Effects of moderate alcohol intake on various lipid fractions

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In this study, effect of moderate alcohol intake on various lipid and lipoproteins, which are risk factors for coronary heart disease , was investigated. Triglyceride and apo A-I levels of moderate drinkers were significantly increased, ($p<0.01$ for triglyceride and $p<0.001$ for apo A-I) total cholesterol, LDL-cholesterol and plasma cholinesterase levels were decreased ($p<0.01$ for both total cholesterol and LDL-cholesterol, $p<0.05$ for chalinesterase). There was no significant difference between phospholipids, HDL-cholesterol and its subtractions ***(HDL2*** *and* ***HDL3)*** *of both groups. Low levels of LDL-cholesterol was thought to result from low activity of cholinesterase. As a result, moderate alcohol intake was found to have no significant useful effect on the risk factors of CHD. Also, the changes found in lipid parameters was thought to result from other metabolic products of alcohol other than alcohol itself. Our findings are discussed in view of literature [Turk J Med Res 1994; 12(2): 108-112]*

Key Words: Alcohol, Coronary heart disease, Apolipoproteins, Cholesterol

Effects of alcohol on atherosclerosis and its major complications such as coronary heart diseases (CHD) have been one of the most popular discussion in this century. Some epidemiological studies showed a negative correlation between consumption of alcohol and CHD (1) while others do not (2). There are also some studies that alcohol increases risk of CHD (3). However, the claim that consumpiton of moderate level of acohol may decrease the risk of CHD in man is becoming more common in recent years (4,5). The negative correlation between CHD and consumption of moderate level of alcohol is attributed to increasing of HDL-cholesterol (HDL-C) level by some researchers (6.7) while others think this is because of Apo A-I levels (8-10). On the other hand, there have been a number of literature related to the health and social problems of alcoholism. At present there are many diseases caused by alcohol (11).

In this study, the effects of moderate alcohol intake on some lipid fractions, which are the risk factors for CHD, were investigated. For this reason, total cholesterol (T-chol), triglyceride (TG), LDL-cholesterol (LDL-chol), HDL-cholesterol (HDL-chol) and its subsfractions, phospholipid (PL),, apo A-I and plasma cholinesterase activities were analyzed.

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MATERIALS AND METHODS

This study was performed on 58 healthy male aged 23 to 51 (mean 34.74) years old, as a control group, and 73 male who had been drinking moderate levels of alcohol for two years but had no clinical sign and symptoms such as neuropathy and chronic hepatitis. "The study group" were 21 to 61 (mean 36.7) years old living in Konya. This group was chosen carefully making sure that they were drinkers of moderate level of alcohol, called "social drinkers". For this purpose, classification of Andrade et al (8) was taken as a reference. According to that classification, persons having 30 to 65 gr of alcohol per day were accepted as "moderate alcohol drinkers". Calculations were roughly made by the types of alcohol consumed and by the information given by the drinkers. It was estimated that (a) half bottle of beer and wine and a double of spirit contained 10 g of alcohol (8,11).

Table 1. Results of both controls and patients

Parameters		Controls mean±SD	Alcoholics meamSD	t	P
Total chol.	(mg/dl)	200.87+5.14	178.04+4.85	3.230	p<0.01
HDL-chol	(mg/dl)	47.45+1:66	47.50+1.55	0.022	p>0.05
HDL2-chol.	(mg/dl)	10.53±1.21	11.52+1.25	0.569	p>0.05
HDL3-chol.	(mg/dl)	45.09+1.87	42.72+2.20	0.820	p>0.05
LDL-chol.	(mg/dl)	123.17±5.48	102.03+4.30	3.034	p<0.01
TG.	(mg/ml)	135.62±6.78	170.53±8.36	3.243	p<0.01
Phos.lipid	(mg/dl)	187.75±4.04	188.47+3.64	0.132	p>0.05
Apo A-I	(mg/dl)	134.28+2.95	152.67±3.70	3.886	p<0.01
PCHE.	(U/L)	7421.9+173.5	6863.4±205.7	2.074	p<0.05

From the both groups, venous blood was taken by venapuncture after an overnight fasting. All assays were performed with commercial kits.

