

# Metabolic syndrome and its clinical correlates among patients attending a tertiary referral hospital in central Kerala, India

### Megha Isac,<sup>1</sup> Asha Biju,<sup>1</sup>\* Arun Basil Mathew,<sup>2</sup> Thundil Simon Francis<sup>3</sup>

#### ABSTRACT

#### Background

Metabolic syndrome denotes a clustering of cardiovascular risk factors. This includes obesity, dyslipidaemia and hypertension. This study was undertaken to understand the prevalence of metabolic syndrome and its clinical correlates in a tertiary care hospital in central Kerala, India.

### Methods

This cross-sectional study was carried out among inpatients. Information was collected by personal interview, physical examination and by analysing blood samples. We evaluated the patients for common socioeconomic and disease factors that may influence the development of metabolic syndrome. Data was analysed using chi-squared test/Fisher's exact test.

#### GJMEDPH 2020; Vol. 9, issue 4

 <sup>1</sup> Department of General Medicine, MOSC Medical College, Kolenchery, India
 <sup>2</sup> Department of CTVS, Amrita Institute of Medical Sciences, Kochi
 <sup>3</sup> Head of Department, MOSC Medical College, Kolenchery, India

#### \*Corresponding Author

Asha Biju, Associate Professor Department of General Medicine, MOSC Medical College, Kolenchery, India ashabiju@hotmail.com Phone No: 9946579222

Conflict of Interest-none

Funding—none

### Results

Overall prevalence of metabolic syndrome was found to be 38.8% (47.5% among females and 31.1% in males). The prevelance of metabolic syndrome among diabetic, hypertensive and dyslipidaemia patients was 76.4%, 83.3% and 89.2% respectively. Prevalence of metabolic syndrome was higher among subjects with a family history diabetes, hypertension and dyslipidaemia. Prevalence in people with abdominal obesity was high (42.95%), whilst prevalence was low in people who reported exercising regularly and amongst more highly educated patients.

### Conclusions

Our study records a higher prevalence of metabolic syndrome than previous studies carried out in India. We recorded a higher prevalence of metabolic syndrome in women, which contradicts some previous studies carried out in India. Diabetes, hypertension, dyslipidaemia and a family history of diabetes may be risk factors for metabolic syndrome, whilst regular exercise and a higher levels of education may reduce the risk.

Keywords: Metabolic Syndrome, Cardiovascular, Diabetes, Hypertension

### INTRODUCTION

Metabolic syndrome is a term used to indicate the presence of a combination of conditons including hypertension, abdominal (central) fat accumulation, impaired glucose tolerance, insulin resistance and atherogenic dyslipidaemia, as well as prothrombotic and/or inflammatory states.<sup>1,2</sup> The pathogenesis of metabolic syndrome is complex and incompletely understood, though obesity, sedentary lifestyle, poor diet, environmental conditions, ethnicity, advancing

age, endocrine dysfunction and genetic factors are all known to contribute to its development.<sup>3,4</sup> Large prospective population-based studies such as the Framingham Offspring Study,<sup>5</sup> the Botnia Study,<sup>6</sup> the Kuopio Ischemic Heart Disease Study,<sup>7</sup> the Italian Study,<sup>8</sup> and the Atherosclerosis Risk in Communities (ARIC) study,<sup>9,10</sup> have confirmed metabolic syndrome to be significantly associated with an increased risk of cardiovascular disease, morbidity and mortality.<sup>1</sup>



The U.S. National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP III)<sup>11</sup> is a set of quidelines that provides clinicians with evidencebased recommendations on the classification, diagnosis and treatment of lipid disorders. The quidelines have identified six components of metabolic syndrome. These are abdominal obesity, high blood pressure, insulin resistance, glucose intolerance, proinflammatory state (C-Reactive Protein), prothrombotic state (plasma plasminogen inhibitor activator and fibrinogen)<sup>3</sup> and atherogenic dyslipidaemia, (increased triglycerides, decreased high density lipoproteins (HDL), increased remnant lipoproteins, increased apolipoprotein B and increased small low density lipoproteing (LDL) particles). Each of these in turn has complex risk factors. Multiple genetic and environmental factors are thought to influence the manifestation of abdominal obesity, for example. Intra-abdominal fat increases with age in both overweight and normal weight individuals, independent of changes in total body fat. Sex hormones also appear to contribute to body fat distribution, as men have twice as much abdominal fat as women<sup>12,13</sup> and oestrogen deficiency after menopause is associated with an increase in intra-abdominal fat, which can be eased by oestrogen replacement therapy.<sup>14,15</sup> Some ethnic groups have higher predisposition to abdominal obesity than others. Asian populations display more metabolic abnormalities with the same level obesity than do Caucasians.<sup>16</sup> There is also evidence that increased abdominal adipose tissue is associated with physical inactivity and that increased plasma cortisol levels can be influenced throughout life by conditons in the intrauterine environment.<sup>17</sup> Genetic factors clearly play a role in body fat distribution: family studies have shown that genetic factors account for 50% of the variance in intra-abdominal fat after adjusting for age, sex and total body fat.<sup>18</sup> Genetic factors that predispose some individuals to gain abdominal weight may explain the susceptibility of certain ethnic groups to diabetes mellitus type-2.<sup>19,20</sup>

