ASIAN JOURNAL OF PHARMACEUTICAL AND CLINICAL RESEARCH

INNOVARE ACADEMIC SCIENCES
Knowledge to Innovation

Vol 15, Issue 9, 2022

Online - 2455-3891
Print - 0974-2441

Research Article

# STUDY OF METABOLIC SYNDROME IN INDIAN POPULATION WITH COMPARISON OF TWO DEFINITIONS

**NEHA RAJWAL[1], JASJOT SINGH[2], NURAKANT NEUPANE[3]***

**[1]Department of Laboratory Medicine, Shri Mata Vaishno Devi Narayana Superspeciality Hospital, Katra, Jammu and Kashmir, India. [2]American Society for Clinical Pathology, USA. [3]Medical Genetics Unit, National Academy of Medical Sciences, Bir Hospital, Kathmandu, Nepal. Email: nurakantn@gmail.com**

***Received: 29 March 2022, Revised and Accepted: 21 June 2022***

**ABSTRACT**

**Objectives:** Metabolic syndrome (MetS) is described as the collection of risk factors for cardiovascular disease such as hypertension, hyperglycemia/insulin resistance, abdominal obesity, and dyslipidemia. In developed countries, MetS is highly prevalent among adults and is an emerging health problem in developing countries. In this study, we used the International Diabetes Federation (IDF) and National Cholesterol Education Program-Adult Treatment Panel-III (NCEP-ATP III) to define the Mets. The aim of this research was to evaluate the prevalence of MetS, its components, and its major risk factors among adults ≥20 in Jammu and Kashmir according to IDF and the NCEP ATP III criteria.

**Methods:** The project was conducted in the Department of Biochemistry, Laboratory Medicine, Shri Mata Vaishno Devi Narayana Superspeciality Hospital, Katra, and Jammu and Kashmir in 100 subjects between the age groups of 20 and 80 years old attending the OPD from 2 January to 30 April 2017.

**Results:** MetS was diagnosed in 57% and 55%, gender-wise distribution came out to be 45.6% and 52.7% in men, and in women, we found 54.3% and 47.3%, according to IDF and the NCEP ATP III definition, respectively.

**Conclusion:** In our study, IDF criteria were better for the early diagnosis of MetS. On the basis of gender prevalence in all parameters in IDF, females are at risk, and in NCEP ATP III, males are more at risk. According to the IDF, central obesity is the only the risk for women.

**Keywords:** Metabolic syndrome, International diabetes federation, National Cholesterol Education Program-Adult Treatment Panel-III, Obesity, Cardiovascular diseases.

 DOI: http://dx.doi.org/10.22159/ajpcr.2022v15i9.44760. Journal homepage: https://innovareacademics.in/journals/index.php/ajpcr

## INTRODUCTION

The metabolic syndrome (MetS) is described as a cluster of several risk factors for cardiovascular diseases (CVD), such as high blood pressure (BP) (hypertension), high blood glucose (hyperglycemia), insulin resistance, abdominal obesity, and dyslipidemia [1]. Syndrome X, Insulin Resistance Syndrome, Dysmetabolic Syndrome X, Reaven Syndrome, and Metabolic Cardiovascular Syndrome are other names for MetS. Obesity, insulin resistance, dyslipidemia, and hypertension are the common features of it [2,3]. The importance of MetS is emphasized for several reasons. Patients with MetS have a high risk of developing type 2 diabetes (T2D) and atherosclerotic CVD. Second, by comparing the components of MetS, we may be able to better understand the mechanism that connects them and the increases the risk of CVD [4]. People with MetS are 3 times as likely to have a heart attack or stroke and have a twofold risk of developing CVD over the next 5–10 years [5]. The prevalence of MetS and its components is influenced by differences in genetic background, diet, level of physical activity, age, and sex structure [6]. Cardiovascular risk factors such as high BP, deficiency in glucose tolerance, hypertriglyceridemia, and low levels of high-density lipoprotein (HDL) are risk factors of cardiovascular disease and are associated with MetS [7].

Reports of clustering of metabolic risk factors are not new and date back to the early 1920s [8]. In 1920, Kylin, a Swedish physician, demonstrated the relationship between hypertension, hyperglycemia, and gout [9]. Later in 1947, Vague described that visceral obesity was commonly associated with the metabolic abnormalities found in CVD and T2DM [10]. Reaven described "a cluster of risk factors for diabetes and cardiovascular disease" and named it "Syndrome X" a in his Banting lecture in 1988 [7]. Number of researchers have attempt to developed diagnostic criteria for the diagnosis of the MetS [11]. The WHO proposed the label "MetS" for the syndrome in1998 [12]. The National Cholesterol Program Adult Treatment Panel (NCEP/ATP) defines one of the most widely used criteria for MetS. In April 2005, the International Diabetes Federation (IDF) proposed a new definition of the MetS [13]. Although it includes the same general criteria as the other definitions, it requires obesity but not necessarily insulin resistance [4]. In the pathophysiology of obesity, visceral obesity is now recognized as an important factor in the IDF definition rather than insulin resistance [14].

The worldwide prevalence of MetS ranges from <10% to as much as 84%. It depends on the region, urban or rural environment, the composition (sex, age, race, and ethnicity) of the population studied and the definition of the syndrome used [15,16]. High body mass index, higher socioeconomic status, and sedentary lifestyle are significantly associated with MetS. Cameron *et al*. concluded that the differences in genetic background, diet, levels of physical activity, smoking, family history of diabetes, and education all influence the prevalence of the MetS and its components [6]. The observance of MetS prevalence, a study conducted by the National Health and Nutrition Examination Survey, found 5% among the subjects of normal weight, 22% among the overweight, and 60% among the obese [17]. It, further, increases with age (10% in individuals aged 60–69) [18]. The prevalence of MetS (based on NCEP-ATP III criteria, 2001) varied from 8% to 43% in men and from 7% to 56% in women around the world [6,18]. The risk increases with the number of MetS components present. MetS can vary due to multiple factors predisposing to metabolic susceptibility, such as genetic defects in insulin signaling pathways, physical

inactivity, and certain ethnic groups [19]. More recent investigation shows that visceral adiposity is a significant independent predictor of insulin sensitivity, elevated BP, and dyslipidemia seen in MetS [20-34]. Furthermore, reduction in visceral fat by weight loss or surgical removal is associated with increases in insulin sensitivity and HDL cholesterol and decreases in TG and BP [22-26,31-38]. To make an effective plan and execute preventive strategies for MetS, we need comprehensive information about the prevalence of MetS. In this paper, we study MetS in Jammu and Kashmir by comparing two international definitions, that is, National Cholesterol Education Program, Adult Treatment Panel III (NCEP ATP III), and IDF.

