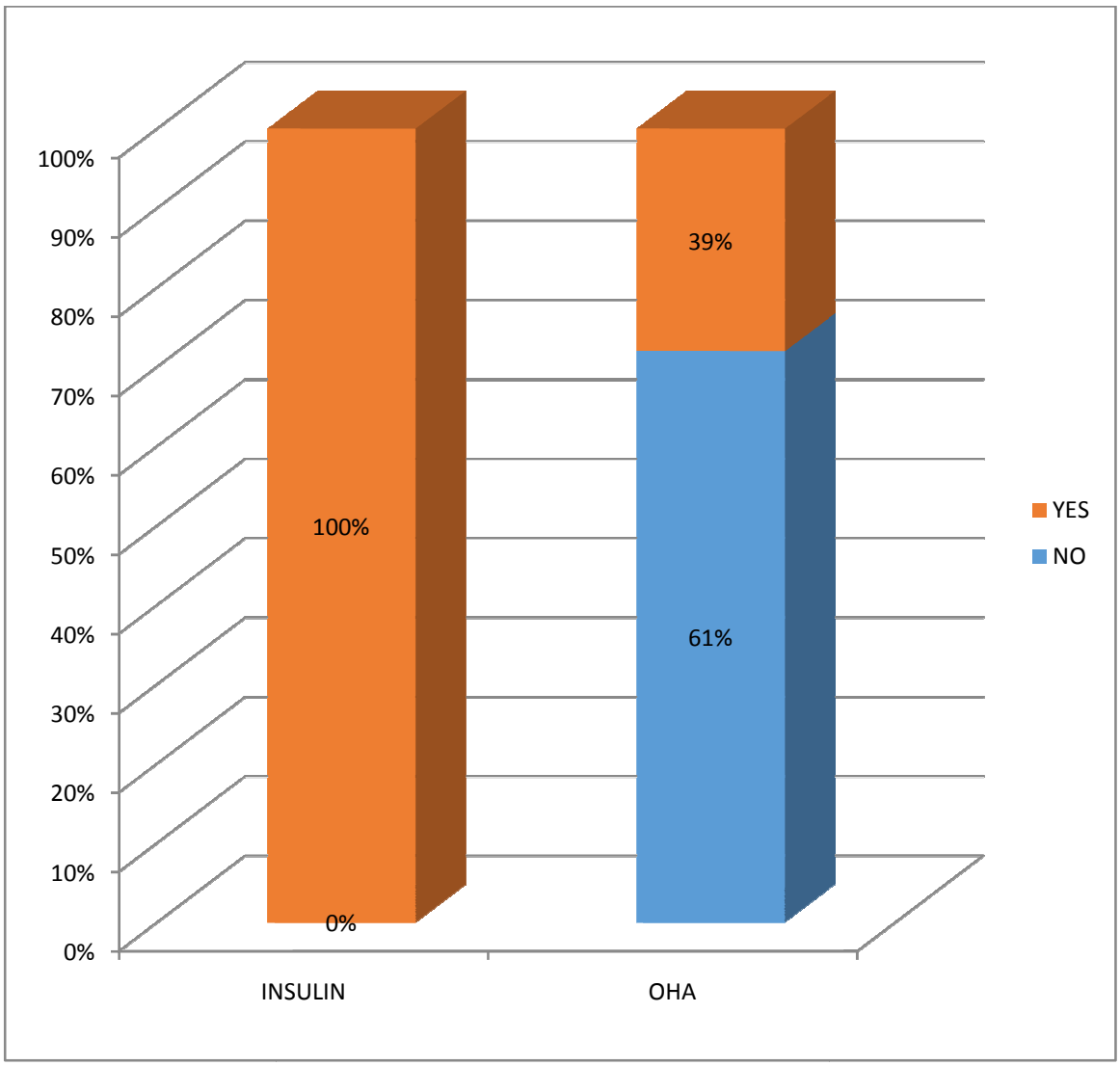


TABLE SHOWING PERCENTAGE OF PATIENTS IDENTIFIED ON PERRECTAL EXAMINATION AS HAVING PROSTATOMEGALY

CROSSTAB			PR		Total
			NO	YES	
Prostatomegaly	NO	Count	49	0	49
		% within PROSTATOMEGALY	100.0%	0.0%	100.0%
	YES	Count	38	13	51
		% within PROSTATOMEGALY	74.5%	25.5%	100.0%
Total		Count	87	13	100
		% within PROSTATOMEGALY	87.0%	13.0%	100.0%

Chi-Square=14.357**P<0.001

It has been evident from the above table, among patients having prostatomegaly about 25% patients are identified by per rectal examination. It is found to be correlating significantly as the p value is < 0.001.

