

International Journal of Medical Science and Innovative Research (IJMSIR)

# IJMSIR : A Medical Publication Hub

Available Online at: www.ijmsir.com Volume – 7, Issue – 5, September – 2022, Page No. : 46 – 57

To study the prevalence of prediabetes in tertiary care centre & to see the association of prediabetes with cardio metabolic risk factors

<sup>1</sup>Dr. Mukesh Patel, Department of Gen. Medicine, N.S.C.B. Medical College & Hospital, Jabalpur, M.P.

<sup>2</sup>Dr. Shivendra Nagiya, Department of Gen. Medicine, N.S.C.B. Medical College & Hospital, Jabalpur, M.P.

<sup>3</sup>Dr. Deepshikha Evane, Department of Gen. Medicine, N.S.C.B. Medical College & Hospital, Jabalpur, M.P.

**Corresponding Author:** Dr. Mukesh Patel, Department of Gen. Medicine, N.S.C.B. Medical College & Hospital, Jabalpur, M.P.

**Citation this Article:** Dr. Mukesh Patel, Dr. Shivendra Nagiya, Dr. Deepshikha Evane, "To study the prevalence of prediabetes in tertiary care centre & to see the association of prediabetes with cardio metabolic risk factors", IJMSIR-September - 2022, Vol – 7, Issue - 5, P. No. 46 - 57.

Type of Publication: Original Research Article

**Conflicts of Interest:** Nil

# Abstract

**Introduction:** The term prediabetic is an intermediate stage used to describe a person with impaired blood glucose tolerance levels of fasting between 100 and 126 mg/dl of blood or whose 2-hour postprandial blood glucose was 140-200 mg/dl. And HbA1c 5.6 % to 6.4 % according to American Diabetes Association (ADA) criteria. Considerable number of these people in the prediabetic stage will go on to develop type 2 diabetes. Studies in India had shown that nearly 40- 55% of the people with prediabetic stage will develop to type 2 diabetes mellitus over a period of 3-5 years.

**Background:** Considerable number of people in the prediabetic stage will go on to develop type 2 diabetes. Early diagnosis and intervention of prediabetic and their cluster of risk factors can prevent the cardiovascular events and other complications of diabetes. Aims & Objectives: Primary

**objective:** To Study the Prevalence of Prediabetes in Tertiary Care Centre. Secondary Objective: To Study

Association of Prediabetes with Cardio metabolic Risk Factors.

**Material and Methods:** Study area and target Population: The present study is carrying out from Department of Medicine, Netaji Subhash Chandra Bose Medical College & Hospital, Jabalpur (M.P.). Sample size: 287, Study design: Observational and Prospective study

Inclusion criteria: Age 18 - 80 who are not diabetic. Patient should give written informed consent to participate in the study

Exclusion criteria: Patient with diabetes mellitus. Any treatment with antidiabetic. Pregnancy.

Hemolytic Anemia. Drugs that affect glucose level. American Diabetes Association (ADA) guideline Cut-off point of FBS 100-125 mg/dl & HbA1C 5.7-6.4 %. Which be used in study. Result: 287 patients are selected for my study, out of which 34 patients have prediabetes. In these 34 pre diabetes patients we study cardiometabolic risk factors. So prevalence of prediabetes 15.6%. Total 34 patients included in the study based on the inclusion and exclusion criteria. Among them 16 were women and 18 were men. Women constitute around 47.1% of total cases and rest 52.9% by men. It is found that in 34 prediabetes patients, mean of BMI is 24.83 and standard deviation is 2.37.

So high risk of development of prediabetes is seen in patient with high values of BMI. Mean values of FBS in study subjects is 109.02 and standard deviation is 9.30. Mean values of HBA1c is 5.95 and standard deviation is 0.28. Mean value of triglyceride is 170.1512 and standard deviation is 40.45447. Mean values of Total serum cholesterol is 227.0029 and Standard deviation is 56.798. it is observed that High levels of Cholesterol and Triglycerides are associated with prediabetes. FBS have significant association with Age, Statistical analysis reveal significant trend in distribution with P < 0.05 (P = 0.016). FBS have significant association with Systolic Blood pressure, Statistical analysis reveal significant trend in distribution with P < 0.05 (P = 0.04). FBS have significant association with Diastolic Blood pressure, Statistical analysis reveal significant trend in distribution with P < 0.05 (P = 0.049). HBA1C have positive correlation with BMI, age, systolic Blood pressure and Diastolic blood pressure. HBA1C have significant association with BMI. Statistical analysis reveal significant trend in distribution with P < 0.05 (P = 0.004). Conclusion: Study done in 287 patients from general population. Out of which 34 have prediabetes. So prevalence of prediabetes is 15.6% in our study. In our study Prevalence of prediabetes is more common in males than females. In our study Prediabetes is more common in 36-55 age groups.

