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Original Research Article

Association of Metabolic Syndrome with Coronary Artery Disease in Andhra Pradesh Population

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Conflict of interest: Nil

Abstract:

Background: Metabolic syndrome is defined to investigate the cardiovascular effects of metabolic syndrome.

Method: 150 metabolic syndrome patients having positive angiography of CAD was compared with 150 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.

Results: 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, 23 (15.3) MS groups had type-II DM, and 120 (80%) HTN were noted, and 12 (8%) had type-II DM, and 18 (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: In the present pragmatic study there is a strong correlation of metabolic syndrome (MS) with coronary artery disease (CHD). It will help the clinician to treat such patients efficiently to avoid morbidity and mortality.

Keywords: Metabolic Syndrome, Coronary Artery Disease, Inflammatory Markers, Insulin Resistance, Lipid Profile.

Introduction

Ever since metabolic syndrome was first described by Reaven in 1988, and later on, the National Cholesterol Education Program's (NCEP) definition of metabolic syndrome is to investigate the cardiovascular effects of the metabolic syndrome.

Globally, the metabolic syndrome (MS) is a major health problem associated with morbidity and mortality because it is associated with coronary artery disease [1]. Metabolic syndrome (MS) represents a host of metabolic abnormalities, including glucose intolerance, dyslipidemia, hypertension, hyperinsulinemia, and abdominal obesity, which predispose an individual to coronary artery disease (CAD) [2].

As per the WHO prediction report, by 2030 India will have over 79 million diabetic subjects, which will contribute to more than 20% of the world diabetic population [3]. However, genetic factors 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 artery disease.

Material and Methods: 150 (one hundred fifty) adult patients aged between 26 to 60 years regularly visited to Kurnool Medical College Kurnool, Andhra Pradesh-518002 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 (150) 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), Interleukin-6 (IL-6), and high-sensitivity C-reactive protein (HS CRP) were measured by the enzyme-linked immunosorbent assay method with kits manufactured by Gen-Probe Diaclone, France, and Bio-Check CA, USA. Insulin estimation was done by micro particle 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 study was January 2023 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.

Observation and Results

Table 1: Comparative study of biochemical analysis in metabolic syndrome and non-metabolic (NMS) syndrome

- TG: 8 (± 1.6) in MS group, 168.3 (± 1.68) in non-MS group, t test was 55.4 and p<0.001
- HDL: 06 (± 5.54) in MS, 40.5 (± 8.30) in Non-MS, t test 6.6 and p<0.001.
- VLDL: 8 (± 7.8) in MS group, 32.8 (± 0.30) in non-MS group, t test 7.9 and p<0.001.
- LDL – 05 (± 9.4) in MS group, 98.6 (± 1.28) in non-MS group, t test 9.6 and p<0.001

Table 2: Comparison of clinical manifestations in both groups

- Type-II DM: 23 (15.3%) in MS group, 12 (8%) in non-MS group.
- Hypertension: 120 (80%) in the MS group, 18 (12%) in the non-MS group

Table 3: Comparison of anthropometric biochemical and inflammatory markers

- Insulin M4/L: 54.2 (± 3.6) in MS group, 17.8 (± 2.2) in non-MS group, t test 10.5 and p<0.001.
- IL-6 (interleukin-6): 36.63 (± 9) in MS group 12.11 (± 0.4) in non-MS group, t test was 30.4 and p<0.001
- TNF-α (pg/ml): 13.2 (± 0.7) in MS group, 7.32 (± 0.2) in non-MS group, t test 9.89 and p<0.001.
- HS CRP (Mg/L): 15.8 (± 0.8) in MS group, 3.3 (± 0.2) in non-MS group, t test 18.5 and p<0.001
- HOMA-IR: 18.8 (± 0.58) in MS group, 5.4 (± 0.3) in non-MS group, t test was 25.1 and p<0.001.
- QUICKI: 0.28 (± 0.3) in MS group, 0.30 (± 0.4) in non-MS group, t test 0.95, and p>0.33 (p value is insignificant).
- Single vessel disease: 20 (± 4) in MS group, 12 (± 2.2) in non-MS group, t test 24.1 and p<0.001.
- Double vessel disease: 24 (± 8.4) in MS group, 15 (± 4.4) in non-MS group; t test was 11.6 and p<0.001.
- Triple vessel disease: 30 (± 3.8) in MS group, 19 (± 4.8) in non-MS group, t test 2.8 and p<0.001.
- BMI: 27.4 (± 2) in MS group, 26.5 (± 3.2) in non-MS group, t test 2.8 and p<0.001.

