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Original Research Article

Relationship between Lipid Profile and Thyroid Disturbance and Ischemic Heart Disease in Iraqi Patients

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Abstract: The current study is a case-control study conducted in Baghdad city between 1st of January to the end of, 2023. The study focused on patients admitted to the Coronary Care Unit of Ibn Al Nafees Hospital and Ibn Al Bitar Hospital in Baghdad. And Alyarmok Hospital in Baghdad. The study included 60 Iraqi patients with coronary heart disease, ranging in age from 37 to 66 years. A control group of 30 apparently healthy individuals (15 men and 15 women) was included in the study. The control group was matched with the patients in terms of gender and age to improve the accuracy of the results. Blood samples were collected from both patients and control individuals after 12 hours of fasting. Biochemical colorimetric methods by spectrophotometer were used to determine total cholesterol (TC), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein (LDL), and Immunofluorescence technique by Afias was used to determine triiodothyronine (T3), thyroxine (T4), and TSH levels and HbA1C levels in the blood. The study analyzed the demographic characteristics of patients with ischemic heart disease (IHD) and the control group, including age groups, sex distribution, and BMI. The majority of IHD patients were non-smokers, with 26.67% being smokers and the majority being non-smokers. The majority of IHD patients had hypertension, while 58.33% had diabetes. The cholesterol levels in IHD patients were significantly higher than the control group, with a p-value of 0.001. The triglyceride levels were also significantly elevated in IHD patients, with a p-value of 0.001. LDL-c levels were significantly elevated in IHD patients, while very low-density lipoprotein cholesterol (VLDLc) levels were significantly elevated in IHD patients. IHD patients had significantly higher T3 levels (1.701 ± 1.21) than the control group (0.87 ± 0.16), suggesting potential differences. T4 levels were also significantly higher in IHD patients with thyroid dysfunction (106.7 ±86.5) compared to the control group (78.5 ±13.65). On the other hand, TSH levels did not show a significant difference between IHD patients with thyroid dysfunction (3.29 ± 2.55) and the control group (3.18 ±2.078). The study found no significant differences in thyroid function parameters among the different age groups of individuals with hypothyroidism. The mean T3 level in the 40-49 age group was 0.49±0.02, the mean T4 level was 43.5±0.7, and the mean TSH level was 4.88±0.38. For the 50-59 age group, the mean T3 level was 0.36±0.17, the mean T4 level was 44.6±0.54, and the mean TSH level was 5.41±1.07. In the >59 age group, the mean T3 level was 0.51±0.03, the mean T4 level was 45.8±6.9, and the mean TSH level was 5.24±0.66. The study showed that hypothyroidism IHD patients tend to be associated with higher levels of cholesterol, triglycerides, LDL-c, and VLDL-c. The highest prevalence of hyperthyroidism among individuals with hypertension was 72.73%, while the highest spread was seen in hypothyroidism among individuals with diabetes. The highest mean value for HbA1c was observed in patients with hypothyroidism (6.99±0.82%), followed by patients with hyperthyroidism (6.46±0.51), and the lowest mean value was seen in patients with normal thyroid function (4.36±0.56%). These differences in mean HbA1c levels among the three groups are statistically significant, as indicated by the p-value: of 0.017.

Keywords: Baghdad city, Ibn Al Nafees Hospital, Patients, Lipoprotein cholesterol.

