# Estimation of Biochemical Parameter with Co-Relation of HbA1c in Diabetic Patient

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**ABSTRACT:** Diabetes is a chronic endocrine disease characterized by persistent hyperglycemia and associated with abnormalities of carbohydrate, protein, and lipid metabolism. This disease is caused by a decreased and/or increased insulin secretion. Blood glucose levels of healthy man are 80 mg/dl on fasting and up to 180 mg/dl in the postprandial state. According to national diabetes data group of the national institute of health, diabetes is diagnosed when fasting plasma glucose concentration becomes >140 mg/dl on at least two separate occasions and after ingestion of 75 g of glucose it is >200 mg/dl at 2 h and at least one other occasion during the 2 h test (i.e. two values >200 mg/dl must be obtained for diagnosis). Glycosylated hemoglobin (HbA1c) is important for the evaluation and management of patients with diabetes mellitus. HbA1c assesses long-term glycemic control and predicts micro-vascular complications in diabetes. The present work summarizes Diabetes mellitus, causes, and complication of this disorder.

**Key Words**: HbA1C, diabetes mellitus, uric acid and complication.

**Introduction:** Diabetes poses a serious threat to health worldwide due to its severe effects on the micro-macro vascular systems. Specifically, diabetes can cause damage and/or dysfunction of multiple organs and tissues, especially eyes, kidneys, nerves, heart, and blood vessels. About 171 million people are convicted with diabetes and are projected to rise from in 2000 to 366 million in 2030 according to a survey report of Wild *et al*., 2004. Controlling blood glucose levels is essential for preventing diabetic complications and for improving the health of patients with diabetes. Prevalence of diabetes mellitus in World population is increasing in epidemic. During year 1980 to 2003, prevalence of diabetes more than doubled and rising from 5.8 to 13.8 million diagnosed individuals (National Center for Health Statistics, 2005).

Diabetes mellitus is caused by pronounced changes in human environment and in human behavior and lifestyle, which have been a part and parcel of globalization and these, have resulted in escalating rates of both obesity and diabetes. Hence the recent adoption of the term "diabesity" was first suggested by Shafrir, 1982 several decades ago.

**Types of Diabetes**

**Type I** (Insulin dependent diabetes mellitus, IDDM):
Insulin dependent diabetes mellitus is caused by absolute deficiency of insulin resulting from reduction of beta cell mass. The patients therefore respond to exogenously administered insulin. In IDDM some environmental factor (Viral infection, geographic variation) initiates the autoimmune destruction of beta cells in genetically susceptible individuals. It is also known as juvenile onset diabetes.
**Type II** (Non-insulin dependent diabetes mellitus, NIDDM):
Non- Insulin dependent diabetes mellitus is a complex multi factorial disease involving deranged insulin secretion and insulin resistance with possible genetic defects, obesity and fault in the insulin receptors. It is also known as maturity onset diabetes (Mohan, 2003).
In nutshell, all diagnosis methods and research in diabetes, there are less research done in biochemical diagnosis and risk of diabetes. This is a study done for such correlation in uric acid and HbA1c of diabetic patients.

**METHODOLOGY:** Aim of this study was to provide scientific evidence with significant associations, correlations and regression between uric acid, HbA1c, and serum insulin among diabetic patients.

**Samples:** 200 known diabetic patients and 200 healthy non-diabetic people were randomly taken for this study that has no any other clinical history. Institutional Human Ethical Committee, RKDF University Bhopal approves this research for human welfare. Informed consents were taken from all the subjects who were included in the study. A detailed history was taken followed by thorough clinical examination. Demographic data viz. Age, Sex, weight, height, residence and birth place were collected in a Performa with informed consent.

**Blood sample:** 2ml peripheral blood samples were drawn from diabetes type-II patients as well as non-diabetic healthy people in fasting with the help of laboratory technician. These samples were then investigated for serum insulin, uric acid, blood sugar and HbA1C.

**Sampling area:** Clinics and Hospitals of Bhopal and adjoining area.

**Methods:** Samples were divided in two groups as follows:

**Group A -** 200 healthy non-diabetic individuals without any previous clinical history.

**Group B** - 200 diabetic diagnosed patients having and type-II diabetes.

**Estimation of Total Cholesterol (TC):** Cholesterol esterasehydrolyzes Cholesterol esters and Cholesterol oxidise into cholest-4-en-3-onand $H_2O_2$ by bacterial cholesterol oxydase. In presence of phenol and amino-4- antipyrin $H_2O_2$forms a complex of red color, which showing maximum absorption between 500-550 nm.

**Estimation of Triglycerides(TG):** Triglycerides broken down to glycerol and fatty acid in presence of lipoproteine lipase. This Glycerol and ATP then make Glycerol-3-phosphate and ADP in presence of glycerol kinase and $Mg^{2+}$. Found Glycerol -3-phosphate and $O_2$ breaks down to dihydroxi-acetonephospate and $H_2O_2$ in presence of Glycerol -3-phosphate oxydase. Finally H2O2 + amino-4-antipyrine + ESPAS (N-ethyl-N-sulfopropyl-m-anizidine gives red derivative of quinone+ 4 H2O in presence of peroxydase.

**Estimation of High Density Lipoproteins-Cholesterol (HDLC):** HDL fraction will be precipitated in the presence of phosphotungstic acid-$MgCl_2$. After centrifugation HDLcholesterol content of the supernatants will be determined

**Estimation of Low Density Lipoproteins (LDL):** Serum LDL cholesterol (mg/dl) = (Serum total cholesterol - Serum HDL cholesterol) + (Triglyceride / 5).