## METHODS

The study was conducted in the Department of Biochemistry, Laboratory Medicine, Shri Mata Vaishno Devi Narayana Superspeciality Hospital, Katra, and Jammu and Kashmir from January 2 to April 30, 2017.

Physical examination of patients was measured, which included patients' weight, height, waist circumference (WC), and BP. BP was measured using a calibrated Sphygmomanometer and WC was measured with a measuring tape, placed at the level of the umbilicus. Laboratory analysis was done for biochemical parameters such as HDL, TGL, and FBS and Clinical diagnosis based on the diagnosis criteria listed in Table 1. Two milliliters of venous sample were drawn from each patient; 12 h overnight fasting, from antecubital vein using vacutainer. Enzymatic analysis was performed on fasting lipid profile (Triglycerides and HDL) and blood glucose.

A total of 100 subjects were between the age groups of 20 and 80 years and were attending the outpatient department (OPD). The subjects were selected by a simple random sampling method for the duration of 4 months, from January 2 to April 30, 2017. On the consent form, information about patient's age, sex, life style, and family history of diabetes and hypertension were recorded. All the patients who came to the OPD for general health check-up were included, with the exception of pregnant women, hypothyroidism patients, patients having Cushing's syndrome, and patients on steroid medication.

### Statistical analysis

The study's findings were presented as number, percentage and Chi-square test as per necessities. The values presented in percentage form and ranges given of all parameters included for study. All data were calculated and graphically presented using Microsoft Excel 2010 capabilities.

## RESULTS AND DISCUSSION

The present study was conducted on 100 patients who attended the OPD for their health check-ups in Shri Mata Vaishno Devi Narayana Superspeciality Hospital, Katra, and Jammu and Kashmir to assess the MetS on the basis of two definitions given by NCEP-ATP III and IDF. In our study, the patients were between 20 and 80 years of age, and it was found the prevalence of Mets came out to be 64% out of 100 patients.

The percentage of MetS is shown in Table 2, which indicates that out of 100 patients, (64%) have MetS (diagnosed using the NCEP ATP III and IDF criteria). However, this trend varies among different countries. Studies in France reported a Mets prevalence of 21.5%, 33.5% in Turkey, 34.1% among American adults, and 54.8% in Mexico [39-42]. Several study groups have developed diagnostic criteria for MetS; the NCEP ATP III and IDF stand out due to their wide use [43].

Table 3 indicates the comparison between the two definitions, that is, IDF and NCEP ATP III, where IDF was seen in the maximum percentage of patients (57%), whereas ATP III had (55%). In our study, IDF was 57% as compared to NCEP ATP III's 55%, which suggests that IDF is a better criteria for diagnosing MetS. This result trend is similar to American, Brazilian, and Iranian people, where 39%, 35.1%, and 43.5% of the IDF and 35%, 29.5%, and 38.3% of the NCEP [44-46].

**Table 1: Diagnostic criteria proposed for the clinical diagnosis of the mets**

| Clinical measures | ATPIII (2001) | IDF (2005) |
|---|---|---|
| Body weight | WC≥102 cm in men or≥88 cm in women | *Increased WC (ethnicity specific values) |
| Lipids | TGs≥150mg/dL HDL-C<40 mg/dL in men or<50 mg/dL in women | TGs≥150 mg/dL or on TGs Rx. HDL-C<40 mg/dL in men or<50 mg/dL in women or on HDL-C Rx |
| Blood pressure | ≥130/85 mm Hg | ≥130 mm Hg systolic or≥85 mm Hg diastolic or on hypertension Rx |
| Glucose | >100 mg/dL (includes diabetes) | ≥100 mg/dL (includes diabetes)[a] |

*Country/ethnic group waist circumference: Europids: Male ≥94 cm. In the USA, the ATP III values (102 cm male; 88 cm female) are likely to continue to be used for clinical purposes Female≥80 cm South Asians: Male ≥90 cm based on a Chinese, Malay and Asian-Indian population Females ≥80cm

**Table 2: Metabolic syndrome**

| Metabolic Syndrome | Percent |
|---|---|
| Present | 64.00 |
| Absent | 36.00 |
| Total | 100.00 |

**Table 3: Comparison between IDF and NCEP ATP III**

| Definition | Patients | |
|---|---|---|
| | Present (%) | Absent (%) |
| IDF | 57 | 43 |
| NCEP ATP III | 55 | 45 |

IDF: International Diabetes Federation, NCEP ATP III: National Cholesterol Education Program-Adult Treatment Panel-III

**Table 4: Overall and gender-specific percentage prevalence of the metabolic syndrome**

| Metabolic syndrome definition | Overall (%) | Males (%) | Females (%) |
|---|---|---|---|
| IDF | 57 | 45.60 | 54.30 |
| NCEP ATP III | 55 | 52.70 | 47.30 |

IDF: International Diabetes Federation, NCEP ATP III: National Cholesterol Education Program-Adult Treatment Panel-III

Overall and gender-specific percentage prevalence of the MetS using two definitions is illustrated by Table 4. Out of 57% of IDF patients, the maximum percentage was seen in females, at 54.3% (31) and at 45.6% (26) in males. While in the case of NCEP ATP III, the maximum percentages seen in males were 52.7% (29) and 47.3% (26) in females. Gender-specific percentages of males and females have an equal percentage of MetS. This type of different result is also found in the UAE and in Bangladesh. The prevalence of MetS in this study was higher among men than among women, as found in the studies of Ibrahim *et al*. in the UAE [47] and in Bangladesh studied by Gupta *et al*., which found a higher prevalence in females according to both the IDF and NCEP [48].

