Research Article

A study of serum TG/HDL cholesterol ratio in patients with and without metabolic syndrome

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Abstract

Objectives: To estimate lipid profile in patients selected for the study and to determine the ability of TG/HDL-c ratio in identifying patients with metabolic syndrome. **Methodology:** 290 subjects of age group 20-65yrs, (women=70,men=220) attending General medicine OPD at RRMCH and satisfying inclusion and exclusion criteria were selected for the study. Anthropometric examinations were made and relevant biochemical parameters were analysed. The study subjects were divided into 2 Groups based on TG/HDL-c ratio ≥3 and Group B<3 (Group A and B).Further Revised NCEP ATP III criteria was used to categorise patients into Group 1 MetS +VE and Group II MetS –VE. Statistical analysis was performed using Student's t test and results tabulated. **Results:** The subjects consisted 220 males and 70 females, in the mean age group of 49.33 ± 12.54.Of 290 subjects 178 had TG/HDL-c ratio≥3(Group A) and in 112 subjects had TG/HDL-c of <3 (Group B). The Triglycerides, Fasting blood sugar, Waist circumference were significantly higher in Group A compared to Group B (P Value for each < 0.0001). HDL was significantly lower in Group A than Group B (P Value for each < 0.0001). And blood pressure was not significantly, different between the two groups. Overall 80% of group A patients and 25% of group B patients had metabolic syndrome. According to Revised NCEP ATP III criteria we have devided into 2 groups as MetS +VE Group 1 and Group II MetS –VE. Of the total 290 subjects, 170 were MetS +VE and 120 were MetS –VE. All the components of the cardiovascular risk profile like WC, TG, FBS, were higher in MetS+VE subjects P value 0.0001 and unlike Group A and B Whereas here we found a statistical significance with both SBP and DBP in these groups (1 and II) **Conclusion:** TG/HDL-c ratio can be used as a reliable marker to identify patients with Metabolic syndrome.

Keywords: HDL-C; MetS; TG; TG-to-HDL cholesterol ratio;

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INTRODUCTION

Cardiovascular disease is the number one cause of mortality worldwide, representing about 31% of the deaths globally. According to World Health Report 2002, CVD will be the largest cause of death and disability in India by 2020.[1] Data from several cross sectional studies confirm the high prevalence of cvrisk factors among Indians. Dyslipidemia has been identified as an important and main modifiable factor, which is independently associated with an increased risk of CV events.[2]. Strategies aimed at its early detection provide an outstanding opportunity for reducing the burden of CVD. Timely detection of atherogenic dyslipidemia and hence CVrisk has hence become the cornerstone of CVD prevention. Efforts have been made in identifying markers for early and improved disease prediction. Metabolic syndrome and isolated lipid parameters like LDL are commonly being used as CV risk predictors. Recently, lipoprotein ratios are recognised as being more useful than isolated lipid values for CVD risk assessment, as it reflects more effectively the metabolic and clinical interactions between the different lipid fractions.[3]

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TG/HDL ratio has recently been proposed as a CV risk index due to its association between dyslipidemia and insulin resistance.[4] The association between triglycerides and HDL cholesterol reflected by its ratio depicts the balance between atherogenic and protective lipoproteins known as the atherogenic plasma index. The simultaneous use of triglycerides and HDL cholesterol in this ratio reflects the complex interactions of lipoprotein metabolism overall and can be useful for predicting atherogenicity. It is a simple, inexpensive and readily available calculated measure of cardiovascular risk and simplifies the task of clinicians in identifying subjects with high risk of coronary artery disease.[3] The aim of the present study is to estimate the lipid profile and to determine the TG/HDL ratio and to compare the ability of an elevated TG/HDL-c ratio to identify subjects with high cardio metabolic risk profile with that obtained using diagnostic criteria of MetS using revised NCEP-ATP III guidelines.