**Estimation of Very Low Density Lipoproteins (VLDL):** Serum VLDL cholesterol level (mg/dL) = Triglyceride / 5

**Estimation of Blood Glucose**

Glucose oxidase is a FAD coferment containing enzyme that -D glucose to gluconolactone. It is isolated from molds, which also containsβcatalyzes the oxidation of D glucose into the ß-D glucose form. As shown in theαmutarotase enzyme which enhances the conversion of reaction, stoichiometric amount of $H_20_2$ is also formed in the

reaction. With the use of a third enzyme peroxydase in a coupled reaction $H_20_2$ is transformed into $H_20$ while the necessary hydrogens are removed from an organic substrate molecule (e.g. ortho-dianisidine). The oxidized form of orthodianisidine is a coloured compound and its amount can be determined spectrophotometrically.

**Uric Acid** (Witte, 2004): Serum urea level was determined according to the method of Varley *et al.*, 1980.

**Glycosylated haemoglobin (HbA1c):** This test was performed according to the method of Steffes, 2008. HbA1c is formed by the non-enzymatic glycation of free amino groups at the N-terminus of the β-chain of hemoglobin $A_0$. The level of HbA1c is proportional to the level of glucose in the blood. As the glucose remains bound to the red cell throughout its life cycle, measurement of HbA1cprovides an indication of the mean daily blood glucose concentration over the preceding two months. Measurement of HbA1c is, therefore, considered to be an important diagnostic tool in the monitoring of dietary control and therapeutic regimes during the treatment of diabetes. Effective control of blood glucose levels is important in the prevention of ketosis and hyperglycemia, and may reduce the prevalence and severity of late diabetic complications such as retinopathy, neuropathy, nephropathy, and cardiac disease.

**Estimation of SGPT /SGOT:** SGOT (AST) and SGPT (ALT) were determined according to prescribed methods of Reitman and Frankel, 1957.

**Assessment of Blood Pressure:** Arterial blood pressure is the force exerted by the blood on the wall of a blood vessel as the heart pumps (contracts) and relaxes. Systolic blood pressure is the degree of force when the heart is pumping (contracting). The diastolic blood pressure is the degree of force when the hearts relaxed.

**Statistical analysis:** Variables of interest were entered and all data analyzed using Microsoft Excel powered by Microsoft Corporation. All statistical tests were performed by standardized methodology in accordance with the reference to Kleinbaum *et al.,* 1998.

## RESULTS

Diabetes mellitus is an endocrinological disorder which is categorized by metabolic abnormalities. It gives long term complications. In this study human samples were

collected from different hospitals and clinics of Bhopal with proper consent and after ethical approval from Institutional Human Ethical Committee and analyse.

**Sex:** Table R1. and Fig. R1. showing percentage population taken for this study according to their sex. In this study there was 56.25% male and 43.75% female population found in healthy group whereas 52.5% male and 47.5% female found in diabetic population.

| Sex | Healthy (In %) | Diabetic (In %) |
|---|---|---|
| Male | 56.25 | 52.5 |
| Female | 43.75 | 47.5 |

Table R1. Healthy and Diabetic population according to their sex.

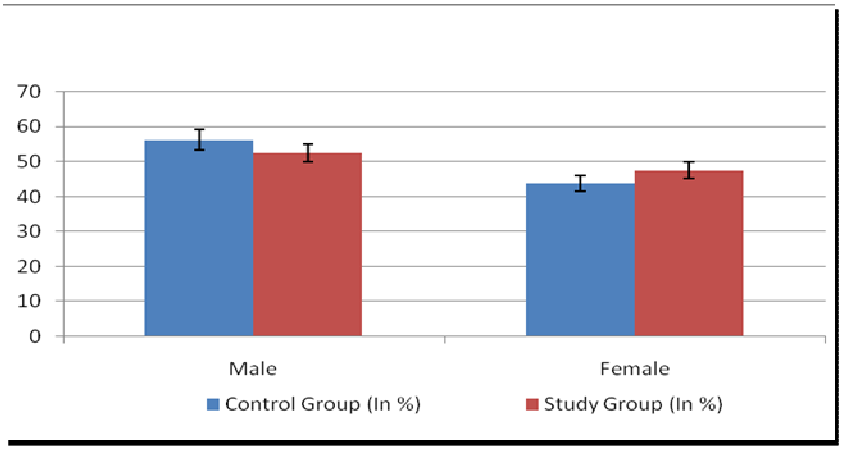


Fig. R1.Healthy and Diabetic population according to their sex.

**Serum uric Acid:** Values of serum uric acids in female and male are different.

**Female:** Study of female population reports serum uric acid (mg/dL) 20.00±1.94 % in 2.4-3.6 range, 54.29±5.20% in 3.6-4.8 range and 25.71±2.43% in 4.8-6.0 range in healthy population whereas 52.63±4.98% in 6.0-7.2 range, 44.74±4.23% in 6.0-7.2 range and 2.63±0.25% in 6.0-7.2 range diabetic population presented in Table R2 and Fig. R2.

**Male:** Study of female population reports serum uric acid (mg/dL) 22.22±2.13% in 3.4-4.6 range, 48.89±4.63% in 4.6-5.8 range and 28.89±2.70% in 5.8-7.0 range in healthy

population whereas 38.10±3.57% in 7.0-8.2 range, 57.14±5.35% in 8.2-9.4 range and 4.76±0.45% in 9.4-10.6 range diabetic population presented in Table R3 and Fig. R3.

| Serum uric Acid (mg/dL) | Healthy (Female in %) | Diabetic (Female in %) |
|---|---|---|
| 2.4 - 3.6 | 20.00±1.94 | 0.00 |
| 3.6 - 4.8 | 54.29±5.20 | 0.00 |
| 4.8 - 6.0 | 25.71±2.43 | 0.00 |
| 6.0 - 7.2 | 0.00 | 52.63±4.98 |
| 7.2 - 8.4 | 0.00 | 44.74±4.23 |
| 8.4 - 9.6 | 0.00 | 2.63±0.25 |

