

CORRELATION OF GLYCATED HAEMOGLOBIN, BLOOD GLUCOSE LEVEL AND LIPID PROFILE IN DIABETIC, PREDIABETIC AND NORMAL HEALTHY INDIVIDUALS

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ABSTRACT:

Introduction: Lipid abnormalities are one of the major risk factors for cardiovascular disease in diabetes mellitus Type 2. The aim of the study was to understand the correlation between FBS, PPBS, HBA1c and lipid parameters among the normal, prediabetic and diabetic patients.

Materials and Methods: This is a retrospective cross-sectional study carried out at Laboratory, Ruby Hall Clinic, Pune, Maharashtra, India, to assess the relationship between glycemic control (as reflected by HBA1C) and serum lipid profile in normal ,prediabetic and diabetic subjects which included a total of 110 individuals (75 males; 35 females; mean age years). Venous blood samples were collected from all the patients after at least 8 h fasting.

Results: The sera were analyzed for HBA1C, fasting blood glucose (FBG) Post prandial glucose (PPBG), total cholesterol, triglycerides (TG), high-density lipoprotein (HDL) cholesterol, and low-density lipoprotein (LDL) cholesterol. Significant positive correlation was noted in FBS,PPBS, HbA1c and lipid parameters in diabetic males. No correlation was noted in diabetic and prediabetic females.

Conclusion: The findings of this study clearly indicate that HBA1C is not only a useful biomarker of long-term glycemic control but can be used as predictor of lipid profile in diabetic males.

KEYWORDS: Cholesterol, Diabetes mellitus, Dyslipidemia, Glycated hemoglobin, High-density lipoprotein cholesterol, Low-density lipoprotein cholesterol, Triglycerides

I. INTRODUCTION

In the ancient Sanskrit literature, diabetes mellitus was described as "honey-urine disease," associated with gross emaciation and wasting. Diabetes Mellitus (DM) comprises a group of common metabolic disorders that share the phenotype of hyperglycemia. It is a global endemic with rapidly increasing prevalence in both developing and developed countries [1].Diabetes mellitus (DM) is a group of metabolic diseases characterized by increase blood glucose level resulting from defects in insulin secretion, insulin action or both. Diabetes mellitus is the commonest endocrine metabolic disorder affecting the people all over the world. On the global arena, even though diabetes has been known since antiquity and in the last five decades new discoveries have provided great hopes and there are continuing research efforts to find ways and means to minimize morbidity and mortality which continue unabated [2].The chronic hyperglycemia of diabetes is associated with long- term damage, dysfunction and disturbance in failure of various organs, especially the eyes, kidneys, nerves, heart and blood vessels.End organ damage is the expected final

International Journal of Innovative Research in Science, Engineering and Technology

(An ISO 3297: 2007 Certified Organization)

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consequence of chronic hyperglycemia, which occurs over the long term. The most frequently affected organs being: coronary arteries, cerebrovascular circulation, retinal arteries, renal microvasculature and tiny arteries of the nerves (vasa nervosa). Patients with type-2 diabetes have increased risk of cardiovascular disease (CVD) associated with atherogenic abnormalities and dyslipidemia. Coronary artery disease, especially myocardial infarction is the leading cause of morbidity and mortality worldwide [3]. CVDs are listed as the cause of death in 65% of persons with diabetes. Diabetes acts as an independent risk factor for several forms of CVD. To make matters worse, when patients with diabetes develop clinical CVD, they sustain a worse prognosis for survival than do CVD patients without diabetes. Dyslipidemia is one of the major cardiovascular disease (CVD) risk factors and plays an important role in the progress of atherosclerosis, the underlying pathology of CVD. The prevalence of dyslipidemia in type 2 diabetes is double with respect to the general population (Haffner, 1998)[4]. Dyslipidemia is characterized by raised triglycerides, low high density lipoprotein and high small dense low density lipoprotein particles. It may be present at the diagnosis of type 2 Diabetes mellitus and is a component of the metabolic syndrome. Abnormal serum lipids are likely to contribute to the risk of coronary artery disease in diabetic patients[5]. The body uses cholesterol to help build cells and produce hormones. Too much cholesterol in the blood can build up inside arteries, forming what is known as plaque. Large amounts of plaque increase the chances of having a heart attack or stroke. High-density lipoprotein (HDL) helps remove fat from the body by binding with it in the bloodstream and carrying it back to the liver for disposal. It is sometimes called "good" cholesterol. A high level of HDL cholesterol may lower the chances of developing heart disease or stroke. Low-density lipoprotein (LDL) carries mostly fat and only a small amount of protein from the liver to other parts of the body. It is sometimes called "bad cholesterol." A high LDL cholesterol level may increase the chances of developing heart disease [6]. Very low-density lipoprotein (VLDL) contains very little protein. The main purpose of VLDL is to distribute the triglyceride produced by your liver. A high VLDL cholesterol level can cause the build-up of atherosclerosis in the arteries and increases the risk of heart disease and stroke. Triglycerides are a type of fat the body uses to store energy and give energy to muscles. Only small amounts are found in the blood. Having a high triglyceride level along with high LDL cholesterol may increase the chances of having heart disease more than having only a high LDL cholesterol level [7, 8, 9]. Abnormal serum lipids are likely to contribute to the risk of coronary artery disease in diabetic patients and the determination of the serum lipid levels in people with diabetes is now considered as a standard of the diabetes care [10]. Abnormal lipid profiles and lipoprotein oxidation (especially LDL-C) are more common in diabetes and are aggravated with a poor glycaemic control. The measurement of the lipid profile of diabetic patients is needed to investigate how their lipid metabolism is affected by diabetes, as they have different genetic compositions and life- styles. Glycation occurs over the entire 90-120 day life span of the red blood cell. HbA1c can consequently be interpreted as an average of the blood glucose present over the past 3-4 months. [11] An elevated Hb1Ac levels indicates poor control of blood glucose levels or poor glycemic index. HbA1c is formed by the condensation of glucose with the N-terminal Valine residue of each β -chain of HbA to form an unstable Schiff-base, which is the most widely used biomarker for long-term glycemic status, as well as an independent risk factor for coronary heart disease (CHD) and stroke [12]. Glycated hemoglobin (HbA1c) is routinely used as a diagnostic tool for screening and measuring long term control in diabetic patient. It is an indicator for the mean blood glucose level in diabetic patients, HbA1c predicts the risk for the development of severe diabetic complications in diabetic patients [13]. As per ADA 2017 guidelines, looking at HbA1c values, patients can be categorized into prediabetic (5.7-6.4), diabetic (≥ 6.5) and normal (< 5.7). Hence, present study was undertaken to determine the impact of glycemic control on lipid profile and to know utility of HbA1c as an indirect indicator of dyslipidemia in normal, prediabetic and diabetic individuals.

