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PREVALENCE OF CARDIOVASCULAR RISK FACTORS AMONG PATIENTS WITH METABOLIC SYNDROME- A CASE CONTROL STUDY IN SOUTH INDIA

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ABSTRACT

Background: Cardiovascular disease is a significant concern in patients with metabolic syndrome. These patients have a higher risk of developing various cardiovascular conditions, such as heart disease, stroke, and peripheral artery disease. This increased risk is due to the combination of factors associated with metabolic syndrome, including obesity, high blood pressure, high cholesterol levels, and insulin resistance. Furthermore, the presence of type 2 diabetes mellitus in patients with metabolic syndrome further exacerbates the risk of cardiovascular disease. Research has shown that individuals with metabolic syndrome have a twofold to fourfold increased risk of developing cardiovascular disease compared to those without metabolic syndrome.The underlying mechanisms linking metabolic syndrome and cardiovascular disease are complex and multi factorial. We sought to determine whether the prevalence of the cardiovascular risk factors in patients with metabolic syndrome.

Materials and Methods: In the present study 50 patients attended to the OPD of Government general hospital, Eluru, and were diagnosed as metabolic syndrome and 50 healthy, age matched controls were included. The present study was conducted from June 2026 to August 2026. The assay was performed using the commercial kits.

Results: Patients with metabolic syndrome had significantly higher levels of Fasting blood glucose, serum triglyceride levels and blood pressure where as the serum HDL levels were lower. Central obesity, impaired glucose tolerance and hypertriglyceridaemia were the predominant components of metabolic syndrome. Patients with Metabolic syndrome had significantly elevated levels of Fibrinogen ($p<0.001$) and Homocysteine ($p<0.05$) compared to the control group whereas no difference was observed the levels of Lp (a) and Uric acid. Hyperfibrinogenaemia (odds ratio 90.75, $p<0.001$) and hyperhomocysteinamia (odds ratio 3.00, $p= 0.015$) were found to be associated with metabolic syndrome.

Conclusion: In conclusion, addressing the underlying risk factors associated with metabolic syndrome, such as obesity, insulin resistance, physical inactivity, and dyslipidemia, is plays a pivotal role of risk of cardiovascular disease in metabolic syndromepatients. Therefore, healthcare professionals should targeted interventions that focus on reducing cardiovascular risk factors, such as obesity, hypertension, and hyperglycemia, are crucial in preventing or slowing the progression of cardiovascular disease in patients with metabolic disorders.

Keywords: CVD, Metabolic syndrome, Obesity, Homocysteine, Cholesterol.

INTRODUCTION

Cardiovascular disease is a significant concern in patients with metabolic syndrome. These patients have a higher risk of developing various cardiovascular conditions, such as heart disease, stroke, and peripheral artery disease.[1] This increased risk is due to the combination of factors associated with metabolic syndrome, including obesity, high blood pressure, high cholesterol levels, and insulin resistance. Furthermore, the presence of type 2 diabetes mellitus in patients with metabolic syndrome further exacerbates the risk of cardiovascular disease. Research has shown that individuals with metabolic syndrome have a twofold to fourfold increased risk of developing cardiovascular disease compared to those without metabolic syndrome. The underlying mechanisms linking metabolic syndrome and cardiovascular disease are complex and multifactorial.[2-4] These include chronic inflammation, oxidative stress, endothelial dysfunction, and dyslipidemia. Additionally, lifestyle factors such as poor diet, sedentary behavior, and smoking contribute to the development and progression of cardiovascular disease in patients with metabolic syndrome. Therefore, it is crucial for healthcare professionals to closely monitor and manage cardiovascular risk factors in patients with metabolic syndrome. Additionally, there is ongoing research exploring the role of micro RNAs in the development and progression of cardiovascular disease in patients with metabolic disorders. These patients have an increased risk of developing various cardiovascular conditions, and close monitoring and management of cardiovascular risk factors are crucial for their overall health.[4-6]

