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SERUM LIPID PROFILE IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

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ABSTRACT

Serum lipids and lipoproteins were estimated in 29 patients with acute myocardial infarction during acute phase (day 1,2,3), predischarge and after three months. Serum total lipids, total cholesterol (TC) and LDL-cholesterol (LDLc) showed no significant change during the hospital stay and three months followup. HDL-cholesterol (HDLc), however, started falling from day 2 onwards with statistically significant reduction at pre-discharge and remained so at 3 months. The ratios of TC/ HDLc and LDLc/HDLc showed significant increase on predischarge day as compared to day 1. Serum triglycerides also showed an increasing trend after myocardial infarction with a significant increase on day 3 and predischarge as compared to day 1. it is concluded that the optimum time for assessment of serum lipid profile in patients with myocardial infarction seems to be within 24 hours of the acute episode.

KEY WORDS

Lipids, Lipoproteins, Myocardial Infarction

INTRODUCTION

Opinion is divided on the changes that occur in serum lipids and lipoproteins following myocardial infarction (MI). Most workers have reported a reduction in total cholesterol (1-10), HDL-cholesterol (9) and LDL-cholesterol (7-9) after acute MI. Others have, however, reported no change in serum total cholesterol (11) and HDL-cholesterol (7,11) .Similar variations have also been noted in serum triglycerides levels(4, 11-13) . From these reports it is clear that phasic changes do occur in patients following MI and therefore there is a recommendation for detection of hyperlipidaemia in patients with acute MI that the serum lipids should be assessed either within 24 hours after infarction or after 2-3 months of acute MI(10, 14-16) .While the recommendation may hold true for absolute levels there is no consensus on when ratios of various fractions of lipids should be assessed. Further, the magnitude, pattern and mechanism of these phasic changes in lipids is also not clearly outlined for our Indian subjects.

The present study was, therefore, undertaken to examine the changes in serum lipids and lipoproteins including ratios of total cholesterol/HDL-cholesterol and LDL-cholesterol/HDL- cholesterol in our Indian subjects with acute myocardial infarction.

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MATERIALS AND METHODS

The study was carried our in 29 patients (24 male and 5 female) aged between 40 to 70 years, admitted to our intensive coronary care unit with acute myocardial infarction. The diagnosis of MI was established by clinical, ECG and serum cardiac enzymes examination. None of the patients had thyroid dysfunction, liver or kidney disease. No separate control group of patients was taken as in a sequential study like this each patient is his/her own control (15).

Fasting blood samples were taken as soon as possible after admission (day 1, average 14 hours after chest pain), 24 hours later (day 2), 48 hours later (day 3), pre discharge (average day 7) and after 3 months. The dietary and life style changes where noted at 3 months followup. Only those patients where finally included who were not taking any hypolipidemic drug.

Serum total lipid concentration was measured according to the method of Zollner and Kirsch(17) whereas serum total cholesterol, HDL-cholesterol and triglycerides were estimated by readymade kits using enzymatic methods. LDL-cholesterol was calculated by Friedewald equation(18). Inter-assay coefficients of variation for total lipids, total cholesterol, HDL-cholesterol and triglycerides were 1.08%, 1.01%, 6.28% and 1.52% respectively.

RESULTS AND DISCUSSION

The results of the present study have been shown in table 1. We found no significant change in serum total lipid and total cholesterol levels either during the acute phase or after 3 months of MI. While confirming the findings of Berlin (19) and Heldenberg *et al.* (11), our

study provides information which is in direct contrast to that by others who found either a decrease (2,4,6,9,10) or an increase (20) during the acute phase of MI. Although there was a decline in total cholesterol level on day 3 but it failed to reach statistical significance. Moreover, the pattern was almost same in males and females. Considering the pattern in individual patients the TC either did not change or decreased a bit (about 10%) on day 3.

HDL-cholesterol levels, in our study, started falling from day 2 onwards, the reduction achieving statistical significance in pre-discharge specimens ($p<0.001$) and remained so at 3 months follow up ($p<0.01$) as compared to day 1. Ryder *et al.* (9) have also reported a significant reduction in HDL-cholesterol on predischarge day but in their study it tended to normalize after 3 months. In contrast to our findings of reduction in HDLc concentration other studies have shown either a rise (10) or no change (11) in HDLc. An inverse relationship between TG and HDLc has been reported (27) and in our subjects too, this appears true. However, the low levels of HDLc at 3 months in our study indicate an irreversible decrement rather than an acute reversible change and since there were no preinfarction values of HDLc known, the effect of infarction can only be assumed and by no means proven (11)

LDL cholesterol recorded no significant change throughout the study period 1(Table 1) in our subjects. However, a significant decrease in LDL-cholesterol following MI has been reported by others (8,9).

