“THE RELATIONSHIP BETWEEN GLYCOSYLATED HEMOGLOBIN AND MEAN PLATELET VOLUME IN TYPE 2 DIABETES MELLITUS PATIENTS – A ONE YEAR CROSS SECTIONAL HOSPITAL BASED STUDY AT KLES DR PRABHAKAR KORE HOSPITAL, BELAGAVI.”

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ENDORSEMENT

This is to certify that the dissertation entitled **"THE RELATIONSHIP BETWEEN GLYCOSYLATED HEMOGLOBIN AND MEAN PLATELET VOLUME IN TYPE 2 DIABETES MELLITUS PATIENTS – A ONE YEAR CROSS SECTIONAL HOSPITAL BASED STUDY AT KLES DR. PRABHAKAR KORE HOSPITAL, BELAGAVI."** is a bonafide research work done by **Reg. No. BG0116014.**

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LIST OF ABBREVIATIONS

2-h PG	-	2-h plasma glucose
ADA	-	American Diabetes Association
BMI	-	Body mass index
CI	-	Confidence interval
DCCT	-	Diabetes control and Complications Trial
DKA	-	Diabetic ketoacidosis
DM	-	Diabetes mellitus
e.g.	-	Exmplia gratia (for example)
ECG	-	Electrocardiogram
EDTA	-	Ethylenediaminetetraacetic acid
ESRD	-	End-stage renal disease
FBS	-	Fasting blood sugar
FGT	-	Fasting glucose test
fL	-	Femto litre
FPG	-	Fasting plasma glucose
FSG	-	Fasting serum glucose
g/dL	-	Grams per decilitre
GDM	-	Gstational diabetes mellitus
HbA1c	-	Glycosylated hemoglobin
HDL	-	High density lipoprotein
HDL-C	-	High-density lipoprotein-cholesterol
HNF	-	Hepatocyte nuclear transcription actor
IDF	-	International Diabetes Federation
ie,	-	That is,

IFG	-	Impaired fasting glucose
IGT	-	Impaired glucose tolerance
IPD	-	In patients department
IPF-1	-	Insulin promoter factor-1
kg/m^2	-	Kilograms per square meter
lakh/µL	-	Lakhs per micro litre
LDL	-	Low-density lipoprotein
mg	-	Milligrams
mg/dL	-	Milligrams per deciliter
mmHg	-	Millimeters of mercury
mmol/L	-	Millimole per litre
MODY	-	Maturity onset diabetes of the young
mol/L	-	Mole per liter
MPV	-	Mean platelet volume
NGSP	-	National Glycohemoglobin Standardization Program
NHANES	-	National Health and Nutrition Examination Survey
OGTT	-	Oral glucose tolerance test
OHA	-	Oral hypoglycaemic agents
OPD	-	Out patients department
p	-	Probability value
PAF	-	Paroxysmal atrial fibrillation
PAI-1	-	Plasminogen activator inhibitor
PDW	-	Platelet distribution width
r	-	Pearson correlation coefficient
RBCs	-	Red blood cells

RBS	-	Random blood sugar
SD	-	Standard deviation
SEA	-	South East Asia
SG	-	Study group
SOC	-	Standard of care
T1DM	-	Type 1 diabetes mellitus
T2DM	-	Type 2 diabetes mellitus
U.S.	-	United States
UK	-	United Kingdom
USA	-	United States of America
UTI	-	Urinary tract infection
WHO	-	World Health Organization

ABSTRACT

Background and objectives

Mean Platelet volume (MPV) being a determinant of platelet functionality. Increased MPV levels are associated with increased risk for hyperglycemic complications. The present study was aimed to find the relationship between glycosylated hemoglobin (HbA1c) and MPV in patients with type 2 diabetes mellitus (T2DM).

Methodology

This one year hospital based cross sectional study was conducted in the Department of General Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi from January 2017 to December 2017. A total of 100 patients presenting with T2DM, fulfilling the selection criteria were studied. The selected patients evaluated for HbA1c, MPV, fasting blood sugar (FBS) and other relevant tests.

Results

The male to female ratio was 1:1.04 and 51% of the patients were females. The common age group was 51 to 60 years and 61 to 70 years (30% each). The mean age was 55.83±10.84 years. The mean duration of diabetes was 5.94±5.03 years and most of the patients (45%) reported duration of diabetes between one to five years. Treatment with oral hypoglycaemic agents was reported by 43% of the patients. Majority of the patients (74%) reported good treatment compliance Most of the patients had (61%) microvascular complications and abnormal fundoscopy was noted in 81% of the patients and 55% of the patients had abnormal ECG findings. 48% of

the patients had MPV levels between 7.01 to 9.00 fL and 42% of the patients had MPV levels between 9.01 to 11.00 the mean MPV levels were noted as 9.08±1.20 fL. HbA1c of ≤7.0 percent was noted in 23% of the patients and 77% of the patients had HbA1c levels of > 7.00 percent. The mean HbA1c levels were noted as 9.86±3.02 percent. All the patients with MPV of > 11 had HbA1c levels of > 7 percent and all the patients with MPV of < 7 had HbA1c levels of ≤7 percent. (p=0.002). The mean MPV levels in patients with HbA1c levels of ≤ 7 percent were significantly low compared to those with HbA1c of >7 percent (8.33±1.09 fL vs 9.4±0.92; p<0.001). There was significant weak positive correlation between MPV and HbA1c (*r*=0.287; R^2=0.0823; p=0.004).

Conclusion and interpretation

Based on the findings of this study it may be concluded that, There is strong association and relationship between HbA1c and MPV in patients with type 2 diabetes mellitus.

Keywords

Diabetes mellitus; Glycosylated haemoglobin; Mean platelet volume; Type 2 Diabetes mellitus;

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INTRODUCTION

Diabetes mellitus (DM) refers to a group of common metabolic disorders that share the phenotype of hyperglycemia. It results from defects in insulin secretion, insulin action or both. The effect of diabetes mellitus include long term damage, dysfunction and failure of various organs, eyes, kidneys, nerves and heart and blood vessels. Several distinct types of DM are caused by complex interaction between genetics and environmental factors. Depending on etiology of the DM factors contributing to hyperglycemia include reduced insulin secretion, decreased glucose utilization, and increased glucose production.[1]

Type 2 diabetes mellitus (T2DM) is a metabolic and endocrine disease characterised by hyperglycemia associated with insulin resistance and/or defective insulin secretion.[2] Type 2 Diabetes mellitus accounts for approximately 90-95% of all diagnosed cases of diabetes.[3]

The prevalence of diabetes is rapidly rising all over the globe at an alarming rate.[4] It is important to note that the rise in prevalence is seen in all six inhabited continents of the globe.[5] Over the past 30 years, the status of diabetes has changed from being considered as a mild disorder of the elderly to one of the major causes of morbidity and mortality affecting the youth and middle aged people. Although there is an increase in the prevalence of type 1 diabetes also, the major driver of the epidemic is the more common form of diabetes, namely type 2 diabetes, which accounts for more than 90% of all diabetes cases.[6]

The estimated number of diabetic patients worldwide was 171 million in 2000, which is expected to increase to 366 million by 2030 and the percentage of diabetics

living in developing countries is projected to increase from 74% in 2000 to 81% in 2030.[5,7] Countries such as United Kingdom (UK), United States of America (USA), Mauritius, Fiji, Malaysia, Singapore, South Africa and countries in the Gulf region of the Middle East are home to a large diaspora of Asian Indian individuals, who have been found to have a much higher prevalence of diabetes mellitus than the native populations of the respective countries.[7-9] The countries of the south Asian region, such as Bangladesh, Pakistan, Sri Lanka and Nepal, also have large numbers of individuals with diabetes mellitus.[10-13]

Asian Indian people, which we broadly define as individuals originating from the Indian sub continent (the countries of India, Pakistan, Bangladesh, Sri Lanka, Afghanistan, Nepal, Bhutan and the Maldives) constitute >17% of the world's population and also have a specific phenotype, characterized by high levels of intra-abdominal fat and insulin resistance in spite of a low BMI, which predisposes them to T2DM and premature coronary heart disease.[13,14]

India is one of the epicentres of the global diabetes mellitus epidemic and has the second highest number of people with the disease in the world (~69 million individuals as of 2015).[13,15]

Diabetes mellitus leads to impaired metabolism of carbohydrates, proteins, fats, water and electrolytes. The persistence of these metabolic disturbances lead to permanent and irreversible functional and structural changes in the cells of the body which in turn lead to the development of "diabetic complications", characteristically affecting the cardiovascular system, eye, kidney and nervous system mainly.[16]

Diabetes mellitus is associated with accelerated atherosclerosis leading to coronary, cerebrovascular, retinal and peripheral arterial diseases. Platelet activation

plays a key role in the initiation and progression of atherothrombosis[17,18] and platelet activation and aggregation together play a fundamental role in thrombotic complications associated with type 2 DM.[17,19] Hyperglycemia plays an independent and important role in the prothrombotic state associated with DM.[17,20,21] Induction of hyperglycemia and hyper-insulinemia in healthy subjects without diabetes has been seen to increase platelet reactivity.[17,22] Consistent with this observation, well-controlled DM has been associated with decreased platelet reactivity.[17,23]

Although there are several measurements to show the platelet activity (platelet aggregometry, platelet surface p-selectin, platelet surface-activated glycoprotein IIb/IIIa, platelet function analyzer-100, serum thromboxane B2, and urinary 11-dehydro-thromboxane B2), almost all of these measurements are time-consuming, expensive or they require special training.[24-26] Mean platelet volume (MPV) is an alternative marker for platelet activity. It can be determined on routine automated hemograms as part of the whole blood count, with a relatively low cost.[26-28] Higher MPV values indicate larger platelets, which are metabolically and enzymatically more active, with a greater prothrombotic potential.[28-30]

Increase in MPV has been recognized in patients with the metabolic syndrome, stroke and DM.[17,31] Moreover, studies have shown that increased MPV is one of the risk factors for myocardial infarction, cerebral ischemia and transient ischemic attacks.[17,32] Furthermore, a significant association between poor glycemic control and increased platelet activity in patients with T2DM has been reported.[17]

Hemoglobin A1c (HbA1c), which indicates glucose tolerance and glucose regulation in diabetes, is a marker formed by slow and non-enzymatic glycosylation of hemoglobin. Additionally, it shows glycemic control in people with diabetes and is

closely associated with the risk of developing DM complications.[33,34] Epidemiologic studies in type-2 diabetes have shown that higher glucose levels, as determined by HbA1c are associated with increased risk of diabetic retinopathy, nephropathy, and neuropathy.[33,35] Guidelines suggest a target ≤ HbA1c 6.5% (American Association of Clinical Endocrinologists) or <7.0% (American Diabetes Association) for patients with short diabetes duration, long life expectancy and at low hypoglycemia risk.[33,36,37]

However, the studies reporting association between HbA1c and MPV are limited especially in Indian settings. Hence there is an increased need for finding the relationship between MPV and glycosylated hemoglobin (HbA1c) since some studies have not shown any correlation between the two. This prompted us to evaluate the relationship between HbA1c and mean platelet volume in patients with type 2 diabetes mellitus.

OBJECTIVE

The objectives of this study were to study the relationship between glycosylated hemoglobin and mean platelet volume in patients with type 2 diabetes mellitus.

REVIEW OF LITERATURE

DIABETES MELLITUS

Diabetes mellitus is defined by a group of common metabolic disorders that share the phenotype of hyperglycemia. Various types of diabetes mellitus exist and are caused by a complex interaction of genetics, environmental factors, and different lifestyles. The factors that contribute to hyperglycemia include reduced insulin secretion, decreased glucose utilization, and increased glucose production. The metabolic dysregulation associated with diabetes mellitus causes secondary pathophysiologic changes in multiple organ systems. Diabetes mellitus is the leading cause of end-stage renal disease (ESRD), non-traumatic lower extremity amputation and adult blindness. With an increasing incidence worldwide, diabetes mellitus will be a leading cause of morbidity and mortality in the future.[1]

ETIOLOGIC CLASSIFICATION OF DIABETES MELLITUS[1]

I. Type 1 diabetes (T1DM) (Absolute insulin deficiency resulting from beta cell destruction)
    A. Immune-mediated
    B. Idiopathic

II. Type 2 diabetes (may have predominant insulin resistance with relative insulin deficiency or predominant insulin secretory defect with insulin resistance)

III. Other specific types of diabetes
    A. Genetic defects of beta cell development or function characterized by mutations in:
        1. Hepatocyte nuclear transcription actor (HNF) 4α – M

2. Glucokinase - Maturity onset diabetes of the young (MODY) 2
3. HNF-1α - MODY 3
4. Insulin promoter factor-1 (IPF-1) - MODY 4
5. HNF-1β - MODY 5
6. NeuroD1 - MODY 6
7. Mitochondrial DNA
8. Subunits of ATP-sensitive potassium channel
9. Proinsulin or insulin
10. Other pancreatic islet regulators/proteins such as KLF11, PAX4, BLK, GATA4, GATA6, RFX6, GLIS3, SLC2A2 (GLUT2).

B. Genetic defects in insulin action
   1. Type A insulin resistance
   2. Leprechaunism
   3. Robson-Mendenhall syndrome
   4. Lipodystrophy syndromes

C. Diseases of Exocrine Pancreas—Pancreatitis, pancreatectomy, neoplasia, Fibrocalculous pancreatopathy, cystic fibrosis, hemochromatosis, mutations in carboxyl ester lipase.

D. Endocrinopathies — Acromegaly, Cushing's syndrome, Glucagonoma, Pheochromocytoma, Somatostatinoma, Hyperthyroidism, Aldosteronoma.

E. Drug or chemical induced—Glucocorticoids, vacor (a rodenticide), pentamidine, nicotinic acid, diazoxide, β-adrenergic agonists, thiazides, calcineurin and mTOR inhibitors, hydantoins, asparaginase, α-intereron, protease inhibitors, antipsychotics (atypicals and others), epinephrine

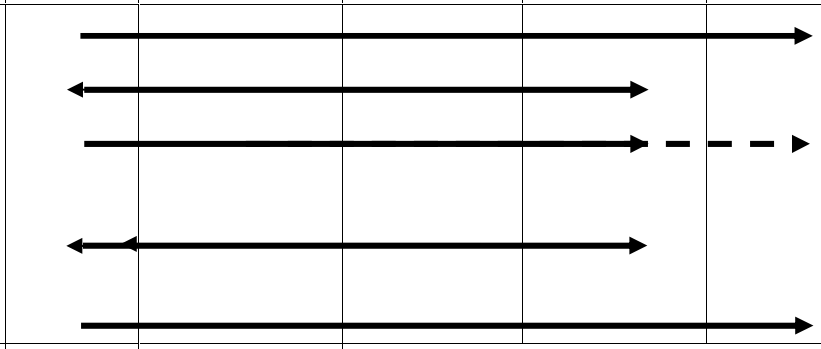
F. Infections—Congenital rubella, cytomegalovirus, coxsackievirus.

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

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

IV. Gestational Diabetes Mellitus

Spectrum of glucose homeostasis and diabetes mellitus[1]

Type of diabetes	**Normal glucose tolerance**	**Hyperglycemia**			
		Pre Diabetes	**Diabetes mellitus**		
		Impaired fasting glucose or impaired glucose tolerance	**Not insulin required**	**Insulin required for control**	**Insulin required for survival**
Type 1 **Type 2** **Other Specific types** **Gestational diabetes** **Time (years)**					
FPG (mg/dL)	< 110	110-125	≥ 126		
2-HPG (mg/dL)	< 140	140 – 199	≥ 200		

Type 1 diabetes (T1DM) and T2DM are heterogeneous diseases in which clinical presentation and disease progression may vary considerably. Classification is important for determining therapy, but some individuals cannot be clearly classified as having type 1 or type 2 diabetes at the time of diagnosis. The traditional paradigms

of type 2 diabetes occurring only in adults and type 1 diabetes only in children are no longer accurate, as both diseases occur in both age-groups. Children with type 1 diabetes typically present with the hallmark symptoms of polyuria/polydipsia, and approximately one-third present with diabetic ketoacidosis (DKA). The onset of type 1 diabetes may be more variable in adults, and they may not present with the classic symptoms seen in children. Occasionally, patients with type 2 diabetes may present with DKA, particularly ethnic minorities. Although difficulties in distinguishing diabetes type may occur in all age-groups at onset, the true diagnosis becomes more obvious over time. In both type 1 and type 2 diabetes, various genetic and environmental factors can result in the progressive loss of b-cell mass and/or function that manifests clinically as hyperglycemia. Once hyperglycemia occurs, patients with all forms of diabetes are at risk for developing the same chronic complications, although rates of progression may differ. The identification of individualized therapies for diabetes in the future will require better characterization of the many paths to b-cell demise or dysfunction. Characterization of the underlying pathophysiology is more developed in type 1 diabetes than in type 2 diabetes. It is now clear from studies of first-degree relatives of patients with type 1 diabetes that the persistent presence of two or more autoantibodies is an almost certain predictor of clinical hyperglycemia and diabetes. The rate of progression is dependent on the age at first detection of antibody, number of antibodies, antibody specificity, and antibody titer. Glucose and A1C levels rise well before the clinical onset of diabetes, making diagnosis feasible well before the onset of DKA. Three distinct stages of type 1 diabetes can be identified and serve as a framework for future research and regulatory decision-making. The paths to b-cell demise and dysfunction are less well defined in type 2 diabetes, but deficient b-cell insulin secretion, frequently in the setting of

insulin resistance, appears to be the common denominator. Characterization of subtypes of this heterogeneous disorder have been developed and validated in Scandinavian and Northern European populations but have not been confirmed in other ethnic and racial groups.

Type 2 diabetes is primarily associated with insulin secretory defects related to inflammation and metabolic stress among other contributors, including genetic factors. Future classification schemes for diabetes will likely focus on the pathophysiology of the underlying b-cell dysfunction and the stage of disease as indicated by glucose status (normal, impaired, or diabetes).[38]

TYPE 2 DIABETES MELLITUS

This form of diabetes, accounts for ~90-95% of those with diabetes, and encompasses those individuals, who have insulin resistance and thus have relative (rather than absolute) insulin deficiency.[1]

There are probably many different causes of this form of diabetes. Specific etiologies are not identified. Most patients with type-2 diabetes mellitus are obese, and obesity itself causes some degree of insulin resistance. This form of diabetes frequently goes undiagnosed for many years, as the hyperglycemia develops gradually. Nevertheless, such patients are at increased risk of developing macrovascular and microvascular complications. The risk of developing type 2 diabetes increases with age, obesity, and lack of physical activity. It occurs more frequently in women with prior GDM (Gestational diabetes mellitus),[39] and individuals with hypertension or dyslipidemia. It is often associated with a strong genetic predisposition, more so than type 1 diabetes mellitus.[40]

Epidemiology

Worldwide

Type 2 diabetes mellitus (T2DM) ranks highly on the international health agenda as a global pandemic and as a threat to human health and global economies.[41] The number of people with T2DM worldwide has more than doubled during the past 20 years.[41,42] According to the International Diabetes Federation, 415 million people are living with T2DM in 2015, and by 2040 the number will be almost 642 million.[15,41] These estimates correspond to a global prevalence of 8.8% (95% confidence interval, 7.2–11.4%) in 2015, and a projected global prevalence of 10.4% (95% confidence interval, 8.5–13.5%) in 2040.[15,41]

The first World Health Organization (WHO) global report on diabetes demonstrates that the number of adults living with diabetes has almost quadrupled since 1980 to 422 million adults.[43,44] Global age-standardized adult diabetes prevalence was 9.8% among men and 9.2% among women in 2008, up from 8.3% and 7.5% in 1980.[43,45] Diabetes has become one of the leading causes of premature illness and deaths in most countries, mainly through the increased risk of cardiovascular disease which is responsible for over 50% of deaths in persons with diabetes.[43,46] Although diabetes is sometimes considered the major concern for developed nations, the loss of life from premature death among persons with diabetes is greatest in developing countries. Nearly 80% of the total adult diabetics are in low- or middle-income countries.[43,46]

Recent data suggests that, Globally, about 1 in 11 adults have diabetes mellitus (90% have type 2 diabetes mellitus (T2DM)), and Asia is the epicentre of this global T2DM epidemic.[47]

According to National Diabetes Statistics Report, 2017 on Estimates of Diabetes and Its Burden in the United States estimated that, 30.3 million people have diabetes that 9.4% of the United States (U.S.) population.[48]

The prevalence of diabetes for all age-groups worldwide was estimated to be 2.8% in 2000 and 4.4% in 2030. There are 382 million people living with diabetes worldwide. The worldwide prevalence of diabetes in adults (aged 20-79 years) was estimated to be 135, 285 million in 1995 and 2010 respectively and is expected 300 million in 2025 and 439 million in 2030.[5,49-55]

Sex predilection

The prevalence of diabetes is higher in men than women.[49-55]

Mortality

In 2012, it resulted in 1.5 million deaths worldwide making it the 8th leading cause of death and more than 80% of diabetic deaths occurring in low and middle-income countries. More than 21 million live births were affected by diabetes during pregnancy and > 79,000 children developed type 1 diabetes in 2013.[49-55]

Indian scenario

According to The International Diabetes Federation (IDF) estimation, India will have increase in people living with diabetes up to 87.0 million by 2030 from 50.8 million (2010), making it the 'Diabetes Capital' of the world.[56-58]

Recent estimates by IDF ranks India as one of the six countries of the IDF South East Asia (SEA) region. India stands at the second position after China, with over 72.946.400 cases of diabetes in India in 2017.[59] With prevalence of 8.8%. The WHO estimated every 26 per 100,000 persons die due to diabetes in India.[60]

Risk factors for Type 2 Diabetes Mellitus[56]

- Family history of diabetes (i.e. Parent or sibling with type 2 diabetes)
- Obesity (BMI≥25 kg/m2)
- Habitual physical inactivity
- Race/ethnicity (e.g. African, American, Hispanic American, Native American, Asian American, Pacific Islander)
- Previously identified impaired fasting glucose (IFG) or impaired glucose tolerance (IGT)
- History of GDM or delivery of baby > 4kg
- Hypertension (Blood pressure ≥ 140/90 mmHg)
- High density lipoprotein (HDL) cholesterol level ≤ 35mg/dL (0.90mmol/L) and / or a triglyceride level ≥ 250mg/dL (2.82 mol/L)
- Polycystic ovary syndrome or Acanthosis nigricans.
- History of vascular disease.

Symptoms

Symptoms are similar in both types of diabetes but vary in their intensity. Symptoms develop more rapidly in type 1 diabetes. The symptoms of diabetes include polyuria, polydipsia, polyphagia, weight loss, fatigue. People with diabetes have an increased risk of developing a number of serious health problems.[56]

Pathophysiology

Hyperglycemia results from lack of endogenous insulin, the deficiency of which, is either absolute, as in type 1 diabetes mellitus, or relative as in type 2 diabetes mellitus. Relative insulin deficiency usually occurs because of resistance to the actions of insulin in muscle, fat, and the liver and an inadequate response by the pancreatic beta cell. Insulin resistance has been attributed to elevated levels of free fatty acids in plasma,[61] and can lead to decreased glucose transport in muscle, elevated hepatic gluconeogenesis, and increased fat breakdown.

Presumably, the defects of type 2 diabetes mellitus occur when a sedentary lifestyle is superimposed on a susceptible genotype. The body mass index at which the risk for diabetes increases varies with different racial groups. For example, compared with persons of European ancestry, persons of Asian ancestry are at increased risk for diabetes at lower levels of being overweight.[62] A simplified scheme for the pathophysiology of abnormal glucose metabolism in type 2 diabetes mellitus is depicted in the image below.