II. REVIEW OF LITERATURE

Historical Aspects

Glycosylated Hemoglobin (HbA1c)

1958: Hemoglobin A1c was first separated from other forms of hemoglobin by Huisman and Meyering in 1958 using a chromatographic column.

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1966:Holmquist and Schroeder identify five subtypes of hemoglobin A, including HbA1c. **1968:**Rahbar recognizes that HbA1c is elevated in people with diabetes.

1975: Koenig and Cerami suggest that HbA1c is related to metabolic control.

1993: DCCT (Diabetes Control and Complications trial) establishes HbA1c as a valuable clinical marker in people with type 1 diabetes.

1998: UKPDS (The UK Prospective Diabetes Study) establishes HbA1c as a valuable clinical marker in people with type 2 diabetes.

2010: ADA (American Diabetes Association) recommends using the HbA1c test to diagnose diabetes and prediabetes.

One of the first systematic demonstration of HbA1c increase in diabetic patients was made by Trivelli et al. in **1971**. During this period, a number of methods based on various principles (i.e., ion-exchange chromatography, affinity chromatography, electrophoresis, colorimetric assay, immunoassay) and methodologies [i.e., micro- and minicolumns of chromatography, low pressure (LPLC), high pressure liquid chromatography liquid chromatography(HPLC), manual assays, automated assays] were proposed for assaying HbA1c , evaluated and compared[14,15,16,17].

Lipid Profile

In **1815**, French chemist Michel Eugene Chevreul isolated and purified sterol from gallstones. And he named it as "cholesterol."

In **1838**, Louis Rene Lecanu identified the presence of cholesterol in human blood.

In **1852**, Irish Physician Richard Quain observed fatty material deposition in the blood vessels. He attributed this to local modification of nutrition. He linked the fatty heart to lacking energy, improper circulation, and distress breathing. Additionally, chest oppression or uneasiness, coma, syncope, angina pectoris, and sudden death.

In **1854**, Dr. Rudolf Virchow described atherosclerosis as a disease. He considered arterial clog is due to excess cholesterol deposition.

In **1927**, Wieland & Adolf Windaus got Nobel Prize for cholesterol & bile acids structure.

In **1934**, a blood test for cholesterol developed.

In **1932**, Adolf Windaus clarified the structure of cholesterol.

In **1954**, Dr. John Gofman found patients with heart disease had high small density LDL and reduced large HDL.

In **1955**, Doctors Richard Havel, Howard Eder, and Carl Bragdon developed ultracentrifugation technique. They used it to isolate plasma lipoproteins.

Around **1959**, John William Gofman researched on identification, quantification and clinical implications of lipoproteins. He identified three major classes of lipoproteins in serum; they are VLDL, LDL, and HDL.

Kare Berg discovered lipoprotein (a) or Lp (a) in **1963**

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In **1964**, Dr. Konrad Bloch received the Nobel Prize for explaining the cholesterol synthesis. He showed HMG-CoA reductase was the enzyme in this complex synthesis. The cholesterol serves as a precursor for bile acids, sex hormones, and cortisol. Konrad Bloch and Feodor Lynen awarded Nobel Prize for cholesterol and fatty acids metabolism

In **1951**, Doctors David Barr, Edward Russ & Howard Eder analyzed heart patients . They found heart patients have elevated LDL and decreased HDL levels.

In a 10-year study, Gofman and colleagues studied ischemic heart disease patients. They found lower HDL and higher levels of LDL, IDL, and small VLDL.