# Introduction

The term prediabetic is an intermediate stage used to describe a person with impaired blood glucose tolerance levels of fasting between 100 and 126 mg/dl of blood or

whose 2-hour postprandial blood glucose was 140-200 mg/dl. And HbA1c 5.6 % to 6.4 % according to American Diabetes Association (ADA) criteria. Considerable number of these people in the prediabetic stage will go on to develop type 2 diabetes. Studies in India had shown that nearly 40- 55% of the people with prediabetic stage will develop to type 2 diabetes mellitus over a period of 3-5 years.<sup>1,2</sup> It has been established by few studies that there is a clear link between type 2 diabetes mellitus and development of cardiovascular risk factors. In this study, we diagnose patients in prediabetic stage and their clustering with the other cardiometabolic risk factors. The clustering of risk factors such as overweight and obesity, dyslipidemia, being older than 40 years, sedentary habits, smoking, alcoholism, hypertension, and intake of fruits and vegetables were studied. Diabetic represents the tip of the iceberg. Early diagnosis and intervention of prediabetic and their cluster of risk factor can prevent the cardiovascular events and complications of diabetes such as diabetic retinopathy, neuropathy, and nephropathy.

In a cohort of individuals from the Diabetes Prevention Program (DPP), who were at high risk for developing diabetes, the prevalence of diabetic retinopathy was 7.9%<sup>3</sup>. In a different study, the prevalence of peripheral neuropathy was higher in those with prediabetes than in those with normal glucose tolerance, and was similar to that in participants with recently diagnosed diabetes<sup>4</sup>. An association between prediabetes and increased risk of chronic kidney disease (CKD) has also been reported, based on results from a meta-analysis<sup>5</sup>. Another metaanalysis showed that, compared with normo glycaemic individuals, there is an increased risk of cardiovascular disease, coronary heart disease, and stroke and all-cause mortality in those with prediabetes<sup>6</sup>. In addition, elevated plasma glucose levels indicative of prediabetes in early pregnancy are associated with increased risk of adverse pregnancy outcomes, and may also lead to gestational diabetes in later pregnancy<sup>7</sup>.

increased risk of adverse pregnancy outcomes, and may also lead to gestational diabetes in later pregnancy7. As a chronic disease, the long-term implications of diabetes contribute to poor quality of life and greatly increase health care expenditure8. Prediabetes may however be reversible, through the implementation of lifestyle modification programmes based around the adoption of healthier diet and increased levels of physical activity9.

## **Global scenario**

Prediabetes is a high-risk state for diabetes mellitus with an annualized conversion rate of 5%-10%. The prevalence of prediabetes is increasing worldwide and it is projected that >470 million people will have prediabetes in 2030. The current medical literature has highlighted the importance of non-glycemic factors such as arterial hyper tension, dyslipidemia in escalating atherosclerosis.10Even though diabetes mellitus is now evident across all sections of society within India, there are very few studies reported from the rural population. Misra et al11 in a review of diabetic studies in rural India found that prevalence of diabetes increased from 1.9% in 1994 to upwards of 12% in 2009. As there are no regional study done in central part of India regarding pre diabetes so we are conducting this study to see the prevalence of prediabetes in our region.

## **Aims & Objectives**

• Primary objective: To Study the Prevalence of Prediabetes in Tertiary Care Centre.

• Secondary Objective: To Study Association of Prediabetes with Cardiometabolic Risk Factors.

# **Background History of diabetes**

Diabetes is one of the oldest known diseases.12 An Egyptian manuscript from c. 1550 BC mentions the

phrase —the passing of too much urine12 The great Indian physician Sushruta (fl. 6th century BC)18 identified the disease and classified it as Medhumeha.13 He further identified it with obesity and sedentary lifestyle, advising exercises to help "cure" it.13 The ancient Indians tested for diabetes by observing whether ants were attracted to a person's urine, and called the ailment "sweet urine disease" (Madhumeh).

The first complete clinical description of diabetes was given by the Ancient Greek physician Are taeus of Cappadocia (fl. 1st century CE), who noted the excessive amount of urine which passed through the kidneys and gave the disease the name —diabetes.<sup>12</sup>

## **Prediabetes**

Prediabetes is the precursor stage before diabetes mellitus in which not all of the symptoms required to diagnose diabetes are present, but blood sugar is abnormally high. Prediabetes represents the tip of the iceberg. This stage is often referred to as the "grev area.<sup>14</sup> It is not a disease: the American Diabetes Association says,15Prediabetes should not be viewed as a clinical entity in its own right but rather as an increased risk for diabetes and cardiovascular disease (CVD). Prediabetes is associated with obesity (especially abdominal or visceral obesity), dyslipidemia with high triglycerides and/or low HDL cholesterol, and hypertension.<sup>15</sup> It is thus a metabolic diathesis or syndrome, and it usually involves no symptoms and only high blood sugar as the sole sign. With a range of criteria available for prediabetes identification, it is not surprising that populations with prediabetes identified by each method vary widely and have limited overlap16. These differences in screening criteria for prediabetes may result in incorrect diagnoses, leading to some people being unnecessarily treated, and others left without treatment to prevent or delay the onset

of overt type 2 diabetes. Similarly, it is difficult to assess the global burden of prediabetes.

