

Original Research Article

Assessment of ApoB and TG/HDL-C ratio as indicators of insulin resistance determined by HOMA IR in patients with the metabolic syndrome

Formatted: Font: Font color: Auto

Objectives:

The aim of the present study was to assess Apolipoprotein B (Apo-B) and Triglyceride/High Density Lipoprotein Cholesterol (TG/HDL-C) ratio as indicators of insulin resistance (IR) with Homeostasis Model of assessment of insulin resistance (HOMA IR) in metabolic syndrome patients.

Study design: Observational and prospective

Place and duration of study: The study was carried out in Department of Biochemistry and Department of Medicine, MGM Medical College, Navi-Mumbai from March 2012 to June 2013

Methodology

Total 110 normal subjects and patients were recruited in the study after obtaining informed written consents. They were divided into two groups. Group I was healthy controls (n=50) and Group II was included subjects with MS (n=60) as per NCEP ATP III criteria. Anthropometric measurements & biochemical analysis was performed in all subjects. IR was defined by HOMA IR. Simple & multiple regression analysis was used to obtain relationship between IR (HOMA IR) using TG/HDL-C (model -1) and Apo B (Model-2) as independent variables.

Result

There were statistically significant differences in anthropometric, glycemic and lipid parameters between the control and study groups ($p < 0.0001$). The regression model between HOMA IR and TG/HDL-C ratio showed a positive correlation ($r = 0.29$, $p < 0.05$), while between HOMA IR & Apo B also showed a significantly positive correlation ($r = 0.41$, $p < 0.001$). But combined multivariate analysis indicated that Apo B is a better predictor of IR as compared to TG/HDL-C ratio.

Conclusion

We conclude in our study that ApoB may be a more significant and a better predictor of IR than TG/HDL-C and hence could be adopted in routine laboratory practice as a lipid marker for prediction of insulin resistance (IR) in metabolic syndrome patients. So that this parameter could contribute to the identification of those patients with the metabolic syndrome may be identified at an early stage.

1. INTRODUCTION

Insulin resistance (IR), is a major finding in the metabolic syndrome (MS) [1] and a contributing factor for risk of development of type 2 diabetes and cardiovascular diseases (CVD). Therefore, a reliable

37 measure of insulin resistance is important for investigating its link with metabolic syndrome (MS).[2,3] For
38 this, the homeostasis model assessment of insulin resistance (HOMA-IR) [4], is a widely accepted
39 method as an alternative to the glucose clamp which is laborious. HOMA-IR is comparable to the glucose
40 clamp technique, in terms of precision but not accuracy and hence it is possible to study a large number
41 of

42 subjects using a single measurement of glucose and insulin in the fasting state [5]. However, as it is Since
43 it is empirical, additional tests have been used for assessment of IR.[6]

44
45 Another alternative proposed for the identification of IR is the determination of fasting triglycerides (TG)
46 and glucose.[7]. Since elevated triglycerides is one of the NCEP ATP III criteria for MS, the
47 hypertriglyceridemic state which may accompany either normal and impaired fasting glucose should
48 contribute to promoting surveillance for IR.[8,9]

49
50 Recently Triglycerides/High density lipoprotein cholesterol (HDL-C) ratio has been proposed as one of
51 the indices to evaluate the atherogenic state due to the association between dyslipidemia and IR. [10,11]
52 IR significantly impacts lipoprotein metabolism and is associated with increased TG levels and depressed
53 decreased HDL-C levels.[12,13].