BAR CHART SHOWING PERCENTAGE OF PATIENTS IDENTIFIED ON PERRECTAL EXAMINATION AS HAVING PROSTATOMEGALY

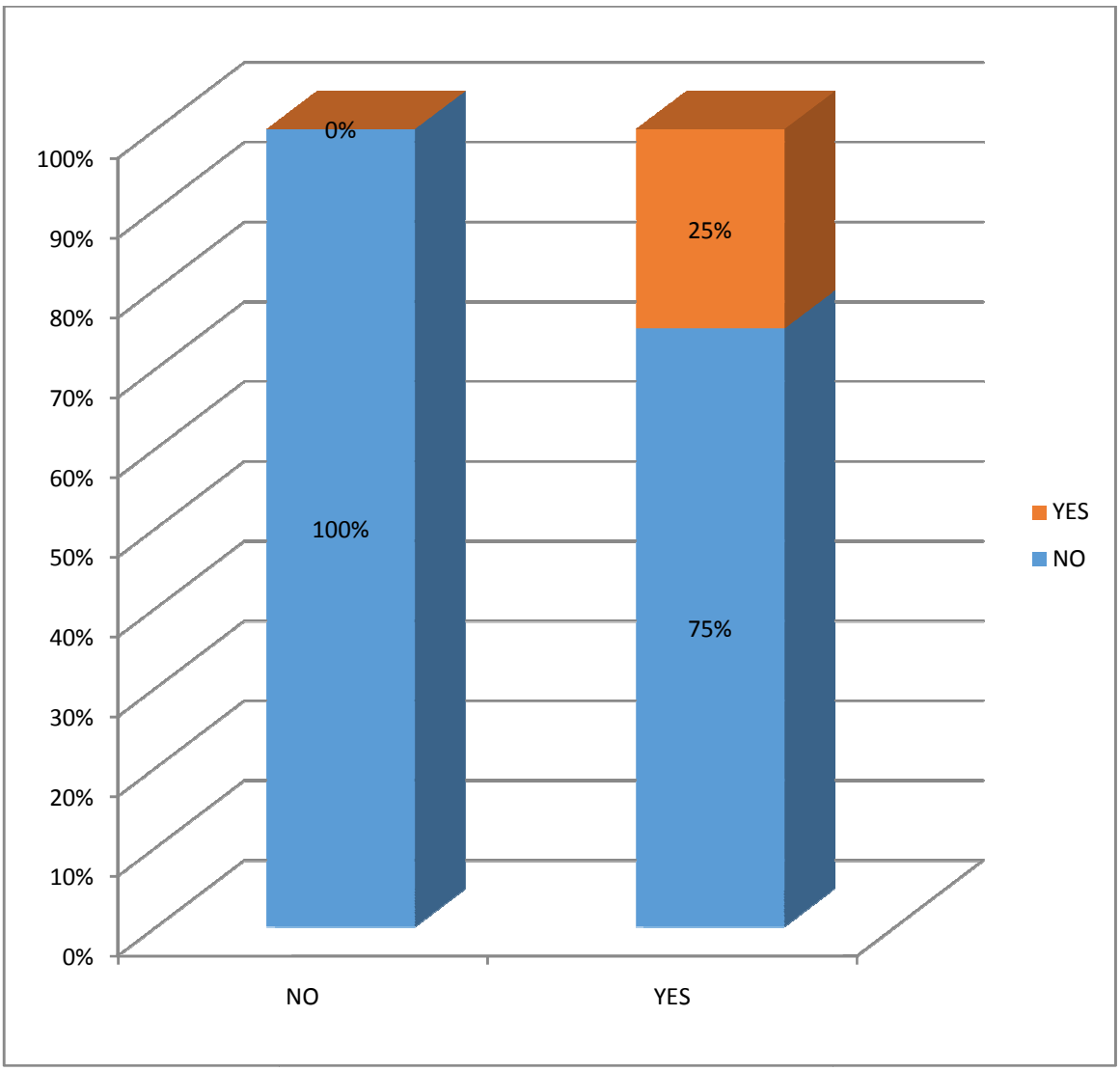


TABLE DEPICTING RELATIONSHIP BETWEEN CONTROL OF DIABETES AND PRESENCE OF PROSTATOMEGALY

CROSSTAB			FBS / PPBS		Total
			ABNORMAL	NORMAL	
Prostatomegly	NO	Count	18	31	49
		% with PROSTATOMEGALY	36.7%	63.3%	100.0%
	YES	Count	34	17	51
		% with PROSTATOMEGALY	66.7%	33.3%	100.0%
Total		Count	52	48	100
		% with PROSTATOMEGALY	52.0%	48.0%	100.0%

Chi-Square=8.970*P=0.003

In our study, it is evidenced that those patients who had Diabetes of poorer control over a period of time has increasing number of prostatomegaly . Similarly, there is very less number of patients with prostatomegaly in those who have strict control of Diabetes.

It is found to be correlating significantly as the p value is < 0.001.

BAR CHART DEPICTING RELATIONSHIP BETWEEN CONTROL OF DIABETES AND PRESENCE OF PROSTATOMEGALY

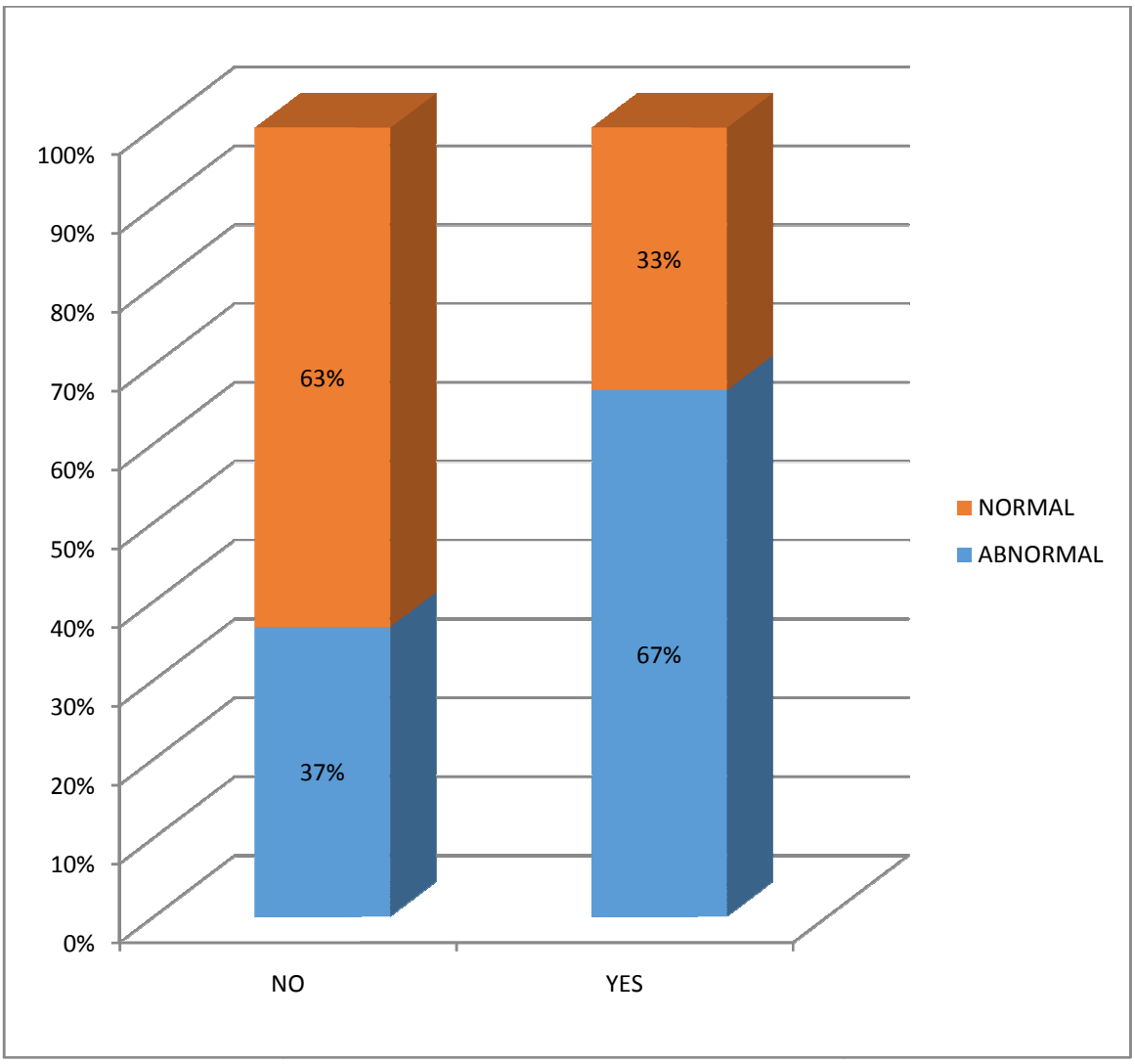


TABLE CORRELATING REGULARITY OF TREATMENT WITH PREVALENCE OF PROSTATOMEGALY

CROSSTAB			REGULAR_TT		Total
			IRREGULAR	REGULAR	
Prostatomegaly	NO	Count	0	49	49
		% with PROSTATOMEGALY	0.0%	100.0%	100.0 %
	YES	Count	14	37	51
		% with PROSTATOMEGALY	27.5%	72.5%	100.0 %
Total		Count	14	86	100
		% with PROSTATOMEGALY	14.0%	86.0%	100.0 %

Chi-Square=15.641**P<0.001

The table depicts that increasing percentage of patients found to have prostatomegaly among those on irregular treatment compared to patients on regular treatment. Similarly, there is less number of patients with Prostatomegaly among those on regular treatment.