#### **Prevalence of Prediabetes**

Prevalence estimates of prediabetes reported in the literature vary greatly, due to the diagnostic criteria used, Guidelines lead to much higher prevalence rates compared with those defined by WHO guidelines; in a cohort of 1547 American adults without diabetes, changing the lower IFG threshold from 110 mg/dL to 100 mg/dL resulted in an increase in prediabetes prevalence from 19.8 to 34.6% 17. A large meta-analysis of studies that reported prevalence in Caucasian and Asian cohorts estimated IFG prevalence at 36.0% using WHO guidelines and 53.1% using ADA guidelines. By contrast, the same meta-analysis described similar prevalence rates for patients who have both IFG and IGT; 15.8% for WHO and 20.2% for ADA guidelines18. According to the ADA guidelines, an abnormal finding of any of the three criteria (IFG, IGT and HbA1C) is sufficient to confirm prediabetes19. Relying on one test may underestimate prevalence. Pathophysiology of prediabetes: Trajectories of glycemic changes in prediabetes Blood glucose levels are strictly regulated in healthy individuals. With the evolution toward type 2 abnormalities diabetes. in glucose and insulin concentrations and dynamics occur continuously and insidiously over many years20. Trajectories of fasting and post-load glucose levels as well as insulin sensitivity and insulin secretion (b-cell function) preceded the development of type 2 diabetes in the British Whitehall II study21. Pointed out that individuals with glucose levels approximating 94 mg/dl (5.2 mmol. 1), considered as normal and below the IFG threshold, are at increased risk for developing diabetes, it is conceivable that postprandial glucose levels below 140 mg/dl (7.8 mmol/l), the cut point for defining IGT, confer increased

risk for developing diabetes. Therefore, individuals with even subtle abnormalities in either fasting or post-load glucose levels should be identified as early as possible, well before achieving critical thresholds for IFG or IGT especially if they have associated risk factors for progression to prediabetes.

#### Multistage model of diabetes development

Weir40 described a multistage model of diabetes development that corresponds to progression of diabetes, each stage marked by changes in b-cell mass, phenotype and function. The first stage is defined by a long period of insulin resistance accompanied by a compensatory increased rate of insulin secretion and increased b-cell mass. The second stage constitutes the stable adaptation period when b cells no longer fully compensate for increased insulin resistance and is accompanied by changes in b-cell phenotype demonstrated by changes in gene and protein expression; thus, fasting and post-load glucose values are not completely maintained. Glucose levels rise to 5.0–6.5 mmol/1 (89–116 mg/dl).

# **Glucose Dysregulation**

Glucose dysregulation has been reviewed by Vasudevan and Garber<sup>22</sup>. In the fasting state, hepatic glucose output (HGO) results from both glycogenolysis and gluconeogenesis accounting for approximately 90 % of the glucose released into the circulation. Conversely, in the postprandial state, HGO is suppressed to help limit the rise in plasma glucose levels and the liver stores fuel by conversion to glycogen. Studies using different measures of b-cell function have reported severely abnormal (up to 80 % decreased) insulin secretion in prediabetes, findings supported by autopsy reports describing a 50 % decrease in b-cell volume in IFG.

## Skeletal muscle glucose metabolism

Aside from impaired insulin action, kinetic defects in insulin action in obesity have also been demonstrated

whereby the rate of activation of insulin's effect to stimulate glucose disposal is decreased and the rate of deactivation of insulin's effect is increased<sup>23</sup>. In physiologic circumstances, insulin is secreted postprandially in a phasic rather than a steady-state manner. It is therefore likely that kinetic defects in insulin action are of functional importance and steady-state measurements of insulin action underestimate alterations in insulin sensitivity.

## Glucolipotoxicity

The relation between glucose and lipid toxicity with insulin resistance and b-cell toxicity has been reviewed<sup>24</sup>. Lipid accumulation in the liver appears to be a principal mechanism associated with obesity-related insulin resistance and type 2 diabetes mellitus<sup>25</sup>. Altered metabolism of triglyceride- rich lipoproteins is an integral part of the atherogenic dyslipidemia in insulin resistant prediabetic individuals and in type 2 diabetes mellitus, and is characterized typically by elevated serum triglyceride levels and decreased high-density lipoprotein cholesterol (HDL-c).

# Altered fat distribution

Body fat distribution is an additional factor that mitigates insulin resistance. Independent total body fat mass, accumulation of adipose within tissue the visceral/abdominal region and liver accentuates insulin resistance. The latter may be related to inflammatory changes in adipose depots with release of cytokines<sup>26</sup>. Intraperitoneal (visceral) adipose tissue may be particularly deleterious as it drains directly to the liver via the portal vein, therefore exposing the liver to high  $FFA^{23}$ . of Furthermore. concentrations visceral adipocytes appear to be more responsive to catecholamine-stimulated lipolysis and less so to suppression of lipolysis by insulin.

