



The Prevalence of Prediabetes and Its Association with Metabolic Syndrome and Insulin Resistance in the Central Indian Population

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Author's contribution

The sole author designed, analyzed, interpreted and prepared the manuscript.

Article Information

DOI: 10.9734/AJMAH/2018/42567

Editor(s):

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Reviewers:

(1) Fulden Sarac, Ege University, Turkey.

(2) Sembol Yildirmak, Giresun University, Turkey.

Complete Peer review History: <http://www.sciedomain.org/review-history/26085>

Received 4th June 2018

Accepted 12th August 2018

Published 3rd September 2018

Original Research Article

ABSTRACT

Pre-diabetes can be recognized as either impaired fasting glucose (IFG) or impaired glucose tolerance (IGT). One hundred forty subjects participating in the different medical hospital out patient's department (OPDs) were selected. The study comprised sixty control and sixty prediabetic individuals aged 30-60 years. A positive correlation was observed between fasting serum glucose, anthropometric parameters cardiovascular parameters and biochemical parameters except for HDL cholesterol which showed a negative correlation. This study has important implications for identification of subjects at higher risk for future type-2 diabetes and suggested that mass screening of aggressive risk modification and close follow-up should be considered for prediabetic subjects with metabolic syndrome. Appropriate intercession in the form of weight reduction, changes in dietary habits and increased physical activity could help to prevent, or at least delay the onset of diabetes and thus reduce the burden due to noncommunicable diseases in India.

Keywords: *Hyperglycemia; glucose tolerance; kidney failure; cholesterol; anthropometric parameter.*

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ABBREVIATIONS

HDL : High density lipoprotein
EDTA : Ethylenediaminetetraacetic acid
BP : Blood pressure
ELISA : Enzyme linked immuno sorbent assay
GOD : Glucose oxidase method
POD : Peroxidase
T2DM : Diabetics mellitus type-2
IGT : Impaired glucose tolerance
IFG : impaired fasting glucose
IDRS : Indian diabetes risk scale
IR : Insulin resistance

1. INTRODUCTION

Worldwide, an estimated 422 million adult's population was living with diabetes in 2014, compared to 108 million in 1980. The intercontinental prevalence (age-standardized) of diabetes has nearly doubled since 1980, rising from 4.7% to 8.5% in the adult population [1]. This considers an increase in associated risk factors such as being overweight or obese. Over the past decade, diabetes prevalence has risen faster in low- and middle-income countries than in high-income countries [2]. Diabetes of all types can lead to complications in many parts of the body and can increase the overall risk of dying prematurely. Possible complications include leg amputation, Kidney failure, stroke, heart attack, vision loss and nerve damage. In pregnancy, poorly controlled diabetes increases the risk of fetal death and other complications [3]. Pre-diabetes is a state in which blood glucose levels are higher than normal but not high enough for a diagnosis of diabetes. This order is sometimes called impaired fasting glucose (IFG) or impaired glucose tolerance (IGT), depending on the test used to diagnose it [4]. The prime risk factor for type 2 diabetes is a condition called pre-diabetes. People with pre-diabetes are at increased risk of evolve type 2 diabetes, formerly called adult-onset diabetes or non-insulin-dependent diabetes. Studies have shown that most people with pre-diabetes develop type 2 diabetes within 10 years, unless they lose 5 to 7 percent of their body weight—about 10 to 15 pounds for someone who weighs 200 pounds—by making changes in their diet and level of physical activity [5]. People with pre-diabetes also are at increased risk of progressing cardiovascular disease. The present study was conducted to deduce prediabetes in the study population and coalition with metabolic syndrome and insulin resistance.

2. MATERIALS AND METHODS

2.1 Materials and Chemicals

All the useful materials, chemicals, and reagents used in this study were of the standard analytical grade.

2.2 Subjects and Sampling

The patients were contacted and convinced from two Government Hospitals- Hamidia Hospital, Bhopal, and K. N. Katju Hospital, Bhopal, M. P. (INDIA). The patients were examined at the time of their visit to the outpatient department (OPD). The biochemical investigation was done in the department of biotechnology and microbiology of the institute. It was cross-sectional study. Blood was collected from peripheral vein. Collected blood sample kept in EDTA containing vials and blood was centrifuged at 3000rpm for 10 min. The sample was stored and frozen at (-20*c) for further biochemical investigation.

2.3 Ethical Considerations

The study protocol and the procedure were approved by the Ethics Committee of Barkatullah University, Bhopal, and M.P. and also it is submitted in center ethics committee on human research (CECHR) and local ethics committee. At the beginning of the study after explaining all steps of the study for patients a written consent was obtained from all participants, and they were assured that their information will be kept totally secret. All of them were free to leave the study whenever they want. No patients had any severe infection nor were on any routine medication.