It is found to correlate well significantly as the p value is < 0.001.

BAR DIAGRAM CORRELATING REGULARITY OF TREATMENT WITH PREVALENCE OF PROSTATOMEGALY

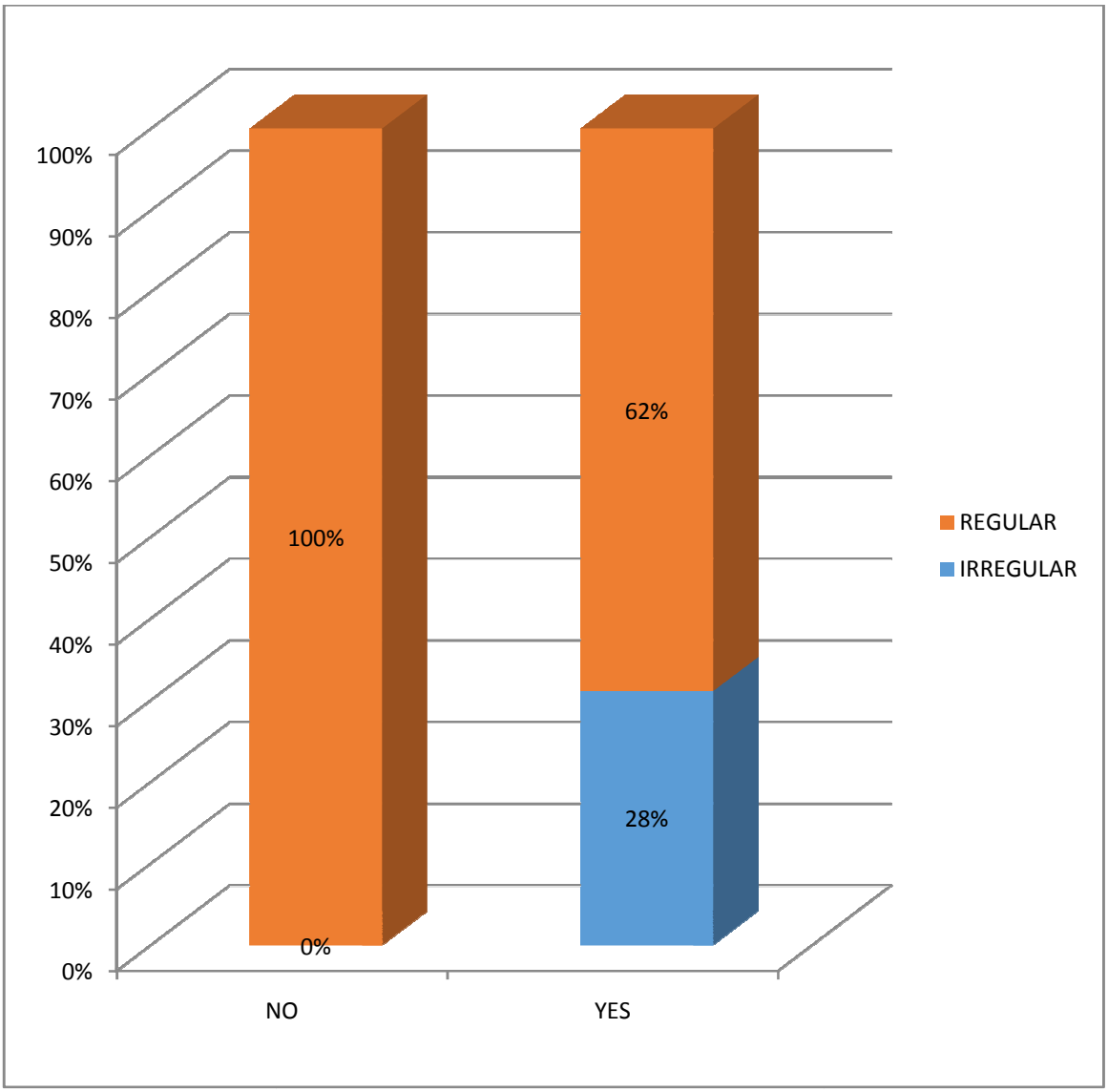


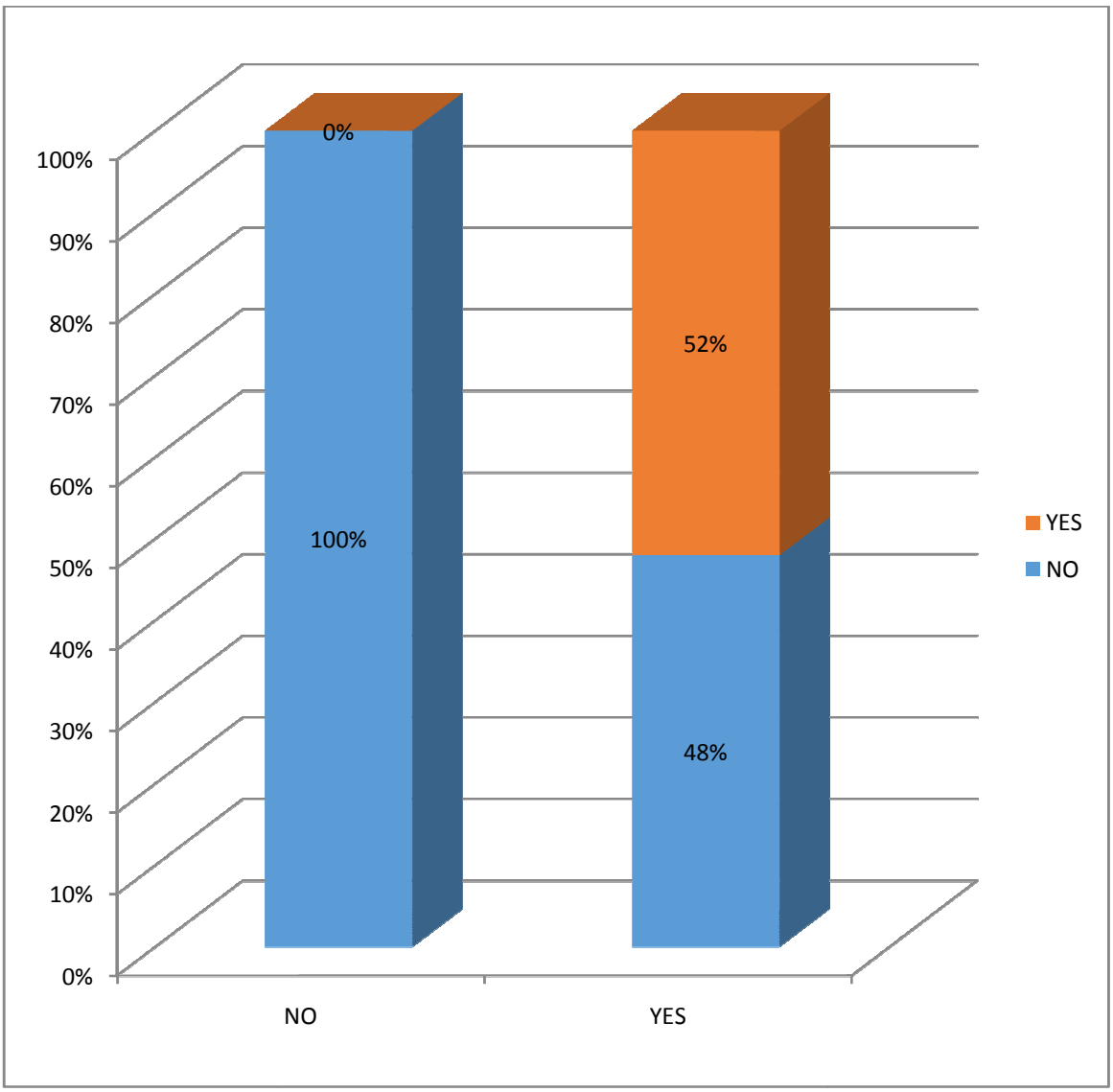
TABLE DEPICTING PERCENTAGE OF PATIENTS HAVING LOWER URINARY TRACT SYMPTOMS AMONG THOSE WITH PROSTATOMEGALY

CROSSTAB			LUTS SYMPTOMS		Total
			NO	YES	
Prostatomegaly	NO	Count	49	0	49
		% with PROSTATOMEGALY	100.0%	0.0%	100.0%
	YES	Count	24	27	51
		% with PROSTATOMEGALY	47.1%	52.9%	100.0%
Total		Count	73	27	100
		% with PROSTATOMEGALY	73.0%	27.0%	100.0%

Chi-Square=36.536**P<0.001

