Section: General Medicine

Original Research Article

ASSOCIATION OF METABOLIC SYNDROME WITH CORONARY ARTERY DISEASE IN NORTH KARNATAKA POPULATION

Uday Kumar Patil[1], Ambrish Avate[1]

[1]Assistant Professor, Department of General Medicine, Bidar Institute of Medical Sciences, BRIMS Bidar, Karnataka, India

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Corresponding Author:
Dr. Ambrish Avate,
Email: udaypatil3046@gmail.com

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Abstract

Background: Metabolic syndrome is associated with coronary artery disease which includes hypertension, glucose intolerance, dyslipidemia, which are lethal complications. **Materials and Methods:** We compared 90 metabolic syndrome patients with positive CAD angiography with 90 non-Mets volunteers (controlled). Blood investigation, i.e., lipid profile, insulin levels, IL-6, TNF-α, Hs-CRP, HOME-IR, Quickie, and angiological findings showing single vessel disease, double vessel disease, and triple vessel diseases, were analyzed. BMI, HTN, and DM were also noted. **Result:** Comparison of biochemical analysis in metabolic syndrome (MS) with non-metabolic syndrome (Non-MS): TG, HDL, VLDL, and LDL had a significant p value ($p<0.001$). In the present study, 14 (15.3) MS groups had type-II DM, and 72 (80%) HTN were noted, and 8 (8%) had type-II DM, and 11 (12%) HTN was observed in the non-MS group. A comparative study of anthropometric, biochemical, and inflammatory biomarkers had a significant p value ($p<0.001$). **Conclusion:** It was concluded that there is a strong correlation of metabolic syndrome with coronary artery diseases. This study will help the clinician treat patients to avoid morbidity and mortality.

INTRODUCTION

Poineer to describe metabolic syndrome was Reaven in 1988 and later on confirmed by the National Cholesterol Education Program (NCEP), and the definition of metabolic syndrome is to investigate cardiovascular effects of the metabolic syndrome. Worldwide metabolic syndrome is a major health problem associated with morbidity and mortality because of the association with coronary artery disease.[1] Metabolic syndrome represents a host of metabolic abnormalities, including glucose intolerance, dyslipidemia, hypertension, hyperinsulinemia, and abdominal obesity, i.e., increased. Body mass index, which predisposes an individual to coronary artery disease (CAD).[2] As per the prediction of the WHO report, by 2030 India will have 79 million diabetic subjects, which will contribute to more than 20% of the world diabetic population.[3] However, genetic factors and sedentary lifestyles are strongly related in type II DM patients. It is also reported that inflammatory markers and insulin resistance correlate with severity of disease.[4,5] Hence, an attempt is made to evaluate the prevalence of metabolic syndrome association with coronary diseases.

MATERIALS AND METHODS

90 (ninety) adult patients aged between 26 to 60 years regularly visited the Bidar Institute of Medical Sciences, BRIMS Bidar, Karnataka-585401, were studied.

Inclusive Criteria

Patients having chest pain and positive angiography and given written consent for study were selected.

Exclusion Criteria

Patients of chronic kidney diseases, hepatic dysfunction, endocrinal disorders, rheumatological diseases, and immune compromised patients were excluded from this study.

Method: The same number of (90) healthy volunteers (controlled) or non-MS were also studied for comparison. Blood investigations were done for all of them. Fasting blood samples were collected after 12 hours of fasting. Triglycerides (TG) and high-density lipoprotein (HDL) were measured by the cholesterol oxidize phenol 4-aminoantipyrine (CHOD PAP) and lipase-glycerol Glycerol Kinase (LIP/GK) enzymatic clearance methods, respectively, whereas LDL and VLDL were calculated by the Friedewald formula. Tumor necrosis factor-α (TNF-α), interlukin-6 (IL-6), and high-sensitivity C-reactive protein (HS CRP) were measured by the enzyme-linked immunosorbent

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assay method with kits manufactured by Gen-Probe Diaclone, France, and Bio-Check CA, USA.

Insulin estimation was done by microparticle enzyme immune assay with commercial kits supplied by Abbott Laboratory. Insulin resistance and sensitivity were calculated by using homeostatic model analysis of insulin resistance (HOMAIR) fasting insulin (NIU/ml).

IDF criteria for Metabolic Syndrome

1. Central obesity (waist circumference male > 90 cm, female > 80 cm)
2. Raised triglyceride (> 150 mg/ml or on treatment).
3. Reduced HDL cholesterol (< 40 mg/dl in men or < 50 mg/dl in women)
4. Raise blood pressure (systolic ≥130 mm Hg or diastolic ≥ 85 mm Hg).
5. Raised fasting plasma glucose (fasting plasma glucose ≥100 mg/dl or on treatment)

The duration of the study was February 2024 to August 2024.

Statistical analysis: various parameters of MS and non-MS were compared with the z test, and significant values were recorded. The statistical data was calculated in SPSS software. The ratio of males and females was 2:1.

