"ASSESSMENT OF ASSOCIATION BETWEEN COGNITIVE DEFICITS AND GLYCAEMIC CONTROL AND DURATION OF DIABETES IN ELDERLY DIABETIC PATIENTS ATTENDING TERTIARY CARE HOSPITAL, KOLAR, KARNATAKA"

By

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Dissertation Submitted To

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IN

GENERAL MEDICINE

Under the Guidance of

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DEPARTMENT OF GENERAL MEDICINE
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I hereby declare that this dissertation entitled **"ASSESSMENT OF ASSOCIATION BETWEEN COGNITIVE DEFICITS AND GLYCAEMIC CONTROL AND DURATION OF DIABETES IN ELDERLY DIABETIC PATIENTS ATTENDING TERTIARY CARE HOSPITAL, KOLAR, KARNATAKA"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. P. N. VENKATARATHNAMMA,** M.D. Professor, **DEPT. OF GENERAL MEDICINE** Sri Devaraj Urs Medical College, Kolar.

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Signature of the candidate

Dr. SPANDANA PEDDAREDDY

ABSTRACT

BACKGROUND: Diabetes Mellitus (DM) is a global epidemic in this millennium. It has been reported that the highest increase in the Diabetes Mellitus prevalence is amongst low and middle-income countries, predominantly within 40-59 years age group. According to the International Diabetes Federation (IDF) estimates, India had 62 million diabetic subjects in the year 2013 which is more than 7.1% of India's adult population. The prevalence of DM and impaired glucose tolerance increased with age. Older patients have more risk of hyperglycemia, which is related to the decrease in pancreatic function. Diabetes affects virtually every tissue in almost all the systems in human body and it's complications cause huge socioeconomic burden. Longstanding hyperglycemia is an independent risk for complications of Diabetes especially neurological manifestations. According to the recent estimates, around 24.3 million people have Dementia worldwide with an incidence of 4.6 million new cases every year. Among them around 60% live in the developing countries where it has been projected to increase by more than 300 percent by 2040. In India prevalence of Dementia was found to be 33.6 per thousand. Prevalence of Mild Cognitive Impairment is found to be 3% to 19% in adults older than 65 years. Conversion rate from Mild Cognitive Impairment to Alzheimer's Disease (AD) is 12% per year.

Hence managing diabetes effectively can modify the course of Alzheimer's disease. This will reduce the disability of the patients as well as the burden to the care-givers.

OBJECTIVES: 1. To identify cognitive impairment in elderly diabetic patients.

2. To determine relationship between duration of diabetes and diabetes control with cognitive impairment.

MATERIAL AND METHODS: An analysis of elderly diabetic patients who were admitted and who visited on outpatient basis to Department of General Medicine at R.L.Jalappa hospital and research centre attached to Sri Devaraj Urs medical college, Tamaka,Kolar-563101, was taken up for study between January 2015 to June 2016.

A structured and pretested proforma was used to collect data regarding history, complaints, past history, family history, treatment history from elderly diabetics who are

both inpatients and outpatients in the department of General Medicine at R.L.Jalappa hospital and research centre.

Written informed consent was taken.

Cognitive functions of the selected patients who have fulfilled the inclusion criteria and exclusion criteria was assessed by Mini mental state examination.

Investigations such as CBC, FBS, PPBS, HbA1c,serum electrolytes, liver function tests, renal function tests and thyroid function tests were done.

RESULTS: A total of 200 elderly diabetic patients satisfying the inclusion and exclusion criteria were included in the study. 121 patients (60.5%) were in the age group 60-65 years, followed by 42 patients (21%) were in the age group 66-70 years. In the study total males were 113 patients (56.5%) and females were 87 patients(43.5%). Among the patients 93 patients (46.5%) were illiterates, 117 patients (53.5%) were literates. Among the literates 43 patients (21.5%) had UPPER PRIMARY EDUCATION. In the study 19 patients(9.5%) had duration of diabetes for 20 years. 159 patients (79.5%) of patients were regularly undergoing treatment for diabetes. This signifies that even in patients were adherent to treatment, still cognitive impairment was seen. In our study 90 patients (45%) had mild cognitive impairment followed by 40 patients (20%) had severe cognitive impairment. In the study majority of patients had cognitive impairment. The study showed 71 patients (35.5 %) had FBS range of 161-200mg/dl. In the study 46 patients (23%) had PPBS range of 161-200mg/dl. The study showed that 31 patients (15.5%) had MMSE score of 23. It indicates that mild cognitive impairment was more common. The descriptive statistics done for the study showed following values:

Mean HbA1c 8.76%- which was significantly high

Mean MMSE score 22-indicating Mild Cognitive Impairment.

Mean duration of diabetes was 13.37 years.

The Chi square test showed the association between FBS and severe cognitive impairment was statistically significant. ($p<0.05$). Chi square test signifies that the association between FBS and MMSE was statistically significant. ($p<0.05$) Chi square test between PPBS and Severe Cognitive Impairment and MMSE Score was statistically significant. ($p<0.05$) In the study the association between HbA1c and Severe Cognitive

Impairment and MMSE Score was statistically significant.($p<0.05$) The study showed that FBS, PPBS and HbA1c had strong association with MMSE SCORE and Cognitive Impairment. Co-morbid conditions had some additive effect on the cognition. This study signifies that association between Diabetes and Cognition was merely significant not by chance.

CONCLUSION: It was an Observational study conducted in the tertiary care hospital. The objectives of the study were to identify cognitive impairment in elderly diabetic patients and to determine the relationship between duration of diabetes and diabetes control with cognitive impairment. Most of the patients belonged to 60-65 years of age. A mean duration of diabetes was found to be around 20 years from the study. Even though patients were adherent to treatment the cognitive impairment was present in the patients. Majority of patients had mild cognitive impairment. The study showed that FBS, PPBS and HbA1c had strong association with MMSE SCORE and Cognitive Impairment. Co-morbid conditions had some additive effect on the cognition. This study signifies that association between Diabetes and Cognitive Dysfunction.

KEY WORDS: Type 2 DM, MMSE, Cognitive Impairment.

LIST OF ABBREVIATIONS

DM	Diabetes Mellitus.
IDF	International Diabetes Federation
IDDM	Insulin-dependent Diabetes Mellitus
NIDDM	Non Insulin-dependent Diabetes Mellitus
T2DM	Type 2 Diabetes Mellitus
GDM	Gestational Diabetes Mellitus
MODY	Maturity Onset Diabetes in Youth
IR	Insulin Resistance
DKA	Diabetes Ketoacidosis
HHS	Hyperglycemic Hyperosmolar state
ADAM	Androgen Deficiency in aging men
FBS	Fasting Blood Sugar
PPBS	Post Prandial Blood Sugar
HbA1c	Glycated hemoglobin
FPG	Fasting Plasma Glucose
WHO	World Health Organization
RBCs	Red Blood Cells
EAG	Estimated Average Glucose
MPG	Mean Plasma Glucose
ADA	American Diabetic Association
MMSE	Mini Mental State Examination
SMMSE	Standardised Mini-Mental State Examination
ADL	Activity Of Daily Living

GDS	Global Deterioration Scale
AD	Alzheimer’s Disease
VD	Vascular Dementia
BBB	Blood Brain Barrier
CNS	Central Nervous System
AGEs	Advanced Glycation End Products
IDE	Insulin Degrading Enzyme
FC	Functional Connectivity
DMN	Default mode network
WM	Working Memory

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INTRODUCTION

Diabetes Mellitus (DM) is a global epidemic in this millennium. It has been reported that the highest increase in Diabetes Mellitus prevalence is amongst low and middle-income countries, predominantly within the 40-59 years age group.

Diabetes Mellitus is emerging as a major health-care challenge for India. According to the International Diabetes Federation (IDF) estimates, India had 62 million diabetic subjects in the year 2013 which is more than 7.1% of India's adult population. An estimate shows that nearly 1 million Indians die due to Diabetes Mellitus every year.

There is a greatest increase in prevalence expected to occur in Asia and Africa. The increase in incidence in developing countries follows the trend of urbanization and lifestyle changes, perhaps most importantly a" Western-style" diet.[1]

The prevalence of DM and impaired glucose tolerance increased with age. Older patients have more risk of hyperglycemia, which is related to the decrease in pancreatic function.

Diabetic patients require special care, and a multidisciplinary approach for the treatment and prevention of the complications.[2] Diabetes affects virtually every tissue in almost all the systems of the human body and its complications cause huge socioeconomic burden.

Type 2 Diabetes Mellitus is commonly associated with obesity, dyslipidaemia and constitutes an important component of the so-called metabolic syndrome. These co-morbidities increase the complications by several-folds.

Longstanding hyperglycemia is an independent risk for complications of diabetes especially neurological manifestations. According to the recent estimates, around 24.3 million people have Dementia worldwide with an incidence of 4.6 million new cases every year. Among them around 60% live in the developing countries where it has been projected to increase by more than 300 percent by 2040.

In India the prevalence of Dementia was found to be 33.6 per thousand. With increase in life expectancy in India the burden and care of people with Dementia would be a challenge.

Dementia is one of the leading causes of death and most important cause of disability above 50 years of age. It is a syndrome characterized by cognitive impairment and executive dysfunction.

Mild Cognitive Impairment is a clinical label which includes elderly subjects with short-term or long-term memory impairment and with no significant daily functional disability. The diagnosis of Mild Cognitive Impairment is made when a subject reports a gradual decline of cognitive functions for at least a six month period.

Prevalence of Mild Cognitive Impairment is found to be 3% to 19% in adults older than 65 years. Conversion rate from Mild Cognitive Impairment to Alzheimer's disease (AD) is 12% per year. Cognitive dysfunction in T2DM may start at a relatively young age; thus, starting the management at an early age may be important in terms of preventing not only dementia but also other complications.

Hence managing diabetes effectively can modify the course of Alzheimer's disease. This will reduce the disability of the patients as well as the burden to the care-givers.[3]

OBJECTIVES

1. To identify cognitive impairment in elderly diabetic patients.

2. To determine the relationship between duration of diabetes and diabetes control with cognitive impairment.

REVIEW OF LITERATURE

HISTORY OF DIABETES

A disease characterized by "too great emptying of urine" finds its place in Egyptian manuscripts dating back to 1500 B.C. Indian physicians called it "MADHUMEHA" honey urine because it attracted ants.[4] The ancient Indian physicians Sushrutha and surgeon Charaka were able to identify two types of diabetes in 400-500 A.D.[5,6] Arataeus and Cappadocian coined the word Diabetes (Greek-Siphon) and stated that " no essential part of the drink is absorbed by the body while great masses of the flesh are liquefied into urine".[6,7,8]

The term "mellitus" (Latin-sweet like honey) was coined by the british surgeon general , John Rollo in 1798, to distinguish this diabetes from other form of diabetes in which urine was tasteless.[9] In 1869, Paul Langerhans, identified the cells that came to be known as 'Islets of Langerhans'.[10]

In 1921, Banting, Best and Collip, working in Macleod's laboratory, ligated the pancreatic duct in animal experiments, causing destruction of the exocrine pancreas. In their animal experiments, by using canine insulin extracts to reverse induced diabetes, they established that the deficiency of insulin was the cause of diabetes.

They first administered this life saving bovine extract of insulin to a 14 year old boy, Leonardo Thompson, in 1922 at the Toronto General Hospital proving a sensation in diabetic therapy.[11] It won Banting and Macleod the Nobel Prize in Physiology and medicine in 1923.[11]

Willis a London physician, made a bold move and became the first person to taste the urine of the patients-because passing copious urine was the hall mark of the disease.[12]

The early remedies for diabetes included diverse and interesting prescriptions like "oil of roses, dates, raw quinces, gruel, sweet almonds, fresh flowers of blind nettles.[6]

Diet and exercise was the hallmark of treatment by 19th century physicians led by Joslin and Fitz from the Massachusettes General Hospital.[13] The first oral antidiabetic drugs were given as treatment in 1950s. In 1980, the first human insulin was manufactured by Graham Bell. In 1982, the first biosynthetic insulin (humulin) was developed.[14]

The refusal to patent insulin, but to share this miraculous therapy freely with the world will remain an outstanding example of unreserved generosity towards mankind in the history of medical diseases.

Since 2007, Banting's birthday 14th November is celebrated as "WORLD DIABETES DAY".

DIABETES MELLITUS

DEFINITION:

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

The chronic hyperglycemia of diabetes is associated with long term damage, dysfunction, and failure of different organs like the eyes, kidney, nerves, heart and blood vessels.

Several pathogenic processes are involved in the development of diabetes. These processes can range from autoimmune destruction of the beta cells of the pancreas with consequent insulin deficiency to abnormalities that result in resistance to insulin action.

The basis of the abnormalities in carbohydrate, fat, protein metabolism in diabetes is deficient action of insulin on target tissue.[15]

CLASSIFICATION

The old terms of insulin-dependent (IDDM) or non-insulin-dependent (NIDDM) which were proposed by WHO in1980 and 1985 have disappeared and the terms of new classification system identifies four types of diabetes mellitus: type 1, type 2, "other specific types" and gestational diabetes.[16]

Type 1 diabetes mellitus

Type 1 diabetes mellitus (juvenile diabetes) is characterized by beta cell destruction caused by an autoimmune process, usually leading to absolute insulin deficiency.[17]

Type 1 is usually characterized by the presence of anti–glutamic acid decarboxylase, islet cell or insulin antibodies which identify the autoimmune processes that lead to beta cell destruction. Eventually, all type1 diabetic patients will require insulin therapy to maintain normglycemia.

Type 2 diabetes mellitus(T2DM)

T2DM comprises 80% to 90% of all cases of DM. Most individuals with Type 2 diabetes exhibit intra-abdominal (visceral) obesity, which is closely related to the presence of insulin resistance. In addition, hypertension and dyslipidemia are often present in these individuals.

This is the most common form of diabetes mellitus and is highly associated with a family history of diabetes, older age, obesity and lack of exercise.

Gestational Diabetes Mellitus (GDM)

Gestational diabetes mellitus is an operational classification identifying women who develop diabetes mellitus during gestation. Women who develop Type 1 diabetes mellitus during pregnancy and women with undiagnosed asymptomatic Type 2 diabetes mellitus that is discovered during pregnancy are classified with Gestational Diabetes Mellitus (GDM). In most women who develop GDM; the disorder has its onset in the third trimester of pregnancy.

Other specific type (Monogenic diabetes)

Types of diabetes mellitus of various known etiologies are grouped together to form the classification called "Other Specific Types". This group includes persons with genetic defects of beta-cell function (this type of diabetes was formerly called MODY or maturity-onset diabetes in youth) or with defects of insulin action; persons with diseases of the exocrine pancreas, such as pancreatitis or cystic fibrosis; persons with dysfunction associated with other endocrinopathies (e.g. acromegaly); and persons with pancreatic dysfunction caused by drugs, chemicals or infections and they comprise less than 10% of DM cases.[18]

TABLE 1: Classification of Diabetes

I. type 1Diabetes mellitus A. Autoimmune B. Idiopathic **II. type 2 Diabetes mellitus** Ranges from relative insulin deficiency to disorders of insulin secretion and insulin resistance **III. Other specific types of diabetes mellitus** A. Genetic defects in β-cell function 1. Chromosome 12, HNF-1α (MODY 3) 2. Chromosome 7, glycosidase (MODY 2) 3. Chromosome 20, HNF-4α (MODY 1) 4. Mitochondrial DNA 5. Monogenic diabetes B. Genetic defects in insulin action 1. Type A insulin resistance 2. Leprechaunism 3. Rabson-Mendenhall syndrome 4. Lipotrophic diabetes C. Disease of the exocrine pancreas 1. Pancreatitis 2. Pancreatectomy/trauma 3. Neoplasia 4. Cystic fibrosis 5. Hemochromatosis 6. Fibrocalcific pancreatopathy D. Endocrinopathies 1. Acromegaly 2. Cushing syndrome 3. Glucagonoma 4. Pheochromocytoma 5. Hyperthyroidism 6. Somatostatinoma 7. Aldosteronoma i. Pharmacologically or chemically induced 1. Vacor 2. Pentamidine	3. Nicotinic acid 4. Glucocorticoids 5. Thyroid hormones 6. Diazoxide 7. β-adrenergic agonists 8. Tiazides 9. Dilantin 10. α interferon ii. Infections 1. Congenital rubeola 2. Cytomegalovirus iii. Infrequent forms of autoimmune diabetes 1. Stiff-man syndrome) 2. Antibodies against insulin receptors iv. Other syndromes occasionally associated with diabetes 1. Down syndrome 2. Klinefelter syndrome 3. Turner syndrome 4. Wolfram syndrome 5. Friedreich ataxia 6. Huntington's chorea 7. Lawrence-Moon-Biedel syndrome 8. Myotonic dystrophy 9. Porphyria 10. Prader-Willi syndrome **IV. Gestational diabetes mellitus** Occurs in mostly in women during gestation.

Table 2: Etiologic Classification of Diabetes Mellitus. Adapted from WHO and ADA [6,19].

Adapted from WHO and ADA

PATHOLOGY AND PATHOGENESIS

INSULIN SECRETION, FUNCTION AND MECHANISM OF ACTION

Insulin is a polypeptide hormone synthesized in humans and other mammals within the beta cells of the islets of Langerhans in the pancreas. The islets of Langerhans form the endocrine part of pancreas, accounting for 2% of the total mass of the pancreas, with beta cells constituting 60-80% of all the cells of islets of Langerhans.

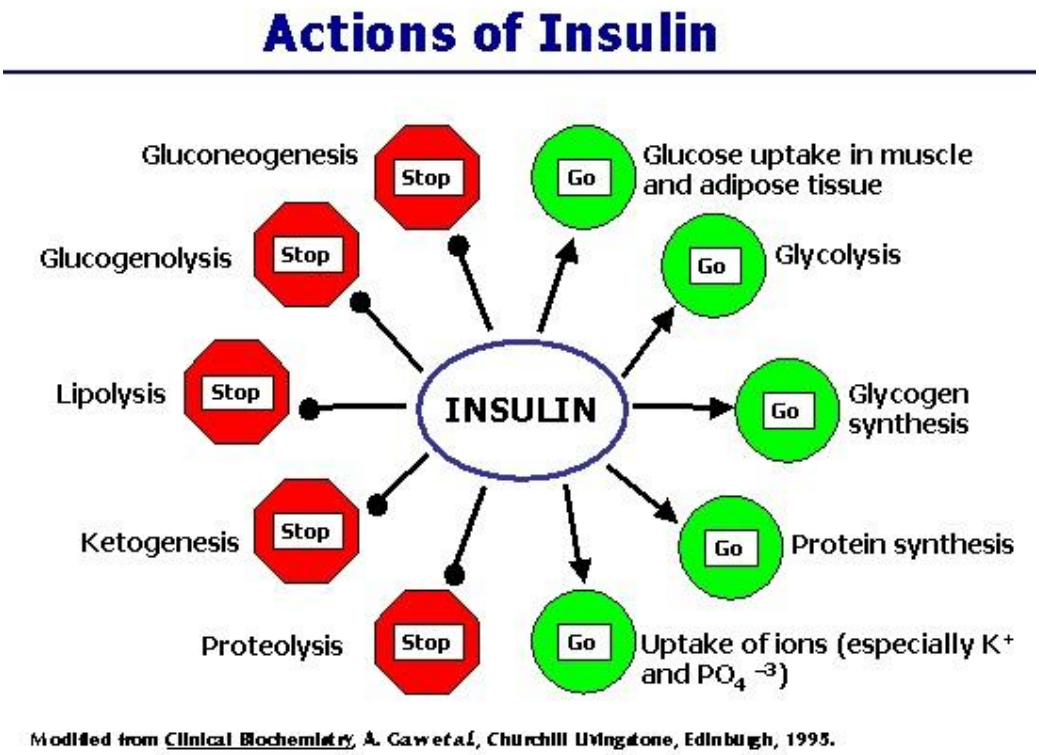


FIG 1: INSULIN AND ITS MECHANISM OF ACTION

Adapted from Caw A et al, clinical biochemistry 1995.

Insulin exhibits a multitude of effects in many tissues, with liver, muscle, and adipose tissue being the most important target organs for insulin action. The basic physiological function of insulin is promoting the synthesis of carbohydrates, proteins, lipids, and nucleic acids. [19]

Insulin is more of an anabolic hormone rather than catabolic. Insufficient amounts of insulin or poor cellular response to insulin as well as defective insulin leads to improper handling of glucose by body cells or appropriate glucose storage in the liver and muscles.

The effects of insulin on carbohydrate metabolism include stimulation of glucose transport across muscle and adipocyte cell membranes, regulation of hepatic glycogen synthesis, and inhibition of glycogenolysis and gluconeogenesis . The end result of these actions is a reduction in blood glucose concentration.[20]

With regard to protein metabolism, insulin promotes transfer of amino acids across membranes, stimulates protein synthesis, and inhibits proteolysis. Incorporation of fatty acids from circulating triglyceride into adipose triglyceride and lipid synthesis are stimulated by insulin; lipolysis is inhibited. Insulin contributes to nucleic acid synthesis by stimulating the formation of ATP, DNA, and RNA.[21]

MECHANISM OF INSULIN ACTION

Insulin initiates its physiological effects by binding to a high affinity specific receptor located on the plasma membrane. The binding capacity and the biological activity of insulin are maximal at a plasma insulin concentration of 20 to 30 μU/ml.

After binding to the receptor, insulin transmits its signal to the interior of the cell through a second messenger that influences enzymatic processes. Thus, the hormone carries out its actions without entering the cell.

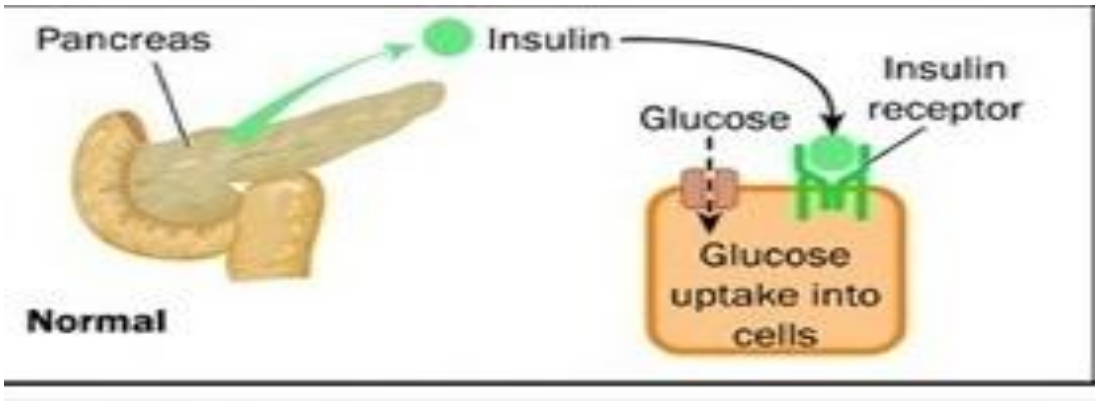


Fig 2: Insulin Uptake by cells

Adapted from Cahill GF Jr and Boston MD. Physiology of insulin in man. Diabetes 1971;12:785-799.