The above table projects the fact that among those patients with prostatomegaly, only about 52% patients will present with symptoms of lower urinary tract. It is found to correlate well significantly as the p value is < 0.001.

BAR DIAGRAM DEPICTING PERCENTAGE OF PATIENTS HAVING LOWER URINARY TRACT SYMPTOMS AMONG THOSE WITH PROSTATOMEGALY



CROSSTABULATION DEPICTING AGE WISE DISTRIBUTION OF PROSTATOMEGALY

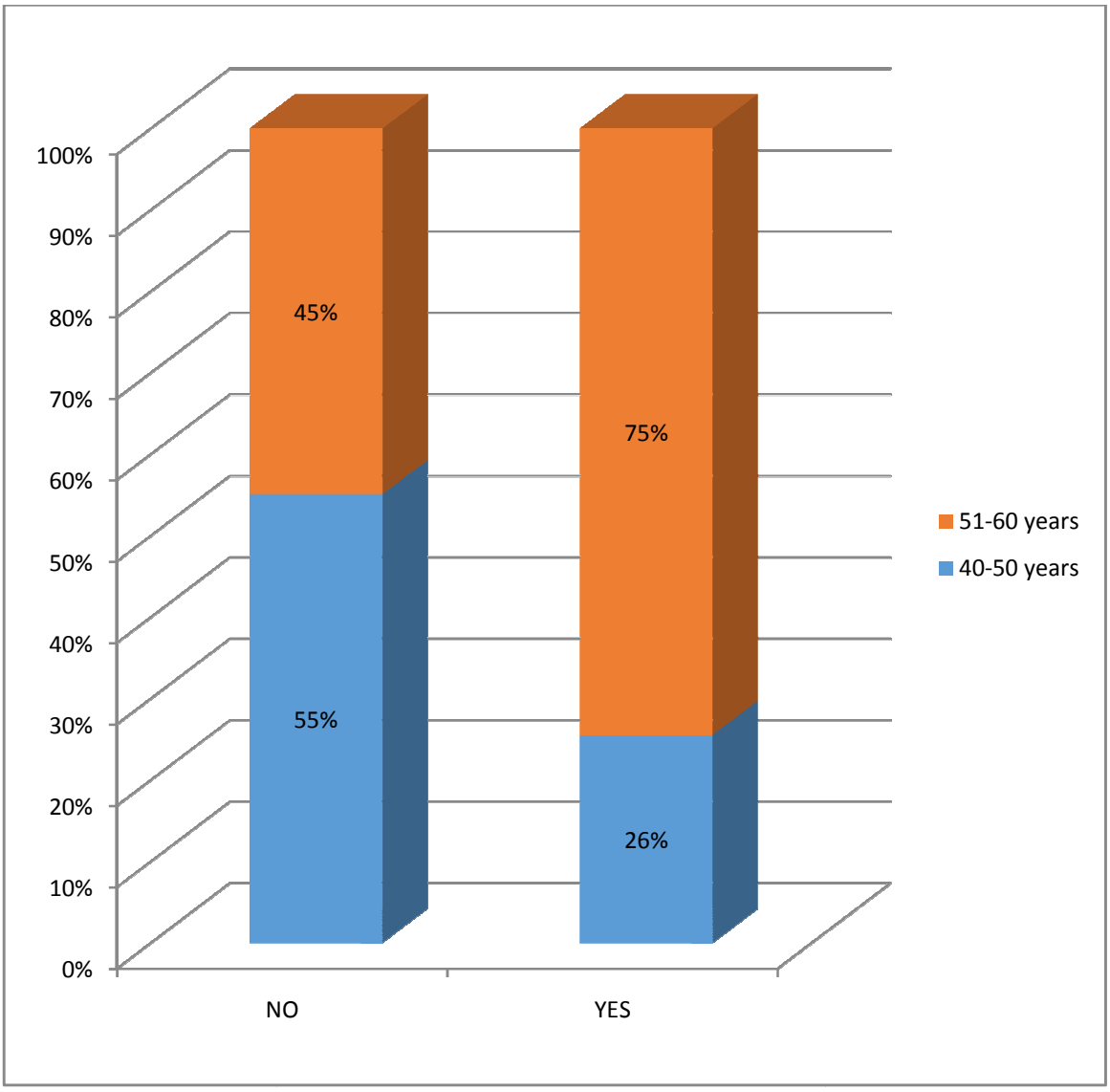
PROSTATOMEGALY * age group Crosstabulation			Age Group		Total
			40-50 years	51-60 years	
Prostatomegaly	NO	Count	27	22	49
		% within PROSTATOMEGALY	55.1%	44.9%	100.0%
	YES	Count	13	38	51
		% within PROSTATOMEGALY	25.5%	74.5%	100.0%
Total		Count	40	60	100
		% within PROSTATOMEGALY	40.0%	60.0%	100.0%