54
55 But TG/HDL-C ratio is known to be affected by gender and ethnic variabilities. Beside, TG itself may also
56 be influenced by biological variabilities variability such as lifestyle, medications, and metabolic
57 abnormalities. Hence TG/HDL-C ratio may not be a stable variable predictor of IR.[14] Recent studies
58 have suggested the use of Apo B as a direct marker of atherogenic state & IR.[14,19]

59
60 ApoB is a structural protein of atherogenic lipoproteins such as very-low-density lipoprotein [VLDL],
61 intermediate-density lipoprotein [IDL] as well as both large, buoyant low density lipoproteins (LDL-C)
62 cholesterol and small, dense LDL.[15,16] Cardiovascular risk is more directly related to the number and
63 size of circulating atherogenic particles than to the concentration of cholesterol in these particles.[17,18].
64 In hypertriglyceridemic patients, the vast majority of total plasma Apo B is strongly associated with LDL-C,
65 making Apo-B100 an effective surrogate for LDL particle concentration.[19]

66
67 Apo B may be particularly relevant in the assessment of insulin resistant states, such as diabetes and
68 metabolic syndrome, as patients with these disorders often manifest normal LDL-C values, but have a
69 preponderance of small, dense LDL-C particles and higher Apo-B levels.

70 Hence this study was undertaken to compare the existing model of HOMA IR with both TG/HDL-C ratio
71 and Apo B levels for assessment of insulin resistance in MS patients.

72 73 2. MATERIALS AND METHODS

74
75 This was an observational prospective study approved by Institutional ethics review committee. A total of
76 110 subjects between age group of 35-65 years were recruited for the study after obtaining their informed
77 consent. The subjects were divided in to two groups. Group I (n=50) included healthy controls and
78 Group II (n=60) included subjects with metabolic syndrome. The diagnosis of metabolic syndrome was
79 based on NCEP ATP III criteria which is based on presence of three or more of the risk factors such as:
80 Waist circumference: men > 102 cm (40 in); women > 88 cm (35 in); Triglycerides $\geq$ 150 mg/dl; HDL-C
81 cholesterol: men < 40 mg/dl; women < 50 mg/dl; Blood pressure $\geq$ 130/ $\geq$ 85 mmHg; Fasting glucose $\geq$ 110
82 mg/dl. All the subjects were matched for age, gender and were non smokers, non alcoholic and of same
83 socioeconomic status. The subjects with chronic diseases of kidney, liver, cancer and diabetes mellitus
84 were excluded from study.

85
86 Measurements of height and weight were done with the subjects standing, without shoes and with light
87 clothing. BMI was calculated as weight in kg divided by height in meter squared. Waist circumference
88 (WC) was measured at the level of the umbilicus with a tape in centimeter scale.

89
90 Venous blood samples were collected after 12 hrs of fasting by closed collection system. For fasting
91 plasma glucose samples were collected in fluoride vacutainer and for serum lipid profile and Apo B
92 samples were collected in plain vacutainer. All the samples were processed on the same day for
93 biochemical analysis. Fasting blood glucose, TG, Total cholesterol and HDL-C -cholesterol levels were
94 measured by enzymatic methods using kits supplied by Beckman and Coulter. All the assays were
95 performed on fully automated analyzer, AU 480 of Beckman and Coulter. TG values of Group-II subjects
96 were between 150 to 192 mg/dl.

97
98 LDL-C and VLDL values were obtained by using Friedewald's formula.[20] Serum Apo-B levels were
99 estimated by immunoturbidometry method using kits supplied by Beckman Coulter. Fasting plasma
100 insulin was measured by ELISA technique using kits supplied by Immunoshop India Pvt Ltd. Insulin
101 Resistance was determined by means of homeostasis model assessment index (Homeostasis Model
102 Assessment–Insulin Resistance, HOMA–IR) using the formula,

103
104
$$\text{HOMA-IR} = \text{fasting insulin } (\mu\text{U/ml}) \times \text{fasting glucose (mg/dl)} / 405. \text{ [21]}$$

105 The TG/HDL-C ratio was calculated using the formula: Fasting TG (mg/dl)/HDL-cholesterol (mg/dl).

106 107 3. Statistical analysis

108 Data are presented as means $\pm$ SDs, student t-test was used to compare age, BMI, W/H ratio, SBP,
109 DBP, FPG, FPI, Lipid profile and Apo B levels between patients and controls. The correlation of HOMA IR
110 was correlated with both TG/HDL-C ratio and Apo B was determined by and Pearson correlation

coefficient was found. Multiple regression analysis was performed when appropriate. Statistical Package for Social Sciences- (version 17.0) was used for calculations. P values < 0.05 were considered statistically significant.

