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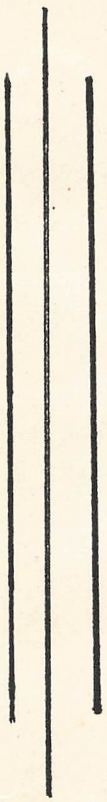

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Measurement of HDL-C (High Density Lipoprotein Cholesterol) concentration had been undertaken for 30 acute Myocardial Infarction and 31 control subjects. The concentration of HDL-C was measured by the precipitation method due to BURSTEIN SAMAILLE, which is modified by LOPES-VIRELLA et al; cholesterol concentration was measured by enzymatic method due to ROSCHLAU GRUBER, using BOEHRINGER-MANHEIM REAGENT KIT no. 400971 and no. 231347.

It had been found that the concentration of HDL-C was higher in acute myocardial infarction (44,45 mg/dl) than control (34,55 mg/dl) and the difference was significant ($p < 0,02$). Other measurements were performed, total cholesterol was lower in acute myocardial infarction than control subjects ($p > 0,05$); Triglyceride was also lower than the control subjects and the difference was significant ($p < 0,05$).

Risk factors for cardiovascular disease have been discussed in the medical literature for many years. Their significant in myocardial infarction has been intensively investigated and graded in numerous studies. The factors are : hypercholesterolemia, cigaret smoking, hypertension, overweight, lack of exercise, diabetes mellitus, gout and hyperuricaemia, policythemia It must nowadays be regarded as definite that risk factors accelerate the development of arteriosclerotic proceses.

Not only the lipids, but also their transport-forms, the lipoproteins, are of significance for the atherogenic potential. Particularly feared because of their high atherogenic potential are the low density lipoproteins (LDL), which transport the larger part of the cholesterol in blood. However, the lipoproteins of the very low density (VLDL), which mainly transport triglycerides, also facilitate the development of atherosclerotic changes. Risk factors represent, however, only a part of the problem. (1,4,6,8,9,10, 12,13,14,16).

Since Miller and Miller 1975 pointed out that in cases of increased myo - cardial infarction risk, the concentration of the high density lipo- - proteins (HDL) in blood is lowered.

One speaks of protective factors of HDL-Cholesterol for the heart and vessels (anti-risk factors).

Miller and Miller proposed the hypothesis that this lipoprotein fraction transports cholesterol from the peripheral tissue to the liver, where it is broken down and excreted. A deficiancy in HDL would hence lead to deposition of cholesterol in the tissue of the arteries (atheroma).(7,8, 12,13.)

During acute myocardial infarction, what is the concentration of HDL-Cholesterol ?

According to Shulman et al (15) and Bellantyne et al (2), they had shown an increased concentration of HDL-Cholesterol in acute myocardial infarction. The purpose of this paper is to evaluate HDL-Cholesterol concentration during acute myocardial infarction.

MATERIALS AND METHODS.

30 acute myocardial infarction patients of Indonesian nation, aged 35 years and over, with the mean age was 54 years, admitted to the Intensive Care Coronary Unit at Ciptomangunkusumo Hospital, between February and July 1980, were the materials in this paper.

The diagnosis of acute myocardial infarction was based on the criteria of WHO (typical chest pain, significant q-wave and ST elevation, and increase in blood enzymes- CPK - SGOT - LDH ∝ HBDH).

31 healthy men who had undertaken medical check-up, including treadmill test with negative result, at Cipto Mangunkusumo Hospital, were taken as control subjects; and the mean age was 49 years.

5 ml of venous blood samples mixed with EDTA were taken after an overnight fast of 12 hours, always after one day of hospitalization.

The concentration of HDL-C was determined by the precipitation method due to BURSTEIN-SAMAILLE, which is modified by LOPES-VIRELLA et al and cholesterol concentration was determined by enzymatic method due to ROSCHLAN-GRUBER, using BOEHRINGER-MANNHEIM REAGENT KIT no. 400971 and no. 231347. Total cholesterol and triglyceride were also determined by the Lieberman-Buchard method and enzymatic method (3,11).