MATERIALS AND METHODS

Subjects: The study was conducted in Raja Rajeswari Medical College and Research Hospital, Kambipura, Bangaluru. The study was approved by the Ethical Committee RRMCH-Bangalore. All participants provided written informed consent issued by RRMCH-Bangalore. We enrolled 290 subjects for the study, out of which 70 were women and 220were men .Subjects with acute medical and surgical illnesses and pregnant women were excluded from the study. Fasting blood samples were collected from all the subjects after overnight fasting under full aseptic precautions for estimation of blood glucose and lipid profiles in fully automated analyser. Blood glucose (FBS) was estimated by glucose oxidase-peroxidase method (GOD-POD). Total cholesterol (TC) by cholesterol oxidase peroxidase method, High density lipoprotein (HDL) was estimated by direct method, Triglyceride (TG) was estimated by glycerol 3-phosphate oxidase method. VLDL was calculated by the formula VLDL=TGL/5. LDL was calculated using Freidwald's equation. Medical history was taken and basic physical examination was done. Waist circumference (WC) was measured according to WHO guidelines using a stretch resistant tape after normal expiration, at the midpoint between the lowest rib and the iliac crest. The measurement was made with the tape held snugly, but not constricting, and at a level parallel to the floor. Blood pressure was measured in the sitting position, after a minimum resting period of 5 min, using a mercury sphygmomanometer. Criteria for subject classification: The NCEP ATP III definition is one of the most widely used criteria of metabolic syndrome. It uses measurements and laboratory results that are readily available to physicians, facilitating its clinical and epidemiological application. It is also simple and easy to remember. Importantly, it does not require that any specific criterion be met; only that at least three of the below five criteria are met.[5] Metabolic Syndrome was diagnosed according to the 2005 revised NCEP ATPIII criteria which requires at least 3 of the following components i.e. Waist circumferance $\geq$ 90 cm and $\geq$ 80 cm of men and women respectively, triglycerides $\geq$ 150 mg/dl, HDL $\leq$ 40 and $\leq$ 50 mg/dl in men and women, Blood Pressure $\geq$ 130/85mm/Hg, FBS $\geq$ 100 mg/dl.[6] On the basis of TG/HDL concentration ratios our study subjects were classified into two groups. Group A TG/HDL-c $\geq$3 and Group B TG/HDL-c <3. Further Revised NCEP ATP III criteria was used to categorise patients into Group 1 MetS +VE and Group II MetS-VE.

Statistical Analysis

Values were expressed as Mean±SD. Statistical comparisons were carried out by student 't' test. All statistical analysis was done at 5% level of significance using statistical software SPSS 15.0**.** All analyses were two-tailed, and p values < 0.05 were considered to be statistically significant.

RESULTS

290 subjects were included in the study based on selection criteria out of which, 220 were men and 70were women. Table 1presents the gender distribution of the study population. The mean age for the study group was 49.33 ± 12.54 years. Table 2 compares the cardiometabolic risk profile of subjects with TG/HDL-C ratio $\geq$3 (group A) versus TG/HDL ratio <3 (group B). Except age and blood pressure all the other risk factors were significantly accentuated in subjects with elevated TG/HDL-C ratio $\geq$3.0. 75% of subjects in group A had hypertriglyceridemia, 71% had higher FBS and 56% had low HDL. Table 3 compares the cardiometabolic risk profile of subjects with and without MetS. All the risk factors were significantly accentuated in subjects with MetS+VE (group 1) in comparison to those without MetS-VE(group II),except for HDL. About 76% in group I had high FBS, 71% had hypertriglyceridemia and 60% had low HDL. Table 4. Shows the distribution of metabolic syndrome diagnosed according to the revised ATP III criteria in group A (TG/HDL ratio $\geq$3) and group B (TG/HDL ratio <3). About 80 % of subjects in group A were MetS +VE whereas only 27% of Group B were Mets+ve.