4. Results:

Table 1 shows anthropometric and clinical characteristics of control and study group (mean and standard deviation).

There were as-significant differences in the values of anthropometric parameters ($p < 0.0001$). The BMI was between 25 to 30 kg/m². The average W/H ratio for the patients exceeded the NCEP ATP III criteria in both the genders. The difference in fasting plasma glucose and LDL- C was also statistically significant in-between the control and study group ($p < 0.0001$). The HOMA IR was elevated in the metabolic syndrome group compared to controls. The mean high density lipoprotein-cholesterol (HDL-C) level was significantly lower in group of the patients ($p < 0.0001$) whereas Apo B levels were significantly higher ($p < 0.0001$). Regression analysis was performed for patients using HOMA IR as dependent variable and TG/HDL-C as an independent variable in first model. The model showed positive correlation with $r = 0.29$ value as 0.29 which is statistically significant ($p < 0.05$) as shown in graph 1. In Model 2, Apo B was used as independent variable with HOMA IR Which also showed positive correlation ($r=0.41$, $P<0.001$) with statistical significance in graph 2. The combined multivariate analysis of HOMA IR with TG/HDL-C ratio and Apo-B which is depicted in table 2 showed that Apo-B variable is statistically significant ($p < 0.005$) than while TG/HDL-C ratio was not significant. The regression model of control group did not reveal any statistical significance for both measured independent parameters as predictors of IR.

5. DISCUSSION:

In our study we have used HOMA IR, the widely accepted Model for assessment of IR and compared the lipid profile markers TG/HDL-C and Apo B , as predictors of IR.

The TG/HDL-C ratio was calculated in metabolic syndrome patients and control group and we found that TG/HDL-C ratio was higher in the study group. There was a significant positive correlation between HOMA IR and TG/HDL-C ratio ($r=0.29$; $p<0.05$) in the study group, refer to graph 1. The regression model showed R^2 value of 0.087, indicating 8.7 percent variation in the HOMA IR due to TG/HDL-C ratio. We also performed regression analysis for control group and we did not get any significant correlation between HOMA IR and TG/HDL-C or Apo B.

Our results are in accordance with Marotta et al [22] and Brehm et al [23] who demonstrated positive correlation between TG/HDL-C ratio and insulin resistance, confirming that TG/HDL-C ratio predicts

147 insulin resistance in metabolic syndrome. But an independent study by Knight et al [24] stated that,
148 TG/HDL-C ratio fails to predict insulin resistance in African American women.

149
150 The predictive level and significance of correlation between HOMA IR and Apo B was assessed by
151 regression analysis taking HOMA IR as dependent variable. As shown in graph 2, a significant positive
152 correlation (r=0.41; p < 0.001) was obtained between HOMA IR and Apo-B in metabolic syndrome
153 patients. The R² value showed by regression model was 0.17 showing 17 percent variation in HOMA IR
154 due to Apo B levels.

155
156 When combined multivariate analysis of TG/HDL-C and Apo B with HOMA IR was performed, the
157 regression model indicated that TG/HDL-C ratio is a non significant (p> 0.05) variable whereas Apo B
158 was statistically significant (p<0.05) as shown in table 2. Most of the studies have compared HOMA IR
159 with either TG alone or withor with TG/HDL-C ratio.[25,21] Some studies have estimated LDL and Apo B
160 levels or Apo B to Apo AI ratio in metabolic syndrome or diabetic patients. We have performed combined
161 model of HOMA IR with either TG/HDL-C or Apo B studied by multivariate analysis. The results of our
162 study support that measurement of Apo B is superior to TG/HDL-C ratio as a predictor of IR. Although the
163 TG/HDL-C ratio is cheap and easy to calculate [26] and is also a good predictor of LDL size. However, it
164 does not necessarily identify most of the patients with hyper ApoB. Moreover, in addition to the
165 requirement of a fasting state it shows high biological variability inherent to triglycerides [27],
166 as reflected by the wide range of recommended cutoff points and it necessitates fasting state. On the
167 other hand, Apo B reflects the total number of atherogenic particles and its determination can also be
168 made in a non fasting state.[28,29] Its biological variability is also known to be lower than other lipid
169 components.