Chi-Square=9.130**P<0.001

From the given table, it is evident that among patients with prostatomegaly 75% falls within the age group of 50-60 years , compared to 25 % in 40- 50 years patients . It shows that incidence of prostatomegaly increases as the age of the patient increases and as the control of Diabetes becomes poor.

It is found to correlate well significantly as the p value is < 0.001.

BAR DIAGRAM DEPICTING AGE WISE DISTRIBUTION OF PROSTATOMEGALY



DISCUSSION

DISCUSSION

The study being conducted as a prospective and retrospective observational study among patients attending Diabetology OPD /Institute of internal medicine at Madras Medical College and Rajiv Gandhi Government General Hospital. The sample size was 100. After getting the informed consent of the patients and their attending close relatives, the patients were subjected to history taking, physical examination and relevant laboratory testing and imging. These were done to identify the presence of prostatomegaly and to correlate it with the glycemic profile and duration of Diabetes in the patients.

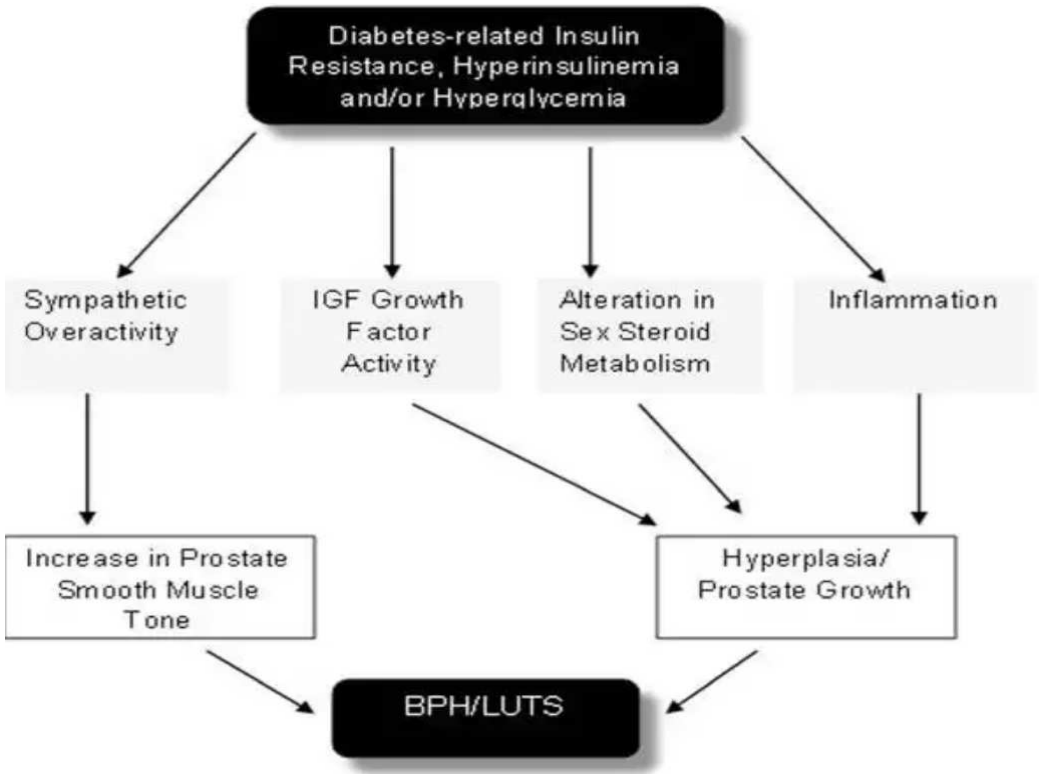
Type of Diabetes and associated diabetic complication does not seem to have any impact on risk of prostatomegaly and LUTS. Patients with Diabetes mellitus more than 6 years duration are found to be associated with a higher prevalence of UTI and LUTS secondary to prostatomegaly.

Benign prostatic hyperplasia (BPH) is a representative urological disease in males at senescence. The etiology of BPH being multifactorial, with chronic conditions, like hypertension, overweight , raised low density lipoprotein (LDL) cholesterol levels and a high insulin levels, constitutes for the development of BPH. Prevalence of hyperglycemia and resistance to insulin in a BPH population was

investigated and the relationship among prostatomegly and metabolic syndrome tends to correlate well.

A study done by Kallol Bhattacharjee et al also correlates the presence of prostatomegaly with duration of DIABETES and glycemic profile.

The probability of males with BPH in the presence of metabolic syndrome is high compared to those without metabolic syndrome. Consequently, in BPH patients, a very careful evaluation for metabolic syndrome is needed, with the early diagnosis and a proper management of metabolic syndrome should accompany the treatment of BPH.



The predominant feature involving the genitourinary system in males with Diabetes mellitus are lower urinry tract symptoms, retrograde ejaculation , erectile dysfunction (ED) and, and dysfunction of bladder. Prevalence of 51% prostatomegaly has been recorded in our study in patients of age group 40-60 years with Diabetes which is significantly increased than the prevalence (35%) found among general population . Since ,plenty of other factors contribute for the development of prostatomegaly, it is prime that males with Diabetes are instructed to maintain glycemic control, lifestyle changes such as smoking contributing to the progression of prostatomegaly.

Reduced bladder sensation , increment in bladder capacity, impaired contractility of detrusor muscles are all the charachteristic features of diabetic bladder dysfunction. Management strategies are aimed at voiding strategies aimed at symptom relief , prevention of infections and maintaining continence.

LIMITATIONS

LIMITATIONS OF THE STUDY

A multi centric study with a larger sample size and further longer follow up is essential to assess the predictive power of these prognostication tools in a more comprehensive manner.

CONCLUSION

CONCLUSION

Benign prostatic hyperplasia and symptoms of lower urinary tract are frequently encountered in type 2 diabetic patients.