INTRODUCTION

The most common form of heart disease. It is the result of atheromatous changes in the vessels supplying the heart. CAD is used to describe a range of clinical disorders from asymptomatic atherosclerosis and stable angina to acute coronary syndrome (unstable angina) [1]. A common symptom of coronary artery disease (CAD) is angina, Angina is chest

pain or discomfort that occurs if an area of the heart muscle doesn't get enough oxygen-rich blood [2]. High total cholesterol, low density lipoprotein (LDL)-cholesterol and triglyceride levels, and low levels of HDL cholesterol increase risk of coronary heart disease and ischaemic stroke. Obesity and its associated comorbidities, such as cardiovascular and metabolic disorders, are promoted early in childhood and adolescence by changes in lifestyle and physical activity levels. Knowing the CVD risk factors is important for developing preventive and treatment methods. Excess weight, which is common in children and teenagers, is one of them. Dyslipidemia is linked to excess weight [3]. The primary predictors of CVD include hypercholesterolemia, namely elevated LDL and reduced HDL values. Another risk factor for CVD is a sedentary lifestyle, which is prevalent in childhood and adolescence and is justified by changing behaviors. Obesity development and maintenance are closely linked to an unhealthy lifestyle that includes less physical exercise and more sedentary behavior [1]. THs exert a significant impact on the cardiovascular system via both genomic and non-genomic mechanisms. The major effects of THs on the myocardium are mediated by T3, which stimulates nearly all of the transporters and ion channels involved in calcium myocardial fluxes, upregulating sarcoplasmic reticulum calcium-activated ATPase 2 (SERCA2) and Na^+/K^+-ATPase and downregulating phospholamban [4]. These changes enhance calcium uptake and release by the sarcoplasmatic reticulum, stimulating both diastolic myocardial relaxation and systolic myocardial contraction [5]. The contractile apparatus of the cardiac myocyte has two subtypes of myosin heavy chains (MHCs): α-MHC and β-MHC – fast and slow myosin, respectively. T3 can upregulate α-MHC and downregulate β-MHC [6]. Therefore, the myocardial hypothyroid state induces a so-called fetal gene reprogramming which increases the expression of β-MHC and decreases the expression of α-MHC and SERCA2 [7].

This phenotype presents substantial implications on myocardial function and subsequent progression to heart failure. The fetal pattern is characterized by a preference for glucose over fatty acids as a substrate. Although such changes may lower oxygen demands, the yield of ATP per substrate also decreases, resulting in metabolic inefficiencies that decrease metabolic reserves – perhaps leading to cardiac pump dysfunction [8]. Nevertheless, this phenotype seems to be reversible with the appropriate therapy. T3 is able to modify the activity of sodium, potassium and calcium channels, altering a variety of intracellular pathways in cardiac and vascular smooth-muscle cells. Consequently, THs can increase resting heart rate, cardiac contractility and venous tone almost immediately, increasing cardiac preload and cardiac output [9]. T3 increases myocardial sensitivity to the adrenergic system by increasing the number of adrenergic membrane receptors. Additionally, T3 decreases systemic vascular resistance through vascular smooth-muscle relaxation, which in turn decreases renal perfusion and leads to renin-angiotensin-aldosterone axis activation [10]. Table 4.2 presents the lipid profile of individuals with ischemic heart disease (IHD) and a control group. The cholesterol levels in IHD patients (245.6±81.2) were significantly higher compared to the control group (167.7±81.2) with a p-value of 0.001. Similarly, the triglyceride levels were substantially elevated in IHD patients (299.8±153.4) compared to the control group (115.6±23.5), also showing a p-value of 0.001. Regarding LDL-c levels, IHD patients (126.54±41.5) exhibited higher values compared to the control group (61.6±24.6) with a p-value of 0.001. Similarly, VLDLc levels were significantly elevated in IHD patients (59.97±12.65) in contrast to the control group (13.5±1.76) with a p-value of 0.001. In contrast, the HDL-c levels in IHD patients (26.35±7.8) were notably lower than those observed in the control group (65.5±8.56) with a p-value of 0.001.