#### Genetics

Type 2 DM, which is the condition for which prediabetes is a precursor, has 90–100% concordance in twins; there is no HLA association.<sup>27</sup> Genetics play a relatively small role, however, in the widespread occurrence of type 2 diabetes mellitus. This may be deduced logically from the huge increase in the occurrence of type 2 diabetes mellitus that has correlated with the significant change in western lifestyle and diet. As the human genome is further explored, it is possible that multiple genetic anomalies at different loci will be found that confer varying degrees of predisposition to type 2 diabetes mellitus.<sup>28</sup>

# **DPP-IV** Inhibitors

Based on their mechanism of action discussed earlier, agents in this class could in principle be beneficial in diabetes prevention although there are no long-term studies available examining the enhancement of insulin secretion into preservation of b-cell function29. Hypoglycemia, weight loss or appetite suppression is not associated with their use. Cost effectiveness of these agents need to be taken into consideration as well.

## Cardiometabolic

Definitions of cardiometabolic risk factors The International Federation of Diabetes criteria were used to determine whether participants had any of the four conditions that are part of the metabolic syndrome (i.e., central obesity, high triglycerides, low HDL cholesterol, and high blood pressure) (122,123). For all adolescents, a high triglyceride level was defined as >1.7 mmol/l (150 mg/dl), and high blood pressure was defined as a systolic blood pressure >130 mmHg or a diastolic blood pressure >85 mmHg (122,123). For adolescents aged 12–15 years, central obesity was defined as a waist circumference >90th percentile, and low HDL cholesterol was defined as <1.03 mmol/l (40 mg/dl). For adolescents aged 16–19

years, the International Federation of Diabetes sex- and race/ ethnicity specific cutoff values of waist circumference (i.e., white males >94 cm, African American males >94 cm, Mexican-American males >90 cm, white females >80 cm, African American females>80 cm, and Mexican- American females 80 cm) were used to define central obesity. Sex-specific cutoff values of HDL cholesterol (i.e., <1.03 mmol/l [40 mg/ dl] in males and <1.29 mmol/l [50 mg/ dl] in females) were used to define low HDL cholesterol (122). Current use of prescribed antihypertensive medicines for the treatment of previously diagnosed hypertension was considered to be high blood pressure.

#### **Material and Methods**

#### **Study area and target Population**

The present study is carrying out from Department of Medicine, Netaji Subhash Chandra Bose Medical College & Hospital, Jabalpur (M.P.).

• Sample size: 287, Study design: Observational and Prospective study.

• Inclusion criteria: Age 18 – 80 who are not diabetic. Patient should give written informed consent to participate in the study.

Exclusion criteria: Patient with diabetes mellitus. Any treatment with antidiabetic. Pregnancy. Hemolytic Anemia. Drugs that affect glucose level. American Diabetes Association (ADA) guideline Cut-off point of FBS 100-125 mg/dl & HbA1C 5.7-6.4 %. Which be used in study

• Data collection method: All the patient information will be recorded by using structured schedule (case report form) and entered in Microsoft Excel Sheet.

• Tools to be used: Hematological & biochemical investigations to be done at Department of Pathology, Microbiology, & Biochemistry at NSCB MCH Jabalpur (M.P.)

Techniques to be used: With all aseptic precautions, 4ml blood sample from antecubital vein of patient will be taken in 1 EDTA vial for HbA1c, and 1 vial for FBS

• Laboratory procedures: Hba1c-Latex enhanced immuno agglutination methods

FBS- Glucose oxidase-phenol and 4 aminophenazo methods

Sampling method: Considering the best availability of the patients by reviewing the previous records of this health facility, to achieve the maximum sample size we will randomly screen all patients and select those who fulfil the inclusion and exclusion criteria and are ready to give the written informed consent. Informed and written consent would be obtained from each individual and the participation in the project would be on voluntary basis.

#### Result

287 patients are selected for my study, out of which 34 patients have prediabetes. In these 34 pre diabetes patients we study cardiometabolic risk factors. So prevalence of prediabetes 15.6%.

Table 1: Sex wise distribution amoung patients

| Sex    | No of patients | Percentage % |
|--------|----------------|--------------|
| Female | 16             | 47.1         |
| Male   | 18             | 52.9         |
| Total  | 34             | 100.0        |

Total 34 patients included in the study based on the inclusion and exclusion criteria. Among them 16 were women and 18 were men. Women constitute around 47.1% of total cases and rest 52.9% by men.

Table 2: Age wise population Distribution AmoungPatient

| Age Group | No. of   | Percentage % | Female | Male |        |
|-----------|----------|--------------|--------|------|--------|
|           | patients |              |        |      |        |
| 24-35 yrs | 8        | 23.5         | 5      | 3    |        |
| 36-45 yrs | 12       | 35.3         | 3      | 9    |        |
| 46-55 yrs | 8        | 23.5         | 4      | 4    | т<br>Ц |

Dr. Mukesh Patel, et al. International Journal of Medical Sciences and Innovative Research (IJMSIR)

| <br>56-65 yrs | 4  | 11.8  | 2  | 2  |
|---------------|----|-------|----|----|
| 66-75 yrs     | 2  | 5.9   | 2  | 0  |
| Total         | 34 | 100.0 | 16 | 18 |

According to age, 8 patients were less than 35 years old. 12 patients were in 36-45 age group, 8 were in 46-55 age group, 4 were in 56-65 age group and 2 patients were above 66 years. Population distribution shown in the Table 2. As we can see most of patients fall in age group 36-55 years, have high chances developing diabetes in later stage of life. Age of patients ranges from 24 to 75 years with mean age 45.03 and Standard Deviation 12.296.

