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Original Research Article

Comparison between lipid profile and atherogenic index of plasma (AIP) in alcoholic and non-alcoholic diabetic patients in Mangalore, Southern India

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Abstract

Diabetes is a complex disorder, caused due to deficiency or complete lack of insulin resulting in derangement in body's lipid, protein and carbohydrate metabolism. Alcoholism is a widespread problem among the diabetics as well as non-diabetics and alcoholism is associated with an increase in the serum concentrations of triglycerides, high-density lipoprotein and a decrease in serum low-density-lipoprotein concentrations. Atherogenic index of plasma (AIP) is found to be an useful marker in patients with diabetes as it predicts the risk of cardiovascular disease. The present study was conducted in 74 diabetic patients including both alcoholics and non alcoholics to compare their lipid profile and AIP. According to our study, AIP could be used as a simple parameter to early assessment of risk for development of atherosclerosis and also it gives as an idea about overall glycaemic status in both alcoholic and non alcoholic diabetic patients.

Keywords: Diabetes mellitus; Atherosclerosis; Atherogenic index of plasma; Alcoholism

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1. Introduction

Diabetes mellitus is one of the most common chronic diseases affecting the major population around the world.[1] The World Health Organization recognizes three main forms of diabetes: type 1, type 2, and gestational diabetes (occurring during pregnancy), which have similar signs, symptoms, and consequences, but different causes and population distributions. The disease is caused in most cases by a deficiency or complete lack of the hormone insulin, which is produced in the pancreas, or by an inability of the body to respond appropriately to insulin (i.e., insulin resistance).[2] The results of both conditions can include chronically elevated blood sugar levels, excessive excretion of sugar in the urine, and the accumulation of certain acidic substances in the blood. If not prevented or treated properly, these changes can lead to coma and even death. Other adverse events associated with diabetes affect the eyes, kidneys, nervous system, skin, and circulatory system.[3]

Abnormalities in the levels and metabolism of lipids are extremely common in people with either type 1 or type 2 diabetes and may contribute to those patients' risk of developing cardiovascular disease. Plasma lipid abnormality, hallmarked by low plasma HDL Cholesterol (HDLc) and high cholesterol, LDL-C and triacylglycerol levels, is very common in patients with diabetes mellitus. High serum triglyceride levels are associated with the risk of developing cardiovascular disease independently of other major measured risk factors.[4]

Although a family history of diabetes is an established risk factor for type 2 diabetes, lifestyle factors also play an important role in its cause. For example, the incidence of diabetes has been associated with physical inactivity and obesity, both of which can be modified to decrease the risk for diabetes.[5] Alcohol consumption is also prevalent in both affluent societies and people with lower socioeconomic status. Alcohol consumption can exacerbate the diabetes-related lipid abnormalities, because numerous studies have shown that heavy drinking can alter lipid levels even in nondiabetics. Alcohol can induce several types of lipid alterations, including elevated triglyceride levels in the blood (i.e., hypertriglyceridemia), reduced levels of low-density lipoprotein (LDL) cholesterol, and elevated levels of high-density lipoprotein (HDL) cholesterol.[6]

Atherogenic index of plasma (AIP) predicts the risk of development of atherosclerosis in diabetic patients as it considers the elevated levels of triglyceride as an important risk factor.[7] The association of TGs and HDLc in this simple ratio theoretically reflects the balance between risk and protective lipoprotein forces, and both TGs and HDLc are widely measured and available. Recent studies have shown that serum triglyceride level is an independent determinant of cardiovascular risk across a broad population group with abdominal obesity.[8, 9]

With this respect, the present study was done to determine and compare the levels of fasting plasma glucose, fasting lipid profile, TC / HDLc and AIP in diabetic non-alcoholic and diabetic patients with chronic alcoholism. To correlate AIP with fasting plasma glucose (FPG) and lipid profile parameters.