Patients with metabolic disorder syndrome are at a higher risk of developing cardiovascular disease, including heart disease, stroke, and peripheral artery disease. These patients have a twofold to fourfold increased risk compared to those without metabolic syndrome. The underlying mechanisms linking metabolic syndrome and cardiovascular disease involve chronic inflammation, oxidative stress, endothelial dysfunction, and dyslipidemia.[7] In addition to lifestyle factors, such as poor diet and sedentary behavior, other factors associated with metabolic syndrome, such as obesity, high blood pressure, and hyperglycaemia, contribute to the development and progression of cardiovascular disease. Therefore, effective management and control of these risk factors are crucial in reducing the incidence and burden of cardiovascular disease in patients with metabolic syndrome. In patients with metabolic disorder syndrome, the risk of developing cardiovascular disease is significantly higher compared to those without metabolic syndrome.[8]These patients have multiple risk factors including chronic inflammation, oxidative stress, endothelial dysfunction, dyslipidemia, poor diet, sedentary behavior, and smoking. It is essential for healthcare professionals to closely monitor and manage these risk factors in order to prevent and mitigate the development of cardiovascular disease in this population. Additionally, ongoing research is exploring the role of micro RNAs in the development and progression of cardiovascular disease in patients with metabolic disorders.

This research aims to uncover the molecular mechanisms involved in the interplay between metabolic disorders and cardiovascular disease. Furthermore, targeted interventions that focus on reducing cardiovascular risk factors, such as obesity, hypertension, and hyperglycemia, are crucial in preventing or slowing the progression of cardiovascular disease in patients with metabolic disorders.

MATERIALS AND METHODS

The present case control study was done with 50 patients (n=50), who were attended to OPD of Government general hospital of Eluru, and were diagnosed as metabolic syndrome and were included after informed consent and compared with 50 (n=50) age and gender matched controls. The study was conducted from June 2026 to August 2026. A total of 100 participants were included into the study. patients (n=50),(males were n=29 and females were n=21), andcontrol group (n=50)(males were n=35 and females were n=15)

Inclusion Criteria: Patients with Fasting blood glucose was $\geq$ 110 mg / dL; Waist circumference (as defined for Asians)>90cms (men), >80cms (women); Triglycerides- □ 150 mg/dL; HDL cholesterol <40 mg/dL (men), <50 mg/dL (women); Blood pressure -□ 130 / 85 mmHg.

Exclusion Criteria: Patients who were having habit of smoking, alcoholism, lipid lowering drugs, vitamin supplements, hormone replacement Infections, abnormal renal function, Malignancy, Lipemic and Icteric samples were excluded from the study.

Sample collection and biochemical analysis:

5ml of venous blood sample from the patients were collected in two separate bulbs (2 ml in EDTA for fibrinogen estimation and 3ml in plain bulb for other parameters). The sample was then centrifuged at 2000 rpm for 20 minutes. The separated plasma was stored at -80^{0}C until further analysis. Analysis of various parameters was done using different methods. They are for estimation of glucose, cholesterol, and triglycerides, used oxidase peroxidase method, for HDL cholesterol used Immunoinhibition method, for Fibrinogen estimation used Immunoturbidometric method, for Uric acid used uricase method, for Lipoprotein (a) estimation, used Agglutination, turbidometric method and to find out the elevated levels of homocystiene, used enzyme linked immunosarbent assay method.

Statistical Analysis

All values were expressed as mean ± SD. Student's unpaired two-tailed t-test were used to test the significance of difference in means between controls and study group. Binary logistic regression was used to know the association between parameters. P value <0.05 was considered as statistically significant. Statistical analysis was done using SPSS ver16.0.