Table 1: Gender distribution of patients studied

Gender	No. of patients	%
Male	220	75.8
Female	70	24.1
Total	**290**	**100.0**

Table 2: Compares the cardiometabolic risk profile of subjects with TG/HDL ratio ≥ 3 (group A) versus TG/HDL ratio <3 (group B)

	≥ 3 (group A) 178	<3 (group B) 112	
Age	49.19 ± 11.69	49.63 ± 13.7	=0.7696
Waist circumference	39 ± 2.8	37 ± 3.6	<0.0001
Triglycerides	217.3 ± 94.4	97.7 ± 32.6	<0.0001
HDL	40.8 ± 7.6	47.6 ± 16.3	<0.0001
FBS	131.3 ± 55.1	109.8 ± 37.9	<0.0003
SBP	130.5 ± 13.9	128.8 ± 15.9	=0.3362
DBP	83.1 ± 7.6	81.8 ± 7.1	=0.1446
TG/HDL-C ratio	5.5 ± 2.7	2.1 ± 0.5	<0.0001

Table 3: Compares the cardiometabolic risk profile of subjects with and without Mets

	MetS +VE (group 1) (170)	MetS –VE (group II) (120)	
W.C.	40.9 ± 2.7	37.3 ± 4.1	=0.0001
TG	206.6 ± 101.0	119.4 ± 58.2	<0.0001
HDL	42.1 ± 12.7	45.5 ± 11.2	=0.0189
FBS	136.6 ± 54.6	103.2 ± 34.8	<0.0001
SBP	133.8 ± 14.8	124.9 ± 13.0	<0.0001
DBP	84.3 ± 7.5	80.2 ± 6.7	<0.0001
TG/HDL-C ratio	5.1 ± 2.9	2.7 ± 1.4	<0.0001

Table 4: The distribution of MetS diagnosed according to the revised NCEP ATP III criteria in group A (TG/HDL ratio ≥ 3) and group B (TG/HDL ratio <3)

	≥ 3.0	<3.0	Total
MetS +VE	142	28	170
MetS -VE	36	84	120
Total	**178**	**112**	**290**

DISCUSSION

Metabolic syndrome is defined as a cluster of cardiovascular risk factors, including low HDL cholesterol, high triglycerides, insulin resistance, impaired carbohydrate metabolism, raised blood pressure and central obesity.[7] It incorporates the key features of hyperglycemia, visceral obesity, atherogenic dyslipidemia. Metabolic syndrome seems to have 3 potential etiological categories: obesity and disorders of adipose tissue; insulin resistance; and a constellation of independent factors (eg, molecules of hepatic, vascular, and immunologic origin) that mediate specific components of the metabolic syndrome.[8] It was estimated that 20%-25% of South Asians have developed MetS, and many more may be prone to it.[9] Individuals with MS have a 30-40% probability of developing CVD in 20 years, depending on the number of components present.[6] The National Cholesterol Education Program (NCEP) diagnostic criteria of the metabolic syndrome was proposed as a practical tool for identifying a high-risk cardiovascular disease (CVD) phenotype, and several studies have subsequently confirmed that the metabolic syndrome predicts incident CVD.[10.] Atherogenic dyslipidemia, a combination of high triglycerides and low HDL in association with elevated apolipoprotein B (ApoB) and small dense LDL particles, is an important component of MetS and a strong predictor of cardiovascular disease.[2] The independent role of triglycerides (TG) and HDL-C as predictors of CVD has already been established.[11] A meta-analysis has shown that an increase in plasma TG concentration by 1 mmol/L results in a 32% increased risk of CVD in men and 76% in women demonstrating that fasting hypertriglyceridemia is an independent risk factor for cardiovascular disease.[12] Multivariate analysis has revealed serum triglyceride concentration as the most important determinant of the presence of small, dense LDL particles.[13] High triglycerides cause extensive intravascular remodelling, resulting in alteration in the composition and size of the different lipoproteins. The atherogenic lipoprotein phenotype consisting of small dense LDL, low HDL is the result of this intravascular remodelling.[14] Increased atherogenic potential of sdLDL is suggested by the greater propensity to invade the subendothelial space,