Table R2. Serum uric acid level in percent female population

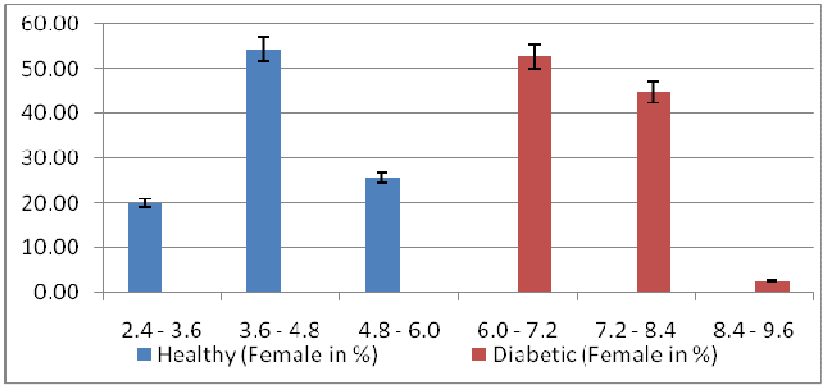


Fig. R2. Serum uric acid level in percent female population

| Serum uric Acid (mg/dL) | Healthy (Male in %) | Diabetic (Male in %) |
|---|---|---|
| 3.4 - 4.6 | 22.22±2.13 | 0.00 |
| 4.6 - 5.8 | 48.89±4.63 | 0.00 |
| 5.8 - 7.0 | 28.89±2.70 | 0.00 |
| 7.0 - 8.2 | 0.00 | 38.10±3.57 |
| 8.2 - 9.4 | 0.00 | 57.14±5.35 |
| 9.4 - 10.6 | 0.00 | 4.76±0.45 |

Table R3. Serum uric acid level in percent male population

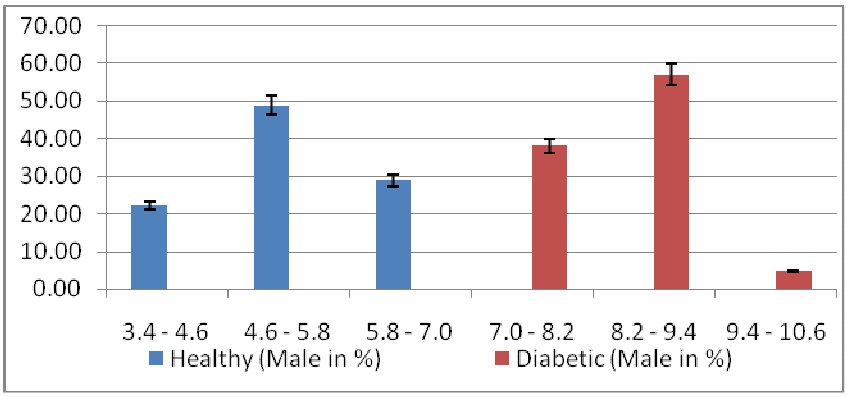


Fig. R3. Serum uric acid level in percent female population

**Glycated Hemoglobin/HbA1c:** In present study, it was observed that the HbA1c value in healthy population 4.38±0.43% in range <5, 81.88±7.74% in range 5-6, 13.75±1.29% in range 6-7 whereas in diabetic population 35.00±3.27 in range 7-8, 60.63±5.67 in range 8-9 and 4.38±0.41 in range >9. This was shows HbA1c in diabetic population had statistically higher than that of healthy population (Table R4 and Fig. R4).

| **Glycated Hemoglobin/HbA1c (%)** | **Healthy (in %)** | **Diabetic (In %)** |
|---|---|---|
| <5 | 4.38±0.43 | 0.00 |
| 5-6 | 81.88±7.74 | 0.00 |
| 6-7 | 13.75±1.29 | 0.00 |
| 7-8 | 0.00 | 35.00±3.27 |
| 8-9 | 0.00 | 60.63±5.67 |
| >9 | 0.00 | 4.38±0.41 |

Table R4. HbA1c values found in different population

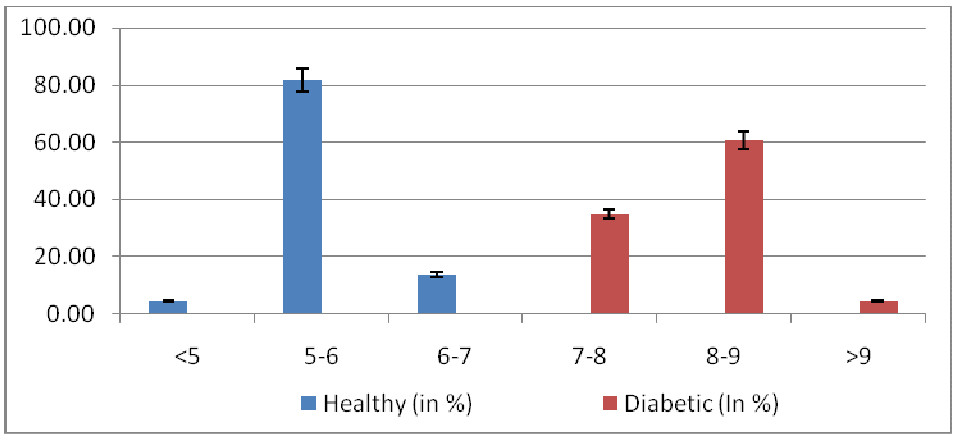


Fig. R4. HbA1c values found in different population

## 7.4 Fasting Blood Sugar:

Fasting Blood Sugar was observed <80 range in 5.00±0.30%, 80-100 range in 69.38±0.69%, 100-120 range in 25.63±0.26 % healthy population. Range and population recorded in diabetic was 120-140 range in 15.00±0.15%, 140-160 range in 38.13±0.38, 160-180 range in 43.13±0.43, 180-200 range in 2.50±0.03 and >200 range in 1.25±0.01% population showed in table R5 and Fig R5.