Two membrane-bound enzyme systems are associated with the insulin signal: the adenyl cyclase-cAMP and the Magnesium - activated Sodium – Potassium - ATPase systems.[22]

After an overnight fast, the 8.00 am normal plasma insulin concentration is 5 to 15 μU/ml. Postprandial values, 100g glucose, can be 5 to 10 times higher than the baseline. Insulin output under basal condition approximates 0.5 to 1.0 U/h and increases about 5 times after food ingestion.[23] The ability of insulin to mediate tissue glucose uptake is a critical step in maintaining glucose homeostasis and in clearing the postprandial glucose load.

The insulin production is directly proportional to the amount of sugar (carbohydrate) consumed. The more sugar one consumes, the more insulin the body will have to produce, but, the tiny pancreatic beta cells were never designed to produce this level of insulin.

With a limited capacity to produce insulin, the forced over-production of insulin will eventually exhaust that capacity and the cells will cease to operate.[24]

Insulin is a major hormone that enables cells to uptake glucose from the bloodstream. Furthermore, insulin is also the major regulatory signal for glycogenesis in the hepatocytes and myocytes.

BIOCHEMICAL BACKGROUND OF DIABETES MELLITUS

Under normal physiological conditions, plasma glucose concentrations are maintained within a narrow range, despite wide fluctuations in supply and demand, through a tightly regulated and dynamic interaction between tissue sensitivity to insulin (especially in liver) and insulin secretion.[25]

In type 2 diabetes these mechanisms break down and the two main pathological defects in type 2 diabetes are impaired insulin secretion through a dysfunction of the pancreatic β-cell, and impaired insulin action through insulin resistance(IR).[26]

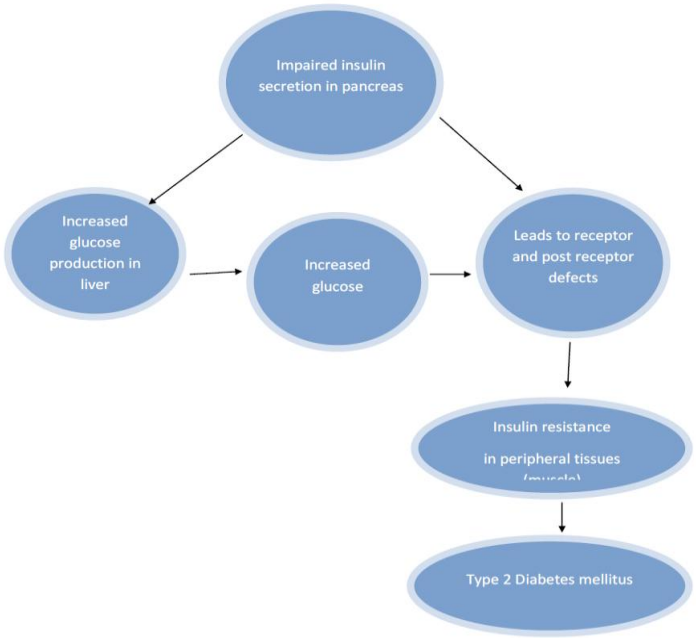


FIG 3: MECHANISM OF INSULIN RESISTANCE AND TYPE 2 DM

Adapted from Defronzo RA, Ferrannini E. Lily Lecture1987.

The Triumvirate: Beta Cell, Muscle, Liver. A Collusion Responsible for NIDDM. Diabetes 1988;37:667-687

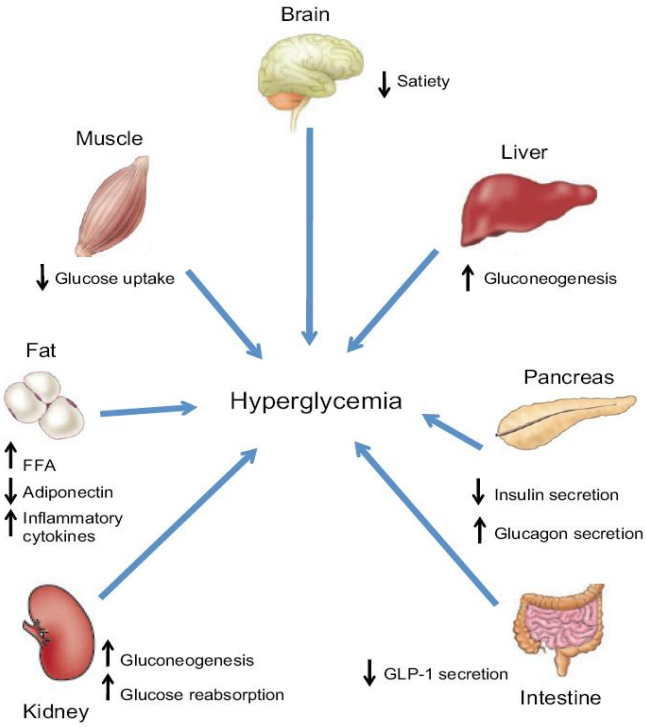


Figure 1 Multiorgan and tissue pathophysiology of type 2 diabetes.
Notes: Adapted with permission from DeFronzo RA. Banting Lecture. From the triumvirate to the ominous octet: a new paradigm for the treatment of type 2 diabetes mellitus. *Diabetes.* 2009;58:773–795.[4]
Abbreviations: FFA, free fatty acids; GLP-1, glucagon-like peptide-1.

Fig 4: Changes in various organs leading to Hyperglycemia.

Adapted from DeFronzo RA.Banting lecture.From the triumvirate to the ominous octet:a new paradigm for the treatment of type 2 Diabetes mellitus.Diabetes 2009;58:773-779.

The body attempts to arrest hyperglycemia, by drawing water out of the cells and into the bloodstream. The excess sugar is excreted in the urine. This is why diabetics present with constant thirst, drinking large amounts of water, and polyuria as the cells try to get rid of the extra glucose. This subsequently leads to glucosuria.

As hyperglycemia prolongs, the body cells are devoid of glucose due to the lack of insulin. This forces the cells to seek alternative energy sources.

The cells turn to fatty acids stored in adipose tissue. The fats are not fuel sources for the red blood cells, kidney cortex and the brain. The red blood cells lack

mitochondria in which beta-oxidation pathway rests. The fatty acids cannot pass the blood-brain barrier.

To avail energy to such cells and tissues, the acetyl-CoA arising from catabolism of fatty acids is diverted to ketogenesis to generate ketone bodies, which can serve as alternative fuel sources for such cells and tissues. These ketone bodies are also passed in the urine, thereby leading to ketonuria, which characterizes diabetes mellitus.[27]

Build up of ketone bodies in the blood produces ketosis. Ketone bodies are acidic in nature and therefore, their build up in blood lowers blood pH, leading to acidosis. A combination of ketosis and acidosis lead to a condition called ketoacidosis.

If left untreated, ketoacidosis leads to condition called ketoacidosis. If left untreated, ketoacidosis leads to coma and death.

RISK FACTORS OF T2DM

- Aging
- Genetic factors
- Stress related factors
- Over eating- especially excessive intake of simple sugars
- Obesity- mild obesity causes 4 to 5 fold increase in developing diabetes.
- Alcohol drinking
- Smoking
- Lack of exercise.[28]

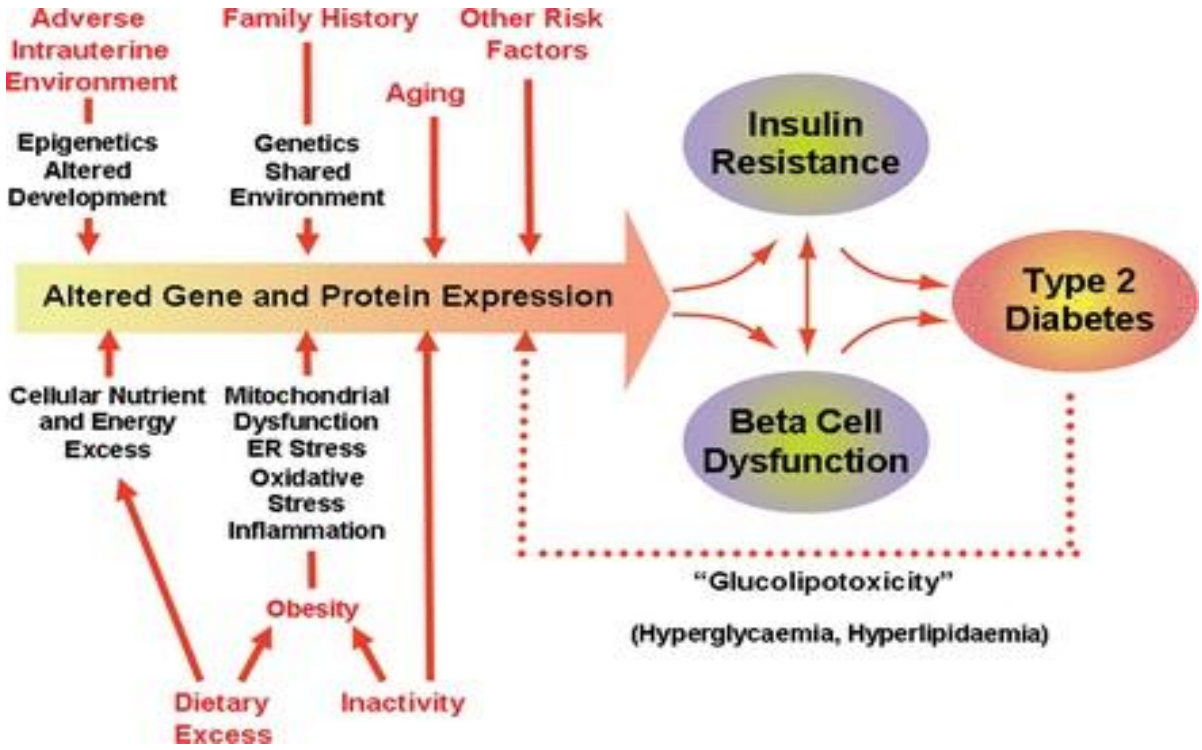


Fig 5: Risk factors for T2DM

Adapted from Belinda R.Gale Encyclopaedia of Alternative Medicine.2004: 2603-2605.

COMPLICATIONS OF DIABETES:

1. **ACUTE COMPLICATIONS**

- Hypoglycemia.
- Hyperglycemic crisis

  -Diabetes ketoacidosis(DKA)

  -Hyperglycemic hyperosmolar state (HSS)

2. **CHRONIC COMPLICATIONS**

- Microvascular:
- Diabetic retinopathy
- Diabetic nephropathy
- Diabetic neuropathy
- Macrovascular diseases.
- Other complications and associated conditions:
- Hypothyroidism
- Hyperthyroidism
- Celiac disease
- Vitiligo
- Addison’s disease
- Lipodystrophy
- Necrobiosis lipoidica diabeticorum
- Non-alcoholic fatty liver disease
- Infections
- Limited joint mobility
- Edema

DIABETES IN OLDER INDIVIDUALS

Hyperglycemia is associated with a deterioration in cognitive function in older people.[29] Depression occurs more commonly in people with diabetes than in non-diabetic people, particularly in those patients with complications. In people with diabetes, it was found that the presence of depression was strongly associated with hospitalization and mortality.

Depression needs to be treated aggressively in older people with diabetes to improve compliance and reduce the risk of suicide.[30] Pressure ulcers occur more commonly in people with diabetes and, although the reasons for this are unclear, poor tissue blood flow may be a contributory factor.

In addition, diabetic patients tend to loose zinc in the urine as a result of elevated glucose levels. Loss of zinc leads to zinc deficiency, which is associated with poor wound healing.

Alterations in blood flow to the microvascular structures of the feet, as well as changes in autonomic nervous system function, are the major causative factors in the pathogenesis of foot ulcers, and eventually infection and amputation.[31,32]

Diabetic patients experience pain more frequently than people with other chronic conditions and older diabetic patients have a decreased pain threshold despite having early signs of peripheral neuropathy.[33]

Results from experimental studies suggest that the lowering of the pain threshold may be caused by glucose blocking the capacity of beta-endorphin to combine with its receptor.[34]

Older people with elevated glucose levels are at increased risk for developing atypical infections. In particular, they have an increased risk of developing tuberculosis, partly as a result of memory T-cell defects that occur with ageing.[35]

The risk of developing dehydration is also increased in older people as a result of m-opioid thirst drive failure. Thus, when older diabetic people develop an osmotic diuresis, they often do not recognize that they are thirsty and become dehydrated and develop hyperosmolar coma.[36]

Diabetes produces a decrease in testosterone levels in men that is secondary to a failure of the hypothalamic-pituitary axis. Low testosterone levels are often seen in older diabetic people, which accelerate the onset of the androgen deficiency in aging men (ADAM) syndrome.[37]

Increased osmolality in the vitreous humour, increased cataract formation and increased propensity to develop glaucoma and retinopathy all interact to increase the visual acuity problems seen in the older diabetic person.

Older people have declining glomerular filtration rates, which can be accelerated by diabetes, resulting in premature renal failure.[38]

Table 2: Aspects of Diabetes Mellitus in the elderly

Syndrome	Preventive measures
Cognitive impairment	Control hyperglycaemia Provide written instructions
Depression/suicide	Screen using Geriatric Depression Scale Treat depression
Pressure ulcers	Control hyperglycaemia Consider zinc deficiency
Amputations	Pay careful attention to foot care
Decreased pain threshold	Control hyperglycaemia
Functional impairment	Control hyperglycaemia
Falls	Balance exercises Monitor orthostatic blood pressure
Incontinence	Control hyperglycaemia
Dehydration	Drink fluids regularly Control hyperglycaemia
Tuberculosis	Control hyperglycaemia
Hypogonadism	Control hyperglycaemia

Adapted from Morley JE, Charlton E, Patrick P, et al. Validation of the androgen deficiency in aging males (ADAM) screening questionnaire. Endo Soc 1998.

DIAGNOSIS

FBS, PPBS & HbA1c

Diabetes mellitus is a chronic illness that requires continuing medical care, patient education, and support to prevent acute complications and to reduce the risk of long-term complications. Control of blood glucose in patients with diabetes can be assessed by several methods.

These include assessment of glycated hemoglobin (HBA1c), fasting blood sugar (FBS), and postprandial blood sugar (PPBS). The gold standard for assessment of glycaemic control at follow up is the glycated haemoglobin level.[39]

Many randomized, prospective clinical trials in type 1 and 2 diabetes have clearly shown that achieving glycemic control or reducing hyperglycemia significantly decrease the microvascular complications of diabetes.

Each 1% reduction in haemoglobin A1c was associated with a 37% decrease in risk for microvascular complications and a 21% decrease in the risk of any end point or death related to diabetes.[40,41]

The ADA has recognized the fasting plasma glucose (FPG), as the diagnostic test of choice. PPBS values can change due to many variables, such as physical activity, insulin sensitivity, gastric emptying rate, and meal composition etc.[42]

HbA1c is not recommended as a diagnostic or a screening test because it is considered that HbA1c is inferior to FPG or post-load glucose values at predicting type 2 diabetes because the existence of hemoglobin or red cell abnormalities that can increase the variability ofHbA1c values. This variability may contribute to its inferior prediction of diabetes compared with fasting or post-load glucose values.

FPG and HbA1c may reflect different aspects glucose metabolism. While HbA1c can reflect a variety of factors in glucose metabolism, FPG levels mainly depend on insulin resistance and hepatic glucose production.[43]

As per the World Health Organization (WHO) people with fasting glucose levels from 6.1 to 6.9 mmol/l (110 to 125 mg/dl) are considered to have impaired fasting glucose.

People with plasma glucose at or above 7.8 mmol/l (140 mg/dl), but not over 11.1 mmol/l (200 mg/dl), two hours after a 75 g oral glucose load are considered to have impaired glucose tolerance.

Acceptable control level of blood glucose were defined as FBS value equal or less than 120 mg/dL and PPBS value equal or less than 160 mg/dL.

Isolated postprandial hyperglycemia was defined as FBS equal or less than 120 mg/dL with PPBS equal or more than 160 mg/dL.[44]

Glycated (Glycosylated) Hemoglobin is a form of hemoglobin used primarily to identify the average plasma glucose concentration over a prolonged period of time. Increased levels of glycated hemoglobin have been associated with cardiovascular disease, nephropathy, and retinopathy in diabetes mellitus.

During the normal 120-day life span of the red blood cell (RBC), glucose molecules react with hemoglobin, forming glycated hemoglobin. Once a hemoglobin molecule is glycated, it remains in this form. In people without diabetes, about 4% to 6% of their hemoglobin is glycosylated. RBCs that contain the hemoglobin circulate in the bloodstream for three to four months before being broken down and replaced.

During that time, the RBC can bond, irreversibly, to glucose in the bloodstream. A buildup of glycated hemoglobin within the red blood cell therefore reflects the average level of glucose to which the cell has been exposed during its life cycle. Thus, A1C readings higher than about 6% indicate higher than normal amounts of glucose roaming the blood stream in the past 120 days.[45]

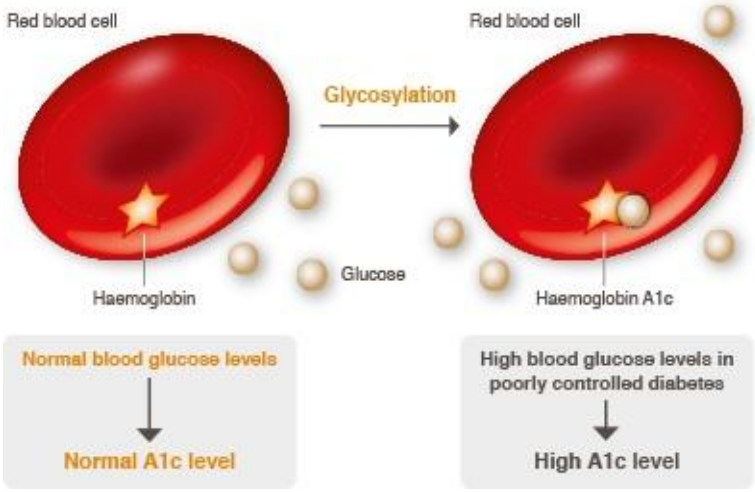


Fig 6: formation of Glycosylated Hemoglobin

Adapted from Balatbat J.Glycated (Glycosylated) Hemoglobin: HbA1c New Directions to diagnose diabetes. 2010.

A 1 percent change in an A1C result reflects a change of about 30mg/dL (1.67 mmol/L) in average blood glucose. Glycated hemoglobin values reflect the 2-3 month average endogenous exposure to glucose including postprandial spikes in the blood glucose level, and have low intraindividual variability, particularly in persons without diabetes.

Table 3: HbA1c values and estimated average glucose levels

HbA1c	Estimated Average Glucose (EAG) Mean Plasma Glucose (MPG)	
%	mg/dL	Mmol/L
5	97 (76-120)	5.4
6	126 (100-152)	7.0
7	154 (123-185)	8.6
8	183 (147-217)	10.2
9	212 (170-249)	11.8
10	240 (193-282)	13.4
11	269 (217-314)	14.9
12	298 (240-347)	16.5

Adapted: American Diabetes Association Standards of Medical Care in Diabetes- 2009, *Diabetes Care;* 32: Suppl 1:S13-S61.14

Adapted from American Diabetes Association Standards of Medical Care in Diabetes. Diabetes Care 2009;32:13-61.

Other advantages of A1C include the fact that it is a better indication of overall glycemic exposure over time and that there is substantially less day-to-day variability. The expert panel has suggested that the A1C assay may be used to diagnose diabetes, recommending 6.5% as the diagnostic threshold, and the ADA has now accepted the suggestion.[46,47]

People of African, Mediterranean or South east Asian descent, or people with family members with sickle cell anemia or thalassemia have less common type of hemoglobin, known as hemoglobin variant, and can give false HbA1c levels. Falsely low A1c results can occur in people with anemia, heavy bleeding. Falsely high A1c results can be seen in iron deficiency anemia. Other causes of abnormal A1c results include kidney failure, liver disease.[48]

STANDARDISED MINI-MENTAL STATE EXAMINATION(SMMSE)

Dr. Marshall Folstein first developed the Mini- Mental State Examination (MMSE) in 1975. Since then it has become widely used as a screening test for cognitive impairment and it is routinely used as an inclusion / exclusion criterion and outcome measure in clinical trials.

The test covers a variety of cognitive domains, including orientation to time and place, short and long term memory, registration, recall, constructional ability, language and the ability to understand and follow commands.

This test should never be used alone. It is used in conjunction with a corroborative history. The test usually takes about ten minutes to complete and can be used reliably after a short training period by physicians, nurses and other health care professionals.

MMSE has overall sensitivity of 64% and specificity of 96%.[3] The original MMSE had few instructions for administration and scoring. Different raters developed their own unique styles and techniques of administration and scoring. This led to wide differences and lowered the reliability of the test.[49]

DIRECTIONS FOR ADMINISTRATION OF SMMSE:

Assess the person's ability to hear and understand very simple conversation, e.g. what is your name? If the person uses hearing or visual aids, provide these before starting.

Before you begin, get the person's permission to ask questions, e.g. would it be all right to ask you some questions about your memory? This helps to avoid catastrophic reactions.

Ask each question a maximum of three times. If the person does not respond, score zero. If the person answers incorrectly, score zero.

Following equipment is required to administer the instrument: A watch, a pencil, reverse of the SMMSE score sheet with CLOSE YOUR EYES written in large letters and two five-sided figures intersecting to make a four-sided figure, and a space for the person to write down a sentence. Repeat the same directions a maximum of three times.

