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Co-relation between Thyroid Profile and Lipid Profile in AMI Patients – A Hospital Based Study

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ABSTRACT
MI or AMI commonly referred as a heart attack occurs where flow of blood stops to part of the heart causing damage to the heart muscle. Increased Lipid profile levels are the major cause of Myocardial Infarction (MI). Myocardial infarction associated with Hyperthyroidism and patients with ischemic heart disease was a very high prevalence of hyperthyroidism. Patient having chest pain and increasing shortness of breath for many days are included in our study.
Purpose of the study: *This study is designed to measure the level of thyroid hormone i.e. T3, T4, TSH and its correlation ship with lipid profile Ch, TG, LDL and VLDL in patients with acute myocardial infarction and control groups. The present showed that the level of total CH, TG, LDL-C and VLDL-C were significantly increased and significantly decreased in activity of HDL in AMI patients when compared to normal healthy control groups.*
Similarly T3 and T4 levels were significantly increased in AMI patients when compared to control groups. Present study showed that the value of TSH decreased as compared to control groups. There was positive significant co-relation between T4 with CH and VLDL but there is no correlation found between T4 and HDL. The degree of T3 decrease is proportional to the severity of cardiac damage and may have a possible prognostic value.
Keywords: AMI, T3, *T4, CH, TG, VLDL, HDL, CVD, CHD, Euthyroid* sick syndrome, Coronary Artery Disease, CAD: TSH: Thyroid Stimulating Hormone, ELISA, RIA, MIS.

Introduction

Myocardial infarction (MI) means the stops blood flow to distinct part of the heart causing damage of heart muscle. Acute Myocardial infarction (AMI) or MI is a condition where circulation to the heart is compromised [(1)].

Low-density lipoprotein (LDL) also called as bad cholesterol and it is one of the main causes of a blockage in the arteries. Not all cholesterol is bad, but LDL cholesterol can stick to the walls of arteries and produce plaque. Plaque is a hard substance that blocks blood flow in the arteries. Blood platelets, which help the blood to clot, may

stick to the plaque and build up over time. Saturated fats may also contribute to the buildup of plaque in the coronary arteries. Saturated fats may lead to an arterial blockage by increasing the amount of LDL-C in blood system and reducing the amount of HDL-C.. Tran's fat is usually artificially produced and can be found in a variety of processed foods like hydrogenated oil or partially hydrogenated oil.

The symptoms in myocardial infarction (MI) is usually gradual, over few minutes, and rarely instantaneous[2] chest pain is the most common symptom of acute MI and is described often a sensation of tightness, pressure, or squeezing[3].

Chest pain due to ischemia of the heart muscle is termed as angina pectoris. The pain radiates most often to the left arm , but may also radiate to the lower jaw, neck, right arm, back & upper abdomen[4] where it may mimic heart other symptoms may includes shortness of blood , excessive form of sweating , nausea, weakness, Vomiting, feeling faint and palpitations[5]. Loss of Consciousness (due to in adequate blood flow to the brain and cardiogenic shock) & sudden death (frequently due to ventricular fibrillation) can occur in MIs.

About 30% of the People have typical symptoms. A typical symptoms are more frequently reported by women, the elderly and those with diabetes where compared to their male & young ones[6]. Myocardial Infarction (MI) or Acute Myocardial infarction (AMI) is the most; contributor of morbidity and mortality worldwide. MIS is rising drastically in large proportion in developing countries and has turned out to be the leading cause of death in India. Acute myocardial infarction is caused by acute plaque rupture and thrombus formation in the coronary artery resulting in a sudden disruption in blood flow to the heart muscle and death of heart tissue. AMI can be classified into ST-segment elevation myocardial infarction (STEMI) and Non-STEMI (NSTEMI), which is distinguished based on the findings from a diagnostic electrocardiogram, ECG [7].

Myocardial infarction is a major cause of morbidity and mortality worldwide. Each year More than 3 million people are estimated to have an acute ST-elevation myocardial infarction (STEMI), while more than 4 million having a non-ST-elevation Myocardial Infarction (NSTEMI). From being an illness seen predominantly in developed countries, myocardial infarction is now becoming increasingly more common in developing countries [8].

The incidence of MI in India is around 23%. The epidemic cardiovascular disease is rapidly advancing in India from 10-23 % in last two decades. About 1.1 million cases occur every year in US with about 30% mortality and more than 50% of death occurs on way to the hospital. In India 31.7%of deaths occur due to MI [9,10]. Incidence rate of cardiovascular disease was about 7% in 1970 and increased upto 32% in 2011 in India. The main cause of CVD in India is the consequence of large population and high prevalence of cardiovascular risk factors like smoking, alcohol intake, low fruit diet and vegetable intake, regular exercise, abdomen obesity, high blood pressure and abnormal lipids and diabetes [11 12].