### METHOD AND MATERIALS

We undertook a cross-sectional study of 170 participants selected from inpatients in a medical college situated in rural area of central Kerala.

Inclusion criteria was patients aged 20–80 years, who were admitted to the Department of Medicine wards during the study period, who were willing to be the part of the study. Critically ill patients and patients presenting with advanced malignancies or chronic infections such as tuberculosis were excluded. So too were patients with an established cause of secondary hyperglycemia, such as endocrinopathies, or druginduced and secondary hypertension such as renal artery stenosis or pheochromocytoma. Pregnant patients and patients with ascites were also excluded. The study period was March 2013 to April 2014.

# **Ethical considerations**

Permission for the study was obtained from the Head of Department of the General Medicine Department and Hospital Administration. The institutional ethics committee provided institutional clearance. Informed consent was obtained from every patient.

# Data collection and analysis

Once eligible patients were identified and informed consent had been given, baseline demographic information and clinical variables were obtained through personal interviews and by using a detailed proforma. Age, gender, religion, domicile, marital status, education, occupation and average monthly family income were recorded.

Patients' medical histories were noted and a detailed physical examination was carried out to give an initial evaluation of general health. Relevant information regarding known risk factors for metabolic syndrome were recorded, including daily and weekly exercise habits, alcohol use, smoking habits and dietary preferences, as well as any previous diagnosis of diabetes mellitus type-2, systemic hypertension, dyslipidaemia or a family history of the same. Additional information about depression or other mental illnesses that could affect compliance with treatment recommendations was noted. Patients were classified as pure vegetarian or non-vegetarian according to Indian cultural dietary norms.

Patients' exercise habits were recorded and defined as low, moderate or vigorous intensity. Moderate intensity was defined as 30 minutes per day bicycling,



walking at a speed of 3-4km per hour (continuous or intermittent), or aerobics activity. Vigorous exercise included jogging, skipping, push-ups, sit-ups, pull-ups, jumping jacks etc. The American Heart Association recommends at least 150 minutes of moderate exercise or 75 minutes of vigorous exercise per week, or a combination of both. Patients were divided into four groups depending on their exercise habits: those who exercised regularly; those who exercised two times or fewer in a week; and those who never exercised.

Patients were divided into three groups depending on alcohol consumption: patients who admiited to consuming alcohol; patients who had regularly consumed alcohol in the past but who no longer did so; and patients who had never regularly consumed alcohol. The same three categories were recorded for smoking habits.

Patients' body weight was measured in light outdoor clothing, without shoes, on standard weighing scales. Weight was recorded to the nearest o.5kg. Height was measured in metres on a wall stadiometer to the nearest o.1cm. BMI was calculated as body weight in kg/m<sup>2</sup> of height. Waist circumference was measured at the midpoint, between the lowest margin of the ribs and the lateral border of the iliac crest, during minimal respiration. Hip girth was measured at the maximum circumference of the buttocks with the subject wearing minimum clothing. From this, waist:hip ratio (WHR) was calculated.

Blood pressure was measured three times per participant, in a seated position after at least five minutes rest, using a calibrated sphygmomanometer. Participants were advised to avoid smoking, caffeinated beverages, alcohol and exercise for at least 30 minutes before having their blood pressure measured. Any abnormalities were noted. Blood specimens were obtained by venepuncture after eight hours of overnight fasting and evaluated for fasting blood sugars and serum lipids, including total cholesterol, triglycerides, high density lipoproteins (HDL), low-density lipoproteins (LDL) and very lowdensity lipoproteins. Blood specimens were centrifuged and plasma was analysed using glucose oxidase peroxidise method. Serum lipids were measured using enzymatic method in a dry chemistry analyser (Vitrios 250). Results of other blood investigations were taken from patient records if available, such as complete blood counts, HBA1c, liver function tests, renal function tests, thyroid function test and reports of ECG, chest X-ray, abdomen ultrasonography or echo cardiogram.

