# Evaluation of Thyroid hormone and lipid profile in patients with myocardial infarction and find the relation between them

Israa Burhan Raoof, Raghad abdulmahdi, Ayad Kareem Khan

**Abstract:** Myocardial infarction (MI) occurs when blood flow stops to part of the heart causing damage to the heart muscle. lipid profile increased are the major causes of myocardial infarction (MI). Patient presented with chest pain and increasing shortness of breath for several days. Myocardial infarction associated with Hyperthyroidism and patients with ischaemic heart disease was a very high prevalence of hyperthyroidism. This work aimed to measure level of lipid profile and T3, T4, TSH and correlation between them in patients with myocardial infarction and control groups. The results showed significantly increased in mean value of Ch ,TG, LDL and VLDL and significantly decreased in mean value of HDL in patient with myocardial infarction when compared to control groups. Also the result appeared the mean value of T3 and T4 levels was significantly increased in patients with myocardial infarction when compared to control groups. In addition of that, the study showed the mean value of TSH decreased as compared to control groups, also there was positive significant correlation between T4 with (Ch , VLDL) but negative correlation betweenT4 and HDL .

Key wards: Myocardial Infarction, Thyroid hormone.

## 1 Introduction

 $\mathbf{M}_{\mathrm{vocardial}}$  infarction (MI) associated with iatrogenic hyperthyroidism where myocardial bridge can be possibly life threatening [1,2]. The most common cardiovascular manifestations of thyrotoxicosis recognized as angina pectoris, atrial fibrillation, myocardial infarction and heart failure [3]. MI known as a heart attack, results from the interruption of blood supply to a part of the heart, that causing heart cells to die. This is most commonly due to occlusion of a coronary artery following the rupture of vulnerable atherosclerotic plaque which collection of lipids in the walls of artery [4]. The diagnosis of MI is usually based on clinical symptoms and on electrocardiographic (ECG) findings of the patient [5]. Also myocardial infarction refers to coronary syndrome [6] that due to coronary artery disease.[7] the Risk factors of Myocardial infarction include high blood pressure, smoking obesity, high blood cholesterol, poor diet, and excessive alcohol[8] low-density lipoprotein (LDL) cholesterol, endothelial dysfunction and increased central arterial stiffness [9,10]. Lipids consider the primary targets of oxidative stress. Lipid peroxidation of the cellular structures, a consequence of increased oxygen free radicals, that play an important role in atherosclerosis and

microvascular complications of DM [11]. Hyperthyroidism is an overproduction of the thyroid hormones  $T_3$  and  $T_4$ . This condition is most commonly caused by the development of Graves' disease, an autoimmune disease in which anomalous antibodies stimulate the thyroid to secrete excessive quantities of thyroid hormones.[12]

## 2 Aim of study

The aim of this study was to measured the level of  $T_3, T_4, TSH$  in patients with myocardial infarction and find the correlation between these parameters in sera of control groups and patients with myocardial infarction.

#### 3 Materials and Methods:

Blood sampling were taken from 12 patients with myocardial infarction and 12 controls. 5 ml of Blood sample were collected from all patients. A questionnaire was designed with different questions including duration of myocardial infarction, heart disease, family history, drug duration, weight, height, smoking, usage of drugs , and hyperthyroidism for all patients groups, and controls group. The distribution of patients according to hypertension, smoking and and drugs was in Table (1):

Israa Burhan Raoof, Raghad abdulmahdi,Ayad Kareem Khan College of Pharmacy, University of Al-Mustansiriyah Table (1) Distribution of patients according tohypertension, Respiratory diseases smoking and drugs

Disease	%
Hypertension	85%
smoking	37%
Respiratory diseases	21%
statin	50%

**1-Cholesterol (Ch)** :Serum cholesterol was determine by using enzymatic method [13]

**2- Triglyceride (TG) :** Serum triacylglycerol was determine by using enzymatic method[14]

**3-High Density Lipoprotein( HDL):** Serum HDL was determine by HDL kit [15]

**4-** Low Density Lipoprotein (LDL): LDL level was usually derived by friedwalds formula [16]

LDL = Total cholesterol – [HDL + TG/5]