1.SCORING WORLD BACKWARDS:

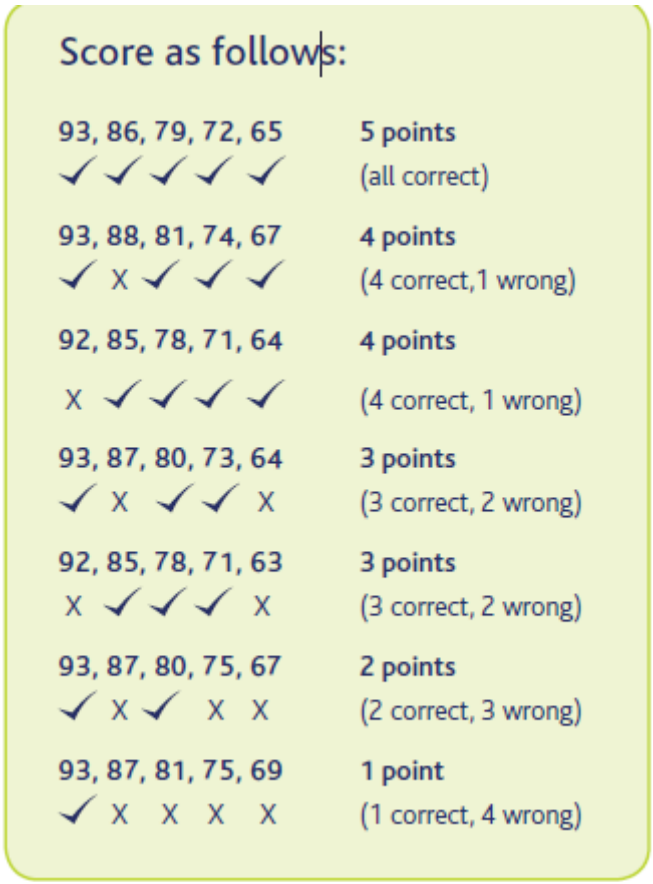
This task accounts for 17% of the total score. It is essential to score it reliably. There are many different ways and "systems" for scoring WORLD backwards. Originally it was advised that the score is "the number of letters in the correct order." i.e score ORDER not SEQUENCE.

The serial sevens task is presented as an alternative to spelling ‘world’ backwards. The two tasks are not equivalent.

The serial sevens is an easier task, and the scoring is easier. It can be used as an alternate to spelling world backwards in people who are illiterate.

Once person starts – do not interrupt – allow him/her to proceed until five subtractions have been made. If person stops before five subtractions have been made, repeat the original instruction to keep subtracting seven (maximum three times).

Table 4: Scoring for serial seven subtraction



Adapted from Molloy et al. Standardized Mini-Mental State Examination, A User’s Guide.1991.

2.SCORING THE OVERLAPPING PENTAGONS

Give the person the pencil, with the eraser, and a clean piece of paper. Many older adults draw shaky, wiggly lines with unclear angles that are more curved than straight. These are acceptable, as long as the person has two five-sided figures intersecting to form a four-sided figure.

People who have physical, non-cognitive disabilities should not score lower just because they are physically unable to perform certain tasks.

If the test cannot be modified, then omit the task. If an item has been omitted because of physical disability, it is important to take this into account when scoring the test. The score from this task is subtracted from the total score (30) to give a new total.

EXAMPLES OF DISABILITIES THAT CAN EXEMPT PEOPLE FROM CERTAIN TASKS

1. PHYSICAL DISABILITIES

The disability should be permanent. Some physical problems may take months to resolve and it may not be practical to wait. In these cases, carefully document the situation and proceed.

Examples of physical disabilities include: amputation, chronic deformity from arthritis, paralysis of limbs, blindness/poor vision even with glasses, permanent hearing loss even with functioning hearing aid.

2. LANGUAGE

Sometimes language difficulties impair a person's ability to perform certain tasks on the SMMSE. If English is not the person's first language, try to score the person in his or her first language.

If the person seems to understand some questions easily and others not, this is likely due to cognitive impairment.

If the person has consistent problems understanding the questions, it is likely due to language difficulties and the score can be adjusted accordingly.

If in doubt, get a translator or give the test in his or her native language. Hearing impairment should not be missed.

3. SPEECH:

Some people have severe speech problems. It is important to be consistent and adhere to the rules of administration, observing the time limits and scoring guidelines. Note should be made of these factors and performance in non-cognitive tests, like activity of daily living (ADL) function, should be assessed.

4. EDUCATION:

The person's disability should be clearly noted on the SMMSE score sheet. Items that are affected by this disability should also be clearly noted.

The calculation of the adjusted score should be done at the bottom of the SMMSE score sheet.[50]

Serial 100-7s subtraction has the highest correlation coefficient to the Global Deterioration Scale (GDS) score in the educated group.

Serial 40-4s subtraction shows the highest correlation to the GDS score in the uneducated group.

Therefore, 'serial 100-7s subtraction' should be considered for replacement with 'serial 40-4s subtraction' when MMSE assessment is made for the uneducated group.[51]

Table 5: SMMSE scores, stages of disease and area of impairment in alzheimer's disease

SMMSE Scores	30 - 25	24 - 21	21 - 10	9 - 0
Stage	**May be Normal**	**Mild/Early**	**Moderate**	**Severe**
ADL		problems with driving, finances, shopping	assistance with dressing, grooming, toileting	problems with eating, walking
Communication		word-finding, repeating, goes off topic, loses track	sentence fragments, "empty" speech, vague terms (i.e. this, that)	speech disturbances (i.e. slurring, stuttering)
Memory	personive problems with names or misplacing objects	three item recall orientation (time then place)	WORLD spelling, language and three step command	all areas show obvious deficits
Years		2-4 years	2-3 years	2-3 years

Adapted from Molloy et al. Standardized Mini-Mental State Examination, A User's Guide.1991.

Education is not the only important factor for all of the tasks contained in the MMSE.[52] A study which was able to retrieve data from survivors of the Scottish Mental Survey in 1932 found no association between features of the sentence writing component of the MMSE and current cognitive ability, functional ability or depression score.[53]

Nonverbal and visual-constructive abilities are learnt and applied not only according to innate abilities but also as a response to the needs of the individual to adapt to the environment. Some of the skills which are necessary in order to cope with

the everyday challenges in a city are continuously practiced without any formal education.

This may be one of the possible explanations why certain MMSE items simply do not show any difference depending on the level of educational achievement.[54]

MECHANISM OF COGNITIVE DYSFUNCTION IN DIABETES MELLITUS

With the progressive aging of the population, an increasing number of patients suffer from dementia. Dementia is a disease condition that precludes the activities of daily life and self-care behaviors, and it constitutes a great burden on the patients themselves, their careers, and society.[55]

It was established that T2DM is associated with cognitive dysfunction. Studies have indicated that older people with T2DM have a higher risk of cognitive dysfunction or dementia.[57]

While a wide range of cognitive domains was reportedly impaired in older patients with T2DM, one of the most frequently reported cognitive functional deficiencies in T2DM is impaired psychomotor speed.[56]

Ample evidence has indicated that T2DM is related not only to vascular dementia (VD) but also to the clinical diagnosis of Alzheimer's disease (AD)-type dementia.[58]

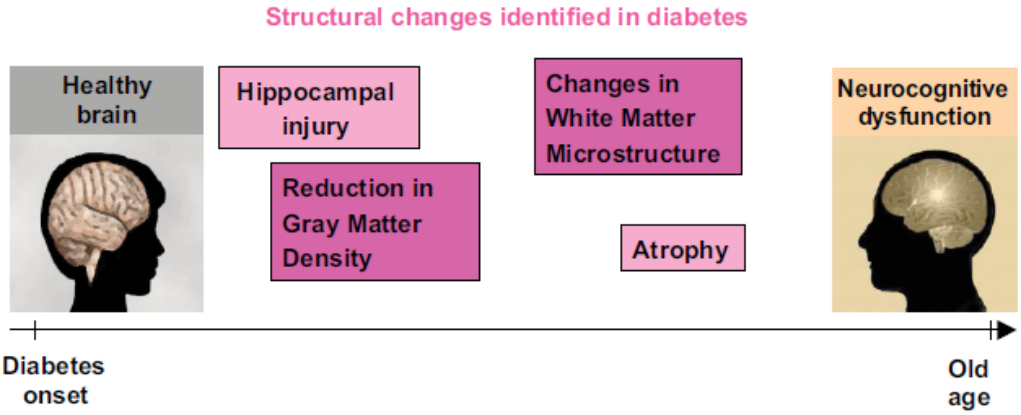


Fig 7: Structural changes seen in brain in diabetes mellitus

Adapted from Li L, Hölscher C. Common pathological processes in Alzheimer disease and type 2 diabetes: a review. Brain Res Rev. 2007;56:384–402

1.NEUROGENESIS

Neurogenesis in the hippocampus plays a role in learning and memory, and age-associated decline in neurogenesis has been reported.[59] The impaired neurogenesis in T2DM subjects may underlie an associated cognitive impairment and brain atrophy.[60, 61]

2.BLOOD-BRAIN BARRIER

The blood–brain barrier (BBB) consists of tight junctions between endothelial cells and astrocytic projections, which regulate paracellular and transcellular flow into the central nervous system (CNS).

Previous observations in brain tissue biopsied from AD subjects have indicated BBB breakdown in several respects.[62,63]

These include thinning of the endothelium, loss of mitochondria, and thickening of the basement membranes, the latter of which increase the accumulation of focal Aβ peptides. A break in the BBB also leads to potentially toxic substances

and metabolites gaining access to the brain. Diabetes is associated with changes in both the barrier and transport functions of the cerebral microvessels.Dysfunction in the BBB may be associated with cognitive impairment and/or the incidence of dementia.[64]

3.HYPERGLYCAEMIA

High glucose concentration, a major pathological characteristic of diabetes, may have toxic effects on neurons in the brain through several mechanisms.

Osmotic insults and oxidative stress may be involved in the mechanism, and the presence of chronic high glucose leads to the enhanced formation of advanced glycation end products (AGEs) through increased polyol pathway which have potentially toxic effects on neurons.[65]

AD patients with T2DM have increased levels of AGEs and microglial activation in the CNS compared to AD patients without T2DM. AGEs couple with free radicals and create oxidative damage, which in turn leads to neuronal injury.[66]

In addition to their direct toxicity, AGEs also activate microglia in the CNS. It is seen that microglia, the resident innate immune cells in the brain, can become deleterious and damage neurons. This process is implicated as an underlying mechanism in diverse neurodegenerative diseases, including AD.

While microglial function is beneficial and mandatory for normal CNS functioning, unregulated overactivation of microglia causes damage to neurons.[67]

In diabetes, oxidative stress also increases because of reduced antioxidant capacity. Oxidative stress has been suggested to lead to neuronal injury through mitochondrial dysfunction.[68]

4. INFLAMMATORY MECHANISM

It has been suggested that inflammation is associated with the pathogenesis of AD. Chronic low-grade inflammation may be a contributor to the disease process of AD. Proinflammatory cytokines such as tumor necrosis factor alpha are known to be involved in the pathogenesis of both T2DM and AD.

The activation of glia by inflammatory cytokines damages the neurons. Therefore, inflammation can be linked between T2DM and dementia, especially AD.[69]

5.INSULIN RESISTANCE(IR)

Insulin working within the brain is presumably of pancreatic origin, and it has passed the BBB through a saturable transporter mechanism from the systemic circulation.[70]

Insulin has multiple important functions in the brain including the control of food intake (via insulin receptors located in the olfactory bulb and thalamus) and effects on cognitive functions, including memory.

The critical pathological mechanism in AD is an accumulation of Aβ. Overproduction of Aβ may be one of the mechanisms by which this accumulation occurs; however, the impairment of clearance of Aβ also play a role.

The desensitization of insulin receptors, ie, IR, reduces the synthesis of several proteins, including Insulin-degrading Enzyme (IDE).IDE degrades Aβ as well as insulin, and reduced amounts of IDE may result in greater amyloid deposition. Research has indicated that insulin controls the expression of IDE in the brain, and IR in the brain may down regulate IDE expression.[71]

Another critical pathology in AD is the hyperphosphorylation of tau. Insulin seems to be an important determinant of tau protein phosphorylation. Impaired insulin signaling can result in the inhibition of PI3K/Akt and the activation of glycogen synthase kinase-3β.

The activation of glycogen synthase kinase-3β leads to the enhanced phosphorylation of tau protein and the formation of neurofibrillary tangles.[72]

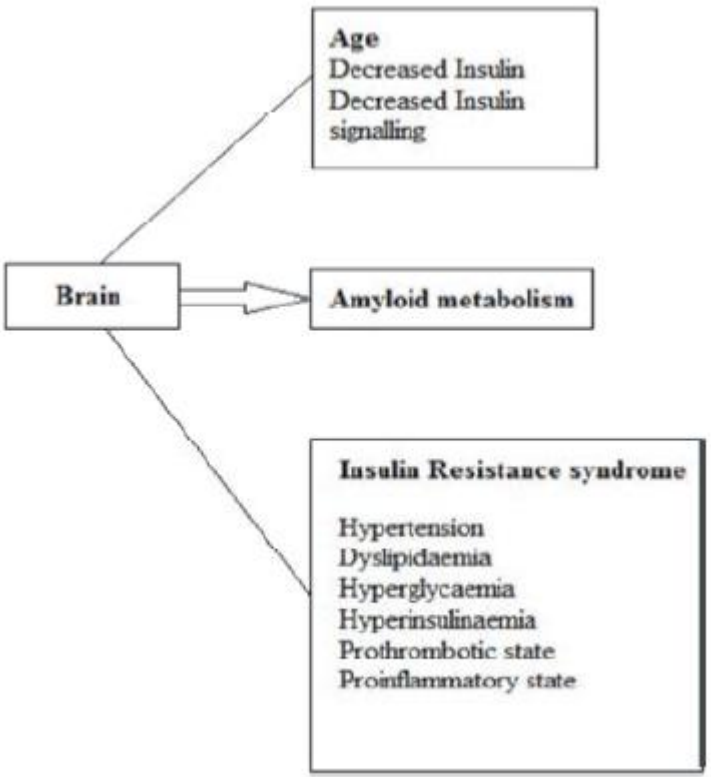


Fig 8: Role of insulin in pathogenesis of cognitive impairment.

Adapted from Umegaki H. Pathophysiology of cognitive dysfunction in older people with type 2 diabetes: vascular changes or neurodegeneration? Age Ageing. 2010;39:8–10.

6. VASCULAR DYSFUNCTION

Microalbuminuria, a marker of vascular dysfunction, predict the accelerated cognitive decline in T2DM subjects.[73]

These findings suggest that a deficit of vascular endothelial cells can lead to impairment of the functional coordination of the vascular supply in a timely response to the demand created by nervous activity.

Neural activity requires a strong increase of cerebral blood flow and an acute increase in neuronal glucose.

Dysfunction of cerebral auto regulation with increasing age along with structural and functional alterations in cerebral blood vessels due to diabetes mellitus impairs the functioning of neurovascular units.

These phenomena may induce functional deficits in neurons and increase neuronal degeneration and the susceptibility to hypoxia and ischemia.

Impaired neurovascular units would also induce BBB leakage.[74]

Recently, the hypothesis that vascular dysfunction may impair the drainage pathways of Aβ from the brain parenchyma and thus increase the deposition of Aβ has been proposed.

Vascular dysfunction could be associated with the progression of amyloid pathology.[75]

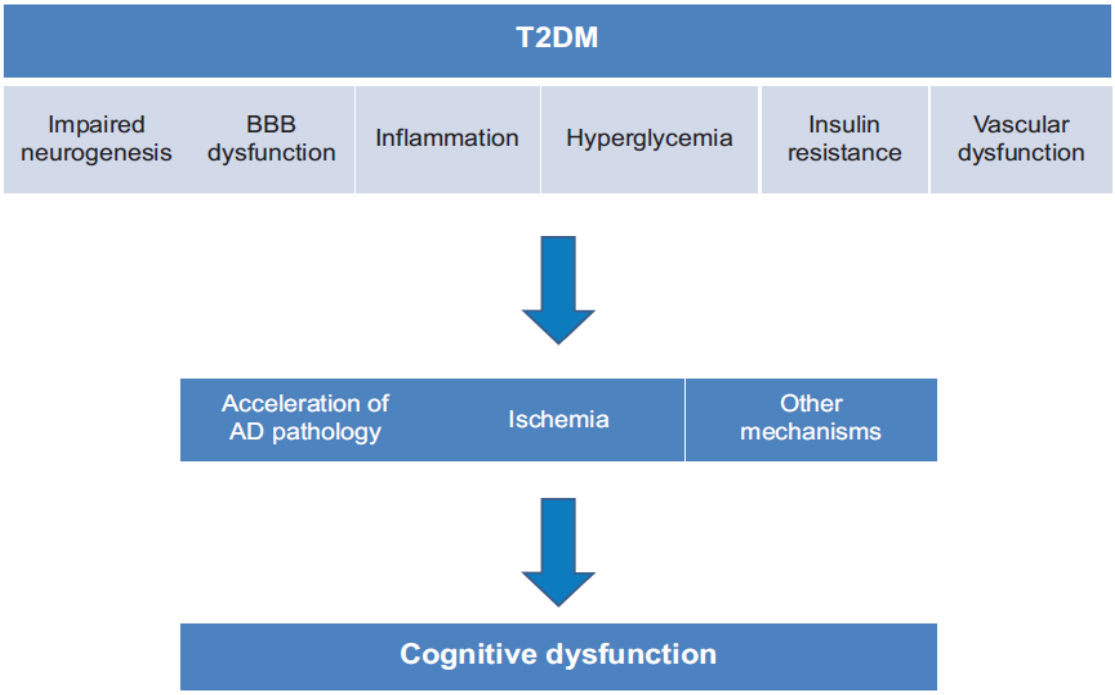


Fig 9: Mechanism of T2dm associated cognitive dysfunction

Adapted from Umegaki H. Pathophysiology of cognitive dysfunction in older people with type 2 diabetes: vascular changes or neurodegeneration? Age Ageing. 2010;39:8–10.

T2DM is an established risk factor for microvascular and macrovascular complications throughout the body, including brain stroke and small vessel disease.[76]

Therefore, vascular damage is likely to be one of the main reasons for the cognitive impairment in T2DM subjects, including VD subjects.

Studies have reported T2DM to be associated with microstructural abnormalities in the white matter in various pathways in the brain independent of small vessel diseases, and these abnormalities are also associated with cognitive impairment.[77,78]

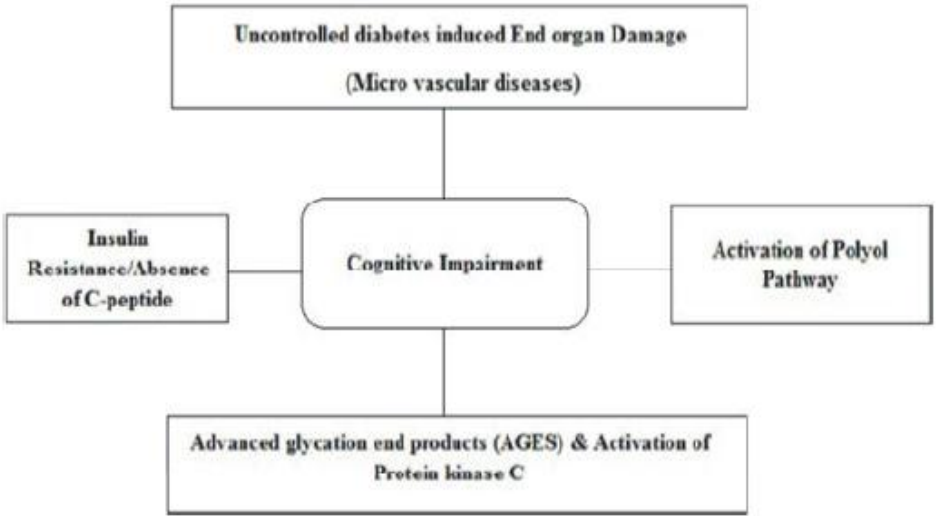


Fig 10: Mechanism of cognitive dysfunction in T2DM

Adapted from Umegaki H. Pathophysiology of cognitive dysfunction in older people with type 2 diabetes: vascular changes or neurodegeneration? Age Ageing. 2010;39:8–10.

7. RECURRENT EPISODES OF HYPOGLYCEMIA

Disruption of the supply of exogenous glucose causes functional disturbances.[79] Repetitive episodes of moderate to severe hypoglycemia have been implicated as possible etiology of cognitive dysfunction in diabetes.

During acute hypoglycemia episodes, it has been shown that performance on immediate verbal memory, immediate visual memory, working memory, delayed memory, visual motor skills, visual-spatial skills and global cognitive dysfunction are all impaired.[80,81]

T2DM patients have grey matter atrophy in the hippocampus, amygdala and prefrontal and parietal cortices which might contribute to cognitive impairment.[82] It was found that the resting-state functional connectivity (FC) in the default mode network (DMN) was reduced in T2DM patients.[83]

Within the impaired cognitive domains, memory and executive functions are often involved at an early stage of T2DM. Working memory (WM) is an important fundamental cognitive process for the temporary storage and manipulation of information in the brain, and it is crucially important for most higher-order cognitive functions. WM impairments have been detected in T2DM patients.[84,85]

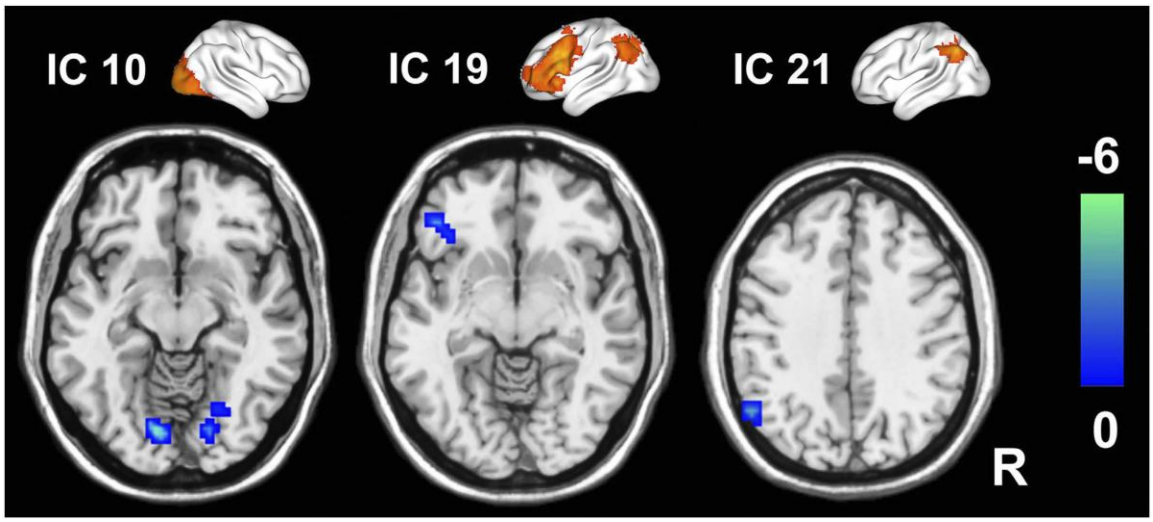


Fig 11: Brain regions showing decreased working memory (WM) in T2DM patients. Adapted from Musen, G. et al. Resting-state brain functional connectivity is altered in type 2 diabetes. Diabetes 2012;61:2375–2379.

MATERIALS AND METHODS

An analysis of elderly diabetic patients who are admitted and who visit on outpatient basis to Department of General Medicine at R.L.Jalappa hospital and research centre attached to Sri Devaraj Urs medical college, Tamaka,Kolar-563101, were taken up for study between January 2015 to June 2016.

