Serum lipids and lipoprotein profile in ischemic heart disease patients with and without diabetes mellitus

Sharanbasappa.M.Awanti*, Mungli Prakash, Prasamit S.Baruah, Nitika
Department of Biochemistry, Kasturba Medical College, Manipal, 576104, (INDIA)
Tel : 0091-0820-2922326
E-mail : drawanti73@yahoo.co.in
Received: 21ˢᵗ June, 2008 ; Accepted: 26ᵗʰ June, 2008

INTRODUCTION

Atherosclerosis is a leading cause of morbidity and mortality in humans because of its effect upon heart, brain, kidney and other vital organs[1]. Atherosclerosis in diabetic population tends to occur at an earlier age and with greater severity than in the non-diabetic population[2]. The concept of a "multiple risk factors" for ischemic heart disease" (IHD) was suggested by the investigators from the Framingham Heart Study[3]. Their analysis shown that no single cause could be found to explain the reason why some people had IHD while others did not[3]. The established risk factors are sex[4,5], age[4,5], family history of premature IHD[4,5], cigarette smoking[4,5], hypertension[4,5], elevated serum total cholesterol (TC)[4,5], elevated low density lipoprotein-cholesterol (LDL-c)[4,5], physical inactivity[5], obesity[5,6] diabetes mellitus[4,5]. In recent times diabetes is considered epidemiologically the most common disease both in developed[7] and developing countries[5]. One of the common biochemical finding in diabetes patients is abnormal lipid and lipoprotein profile[8]. It has been suggested by previous authors that the incidence of IHD is predominantly more in diabetes patients as compared to non diabetes individuals[9]. In this context, we have designed the current study to know the levels of lipids and lipoproteins in IHD patients with and without diabetes mellitus and also in healthy controls.

Subjects and methods

The study was carried out on 30 IHD patients with diabetes and 30 IHD patients without diabetes who were hospitalized to Kasturba hospital, Manipal and 30 healthy controls. All the diabetes patients having pre-existing familial hyperlipidemias were excluded from the study to minimize the bias. This study was approved by institutional ethical committee and informed consent was obtained from all subjects involved in the study.

Under aseptic conditions blood samples (5 ml) were drawn into plain vacutainers from ante-cubital veins of controls and patient groups. The collected blood was allowed to clot for 30 minutes, and then centrifuged at 2000 g for 15 minutes for clear separation of serum. All assays were performed immediately after serum was separated.

Serum obtained from venous blood sample are used for estimating total cholesterol (TC), triglyceride (TG), high density lipoprotein cholesterol (HDL-c), low density lipoprotein cholesterol (LDL-c), fasting plasma glucose (FPG) using automated analyser (Hitachi 911). Serum total phospholipids were determined by method
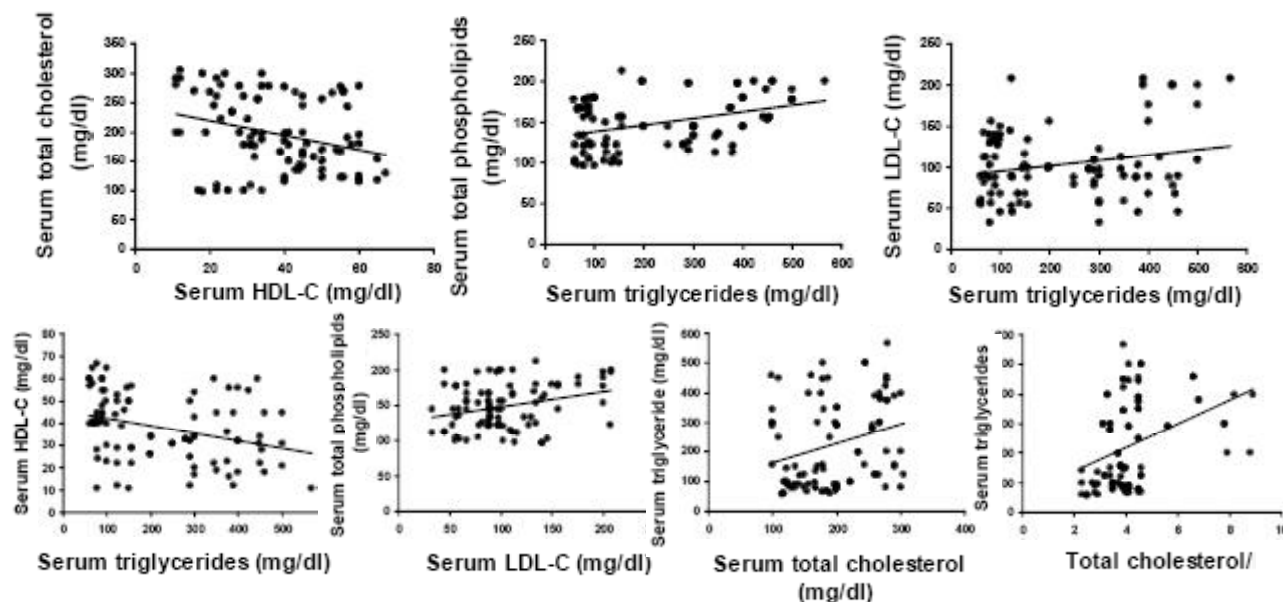
Figure 1: Correlation between different parameters of lipid profile in controls and patient groups