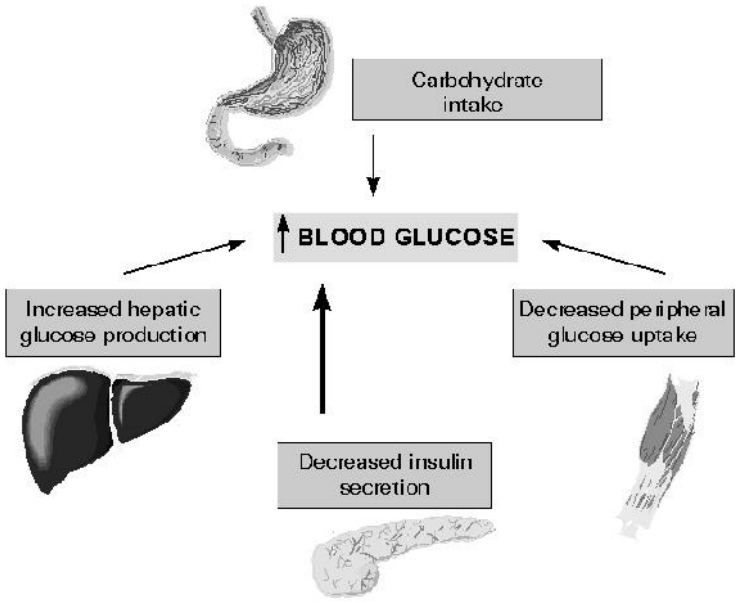


Figure 1. Pathophysiology of type 2 diabetes mellitus[63]

Hyperglycemia is known to be a major determinant of microvascular and metabolic complications. However, glycemia has a lesser effect on the macrovascular complications. Insulin resistance with associated abnormalities in lipid metabolism (i.e., small dense low-density lipoprotein [LDL] particles, low high-density lipoprotein-cholesterol [HDL-C] levels, elevated triglyceride-rich remnant lipoproteins) and prothrombotic states (ie, elevated type-1 plasminogen activator inhibitor [PAI-1], elevated fibrinogen), as well as the conventional atherosclerotic risk factors (e.g., family history, smoking, hypertension, elevated low-density lipoprotein-cholesterol [LDL-C], low HDL-C), determine cardiovascular risk.[63]

Diagnosis[38]

Diabetes may be diagnosed based on plasma glucose criteria, either the fasting plasma glucose (FPG) or the 2-h plasma glucose (2-h PG) value during a 75-g oral glucose tolerance test (OGTT), or A1C criteria as below.

Criteria for the diagnosis of diabetes

- FPG ≥126 mg/dL (7.0 mmol/L). Fasting is defined as no caloric intake for at least 8 h.*

OR

- 2-h PG ≥200 mg/dL (11.1 mmol/L) during OGTT. The test should be performed as described by the WHO, using a glucose load containing the equivalent of 75-g anhydrous glucose dissolved in water.*

OR

- A1C ≥6.5% (48 mmol/mol). The test should be performed in a laboratory using a method that is National Glycohemoglobin Standardization Program (NGSP) certified and standardized to the DCCT assay.*

OR

- In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose more than 200 mg/dL (11.1 mmol/L).

**In the absence of unequivocal hyperglycemia, results should be confirmed by repeat testing.*

Generally, FPG, 2-h PG during 75-g OGTT, and A1C are equally appropriate for diagnostic testing. It should be noted that the tests do not necessarily detect diabetes in the same individuals. The efficacy of interventions for primary prevention of type 2 diabetes has primarily been demonstrated among individuals who have impaired glucose tolerance (IGT) with or without elevated fasting glucose, not for individuals with isolated impaired fasting glucose (IFG) or for those with prediabetes defined by A1C criteria. The same tests may be used to screen for and diagnose diabetes and to detect individuals with prediabetes. Diabetes may be identified anywhere along the spectrum of clinical scenarios: in seemingly low-risk individuals who happen to have glucose testing, in individuals tested based on diabetes risk assessment and in symptomatic patients.

Fasting and 2-Hour Plasma Glucose may be used to diagnose diabetes. The concordance between the FPG and 2-h PG tests is imperfect. Numerous studies have confirmed that compared with FPG and A1C cut points, the 2-h Post Glucose value diagnoses more people with diabetes.[38]

Glycosylated Hemoglobin (HbA1c)

Analysis of glycated hemoglobin (HbA1c) in blood provides evidence about an individual's average blood glucose levels during the previous two to three months, which is the predicted half-life of red blood cells (RBCs). The HbA1c is now recommended as a standard of care (SOC) for testing and monitoring diabetes, specifically the type 2 diabetes.[64]

Historically, HbA1c was first isolated by Huisman et al.[65] in 1958 and characterized by Bookchin and Gallop[66] in 1968, as a glycoprotein. The elevated levels of HbA1c in diabetic patients were reported by Rahbar et al.[67] in 1969. Bunn et al.[68] identified the pathway leading to the formation of HbA1c in 1975. Using the HbA1c as a biomarker for monitoring the levels of glucose among diabetic patients was first proposed by Koenig et al.[69] in 1976.

The ADA has recently recommended HbA1c with a cut-point ≥ 6.5% for diagnosing diabetes as an alternative to fasting plasma glucose (FPG ≥7.0 mmol/L)-based criteria.[70] The levels of HbA1c are strongly correlated with FPG.[71]

FPG, 2-hour post-glucose load plasma glucose, and oral glucose tolerance tests are recommended for the diagnosis of diabetes only if HbA1c testing is not possible due to unavailability of the assay, patient factors that preclude its interpretation, and during pregnancy.[34,72]

HbA1c provides a reliable measure of chronic glycemia and correlates well with the risk of long-term diabetes complications, so that it is currently considered the test of choice for monitoring and chronic management of diabetes. However, the cut-point of HbA1c from the diagnostic point of view is still controversial. Among

diabetics, the blood glucose levels increase in the blood and the glucose attaches to the hemoglobin molecule in a concentration-dependent manner. The glucose-bound (glycated) hemoglobin or HbA1c provides the average glucose levels in an individual's blood as it becomes glycated with the hemoglobin. It is important to note that the HbA1c levels are directly proportional to the blood glucose levels. A simple blood glucose test such as a fasting glucose test (FGT) is a measure of glucose concentration present in an individual's blood at a given point of time.[73]

The FGT is an excellent test for "in the moment" glucose levels, but it does not provide detailed information about the time course trend of the glucose levels. The HbA1c test, however, is a marker of the average glucose levels spread over a two- to three-month period. Contrary to popular belief, along with the type 2 diabetes, the HbA1c is also used to diagnose, manage, and monitor the type 1 diabetes as well.[74]

In a series of 12,785 male diabetic patients, Khan et al.[75] have shown that the HbA1c cut-point of 6.5% was associated with 3.78% false-negative predictions, while majority of the false-negative patients had borderline FPG (7.0–8.0 mmol/L) and HbA1c (6.0%–6.5%), and therefore belonged to at-risk category on the basis of HbA1c alone criteria. These findings suggest that the status of individuals with HbA1c between 6.0% and 6.5% should be verified by combined FPG and HbA1c criteria.

Recently, Khan et al.[76] have provided regression equations for interconversions between the levels of FGT and HbA1c for predicting their expected values in diabetic patients.

The HbA1c levels differ for different diabetes patients, depending on their history of diabetes and whether they are on tablets or long-term and/or short-term

insulin dosage. Type 2 diabetes mellitus (DM) manifests itself in terms of hyperglycemia due to compromised insulin production (no production or unavailability). The significance of the HbA1c test lies in the diagnosis and the prognosis of the diabetes patients, which lends it to a detailed understanding of insulin and insulin resistance. There is a direct correlation between HbA1c and insulin resistance, where HbA1c has been shown to be more strongly associated with the insulin sensitivity in healthy subjects with normal glucose tolerance. The HbA1c test has revealed minimal overlap in values between normal glucose tolerance in subjects with type 2 diabetes while comparing the glycemic spectrum for insulin resistance. As a result, HbA1c is a reliable biomarker and an excellent indicator of insulin resistance for testing individuals for diabetes and prediabetes.[64]

Kwon et al.[77] evaluated the clinical usefulness of HbA1c in diagnosing gestational diabetes mellitus (GDM) and predicting the risk of future type 2 DM development among GDM patients. HbA1c showed high sensitivity with relatively low specificity for diagnosis of GDM in pregnant women but was a potential predictor of postpartum DM. The prognostic value of HbA1c for postpartum DM was evaluated by receiver-operating characteristic curve analysis, with a sensitivity of 78.6% and a specificity of 72.5% at a cut-off value of 5.55%.

HbA1c is not only a useful biomarker of long-term glycemic control but also a good predictor of lipid profile; thus, monitoring of glycemic control using HbA1c could have additional benefits of identifying diabetes patients who are at a greater risk of cardiovascular complications.[64] Thus, a single HbA1c test provides valuable information that can be used for the management of chronic diseases.[64]

In a series of 1,011 type 2 diabetic patients,[71] HbA1c exhibited direct correlations with cholesterol, triglycerides, and low density lipoprotein cholesterol and inverse correlation with high-density lipoprotein cholesterol. There was a linear relationship between HbA1c and dyslipidemia as the levels of serum cholesterol and triglycerides were significantly higher and that of high-density lipo-protein cholesterol were significantly lower in patients with worse glycemic control as compared to patients with good glycemic control.[71]

Recommendations by ADA 2018 for HbA1c[38]

- To avoid misdiagnosis or missed diagnosis, the A1C test should be performed using a method that is certified by the NGSP and standardized to the Diabetes Control and Complications Trial (DCCT) assay.

- Marked discordance between measured A1C and plasma glucose levels should raise the possibility of A1C assay interference due to hemoglobin variants (i.e., hemoglobinopathies) and consideration of using an assay without interference or plasma blood glucose criteria to diagnose diabetes.

- In conditions associated with increased red blood cell turnover, such as sickle cell disease, pregnancy (second and third trimesters), hemodialysis, recent blood loss or transfusion and erythropoietin therapy monitoring of only plasma blood glucose criteria to be used to diagnose diabetes.

The A1C test should be performed using a method that is certified by the NGSP and standardized or traceable to the Diabetes Control and Complications Trial (DCCT) reference assay. Although point-of-care A1C assays may be NGSP certified, proficiency testing is not mandated for performing the test, so use of point-of-care

assays for diagnostic purposes is not recommended but may be considered in the future if proficiency testing is performed, documented, and deemed acceptable. The A1C has several advantages compared with the FPG and OGTT, including greater convenience (fasting not required), greater preanalytical stability, and less day-to-day perturbations during stress and illness. However, these advantages may be limited by the lower sensitivity of A1C at the designated cut point, greater cost, limited availability of A1C testing in certain regions of the developing world, and the imperfect correlation between A1C and average glucose in certain individuals. National Health and Nutrition Examination Survey (NHANES) data indicate that an A1C cut point of 6.5% (48 mmol/mol) identifies a prevalence of undiagnosed diabetes that is one-third of that using glucose criteria. When using A1C to diagnose diabetes, it is important to recognize that A1C is an indirect measure of average blood glucose levels and to take other factors into consideration that may impact hemoglobin glycation independently of glycemia including age, race/ethnicity, and anemia/ hemoglobinopathies.[38]

Age

The epidemiological studies that formed the basis for recommending A1C to diagnose diabetes included only adult populations. Therefore, it remains unclear whether A1C and the same A1C cut point should be used to diagnose diabetes in children and adolescents.[38]

Race/Ethnicity/Hemoglobinopathies

Hemoglobin variants can interfere with the measurement of A1C, although most assays in use in the U.S. are unaffected by the most common variants. Marked discrepancies between measured A1C and plasma glucose levels should prompt

consideration that the A1C assay may not be reliable for that individual. For patients with a hemoglobin variant but normal red blood cell turnover, such as those with the sickle cell trait, an A1C assay without interference from hemoglobin variants should be used. African Americans heterozygous for the common hemoglobin variant HbS may have, for any given level of mean glycemia, lower A1C by about 0.3% than those without the trait. Another genetic variant, X-linked glucose-6-phosphate dehydrogenase G202A, carried by 11% of African Americans, was associated with a decrease in A1C of about 0.8% in hemizygous men and 0.7% in homozygous women compared with those without the variant. Even in the absence of hemoglobin variants, A1C levels may vary with race/ ethnicity independently of glycemia. For example, African Americans may have higher A1C levels than non Hispanic whites with similar fasting and post glucose load glucose levels, and A1C levels may be higher for a given mean glucose concentration when measured with continuous glucose monitoring. Though conflicting data exists, African Americans may also have higher levels of fructosamine and glycated albumin and lower levels of 1,5-anhydroglucitol, suggesting that their glycemic burden (particularly post prandially) may be higher. The association of A1C with risk for complications appears to be similar in African Americans and non-Hispanic whites.[38]

Red Blood Cell Turnover

In conditions associated with increased red blood cell turnover, such as sickle cell disease, pregnancy (second and third trimesters), hemodialysis, recent blood loss or transfusion and erythropoietin therapy, only plasma blood glucose criteria should be used to diagnose diabetes.[38]

Confirming the Diagnosis unless there is a clear clinical diagnosis (e.g., patient in a hyperglycemic crisis or with classic symptoms of hyperglycemia and a random plasma glucose ≥200 mg/dL [11.1 mmol/L]), a second test is required for confirmation. It is recommended that the same test be repeated or a different test be performed without delay using a new blood sample for confirmation. For example, if the A1C is 7.0% (53mmol/mol) and a repeat result is 6.8% (51 mmol/mol), the diagnosis of diabetes is confirmed. If two such tests (such as A1C and FPG) are both above the diagnostic threshold, this also confirms the diagnosis. On the other hand, if a patient has discordant results from two different tests, then the test result that is above the diagnostic cut point should be repeated, with consideration of the possibility of A1C assay interference. The diagnosis is made on the basis of the confirmed test. For example, if a patient meets the diabetes criterion of the A1C (two results 6.5% [48 mmol/mol]) but not FPG (<126mg/dL [7.0 mmol/L]), that person should nevertheless be considered to have diabetes.[38]

Since all the tests have pre-analytic and analytic variability, it is possible that an abnormal result (i.e., above the diagnostic threshold) when repeated, will produce a value below the diagnostic cut point. This scenario is likely for FPG and 2-h PG if the glucose samples remain at room temperature and are not centrifuged promptly. It is pivotal that samples for plasma glucose be spun and separated immediately after they are drawn. If patients results are inconclusive, the healthcare professional should follow the patient closely and repeat the test in 3–6 months.[38]

Complications

Uncontrolled diabetes can lead to an increased risk of heart disease, hypertension, stroke, neuropathy, renal failure, gum disease, blindness, foot and leg

infections, sexual dysfunctions, pregnancy complications. It can also lead to acute biochemical imbalances that cause life-threatening complications, such as diabetes ketoacidosis and hyperosmolar coma.[49,78,79]

***Acute complications*[1]**

- Diabetic ketoacidosis
- Hyperglycemic Hyperosmolar state
- Hypoglycemia

Chronic Complications [80-86]

The chronic complications of DM affect many organ systems and those may be responsible for the majority of morbidity and/or mortality associated with the disease.

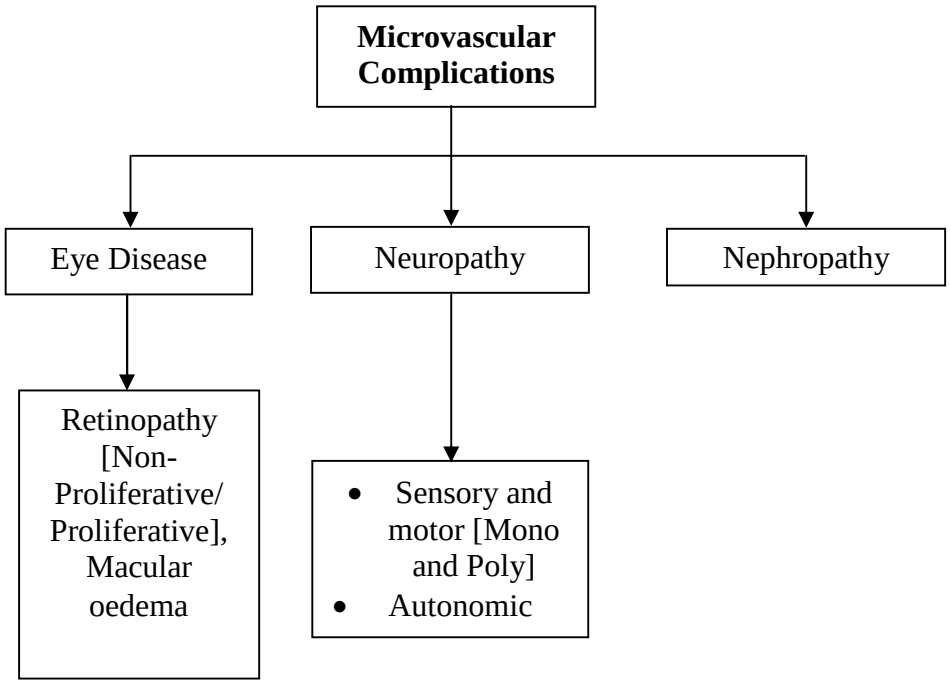


Figure 2. Microvascular complications seen in diabetes mellitus

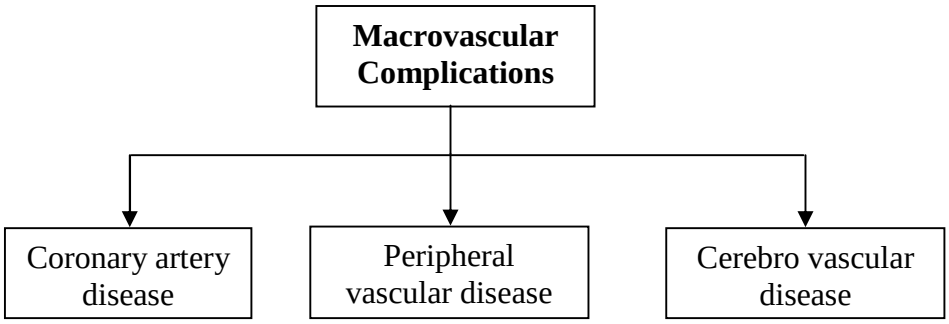


Figure 3. Macrovascular complications seen in diabetes mellitus

Other complications seen in diabetes mellitus:[80-86]

- Gastro-intestinal problems [Gastroparesis, diarrhea]
- Genitor-urinary problems [Uropathy / Sexual dysfunction]
- Dermatologic problems.
- Infections.
    - Urinary tract infection (UTI)
    - Tuberculosis
    - Candidiasis – oral / vulvovaginal
    - Mucormycosis
    - Necrotising fasciitis
    - Periodontitis
- Cataracts and Glaucomas.
- Dupuytren's contracture, Pseudogout

Mean Platelet volume

The key role of Platelet is well known and studied in both physiological and clinical point of view in presumption of numerous aberrances. Moreover, it is the most indispensable and constitutional component of blood whose participation and activity is needed for the normal regulation of human physiology. The building

mechanism of haemostatic plug at the site of injury is a quite interesting phenomenon, executed by these small platelets to stop blood shedding. Considering this event of bleeding arrestment, several works have been put forwarded to demonstrate the role of platelets and platelet related parameters. Platelet count and Mean Platelet Volume (MPV) are found to be the most significant and extraordinary components of blood in assumption of various atypical facets interlinked with these hematological indices. A number of platelet defects have been recognized by many researchers to expose the various atherosclerotic complications arising due to increased platelet activity. Apart from the size and morphology of platelet, the receptors provided on its surface unlocks many mysteries behind the diseases that can be categorized as; immune deficiencies, inborn and acquired anomalies. Hence, it provides information in correlating both the genotypic and phenotypic constitutions of an individual, associated with variable bleeding abnormalities.[87]

Human physiology is crucially driven by the extraordinary role of platelet.[88] These multifunctional anucleated tiny cells are 2 to 4 µm in diameter and 0.5 to 1.0 µm in thickness. In normal healthy conditions, platelet count in peripheral blood ranges from 1.5 lakhs to 4.5 lakhs per microliter. The precursor cells of platelets known as Megakaryocytes, originated from hematopoietic stem cells and undergo successive commitment steps to produce circulating platelets. The inducer in development of Megakaryocyte is thrombopoietin, also called hematopoietic growth factor which drives the production of platelet and act as a primary growth regulator. The arrestment of blood flow at the site of damage and building of haemostatic plug, incorporates a set of different physiological events, performed by these platelets. The procedure of thrombus formation includes adherence of platelet to the damaged endothelium, expansion and degranulation of containments. Degranulation recruits

several other micro particles and procoagulants which enable huge platelet aggregation to stop bleeding. Hence, any malformation or functional break down in platelet conclusively affects hemostasis and maximizes the risk of bleeding. Instead, a pronounced rise in platelet count and activity evokes inappropriate thrombus formation, consequences several atherosclerotic conditions. In platelet related aberrances, another essentially prominent hematologic parameter is the size of the platelet.[87] Mean platelet volume (MPV) elucidates the average dimension of the platelets, ranges from 7.0-11.0 fl in apparently healthy individuals.[89] An elevated value for MPV results into uneventful platelet aggregation, as the surface of platelet is provided with receptors for numerous adhesive molecules. Hence, in determination of various micro and macro vascular complications, this MPV works as an indicator, uncovering wide range of platelet activity.[87]

The etiological perspective behind the quantitative defect in platelet may either be acquired or inheritable. Along with low platelet count, the value for MPV also deviates in different level of hemorrhagic incidents in severe to mild thrombocytopenia. It has already stated that larger platelets display more receptors on their surface and hence turned as more functional and reactive.[87]

A study revealed that, patients affected with severe thrombocytopenia, suffers recurrent hemorrhagic episodes accompanied with a high MPV value.[90] However, this provision is not so intense for those having mild or lower thrombocytopenic conditions. High value for MPV is also discovered and radically perceived in patients with Diabetes mellitus Type 2 and in patients with paroxysmal atrial fibrillation (PAF). Thus it acts as marker in suspecting both these two diseased conditions.

Atypical platelet count and its activity are also influenced by some transmissible disorders.[87]

Platelet count and Mean Platelet volume (MPV) are the utmost remarkable hematological indices, well noticed in a series of clinical evaluations that proportionately influences the bleeding physiology of human body. They also provide knowledge related to both acquired and congenital defects and the mutation patterns that co-associate various other diseases and sometimes coexist heterogenetically in clinical manifestations. Hence, in assessment of various symptomatic and asymptomatic features, platelet indices serve as an extraordinary mean, as well as marker in understanding and diagnosing the abnormality. Even so, this area of hematology is still needed deep researches in various prospects to demonstrate the role of every constituents of platelet responsible for any cause.[87]

Platelets play a key role in the development of atherothrombosis, a major contributor of cardiovascular events.[91] The contribution of platelets to cardiovascular events has been noted for decades. Since then, there have been numerous studies underlying the importance of platelets in thrombotic complications.[91] To further solidify the importance of platelets in cardiovascular disease, medicines aimed at inhibiting platelet activity have been demonstrated to be very effective at decreasing myocardial infarction, stroke, and death.[92,93]

In a landmark study, Davì et al.[94] noted that thromboxane biosynthesis was elevated in subjects with cardiovascular disease who had inadequately controlled diabetes compared with healthy volunteers.

Several studies since then have suggested that diabetic patients have altered platelet morphology and increased platelet activity. However, the importance of

platelet activity in the setting of diabetes was noted in small selected populations, and specialized laboratory techniques were used to measure platelet activity.[93]

Although several measurements of platelet activity have emerged as potential contributors to atherothrombosis, many of these measurements are time-consuming, expensive, use a high sample volume, or require specialty training. Alternatively, mean platelet volume (MPV) is a marker of platelet size that is easily determined on routine automated hemograms and routinely available at a relatively low cost. Subjects with a higher MPV have larger platelets that are metabolically and enzymatically more active and have greater prothrombotic potential than smaller platelets.[93]

DM is characterized by enhanced platelets activation and coagulation proteins and reduced fibrinolytic activity. This pro thrombotic state precede the development of cardiovascular and atherosclerotic complications associated with DM. Type 2 diabetes mellitus patients have two-four folds increase in risk of atherosclerosis.[95]

Luscher et al.[96] also documented an increased risk of coronary artery disease and cerebrovascular disease as a result of accelerated atherosclerosis in DM.