# Diagnostic criteria for metabolic syndrome

Metalbolic syndrome was diagnosed according to the Revised NCEP ATP-III definition<sup>29</sup> with the waist circumference adjusted according to ethnicity<sup>22</sup>. For metabolic syndrome to be diagnosed, any of the following three criteria had to be present:

- Abdominal (central) obesity, males > 90cm and females > 80cm
- Raised trigylcerides (Tg) >/= 150 mg/dL or on specific treatment for this lipid abnormality.
- Reduced HDL < 40mg/dl in males, <50 mg/dL in females or on treatment specific for this lipid abnormality.
- 4) Raised blood pressure (BP), systolic BP >/= 130mm of Hg or diastolic BP >/= 85mm of Hg or on treatment for previously diagnosed hypertension.
- 5) Raised fasting plasma glucose/fasting blood sugar >/= 100 mg/dl or previously diagnosed type-2 diabetes.

# Statistical analysis

Sample size = 
$$\frac{Z^2 * (p) * (1-p)}{C^2}$$

Z = Z value (standard normal distribution) p = percentage, expressed as decimal (0.5 used for

sample size needed), and c = confidence interval, expressed as decimal.

# Correction for finite population

A sample size of 170 was calculated using an online calculator – Creative Research Systems Version 11.0 (<u>www.surveysystem.com/sscalc.htm</u>) assuming a population size of 14,000 inpatients in the Department of General Medicine in one year with an

expected frequency of 50%. The confidence level was set at 95%. All data were transferred from the proforma into a Microsoft Excel spreadsheet for analysis. The prevalence of metabolic syndrome was analyzed from the collected data using chi-squared test/Fisher's exact test. Any association with a p value <0.05 was taken to be statistically significant.

# RESULTS

Our study found that metabolic syndrome (MetS) was highest in the 61-80 age group: 57.1% of our study group in this age range had MetS (table 1). This was slightly lower in the 47% of the study group who were female; 47.5% of female participants had MetS (table 2). Comparison between diabetics and non-diabetics showed 76.4% of diabetics had MetS compared with 20.9% of non-diabetics (table 3) and 83.3% of hypertensives had MetS in contrast to 24.2% of non-hypertensives (table 4). A family history of diabetes and dyslipidaemia was recorded for 46.4% of patients; of these, 62% had MetS (table 5) compared with only 18.7% of patients with no family history; of the 27.5% with a family history of dyslipidaemia, 62% of had MetS (table 6).

There was a strong correlation with exercise: only 7.1% people who reported exercising daily had MetS, compared with 60.3% of people who reported never exercising (table 7). The relationship with MetS and alcohol was unclear, however, as while 77.8% people who reported consuming alcohol regularly had MetS,

compared with 62.9% of people who said they had never consumed alcohol (table 8) (p=0.52). The lowest incidence (57.1%) was found in people who had consumed alcohol in the past but did not do so currently. Current smokers were more likely to have MetS (75%) than non-smokers (64.4%) (table 9). The study group consisted of 74.1% non-vegetarians, of whom 36.5% suffered from MetS, compared with 45.4% of vegetarians (table 10) (P 0.294). None of these differences were stastistically significant. As expected, abdominal obesity was a statistically significant risk factor. Only 20.6% of patients who were not obese had MetS, compared with 63% of patients with abdominal obesity (table 11). MetS was recorded in a high percentage of hypertriglyceridemic patients (85.5%) (table 12), and 80.3% of patients with low HDL (table 13).

relationship with education was highly The significant. The study sample contained 25.8% postgraduates, 28.2% graduates, 24.1% who had completed 10<sup>th</sup> Standard (secondary education) and 21.7% who had not completed 10<sup>th</sup> Standard. MetS prevalence among postgraduates and graduates was 22.7% and 29.2% respectively (table 14). The study participants were divided into three groups depending on annual income; MetS prevalence was 41.7% among the lowest income groups (<500,000INR), 45.1% among middle income (500,000-1,000,000INR) and 27% among the high income (>1,000,000INR) group (p=0.366).

Age	MetS		Total	P-value		
	No	Yes				
20 – 40	39 (90.7%)	4 (9.3%)	43	_		
41 – 60	38 (59.4%)	26 (40.6%)	64	0.000		
61 – 80	27 (42.9%)	36 (57.1%)	63	0.000		
Total	104 (61.2%)	66 (38.8%)	170			

### Table 1 Prevalence of metabolic syndrome (MetS) according to age

#### Table 2 Prevalence of metabolic syndrome (MetS) among males and females

Gender	MetS		Total	P-value
	No	Yes		
Male	62 (68.9%)	28 (31.1%)	90	
Female	42 (52.5%)	38 (47.5%)	80	0.029
Total	104 (61.2%)	66 (38.8%)	170	

www.gjmedph.com Vol. 9, No. 4, 2020

DM	MetS		Total	P-value
	No	Yes		
No	91 (79.1%)	24 (20.9%)	115	
Yes	13 (23.6%)	42 (76.4%)	55	0.000
Total	104 (61.2%)	66 (38.8%)	170	

