Total Cholesterol and High Density Lipoprotein Cholesterol in Myocardial Infarction Patients and Controls.

WAHEED A. SAHIBZADA *
ZAHOOR AHMAD
TASLIM AKHTAR

SUMMARY

Total Cholesterol (TC) and high density lipoprotein Cholesterol (HDL—C) was measured in a group of Myocardial Infarction patients and controls. The mean TC was 206 ± 4.49 SEM in the patient group and 209.7 ± 3.54 SEM in the control group. In patients less than 40 years of age there was a statistical difference between the TC of the two groups at the .01 level the values being 225 and 207.5; also there was a difference in the HDL—C values but it did not reach statistical significance. Lipoproteins may not be a major contributory factor to coronary artery disease in our population or may be operative at a lower level.

INTRODUCTION

Atherosclerotic Coronary heart disease has reached epidemic proportions in the developed countries of the Western world where mortality from it is approximately 1000 per 100,000 population (1). The prevalence of the disease in the tropical countries including Pakistan was thought to be low (2,3,4). However, the hospital admissions from Coronary Heart Disease, appear to have increased dramatically over the past few decades (5,6); even more disconcerting is the relatively high proportion of young individuals, less than forty years of age, afflicted by the disease.

The aetiology of Atherosclerotic Coronary artery disease (CAD) still eludes us. Epidemiologic studies in the west have identified certain risk factors that are associated with CAD. These include a high cholesterol level, smoking and hypertension (18). Recently subfractionation of the total cholesterol has allowed separate evaluation of the low density lipoprotein fraction (LDL—C) and high density lipoprotein fraction (HDL—C) vis-a-vis CAD. The LDL—C is thought to have a deleterious effect and the HDL—C a protective effect.

Data regarding risk factors in CAD in hospitalised patients suffering from acute Myocardial Infarction, in Pakistan, was collected in a multicentre study by the Pakistan Medical Research Council (PMRC) (7). This, however, did not include fractionation of the cholesterol into LDL—C and HDL—C fraction. Another smaller study from Lahore(8), has looked at the lipoprotein profile in hospitalised CAD patients. The value of cholesterol in the PMRC study was high while that in the study from Lahore was low. The ratio of total cholesterol to HDL—C was not done in the PMRC study while it was low in the Lahore study. The values from Lahore are closer to the values reported in a community study by Knuiman (9) and others (10,11).

Since the T. C. and TC/HDL—C ratio from our country has been reported to be low, yet the number of patients afflicted by the disease is increasing, we decided to take another look at the lipoprotein profile in CAD with special emphasis on the younger age group. It was our

*From the Deptt. of Cardiology
Khyber Medical College, Peshawar

feeling that relative affluence and a relatively higher caloric intake from saturated fats may be changing the lipoprotein profile in the younger age group thereby contributing to a higher incidence of CAD.

METHODS

All patients who presented to the Coronary Care unit of Khyber Hospital Peshawar and who were diagnosed as having Myocardial Infarction were included in the study. The diagnosis of Myocardial Infarction was made on the basis of at least two of the following Criteria: 1) The presence of typical constricting chest pain, at least half an hour in duration. 2) Elevation of serum Enzymes CPK, SGOT LDH 3) Typical ECG changes of Acute Myocardial Infarction – Q waves, QS complexes and ST and T wave changes.

The control group was taken from the attendants of the patients, matched for age and from the same geographic location. A complete cardiovascular examination of the control group was performed. All those with an abnormal examination were excluded. None in the control group had a history of Angina or previous Myocardial Infarction.

Blood was taken for biochemical analysis after a twelve hour fast. All samples were sent to the Pakistan Medical Research Council cell at Khyber Mdical College for analysis. Serum Cholesterol was determined by the Modified Method of Lieberman Burchard; the HDL cholesterol was determined by the Phospho Tungstate/Magnesium Precipitation Method.

Results of the tests were given in mgs%. Statistical analysis were carried out and the student–T test used for calculation of the P value.

A total of ninety five cases was studied. There were only 12 females in the whole group and hence they were excluded from the calculations. The male patients were divided into three groups. A) 40 years and less in age, B) 41–60 years in age and C) greater than 60 years in age.

Because of the effect of Myocardial Infarction on the Blood Pressure this was not looked at as a risk factor.

Cigarette smoking and other forms of tobacco use were also looked at in the control and patient population.

RESULTS

Age:– The mean age of the patient group was 53.9 ± 10.3 years: of the total number of 82 cases there were ten (12.2%) aged 40 years OR less: 54 (65.8%) in the 41 – 60 years age group and 18 (22%) in the over sixty group. As is apparent from figure 1 the age distribution is similar to other reported series where the majority of patients fall in the middle aged category (7). Also a fairly significant proportion fall in the less than 40 years bracket.