170
171 Thus, our study shows that measurement of Apo-B levels may be used as a more stable, predictive
172 atherogenic biomarker and IR predictor in metabolic syndrome patients compared to biomarker than
173 TG/HDL-C ratio in metabolic syndrome patients with insulin resistance. Miller et al [14] in a recent review
174 his review has compared the predictive value of Apo-B with non-HDL-C for CVD outcomes and reported
175 that Apo B has been commonly identified as either superior marker or equivalent to non-HDL-C. While
176 non-HDL-C alone has been more predictive only in limited cases.[30] Yet in studies that demonstrated
177 statistically significant differences between Apo-B and non-HDL-C, the differences in point estimates
178 were often quite small and therefore unlikely to have a major impact of day-to-day clinical practice.[31].

179
180 As per NCEP ATP III final guidelines of 2002 ApoB apolipoprotein B is recommended as a potential
181 marker for MS and it has been proposed as an alternative to LDL cholesterol. Thus, Apo-B may be linked

182 | to metabolic risk factor clustering phenomena. As [nNon-HDL-C-cholesterol](#) is significantly correlated with
183 [Apoapolipoprotein B](#), it can serve as a “surrogate” for metabolic syndrome [\[32\]](#).
184

185 This study is limited by the fact that HOMA IR index was not correlated with insulin sensitivity by the gold
186 standard method such as euglycemic clamp.
187

188 6.CONCLUSION

189 In conclusion, we propose that Apo B estimation be used to predict long term **lipid** control for diagnosing
190 IR in line with the current use of HbA1C as long term glycemic control marker.
191

192 ACKNOWLEDGEMENT

193 This work was supported by MGM Medical College and Hoapital, Kamothe, Navi-Mumbai, India. We
194 acknowledge **Mr. Pandurang Tihare for helping with data analysis.**
195

196 COMPETING INTERESTS

196 The authors have no competing interests.
197

198 CONSENT

199 | **Authors declare that written informed consents [was were](#) obtained from subjects ([normal-controls](#)) and**
200 **patients before their participation in the study.**
201

202 ETHICAL APPROVAL

203 All authors hereby declare that approval from Institutional ethics review committee was
204 obtained and study was carried out as per standards.
205

206 REFERENCES:

- 207
- 208 1.Martin BC, Warram JH, Krolewski AS, Bergman RN, Soeldner JS, Kahn CR: Role of glucose and insulin
209 resistance in development of type 2 diabetes mellitus: results of a 25-year follow-up study. Lancet. 1992;
210 340:925-929.
211
 - 212 2. Hanley AJ, Williams K, Stern MP, Haffner SM: Homeostasis model assessment of insulin resistance in
213 relation to the incidence of cardiovascular disease: the San Antonio Heart Study. Diabetes Care. 2002;
214 25:1177-1184.
215
 - 216 3. Rutter MK, Meigs JB, Sullivan LM, D’Agostino RB Sr, Wilson PW: Insulin resistance, the metabolic
217 syndrome, and incident cardiovascular events in the Framingham Offspring Study. Diabetes. 2005;
218 54:3252-3257.

219

220 4. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC: Turner, Homeostasis

221 model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin

222 concentrations in man. *Diabetologia*. 1985; 28:412-419.

223

224 5. Bonora E, Targher G, Alberiche M, Bonadonna RC, Saggiani F, Zenere MB, Monauni T, Muggeo M:

225 Homeostasis model assessment closely mirrors the glucose clamp technique in the assessment of insulin

226 sensitivity: studies in subjects with various degrees of glucose tolerance and insulin sensitivity. *Diabetes*

227 *Care*. 2000; 23(1):57-63.

