Thyroid dysfunction in patients with unstable angina

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Abstract

Background

Unstable angina has considerable effect on homeostasis of thyroid gland with consequences effect in form of mortality and morbidity. Thyroid hormone has an important effect in the cardiovascular system. Small changes of the thyroid hormone within the normal physiological response have been an adverse effect on cardiovascular results.

Aim of study

The study was designed to assess the profile of thyroid hormonein patients with unstable angina who wereadmitted to the Coronary care unit and toevaluate short term affection.

Patients and methods

An observational study was enrolled 40 patients were admitted to the CCU of AL-Diwanyia Teaching Hospital during period from 1st of March 2017 to the 30 of November 2017. Thyroid function tests (FT3, FT4, TSH) were done by ELISA method within 48 hours of admission.

Results

Sick euothyroid syndrome was observed in 42%, subclinical hypothyroidism in 35% and subclinical hyperthyroidism in 23% of patients with abnormal thyroid hormone profile in 65%. A higher prevalence of abnormal thyroid hormone profile (54%) was seen in elderly age group >60 years.

Conclusion

There is statistical significant value of thyroid dysfunction in patients with unstable angina and age groups >60 years, but withno significant difference regarding the gender.

Introduction

1. Atherosclerosis

It is an inflammatory process started in early age and remains the major cause of early disability and death in developed countries. Current estimation by the year 2020 regarding cardiovascular diseases, noted that atherosclerosis, will become the main leading cause of total burden of disease[1]. Atherosclerosis affects different areas of the circulation with different risk factors may predispose to its development and has clear clinical manifestations that depend on the affected circulatory bed. Atherosclerosis of the coronaries commonly causes MIand angina[1].Early lesions have been found in the arteries in persons with sudden death in the 2 and 3 decades.Otherwise clinical presentation often do not manifest until the 6, 7 or 8 decade[2].

2. Ischemic heart disease

2.1.Chronic stable angina

Angina pectoris is the term used to describe the syndrome of chest discomfort resulting from myocardial ischemia during exercise. Anginal symptoms are defined as stable if there is no substantial change in symptoms over several weeks[3].

For some patients with new-onset angina that has been stable over afew weeks, clear distinction between stable and unstable angina is not possible. These patients can be considered to be in an intermediate stage between unstable and stable angina, althoughangina may be considered as unstable when the symptom pattern

worsens abruptly (increase in frequency and duration) without an obvious cause of increased myocardial oxygen consumption [3].

2.2.Acute coronary syndrome

Symptoms of ACS can fluctuate from time to time, depending on myocardial oxygen consumption, emotional stress, or change in ambient temperature[3].

UA and NSTEMI remain the leading causes of morbidity and mortality in the USA accounting for more than 1.5 million hospital admission in the year 2004 alone. these conditions are part of continuum of ACS that range from UA and NSTEMI to STEMIserial measurementof cardiac biomarkers in patients with ACS should be performed.With improvement in the diagnosis and risk stratification of patient with UA and NSTEMI,therapeutic approaches to UA have continued to evolve[4].UA is defined as angina or equivalent ischemic discomfort with at least 1 of 3 features:

a. It occurs at rest (or with minimal exertion), usually lasting more 10 minutes.
b. It is severe and of new onset (i.e., within the prior 4-6 weeks).
c. It occurs with a crescendo pattern (i.e., distinctly more severe, prolonged, or frequent than previously)[4].

2.2.1. Electrocardiography

The first ECG can help risk-stratify patients with UA.Ideally, this should be performed within 10 minutes of arrival to the ED. Patients with ST-segment deviation(i.e.ST-depression or transient ST-elevation), or with pre-existing LBBB are at increased risk for death or MI at 1 year after presentation.T-wave inversions alone are generally not predictive of adverse ischemic events[3].

2.2.2. Clinical risk classification systems of UA

Many scores have been derived to facilitate risk assessment and guide medical therapy in patients with UA.It is essential to note that these scores can also be used to determine which patients may benefit most from early invasive therapy as opposed to amore conservative approach. The Braunwald`s classification system risk–stratifies patients with UA at presentation[3].

Braunwald`s classification of UA

Class	Characteristics
I	Exertional angina, new onset, sever, or accelerated Angina <2 month duration, more frequent angina, angina precipitated by less exertion No rest angina in the last 2 month
II	Restangina , subacuterest angina within the last month but non within 48 hrof presentation
II	Rest angina,acute Rest angina within 48 hr of presentation
Clinical circumstances	
A	Secondary UA caused by anon-cardiac condition,suchas anemia,infection, thyrotoxicosis,or hypoxemia
B	Primary UA
C	Post-infarction UA, within 2 wk of documentedMI[3]

2.2.3. TIMI risk score of UA

Incorporates the combination of age,clinical characteristics, ECG changes,and cardiac biomarkers for risk stratification.