increased binding to arterial proteoglycans and susceptibility to oxidative modification.[15] Low HDL cholesterol counts as another component of atherogenic dyslipidemia. Epidemiological studies reveal a strong inverse relation between HDL-cholesterol levels and CHD risk.[8] It is postulated that TG enrichment of HDL particles secondary to enhanced CETP mediated exchange of TG and CE between HDL and TG rich lipoproteins, combined with the lipolytic action of hepatic lipase are the driving forces in the reduction of plasma HDL concentration.[16] Patients with metabolic syndrome exhibit higher concentrations of small, dense LDL subfractions than individuals who do not fulfill the criteria for the diagnosis of this syndrome, as triglyceridemia and low HDL are among the other components of metabolic syndrome. This increase is directly related to the number of components of metabolic syndrome and is mainly determined by the serum concentrations of triglycerides.[8] In the present study, the cardiometabolic risk profile of individuals identified by the use of TG/HDL-c ratio (Table 1) or MetS (Table 2) did not differ substantially irrespective of whether they were high risk or low risk individuals. The minor differences that exist in absolute values is consistent with the criteria used to classify them into Mets +VE and –VE group (Group I and II) and abnormal and normal TG/HDL-c ratio (Group A and B). Mets+VE group (Group I) had a higher waist circumference, higher fasting blood sugar, higher systolic and diastolic blood pressure compared to subjects in group A (TG/HDL $\geq$ 3) who had higher mean values of triglycerides and lower values of HDL.

Subjects who had a high TG/HDL-c ratio and were Mets+VE showed the worst cardio metabolic risk profile. Our data suggest that these subjects should be considered at high risk of CVD and should be closely followed clinically, even in the absence of signs and symptoms of cardiovascular disease. The findings of our study correlated with the findings of Teodoro Marotta *et al*, 2012 and Kimm *et al*, 2010 and Kawamoto R and Tabara Y *et al*, 2011. who showed that high TG/HDL-c ratio were consistently associated with Metabolic syndrome and Patients with Met S and TG/HDL-c ratio $\geq$ 3 had the worst cardiovascular risk profile.[17] Parineetha S, Padma C *et al* in their study have evaluated TG/HDL-c ratio as a surrogate marker of IR in Metabolic Syndrome.[18] The ratio TG/HDL-c, initially proposed by Gaziano *et al*, is an atherogenic index that has proven to be a highly significant independent predictor of myocardial infarction, even stronger than TC/HDL-c and LDL-c/HDL-c.Studies by Protasio lemos da luz *et al*, have shown that elevation in the ratio of TG to HDL-c was the single most powerful predictor of extensive coronary heart disease among all the lipid variables examined. And also in a multivariate model that included, the ratio of triglycerides to HDL-cholesterol was found to be a powerful independent indicator of cardiovascular disease. Several studies have attempted to determine the risk levels of CVD using lipid indexes or formulas.[19] The goal of this work is to manage patients better in order to prevent cardiac events. Study of J. Michael Gaziono *et al*, stated that elevated fasting TG represent a useful marker for risk of CHD, particularly when HDL levels are considered. And also the strong association of the ratio of TG/HDL with risk of CHD suggest a metabolic interaction between the TG and cholesterol ester-rich lipoproteins in increasing risk of MI.[20] Forouhi *et al*. showed strong association of the MetS with cardiovascular risk in South Asians as compared to Caucasians, which could not be explained by presence of conventional risk factors.[21] Numerous studies have confirmed that presence of the metabolic syndrome increases the risk of subsequent development of CVD.[22-27] However, there is published evidence that the TG/HDL-c ratio has been successfully used in predicting the development of cardiovascular events and all-cause mortality.[27] Finally several studies have suggested a number of other unique risk factors that may be responsible for a stronger association between the metabolic syndrome and cardiovascular risk in women. These risk factors include polycystic ovary syndrome, hormonal contraceptive use and gestational diabetes.[28-32]

LIMITATION

The obvious weakness of the study is that it is cross sectional and does not have an outcome data and sex specific cut points have not been used to improve the efficiency of TG/HD-c ratio. In spite of these limitations, it is very economical and simple which is an obvious advantage. and we hope that our results will encourage the clinicians to use it to identify individuals with high cardiometabolic risk. In the present study, we have compared the relative abilities of MetS and TG/HDL-c ratio to accomplish this goal and the clinicians can use the criteria which is relevant for their research.