RESULTS

By using student's t-test, the results are as follow : (Table I and II)

1. There is significant difference of HDL-Cholesterol concentration between

Acute Myocardial Infarction (AMI) and control subjects ($p < 0,02$). HDL-C concentration is higher in AMI than in control subjects.

2. There is no significant difference of total cholesterol concentration between AMI and control subjects ($p > 0,05$). Total cholesterol is lower in AMI than control subjects.
3. There is a significant difference of Triglyceride concentration between AMI and control subjects ($p < 0,05$). Triglyceride concentration is lower in AMI than in control subjects.

DISCUSSION :

It had been emphasized in the Framingham Study, that those people with low HDL-C concentration, had propensity to sustain myocardial infarction (6). The result found here in acute myocardial infarction differed, the concentration of HDL-C was higher than in control subjects ($p < 0,02$) RICHARD SHULMAN et al (15) had shown that on the first day of AMI had the highest concentration of HDL-C, then decreased on the second day, and increased again on the 28^{th} and 56^{th} day to nearly the same value as the first day after AMI. (See Table III).

The concentration of total serum cholesterol decreased on 72 hours after admission, and HDL-C decreased on the 5^{th} and 7^{th} day after AMI. Suppression of HDL-C levels persisted long after total cholesterol approached admission levels (15).

According to Bellantyne Table IV, The concentration of HDL-C on the first day was higher in AMI than in control subjects ($p < 0,01$); on the third day about the same, and after 14^{th} day decreased significantly ($p < 0,05$); after one month and three months HDL-C concentration were nearly the same as the first day after admission.

Total serum cholesterol concentration increased significantly on the first day and the third day after admission, and then no change until 3 months. Triglyceride concentration was lower in AMI than in control subjects and the difference was significant ($p < 0,01$). (2)

The changes in the lipids and lipoproteins are probably part of the general metabolic response to trauma. So the determination of HDL-C concentration during acute myocardial infarction can not be regarded as diagnostic and basic for therapy and prognosis.

SUMMARY.

Serum HDL-C, Triglyceride and total cholesterol were performed in 30 patients with acute myocardial infarction and in 31 patients with healthy control subjects, above 35 years old, the mean age for the AMI group was 54 years and the control subjects was 49 years old. HDL-C concentration was higher in AMI than in control subjects ($p < 0,02$). This result is in accordance with the previous findings of RICHARD SHULMAN et al (15) and FIONA C. BELLANTYNE et al (2).

Total serum cholesterol was lower in AMI than the control subjects event hough the difference was not significant ($p > 0,05$).

This result is not in accordance with the previous findings of FIONA C. BELLANTYNE (2), but is in accordance with the findings of fyfe (5) and Pomerant (14).

Serum triglyceride concentration was lower in AMI than in control subjects ($p < 0,05$). This result is in accordance with the previous findings of FIONA C. BELLANTYNE (2).

The changes of lipids and lipoproteins concentration in acute myocardial infarction are probably part of the metabolic response to trauma.

The evaluation of HDL-C concentration during acute myocardial infarction

can not be regarded as real value as before AMI, where those who had predilection to sustain myocardial infarction, their HDL-C concentration were low. (1,4,8,10,12,13,16,)

The concentration of total cholesterol and triglyceride can not also be regarded as real value. The changes of HDL-C, triglyceride and total cholesterol occurred during and after AMI.

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Table I.

The Concentration of HDL-Cholesterol (HDL-C), Triglyceride (TG), and Total Cholesterol (C) in ACUTE MYOCARDIAL INFARCTION.

LABOR EXAM. / STATISTICS	C	TG	HDL-C
X	220.53	103,45	44,45
SD	58,13	56,47	7,71
N	30	30	30

Table II.

The Concentration of HDL-Cholesterol, Triglyceride and Total Cholesterol in Control Subjects.

LABOR EXAM. / STATISTICS	C	TG	HDL-C
X	223,94	139,68	34,55
SD	41,68	55,99	7,81
N	31	31	31

Table III.