In **1969**, Dr. Meyer Burstein developed a technique to precipitate chylomicrons, VLDL, and LDL. This method can allow for the measurement of HDL cholesterol (HDL-C). Doctors William Friedewald, Robert Levy, and Donald Fredrickson developed the Friedewald formula. This approach calculates LDL cholesterol (LCL-C) based on fasting total cholesterol, HDL-C, and triglyceride [18].

Haemoglobin A1C (Hba1c)

Glycated Hb is derived from the nonenzymatic addition of glucose to amino groups of haemoglobin.HbA1c is a specific glycated haemoglobin that results from the attachment of glucose to the N-terminal valine of the haemoglobin beta chain.

Total glycated Hb includes all glycated fractions including Hba1c as well as haemoglobin glycated at sites other than the N-terminus of the beta chain.

Glucose combines with Hb continuously and nearly irreversibly during the life span of RBC (120 days).Therefore, glycosylated Hb will be proportional to mean plasma glucose level during previous 6-12 weeks. Hence.it id therefore, free of the large fluctuations that occur daily in blood glucose concentrations[19].

Measurement of Hba1c

Numerous assays have been developed to measure glycated haemoglobin.

The principle of all methods is to separate the glycated and nonglycated forms of haemoglobin. This can be accomplished based on differences in charge (usually by HPLC) or structure (usually immunoassays or boronated affinity chromatography)

The interpretation of Hba1c levels is not intuitively obvious to many patients with DM, who are accustomed to thinking in terms of blood glucose levels.

The American Diabetes Association has called for laboratories to express Hba1c results as estimated average blood glucose (eAG).ADA believes that eAG will be easier for patients to understand and will lead to improved management of DM.

Formula recommended to calculated eAG is

$$eAG \text{ (mg/dl)} = 28.7 \times \text{Hba1c} - 46.7$$

Normal range (ADA2017) guidelines

Hba1c levels for diagnosis of Diabetes Mellitus

- < 5.7% - Normal

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- 5.7-6.4% - High risk / Prediabetes
- $>/=$: Diabetes (to be confirmed by repeating test test on different day).

For monitoring of Diabetes mellitus,HbA1C target goal should be $< 7\%$.

Uses

- Monitoring compliance and long term glucose level control in patients with diabetes
- Index of diabetic control (Direct relationship between poor control and development of complications)
- Predicting development and progression of diabetic microvascular complications
- Possibly for diagnosis of DM.

Increased in

- Chronic renal failure
- Iron deficiency anemia
- Splenectomy
- Increased serum triglycerides
- Alcohol ingestion
- Fetal Hb above normal or 0.5%

Decreased in

- Shortened RBC life span (e.g hemolytic anemia, blood loss)
- Following blood transfusions
- Pregnancy
- Hemoglobinopathies (e.g spherocytes)

Importance and prerequisite for testing

- Fasting or dietary preparation is not required
- One percent increase in HbA1c is related approximately 30 mg/dl increase in glucose levels
- Test should be performed at least twice in a year to keep check on glycemic control.

III. AIM AND OBJECTIVES

1. To estimate blood glucose level (fasting and postprandial), HbA1c and lipid profile in diabetic, prediabetic and healthy individuals.
2. To study the correlation of Glycated Haemoglobin (HbA1c) , Blood glucose level and Lipid Profile in diabetic, prediabetic and healthy individuals.

IV. MATERIALS AND METHODS

1. Selection of subjects

Patient group

In this study 110 individuals were selected (35 Women and 75 Men). Subjects of this study were randomly selected from patients coming to Laboratory, Ruby Hall Clinic, Pune, India. Measurements of HbA1c (glycated haemoglobin), Fasting and Post Prandial Blood Sugar and lipid profile were easily accepted by patients.

Clinical information and data of these subjects were obtained according to the questionnaire in the proposal that including age, sex, clinical complaints.

They were categorized into diabetic, prediabetic and normal healthy individuals, after doing glycosylated hemoglobin.

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HbA1c <5.7 : Normal ,5.7 – 6.4 : Prediabetic ,≥ 6.5 : Diabetic stage

Total number of 38 healthy subjects were recruited to represent the control group with no history of diabetes mellitus this group contains N women and N men. The non diabetic volunteers selected as controls proved to have no complication of diabetes and no history of dyslipidemia at the time of investigations.

2. Blood samples collection and Serum preparation

A minimum of 8 ml of blood was collected by vein puncture using vacutainer. 3 ml of the blood sample was transferred to tubes containing EDTA for estimation of HbA1c , 3 ml for Fasting blood sugar and estimation of Lipid profile (Chole, TG, HDL and LDL) and ,2 ml for postprandial

For estimation of HbA1c we used High performance liquid chromatography HPLC (Bio rad D10) method and for estimation of BSL fasting and postprandial, Cholesterol, TG, HDL and LDL, Vitros 5600 and Roche Cobas 6000 was used

Sample Processing and estimation of Hb1Ac, Glucose and lipid parameters.

Hb1AC (Glycosylated Hb):

HPLC method

Bio-Rad D-10 Hemoglobin A1c program which is intended for the percent determination of HbA1c in human whole blood was used for estimation of Hb1AC.

The D-10 Hemoglobin A1c Program is based on chromatographic separation of HbA1c on a cation exchange cartridge. Separation is optimized to minimize interferences from hemoglobin variants, labile A1c and carbamylated hemoglobin. It also offers automatic sampling from a primary whole blood tube, followed by sample dilution and an analysis time of three minutes per sample [20].