In NCEP ATP III, Table 5 shows the gender-based prevalence of individual parameters in NCEP ATP III, with FPG being highest in males (89.7) and lowest in females (88.5) out of 29 males and 26 females. In the case of TGL, the maximum percentage was observed in females (76.9%). In HDL, 69.2% of females and 69% of males were found. In WC, there was a huge difference between females (92.3%) and males

**Table 5: Gender-wise prevalence of individual parameters in NCEP ATP III**

| Parameters | NCEP ATP III | |
|---|---|---|
| | Male in percentage | Female in percentage |
| FPG | 89.7 | 88.5 |
| TGL | 75.9 | 76.9 |
| HDL | 69.0 | 69.2 |
| WC | 48.3 | 92.3 |
| BP | 69.0 | 61.5 |

NCEP ATP III: National Cholesterol Education Programme-Adult Treatment Panel-III, HDL: High-density lipoprotein, BP: Blood pressure, WC: Waist circumference

(48.3%), and in the case of BP, the maximum percentages were seen in males (69%), which are 7.5% higher than females.

Table 6 shows the gender distribution of individual parameters in the IDF; the WC of all the males and females was 100%, with 26 males and 31 females having MetS. While in the case of FPG, maximum percentages were seen in males 88.5% and in females 87.1%, in the case of TGL, maximum percentages were seen in females 64.5% and in males 61.5%, in the case of HDL, maximum percentages were seen in males 92.3% and in females 71%, and in the case of BP, maximum percentages were seen in males 76.9% and in females 48.4%.

Table 7 describes the demographic profile that includes sex, age, and life style of 100 patients (study population) and 64 patients (MetS present). The age distribution of 100 patients shows that the age group of 40–60 years had the highest percentage of patients (66%), followed by 60–80 years (22%) and 20–40 years (12%). In subjects having MetS, the maximum percentage of patients was in the age groups of 40–60 years (34%) followed by the age group of 60–80 years (10.9%) and 20–40 years (4.6%). In our study, there was considerable change demographically in Mets patients. The maximum percentage was seen in the age group of 40–60 years (34.4%), but it was eventually reduced to 10.9% in patients aged group 60–80 years, and in the 20–40 year age group of patients (4.6%). There is an increase in the prevalence of MetS from 20 years old through the sixth and seventh decades of life for males and females, as noticed by Park *et al*. [17]. Mets were shown to be more prevalent in the 55–64 age bracket in Iranian studies, and the same tendency was discovered in Turkey [45,49].

Similarly, for sex, it was seen that out of a total of 100 patients, male and female percentage were 53.0% and 47.0%, respectively. Males had a prevalence of 32.0% in participants with MetS, followed by females at 32.0%. The prevalence of males and females is equal in 100 patients.

Based on the lifestyle of patients, we observed that out of a total of 100 patients, moderate and sedentary patients' percentage were 51% and 49%, respectively. Equal numbers of moderate and sedentary were seen, that is, 32% and 32% respectively, in patients having MetS. The percentage of moderate and sedentary lifestyle was also the same, with no significant difference. MetS had no association with age, sex, or lifestyle. This is may be due to the sample size, and we recommend study in a large population.

The clinical profile that includes WC and BP of both 100 subjects (study population) and 64 patients (MetS present) is represented by Table 8. Increased WC levels (>90 cm) among 100 patients were found in 69% of patients. A further account of subjects having WC within the range of 80–90 and 70–80 cm was 26% and 5%, respectively. About 78.2% of MetS patients had elevated levels of WC (>90 cm). It decreased significantly from 20.3% (80–90) to 1.6% (70–80) of the patients. Recent studies have reported that a strong correlation also exists between WC and insulin resistance [50,51]. The metabolic characteristics of fat tissue present in omental and paraintestinal areas promote insulin resistance

**Table 6: Gender-wise prevalence of individual parameters in IDF**

| Parameters | IDF | |
|---|---|---|
| | Male in percentage | Female in percentage |
| FPG | 88.5 | 87.1 |
| TGL | 61.5 | 64.5 |
| HDL | 92.3 | 71.0 |
| WC | 100 | 100 |
| BP | 76.9 | 48.4 |

IDF: International Diabetes Federation, HDL: High-density lipoprotein, BP: Blood pressure, WC: Waist circumference

**Table 7: Demographic profile metabolic syndrome**

| Demographic | In total patients | | Metabolic syndrome | |
|---|---|---|---|---|
| | Frequency | Percentage | Frequency | Percentage |
| Age Group | | | | |
| 20–40 | 12 | 12 | 3 | 4.6 |
| 40–60 | 66 | 66 | 22 | 34.4 |
| 60–80 | 22 | 22 | 7 | 10.9 |
| Sex | | | | |
| Male | 53 | 53 | 32 | 50 |
| Female | 47 | 47 | 32 | 50 |
| Life style | | | | |
| Moderate | 51 | 51 | 32 | 50 |
| Sedentary | 49 | 49 | 32 | 50 |

**Table 8: Clinical profile of metabolic syndrome patients**

| Clinical profile | In total patients | | Metabolic syndrome | |
|---|---|---|---|---|
| | Frequency | Percentage | Frequency | Percentage |
| Waist Circumference (cm) | | | | |
| 70–80 | 5 | 5 | 1 | 1.6 |
| 80–90 | 26 | 26 | 13 | 20.3 |
| 90–100 | 32 | 32 | 22 | 34.4 |
| >100 | 37 | 37 | 28 | 43.8 |
| BP (mmHg) | | | | |
| 100–110 or 60–70 | 6 | 6 | 1 | 1.6 |
| 110–120 or 70–80 | 23 | 23 | 13 | 20.3 |
| 120–130 or 80–90 | 34 | 34 | 20 | 31.3 |
| 130–140 or 90–100 | 15 | 15 | 11 | 17.2 |
| ≥140 or ≥100 | 22 | 22 | 19 | 29.7 |

and hyperinsulinemia [52]. Obesity, along with insulin resistance and hyperinsulinemia, has been link to an increased risk of cardiovascular disease and stroke [53].