### Table 3 Prevalence of metabolic syndrome (MetS) among diabetic and non-diabetic patients

Table 4 Prevalence of metabolic syndrome (MetS) among hypertensive and non-hypertensive patients

HTN	MetS		Total	P-value
	No	Yes		
No	97 (75.8%)	31 (24.2%)	128	
Yes	7 (16.7%)	35 (83.3%)	42	0.000
Total	104 (61.2%)	66 (38.8%)	170	

# Table 5 Prevalence of metabolic syndrome (MetS) among patients with and without family history of diabetes

Family history of	MetS		Total	P-value
DM	No	Yes		
No	74 (81.3%)	17 (18.7%)	91	
Yes	30 (38.0%)	49 (62.0%)	79	0.000
Total	104 (61.2%)	66 (38.8%)	170	

# Table 6 Prevalence of metabolic syndrome among patients with and without family history of DLP

Family history of	MetS		Total	P-value
DLP	No	Yes		
No	95 (64.6%)	52 (35.4%)	147	
Yes	9 (39.1%)	14 (60.9%)	23	0.020
Total	104 (61.2%)	66 (38.8%)	170	

#### Table 7 Physical activity and metabolic syndrome (MetS)

Physical Activity	MetS		Total	P-value
	No	Yes		
Daily	26 (92.9%)	2 (7.1%)	28	
3 Times a week	19 (76.0%)	6 (24.0%)	25	
2 Times a week	32 (65.3%)	17 (34.7%)	49	0.000
Never	27 (39.7%)	41 (60.3%)	68	
Total	104 (61.2%)	66 (38.8%)	170	

#### Table 8 Alcohol intake and metabolic syndrome (MetS)

Alcohol	MetS		Total	P-value
	No	Yes		
Current	14 (77.8%)	4 (22.2%)	18	
Quit	12 (42.9%)	16 (57.1%)	28	0.050
Never	78 (62.9%)	46 (37.1%)	124	0.052
Total	104 (61.2%)	66 (38.8%)	170	

www.gjmedph.com Vol. 9, No. 4, 2020

ISSN#- 2277-9604

Table 9 Smoking habits and metabolic syndrome	(MetS)
-----------------------------------------------	--------

Smoking	MetS		Total	P-value
	No	Yes		
Current	9 (75.0%)	3 (25.0%)	12	
Quit	8 (34.8%)	15 (65.2%)	23	0.017
Never	87 (64.4%)	48 (35.6%)	135	0.017
Total	104 (61.2%)	66 (38.8%)	170	

# Table 10 Dietary habits and metabolic syndrome (MetS)

Dietary habits	MetS		Total	P-value
	No	Yes		
Vegetarian	24 (54.5%)	20 (45.5%)	44	
Non-vegetarian	80 (63.5%)	46 (36.5%)	126	0.294
Total	104 (61.2%)	66 (38.8%)	170	

### Table 11 Central obesity and metabolic syndrome (MetS)

Central obesity	MetS		Total	P-value
	No	Yes		
No	77 (79.4%)	20 (20.6%)	97	
Yes	27 (37.0%)	46 (63.0%)	73	0.000
Total	104 (61.2%)	66 (38.8%)	170	

#### Table 12 Hypertriglyceridemia and metabolic syndrome (MetS)

Hypertriglyceridemia	MetS		Total	P-Value	
	No	Yes			
Νο	96 (83.5%)	19 (16.5%)	115	0.000	
Yes	8 (14.5%)	47 (85.5%)	55	0.000	

### Table 13 High Density Lipoprotein (HDL) and metabolic syndrome (MetS)

Low HDL	MetS		Total	P-value
	No	Yes		
Νο	89 (94.7%)	5 (5.3%)	94	
Yes	15 (19.7%)	61 (80.3%)	76	0.000
Total	104 (61.2%)	66 (38.8%)	170	

### Table 14 Comparison of education and metabolic syndrome (MetS)

Education	MetS		Total	P-value	
	No	Yes			
Below 10 Standard	20 (54.1%)	17 (45.9%)	37		
Above 10 Standard	16 (39.0%)	25 (61.0%)	41		
Graduate	34 (70.8%)	14 (29.2%)	48	0.001	
Postgraduate	34 (77.3%)	10 (22.7%)	44		
Total	104 (61.2%)	66 (38.8%)	170		