RESULTS

[Table 1] Comparative study of biochemical analysis in metabolic syndrome and non-metabolic syndrome groups –

- TG: 177.7 (± 1.5) in MS group, 168.2 (± 1.66) in non-MS group, t test was 40.2 and p<0.001
- HDL: 35.08 (± 5.52) in MS, 40.4 (± 8.20) in Non MS group, t test 5.1 and p<0.001.
- VLDL: 36.8 (± 7.7) in MS group, 32.66 (± 0.28) in non-MS group, t test 5.09 and p<0.001.
- LDL – 104.06 (± 9.2) in MS group, 98.4 (± 1.26) in non-MS group, t test 5.78 and p<0.001

[Table 2] Comparison of clinical manifestations in both groups –

- Type-II DM: 14 (15.5%) in MS group, 8 (8%) in non-MS group.
- Hypertension: 72 (80%) in the MS group, 11 (12%) in the non-MS group

[Table 3] Comparison of anthropometric biochemical and inflammatory markers in Ms and Non-MS Groups

- Insulin: 52.2 (± 3.2) in MS group, 17.4 (± 2.2) in non-MS group, t test 8.5 and p<0.001.
- IL-6 (interleukin-6): 34.6 (± 8) in MS group, 12.10 (± 0.2) in non-MS group, t test was 25.8 and p<0.001
- TNF-α: 12.2 (± 0.5) in MS group, 7.30 (± 0.2) in non-MS group, t test 8.63 and p<0.001.
- HS CRP: 14.6 (± 0.6) in MS group, 3.3 (± 0.2) in non-MS group, t test 16.9 and p<0.001
- HOMA-IR: 17.7 (± 0.50) in MS group, 5.4 (± 0.2) in non-MS group, t test was 19.6 and p<0.001.
- Quicki: 0.26 (± 0.2) in MS group, 0.32 (± 0.3) in non-MS group, t test 1.57 and p<0.001.
- Single vessel disease: 18 (± 3.2) in MS group, 10 (± 2.2) in non-MS group, t test 19.5 and p<0.001.
- Double vessel disease: 22 (± 8.2) in MS group, 13 (± 4.2) in non-MS group; t test was 9.26 and p<0.001.
- Triple vessel disease: 28 (± 3.5) in MS group, 17 (± 4.6) in non-MS group, t test 17.8 and p<0.001.
- BMI: 24.5 (± 3.2) in MS group, 24.5 (± 3.2) in non-MS group, t test 4.1 and p<0.001.

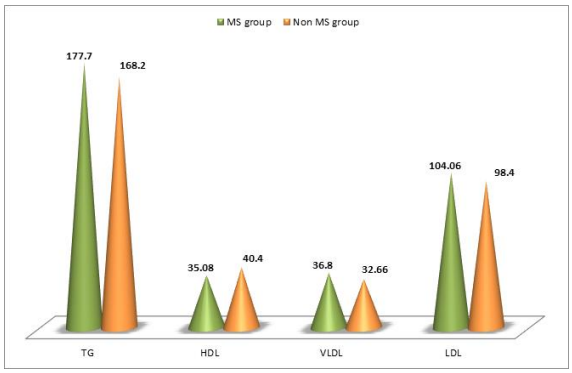

Figure 1: Comparative study of Biochemical analysis in Metabolic syndrome and non-metabolic syndrome Groups

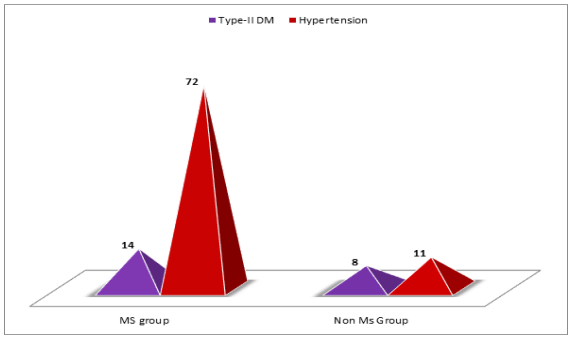

Figure 2: Comparison of clinical manifestations in both groups

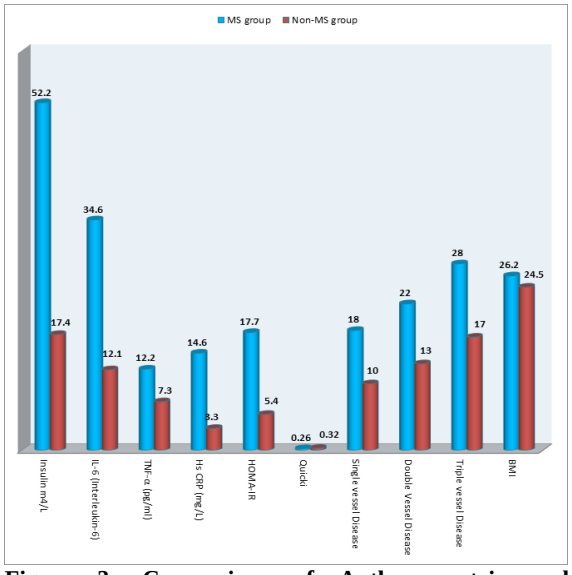

Figure 3: Comparison of Anthropometric and Biochemical and inflammatory markers

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Table 1: Comparative study of Biochemical analysis in Metabolic syndrome and Non- Metabolic syndrome Groups.