| **Fasting Blood Sugar (mg/dL)** | **Healthy (in %)** | **Diabetic (In %)** |
|---|---|---|
| <80 | 5.00±0.30 | 0.00 |
| 80-100 | 69.38±0.69 | 0.00 |
| 100-120 | 25.63±0.26 | 0.00 |
| 120-140 | 0.00 | 15.00±0.15 |
| 140-160 | 0.00 | 38.13±0.38 |
| 160-180 | 0.00 | 43.13±0.43 |
| 180-200 | 0.00 | 2.50±0.03 |
| >200 | 0.00 | 1.25±0.01 |

Table R5. Comparison of fasting blood sugar in the both the groups

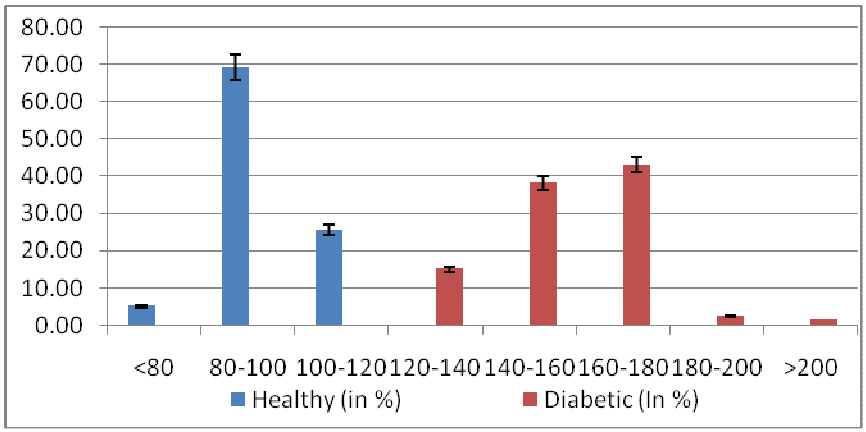


Fig. R5. Comparison of fasting blood sugar in the both the groups

**Serum Insulin Level:** Obtained results shown in table R6 and Fig.R6.Serum insulin level in healthy group was recorded 0.00% in range <10, 1.88±0.16 in range 10-15, 85.63±7.06 in range 15-20, 12.50±0.97 in range 20-25. Findings of serum insulin in diabetic population was as 25.63±1.99 in 25-25 range, 70.00±15.75 in 25-30 range and 4.38±0.99 in >35µlU/mL range.

| Serum Insulin (µlU/mL) | Healthy (in %) | Diabetic (In %) |
|---|---|---|
| <10 | 0.00 | 0.00 |
| 10-15 | 1.88±0.16 | 0.00 |
| 15-20 | 85.63±7.06 | 0.00 |
| 20-25 | 12.50±0.97 | 0.00 |
| 25-30 | 0.00 | 25.63±1.99 |
| 30-35 | 0.00 | 70.00±15.75 |
| >35 | 0.00 | 4.38±0.99 |

**Table R6. Serum insulinlevel in % population of both groups**

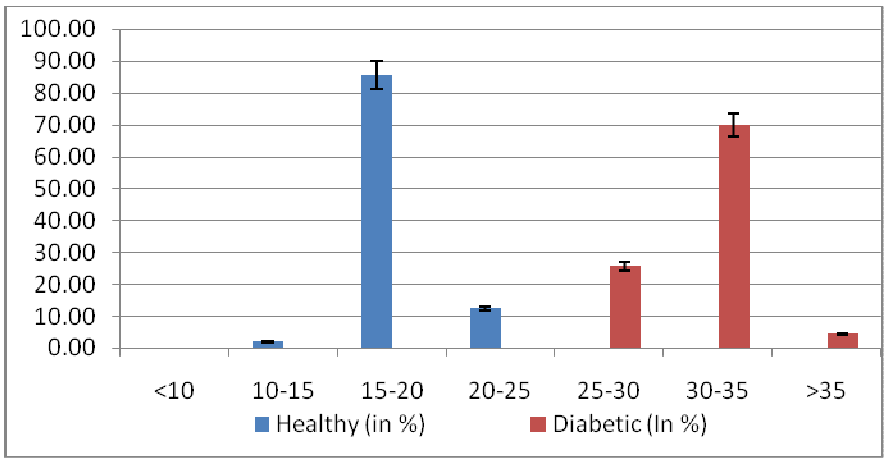


**Fig.R6. Serum insulinlevel in % population of both groups**

**Total Cholesterol:** Total cholesterol was observed 100-120 range in 31.88±0.32%, 120-140 range in 51.25±1.43%, 140-160 range in 16.88±0.84% healthy population. Range and population recorded in diabetic was 160-180 range in 31.881.59%, 180-200 range in 68.13±3.41 and 200-220 range in 0.00% population showed in table R7 and Fig R7.

| Total Cholesterol (mg/dL) | Healthy (in %) | Diabetic (In %) |
|---|---|---|
| 100-120 | 31.88±0.32 | 0.00 |
| 120-140 | 51.25±1.43 | 0.00 |
| 140-160 | 16.88±0.84 | 0.00 |
| 160-180 | 0.00 | 31.881.59 |
| 180-200 | 0.00 | 68.13±3.41 |
| 200-220 | 0.00 | 0.00 |