228

229 6. Wallace TM, Levy JC, Matthews DR: Use and abuse of HOMA modeling. *Diabetes Care*. 2004;

230 27:1487-1495.

231

232 7. Simental-Mendía LE, Rodríguez-Morán M, Guerrero-Romero F. The product of fasting glucose and

233 triglycerides as surrogate for identifying insulin resistance in apparently healthy subjects. *Metabolic Syndr*

234 *ome Related Disorders*. 2008;6:299-304

235

236 8 Haffner SM, Stern MP, Hazuda HP, Mitchell BD, Patterson JK. Cardiovascular

237 risk factors in confirmed prediabetic individuals: does the clock for coronary heart disease start ticking

238 before the onset of clinical diabetes? *Journal of the American Medical Association*. 1990;263:2893–2898

239

240 9. D'Agostino RB Jr, Hamman RF, Karter AJ, Mykkanen L, Wagenknecht

241 LE, Haffner SM; Insulin Resistance Atherosclerosis Study Investigators. Cardiovascular disease risk

242 factors predict the development of type 2 diabetes: the Insulin Resistance Atherosclerosis Study.

243 *Diabetes Care*. 2004;27:2234 –2240.

244

245 10. McLaughlin T, Abbasi F, Cheal K, Chu J, Lamendola C, Reaven G: Use of metabolic markers to

246 identify overweight individuals who are insulin resistant. *Annals of Internal Medicine*. 2003; 139:802-809.

247

248 11. McLaughlin T, Reaven G, Abbasi F, Lamendola C, Saad M, Waters D, Simon J, Krauss RM: Is there a

249 simple way to identify insulin-resistant individuals at increased risk of cardiovascular disease? *American*

250 *Journal of Cardiology*. 2005;96:399-404.

251

252 12. M. Laakso and E. Barrett-Connor, "Asymptomatic hyperglycemia is associated with lipid and

253 lipoprotein changes favoring atherosclerosis," *Arteriosclerosis*. 1989;9(5):665–672.

254

255 13. B. V. Howard, "Insulin resistance and lipid metabolism," American Journal of Cardiology. 1999; 84:
 256 28J–32J.
 257

258 14. Michael Miller, Neil J. Stone, Christie Ballantyne, Vera Bittner, Michael H. Criqui, Henry N. Ginsberg,
 259 Anne Carol Goldberg Triglycerides and Cardiovascular Disease: A Scientific Statement From the
 260 American Heart Association. *Circulation*, 2013;2293-2305.
 261

262 15. Marcovina S, Packard CJ. Measurement and meaning of apolipoprotein AI and apolipoprotein B
 263 plasma levels. *Journal of Internal Medicine*. 2006;259:437–446.
 264

265 16. Walldius G, Jungner I. The apoB/apoA-I ratio: a strong, new risk factor for cardiovascular disease and
 266 a target for lipid-lowering therapy—a review of the evidence. *Journal of Internal Medicine*. 2006;259:493–
 267 519.
 268

269 17. Barter PJ, Ballantyne CM, Carmena R, et al. Apo B versus cholesterol in estimating cardiovascular
 270 risk and in guiding therapy: report of the thirty-person/ten-country panel. *Journal of Internal Medicine*.
 271 2006;259:247–258
 272

273 18. Kathiresan S, Otvos JD, Sullivan LM, et al. Increased small low-density lipoprotein particle number: a
 274 prominent feature of the metabolic syndrome in the Framingham Heart Study. *Circulation*. 2006;113:20–
 275 29.
 276

277 19. Michael H. Davidson. Apolipoprotein Measurements: Is More Widespread Use Clinically Indicated?
 278 *Clinical Cardiology*. 2009;32, 9, 482–486.
 279

280 20. Friedewald W.T., Levy R.I., and Fredrickson D.S.: Estimation of the concentration of
 281 low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clinical*
 282 *Chemistry*. 1972;18:499–502.
 283