RESULTS

Patients with metabolic syndrome had a higher systolic and diastolic blood pressures compared to the control group. [Table: 1] The average age of the patients was 44.94 ± 13.11 and the average age of control group was 39.89 ± 12.79

The average age of the patients was 44.94 ± 13.11 and the average age of control group was 39.89 ± 12.79. Similarly, metabolic syndrome patients had a statistically significant higher waist circumference both in males as well as females when compared to the control group. Among the other components of metabolic syndrome, patients with metabolic syndrome had significantly higher fasting blood glucose (p= 0.001), higher serum total cholesterol (p=0.016), serum triglycerides (p=0.001) and lower HDL Cholesterol (p=0.015) levels compared to the control group. Of the components of metabolic syndrome, increased fasting blood sugar levels (82%) central obesity (89% in males and 94% in females) and hypertriglyceridaemia (72%) were the predominant features when compared to hypertension (50%) and lower HDL levels (16% in males and 28% in females). The scatter plots presented in Fig-1 show the distribution of all the components of metabolic syndrome in patient and control groups.

Table: 2 shows the levels of novel risk factors i.e. Fibrinogen, Homocysteine, Lipoprotein (a) and Uric acid in the Patient group and the control group. As shown in the Table:7, Patients with metabolic syndrome had increased Fibrinogen levels (587.66±296.12 mg/dL) compared to the controls (145.84 ± 72.48 mg/dL). The difference was found to be statistically significant (p=0.001). Similarly, metabolic syndrome patients had increased Homocysteine levels (16.42 ± 8.94 mg/dL) compared to the control group (13.11 ± 4.33 mg/dL). The statistically significant difference was found (p=0.026). However, no significant difference was found in the levels of Lipoprotein (a) (p=0.602) and uric acid (p=0.915) among the Patient group and control group. This shows that among the novel risk.

Table 1: Metabolic syndrome components in Patient & Control groups

Parameter	Patientgroup(n=50) (Mean±SD)	Controlgroup(n=50) Mean± SD)	Pvalue
SBP(mmof Hg)	132.64±17.86	118.54±10.51	0.001*
DBP (mmofHg)	85.16±8.90	74.37±6.81	0.001*
Waist circumference(cm)	M:99.93±13.83	M:83.93±15.60	0.001*
	F:104.76 ± 20.20	F:80.65.67	0.001*
FBS(mg/dl)	170.28±72.72	92.04±16.14	0.001*
TCHOL (mg/dl)	197.97±48.74	175.15±38.41	0.016*
HDL-C(mg/dl)	M:44.86± 6.86	50.05±7.92	0.015*
	F:49.11± 1.61	50.21±2.86	0.189
TGL (mg/dl)	260.19±156.06	141.56±61.76	0.001*

N= number of patients and controls, *Statistically significant, SBP- systolic blood pressure, DBP-diastolic blood pressure, FBS-fasting blood sugar, T CHOL= Total Cholesterol, HDL-C-HDL Cholesterol, TGL-Triglycerides

Table 2: Novel cardiovascular risk factors in Patient & Control groups

Parameter	Patientgroup (Mean±SD)	Controlgroup(Mean± SD)	P-Value
Fibrinogen(mg/dL)	587.66±296.12	145.84±72.48	0.001*
Homocysteine(µmol/L)	16.42±8.94	13.11±4.33	0.026*
Lipoprotein(a)(mg/dL)	15.65±13.86	14.31±9.33	0.602
Uricacid(mg/dL)	4.34±1.16	4.37±1.18	0.915

Statistically significant

The Prevalence of the novel rsik factors studied in the patient population is shown in Figure: 6. hyperfibrinoginemia and hyperhomocysteinemia were found to be 66% and 48% respectively as against only 10% and 2% for Lp(a) and Uric acid respectively. The scatter plots presented in Figure: 2 show the distinct distribution of higher Fibrinogen and Homocysteine levels in the patient group when compared to control group.

The association between marker positivity and disease was assessed by logistic regression analysis (Table-3). Association was assessed taking into account the upper limit of the reference range as the cut off value. Plasma Fibrinogen and Homocysteine levels were positively associated with metabolic syndrome. No significant association was found with Lp (a) and Uric acid.