2.4 Clinical Measurements

2.4.1 Anthropometric measurement

2.4.1.1 *Body weight (kg)*

The body weight of each subject was recorded on the same platform beam balance, barefooted with the minimum clothing on the body. The subject was made to stand erect on the centre of the platform without touching anything else weight was recorded in kilogram up to the accuracy of 100 gm. Steps were taken to minimize zero error. Machine was checked repeatedly during the study.

2.4.1.2 Height (cm)

Using a vertical measuring rod, height was measured without shoes; subject was made to stand on a flat floor with feet parallel and with heels, buttock, shoulder, and back of head touching the rod. The head was held perfectly erect with the lower border of the orbit in the same horizontal plane as the external auditory meter and arm hanging at the side in a natural manner. Metallic block was lowered gently to make contact with scalp and reading was taken.

2.4.1.3 Body mass index (kg/m²)

$$\text{BMI} = (\text{Weight in kg} / \text{Height in meter}^2)$$

2.4.1.4 Waist circumference (Hip circumference (cm)

Waist circumference was measured at the level of umbilicus with nearest 0.1 cm and person breathing silently; Hip circumference was measured at the level of inter-trochanteric girth according to WHO guide line with the help of non-stretchable tape [6].

2.5 Blood Pressure Measurements

Blood pressure (BP) was measured using sphygmomanometer. The subject was allowed 5 min complete physical and mental rest in calm and quiet surrounding to minimize the effect of anxiety. Blood pressure was recorded in the sitting position in the right arm using the sphygmomanometer (Diamond Deluxe Blood Pressure apparatus). Blood pressure readings were normally recorded to the nearest 2 mm Hg from the top of the mercury meniscus. Systolic pressure was recorded at the first appearance of sound, and diastolic pressure is measured at the disappearance of sound [7]. After the measurement, the cuff was deflated, and the measurement is then repeated after a period of 5 minutes rest and the mean of the two was recorded as the blood pressure reading

2.6 Biochemical Investigations

2.6.1 Fasting blood sugar (GOD/POD METHOD)

The blood glucose level was measured in all the groups by using GOD/POD METHOD (Autoanalyser method) [8]. The blood glucose values were expressed as mg/dl.

2.6.2 Lipid profile

Serum Cholesterol (CHOD – PAP Method)

Cholesterol esters are hydrolyzed by cholesterol esterase. The free cholesterol produced is oxidized by cholesterol oxidase to form cholest-4en-3-one with simultaneous production of hydrogen peroxide which oxidatively couple with 4 aminoantipyrine and phenol in presence of peroxidase to yield a red colored quinoneimine dye complex. Intensity of the color formed is directly proportional to the amount of cholesterol present in the sample [9]. The values were expressed as mg/dl.

2.6.3 HDL cholesterol [PTA method]

High density lipoproteins are separated from other protein fractions by treating serum with phosphotungstic acid and magnesium chloride. High density lipoproteins fractions are precipitate; cholesterol content of which is estimated by enzymatic method [10].

2.6.4 SERUM triglycerides

Lipoprotein lipase hydrolyzes triglycerides to glycerol and free fatty acids. The glycerol formed with ATP in the presence of glycerol kinase forms glycerol- 3-phosphate which is oxidized by the enzyme glycerol phosphate oxidase to form hydrogen peroxide. The hydrogen peroxide further reacts with phenolic compound and 4 aminoantipyrine by the catalytic action of peroxidase to form a cooled quinoneimine dye complex. The intensity of the color formed is directly proportional to the amount of triglycerides present in the sample [11].

2.7 Fasting Insulin (by Elisa Method)

The insulin ELISA test is based on simultaneous binding of human insulin by two monoclonal antibodies, one immobilized on micro well plates and the other conjugates with horseradish peroxidase (HRP). Insulin resistance was assessed using the homeostasis model assessment. [12]. HOMA-IR was calculated using the following formula. HOMA-IR = fasting glucose (mmol/L) x fasting insulin (μU/ml)/22.5

2.8 Statistical Analysis

Statistical analysis was carried out with appropriate statistical software. Data obtained from the study groups were compared by

student't' tests. Correlation analysis between variables was made by Pearson's test: $p < 0.05$ were considered as statistically significant [13]. All results were expressed as mean with their standard deviation (Mean=SD).