Table R7. Total cholesterol in both groups

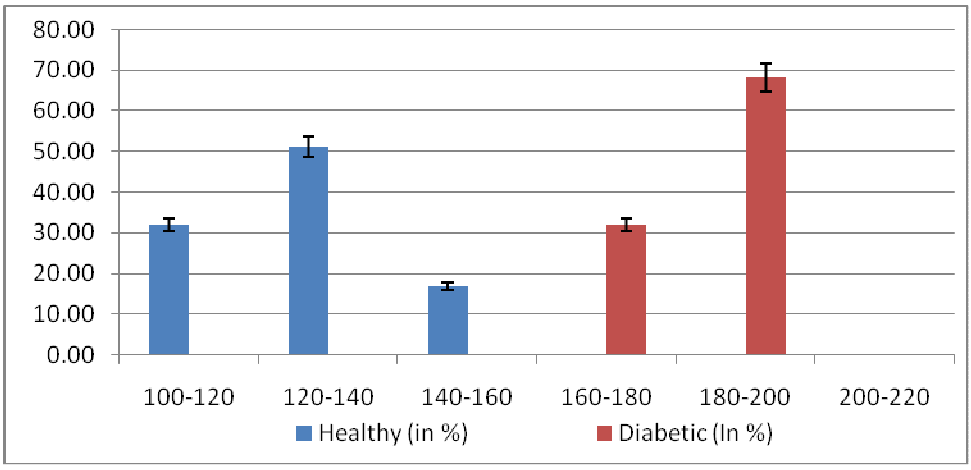


Fig. R7. Total cholesterol in both groups

**Triglycerides:** Obtained results shown in table R8 and Fig.R8.Triglycerides (mg/dL)level in healthy group was recorded 0.00% in range <100, 88.75±2.22 % in range 100-150 and 11.25±0.84 % in range 150-200. Whereas, findings of triglycerides in diabetic population was 28.75±0.72 % in 100-150 range, 60.63±4.55 % in 150-200 range, 3.75±0.28 % in 200-250 range, 3.13±0.23 % in 250-300, 1.88±0.14 % in 300-350, 1.88±0.14 % in 350-400 range and 0% in >400mg/dL range. Study revealed that the comparison of triglycerides level of group diabetic population was significantly higher than that of healthy group population where p< 0.001).

| Triglycerides (mg/dL) | Healthy (in %) | Diabetic (In %) |
|---|---|---|
| <100 | 0.00 | 0.00 |
| 100-150 | 88.75±2.22 | 28.75±0.72 |
| 150-200 | 11.25±0.84 | 60.63±4.55 |
| 200-250 | 0.00 | 3.75±0.28 |
| 250-300 | 0.00 | 3.13±0.23 |
| 300-350 | 0.00 | 1.88±0.14 |
| 350-400 | 0.00 | 1.88±0.14 |
| 400-450 | 0.00 | 0.00 |
| 450-500 | 0.00 | 0.00 |
| >500 | 0.00 | 0.00 |

Table R8. Showing triglycerides levels in healthy and diabetic groups

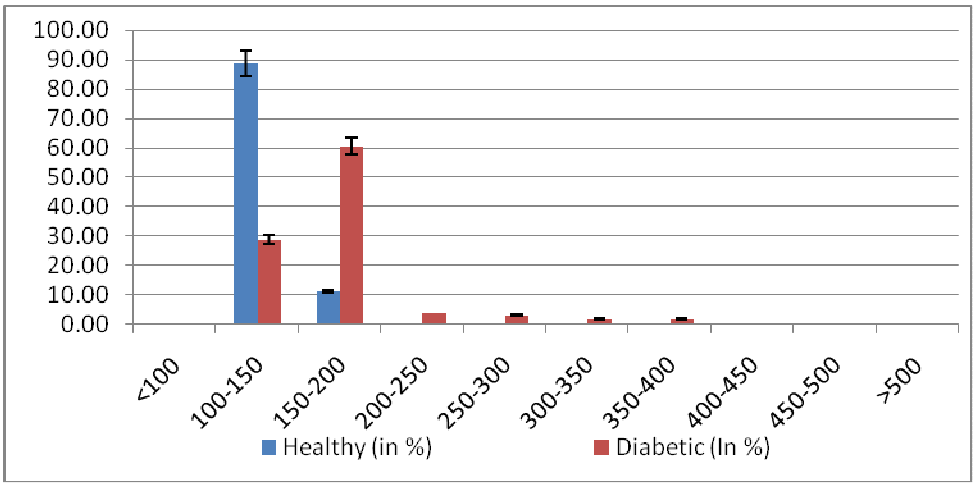


Fig. R8. Showing triglycerides levels in healthy and diabetic groups

**High-density lipoprotein cholesterol:** In the present study, it was observed that when a comparison was made high-density lipoprotein cholesterol between both the groups, the comparison of HDL level of diabetic group was significantly higher than healthy group population where p< 0.001 showed in Table-R9/Fig. R9. Results shown healthy population with 31.88±0.32% in <120 range, 51.25±1.54% in 120-140 and 16.88±0.84% in 140-160 range. Significantly 31.88±1.59% in 160-180, 68.13±3.41% in 180-200 range and 0% in greater than 200mg/dL HDL in diabetic population.

| **High-density lipoprotein HDL (mg/dL)** | **Healthy (in %)** | **Diabetic (In %)** |
|---|---|---|
| <120 | 31.88±0.32 | 0.00 |
| 120-140 | 51.25±1.54 | 0.00 |
| 140-160 | 16.88±0.84 | 0.00 |
| 160-180 | 0.00 | 31.88±1.59 |
| 180-200 | 0.00 | 68.13±3.41 |
| 200-220 | 0.00 | 0.00 |