Several studies have advocated the value of ratios of LDLc/HDLc and TC/HDLc as a correlate of the severity and extent of coronary artery stenosis (22,24,25). The present study showed an increase in the ratios of TC/HDLc and LDLc/HDLc on predischarge day as compared to day 1($p<0.01$ and <0.05 respectively). This was due to significant decrease in HDLc on predischarge day.

Serum triglycerides showed an increasing trend after MI with a significant increase on day 3($p<0.01$) and predischarge ($p<0.001$) as compared to day 1. The values at 3 months were higher than day 1 but the difference was not statistically significant. This finding was in accordance with those by others(8,11). On the contrary, Vetter *et al.* (23) recorded a progressive fall in triglycerides levels from the second hour after MI and Ryder *et al.* (9) found no significant change in triglycerides. The mechanism of increase in triglycerides after MI may be due to elevated flux of fatty acids and impaired removal of VLDL from the plasma (12). Another possible mechanism for elevated TG levels may be the effect of β-blockers but this contention seems to be invalid for increased TG levels on day 3 and pre-discharge (day 7) as β-blockers take about 2 weeks to show their effect on serum lipids (27). Moreover, the TG levels came down to near day 1 values even in patients taking β-blockers. However, the normalization of TG levels at 3 months nearing day 1 levels could be due to diet restrictions and changes in overall lifestyle of the patients.

Thus, our study reveals some significant alterations in serum HDL- cholesterol, triglycerides and ratios of TC/HDLc and LDLc/HDLc after MI. However, we did not find significant changes in serum total lipids, total cholesterol and LDL-cholesterol. Therefore, the optimum time for estimation of serum lipids in patients with MI appears to be within 24 hours of acute episode. To the best of our knowledge there is no such study

Table 1: Serum lipids and lipoproteins in patients with acute myocardial infarction

Schedule	Total Lipids (mg/dl)	Total cholesterol (mg/dl)	HDLc (mg/dl)	LDLc (mg/dl)	Triglycerides (mg/dl)	Tc/HDLc	LDLc/HDLc
Day-1	484.8±142.6	187.9±38.7	41.7±8.3	123.7±55.8	112.7±55.8	4.68±1.44	3.11±1.13
Day-2	478.9±135.05	187.3±42.9	40.9±7.6	118.4±7.6	137.7±52.8	4.70±1.30	2.97±1.11
Day-3	481.4±137.0	176.9±40.9	38.1±7.65	106.6±33.5	161.7±57.4**	4.78±1.26	2.90±1.01
Pre-discharge	496.5±142.6	190.5±38.1	33.8±7.46***	123.0±33.5	168.2±54.6***	5.85±1.65**	3.83±1.35*
3 months	462.3±112.9	183.2±37.4	35.3±6.0**	121.8±6.0	130.8±51.8	5.33±1.44	3.55±1.19
	n = 29	n = 29	n = 29	n = 29	n = 29	n = 29	n = 29

Values are mean ± S.D.

*, **, *** significance of difference as compared to Day 1: *p<0.05, **p<0.01, *** p<0.001

available in Indian subjects residing in India. The mechanism of these changes is still not clear. Could it be a metabolic effect of stress, hormones etc.? One recent study has shown that acute myocardial infraction causes a profound up regulation of cholesterol synthesis as acute phase response and the observed decrease of plasma cholesterol levels after acute myocardial infarction must, therefore, be explained by the parallel increase of LDL receptor activity and thus increased cholesterol catabolism (26). The mechanistic aspect of these changes deserves further investigations with larger number of patients.

