JAMC, Vol. 8, No. 1, 1996

COFFEE INTAKE AND ELEVATED LIPID PROFILE IN AMI PATIENTS

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Coffee intake from diet records was studied in association with serum lipid profile concentrations in a cross-sectional sample of 60 middle aged men and women suffering from acute myocardial infarction to determine the significance and form of their interrelationships. All the subjects were evaluated for diabetes mellitus, nephrotic syndrome and hyper lipid - anemia.

Heavy drinking of coffee revealed a highly significant ($p < 0.001$) increase in levels of total cholesterol and LDL-cholesterol. Graphic analysis revealed that serum concentrations of total cholesterol and LDL-cholesterol were positively related, which were statistically less significant ($p < 0.01$) to intake of upto 3 cups of coffee per day and more significant ($p<0.001$) with intake exceeding 3-4 cups. These results suggest that heavy coffee drinking patients of acute myocardial infarction have significant increases in levels of lipoprotein profiles when compared to those faking no coffee.

INTRODUCTION

Epidemiological studies have demonstrated that serum lipoprotein concentrations are strongly predictive of the risk of atherosclerotic heart disease. The incidence of coronary events has been shown to the positively associated with low density lipoprotein (LDL) cholesterol both in population comparisons and in studies conducted within populations[1]. The relation between high density lipoprotein (HDL) cholesterol and coronary disease is more complex. A negative correlation between the HDL cholesterol level and coronary mortality has been a consistent finding in studies within population [2]. On the other hand, the mean level of HDL cholesterol appears to be high in populations with a high incidence of coronary disease and low in populations in which ischemic heart disease is rare [3,4].

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Environmental factors known to influence plasma lipoprotein levels include physical exercise, smoking, consumption of alcohol, hormones, drugs and the composition of the diet [2"]. It has been amply documented that a diet rich in saturated fats and cholesterol raises the level of LDL-cholesterol, whereas an opposite change is observed when the proportion of polyunsaturated fatty acids is increased [6-s]. The data on the effects of diet on HDLs are more conflicting. Although a decrease in the ratio of polyunsaturated fatty acids to saturated fatty acids (P/S ratio) has usually been associated with an elevation in HDL cholesterol, the change has not been observed in all investigations[2].

Coffee intake is reported to be associated with plasma cholesterol concentrations in some European and Commonwealth population. Studies on Americans, however, have generally failed to substantiate the coffee cholesterol connection [14] \ It has been proposed that the lower levels of coffee consumption or that differences in brewing procedures of Americans vs Europeans may partially account for these discrepant results.

Scandinavians have traditionally made their coffee by boiling ground coffee beans and water in a sauce pan and decanting the fluid into a cup. In 1983, a study from Norway showed a strongly significant relation between the amount of coffee ingested and serum total cholesterol [14]. Recently, it is found that this association is

specially due to boiled coffee[17]. Controlled experiments [18'19] showed that abstaining from boiled coffee caused a large decrease in serum cholesterol in healthy volunteers [1] and in hypercholesterolemic men. In a Finnish trial [20], volunteers showed a rise in cholesterol of 0.64 mmol/1 after four weeks of consumption of 8 cups of boiled coffee per day but a fall alter consumption of regular drip filter coffee or tea. A similar cholesterol-raising effect of boiled but not of drip coffee was found in a Dutch experiment. A switch from boiled to drip coffee consumption is believed to have contributed to the fall in serum cholesterol and coronary heart disease observed in Finland m the past 20 years However, it is totally unclear why boiled coffee should cause hypercholesterolemia. Nor do we know to what extent other types of coffee such as Turkish, Greek or Espresso coffee will raise cholesterol. A report that decaffeinated coffee raises cholesterol may also indicate that the relevance of the putative hypercholesterolemic factor extends beyond Scandinavia [22]. Boiled coffee thus contains a lipid that powerfully raises serum cholesterol [23].

A relationship between coffee intake and the potential for increased cardiovascular risk would be more strongly supported if the level of consumption was shown to be associated with elevated plasma levels of apolipoprotein-B (apo) or low density lipoprotein (LDL) cholesterol, because these lipoprotein components are thought to be more directly involved with the atherosclerotic process than in the total amount of cholesterol in the plasma [24]. Thus far, coffee studies have primarily focused only on total cholesterol levels as a risk factor.

In this report we demonstrate a significant association between coffee intake and elevated serum concentrations of total cholesterol and LDL-cholesterol in Pakistani people suffering from acute myocardial infarction.

MATERIALS AND METHODS

The present study of lipid profile was undertaken on 60 subjects suffering from acute myocardial

infarction. The subjects were selected to be free of known diabetes mellitus, nephrotic syndrome, acute illness, active chronic systemic diseases and medicinal use likely to interfere with their lipid metabolism. In each case a detailed history including age, sex, socio-economic status, dietary history of activity in leisure time, family background, history of past illness and previous treatment if any, was taken and thorough clinical physical examinations were performed.

Only non-smokers and patients of acute myocardial infarction were included in the study. The diagnosis of acute myocardial infarction was made by the Professor of Cardiology (CCU, Mayo Hospital, Lahore) on the triad criteria of clinical presentation, ECG changes and abnormalities of cardiac enzymes. Patients with acute myocardial infarction involving any area of heart within 48 hours of onset of symptoms were included in the study. Subjects were divided into three groups:

Group-I: Group 1 was the Control Group which included 20 patients of acute myocardial infarction taking no coffee.