Elderly patients of age >50 years, longer duration of Diabetes, non-adherence to treatment, poor glycemic profile, insulin therapy are all a significant risk factor for the development of prostatic enlargement and thereby, leading on to benign prostatic hyperplasia and lower urinary tact symptoms. Uncontrolled blood glucose (FBS/ PPBS) with HbA1 C>7%, post void residue >150 ml and prostate volume >40 cc correlates well.

It has become evident clearly from our study that, the presence of prostatomegaly correlates well with the glycemic profile and the duration of Diabetes mellitus .The prevalence of prostatomegaly is thus, found to be increased in type 2 diabetic patients aged 40-60 years.

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BIBLIOGRAPHY

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ABBREVIATIONS

DM	-	DIABETES MELLITUS
BPH	-	BENIGN PROSTATIC HYPERPLASIA
MODY	-	MATURITY ONSET DIABETES OF YOUNG
GDM	-	GESTATIONAL DIABETES MELLITUS
ADA	-	AMERICAN DIABETES ASSOCIATION
IDF	-	INTERNATIONAL DIABETES FEDERATION
FPG	-	FASTING PLASMA GLUCOSE
HBA1c	-	GLYCOSYLATED HEMOGLOBIN
IGT	-	IMPAIRED GLUCOSE TOLERANCE
IFG	-	IMPAIRED FASTING GLUCOSE
BMI	-	BODY MASS INDEX
CRP	-	C REACTIVE PROTEIN
IAPP	-	ISLET AMYLOID POLYPEPTIDE
GLUT4	-	GLUCOSE TRANSPORTER 4
CDC	-	CENTER FOR DISEASE CONTROL
TNF	-	TUMOUR NECROSIS FACTOR ALPHA
HDL	-	HIGH DENSITY LIPOPROTEIN
LDL	-	LOW DENSITY LIPOPROTEIN
BOO	-	BLDDER OUTLET OBSTRUCTION
LUTS	-	LOWER URINARY TRACT SYMPTOMS

ANNEXURES

PROFORMA

NAME OF THE PATIENT :

AGE / SEX :

OP/ NUMBER :

OCCUPATION :

ADDRESS :

CONTACT NUMBER :

COMPLAINTS

- Urinary frequency
- Urinary urgency
- Hesitancy
- Interrupted, weak stream
- Incomplete bladder emptying
- Straining
- Dribbling

PAST HISTORY

K/C/O PROSTATIC CARCINOMA

H/O DIABETES

TYPE 1 / TYPE 2 -

DURATION -

ON OHA / INSULIN -

REGULAR / IRREGULAR TREATMENT -

GENERAL EXAMINATION

Pallor: Icterus: Cyanosis: Clubbing:

Lymphadenopathy: Edema:

VITALS

Pulse Rate: BP: Respiratory rate: Temperature:

SYSTEMIC EXAMINATION

CARDIOVASCULAR SYSTEM :

RESPIRATORY SYSTEM :

ABDOMEN :

CENTRAL NERVOUS SYSTEM :

INVESTIGATIONS:

FASTING BLOOD GLUCOSE

POSTPRANDIAL BLOOD GLUCOSE

HBA1c

SERUM PROSTRATE SPECIFIC ANTIGEN

(IF APPLICABLE)

ULTRASONOGRAM OF PELVIS

- PROSTRATE SIZE/VOLUME
- POST-RESIDUAL VOLUME

THESIS APPROVAL CERTIFICATE

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.S.Prabhakaran
Post Graduate in M.D. (General Medicine)
Institute of Internal Medicine
Madras Medical College
Chennai 600 003

Dear Dr.S.Prabhakaran,

The Institutional Ethics Committee has considered your request and approved your study titled **"A STUDY ON PREVALANCE OF PROSTATOMEGALY AND ITS CORRELATION WITH DURATION AND GLYCEMIC CONTROL IN PATIENTS WITH TYPE 2 DIABETES MELLITUS " - NO.(II) 10032016.**

The following members of Ethics Committee were present in the meeting hold on **22.03.2016** conducted at Madras Medical College, Chennai 3

1.Dr.C.Rajendran, MD.,	:Chairperson
2.Dr.R.Vimala,MD.,Dean,MMC,Ch-3	:Deputy Chairperson
3.Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	: Member Secretary
4.Prof.P.Raghumani,MS, Dept.of Surgery,RGGGH,Ch-3	: Member
5.Dr.Baby Vasumathi, Director, Inst. of O&G,Ch-8	: Member
6.Prof.M.Saraswathi,MD.,Director, Inst.of Path,MMC,Ch-3	: Member
7.Prof.Srinivasagalu,Director,Inst.of Int.Med.,MMC,Ch-3	: Member
8.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person
9.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
10.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

MADRAS MEDICAL COLLEGE
31 MAR 2016
CHENNAI-600 003

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE.
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

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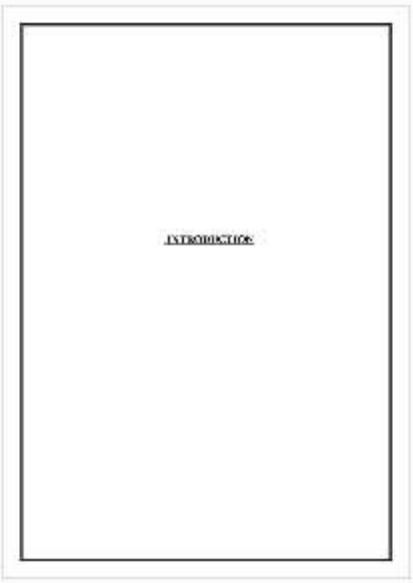