**5- Very Low Density Lipoprotein (VLDL)**: Very lowdensity lipoprotein was determine by using formula of friedwalds: [17]

VLDL-Ch = TG/5

**6- T<sub>3</sub> (Triiodothyronine) level:** Serum T<sub>3</sub> was determine by Human Free T<sub>3</sub> ELISA Kit [18]

**7-T**<sup>4</sup> (Thyroxine) level: Serum T<sup>4</sup> was determine by Human Free T4 ELISA Kit [19]

**8- TSH (Thyroid-Stimulating Hormone) level:** Serum TSH was determine by Human Free T4 ELISA Kit [20]

# **4 Statistical Analysis**

Results are expressed as Mean±SD. and significant differences between means were assessed by student t-test using the available statistical software packages (Microsoft SPSS), statistical significance was set at  $P \le 0.05$ ,  $P \le 0.01$ ,...

# 5 Results and Discussions:

Table (1) showed mean value of Age and BMI was significantly increase in patients with myocardial infarction compared with control groups.

Table (1): Mean value of Age and BMI in myocardial infarction and control groups.

marcaon and condor groups.			
	Mean <u>+</u> st control	Mean <u>+</u> st	p.value
		Patients	
Age	37.16 <u>+</u> 5.27	57.00 <u>+</u> 6.60	0.001
BMI (Kg/m2)	23.19 <u>+</u> 2.40	32 <u>+</u> 4.41	0.002
*Significant using spss for two independent means at significance * (P $\leq$			
0.05), ** (P≤0.01)			

Table (2): Mean value of Lipid profile in myocardial	
infarction and control groups.	

parameters	Mean <u>+</u> st control	Mean <u>+</u> st Patients	p.value
Ch (mg/dl)	116.81 <u>+</u> 19.54	250.81 <u>+</u> 39 .19	0.001
TG (mg/dl)	92.63 <u>+</u> 3.64	160.81 <u>+</u> 10 0.55	0.027
HDL (mg/dl)	53.36 <u>+</u> 3.90	43.36 <u>+</u> 3.9 0	0.001
LDL (mg/dl)	79.63 <u>+</u> 15.08	166.18 <u>+</u> 41 .43	0.000
VLDL (mg/dl)	18.36 <u>+</u> 0.08	37.54 <u>+</u> 21. 73	0.003
*Significant using spss for two independent means at significance * (P≤ 0.05), ** (P≤ 0.01)			

The results showed that significantly increased in Cholesterol, TG, LDL and VLDL also significantly decreased in HDL in patient with myocardial infarction when compared to control groups[21] Table (2). High serum cholesterol level considered as a risk factor for cardiovascular disease [22]. Also Triglyceride another strong risk factor but it found that triglyceride levels stratifying led to more accurate detection of increased risk of coronary disease. [23].In addition elevated LDL is associated with 3-fold increase in the risk of myocardial infarction.[24] Increased VLDL in Myocardial infarction patients and the role of low HDL in the CHD development has been widely accepted.[25].The study showed levels of T3 and T4 was significantly increased decreased in patient with myocardial while TSH infarction when compared to control groups<sup>(26)</sup> as shown in Table (3).

Hyperthyroidism is the clinical syndrome caused by increase of circulating free Thyroxine T<sub>4</sub>, free Triiodothyronine T<sub>3</sub>, or both. It is a common disorder that affects approximately 2% of women and 0.2% of men [26]. Also the diagnosis of hyperthyroidism is confirmed by blood tests that show decreased of thyroidstimulating hormone (TSH) [27].

parameters	Mean <u>+</u> st control	Mean <u>+</u> st Patients	p.value
T₃ (ng/ml)	1.05 <u>+</u> 0.08	1.11 <u>+</u> 0.08	0.008
T₄ (mg/dl)	7.07 <u>+</u> 0.56	8.36 <u>+</u> 0.54	0.001
TSH (mIU/l)	1.58 <u>+</u> 0.63	1.22 <u>+</u> 0.97	0.226
*Significant using spss for two independent means at significance * (P≤ 0.05) and ** (P≤ 0.01)			

Table (3): Mean value of T<sub>3</sub> , T<sub>4</sub> and TSH in myocardial infarction and control groups.