In type 2 DM, platelet function is of pathophysiological importance in atherothrombosis Several authors have documented that increased morbidity and mortality in type 2 DM are associated with macro vascular (cardiovascular diseases, stroke, and peripheral arterial disease) and micro vascular (nephropathy, neuropathy and retinopathy) complications due to platelet dysfunction. Also, an increased platelet counts and activity have been reported in diabetics as demonstrated by increases in GPs IIb/IIIa, 1b-IX, and 1a/IIa, CD62 and CD63 Mean Platelet Volume (MPV), the average volume of platelets, a parameter in full blood count measures platelet size

distribution, and is not influenced by glycemic control. An increased MPV has been associated with high incidence of proliferative diabetic retinopathy and myocardial infarction. An activated megakaryocyte-platelet system in diabetes mellitus has been reported to be responsible for larger than normal platelets circulating in DM patients. Platelet count and MPV are simple, effective and cheap tests that may be used to predict angiopathy in type 2 DM. Elevated MPV has been documented to predict bad outcome for acute ischaemic cerebrovascular events independent of other clinical parameters.[95]

Several clinical studies have shown that patients with diabetes have an altered population of circulating platelets compared with non diabetics. Type 2 DM is caused by a combination of peripheral resistance to insulin action and relative insulin deficiency. Sustained hyperglycemia leads to formation of advanced glycation end products, activation of protein kinase C and disturbance in polyol pathway resulting in damage of endothelium and vascular smooth muscles. This probably explains increased prevalence of diabetic micro vascular complications in individuals with poor glycemic control and longer duration of DM. Blood platelets are highly complex, discoid, anucleate cells derived from bone marrow megakaryocytes that participate in critical reactions central to homeostasis and thrombosis. In response to stimuli generated by the endothelium of blood vessels, platelets change shape, adhere to sub endothelial surfaces, secrete the contents of intracellular organelles, and aggregate to form a thrombus. These pro-aggregatory stimuli include thrombin, collagen, epinephrine, ADP (dense storage granules), and thromboxane A2 (activated platelets). Thus, platelets may assume an important role in signaling of the development of advanced atherosclerosis in diabetes.[97]

Functional and morphological abnormalities of platelets in diabetes mellitus are reported. Large platelets contain more dense granules, metabolically and enzymatically more active than small platelets and show more dense granules producing more procoagulant factors like serotonin β-thromboglobulin and thromboxane A2, thus playing an important role in intracoronary thrombus formation and acute thrombotic events. This suggests a relationship between platelet size and function with diabetic vascular complications and thus changes in MPV reflect the state of thrombogenesis. Mean platelet volume is expected as a possible marker for platelet size and function; hence individuals with higher MPV are more thrombotic. It is also shown that MPV values are high in patients with diabetes mellitus.[97]

In recent decades, an increasing number of evidence showed that platelets were also related to inflammation. Mean platelet volume (MPV), an important morphological parameters of platelets and an easily accessible indice in routine blood test, could reflect the size and activity of platelet. Higher MPV level indicates larger platelets, which are metabolically and enzymatically more active. In recent decade, MPV was defined as an inflammatory marker in different diseases such as fever and inflammatory bowel disease.[98]

Studies showed that DM cases have increased platelet activity. A latest meta-analysis[99] which included 30 case–control and cross-sectional studies found that MPV was significantly higher in T2DM cases than study participants without DM. Nevertheless, other cross-sectional studies[93,100] and a cohort study[101] exploring the relationship between MPV and diabetes obtained inconsistent findings. In the National Health and Nutrition Examination Survey the researchers reported that MPV was strongly and independently associated with the presence and severity of diabetes

in study participants with diabetes.[93] However, the large prospective study to evaluate the impact of diabetes on MPV found no associations between DM and MPV.[101]

Earlier in 1993, Sharp PC and Trinick T.[102] reported a study where they measured mean platelet volume (MPV) in patients with diabetes mellitus, compared with MPV in non-diabetic control subjects. Mean MPV was significantly increased in the diabetic subjects (8.9 ± 0.07 fL, mean ± SEM) compared with nondiabetic subjects (8.0 ± 0.05) ($p < 0.001$). Since platelet size is a determinant of platelet function, with larger platelets being more reactive per unit volume, we believe platelets may play a part in the micro- and macro-vascular complications of diabetes mellitus.

Study conducted by Demirtunc, DD, et al.[103] in 2009 involving 70 patients (Group A with HbA1C > 7% and Group B with HbA1C < or = 7%) and 40 controls showed that MPV was significantly higher in patients with Diabetes Mellitus than in controls. Diabetic patients showed significant positive correlation between MPV and HbA1C levels (r=0.39, P-0.001) but not with diabetic vascular complications.

Kodiatte TA et al.[104] reported a study in 2012 to determine the MPV in diabetics compared to nondiabetics, to see if there is a difference in MPV between diabetics with and without vascular complications, and to determine the correlation of MPV with fasting blood glucose, glycosylated hemoglobin (HbA1c), body-mass index, and duration of diabetes in the diabetic patients. Platelet counts and MPV were measured in 300 Type 2 diabetic patients and 300 nondiabetic subjects using an automated blood cell counter. The blood glucose levels and HbA1c levels were also measured. Statistical evaluation was performed by SPSS using Student's t test and Pearson correlation tests. The mean platelet counts and MPV were higher in diabetics

compared to the nondiabetic subjects [277.46 ± 81 X 109/l vs. 269.79 ± 78 X 109/l (*P*= 0.256)], 8.29 ± 0.74 fl versus 7.47 ± 0.73 fl (*P*= 0.001), respectively. MPV showed a strong positive correlation with fasting blood glucose, postprandial glucose and HbA1C levels (*P*=0.001). This study showed that, showed significantly higher MPV in diabetic patients than in the nondiabetic subjects which indicates that elevated MPV could be either the cause for or due to the effect of the vascular complications. Authors also commented that, platelets may play a role and MPV can be used as a simple parameter to assess the vascular events in diabetes.

Ulutas KT et al.[105] in 2014 reported a study to investigate if platelets were activated in diabetes and its associated vascular complications by measuring the MPV in the diabetics compared to the non-diabetics, and to determine the correlation of MPV with fasting serum glucose (FSG), HbA1c and duration of diabetes in the diabetic patients, respectively. The study carried out in 65 patients with type 2 DM and 40 nondiabetic subjects. In addition to non-diabetic patients, all diabetic patients were divided into two groups according to their HbA1c levels: group A consisted of patients with HbA1c levels ≤7% and group B consisted of patients with HbA1c levels >7%. MPV was significantly higher in Group B as compared to both non-diabetics and Group A. MPV had a high positive correlation with HbA1c and FSG, as with diabetes duration. It is found that MPV was increased in type 2 DM. The study concluded an association between MPV and HbA1c. Further authors recommended that, MPV would be a beneficial prognostic marker of cardio-vascular complications in patients with type 2 DM.

Muhammad I et al.[17] in 2015 reported a study to determine the correlation between glycosylated hemoglobin level and platelet activity among patients with type

2 diabetes mellitus (T2DM). This study was conducted in the Department of Medicine, Khyber Teaching Hospital, Peshawar from January to June 2012. Through a Comparative Cross Sectional Study Design, a total of 80 patients with T2DM were selected from Medical Wards and OPDs and were grouped into those with glycated hemoglobin (HbA1c) levels < 7% (Group A, n=33) and those with HbA1c ≥ 7% (Group B, n=47 patients). Both the groups were compared with regards to MPV and HbA1c. The mean age of patients was 47.41 ± 6.74 years. In group A there were 45.5% (15) males and 54.5% (18) females. In group B there were 48.9% (23) males and 51.1% (24) female patients. MPV was significantly higher in group B as compared to group A (9.21±0.76 fl vs. 8.29±0.46 fl; P < 0.001). Among the group B patients, a positive statistical Pearson's correlation was seen between MPV and HbA1c levels (r = 0.589; p < 0.001). However, no statistical correlation was seen between MPV and the duration of DM and BMI. The authors commented that, there is a significant association between poor glycemic control and increased platelet activity in patients with uncontrolled T2DM.

Kurt H and Demikiran D. in the year 2016 reported a study which examined whether there is a correlation between MPV and Hemoglobin A1c (HbA1c) and fasting plasma glucose (FPG). Authors examined baseline and final HbA1c, MPV and FPG values of 343 patients. Including the study group (SG) consisted of 169 patients with diabetes whose HbA1c levels decreased 1 % and the control group involving 174 patients whose HbA1c levels did not change. Similar to CG, HbA1c level of SG decreased to 7.58, and MPV level reduced to 8.68. There existed a positive correlation between MPV and HbA1c levels (r:154; p<0.005). Additionally, positive correlation was found between MPV and HbA1c changes (r:216; p:0.005), and MPV and FPG changes (r:245; p:0.001) in SG. Moreover, there was a positive correlation between

MPV and HbA1c changes (r:306; p<0.005), and MPV and FPG changes (r:306; p<0.005) in patients underwent insulin treatment. The study concluded that, the change in HbA1c level is similar to MPV level change. The effect of insulin therapy on MPV is significant.[33]

In another Case-control Study reported by Vaddatti, T. et al.[97] in 2016 showed that MPV was significantly higher in diabetics when compared to non diabetics and also showed a statistically significant positive correlation between HbA1c and MPV (r value – 0.5 , p value – 0.4).

Dayal A et al.[106] in 2016 reported a study which involved a total of 211 subjects, 105 diabetics and 106 nondiabetic controls. MPV, platelet count, blood glucose levels, lipid profile and HbA1c levels were measured. Mean MPV was significantly higher in the diabetic group compared to the controls (9.94 ± 1.07 fl versus 9.36 ± 0.96 fl; p=.00003). Mean BMI, cholesterol and triglycerides were also significantly higher in the diabetic group. Within the diabetic group, mean MPV, cholesterol and triglycerides were significantly higher in subjects with HbA1c ≥ 6.5% (10.30±0.95fl vs 9.82±1.08fl, p=.03; 206.28±37.17 mg/dl vs 185.07±41.91 mg/dl, p=.026; 176.96 ± 88.54 mg/dl vs 133.19 ± 46.30 mg/dl, p=.002 respectively). It was concluded that, High MPV in diabetics may indicate platelet hyperactivity, which may contribute to the vascular complications of type II DM. Thus MPV can be used as a simple, cost effective parameter to assess the probability of developing vascular complications in diabetes.

Hasan Z. et al.[107] reported a descriptive and prospective study in the year 2016 to evaluate MPV in patients with type II DM in comparison with a healthy control group and prediabetes group. A total of 77 patients with type II DM, 25 prediabetes

subjects, and 38 healthy subjects attending a teaching hospital of Bangalore constituted the study population. The study subjects were evaluated by performing Complete blood count including MPV, Fasting Blood Glucose levels and Lipid profile. The diabetic subjects were interviewed for duration of disease and examined for presence of microvascular and macrovascular complications apart from noting the HbA1C levels. Mean platelet volume was compared between diabetic patients, prediabetes and healthy counterparts. Within the diabetic group, MPV was compared between this and without vascular complications. The exclusion criteria employed in the study was subjects with anaemia (Hb<11g/dl for females and Hb<12g/dl for males) and thrombocytopenia (<1.5 lakh/μL). MPV of diabetic patients was not significantly different when compared to prediabetes and non-diabetic individuals. Similarly, the MPV had no significant relation to FBS, PPBS, HbA1c and Body mass index. MPV had an inverse relationship with the platelet count. The study showed that MPV of diabetic patients was not significantly different when compared to prediabetes and non-diabetic individuals. Though the MPV in Diabetics with complications was higher than those without complications, it was not statistically significantly.

In a study reported by Swaminathan AL et al.[108] in the year 2017 at the Diabetology Dept of SRM Medical College Hospital and Research Center showed that MPV and Platelet distribution width (PDW) showed a significant increase in Diabetic's with HbA1c > 7% and MPV was increased in Diabetics with > 10 years duration of Diabetes.

METHODOLOGY

The present study was undertaken in the Department of General Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi from January 2017 to December 2017.

Study design

The study design was a one year hospital based cross sectional study.

Study period and duration

The present was done for a period of one year from January 2017 to December 2017.

Setting

The present study was carried out in the Department of General Medicine KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi a tertiary care teaching hospital attached to KLE University's Jawaharlal Nehru Medical College, Belagavi.

Source of Data

Patients presenting with type 2 diabetes mellitus to the Department of General Medicine KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi were enrolled.

Sample size

A total of 100 patients presenting with type 2 diabetes mellitus fulfilling the selection criteria were selected for the study.

Sampling procedure

The sample size was calculated based on the formula as mentioned below.

$n = 4 \times p \times q / d^2$

Where, p = Prevalence of the disease was considered as 50%

due to scarcity of data on role of MPV in T2DM

q = $100 - p$ =100-50 = 50

d = Absolute error taken as 10%

Therefore, $n = 4 \times 50 \times 50 / 10^2$

n = 100

Hence a sample size of 100 was considered for the study.

Sampling method

Patients fulfilling the inclusion criteria were enrolled considering simple random sampling.

Selection criteria

Inclusion Criteria

- Patients presenting with type 2 diabetes mellitus to the Department of General Medicine at KLES, Dr Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

Exclusion Criteria

- Male patients with Hb < 13g/dL and Females with Hb < 11g/dL.
- Patients with history of anti-thrombotic medications.
- Patients with history of coagulation disorders, peripheral vascular disease, and those having habits of smoking, tobacco chewing and alcohol intake.

Ethical clearance

Prior to the commencement, the study was approved by the Institutional Ethics Committee of Jawaharlal Nehru Medical College, Belagavi.

Informed consent

Patients presenting to the Department of General Medicine at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. were screened for eligibility based on selection criteria. Those who fulfill the selection criteria were briefed about the nature of the study and a written informed consent was obtained (Annexure–I) prior to the enrollment.

Data collection

Patients were interviewed and demographic data like gender and age were noted. Detailed diabetic history like duration of diabetes, medication, treatment

compliance along with history of other comorbid conditions and personal history were obtained and recorded. A thorough general physical examination was conducted to assess vital parameters followed by systemic examination. All these findings were recorded on a predesigned and pretested proforma (Annexure-II).

Investigations

The selected patients were subjected to the following investigations

- Haemoglobin
- Fasting blood sugar
- Glycosylated hemoglobin (HbA1C)
- Mean platelet volume (MPV)
- Serum Urea and creatinine
- Electrocardiogram (ECG)

Under aseptic conditions, 2 mL of venous blood was collected in EDTA vials for complete blood counts and HbA1c. Early morning samples were drawn for estimating fasting blood glucose levels. Samples were maintained at room temperature and analyzed within one hour of collection.

Study variables

Glycosylated hemoglobin (HbA1c)

HbA1c was calculated by high performance liquid chromatography. Based on ADA 2018 recommended target for HbA1c, a value of ≤7 was considered as optimal.[38,109]

Mean platelet volume

MPV and platelet counts were analyzed in fully automated hematology analyzer. The mean platelet volume was estimated on Beckman and Coulter LH 780 hematology analyzer using electrical impedance method[110] and normal reference range was considered between 7 to 11fL.[111]

Fasting blood sugar

The FBS was analyzed in samples collected in sodium fluoride. FBS was estimated by hexokinase method.

Statistical analysis

The data thus obtained was tabulated on Microsoft Excel spread sheet. The categorical data was expressed as ratios and percentages. Chi-square test was used to find the association. Continuous data was expressed as mean ± standard deviation (SD) and then the comparison was done using independent sample‘t’ test. Pearson’s correlation coefficient was used to determine correlation between MPV and glycosylated hemoglobin, fasting blood sugar and duration of diabetes. At 95% confidence interval (CI), a probability value (‘p’ value) of less than or equal to 0.050 was considered to be statistically significant.

RESULTS

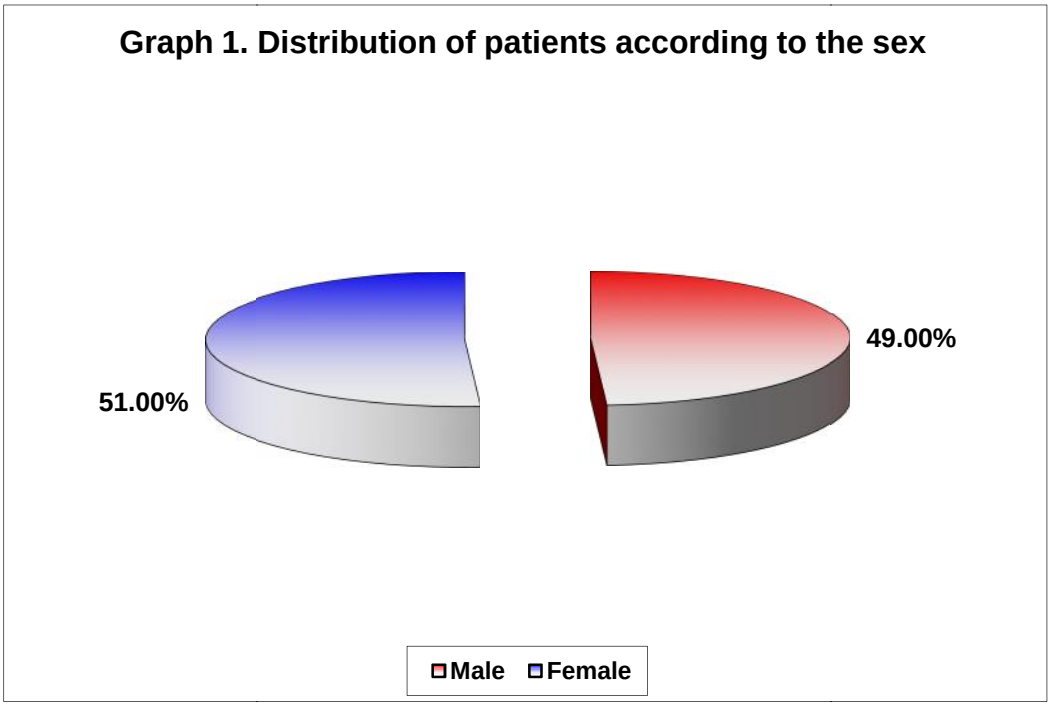
This one year hospital based cross sectional study was conducted in the Department of General Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi from January 2017 to December 2017. A total of 100 patients presenting with type 2 diabetes mellitus fulfilling the selection criteria were studied. The selected patients underwent tests for HbA1c, MPV and FBS

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

Table 1. Distribution of patients according to the sex

Sex	Distribution (n=100)	
	Number	Percentage
Male	49	49.00
Female	51	51.00
Total	**100**	**100.00**

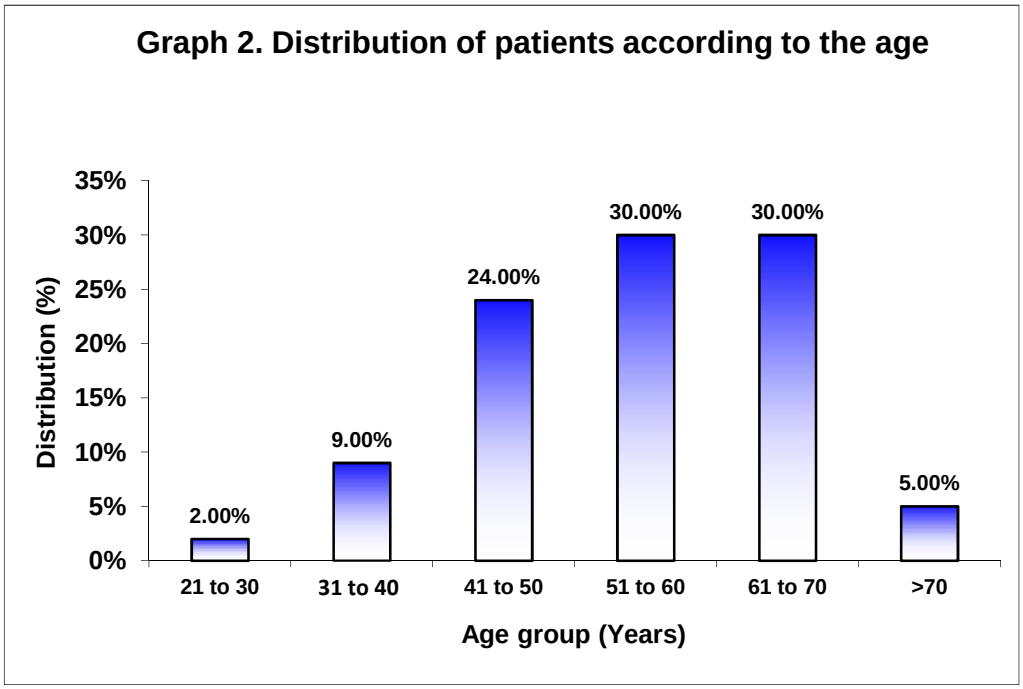
Graph 1. Distribution of patients according to the sex



In the present study 51% of the patients were females and 49% of the patients were males (49%). The male to female ratio was 1:1.04

Table 2. Distribution of patients according to the age

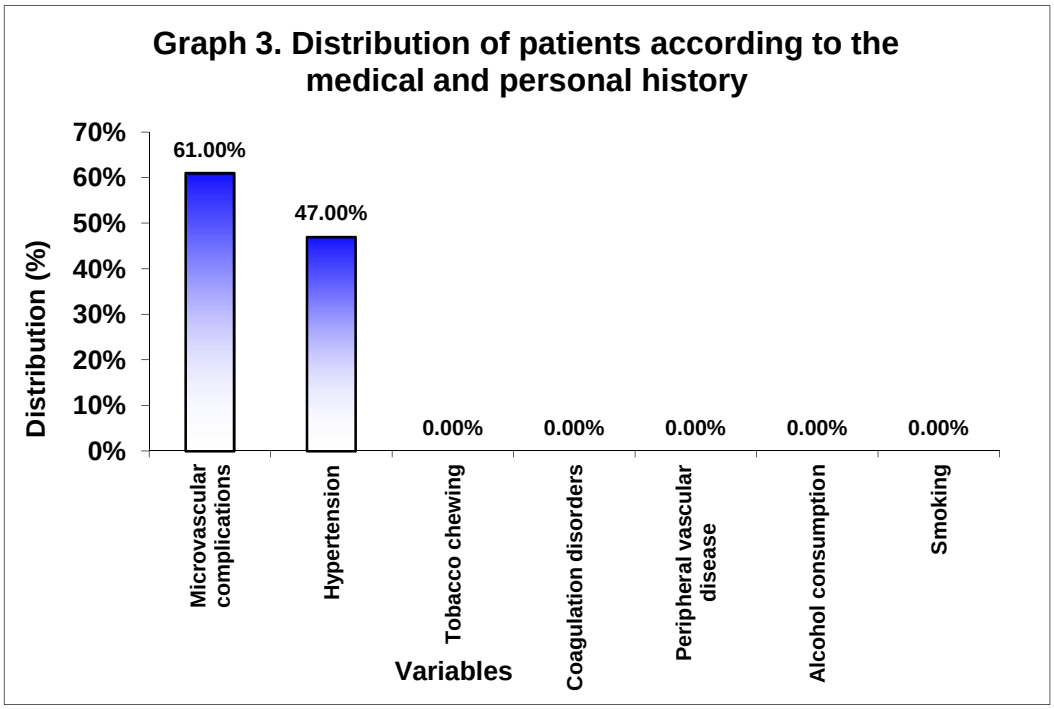
Age group (Years)	Distribution (n=100)	
	Number	Percentage
21 to 30	2	2.00
31 to 40	9	9.00
41 to 50	24	24.00
51 to 60	30	30.00
61 to 70	30	30.00
> 70	5	5.00
Total	**100**	**100.00**



In this study 30% of the patients each were aged between 51 to 60 years and 61 to 70 years. The mean age was 55.83±10.84 years and the median age was 57.50 years and ranged between 29 to 74 years.

Table 3. Distribution of patients according to the medical and personal history

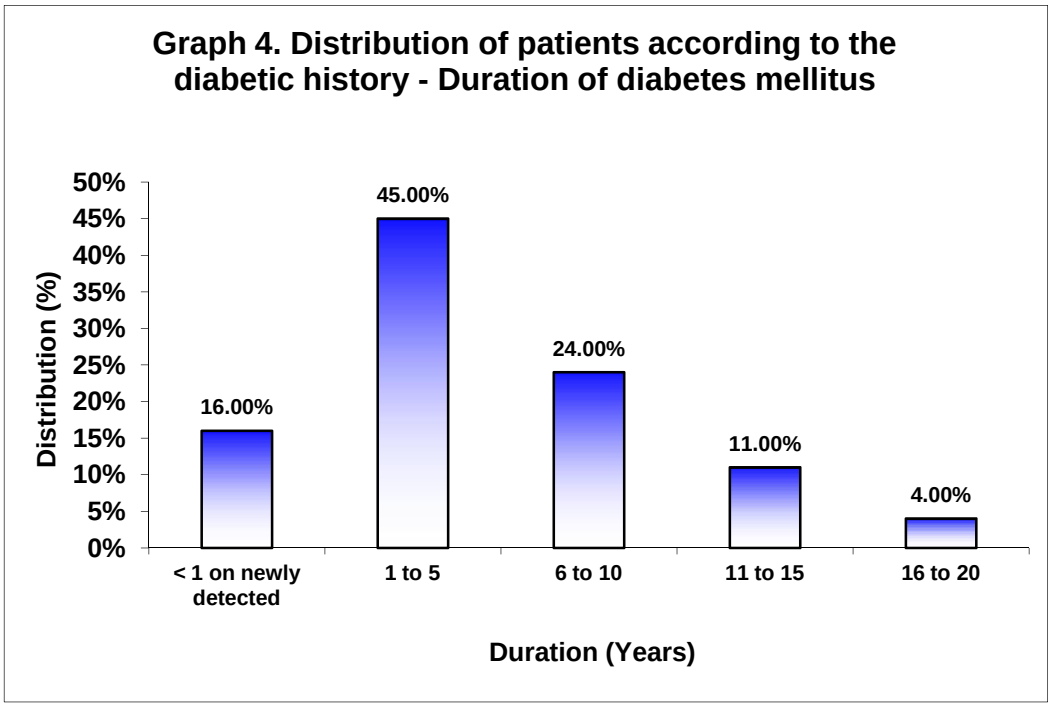
Variables	Distribution (n=100)	
	Number	Percentage
Microvascular complications	61	61.00
Hypertension	47	47.00
Tobacco chewing	0	0.00
Coagulation disorders	0	0.00
Peripheral vascular disease	0	0.00
Alcohol consumption	0	0.00
Smoking	0	0.00



In the present study 61% of the patients had microvascular complications and 47% of the patients reported history of hypertension.