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

Parameters	MS group (150)	Non-MS group (150)	t test	p value
TG	178.8 (± 1.6)	168.3 (± 1.68)	55.4	P<0.001
HDL	35.06 (± 5.54)	40.5 (± 8.30)	6.6	P<0.001
VLDL	38.8 (± 7.8)	32.68 (± 0.30)	9.7	P<0.001
LDL	106.08 (± 9.4)	98.6 (± 1.28)	9.6	P< 0.001

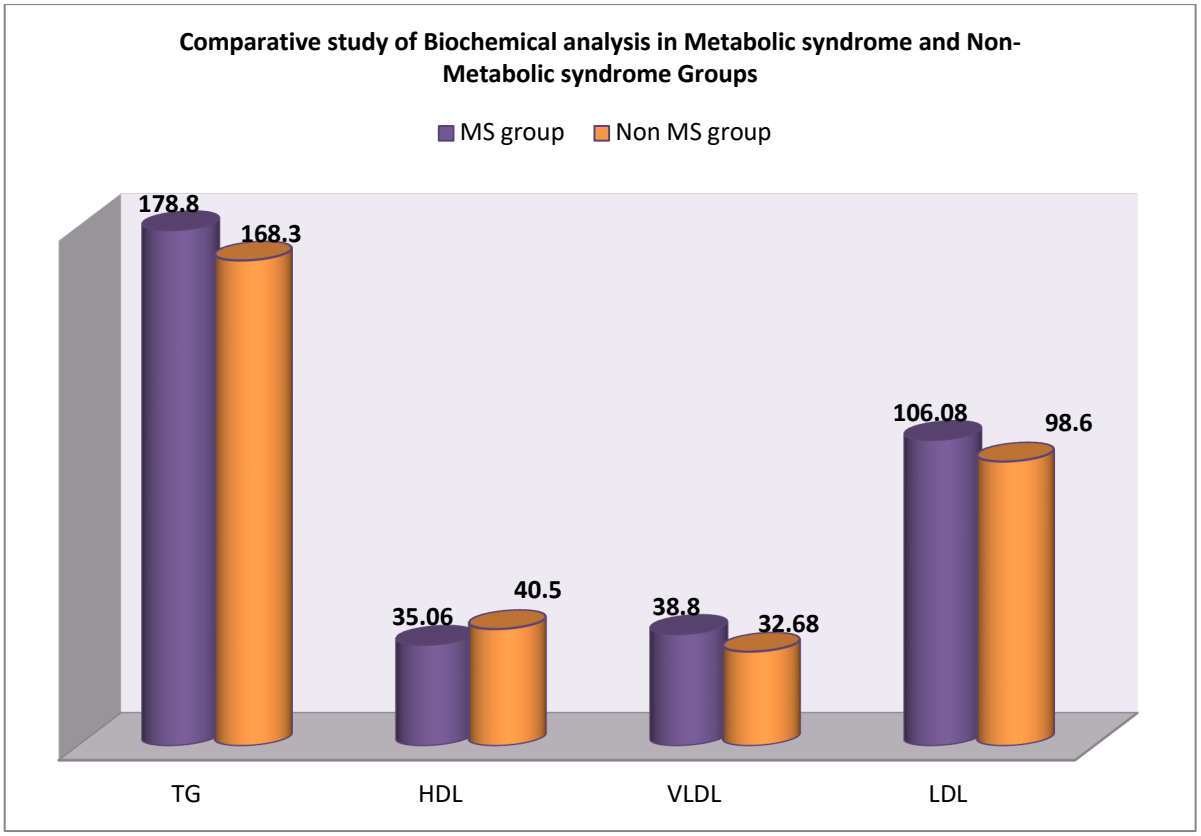

Figure 1: Comparative study of Biochemical analysis in Metabolic syndrome and Non- Metabolic syndrome Groups

Table 2: Comparison of clinical manifestations in both groups

Clinical Manifestation	MS group (150)	Percentage (%)	Non-Ms Group (150)	Percentage (%)
Type-II DM	23	15.3 %	12	8 %
Hypertension	120	80 %	18	12 %