#### Table 15 Comparison of annual income in metabolic syndrome (MetS) and non-metabolic syndrome groups

Annual Income	MetS		Total	P-value	
	No	Yes			
Below Rs. 5,00,000	39 (54.9%)	32 (45.1%)	71		
Rs. 5,00,000 – 10,00,000	35 (66.0%)	18 (34.0%)	53	0.366	
Above Rs. 10,00,000	30 (65.2%)	16 (34.8%)	46	0.300	
Total	104 (61.2%)	66 (38.8%)	170		

#### DISCUSSION

The original World Health Organization definition of metabolic syndrome laid special emphasis on insulin resistance but the more recent definition from the U.S. National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP-III) has given equal importance to individual components. The International Diabetes Federation (IDF) takes abdominal (central) obesity as a key risk factor. For the patients admitted to the medical wards during our study period, who satisfied the inclusion criteria for the study, we estimated the prevalence of metabolic syndrome as per the modified NCEP ATP-III definition. Waist circumference was adjusted to the appropriate standards for the Indian population.<sup>22</sup>

The worldwide prevalence of reported metabolic syndrome in populations ranges from 3.5% to 50%<sup>23-25</sup>; in our study it was 38.8%. Prevalence of metabolic syndrome may vary with ethnic background<sup>26</sup> and the reasonably high prevalence observed in our study suggests that Indian Asians may be more prone to it than populations in other parts of the world. However, reports from different parts of India have observed significant differences in the prevalence of metabolic syndrome even within the same ethnic population group<sup>27,28</sup>.

In our study, there was significant difference in the prevalence of metabolic syndrome in different age groups. Prevalence increased with age, with the highest prevalence observed in participants aged between 61 and 80 years. Previously, Nalia Hamid et al conducted a hospital-based study in Pakistan on the association between metabolic syndrome and age, and observed it to be more common in the 51-60 years age group.<sup>29</sup> This difference could be because our study population had adopted a more sedentary lifestyle at a later age, probably after retirement.

In the present study, the prevalence of metabolic syndrome was found to be higher in women (47.5%) than in men (31.1%). This is similar to the findings of a survey from Chandigarh in North India<sup>30</sup>, and in the Jaipur Heart Watch Studies, both of which found women to be at greater risk<sup>31</sup>. However, other studies have found men to be at higher risk: Chow et al found

prevalence of metabolic syndrome to be 26.9% in men but only 18.4% in women in a study from a developing region of rural Andra Pradesh<sup>32.</sup> Another study from urban areas in India has also reported higher prevalence in men than women.<sup>33</sup> Worldwide reports on gender differences in metabolic syndrome rates are equally inconsistent. In a hospital-based study of type-2 diabetics in Nigeria, men and women in the 35-80 years age group showed similar prevalence.<sup>35</sup> In the US population overall, ageadjusted prevalence is similar for women and men, but African-American women have a 57% higher prevalence than African-American men and Mexican-American women have a 26% higher prevalence than Mexican-American men.<sup>34</sup>

Higher male prevalence is also reported in Gulf Cooperation Council Countries<sup>36</sup> and in Taiwan, where a nationwide population-based survey of metabolic syndrome recorded 20.4% prevalence in men and 15.3% in women<sup>37</sup>. A report of 11 prospective European cohort studies that surveyed 6,156 men and 5,356 women aged 30-89 years without diabetes also reported slightly male higher prevalence<sup>38</sup> and a study from Peshawar, Pakistan reported metabolic syndrome in 66% of men but only 34% of women.<sup>31</sup> It appears that metabolic syndrome afflicts both men and women of all races but prevalence rates between genders differ significantly between different populations. In our study, women were more susceptible to metabolic syndrome. We hypothesise that in our study group the reason may be that women tend to exercise less, but we have not analysed that data.

The presence of hypertension constitutes one criterion for metabolic syndrome but is not a prerequisite by the Asia Diabetes Federation definition. In our study, prevalence of hypertension was significantly higher in the group with metabolic syndrome compared to those without. Our finding is in accordance with those from a recent evaluation of the Framingham Heart Study, which found that hypertension was the risk factor most often associated with the diagnosis of metabolic syndrome.<sup>39</sup>



In the present study, among of the 55 subjects with diabetes mellitus type-2, 42 were diagnosed with metabolic syndrome and 13 were not. Among the 37 study subjects with dyslipidaemia, 33 had metabolic syndrome; 76 study subjects had low HDL levels and 55 had hypertriglyceridemia.

The prevalence of abdominal (central) obesity was found in 73 out of the total 170 subjects. In the present study, the disease load of dyslipidaemia, diabetes mellitus type-2 and hypertension were significantly higher in subjects with metabolic syndrome. The majority of these patients were on pharmacotherapy, suggesting that pharmacotherapy to control diabetes mellitus type-2 does not necessarily facilitate reduced central obesity.