HDL-Cholesterol and Cholesterol Concentration Changes In Peri-Infarction (R. SHULMAN).

D A Y	C	HDL-C
1	209	50
2	283	49
3	175	47
5	173	39
7	173	36
9	188	37
11	193	37
13	202	38
28	203	41
56	211	45

Table IV.

Lipid Concentration In Survivors Of Myocardial Infarction And In Control Subjects (FIONA C. BELLANTYNE).

Results as mean ± S.E.M. Lipids m mol/L.

	DAY: 1	DAY: 3	DAY: 14	1 MONTH	3 MONTHS	CONTROL SUBJECTS.
Total Cholesterol	6,17±0,24	5,62±0,21	5,75±0,26	6,57±0,24	6,30±0,22	4,92±0,20
Total Triglyceride	1,20±0,12	1,65±0,07	1,62±0,08	1,86±0,15	1,68±0,15	1,40±0,09
HDL-Cholesterol	1,30±0,07	1,35±0,06	1,18±0,06	1,27±0,06	1,33±0,08	1,06±0,06

REFERENCES.

1.Assmann G. Schriewer H., Role of Low Density and High Density Lipoproteins in Atherogenesis, Boehringer Manheim, 1-7, 1979.

2.Bellantyne F.C.,et al :
Response of Plasma Lipoproteins and Acute Phase Proteins to Myocardial Infarction, Clinical Chemica Acuta, 99 : 85-92, 1979. Elsevier / North Holland Biomedical Press.

3.Boehringer Manheim, HDL-Cholesterol, The Key to Differential Diagnosis of Lipid Disorders.

4.Castelli W.P. Doyle J.T. et al. :
HDL-Cholesterol and Other Lipids in Coronary Heart Disease.
Circulation 55 : 767-772, May 1977.

5.Fyfe T., et al :
Plasma Lipid Changes After Myocardial Infarction.
Lancet, 997-1001, November 1971.

6.Gordon T. Casteli W.P. et al:
High Density Lipoprotein as a Protective Factor Agains Coronary Heart Disease.
Am J Med 62 : 707-714, May 1977.

7.Hurst J.W. The Heart, Arteries and Veins, Etiology of Coronary Atherosclerosis , 987-1002, Mc Graw Hill, KOGAKUSHA ltd, 1974.

8.Jenkins P.J. et al :
Severity of Coronary Atherosclerosis Related to Lipoprotein Concentration.
Br. J. 2 : 388-391, 1978.

9.Jones R.J. :
The Hyperlipoproteinemis, Detection, Diagnosis and Management Symposium on Coronary Heart Disease.
Medical Clinics of North America, Vol 57, no 4 : 47-61, Jan 1973.

10. Kannel W.B. Castelli W.P. Gordon T,
Cholesterol in The Prediction of Atherosclerotic Disease.
An. Int. Med. 90 : 85-91, 1979.

11. Lopez-Virella M.F. et al :
Cholesterol Determination in High Density Lipoproteins Seperated by Three Different Methods, Clin, Chemistry 22: 882-884, no.5, 1977.

12. Miller N.E. Weinstein D.B., et al :
Interaction Between High Density and Low Density Lipo - proteins During Uptake and Degradation by Cultured Human Fibroblasts,
J Clin Investigation 60 : 78-87, July 1977.

13. Miller N.E. Plasma Lipoproteins, Lipid Transport and Atherosclerosis Recent Advance, J.F. Clin Path 32 : 639-650, 1979.

14. Pomerantz H.Z. MD,:
Effect of Acute Myocardial Infarction Upon Serum Cholesterol Levels.
The Canadian Med Assoc. Vol 86, no.8 : 355-359, Feb 24,1962.

15. Shulman R. et al :
Time course of Peri-Infarction Changes In Cholesterol and HDL-Cholesterol, Abstracts of the 2nd Scientific Sessions Circulation, Vol 59 and 60, Supp II, Oct. 1979.

16. Williams P, Robinson D., ALan Bailey, High DensityLipoprotein and Coronary Risk Factors in Normal Men.
Lancet 72-75, January 13, 1979.