REFERENCES

1. Biorch, G., Blomquist, G. and Sievers, J. (1957) Cholesterol values in patients with myocardial infarction and normal control group. Acta. Med. Scand. 156,493-497.
2. Bjorntrop, P. and Malmcrone, R. (1960) Serum cholesterol *in* patient with myocardial infarction in younger ages. Acta. Med. Scand. 168,151-155.
3. Dodds, C. and Mills, G.L. (1959) Influence of myocardial infarction on plasma lipoprotein concentration. Lancet i, 1160 -1163.
4. Tibblin, G. and Cramer, K. (1963) Serum lipids during the course of acute myocardial infarction and one year afterwards Acta Med. Scand. 174, 451-455.
5. Fyfe, T., Baxter, R.H, Cochran, K.M. and Booth, E.M. (1971) Plasma lipid changes'after myocardial infarction. Lancet ii, 997 - 1001.
6. Kirkeby, K. (1972) Disturbances in serum lipids and in their fatty' acid composition following acute myocardial infarction. Acta Med. Scand. 192,523-528.
7. Avogaro, A., Bittilin Bon, G., Cazzalato, C., Quinci, G.B., Sanson, A., Sparla, H. and Zagatti, G.C. (1978). Variations in apolipoproteins B and A during the course of myocardial infarction. Eur. J. Clin. Invest. 8,121-129.
8. Ballantyne, F.C., Melville, D.A., Mc Kenna, J.P., Morrison, B.A. and Ballantyne, D. (1979) Response of plasma lipoproteins and acute phase proteins to myocardial infarction. Clin. Chim. Acta 99,85-92.
9. Ryder, R.E.J., Hayes, T.M., Mulligan, I.P., Kingswood, J.C., Williams, S. and Owens, D.R. (1984) How soon after myocardial infarction should plasma lipid values be assessed ? Br. Med. J. 289, 1651-1653..
10. Jackson, R., Scragg, R., Marshall, R, White, O'Brien, K. and Small, C. (1987) Changes is serum lipid concentrations during first 24, hours after myocardial infarction. Br. Med. J. 294, 1588-1589.
11. Heldenburg, D., Rubenstein, A., Levtov, 0., Berns, L., Werbin, B. and Tamir, L. (1980) Serum lipids and lipoprotein concentrations during the acute phase of myocardial infarction. Atherosclerosis 35,433-437.
12. Fredrickson, D.S. (1969) The role of lipids in acute myocardial infarction. Circulation 39,Suppl. IV-99-IV-III.
13. Swedarsen, M., Vythilingum, S., Jialal, I. and Nadar, R. (1988). Plasma lipids can be reliably assessed within 24 hours after acute myocardial infarction. Postgrad. Med. J. 64, 352-356.
14. Buckley, B.M. and Bold, A.M. (1982) Managing hyperlipidaemias, Br. Med. J. 285, 1293-1294.
15. Watson, W.C., Buchman, K.D. and Dickson, C. (1963) Serum cholesterol levels after myocardial infraction. Br. Med. J. 2, 709-712.
16. Schlant, R.C. and Digirolamo, M. (1978) Modification of risk factor in the prevention and management of coronary atherosclerotic heart disease: The Heart, 4th Ed., Hurst J.W., Logue R.B., Schlant R.C., Wenger N.K., New York: McGraw Hill Book *Co.*, p 1311-1344.
17. Zollner, N. and Kirsch, K. (1962) Absorptimetric determination of total lipids in serum. Z. Ges. Exp. Med. 135, 544-549.
18. Friedewald, W.T., Levy, R.I. and Fredrickson, D.S., (1972) Estima,tion of the concentration of low-density lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. Clin. Chem. 18, 499-502.
19. Berlin, R., Oldfelt, C.O. and Vikrot, 0. (1969) Acute myocardial infraction and plasma phospholipid levels. Acta Med. Scand. 185, 439-442.
20. Dyerberg, J., Bang, H.O. and Neilsen, J.A. (1970) Plasma lipids and lipoproteins in patients with myocardial infarction and in a control material. Acta Med. Scand. 187, 353-359.
21. Glomset, J.A. (1968) the plasma lecithin: cholesterol acy]transferase reaction. J. Lipid Res. 9, 155-167.
22. Natio, H.K. (1985) ,The association of serum-lipids, lipoproteins and apolipoproteins with coronary artery disease assessed by coronary arteriography. Ann. N.Y. Acad. Sci. 454, 230-238.

23. Vetter, N.J., Adams, W., Strange, R.C. and Oliver, M.F. (1974) Initial metabolic and hormonal response to acute myocardial infarction. Lancet i, 284-289.

24. Zampogna, A., Luria, M.H., Mancibens, S.J. and Luria, M.A. (1980) Relationship between lipids and occlusive coronary disease. Arch. Intern Med. 140, 1067-1069.

25. Luria, M.H., Erel, J., Sapoznikov, D., Gotsman, M.S. (1991) Cardiovascular risk factor clustering and ratio of total cholesterol to high-density lipoprotein cholesterol in angiographically documented coronary artery disease. Am. J. Cardiol 67, 31-36.

26. Pfohl, M., Schreiber, I, Liebich, H.M., Hairing, H.U. and Haffmeister, H.M. (1999) Upregulation of cholesterol synthesis after acute myocardial infarction-is cholesterol a positive acute phase reactant ? Atherosclerosis 142, 389-393.

27. Clucas, A. and Miller, N. (1988) Effects of Acebutolol on the serum lipid profile. Drugs 36 (suppl.2), 41-50.