The samples are automatically diluted on the D-10 and injected into the analytical cartridge. The D-10 delivers a programmed buffer gradient of increasing ionic strength to the cartridge where the hemoglobin is separated based on their ionic interactions with the cartridge material. The separated hemoglobin then passes through the flow cell of the filter photometer where changes in the absorbance are measured at 415 nanometer wavelength. The D-10 software performs reduction of raw data collected from each analysis. Two-level calibration is used for quantification of the HbA1c values. A sample report and a chromatogram are generated for each sample.

The A1c area is calculated using an exponentially modified Gaussian (EMG) algorithm that excludes the labile HbA1c and carbamylated peak area from the A1c peak area.

Materials:-

- **Elution Buffer 1.** Two bottles containing 2000 mL of a Bis-Tris/Phosphate buffer, pH 6.0. Contains <0.05% sodium azide as a preservative.
- **Elution Buffer 2.** One bottle containing 1000 mL of a Bis-Tris/Phosphate buffer, pH 6.7. Contains <0.05% sodium azide as a preservative.
- **Wash/Diluent Solution.** One bottle containing 1600 mL of deionized water with <0.05% sodium azide as a preservative.
- **Analytical Cartridge.** One cation exchange cartridge, 4.0 mm ID x 30 mm.
- **Floppy Diskette** with D-10 Hemoglobin A1c Program parameters.
- **Calibrator/Diluent Set.** One set consisting of three vials of Calibrator Level 1, three vials of Calibrator Level 2, and one bottle of Calibrator Diluent. The calibrator vials contain lyophilized human red blood cell hemolysate with gentamicin, tobramycin, and EDTA as preservatives. Reconstituted volume is 7 mL per vial. Calibrator Diluent

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contains 100 mL of deionized water with <0.05% sodium azide as a preservative.

- **Whole Blood Primer.** Four vials of lyophilized human red blood cell hemolysate with gentamicin, tobramycin, and EDTA as preservatives. Reconstituted volume is 1.0 mL per vial.
- **Sample Vials.** 100 polypropylene vials with pierceable caps, 1.5 mL.
- **Thermal Paper.** One roll.
- **Specimen :** Whole blood specimen in EDTA
Sample can be stored up to 7 days at 2–8 °C or 3 days at room temperature (15–30 °C).

Sample Preparation

1. Allow sample tubes to reach room temperature (15–30 °C) before performing the assay. No sample preparation is required. Mixing has no impact on HbA1c value as long as the total area is in range. The sample tubes are loaded into the D-10 sample rack and placed in the D-10. Ensure that the sample barcodes are facing towards the back of the instrument. Use special rack inserts for 12, 13, and 14 mm diameter tubes. Remove all inserts for 16 mm diameter tubes. Tubes with a height of 75 mm to 100 mm are acceptable for use.

2. If the sample is in an abnormal size/type tube, or if there is less than 2.0 mL of sample in the tube, then the sample must be prediluted. Before pipetting, thoroughly mix the sample by gently inverting the tube. To predilute, pipet 1.5 mL of Wash/Diluent Solution into a labeled 1.5 mL vial, followed by 5 µL of the whole blood sample. Cap the sample vial and mix thoroughly. Use a sample vial adapter for 1.5 mL vials.

Procedure:-

Pipet 1 mL of reconstituted Whole Blood Primer into a sample vial. Put the sample vial into a sample vial adapter labelled with a Primer barcode, then place the adapter into sample rack position 1. Start a run. When finished, the cartridge is ready for calibration.

Calibration:-

Calibration must be performed once, following the installation and priming of every new analytical cartridge. Additional calibration may be performed at the discretion of the laboratory.

Prepare samples as described in **Sample Preparation** section.

Place calibrators and controls on the D-10. Use sample vial adapters for 1.5 mL vials with calibrators and controls. The adapters should have barcodes identifying sample type.

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Biorad D10 HPLC (Hb1Ac) Analyser

Glucose/Blood Sugar level

Glucose is the major carbohydrate present in the peripheral blood. Oxidation of glucose is the major source of cellular energy in the body. Glucose derived from dietary sources is converted to glycogen for storage in the liver or to fatty acids for storage in adipose tissue. The concentration of glucose in blood is controlled within narrow limits by many hormones, the most important of which are produced by the pancreas.

The most frequent cause of hyperglycemia is diabetes mellitus resulting from a deficiency in insulin secretion or action. A number of secondary factors also contribute to elevated blood glucose levels. These include pancreatitis, thyroid dysfunction, renal failure and liver disease.

Hypoglycemia is less frequently observed. A variety of conditions may cause low blood glucose levels such as insulinoma, hypopituitarism or insulin induced hypoglycemia.

Glucose measurement in urine is used as a diabetes screening procedure and to aid in the evaluation of glycosuria, to detect renal tubular defects, and in the management of diabetes mellitus.

Thus, Glucose is a primary cellular energy source. Fasting plasma glucose concentrations and tolerance to a dose of glucose are used to establish the diagnosis of diabetes mellitus and disorders of carbohydrate metabolism. Glucose measurements are used to monitor therapy in diabetics and in patients with dehydration, coma, hypoglycemia, insulinoma, acidosis, and ketoacidosis.