CONCLUSION

Calculation of TG/HDL ratio is simple and inexpensive and helps to identify people at increased risk of developing cardiovascular disease. It relies on two simple lipid measurements which is done as a routine in the clinical laboratories and does not depend on waist measurements that is neither routinely performed nor has a good degree of reproducibility.

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REFERENCES

1. Global status report on Noncommunicable diseases. WHO. 2015 Jan.
2. Vera B, B. Delia. J, Issam Z, William J R, Diane V, *et al*.: The TG/HDL Cholesterol Ratio Predicts All Cause Mortality in Women with Suspected Myocardial Ischemia A Report from the women's Ischemia Syndrome Evaluation (WISE): Am Heart J. 2010 March; 157(3):548-555.
3. Jesus M, Xavier P, Anna M, Manuel Z, Joan Rubies P, *et al*.: Lipoprotein ratios: Physiological significance and clinical usefulness in cardiovascular prevention: Vascular Health and Risk Management. 2009:5: 757-765.
4. Honard B.V. Insulin Resistance and lipid Metabolism: American Journal Of cardiology. 1999, 84; 28j-32j.
5. Paul L.H.: A compressive definition for metabolic syndrome: Disease Models Mechanisms. 2009 May-Jun; 2(5-6): 231-237.
6. S. Chackrewarthy, D. Gunasekera, A.Pathmeswaren, C.N. Wijekoon, U.K. Ranawaka, *et al*.: A Comprison between Revised NCE ATP III and IDF Definitions in Diagnosing Metabolic Syndrome in an Urban Sri Lanka Populations: The Ragama Health Study: ISRN Endocrinology. 2013 January; Article ID 320176.
7. Ulf L, Robert DL, Deborah L. Wingard, Ronald G.T, Elizabeth L, *et al*.: Metabolic syndrome and Ischemic Heart Disease in Elderly Men and Women: American Journal of Epidemiology. 2001: Vol.153, No. 5. 481-489.
8. Scott M. Grundy: Small LDL, Atherogenic Dyslipidemia, and the Metabolic Syndrome: American heart association journals. 1997; 95:1-4.
9. Anoop Mishra, Ranjita M, Mahen W, Dipanjan B: The metabolic syndrome in south Asians: Continuing escalation and possible solutions: Indian J Med Res. 2007 March, 125: 345-354.
10. Martin k, Rutter, James B. M, Lisa M.S, Ralph B.D.A, Peter W.W, *et al*.: Insulin Resistance, the Metabolic Syndrome, and Incident Cardiovascular Events in the Framingham Offspring Study: Diabetesjournals. 2005 November; vol 54 no 11 3252-3257.
11. Ryuichi K, Yasuhara T, Katsuhiko K, Tetsuro M, Tomo K, *et al*.: Relationship between lipid profiles and metabolic syndrome, insulin resistance and serum high molecular adiponectin in Japanese community-dwelling adults: Lipids in Health and Disease. 2011. 10:79.
12. Geesje M, Dallinga-T, *et al*.: Derangements of Intravascular Remodelling of Lipoproteins in Type 2 Diabetes Mellitus: Consequences for Atherosclerosis Development: Current Diabetes Reports. 2008. 8:65-70
13. Ireni G, Vasilis T, *et al*.: Concentration and relative distribution of low-density lipoprotein subfractions in patients with metabolic syndrome defined according to the National Cholesterol Education Program Criteria: Metabolism-clinical and experimental. 2006 July; vol 55, issue 7, 885-891.
14. T.B. Twickler, G.M. Dallinga-T, J.S. Cohn, and M.J. Chapman, *et al*.: Elevated Remnant-Like Particle Cholesterol Concentraion A Characteristic Feature of the Atherogenic Lipoprotein Phenotype: Journal of American Heart Association. 2004. April; 109: 1918-1925.
15. Kaspar k B, Ronald M K: Metabolic origins and clinical significance of LDL heterogeneity: Journal lipid research.2002 May; 43, 1363-1379.
16. Shirya Rashid, Takehiko W, Taro S, Gary F L: Mechanisms of HDL Lowering in insulin resistant, hypertryglyceridemic states: the combined effect of HDL triglycerides enrichment and elevated hepatic lipase activity: Clinical Biochemistry.2003. Oct; 36(6):421-9.
17. Teodoro M, Barbara F, *et al*.: Triglyceride-to-HDL-cholesterol Ratio and Metabolic Syndrome as Contributors to Cardiovascular Risk in Overweight Patients: Intervention And Prevention. 2010 August; volume 18 No.8:1608-1618.
18. Parineeta S, Padma C, Sandeep R, *et al*.: TG/HDL-C Ratio: A Surrogate Marker of Insulin Resistance in Patients with Metabolic Syndrome: MGMJMS. 2014 Jan-March; 1(1):18-21.
19. Protasio Lemos d L, Desiderio F, Jose Rocha F, Pedro Lemos, Antonio Carlos P, *et al*.: High Ratio of Triglycerides to HDL-Cholesterol Predicts Extensive Coronary Disease: Clinical science. 2008 April; 64:427-32.
20. Michael J G, Charles H H, Christopher J O, Jan L B, Julie E: Fasting Triglycerides, High-Density Lipoprotein, and Risk of Myocardial Infarction: Circulation American Heart Association. 1997. 96:2520-2525.
21. Kaushik P, Soumik G, Sujoy G, Pradip M, Subhankar C, *et al*.: Metabolic Syndrome in South Asians: International