2. Materials and methods

The study was carried out on total 74 patients and they were grouped into, group I consisting of 37 diabetic patients (mean age of 57 years) who are non alcoholics and group II consisting of 37 diabetic patients (mean age of 53 years) who are alcoholics. Average duration of alcoholism in group II is about 7-8 years. Patients involved in the study were recruited from Kasturba Medical College Hospital, Mangalore, India during 2009-2010, with average history of diabetes mellitus from last 4-6 years. Informed consent was taken from all subjects involved in the study and the study was approved by the institutional ethics committee. Fasting blood samples were also collected from group I and group II patients and serum was separated subsequently and processed for determination of fasting lipid profile and fasting plasma glucose.

Fasting lipid profile (TC, TG, HDLc) and FPG were estimated by enzymatic method using automated analyzer Hitachi 917. FPG was determined by modified glucose oxidase/peroxidase method.[10] Total cholesterol estimation was done by cholesterol oxidase method; high density lipoprotein was estimated by the same method after precipitating the low density lipoproteins, very low density lipoproteins and chylomicrons.[11] Triglycerides were estimated by enzymatic mixture containing lipoprotein lipase, glycerol kinase and glycerol-3-phosphate oxidase and peroxidase.[12] LDL was calculated by Friedewald's formula (LDL = TC - [HDL + TG/5]), TC/HDLc was calculated by dividing total cholesterol by HDLc.[13] AIP was calculated using the formula, log TG /HDL, with TG and HDL expressed in molar concentrations. To convert mg/dl to mmol/L, the HDLc was devided by 39 and triglyceride by 89.[14]

The results were expressed as mean ± standard deviation. A $p<0.05$ was considered statistically significant. Statistical analysis was performed using the statistical package for social sciences (SPSS-16, Chicago, USA). Independent sample t test was used to compare the mean values between cases and controls. Pearson correlation was applied to correlate between the parameters.

3. Results

As depicted in table 1, there was significant increase in fasting plasma glucose ($p<0.01$), total cholesterol ($p<0.01$), triglycerides ($p<0.001$), TC/HC ratio ($p<0.001$) and AIP ($p<0.001$) and significant decrease in HDL ($p<0.01$) in group II patients compared to group I patients. As shown in fig. 1 on applying Pearson's correlation we observed significant positive correlation between FBS and AIP ($r = 0.472$, $p< 0.01$) in diabetic alcoholics.

Table 1: Comparison of lipid profile and AIP between non alcoholic and alcoholic diabetic patients (Values are expressed as mean ± SD)

Parameters	Non alcoholics (n = 37)	Alcoholics (n = 37)
Age (years)	57 ± 8	53 ± 9
Weight (kg)	68 ± 7.5	70.5 ± 7.5
Duration of diabetes (years)	5.6 ± 4.5	4.7 ± 4.5
Total Cholesterol (mg/dl)	187 ± 37	220 ± 60*
Triglycerides (mg/dl)	130 ± 46	218 ± 121**
HDL (mg/dl)	37 ± 7	32 ± 6*
LDL (mg/dl)	123 ± 37	142 ± 56
TC/HC ratio	5.0 ± 1.3	6.8 ± 2.3**
AIP	0.162 ± 0.189	0.406 ± 0.224**
Fasting plasma glucose (mg/dl)	143 ± 42	174 ± 73*

*p<0.01, **p<0.001

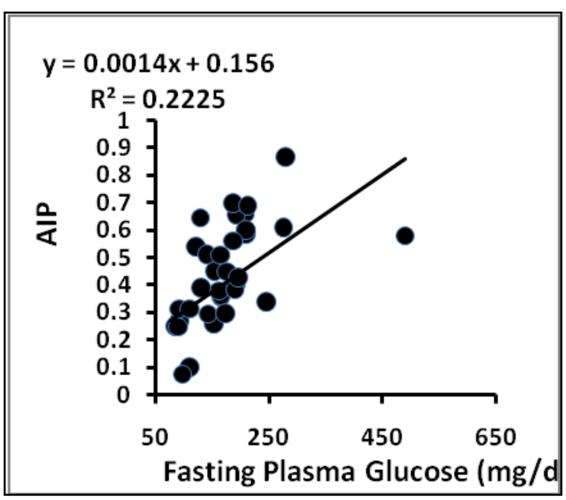

Fig. 1: Correlation graph between fasting plasma glucose and AIP in diabetic alcoholics