The AMI is triggered by increased oxygen demands such as fever, thyrotoxicosis, hyperlip-idemia; hyper coagulation states, obesity, smoking and cocaine abuse. At least one quarter of all MIS is silent, without chest pain or other symptoms.

Estimates of the prevalence of silent MIS vary between 22 & 64% [13]. An MI may cause heart failure an irregular heartbeat, or cardiac arrest. Most MIs Occur mainly due to Coronary Artery disease (CAD) Or Coronary Artery syndrome (CAS) [15]. Patients with thyrotoxicosis induced acute myocardial infarction are rare. The exact mechanism is still not known.

The most common cardiovascular manifestation of thyrotoxicosis has been recognized as angina pectoris, arterial fibrillation, myocardial infarction and heart failure [16].

Thyrotoxicosis is the endocrine disorder which increase oxygen demand and at the same time can

induce coronary vasospasm leading to AMI. AMI with little or no history of symptoms in women chest pain may be less predictive of coronary ischemia than in male. AMI was defined as typical chest pain lasting minimum half an hour and an ECG showing ST elevation of at least 2 mm. Many risk factors for myocardial infarction include high Blood pressure smoking, lack of exercise, obesity, diabetes, poor diet excessive alcohol intake, among other. The mechanism of an MI involves the rupture of an atherosclerotic plaque, lieder to complete blockage of coronary artery. MIS are commonly caused by coronary artery spasms, which may be due to cocaine, artery spasms, which may be extreme cold & among other [17].

In view of this present study was under taken to assess, the correlation between thyroid profile and lipid profile in AMI patients.

Following are the Objectives of study

1) To measure the level of T3, T4, and TSH in patients with MI or Thyroid profile in AMI patients.
2) To measure the lipid profile or dyslipidemia i.e. TC, TG, HDL, LDL and VLDL in AMI patients.
3) To study the correlation of thyroid profile and lipid profile in AMI patients and to establish a significant relationship in causation of AMI.

Material and Methods

Total 20 acute myocardial infection patients were selected for study group and compare with 25 normal healthy control groups. The study will be conducted in the patients admitted in the Department of Biochemistry in collaboration with Dept. of Medicine, Intensive Coronary Care Unit at Chandulal Chandrakar Memorial Medical College, Kachandur, Durg. All patients of acute myocardial infection are diagnosed by clinician and admitted in our hospital for treatment. Informed consent was taken from all patients, who participated in our study and the study was approved by the hospital ethics committee.

Collection of Blood sample

5 ml fasting blood sample was collected in a dry, clean, plane tube from AMI patients. After clotting of blood, it is centrifuged at 3000 r.p.m. for 10 minutes. Serum will separated for the analysis of thyroid profile and lipid profile such as TSH, T3, T4, Total cholesterol, Triglyceride and HDL by enzymatic method and LDL-C and VLDL-C calculated by Friedewald equation.

The activity of TSH, T_4, T_3, estimated by using ELISA and reagents kits will purchased from accu-bind [17,18].

Total cholesterol, triglycerides and HDL were analyzed by enzymatic method using auto-analyzer. Serum Total Cholesterol estimated by enzymatic kit method[19], Triglyceride estimated by bioluminescent assay method[20] and HDL-cholesterol estimated by phasphotungstate precipitation method[21] manufactured by ERBS Transasia. LDL-C and VLDL-C calculated by Friedewald formula.

Following formulae were used[22]:

For VLDL Cholesterol in mg % =Serum Triglyceride / 5

Serum LDL = Serum total cholesterol - (serum VLDL + Serum HDL).

Control Group

Total 25 normal healthy 20-45 years adult, age & sex matched subject comprises for control group.

Data Analysis

Data were expressed as mean ±SD. Mean values were assessed for significance by unpaired student –t test. A statistical analysis was performed using the Stastical Package for the Social Science program (SPSS, 16.0). Frequencies and percentages were used for the categorical measures. Probability values $p < 0.05$ were considered statistically significant.

Exclusion criteria

Previous MI, cardiac disease, thyroid abnormalities, chronic illness and use of medication before admission.

Blood samples were collected at a time of admission, before any treatment (4h) & at 12h, 24h, 36h, 48h and 7 days after onset.

Table 1 Showed mean value of Age and BMI was significantly increased in patients with myocardial infarction compared with control groups.

	Control Group Mean ± SD	AMI Patients Mean ± SD	'P' value
Age	34.73 ± 6.29	59.93 ± 8.63	0.001
BMI (Kg/m2)	22.57 ± 2.98	33.57 ± 5.12	0.002
Significant using SPSS for two independent means at significance * ($P \leq 0.05$), ** ($P \leq 0.01$)			

Table 2 Showed distribution of patients according to hypertension, respiratory disease, smoking and drugs.