The prevalence of metabolic syndrome was significantly lower in people who exercised regularly (7.1%) and highest among those who never exercised (60.3%). Further studies with fewer confounding factors and a bigger sample size are required to study the importance of exercise, as the percentage of subjects who exercised regularly in this study was limited – 68 subjects out of the total 170 (40%).

Most of the study subjects were non-vegetarians; 44 were vegetarians. The prevalence of metabolic syndrome was 36.5% among non-vegetarians and 45.5% among vegetarians. The p-value was 0.294, however, so significant association could not be established between diet and prevalence of metabolic syndrome from this study.

According to reported behaviour collected from interviews, 124 out of 170 subjects had never consumed alcohol and the prevalence of metabolic syndrome in this group was 37.1%. The prevalence was 22.2% among those who consumed alcohol regularly and 57.1% among those had previously done so but had quit (p-value = 0.052). 135 subjects reported no history of smoking. The prevalence of metabolic syndrome among non-smokers and smokers was 35.6% and 25% respectively. The prevalence among those who had quit smoking was 65.2% (p-value=0.017). This raises questions over whether alcohol may decrease metabolic syndrome? We suggest that the results in this study may be due to the nutritional status of alcohol: subjects with poor nutrition may gain some macro and micronutrients from alcoholic drinks. They may be prone to other type of complications, however.

According to this study there is no statistically significant association between annual income and prevalence of metabolic syndrome. The prevalence was 45.1% among those with annual income less than 5 lakh rupees (500,000 INR), 34% among those with an annual income between 5 lakh rupees and 10 lakh rupees, and 34.8 % among those with annual income of more than 10 lakh rupees. There was a significant difference in the prevlance of metabolic syndrome among various groups based on educational qualification, on the other hand. The prevalence was among postgraduates, 22.7% 29.2% among graduates, 61% among those who passed 10<sup>th</sup> Standard (completed secondary education) and 45.9% in those who had left school before completing 10<sup>th</sup> Standard. Education seems to improve the health of the subjects in relation to metabolic syndrome, however not in a linear manner. This could be due to the type of jobs people engaged in; subjects who complete 10<sup>th</sup> standard may be less likely to be employed as physical labourers compared to the subjects who had lower levels of education.

The prevalence of metabolic syndrome was higher among subjects with a positive family history of diabete mellitus type-2, systemic hypertension and dyslipidaemia. These findings emphasize some key risk factors for metabolic syndrome and stress and the need for each component condition (obesity, hypertension, insulin resistance dyslipidaemia and hypercoagulable disorders) to be treated separately. Lifestyle modification should be the firstline approach to the management of patients with metabolic syndrome and insulin resistance. Women may need to concentrate on routine exercises, which is not a common practice in the study area; women may not be getting sufficient time for outdoor exercise. The climate may not be ideal for outdoor exercise for both men and women. If so, it may be beneficial for the patients to concentrate on indoor aerobic exercises such as skipping and dancing.

#### CONCLUSION

In the present study, the prevalence of metabolic syndrome is 38.8%, which is higher than has been observed in other Indian studies. Peak prevalence is observed in subjects aged 61-80 years and prevalence decreased with age. Prevalence of metabolic syndrome is found to be relatively higher in women (47.5%) compared with men (31.1%). Frequency of diabetes mellitus type-2, systemic hypertension and dyslipidaemia is significantly higher in the metabolic syndrome group compared to the study subjects who do not have metabolic syndrome. The prevalence of abdominal (central obesity) is high: 73 out of the total 170 subjects (42.9%), but is significantly lower in people who exercise regularly. Prevalence is higher among subjects with a positive family history of diabetes mellitus type-2, systemic hypertension and dyslipidaemia and lower among those who have higher levels of education.

#### LIMITATIONS OF THE STUDY

The present study is not a population-based study and so several inevitable biases are present. The results might show seasonal variations: for example, a higher percentage of younger individuals with fewer co-morbidities may be admitted during rainy seasons, when they are more prone to fever endemics. Lifestyle modification is considered to be one of the most effective remedial measures for

### REFERENCES

- 1. Meigs JB: Epidemiology of the metabolic syndrome, 2002. Am J Manag Care 2002;8: S283-292; quiz S293-286.
- Hu G, Qiao Q, Tuomilehto J, Balkau B, Borch-Johnsen K, Pyorala K: Prevalence of the metabolic syndrome and its relation to all-cause and cardiovascular mortality in nondiabetic European men and women. Arch Intern Med 2004; 164:1066-1076.
- Reaven GM: Banting lecture 1988. Role of insulin resistance in human disease. Diabetes 1988; 37:1595-1607.
- Liese AD, Mayer-Davis EJ, Haffner SM: Development of the multiple metabolic syndrome: an epidemiologic perspective. Epidemiol Rev 1998; 20:157-172.

metabolic syndrome. The study includes indicators incuding smoking, alcohol consumption, diet and exercise habits for which the details are obtained from interview alone. This may be subject to reporting bias and so results may not be accurate. They study was conducted over a limited period and was a cross-sectional study with a low sample size.