Table 3: Logistic Regression analysis value

Parameter	Cut off	Regression coefficient (B) ± SEM	Exp(B) Oddsratio	95%CI	P-Value
Fibrinogen■	**400 mg/dL**	**4.808± 1.056**	**90.750**	**11.450– 719.241**	0.001*
Homocysteine■	**15 µmol/L**	**1.099±0.452**	**3.000**	**1.236– 7.279**	0.015*
Lp(a)■	**30mg/dL**	**0.593±0.742**	**1.810**	**0.423– 7.743**	0.423
Uricacid■	**7.0mg/dL**	**-0.042±1.429**	**0.959**	**0.058– 15.782**	0.977
W.C:M*	**90cms**	**19.480±40192.976**	**288477684.444**	**0.000**	1.000
W.C:M#	**90cms**	**1.253±1.188**	**3.500**	**0.341-35.898**	0.292
W.C:F*	**80cms**	**17.706±28420.721**	**48953783.277**	**0.000**	1.000
W.C:F#	**80cms**	**-0.236±0.993**	**0.789**	**0.113-5.528**	0.789
Sex*		**0.539±1.430**	**1.714**	**0.104-28.275**	0.706
Sex#		0.807±710	2.241	0.558-9.008	0.255

Statistically Significant

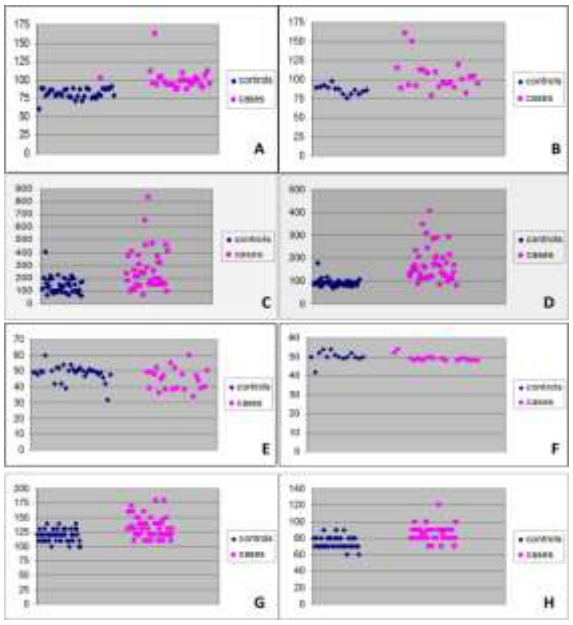

Figure 1: Scatter plots of components of metabolic syndrome

A. Waist Circumference (Males), B. Waist Circumference (Females), C. Triglycerides, D. Fasting Glucose, E. HDL (males), F. HDL(females), G. Systolic Blood Pressure, H. Diastolic Blood Pressure

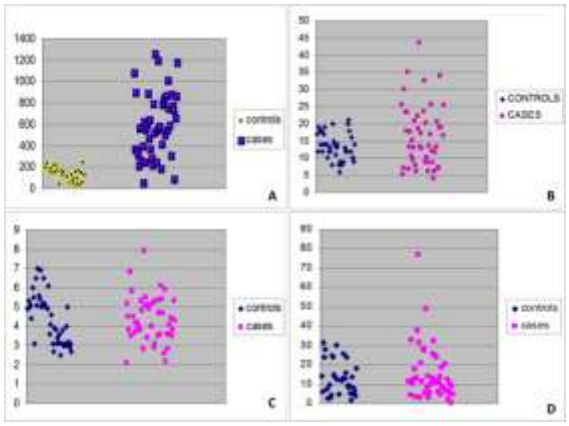

Figure 2: Scatter plots of novel risk factors studied

A. Fibrinogen, B. Homocysteine, C. Uric Acid, D. Lp (a)

DISCUSSION

Each of the components of the metabolic syndrome is an independent risk factor for development of CAD. Regardless of the level of LDL cholesterol and other risk factors, MS confers an increased risk for CAD development.[1-2] The ATP III recognizes that CAD risk is influenced by two groups of risk factors: the first includes those that are related to lifestyle including physical inactivity, and atherogenic diet and obesity. The second group includes high levels of Lp(a), Hcy, prothrombotic, and proinflammatory state.[3]Some of these risk factors are modifiable with simple precautions, while others are not.