3. RESULTS

The study constitutes 60 control and 60 prediabetic subjects aged 30-60 years with average age (42.61±8.70) and (45.43±8.77) years respectively. (Table -1 graph -1). Out of 60 controls having fasting serum glucose (FSG) (>100-125 mg/dl) labeled as prediabetic 38% subjects (23) fell into moderate risk group (IDRS 30-50), and 55% (33) subjects raze into a high-risk group (IDRS >60).(Table 2, Graph 2) In the present study, 28% control subjects and 72% prediabetic subjects were categorized as obese based on BMI. 79% control subjects and 84% prediabetic subjects were categorized as having central obesity based on waist circumference. (Table 3, Graph 3). The mean values of BMI (25.94±2.61 kg/m²) and waist circumference (94.15±11.07 cm) of prediabetic subjects were significantly higher than the control subjects (23.69±7.98 kg/m², 87.45±7.8 cm). (Table 3,

Graph 3). An attempt was made to compare the cardiovascular parameters in study population it was observed that both the systolic and diastolic blood pressure significantly higher in prediabetic subjects as compared to the age and sex match control.(Table 4,5, Graphs 4,5). In the present study in the prediabetic subjects the fasting glucose values (110.4±7.9 mg/dl) as well as fasting insulin values found to be higher than control group (fasting serum glucose 82.9±7.3 mg/dl, fasting insulin 15.6±3.2 mg/dl) indicating normal β cell function in these subjects. (Table 6, Graph 6). On correlation analysis, positive correlation was observed between fasting serum glucose, anthropometric parameters, cardiovascular parameters and biochemical parameters except for HDL cholesterol which showed a negative correlation. The positive association between fasting serum glucose and dyslipidemia with the development of insulin resistance signified that persistent hyperglycemia and dyslipidemia might lead to the development of insulin resistance. Both the general obesity and central obesity were associated with higher risk of development of dyslipidemia, insulin resistance, and altered cardiovascular parameters.

Table 1. Age wise distribution of study population

Variable	Control group (n=70)			Prediabetic group (n=70)		
	Men (n=36)	Women (n=34)	Overall (n=60)	Men (n=36)	Women (n=34)	Overall (n=60)
30 -40	31.2±1.5 (29)	35.5±4.3 (38)	32.3±3.4 (33)	34.8±3.70 (29)	34.3±2.9 (38)	35.4±2.9 (33)
	11	14	25	12	15	27
>40 – 50	45.1±2.9 (48)	43.5±2.7 (38)	44.4±2.9 (43)	47±3.0 (48)	46.8±3.2 (38)	46.9±3.0 (43)
	16	13	29	16	13	29
>50- 60	54.3±1.4 (23)	53.6±1.5 (24)	54±1.41 (24)	56.7±3.5 (23)	57.4±3.5 (24)	57.1±3.4 (24)
	9	7	16	8	7	15

Table 2. Distribution of study population based (IDRS) Indian diabetic risk score

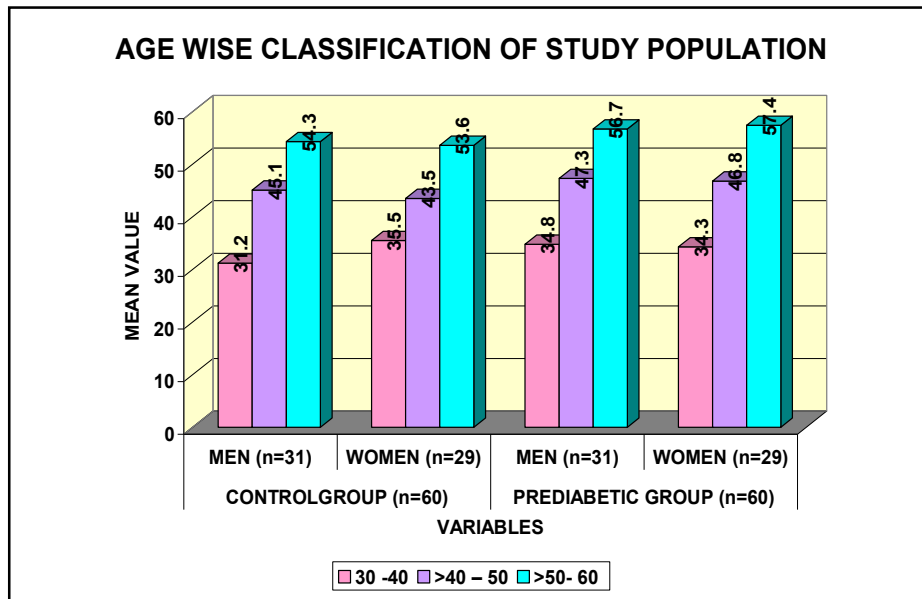
Indian diabetic risk score	Control group		Overall (n=70)	Prediabetic group		Overall (n=70)
	Men (n=36)	Women (n=34)		Men (n=36)	Women (n=34)	
<30 (Low risk)	7 (23)	4 (14)	11 (18)	2 (6)	2 (7)	4 (7)
30-50 (Moderate risk)	19 (61)	15 (52)	34 (57)	14 (45)	9 (31)	23 (38)
>60 (High risk)	5 (16)	10 (34)	15 (25)	15 (49)	18 (62)	33 (55)