Further, larger studies are required to confirm these results. Genetic predisposition to metabolic syndrome was studied only from available family history, which is not necessarily a reliable indicator. The highest incidence was noted in the age group 61-80; subjects >80 years were not included in the study.

### ACKNOWLEDGEMENTS

We express our sincere gratitude to Dr Anna Mathew, Professor in the Department of Pharmacology and Dr Marina Rajan Joseph, Professor in the Department of Community Medicine, formerly working in the same institution, for their valuable suggestions and support during the course of the study.

We wish to express our sincere gratitude to Professor Dr Mariamma Kuriakose and Professor Dr Abraham Ittyachan M for all their motivation during the course of study. We acknowledge all the help extended by the Department of Biostatistics.

- 5. Wilson PW, Kannel WB, Silbershatz H, D'Agostino RB: Clustering of metabolic factors and coronary heart disease. Arch Intern Med 1999; 159:1104-1109.
- Isomaa B, Almgren P, Tuomi T, Forsen B, Lahti K, Nissen M, Taskinen MR, Groop L: Cardiovascular morbidity and mortality associated with the metabolic syndrome. Diabetes Care 2001; 24:683-689.
- Lakka HM, Laaksonen DE, Lakka TA, Niskanen LK, Kumpusalo E, Tuomilehto J, Salonen JT: The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. Jama 2002;288: 2709-2716.
- 8. Trevisan M, Liu J, Bahsas FB, Menotti A: Syndrome X and mortality: a population-based study. Risk Factor



and Life Expectancy Research Group. Am J Epidemiol 1998;148: 958-966.

- 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; 287:356-359.
- 10. Schmidt MI, Watson RL, Duncan BB, Metcalf P, Brancati FL, Sharrett AR, Davis CE, Heiss G: Clustering of dyslipidemia, hyperuricemia, diabetes, and hypertension and its association with fasting insulin and central and overall obesity in a general population. Atherosclerosis Risk in Communities Study Investigators. Metabolism1996; 45:699-706.
- 11. Lorenzo C, Williams K, Hunt KJ, Haffner SM. The National Cholesterol Education Program–Adult Treatment Panel III, International Diabetes Federation, and World Health Organization definitions of the metabolic syndrome as predictors of incident cardiovascular disease and diabetes. Diabetes care. 2007 Jan 1;30(1):8-13.
- 12. Lemieux S, Prud'homme D, Bouchard C, Tremblay A, Despr'es JP 1993 Sex differences in the relation of visceral adipose tissue accumulation to total body fatness. Am J Clin Nutr 58:463–467.
- 13. Carr MC, Hokanson JE, Zambon A, Deeb SS, Barrett PH, Purnell JQ, Brunzell JD 2001 The contribution of intraabdominal fat to gender differences in hepatic lipase activity and low/high density lipoprotein heterogeneity. J Clin Endocrinol Metab 86:2831–2837
- 14. Haarbo J, Marslew U, Gotfredsen A, Christiansen C 1991 Postmenopausal hormone replacement therapy prevents central distribution of body fat after menopause. Metabolism 40:1323–1326
- 15. Carr MC, Brunzell JD, increased hepatic lipase activity and intraabdominal fat across the transition from pre- to post menopause. Program of the 85th Annual Meeting of The Endocrine Society, Philadelphia, PA, 2003, p 374 (Abstact P 2-280)
- Liu J, Grundy SM, Wang W, Smith SC, Jr., Vega GL, Wu Z, Zeng Z, Wang W, Zhao D: Ethnic-specific criteria for the metabolic syndrome: evidence from China. Diabetes Care 2006; 29:1414-1416.
- 17. Wajchenberg BL 2000 Subcutaneous and visceral adipose tissue: their relation to the metabolic syndrome. Endocr Rev 21:697–738.
- 18. Perusse L, Despres JP, Lemieux S, Rice T, Rao DC, Bouchard C 1996 Familial aggregation of abdominal

visceral fat level: results from the Quebec family study. Metabolism 45:378–382.