Table 3: Frequency Table for Smoking.

| Smoking | Ν  | %     |
|---------|----|-------|
| No      | 22 | 64.7  |
| Yes     | 12 | 35.3  |
| Total   | 34 | 100.0 |

Table 4: Descriptive statistics of the variables in study population.

| Variables          | Ν  | Minimum | Maximum | Mean     | SD       |
|--------------------|----|---------|---------|----------|----------|
| Age                | 34 | 24      | 75      | 45.03    | 12.296   |
| PR                 | 34 | 69      | 102     | 78.53    | 6.561    |
| RR                 | 34 | 10      | 19      | 14.91    | 2.109    |
| Systolic BP        | 34 | 78      | 128     | 113.76   | 9.869    |
| Diastolic BP       | 34 | 54      | 86      | 73.71    | 5.932    |
| Asian BMI Criteria | 34 | 19.30   | 29.00   | 24.8332  | 2.37041  |
| FBS                | 34 | 68.00   | 123.89  | 109.0276 | 9.30809  |
| HBA1C              | 34 | 5.60    | 6.80    | 5.9544   | .28990   |
| SERUM CHOLESTEROL  | 34 | 103.00  | 318.00  | 227.0029 | 56.79822 |
| SERUM LDL          | 34 | 73.60   | 206.00  | 126.8324 | 31.80110 |
| HDL                | 34 | 21.50   | 70.60   | 40.0853  | 13.84317 |
| TRIGLYCERIDE       | 34 | 89.00   | 269.90  | 170.1512 | 40.45447 |
| WBC                | 34 | 3800    | 18800   | 8488.53  | 3613.265 |
| Hemoglobin         | 34 | 5.20    | 16.50   | 11.4688  | 2.47091  |
| Platelet           | 34 | 1.25    | 4.35    | 2.2124   | .73606   |
| SGOT               | 34 | 18.00   | 120.00  | 48.7232  | 23.74249 |
| SGPT               | 34 | 14.00   | 240.00  | 48.8694  | 38.41353 |
| Total Bilirubin    | 34 | .23     | 1.87    | .7650    | .34111   |
| Uric acid          | 34 | 3.43    | 7.60    | 5.6618   | 1.05414  |

It is found that in 34 prediabetes patients, mean of BMI is 24.83 and standard deviation is 2.37. So high risk of development of prediabetes is seen in patient with high values of BMI. Mean values of FBS in study subjects is 109.02 and standard deviation is 9.30. Mean values of

HBA1c is 5.95 and standard deviation is 0.28. Mean value of triglyceride is 170.1512 and standard deviation is 40.45447. Mean values of Total serum cholesterol is 227.0029 and Standard deviation is 56.798. it is observed

that High levels of Cholesterol and Triglycerides are

associated with prediabetes.

|            |                             | Age         | PR     | RR     | Systolic | Diastolic | Asian BMI Criteria |
|------------|-----------------------------|-------------|--------|--------|----------|-----------|--------------------|
|            | Pearson Correlation         | .411*       | -0.265 | -0.226 | .354*    | .340*     | .346*              |
| FBS        | P value                     | 0.016       | 0.13   | 0.198  | 0.04     | 0.049     | 0.045              |
|            | Pearson Correlation         | 0.153       | -0.226 | 368*   | 0.301    | 0.265     | .477**             |
|            | P value                     | 0.388       | 0.198  | 0.032  | 0.084    | 0.13      | 0.004              |
|            | ation is significant at the | 0.01 level. |        |        |          |           |                    |
| * Correlat | ion is significant at the ( | ).05 level. |        |        |          |           |                    |

Table 5: Correlation of FBS & HbA1C with age, PR, RR, systolic BP diastolic BP & Asian BMI criteria

FBS have positive correlation with BMI, age, systolic Blood pressure and Diastolic blood pressure. FBS have significant association with BMI. Statistical analysis reveal significant trend in distribution with P < 0.05 (P =0.045). FBS have significant association with Age, Statistical analysis reveal significant trend in distribution with P < 0.05 (P = 0.016). FBS have significant association with Systolic Blood pressure, Statistical analysis reveal significant trend in distribution with P < 0.05 (P = 0.04). FBS have significant association with Diastolic Blood pressure, Statistical analysis reveal significant trend in distribution with P < 0.05 (P = 0.049). HBA1C have positive correlation with BMI, age, systolic Blood pressure and Diastolic blood pressure. HBA1C have significant association with BMI. Statistical analysis reveal significant trend in distribution with P < 0.05 (P = 0.004).