1.Inclusion Criteria

- Patients more than 60 years of age
- Type 2 Diabetes Mellitus

2.Exclusion Criteria

- Dementia
- Type 1 Diabetes Mellitus
- Past or current history of CVA
- Epilepsy
- Depression
- Hypothyroidism
- Vitamin B12 deficiency
- Alzheimer's disease
- Chronic liver disease
- Chronic neuroinfections
- Chronic kidney disease
- Alcoholics

A structured and pretested proforma was used to collect data regarding history, complaints, past history, family history, treatment history from elderly diabetics who are both inpatients and outpatients in the department of General Medicine at R.L.Jalappa hospital and research centre. Written informed consent was taken. Cognitive functions of the selected patients who fulfilled the inclusion criteria and exclusion criteria were assessed by Mini mental state examination.

CBC ANALYSIS:

5ml blood was collected in the EDTA tube and was analyzed using SYSMAX CBC analyzer.

SERUM ELECTROLYTES,RENAL FUNCTION TESTS AND THYROID FUNCTION TESTS:

5ml blood was collected in plain red tube and was analyzed using VITROS 5.1. analyzer.

FBS AND PPBS:

5ml blood was collected in Sodium Fluoride tube and was analyzed using VITROS 5.1. analyzer.

HbA1c:

5ml blood was collected in EDTA tube and was analyzed using BIO-RAD D-10 analyzer.

Over night fasting samples were sent for estimating FBS and Thyroid profile estimation.

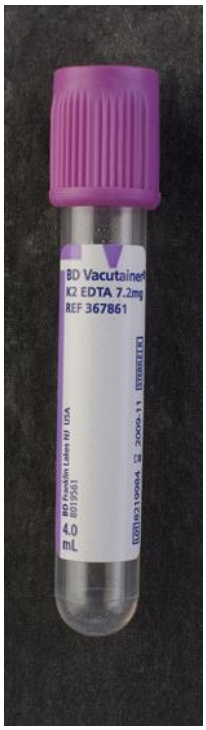


Fig. 12: EDTA Tube

Fig. 13: Sysmax CBC Analyzer

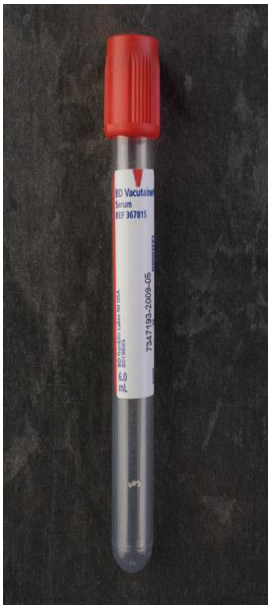


Fig. 14: Plain Red Tube

Fig. 15: VITROS 5.1

Fig 16: Sodium Fluoride Tube

Fig 17: Bio-Rad D-10

RESULTS AND OBSERVATIONS

A total of 200 elderly diabetic patients satisfying the inclusion and exclusion criteria were taken up for the study.

TABLE 6: AGE DISTRIBUTION

AGE RANGE

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	60-65 YEARS	121	60.5	60.5	60.5
	66-70 YEARS	42	21.0	21.0	81.5
	71-75 YEARS	19	9.5	9.5	91.0
	76-80 YEARS	14	7.0	7.0	98.0
	81-85 YEARS	2	1.0	1.0	99.0
	86-90 YEARS	2	1.0	1.0	100.0
	Total	200	100.0	100.0	

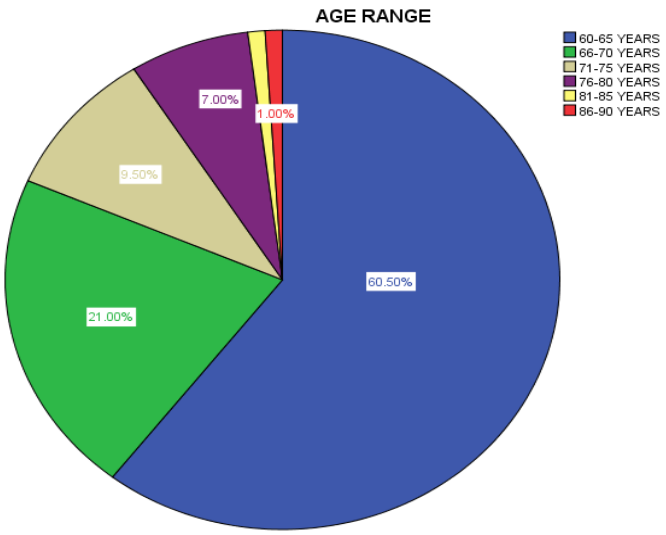


Chart 1: Age Distribution

- 121 patients (60.5%) are in the age group 60-65 years.
- 42 patients (21%) are in the age group 66-70 years.
- 19 patients (9.5%) are in the age group 71-75 years.

Table 7: Sex Distribution

SEX

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	FEMALE	87	43.5	43.5	43.5
	MALE	113	56.5	56.5	100.0
	Total	200	100.0	100.0	

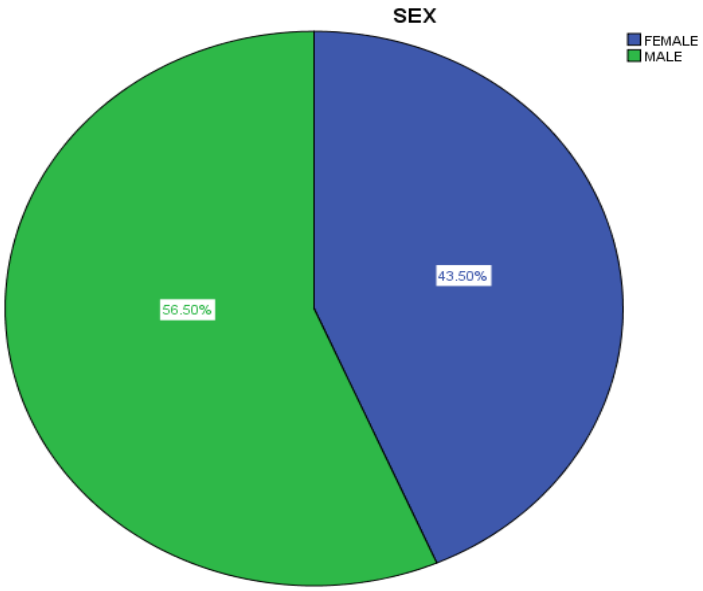


Chart 2: Sex Distribution

- Out of the 200 patients males are 113 (56.5%) and females are 87 (43.5%).

Table 8: Education Status

EDUCATION

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Degree	1	.5	.5	.5
	Graduate	11	5.5	5.5	6.0
	Higher secondary	8	4.0	4.0	10.0
	Lower secondary	30	15.0	15.0	25.0
	Primary	14	7.0	7.0	32.0
	Uneducated	93	46.5	46.5	78.5
	Upper primary	43	21.5	21.5	100.0
	Total	200	100.0	100.0	

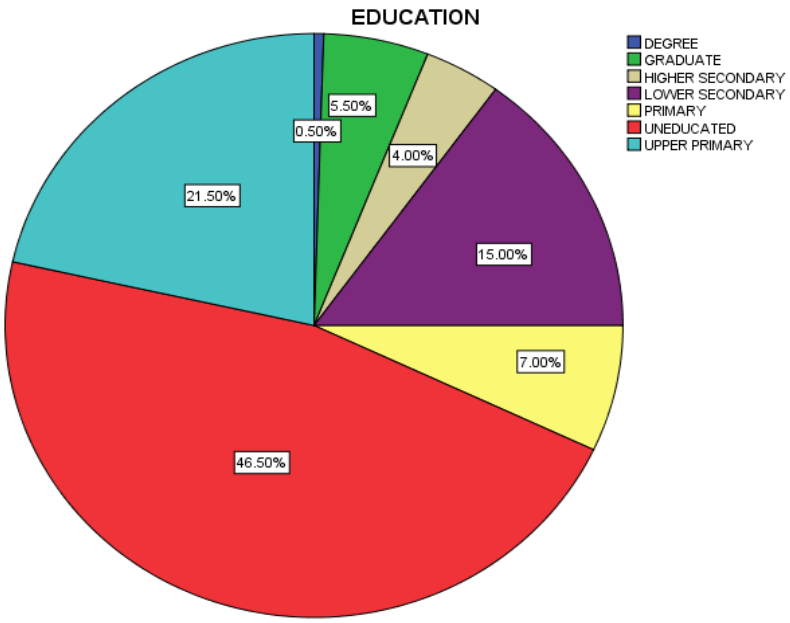


Chart 3: Education Status

- 93 patients (46.5%) patients are illiterates,117 (53.5%) patients are literates.
- In literate patients 43 patients (21.5%) had UPPER PRIMARY EDUCATION.

Table 9: Duration of Diabetes

DURATIONOFDIABETES

	Frequency	Percent	Valid Percent	Cumulative Percent
0	1	.5	.5	.5
0	1	.5	.5	1.0
0	1	.5	.5	1.5
0	3	1.5	1.5	3.0
1	2	1.0	1.0	4.0
1	1	.5	.5	4.5
1	1	.5	.5	5.0
1	5	2.5	2.5	7.5
2	4	2.0	2.0	9.5
2	5	2.5	2.5	12.0
3	1	.5	.5	12.5
3	5	2.5	2.5	15.0
4	1	.5	.5	15.5
4	10	5.0	5.0	20.5
5	10	5.0	5.0	25.5
6	6	3.0	3.0	28.5
7	7	3.5	3.5	32.0
8	10	5.0	5.0	37.0
9	4	2.0	2.0	39.0
10	18	9.0	9.0	48.0
11	3	1.5	1.5	49.5
12	7	3.5	3.5	53.0
13	3	1.5	1.5	54.5
14	5	2.5	2.5	57.0
15	11	5.5	5.5	62.5
16	7	3.5	3.5	66.0

17	4	2.0	2.0	68.0
18	10	5.0	5.0	73.0
19	1	.5	.5	73.5
20	19	9.5	9.5	83.0
22	5	2.5	2.5	85.5
23	1	.5	.5	86.0
25	3	1.5	1.5	87.5
26	4	2.0	2.0	89.5
28	2	1.0	1.0	90.5
29	1	.5	.5	91.0
30	5	2.5	2.5	93.5
32	2	1.0	1.0	94.5
35	8	4.0	4.0	98.5
38	2	1.0	1.0	99.5
40	1	.5	.5	100.0
Total	200	100.0	100.0	

- 19 patients(9.5%) have duration of diabetes for 20 years. This is the highest frequency.

Table 10: Adherence to Diabetic Treatment

DIABETICTREATMENTTAKEN

	Frequency	Percent	Valid Percent	Cumulative Percent
IRREGULAR	41	20.5	20.5	20.5
REGULAR	159	79.5	79.5	100.0
Total	200	100.0	100.0	

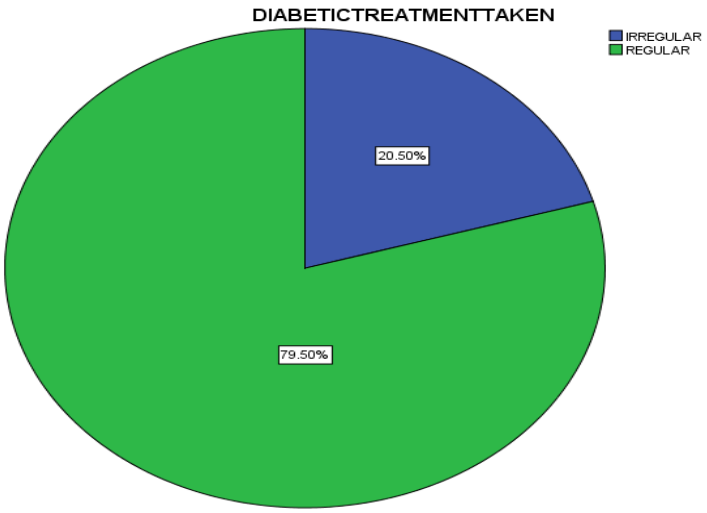


Chart 4: Adherence to Diabetic Treatment

- 159 patients (79.5%) are regularly undergoing treatment for diabetes.
- 41 patients (20.5%) are not regularly undergoing treatment for diabetes.

Table 11: Range Of FBS (mg/dl)

FBS RANGE

	Frequency	Percent	Valid Percent	Cumulative Percent
81-120mg/dl	16	8.0	8.0	8.0
121-160mg/dl	47	23.5	23.5	31.5
161-200mg/dl	71	35.5	35.5	67.0
201-240mg/dl	31	15.5	15.5	82.5
241-280mg/dl	16	8.0	8.0	90.5
281-320mg/dl	12	6.0	6.0	96.5
321-360mg/dl	3	1.5	1.5	98.0
361-400mg/dl	3	1.5	1.5	99.5
521-560mg/dl	1	.5	.5	100.0
Total	200	100.0	100.0	

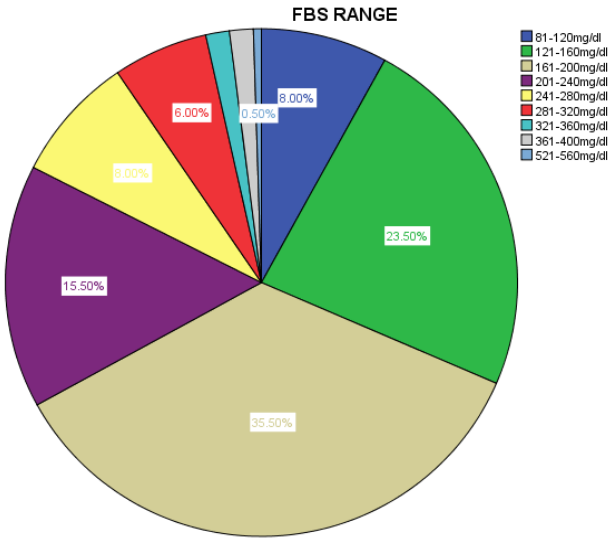


Chart 5: Range of FBS (mg/dl)

- 71 patients (35.5 %) in the study have FBS range 161-200mg/dl.

Table 12: Range of PPBS (mg/dl)

PPBS RANGE

	Frequency	Percent	Valid Percent	Cumulative Percent
81-120mg/dl	2	1.0	1.0	1.0
121-160mg/dl	14	7.0	7.0	8.0
161-200mg/dl	46	23.0	23.0	31.0
201-240mg/dl	37	18.5	18.5	49.5
241-280mg/dl	32	16.0	16.0	65.5
281-320mg/dl	36	18.0	18.0	83.5
321-360mg/dl	18	9.0	9.0	92.5
361-400mg/dl	5	2.5	2.5	95.0
401-440mg/dl	5	2.5	2.5	97.5
441-480mg/dl	2	1.0	1.0	98.5
521-560mg/dl	2	1.0	1.0	99.5
601-640mg/dl	1	.5	.5	100.0
Total	200	100.0	100.0	

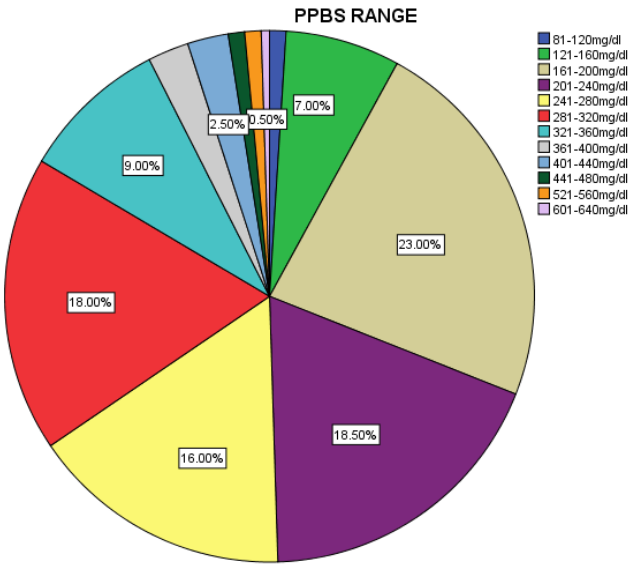


Chart 6: Range of PPBS (mg/dl)

- 46 patients (23%) in the study have PPBS range 161-200mg/dl.

Table 13: Patients with no Cognitive Impairment

NO COGNITIVE IMPAIREMENT

	Frequency	Percent	Valid Percent	Cumulative Percent
N/A	130	65.0	65.0	65.0
NORMAL	70	35.0	35.0	100.0
Total	200	100.0	100.0	

Table 14: Patients with mild cognitive Impairment

MILD COGNITIVE IMPAIRMENT

	Frequency	Percent	Valid Percent	Cumulative Percent
ABSENT	110	55.0	55.0	55.0
PRESENT	90	45.0	45.0	100.0
Total	200	100.0	100.0	

Table 15: Patients with severe cognitive impairment

SEVERE COGNITIVE IMPAIRMENT

	Frequency	Percent	Valid Percent	Cumulative Percent
ABSENT	160	80.0	80.0	80.0
PRESENT	40	20.0	20.0	100.0
Total	200	100.0	100.0	

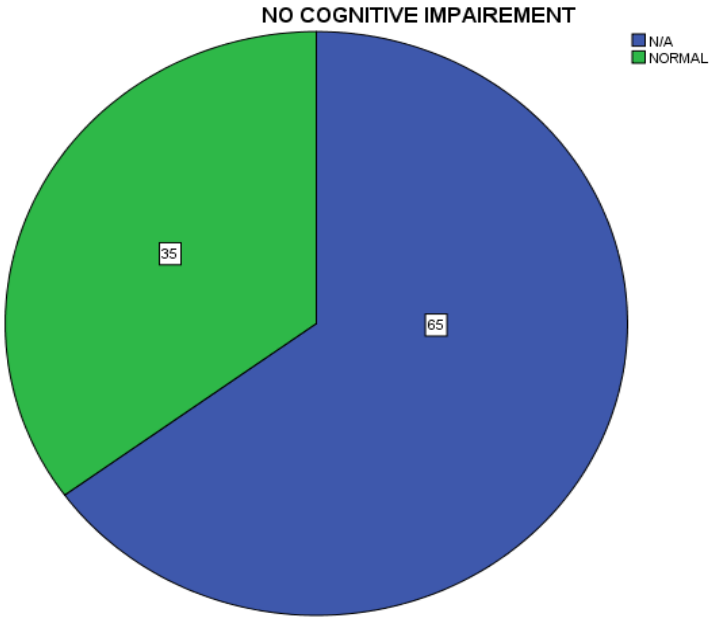


Chart 7: Patients with no cognitive impairment

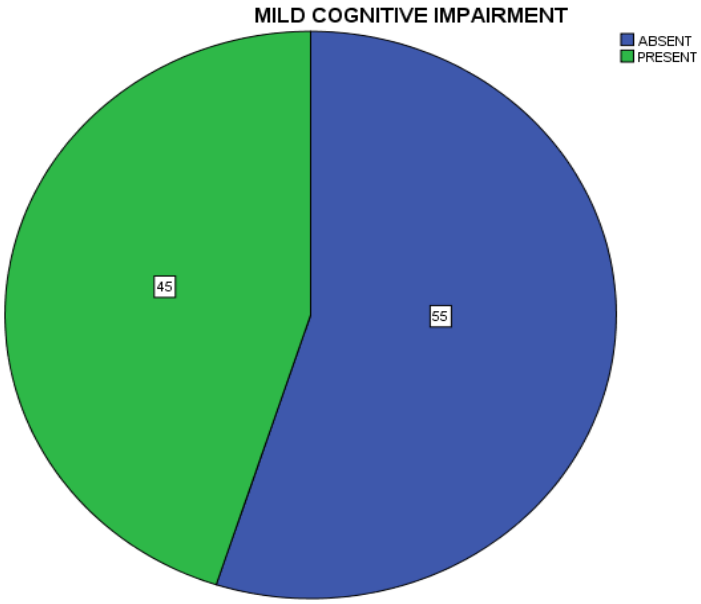


Chart 8: Patients with mild cognitive impairment

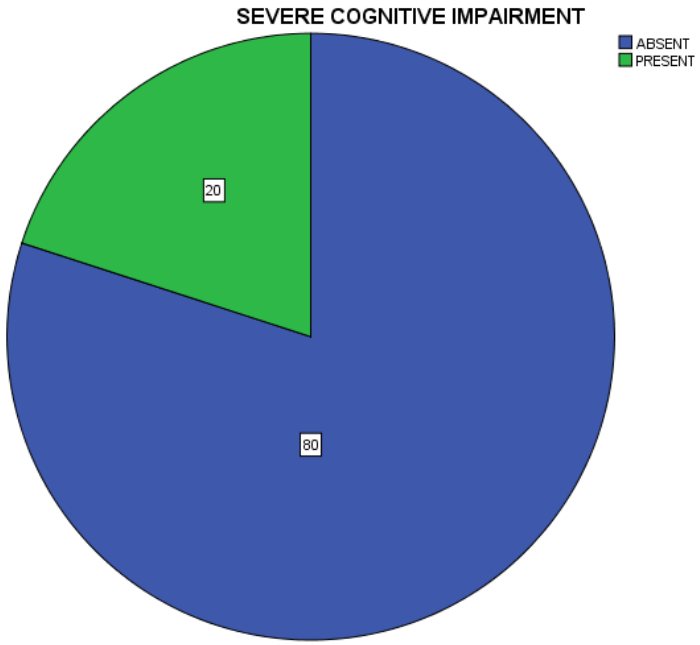


Chart 9: Patients with severe cognitive impairment

- Out of 200 patients in the study
- 70 patients(35%) have normal cognition
- 90 patients(45%) have mild cognitive impairment
- 40 patients(20%) have severe cognitive impairment.
- Around 65% of the patients in the study have Cognitive Impairment and majority had Mild Cognitive Impairment.

Table 16: MMSE Scores

MMSESCORE

	Frequency	Percent	Valid Percent	Cumulative Percent
12	1	.5	.5	.5
14	7	3.5	3.5	4.0
15	4	2.0	2.0	6.0
16	16	8.0	8.0	14.0
17	11	5.5	5.5	19.5
18	5	2.5	2.5	22.0
19	13	6.5	6.5	28.5
20	8	4.0	4.0	32.5
21	20	10.0	10.0	42.5
22	14	7.0	7.0	49.5
23	31	15.5	15.5	65.0
24	6	3.0	3.0	68.0
25	2	1.0	1.0	69.0
26	20	10.0	10.0	79.0
27	4	2.0	2.0	81.0
28	17	8.5	8.5	89.5
29	2	1.0	1.0	90.5
30	19	9.5	9.5	100.0
Total	200	100.0	100.0	

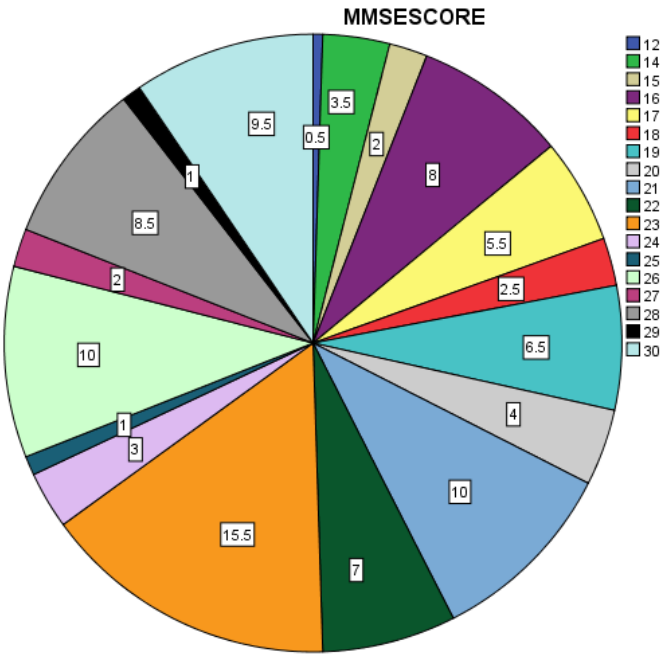


Chart 10 : MMSE Scores

- 31 patients (15.5%) have MMSE score of 23.
- It indicates that mild cognitive impairment was more common.