journal of Endocrinology and Metabolism. 2012 Jan-Feb; 16(1): 44-45.
22. Knut Borch-Johnsen: Epidemiology of the Metabolic Syndrome: Springer-Verlag Wein. 2013; 10.1007/978-3-7091-1331-8_2.
23. Lakka HM, Laaksonen DE, The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men: JAMA. 2002; 288:2709-2716.
24. Hu G, Qiao Q, *et al*.: Prevalence of the metabolic syndrome and its relation to all-cause mortality in non-diabetic European men and women: Arch Intern Med. 2004; 164:1066-1076.
25. Pyorala M, Miettinen H, *et al*.: Insulin resistance syndrome predicts the risk of coronary heart disease and stroke in healthy middle-aged men: the 22-year follow-up results of Helsinki Policemen study: Arterioscaler Thromb Vasc Biol. 2000; 20:538-544.
26. Isomaa B, Almgren P, Tuomi T, Forse´n B, Lahti K, *et al*.: Cardiovascular morbidity and mortality associated with the metabolic syndrome. Diabetescare.2001; 24:683-689.
27. Martin R S, Horacio A C, Walter G E, Carlos E, CARLOS E March, Eduardo Balbin, *et al*.: Comparison of the abilities of the plasma triglyceride/high-density lipoprotein cholesterol ratio and the metabolic syndrome to identify insulin resistance: SAGE Journals.2013…Vol 10 no. 4. 346-352.
28. Girman CJ, Rhodes T, *et al*.: The metabolic syndrome and risk of major coronary events in the Scandinavian Simvastatin Survival study and the Air Force/Texas Coronay Atherosclerosis Prevention Study: Am J Cardiol. 2004; 93:136-141.
29. Johnson L W, Weinstock: The Metabolic syndrome: Concepts and Controversy: Mayo Clinic Proc. 2006; 81:1615-1620.
30. Hunt k J, Resendez R G, Williams K, Haffner S M, Stem M P: National Cholesterol Education Program Versus World Health Organization metabolic syndrome in relation to all cause and cardiovascular mortality in the San Antoino Heart Study: Circulation. 2004; 110: 1251-1257.
31. Bayturan O, Tuzcu E M, Lavoie A, *et al*.: The Metabolic Syndrome, its component risk factor, and progression of coronary atherosclerosis: Arch Intern Med. 2010; 170: 478-484.
32. Salvatore M, Kristian B F, Jacques G, Lawrence J, Louise P, *et al*.: The Metabolic Syndrome and Cardiovascular Risk: Journal of the American College of Cardiology. 2010;56(14):113-1132

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