Table 3. Comparison of anthropometric parameters in study group

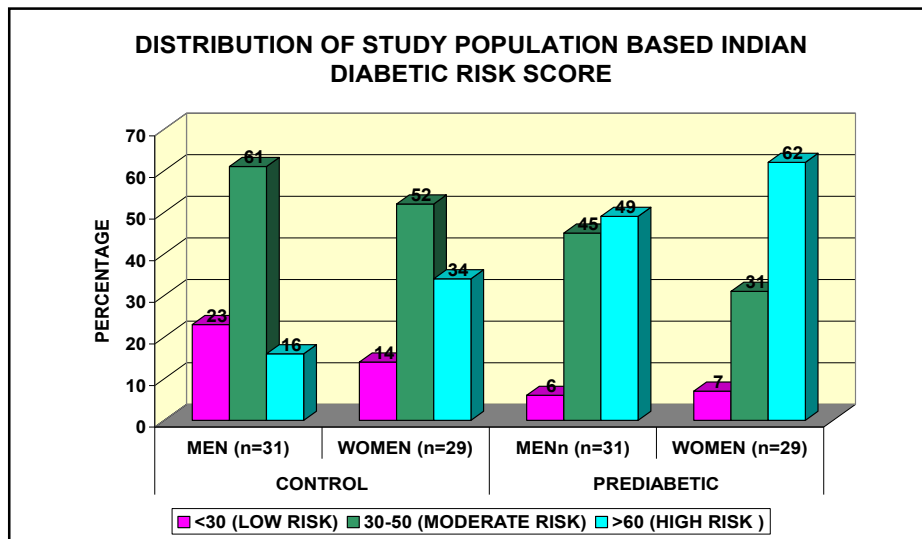
Parameters	Prediabetic group (n=70)	Control group (n=70)	't' value	'p' value
Age(years)	45.43±8.77	42.61±8.70	1.876	NS
BMI(kg/m ²)	25.74±2.61	23.69±7.98	4.213	<0.001
WC(cm)	94.15±11.07	87.45±7.8	3.809	<0.001

Table 4. Comparison of cardiovascular parameters in study population (Student 'T' Test)

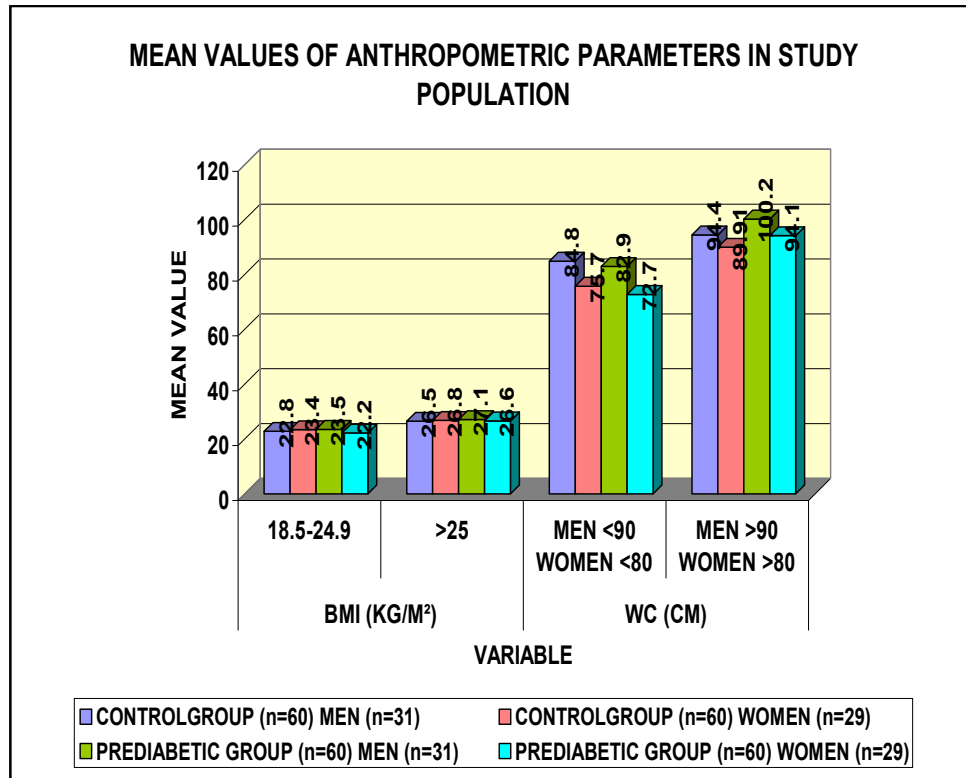
Parameter	Control group (n=70)	Prediabetic group (n=70)	'T' value	P value
SBP(mmHg)	119.7±16.2	133.9±16	4.79	<0.001
DBP(mmHg)	80.2±10	88.1±12.7	3.81	<0.001
PULSE (/min)	79.3±7.4	82.3±7.6	2.19	NS