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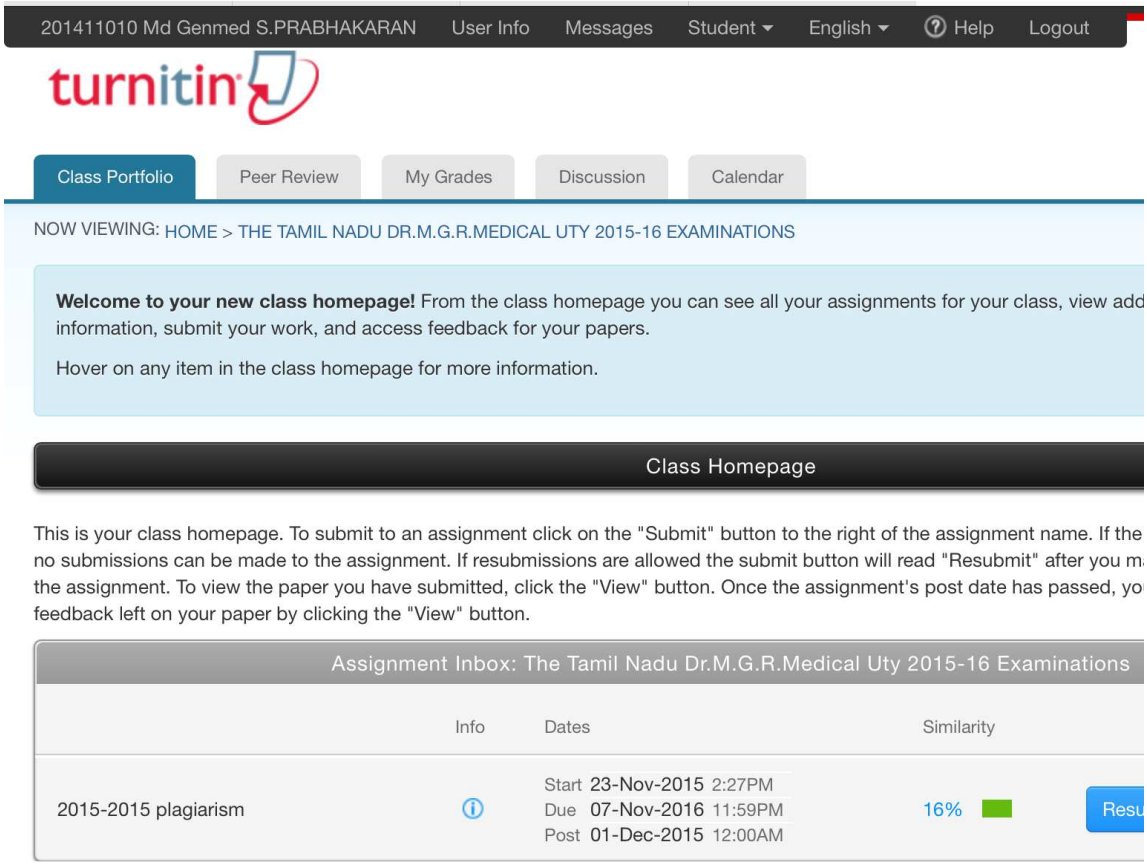

INFORMATIONSHEET

We are conducting a study on "**A STUDY ON PREVALANCE OF PROSTATOMEGALY AND ITS CORRELATION WITH DURATION AND GLYCEMIC CONTROL IN PATIENTS WITH TYPE 2 DIABETES MELLITUS** " among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your co-operation to undergo FBS/ PPBS , HBA1C , USG OF PELVIS, SERUM PSA (if applicable) may be valuable to us.

The purpose of this study is to identify the presence of prostatomegaly in patients with type 2 diabetes mellitus and correlating it with duration and glycemic profile of diabetes .

We are selecting certain cases and if you are found eligible, we would like to perform extra tests and special studies which in any way do not affect your final report or management.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator	Signature/left thumb impression of Participant

Date :
Place :

ஆராய்ச்சி தகவல் தாள்

சென்னை ராஜீவ்காந்தி அரசு பொது மருத்துவமனையின் பொது மருத்துவத்துறையில் "அப்பாச்சி-2 மற்றும் சோஃபா அளவீடுகளை குறுதி நஞ்சு-பல்லுறுப்பு செயல் பிறழ்ச்சியின் இறப்பு விகிதத்தின் குறிகாட்டிகளாய் ஒப்பிட்டு ஆராய்தல்" பற்றிய ஆய்வு நடைபெறுகிறது.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். இதனால் தங்களது சிகிச்சையில் பாதிப்பு ஏற்படாது என்பதையும் தெரிவித்துக்கொள்கிறோம்.

இந்த ஆய்வில் தங்களுக்கு மருத்துவபரிசோதனை, இரத்தப் பரிசோதனை, ஸ்கேன், சிறுநீர் பரிசோதனை மற்றும் எக்ஸ்ரே (X-Ray) பரிசோதனை செய்யப்படும்.

முடிவுகளை அல்லது கருத்துக்களை வெளியிடும்போதோ அல்லது ஆராய்ச்சியின்போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிட மாட்டோம் என்பதை தெரிவித்துக்கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின்பேரில்தான் இருக்கிறது. மேலும் நீங்கள் எந்த நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

இந்த சிறப்பு பரிசோதனைகளின் முடிவுகளையும் நோயின் தன்மை பற்றியும் ஆராய்ச்சியின்போது அல்லது ஆராய்ச்சியின் முடிவின்போது தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

பங்கேற்பாளர் கையொப்பம்

ஆராய்ச்சியாளர் கையொப்பம்

நாள் :
இடம் :

PATIENT CONSENT FORM

Study Detail	:	"**A STUDY ON PREVALANCE OF PROSTATOMEGALY AND ITS CORRELATION WITH DURATION AND GLYCEMIC CONTROL IN PATIENTS WITH TYPE 2 DIABETES MELLITUS**"
Study Centre	:	Department of Diabetology,Institute of Internal Medicine, Rajiv Gandhi Government General Hospital, Chennai.
Patient's Name	:	
Patient's Age	:	
Identification Number	:	

Patient may check (☑) these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. •

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. •

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. •

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. •

I hereby consent to participate in this study. •

I hereby give permission to undergo complete clinical examination, and necessary investigations. •

Signature of Investigator Signature/thumb impression

Investigator's Name:

Patient's Name and Address:

Dr.S.PRABHAKARAN

சுய ஒப்புதல் படிவம்

ஆராய்ச்சி தலைப்பு :

அப்பாச்சி 2 மற்றும் சோபா அளவீடுகளை, குறுதி நஞ்சு - பல்லுறுப்பு செயல் பிறழ்ச்சி நோயின் குறிகாட்டிகளாக ஒப்பிட்டு ஆராய்தல்

பெயர்	வயது
பால்	தேதி
உள் நோயாளி எண்	ஆராய்ச்சி சேர்க்கை எண்

இந்த ஆராய்ச்சியின் விவரங்களும் நோக்கங்களும் எனக்கு முழுமையாகவும் தெளிவாகவும் விளக்கப்பட்டன. எனக்கு விளக்கப் பட்ட விஷயங்களை நான் புரிந்து கொண்டு எனது சம்மதத்தை தெரிவிக்கிறேன்.

மேற்கொண்ட பரிசோதனையின் பொழுது ஏற்படக்கூடிய பின் விளைவுகளை உணர்ந்து இந்த பரிசோதனைக்கு மனமாற சம்மதிக்கிறேன்.

இந்த ஆய்வுக்கான பரிசோதனைகளை செய்து கொள்ள சம்மதிக்கிறேன். இந்த ஆராய்ச்சியின் விளக்க தாளை பெற்றுக்கொண்டேன். இந்த ஆய்வில் முழு சுதந்திரத்துடன் மற்றும் சுய நினைவுடன் பங்கு கொள்ள சம்மதிக்கிறேன்.