Table 17: Descriptive Statistics

Descriptive Statistics

	N	Minimum	Maximum	Mean		Std. Deviation
	Statistic	Statistic	Statistic	Statistic	Std. Error	Statistic
HbA1C	200	5.00%	14.90%	8.7620%	0.14925%	2.11069%
MMSE Score	200	12	30	22.39	.326	4.612
Age Range	200	1	6	1.70	.076	1.070
Duration of diabetes	200	0	40	13.37	.679	9.602
N	200					

- The descriptive statistics done for the study showed following values:
- Mean HbA1c 8.76%- which is significantly high
- Mean MMSE score 22.39-indicating Mild Cognitive Impairment
- Mean duration of diabetes 13.37 years.

Table 18a: FBS and severe cognitive impairment

Crosstab

		Severe cognitive impairment		Total
		Absent	Present	
FBS RANGE	81-120mg/dl	13	3	16
	121-160mg/dl	43	4	47
	161-200mg/dl	62	9	71
	201-240mg/dl	25	6	31
	241-280mg/dl	8	8	16
	281-320mg/dl	7	5	12
	321-360mg/dl	1	2	3
	361-400mg/dl	1	2	3
	521-560mg/dl	0	1	1
Total		160	40	200

Table 18b: FBS and severe cognitive impairment

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	30.969[a]	8	.000
Likelihood Ratio	26.791	8	.001
Linear-by-Linear Association	22.678	1	.000
N of Valid Cases	200		

9 cells (50.0%) have expected count less than 5. The minimum expected count is .20.

Chi square test signifies the association between FBS and severe cognitive impairment as statistically significant. ($p<0.05$).

Table 18c : FBS and severe cognitive impairment

Symmetric Measures

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Interval by Interval	Pearson's R	.338	.076	5.046	.000[c]
Ordinal by Ordinal	Spearman Correlation	.284	.074	4.161	.000[c]
N of Valid Cases		200			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

c. Based on normal approximation.

Pearson and spearman correlation signifies the association between FBS and severe cognitive impairment as statistically significant. ($p<0.05$).

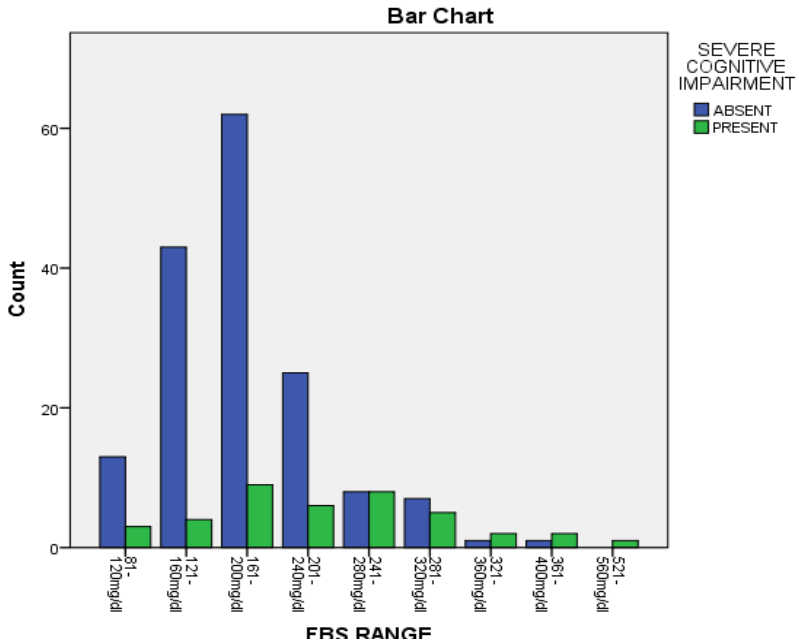


Chart 11: FBS and severe cognitive impairment

Table 19a: FBS and MMSE score

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	55.764[a]	24	.000
Likelihood Ratio	60.249	24	.000
Linear-by-Linear Association	27.915	1	.000
N of Valid Cases	200		

a. 25 cells (69.4%) have expected count less than 5. The minimum expected count is .06.

Chi square test signifies that the association between FBS and MMSE is statistically significant. ($p<0.05$)

Table 19b: FBS and MMSE score

Symmetric Measures

			Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Interval by Interval		Pearson's R	-.375	.058	-5.684	.000[c]
Ordinal by Ordinal		Spearman Correlation	-.365	.066	-5.511	.000[c]
N of Valid Cases			200			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

c. Based on normal approximation.

Pearson and spearman correlation statistically signify the association between FBS and MMSE ($p<0.05$)

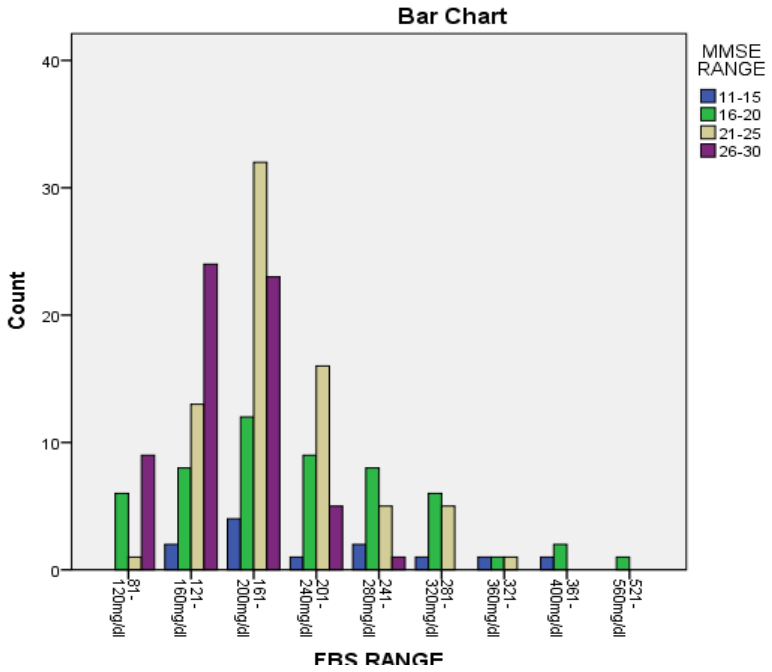


Chart 12: FBS and MMSE score

INFERENCE :

High FBS values were associated with cognitive impairment.

Table 20a: PPBS and MMSE score

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	67.437[a]	33	.000
Likelihood Ratio	67.478	33	.000
Linear-by-Linear Association	31.843	1	.000
N of Valid Cases	200		

a. 33 cells (68.8%) have expected count less than 5. The minimum expected count is .06.

Chi square test between PPBS and MMSE Score shows statistical significance. ($p<0.05$)

Table 20b: PPBS and MMSE score

Symmetric Measures

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Interval by Interval	Pearson's R	-.400	.058	-6.142	.000[c]
Ordinal by Ordinal	Spearman Correlation	-.420	.061	-6.504	.000[c]
N of Valid Cases		200			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

c. Based on normal approximation.

Pearson and spearman correlation shows statistical significance between PPBS and MMSE ($p<0.05$)

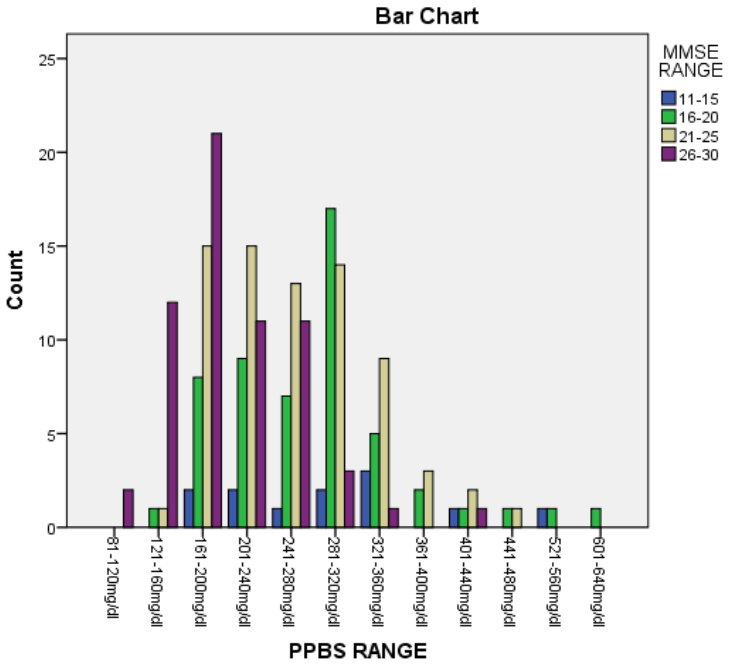


Chart 13: PPBS and MMSE score

Table 21a: PPBS and mild cognitive impairment

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	20.044[a]	11	.045
Likelihood Ratio	24.508	11	.011
Linear-by-Linear Association	5.758	1	.016
N of Valid Cases	200		

a. 12 cells (50.0%) have expected count less than 5. The minimum expected count is .45.

Chi square test between PPBS and mild cognitive impairment shows statistical significance. ($p<0.05$)

Table 21b: PPBS and mild cognitive impairment

Symmetric Measures

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Interval by Interval	Pearson's R	.170	.072	2.429	.016[c]
Ordinal by Ordinal	Spearman Correlation	.223	.067	3.214	.002[c]
N of Valid Cases		200			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

c. Based on normal approximation.

Pearson and spearman correlation shows statistical significance between PPBS and Mild Cognitive impairment.($p<0.05$)

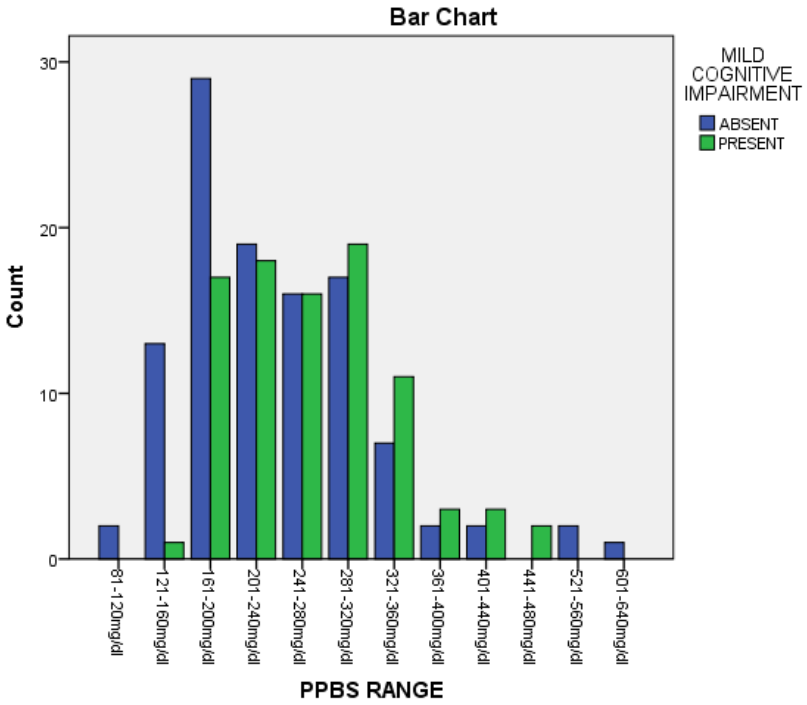


Chart 14: PPBS and mild cognitive impairment.

Table 22a: PPBS and severe cognitive impairment

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	28.159[a]	11	.003
Likelihood Ratio	26.373	11	.006
Linear-by-Linear Association	15.652	1	.000
N of Valid Cases	200		

a. 14 cells (58.3%) have expected count less than 5. The minimum expected count is .20.

Chi square test between PPBS and severe cognitive impairment shows statistical significance. ($p<0.05$)

Table 22b: PPBS and severe cognitive impairment

Symmetric Measures

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Interval by Interval	Pearson's R	.280	.073	4.111	.000[c]
Ordinal by Ordinal	Spearman Correlation	.252	.068	3.664	.000[c]
N of Valid Cases		200			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

c. Based on normal approximation.

Pearson and spearman correlation shows statistical significance between PPBS and Severe Cognitive Impairment ($p<0.05$)

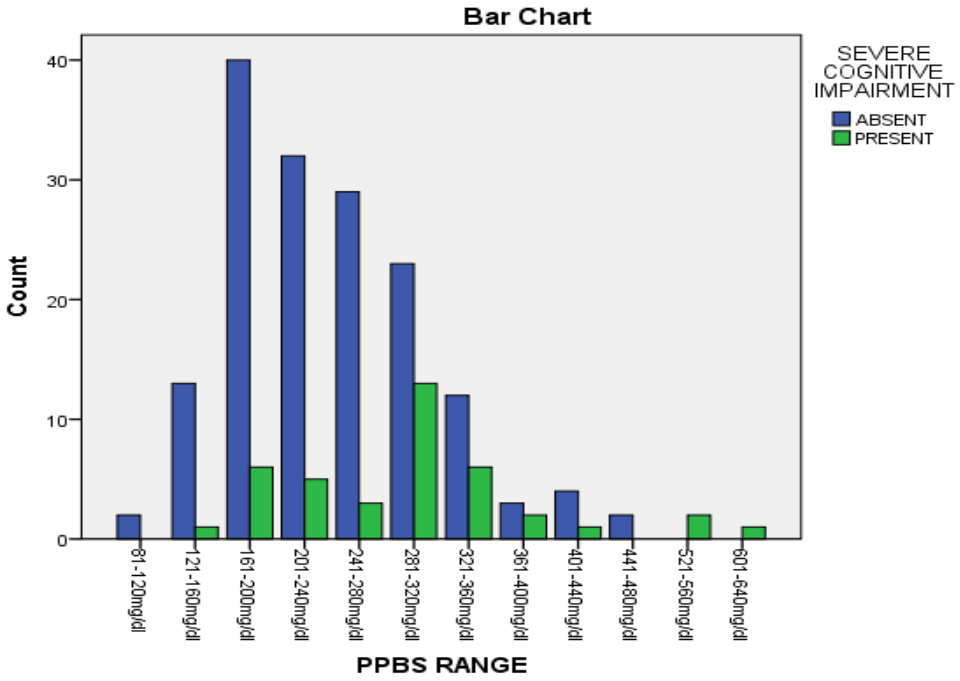


Chart 15: PPBS and severe cognitive impairment

INFERENCE:

- High PPBS values were associated with cognitive impairment.

Table 23a: HbA1c and MMSE score

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	34.893[a]	3	.000
Likelihood Ratio	36.968	3	.000
Linear-by-Linear Association	34.417	1	.000
N of Valid Cases	181		

a. 1 cells (12.5%) have expected count less than 5. The minimum expected count is 2.04.

Chi-square test between HbA1c and MMSE shows statistical significance. ($p<0.05$)

Table 23b:HbA1c and MMSE score

Symmetric Measures

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Interval by Interval	Pearson's R	-.437	.061	-6.505	.000[c]
Ordinal by Ordinal	Spearman Correlation	-.430	.058	-6.365	.000[c]
N of Valid Cases		181			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

c. Based on normal approximation.

Pearson and spearman correlation shows statistical significance between HbA1c and MMSE ($p<0.05$)

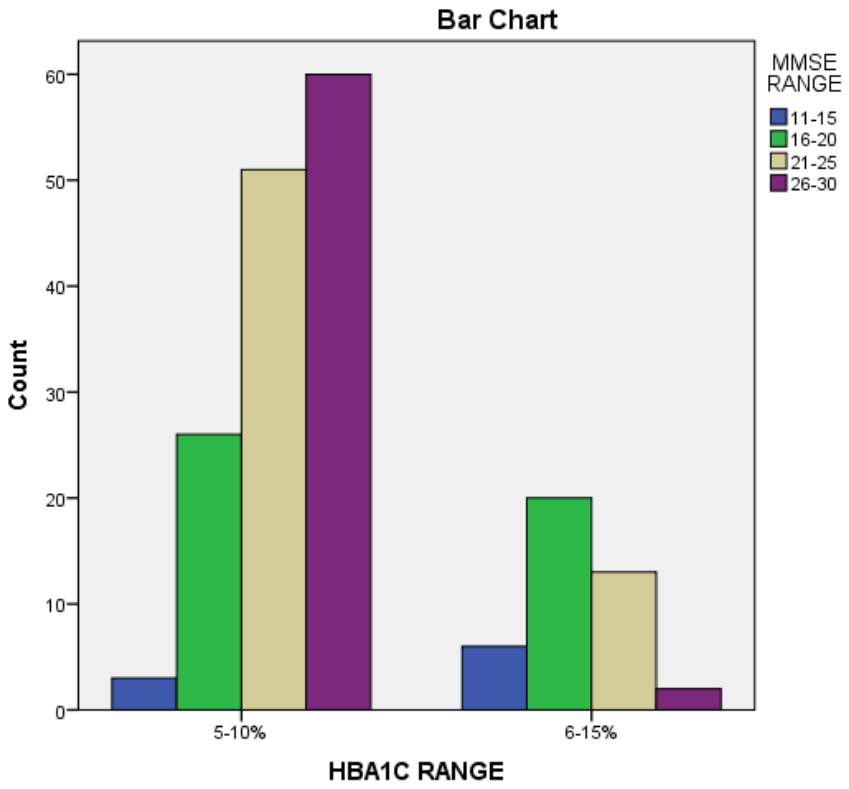


Chart 16: HbA1c and MMSE score

Table 24a: HbA1c and severe cognitive impairment

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	19.321[a]	1	.000		
Continuity Correction[b]	17.278	1	.000		
Likelihood Ratio	16.696	1	.000		
Fisher's Exact Test				.000	.000
Linear-by-Linear Association	19.214	1	.000		
N of Valid Cases	181				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 6.80.

b. Computed only for a 2x2 table

Chi-square test between HbA1c and severe cognitive impairment shows statistical significance. ($p<0.05$)

Table 24b: HbA1c and severe cognitive impairment

Symmetric Measures

		Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.
Interval by Interval	Pearson's R	.327	.085	4.625	.000[c]
Ordinal by Ordinal	Spearman Correlation	.327	.085	4.625	.000[c]
N of Valid Cases		181			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

c. Based on normal approximation.

Pearson and spearman correlation shows statistical significance between HbA1c and Severe Cognitive impairment ($p<0.05$)

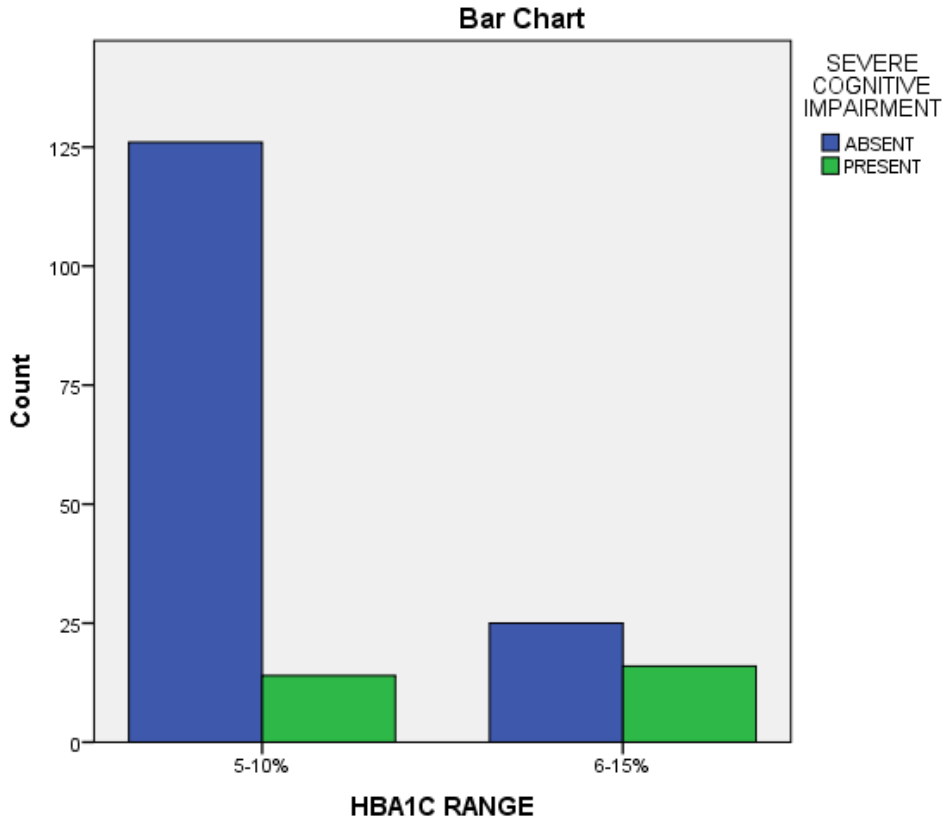


Chart 17: HbA1c and severe cognitive impairment

INFERENCE:

- High HbA1c values are associated with cognitive impairment.

Table 25: Anova Table

			Sum of Squares	df	Mean Square	F	Sig.
MMSE RANGE * HBA1C RANGE	Between Groups	(Combined)	27.529	1	27.529	42.317	.000
	Within Groups		116.449	179	.651		
	Total		143.978	180			
NO COGNITIVE IMPAIREMENT * HBA1C RANGE	Between Groups	(Combined)	6.054	1	6.054	29.390	.000
	Within Groups		36.874	179	.206		
	Total		42.928	180			
MILD COGNITIVE IMPAIRMENT * HBA1C RANGE	Between Groups	(Combined)	.682	1	.682	2.772	.098
	Within Groups		44.069	179	.246		
	Total		44.751	180			
SEVERE COGNITIVE IMPAIRMENT * HBA1C RANGE	Between Groups	(Combined)	2.672	1	2.672	21.390	.000
	Within Groups		22.356	179	.125		
	Total		25.028	180			

a. With fewer than three groups, linearity measures for MMSE RANGE * HBA1C RANGE cannot be computed.

b. With fewer than three groups, linearity measures for NO COGNITIVE IMPAIREMENT * HBA1C RANGE cannot be computed.

c. With fewer than three groups, linearity measures for MILD COGNITIVE IMPAIRMENT * HBA1C RANGE cannot be computed.

d. With fewer than three groups, linearity measures for SEVERE COGNITIVE IMPAIRMENT * HBA1C RANGE cannot be computed.