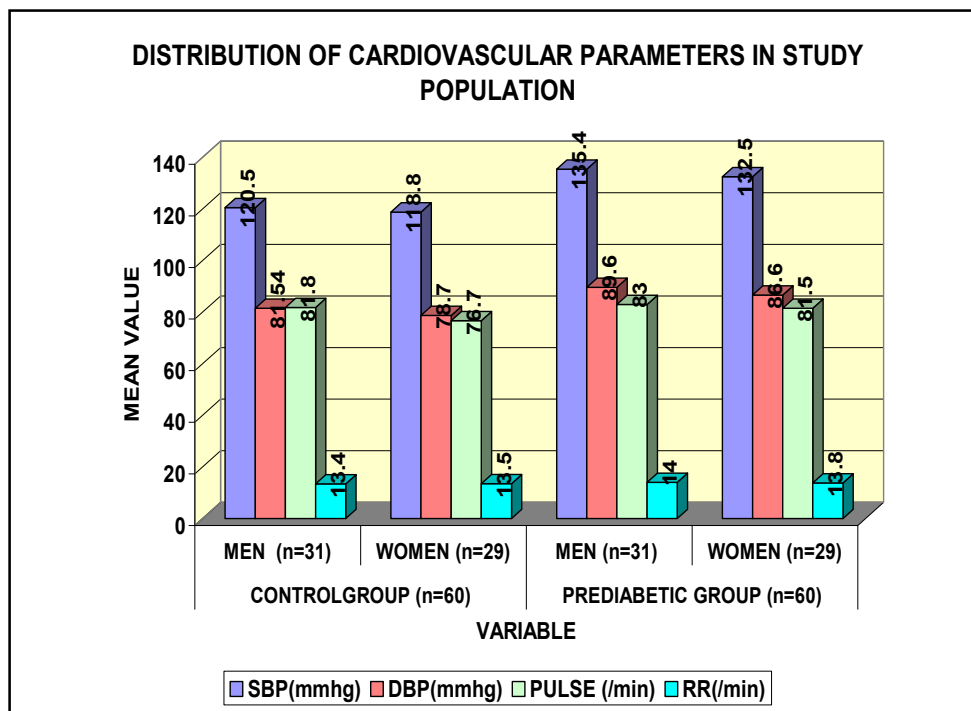
Graph 1. Age wise classification study population



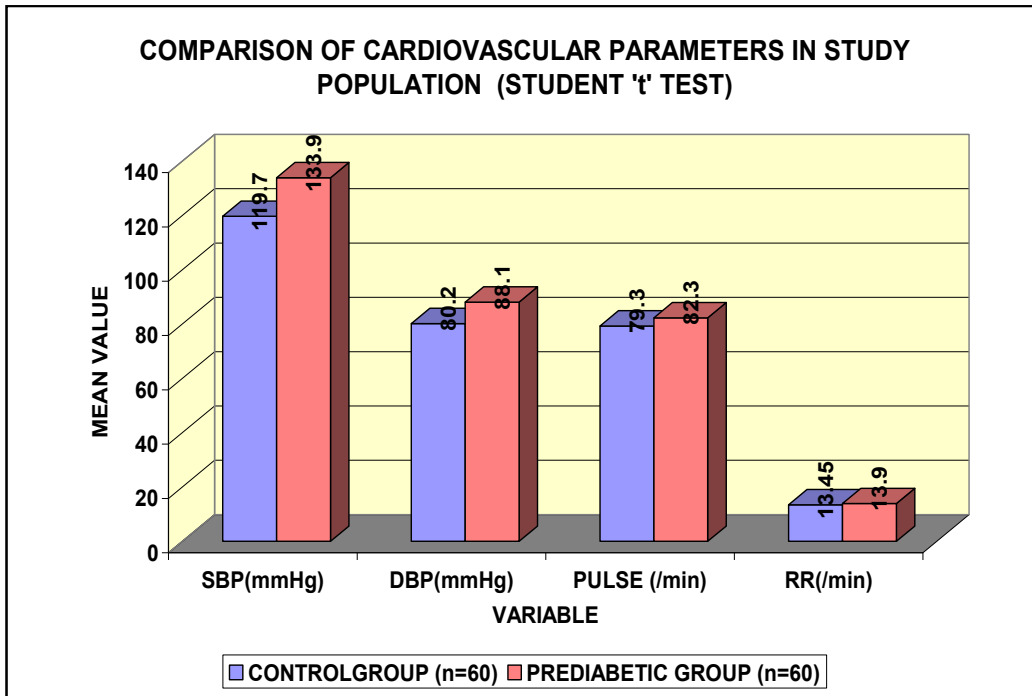
Graph 2. Distrubation of study population based Indian diabetic risk score