பங்கேற்ப்பாளர் / பாதுகாவலர் கையொப்பம்

தேதி

MASTER CHART

S.NO.	AGE	H/O LUTS	HYPERTENSION	DYSLIPIDEMIA	DURATION OF DM	TREATMENT	REGULAR TT	PR	FBS/PPBS	7-8 HBAIC	8-10 HBA1c	SERUM PSA	PROSTATOMEGLY
1	41	YES			8 years	OHA			ABNORMAL	PRESENT		<4	YES
2	45	YES			8	INSULIN	IRREGULAR	YES	ABNORMAL	PRESENT		<4	YES
3	43	YES			12	INSULIN		YES	ABNORMAL	PRESENT		<4	YES
4	46	YES	YES	YES	10	INSULIN		YES	ABNORMAL	PRESENT		<4	YES
5	47	YES	YES		3	OHA	IRREGULAR		ABNORMAL			<4	YES
6	48				4	INSULIN	IRREGULAR		ABNORMAL		PRESENT	<4	YES
7	49		YES		10	INSULIN	IRREGULAR		ABNORMAL		PRESENT	<4	YES
8	43				12	OHA	IRREGULAR		ABNORMAL		PRESENT	<4	YES
9	44		YES	YES	4	INSULIN			ABNORMAL		PRESENT	<4	YES
10	46				9	INSULIN					PRESENT	<4	YES
11	48		YES		8	INSULIN					PRESENT	<4	YES
12	42				11	OHA						<4	YES
13	41				3	OHA			ABNORMAL				
14	42		YES		6	OHA			ABNORMAL		PRESENT		
15	47				1	OHA							
16	49		YES		4	OHA							
17	46			YES	3	OHA							
18	47				2	OHA							
19	45			YES	7	OHA							
20	44				11	OHA			ABNORMAL	PRESENT			
21	48		YES	YES	8	OHA			ABNORMAL	PRESENT			
22	48				5	OHA			ABNORMAL	PRESENT			
23	42				2	OHA			ABNORMAL	PRESENT			
24	41				4	OHA							
25	42				1	OHA							
26	45				5	OHA							
27	48		YES	YES	3	OHA							
28	45				2	OHA			ABNORMAL				
29	43				6	OHA			ABNORMAL	PRESENT			
30	41				1	OHA							
31	47		YES		3	OHA							
32	49		YES	YES	2	OHA							
33	48				3	OHA							
34	45				5	OHA							

35	44				3	OHA							
36	41		YES		2	OHA							
37	40				5	OHA							
38	47				13	OHA							
39	49				8	OHA							
40	52	YES			18	INSULIN			ABNORMAL	PRESENT		<4	YES
41	57	YES			13	INSULIN			ABNORMAL			<4	YES
42	58	YES	YES	YES	15	INSULIN			ABNORMAL		PRESENT	<4	YES
43	56	YES			11	INSULIN			ABNORMAL		PRESENT	<4	YES
44	51	YES			13	OHA	IRREGULAR		ABNORMAL		PRESENT	<4	YES
45	54	YES		YES	13	INSULIN	IRREGULAR		ABNORMAL		PRESENT	<4	YES
46	54	YES			12	INSULIN	IRREGULAR		ABNORMAL		PRESENT	<4	YES
47	57	YES			15	INSULIN	IRREGULAR	YES	ABNORMAL		PRESENT	<4	YES
48	57	YES	YES		16	INSULIN			ABNORMAL		PRESENT	<4	YES
49	59	YES			14	OHA			ABNORMAL		PRESENT	<4	YES
50	53	YES			15	INSULIN		YES	ABNORMAL		PRESENT	<4	YES
51	55	YES	YES		13	INSULIN	IRREGULAR		ABNORMAL		PRESENT	<4	YES
52	51	YES			14	OHA	IRREGULAR		ABNORMAL			<4	YES
53	52	YES		YES	12	OHA	IRREGULAR	YES	ABNORMAL		PRESENT	<4	YES
54	55	YES			10	INSULIN			ABNORMAL		PRESENT	<4	YES
55	54	YES	YES	YES	11	INSULIN	IRREGULAR	YES	ABNORMAL		PRESENT	<4	YES
56	57	YES			14	INSULIN	IRREGULAR	YES	ABNORMAL		PRESENT	<4	YES
57	56	YES	YES	YES	16	OHA		YES	ABNORMAL			<4	YES
58	56	YES			14	OHA			ABNORMAL	PRESENT		<4	YES
59	56	YES	YES		15	INSULIN		YES	ABNORMAL	PRESENT		<4	YES
60	55	YES			17	OHA		YES	ABNORMAL	PRESENT		<4	YES
61	54	YES	YES	YES	12	INSULIN		YES	ABNORMAL	PRESENT		<4	YES
62	54				14	INSULIN			ABNORMAL	PRESENT		<4	YES
63	52				16	INSULIN		YES	ABNORMAL	PRESENT		<4	YES
64	52		YES	YES	13	OHA			ABNORMAL	PRESENT		<4	YES
65	56				15	INSULIN						<4	YES
66	57				16	INSULIN						<4	YES
67	58		YES	YES	13	INSULIN				PRESENT		<4	YES
68	53				12	INSULIN				PRESENT		<4	YES
69	52				15	INSULIN						<4	YES

70	51				8	INSULIN						<4	YES
71	50				3	OHA						<4	YES
72	54		YES	YES	5	OHA						<4	YES
73	56		YES		2	OHA						<4	YES
74	55				1	OHA						<4	YES
75	56				5	OHA						<4	YES
76	56			YES	3	OHA						<4	YES
77	58				2	OHA						<4	YES
78	54				6	OHA						<4	YES
79	52		YES		2	OHA							
80	51			YES	11	OHA							
81	54				14	OHA			ABNORMAL		PRESENT		
82	53			YES	10	OHA			ABNORMAL	PRESENT			
83	57				15	OHA			ABNORMAL	PRESENT			
84	59				2	OHA			ABNORMAL				
85	56				5	OHA			ABNORMAL		PRESENT		
86	55			YES	1	OHA							
87	54				4	OHA							
88	55		YES		2	OHA							
89	53			YES	4	OHA							
90	53		YES		1	OHA			ABNORMAL				
91	52				3	OHA			ABNORMAL		PRESENT		
92	51		YES	YES	2	OHA			ABNORMAL				
93	56				4	OHA			ABNORMAL		PRESENT		
94	53				2	OHA			ABNORMAL		PRESENT		
95	55				1	OHA							
96	54				1	OHA							
97	52		YES		1	OHA							
98	53				1	OHA							
99	54				2	OHA							
100	57												