Table 4. Distribution of patients according to the diabetic history - Duration of diabetes mellitus

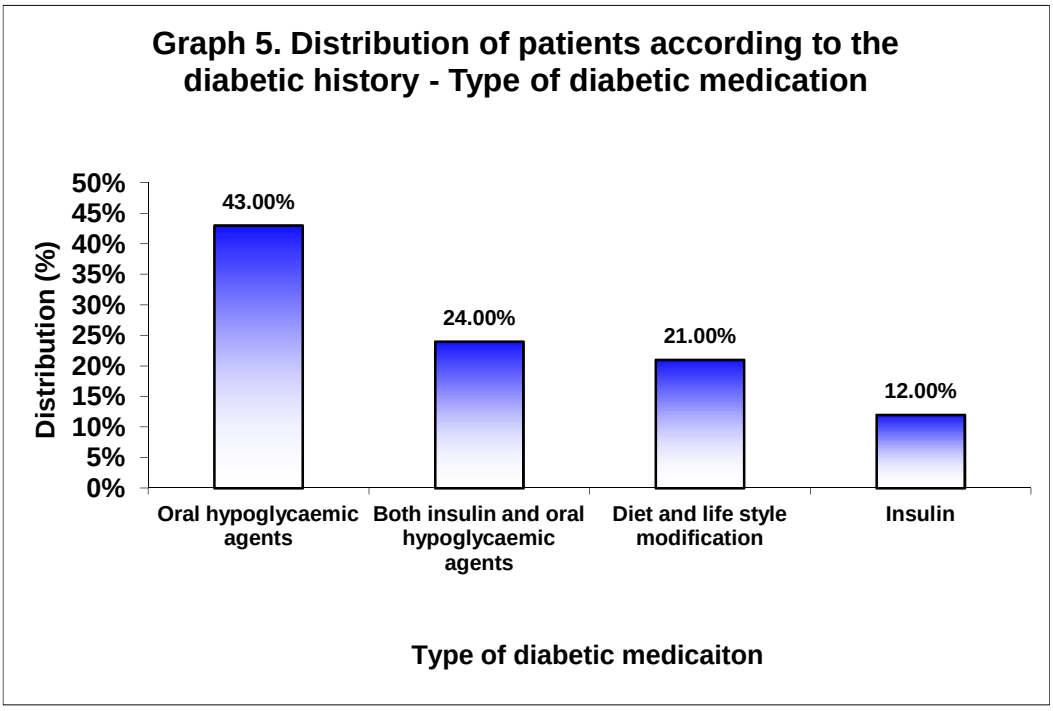
Duration of diabetes	Distribution (n=100)	
	Number	Percentage
< 1 year or Newly detected	16	16.00
1 to 5 years	45	45.00
6 to 10 years	24	24.00
11 to 15 years	11	11.00
16 to 20 years	4	4.00
Total	**100**	**100.00**



In this study 45% of the patients reported duration of diabetes between one to five years and 24% had duration between 6 to 10 years. However 16% of the patients were newly diagnosed and 4% of the patients had duration of 16 to 20 years. The mean duration of diabetes was noted as 5.94±5.03 years and the median duration of diabetes was noted as 5 years and ranged up to 20 years.

Table 5. Distribution of patients according to the diabetic history - Type of diabetic medication

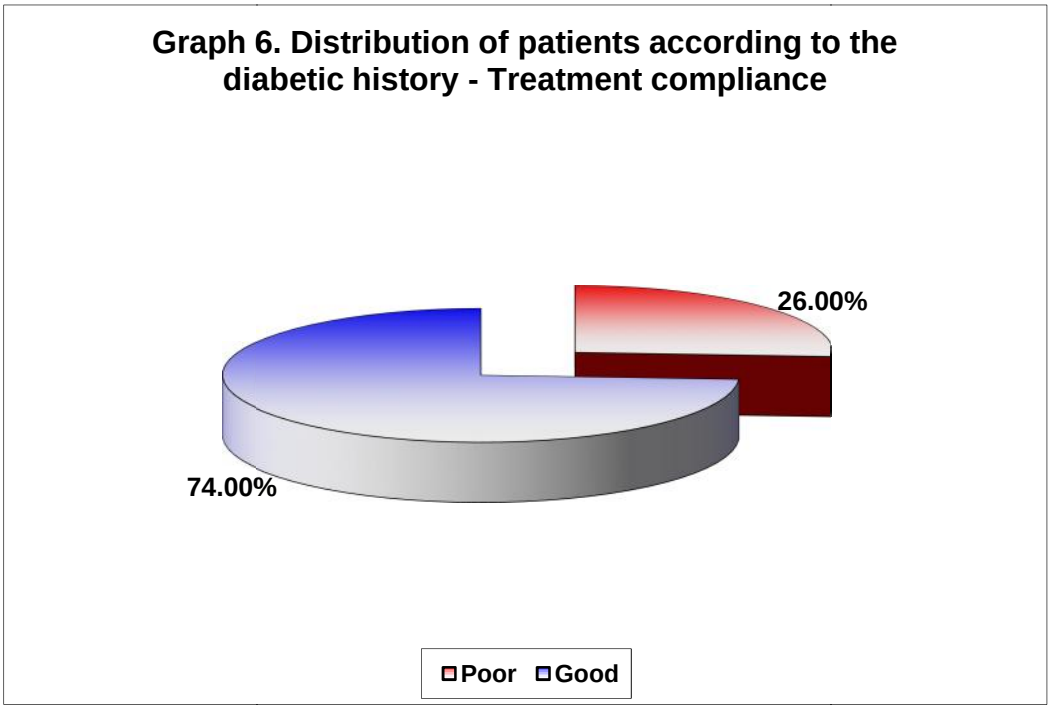
Type of Diabetic Medication	Distribution (n=100)	
	Number	Percentage
Oral Hypoglycaemic agents	43	43.00
Both Insulin and oral hypoglycaemic agents	24	24.00
Diet and life style modification	21	21.00
Insulin	12	12.00
Total	**100**	**100.00**



In the present study 43% of the patients were on treatment with oral hypoglycaemic agents and 24% were on both insulin and OHA while 21% of the patients reported diet and life style modification and 12% were on treatment with insulin.

Table 6. Distribution of patients according to the diabetic history - Treatment compliance

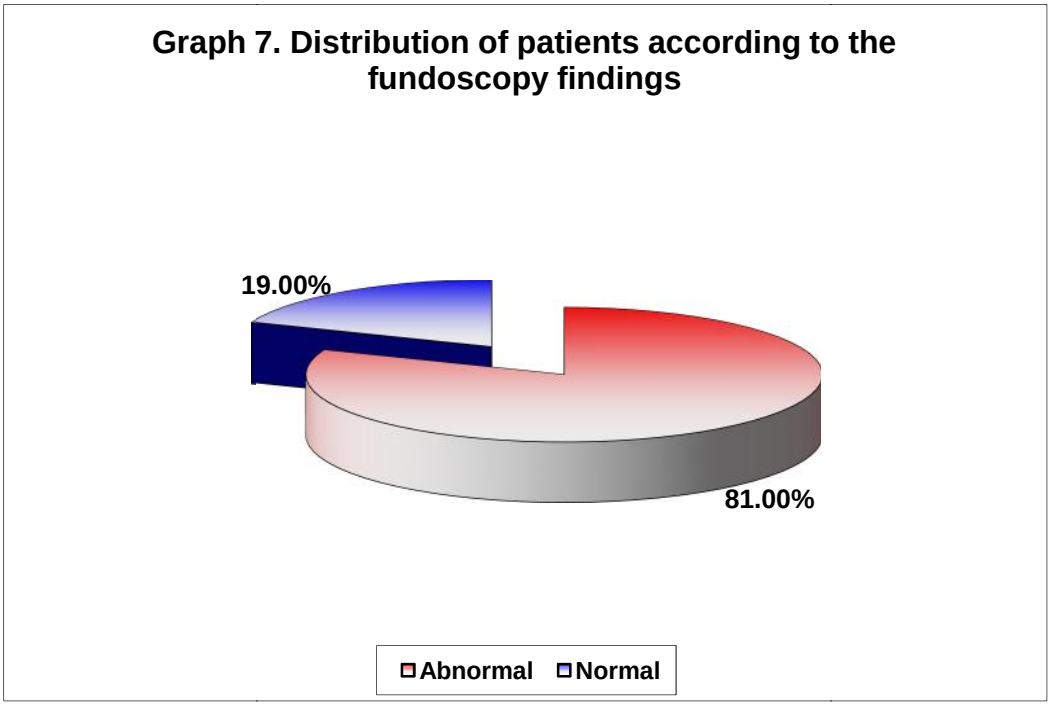
Treatment compliance	Distribution (n=100)	
	Number	**Percentage**
Good	74	74.00
Poor	26	26.00
Total	**100**	**100.00**



In the present study 74% of the patients reported good treatment compliance while poor treatment compliance was noted in 26% of the patients.

Table 7. Distribution of patients according to the fundoscopy findings

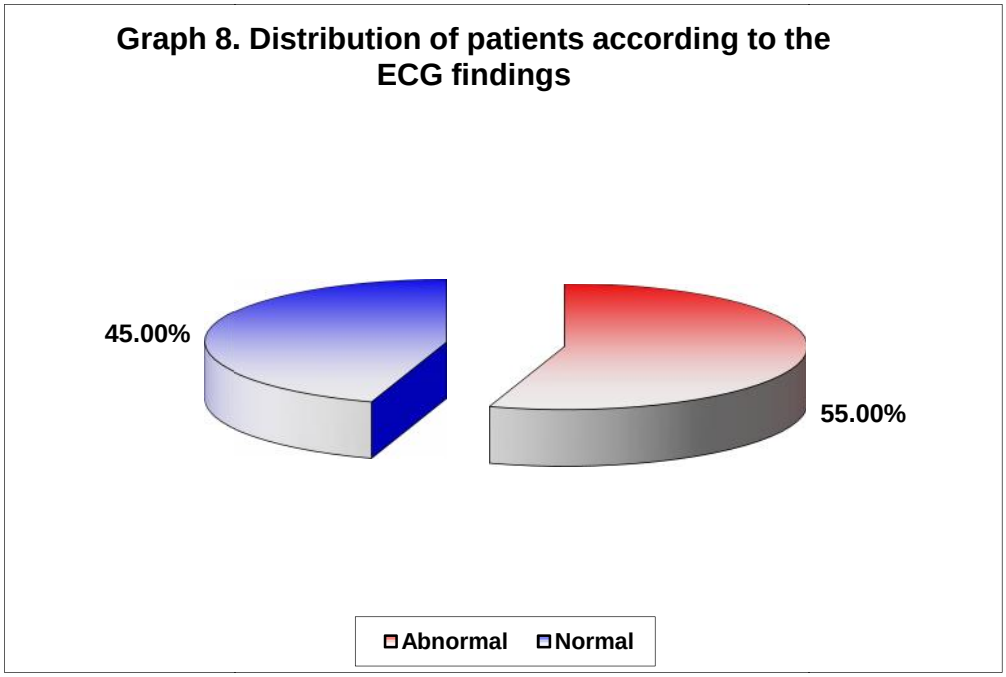
Fundoscopy findings	Distribution (n=100)	
	Number	**Percentage**
Normal	19	19.00
Abnormal	81	81.00
Total	**100**	**100.00**



In the present study abnormal fundoscopy was noted in 81% of the patients while 19% of the patients had a normal fundus exam.

Table 8. Distribution of patients according to the ECG findings

ECG findings	Distribution (n=100)	
	Number	Percentage
Normal	45	45.00
Abnormal	55	55.00
Total	**100**	**100.00**



In this study 55% of the patients had abnormal ECG findings.

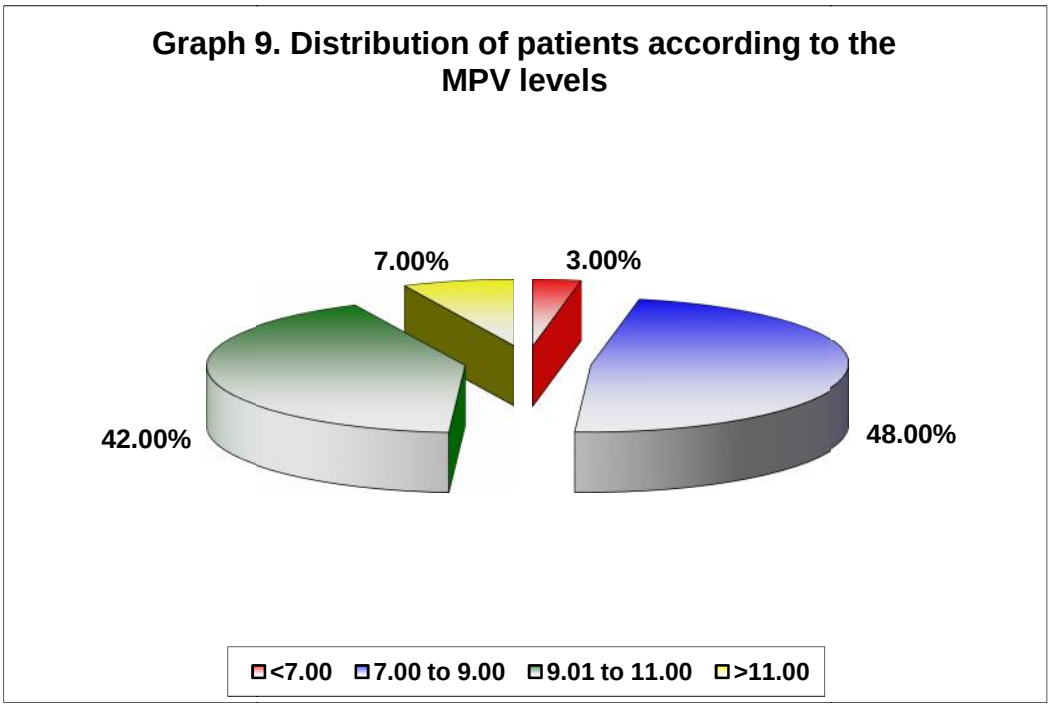
Table 9. Clinical profile of the study population

Parameters	Mean (n=100)		Median	Range	
	Mean	SD		Minimum	Maximum
Age (Years)	55.83	10.84	57.50	29.00	74.00
Duration of Diabetes (Years)	5.94	5.03	5.00	0.00	20.00
Pulse Rate (bpm)	81.45	11.06	80.00	54.00	114.00
Systolic blood pressure (mm Hg)	126.50	21.70	123.00	86.00	180.00
Diastolic blood pressure (mm Hg)	79.10	9.99	80.00	60.00	100.00
Respiratory rate (Per minute)	17.11	2.20	18.00	14.00	24.00
Mean platelet volume (fL)	9.08	1.20	9.00	5.60	12.00
Mean haemoglobin in males (gm%)	13.68	0.45	13.70	13.00	14.60
Mean haemoglobin in females (gm%)	11.88	0.49	11.90	11.00	12.90
HbA1C (%)	9.86	3.02	9.75	4.80	19.90
Urea (mg/dl)	39.92	30.71	28.00	10.00	201.00
Creatinine (mg/dl)	1.43	0.89	1.10	0.37	5.00
Fasting blood sugar (mg/dL)	139.09	32.05	126.50	94.00	250.00

The clinical and diabetic profile of the study population is as shown in Table 9.

Table 10. Distribution of patients according to the MPV levels

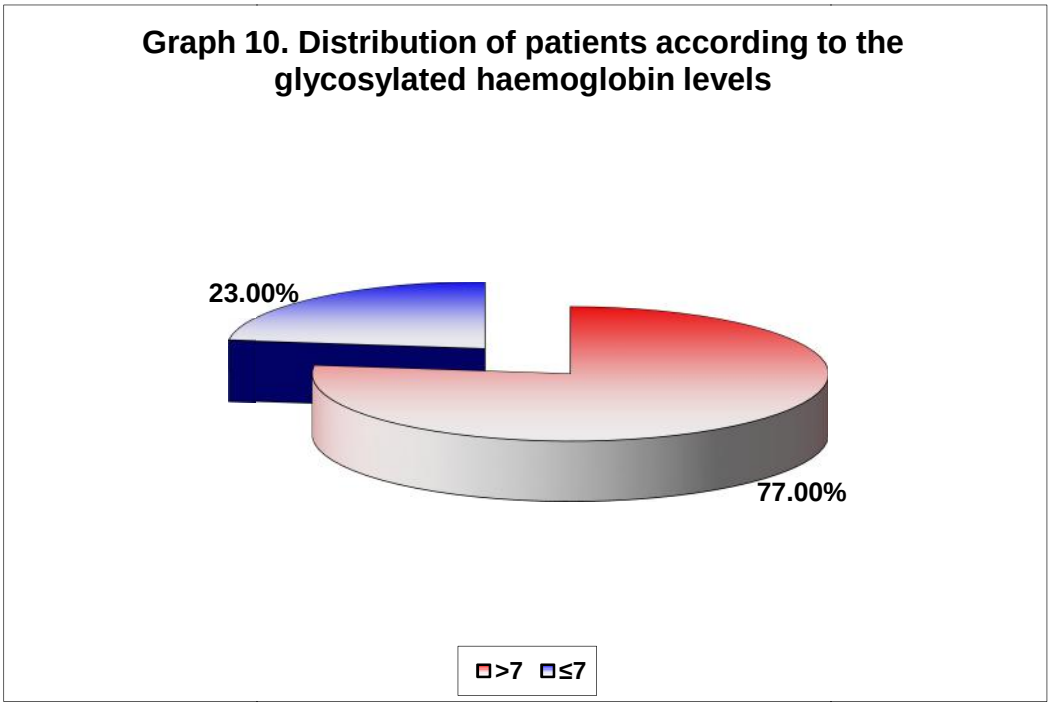
MPV levels (fL)	Distribution (n=100)	
	Number	Percentage
<7.00	3	3.00
7.00 to 9.00	48	48.00
9.01 to 11.00	42	42.00
>11.00	7	7.00
Total	**100**	**100.00**



In the present study 48% of the patients had MPV levels between 7.01 to 9.00 fL and 42% of the patients had MPV levels between 9.01 to 11.00 the mean MPV levels were noted as 9.08±1.20 fL and Median MPV levels were 9.00 fL and ranged between 5.6 to 12.00 fL.

Table 11. Distribution of patients according to the Glycosylated Hemoglobin levels

Glycosylated Hemoglobin levels (%)	Distribution (n=100)	
	Number	**Percentage**
≤7	23	23.00
> 7	77	77.00
Total	**100**	**100.00**

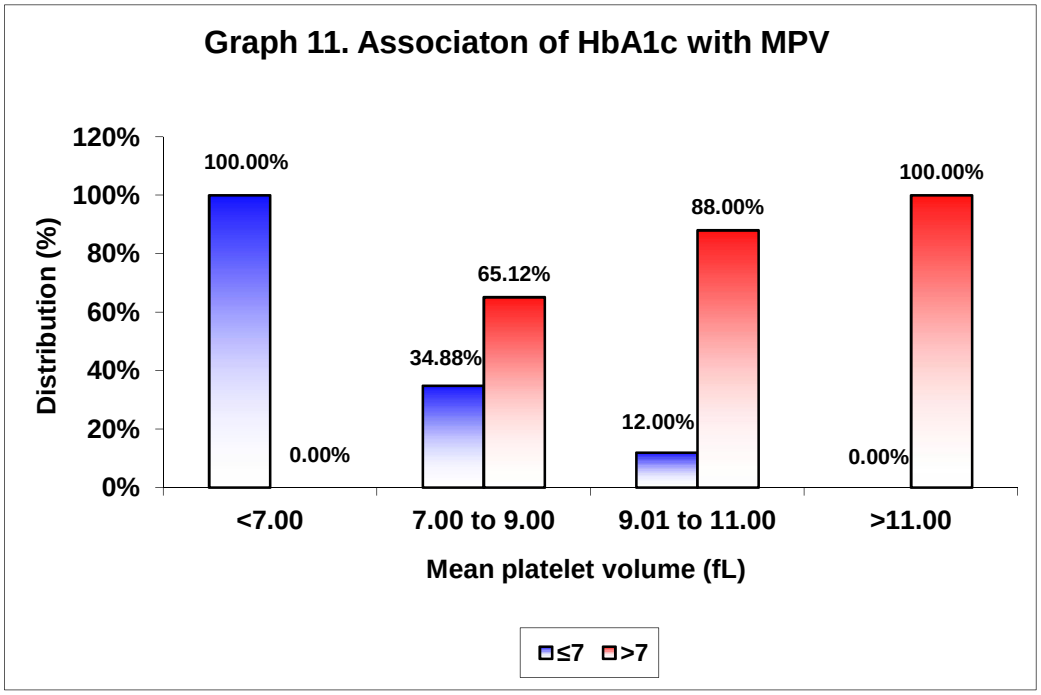


In the present study HbA1c of ≤ 7.0 percent was noted in 23% of the patients and 77% of the patients had HbA1c levels of > 7.00 percent. The mean HbA1c levels were noted as 9.86±3.02 percent and median HbA1c levels were 9.75 percent and ranged between 4.8 to 19.90 percent.

Table 12. Association of HbA1C with MPV

Mean platelet volume (fL)	HbA1c (%) ≤7		HbA1c (%) >7		Total (n=100)	
	No.	%	No.	%	No.	%
<7.00	2	100.00	0	0.00	2	2.00
7.00 to 9.00	15	34.88	28	65.12	43	43.00
9.01 to 11.00	6	12.00	44	88.00	50	50.00
>11.00	0	0.00	5	100.00	5	5.00
Total	**23**	**23.00**	**77**	**77.00**	**100**	**100.00**

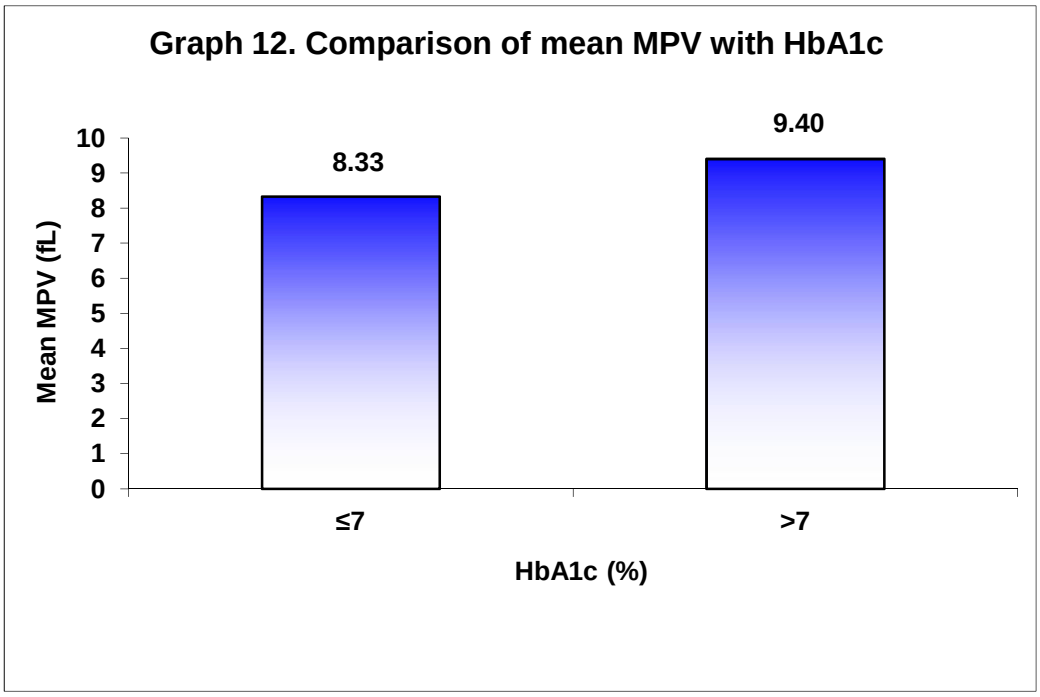
p = 0.002



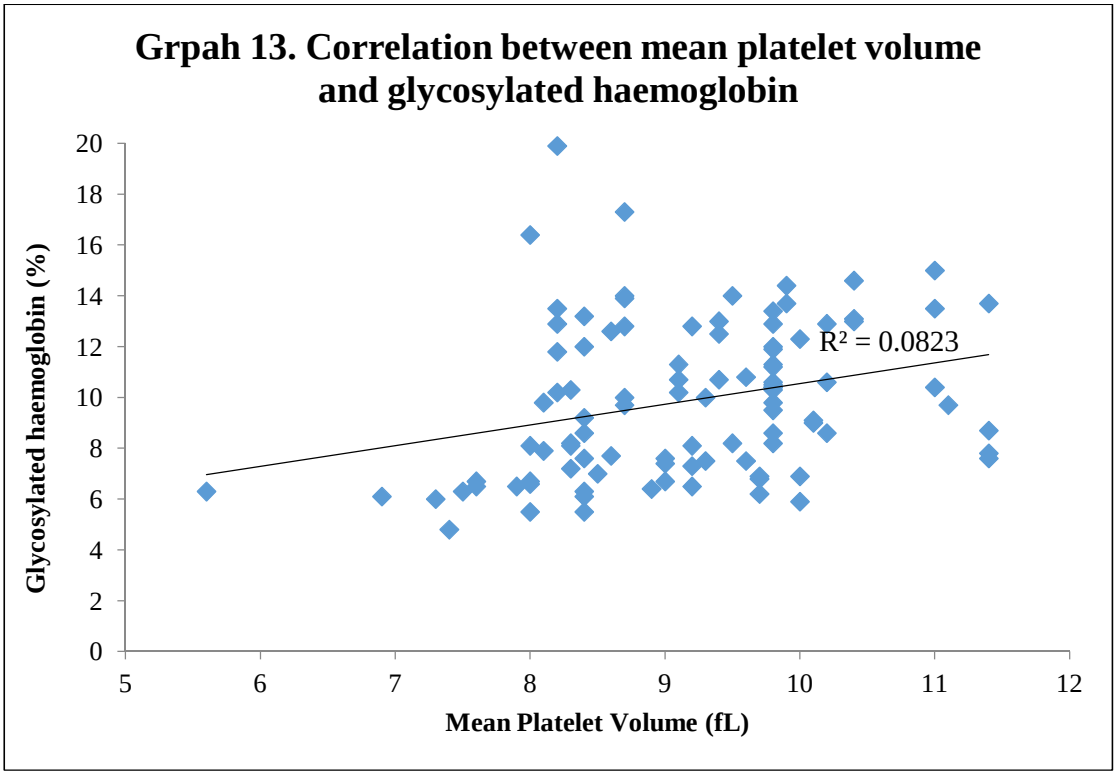
In this study all the patients with MPV of > 11 had HbA1c levels of > 7 percent and all the patients with MPV of < 7 had HbA1c levels of ≤ 7 percent. This difference was statistically significant (p=0.002).