Group-II: This group consisted of 20 patients of acute myocardial infarction taking 1-3 cups of coffee daily.

Group-III: This group included 20 patients of acute myocardial infarction taking more than 3 cups of coffee daily.

The methods of dietary interview and consumption of nutrient content have been reported [2""2A]. Information of consumption of coffee included details of additives (mainly milk and sugar) but not of methods of brewing or strength of the beverage. We coded the number of cups of coffee consumed in the 24-hour period of recall according to whether the coffee was drunk with or without added milk.

Plasma lipid and lipoprotein concentrations were measured according to the protocol of the Lipid Research Clinics m blood samples drawn alter a 12 hour's fast[27] Serum cholesterol concentrations were determined with oxidase method [2S]. using a commercial kit (Human). High density lipoprotein was measured directly from the serum after precipitation of the apolipoprotein B containing lipoproteins with heparin and manganese chloride[29].The concentration of low density lipoprotein cholesterol was calculated by Friedewald formula[30]. Very low density lipoprotein in the serum was determined by Nephelometric method by using BLF Eiken [29-31]. Serum triglycerides were estimated by enzymatic method[28] Total

lipids were measured by sulphophosphovanillin reaction [33].

RESULTS

The present study reports physical, clinical and laboratory data in sixty patients of acute myocardial infarction divided into three groups. 83% of the patients were male and 17 % were females (Table 1). 70% of the subjects were between 30-60 years of age and 30% were between 60-70 years (Table 1). Age, weight, height and body mass index of the subjects of different groups are shown in Table 2 ($p > 0.05$).

Table 3 shows the mean ± S.D. levels and range values of serum lipids in different groups. Table 4 shows ratios of total cholesterol: HDL- cholesterol and LDL-cholesterol: HDL-cholesterol according to coffee consumption ($p > 0.05$). Total cholesterol levels increased with increasing coffee intake. From no coffee consumption to the highest coffee consumption category, the mean serum cholesterol level increased by 51.4 mg/dl ($p<0.001$). HDL-cholesterol level increased by 5.9 mg/dl ($P>0.05$). LDL-cholesterol level increased with increasing coffee intake. The mean LDL-cholesterol level increased by 46.2 mg/dl ($P<0.001$) from no coffee consumption to highest coffee consumption category. Serum VLDL levels decreased by 44.2 mg/dl ($p > 0.05$). The mean serum triglyceride level was decreased but showed an inconsistent pattern. The mean serum total lipids decreased by 45.3 mg/dl ($p>0.05$).

From the lowest to the highest coffee consumption category', the mean serum cholesterol level increased by 21.5 mg/dl ($p < 0.05$), the mean LDL- cholesterol level increased by 46.2 mg/dl ($p <0.01$) while the mean HDL-cholesterol level increase was statistically insignificant. The mean VLDL level increased by 41.0 mg/dl ($p>0.05$), the mean triglyceride level increased by 4.6 mg/dl ($p> 0.05$) and mean serum total lipids decreased by 12.2 mg/dl ($p > 0.05$).

DISCUSSION

Our attitude towards the present finding of a coffee- cholesterol association is strong and consistent, and its magnitude makes coffee one of the strongest determinants of serum cholesterol

levels. Why is this strong association not well established in the literature?

One of the few studies with findings that were similar to ours was reported from Norway by Bejelke in 1974 [l0]. He observed a relationship between serum cholesterol levels and coffee consumption in middle aged men. Earlier, Little et al, had found a possible relation between serum cholesterol levels and coffee consumption [n]. In a comparison between patients with myocardial infarction and controls, these workers reported consistently positive and statistically significant correlation between coffee and each of the serum lipid fractions in the patients with coronary disease. They suggested that coffee consumption elevated total cholesterol levels in susceptible subjects by stimulating lipolysis, with elevation of unesterified fatty acids, particularly in subjects prone to coronary bean disease. The coffee-cholesterol relation was also exposed in the Framingham study, but no association was observed [l6]. However, in another study comparing vegetarians with a control group randomly selected from the offspring of the Framingham population, raised levels of total cholesterol and LDL-cholesterol were associated with consumption of more than two cups of coffee a week [34]. Scandinavian style boiled coffee which raises serum cholesterol was found to contain more lipid material than (non-cholesterol-raising) drip filter coffee [23].

Thus the existing documentation of a coffee- lipid link is sparse and inconsistent. Coffee contains many substances other than caffeine that might influence lipid levels, and any of these may be the responsible component(s). The lack of association between tea and serum cholesterol levels, shown by- Little et al [11], suggests that other components maybe involved. On the other hand, even if caffeine is the component of coffee responsible for increased cholesterol levels, comparison both within and between different populations may be muddled. Caffeine is a compound found not only in coffee and tea but also in drugs and soft drinks. This, in addition to the use of decaffeinated coffee which is frequent in the United States but virtually non-existent in Norway, introduces confounding factors that are difficult to control. The frequency of adding sugar or cream or both in coffee is not known in the Tromso population, but information from the Norwegian Coffee Promotion Committee indicates that 80% of the coffee consumed in Northern Norway is black. The cholesterol-raising effect of sugar and cream in coffee is therefore thought to be minimal. It is possible that other unknown variables either

interact with coffee or act concomitantly with it in producing the cholesterol- coffee relation, but our data do not allow further exploration. In any case, it is one thing to show that a frequently occurring habit is a determinant of an established coronary risk factor and another to relate this habit to the occurrence of disease.

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