Principle and method

1) Vitros 5600

Method: Glucose Oxidase

The VITROS GLU Slide is a multilayered, analytical element coated on a polyester support. A drop of patient sample is deposited on the slide and is evenly distributed by the spreading layer to the underlying layers.

The oxidation of sample glucose is catalyzed by glucose oxidase to form hydrogen peroxide and gluconate.

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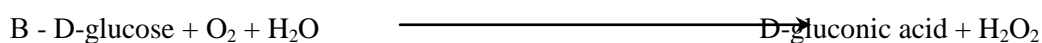
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This reaction is followed by an oxidative coupling catalyzed by peroxidase in the presence of dye precursors to produce a dye. The intensity of the dye is measured by reflected light.

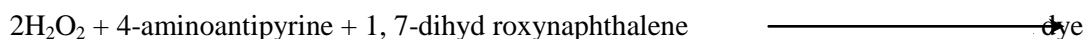
The dye system used is closely related to that first reported by Trinder. The chemistry of the glucose slides has been described by Curme, et al.

Reaction Sequence

Glucose oxidase



peroxidase

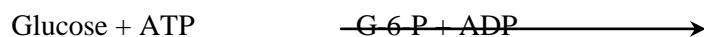


2) Cobas 6000:

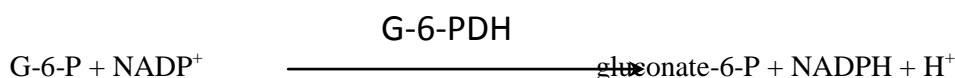
Method: Hexokinase

Hexokinase catalyzes the phosphorylation of glucose to glucose-6-phosphate by ATP.

HK



Glucose-6-phosphate dehydrogenase oxidizes glucose-6-phosphate in the presence of NADP to gluconate-6-phosphate. No other carbohydrate is oxidized. The rate of NADPH formation during the reaction is directly proportional to the glucose concentration and is measured photometrically.



After performing the tests, the data was entered in the sheet and necessary statistical test were applied. Regression coefficient and p value were calculated wherever required.

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Lipid Profile

Lipid profile tests are a group of tests that involved Cholesterol, Triglycerides , High density lipoproteins (HDL), Low density lipoproteins (LDL) and very low density lipoproteins (VLDL).

Cholesterol is measured enzymatically in serum or plasma in a series of coupled reactions .In this enzymatic method esterified cholesterol is converted to cholesterol by cholesterol esterase. The resulting cholesterol is then acted upon by cholesterol oxidase to produce cholest-4-en-3-one and hydrogen peroxide. The hydrogen peroxide then reacts with 4-aminophenazone in the presence of peroxidase to produce a colored product that is measured at 505 nm (secondary wavelength = 700 nm). The final step is known as the Trinder reaction. This method is a single reagent, endpoint reaction that is specific for cholesterol.

Triglycerides are measured enzymatically in serum or plasma using a series of coupled reactions in which triglycerides are hydrolyzed to produce glycerol. Glycerol is then oxidized using glycerol oxidase, and H_2O_2 , one of the reaction products, is measured as described above for cholesterol. Absorbance is measured at 500 nm.

For HDL measurement, magnesium/dextran sulfate solution is first added to the specimen to form water-soluble complexes with non-HDL cholesterol fractions. These complexes are not reactive with the measuring reagents added in the second step. With addition of reagent 2, HDL-cholesterol esters are converted to HDL-cholesterol by PEG-cholesterol esterase. The HDL-cholesterol is acted upon by PEG-cholesterol oxidase, and the hydrogen peroxide produced from this reaction combines with 4-amino-antipyrine and HSDA under the action of peroxidase to form a purple/blue pigment that is measured photometrically at 600 nm (secondary wavelength = 700 nm).

LDL-cholesterol is calculated from measured values of total cholesterol, triglycerides and HDL-cholesterol according to the relationship:

$$\text{LDL-cholesterol} = \text{Total cholesterol} - [\text{HDL-cho}] - [\text{TG}] / 5$$

where $[\text{TG}] / 5$ is an estimate of VLDL-cholesterol and all values are expressed in mg/dL.

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After sample testing, data was formulated in the table and necessary statistical tests were applied like regression coefficient (p value) and correlation coefficient(r value)

