Lipid Profile and Coronary Heart Disease

Keywords: Lipid profile; coronary heart disease

ABSTRACT

Background: Coronary artery disease (CAD) remains the major cause of mortality and morbidity in the entire world population despite therapeutic advances that control many risk factors. Lipid profile is regarded as an important factor in the development of Coronary heart disease. There have been numerous studies confirming the association of hyperlipidemias with Coronary Heart Disease in most of the Western and Asian countries of the world. Many studies demonstrated the relation between coronary heart disease (CHD) prevalence and lipid levels. The aim of this study was to determine the prevalence of abnormal lipid levels and its association with coronary heart disease (CHD).

Methods and Results: A case-control study was conducted to find the relationship between lipid profile and coronary heart disease. This study involved 150 CHD patients and 150 controls group. The serum total cholesterol, HDL cholesterol, LDL, VLDL, Triglyceride, and body mass index were determined. The mean age of subjects was (28.71 ± 4.12) years in the CHD group and (30.14 ± 3.29) years in the control group. The lipid profile parameters were comparable between patients and control subjects. There were statistically significant in the BMI, total cholesterol, Triglyceride, low-density lipoprotein cholesterol and high-density lipoprotein cholesterol in CHD group when compared with the control group.

Conclusion: The changed lipid profile levels may contribute to increased risk of Coronary Heart Disease(CHD) patients.
INTRODUCTION

Coronary heart disease (CHD) is a leading cause of morbidity and mortality in many countries worldwide (WHO, 2015). CHD refers to a group of a closely related syndrome caused by the imbalance between the myocardial oxygen demand and the blood supply. Depending on the rated severity of coronary artery narrowing and the myocardial response, which is divided into angina pectoris (chest pain), acute myocardial infarction, sudden cardiac death and chronic ischemic heart disease (Kumar et al., 2015). The most common risk factors of CHD are hypertension (Jindrich, 2012), smoking, obesity (Paratz et al., 2015), diabetes (Go., 2014), stress, gender, age (Ashif, 2011) and dyslipidemia (Sarwar et al., 2007; de Campos et al., 2010). These are high levels of total cholesterol, Triacylglycerols (TAG), low-density lipoprotein cholesterol (LDL-C) and very low-density lipoprotein (VLDL) and with low levels of High density lipoprotein cholesterol (HDL-C) (Ahmed et al., 1998; Kullisaar et al., 2016). That considered as one of the most common modifiable risk factors for CHD (Di Angelantonio, 2009).

MATERIALS AND METHODS

Study design:-

A case-control study of 300 persons included two groups (150 CHD patients and 150 Control) randomly selected. All patients were diagnosed by specialist physicians as having CHD, were based on WHO guidelines.

Phenotype Measurements:-

We collected clinical data, such as weight, height, and other data. The Body Mass Index was calculated as weight (in kg) divided by the square of height (in m). Blood was collected after an overnight fast (for 12-14 hours) in anticoagulant coated tubes and centrifuged after 15 minutes, the plasma was separated and lipid profile was measured according to the procedures provided with Biolabo kits. Total cholesterol (TC) was estimated using the Cholesterol Oxidase Phenol 4-Amino antipyrine peroxidase (CHOD-PAP) method. (Russel et al., 1996; Michael et al., 1996; Allain et al., 1974). Triacylglycerol’s (TAG) levels were measured by GPO-PAP enzymatic method (Russel et al., 1996; Werner et al., 1981) High-density lipoprotein cholesterol (HDL-C)
and low-density lipoprotein cholesterol (LDL-C) were estimated using a two-step procedure: (i) precipitation and (ii) enzymatic determination (Russel et al., 1996).

**Statistical analysis:-**

Data are presented descriptively as means and standard deviation (M ± SD). Student T-test was used to compare phenotypic data between control and CHD groups using SPSS windows software (SPSS Inc., Chicago, IL). Values of P (two-tailed) less than 0.05 were considered statistically significant for all of the statistical analysis.

**RESULTS:-**

300 subjects aged between 40 to 69 years were included in this study (Figure 1). 54.6% were male and 45.3% female (Figure 2).

**Figure 1: Age distribution of study individuals.**

**Figure 2: Gender distribution of study individuals.**
Clinical and biochemical characteristics of study subjects are presented in [Table1]. 150 patients with CHD and 150 control were included in the study with a mean age of (57.41±6.38) and (49.16±4.6) years. Other basic characteristics of the study participants such as BMI of the CHD patients were statistically significantly (P = 0.001) decreased when compared with control group (22.76±2.91 vs. 28±3). There were statistically significantly of total cholesterol (P=0.001), TG (P=0.03) LDL-C (0.001) and HDL-C (P=0.013) in CHD group when compared with the control group.