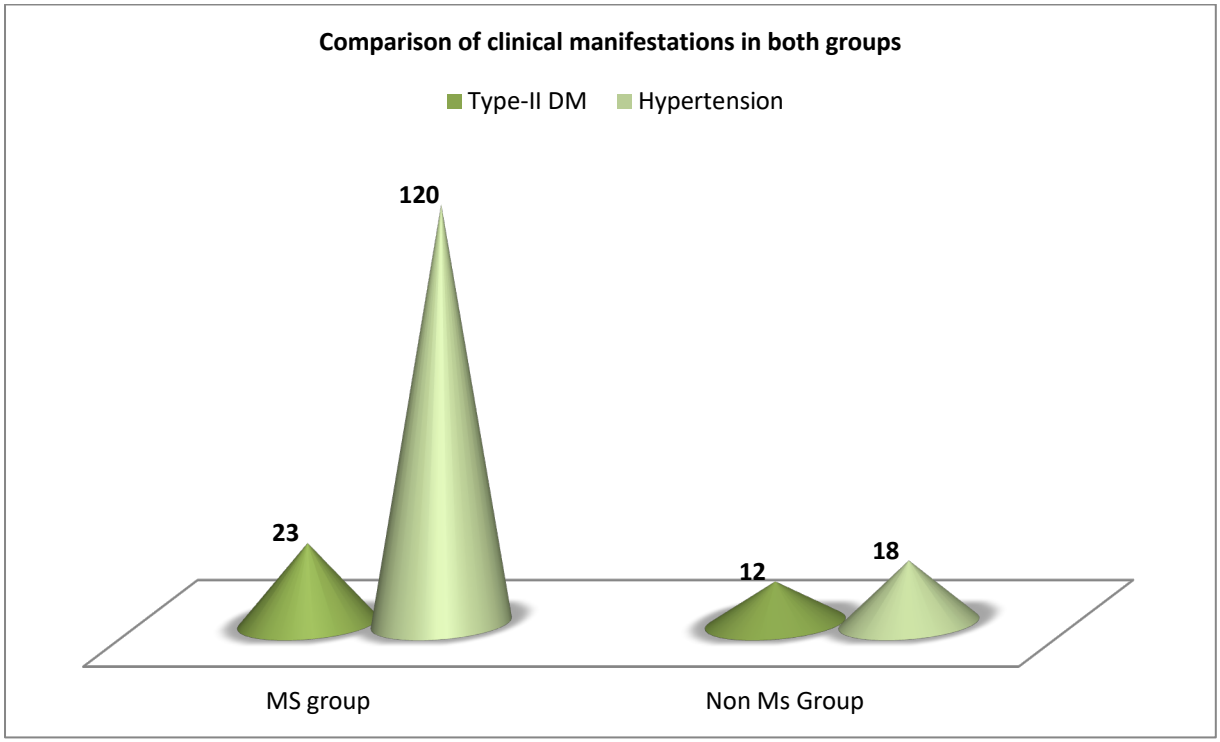

Figure 2: Comparison of clinical manifestations in both groups

Table 3: Comparison of Anthropometric and Biochemical and inflammatory markers

Parameters	MS group (150)	Non-MS group (150)	t test	p value
Insulin m4/L	54.2 (± 3.6)	17.8 (± 2.2)	10.5	P<0.001
IL-6 (Interleukin-6) pg/ml	36.63 (± 0.9)	12.11 (± 0.4)	30.4	P<0.001
TNF-α (pg/ml)	13.2 (± 0.7)	7.32 (± 0.2)	9.89	P<0.001
Hs CRP (mg/L)	15.8 (± 0.8)	3.3 (± 0.2)	18.5	P<0.001
HOMA-IR	18.8 (± 0.58)	5.4 (± 0.3)	25.1	P<0.001
Quicki	0.28 (± 0.3)	0.30 (± 0.4)	0.95	P>0.33
Single vessel Disease	20 (± 3.4)	12 (± 2.2)	24.1	P<0.001
Double Vessel Disease	24 (± 8.4)	15 (± 4.4)	11.6	P<0.001
Triple vessel Disease	30 (± 3.8)	19 (± 4.8)	22	P<0.001
BMI	27.4 (± 2.2)	26.5 (± 3.2)	2.8	P<0.001