Metabolic syndrome is diagnosed based upon the presence of at least three of the five risk factors as per the NCEP-ATP III guidelines. In the present study elevated fasting glucose, hypertriglyceridemia and central obesity were found to predominate in metabolic syndrome patients compared to controls. The combination of these three risk factors also predominated compared to the triad of elevated fasting glucose, hypertension and increased waist circumference. Prevalence of decreased HDL levels was found to be lower. Impaired glucose tolerance, hypertriglyceridemia and central obesity are the key features that characterize metabolic syndrome in south Indian population. The clustering of insulin resistance, central obesity, hypertension and atherogenicdyslipidemia has been to shown to predispose Asian Indians to increased cardiovascular risk.[4-7] The Chennai Urban Population Study showed that polymorphisms in the fatty acid binding protein-2 and apo-III genes to be associated with MS in south Indian population. They suggested that these polymorphisms result in hypertriglyceridemia through their concerted action of increased chylomicron production and decreased lipolysis/clearance of TRL.[8]

In The Chennai Urban Population Study" MS diagnosed on the basis of ATP III, M. Deepa et al, found only 20.9% of the individuals to have impaired fasting glucose (IFG and DM were taken together in the present study). Abdominal obesity was present in 18.5% of the cases.[8] The major components in their study were hypertriglyceridemia (25.2%)and low HDL levels(63.5%). Achari V et al, reported

hypertriglyceridemia followed by obesity and hypertension in metabolic syndrome patients with type 2 Diabetes mellitus from Patna, East of India. Low HDL was the least common finding.[9] The findings are similar to the present study, however the reported percentage was lower compared to the present study. The difference could possibly be due to regional variations and lower sample size in the present study.

A number of cross sectional and case-control studies have identified other biochemical cardiovascular risk markers with statistically significant association with metabolic syndrome. This especially concerns Homocysteine and Fibrinogen.[10]In this study, the prevalence of the novel risk markers i.e. Fibrinogen, Homocysteine, Lp (a) and Uric acid in Metabolic syndrome patients was studied. It was observed that there was significant difference (P<0.05) in plasma Fibrinogen and Homocysteine levels in Metabolic syndrome patients compared to controls. However, no significant difference was observed with plasma Lp(a) and uric acid levels.

Fibrinogen is considered to be a novel risk marker for metabolic syndrome. In the present study, a significant increase in plasma fibrinogen levels (p=0.001) in Metabolic syndrome patients compared to controls (587.66±296.12 mg/dl in cases vs. 145.84 ± 72.48 mg/dL in controls) was observed. This is in line with those reported by Imperatore G et al, and the National Health and Nutrition Examination Survey.[10-11] In this survey (1988-1994) participants with the metabolic syndrome had higher fibrinogen concentrations and white blood cell counts than those without this syndrome. Onat A et al observed that plasma Fibrinogen predicts metabolic syndrome independent of its components in men but not in women.[12]Fibrinogen is an acute phase reactant. Elevated Fibrinogen levels represent low grade inflammation in patients with metabolic syndrome. The acute manifestations of CAD such as acute myocardial infarction are triggered by complications of atherosclerotic plaque—thrombosis, occlusion, or rupture because of alteration in the delicate balance between fibrinogenesis and fibrinolysis.[13-14] Fibrinolysis breaks down fibrin and maintains vessel patency, and in tissues it breaks down the extracellular matrix and controls cell adhesion and migration thereby participating in tissue remodeling.Plasminogen activator inhibitor type-1 (PAI-1) regulated the Fibrinolysis which prevents the escape of this potentially destructive protease system.[14,15] High levels ofplasma PAI-1 levels have been linked with the severity of Metabolic syndrome.[16] Increased PAI-1 levels may predispose patients to the formation of atherosclerotic plaques prone to rupture with a high lipid-to-vascular smooth muscle cells ratio as a result of decreased cell migration.[17]Hypertriglyceridaemia seen in these patients impair fibrinolysis and also hyperglycemia is known to induce coagulation, further altering the hemostatic function.[16-17]

Lohsoonthorn Vet al, reported that from the lowest to the highest quartile of level of plasma fibrinogen, the prevalence of metabolic syndrome (39%, 46%, 47%, and 64%) also increased. (18) The present study found elevated levels of fibrinogen in 62 % of the patients with metabolic syndrome (Figure:2).