4. Discussion

In our study we have found a significant increase in fasting plasma glucose in diabetic alcoholics compared to diabetic non alcoholics. The mechanism underlying the increasing hyperglycaemia in chronically drinking diabetics are still unknown. The most common form of diabetes, type 2 diabetes, is associated with both insufficient insulin secretion and insulin resistance. Previous studies have indicated that alcohol consumption increases the insulin resistance which in turn leads to chronic hyperglycaemia. Along with insulin resistance, chronically drinking diabetics may show worse compliance with their dietary and pharmacological treatment regimens, which also may result in uncontrolled blood sugar levels.[15]

According to our study there was significant increase in TC, TG, LDLc, TC/HC ratio and a significant decrease in HDLc in diabetic alcoholics compared to diabetic non alcoholics. Previous studies have shown dyslipidemia in diabetes mellitus being closely associated with insulin resistance and hyperglycaemia, which in turn increases the glycation of proteins and generation of reactive oxygen species together contributing for oxidation of LDL and atherosclerosis.[16] Also previous studies have shown that elevated free radicals can enhance the oxidation of LDL and atherosclerosis.[17] Even chronic alcoholism induced liver damage may cause decreased synthesis of paroxonase, an enzyme that protects against LDL oxidation.[18]

Although an independent, inverse relationship between HDLc and cardiovascular risk has been demonstrated beyond any doubt, the contribution of TGs to cardiovascular risk has been underestimated. To overcome this AIP was used, which takes both triglyceride and HDL levels in to consideration while calculating the atherogenicity and cardiovascular risk. Previous studies have indicated the role of AIP as early predictor of risk for development of atherosclerosis in diabetic patients. In line with previous studies, we also observed significant increase in AIP in diabetic patients, which is further enhanced in alcoholics as compared to diabetic non alcoholics.[7]

Studies conducted by Dobiasova and Frohlich have shown that AIP is positively correlated with the fractional esterification rate of HDL (FERHDL), and that AIP is inversely correlated with LDL particle size. Because FERHDL predicts particle size in HDL and LDL, which in turn predicts coronary artery disease risk, the simultaneous use of TGs and HDLc (both readily available in a plasma lipoprotein profile) as AIP may be useful in predicting plasma atherogenicity. Furthermore, insulin resistance (decreased insulin sensitivity), which is often accompanied by increased coronary artery disease risk, is also often associated with increased TG and decreased HDLc concentrations and a predominance of small, dense LDL particles. Thus AIP can give a better idea in predicting the risk for development of atherosclerosis in diabetic patients.[14]

We have observed a significant positive correlation between FBS and AIP ($r = 0.472$, $p < 0.01$) in diabetic alcoholics. In diabetic alcoholics, prolonged hyperglycaemia and insulin resistance increases the lipogenesis and increases the triglyceride concentrations and decreases the HDLc and thus may contribute to elevation of AIP levels. Several mechanisms may contribute to alcohol-induced increases in triglyceride levels. First, alcohol likely stimulates the generation of VLDL particles in the liver, which are rich in triglycerides. Second, alcohol may inhibit VLDL particle breakdown. Third, alcohol may enhance the increase in triglyceride levels in the blood that usually occurs after a meal. Thus diabetic persons with chronic alcoholic history have got increased risk for development of atherosclerosis and coronary artery disease.[1]

In conclusion, by using AIP, risk for development of atherosclerosis and coronary artery can be identified early and prevented.

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