Table 6: Correlation of FBS & Hba1C with serum cholesterol, serum LDL, HDL & triglyceride.

|                 |                   | Serum           | Serum  | HDL   | Triglyceride |
|-----------------|-------------------|-----------------|--------|-------|--------------|
|                 | Pearson           | 0.324           | 0.006  | 0.203 | 0.138        |
|                 | P value           | 0.061           | 0.973  | 0.250 | 0.436        |
|                 | Pearson           | 0.338           | -0.118 | 0.343 | 0.230        |
| HBA1 C          | P value           | 0.049           | 0.508  | 0.047 | 0.190        |
| ** Correlation  | is significant at | the 0.01 level. |        | -     |              |
| * Correlation i | s significant at  | he 0.05 level.  |        |       |              |

HBA1C have positive correlation with Serum cholesterol, HDL, triglycerides. HBA1C have significant association with Serum cholesterol, Statistical analysis reveal significant trend in distribution with P < 0.05 (P =

0.049). HBA1C have significant association with HDL, Statistical analysis reveal significant trend in distribution with P < 0.05 (P = 0.047).

Table 7: Correlation of FBS & Hba1C with WBC, HB, platelet, SGOT, SGPT, t. Bilirubin, uric acid

| 374*  | 0.308  | 0.227        |                    |                           |                                 |
|-------|--------|--------------|--------------------|---------------------------|---------------------------------|
|       | 0.500  | - 0.337      | 686**              | -0.026                    | -0.213                          |
| 0.029 | 0.076  | 0.051        | < 0.0001           | 0.884                     | 0.228                           |
| 0.149 | -0.055 | 0.025        | -0.218             | 0.275                     | -0.047                          |
| 0.399 | 0.759  | 0.89         | 0.216              | 0.115                     | 0.79                            |
|       | 0.149  | 0.149 -0.055 | 0.149 -0.055 0.025 | 0.149 -0.055 0.025 -0.218 | 0.149 -0.055 0.025 -0.218 0.275 |

FBS have significant association with Hemoglobin, Statistical analysis reveal significant trend in distribution with P < 0.05 (P = 0.029).

# Discussion

The present study entitled —To study the Prevalence of Prediabetes in Tertiary Care Centre and to see the Association of Prediabetes with Cardio Metabolic Risk Factors was carried out in the Department of General Medicine, Netaji Subhash Chandra Bose Medical College and Hospital, Jabalpur (M.P), after taking ethical clearance from institutional ethics committee. The study duration was one and half year from 1<sup>st</sup> March 2019 to 31<sup>st</sup> August 2020.

In this study, we selected 287 patients out of which 34 found to have prediabetes. Prevalence of prediabetes in my study group was 15.6%. According to study done by<sup>30</sup>, prevalence of prediabetes is 8.5% and 22.7% respectively. Another study done by<sup>31</sup>.Showing prevalence of prediabetes in general population is 25%.

In this study, prediabetes is more commonly involving men. 52.9% of patients are males and 47.1% is female. According to study done by  $^{30}$ , 57.2% are males and 42.8% are females. Another study done by  $^{31}$ , 60% is males and 40% are female.

In this study most of patients fall in age group 36-55 years, have high chances developing diabetes in later stage of life. In study subjects mean age  $45.03\pm12.296$ . According study done by<sup>30</sup>mean age is  $46.43\pm13.31$ . Another study done by<sup>31</sup>, mean age is  $49.60\pm8.00$ . In these studies maximum number of patients were in 4<sup>th</sup> and 5<sup>th</sup> decade.

In this study prevalence of family history of diabetes was 32.35% and family history of Hypertension was 14.70%. According to study done by<sup>32</sup>prevalence of diabetes in family is 37.10 and Hypertension is 47.70. It is observed

that, family history of diabetes is more important in development of prediabetes in my study subjects.

In this study prevalence of smoking and alcoholism in prediabetes was 35.29% and 50% respectively<sup>33</sup>, showing prevalence of smokers in prediabetes is 3.5%.

In the present study mean value of BMI is  $24.83\pm2.37$ , this is very similar to the study done by<sup>30</sup>in which mean BMI is  $24.47\pm5.45$ . In the study done by<sup>31</sup>, mean of BMI is  $24.20\pm3.49$ . It is observed that prediabetes is more common in patients with high BMI.

In the present study mean value of triglycerides is  $170.15\pm40.45$ . In the study done study done by<sup>32</sup>in which mean of triglycerides is  $129.14\pm36.60$ . Study done by<sup>31</sup>.Mean values of triglycerides are  $142.95\pm47.97$  and  $169.84\pm105.17$ . It is observed high triglycerides levels are associated with prediabets. In the present study mean value of serum Cholesterol is  $227.00\pm56.79$ . In the study done study done by<sup>32</sup>in which mean of serum cholesterol is  $190.58\pm47.05$ . Study done by<sup>31</sup> mean values of cholesterol are  $199.65\pm51.96$  and  $200.02\pm40.54$ . It is observed high serum cholesterol levels are associated with prediabets.