of Connerty et al.[10].

Statistical analysis

The results were expressed as mean ±standard deviation (SD). A p value of <0.05 was considered statistically significant. Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS-10, Chicago, USA). Descriptive analysis of variance (ANOVA) followed by multiple comparison by post-hoc test was used to compare mean values. Pearson's correlation was applied to correlate between the parameters.

RESULTS

The mean±SD values for various lipid and lipoprotein levels in healthy controls and patient groups are given in TABLE 1. IHD patients with and without diabetes shown significant increase in serum total cholesterol (p<0.01), triglycerides (p<0.01), LDL-c (p<0.01), total phospholipids (p<0.01), TC/HDL-c (p<0.01) compared to healthy controls. The HDL-cholesterol levels were significantly lower (p<0.01) in IHD patients with and without diabetes compared to healthy controls. On applying Pearson's correlation (figure 1) there was significant positive correlation between TG with total phospholipids (r=0.367, p<0.01), LDL-c (r=0.223, p<0.05), TC (r=0.278, p<0.01) and TC/HDL-c (r=0.388, p<0.01), and total phospholipids with

TABLE 1: Demographic characteristics and biochemical parameters of IHD patients with and without diabetes, and healthy controls (expressed in mean ± SD)

Parameters	Healthy controls (n=30)	IHD with diabetes (n=30)	IHD without diabetes (n=30)
Total cholesterol(TC) mg/dl	150.2±27.6	213.8±78.2*	227.2 ±41.3*
Triglycerides (TG) mg/dl	93.5±28.0	298.2±138.4*	288.5±132.1*
HDL-cholesterol (HDL)-c mg/dl	50.4±8.7	21.9±7.32*	41.4±10.9**
LDL-cholesterol (LDL)-c mg/dl	90.4±50.1	103.9±30.9*	117.3±43.6*
Total phospholipids mg/dl	133.03±29.9	153.9±30.41*	159.5±28.7*
TC/HDL-C	3.49±0.75	4.63±1.57*	4.57±1.43*

*P<0.0l compared to healthy controls, **p<0.01 compared to IHD with diabetes

LDL-c (r=0.291, p<0.01). There was significant negative correlation between HDL-c with TC (r=- 0.300, p<0.01) and TG (r=- 0.334, p<0.01).

DISCUSSION

Hypercholesterolemia is a major risk factor for coronary heart disease[11,12] but total cholesterol values have poor discriminating power in the assessment of risk for coronary heart disease (CHD)[13]. In line with the earlier studies our data has shown no difference in TC values in IHD patients with and without diabetes supporting poor discriminating power of TC in assessing risk

Regular Paper

of CHD. The association of increased LDL-c and decreased HDL-c is well established[14]. In the present study the LDL-c levels were found to be increased and HDL-c levels were decreased in the IHD patients with and without diabetes. Our findings supports previous authors reporting no difference in lipid profile in diabetes patients with and without IHD[8]. This suggests possible abnormalities of cholesterol efflux over cholesterol influx in IHD patients.

It has been shown that, the ratio between the TC to HDL-c is clinically more useful in assessment of coronary risk rather than emphasizing exclusively on either HDL-cholesterol or LDL-cholesterol[15,16]. Results from the present study has shown that ratio between the TC to HDL-c is significantly increased in both the patient groups compared to controls. Thus the results indicate that the ratio between TC to HDL-c has more predictive value.