Table 13. Comparison of Mean MPV with HbA1c

	Mean HbA1c (%)				
	≤7 (n=23)		>7 (n=77)		p value
	Mean	SD	Mean	SD	
MPV (fL)	8.33	1.09	9.4	0.92	<**0.001**

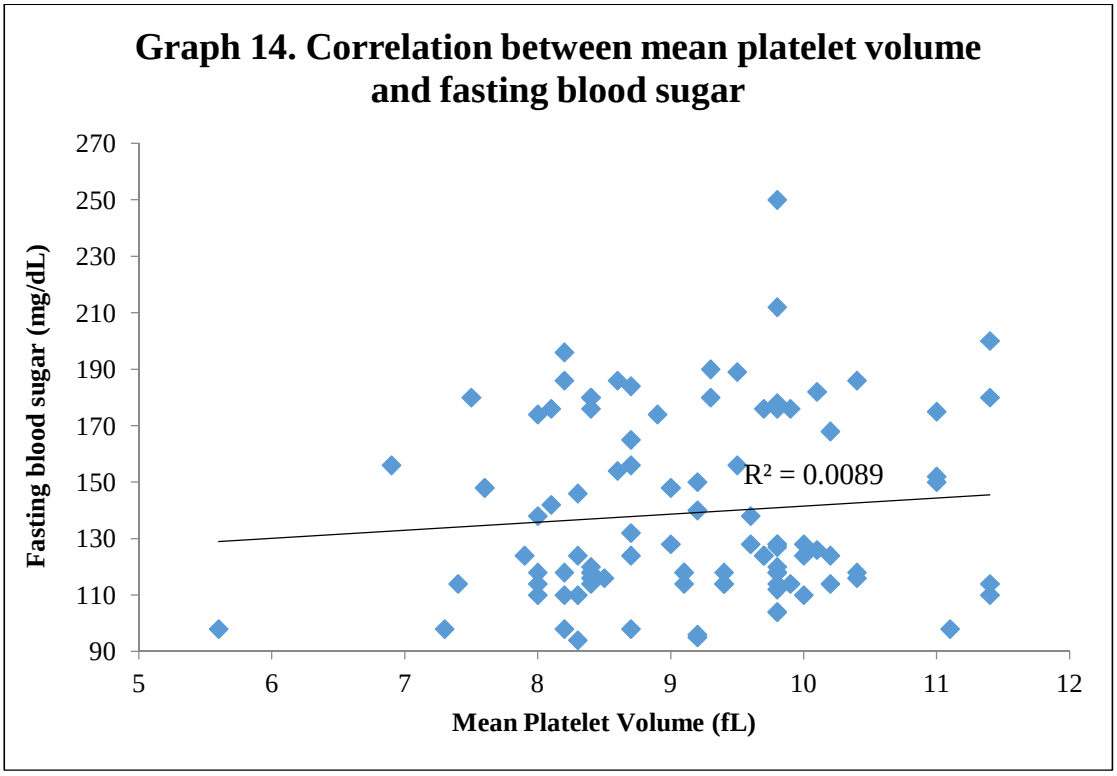


In the present study the mean MPV levels in patients with HbA1c levels of ≤ 7% were significantly low (8.33±1.09 fL) compared to those with HbA1c of > 7 (9.4±0.92) ($p<0.001$).



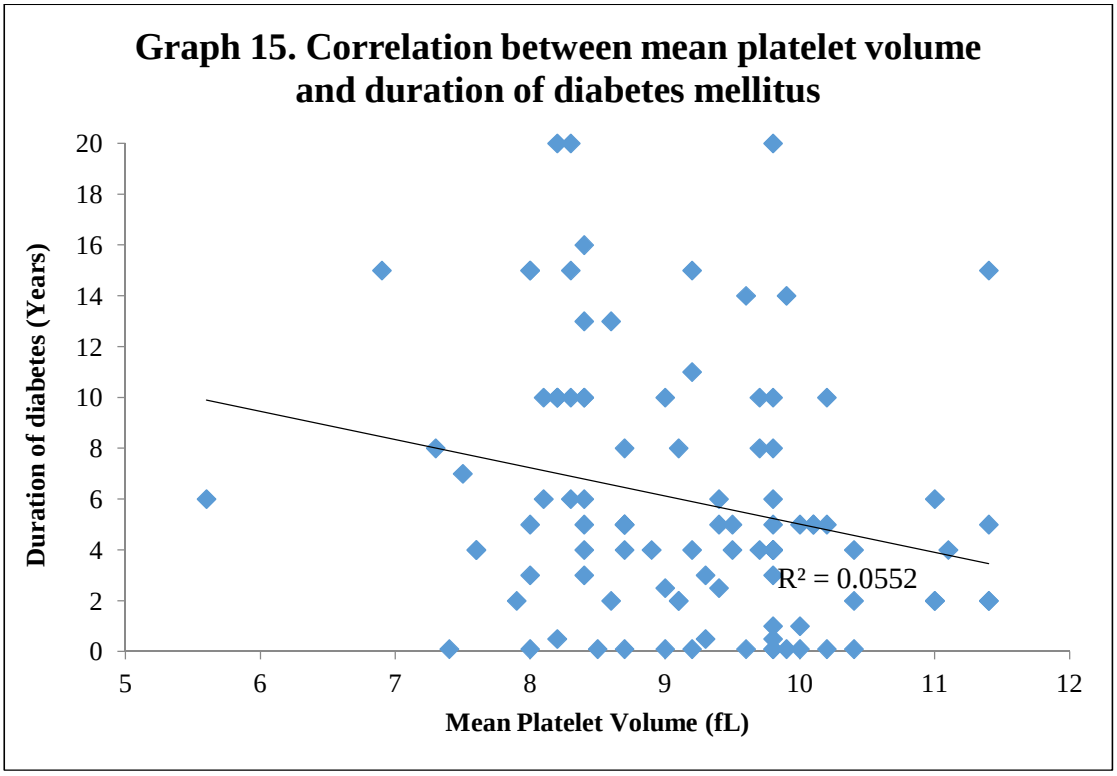
***r*= 0.287; R^2=0.0823; p=0.004**

The correlation between MPV and HbA1c is as shown in Graph 14. There was significant weak positive correlation between MPV and HbA1c (*r*= 0.287; R^2=0.0823; p=0.004) .



***r*= 0.094; R^2=0.0089; p=0.350**

The correlation between MPV and FBS is as shown in Graph 15. There was non significant weak positive correlation between MPV and FBS (*r*= 0.094; R^2=0.0089; p=0.350).



***r*= -0.235; R^2=0.0552; p=0.019**

The correlation between MPV and duratrion of diabetes is as shown in Graph 16. There was significant weak negative correlation between MPV and duration of diabetes (*r*=-0.235; R^2=0.0552; p=0.019).

DISCUSSION

Diabetes mellitus (DM) is the most common endocrine disease characterized by metabolic abnormalities, hyperglycemia resulting in long term complications.[106] Thrombosis, atherosclerosis and other vascular diseases are common complications of diabetes mellitus.[112]

Platelets play an important role in the normal hemostasis; the Mean Platelet Volume (MPV), an accurate measure of the platelet size is considered a marker and determinant of platelet function. It is postulated that, larger platelets with higher MPV are more reactive and produce higher amounts of the prothrombotic factor Thromboxane A2, increasing a propensity to thrombosis.[113] Platelets from subjects with DM, particularly from those with type 2 diabetes, exhibit increased reactivity. Factors that may contribute to this greater platelet reactivity are not completely elucidated and include metabolic abnormalities such as hyperglycemia, hyperlipidemia, insulin resistance, and conditions as oxidative stress, inflammation, and endothelial dysfunction.[114]

MPV, a determinant of platelet function, is a newly emerging risk factor for atherothrombosis. A large proportion of persons with type 2 DM suffer from preventable macrovascular complications. Hence, there is a need to develop risk factor modification interventions in order to reduce the impact of long term complications. MPV can be easily measured during routine hematological analysis. Thus, MPV can emerge as an important, simple, effortless, and cost-effective tool for monitoring and for early recognition of patients that could possibly benefit from preventive treatment in order to avoid long term complications.[106] Considering these

facts the present study was designed to evaluate the relationship between glycosylated hemoglobin and mean platelet volume in patients with type 2 diabetes mellitus.

The present one year hospital based cross sectional study was carried out in the Department of General Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi from January 2017 to December 2017. A total of 100 patients diagnosed to have type 2 diabetes mellitus were studied. The selected patients were subjected to estimation of HbA1c, MPV and FBS and other routine investigations.

It is reported that, the prevalence of diabetes is higher in men than women.[49-55] However, in the present study 51% of the patients were females and 49% of the patients were males. The male to female ratio was 1:1.04 suggesting almost equal number of males and females with T2DM.

Unlike in the West, where older persons are most affected, diabetes in Asian countries is disproportionately high in young to middle-aged adults.[49-55] In this study age ranged between 29 to 74 years. 30% of the patients each were aged between 51 to 60 years and 61 to 70 years. The mean age was 55.83±10.84 years and the median age was 57.50 years. These findings suggest that, most of the patients in the present study were middle aged individuals or elderly.

With regard to diabetic characteristics, the duration of diabetes ranged from less than a year as 16% of the patients were newly diagnosed to 20 years. Most of the patients (45%) reported duration of diabetes between one to five years and 24% had duration between 6 to 10 years. However 4% of the patients had duration of 16 to 20 years. The mean duration of diabetes was noted as 5.94±5.03 years. Looking at the

mean age and duration of diabetes it was evident that most of the individuals had shorter duration of diabetes and only few patients had long standing diabetes. With respect to the diabetic medication, 43% of the patients had treatment with oral hypoglycaemic agents (OHA) and 24% were on both insulin and OHA. Treatment with diet and life style modification was noted in 21% of the patients and 12% were on treatment with only insulin. Majority of the patients (74%) reported good treatment compliance while poor treatment compliance was noted in 26% of the patients. Also, 61% of the patients had microvascular complications and 47% of the patients reported history of hypertension. Abnormal fundoscopy was noted in 81% of the patients and 55% of the patients had abnormal ECG findings.

In the present study MPV levels in patients with T2 DM ranged between 5.6 to 12.00 fL. Nearly half of the patients with T2DM (48%) had MPV levels between 7.01 to 9.00 fL while more than one third (42%) of the patients had higher MPV levels that is, between 9.01 to 11.00, the mean MPV levels were noted as 9.08±1.20 fL and median MPV levels were 9.00 fL. These findings suggest that, patients with T2DM are likely to have high normal or intermediate MPV levels.

In this study, HbA1c levels ranged between 4.8 to 19.90 percent and the mean HbA1c levels were noted as 9.86±3.02 percent and median HbA1c levels were 9.75 percent. Further, majority of the patients that is, 77% had poor diabetic control with HbA1c levels of > 7.00 percent and only 23% of the patients had optimal diabetic control (HbA1c ≤7.0 percent). Looking at the mean HbA1c levels and the proportion of patients with HbA1c > 7 percent it was evident that, majority of the patients in the present study had poor glycemic control.

In the present study all the patients (100%) with MPV of > 11 fL had HbA1c levels of > 7 percent. All the patients with MPV of <7 fL had HbA1c levels of ≤7 percent. This difference was statistically significant (p=0.002). Further, the mean MPV levels in patients with HbA1c levels of ≤ 7 percent were significantly low (8.33±1.09 fL) compared to those with HbA1c of > 7 (9.4±0.92) (p<0.001). Also, there was weak positive correlation between MPV and HbA1c (*r*= 0.287; R^2=0.0823; p=0.004) but this correlation was statistically significant (p=0.004). These findings not only suggest strong association between MPV and HbA1c but also strong linear relationship between MPV and HbA1c in patients with T2DM. These findings were consistent with several other studies in literature.

The findings of the present study were consistent with the observations reported by Sharpe and Trinick[102] (1993) who were first to report the association between MPV and T2DM and reported significant increase of MPV in diabetic compared with non diabetic patients. This study was followed by numerous other small studies by Papanas N. et al.[115] (2004), Demirtunc R et al.[103] (2009), Kodiatte TA et al.[104] (2012), Dindar S. et al.[116] (2013) and Zaccardi F. et al.[99] (2015) and with the largest study to date, conducted on 13,021 diabetic patients by Shah B et al.[93] (2012) with similar findings. More recently, the Dongfeng–Tongji cohort study by Zhaoyang Li et al.[98] (2018) showed that higher levels of MPV were independently associated with increased incident risk of T2DM in a middle aged and older Chinese population. In contrast to the observations noted in our study, Bayram SM et al.[117] (2015) reported that MPV levels did not correlate with HbA1c.

A series of interrelated alterations occur following sustained hyperglycaemia which cause endothelial dysfunction and vascular lesions leading to complications in

diabetics.[107] Kodiatte et al.[104] (2012) reported that increased platelet activity have an important role in the development of vascular complications in type 2 DM. It can be suggested that increased platelet volume may be an important factor in the enhanced risk of vascular complications in these cases.[105] In this respect, MPV can be used as a favorable test in the monitoring of type 2 DM in terms of atherosclerosis development.[105]

Increased platelet reactivity and consequently higher MPV in T2 DM patients is caused by the multifactorial causes, e.g. hyperglycemia, hypertriglyceridemia, oxidative stress, inflammation and absolute or relative insulin deficiency creating a favorable milieu for the development of vascular complications. It is proposed that, effects on coagulation system in those patients cannot be attributed to only one of these factors, individually. Since T2DM is primarily defined by hyperglycemia, its impact on coagulation has been studied quite extensively. Hyperglycemia induces nonenzymatic glycation of proteins on the surface of the platelets, which decreases membrane fluidity and increases its reactivity. Hyperglycemia also increases platelet reactivity due to its direct osmotic effects on platelets causing osmotic swelling. Additionally, higher MPV values may be the result of the increased production of young platelets due to a higher platelet turnover rate.[118]

Bayram SM et al.[117] (2016) suggested that, increased MPV in diabetes may be due to osmotic swelling of the platelets or due to insulin effect, which forces megakaryocytes to produce platelets with large sizes. Another postulated theory may reflect increased platelet turnover and increased presence of younger thrombocytes.

Though, it is reported that, increased platelet reactivity is strongly associated with hyperglycaemia, interestingly, the present study showed weak positive

correlation between MPV and FBS (r=0.094; R^2=0.0089; p=0.350) and the correlation observed was statistically not significant which was consistent with a study by Bayram SM et al. (2015) where MPV levels did not correlate with FBG.[117] Similarly Hekimsoy Z et al.[29] (2004) also did not find any correlation between MPV and fasting serum glucose (FSG) in patients with type 2 diabetes mellitus. In contrast, Ulutas KT et al.[105] (2014) found a correlation between MPV and FSG. Shimodaira et al.[119](2013) also reported relationship between MPV and FSG in prediabetic patients.

In the present study weak negative correlation between MPV and duration of diabetes was observed (r=-0.235; R^2=0.0552; p=0.019). Though the correlation observed was statistically significant the correlation observed was weak. Bayram SM et al.[117] (2016) Hekimsoy Z. et al.[29] (2004), Papanas N. et al.[115] (2004), and Sharpe PC, Trinick T. (1993) also did not find any correlation between MPV and duration of diabetes. Bayram SM et al.[117] (2016) stated that, MPV increase may only be related to diabetes. It was shown that an increase in MPV occurred at the beginning of the disease and persisted for the duration of the disease. It can be said that when the damage in vasculature starts, it may be constant, and it continues for the duration of the disease independent of the control of diabetes. In concordance with this thought, we also think that the type of diabetes treatment (either insulin or oral hypoglycemics) may not affect MPV levels. However, in Vernekar and Vaidya's study[120] (2013), early initiation of insulin treatment was not only found to help in controlling blood glucose levels but also helped in keeping MPV levels low. Dolasık et al.[121] (2013) also found that metformin treatment significantly decreased MPV levels. Keeping in mind that control of diabetes did not affect MPV levels, Bayram SM et al.[117] (2016) postulated that insulin and metformin have specific effects on MPV levels, and they do not act solely by controlling diabetes.

Overall, the present study showed strong association and relationship between MPV and HbA1c in patients with T2DM but no relation was noted between MPV with FBS and duration of diabetes as well. However these findings require further validation due to potential limitations of the study. That is, relatively smaller sample size and the study was from a single institution thus can't be generalised to the entire population. Also in the present study 16% of the patients were newly diagnosed to have T2DM and they were managed by diet and lifestyle modification which may raise the concern about the increase in MPV occurred at the beginning of the disease as proposed by Bayram SM et al.[117] (2016) These factors could inevitably lead to potential bias in the evaluation of the relation between MPV and HbA1c in patients with T2DM. Furthermore, this study did not ascertained a causal relationship between MPV and vascular complications in diabetes which was beyond the scope of this study.

Hence further multicentric studies involving large sample size with pertinent selection criteria related to duration and treatment of diabetes and complications may provide useful insights about the true relationship between MPV and HbA1c in patients with T2DM.

CONCLUSION

The present study demonstrates not only strong association but also strong relationship between HbA1c and MPV in patients with type 2 diabetes mellitus. However, no relation of MPV was found with FBS.

SUMMARY

Since it is postulated that, MPV being a determinant of platelet functionality and increased MPV levels are associated with increased risk for hyperglycemic complications, this study aimed to find the relationship between glycosylated hemoglobin and mean platelet volume in patients with type 2 diabetes mellitus.

This one year hospital based cross sectional study was conducted in the Department of General Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi from January 2017 to December 2017. A total of 100 patients presenting with type 2 diabetes mellitus fulfilling the selection criteria were studied. The selected patients underwent tests for HbA1c, MPV, FBS and other relevant tests. The salient findings of the study are as summarized below.

- The male to female ratio was 1:1.04 with 51% of the patients being females and 49% being males.

- The common age group was 51 to 60 years and 61 to 70 years comprised of 30% of the patients each. The mean age was 55.83±10.84 years.

- Most of the patients had (61%) microvascular complications.

- The mean duration of diabetes was noted as 5.94±5.03 years. Most of the patients (45%) reported duration of diabetes between one to five years. However 16% of the patients were newly diagnosed and 4% of the patients had duration of 16 to 20 years.

- Treatment with oral hypoglycaemic agents was reported by 43% of the patients and 24% were on both insulin and OHA while 21% of the patients reported diet and life style modification and 12% were on treatment with insulin.

- Majority of the patients (74%) reported good treatment compliance and poor treatment compliance was noted in 26% of the patients.

- Abnormal fundoscopy was noted in 81% of the patients and 55% of the patients had abnormal ECG findings

- 48% of the patients had MPV levels between 7.01 to 9.00 fL and 42% of the patients had MPV levels between 9.01 to 11.00 the mean MPV levels were noted as 9.08±1.20 fL.

- HbA1c of ≤7.0 percent was noted in 23% of the patients and 77% of the patients had HbA1c levels of > 7.00 percent. The mean HbA1c levels were noted as 9.86±3.02 percent.

- All the patients with MPV of > 11 had HbA1c levels of > 7 percent and all the patients with MPV of < 7 had HbA1c levels of ≤7 percent. (p=0.002).

- The mean MPV levels in patients with HbA1c levels of ≤ 7 percent were significantly low (8.33±1.09 fL) compared to those with HbA1c of >7 percent (9.4±0.92) (p<0.001).

- There was significant weak positive correlation between MPV and HbA1c (r=0.287; R^2=0.0823; p=0.004) and non significant weak positive correlation between MPV and FBS (r= 0.094; R^2=0.0089; p=0.350).

- There was significant weak negative correlation between MPV and duration of diabetes (*r*=-0.235; R^2=0.0552; p=0.019).

Based on the findings of this study it may be concluded that, there is strong association and relationship between HbA1c and MPV in patients with type 2 diabetes mellitus.

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INFORMED CONSENT FORM

THE RELATIONSHIP BETWEEN GLYCOSYLATED HEMOGLOBIN AND MEAN PLATELET VOLUME IN TYPE 2 DIABETES MELLITUS – ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY

Objective and purpose of the study:

This research is intended to study the relationship between glycosylated hemoglobin and mean Platelet Volume. The principal investigator of the study is **Dr.** _____ under the guidance of **Dr** _____**.** The study aims to establish the relationship between Mean platelet volume and Glycosylated Hemoglobin in patients who have Type 2 Diabetes Mellitus. This will help in assessing the vascular complications of Diabetes Mellitus owing to poor glycemic control and help to intervene at the primary level to curtail such complications

Procedure

If you agree to be part of the research study you will be asked history in brief and subjected to clinical examination and Blood investigations like Mean Platelet volume and Glycosylated Hemoglobin and other relevant investigations.

Risk and Benefits

There is no risk associated with study.

Alternatives

Taking part in this study is voluntary. You may choose not to take part in this study, or if you decide to take part you can later change my mind and withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsorer may stop your participation in this study any time. If you choose not to take part in the study you will receive the standard treatment for patients with your condition

Privacy and Confidentiality

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

Institution / Sponsor's policy

Does not apply to this research

Voluntary participation/ withdrawal

Your participation in this study is entirely voluntary and you may withdraw from the study at any time.

Financial incentives for participation

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

Authorization to publish the results

The results of the study would be forwarded to the KLE University, Belagavi as part of requirement towards the completion of MD degree, review and publishing.

Questions

If you have any questions about study you may call In case of queries regarding your right as a participant you may contact:

Dr. _____	Dr _____
Professor of Medicine	Investigator,
Department of General Medicine	PG in General Medicine,
JNMC, Belagavi.	JNMC, Belagavi
Phone No: _____	Phone No.: _____

Dr. Ganga S. Pilli,
Chairman, J.N.M.C Ethical Committee for Human Research,
Professor and Head,
Department of Pathology,
JNMC Belagavi
Phone number: 0831-2471350, Extn: 1527

CONSENT FORM

I voluntarily agree to take part in this study by signing on the line below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicated that I have read this entire consent form or it has been read to me, and has been explained to me in my vernacular language and had all my questions answered. I will be given a copy of this consent form.

Signature /Left Thumb print of the Participant or legally authorized representative.

Participant's Name/ : …….

Signature/ Left Thumb

Impression of the participant's : …….