Total Cholesterol (T.C)

The mean T.C in the patient group was 206.5± 4.49SEM and 209.7 ± 3.54 in the control group. These figures are substantially lower than those reported by PMRC(7) and closer to the figures reported from Lahore (8). The range of Cholesterol in both the patient and control group was quite wide. In the patient group it extended from a low of 100 mgs% to a maximum of 345 mgs% while in the control group it was 121-320 mg% respectively.

Recommended cholesterol values for adults are 200 mgs% and below; 201-260mgs% are probably abnormal and greater than 260 mg% definitely abnormal (12). (12). Analysing our data according to these values we had eight

AGE DISTRIBUTION

Total No of Cases = 82

Mean AGE (yrs.) = 53·9 ± 10·3

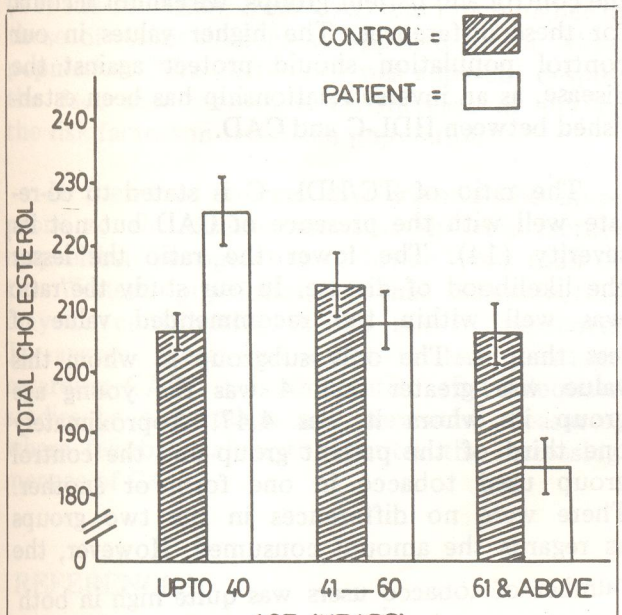
AGE GROUP	No. of Cases	%
Less than 40	10	12·2
41–60	54	65·8
61 and greater	18	22.0

Figure – I

Age distribution and percentages.

patients (Less than 10%) in the definitely abnormal category of whom three were forty years or less in age. Equal numbers, that is 37 patients respectively were in the 201-260 mg% and < 200mg%.

The mean cholesterol in the three patients categories shows a fall in the patient group as the age increases whereas in the control group there is no significant age related change as is shown in figure 2.



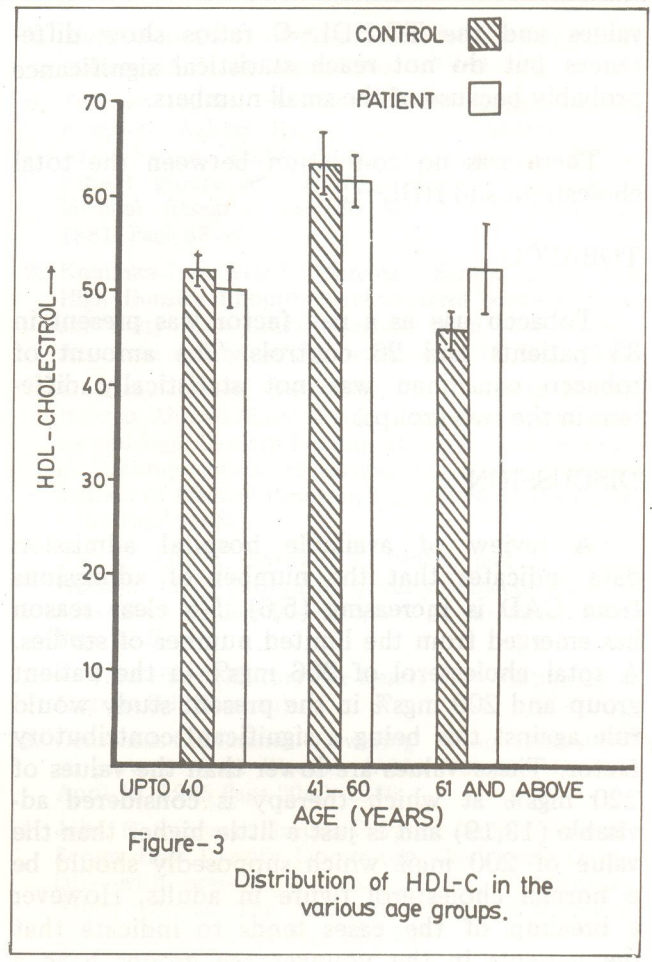
Figure–2

Distribution of total cholesterol in the various age groups.