In the present study mean value of HDL is  $40.08\pm13.84$ . In the study done study done by <sup>32</sup>in which mean HDL is  $27.28\pm9.55$ . Study done by<sup>31</sup> mean values of HDL are  $55.18\pm12.21$  and  $45.08\pm11.97$ .

In the present study mean value of LDL is  $126.83\pm31.80$ . In the study done study done by<sup>32</sup>in which mean of LDL is  $124.04\pm32.49$ . study done by<sup>33</sup>, mean values of LDL is  $122.03\pm35.06$  Prediabetes has dear ranged lipid profile according to study done by<sup>34</sup>.

In the present study mean value of SBP and DBP is  $113.76\pm9.86$  and  $73.71\pm5.93$ . In the study done study done by<sup>30</sup>in which mean of SBP and DBP is  $121\pm18.60$  and  $79.15\pm11.79$ .

# Conclusion

Study done in 287 patients from general population. Out of which 34 have prediabetes. So prevalence of prediabetes is 15.6% in our study. In our study Prevalence of prediabetes is more common in males than females. In our study Prediabetes is more common in 36-55 age group. In our study Family history of diabetes mellitus have more association in development of prediabetes than family history of Hypertension. In our study smoking and alcoholism have strong association with the development of prediabetes. In our study patients with high BMI have more risk of developing Prediabetes. In our study prediabetes has dear ranged lipid profile, especially prediabetes have high values of Triglycerides and Total Cholesterol. In our study HBA1C have positive correlation with BMI, age, systolic Blood pressure and Diastolic blood pressure, Serum cholesterol, HDL, triglycerides. In our study FBS have positive correlation with BMI, age, systolic Blood pressure and Diastolic blood pressure.

# Limitations

Due to COVID 19 pandemic, limited number of study participants could be taken. The study participants are taken only from the tertiary care centre, hence these are not representatives of the general population and therefore the results cannot be generalized. Small sample size was another major limitation.

#### References

1. Viswanathan V, Clementina M, Nair BM, Satya Vani K. Risk of future diabetes is as high with abnormal intermediate post-glucose response as with impaired glucose tolerance. J Assoc Physicians India 2007; 55:833-7.

2. Ramachandran A, Snehalatha C, Mary S, Mukesh B, Bhaskar AD, Vijay V, et al. The Indian diabetes prevention programme shows that lifestyle modification and metformin prevent type 2 diabetes in Asian Indian subjects with impaired glucose tolerance (IDPP-1). Diabetologia 2006; 49:289-97.

3. Diabetes Prevention Program Research Group. The prevalence of retinopathy in impaired glucose tolerance and recent-onset diabetes in the diabetes prevention program. Diabet Med. 2007; 24:137–44.

4. Lee CC, Perkins BA, Kayaniyil S, Harris SB, Retnakaran R, Gerstein HC, Zinman B, Hanley AJ. Peripheral neuropathy and nerve dysfunction in individuals at high risk for type 2 diabetes: the PROMISE cohort. Diabetes Care. 2015; 38:793–800.

5. Echouffo-Tcheugui JB, Narayan KM, Weisman D, Golden SH, Jaar BG. Association between prediabetes and risk of chronic kidney disease: a systematic review and meta-analysis. Di abet Med. 2016; 33:1615–24

6. Huang Y, Cai X, Mai W, Li M, Hu Y. Association between prediabetes and risk of cardiovascular disease and all-cause mortality: systematic review and metaanalysis. Bmj. 2016;355: i5953.

7. Metzger BE, Lowe LP, Dyer AR, Trimble ER, Chaovarindr U, Coustan DR, Hadden DR, McCance DR, Hod M, McIntyre HD, et al. Hyper glycemia and adverse pregnancy outcomes. N Engl J Med. 2008; 358:1991–2002.

International diabetes federation: IDF diabetes atlas
 8th edition, 2017.

9. 2017 IDF: IDF clinical practice recommendations for managing type 2diabetes in primary care.

10. Milicevic Z, Raz I, Beattie SD, Campaigne BN, Sarwat's, Gromniak E, et al. Natural history of cardiovascular disease in patients with diabetes: role of hyperglycemia. Diabetes Care. 2008;31: S155-60.

11. Misra P, Upadhyay RP, Misra A, Anand K. A review of the epidemiology of diabetes in rural India. Diabetes Res Clin Pract. 2011; 92:303–11.

12. Dobson, M. (1776). "Nature of the urine in diabetes". Medical Observations and Inquiries 5: 298–310.

13. Medvei, Victor Cornelius (1993). The history of clinical endocrinology. Carn forth, Lancs., U.K: Parthenon Pub. Group. pp. 23–34. ISBN 1-85070-427-9.

14. "Prediabetes" at Dorland's Medical Dictionary.

15. American Diabetes Association (2017), "2.
Classification and diagnosis of diabetes", Diabetes Care,
40 (Suppl 1): S11–S24, PMID 2797 9889, doi: 10. 2337
/dc17- S005.