- 19. FujimotoWY1992 the growing prevalence of noninsulin-dependent diabetes in migrant Asian populations and its implications for Asia. Diabetes Res Clin Pract 15:167–183.
- 20. Haffner SM, D'Agostino R, Saad MF, Rewers M, Mykkanen L, Selby J, Howard G, Savage PJ, Hamman RF, Wagenknecht LE, Bergman RN 1996 Increased insulin resistance and insulin secretion in nondiabetic African- Americans and Hispanics compared with non-Hispanic whites. The Insulin Resistance Atherosclerosis Study. Diabetes 45:742–748.
- 21. Reaven G: The metabolic syndrome or the insulin resistance syndrome? Different names, different concepts, and different goals. Endocrinol Metab Clin North Am 2004; 33:283-303.
- 22. Mishra A, Vikram NK, Gupta R, Pandey RM, Wasir JS, Gupta VP. Waist circumference cut-off points and action levels for Asian Indians for identification of abdominal obesifying J Obes (Lond) 2006; 30:106-11.
- 23. Assmann G, Cullen P, Schulte H: Simple scoring scheme for calculating the risk of acute coronary events based on the 10-year follow-up of the prospective cardiovascular Munster (PROCAM) study. Circulation 2002; 105:310-315. (22) Despres JP, Lemieux I: Abdominal obesity and metabolic syndrome. Nature 2006; 444:881-887.
- 24. Cameron AJ, Shaw JE, Zimmet PZ. The Metabolic Syndrome: Prevalence in Worldwide Populations. Endocrinol Metab Clin North Am 2004; 33: 351–76.
- 25. Ford ES. Prevalence of the metabolic syndrome defined by the International Diabetes Federation among adults in the US. Diabetes Care 2005; 28: 2745-9.
- 26. Adams RJ, Appleton S, Wilson DH, et al. Population comparison of two clinical approaches to the metabolic syndrome: implications of the new International Diabetes Federation consensus definition. Diabetes Care 2005; 28: 2777-79.
- 27. Ramachandran A, Snehalatha C, Satyavani K, Sivasankari S, Vijay V. Metabolic syndrome in urban Asian Indian adults-a population study using modified ATP III criteria. Diabetes Res Clin Pract 2003; 60:199-204.
- 28. Gupta A, Gupta R, Sarna M, Rastogi S, Gupta VP, Kothari K. Prevalence of diabetes, impaired fasting



glucose and insulin resistance syndrome in an urban Indian population. Diab Res Clin Pract 2003; 61:69-76.

- 29. Hamid N, Jilliani G, Parveen N, I Haq IU, Arif S, Hussain H. Metabolic syndrome and its relationship with associated risk factors. J. Med. Sci. 2010; 18: 186-90
- 30. Ravikiran M, Bhansali A, Ravikumar P, Bhansali S, Dutta P, Thakur JS, Sachdeva N, Bhadada S, Walia R. Prevalence and risk factors of metabolic syndrome among Asian Indians: a community survey. Diabetes Res Clin Pract. 2010; 89:181-8.
- Gupta R, Deedwania PC, Gupta A, Rastogi S, Panwar RB, Kothari K: Prevalence of metabolic syndrome in an Indian urban population. Int J Cardiol 2004; 97:257-61.
- 32. Chow CK, Naidu S, Raju K, Raju R, Joshi R, Sullivan D, Celermajer DS, Neal BC. Significant lipid, adiposity and metabolic abnormalities amongst 4535 Indians from a developing region of rural Andhra Pradesh. Atherosclerosis 2008; 196: 943–52.
- 33. Apurva Sawant, Ranjit Mankeshwar, Swarup Shah, Rani Raghavan, Gargi Dhongde, Himanshu Raje, Shoba D'souza, Aarti Subramanium, Pradnya Dhairyawan, Seema Todur, and Tester F. Ashavaid. Prevalence of Metabolic Syndrome in Urban India. Cholesterol 2011; 2011:1-7.

- 34. 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.
- 35. Ogbera AO, Prevalence and gender distribution of the metabolic syndrome. Diabetology & Metabolic Syndrome 2010, 2:1
- 36. Mabry RM Reeves MM, Eakin EG, Owen N. Gender differences in prevalence of the metabolic syndrome in Gulf Cooperation Council Countries: a systematic review. Diabet Med. 2010; 27:593-7.
- 37. Hwang LC, Bai CH, Chen CJ, Chien KL. Gender difference on the development of metabolic syndrome: a population-based study in Taiwan. Eur J Epidemiol 2007; 22:899- 906.
- 38. Hu G, Qiao Q, Tuomilehto J, Balkau B, Borch-Johnsen K, Pyorala K; DECODE Study Group. Prevalence of the metabolic syndrome and its relation to all-cause and cardiovascular mortality in nondiabetic European men and women. Arch Intern Med. 2004; 164:1066-76.
- 39. Franco OH, Massaro JM, Civil J, Cobain MR, O'Malley B, D'Agostine RB Sr. Trajectories of entering the metabolic syndrome: the Framingham Heart Study. Circulation.2009; 120:1943-50