Table R9. High-density lipoprotein cholesterol levels in both groups

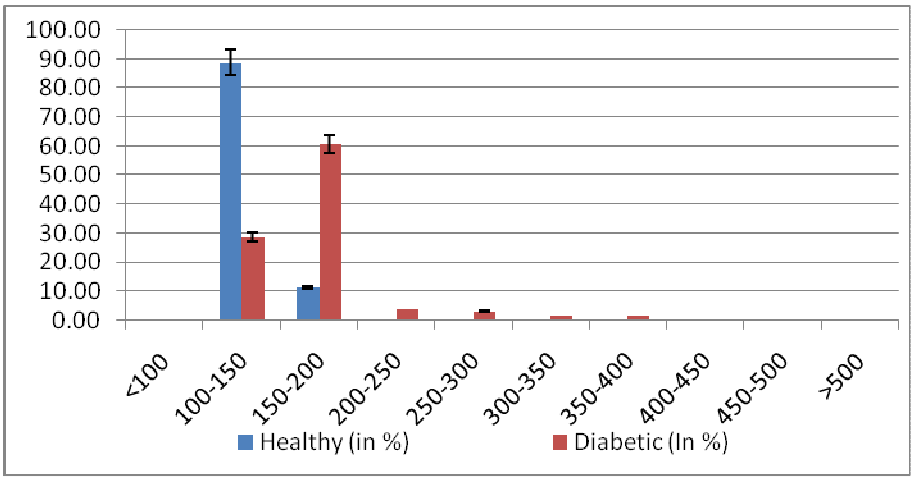


Table R9. High-density lipoprotein cholesterol levels in both groups

**Low-density lipoprotein cholesterol:** As shown in Table-R10/Fig. R10, Results shown healthy population with 31.88±0.32% in <120 range, 51.25±1.54% in 120-140 and 16.88±0.84% in 140-160 range. Significantly 31.88±1.59% in 160-180, 68.13±3.41% in 180-200 range and 0% in greater than 200mg/dL LDL in diabetic population.Comparison was made and found low-density lipoprotein cholesterol between both the groups, the comparison of LDL level of diabetic group was significantly higher than healthy group population where p< 0.001.

| **Low-density lipoprotein cholesterol LDL (mg/dL)** | **Healthy (in %)** | **Diabetic (In %)** |
|---|---|---|
| <120 | 31.88±0.32 | 0.00 |
| 120-140 | 51.25±1.54 | 0.00 |
| 140-160 | 16.88±0.84 | 0.00 |
| 160-180 | 0.00 | 31.88±1.59 |
| 180-200 | 0.00 | 68.13±3.41 |
| 200-220 | 0.00 | 0.00 |

Table R10. Showing low-density lipoprotein cholesterol levels

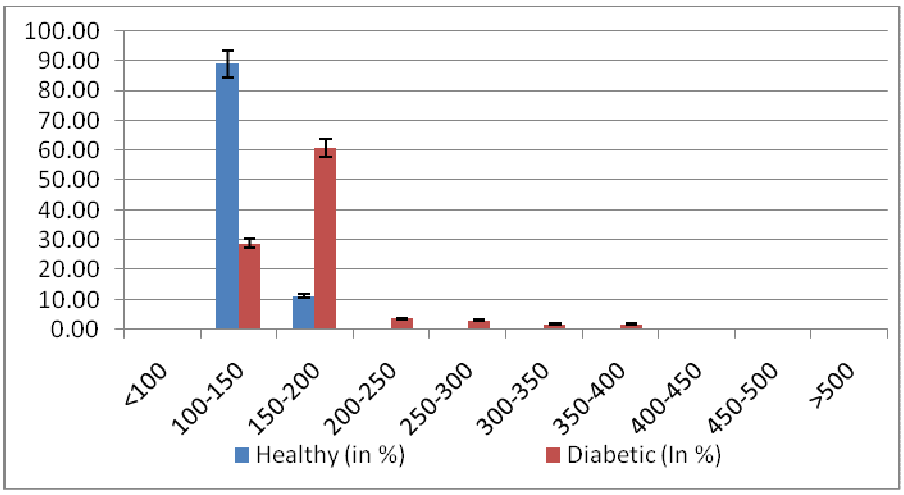


Fig.R10. Showing low-density lipoprotein cholesterol levels

**SGPT/AST (Aspartate aminotransferase):** Obtained results shown in table R11 and Fig.R11. SGPT/AST (Aspartate aminotransferase) level in healthy group was recorded 0.63±0.06% in range <7, 88.75±2.22 % in range 7-31 and 31.25±1.77 % in range 31.56. Whereas, findings of SGPT/AST (Aspartate aminotransferase) in diabetic population was 3.75±0.30% in 7-31range, 36.88±2.08% in 31-56 range, 57.50±1.84% in 56-80range and1.88±0.06 in >80 IU/dL range. In the present study, it was also observed that SGPTlevel of diabetic group was significantly higher than healthy group where p< 0.001.

| SGPT (IU/L) | Healthy (in %) | Diabetic (In %) |
|---|---|---|
| <7 | 0.63±0.06 | 0.00 |
| 7-31 | 68.13±5.52 | 3.75±0.30 |
| 31-56 | 31.25±1.77 | 36.88±2.08 |
| 56-80 | 0.00 | 57.50±1.84 |
| >80 | 0.00 | 1.88±0.06 |