Table (1): Anthropometric and biochemical characteristics of the patient and control groups

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control subjects</th>
<th>IHD subjects</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(Mean± SD)</td>
<td>(Mean± SD)</td>
<td></td>
</tr>
<tr>
<td>No (M/F)</td>
<td>150(74/76)</td>
<td>150(90/60)</td>
<td>0.064</td>
</tr>
<tr>
<td>Age (y)</td>
<td>49.16±4.6</td>
<td>57.41±6.38</td>
<td>0.001</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>22.76±2.91</td>
<td>28±3</td>
<td>0.001</td>
</tr>
<tr>
<td>Cholesterol (mg/dl)</td>
<td>168.71±23.86</td>
<td>186.12±25.89</td>
<td>0.001</td>
</tr>
<tr>
<td>Triglycerides (mg/dl)</td>
<td>116.78±34.73</td>
<td>124.39±27.82</td>
<td>0.033</td>
</tr>
<tr>
<td>VLDL (mg/dl)</td>
<td>24.00±7.54</td>
<td>25.00±6.00</td>
<td>0.231</td>
</tr>
<tr>
<td>LDL (mg/dl)</td>
<td>92.67±25.91</td>
<td>110.00±24.00</td>
<td>0.001</td>
</tr>
<tr>
<td>HDL (mg/dl)</td>
<td>57.94±15.17</td>
<td>54.5±7.18</td>
<td>0.013</td>
</tr>
</tbody>
</table>

Values are expressed as a mean ± standard deviation. BMI: body mass index; TG: triglyceride; LDL-C: low-density lipoprotein cholesterol; VLDL-C: very low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol.

DISCUSSION

Dyslipidemia was an important risk factor for atherosclerosis and Coronary heart disease (Fox, 2002). Where high levels of cholesterol, triglycerides, (LDL-C), and decreased HDL cholesterol (HDL-C) in Plasma are associated with increased risk for CHD(Molly et al., 2004; Sherbet et al., 2013;Kullisaar et al., 2016) Several mechanisms could explain the strong effect of lipid profile on the rate of CHD. Based on known the physiological functions of lipoproteins (Ferrari
et al., 2016). Atherogenic Lipoprotein that contains apoB (LDL- cholesterol), is known to promote cholesterol collection in macrophages as well as inflammatory responses inside the vessel wall, leading to atherosclerosis progression. Lipid deposition starts with the mobility of LDL from the blood into the wall of the vessel. Once within the media of the blood vessels it may move back into the bloodstream (a sign of lesional regression and a process that may be expedited by some lipid reducing strategies), or become oxidized (through direct activity of leukocytes or action of free radicals) or taken up by monocyte/macrophages which eventually become foam cells (Crowther.,2005).

High density lipoproteins (HDL) major apolipoprotein, apoA-I , which ordinarily carry about 20% of the total plasma cholesterol, transporting excess cholesterol from the arterial wall's foam macrophages to the liver(G.F.Lewis et al.,2005). The efflux of cholesterol from peripheral cells is largely facilitated through adenosine triphosphate-binding cassette transporters (ABC) and cholesterol esterification by lecithin-cholesterol acyltransferase (LCAT) helps modify these particles into more mature HDL2 and HDL3 particles (Huang., 2013; Bobom., 2017). Cholesterol ester transfer protein (CETP) transfers additional cholesterol esters from apolipoprotein B-containing particles to HDL (G.F. Lewis et al.,2005; Zhang.,2012). Subsequently, these HDL particles are taken up by hepatocytes through scavenger receptor B1 (SR-B1) (G.F. Lewis et al.,2005; Zhou.,2015 ). So, The strong connection between low levels of HDL and the risk for atherosclerosis (Shao et al.,2009).

In this study observed that their elevated Triglyceride, Low Density Lipoprotein (LDL) , cholesterol and decrease High Density lipoprotein (HLD) in patient of CHD that correspond with (Haddad et al.,2002) in Jordan; (Hassan et al.,2013) in Sudan;( M.Mohsen et al., 2013) in Egyptian that show the abnormal lipid profile remain strong risk factor of CHD.

CONCLUSION

In this background, it is concluded that Patients with CHD have altered lipid profile, with higher levels of TGs, total cholesterol, VLDL and LDL and low level of serum HDL; this difference may play a role in the pathophysiology found in Patients with CHD.
REFERENCES