The association between marker positivity (value above cut off) and presence of metabolic syndrome was assessed using logistic regression analysis. With the cut off value taken at the upper limit of the reference range (≥ 400 mg/dL) a significant odds ratio of 90.750 (95% CI: 11.450-719.241, p=0.001) was observed indicating a definite occurrence of increased fibrinogen levels in patients with metabolic syndrome.In accordance with this finding, Onat A et al,observed that plasma fibrinogen in men independently predicted metabolic syndrome, but not in women.[18] In a population based study, Bruno G. et al, reported that fibrinogen increases with age, HbA1c, smoking, hypertension and a number of components of the metabolic syndrome, independent of major confounders.[19] In another study assessed the levels of plasma fibrinogen and other markers of metabolic syndrome in type 2 Diabetes mellitus and found that though the levels of fibrinogen were elevated they were not different among those with or without metabolic syndrome.[20]

Hyperhomocysteinemia is another novel risk factor for atherosclerosis. High homocysteine concentrations are thought to cause endothelial damage, inducing atherosclerosis.[21] The relationship between elevated serum homocysteine levels and CAD has been established.[22] However, reports on the relationship between hyperhomocysteinemia and metabolic syndrome are not well established.

The present study observed significant difference (P=0.026) in homocysteine levels in patient group when compared to controls. The homocysteine levels were 16.42±8.94µmol/L in patients as compared to 13.11 ± 4.33µmol/L in the controls. This is in agreement with Hajer et al, and Guven A et al, who found increased plasma homocysteine in metabolic syndrome patients compared to those who did not have metabolic syndrome.[23-24] However, Soysal D et al, and Jermendy et al, found homocysteine levels to be similar in patients with Metabolic syndrome compared to controls, though they found an increased incidence of hyperhomocysteinemia in CAD patients. In Framingham Offspring Study, increased total homocysteine levels were shown to increase the risk of cardiovascular disease only in the presence of abnormal proteinuria in patients with insulin resistance.[25-27]

The findings of the regression analysis to assess the association between hyperhomocysteinaemia and metabolic syndrome were similar to that of fibrinogen. Taking the upper limit of reference range as the cut off (15 µmol/L) for homocysteine, a significant odds ratio of 3.0(95%CI 1.236-7.279 p =

0.015) was observed. This is in agreement with Bellia C et al. other authors have studied the relationship of homocysteine with individual components of metabolic syndrome.[28]Budak et al, in their study found that Hcy level was negative and weakly correlated with insulin resistance in females and negative strongly correlated with SBP in males. No associations were found between Hcy level and other components of metabolic syndrome in their study. They found that 7.7% of adolescents had homocysteine levels of ≥ 15 micromol/L.[29]

In the present study, hyperhomocysteinemia was present in almost 50% of the patients with metabolic syndrome (Figure:2). Similarly Bellia C et al, reported a prevalence of 67.2% of hyperhomocysteinemia in their study.[28]

Homocysteine levels are influenced by a number of variables such as smoking, renal function, enzyme dysfunction states. Patients with history of smoking and all the patients with abnormal renal function were excluded from the present study. However, Vitamin B 12 status and enzyme activities were not evaluated in our study. Low levels of vitamin B12 have been reported in metabolic syndrome.[24]Folate and vitamin B12 treatment improve insulin resistance and endothelial dysfunction, along with decreasing homocysteine levels, in patients with metabolic syndrome.[30] Also, plasma levels of insulin also seem to influence homocysteine metabolism, possibly through effects on glomerular filtration or by influencing activity of key enzymes in homocysteine metabolism, including 5,10-methylenetetrahydrofolate reductase (MTHFR) orcystathione b-synthase (CBS).[31-32] At the same time, hyperhomocysteinemia can induce insulin resistance, leading to compensatory hyperinsulinemia, which may impair activity of the MTHFR and CBS enzymes, leading to accumulation of homocysteine in plasma. Therefore, insulin resistance and hyperhomocysteinemia may create a deleterious feedback loop, each promoting the development and propagation of the other.[33]