Similarly, out of 100 patients, the majority (59%) were in the pre-hypertensive stage, that is, 34% and 15% (120–130 or/80–90 mmHg and 130–140 or/90–100 mmHg). About 22% had stage I hypertension (≥140 or ≥100 mmHg), while 29% were normotensive (100–110 or 60–70 mmHg and 110–120 or 70–80 mmHg). Similarly, 48.5% of MetS patients had pre-hypertensive BP, with 29.7% having stage I hypertension, and 21.9% have post-hypertensive BP. The most commonly researched condition in connection to insulin resistance is hypertension (BP), which is a critical component of the MetS [54,55]. However, the relationship between the MetS and increased BP is controversial as not all people who meet the definition of the MetS have an elevated BP. Due to the association between BP and body weight, obesity could be a major confounder in the

association between elevated BP and insulin resistance [56]. There are three basic processes that link high BP to insulin resistance: (1) high BP causes insulin resistance; (2) insulin resistance causes higher BP; and (3) both (elevated BP and insulin resistance).

The status of the biochemical profile of the patients is shown in Table 9 which comprise fasting plasma glucose, HDL, and triglycerides in both 100 patients (study population) and 64 patients (MetS present). Fasting plasma glucose distribution among 100 patients shows that the highest percentage was seen in the range of 90–110 mg/dl (53%), followed by the levels between ≥130 mg/dl (25%), 110–130 mg/dl (19%), and 70–90 mg/dl (3%), respectively. While among the 64% of MetS patients, increased levels of FPG (90–110) were seen in 42.2% of patients, and the rest of the patients had levels between ≥130 mg/dl (29.7%), 110–130 mg/dl (26.6%), and 70–90 mg/dl (1.60%), respectively. Insulin resistance, especially among those with diabetes and the obese, is believed to result from multiple mechanisms, including defective insulin signaling and abnormalities in glucose transport [57]. Dysglycemia, or an elevated blood glucose range in non-diabetic, is an imperative component of the MetS, predominantly in IGT [7]. It is believed that hyperglycemia predates overt T2D by many years [58]. Insulin resistance thus thought to be vital to the progression from "normoglycemia" to "dysglycemia" (elevated glucose in the non-diabetic range) and from IGT to overt T2D. Dysglycemia in the non-diabetic range has been linked to the progression of atherosclerosis despite the absence of overt diabetes [59,60]. In the San Antonio Heart Study, hyperinsulinemia predicted the development of T2D, dyslipidemia, and hypertension over an 8 year follow-up period [61].

Our study showed the percentage of patients with MetS having elevated FPG was 56.3%. Also, 1.60% of patients had FPG within the range of 70–90 mg/dl, followed by 42.2% within the range of 90–110 mg/dl. The association of levels was validated as significant with MetS ($p<0.05$). The prevalence of FPG is higher in males (89.7%) than females (88.5%) in NCEP ATP III, while in the case of IDF, males (88.5%) are more than females (87.1%).

Similarly, 67 % had low HDL levels (<50 mg/dl), 21% had 50–60 mg/dl, and 12% had levels >60 mg/dl. Low levels of HDL (<50 mg/dl) were found in 81.2% of patients, with 34.3% and 7.8% of subjects having levels between 50–60 mg/dl and >60 mg/dl, respectively. Primarily, lipid abnormalities comprise hypertriglyceridemia and low HDL-cholesterol concentration. In the metabolism of free fatty acids, insulin plays a crucial role by suppressing lipolysis in adipocytes. As a result, impaired insulin signaling increases lipolysis, which leads to increased FFA levels [62,63]. Significant amounts of plasma FFA lead to an increased flux of free fatty acids to the liver, which results in an accumulation of hepatic triglyceride VLDL and cholesterol ester synthesis and secretion [63-65].

**Table 9: Biochemical profile of metabolic syndrome patients**

| Biochemical profile | In total patients | | Metabolic syndrome | |
|---|---|---|---|---|
| | Frequency | Percentage | Frequency | Percentage |
| Fasting plasma glucose (mg/dl) | | | | |
| 70–90 | 3 | 3 | 1 | 1.60 |
| 90–110 | 53 | 53 | 27 | 42.2 |
| 110–130 | 19 | 19 | 17 | 26.6 |
| ≥130 | 25 | 25 | 19 | 29.7 |
| HDL (mg/dl) | | | | |
| 20–30 | 6 | 6 | 4 | 6.3 |
| 30–40 | 31 | 31 | 26 | 40.6 |
| 40–50 | 30 | 30 | 22 | 34.3 |
| 50–60 | 21 | 21 | 7 | 10.9 |
| ≥60 | 12 | 12 | 5 | 7.8 |
| Triglycerides (mg/dl) | | | | |
| <70 | 8 | 8 | 2 | 3.12 |
| 70–100 | 19 | 19 | 4 | 6.25 |
| 100–130 | 12 | 12 | 5 | 7.8 |
| 130–160 | 15 | 15 | 13 | 20.3 |
| ≥160 | 46 | 46 | 40 | 62.5 |

Another component that is TGL was elevated in females (76.9%) than in males (75.9%) in NCEP ATP III, while in IDF, it was also (64.5%) more in females than in males (61.5%). The overall percentage of patients having MetS with elevated TGL was 82.8%. The level was found to be significant with MetS ($p<0.05$).