Controversy exits with regard to TG levels as an independent risk factor[17]. Decreased lipoprotein lipase activity and HDL-c concentration has shown inverse relationship to TG and LDL-c[18]. The result of the present study has shown that there is an increased level of TG and decreased level of HDL-c in both the patient groups and we found significantly negative correlation of HDL-C with both TG and TC supporting the findings of previous studies[18]. Thus the results indicate that there is a combined abnormality in lipid and lipoprotein metabolism in these patients. Recently the phospholipids have gained importance as a risk factor for coronary heart diseases. It has been reported that total phospholipids content is increased in patients with atherosclerosis[19,20]. Our results have shown that there is no such significant difference in the total phospholipids levels within the patient groups.

To conclude there is combined abnormalities of lipid and lipoproteins in patients with IHD with and without diabetes mellitus.

REFERENCES

[1] R.Ross; N.Engl.J.Med., **314(8)**, 488-500 **(1986)**.

[2] K.Pyorala, M.Laakso, M.Uusitupa; Diabetes Metab Rev., **3(2)**, 463-524 **(1987)**.

[3] W.B.Kannel, W.P.Castelli, T.Gordon; Ann.Intern Med., **90(1)**, 85-91 **(1979)**.

[4] M.Pietrzak-Nowacka, K.Safranow, H.Rulkowska, M.Brzosko, L.Domanski, G.Dutkiewicz, M. Lawniczak, E.Plonska, E.Szmatloch; Ann.Acad. Med.Stetin., **52(2)**, 71-7 **(2006)**.

[5] S.Dodani, D.D.MacLean, R.E.LaPorte, M.Joffres; Ethn.Dis.Summer., **15(3)**, 429-35 **(2005)**.

[6] S.H.Jee, R.Pastor-Barriuso, L.J.Appel , I.Suh , E.R. Miller, E.Guallar; Am.J.Epidemiol., **163(1)**, 99 **(2006)**.

[7] G.A.Kaplan, J.E.Keil; Circulation, **88**, 1973-1998 **(1993)**.

[8] M.Zamaklar, K.Lalic, N.Rajkovic, D.Trifunovic, M. Dragasevic, L.Popovic, D.Draskovic, N.Laic, A.Jotic, O.Vasovic, L.Lukic, T.Milicic, T.Potpara; Vojnosanit Pregl., **62(7-8)**, 529-36 **(2005)**.

[9] K.Arvind, R.Pradeepa, R.Deepa, V.Mohan; Indian J.Med.Res., **116**, 163-76 **(2002)**.

[10] H.V.Connerty, A.R.Briggs, E.H.Eaton (Jr.); Clin. Chem., **7**, 37-53 **(1961)**.

[11] L.L.Rudel, R.Shah, D.G.Greene; J.Lipid.Res., **20(1)**, 55-65 **(1979)**.

[12] J.Stokes3rd, W.B.Kannel, P.A.Wolf, L.A.Cupples, R.B.D'Agostino; Circulation, **75**, 65-73 **(1987)**.

[13] W.P.Castelli; Am.J.Med., **76(2A)**, 4-12 **(1984)**.

[14] W.P.Castelli, R.J.Garrison, P.W.Wilson, R.D.Abbott, S.Kalousdian, W.B.Kannel; JAMA, **256(20)**, 2835-2838 **(1986)**.

[15] S.Hulley, P.Ashman, L.Kuller, Lasser, R.Sherwin; Lipids, **14(1)**, 119-123 **(1979)**.

[16] A.Rivellese, G.Riccardi, G.Romano, R.Giacco, L. Patti ,G.Marotta, G.Annuzzi; Diabetologia, **31(12)**, 884-8 **(1988)**.

[17] H.Aberg, H.Lithell, I.Selinus, H.Hedstrand; Atherosclerosis., **54(1)**, 89-97 **(1985)**.

[18] H.Riess, W.Merk, C.Falkner, E.Hiller; Thromb. Res., **41(3)**, 281-9 **(1986)**.

[19] A.Zak, M.Zeman, E.Tvrzicka, S.Stipek, M. Buchtikova, L.Pousek, E.Novakova; Cas Lek Cesk., **139(1)**, 18-23 **(2000)**.

[20] S.K.Jain, R.McVie, Z.D.Meachum, T.Smith; Atherosclerosis., **149(1)**, 69-73 **(2000)**.