Table 1: Lipid profile parameters among the studied Control groups

Variable	IHD patients	Control group	P-value
Cholesterol mg/dl	245.6±81.2	167.7±81.2	0.001
Triglyceride mg/dl	299.8±153.4	115.6±23.5	0.001
LDL-c mg/dl	126.54±41.5	61.6±24.6	0.001
VLDLc mg/dl	59.97±12.65	13.5±1.76	0.001
HDL-c mg/dl	26.35±7.8	65.5±8.56	0.001

The finding that individuals with ischemic heart disease (IHD) exhibit significantly higher levels of cholesterol, triglycerides, LDL (low-density lipoprotein), and VLDL (very low-density lipoprotein), as well as lower levels of HDL (high-density lipoprotein), compared to a healthy control group aligns with numerous previous studies. These findings consistently indicate a robust association between dyslipidemia (abnormal lipid profiles) and the development of IHD. For example, the Yusuf *et al.* [11] involving a large sample size of over 29,000 participants from 52 countries, discovered a robust association between high levels of total cholesterol and LDL and the risk of development of IHD. Additionally, the Prospective Studies Collaboration, which encompassed a vast cohort of more than 900,000 individuals, further confirmed the link between dyslipidemia and coronary heart disease (CHD). This collaboration revealed that higher levels of total cholesterol, LDL, and triglycerides were associated with an increased risk of developing CHD. The findings from this large-scale study provide strong evidence supporting the role of dyslipidemia as a significant risk factor for the development of CHD [12]. Other studies have also demonstrated the effectiveness of lipid-lowering therapy, such as statins, in reducing the risk of IHD [13, 14]. In fact, the use of statins is recommended in current guidelines for the management of dyslipidemia and prevention of cardiovascular disease [15]. Overall, the finding that IHD patients have significantly higher levels of cholesterol, triglycerides, and LDL reinforces the importance of dyslipidemia management in reducing the risk of IHD and

highlights the need for early identification and treatment of dyslipidemia in high-risk individuals [16]. The study showed Hypothyroidism IHD patients tends to be associated with higher levels of cholesterol, triglycerides, LDL-c, and VLDL-c. Table 4.10.

Table 2: Mean of lipid profile in IHD patients in relation to thyroid dysfunction

Lipid profile	Thyroid dysfunction			P-value
	Hypothyroidism	Hyperthyroidism	Normal	
Cholesterol mg/dl	303.7±83.5	264.2±47.9	215.8±82.5	0.003
Triglyceride mg/dl	391.4±169.5	308.3±110.4	263.9±157.7	0.014
LDL-c mg/dl	151.8±34	137.42±20.5	112.06±47.3	0.018
VLDL-c mg/dl	78.3±33.9	61.65±22.1	52.78±31.5	0.015
HDL-c mg/dl	21.08±3.63	22.886±2.52	30.1±8.77	0.016

In agreement with these finding, Yeza *et al* [16] found that dyslipidemia was found in highest rate of the population sample, in which more than half had thyroid disorders, with a significant association with hypothyroidism and no association was found with hyperthyroidism. The study also in agreement with some authors who refer to hypothyroidism as a recognized cause of secondary dyslipidemia [17,18]. Hypothyroidism is known to affect lipid metabolism, resulting in increased total cholesterol, LDL cholesterol (LDL-c), triglycerides, and potentially very low-density lipoprotein cholesterol (VLDL-c). These lipid abnormalities can contribute to the development and progression of cardiovascular diseases such as IHD [19]. This can be attributed primarily to the increase in LDL-chol and secondarily to the increase in LDL and VLDL-c fractions. The main cause of hypercholesterolaemia in these patients is the decreased clearance of LDL by its receptor, based on decreased LDL receptor gene expression in fibroblasts, hepatocytes and other tissues [20]. In hypothyroidism there is a predisposition to cardiovascular problems, as there is a decrease in myocardial contractility which favors atherosclerotic processes, there is a reduction in lipolysis and a consequent increase in serum lipid levels [21-22].

CONCLUSION

In conclusion, this study highlights the significant differences in T3 and T4 levels between IHD patients and the control group, suggesting potential alterations in thyroid function associated with ischemic heart disease. These findings contribute to our understanding of the relationship between thyroid function and ischemic heart disease, emphasizing the need for further research to elucidate the clinical implications and underlying mechanisms involved.

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