Signature/ Left Thumb

Impression. : …….

Witness's Name : …….

Signature/ Left Thumb

Impression. :…….

Investigators name and Signature : …….

Date and Place : …….

Date: Place :

ತಿಳುವಳಿಕೆ ಒಪ್ಪಿಗೆ ಪತ್ರ

ಸಂಶೋಧನೆಯ ಶೀರ್ಷಿಕೆ: ಎರಡನೇ ಮಧುಮೇಹ ಮಲ್ಲಿಟಸ್ ನಲ್ಲಿ ಗ್ಲೈಕೂಸ್ಯಲೇಟೆಡ್ ಹಿಮೊಗ್ಲಬೀನ್ ಮತ್ತು ಮೀನ್ ಪ್ಲೇಟ್ ಲೆಟ್ ಗಳ ಸಂಬಂಧ ಮತ್ತು ಪ್ರಮಾಣ.

ಸಂಶೋಧನೆಯ ಪರಿಚಯ ಮತ್ತು ಉದ್ದೇಶ :

ಈ ಅಧ್ಯಯನದ ಉದೇಶವು ಎರಡನೇ ಮಾದರಿ ಮಧುಮೇಹ ಮಲ್ಲಿಟಸ್ ನ ಗ್ಲೈಕೂಸ್ಯಲೇಟೆಡ್ ಹಿಮೊಗ್ಲೊಬಿನ್ ಮತ್ತು ಮೀನ್ ಪ್ಲೇಟ್ಲೆಟ್ ಗಳ ಸಂಬಂಧ ಮತ್ತು ಪ್ರಮಾಣವನ್ನು ತಿಳಿಯುವುದಾಗಿದೆ. ಡಾ. _____ ಇವರ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ಡಾ. _____ ಇವರ ಸಂಶೋಧನೆಯನ್ನು ಮಾಡಿರುತ್ತಾರೆ.

ಈ ಅಧ್ಯಯನದ ಮುಖ್ಯ ಉದ್ದೇಶ ಎರಡನೇ ಮದರಿ ಮಧುಮಹ ಮಲ್ಲಿಟಸ್ ನಲ್ಲಿ ಗ್ಲೈಕೂಸ್ಯಲೇಟೆಡ್ ಹಿಮೊಗ್ಲೊಬಿನ್ ಮತ್ತು ಮೀನ್ ಪ್ಲೇಟ್ಲೆಟ್ ಗಳ ಸಂಬಂಧ ಮತ್ತು ಪ್ರಮಾಣವನ್ನು ತಿಳಿಯುವುದೇಗಿದೆ. ಇವರಿಂದ ಎರಡನೇ ಮಾದರಿ ಮಧುಮೇಹ ಮಲ್ಲಿಟಸ್ ಗೆ ಕಾರಣಗಳನ್ನು ತಿಳಿದುಕೊಂಡು, ಅದನ್ನು ನಿಯಂತ್ರಿಸಲು ಮತ್ತು ಉಪಚರಿಸಲು ಸಹಯವಗುತ್ತದೆ.

«zsÁ£À:

¤ÃªÀÅ F ಸಂಶೋಧನೆಯ ¨sÁUÀªÁUÀ®Ä M¦àPÉÆAqÀ°è ¤ªÀÄUÉ SÁAiÉÄ°UÉ ¸ÀA§A¢ü¹zÀ ¥Àæ±ÀßUÀ¼À£ÀÄß PÉÃ¼ÀèUÀÄªÀÅzÀÄ ºÁUÀÄ *zÀåQÃAiÀÄ vÀ¥Á¸ÀtÂUÉ M¼ÀqÀ¨ÉPÁUÀÄvÀÛzÉ. EªÀgÀ eÉÆwUÉ ¤ÃªÀÅ ¤ªÀÄä gÀPÀÛ ºÁUÀÄ ªÀiÁzÀjAiÀÄ£ÀÄß ¤ÃqÀ¨ÉPÁUÀÄvÀÛzÉ.

C£ÀÄPÀÄ® ºÁUÀÄ C£Á£ÀÄPÀÆ®vÉUÀ¼ÀÄ:

F ಸಂಶೋಧನೆಯ ¸ÀªÀÄAiÀÄzÀ°è AiÀiÁªÀzÉ jÃwAiÀÄ C£Á£ÀÄPÀÆ¯ÉÚUÀ¼ÁUÀ°Ã CxÀªÁ vÉÆzÀgÉUÀ¼ÁUÀ° EgÀÄªÀÅ¢®è.

¥ÀAiÀiÁðAiÀÄUÀ¼ÀÄ:

F ¸ÀA±ÉÆzsÀ£Á CzsÀåAiÀÄ£ÀzÀ°è ¤ÃªÀÅ ¸Àé EZÀÑ¬ÄAzÀ ªÀÄvÀÄÛ M¦àUÉ¬ÄAzÀ ¨sÁUÀªÀ»¸À§ºÀÄzÀÄ DxÀªÁ ¤ÃgÁPÀj¸À®åº§ºÀÄzÀÄ. MAzÀÄ ªÉÃ¼É ¤ÃªÀÅ ¸ÀA±ÉÆzsÀ£ÉAiÀÄ ¨sÁUÀªÁ®Ä M¦àPÉÆAqÀÄ £ÀAvÀgÀzÀ ¢£ÀUÀ¼À°è ªÀÄ£É¸ÀÄì §zÀ¯Á¬Ä¹zÀgÉ ¤ÃªÀÅ JCzÀjAzÀ ºÉÆgÀ§gÀ§ºÀÄzÀÄ. ¤ÃªÀÄä F ¤zÁðgÀªÀÅ ¤ªÀÄUÉ FUÀ zÀÆgÀAiÀÄÄwÛgÀÄªÀ ªÉÊzÀåQÃAiÀÄ ºÁUÀÄE£ÉßvÀgÀ ¸ÉÃªÉUÀ¼À ªÉÄÃ¯É AiÀiÁªÀÅzÉ ¥Àæ¨sÁ¸À ©ÃgÀÄªÀÅ¢®è ºÁUÀÄ £ÀªÀÄUÉ ¸ÀºÀ EgÀgÉ gÉÆÃVUÀ¼ÀAvÀ ¤ªÀÄVgÀÄªÀ SÁ¬Ä¯ÉUÀ£ÀÄ¸ÁgÀªÀV aQvÉì zÀÆgÉAiÀÄÄªÀÅzÀÄ.

UË¥ÀåvÉ:

¸ÀA±ÉÆzsÀ£ÉAiÀÄ ¸ÀAzsÀ§ðzÀ°è ¸ÀAUÀæ»¹gÀÄªÀ J¯Áè ªÀiÁ»wAiÀÄ£ÀÄß PÁ£ÉÆ¤UÀ£ÀÄ¸ÁgÀªÁV UË¥ÀåªÁVqÀ¯ÁUÀÄªÀÅzÀÄ, J°èAiÀÄÆ ¤ßªÀÄä ºÉ¸ÀgÀÄ ¥ÀæPÀlªÀUÀzÉÃ ¤ßªÀiË£ÀÄß UË¥ïà ¸ÀASÉå¬ÄÃAzÀ

UÀÄgÀÄw¸À®UÀÄªÀÅzÀÄ. ªÀÄÄA¢£À ¢£ÀUÀ¼À°è F ¸ÀA±ÀÆzsÀ£Á ªÀgÀ¢AiÀÄÄ ¥ÀæPÀlUÉÆAqÁUÀ®Ä ¸ÀºÀ ¤ªÀÄä ºÉ¸ÀgÀ£ÀÄß UË¥ÀåªÀUÉqÀ¯ÁUÀÄªÀÅzÀÄ.

¸ÀA¸ÉÜ DxÀªÁ ¥ÁæAiÉÆfPÀgÀ ¤ßwUÀ¼ÀÄ :

F ¸ÀA±ÉÆzsÀ£Á DzsËåC£ÀzÀ°è ¤ÃªÀÅ ¸Àé EaÑ¬ÄAzÀ ªÀÄvÀÄÛ M¦àUÉ¬ÄAzÀ ¨sÀUÀªÀ»¸À§ºÀÄzÀÄ CxÀªÁ »AzÀ ¸ÀjAiÀÄ®Ä§ºÀÄzÀÄ.

¥sÀÇævÁìºÀ zsÀ£À:

¤ªÀÄUÉ F ಸಂಶೋಧನಯ ¥sÀ°vÁA±ÀªÀ£ÀÄß PÉ.J¯ï.E «±Àé«zÁå®AiÀÄ ¨É¼ÀUÁ« EªÀjUÉ JªÀiï. r. ¥ÀzÀ« ¥ÀÅtðUÉÆ½¸À®Ä, «ªÀÄ²ð¸À®Ä ºÁUÀÄ ¥ÀæPÀn¸À®Ä PÀ¼ÀÄ»¹PÀÆqÀ®UÀÄªÀÅzÀÄ.

¸ÀA±ÉÆzsÀ£ÉAiÀÄ ¸ÀAzÀ¨sÀðzÀ°è CxÀªÁ ªÀÄÄAzÉ AiÀiÁªÀÅzÁzÀ®Ä ¥Àæ±ïßUÀ¼ÀÄ CxÀªÁ C£ÀÄªÀiÁ£ÀUÀ½zÀÝ°è F PÉ¼ÀV£ÀªÀgÀ£ÀÄß ¸ÀA¥ÀQð¸À§ºÀÄzÁVzÉ.

M¦àUÉ ¥ÀvÀæ

£Á£ÀÄ £À£Àß ¸Àé-EZÉÑ¬ÄA¤ßÃ ¸ÀªÀÄäw ¥ÀvÀæPÉÌ ¸À» ªÀiÁrgÀÄvÉÛÃ£É, £Á£ÀÄ £À£Àß ¸ÀªÀÄäwAiÀÄ£ÀÄß AiÀiÁªÁUÀ ¨ÉÃPÁzÀgÀÆ »A¥ÀqÉAiÀÄ§ºÀÄzÀÄ, PÉÃªÀ® ¸À» ªÀiÁrzÀ ªÀÄvÀæPÉÌ £À£Àß PÁ£ÀÆ£ÁvÀäPÀ ºÀPÀÄÌUÀ¼Å£ÀÄß ©lÄÖ PÉÆlÖAvÁUÀÄªÀÅ¢®è. £Á£ÀÄ F ¸ÀªÀÄw ¥ÀvÀæPÉÌ ¸À» ªÀiÁrzÀ £ÀAvÀgÀ EªÀgÀ MAzÀÄ ¥ÀæwAiÀÄ£ÀÄß £Á£ÀÄ PÁ¬ÄÝj¹PÉÆ¼ÀÆîvÉÛÃ£É.

¨sÁVzÁgÀgÀ ¸À» / JqÀUÉÊ ºÉ¨ÉâgÀ½£À UÀÄgÀÄvÀÄ

¸À» / JqÀUÉÊ ºÉ¨ÉâgÀ½£ï UÀÄgÀÄvÀÄ

_____

¸ÁQëzÁgÀgÀ ¸À» _____

¸À» / JqÀUÉÊ ºÉ¨ÉâgÀ½£À UÀÄgÀÄvÀÄ

_____

vÀ¤¢üPÀjAiÀÄ ºÉÃ¸ÀgÀÄ ªÀÄvÀÄÛ ¸À»

_____

¸ÀÜ¼À ªÀÄvÀÄÛ ¢£ÁAPÀ

_____

¢£ÁAPÀ

¸ÀÜ¼À

eÉÉlÉMüÉUÏ mÉëÉmiÉ WûÉålÉå Måü oÉÉS SÏ aÉD xÉqqÉÌiÉ

OûÉDmÉ- 2 qÉkÉÑqÉåWû qÉåÇ U£ü zÉMïüUÉ AÉæU U£ü Måü mÉåOûsÉåO CxÉ NûÉåOåû MühÉÉåÇMüÐ qÉÉ§ÉÉ!

xÉÇZrÉÉ CxÉMüÉ xÉÇoÉÇkÉ – LMü xÉÉsÉ MüÉ WûÉxmÉÏOûsÉ qÉåÇ ÌMürÉÉ aÉrÉÉ xÉÉqÉÔÌWûMü AprÉÉxÉ¢üqÉ

AprÉÉxÉMüÉ E¬åzÉ AÉæU WåûiÉÑ

CxÉ xÉÇzÉÉåkÉlÉ AprÉxÉqÉåÇ U£ü zÉMïüUÉ AÉæU U£ü qÉåÇ msÉåOèsÉåOû ClÉ NûÉåOåû MühÉÉåÇMüÐ xÉÇZrÉÉ/qÉÉ§ÉÉ CxÉMüÉ xÉÇoÉÇkÉ YrÉÉ Wæû rÉWû eÉÉlÉMüÉUÏ mÉëÉmiÉ MüUlÉÉ rÉWû Wæû!

CxÉ AprÉÉxÉ¢üqÉ Måü mÉëqÉÑZÉ xÉÇzÉÉåkÉMü *ಸ್ವೀ.*

AÉæU ExÉMåü qÉÉaÉïSzÉïMü QûÉ.

!

OûÉDmÉ- 2 qÉkÉÑqÉåWû UÉåaÉÏrÉÉåÇ qÉåÇ U£ü qÉåÇ msÉåOèsÉåOû Måü NûÉåOåû MühÉÉåÇMüÐ xÉÇZrÉÉ! qÉÉ§ÉÉ AÉæU

U£üzÉMïüUÉ CxÉMüÉ YrÉÉ xÉÇoÉkÉ Wæû CxÉMüÉ AprÉÉxÉ MüUlÉÉ Wæû! qÉkÉÑqÉåÇWû Måü UÉåaÉÏrÉÉåÇ qÉåÇ MüqÉ U£ü zÉMïüUÉ WûÉålÉå MüÐ uÉeÉWûxÉå U£ü uÉÉÌWûlÉÏrÉÉåÇ qÉåÇ, ÌMüxÉ mÉëMüÉUxÉå ElÉMåü qÉÉaÉï qÉåÇ mÉëÌiÉoÉÇkÉ ÌlÉqÉÉïhÉ WûÉåiÉÉ Wæû CxÉMüÐ eÉÉlÉMüÉUÏ WûÉxÉÏsÉ MüUlÉÉ AÉæU mÉëÉjÉÍqÉMü ÎxjÉÌiÉqÉåÇ CxÉ mÉëMüÉU Måü mÉëÌiÉoÉÇkÉ ÌlÉqÉÉïhÉ WûÉålÉåmÉU MüÉælÉxÉå EmÉcÉÉU MüUlÉÉ Wæû, CxÉMüÐ eÉÉlÉMüÉUÏ WûÉxÉÏsÉ MüUlÉÉ Wæû!

mÉkSiÉ

CxÉ AprÉÉxÉ qÉåÇ AÉmÉ rÉSÏ xÉÉÍqÉsÉ WûÉåiÉå Wæû iÉÉå AÉmÉMüÉå AÉmÉMåü oÉÉUå qÉåÇ eÉÉlÉMüÉUÏ mÉÔNûÏ eÉÉrÉåaÉÏ AÉæU uÉæ±ÌMürÉ eÉÉÇcÉ MüUMåü AÉæU U£üqÉåÇ msÉåOèsÉåOû Måü NûÉåOåû MühÉÉåÇMüÐ xÉÇZrÉÉ AÉæU U£üzÉMïüUÉ CxÉMüÉ xÉÇoÉkÉ Måü oÉÉUå qÉåÇ xÉÇzÉÉåkÉlÉ ÌMürÉÉ eÉÉrÉåaÉÉ!

kÉÉåMüÉ AÉæU qÉÑlÉÉTüÉ

CxÉ AprÉÉxÉ¢üqÉ qÉåÇ kÉÉåMüÉ lÉWûÏ Wæû!

mÉrÉÉïrÉ

CxÉ xÉÇzÉÉåkÉlÉ AprÉÉxÉqÉåÇ AÉmÉ ZÉÑS xÉqqÉÍsÉiÉ WûÉå xÉMüiÉå Wæû! AÉmÉ CxÉ AprÉÉxÉqÉåÇ xÉqqÉÍsÉiÉ lÉWûÏ WûÉålÉÉ cÉÉWûiÉå rÉWû pÉÏ AÉmÉ oÉlÉÉ xÉMüiÉå Wæû! rÉÌS AÉmÉ CxÉ AprÉÉxÉqÉåÇ ZÉÑS xÉqqÉÍsÉiÉ WûÉå aÉrÉå iÉÉå AÉmÉMüÉå CxÉMåü oÉÉS pÉÏ qÉlÉqÉåÇ zÉÇMüÉ EimÉ³É WûÉå aÉrÉÏ iÉÉå, AÉmÉMüÉ lÉÉqÉ CxÉ AprÉÉxÉ¢üqÉ xÉå AÉmÉ MüqÉ MüU xÉMüiÉå Wæû! CxÉ ÌlÉhÉïrÉ xÉå AÉmÉMüÉå pÉÌuÉzrÉ qÉåÇ ÍqÉsÉlÉåuÉÉsÉå uÉæ±ÌMürÉ qÉSiÉ AjÉuÉÉ xÉåuÉÉAÉåÇ mÉU MüÉåC AxÉU lÉWûÏ WûÉåaÉÉ!

rÉWû AprÉÉxÉ xÉÇzÉÉåkÉlÉ MüpÉÏ pÉÏ xÉÇzÉÉåkÉMü MüUlÉåuÉÉsÉå ƷŸYOûU rÉÉ CxÉMåü mÉërÉÉåeÉMü, CxÉMüÉå xÉqÉÉmiÉ MüU xÉMüiÉå Wæû! rÉSÏ AÉmÉ CxÉ AprÉÉxÉ¢üqÉxÉå xÉqqÉÍsÉiÉ lÉWûÏ WûÉåiÉå iÉÉå pÉÏ AÉmÉMüÉå CxÉ eÉaÉWû xÉå rÉÉæarÉ uÉæ±ÌMürÉ ÍcÉÌMüixÉÉ ÍqÉsÉåaÉÏ!

aÉÉåmÉÌlÉrÉiÉÉ

CxÉ xÉÇzÉÉåkÉlÉqÉåÇ AÉmÉMåü ²ÉUÉ mÉëÉmiÉ MüÐ WÒûD eÉÉlÉMüÉUÏ MüÉlÉÔlÉ Måü qÉÑiÉÉÌoÉMü aÉÉåmÉÌlÉrÉ UZÉÏ eÉÉrÉåaÉÏ! CxÉ xÉÇzÉÉåkÉlÉ AprÉÉxÉ¢üqÉqÉåÇ AÉmÉMüÉå LMü MüÉåQû lÉÇoÉU ÌSrÉÉ eÉÉrÉåaÉÉ AÉæU MüÉåQû lÉÇoÉU ÌMü qÉSiÉ xÉå AÉmÉMüÐ mÉWûcÉÉlÉ MüÐ eÉÉrÉåaÉÏ! CxÉ AprÉÉxÉ¢üqÉ qÉåÇ ÍqÉsÉÏ WÒûD eÉÉlÉMüÉUÏ mÉëÍxÉkSÏ qÉåÇ aÉÉåmÉÌlÉrÉ UZÉÏ eÉÉLaÉÏ.

xÉÇxjÉÉMüÉ rÉÉ mÉërÉÉåeÉlÉ MüUlÉåuÉÉsÉå MüÉ WåûiÉÑ

CxÉ xÉÇzÉÉåkÉlÉ Måü ÍsÉL rÉWû sÉÉaÉÔ lÉWûÏ Wæû!

AÉmÉlÉå SÏ WÒûD xÉqqÉÌiÉ rÉÉ xÉqqÉÍsÉiÉ lÉ WûÉålÉå MüÐ mÉëÌ¢ürÉÉ

AÉmÉMüÉ CxÉ AprÉÉxÉ qÉåÇ xÉqqÉÏsÉÏiÉ WûÉålÉÉ AÉmÉMåü EmÉU ÌlÉpÉïU MüUiÉÉ Wæû, AÉæU AÉmÉ CxÉ xÉÇzÉÉåkÉlÉ AprÉÉxÉxÉå AÉmÉMüÉ lÉÉqÉ ÌMüxÉÏ pÉÏ xÉqÉrÉ MüqÉ MüU xÉMüiÉå Wæû!

xÉÎqqÉÍsÉiÉ WûÉålÉåuÉÉsÉå MüÉå ÍqÉsÉlÉåuÉÉsÉÏ AÉÍjÉïMü xÉWûÉrÉiÉÉ

CxÉ AprÉÉxÉ¢üqÉ qÉåÇ xÉÎqqÉÍsÉiÉ WûÉålÉåuÉÉsÉå MüÉå MüÉåD pÉÏ AÉÍjÉïMü xÉWûÉrÉ¨ÉÉ rÉÉ ClÉÉqÉ lÉWûÏ ÍqÉsÉåaÉÉ!

AprÉÉxÉ Måü ÌlÉwMüwÉï mÉëÍxÉkS MüUlÉå Måü AÉÍkÉMüÉU

CxÉ AprÉÉxÉ Måü ÌlÉwMüwÉï Måü Måü. LsÉ C rÉÑÌlÉuWûÍxÉïÌOû oÉåsÉaÉÉqÉ MüÉå pÉåeÉå eÉÉrÉåaÉå AÉæU CxÉ AprÉAxÉ¢üqÉ MüÉå mÉëÉmiÉ MüUMåü iÉjÉÉ ExÉMüÉå mÉëÍxÉkSÏ SåMüU LqÉ. QûÏ. mÉSuÉÏ mÉëÉmiÉ MüUlÉÉ rÉWû LMü CxÉ xÉÇzÉÉåkÉlÉ MüÉ ÌWûxxÉÉ Wæû!

xÉqqWûiÉÏMüÉ lÉqÉÑlÉÉ

qÉæ CxÉ AprÉÉxÉ qÉåÇ, ÌlÉcÉå ÌMürÉå WÒûL qÉåUå ÍsÉrÉå sÉÉaÉÔ WûÉålÉåuÉÉsÉå sÉÉDlÉuÉU xÉWûÏ MüUMåü xuÉÇqÉ CxÉqÉåÇ pÉÉaÉ sÉå UWûÉ WÕÇû! qÉæÇ CxÉ AprÉÉxÉ ¢üqÉ xÉå MüpÉÏ pÉÏ, ÌMüxÉÏ pÉÏ uÉ£ü qÉÑÄ£ü WûÉ xÉMüiÉÉ WÕÇû! qÉælÉå qÉåUå MüÉåD pÉÏ MüÉlÉÔlÉÏ AÍkÉMüÉU CxÉ xÉqqÉÌiÉ lÉqÉÔlÉåmÉU yxiÉÉ¤ÉU MüUMåü lÉWûÏ aÉqÉÉrÉå WÕûL Wæû! qÉæÇ ÌlÉcÉå xÉWûÏ MüUlÉåuÉÉsÉÉ rÉWû oÉiÉÉlÉÉ NûÉWûiÉÉ WÕÇû ÌMü, rÉWû lÉqÉÑlÉÉ ÄTüÉqÉï qÉæÇlÉå mÉRûMüU xÉqÉfÉÉrÉÉ aÉrÉÉ Wæû AÉæU qÉÔfÉå qÉåUÏ pÉÉwÉÉqÉåÇ rÉWû xÉqÉfÉÉrÉÉ Wæû AÉæU qÉåUÉ mÉÔUÉ zÉÇMüÉxÉqÉÉkÉÉlÉ ÌMürÉÉ Wæû! qÉÔfÉå CxÉ lÉqÉÑlÉÉ ÄTüÉqÉï MüÐ MüÉmÉÏ SÏ aÉD Wæû!