HDL-CHOLESTEROL (HDL—C)

The mean HDL—C in the patient group was 56.0± 4.49 SEM while that in the control group was 56.2 ± 1.53 SEM. The values obtained in our patient group are surprisingly high compared to those reported by Zulfiqar Haider from Lahore (8). The values reported for the control group are comparable to those published in the literature.

The range of HDL—C in both the patient and control group was from 20-102 mgs%. There was no co-relation between age and the HDL—C even though the values in both controls and patients was higher in the middle age group (Figure 3).



Figure–3

Distribution of HDL-C in the various age groups.

RATIO OF T. C. TO HDL—C

The overall ratio in the patient group was 3.69 while it was 3.73 in the control group. Ratios obtained in the West indicate that the lower the ratio the lesser the likelihood of disease. Values greater than four are associated with a higher risk. In both our groups the values are in the normal category.

AGE GROUP LESS THAN 40 YEARS

The total cholesterol in the patient group was 225 ± 15.5 mg whereas that in the control group was 207.5 ± 2.1 mgs. The difference between the two values is significant at the 0.01 level. Only four patients had a total cholesterol of less than 200 mg%. Similarly the HDL—C in the patient group was 50.40 ± 5.89 as against 55.31 ± 2.76 in the control group; the RESULTANT TC/HDL—C ratios were 4.47 and 3.76 in the two groups respectively. Both the HDL—C

values and the TC/HDL–C ratios show differences but do not reach statistical significance probably because of the small numbers.

There was no co-relation between the total cholesterol and HDL–C.

TOBACCO

Tobacco use as a risk factor was present in 33 patients and 26 controls. The amount of tobacco consumed was not statistically different in the two groups.

DISCUSSION

A review of available hospital admission data indicates that the number of admissions from CAD is increasing (5,6). No clear reason has emerged from the limited number of studies. A total cholesterol of 206 mgs% in the patient group and 209 mgs% in the present study would rule against this being a significant contributory factor. These values are lower than the values of 220 mgs% at which therapy is considered advisable (13,19) and is just a little higher than the value of 200 mg% which supposedly should be a normal cholesterol figure in adults. However a breakup of the cases tends to indicate that the patients in the younger age groups have a higher cholesterol value which in turn may be secondary to a change in dietary habits as a result of relative affluence in the country over the past few decades (personal observation).

The mean HDL-C observed in our study is comparable to the values reported from other centres abroad (14,15). However these values are greater than those reported from Pakistan (8,9). The values obtained in our patient population are at variance with those already reported in that there is no significant difference between the control and patient groups. We cannot account for these differences. The higher values in our control population should protect against the disease, as an inverse relationship has been established between HDL-C and CAD.

The ratio of TC/HDL–C is stated to co-relate well with the presence of CAD but not its severity (14). The lower the ratio the lesser the likelihood of disease. In our study the ratio was well within the recommended value of less than 4. The only subgroup in whom this value was greater than 4 was the young age group in whom it was 4.47. Approximately one third of the patient group and the control group used tobacco in one form or another. There were no differences in the two groups as regards the amount consumed. However, the number of tobacco users was quite high in both

Age Group	TOTAL CHOLESTEROL		HDL–C		TC/HDL–C	
	Patients	Controls	Patients	Control	Patients	Controls
Less than 40 years.	225 ± 15.5 SEM	207 ± 2.15 SEM	50 ± 1.8 SEM	52 ± 2.5 SEM	4.5	3.9
41-60	208 ± 4.8 SEM	215 ± 3.9 SEM	62 ± 3.0 SEM	63 ± 3.0	3.3	3.4
Greater than 60 years.	185 ± 3.6 SEM	207 ± 3.7. SEM	54 ± 5.0 SEM	47 ± 1.7	3.8	3.9

FIGURE 4 Total Cholesterol, HDL–C, and Ratio of TC/HDL–C

* p = 0.01

groups. This may be more important than the cholesterol values as a risk factor.

Other factors that may be contributing to an increase of CAD may be our race, ethnic groups or socio-cultural patterns. In a recent comparative study from Birmingham, United Kingdom (16), the incidence of CADwas as high amongst Asians as the local white population whereas that in persons of West Indian Origin was much lower. In another comparative study from London (17) the mortality from CAD was much higher in those of Asian descent as opposed to the white population. This difference was apparent despite the fact that there was no increase in any of the risk factors in the Asian population.

It therefore appears that we may be more prone to the disease and that the risk factors may either be operative at lower levels than in the Western population or that other factors may be contributing to the rise in incidence. To answer these and other related questions regarding CAD, it is imperative that adequate studies be undertaken and preventive measures, where feasible, taken to counter the increasing menace of CAD.

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