V.RESULTS

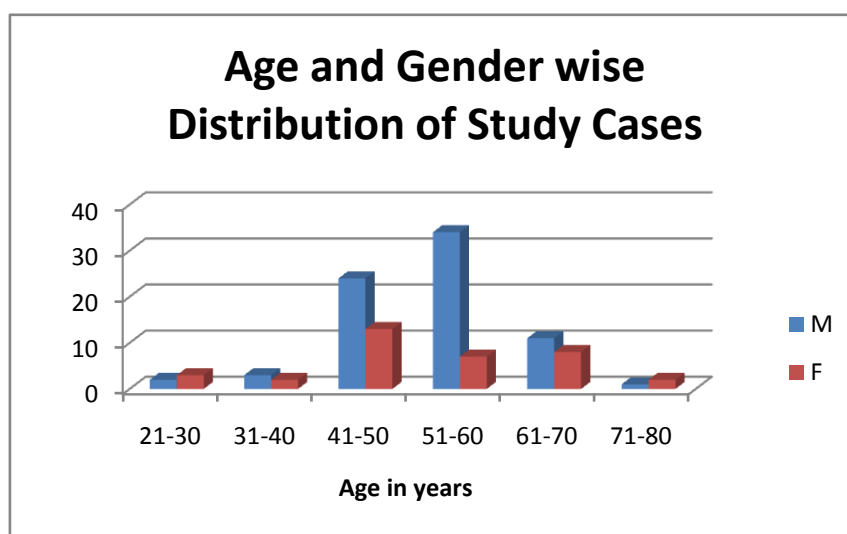
Gender wise separation of sample size

Total 110 subjects were included in the study, out of which 75 were males and 35 were females

Age wise Distribution

AGE	M	F
21-30	2	3
31-40	3	2
41-50	24	13
51-60	34	7
61-70	11	8
71-80	1	2
Total	75	35

Table 1.Age and Gender Wise Distribution of Study Cases



Distribution of cases as per HBA1c values in normal, prediabetic an diabetic subjects

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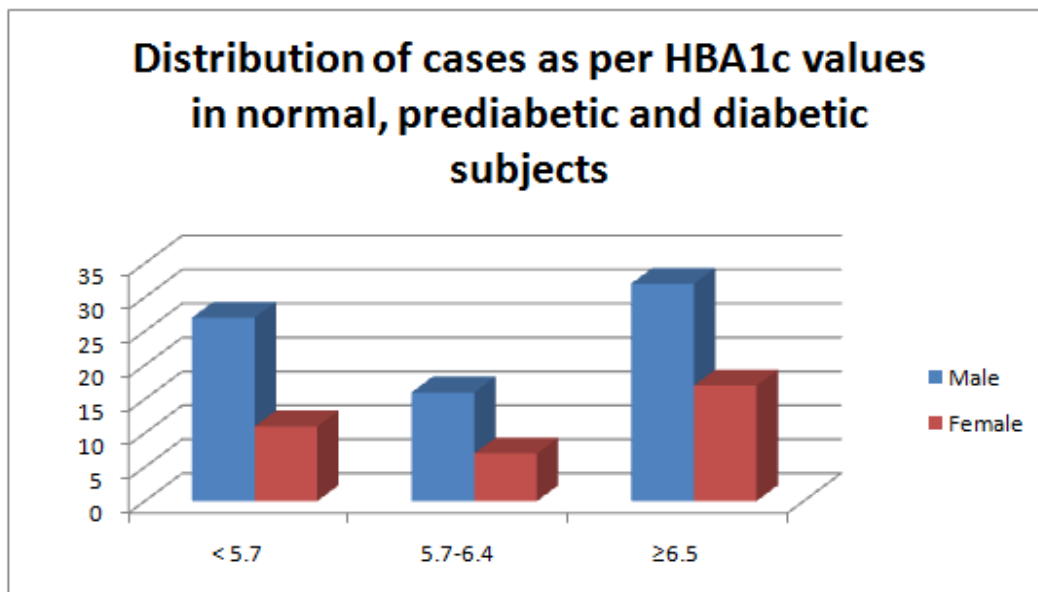
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HbA1c	Male	Female	Total
< 5.7	27	11	38
5.7-6.4	16	7	23
≥6.5	32	17	49
Total	75	35	110

Table 2. Distribution of cases as per HbA1c values in normal, prediabetic and diabetic subjects



Distribution of cases with abnormal lipid parameters in all 3 groups

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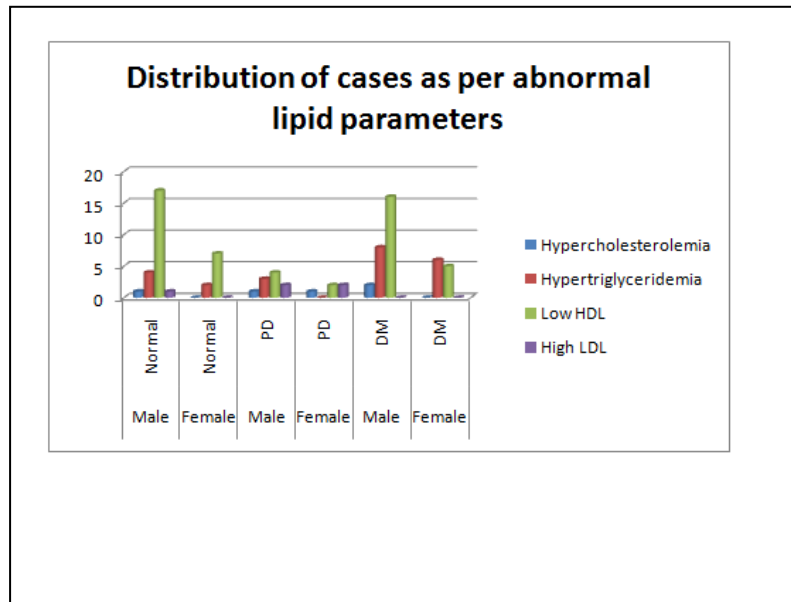
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	Male	Female	Male	Female	Male	Female
	Normal	Normal	Pre Diabetic	Pre Diabetic	DM	DM
Hypercholesterolemia	1	0	1	1	2	0
Hypertriglyceridemia	4	2	3	0	8	6
Low HDL	17	7	4	2	16	5
High LDL	1	0	2	2	0	0