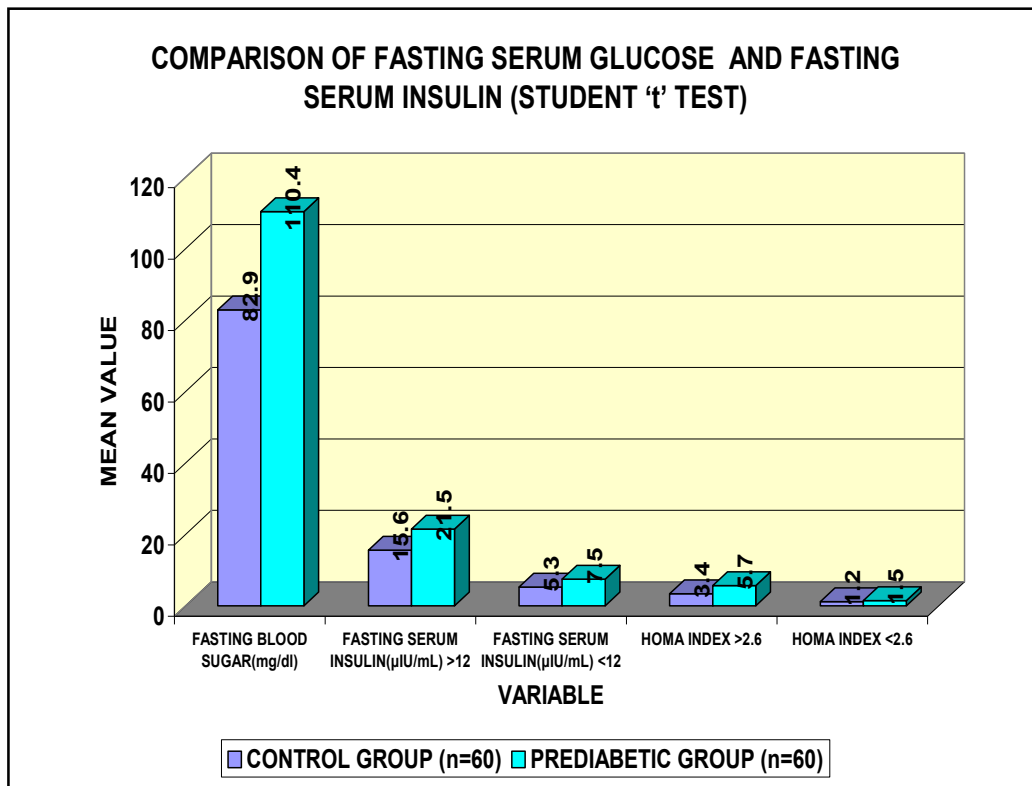
Graph 3. Mean value of anthropometric parameters in study population



Graph 4. Distribution of cardiovascular parameters in study population



Graph 5. Comparison of cardiovascular parameters in study population (student 't' test)



Graph 6. Comparison of fasting serum glucose and fasting serum insulin (student 't' test)

Table 5. Comparison of fasting serum glucose and fasting serum insulin (Student 't' test)

Parameter	Control group (n=70)	Prediabetic group (n=70)	'T' Value	P value
Fasting serum glucose (mg/dl)	82.9±7.3	110.4±7.9	19.80	<0.001
Fasting serum insulin (≥12μU/mL)	15.6±3.2	21.5±11.7	3.50	<0.001
Fasting Serum insulin (<12μU/mL)	5.3±2.7	7.5±2.9	4.40	<0.001
HOMA index ≥2.6	3.4±0.75	5.7±3.2	3.56	<0.001
HOMA index <2.6	1.2±0.6	1.5±0.6	4.017	<0.001