Diseases	Percentage	No of Patients
Respiratory disease	20 %	4
Smoking	35%	7
Hypertension	80%	16
Statin	50%	10

Results and Discussion

The present study was conducted in the Dept. of Biochemistry in collaboration with Dept. of Medicine at CCM Medical College, Kachandur Durg. In severe illness of any cause, how down regulation of the thyroid hormone system affects patients with acute myocardial infarction (AMI) is largely not known.

Table 3 Shows Lipid profile parameters in AMI patients along with Control groups:

Biochemical Parameters	Control	AMI Patients	P value
CH (mg/dl)	113.75+21.47	261.81+41.91	0.001
TG (mg/dl)	98.27+12.53	178.81+93.36	0.019
HDL-C (mg/dl)	48.36+5.96	34.98+5.52	0.001
LDL-C (mg/dl)	74.63+19.78	174.18+47.43	0.000
VLDL-C (mg/dl)	17.69+ 0.96	41.54+19.98	0.005

Table no 3 shows that the activity of lipid profile like CH, TG, LDL and VLDL were significantly increased found in AMI patients than control group. The level of lipid profile in AMI patients were 261.81+41.91, 178.81+93.36, 174.18+47.43 and 41.54+19.98 and in normal control group were 113.75+21.47, 98.27+12.53, 74.63+19.78, 17.69+ 0.96. The level of HDL- cholesterol was significantly decreased found in AMI patients (34.98+5.52) than control subjects (48.36+5.96). It shows that high level of serum cholesterol considered as a risk factor for CVD and high level of TG, LDL and VLDL and low level of HDL stratifying led to accurate detection of CHD.

Table 4 Shows thyroid parameters T_3, T_4 and TSH in AMI patients along with Control groups:

Parameters	Control	AMI Patients	P. value
T_3 (ng/ml)	0.93 ± 0.10	1.27 ± 0.19	0.007
T_4 (mg/dl)	7.16 ± 0.93	8.96 ± 1.09	0.001
TSH (m IU/L)	1.62 ± 0.74	1.03 ± 0.86	0.194
Significant using SPSS for two independent means significance (P<0.05),**(P<0.01)			

Table no 4 shows the level of TSH was decreased in AMI patients but the level of T3 and T4 were significantly increased found than control group. Similar findings were observed by Krikegaard c et. al[23] showed hyperthyroidism is clinical syndrome caused by increase of circulating FT4, FT3 or both. It's a common disorder that affects near about 2% women and 0.2% of men. Pavlou et al [24] and Friberg et al [25] have also reported a rapid down regulation in T3 levels in patients with acute myocardial infarction. It has been postulated that down regulation of the thyroid hormone

system may be an advantage if it is present when AMI strikes because the O_2 demand of the myocardium may be reduced, when metabolic rate is diminished.

When the thyroid hormone system is down regulated in short term in AMI, intracellular calcium handling is affected in a way that may contribute to myocardial stunning and reperfusion injury due to calcium overload. Furthermore, down regulation of the thyroid hormone system leads to increase in systemic vascular resistance and increased cardiac after load. If the heart is unable to cope with this, cardiac output will be reduced.

Our present study results showed that significantly increased the level of CH, TG, LDL-C and VLDL-C and the activity of HDL-C significantly decreased in AMI patient, when compared to control groups show in table no 3. High level of serum cholesterol considered as a risk factor for CVD. Also TG is another strong risk factor but it found that triglyceride levels stratifying led to more accurate detection of increased risk of coronary disease[26]. Increased VLDL in Myocardial infarction patients and the role of low HDL in the CHD development has been widely accepted[27]. The study showed levels of $T_3$and T_4 was significantly increased while TSH decreased in patient with Hyperthyroidism is the clinical syndrome caused by increase of circulating free Thyroxine T_4, free Triiodothyronine T3, or both.

It is a common disorder that affects approximately 2% of women and 0.2% of men [23]. Also the diagnosis of hyperthyroidism is confirmed by blood tests that show decreased of thyroid. Thyrotoxicosis resulting from painless thyroiditis accounts up to 23% of all thyrotoxicosis cases.

The pathophysiology of myocardial bridge is not clearly understood; sometimes it may associates with overt pathology, where as sometime it might be incidental finding without any clinical significance[28]. One of the studies showed that thyrotoxicosis with painless thyroiditis induced acute myocardial infarction in a young man usually has normal Coronary arteries without any coronary risk factor.

In conclusion, the thyroid hormone system is rapidly down regulated in acute myocardial infarction. This may be beneficial during acute ischemia. The degree of T3 decrease is proportional to the severity of cardiac damage and may have a possible prognostic value. Thus, T3 blood levels may contribute to the elaboration of an AMI severity index.

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