ANOVA TABLE

- FACTOR:HbA1C
- DEPENDENT VARIABLES: MMSE, NO COGNITIVE IMPAIRMENT, MILD, SEVERE COGNITIVE IMPAIRMENT
- HbA1c has strong association with MMSE, mild and severe cognitive impairment. ($p<0.05$)
- It has no association with no cognitive impairment.
- This signifies that uncontrolled diabetes can lead to mild or severe cognitive impairment.

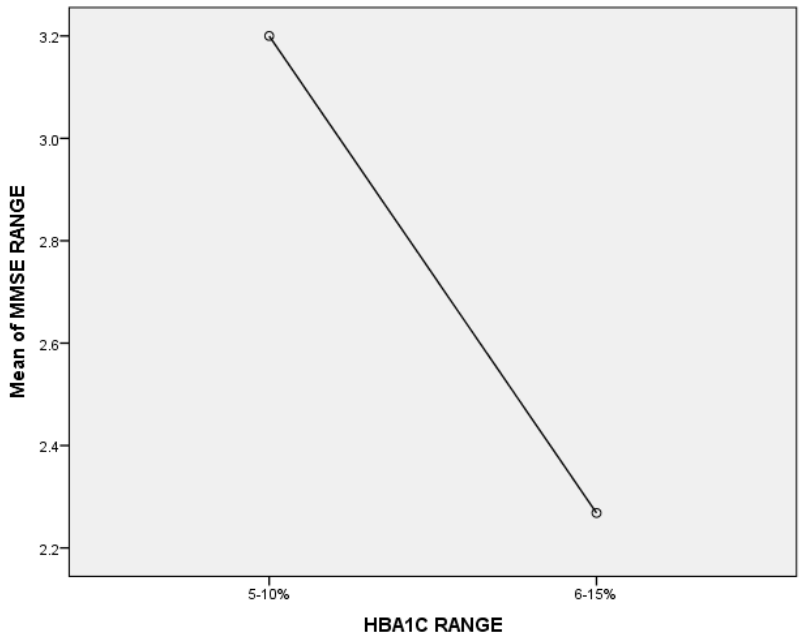


Chart 18: Mean of MMSE and HbA1c range.

- Mean of MMSE decreases with increasing HbA1c levels.

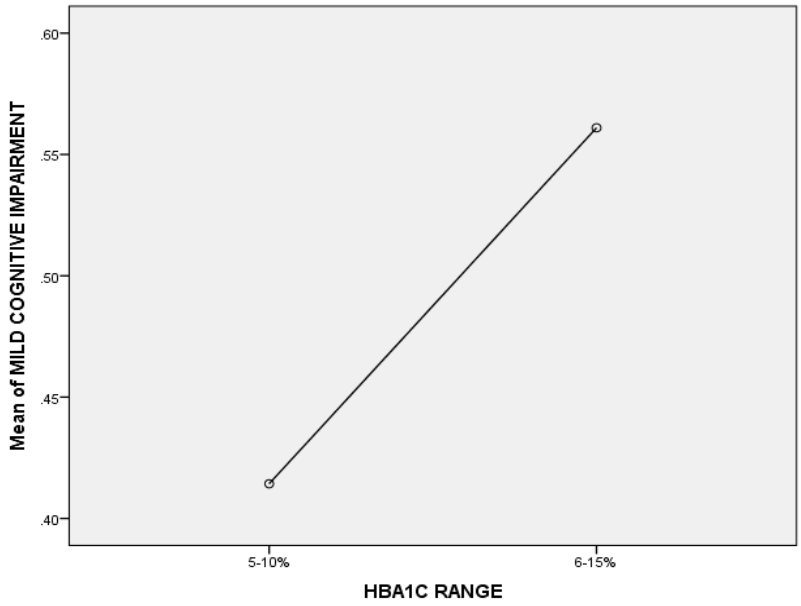


Chart 19:mean of mild cognitive impairment and HbA1c range

- Mean of mild cognitive impairment increases with increase in HbA1c levels.

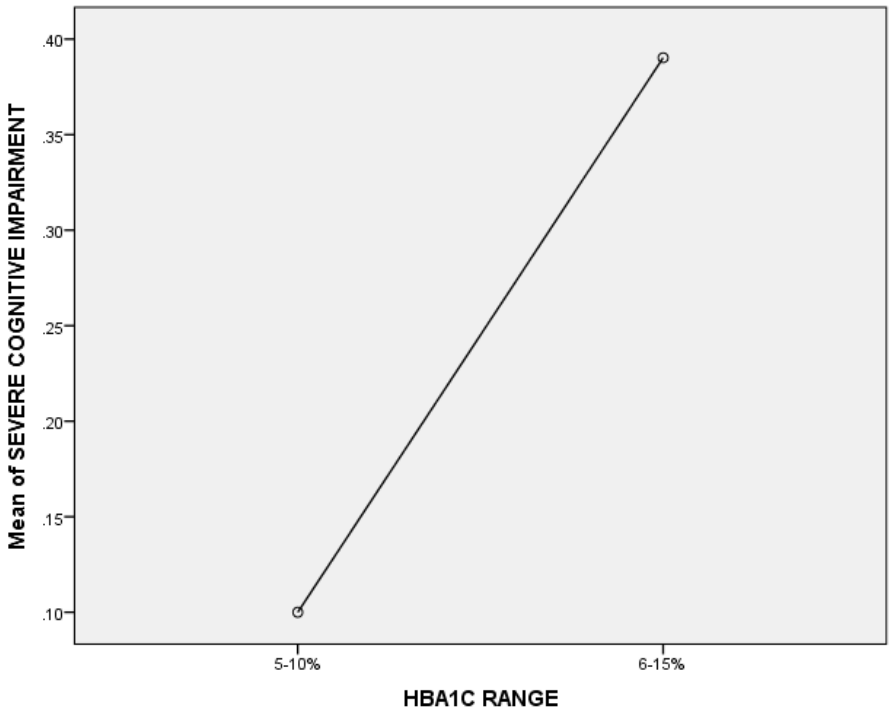


Chart 20:Mean of severe cognitive impairment and HbA1c range

- Mean of severe cognitive impairment increases with increasing HbA1c levels.

INFERENCE

- FBS, PPBS and HbA1c has strong association with MMSE SCORE and Cognitive Impairment.
- Co-morbid conditions also has some additive effect on the cognition.
- This study signifies that association between Diabetes and Cognition is merely significant not by chance.
- Hence NULL HYPOTHESIS can be rejected i.e there is no association between Diabetes and Cognitive Impairment.
- This study proves that Diabetes increases the risk of COGNITIVE IMPAIREMENT.

DISCUSSION

A total of 200 elderly diabetic patients satisfying the inclusion criteria and exclusion criteria were taken for the study.

In our study around 121 patients (60.5%) were in the age group 60-65 years. It was similar to a study conducted by Rajeshkanna et.al, where the mean age of the individuals was 66.8 for males and 63.5 for females.[4]

In our study out of 200 patients, males were 113 (56.5%) and females were 87 (43.5%).This study showed a slight male preponderance towards males which was not similar to the study conducted by Rajeshkanna et.al.[4]

In our study 93 patients (46.5%) were illiterates,117 patients (53.5%) were literates. Among the literates 43 patients (21.5%) had UPPER PRIMARY EDUCATION. It correlates with study conducted by Laks J et.al, which also showed that there was no association between MMSE Scoring and the educational status of the patients.[53]

In our study 19 patients(9.5%) had duration of diabetes for 20 years.It was the highest frequency. It is similar to a study conducted by Ruis et al, which showed that diabetic duration was associated with cognitive decline. The longer the duration of diabetes, more was the cognitive decline.

Another study done by Gregg et.al, also showed that there was a trend of increasing risk of cognitive decline with increasing duration of diabetes. A study conducted by Elias et.al, showed that each 5 year increment between diabetic diagnosis and cognitive assessment was associated with lower scores on tests of logical memory, word fluency. [86]

In our study 71 patients (35.5 %) had FBS range 161-200mg/dl and 46 patients (23%)in the study had PPBS range 161-200mg/dl. A study done by Abbatecola et al. demonstrated that exaggerated postprandial glucose excursions are associated with disturbances in both global executive and attention functioning .

Thus it is possible that a tight control of postprandial glucose may prevent cognitive decline in elder diabetic individividuals.[87]

In our study Chi square test showed significant association between FBS and Severe Cognitive Impairment ($p<0.05$).

There was also significant association between the FBS levels and MMSE score. ($p<0.05$).

Chi square test also showed significant association between PPBS and Mild Cognitive Impairment ($p<0.05$).

The association between PPBS and MMSE Score was also statistically significant.($p<0.05$)

The descriptive statistical analysis in our study showed a mean HbA1c 8.76%-which was significantly high and showed an association with mean MMSE score 22-indicating Mild Cognitive Impairment

These findings were in correlation with study conducted by Rajeshkanna et.al in which it was also noted that complications of diabetes were positively correlated with the level of HbA1c.

HbA1c level had been shown to be associated with cognitive functions even in the non-diabetic population.Yaffe et al had shown that those with HbA1C level ≥7%,

the age-adjusted risk for developing mild cognitive impairment was increased nearly 4-fold and the risk was increased nearly 3-fold for developing dementia.[4]

A study done by Pearson et.al, showed a significant positive correlation between HbA1c and cognitive dysfunction.[86]

In our study 159 patients (79.5%) were regularly undergoing treatment for diabetes. It indicated that even though patient had regular treatment they were prone for cognitive impairment.

It was not similar to study done by Pearson et al, which showed a strong positive correlation between regularity of diabetic treatment and good cognitive performance.[86]

Our study showed that FBS, PPBS and HbA1c statistically had strong association with MMSE SCORE and Cognitive Impairment. Co-morbid conditions like Hypertension also has some additive effect on the cognition.

In a study done by Arvanitakis et al, it was found that diabetes mellitus was associated with double the risk of developing AD.[88]

A study conducted by Munshi et al, also showed that the elderly diabetic population also had cognitive dysfunction.

A study conducted by Cukierman et al, showed that individuals with diabetes were 1.5 times more likely to experience frank dementia than individuals without diabetes.[86]

Workers like Gold et al, showed abnormalities in cognitive functions mediated by frontal lobe involving a number of complex behaviors like problem solving, planning, organization, insight, reasoning and attention.[86]

CONCLUSION

- A total of 200 elderly diabetic patients satisfying the inclusion and exclusion criteria were included in the study.
- 121 patients (60.5%) were in the age group 60-65 years, followed by 42 patients (21%) were in the age group 66-70 years.
- In the study total males were 113 patients (56.5%) and females were 87 patients(43.5%).
- Among the patients 93 patients (46.5%) were illiterates, 117 patients (53.5%) were literates. Among the literates 43 patients (21.5%) had UPPER PRIMARY EDUCATION.
- In the study 19 patients(9.5%) had duration of diabetes for 20 years.
- 159 patients (79.5%) of patients were regularly undergoing treatment for diabetes. This signifies that even in patients were adherent to treatment, still cognitive impairment was seen.
- In our study 90 patients (45%) had mild cognitive impairment followed by 40 patients (20%) had severe cognitive impairment. In the study majority of patients had cognitive impairment.
- The study showed 71 patients (35.5 %) had FBS range of 161-200mg/dl.
- In the study 46 patients (23%) had PPBS range of 161-200mg/dl.
- The study showed that 31 patients (15.5%) had MMSE score of 23.
- It indicates that mild cognitive impairment was more common.

- The descriptive statistics done for the study showed following values:
- Mean HbA1c 8.76%- which was significantly high
- Mean MMSE score 22-indicating Mild Cognitive Impairment.
- Mean duration of diabetes was 13.37 years.
- The Chi square test showed the association between FBS and severe cognitive impairment was statistically significant. ($p<0.05$).
- Chi square test signifies that the association between FBS and MMSE was statistically significant. ($p<0.05$)
- Chi square test between PPBS and Severe Cognitive Impairment and MMSE Score was statistically significant. ($p<0.05$)
- In the study the association between HbA1c and Severe Cognitive Impairment and MMSE Score was statistically significant.($p<0.05$)
- The study showed that FBS, PPBS and HbA1c had strong association with MMSE SCORE and Cognitive Impairment.
- Co-morbid conditions had some additive effect on the cognition.
- This study signifies that association between Diabetes and Cognition was merely significant not by chance.
- Hence NULL HYPOTHESIS can be rejected i.e there is no association between Diabetes and Cognitive Impairment.
- **This study proves that Diabetes increases the risk of COGNITIVE IMPAIREMENT.**

SUMMARY

It was an Observational study conducted in the tertiary care hospital.

The objectives of the study were to identify cognitive impairment in elderly diabetic patients and to determine the relationship between duration of diabetes and diabetes control with cognitive impairment. Most of the patients belonged to 60-65 years of age. A mean duration of diabetes was found to be around 20 years from the study.

Even though patients were adherent to treatment the cognitive impairment was present in the patients. Majority of patients had mild cognitive impairment.

The study showed that FBS, PPBS and HbA1c had strong association with MMSE SCORE and Cognitive Impairment. Co-morbid conditions had some additive effect on the cognition. This study signifies that association between Diabetes and Cognitive Dysfunction.

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PROFORMA

1. OP/IP No.: 2.Date:

3. Serial No.:

4. Name: 5.Age: 6. Gender:

7. Occupation:

8. Date of Admission:

9. Date of Discharge:

10. Socioeconomic status:

11. Address with Phone no.:

12. Chief Complaints:

13. Past history:

14. Family history:

15. Personal History:

16. General Physical Examination:

PR: BP: HEIGHT:

WEIGHT:

Pallor: Icterus: Cyanosis:

Clubbing: Lymphdenopathy: Oedema:

17. Systemic examination:

CVS:

RS:

PA:

CNS:

18. INVESTIGATIONS:

CBC

FBS

PPBS

HbA1c

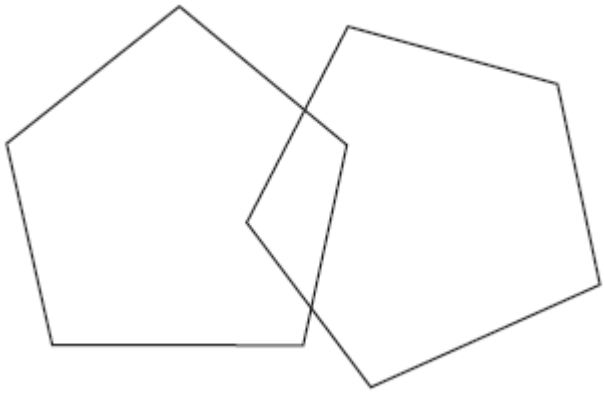
Renal function tests

Thyroid function tests

Liver function tests

Serum electrolytes

STANDARDIZED MINI-MENTAL STATE EXAMINATION (SMMSE) **QUESTION**		**TIME ALLOWED**	**SCORE**
1	a. ***What year is this?***	10 seconds	/1
b. ***Which season is this?***		10 seconds	/1
c. ***What month is this?***		10 seconds	/1
d. ***What is today's date?***		10 seconds	/1
e. ***What day of the week is this?***		10 seconds	/1
2	a. ***What country are we in?***	10 seconds	/1
b. ***What province are we in?***		10 seconds	/1
c. ***What city/town are we in?***		10 seconds	/1
d. ***IN HOME – What is the street address of this house?*** ***IN FACILITY – What is the name of this building?***		10 seconds	/1
e. ***IN HOME – What room are we in? IN FACILITY – What floor are we on?***		10 seconds	/1
3	**SAY:** ***I am going to name three objects. When I am finished, I want you to repeat them. Remember what they are because I am going to ask you to name them again in a few minutes.*** Say the following words slowly at 1-second intervals **- ball/ car/ man**	20 seconds	/3
4	***Spell the word WORLD. Now spell it backwards.***	30 seconds	/5
5	***Now what were the three objects I asked you to remember?***	10 seconds	/3
6	**SHOW** wristwatch. **ASK:** ***What is this called?***	10 seconds	/1
7	**SHOW** pencil. **ASK**: ***What is this called?***	10 seconds	/1
8	**SAY:** ***I would like you to repeat this phrase after me: No ifs, ands or buts.***	10 seconds	/1
9	**SAY:** ***Read the words on the page and then do what it says.*** Then hand the person the sheet with CLOSE YOUR EYES on it. If the subject reads and does not close their eyes, repeat up to three times. Score only if subject closes eyes	10 seconds	/1
10	**HAND** the person a pencil and paper. **SAY:** ***Write any complete sentence on that piece of paper.*** (Note: The sentence must make sense. Ignore spelling errors)	30 seconds	/1
11	**PLACE** design, eraser and pencil in front of the person. **SAY**: ***Copy this design please.***	1 minute	/1

	 Allow multiple tries. Wait until person is finished and hands it back. Score only for correctly copied diagram with a 4-sided figure between two 5-sided figures.		
12	**ASK** the person if he is right or left-handed. Take a piece of paper and hold it up in front of the person. **SAY**: ***Take this paper in your right/left hand*** (whichever is non-dominant), ***fold the paper in half once with both hands and put the paper down on the floor*** . Score 1 point for each instruction executed correctly. Takes paper correctly in hand Folds it in half Puts it on the floor	30 seconds	/1 /1 /1
	TOTAL TEST SCORE	**/30**	

Method	**Score**	**Interpretation**
Single cutoff	<24	Abnormal
Range	<21	Increased odds of Dementia.
	>25	Decreased odds of Dementia.
Severity	24-30	No cognitive impairment.
	18-23	Mild cognitive impairment.
	0-17	Severe cognitive impairment.

INFORMED CONSENT FORM

Cognitive impairment and dementia in patients with T2DM creates a large burden for patients, their families, and society. Hence, early diagnosis and proper glycaemic control is required for halt of cognitive impairment.If you agree to participate in the study we will collect information (as per proforma) from you or a person responsible for you or both. We will collect the treatment and relevant details from your hospital record. This information collected will be used for only dissertation and publication. This study has been reviewed by the institutional ethical committee. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

I understand that I remain free to withdraw from the study at any time and this will not change my future care. I have read or have been read to me and understood the purpose of the study, the procedure that will be used, the risk and benefits associated with my involvement in the study and the nature of information that will be collected and disclosed during the study. I have had the opportunity to ask my questions regarding various aspects of the study and my questions are answered to my satisfaction. I, the undersigned agree to participate in this study and authorize the collection and disclosure of my personal information for dissertation.

Subject name :

Parents / Guardians name :

DATE: SIGNATURE /THUMB IMPRESSION

Name and signature of person taking consent

ಮಾಹಿತಿಯುಕ್ತ ಸಮ್ಮತಿಯ ನಮೂನೆ

ಮಧುಮೇಹ ರೋಗಿಗಳಲ್ಲಿ ಅರಿವಿನ ಕೊರತೆ ಮತ್ತು ಬುದ್ಧಿಮಾಂದ್ಯತೆಯ ರೋಗಿಗಳಿಗೆ, ಅವರ ಕುಟುಂಬಗಳು, ಮತ್ತು ಸಮಾಜಕ್ಕೆ ಒಂದು ದೊಡ್ಡ ಹೊರೆ ಸೃಷ್ಟಿಸುತ್ತದೆ. ಆದ್ದರಿಂದ, ರೋಗ ಆರಂಭಿಕ ಮತ್ತು ಸರಿಯಾದ ಗ್ಲೈಸೆಮಿಕ್ ನಿಯಂತ್ರಣ ಅರಿವಿನ ಜ್ಞಾನದ ನ್ಯೂನತೆಯ ಮಟ್ಟವನ್ನು ತಡೆಯಲು ಅಗತ್ಯವಿದೆ. ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪಿದಲ್ಲಿ ನಿಮ್ಮಿಂದ ಅಥವಾ ನಿಮ್ ಜವಬ್ದಾರಿಯನು ತೆಗದುಕೋಲೊ ವೆಕ್ತೀ ಇಂದ (ಪ್ರೊಫಾರ್ಮ ಪ್ರಕಾರ) ಮಾಹಿತಿಯನ್ನು ಸಂಗ್ರಹಿಸವೆ.ನಾವು ನಿಮ್ಮ ಆಸ್ಪತ್ರೆಯ ದಾಖಲೆ ಚಿಕಿತ್ಸೆ ಮತ್ತು ಸಂಬಂಧಿಸಿದ ವಿವರಗಳನ್ನು ಸಂಗ್ರಹಿಸುತ್ತದೆ. ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಪ್ರೌಢಪ್ರಬಂಧದಲ್ಲಿ ಮತ್ತು ಪ್ರಕಟಣೆಗೆ ಬಳಸಲಾಗುತ್ತದೆ. ಈ ಅಧ್ಯಯನವು ಸಾಂಸ್ಥಿಕ ನೈತಿಕ ಸಮಿತಿಯು ವಿಮರ್ಶಿಸುತ್ತದೆ ಮಾಡಲಾಗಿದೆ. ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನಿರಾಕರಿಸಿದರೆ ಆರೈಕೆ ಮತ್ತು ಚಿಕಿತ್ಸೆ ಚಿಕಿತ್ಸೆಯಿಳಿ ಯಾವುದ್ ಬದಲಾವಣೆ ಯಾಗುವುದಿಲ್ಲ.ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪುತ್ತೀರಿ ಮಾತ್ರ ಹೆಬ್ಬೆಟ್ಟಿನ ಗುರುತು ಒದಗಿಸುವ ಅಗತ್ಯವಿದೆ.

ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನ ಇಂದ ಹಿಂದಕ್ಕೆ ಸರಿಯಬಹುದು ನಿಮ ಚಿಕಿತ್ಯಲಿ ಯಾವುದೇಬದಲಾವಣೆ ಯಾಗುವುದಿಲ್ಲ ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನ ಹಿಂದಕ್ಕೆ ಉಚಿತ ಉಳಿದು ಈ ನನ್ನ ಭವಿಷ್ಯದ ಕಾಳಜಿ ಬದಲಾಗುವುದಿಲ್ಲ ಎಂದು ಅರ್ಥ. ನಾನು ಓದಲು ಅಥವಾ , ಅಪಾಯ ಮತ್ತು ನನ್ನ ಅಧ್ಯಯನದಲ್ಲಿ ತೊಡಗಿರುವ ಮತ್ತು ಸಂಗ್ರಹಿಸಿ ಅಧ್ಯಯನ ಮಾಡುವ ಸಂದರ್ಭದಲ್ಲಿ ಬಹಿರಂಗ ಎಂದು ಮಾಹಿತಿ ಸ್ವರೂಪ ಸವಲತ್ತುಗಳು ಬಳಸಲಾಗುತ್ತದೆ ಎಂದು ವಿಧಾನ ನನಗೆ ಓದಲು ಮತ್ತು ಅಧ್ಯಯನದ ಉದ್ದೇಶ ಗ್ರಹಿಸಲಾಗಿದೆ . ನಾನು ಅಧ್ಯಯನ ವಿವಿಧ ಅಂಶಗಳನ್ನು ಬಗ್ಗೆ ನನ್ನ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ಅವಕಾಶ ಹೊಂದಿದ್ದರು ಮತ್ತು ನನ್ನ ಪ್ರಶ್ನೆಗಳಿಗೆ ನನ್ನ ತೃಪ್ತಿ ಉತ್ತರಿಸುತ್ತದೆ. ನಾನು ರುಜುಮಾಡಿರುವ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಮತ್ತು ಪ್ರೌಢಪ್ರಬಂಧದಲ್ಲಿ ನನ್ನ ವೈಯಕ್ತಿಕ ಮಾಹಿತಿಯ ಸಂಗ್ರಹಣೆ ಮತ್ತು ಬಹಿರಂಗಪಡಿಸುವಿಕೆಯ ಅಧಿಕೃತಗೊಳಿಸಲು ಒಪ್ಪುತ್ತೇನೆ .

ರೋಗಿಯ ಹೆಸರು:

ಪಾಲಕರು / ಗಾರ್ಡಿಯನ್ಸ್ ಹೆಸರು:

ದಿನಾಂಕ:

ಅರ್ಜಿದಾರರ ಸಹಿ / ಹೆಸರು ಮತ್ತು ವ್ಯಕ್ತಿ ತೆಗೆದುಕೊಳ್ಳುವ ಒಪ್ಪಿಗೆ ಸಹಿ

PATIENT INFORMATION SHEET

STUDY TITLE: ASSESSMENT OF ASSOCIATION BETWEEN COGNITIVE DEFICITS AND GLYCAEMIC CONTROL AND DURATION OF DIABETES IN ELDERLY DIABETIC PATIENTS ATTENDING TERTIARY CARE HOSPITAL, KOLAR, KARNATAKA.

STUDY LOCATION: R L Jalappa Hospital and research centre attached to Sri Devraj Urs Medical College. Tamaka, Kolar

DETAILS:

In the study elderly diabetic patient will be taken and they will be subjected to MMSE and the cognitive deficit will be assessed. The duration of diabetes and the glycaemic control will be assessed.

The study requires routine investigations like CBC, RFT, TFT, LFT, FBS, PPBS, HbA1C will be done. The cost of investigations will be taken up by the principal investigator.

Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in the study we will collect information (as per proforma) from you or a person responsible for you or both. Relevant history will be taken. This information collected will be used only for dissertation and publication.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of the Institutional Ethics Committee. There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

For further information contact
Dr Spandana Peddareddy
Post graduate
Department of general medicine
SDUMC , Kolar

MASTER CHART

S.No.	Name	IP No.	Age	Sex	Education	Duration Of Diabetes	Diabetictreatment Taken	Other Comorbidities	FBS	PPBS	HbA1C	Renal Function Tests	Thyroid Function Tests	Liver Function Tests	Serum Electrolytes	MMSE Score	No Cognitive Impairment	Mild Cognitive Impairment	Severe Cognitive Impairment
1	Jayakumar	134918	60 Years	Male	Lower Secondary	16 Years	Irregular	Nil	167 mg/dl	197 mg/dl	7.00%	Normal Limits	Normal Limits	Normal Limits	Normal	30	Normal		
2	Munishamappa	273265	60 Years	Male	Uneducated	1.5 Years	Regular	Nil	152 mg/dl	301 mg/dl	7.20%	Normal Limits	Normal Limits	Normal Limits	Normal	28	Normal		
3	Renukacharya	277624	60 Years	Male	Lower Secondary	15 Years	Regular	Hypertension	168 mg/dl	250 mg/dl	6.80%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		
4	Lakshmi Narayana	279701	60 Years	Male	Uneducated	1 Year	Regular	Hypertension	213 mg/dl	280 mg/dl	8.20%	Normal Limits	Normal Limits	Normal Limits	Normal	20		Present	
5	Sathyamma	257795	60 Years	Female	Uneducated	3 Years	Regular	Nil	124 mg/dl	189 mg/dl	6.60%	Normal Limits	Normal Limits	Normal Limits	Normal	25	Normal		
6	Anjanamma	258839	70 Years	Female	Uneducated	4 Years	Regular	Nil	122 mg/dl	287 mg/dl	8.60%	Normal Limits	Normal Limits	Normal Limits	Normal	18		Present	
7	Shivanna	148582	60 Years	Male	Lower Secondary	20 Years	Irregular	Hypertension	130 mg/dl	148 mg/dl	11.50%	Normal Limits	Normal Limits	Normal Limits	Normal	30	Normal		
8	Sanjeevappa	35081	70 Years	Male	Upper Primary	10 Years	Regular	Nil	160 mg/dl	322 mg/dl	8.70%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
9	Muralidhar	266358	60 Years	Male	Upper Primary	6 Years	Regular	Nil	102 mg/dl	97 mg/dl	9.10%	Normal Limits	Normal Limits	Normal Limits	Normal	30	Normal		
10	Shabeer Pasha	263923	60 Years	Male	Upper Primary	3 Years	Regular	TB 8 years ago treated	90 mg/dl	114 mg/dl	12.10%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		
11	Srinivasaiah Shetty	69612	66 Years	Male	Lower Secondary	13 Years	Regular	Hypertension	117 mg/dl	238 mg/dl	5.00%	Normal Limits	Normal Limits	Normal Limits	Normal	27	Normal		
12	Balaramachari	268148	60 Years	Male	Upper Primary	1.5 Years	Irregular	Nil	161 mg/dl	256 mg/dl	6.90%	Normal Limits	Normal Limits	Normal Limits	Normal	27	Normal		
13	Chandra Reddy	54116	65 Years	Male	Primary	5 Years	Irregular	TB 1 Year ago treated	247 mg/dl	322 mg/dl	7.20%	Normal Limits	Normal Limits	Normal Limits	Normal	29	Normal		
14	Muniyamma	266937	60 Years	Female	Uneducated	2 Years	Irregular	Epilepsy	186 mg/dl	290 mg/dl	8.40%	Normal Limits	Normal Limits	Normal Limits	Normal	15			Present
15	Divakar.R	161550	65 Years	Male	Graduate	20 Years	Regular	Hypertension	174 mg/dl	284 mg/dl	8.90%	Normal Limits	Normal Limits	Normal Limits	Normal	30	Normal		
16	Shanmugam	250528	70 Years	Male	Upper Primary	10 Years	Regular	Nil	109 mg/dl	444 mg/dl	11.10%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
17	Someshwar Rao	251427	61 Years	Male	Upper Primary	20 Years	Irregular	Dilated Cardiomyopathy, Hypertension	118 mg/dl	205 mg/dl	6.40%	Normal Limits	Normal Limits	Normal Limits	Normal	30	Normal		
18	Krishnappa.M	259781	65 Years	Male	Uneducated	15 Years	Regular	Hypertension, Ischaemic Heart Disease, Renal Cell Carcinoma	189 mg/dl	250 mg/dl	7.20%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
19	Sardar	251130	62 Years	Male	Upper Primary	10 Years	Regular	Hypertension	197 mg/dl	327 mg/dl	8.60%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
20	Gulabjan	259096	60 Years	Female	Uneducated	20 Years	Regular	HYPERTENSION, ISCHAEMIC HEART DISEASE,TB 20 Years Ago	187 mg/dl	363 mg/dl	9.40%	Normal Limits	Normal Limits	Normal Limits	Normal	22		Present	

21	Ramakrishna Gowda	260671	62 Years	Male	Graduate	15 Years	Regular	Nil	123 mg/dl	244 mg/dl	11.10%	Normal Limits	Normal Limits	Normal Limits	Normal	20		Present	
22	Zakeerunnisa	257599	60 Years	Female	Uneducated	10 Years	Regular	Hypertension, Ischaemic Heart Disease	165 mg/dl	260 mg/dl	7.50%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
23	Narayana Swamey	259677	60 Years	Male	Lower Secondary	20 Years	Regular	Hypertension, Ischaemic Heart Disease	154 mg/dl	198 mg/dl	6.80%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		
24	Muniyamma	250898	80 Years	Female	Uneducated	10 Years	Irregular	Nil	339 mg/dl	559 mg/dl	11.30%	Normal Limits	Normal Limits	Normal Limits	Normal	12			Present
25	Vijay Kumari	258618	63 Years	Female	Upper Primary	4 Years	Regular	Hypertension	142 mg/dl	164 mg/dl	6.60%	Normal Limits	Normal Limits	Normal Limits	Normal	25	Normal		
26	Ramakrishnappa	251875	61 Years	Male	Upper Primary	10 Years	Regular	Hypertension, Ischaemic Heart Disease, Bronchial Asthma	130 mg/dl	210 mg/dl	10.30%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
27	Lakshmidevamma	267389	65 Years	Female	Graduate	8 Years	Regular	Hypertension	286 mg/dl	379 mg/dl	12.50%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	
28	Munegowda	266758	60 Years	Male	Lower Secondary	1.5 Month	Regular	Nil	125 mg/dl	221 mg/dl	6.80%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		
29	Muniswamappa	259052	60 Years	Male	Lower Secondary	3 Years	Regular	Hypertension	191 mg/dl	238 mg/dl	7.20%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		
30	Sonnegowda	261567	70 Years	Male	Higher Secondary	10 Years	Regular	Hypertension,Ischaemic Heart Disease	129 mg/dl	284 mg/dl	11.10%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
31	Ramappa	171678	60 Years	Male	Uneducated	5 Years	Regular	Nil	549 mg/dl	629 mg/dl	13.10%	Normal Limits	Normal Limits	Normal Limits	Normal	16			Present
32	Muninanjamma	259082	62 Years	Female	Uneducated	3 Years	Regular	Nil	109 mg/dl	182 mg/dl	6.30%	Normal Limits	Normal Limits	Normal Limits	Normal	16			Present
33	Hanumappa	255454	70 Years	Male	Uneducated	15 Years	Regular	Hypertension	131 mg/dl	198 mg/dl	12.50%	Normal Limits	Normal Limits	Normal Limits	Normal	15			Present
34	Pillamma	251006	70 Years	Female	Uneducated	15 Years	Regular	Hypertension	323 mg/dl	164 mg/dl	10.70%	Normal Limits	Normal Limits	Normal Limits	Normal	16			Present
35	Thopamma	253876	75 Years	Female	Uneducated	1 Year	Regular	Hypertension	110 mg/dl	168 mg/dl	7.40%	Normal Limits	Normal Limits	Normal Limits	Normal	16			Present
36	Ujinappa	263042	60 Years	Male	Uneducated	2 Years	Regular	Nil	107 mg/dl	235 mg/dl	7.80%	Normal Limits	Normal Limits	Normal Limits	Normal	20		Present	
37	Lakshmamma	255974	70 Years	Female	Uneducated	9 Months	Regular	Hypertension	143 mg/dl	186 mg/dl	8.40%	Normal Limits	Normal Limits	Normal Limits	Normal	19		Present	
38	Narayana Reddy	11048	60 Years	Male	Uneducated	4 Years	Regular	Nil	159 mg/dl	337 mg/dl	10.10%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	
39	Krishna Reddy	248841	67 Years	Male	Lower Secondary	20 Years	Regular	Hypertension	114 mg/dl	134 mg/dl	7.10%	Normal Limits	Normal Limits	Normal Limits	Normal	30	Normal		
40	Lakshmamma	231999	60 Years	Female	Uneducated	3 Months	Regular	Hypertension	237 mg/dl	307 mg/dl	12.80%	Normal Limits	Normal Limits	Normal Limits	Normal	19		Present	
41	Lakshmi Narasamma	235462	74 Years	Female	Upper Primary	20 Years	Regular	Hypertension	250 mg/dl	356 mg/dl	12.90%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
42	Chotima	236377	65 Years	Female	Uneducated	20 Years	Regular	Nil	279 mg/dl	433 mg/dl	12.30%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	
43	Syed Nazeer Ahmed	239416	65 Years	Male	Lower Secondary	6 Years	Regular	Hypertension	281 mg/dl	326 mg/dl	12.80%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	
44	Veerabadrashetty	229656	65 Years	Male	Uneducated	5 Years	Regular	Nil	221 mg/dl	260 mg/dl	8.40%	Normal Limits	Normal Limits	Normal Limits	Normal	17			Present
45	Shekar Reddy	231101	76 Years	Male	Lower Secondary	10 Years	Regular	Ischaemic Heart Disease	231 mg/dl	330 mg/dl	11.90%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
46	Lakshmidevamma	223247	60 Years	Female	Uneducated	15 Years	Regular	TB 30 years ago	127 mg/dl	291 mg/dl	10.60%	Normal Limits	Normal Limits	Normal Limits	Normal	16			Present

47	Srinivas Gowda	240969	62 Years	Male	Lower Secondary	2 Years	Regular	Nil	108 mg/dl	411 mg/dl	9.40%	Normal Limits	Normal Limits	Normal Limits	Normal	30	Normal		
48	Ramakrishnappa	240675	60 Years	Male	Primary	2 Months	Regular	Hypertension	224 mg/dl	287 mg/dl	14.90%	Normal Limits	Normal Limits	Normal Limits	Normal	19		Present	
49	Appanna	241371	70 Years	Male	Uneducated	5 Years	Regular	Nil	177 mg/dl	342 mg/dl	12.40%	Normal Limits	Normal Limits	Normal Limits	Normal	16			Present
50	Gurrappa	238499	60 Years	Male	Uneducated	1 Week	Regular	Nil	134 mg/dl	331 mg/dl	11.80%	Normal Limits	Normal Limits	Normal Limits	Normal	19		Present	
51	Azeema Bee	240897	65 Years	Female	Uneducated	5 Years	Regular	Hypertension	96 mg/dl	333 mg/dl	6.20%	Normal Limits	Normal Limits	Normal Limits	Normal	19		Present	
52	Lakshmamma	242153	65 Years	Female	Uneducated	4 Years	Regular	Hypertension	186 mg/dl	221 mg/dl	7.20%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	
53	Saraswathamma	240646	70 Years	Female	Uneducated	5 Years	Irregular	Nil	124 mg/dl	197 mg/dl	11.40%	Normal Limits	Normal Limits	Normal Limits	Normal	18		Present	
54	Prabhavathi Bai	240487	60 Years	Female	Upper Primary	5 Years	Irregular	Hypertension	109 mg/dl	186 mg/dl	11.00%	Normal Limits	Normal Limits	Normal Limits	Normal	19		Present	
55	Ramakrishnappa	236038	60 Years	Male	Uneducated	1 Year	Regular	Hypertension	105 mg/dl	136 mg/dl	7.20%	Normal Limits	Normal Limits	Normal Limits	Normal	17			Present
56	Hemavathamma	230986	79 Years	Female	Upper Primary	12 Years	Regular	Nil	130 mg/dl	152 mg/dl	12.70%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
57	Kushedunnisa	225069	71 Years	Female	Uneducated	8 Years	Regular	Hypertension	241 mg/dl	332 mg/dl	10.40%	Normal Limits	Normal Limits	Normal Limits	Normal	14			Present
58	Ramaswamy	234192	63 Years	Male	Lower Secondary	15 Years	Regular	Hypertension	162 mg/dl	227 mg/dl	7.60%	Normal Limits	Normal Limits	Normal Limits	Normal	28	Normal		
59	Narayanaswamy	235005	68 Years	Male	Lower Secondary	4 Years	Irregular	Nil	209 mg/dl	320 mg/dl	10.60%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
60	Nagaraj	235506	61 Years	Male	Lower Secondary	8 Years	Regular	Hypertension	124 mg/dl	148 mg/dl	5.20%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		
61	Bramaramba	236027	85 Years	Female	Upper Primary	9 Years	Regular	Hypertension	142 mg/dl	226 mg/dl	8.40%	Normal Limits	Normal Limits	Normal Limits	Normal	19		Present	
62	Azeemunnisa	235491	68 Years	Female	Uneducated	10 Years	Regular	Hypertension	145 mg/dl	186 mg/dl	10.20%	Normal Limits	Normal Limits	Normal Limits	Normal	14			Present
63	Venkateshappa	222839	60 Years	Male	Uneducated	5 Years	Regular	Nil	162 mg/dl	286 mg/dl	7.80%	Normal Limits	Normal Limits	Normal Limits	Normal	24	Normal		
64	Basavaraj E.N	62729	60 Years	Male	Upper Primary	8 Years	Regular	Nil	236 mg/dl	354 mg/dl	10.40%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	
65	Dodda Chowdappa	142711	60 Years	Male	Primary	15 Years	Regular	Hypertension	166 mg/dl	256 mg/dl	7.40%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		
66	Venkatesh Gowda	209738	60 Years	Male	Uneducated	7 Years	Irregular	Hypertension, Ischaemic Heart Disease	186 mg/dl	210 mg/dl	7.80%	Normal Limits	Normal Limits	Normal Limits	Normal	28	Normal		
67	Jayaramappa	209709	65 Years	Male	Lower Secondary	4 Years	Regular	Nil	198 mg/dl	220 mg/dl	6.80%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		
68	Ramappa	161863	65 Years	Male	Uneducated	20 Years	Irregular	HYPERTENSION, TB 10 Years Ago	260 mg/dl	320 mg/dl	12.40%	Normal Limits	Normal Limits	Normal Limits	Normal	19		Present	
69	Narayanappa	219650	60 Years	Male	Upper Primary	25 Years	Regular	Nil	178 mg/dl	200 mg/dl	8.20%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
70	Munivenkatappa	215166	75 Years	Male	Upper Primary	30 Years	Regular	Hypertension	236 mg/dl	286 mg/dl	10.40%	Normal Limits	Normal Limits	Normal Limits	Normal	18			Present
71	Seenappa	228808	65 Years	Male	Uneducated	18 Years	Regular	Nil	220 mg/dl	286 mg/dl	7.60%	Normal Limits	Normal Limits	Normal Limits	Normal	22		Present	
72	Venkataramana Reddy	234714	70 Years	Male	Lower Secondary	35 Years	Regular	Hypertension, Ischaemic Heart Disease	198 mg/dl	265 mg/dl	7.00%	Normal Limits	Normal Limits	Normal Limits	Normal	28	Normal		
73	Bisappa	238106	60 Years	Male	Upper Primary	6 Years	Regular	Nil	165 mg/dl	182 mg/dl	6.80%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		

74	Chandrappa	239991	70 Years	Male	Upper Primary	35 Years	Regular	Hypertension	175 mg/dl	240 mg/dl	8.20%	Normal Limits	Normal Limits	Normal Limits	Normal	16			Present
75	Ramappa	239792	60 Years	Male	Lower Secondary	22 Years	Irregular	Hypertension	232 mg/dl	284 mg/dl	10.60%	Normal Limits	Normal Limits	Normal Limits	Normal	17			Present
76	Veebadra Reddy	229656	65 Years	Male	Uneducated	10 Years	Regular	Hypertension	174 mg/dl	248 mg/dl	8.20%	Normal Limits	Normal Limits	Normal Limits	Normal	30	Normal		
77	Narasappa	249906	73 Years	Male	Uneducated	30 Years	Regular	Hypertension,Bronchial Asthma	213 mg/dl	268 mg/dl	6.90%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		
78	Munivenkatamma	195003	62 Years	Female	Uneducated	14 Years	Regular	Hypertension	164 mg/dl	186 mg/dl	6.60%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	
79	Krishnamma	195912	60 Years	Female	Uneducated	5 Years	Irregular	Nil	188 mg/dl	220 mg/dl	7.40%	Normal Limits	Normal Limits	Normal Limits	Normal	22		Present	
80	Zaibunnissa	195784	65 Years	Female	Uneducated	10 Years	Regular	Ischaemic Heart Disease	246 mg/dl	333 mg/dl	12.00%	Normal Limits	Normal Limits	Normal Limits	Normal	14			Present
81	Abida	214249	60 Years	Female	Uneducated	12 Years	Regular	Nil	166 mg/dl	182 mg/dl	6.80%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
82	Shanthamma	182878	65 Years	Female	Upper Primary	15 Years	Regular	Hypertension, Ischaemic Heart Disease	150 mg/dl	178 mg/dl	7.60%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
83	Musthafa Sab	280565	90 Years	Male	Uneducated	30 Years	Regular	HYPERTENSION, TB 30 Years Ago	200 mg/dl	256 mg/dl	8.00%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
84	Abdul Sab	282849	60 Years	Male	Uneducated	10 Years	Regular	Nil	254 mg/dl	320 mg/dl	11.10%	Normal Limits	Normal Limits	Normal Limits	Normal	20		Present	
85	Munivenkatappa	273725	60 Years	Male	Upper Primary	6 Years	Irregular	Hypertension, Bronchial Asthma	174 mg/dl	200 mg/dl	9.00%	Normal Limits	Normal Limits	Normal Limits	Normal	18		Present	
86	Ahmed Ali Khan	283258	80 Years	Male	Uneducated	26 Years	Regular	Hypertension, Ischaemic Heart Disease	204 mg/dl	288 mg/dl	10.00%	Normal Limits	Normal Limits	Normal Limits	Normal	16			Present
87	Muniyamma	139172	67 Years	Female	Uneducated	8 Years	Regular	Nil	194 mg/dl	226 mg/dl	6.90%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		
88	Faizunnissa	240380	65 Years	Female	Uneducated	8 Years	Irregular	Nil	256 mg/dl	330 mg/dl	12.40%	Normal Limits	Normal Limits	Normal Limits	Normal	17			Present
89	Krishnamma	231947	76 Years	Female	Upper Primary	10 Years	Regular	Hypertension, Ischaemic Heart Disease	178 mg/dl	248 mg/dl	8.20%	Normal Limits	Normal Limits	Normal Limits	Normal	19		Present	
90	Chennamma	240246	70 Years	Female	Primary	16 Years	Regular	Nil	146 mg/dl	192 mg/dl	6.90%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	
91	Munivenkatamma	253353	60 Years	Female	Uneducated	4 Years	Regular	Nil	210 mg/dl	258 mg/dl	8.80%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		
92	Vijayamma	251439	60 Years	Female	Uneducated	9 Years	Regular	Hypertension, Bronchial Asthma	192 mg/dl	266 mg/dl	7.70%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	
93	Lakshmamma	251357	60 Years	Female	Uneducated	12 Years	Regular	Nil	164 mg/dl	210 mg/dl	7.60%	Normal Limits	Normal Limits	Normal Limits	Normal	24	Normal		
94	Revamma	265474	65 Years	Female	Primary	10 Years	Regular	Hypertension	196 mg/dl	250 mg/dl	10.40%	Normal Limits	Normal Limits	Normal Limits	Normal	16			Present
95	Lakshmamma	266539	67 Years	Female	Uneducated	20 Years	Regular	Nil	230 mg/dl	288 mg/dl	9.60%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
96	Chinamma	270662	60 Years	Female	Upper Primary	18 Years	Regular	Nil	172 mg/dl	252 mg/dl	6.80%	Normal Limits	Normal Limits	Normal Limits	Normal	24	Normal		
97	Sarojamma	276967	60 Years	Female	Lower Secondary	4 Years	Regular	Nil	142 mg/dl	197 mg/dl	6.50%	Normal Limits	Normal Limits	Normal Limits	Normal	28	Normal		
98	Krishnamurthy	334413	64 Years	Male	Higher Secondary	12 Years	Regular	Hypertension	208 mg/dl	296 mg/dl	7.40%	Normal Limits	Normal Limits	Normal Limits	Normal	30	Normal		
99	Krishnappa	182945	65 Years	Male	Primary	6 Years	Irregular	Nil	160 mg/dl	224 mg/dl	10.00%	Normal Limits	Normal Limits	Normal Limits	Normal	17			Present
100	Ramappa	171694	65 Years	Male	Uneducated	16 Years	Regular	Hypertension	236 mg/dl	312 mg/dl	11.40%	Normal Limits	Normal Limits	Normal Limits	Normal	22		Present	