284 21. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC: Turner, Homeostasis
 285 model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin
 286 concentrations in man. *Diabetologia* 1985, 28:412-419
 287

288 22. Teodoro Marotta, Barbara F. Russo² and L. Aldo Ferrara. Triglyceride-to-HDL-cholesterol Ratio and
 289 Metabolic Syndrome as Contributors to Cardiovascular Risk in Overweight Patients. *Obesity*. 2010;
 290 18:1608–1613

Formatted: Portuguese (Brazil)

291
 292 23. Attila Brehm, Georg Pfeiler, Giovanni Pacini, Heinrich Vierhapper, and
 293 Michael Roden. Relationship between Serum Lipoprotein Ratios and Insulin Resistance in Obesity.
 294 Clinical Chemistry. 2004; 50(12):2316–2322
 295
 296 24. Michael G. Knight, Julia H. Goedecke, Madia Ricks, Juliet Evans, Naomi S. Levitt,
 297 Marshall K. Tulloch-Reid, Anne E. Sumner. The TG/HDL ratio does not predict insulin resistance in
 298 overweight women of African descent: A study of south African, African Americans and West African
 299 Women. Ethnicity & Disease, 2011; 21,:490-494
 300
 301 25. Chávez A.G, Simental-Mendía L.E, Elizondo-Argueta S. Elevated triglycerides/HDL-cholesterol ratio
 302 associated with insulin resistance. Circulation 2011;79:115-119
 303
 304 26. Wanger A.M, Perez A.P., Sanchez-Quesada J.L., Ordonez-Linos J. Triglycerides-to-HDL Cholesterol
 305 ratio in the Dyslipidemic state classification of type 2 diabetes mellitus. Diabetic Care. 2005;28(7):1798-
 306 1800.
 307
 308 27. Ortolá J, Castineiras MJ, Fuentes-Arderiu X: Biological variation data applied to the
 309 selection of serum lipid ratios used as risk markers of coronary heart disease. Clinical Chemistry. 1992;
 310 38:56–59.
 311
 312 28. Sniderman AD, Furberg CD, Keech A, Roeters van Lennep JE, Frohlich J, Jungner I, Walldius G:
 313 Apolipoproteins versus lipids as indices of coronary risk and as targets for statin treatment. Lancet. 2003
 314 361:777–780.
 315
 316 29. Walldius G, Jungner I, Holme I, Aastveit AH, Kolar W, Steiner E: High apolipoprotein
 317 B, low apolipoprotein A-I, and improvement in the prediction of fatal myocardial infarction (AMORIS
 318 study): a prospective study. Lancet. 2001 358:2026–2033.
 319
 320 30. Wiesbauer F, Blessberger H, Azar D, Goliash G, Wagner O, Gerhold L, Huber K, Widhalm K,
 321 Abdolvahab F, Sodeck G, Maurer G, Schillinger M. Familial-combined hyperlipidaemia in very young
 322 myocardial infarction survivors (< or = 40 years of age). European Heart Journal. 2009;30:1073–1079
 323
 324 31. De Graaf J, Couture P, Sniderman A. A diagnostic algorithm for the atherogenic apolipoprotein B
 325 dyslipoproteinemias. Nature Clinical Practice Endocrinology & Metabolism. 2008;4:608–618.
 326
 327 32. National Cholesterol Education Program. National Heart, Lung, and Blood Institute