TIMI risk score

Score	% Incidence of recurrent or new MI, recurrent ischemia that requiring revascularization or death
0/1	4.7
2	8.3
3	13.2
4	19.9
5	26.2
6/7	40.9

Scoring system:present risk factor = 1, absent = 0

- Age more than 65 y
- Prior usage of aspirin in past 7 days
- Presence of more than 3 risk factor for CAD
- Presence of ST-segment deviation on admission ECG
- More than 2 anginal attack within past 24 hr
- Prior stenosis of coronary artery $\geq$ 50%
- Elevated cardiac biomarkers[3]

A higher score correlated with an increasing in the incidence of or recurrent or MI, recurrent ischemia that requiring revascularization and death. [3]

2.2.4. Signs and symptomsof UA

Chest pain due to UA may be rest pain or may be triggered with minimal exertion and can be new onset or increased in severity and frequency or precipitated with less effort than prior angina. Compared with stable angina,chest pain in UA is usually more sever and protracted and usually requiring several doses of sublingual nitroglycerin or extended periods of rest for relief.UA and NSEMI cannot be differentiated on the basis of chest pain criteria or ECG abnormalities alone.The only way, differentiation can be made with evidence of myocardial necrosis by measurement of cardiac biomarkers [3].

ACS has considerable effect on thyroid gland function with consequences effect in terms of mortality and morbidity[5].

3. Thyroid gland

3.1. Functional anatomy and physiology

The thyroid gland produces 2 related hormones, T4 and T3 acting through thyroid hormonereceptors (α and β), these hormones play an essential role in cell differentiation during development and help maintain thermogenic and metabolic homeostasis in the adult. Autoimmune disorders of the thyroid gland can stimulate overproduction of thyroid hormones (thyrotoxicosis) or cause glandular destruction and hormone deficiency (hypothyroidism),

TSH (secreted by the anterior lobe of pituitary gland) plays an important role in control of the thyroid secretion [6]. Thyroid hormones are derived from thyroglobulin, a large iodinated glycoprotein, which is iodinated on tyrosine residues. T4 is secreted from the thyroid gland in about 20-fold excess over T3. Both hormones are bound to plasma proteins, including (albumin, TTR, TBGand TBPA). The plasma-binding proteins delay clearance, increase the pool of circulating hormone, and may modulate the delivery to selected tissue sites. When effects of various binding proteins are combined, approximately 99.98% of T4 and 99.7% of T3 are protein bound. T4 is converted to T3 by the deiodinase enzymes.[6] T4 to T3 conversion is affected by fasting, systemic illness and a variety of drugs including propyl thiouracil, amiodarone, propranolol, glucocorticoids. T4 and T3 inactivates by type III deiodinase and

both are the most important source of reverse T3[7].

3.2. Sick euothyroid syndrome or(non-thyroidal illness)

Any severe or acute illness can lead to changes in the level circulating TSH or thyroid hormone in the absence of thyroid abnormalities, making misleading by these measurements. These hormonal changes is mostly related to release of cytokines (such as interleukin-6)[8].The routine testing of thyroid function should be avoided in acutely ill patients unless there is a strong suspicion of thyroid disorders.Decrease in total and unbound T3 levels (low T3 syndrome) with normal levels of T4 and TSH is the most common hormonal profile in SES.There is good correlation between thelevel of decrease in T3 and the severity of illness[8]. There is impairment in the conversion T4 to T3 by peripheral 5′ deiodination,that leading to increase level of rT3 and reduced in its clearance (which is the major basis for increased rT3).T4 is alternately metabolized to inactive T3 sulfate. This low T3 state is adaptive as assumed generally because it can be induced by fasting in normal persons[8].

Very ill patients may appears a obvious decrease in total T4 and T3 levels (low T4 syndrome). With decreased tissue perfusion, muscle and liver expression of the type 3 deiodinase leads to accelerated T4 and T3 metabolism and this has a poor prognosis.Another key factor in the decrease in T4 levels is change in binding to TBG, useful features to consider include previous history of thyroid abnormalities, severity and patient's curse of acute illness, presence of drugs that may affect level of thyroid hormone, rT3, TSH and unbound thyroid hormones[8]. The diagnosis of SES is presumptive, given the clinical manifestation and laboratory test values; only resolution of results with clinical recovery can clearly prove this abnormality. Treatment with thyroid hormone is controversial for SES [8].