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

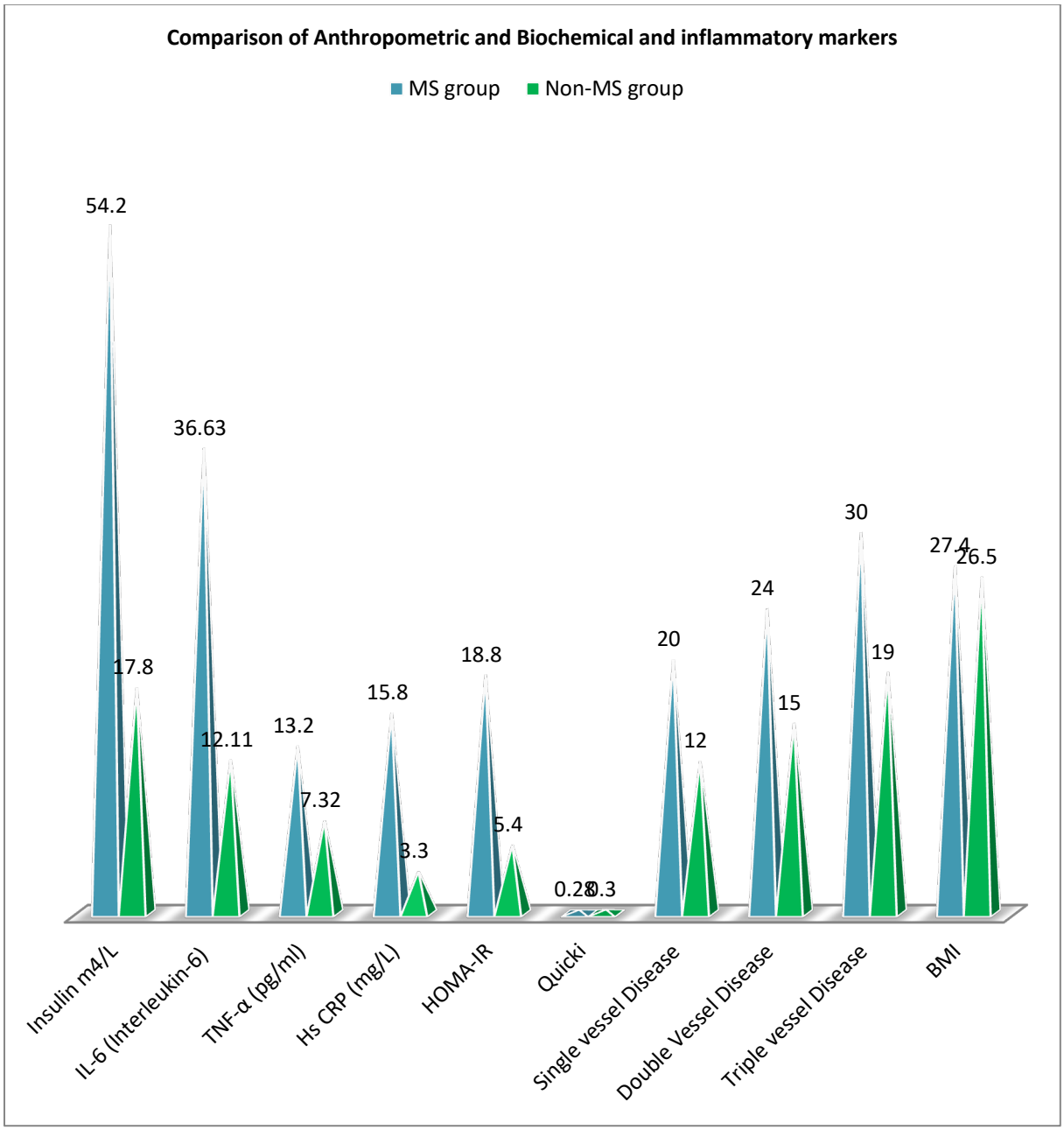

Figure 3: Comparison of Anthropometric and Biochemical and inflammatory markers

Discussion

Present study of Association of Metabolic syndrome in Andhra Pradesh population. In comparative study of Biochemical analysis TG, HDL, VLDL and LDL has significant p value (Table-1). In comparison of clinical manifestation in both groups (MS and Non-MS) type-DM were 23 (15.3%) and hypertensive were 120 (80%) in MS group, but in Non-Ms 12 (8%) type-II DM and 18 (12%) hypertensive (Table-2). In comparison of Anthropometric, biochemical and inflammatory markers had significant p value (Table 3). These findings are more or less in agreement with previous studies [6,7,8].

It is an established fact that insulin resistance is the dominant cause of the syndrome. Hence it prefers to term as "Insulin Resistance Syndrome". According to 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 patho physiological mechanism by which metabolic syndrome increases cardio vascular risk remains under debate because many studies have reported that insulin has independent role underlying component of metabolic syndrome. Insulin resistance progresses towards hyper insulinemia and hyper glycemia 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 (Met-S) carries increased long term risk for atherosclerosis, cardio-vascular diseases and DM-II as well.

It is important to note that though the Met-S 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 Met-S, life style and proper diet should be introduced apart from drug therapy.

Summary and Conclusion

Met-S 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.

This research work paper was approved by the ethical committee of Kurnool Medical College, Kurnool, and Andhra Pradesh-518002.

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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