16. Barry E, Roberts S, Oke J, Vijaya Raghavan S, Norman sell R, Greenhalgh T. Efficacy and effectiveness of screen and treat policies in prevention of type2 diabetes: systematic review and meta-analysis of screening tests and interventions. BMJ. 2017;356.

17. Karve A, Hayward RA. Prevalence, diagnosis, and treatment of impaired fasting glucose and impaired glucose tolerance in non-diabetic U.S. adults. Diabetes Care. 2010; 33:2355–9.

18. Yip WCY, Sequeira IR, Plank LD, Poppitt SD. Prevalence of pre- diabetes across ethnicities: a review of impaired fasting glucose (IFG) and impaired glucose tolerance (IGT) for classification of Dysglycaemia. Nutrients. 2017;9.

19. American Diabetes Association. 2. Classification and diagnosis of diabetes: standards of medical Care in Diabetes-2019. Diabetes Care. 2019, 42: S13–28.

20. C. Weyers, C. Bogardus, D.M. Mott, R.E. Pratley, The natural history of insulin secretory dysfunction and insulin resistance in the pathogenesis if type 2 diabetes mellitus. J. Clin. Invest. 104(6), 787–794 (1999).

21. A.G. Ta 'bak, C. Herder, W. Rathmann, E.J. Brunner, M. Kivima "ki Prediabetes: a high-risk state for diabetes development. Lancet Published online June 9, (2012) doi:10.1016/S0140-6736(12) 60283-9

22. A. Vasudevan, A.J. Garber, Postprandial Hyper glycemia, in Contemporary Endocrinology: Type 2 Diabetes Mellitus: An Evidence- Based Approach to practical Management, ed. by M.N. Feinglos, M.A. Bethel (Humana Press, Totowa, 2008), pp. 97–113.

23. C.H. Courtney, J.M. Olefsky, Insulin Resistance, in Mechanisms of Insulin Action, ed. by A.R. Saltiel, J.E. Pessin (Landes Bioscience and Springer Science? Business Media, New York, 2007), pp. 185–209.

24. D.M. Muoio, T.R. Koves, J. An, C. New Gard, Metabolic Mechanisms of Muscle Insulin Resistance, in Contemporary Endocrinology: Type 2 Diabetes Mellitus: An Evidence-Based Approach to Practical Management, ed. by M.N. Feinglos, M.A. Bethel (Humana Press, Totowa, 2008), pp. 97–1133

25. J.M. Haus, T.P.J. Solomon, C.M. Marchetti, J.M. Edmison, F. Gonzalez, J.P. Kirwan, Free fatty acidinduced hepatic insulin resistance is attenuated following lifestyle intervention in obese individuals with impaired glucose tolerance. J. Clin. Endocrinol. Me tab. 95 (1), 323–327 (2010)

26. E. Ferrannini, A. Gastaldelli, P. Iozzo, Patho physiology of Prediabetes, in Prediabetes and Diabetes Prevention, ed. by M. Bergman (W.B. Saunders Company, Phila del phia, 2011), pp. 327–340.

27. WebMD: Prediabetes. Accessed Jan. 27, 2009.

28. Cotran, Kumar, Collins; Robbins Pathologic Basis of Disease, Saunders Sixth Edition, 1999; 913-926.

29. E. Mus celli, A. Casola Ro, A. Gastaldelli, a Mari, G. Seghieri, B. Astiarraga, Y. Chen, M. alba, J. Holst, E. Ferrannini, Mechanisms for the anti-hyperglycemic effect of sitagliptin in patients with type 2 diabetes. J. Clin. Endocrinol. 97(8), 2818–2819 (2012)

30. Logaraj Muthu Narayanan, Balaji Ramraj, John Kamala Russel; Prevalence of pain among rural adults seeking medical care through medical camps in Tamil Dr. Mukesh Patel, et al. International Journal of Medical Sciences and Innovative Research (IJMSIR) Nadu; Indian Journal of Pain | January-April 2015 | Vol

31. Inderamohan Bisht1, Saurabh Dhanda2, Suman Kumari Chauhan2, Rajinder Yadav2, Suman Yadav1; Prevalence of prediabetes in apparently healthy population of Tehsil Kangra and adjoining areas Bisht I et al. Int J Community Med Public Health. 2018 Nov;5(11):4916-4920

32. Vandana Balgi1, L Harshavardan1, E Sahna2, Shinto K Thomas2; Pattern of Lipid Profile Abnormality in Subjects with Prediabetes International Journal of Scientific Study | February 2017 | Vol 4 | Issue 11.

33. Shahla Safari, Masoud Amini, Ashraf Aminorroaya & Awat Feizi; Patterns of changes in serum lipid profiles in prediabetic subjects: results from a 16-year prospective cohort study among first-degree relatives of type 2 diabetic patients; Published: 23 August 2020.

34. Subodh kausal, T K Kamble; Lipid Profile in Prediabetes; Assoc Physician India. 2016 Mar; 64 (3); 18-21.

<sup>29 |</sup> Issue 1.