Out of 100 patients, increased TGL (>130 mg/dl) was seen in 61%, which was followed by 19%, 12%, and 8% within the levels of 70–100, 100–130, and <70 mg/dl, respectively, while among MetS patients, increased TGL (>130 mg/dl) was seen in 82.8%. In addition, 7.8% have (100–130), 6.3 % have (70–100), and 3.12 % have (<70 mg/dl). Abundant plasma free fatty acids, caused by lipid anomalies, are supposed to reduce glucose utilization by skeletal muscle, decrease insulin removal, and promote gluconeogenesis by the liver. According to Sniderman *et al*., reduced glucose utilization, decreased insulin removal, and increased gluconeogenesis are important in the development of insulin resistance and diabetes mellitus [64].

Low HDL-C levels are thought to be a strong predictor of myocardial infract and stroke, both of which are associated with premature and severe CAD [66]. Our study found out that the gender specific low HDL-C levels prevalence was more in females (62.2%) than in males (69%) in NCEP ATP III, where as in IDF, there was a greater prevalence of males (92.3%) than females (71%). The maximum low HDL-C levels were seen at 30–40 mg/dl (40.6%). The levels were found significant with MetS (<0.05).

Table 10 affirms that the association of MetS, where no significant association was found, with age, sex, and lifestyle of the patients.

Table 11 epitomizes the highly significant association of MetS with clinical parameters that include WC and BP ($p<0.05$). The proportion of subjects with WC abnormal was 69%. The prevalence of WC was elevated in females (92.3%) and males (48.3%) in NCEP ATP III, while in IDF, WC was high in all MetS patients (100%). The association was found to be significant with the Mets ($p<0.05$).

BP changes in our study also had a significant association with MetS ($p<0.05$), respectively. The maximum percentage of patients was seen in the pre-hypertensive stage, followed by 29.7% in stage 1 hypertension. Males (69) have a higher prevalence of BP than females (61.5%) in NCEP ATP III and in IDF prevalence also, males (76.9%) are higher than females (48.4%). Haverinen *et al*. found the same study in Finland [67].

**Table 10: Relation between metabolic syndrome and demographic risk factors**

| Demographic | Metabolic syndrome (frequency) | | $X^2$ value | p-value |
|---|---|---|---|---|
| | Present | Absent | | |
| Sex | | | | |
| Male | 32 | 21 | 0.6423 | 0.4228 |
| Female | 32 | 15 | | |
| Age (years) | | | | |
| 20–40 | 6 | 6 | 0.0711 | 0.789685 |
| 40–60 | 45 | 21 | | |
| 60–80 | 13 | 9 | | |
| Life style | | | | |
| Moderate | 32 | 19 | 1.7519 | 0.4164 |
| Sedentary | 32 | 17 | | |

Significant at *p<0.01, **p<0.05

**Table 11: Association between metabolic syndrome and clinical risk factors**

| Clinical | Metabolic syndrome (frequency) | | $X^2$ value | p-value |
|---|---|---|---|---|
| | Present | Absent | | |
| Waist circumference (cm) | | | | |
| 70-80 | 1 | 4 | 8.9158 | 0.030432** |
| 80-90 | 13 | 13 | | |
| 90-100 | 22 | 10 | | |
| >100 | 28 | 9 | | |
| Blood pressure (mmHg) | | | | |
| 100–110 Or 60–70 | 1 | 5 | 12.1309 | 0.0164** |
| 110–120 Or 70–80 | 13 | 10 | | |
| 120–130 Or 80–90 | 20 | 14 | | |
| 130–140 Or 90–100 | 11 | 4 | | |
| ≥140 Or ≥100 | 19 | 3 | | |

Significant at **p<0.05

**Table 12: Connotation between metabolic syndrome and biochemical risk factors**

| Biochemical profile | Metabolic syndrome (frequency) | | $X^2$ value | p-value |
|---|---|---|---|---|
| | Present | Absent | | |
| Fasting plasma glucose (mg/dl) | | | | |
| 70–90 | 1 | 2 | 12.06 | 0.007** |
| 90–110 | 27 | 26 | | |
| 110–130 | 17 | 2 | | |
| ≥130 | 19 | 6 | | |
| HDL (mg/dl) | | | | |
| 20–30 | 4 | 2 | 17.635 | 0.001454** |
| 30–40 | 26 | 5 | | |
| 40–50 | 22 | 8 | | |
| 50–60 | 7 | 14 | | |
| ≥60 | 5 | 7 | | |
| Triglycerides (mg/dl) | | | | |
| <70 | 2 | 6 | 36.956 | <0.00001** |
| 70–100 | 4 | 15 | | |
| 100–130 | 5 | 7 | | |
| 130–160 | 13 | 2 | | |
| ≥160 | 40 | 6 | | |

Significant at **p<0.05

An association between MetSs with biochemical components is presented in Table 12. Results indicated that the association of MetS with individual biochemical components (fasting plasma glucose, HDL, and triglyceride) was significant (p<0.05).

## CONCLUSION

The overall prevalence of MetS in the Jammu population was estimated to be 57% by IDF criteria and 55% by NCEP ATP-III criteria. Males and females were found to be more affected by MetS according to both IDF and ATPIII criteria (MetS IDF criteria: Males 45.6%, women 54.3%, and Met S NCEP ATP III criteria: Males – 52.7%, women 47.3%). According to both IDF and NCEP ATP III criteria, the prevalence rate increases with >60 years of age in both males and females of the study population. In IDF, females had high levels of FPG, TGL, and WC except BP and HDL levels as compared to males. In NCEP ATP III, males had high levels of FPG, TGL, HDL, and BP except WC as compared to females. According to the criteria of the two definitions, males and females are equally at the risk of causing MetS. There was no relevant association with age, gender, and lifestyle, and a highly relevant association with the biochemical and clinical profile of patients.