Table 3. Distribution of cases with abnormal lipid parameters in all 3 groups



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Mean Values of study parameters in Normal Subject

Parameter	MALES (n=27)	FEMALES(n=11)	TOTAL (n=38)
Age	48.9 ± 7.5	44.8 ± 14.3	47.7 ± 9.9
FBS (mg/dl)	93.3 ± 12.7	102.7 ± 18.1	95.9 ± 14.8
PPBS (mg/dl)	102 ± 29.6	103.6 ± 20.6	102 ± 27.0
HbA1c (%)	5.3 ± 0.2	5.2 ± 0.16	5.3 ± 0.2
Chole (mg/dl)	174.2 ± 34.9	186.2 ± 20.5	177.7 ± 31.6
TG (mg/dl)	122.7 ± 43.6	135.8 ± 58.9	126.5 ± 48.0
HDL (mg/dl)	37.6 ± 9.3	45.8 ± 15.4	40.02 ± 11.9
LDL (mg/dl)	112.1 ± 32.8	113.3 ± 20	112.4 ± 29.4
VLDL (mg/dl)	38.2 ± 8.8	27.2 ± 11.7	25.58 ± 9.6

Table 4. Mean Values of study parameters in Normal Subjects

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Mean values of study parameters in Prediabetic Subjects

Parameter	MALES (n=16)	FEMALES (n=7)	TOTAL (n=23)
Age	51.0 ± 10.6	47.1 ± 5.8	49.8 ± 9.4
FBS (mg/dl)	107.3 ± 12.5	98 ± 9.0	104.4 ± 12.2
PPBS (mg/dl)	131 ± 41.7	109 ± 12.8	124.3 ± 36.6
HbA1c (%)	5.9 ± 0.2	6.02 ± 0.2	5.99 ± 0.2
Chole (mg/dl)	166.1 ± 47.8	196.2 ± 52.3	175.3 ± 46.6
TG (mg/dl)	130.9 ± 55.1	101.9 ± 12.4	122.1 ± 51.6
HDL (mg/dl)	45.9 ± 13.1	45.2 ± 6.6	45.6 ± 11.4
LDL (mg/dl)	99.4 ± 42.4	130.9 ± 45.6	109 ± 44.9
VLDL (mg/dl)	26.2 ± 11.08	20.4 ± 2.5	24.4 ± 9.6

Table 5. Mean values of study parameters in Prediabetic Subjects

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Parameter	MALES (n=32)	FEMALES (n=17)	TOTAL (n=49)
Age	56 ± 7.3	57.4 ± 9.3	56.5 ± 8.0
FBS (mg/dl)	156.9 ± 61.6	193.8 ± 65.2	169.7 ± 64.7
PPBS (mg/dl)	239.7 ± 91.2	303.0 ± 108.4	261.8 ± 101
HbA1c (%)	7.8 ± 1.4	9.26 ± 0.2	8.3 ± 1.8
Chole (mg/dl)	166.2 ± 39.8	171.4 ± 45.1	168.04 ± 41.3
TG (mg/dl)	151.7 ± 161	160.8 ± 65.3	154.8 ± 135.7
HDL (mg/dl)	41.3 ± 11.6	42.7 ± 8.4	41.7 ± 10.5
LDL (mg/dl)	89.1 ± 26.7	96.7 ± 40.7	91.6 ± 32.0
VLDL (mg/dl)	35.9 ± 43.7	32.2 ± 13.0	34.6 ± 35.9

Mean Values of Study Parameters in Diabetic Subjects

Table 6. Mean Values of Study Parameters in Diabetic Subjects

Correlation coefficient (r) and p values in normal males

Variable	Cholestrol		TG		HDL		LDL		VLDL	
	r value	P value	r value	P value	r value	P value	r value	P value	r value	P value
FBS	-0.5522	0.0028	-0.3159	0.1083	0.3953	0.0412	-0.6181	0.0005	0.0183	0.9275
PPBS	-0.4304	0.0249	-0.1474	0.4630	0.1589	0.4284	-0.4657	0.0143	-0.0217	0.9142
HbA1c	-0.0317	0.8750	-0.2649	0.1817	0.2945	0.1359	-0.0475	0.8136	-0.1951	0.3292

Table 7. Correlation coefficient (r) and p values in normal males

In normal males, significant positive correlation was noted in 1) cholesterol, FBS and PPBS 2) HDL and FBS 3) LDL, FBS and PPBS

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Correlation coefficient (r) and p values in prediabetic males

Variable	Cholestrol		TG		HDL		LDL		VLDL	
	r value	P value	r value	P value	r value	P value	r value	P value	r value	P value
FBS	-0.0643	0.8129	-0.1313	0.6276	0.3223	0.2233	-0.0622	0.8189	-0.1268	0.6396
PPBS	-0.1541	0.5686	-0.2929	0.2707	0.7374	0.0011	-0.1672	0.5357	-0.2962	0.2653
HbA1c	-0.1110	0.6821	-0.1045	0.6999	0.3877	0.13783	-0.1146	0.6724	-0.1020	0.7068

Table 8. Correlation coefficient (r) and p values in prediabetic males

In prediabetic males, only significant positive correlation was noted between HDL and PPBS.