Table R11. Showing comparison of SGPT in both groups

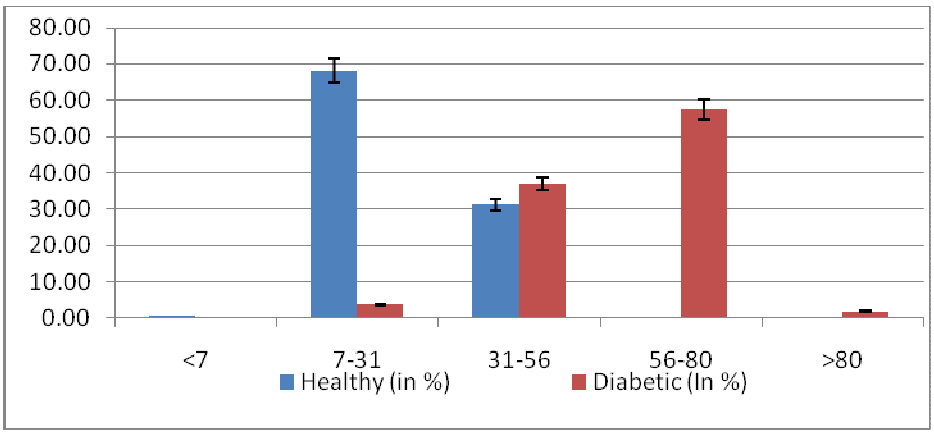


Table R11. Showing comparison of SGPT in both groups

**SGOT/ALT (Alanine aminotransferase):** In the present study, it was observed that when a comparison was made SGOT/ALT (Alanine aminotransferase) between healthy and diabetic groups, thenSGOT/ALT (Alanine aminotransferase) level of diabetic group was significantly higher than healthy group population where $p<0.001$ showed in Table R12/Fig. R12. Found results of healthy population was 0.63±0.06 % in <5 range, 66.25±5.73 % in 5-22 and 33.13±2.29 % in 22-40 IU/L range. Significantly diabetic population 3.75±0.32 % in 5-22, 38.13±2.63 % in 22-40 range, 55.63±2.86 in 40-57 and 2.50±0.13 % in greater than 57IU/L SGOT.

| SGOT (IU/L) | Healthy (in %) | Diabetic (In %) |
|---|---|---|
| <5 | 0.63±0.06 | 0.00 |
| 5-22 | 66.25±5.73 | 3.75±0.32 |
| 22-40 | 33.13±2.29 | 38.13±2.63 |
| 40-57 | 0.00 | 55.63±2.86 |
| >57 | 0.00 | 2.50±0.13 |

Table R12. Showing comparison of SGOT in both groups

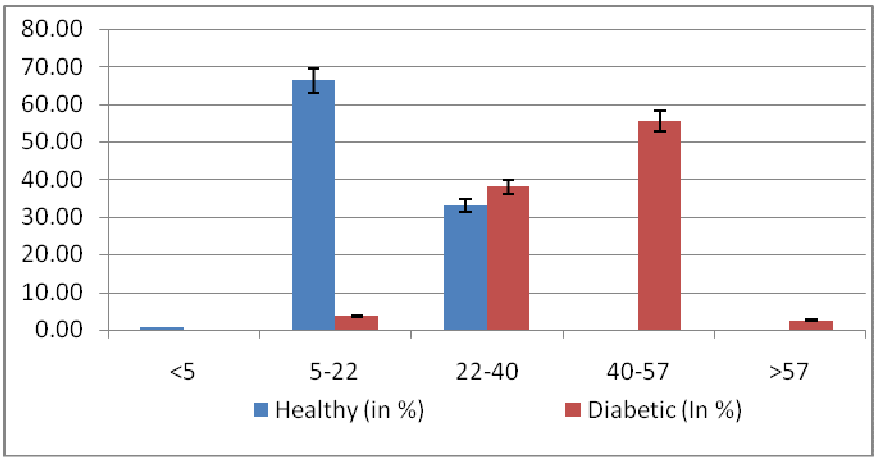


Table R12. Showing comparison of SGOT in both groups

**Blood Pressure:** This study observed that systolic and diastolic pressure of blood level in healthy as well as diabetic group.

**Systolic:** Healthy population reported normal systolic pressure in between 120-140 mmHg. But diabetic population had scattered systolic pressure and reports 1.88±0.08% below 120 ranges, 90.63±2.72 reported normal range between 120-140 and 7.50±0.38% reports high systolic pressure in between range 140-160 mmHg. This reseals clears that there are no much correlation between diabetes and systolic blood pressure. Observation showed in Table R13 and Fig. R13.

| **Blood pressure Systolic (mmHg)** | **Healthy (in %)** | **Diabetic (In %)** |
|---|---|---|
| <120 | 0.00 | 1.88±0.08 |
| 120-140 | 100.00±3.00 | 90.63±2.72 |
| 140-160 | 0.00 | 7.50±0.38 |
| >160 | 0.00 | 0.00 |

Table R13. Systolic pressure of blood in healthy and diabetic group

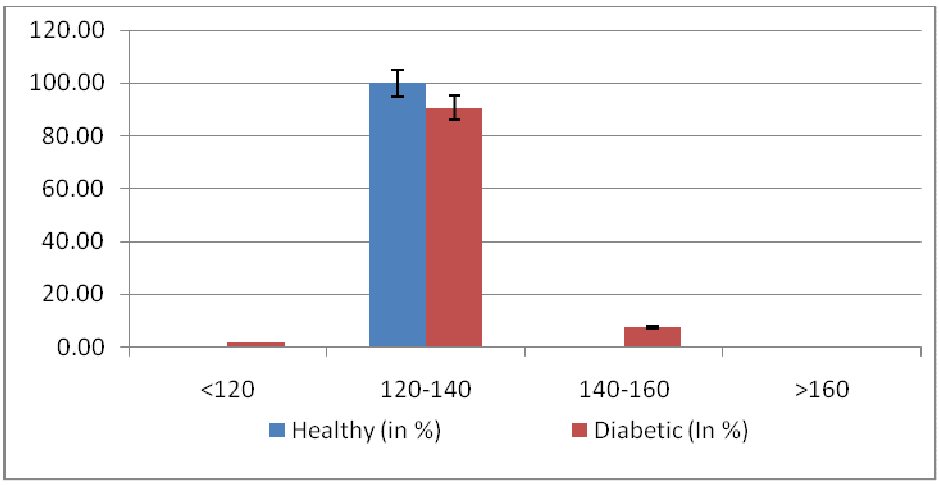


Fig.R13. Systolic pressure of blood in healthy and diabetic group

**Diastolic:** Healthy population reported normal diastolic pressure in between 80-90 mmHg. But diabetic population had scattered diastolic pressure which reports 1.88±0.08% below 80 ranges, 90.63±2.72 reported normal range between 80-90 and 7.50±0.38% reports high systolic pressure in between range 90-100 mmHg. This reseals clears that there are no much correlation between diabetes and diastolic blood pressure. Observation showed in Table R14 and Fig. R14.