Table 6. Association between impaired fasting glucose, insulin resistance and cardiovascular metabolic factors

	FG	BMI	WC	IDRS	FI	IR	TC	LDL	HDL	TG	SBP	DBP	PULSE
FG	1*	.30*	.31*	.38*	.15	.62*	.49*	.44*	-1.31*	.42*	.38*	.31*	.9*
BMI	.3*	1*	.72*	.47*	.05	.36*	.38*	.37*	.16	.29*	.29*	.33*	.02
WC	.31*	.72*	1*	.38*	.05	.42*	.48*	.4*	-.32*	.37*	.32*	.41*	.12
IDRS	.38*	.47*	.54*	1*	.1	.45	.55	.5*	-.36	.48*	.35*	.31*	.16
FI	.15	.05*	.05	.10	1*	.01	.17	.14	-.15	.11	.04	.08*	-.05
IR	.62*	.36*	.42*	.45*	.01	1*	.42	-.30*	-.26	.35*	.27*	.26*	.18
TC	.49*	.38*	.48*	.55*	.17	.42*	1	.84*	.57	.84*	.75*	.50*	.08
LDL	.44*	.37*	.40*	.50*	.14	-.30*	.84	1*	-.52	.71*	.55*	.48*	.03
HDL	-1.31*	-.16	-.32*	-.36*	-.15	-.26*	-.57	-.52*	1	-.55*	-.30*	.33*	-.15
TG	.42*	.29*	.37*	.48*	.11	.35*	.84	.71*	-.55	-.30*	.40*	.43*	.07
SBP	.38*	.29*	.32*	.35*	.04	.27*	.75	.55*	-.30	.49*	1*	.80*	.23**
DBP	.31*	.33*	.41*	.31*	.08	.26*	.50	.48*	-.33	.43*	.80*	1*	.24**
PULSE	.9*	.02	.12	.16	-.05	.18	.08	.03*	-.15	.07	.23**	.24**	1

* Correlation significant at the 0.01 level

**Correlation significant at the 0.05 level

4. DISCUSSION

This study depicted that multiple peril factors are related to T2DM, but not to the pre-diabetes group, including age, female gender and HTN. Generalization to all population could not be due to territorial characteristics. In addition, it does not assess the healthcare services offered in our city. The size of our sample and the cross section type of the study should be of cogitation [14]. Should an effort be made to detect pre-diabetes, and if so, when and how? As desirable as general health screening may be for prevention of chronic disease, there is little inspiration for a universal national program because of costs. However, because most people visit physicians intermittently, opportunities exist to test for pre-diabetes [15]. Several clinical attributes increase the likelihood of a positive finding: advancing age, obesity, another attribute of the metabolic syndrome, family history of diabetes or cardiovascular disease (CVD), signs of atherosclerotic disease. Although higher insulin resistance and lower insulin secretion are known to be the key pathogenic factors in type II diabetes, only a few prospective studies have reported whether or not these are prospectively similar with an increased risk of prediabetes. Clement et al. [16] wrote in their article entitled reduce glucose tolerance and reduce fasting glycaemia that prediabetes (dysglycaemia) is primarily a peril factor for the development of type-2 diabetes. Global status report on non-communicable diseases [17] reported that development of obesity particularly abdominal obesity, promotes insulin resistance and cluster of risk factors for cardiovascular disease including hypertension, atherogenic dyslipidemia inflammation, and altered homeostasis, abdominal obesity and type II diabetes often coexist and patients with type II diabetes are well known to be at elevated risk of first or repeat cardiovascular events compared with their non-diabetic counterparts Association of hypertension and obesity with hyper insulinemia and glucose intolerance in Dutch population. They elucidated the role of hypertension as part of insulin resistance syndrome [18]. They reported that men with diabetes and IGT had significantly high systolic blood pressure and higher prevalence of hypertension than men with normal glucose tolerance test they concluded that hypertension independently associated with insulin resistance. Whether pre-diabetes create atherosclerosis and its obstacle is uncertain. Previous cross-sectional studies have described that multiple possible factors are related to pre-diabetes, Such as

increased age, overweight, obesity, blood pressure, and dyslipidemia [19]. More importantly, reduce glucose sufferance was found to be an independent risk factor for cardiovascular disease, the hazard ratio of death was 2.22 (95% CI = 1.08–4.58), and arterial stiffness and pathological changes in the arterial intima appear in the stage of IGT [20]. The participants in our study with pre-diabetes had higher BMI; more recurrent HTN, higher triglyceride, frequent renal failure and DN than those without pre-diabetes but lower than party with T2DM. logistic regression analysis showed no significant association of any of the covariables with normal glycemic subjects in forepart of the pre-diabetic reference group, whereas the odds of being in the diabetic group gets multiplied by 7.56 for each unitary expansion in the male gender. Also, individuals with hypertension had higher odds of being in the DM group than in the pre-diabetic. Age of subjects had lower odds of being in the DM group than in the pre-diabetic which was consistent with prior studies [21].