328 National Institutes of Health NIH Publication No. 02-5215. 2002.

329

330

331

332

333

334

335

336

337

338

339 Table.1 Showing descriptive and comparative statistics for different groups by student t test

340 Use the correct symbol $\leq$ for the P values in the table for all values

Parameters	Group I <u>Controls</u>	Group II <u>Patients</u>	P Value
SBP(mm/Hg)	115 $\pm$ 6.98	134 $\pm$ 2.86	$p < 0.0001$
DBP (mm/Hg)	76.7 $\pm$ 4.98	96 $\pm$ 0.60	≤ 0.0001
BMI (Kg/m^2)	23.3 $\pm$ 1.39	29.6 $\pm$ 0.53	0.0001
W/H ratio	0.802 $\pm$ 0.03	0.99 $\pm$ 0.01	0.0001
Fasting Plasma Glucose mg/dl	88.1 $\pm$ 5.06	113. $\pm$ 1.19	0.0001
Fasting Plasma insulin $\mu\text{IU/ml}$	10.2 $\pm$ 3.2	22 $\pm$ 0.11	0.0001
HOMA IR	2.23 $\pm$ 0.69	6.14 $\pm$ 0.09	0.0001
Cholesterol mg/dl	146.3 $\pm$ 10.59	174 $\pm$ 15	0.0001
Triglycerides (mg/dl)	110 $\pm$ 17.2	189 $\pm$ 7.5	0.0001
HDL Cholestrol mg/dl	44.5 $\pm$ 3.58	40 $\pm$ 0.6	0.0001
VLDL mg/dl	21.59 $\pm$ 2.72	36 $\pm$ 2.1	0.0001

LDL cholesterol mg/dl	83.92±14.2	103±22.2	0.0001
Apo B mg/dl	82.615±15.34	108.96±23.06	0.0001
TG/HDL-C ratio	2.4653± 0.2094	4.554± 0.152	0.0001

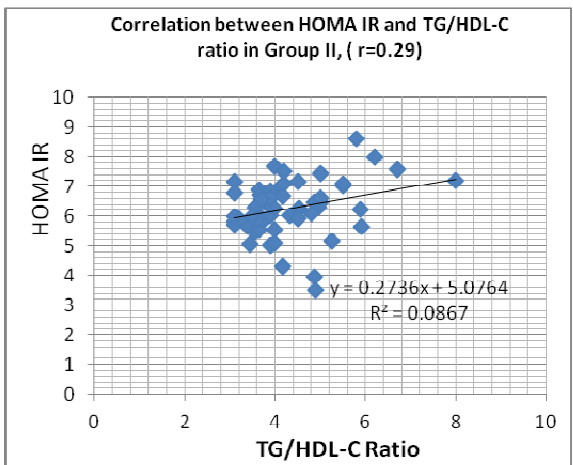
Data is expressed as mean ± sd

P<0.01 is statistically significant

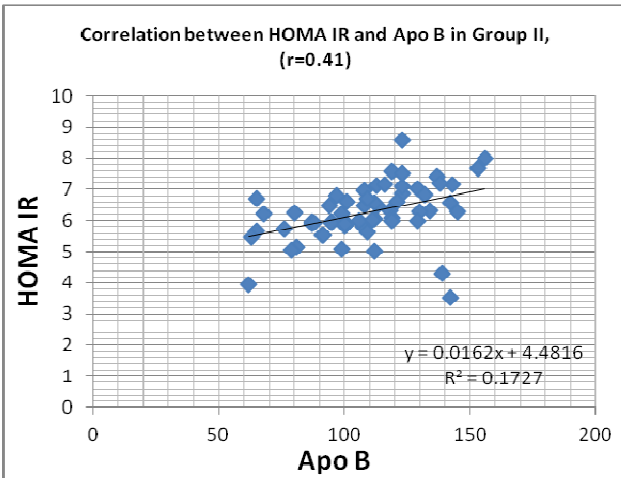
Table 2 Linear Regression Model

Dependent Variable: HOMA IR

Variable	Reg. Coefficient	SE	P-value	Correlation Coefficient	R-square
TG/HDL-C	0.180	0.114	0.120	0.46	0.21
APO B	0.014	0.005	0.005		



Graph 1 showing positive correlation between HOMA IR (Y-axis) and TG/HDL-C (X-axis) ratio in subjects with metabolic syndrome($r=0.29$; $P<0.05$)



Graph 2 showing positive correlation between HOMA IR (Y-axis) and Apo B (X-axis) ratio in subjects with metabolic syndrome($r=0.41$; $P<0.001$)