AprÉÉxÉ qÉåÇ pÉÉaÉ sÉålÉåuÉÉsÉÉåÇ MüÐ rÉÉ ExÉMüÐ iÉUTüxÉå ExÉMåü mÉëÌiÉÌlÉÍkÉiuÉ MüUlÉåuÉÉsÉå MüÐ xÉWûÏ/oÉÉrÉå WûxiÉ Måü AÇaÉÑOåûMüÉ ÌlÉzÉÉlÉ

AprÉÉxÉ qÉåÇ pÉÉaÉ sÉålÉåuÉÉsÉå MüÉ lÉÉqÉ :_____

pÉÉaÉ sÉålÉåuÉÉsÉåMüÐ xÉWûÏ/oÉÉrÉå WûÉiÉ Måü AÇaÉÑPåûMü ÌlÉzÉÉlÉ:_____

xÉÉ¤ÉÏSÉUMüÐ xÉWûÏ:_____

xÉWûÏ rÉÉ oÉÉrÉå WûÉiÉ Måü AÇaÉÑPåûMü ÌlÉzÉÉlÉ:_____

xÉÇzÉÉåkÉlÉ MüUlÉåuÉÉsÉå MüÉ lÉÉqÉ AÉæU xÉWûÏ:_____

ÌSlÉÉÇMü AÉæU xjÉVû/eÉaÉWû:

iÉÉUÏZÉ:

xjÉVû/eÉaÉWû

qÉÉÌWûiÉÏ ÍqÉVûÉsrÉÉlÉÇiÉU ÌSsÉåsÉÏ xÉWûqÉÌiÉ

OûÉDmÉ-2 qÉkÉÔqÉåWûÉqÉkrÉå U£ü zÉMïüUÉ AÉÍhÉ U£üÉiÉÏsÉ msÉåOèsÉåOû rÉÉ NûÉåšÉ MühÉÉcÉÏ qÉÉ§ÉÉ! xÉÇZrÉÉ rÉÉqÉkÉÏsÉ xÉÇoÉÇkÉ- LMü uÉwÉÉïcÉÉ WûÉåxmÉÏOûsÉqÉkÉÏsÉ xÉÉqÉÔÌWûMü AprÉÉxÉ¢üqÉ

AprÉÉxÉÉcÉÉ E¬åzÉ uÉ WåûiÉÑ

WûÉ xÉÇzÉÉåkÉlÉ AprÉÉxÉ¢üqÉ U£ü zÉMïüUÉ uÉ U£üÉiÉÏsÉ rÉÉ NûÉåšÉ MührÉÉcÉÏ xÉÇZrÉÉ rÉÉÇcrÉÉ xÉÇoÉÇkÉÏcÉÏ qÉÉWûÏiÉÏ mÉëÉmiÉ MüUhÉå AxÉÉ AÉWåû. rÉÉ AprÉÉxÉ¢üqÉÉcÉå mÉëqÉÑZÉ xÉÇzÉÉåkÉMü _____ *ST.* _____ AÉÍhÉ irÉÉcÉå qÉÉaÉïSzÉïMü *ST.* Wåû AÉWåûiÉ.

OûÉDmÉ-2 qÉkÉÔqÉåW AxÉsÉåsrÉÉ UÉåaÉÉcrÉqÉkÉå U£üÉiÉÏsÉ msÉåOûsÉåOûcrÉÉ NûÉåšÉ MühÉÉcÉÏ xÉÇZrÉÉ! qÉÉ§ÉÉ uÉ U£ü zÉMïüUÉ rÉÉÇcÉÉ xÉÇoÉkÉ MüÉrÉ AÉWåû. rÉÉ xÉÇoÉÇkÉÏcÉÉ WûÉ AprÉÉxÉ¢üqÉ AÉWåû. qÉkÉÑqÉåWûÉcrÉÉ UÉåaÉÏqÉkrÉå MüqÉÏ mÉëqÉÉhÉÉiÉ U£ü zÉMïüUÉ fÉÉsrÉÉqÉÑVåû U£üuÉÉÌWûhrÉÉqÉkrÉå MüÉåhÉirÉÉ AQûcÉhÉÏ ÌMÇüuÉÉ ÌoÉbÉÉQû WûÉåuÉÔ zÉMüÉiÉÉiÉ irÉÉÌuÉzÉrÉÏ AÉqWûÉxÉ AÇSÉeÉ MüUhrÉÉxÉ qÉSiÉ ÍqÉVûiÉå AÉÍhÉ mÉëÉjÉÍqÉMü AuÉxjÉåiÉ AzÉÉ mÉëMüÉUcÉå ÌoÉbÉÉQû ÌMÇüuÉÉ AQûcÉhÉÏ ÌlÉqÉÉïhÉ fÉÉsrÉÉuÉU MüÉåhÉiÉå EmÉÉrÉ MüUÉuÉå Wåû xÉqÉeÉÑlÉ rÉåDsÉ.

mÉ®iÉÏ

rÉÉ xÉÇzÉÉåkÉlÉ AprÉÉxÉÉiÉ iÉÔqWûÏ xÉWûqÉiÉÏ xÉÉÇaÉiÉ AxÉÉsÉ iÉU, iÉÑqÉcrÉÉ ÌuÉwÉrÉÏcÉÉ mÉÔuÉÉïCÌiÉWûÉxÉ ÌuÉcÉÉUsÉÉ eÉÉDsÉ uÉ iÉÑqÉcÉÏ uÉæ±ÌMürÉ iÉmÉÉxÉhÉÏ MåüsÉÏ eÉÉDsÉ AÉÍhÉ U£üÉqÉkÉÏsÉ msÉåOûsÉåOûcrÉÉ NûÉåšÉ MühÉÉcÉÏ xÉÇZrÉÉ! qÉÉ§ÉÉ uÉ U£üÉiÉÏsÉ xÉÉZÉU rÉÉcrÉÉxÉÇoÉÇkÉÏ xÉÇzÉÉåkÉlÉ MåüsÉå eÉÉDsÉ.

kÉÉåMåü uÉ TüÉrÉSå

rÉÉ AprÉÉxÉ ¢üqÉÉqÉkrÉå MüÉåhÉiÉåWûÏ kÉÉåMåü lÉÉWûÏiÉ.

mÉrÉÉïrÉ

rÉÉ xÉÇzÉÉåkÉlÉ AprÉÉxÉÉqÉkÉå iÉÑqWûÏ xuÉiÉ:WÕûlÉ xÉWûÉpÉÉaÉÏ WûÉåuÉÔ zÉMüiÉÉ. iÉÔqWûÏ rÉÉ AprÉÉxÉ¢üqÉÉqÉkrÉå xÉWûpÉÉaÉÏ WûÉåiÉ lÉÉWûÏ AxÉå xÉÑ®É xÉÉÇaÉÔ zÉMüiÉÉ. eÉU MüÉ iÉÑqWûÏ xuÉiÉ:WÕûlÉ rÉÉ AprÉÉxÉ¢üqÉÉiÉ xÉWûpÉÉaÉÏ fÉÉsÉÉ AxÉÉsÉ lÉÇiÉU iÉÑqÉcrÉÉ qÉlÉÉiÉ iÉÑqÉcÉå lÉÉÇuÉ rÉÉ AprÉÉxÉ¢üqÉÉiÉÑlÉ MüÉRÕûlÉ CÎcNûiÉ AxÉÉsÉ iÉU iÉå xÉÑ®É iÉÑqWûÏ MüÂ zÉMüiÉÉ. rÉÉ iÉÑqÉÉcrÉÉ ÌlÉhÉïrÉÉqÉÑVåû iÉÑqWûÉxÉ pÉÌuÉzrÉÉiÉ ÍqÉVûhÉÉUÏ uÉæ±ÌMürÉ qÉSiÉ ÌMÇüuÉÉ xÉåuÉÉ rÉÉuÉU MüÉåhÉiÉÉWûÏ mÉUÏhÉÉqÉ WûÉåhÉÉU lÉÉWûÏ.

WûÉ AprÉÉxÉ¢üqÉ MüÉåhÉirÉÉWûÏ uÉåVûÏ xÉÇzÉÉåkÉMü MüUhÉÉUå ʒŸOûU ÌMÇüuÉÉ rÉÉ MüÉrÉï¢üqÉÉcÉå mÉërÉÉåeÉMü Wåû WûÉ AprÉÉxÉ¢üqÉ oÉÇS MüÂ zÉMüiÉÉiÉ. eÉU iÉÑqWûÏ rÉÉ AprÉÉxÉÉiÉ xÉWûpÉÉaÉÏ WûÉåiÉ lÉxÉÉsÉ iÉUÏ xÉÑ®É iÉÑqWûÉxÉ rÉÉ ÌPûMüÉhÉÏ rÉÉåarÉ iÉå xÉÇmÉÔhÉï uÉæ±ÌMürÉ EmÉcÉÉU ÍqÉVûiÉÏsÉ.

aÉÑm¨ÉiÉÉ

rÉÉ xÉÇzÉÉåkÉlÉÉqÉkrÉå iÉÑqÉcrÉÉ xÉÇSpÉÉïiÉ ÍqÉVûuÉsÉåsÉÏ qÉÉWûÏiÉÏ WûÏ MüÉrÉ±ÉcrÉÉ iÉUiÉÑSÏlÉÑxÉÉU aÉÑmiÉ PåûuÉhrÉÉiÉ rÉåDsÉ. rÉÉ xÉÇzÉÉåklÉ AprÉÉxÉ¢üqÉÉqÉkrÉå iÉÑqWûÉxÉ MüÉåQû lÉÇoÉU SåuÉÔlÉ iÉÑqÉcÉÏ AÉåVûZÉ MåüsÉÏ eÉÉDsÉ. rÉÉ AprÉÉxÉ¢üqÉÉqÉkrÉå ÍqÉVûuÉsÉåsÉÏ qÉÉÌWû¨ÉÏ mÉëÍxÉkS MåüsÉÏ eÉÉDsÉ. mÉUÇiÉÑ iÉÑqÉcÉå lÉÉÇuÉ uÉ xÉWûpÉÉaÉ WûÉ rÉÉ mÉëÍxÉ®ÏmÉÉxÉÔlÉ aÉÑmiÉ PåûuÉhrÉÉiÉ rÉåDsÉ.

xÉÇxjÉÉcÉå/mÉërÉÉåeÉlÉ MüUhrÉÉUÉcÉå kÉÉåUÉhÉ

rÉÉ xÉÇzÉÉåkÉlÉÉxÉ Wåû sÉÉaÉÔ lÉÉWûÏ.

xuÉiÉ:WÕûlÉ ÌSsÉåsÉÏ xÉWûqÉiÉÏ ÌMÇüuÉÉ/xÉWûÉpÉÉaÉÏ lÉ WûÉåhrÉÉcÉÏ ¢üÏrÉÉ

iÉÑqÉcÉÉ rÉÉ AprÉÉxÉÉqÉkrÉå xÉWûpÉÉaÉ WûÉ xuÉrÉÇpÉÔ AxÉåsÉ AÉÍhÉ rÉÉ AprÉÉxÉ xÉÇzÉÉåkÉlÉÉiÉÔlÉ iÉÑqWûÏ AÉmÉsÉå lÉÉÇuÉ MüÉåhÉirÉÉWûÏ ¤ÉhÉÏ MüÉRÒûlÉ bÉåF zÉMüiÉÉ.

MüÉrÉï¢üqÉÉiÉ pÉÉaÉ bÉåiÉsrÉÉoÉ¬sÉÉ ÍqÉVûhÉÉUÏ AÉÍjÉïMü qÉSiÉ

rÉÉ AprÉÉxÉ¢üqÉÉqÉkrÉå pÉÉaÉ bÉåiÉsrÉÉoÉ¬sÉ iÉÑqWûÉxÉ MüÉåhÉiÉÏWûÏ AÉÍjÉïMü qÉSiÉ ÌMÇüuÉÉ pÉåOû uÉxiÉÑ SåhrÉÉiÉ rÉåhÉÉU lÉÉWûÏ.

AprÉÉxÉÉcÉå ÌlÉwMüwÉï MüUhrÉÉcÉå AÉÍkÉMüÉU

rÉÉ AprÉÉxÉ¢üqÉÉcÉå ÌlÉwMüwÉï Wåû Måü. LsÉ. D rÉÑÌlÉuWûÍxÉïOûÏ oÉåVûaÉÉÇuÉ rÉÉÇlÉÉ mÉÉPûÌuÉhrÉÉiÉ rÉåiÉÏsÉ uÉ WûÉ AprÉÉxÉ¢üqÉ iÉmÉÉxÉÑlÉ uÉ mÉëÍxÉ®ÏxÉ SåFlÉ LqÉ QûÏ. WûÏ mÉSÌuÉ mÉÔhÉï MüUhrÉÉcÉÉ WûÉ LMü AÉqÉcÉÉ pÉÉaÉ AÉWåû.

xÉWûqÉiÉÏcÉÉ lÉqÉÑlÉÉ

qÉÏ rÉÉ AprÉÉxÉ¢üqÉÉqÉkrÉå ZÉÉsÉÏ ÌSsÉåsrÉÉ xÉÇoÉÇkÉÏiÉ UåwÉåuÉU xÉWûÏ MüÂlÉ xuÉiÉ:WÕûlÉ pÉÉaÉ bÉåiÉ AÉWåû. qÉÏ rÉÉ AprÉÉxÉ¢üqÉqÉkÉÔlÉ MüÉåhÉirÉÉWûÏ uÉåVûÏ qÉÑ£ü WûÉåuÉÔ zÉMüiÉÉå. qÉÏ qÉÉfÉå MüÉåhÉiÉåWûÏ MüÉrÉSåzÉÏU AÍkÉMüÉU rÉÉ xÉWûqÉiÉÏcrÉÉ lÉqÉÑlrÉÉuÉU xÉWûÏ MåüsrÉÉlÉå aÉqÉuÉÔlÉ

oÉxÉsÉåsÉÉå lÉÉWûÏ. qÉÏ ZÉÉsÉÏ xÉWûÏ MüUhÉÉU xÉÉÇaÉiÉÉå ÌMü, WûÉ xÉÇmÉÔhÉï lÉqÉÑlÉÉ TüÉqÉï qÉÏ uÉÉcÉÔlÉ mÉÉWûÏsÉÉ AÉWåû ÌMÇüuÉÉ qÉsÉÉ iÉÉå uÉÉcÉÔlÉ SÉZÉÌuÉsÉÉ AÉWåû uÉ qÉÉfrÉÉ pÉÉwÉåqÉkrÉå iÉÉå qÉsÉÉ xÉqÉeÉÉuÉÔlÉ xÉÉÇÌaÉiÉsÉÉ AÉWåû uÉ qÉÉfÉå xÉÇmÉÑhÉï zÉÇMüÉxÉqÉÉkÉÉlÉ fÉÉsÉåsÉå AÉWåû. qÉsÉÉ rÉÉ lÉqÉÑlrÉÉ TüÉqÉï cÉÏ MüÉãmÉÏ SåhrÉÉiÉ rÉåDsÉ.

rÉÉ AprÉÉxÉ¢üqÉÉiÉ pÉÉaÉ bÉåhrÉÉÄrÉÉïcÉÏ ÌMÇüuÉÉ irÉÉcrÉÉiÉTåïü irÉÉcÉå mÉëÌiÉÌlÉkÉÏiuÉ MüUhrÉÉÄrÉÉïÇcÉÏ **xÉWûÏ/QûÉurÉÉ WûÉiÉÉcÉÉ AÇaÉPûÉ**

AprÉÉxÉÉiÉ pÉÉaÉ bÉåhrÉÉÄrÉÉïcÉå lÉÉÇuÉ:

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pÉÉaÉ bÉåhÉÉrÉÉïcÉÏ xÉWûÏ / QûÉurÉÉ WûÉiÉÉcÉÉ

AÇaÉPûÉ:_____

xÉWûÏ ÌMÇüuÉÉ QûÉurÉÉ WûÉiÉÉcÉÉ AÇaÉPûÉ:

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xÉÉ¤ÉÏSÉUÉcÉÏ xÉWûÏ: _____

xÉWûÏ ÌMÇüuÉÉ QûÉurÉÉ WûÉiÉÉcÉÉ AÇaÉPûÉ:

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iÉÉUÏZÉ uÉ xjÉVû:

iÉÉUÏZÉ: **xjÉVû:**

PREFORMA FOR STUDY OF RELATIONSHIP BETWEEN HBA1C AND MEAN PLATELET VOLUME IN TYPE 2 DIABETES MELLITUS PATIENTS-A ONE YEAR CROSS SECTIONAL HOSPITAL BASED STUDY

1. Serial number:
2. Name:
3. Age
4. Sex:
5. Date of admission:
6. Religion:
7. In patient /Out patient number:
8. Address:
9. Occupation:
10. Date of discharge/Outcome:

Chief complaints

History of presenting illness (Including history specific to Type 2 DM)

Past history

Treatment history

Received any treatment for similar complaints in the past

General physical examination

Pallor:	Yes/No
Icterus:	Yes/No
Lymphadenopathy:	Yes/No
Edema:	Yes/No
Markers of Insulin resistance (Acanthosis Nigricans):	Yes/No

Vital signs

Pulse:

Blood Pressure:

Respiratory rate:

Temperature:

Systemic examination

Respiratory system:

Cardiovascular system:

Per abdomen:

Central nervous system:

Investigations like

1. Mean platelet volume:
2. Haemoglobin
3. Urea:
4. Creatinine:
5. Fasting blood sugar:
6. HbA1c
7. Fundoscopy
8. ECG (if required)

ANNEXURE III – KEY TO MASTER CHART

A	-	Absent
AB	-	Abnormal
B	-	Both Oral hypoglycaemic agent and insulin
B/L	-	Bilateral
BP	-	Blood pressure
bpm	-	Beats per minute
DLM	-	Diet and lifestyle modification
DOA	-	Date of admission
DOD	-	Date of Discharge
ECG	-	Electrocardiogram
F	-	Female
fL	-	Femto Litre
gm	-	Gram
Gr	-	Grade
HTN	-	Hypertension
IN	-	Insulin
LL	-	Lower limb
Lt	-	Left
M	-	Male
mg/dL	-	Milligrams per deciliter
min	-	Minute
mm Hg	-	Millimeters of mercury
N	-	Normal

ND	-	Newly detected
NPDR	-	Non proliferative diabetic retinopathy
O	-	Oral hypoglycaemic agent
P	-	Present
Pr	-	Poor
PDR	-	Proliferative diabetic retinopathy
RET	-	Retinopathy
Rt	-	Right