Nonetheless, many persons with pre-diabetes have metabolic syndrome, which undoubtedly is a risk factor for macrovascular disease. Moreover, prediabetes increases with age, and aging itself is accompanied by increased risk. Therefore, it is reasonable to intensively intervene on all CVD risk factors in patients with prediabetes. First-line management is lifestyle intervention: Weight reduction in obese subjects, reduced intakes of dietary saturated and trans-fatty acids, cholesterol, and sodium, and increased physical activity. Changes in the BP precedes abnormal glucose tolerance mean values (110.4 ± 7.9 mg/dl) of FSG in prediabetic subjects was significantly higher ($p < 0.001$) as collate to the control group (82.9 ± 7.3). According to Alexander et al. [22], the pathogenesis of diabetes is convoluted by several metabolisms – related problems. In particular, deterioration in insulin secretion and aggravation of insulin resistance are known to be essential in the primary pathogenesis of type 2 diabetes. Subjects with prediabetes, 64% of men and 42% women had metabolic syndrome [23], and for normal glucose tolerance (NGT) groups, only 15% of men and 10% of women had metabolic syndrome. Deterioration in IGI (insulinogenic index, which means the insulin secretion ability index) and aggravation of HOMA–IR index is both critical in the primary pathogenesis of diabetes in those with metabolic syndrome. However, IGI deterioration may be the only

important factor in the primary pathogenesis of type 2 diabetes in the absence of metabolic syndrome. The only way to prevent (or delay) microvascular disease in patients with pre-diabetes is to prevent (or delay) the development of diabetes. Unfortunately, there is no proven way to prevent the decline in beta cell function in persons destined to have diabetes. Therefore, priority must be given to reducing insulin resistance. This is best attained by lifestyle intervention—weight reduction and increased physical activity. The efficacy of this approach suitably, all persons with pre-diabetes should be encouraged to engage in a lifestyle intervention program (If it is required to be, professional assistance is useful. It was found that metformin therapy also could delay conversion of pre-diabetes to diabetes in about 40% of subjects. This has led to a recommendation on the part of some diabetologists for the use of metformin in persons with IFG plus IGT and other metabolic syndrome risk component [24]. Whether this approach will materially retard the development of microvascular disease would require a major clinical trial that is unlikely in the near future.

Results of our inspection must be interpreted in light of some limitations such as the cross-sectional design, which does not let to establish any causal relation with respect to prediabetic state and only provides mere associations. Moreover, the classification of glycemic state was based on HbA1c, instead of its combination with a glucose tolerance test. Then, it is expected that the lack of glucose tolerance test data leads to a suboptimal estimation of glycemic state because normoglycemic group may include some individuals with impaired glucose tolerance that should have been included in pre diabetic group. Considering the goal population, a larger cohort would have probably provided a greater power of the statistical analyses.

5. CONCLUSION

This study found a good positive correlation was found between prediabetic state and metabolic syndrome, positive association between FSG, BMI, abdominal obesity and dyslipidemia with development of insulin resistance signified that persistent hyperglycemia and dyslipidemia might lead to a development of insulin resistance. Independent significant positive correlation was found between FSG, BMI, abdominal obesity serum total cholesterol, and serum triglyceride, LDL, IR, and BP. This study has important implications for identification of subjects at higher

risk for future type 2 diabetes and suggested that mass screening of prediabetic subjects and aggressive risk modification and close follow-up should be considered for prediabetic subjects with metabolic syndrome. Appropriate intervention in the form of weight reduction, changes in dietary habits and increased physical activity could greatly help to prevent, or at least delay the onset of diabetes and thus reduce the burden due to noncommunicable diseases in India.

CONSENT

At the dawn of the study after describes all steps of the study for patients a written consent was obtained from all participants, and they were assured that their information will be kept totally secret. All of them were free to leave the study whenever they want. No patients had any severe infection nor were on any routine medication.

ETHICAL APPROVAL

The study protocol and the plan were accepted by the Ethics Committee of Barkatullah University, Bhopal, and M.P. and also it is submitted in center ethics committee on human research (CECHR) and local ethics committee.

COMPETING INTERESTS

Author has declared that no competing interests exist.

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