## AUTHORS CONTRIBUTIONS

The manuscript writing and data collection had accomplished by Rajwal research reviewed and edited by Singh and manuscript editing, reviewed, finalized, and submitted for publication by Neupane.

## CONFLICTS OF INTEREST

The authors affirm no conflicts of interest.

## FUNDING

This study was conduct without funding.

## REFERENCES

1. Miranda PJ, DeFronzo RA, Califf RM, Guyton JR. Metabolic syndrome: Definition, pathophysiology, and mechanisms. Am Heart J 2005;149:33-45. doi: https://doi.org/10.1016/j.ahj.2004.07.013
2. Falkner B, Hassink S, Ross J, Gidding S. Dysmetabolic syndrome: Multiple risk factors for premature adult disease in an adolescent girl. Pediatrics 2002;110:e14-4. doi:10.1542/peds.110.1.e14
3. Hjermann I. The metabolic cardiovascular syndrome: Syndrome X, Reaven's syndrome, insulin resistance syndrome, atherothrombogenic syndrome. J Cardiovasc Pharmacol 1992;20 Suppl 8:S5-10. Available from: https://journals.lww.com/cardiovascularpharm/Fulltext/1992/00208/The_Metabolic_Cardiovascular_Syndrome__Syndrome_X.,2.aspx
4. Huang PL. A comprehensive definition for metabolic syndrome. Dis Model Mech 2009;2:231-7. doi:10.1242/dmm.001180
5. Alberti KG, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Donato A, *et al*. Harmonizing the metabolic syndrome. Circulation 2009;120:1640-5. doi:10.1161/CIRCULATIONAHA.109.192644
6. Cameron AJ, Shaw JE, Zimmet PZ. The metabolic syndrome: Prevalence in worldwide populations. Endocrinol Metab Clin North Am 2004;33:351-75. doi: https://doi.org/10.1016/j.ecl.2004.03.005
7. Reaven GM. Role of insulin resistance in human disease. Diabetes 1988;37:1595-607. doi:10.2337/diab.37.12.1595
8. Sarafidis PA, Nilsson PM. The metabolic syndrome: A glance at its history. J Hypertens 2006;24:621-6.
9. Kylin E. Studien ueber das hypertonie-hyperglyka "mie-Hyperurika" miesyndrom. Zentralbl Inn Med. 1923;44:105-27.
10. Vague J. Sexual differentiation, a factor affecting the forms of obesity. Presse Med 1947;30:339-40.
11. Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. Lancet 2005;365:1415-28. doi:10.1016/S0140-6736(05)66378-7
12. Kaur J. A Comprehensive Review on Metabolic Syndrome. Cardiol Res Pract 2014;2014:943162. doi:10.1155/2014/943162
13. International Diabetes Federation. The IDF Consensus Worldwide Definition of the Metabolic Syndrome. Brussels, Belgium: International Diabetes Federation; 2006. p. 1-24.
14. Reaven GM. The metabolic syndrome: Is this diagnosis necessary? Am J Clin Nutr 2006;83:1237-47. doi:10.1093/ajcn/83.6.1237
15. Desroches S, Lamarche B. The evolving definitions and increasing prevalence of the metabolic syndrome. Appl Physiol Nutr Metab. 2007;32(1):23-32. doi:10.1139/h06-095
16. Kolovou GD, Anagnostopoulou KK, Salpea KD, Mikhailidis DP. The prevalence of metabolic syndrome in various populations. Am J Med Sci. 2007;333(6):362-371. doi:10.1097/MAJ.0b013e318065c3a1
17. Park YW, Zhu S, Palaniappan L, Heshka S, Carnethon MR, Heymsfield SB. The metabolic syndrome: Prevalence and associated risk factor findings in the US population from the Third National Health and Nutrition Examination Survey, 1988-1994. Arch Intern Med 2003;163:427-36. doi:10.1001/archinte.163.4.427
18. Ford ES, Giles WH, Dietz WH. Prevalence of the metabolic syndrome among US adults findings from the third national health and nutrition examination survey. JAMA 2002;287:356-9. doi:10.1001/jama.287.3.356
19. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH,