Lipoprotein(a), a macromolecule consisting of a particle of low-density lipoprotein (LDL) linked to apolipoprotein (a), is highly homologous with plasminogen, so the characteristics may contribute to the processes of atherothrombosis. Serum Lp(a) level is considered an independent risk factor for atherosclerotic events such as cardiovascular and cerebrovascular disease.[34] Similarly, metabolic syndrome (MS) has recently been considered a risk factor for atherosclerosis and its sequelae such as cardiovascular and cerebrovascular diseases. The Lp(a) might promote atherosclerosis under presence of other atherogenic pathologies and conditions.

Data regarding Lp(a) levels in patients with MS are very limited and contrasting. In the present study we observed no significant difference (p=0.363) in Lp(a) levels in patient group when compared to controls. The Lp(a) levels were 15.65±13.86mg/dl in Patient group and 14.31 ± 9.33 mg/dl in control group. This is in contrast to those reported by GuvenA et al and Huseyin B et al. Guven A et al found higher concentration of Homocysteine and Lp(a) levels in Turkish patients with metabolic syndrome.[35] Similarly, Huseyin B et al found that patients having metabolic syndrome had significantly higher Lp(a) levels than did patients without this syndrome.[36]

Some studies have evaluated the association between Lp(a) and obesity. Studies have revealed that levels of Lp(a) are higher in obese patients than those in the non-obese subjects [37, 38], while others have shown no such association. (39) However, no association was found with waist circumference in the present study [(M: odd's ratio: 3.500, 95%C.I.: 0.341-35.898, p= 0.292), (F: odd's ratio: 0.789, 95% C.I:0.113-5.528, p=0.789)]. Factors such asgenetic and environmental influences including dietary habits have been proposed to play role in affecting Lp(a) levels in these studies. While renal failure, inflammatory conditions and growth hormone therapy have been proposed as the non-genetic causes of increased Lp(a) levels. The differential results in the present study with respect to Lp(a) and uric acid needs further probing.

Uric acid is yet another newly described coronary risk factor. Studies have shown that high uric acid levels are associated with increased cardiovascular disease and mortality.[40,41] In the present study no significant difference in uric acid levels were found between the patient group and control group. This is in agreement with Huseyin B et al,[36] who found similar levels of uric acid in patients with and without metabolic syndrome. Serum uric acid levels have been found to be associated with BMI, waist/hip ratio, fasting insulin, serum triglycerides, LDL, and diastolic blood pressure in healthy men and women.[42] It has been considered to be an indicator of pre-metabolic syndrome in obese youth.[43] They found that uric acid levels correlated with metabolic syndrome.

Only 2% of the patients with metabolic syndrome had elevated levels of uric acid in the present study. Chen LY et al in a study reported higher prevalence of hyperuricemia in a Chinese population.[44] Similarly Rho YH et al, in Korean subjects found that hyperuricemic subjects tended to have a higher prevalence of the metabolic syndrome and more metabolic abnormalities than subjects with normal levels of uric acid.[45] In a Japanese study by Kawamoto R et al, the prevalence of metabolic syndrome was significantly increased according to serum uric acid values only in women. In men without metabolic syndrome, uric acid levels were found to be an independent risk factor for incidence of carotid atherosclerosis. However, no significant association was found between uric acid levels with either sex in the present study.[46] Similarly, uric acid levels were also not associated with waist circumference in neither males nor females.