Serial number	IP/OP Number	Age (Years)	Sex	Occupation	Chief complaints		History											General physical examination						Systemic examination				Investigations										
							Diabetic			Past		Personal			Treatment							Vital signs																	
					Chief complaint	Duration (days)	Duration of diabetes (Years)	Microvascular complications	Peripheral vascular disease	Hypertension	Coagulation disorders	Alcohol consumption	Smoking	Tobacco chewing	Antithrombotic medications	Diabetic medication	Treatment compliance	Pallor	Icterus	Lymphadenopathy	Edema	Markers of insulin resistance (Acanthosis Nigricans)	Pulse (bpm)	BP Systolic (mm Hg)	BP Diastolic (mm Hg)	Respiratory rate (/min)	Temperature (N/AB)	Cardiovascular system	Respiratory system	Central nervous system	Per abdomen	Haemoglobin (gm%)	Mean platelet volume (fL)	HbA1C (%)	Urea (mg/dl)	Creatinine (mg/dl)	Fasting blood sugar (mg/dl)	Fundoscopy	ECG	DOA	DOD
1	779865	60	F	Housewife	Giddiness	8	20	P	A	A	A	A	A	A	A	B	G	A	A	A	A	A	78	100	70	16	N	N	N	AB	N	11.1	8.2	19.9	63	2.2	186	B/L PDR	AB	1 January 2017	03 January 2017
2	780665	71	F	Business	Giddiness	4	ND	A	A	P	A	A	A	A	A	DLM	G	A	A	A	A	A	80	110	80	19	AB	AB	AB	AB	N	11.5	8	6.7	35	1	110	B/L Gr 2 HTN RET with Rt eye moderate NPDR	AB	1 January 2017	17 January 2017
3	778906	50	F	Business	breathlessness	30	15	A	A	P	A	A	A	A	A	DLM	G	A	A	A	A	A	84	106	60	16	N	N	AB	N	N	11.9	11.4	7.6	62	2.2	114	B/L Gr 3 HTN RET with Moderate NPDR	AB	18 January 2017	24 January 2017
4	785981	42	F	Housewife	Chest pain on and off	90	ND	A	A	A	A	A	A	A	A	DLM	G	A	A	A	A	P	88	130	70	14	N	AB	N	N	N	12.0	10.2	12.9	16	1	114	N	AB	30 January 2017	06 February 2017
5	787740	70	F	Housewife	Loose stool, vomiting	5	11	P	A	P	A	A	A	A	A	B	Pr	A	A	A	A	A	76	130	70	14	N	N	AB	N	N	12.5	9.2	12.8	46	2	96	Left eye mild NPDR	AB	9 February 2017	03 March 2017
6	788577	49	M	Business	B/L LL swelling	30	20	P	A	P	A	A	A	A	A	O	G	A	A	A	A	P	88	140	70	14	N	N	N	N	N	13.5	9.8	12	65	2.5	120	B/L Gr 3 HTN RET with B/L mild NPDR	AB	13 February 2017	05 March 2017
7	775608	47	F	Housewife	B/L LL swelling	90	10	A	A	A	A	A	A	A	A	IN	G	A	A	A	A	A	76	110	80	18	N	N	N	N	N	12.8	8.4	7.6	17	0.6	180	B/L Moderate NPDR	AB	15 February 2017	24 February 2017
8	789853	46	M	Unemployed	Generalised weakness	120	0.5	P	A	A	A	A	A	A	A	O	G	A	A	A	A	A	78	110	80	16	N	N	N	N	N	13.0	9.8	13.4	25	1.4	118	B/L Macular oedema	N	20 February 2017	24 February 2017
9	789819	72	M	Retired	Breathlessness on exertion	120	15	P	A	P	A	A	A	A	A	B	G	A	A	A	A	A	76	130	70	14	N	AB	AB	N	N	13.1	9.2	7.3	125	5	95	Rt eye moderate NPDR with Gr 2 HTN RET	AB	20 February 2017	03 March 2017
10	792389	55	M	Farmer	Loss of appetite	14	6	P	A	A	A	A	A	A	A	IN	G	A	A	A	A	A	88	124	70	18	AB	N	N	N	N	13.3	9.4	12.5	65	3	114	Rt. Eye moderate NPDR	N	6 March 2017	16 March 2017
11	792446	72	F	Housewife	Fever with chills	2	10	P	A	A	A	A	A	A	A	O	G	A	A	A	A	A	98	130	90	14	N	N	N	N	N	12.9	8.3	8.1	95	3	110	B/L Eye moderate NPDR	AB	6 March 2017	17 March 2017
12	794629	29	M	Farmer	Chest pain	7	7	A	A	A	A	A	A	A	A	O	G	A	A	A	A	P	60	122	80	14	N	N	N	N	N	13.4	7.5	6.3	21	0.6	180	N	AB	17 March 2017	20 March 2017
13	796013	74	M	Business	Breathlessness	365	3	A	A	P	A	A	A	A	A	DLM	G	A	A	A	A	A	82	140	90	18	N	N	AB	N	N	13.8	9.3	7.5	29	1	190	B/L severe PDR	AB	24 March 2017	14 April 2017
14	795974	48	F	Housewife	Chest pain	1	3	P	A	A	A	A	A	A	A	O	Pr	A	A	A	A	P	88	130	90	20	N	AB	N	N	N	11.6	9.8	8.6	22	1.2	128	B/L mild NPDR	N	24 March 2017	27 March 2017
15	797192	46	M	Farmer	Fever with chills	20	ND	A	A	A	A	A	A	A	A	DLM	G	A	A	A	A	A	86	120	80	16	N	N	AB	N	N	14.0	8.5	7	31	1.7	116	B/L mild NPDR	N	31 March 2017	08 April 2017
16	797443	59	M	Service	Pain abdomen	1	8	A	A	P	A	A	A	A	A	IN	Pr	A	A	A	A	A	70	110	80	18	N	N	N	N	AB	13.9	8.7	10	50	2.2	124	B/L Gr 1 HTN RET with mild NPDR	AB	1 April 2017	04 April 2017
17	797609	42	M	Farmer	Multiple joint pain	15	ND	A	A	A	A	A	A	A	A	DLM	G	A	A	A	A	A	68	110	70	18	N	AB	N	N	N	13.1	9.8	10.6	23	0.7	112	N	AB	3 April 2017	08 April 2017
18	797674	70	M	Service	Tingling and Numbness	8	ND	P	A	A	A	A	A	A	A	DLM	G	A	A	A	A	A	102	140	80	18	N	N	N	AB	N	13.6	10.4	14.6	45	1.4	118	B/L mild NPDR	AB	3 April 2017	08 April 2017
19	798098	40	M	Service	Backache	2	4	A	A	A	A	A	A	A	A	O	G	A	A	A	A	P	90	140	90	18	N	N	N	N	N	13.4	9.5	8.2	21	0.9	189	B/L mild NPDR	N	5 April 2017	11 April 2017
20	798558	60	F	Housewife	Chest pain	15	2	P	A	A	A	A	A	A	A	O	G	A	A	A	A	A	68	110	70	18	N	AB	N	N	N	11.8	11.4	7.8	23	0.7	180	B/L SENILE IMMATURE CATARACT	AB	7 April 2017	08 April 2017
21	798642	50	F	Housewife	Headache	2	5	A	A	A	A	A	A	A	A	O	G	A	A	A	A	A	80	140	80	18	N	N	N	N	N	11.5	11.4	8.7	24	0.6	200	B/L Gr 1 HTN RET	AB	8 April 2017	10 April 2017
22	798671	70	F	Housewife	Bilateral knee pain	7	10	P	A	P	A	A	A	A	A	O	Pr	A	A	P	A	A	84	160	90	18	AB	N	N	N	N	12.1	10.2	10.6	63	1.6	168	B/L Gr 3 HTN RET with moderate NPDR	AB	8 April 2017	13 April 2017
23	799459	50	M	Service	Dizziness	1	5	A	A	A	A	A	A	A	A	B	G	A	A	P	A	A	84	126	84	16	AB	N	N	N	N	13.2	8.7	13.9	16	0.6	132	B/L moderate NPDR	N	12 April 2017	19 April 2017
24	802276	57	M	Service	Fever with bodyache	2	10	P	A	P	A	A	A	A	A	O	Pr	A	A	A	A	P	90	140	90	20	AB	N	N	AB	N	13.8	9	7.6	48	2.76	148	B/L mild NPDR	AB	24 April 2017	29 April 2017
25	663446	63	F	Housewife	Right knee pain	7	10	P	A	P	A	A	A	A	A	O	G	A	A	A	A	P	90	150	80	18	AB	N	N	N	N	12.5	8.2	11.8	21	0.6	98	B/L Gr 1 HTN RET with mild NPDR	AB	27 April 2017	01 May 2017
26	802423	40	F	Housewife	Fever	4	1	P	A	P	A	A	A	A	A	O	G	A	A	A	A	P	82	152	90	14	N	N	AB	N	N	11.6	9.8	10.3	22	0.57	127	Rt eye mild NNPDR	AB	28 April 2017	29 April 2017
27	805464	65	F	Housewife	Fever	4	6	A	A	A	A	A	A	A	A	O	G	A	A	A	A	P	110	104	80	22	AB	N	AB	N	N	11.1	11	13.5	18	0.52	150	B/L moderate NPDR	N	16 May 2017	23 May 2017
28	805808	46	M	Unemployed	LL stiff	1	5	P	A	A	A	A	A	A	A	IN	G	A	P	A	A	A	88	140	80	16	N	N	N	AB	N	14.1	10.1	9	22	0.9	182	N	N	17 May 2017	20 May 2017
29	806329	54	F	Housewife	Backache	2	5	P	A	A	A	A	A	A	A	O	G	A	A	A	A	P	64	98	60	14	N	N	N	N	N	11.0	8	8.1	22	0.8	138	B/L moderate NPDR	N	18 May 2017	24 May 2017
30	806555	59	F	Housewife	Micturition	7	5	P	A	A	A	A	A	A	A	B	G	A	A	P	P	A	86	106	80	18	N	N	N	N	AB	12.0	10	6.9	18	0.5	128	B/L mild NPDR	N	22 May 2017	26 May 2017
31	807087	40	F	Housewife	Headache (Left sided)	8	4	P	A	A	A	A	A	A	A	O	Pr	A	A	A	A	P	78	140	80	16	AB	N	N	N	N	12.3	9.8	11.3	28	0.6	176	N	N	24 May 2017	29 May 2017
32	810530	54	F	Housewife	Breathlessness on exertion	120	5	P	A	P	A	A	A	A	A	IN	Pr	A	A	A	A	A	72	156	80	16	N	AB	AB	N	N	12.5	9.5	14	26	0.98	156	B/L severe NPDR	AB	9 June 2017	14 June 2017
33	811469	43	M	Professional	Pain abdomen and vomiting	2	5	P	A	P	A	A	A	A	A	B	Pr	A	A	A	A	P	78	134	80	16	N	N	N	N	AB	14.5	8.7	12.8	41	2.5	98	Rt eye Gr 1 HTN RET with moderate NPDR	AB	16 June 2017	19 June 2017
34	811629	70	F	Housewife	Sore throat	2	15	P	A	A	A	A	A	A	A	O	G	A	A	A	A	A	64	98	60	18	N	N	N	N	N	12.4	6.9	6.1	40	0.5	156	B/L moderate NPDR	N	17 June 2017	18 June 2017
35	811747	65	F	Housewife	Fever with chills	5	6	A	A	P	A	A	A	A	A	O	G	A	A	A	A	A	`100	160	98	14	AB	AB	AB	N	N	11.6	8.3	10.3	127	4.07	124	B/L moderate NPDR with Gr 1 HTN RET	AB	17 June 2017	21 June 2017
36	811654	60	M	Farmer	Giddiness	4	ND	A	A	P	A	A	A	A	A	DLM	G	A	A	A	A	P	78	146	80	22	N	N	N	N	N	14.2	8.7	9.7	40	2.4	156	Lt eye mild NPDR	AB	17 June 2017	21 June 2017
37	811911	48	F	Service	Fever	90	2	A	A	A	A	A	A	A	A	DLM	G	A	A	A	P	P	82	130	80	24	AB	N	N	N	AB	11.2	11	15	40	1.1	175	B/L severe NPDR	N	19 June 2017	28 June 2017
38	811959	54	M	Service	Routine check up	0	4	P	A	P	A	A	A	A	A	O	Pr	A	A	A	A	P	82	150	80	16	N	N	N	AB	N	13.1	9.8	9.8	26	1	250	Rt eye Gr 3 HTN RET	AB	19 June 2017	29 June 2017
39	812517	68	F	Housewife	Breathlessness	7	13	A	A	P	A	A	A	A	A	B	G	A	A	A	A	P	82	144	80	14	N	N	AB	N	N	11.3	8.6	7.7	47	1.34	186	B/l mild NPDR Gr 3 HTN RET	AB	21 June 2017	25 June 2017
40	813107	45	F	Housewife	Tingling and Numbness	30	5	P	A	P	A	A	A	A	A	O	Pr	A	A	A	A	P	78	156	80	15	N	N	N	AB	N	11.8	8.7	17.3	17	0.42	184	B/L Gr 2 HTN RET with mild NPDR	AB	25 June 2017	08 July 2017
41	813337	53	F	Housewife	Dry cough	4	ND	P	A	A	A	A	A	A	A	DLM	G	A	A	A	A	P	68	110	60	16	N	N	AB	N	N	11.7	9.9	13.7	76	3.4	114	B/L moderate NPDR (Rt > Lt)	N	27 June 2017	28 June 2017
42	813422	47	M	Business	Chest pain on and off	3	5	P	A	A	A	A	A	A	A	O	Pr	A	A	A	A	P	72	102	80	18	N	AB	N	N	N	13.0	9.8	9.5	29	0.78	178	B/L moderate NPDR	AB	27 June 2017	29 June 2017
43	877508	67	M	Business	Seizures	4	8	P	A	A	A	A	A	A	A	O	G	A	A	A	A	A	64	118	80	16	N	N	N	N	AB	13.9	7.3	6	60	2.8	98	N	N	27 June 2017	30 June 2017
44	813857	61	M	Unemployed	LL pain	7	16	P	A	A	A	A	A	A	A	B	G	A	A	A	A	A	80	110	70	18	AB	N	N	N	N	14.0	8.4	9.2	19	1	180	B/L mild NPDR	N	29 June 2017	19 July 2017
45	814275	60	F	Housewife	Upper abdominal pain	1	14	P	A	P	A	A	A	A	A	B	G	A	A	A	A	A	72	152	96	14	N	N	N	N	AB	11.6	9.6	7.5	43	1.42	128	B/L mild NPDR with Gr 1 HTN RET	AB	1 July 2017	15 July 2017
46	814436	62	M	Service	Exertional dyscomfort	1	10	A	A	P	A	A	A	A	A	IN	G	A	A	A	A	A	86	148	90	18	N	AB	AB	N	N	13.8	9.8	8.2	68	1.57	128	B/L Gr 2 HTN RET with Rt eye moderate NPDR	AB	3 July 2017	11 July 2017
47	813757	47	M	Farmer	Dry cough	7	2.5	P	A	P	A	A	A	A	A	IN	G	A	A	A	A	A	80	156	88	18	N	N	AB	N	N	13.1	9	6.7	26	1.4	148	B/L mild NPDR	AB	4 July 2017	15 July 2017
48	815318	70	M	Unemployed	Generalised weakness	14	10	A	A	A	A	A	A	A	A	IN	G	A	A	A	A	P	76	100	88	18	N	N	N	N	N	13.5	9.7	6.2	21	1.40	124	Gr 1 HTN RET	N	6 July 2017	10 July 2017
49	815581	65	M	Business	Generalised weakness	30	10	P	A	P	A	A	A	A	A	O	Pr	A	A	A	A	A	78	156	94	14	N	N	N	N	AB	13.3	8.2	10.2	24	1.5	196	B/L PDR (Rt > Lt) with Gr 2 HTN RET	AB	8 July 2017	10 July 2017
50	815548	69	M	Professional	Headache (Left sided)	7	4	P	A	P	A	A	A	A	A	O	G	A	A	A	A	A	82	160	80	16	N	N	N	N	AB	13.4	8.9	6.4	82	2.97	174	Rt eye severe NPDR with Gr 1 HTN RET	AB	8 July 2017	27 July 2017
51	815968	54	M	Farmer	Bleeding per rectum	7	4	A	A	A	A	A	A	A	A	O	G	A	A	A	A	A	74	110	80	18	N	N	N	N	N	13.6	7.6	6.7	24	1.03	148	N	N	10 July 2017	23 July 2017
52	814861	59	M	Service	Tingling in both legs	14	4	P	A	P	A	A	A	A	A	B	Pr	A	A	A	P	A	80	150	100	18	N	N	N	N	AB	13.7	8.7	14	18	0.5	165	B/L moderate NPDR with Gr 2 HTN RET	AB	11 July 2017	12 July 2017
53	816258	52	M	Farmer	Fever	14	ND	A	A	P	A	A	A	A	A	DLM	G	A	A	A	A	A	78	162	100	20	N	N	N	N	N	13.1	9.6	10.8	48	1.2	138	N	AB	11 July 2017	14 July 2017
54	816317	50	F	Housewife	Breathlessness	14	2.5	A	A	P	A	A	A	A	A	B	Pr	A	A	A	P	P	110	180	92	16	N	AB	AB	N	N	12.6	9.4	13	32	0.7	114	B/L Gr 3 HTN RET with B/L mild NPDR	AB	11 July 2017	18 July 2017
55	816146	40	F	Housewife	Dry cough	2	ND	A	A	P	A	A	A	A	A	DLM	G	A	A	A	A	P	112	90	64	24	N	N	AB	N	N	12.3	9.8	12.9	16	1.1	114	N	N	11 July 2017	15 July 2017

Serial number	IP/OP Number	Age (Years)	Sex	Occupation	Chief complaints		History											General physical examination						Systemic examination				Investigations											
							Diabetic			Past		Personal			Treatment							Vital signs																	
																						BP																
					Chief complaint	Duration (days)	Duration of diabetes (Years)	Microvascular complications	Peripheral vascular disease	Hypertension	Coagulation disorders	Alcohol consumption	Smoking	Tobacco chewing	Antithrombotic medications	Diabetic medication	Treatment compliance	Pallor	Icterus	Lymphadenopathy	Edema	Markers of insulin resistance (Acanthosis Nigricans)	Pulse (bpm)	Systolic (mm Hg)	Diastolic (mm Hg)	Respiratory rate (/min)	Temperature (N/AB)	Cardiovascular system	Respiratory system	Central nervous system	Per abdomen	Haemoglobin (gm%)	Mean platelet volume (fL)	HbA1C (%)	Urea (mg/dl)	Creatinine (mg/dl)	Fasting blood sugar (mg/dl)	Fundoscopy	ECG	DOA	DOD
56	816842	60	F	Housewife	Lethargy	7	2	A	A	P	A	A	A	A	A	O	Pr	A	A	A	A	A	82	106	82	18	N	N	N	N	N	12.1	9.1	11.3	63	1.5	114	B/L GrADE 2 HTN RET	AB	14 July 2017	17 July 2017
57	817104	58	M	Service	Loose stool	4	4	P	A	A	A	A	A	A	A	B	G	A	A	A	A	A	114	86	60	18	N	N	N	N	AB	13.8	10.4	13	30	0.63	186	B/L moderate NPDR	N	15 July 2017	19 July 2017
58	818243	55	M	Farmer	Chest pain	7	4	A	A	P	A	A	A	A	A	B	G	A	A	A	A	A	78	128	70	16	N	AB	N	N	N	13.6	9.8	10.4	43	1.2	104	B/L mild NPDR	N	21 July 2017	28 July 2017
59	818260	65	M	Farmer	Decreased appetite	60	20	P	A	P	A	A	A	A	A	O	Pr	A	A	A	A	A	68	162	80	16	N	N	N	N	AB	13.9	8.3	8.2	71	2.8	94	B/L Gr 3 HTN RET	AB	21 July 2017	24 July 2017
60	818429	60	F	Housewife	Multiple joint pain	4	15	P	A	A	A	A	A	A	A	B	G	A	A	A	A	A	112	114	70	22	N	N	N	N	N	11.6	8	6.6	35	1	174	B/L mild NPDR	N	22 July 2017	25 July 2017
61	818538	42	M	Service	Altered sensorium	1	2	P	A	A	A	A	A	A	A	B	G	A	A	A	A	A	76	122	86	16	N	N	N	AB	N	14.0	9.1	10.2	10	0.6	118	N	N	23 July 2017	31 July 2017
62	813453	45	F	Housewife	Generalised weakness	14	2	A	A	A	A	A	A	A	A	IN	G	A	A	A	A	P	76	100	70	18	N	N	N	N	N	11.2	11.4	13.7	30	0.90	110	Gr 1 HTN RET	N	24 July 2017	28 July 2017
63	818947	60	F	Housewife	Weight gain	90	6	P	A	P	A	A	A	A	A	IN	G	A	A	A	A	P	54	176	90	16	N	N	N	N	N	12.2	8.1	9.8	40	2.73	142	B/L severe NPDR	AB	25 July 2017	30 July 2017
64	819332	71	M	Unemployed	Dysuria	4	10	P	A	P	A	A	A	A	A	B	G	A	A	A	A	P	80	100	60	20	N	N	N	N	AB	14.5	8.1	7.9	22	0.8	176	B/L Gr 2 HTN RET with moderate NPDR	AB	27 July 2017	01 August 2017
65	819439	55	M	Business	Backache	60	ND	A	A	A	A	A	A	A	A	DLM	G	A	A	A	P	A	88	120	90	18	N	N	N	AB	N	14.6	9	7.4	19	1.6	128	N	N	28 July 2017	05 August 2017
66	819575	60	F	Housewife	Fever	2	4	P	A	A	A	A	A	A	A	IN	G	A	A	A	A	A	82	110	80	16	AB	N	N	N	N	12.6	8.4	6.3	47	0.98	116	mild Rt eye NPDR	N	29 July 2017	31 July 2017
67	819990	57	M	Business	Loose stools and vomiting	2	4	A	A	A	A	A	A	A	A	O	Pr	A	A	A	A	P	86	110	80	16	N	N	N	N	AB	14.2	11.1	9.7	46	2.39	98	N	N	31 July 2017	08 August 2017
68	820097	62	F	Housewife	Upper backache	7	5	A	A	P	A	A	A	A	A	DLM	G	A	A	A	A	P	100	122	80	18	AB	N	N	N	N	12.1	10.2	8.6	18	3.52	124	B/L Gr 1 HTN RET with B/L moderate NPDR	AB	1 August 2017	06 August 2017
69	820616	70	M	Farmer	Aletered sensorium	2	15	P	A	P	A	A	A	A	A	O	Pr	A	A	A	A	A	76	104	60	14	N	N	N	N	AB	14.1	8.3	7.2	10	0/67	146	Rt eye severe NPDR	AB	3 August 2017	07 August 2017
70	820803	65	F	Housewife	Backache	90	15	P	A	A	A	A	A	A	A	B	Pr	A	A	A	A	A	64	104	72	18	N	AB	N	N	N	11.4	8	16.4	28	1.7	114	B/L moderate NPDR	N	4 August 2017	09 August 2017
71	822092	56	F	Housewife	Fever and cough	7	5	P	A	A	A	A	A	A	A	O	Pr	A	A	A	A	A	82	124	80	16	AB	N	AB	N	N	11.9	8.4	8.6	37	1.6	118	Lt eye mild NPDR	N	10 August 2017	15 August 2017
72	755424	65	F	Housewife	Breathlessness	20	8	P	A	P	A	A	A	A	A	B	G	A	A	A	A	A	86	130	80	18	N	AB	AB	AB	N	12.0	9.7	6.8	38	0.57	124	B/L Gr 2 HTN RET with B/L mild NPDR	AB	12 August 2017	17 August 2017
73	822941	70	F	Housewife	Fever with pain abdomen	4	14	P	A	P	A	A	A	A	A	B	Pr	A	A	A	A	A	64	104	60	22	AB	N	N	N	AB	11.1	9.9	14.4	90	2.36	176	B/L moderate NPDR (Rt > Lt)	AB	15 August 2017	16 August 2017
74	823045	70	M	Service	Breathlessness	60	4	P	A	A	A	A	A	A	A	IN	G	A	A	A	P	P	82	100	80	16	N	AB	AB	N	N	13.3	9.2	8.1	32	0.9	150	B/L moderate NPDR	AB	16 August 2017	21 August 2017
75	823962	46	M	Business	Headache	7	8	P	A	P	A	A	A	A	A	O	Pr	A	A	P	A	A	80	110	80	18	N	N	N	AB	N	14.2	9.1	10.7	18	0.72	118	Gr 2 HTN RET	AB	20 August 2017	24 August 2017
76	824319	70	F	Unemployed	Breathlessness	548	ND	A	A	A	A	A	A	A	A	DLM	G	A	A	A	A	P	88	110	80	18	N	AB	N	N	N	12.0	9.2	6.5	27	1.5	140	B/L moderate NPDR	N	22 August 2017	28 July 2017
77	824958	55	M	Unemployed	Retrosternal chest pain	1	4	P	A	P	A	A	A	A	A	O	G	A	A	A	A	P	78	98	60	18	N	AB	AB	N	N	13.6	7.6	6.5	21	0.9	148	B/L mild NPDR	AB	26 August 2017	30 August 2017
78	825607	56	M	Service	Itching all over body	4	6	P	A	A	A	A	A	A	A	B	G	A	A	A	A	A	84	110	70	14	N	N	N	N	N	13.2	5.6	6.3	21	0.74	98	B/L mild NPDR	N	30 August 2017	01 September 2017
79	826410	44	F	Housewife	Pain abdomen	3	1	P	A	A	A	A	A	A	A	O	G	A	A	A	A	P	78	110	80	18	N	N	N	N	AB	12.1	10	12.3	10	0.37	110	B/L mild NPDR	N	3 September 2017	08 September 2017
80	827126	62	F	Housewife	Generalised weakness	3	6	A	A	P	A	A	A	A	A	O	G	A	A	A	A	A	78	156	100	18	N	N	N	N	N	11.6	9.8	11.9	21	0.9	118	B/L severe NPPDR	AB	6 September 2017	08 September 2017
81	812054	65	M	Service	Fever on and off	30	4	P	A	A	A	A	A	A	A	DLM	G	A	A	A	A	A	82	110	70	14	N	N	N	N	N	13.3	9.8	10.5	65	2.3	212	B/L severe NPDR (Rt > Lt)	N	7 September 2017	08 September 2017
82	829153	66	M	labour	Exertional chest discomfort	1	10	P	A	P	A	A	A	A	A	O	Pr	A	A	A	A	A	76	148	92	16	N	AB	N	N	N	14.1	8.4	6.1	201	2.48	176	B/L Gr 2 HTN RET with moderate NPDR	AB	14 September 2017	17 September 2017
83	830285	54	M	Service	Giddiness and Headache	2	4	P	A	A	A	A	A	A	A	O	G	A	A	A	A	A	86	118	90	16	N	N	N	N	AB	13.9	9.7	6.9	72	2.1	176	N	N	23 September 2017	28 September 2017
84	830842	60	M	Service	Upper abdominal pain	2	13	P	A	P	A	A	A	A	A	B	Pr	A	A	A	A	P	80	180	94	16	N	N	N	N	AB	14.6	8.4	12	26	1.4	114	B/L severe NPDR with Gr 2 HTN RET	AB	23 September 2017	03 October 2017
85	841395	38	M	Service	Loose stools and vomiting	5	2	A	A	A	A	A	A	A	A	O	Pr	A	A	A	P	P	76	120	80	16	N	N	N	N	N	14.0	10.4	13.1	16	0.7	116	N	N	15 October 2017	20 October 2017
86	835420	40	M	Service	Altered sensorium	1	3	A	A	P	A	A	A	A	A	DLM	G	A	A	A	A	P	80	140	80	20	N	N	N	AB	N	13.8	8.4	5.5	20	0.56	120	N	N	16 October 2017	21 October 2017
87	834244	40	F	Housewife	Generalised weakness	365	2	P	A	P	A	A	A	A	A	B	G	A	A	P	A	P	70	150	80	18	AB	N	P	N	N	11.1	7.9	6.5	82	2.3	124	N	N	24 October 2017	25 October 2017
88	837478	45	F	Housewife	Generalised weakness	30	5	A	A	P	A	A	A	A	A	DLM	G	A	A	A	A	A	82	156	90	14	N	N	N	N	AB	11.8	10.1	9.1	22	1	126	N	N	26 October 2017	29/20/2017
89	838129	62	F	Service	Fever with cough	4	3	A	A	A	A	A	A	A	A	O	G	A	A	A	P	P	110	100	60	20	AB	N	AB	N	N	11.4	8	5.5	20	0.56	118	B/L moderate NPDR	N	30 October 2017	02 November 2017
90	838585	35	F	Business	Cough	4	0.5	A	A	A	A	A	A	A	A	DLM	G	A	A	A	A	P	88	110	80	18	AB	N	AB	N	N	11.8	9.3	10	137	1.7	180	Rt eye mild NPDR	N	1 November 2017	06 November 2017
91	838656	30	M	Professional	Headache	2	ND	P	A	A	A	A	A	A	A	DLM	G	A	A	A	A	A	92	104	80	16	N	N	N	N	N	13.1	7.4	4.8	16	1	114	N	N	2 November 2017	08 November 2017
92	839642	60	F	Housewife	Pain abdomen	2	6	A	A	A	A	A	A	A	A	O	G	A	A	A	A	A	86	110	70	18	N	N	N	N	AB	11.7	8.4	13.2	26	0.8	118	B/L mild NPDR	N	7 November 2017	18 November 2017
93	842302	58	F	Business	Tingling and Numbness	1095	4	P	A	P	A	A	A	A	A	B	G	A	A	A	A	P	74	140	80	18	N	N	N	N	AB	11.5	9.8	11.2	22	1.6	118	B/L Gr 1 HTN RET	AB	21 November 2017	25 November 2017
94	842610	66	F	Housewife	Decreased vision right eye	90	8	P	A	A	A	A	A	A	A	O	G	A	A	A	A	P	88	140	80	16	N	N	N	AB	N	12.0	9.8	10.4	24	1.7	104	Rt eye moderate NPDR	AB	22 November 2017	29 November 2017
95	843419	45	F	Housewife	Generalised weakness	7	2	P	A	P	A	A	A	A	A	O	G	A	A	A	A	A	78	120	80	18	N	N	N	N	N	12.1	8.6	12.6	20	1	154	Lt eye Gr 1 HTN RET with mild NPDR	AB	27 November 2017	01 December 2017
96	844534	38	M	Business	Headache	3	2	A	A	A	A	A	A	A	A	O	Pr	A	A	A	A	A	82	110	70	14	N	N	N	N	N	13.0	11	10.4	44	1.5	152	Rt eye moderate NPDR	N	1 December 2017	07 December 2017
97	844636	63	M	Business	cough with sputum	10	10	P	A	A	A	A	A	A	A	O	G	A	A	A	A	P	80	110	70	18	N	N	AB	N	N	13.9	8.2	12.9	108	1.44	118	B/L moderate NPDR	N	2 December 2017	04 December 2017
98	877613	67	F	Unemployed	cough with sputum	90	ND	P	A	A	A	A	A	A	A	DLM	G	A	A	A	A	A	80	130	90	16	N	N	N	N	N	12.6	10	5.9	20	0.8	124	N	N	18 December 2017	30 December 2017
99	848714	43	F	Housewife	Fever	8	5	A	A	P	A	A	A	A	A	B	G	A	A	A	A	A	80	130	80	18	N	N	N	N	AB	12.4	9.4	10.7	28	1.3	118	B/L mild NPDR	AB	19 December 2017	30 December 2017
100	779618	70	F	Retired	Itching all over body	1	0.5	P	A	A	A	A	A	A	A	O	G	A	A	A	A	A	70	132	80	17	N	N	N	N	N	12.5	8.2	13.5	25	0.7	110	B/L severe NPDR	AB	26 December 2017	29 December 2017