The method of double precipitation established by Warnick et al (12) was used for determination of HDL subtractions In that method, total HDL-chol was determined by using a precipitation solution containing 10 g of dextran sulphate (MW. 50.000) and 0.5 mol of MgCl2. 6H2O per liter. And then 1 mol (3203.3 gr) of MgCl2. 6H2O was added into the above solution to make the final concentration of 1.5 mol MgCl2/dl. By addition of the latter solution to the former supernatant HDL2 was precipitated and HDL3-chol was determined from supernatant. HDL2-chol was calculated by subtracting HDL-3 chol amount from total HDL-chol. Unfortunately, in some samples no precipitation was obtained. Therefore, only the samples which HDL2-chol precipitation was obtained. Therefore, only the samples which HDL2-chol precipitations occurred were included in the study.

RESULTS

The results from both groups are given in Table-1. As seen from the table, levels of TG and apo A-I in alcohol group were significantly higher than those of controls, whereas T-chol, LDL-chol levels and plasma cholinesterase activities were found to be lower in the study group. There was no stastically significant difference in the other parameters assayed between both groups.

DISCUSSION

Extensive use of alcohol results in severe impairments of lipid and lipoprotein metabolisms (3,13).

There were various findings in the effects of alcohol on T-chol levels. Some investigators showed increased levels of T-chol in serum with ethanol (8,14) while others not (2,7,10,15). These differences may depend on the amount and duration of alcohol consumption. In our study, serum T-chol levels were significantly low (p<0.01) in the alcoholics like the findings of Handa et al (2), Taskinen et al (7) and Danielson et al (16).

The decrease in serum cholesterol of the study group might be caused by changes in the synthesis and absorbtion of cholesterol as well as bile acid synthesis or excretion. Because bile acids partly enter the enterohepatic circulation (17). Any impairment in this circulation decreases serum cholesterol levels. Such an obstruction can be done experimentally to lower serum cholesterol levels (18). Since alcohol causes malnutritions and absorbtion abnormalities (3,19), this decrease may occur with the impairment of bile acid reabsorbtion. In their study, Nestel et al (20) found that 3 hyperlipemic and 4 normolipemic persons having 37 % of calory from alcohol in their diet showed no change in the synthesis and excretion of cholesterol, but the hyperlipemics had increased levels of bile acid excretion.

The level of serum cholesterol may be regarded as the sum of HDL-chol, LDL-chol and, to a lesser extend, VLDL-chol amount (21). In our study, in contrast to HDL-chol levels, LDL-chol levels was found to be low in the study group. This was correlated to the decrease in serum cholesterol levels. On the other hand, this decrease in cholesterol levels might be a resut of negative effects of alcohol on the liver (22,23). We could not find any statistically significant difference in HDL-, HDL2- and HDL3- chol levels of both groups, and that agreed with the findings of Moore et al (10).

Increased levels of HDL-chol in alcoholics has been shown as a reason for the negative correlation btetween alcohol and CHD (6,24,25). However, up to date, that claim has not been proven to be so. In contrast, some investigators found no relation between alcohol and HDL-chol levels (10). On the other hand, Laporte et al (26) found high level of HDL-chol correlated with high levels of liver enzymes, especially SGOT(10).

Even if HDL-chol levels increase with alcohol intake, it is accepted that HDL2-chol is the only fraction having a negative correlation with CHD (6).This situation was confirmed by Ballantyne et al (27).

Taksinen et al (7) showed increases in HDL3-chol as well as HDl_2-chol in alcoholics. Haksel et al (6) investigated changes of HDL-chol and its subtractions when alcohol intake was stopped, and found that alcohol increased HDL-chol and HDL3, but did not change the level of HDL2. This led to the conclusion that the negative correlation between CHD and alcohol was not dependent on rises of HDL2 levels. Moore et al (10) showed that alcohol increased only the levels of Apo A-I, but caused no change in the levels of HDL-chol, HDL2 and HDL3.

As seen from above discussions, the results obtained from various studies on alcohol-HDL-chol correlations are not in aggreement to each other. This may be due to the difference in study groups chosen, since, especially in Western Countries, control groups were mostly chosen among persons stopping alcohol intake for some time. In addition, in these countries study groups were generally heavy drinkers. In our study, controls never had alcohol in their life span. Hence, we believe that our results are more reliable than others. However, we think that more studies for the effects of alcohol on HDL-chol and its subtractions is needed.

In general, a negative correlation was found between alcohol intake and LDL-chol levels (10,14). Taksinnen et al (7) showed that alcoholics had a insignificant decrease in their LDL-chol levels. Also Castelli et al (21) found the same correlation in their broad studies separately. Like these two groups of researchers, we found also low level of LDL-chol in the study group ($p<001$).