Parameters	MS group (90)	Non MS group (90)	t test	p value
TG	177.7 (± 1.5)	168.2 (± 1.66)	40.2	P<0.001
HDL	35.08 (± 5.52)	40.4 (± 8.20)	5.1	P<0.001
VLDL	36.8 (± 7.7)	32.66 (± 0.28)	5.09	P<0.001
LDL	104.06 (± 9.2)	98.4 (± 1.26)	5.78	P< 0.001

Table 2: Comparison of clinical manifestations in both groups

Clinical Manifestation	MS group (90)	Percentage (%)	Non Ms Group (90)	Percentage (%)
Type-II DM	14	15.5 %	8	8 %
Hypertension	72	80 %	11	12 %

Table 3: Comparison of Anthropometric and Biochemical and inflammatory markers

Parameters	MS group (90)	Non-MS group (90)	t test	p value
Insulin m4/L	52.2 (± 3.2)	17.4 (± 2.2)	8.5	P<0.001
IL-6 (Interleukin-6) pg/ml	34.60 (± 0.8)	12.10 (± 0.2)	25.8	P<0.001
TNF-α (pg/ml)	12.2 (± 0.5)	7.30 (± 0.2)	8.63	P<0.001
Hs CRP (mg/L)	14.6 (± 0.6)	3.3 (± 0.2)	16.9	P<0.001
HOMA-IR	17.7 (± 0.56)	5.4 (± 0.2)	19.6	P<0.001
Quicki	0.26 (± 0.2)	0.32 (± 0.3)	1.57	P>0.33
Single vessel Disease	18 (± 3.2)	10 (± 2.2)	19.5	P<0.001
Double Vessel Disease	22 (± 8.2)	13 (± 4.2)	9.22	P<0.001
Triple vessel Disease	28 (± 3.6)	17 (± 4.6)	17.8	P<0.001
BMI	26.2 (± 2.2)	24.5 (± 3.2)	4.1	P<0.001

Quicki = Quantitative Insulin
HOMA = Homeostatic Model Analysis
BMI = Body Mass Index
Hs CRP = highly sensitive check Index
TNFα = Tumour Necrosis Factor alpha

DISCUSSION

Present study of association of metabolic syndrome in North Karnataka population. In a comparative study of biochemical analysis, TG, HDL, VLDL, and LDL have significant p values [Table 1]. In comparison of clinical manifestation in both groups (MS and Non-MS), type-DM was 14 (15.5%) and hypertensive was 72 (80%) in the MS group, but in Non-MS 8 (8%) type-II DM and 11 (12%) hypertensive [Table 2]. In comparison of anthropometric, biochemical, and inflammatory markers had significant p values (Table 3). These findings are more or less in agreement with previous studies.[6-8]

It is an established fact that insulin resistance is the dominant cause of the syndrome. Hence it prefers to be referred to as “Insulin Resistance Syndrome." According to the Insulin Resistance Hypothesis, even obesity elicits the metabolic risk factors through insulin resistance. Moreover, the term pre-diabetes encompasses impaired fasting glucose, and impaired glucose tolerance is meant to identify the elevated risk for type II DM.[9]

ATP III (Third Adult Treatment Panel) indeed defines diabetes itself as a high-risk condition for CAD. It is also reported that metabolic syndrome as defined by ATP-III accounts for the increased risk for congenital heart disease.[10] The pathophysiological mechanism by which metabolic syndrome increases cardiovascular risk remains under debate because many studies have reported that insulin has an independent role as an underlying component of metabolic syndrome. Insulin resistance progresses towards hyperinsulinemia and hyperglycemia; thus, there is a triggering of metabolic syndrome.[11]

Unfortunately, most of the physicians who treat the patients with type II DM fail to recognize the necessity to substantially lower the cholesterol and blood pressure levels and to prescribe aspirin prophylaxis to avoid cardio-vascular risk factors in patients with type II DM, who have features of the metabolic syndrome. Metabolic syndrome (Mets) carries increased long-term risk for atherosclerosis, cardio-vascular diseases, and DM-II as well. It is important to note that the Mets is not a reliable tool to assess the risk of CVD/CAD, but can be a good predictor to start drug therapies for prevention. But once a person is found to be confirmed as Mets, lifestyle and proper diet should be introduced apart from drug therapy.

CONCLUSION

Mets consists of clustering of risk factors of metabolic origin that together are associated with atherosclerotic CVD’s and diabetes. Lifestyle, diet, and drug therapies will dampen the syndrome. But this study demands further genetic, hormonal, angiological, nutritional, and pathophysiological studies as the exact mechanism of insulin resistance and elevation of cholesterol is still not clear.

Limitation of study: Owing to the tertiary location of the research center, the small number of patients and the lack of the latest techniques, we have limited findings and results.

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