Correlation coefficient (r) and p values in diabetic males

Variable	Cholestrol		TG		HDL		LDL		VLDL	
	r value	P value	r value	P value	r value	P value	r value	P value	r value	P value
FBS	0.4965	0.0038	0.3541	0.0467	-0.2593	0.1517	0.0041	0.982	0.5193	0.0023
PPBS	0.3856	0.0292	0.2764	0.1255	-0.2082	0.2527	0.0107	0.9535	0.4004	0.0231
HbA1c	0.4143	0.0183	0.4138	0.0185	-0.3870	0.0286	-0.2259	0.2137	0.6183	0.0001

Table 9. Correlation coefficient (r) and p values in diabetic males

In Diabetic males, significant positive correlation was noted between 1) Cholesterol and FBS, PPBS, HbA1c 2) Triglyceride, FBS and HbA1c 3) VLDL and FBS, PPBS, HbA1c

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Also, significant negative correlation was noted between HbA1c and HDL.

Correlation coefficient (r) and p values in normal females

Variable	Cholestrol		TG		HDL		LDL		VLDL	
	r value	P value	r value	P value	r value	P value	r value	P value	r value	P value
FBS	0.5103	0.1086	0.8074	0.0026	-0.1132	0.7403	0.1235	0.7175	0.8079	0.0026
PPBS	0.6285	0.0383	0.8798	0.0003	-0.3563	0.282	0.3946	0.2297	0.8804	0.0003
HbA1c	0.5945	0.0537	0.3167	0.3426	-0.0199	0.9536	0.4352	0.1809	0.3160	0.3436

Table 10. Correlation coefficient (r) and p values in normal females

In normal females, significant positive correlation was noted between 1) cholesterol, PPBS and HbA1c, 2) Triglyceride, FBS and PPBS, 3) VLDL and FBS, PPBS

Correlation coefficient (r) and p values in prediabetic females

Variable	Cholestrol		TG		HDL		LDL		VLDL	
	r value	P value	r value	P value	r value	P value	r value	P value	r value	P value
FBS	-0.1602	0.7314	-0.0514	0.9127	-0.1456	0.7554	-0.1447	0.7567	-0.0553	0.9062
PPBS	-0.6908	0.0857	0.3787	0.4021	-0.3760	0.4057	-0.6924	0.0846	0.4046	0.3678
HbA1c	-0.4073	0.3644	0.5691	0.1823	-0.1928	0.6786	-0.4333	0.3314	0.5758	0.176

Table 11. Correlation coefficient (r) and p values in prediabetic females

No significant correlation was noted in FBS, PPBS, HbA1c and lipid parameters in prediabetic females in our study.

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Correlation coefficient (r) and p values in diabetic females

Variable	Cholestrol		TG		HDL		LDL		VLDL	
	r value	P value	r value	P value	r value	P value	r value	P value	r value	P value
FBS	0.0496	0.84	0.2460	0.34	-0.2687	0.29	0.0347	0.89	0.2442	0.343
PPBS	0.1622	0.533	0.1667	0.52	0.0395	0.88	0.1229	0.638	0.1660	0.52
HbA1c	0.1650	0.526	0.1332	0.61	0.0231	0.92	0.1394	0.593	0.1316	0.61

Table 12. Correlation coefficient (r) and p values in diabetic females

No significant correlation was noted in FBS, PPBS, HbA1c and lipid parameters in diabetic females in our study.

VII.CONCLUSION

Present observational study show significant positive correlation in FBS, PPBS, HbA1c and lipid parameters in diabetic males. No significant correlation was noted in diabetic and prediabetic females in our study. In normal males group, positive correlation was noted in Cholesterol, FBS and PPBS 2) HDL and FBS 3) LDL, FBS and PPBS whereas in normal females significant positive correlation was noted between 1) cholesterol, PPBS and HbA1c, 2) Triglyceride, FBS and PPBS, 3) VLDL and FBS, PPBS. In prediabetic males, only significant positive correlation was noted between HDL and PPBS. The findings of this study clearly indicate that HbA1c is not only a useful biomarker of long-term glycemic control but can be used as predictor of lipid profile in diabetic males. Thus our study suggests that there is significant variation in lipid abnormalities and glycemic control in males and females. Good glycemic control, proper diet and exercise can help in controlling lipid parameters as well as FBS, PPBS to avoid future complications arising due to diabetes and thereby increasing longevity of person.

VIII.FUTURE PROSPECTS

In developing countries, due to lack of proper infrastructure and laboratory setups, most of the patients lack proper diagnosis. Physicians have to rely on clinical findings for diagnosis. With development of various small but cost effective equipments which measures HbA1c and lipid parameters, actual correlation between such parameters can be found out. This can prevent morbidities and mortality due to diabetes. Normal and prediabetic individuals can be advised to have proper diet and exercise which can reduce the incidence of diabetes in developing countries. Other parameters like Red Cell Distribution width (RDW) and Mean Platelet Volume (MPV) can be used along with HbA1c to predict the future complications of diabetes.

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