However, increasing serum uric acid levels even in the normal range have been shown to be associated with increased CV risk. In our study also, we found

that the uric acid levels of patients were scattered at a higher level than control group as shown in Fig-1. Regression analysis showed no significant association between uric acid levels and presence of metabolic syndrome (odds ratio = 0.959; 95%CI: 0.058 – 15.782; p = 0.977). Previous studies have found a positive association between uric acid and MS.[47,18]They found hyperurecemia to be positively associated with metabolic syndrome. See LC et al reported a strong association between hyperuricemia and metabolic syndrome independent of eGFR.[47]

Though elevated uric acid levels have been reported in MS patients we did not observe any change in uric acid levels in MS patients compared to controls. This could probably be due to the dietary habits followed in south India. Staple diet in this region is mostly vegetarian in nature when compared to the nonvegetarian diet followed in the west which is another reason for increased uric acid levels found in other studies. Uric acid levels are affected by ethnicity, age, sex, BMI, renal function. No significant association with sex (odd's ratio: 1.714, 95% C.I: 0.104-28.275, p=0.706) or abdominal obesity [(M:odd's ratio: 288477684.444, 95% C.I : 0.000, p=1.000), F: 48953783.277, 95% C.I : 0.000, p=1.000)]

was found in the present study. No significant association with either uric acid or Lp(a) was found in patients with MS in the present study. Previous studies have found a positive association between uric acid and MS.[47,18,48]They found hyperuricemia to be positively associated with metabolic syndrome. See LC et al, reported a strong association between hyperuricemia and metabolic syndrome independent of eGFR. We did not find any significant association with sex or abdominal obesity.[47]

The results of the present study show significant association of Novel risk factors especially, Fibrinogen and Homocysteine with Metabolic syndrome. Since hyperhomocysteinemia is easily treatable, early identification by means of screening in these patients for these markers can help in decreasing the morbidity and mortality in these patients. Similarly, appropriate measures can be initiated to establish the hemostatic function.

It was observed that metabolic syndrome can also present with other novel risk factors like Homocysteine, Uric acid, Lipoprotein (a) and Fibrinogen. These novel risk factors are likely to have regional and ethnic variations. Hence, evaluation of these markers, which vary with geographic distribution and life style, helps to identify which of these risk factors predominate in a given geographic area. This add to the primary aim of identifying metabolic syndrome i.e., to decrease the cardiovascular morbidity and mortality. The following findings were observed in the present study patients with metabolic syndrome had significantly higher levels of Fasting blood glucose, serum triglyceride levels and blood pressure where as the serum HDL levels were lower. Central obesity, impaired glucose tolerance and hypertriglyceridaemia were the predominant components of metabolic syndrome. Patients with Metabolic syndrome had significantly elevated levels of Fibrinogen ($p<0.001$) and Homocysteine ($p<0.05$) compared to the control group whereas no difference was observed the levels of Lp(a) and Uric acid. Hyperfibrinogenaemia (odds ratio 90.75, $p<0.001$) and hyperhomocysteinamia (odds ratio 3.00, $p= 0.015$) were found to be associated with metabolic syndrome

Homocysteine is toxic to the endothelial cells as well as it has a role in the pathogenesis of insulin resistance in patients with Metabolic syndrome. At the same time, Fibrinogen represents an acute phase reactant and represents the presence of low grade inflammation in patients with metabolic syndrome. This along with the abnormal hemostatic balance as a result of elevated fibrinogen levels can increase the cardiovascular risk of these patients. Both these markers are amenable to correction with simple measures. Hence, correction of hyperhomocysteneimia and modulation of the hemostatic function can play a key role in decreasing the cardiovascular mortality in these patients. Hence routine assessment of these novel cardiovascular risk markers can add to our ability to predict risk over and above those achievable through simple identification and addressing the presence of metabolic syndrome.

CONCLUSION

In conclusion, addressing the underlying risk factors associated with metabolic syndrome, such as obesity, insulin resistance, physical inactivity, and dyslipidemia, is plays a pivotal role of risk of cardiovascular disease in these patients. Therefore, healthcare professionals should continue to prioritize the implementation of targeted interventions that aim to address these risk factors and promote lifestyle changes to prevent or delay the development of cardiovascular disease in individuals with metabolic disorders.

Conflict of Interest: NIL

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