Levels of  $T_4$  with (Ch,,VLDL) showed positive significant correlation coefficient also there is negative correlation coefficient between  $T_4$  with HDL in patients with myocardial infarction and control groups as shown in Table (4) and Figure (1,2,3).

# Table (4): Baseline Pearson relation coefficients of T<sub>4</sub> levels with various lipid profile (Ch, HDL ,VLDL) in myocardial infarction and control groups

Correlatio n	Ch	HDL	VLDL
$T_4$	0.992 0.000	-0.885 0.001	0.886 0.001
* Correlat	ion is signific	ance * (P≤ 0.05)	, ** (P≤ 0.01)

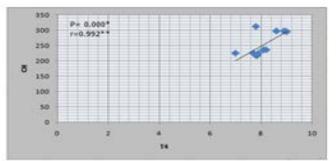


Figure (1): Relationship between T<sub>4</sub> with Ch in myocardial infarction and control groups

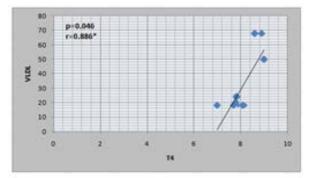
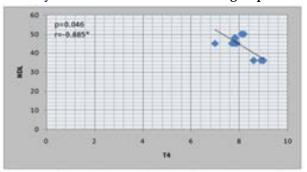


Figure (2): Relationship between T<sub>4</sub> with VLDL in myocardial infarction and control groups



# Figure (3): Relationship between T<sub>4</sub> with HDL in Myocardial infarction and control groups

cardiovascular The manifestations of hyperthyroidism have been recognized for more than two centuries and are a cornerstone for clinical diagnosis [28]. The peroxidation of lipids is basically damaging because the formation of lipid peroxidation products leads to spread of free radicals reactions.[29]. Where hydroxyl radical can initiate lipid peroxidation, which is a free radical chain reaction leading to damage of membrane structure and function. [30] Free radicalmediated oxidative stress (OS) implicated in the pathogenesis of thyroid disorders [31]. In addition Free radicals have the potential to damage the organism, their generation is inevitable for some metabolic process [32]. Variations in the levels of thyroid hormones can be one of the main physiological modulators of in vivo cellular oxidative stress and due to their known effects on mitochondrial respiration. [33].That thyroid hormones are involve in combating the toxicity of oxidative stress in humans [34].

# 6 References

1-Gowda RM et al; Acute myocardial infarction with normal coronary arteries associated with iatrogenic hyperthyroidism. Int J Cardiol; (2003); 90: 327-329.

2. Patanè S, Marte F, Patanè F, et al. ;Acute myocardial infarction in a young patient with myocardial bridge and elevated levels of free triiodothyronine. Int J Cardiol ; ;(2009); 132: 140-142.

3. Irwin K et al;Thyroid Hormone and the Cardiovascular System. N Engl J Med ; ;(2001); 344: 501-509.

4-K. Sathya narayana. et al; Comprehensive levels of serum Enzymes and lipid profile | testing in MI and stable Angina subjects. Indian journal of Basic & Applied | Medical Research :. ;2011;. 1(1). 13-20.

5-Robert.H.christenson et al; Biochemical markers of acute corona syndromes. Clinical Chemistry.;1998; ;44(8). 1855-1864

6-Moe KT et al "Current trends in diagnostic biomarkers of acute coronary syndrome" (PDF). Ann. Acad. Med. Singap. ;2010; 39 (3): 210–5.

7- Munther K. et al ;Tufts-New England Medical Center Spring. 2008;

8-Mehta, PK; Wei, J; Wenger, NK ;Ischemic heart disease in women: A focus on risk factors.". Trends in Cardiovascular Medicine ;(2014);"25 (2): 140–151.

9-Dernellis J et al ; Effects of thyroid replacement therapy on arterial blood pressure in patients with hypertension and hypothyroidism, *Am Heart J*, ;2002; ;143:718–24.

10. Klein I, Danzi S,; Thyroid disease and the heart, *Circulation*, ; 2007; 116:1725–35.

11-Hanachi P., Moghadam R.H.et al. ;Investigation of Lipid Profiles and Lipid Peroxidation in patients with Type 2 Diabetes. Eur J Sci Res., (2009): 28(1):6-13.