| **Blood pressure diastolic (mmHg)** | **Healthy (in %)** | **Diabetic (In %)** |
|---|---|---|
| <80 | 0.00 | 1.88±0.11 |
| 80-90 | 100.00±1.50 | 90.63±1.36 |
| 90-100 | 0.00 | 7.50±0.04 |
| >100 | 0.00 | 0.00 |

Table.R14. Diastolic pressure of blood in healthy and diabetic group

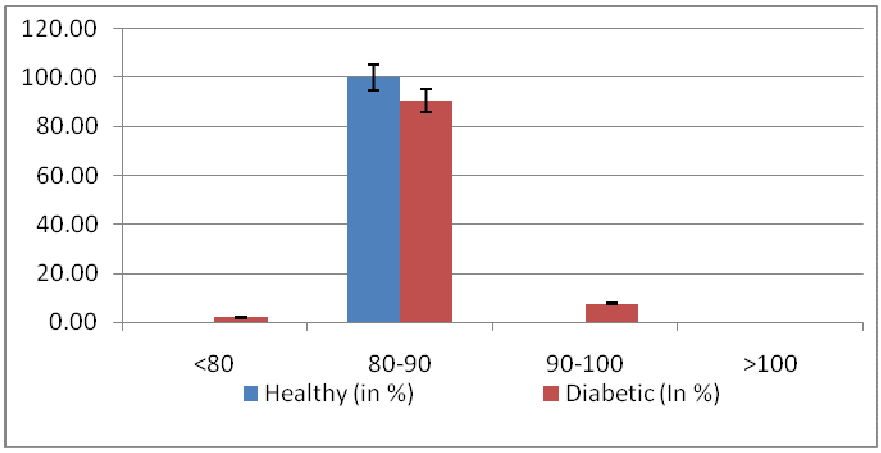


Fig.R14. Diastolic pressure of blood in healthy and diabetic group

**Correlation between uric acid and HbA1c:** A comparison was made between uric acid and HbA1c, This comparison gives a significant positive correlation between uric acid and HbA1c with $p<0.05$, which meant an increment in uric acid must increase HbA1c in diabetic population.

Female and male had different range of uric acid, so in this study a comparison was made individually in female and male with healthy and diabetic population.

**Female:** In present study, it was observed that the HbA1c value in healthy population 4.38±0.43% in range <5, 81.88±7.74% in range 5-6, 13.75±1.29% in range 6-7 whereas in diabetic population 35.00±3.27 in range 7-8, 60.63±5.67 in range 8-9 and 4.38±0.41 in range >9. This was shows HbA1c in diabetic population had statistically higher than that of healthy population (Table R15 and Fig. R15). Similar values in gradually increased range reported for serum uric acid (mg/dL) 20.00±1.94 % in 2.4-3.6 range, 54.29±5.20% in 3.6-4.8 range and 25.71±2.43% in 4.8-6.0 range in healthy population whereas 52.63±4.98% in 6.0-7.2 range, 44.74±4.23% in 6.0-7.2 range and 2.63±0.25% in 6.0-7.2 range diabetic population presented in Table R15 and Fig. R15.

**CONCLUSION**:

Diabetes (Type I (IDDM) and Type 2 (NIDDM) are multi factorial disease. Type 2 is caused by oligo and polygenic genetic factors as well as non-genetic factors (environmental) that result from a lack of balance between the energy intake and output

and other life style related factors. Type 2 diabetes and obesity are major global health problems with increasing incidence and prevalence in both the western world and in the developing countries. Type 2 diabetes is primarily characterized by obesity, insulin resistance and a relative deficient insulin secretion by the pancreatic β-cell and is influenced by lifestyle, environment and genetic factors. Diabetes is considered a state of increased oxidative stress. Persistent hyperglycemia secondary to insulin resistance and diminished insulin secretion in type 2 diabetes leads to progressing organ injuries known as late or chronic diabetes complications. Currently serum uric acid is not considered a metabolic biomarker in diabetes. Purpose of the current study was to find the significant associations, correlations and to develop regression models between uric acid, HbA1c, and serum insulin among diabetic patients. Prevalence of diabetes mellitus in World population is increasing in epidemic. Diabetes is one of the major causes of premature mortality, stroke, cardiovascular disease, peripheral vascular disease, congenital malformations as well as long- and short-term disability.

Increased uric acid levels were associated with increased risk of development of hypertension, cardiovascular disease and progression of chronic kidney disease. Uric acid levels were positively associated with BMI, waist circumference, triglycerides, systolic blood pressure, diastolic blood pressure, glycohemoglobin, fasting plasma glucose, postprandial 2-hour plasma glucose (all $P < 0.05$), and negatively associated with HDL-cholesterol ($P < 0.001$).Uric acid was significantly and inversely correlated with HbA1c ($P < 0.001$) and positively correlated with serum insulin ($P < 0.005$). In Conclusion thehigher levels of uric acid are associated with lower HbA1c both in type-2 diabetic patients. Uric acid is involved in the augmentation of insulin secretion in type-2 subjects. Type 2 diabetes variants explain below 10% of the genetic contribution to risk of disease which is underlined by the poor ability of genetic variants to predict type 2 diabetes. Reasons for the large residual variation are many.

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