101	Chandra	193503	64 Years	Male	Lower Secondary	14 Years	Regular	Hypertension	190 mg/dl	246 mg/dl	8.80%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	
102	Munivenkata Reddy	177297	75 Years	Male	Primary	16 Years	Regular	Nil	210 mg/dl	256 mg/dl	7.20%	Normal Limits	Normal Limits	Normal Limits	Normal	24	Normal		
103	Gowramma	278097	60 Years	Female	Uneducated	2 Years	Irregular	Nil	368 mg/dl	410 mg/dl	11.40%	Normal Limits	Normal Limits	Normal Limits	Normal	20		Present	
104	Venkatesh H.B	321538	65 Years	Male	Uneducated	7 Years	Regular	Nil	188 mg/dl	238 mg/dl	10.10%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
105	Mahaboob Bee	326353	67 Years	Female	Uneducated	18 Years	Regular	Hypertension	170 mg/dl	246 mg/dl	6.90%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		
106	Malarveni	301754	62 Years	Female	Graduate	12 Years	Regular	Nil	222 mg/dl	280 mg/dl	6.60%	Normal Limits	Normal Limits	Normal Limits	Normal	28	Normal		
107	Chikkagovindappa	216799	78 Years	Male	Higher Secondary	22 Years	Regular	Hypertension	130 mg/dl	158 mg/dl	6.20%	Normal Limits	Normal Limits	Normal Limits	Normal	30	Normal		
108	Lakshmakka	307056	75 Years	Female	Upper Primary	35 Years	Regular	Ischaemic Heart Disease	300 mg/dl	480 mg/dl	12%	Normal Limits	Normal Limits	Normal Limits	Normal	19		Present	
109	Narayanappa	315513	60 Years	Male	Upper Primary	8 Years	Regular	Hypertension	166 mg/dl	184 mg/dl	7.80%	Normal Limits	Normal Limits	Normal Limits	Normal	22		Present	
110	Chowdamma	278718	65 Years	Female	Uneducated	1.5 Years	Regular	Hypertension	166 mg/dl	230 mg/dl	7%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		
111	Govind Rao	238672	74 Years	Male	Lower Secondary	40 Years	Regular	Hypertension, Seizures	290 mg/dl	310 mg/dl	10.20%	Normal Limits	Normal Limits	Normal Limits	Normal	17			Present
112	Konappa	265144	67 Years	Male	Uneducated	8 Years	Irregular	Nil	166 mg/dl	186 mg/dl	7.80%	Normal Limits	Normal Limits	Normal Limits	Normal	22		Present	
113	Narayanappa	272040	60 Years	Male	Uneducated	17 Years	Regular	TB 20 years ago	154 mg/dl	199 mg/dl	8.80%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	
114	Lakshmi Narayanappa	272713	64 Years	Male	Upper Primary	8 Years	Regular	Seizures	186 mg/dl	224 mg/dl	7.60%	Normal Limits	Normal Limits	Normal Limits	Normal	18		Present	
115	Subbaiah.D	280559	63 Years	Male	Uneducated	12 Years	Regular	Nil	148 mg/dl	200 mg/dl	6.60%	Normal Limits	Normal Limits	Normal Limits	Normal	27	Normal		
116	Chowdappa	304096	80 Years	Male	Uneducated	28 Years	Irregular	Hypertension	250 mg/dl	292 mg/dl	12.60%	Normal Limits	Normal Limits	Normal Limits	Normal	16			Present
117	Gangamma	186151	78 Years	Female	Uneducated	18 Years	Irregular	Nil	220 mg/dl	258 mg/dl	9.40%	Normal Limits	Normal Limits	Normal Limits	Normal	20		Present	
118	Ramanjanappa	301910	65 Years	Male	Upper Primary	10 Years	Regular	Nil	182 mg/dl	200 mg/dl	6.60%	Normal Limits	Normal Limits	Normal Limits	Normal	28	Normal		
119	Munilakshmamma	344725	75 Years	Female	Uneducated	14 Years	Irregular	Nil	258 mg/dl	290 mg/dl	9.80%	Normal Limits	Normal Limits	Normal Limits	Normal	16			Present
120	Sarojamma	152741	60 Years	Female	Lower Secondary	3 Years	Regular	Nil	166 mg/dl	182 mg/dl	7.00%	Normal Limits	Normal Limits	Normal Limits	Normal	22		Present	
121	Uddedulla Khan	294718	60 Years	Male	Uneducated	19 Years	Irregular	Hypertension	222 mg/dl	248 mg/dl	8.80%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
122	Vasantha	333038	60 Years	Female	Lower Secondary	3.5 Years	Regular	Hypertension, Ischaemic Heart Disease	176 mg/dl	222 mg/dl	8.40%	Normal Limits	Normal Limits	Normal Limits	Normal	24	Normal		
123	Sulochanamma	315626	60 Years	Female	Upper Primary	1.5 Years	Regular	Nil	132 mg/dl	157 mg/dl	6.00%	Normal Limits	Normal Limits	Normal Limits	Normal	28	Normal		
124	Muniyappa	293239	67 Years	Male	Uneducated	13 Years	Regular	Nil	190 mg/dl	238 mg/dl	9.40%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	
125	Rukkamma	188719	70 Years	Female	Upper Primary	20 Years	Regular	Hypertension, Ischaemic Heart Disease	260 mg/dl	296 mg/dl	10.60%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	
126	Reddappa Guptha	287270	60 Years	Male	Lower Secondary	7 Years	Regular	Hypertension	133 mg/dl	184 mg/dl	6.20%	Normal Limits	Normal Limits	Normal Limits	Normal	28	Normal		
127	Rose Mary	330494	63 Years	Female	Lower Secondary	22 Years	Regular	HYPERTENSION, TB 5 Years Ago	190 mg/dl	248 mg/dl	7.40%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	

128	Prabha	333376	60 Years	Female	Uneducated	9 Years	Regular	Hypertension	260 mg/dl	316 mg/dl	10.00%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
129	Alamelamma	338493	66 Years	Female	Higher Secondary	18 Years	Irregular	Nil	340 mg/dl	410 mg/dl	11.00%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	
130	Subramani	284629	62 Years	Male	Graduate	11 Years	Irregular	Hypertension, Ischaemic Heart Disease	186 mg/dl	200 mg/dl	8.10%	Normal Limits	Normal Limits	Normal Limits	Normal	17			Present
131	Rajamma	347871	68 Years	Female	Uneducated	3 Months	Regular	Nil	138 mg/dl	157 mg/dl	6.00%	Normal Limits	Normal Limits	Normal Limits	Normal	28	Normal		
132	Venkataramappa	278251	60 Years	Male	Uneducated	1 Year	Regular	Nil	100 mg/dl	152 mg/dl	6.50%	Normal Limits	Normal Limits	Normal Limits	Normal	27	Normal		
133	Gowramma	199978	60 Years	Female	Uneducated	7 Years	Irregular	Hypertension	188 mg/dl	230 mg/dl	7.60%	Normal Limits	Normal Limits	Normal Limits	Normal	19		Present	
134	Lakshmamma	347629	65 Years	Female	Primary	16 Years	Regular	Nil	208 mg/dl	244 mg/dl	7.40%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
135	Pushpa Bai	192951	65 Years	Female	Uneducated	10 Months	Regular	Nil	126 mg/dl	156 mg/dl	6.10%	Normal Limits	Normal Limits	Normal Limits	Normal	28	Normal		
136	Krishnappa Naidu	283177	75 Years	Male	Primary	18 Years	Regular	Hypertension	154 mg/dl	198 mg/dl	7.00%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		
137	Muniswamappa	288664	80 Years	Male	Uneducated	35 Years	Regular	Nil	184 mg/dl	252 mg/dl	12.40%	Normal Limits	Normal Limits	Normal Limits	Normal	15			Present
138	Seetharamappa	297446	60 Years	Male	Upper Primary	7 Years	Regular	Nil	220 mg/dl	294 mg/dl	10.20%	Normal Limits	Normal Limits	Normal Limits	Normal	14			Present
139	Anjaneyallu	300107	65 Years	Male	Uneducated	10 Years	Regular	Hypertension	166 mg/dl	210 mg/dl	7.20%	Normal Limits	Normal Limits	Normal Limits	Normal	24	Normal		
140	Lakshmaiah	302406	75 Years	Male	Lower Secondary	20 Years	Regular	Hypertension	138 mg/dl	154 mg/dl	6.40%	Normal Limits	Normal Limits	Normal Limits	Normal	28	Normal		
141	Muniswamy	303233	85 Years	Male	Upper Primary	35 Years	Regular	Nil	188 mg/dl	230 mg/dl	8.00%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	
142	Sriramappa	310611	66 Years	Male	Graduate	26 Years	Regular	Nil	130 mg/dl	152 mg/dl	6.00%	Normal Limits	Normal Limits	Normal Limits	Normal	30	Normal		
143	Syed Saniulla	310265	65 Years	Male	Upper Primary	10 Years	Regular	Hypertension, Ischaemic Heart Disease	234 mg/dl	310 mg/dl	11.80%	Normal Limits	Normal Limits	Normal Limits	Normal	22		Present	
144	Venkateshappa	317150	67 Years	Male	Uneducated	8 Years	Irregular	Ischaemic Heart Disease	178 mg/dl	240 mg/dl	9.20%	Normal Limits	Normal Limits	Normal Limits	Normal	15			Present
145	Venkataravanappa	325341	70 Years	Male	Uneducated	9 Years	Regular	Hypertension	200 mg/dl	244 mg/dl	7.60%	Normal Limits	Normal Limits	Normal Limits	Normal	30	Normal		
146	Siddappa	331295	66 Years	Male	Uneducated	14 Years	Regular	Nil	164 mg/dl	228 mg/dl	9.60%	Normal Limits	Normal Limits	Normal Limits	Normal	14			Present
147	Venkatamma	192949	70 Years	Female	Uneducated	23 Years	Regular	Hypertension, Ischaemic Heart Disease	189 mg/dl	236 mg/dl	8.40%	Normal Limits	Normal Limits	Normal Limits	Normal	19		Present	
148	Gowramma	201015	73 Years	Female	Primary	20 Years	Regular	Nil	162 mg/dl	188 mg/dl	6.90%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		
149	Saraswathamma	21004	68 Years	Female	Graduate	20 Years	Regular	Ischaemic Heart Disease, Hypertension	174 mg/dl	196 mg/dl	7.80%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
150	Thimmakka	338884	60 Years	Female	Uneducated	15 Years	Regular	Nil	176 mg/dl	222 mg/dl	8.00%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	
151	Venkatamma	193143	70 Years	Female	Upper Primary	17 Years	Irregular	Nil	279 mg/dl	310 mg/dl	11.60%	Normal Limits	Normal Limits	Normal Limits	Normal	17			Present
152	Venkatamma	206097	70 Years	Female	Uneducated	30 Years	Regular	Bronchial Asthma, Hypertension, Ischamic Heart Disease	198 mg/dl	248 mg/dl	9.20%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
153	Byramma	223848	75 Years	Female	Uneducated	5 Years	Regular	Nil	136 mg/dl	174 mg/dl	6.00%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		

154	Chowdamma	340471	80 Years	Female	Upper Primary	30 Years	Regular	Hypertension	400 mg/dl	542 mg/dl	11.40%	Normal Limits	Normal Limits	Normal Limits	Normal	16			Present
155	Chikkamuniyamma	230354	75 Years	Female	Upper Primary	26 Years	Regular	Hypertension	222 mg/dl	289 mg/dl	7.20%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
156	Bibijan	344564	65 Years	Female	Uneducated	2.5 Years	Irregular	Hypertension	130 mg/dl	164 mg/dl	6.20%	Normal Limits	Normal Limits	Normal Limits	Normal	28	Normal		
157	Mala	238968	78 Years	Female	Higher Secondary	11 Years	Regular	Hypertension	147 mg/dl	181 mg/dl	6.60%	Normal Limits	Normal Limits	Normal Limits	Normal	30	Normal		
158	Lakshmamma	284627	70 Years	Female	Uneducated	26 Years	Irregular	Nil	173 mg/dl	224 mg/dl	7.80%	Normal Limits	Normal Limits	Normal Limits	Normal	22		Present	
159	Lakshmamma	215047	70 Years	Female	Primary	16 Years	Regular	Hypertension	134 mg/dl	162 mg/dl	6.30%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		
160	Chowdamma	288990	65 Years	Female	Upper Primary	6 Months	Regular	Hypertension, Ischaemic Heart Disease	236 mg/dl	267 mg/dl	9.40%	Normal Limits	Normal Limits	Normal Limits	Normal	22		Present	
161	Fathima Bee	239936	65 Years	Female	Upper Primary	15 Years	Regular	Nil	196 mg/dl	220 mg/dl	6.80%	Normal Limits	Normal Limits	Normal Limits	Normal	28	Normal		
162	Parvathamma	302775	75 Years	Female	Lower Secondary	25 Years	Irregular	Hypertension	286 mg/dl	340 mg/dl	10.60%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	
163	Ramappa	276736	72 Years	Male	Uneducated	29 Years	Regular	Nil	137 mg/dl	172 mg/dl	6.00%	Normal Limits	Normal Limits	Normal Limits	Normal	30	Normal		
164	Gulabjan	239982	60 Years	Female	Lower Secondary	18 Years	Regular	Ischaemic Heart Disease	286 mg/dl	388 mg/dl	10.40%	Normal Limits	Normal Limits	Normal Limits	Normal	22		Present	
165	Vasanthachar	274256	86 Years	Male	Graduate	38 Years	Regular	Ischaemic Heart Disease	300 mg/dl	384 mg/dl	12.80%	Normal Limits	Normal Limits	Normal Limits	Normal	17			Present
166	Veerabadra Gowda	249628	65 Years	Male	Uneducated	11 Years	Irregular	Hypertension	183 mg/dl	218 mg/dl	7.70%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
167	Jayasheelan	266880	63 Years	Male	Degree	17 Years	Regular	TB 28 years ago	172 mg/dl	188 mg/dl	6.70%	Normal Limits	Normal Limits	Normal Limits	Normal	28	Normal		
168	Venkatamma	242664	60 Years	Female	Uneducated	4 Years	Regular	Nil	178 mg/dl	210 mg/dl	7.00%	Normal Limits	Normal Limits	Normal Limits	Normal	28	Normal		
169	Ramalingappa	203967	66 Years	Male	Uneducated	13 Years	Regular	Nil	262 mg/dl	294 mg/dl	9.40%	Normal Limits	Normal Limits	Normal Limits	Normal	16			Present
170	Muniyappa	264144	80 Years	Male	Upper Primary	38 Years	Regular	Ischaemic Heart Disease, Hypertension,Seizures	297 mg/dl	343 mg/dl	8.00%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
171	Muniswamy	263998	65 Years	Male	Uneducated	7 Years	Irregular	Nil	282 mg/dl	310 mg/dl	9.80%	Normal Limits	Normal Limits	Normal Limits	Normal	19		Present	
172	Venkatamma	250510	61 Years	Female	Primary	17 Years	Regular	Hypertension	246 mg/dl	344 mg/dl	11.60%	Normal Limits	Normal Limits	Normal Limits	Normal	16			Present
173	Venkatashamappa	81492	65 Years	Male	Lower Secondary	20 Years	Regular	Ischaemic Heart Disease, Hypertension	182 mg/dl	236 mg/dl	9.40%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
174	Gopalappa	259444	75 Years	Male	Primary	22 Years	Irregular	Nil	200 mg/dl	234 mg/dl	12.00%	Normal Limits	Normal Limits	Normal Limits	Normal	16			Present
175	Nagappa	259448	70 Years	Male	Higher Secondary	20 Years	Irregular	Hypertension, Ischaemic Heart Disease, TB 30 Years Ago	288 mg/dl	310 mg/dl	10.20%	Normal Limits	Normal Limits	Normal Limits	Normal	17			Present
176	Sitaramaiah.S.T	253956	68 Years	Male	Upper Primary	14 Years	Regular	Nil	220 mg/dl	298 mg/dl	8.10%	Normal Limits	Normal Limits	Normal Limits	Normal	22		Present	
177	Munivenkatappa	247140	75 Years	Male	Upper Primary	35 Years	Regular	Hypertension	140 mg/dl	178 mg/dl	7.60%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
178	Munilakshmamma	93529	68 Years	Female	Lower Secondary	7 Years	Regular	Hypertension	148 mg/dl	200 mg/dl	7.60%	Normal Limits	Normal Limits	Normal Limits	Normal	29	Normal		
179	Narayana.K	217607	64 Years	Male	Graduate	2 Years	Regular	Nil	118 mg/dl	140 mg/dl	6.00%	Normal Limits	Normal Limits	Normal Limits	Normal	28	Normal		

180	Narayanaswamy	241323	60 Years	Male	Higher Secondary	18 Years	Irregular	Ischaemic Heart Disease	250 mg/dl	320 mg/dl	12.60%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
181	Krushidunnissa	260540	70 Years	Female	Uneducated	25 Years	Regular	Hypertension, Ischaemic Heart Disease	286 mg/dl	332 mg/dl	12.60%	Normal Limits	Normal Limits	Normal Limits	Normal	14			Present
182	Gopalappa	239282	62 Years	Male	Upper Primary	1 Year	Regular	Hypertension, Seizures	166 mg/dl	180 mg/dl	6.50%	Normal Limits	Normal Limits	Normal Limits	Normal	30	Normal		
183	Ajaz Pasha	230123	65 Years	Male	Uneducated	4 Years	Regular	Nil	100 mg/dl	170 mg/dl	5.80%	Normal Limits	Normal Limits	Normal Limits	Normal	30	Normal		
184	Rajanna	222951	61 Years	Male	Lower Secondary	6 Years	Regular	TB 10 years ago	172 mg/dl	230 mg/dl	7.00%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	
185	Chowdamma	285771	60 Years	Female	Uneducated	3 Months	Regular	Nil	210 mg/dl	248 mg/dl	7.80%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		
186	Syed Pasha	222126	65 Years	Male	Uneducated	20 Years	Regular	Hypertension	300 mg/dl	378 mg/dl	11.40%	Normal Limits	Normal Limits	Normal Limits	Normal	16			Present
187	Doddamuniyappa	218960	78 Years	Male	Uneducated	32 Years	Irregular	Hypertension	160 mg/dl	214 mg/dl	7.60%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
188	Narasamma	294157	60 Years	Female	Upper Primary	12 Years	Regular	Hypertension	194 mg/dl	222 mg/dl	7.60%	Normal Limits	Normal Limits	Normal Limits	Normal	20		Present	
189	Byramma	299267	60 Years	Female	Uneducated	10 Years	Regular	Nil	220 mg/dl	284 mg/dl	8.00%	Normal Limits	Normal Limits	Normal Limits	Normal	17			Present
190	Anand	214256	62 Years	Male	Graduate	28 Years	Regular	Nil	136 mg/dl	178 mg/dl	6.40%	Normal Limits	Normal Limits	Normal Limits	Normal	30	Normal		
191	Srinivasaiah	212400	60 Years	Male	Higher Secondary	6 Months	Regular	Nil	138 mg/dl	152 mg/dl	6.00%	Normal Limits	Normal Limits	Normal Limits	Normal	30	Normal		
192	Narasamma	293794	70 Years	Female	Uneducated	16 Years	Irregular	Ischaemic Heart Disease, Hypertension	178 mg/dl	192 mg/dl	9.00%	Normal Limits	Normal Limits	Normal Limits	Normal	22		Present	
193	Ramesh	205145	60 Years	Male	Graduate	18 Years	Regular	Nil	140 mg/dl	186 mg/dl	6.90%	Normal Limits	Normal Limits	Normal Limits	Normal	30	Normal		
194	Narayanaswamappa	205153	80 Years	Male	Upper Primary	35 Years	Regular	Ischamic Heart Disease	196 mg/dl	242 mg/dl	8.80%	Normal Limits	Normal Limits	Normal Limits	Normal	20		Present	
195	Muniyamma	306505	65 Years	Female	Upper Primary	18 Years	Regular	Hypertension	226 mg/dl	290 mg/dl	10.00%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	
196	Munishamappa	333287	60 Years	Male	Uneducated	20 Years	Irregular	Hypertension	174 mg/dl	182 mg/dl	7.30%	Normal Limits	Normal Limits	Normal Limits	Normal	26	Normal		
197	Rayappa	201963	75 Years	Male	Uneducated	35 Years	Regular	HYPERTENSION, ISCHAEMIC HEART DISEASE, TB 26 Years Ago	236 mg/dl	298 mg/dl	12.40%	Normal Limits	Normal Limits	Normal Limits	Normal	22		Present	
198	Rathnamma	325643	63 Years	Female	Primary	20 Years	Irregular	Hypertension, Bronchial Asthma	160 mg/dl	174 mg/dl	6.90%	Normal Limits	Normal Limits	Normal Limits	Normal	21		Present	
199	Pillamma	328192	70 Years	Female	Uneducated	32 Years	Irregular	Hypertension, Ischaemic Heart Disease	362 mg/dl	424 mg/dl	12.20%	Normal Limits	Normal Limits	Normal Limits	Normal	14			Present
200	Seethamma	340377	70 Years	Female	Uneducated	22 Years	Regular	Hypertension, Seizures	222 mg/dl	262 mg/dl	10.40%	Normal Limits	Normal Limits	Normal Limits	Normal	23		Present	