**12-**Siegenthaler, W ; Differential Diagnosis in Internal Medicine: From Symptom to Diagnosis. Thieme. ; 2007;485.

13-Meiattini F. et al; The 4-hydroxybenzoate/4aminophenazone Chromogenic System. Clin Chem; .;1978; 24 (12): 2161-2165

14. Kaplan A et al. Tryglycerides. Clin Chem The C.V. Mosby Co. St Louis. Toronto. Princeton 437 and Lipids; 1984; 1194-1206.

15-Grove T H. ; Effect of reagent pH on Determination of HDL Cholesterol by precipitation with Sodium Phosphotungstate-magnesium Clin Chem;1979; 25:560.

16-WHO., Physical status; "the use and interpretation of anthropometry", J.World Health Organization; (1995); 854:1-452.

17-Friedewald W., Levy R. et al ;"Estimation of the concentration of low-density lipoprotein cholesterol in plasma without use of the ultracentrifuge", ;(1972); Clin. Chem.;

18-Sterling, L., ; Diagnosis and Treatment of Thyroid Disease, Cleveland, CRC Press, 1975;P. 19-51.

19-Young, D.S., Pestoner, L.C., et al, U; Effects of Drugs on Clinical Laboratory Tests, Clinical Chemistry; 1975;21, 3660.

20-Chopra, I.J., Soloman, D.H., et al;; Radioimmunoassay of Thyroxine, J. Clinical Endocrinol1971;. 33, 865.

21- Kulsoom B, Nazrul SH. Association of serum C-reactive protein and LDL:HDL with myocardial infarction. *J Pak* Med Assoc. ;2006; ;56:318

22-Lloyd-Jones D, Adams R, Carnethon M, et al.: Heart disease and stroke statistics. Circulation, 2009; 119:21-181.

23. Jeppesen J, et al ;Triglycerides concentration and ischemic heart disease: an eight-year follow-up in the Copenhagen Male Study.Circulation. ; ; .;1998; 97:1029–1036.

24. Koba S, Hirano T,Kondo T et al.; Significance of small dense low-density lipoproteins and other risk factors in patients with various types of coronary heart diseases. Am Heart J.; 2002; ; 144: 1026-1035.

25. Vera Bittner, B.Delia Johnson et al.; The TG/HDL Cholesterol ratio predicts all cause mortality in women with suspected myocardial Ischemia. A report from women's Ischemia syndrome evaluation. Am.Heart J. 2009;157(3) 548-555

26-Kirkegaard C, Faber J; The role of thyroid hormones in depression". Eur J Endocrinol; 1998;"138 (1): 1–9.

27- http://jnm.snmjournals.org/cgi/content/full/43/1/77

28-Jiunn-Wen Lin, et al; Coronary Vasospasm Associated with Hyperthyroidism; Acta Cardiol Sin; 2010; 26:4851

29-Davies, AG., Griller, D., et al. ; An electron spin resonance study of pentadienyl and related radicals: homolytic fission of cyclobut-2- enylmethyl radicals. J. Chem. Soc. Perkin. Trans., II: 1981; 633-641.

30-Halliwell, B., and Gutteridge, J.M.C. ; Role of free radicals and catalytic metal ions in human disease: an overview. Methods Enzymol., 1990; 186: 1-85

31-S. V. Reddy et al ;Ischemia-modified albumin levels in overt and subclinical hypothyroidism;Journal of Endocrinological Investigation 2015 ;(4);1-6

32-Hauck, J.S. and Bartke, A. ;Effects of growth hormone on hypothalamic catalase and Cu/Zn superoxide dismutase. Free. Rad. Biol. Med., ;(2000); 28: 970-979.

33-Yilmaz, S. et al. ;Oxidative damage and a ntioxidant enzyme activities in experimental hypothyroidism. Cell. Biochem. Funct.; ;(2003);. 21(4): 325-330.

34-Gredilla, R et al.; Thyroid hormone-induced oxidative damage on lipids, glutathione and DNA in the mouse heart. Free Rad. Res.; (2001);. 35(4): 417-425.