Franklin BA, *et al*. Diagnosis and management of the metabolic syndrome. Circulation 2005;112:2735-52. doi:10.1161/CIRCULATIONAHA.105.169404
20. Carey DG, Jenkins AB, Campbell LV, Freund J, Chisholm DJ. Abdominal fat and insulin resistance in normal and overweight women: direct measurements reveal a strong relationship in subjects at both low and high risk of NIDDM. Diabetes 1996;45:633-8. doi:10.2337/diab.45.5.633
21. Cnop M, Landchild MJ, Vidal J, Havel PJ, Knowles NG, Carr DR, *et al*. The concurrent accumulation of intra-abdominal and subcutaneous fat explains the association between insulin resistance and plasma leptin concentrations: Distinct metabolic effects of two fat compartments. Diabetes 2002;51:1005-15. doi:10.2337/diabetes.51.4.1005
22. Pascot A, Després JP, Lemieux I, Bergeron J, Nadeau A, Prud'homme D, *et al*. Contribution of visceral obesity to the deterioration of the metabolic risk profile in men with impaired glucose tolerance. Diabetologia 2000;43:1126-35. doi:10.1007/s001250051503
23. Pascot A, Lemieux I, Prud'homme D, Tremblay A, Nadeau A, Couillard C, *et al*. Reduced HDL particle size as an additional feature of the atherogenic dyslipidemia of abdominal obesity. J Lipid Res 2001;42:2007-14. doi:10.1016/S0022-2275(20)31529-7
24. Nicklas BJ, Penninx BW, Ryan AS, Berman DM, Lynch NA, Dennis KE. Visceral adipose tissue cutoffs associated with metabolic risk factors for coronary heart disease in women. Diabetes Care 2003;26:1413-20. doi:10.2337/diacare.26.5.1413
25. Nieves DJ, Cnop M, Retzlaff B, Walden CE, Brunzell JD, Knopp RH, *et al*. The atherogenic lipoprotein profile associated with obesity and insulin resistance is largely attributable to intra-abdominal fat. Diabetes 2003;52(1):172-9. doi:10.2337/diabetes.52.1.172
26. Phillips GB, Jing T, Heymsfield SB. Relationships in men of sex hormones, insulin, adiposity, and risk factors for myocardial infarction. Metab Clin Exp 2003;52:784-90. doi:10.1016/S0026-0495(03)00072-6
27. Katsuki A, Sumida Y, Urakawa H, Gabazza EC, Murashima S, Maruyama N, *et al*. Increased visceral fat and serum levels of triglyceride are associated with insulin resistance in japanese metabolically obese, normal weight subjects with normal glucose tolerance. Diabetes Care 2003;26:2341-4. doi:10.2337/diacare.26.8.2341
28. Wagenknecht LE, Langefeld CD, Scherzinger AL, Norris JM, Haffner SM, Saad MF, *et al*. Insulin sensitivity, insulin secretion, and abdominal fat: The insulin resistance atherosclerosis study (IRAS) family study. Diabetes 2003;52:2490-6. doi:10.2337/diabetes.52.10.2490
29. Fujimoto WY, Abbate SL, Kahn SE, Hokansno JE, Brunzell JD. The visceral adiposity syndrome in Japanese-American men. Obes Res 1994;2:364-71. https://doi.org/10.1002/j.1550-8528.1994.tb00076.x
30. Rattarasarn C, Leelawattana R, Soonthornpun S, Setasuban W, Thamprasit A, Lim A, *et al*. Regional abdominal fat distribution in lean and obese thai Type 2 diabetic women: Relationships with insulin sensitivity and cardiovascular risk factors. Metab Clin Exp 2003;52:1444-7. doi:10.1016/S0026-0495(03)00257-9
31. Kanai H, Tokunaga K, Fujioka S, Yamashita S, Kameda-Takemura K, Matsuzawa Y. Decrease in intra-abdominal visceral fat may reduce blood pressure in obese hypertensive women. Hypertension 1996;27:125-9. doi:10.1161/01.HYP.27.1.125
32. Bacha F, Saad R, Gungor N, Janosky J, Arslanian SA. Obesity, regional fat distribution, and syndrome X in obese black versus white adolescents: Race differential in diabetogenic and atherogenic risk factors. J Clin Endocrinol Metab 2003;88:2534-40. doi:10.1210/jc.2002-021267
33. Carr MC, Hokanson JE, Deeb SS, Purnell JQ, Mitchell ES, Brunzell JD. A hepatic lipase gene promoter polymorphism attenuates the increase in hepatic lipase activity with increasing intra-abdominal fat in women. Arterioscler Thromb Vasc Biol 1999;19:2701-7. doi:10.1161/01.ATV.19.11.2701
34. Ribeiro-Filho FF, Faria AN, Kohlmann NE, Zanella MT, Ferreira SR. Two-hour insulin determination improves the ability of abdominal fat measurement to identify risk for the metabolic syndrome. Diabetes Care 2003;26:1725-30. doi:10.2337/diacare.26.6.1725
35. de Oliveira RM, do Verreschi IT, Lipay MV, Eça LP, Guedes AD, Bianco B. Y chromosome in Turner syndrome: Review of the literature. Sao Paulo Med J 2009;127:373-8. doi:10.1590/s1516-31802009000600010
36. Barzilai N, She L, Liu BQ, Vuguin P, Cohen P, Wang J, *et al*. Surgical removal of visceral fat reverses hepatic insulin resistance. Diabetes 1999;48:94-8. doi:10.2337/diabetes.48.1.94
37. Brochu M, Tchernof A, Turner AN, Ades PA, Poehlman ET. Is there a threshold of visceral fat loss that improves the metabolic profile in obese postmenopausal women? Metab Clin Exp 2003;52:599-604. doi:10.1053/meta.2003.50095
38. Gabriely I, Ma XH, Yang XM, Atzmon G, Rajala MW, Berg AH, *et al*. Removal of visceral fat prevents insulin resistance and glucose intolerance of aging: An adipokine-mediated process? Diabetes 2002;51:2951-8. doi:10.2337/diabetes.51.10.2951
39. Vernay M, Salanave B, de Peretti C, Druet C, Malon A, Deschamps V, *et al*. Metabolic syndrome and socioeconomic status in France: The French nutrition and health survey (ENNS, 2006-2007). Int J Public Health 2013;58:855-64. doi:10.1007/s00038-013-0501-2
40. Gündogan K, Bayram F, Capak M, Tanriverdi F, Karaman A, Ozturk A, *et al*. Prevalence of metabolic syndrome in the mediterranean region of turkey: Evaluation of hypertension, diabetes mellitus, obesity, and dyslipidemia. Metab Syndr Relat Disord 2009;7:427-34. doi:10.1089/met.2008.0068
41. Mozumdar A, Liguori G. Persistent increase of prevalence of metabolic syndrome among U.S. adults: NHANES III to NHANES 1999-2006. Diabetes Care 2010;34:216-9. doi:10.2337/dc10-0879
42. Salas R, del Bibiloni MM, Ramos E, Villarreal JZ, Pons A, Tur JA, *et al*. Metabolic syndrome prevalence among Northern Mexican adult population. PLoS One 2014;9:e105581. https://doi.org/10.1371/journal.pone.0105581
43. Correia F, Poínhos R, Freitas P, Pinhão S, Maia A, Carvalho D, *et al*. Prevalência da sindrome metabólica comparação entre os critérios ATPIII e IDF numa população feminina com obesidade severa prevalence of the metabolic syndrome comparison between ATPIII and IDF criteria in a feminin population with severe obesity. Acta Med Port 2006;19:286-94.
44. do Vale Moreira NC, Hussain A, Bhowmik B, Mdala I, Siddiquee T, Fernandes VO, *et al*. Prevalence of metabolic syndrome by different definitions, and its association with Type 2 diabetes, pre-diabetes, and cardiovascular disease risk in Brazil. Diabetes Metab Syndr Clin Res Rev 2020;14:1217-24. https://doi.org/10.1016/j.dsx.2020.05.043
45. Tabatabaei-Malazy O, Moghaddam SS, Rezaei N, Sheidaei A, Hajipour MJ, Mahmoudi N, *et al*. A nationwide study of metabolic syndrome prevalence in Iran; a comparative analysis of six definitions. PLoS One 2021;16:e0241926. doi:10.1371/journal.pone.0241926
46. Ford ES. Prevalence of the metabolic syndrome defined by the international diabetes federation among adults in the U.S. Diabetes Care 2005;28:2745-9. doi:10.2337/diacare.28.11.2745
47. Mahmoud I, Sulaiman N. Prevalence of metabolic syndrome and associated risk factors in the United Arab Emirates: A cross-sectional population-based study. Front Public Health 2022;9:811006. doi:10.3389/fpubh.2021.811006
48. Das Gupta R, Tamanna R, Akonde M, Biswas T, Chakraborty P, Hossain MB. Prevalence and associated factors of metabolic syndrome among Bangladeshi adults: Evidence from a nation-wide survey. Diabetes Epidemiol Manag 2021;5:100037. doi:10.1016/j.deman.2021.100037
49. Erem C, Hacıhasanoglu A, Deger O, Topbaş M, Hosver I, Ersoz HO, *et al*. Prevalence of metabolic syndrome and associated risk factors among Turkish adults: Trabzon MetS study. Endocrine 2008;33:9-20. doi:10.1007/s12020-008-9044-3
50. Rankinen T, Kim SY, Pérusse L, Després JP, Bouchard C. The prediction of abdominal visceral fat level from body composition and anthropometry: ROC analysis. Int J Obes 1999;23:801-9. doi:10.1038/sj.ijo.0800929
51. Lemieux S, Prud'homme D, Bouchard C, Tremblay A, Després JP. A single threshold value of waist girth identifies normal-weight and overweight subjects with excess visceral adipose tissue. Am J Clin Nutr 1996;64:685-93. doi:10.1093/ajcn/64.5.685
52. Banerji MA, Lebowitz J, Chaiken RL, Gordon D, Kral JG, Lebovitz HE. Relationship of visceral adipose tissue and glucose disposal is independent of sex in black NIDDM subjects. Am J Physiol Metab 1997;273:E425-32. doi:10.1152/ajpendo.1997.273.2.E425
53. Sowers JR, Epstein M, Frohlich ED. Diabetes, hypertension, and cardiovascular disease. Hypertension 2001;37:1053-9. doi:10.1161/01.HYP.37.4.1053
54. Reaven GM. Syndrome X: 6 years later. J Intern Med Suppl 1994;736:13-22. Available from: http://europepmc.org/abstract/MED/7986303
55. Timar O, Sestier F, Levy E. Metabolic syndrome X: A review. Can J Cardiol 2000;16:779-89.
56. Prabhakaran D, Anand SS. The metabolic syndrome: An emerging risk state for cardiovascular disease. Vasc Med 2004;9:55-68. doi:10.1191/1358863x04vm515ra
57. Kohler HP. Insulin resistance syndrome: Interaction with coagulation and fibrinolysis. Swiss Med Wkly 2002;132:241-52.
58. Haffner SM. The prediabetic problem: Development of non-insulin-