In relation to the effects of alcohol on LDL-chol metabolism, various suggestions have been made. For example, acetaldehyde, an intermediate compound of alcohol metabolism, may decrease VLDL levels (28). In our study, we determined the activity of plasma cholinesterase, which has a role in LDL metabolism, in order to explain this" phenomenia. This enzyme takes role in conversion of VLDL to LDL (29,20). We found that the enzyme activity was very lower in. the study group than controls ($p<0.05$). As a result of this, production of LDL from VLDL, and subsequently levels of LDL-chol decreases.

In literature, we have found no study showing the relationship between cholinesterase activity and LDL-chol levels in alcoholics. So this is the first study showing the correlation between them, but needs further and detailed investigations.

In agreement with other researchers, we found a significant increase in serum triglyceride levels in the alcoholics ($p<0.01$) (2,13,14,22,30).

It is believed that alcohol-induced fatty liver results from the impairments of hepatic triglyceride synthesis and secretion. In this respect, many researchers established that free fatty acid levels rises after experimental alcohol drinking and that leads to rise in TG levels and hence fatty liver (20,26). Moreover, increased triglyceride synthesis with alcohol has also been shown in "in vitro" studies (31).

On the other hand, apo A-I, the major protein portion of HDL-chol, is affected by alcohol intake, and decreases the risks of CHD (28,32). Camargo et al (9) showed a positive correlation between alcohol and apo A-I when moderate level of alcohol was used. Moore et al (23) showed that alcohol increased the level of apo A-I without changing the levels of HDL-chol and its subtractions when low amounts of alcohol was consumed we also have found that Apo A-1 level of alcohol groups was increased with no change in HDL subtractions ($p<0.001$) in the study group. The increase in Apo A-I level was thought to be related to the induction of microsomal enzyme system (28).

These results support the idea that there is a negative correlation between apo A-I and CHD. BBut, it is still not known that the increase in Apo A-I levels occurs in which fraction of HDL. Camago et al (9) claimed that most of Apo A-I present in HDL3, and the decrease in Apo-A-I results from the decrease of HDL3 when alcohol intake ceases. It is well known that there is no correlation between HDL3 and CHD (6,27).

In conclusion, alcohol leads to various Impairement in lipid and lipoprotein metabolism. This situation is due to the changes in synthesis, secretion and catabolism of lipoproteins. Moderate level of alcohol consumption may decrease effects of psychologic stresses rather than making positive effects on metabolism of lipid and lipoproteins related to CHD. Stres is regarded as a possible risk factor for CHD (2,33).. In this respect, alcohol may reduce this risk by decreasing person's stresses when consumed. However, it should not be forgotten that alcohol is a toxic substance causing various damages in whole body (10), especially in liver which is a vital organ. Since usually cigarette is smoked together with alcohol consumption, this also increases the risk for CHD (3). Also, other constituents of alcoholic beverages such as aldehydes, keton bodies, organic acids and esters, aliphatic and aromatic substances may have adverse effects on important metabolic processes and organs (34).

jna derecede alkol alımının çeşitli lipid fraksiyonları üzerine olan etkileri

Bu çalışmada orta derecede alkol içiminin, koroner kalp hastalığı için risk faktörleri olan çeşitli lipid ve lipoproteinler üzerine olan etkileri araştırıldı. Orta derecede alkol içenlerde kan etil alkol, trigliserid ve Apo A-I seviyeleri istatistik/" açıdan önemli oranda yüksek (etil alkol için p<0.05, trigserid için p<0.01, Apo A-I için p<0.001) bulunurken total kor lesterol, LDL kolesterol ve plazma kolinesteraz seviyeleri (total kolesterol ve LDL kolesterol için p<0.01, plazma kolinesteraz için p<0.05) düşük bulundu. Fosfolipid, HDL kolesterol ve alt gruplarına ait değerler arasında istatistiki açıdan bir fark tesbit edilemedi. Ayrıca kan etil alkol seviyesi ile diğer parametreler arasında önemli bir korelasyon belirlenemedi. LDL kolesterol seviyesindeki azalmanın kolinesteraz aktivitesindeki azalmadan kaynaklandığı anlaşıldı. Sonuç olarak, orta derecede alkol tüketikiminin koronerkalp hastalığı risk faktörleri üzerine ciddi bir müsbet etkisinin olmadığı anlaşıldı. Ayrıca, alkol içenlerde görülen değişikliklerin alkolün kendisinden ziyade diğer metabolik ürünlerinin veya içkideki diğer bileşiklerin etkileri sonucu olabileceği kanaatine varıldı. Bulgularımız literatür bulguları ışığında tartışıldı. [Turk J Med Res 1994, 12(3): 108-112]

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