dependent diabetes mellitus and related abnormalities. J Diabetes Complications 1997;11(2):69-76. doi: https://doi.org/10.1016/S1056-8727(96)00099-2

59. Gerstein HC, Prem P, Janice P, Salim Y. Relationship of glucose and insulin levels to the risk of myocardial infarction: A case-control study. J Am Coll Cardiol 1999;33:612-9. doi:10.1016/S0735-1097(98)00637-8
60. Gerstein HC, Yusuf S. Dysglycaemia and risk of cardiovascular disease. Lancet 1996;347:949-50. doi:10.1016/S0140-6736(96)91420-8
61. Mitchell BD, Haffner SM, Hazuda HP, Valdez R, Stern MP. The relation between serum insulin levels and 8-year changes in lipid, lipoprotein, and blood pressure levels. Am J Epidemiol 1992;136:12-22. doi:10.1093/oxfordjournals.aje.a116416
62. McKenney JM. Understanding and treating dyslipidemia associated with noninsulin-dependent diabetes mellitus and hypertension. Pharmacother J Hum Pharmacol Drug Ther 1993;13:340-52. doi: https://doi.org/10.1002/j.1875-9114.1993.tb02741.x
63. Grundy SM. Hypertriglyceridemia, insulin resistance, and the metabolic syndrome. Am J Cardiol 1999;83:25-9. doi:10.1016/S0002-9149(99)00211-8
64. Sniderman AD, Scantlebury T, Cianflone K. Hypertriglyceridemic HyperapoB: The unappreciated atherogenic dyslipoproteinemia in Type 2 diabetes mellitus. Ann Intern Med 2001;135:447-59. doi:10.7326/0003-4819-135-6-200109180-00014
65. Reynisdottir S, Angelin B, Langin D, Lithell H, Eriksson M, Holm C, *et al*. Adipose tissue lipoprotein lipase and hormone-sensitive lipase. Arterioscler Thromb Vasc Biol 1997;17:2287-92. doi:10.1161/01.ATV.17.10.2287
66. Enas EA, Yusuf S, Mehta JL. Prevalence of coronary artery disease in Asian Indians. Am J Cardiol 1992;70:945-9. doi:10.1016/0002-9149(92)90744-J
67. Haverinen E, Paalanen L, Palmieri L, Padron-Monedero A, Noguer-Zambrano I, Suárez RS, *et al*. Comparison of metabolic syndrome prevalence using four different definitions a population-based study in Finland. Arch Public Health 2021;79:231. doi:10.1186/s13690-021-00749-3