3.3. Subclinical hypothyroidism

Serum level of TSHis increased, and T3 and T4 level are at the lower end of the normal value. This may continue for many years, but there is a risk of progression to overt failure of thyroid gland, particularly if thyroid peroxidase antibody are present or TSH level increase more than 10 mU/L[2]. In state of non-specific symptoms, a trial of levothyroxine treatment may be appropriate. In patients with positive autoantibodies or TSH more than 10 mU/L, it is preferable to treat the thyroid failure earlier better than risk of loss the follow up and subsequent manifestation of hypothyroidism. Levothyroxine should be given in a dose enough to restore normal serum TSH level[2].

3.4. Subclinical hyperthyroidism

Serum level of TSH is undetectable, and serum T3 and T4 are at the upper end of normal values. This is most often found in multinodular goiter in elderly patients, which are at increased risk of AF and osteoporosis, and they may have mild thyrotoxicosis and need 131 radioactive iodine therapy. Annual review is important, as overt thyrotoxicosis may appear with elevated T4 and/or T3 level is 5% yearly[2].

According to data from the ministry of health in Brazil, the CVD are the leading cause of death and third leading cause of hospitalization[9]. CVD have similar pathophysiologic mechanisms which lead patient from risk factors (dyslipidemia, high blood pressure and smoking to congestive heart failure and end to death)[10]. T3 and T4 have a major effect on the CVS[11]. Systolic and diastolic cardiac abnormalities, HTN, and disorders of heart rhythm can be attributed to thyroid disease.Overt hyperthyroidism and hypothyroidism impact results in patients with CAD.

Subclinical hyperthyroidism can be act as an independent risk factor for all cause of and cardiovascular mortality[12]. Subclinical hypothyroidism was considered as an independent risk factorin elderly female for atherosclerosis and MI[13].

Thyroid hormone has an important effect in the function of CVS and cardiac hemodynamic [14, 15], and to maintain the cardiovascular function[16]. A minor change in thyroid condition affects ventricular function, cholesterol level, heart rate and rhythm, with increases risk of CAD and cardiovascular mortality [17]. Decrease in thyroid hormones is a frequent observation and is considered as an energy adaptation to acute stress which may lead to cardio protection[18] .

Characteristics and clinical signs of hyperthyroidism (tachycardia, increase cardiac output, myocardial contractility, systolic blood pressure, and basal metabolism, as well as tremor) suggest a hyper adrenergic state. This is all because the sensitivity to catecholamine[19-20]. Hypothyroidism represent a hypoadrenergic state due to the presence of (bradycardia, decrease basal metabolism and cardiac output), and the intracellular catecholamine which has been found to be decrease during hypothyroidism[21-22] .

Subclinical hyperthyroidism and hypothyroidism have recently been proved as clinical issues with negative effects on the CVS[23,24].
Subclinical hypothyroidism is identified by normal levels of FT3 and FT4, and slightly elevated serum level of TSH. This is associated with initial reduced cardiac systolic function, diastolic HT, elevated systemic resistance of vascular system, anatherogenic profile of serum lipid, and inflammatory condition [25]. Subclinical hyperthyroidism is confined to hypercoagulable state, mild reduce of coronary reserve and increased risk of supraventricular arrhythmias.

Recent studies define a correlation between both subclinical hyperthyroidism and hypothyroidism with elevated cardiovascular mortality[26,27] and affecting prognosis in AMI. This changes in thyroid function is thought to be associated with altered systemic homeostasis that caused by the acute ischemic attack or directly related to inflammatory cytokines. The thyroid hormone is rapidly down-regulated in AMI.This may be beneficial during acute ischemic event. Patients with IHD had higher levels of IL-6 and CRP and more reduction of thyroid hormone level in early stage[28].Low T3 level are considered to be major independent indicators of mortality in hospitalized patients with cardiac events[29]. Determination of rT3 levels may be a valuable and simple way to improve identification of patients with AMI who are at high risk of subsequent mortality[30]. The importance of recognizing SES in patients with CSA suggesting bad prognosis[31].

Aim of the study

Our study was designed to assess the potential changes of thyroid profile in patients with UA and to study the impact of these changes on morbidity and mortality for short term affect among those patients.

Patients and Methods

Exclusion criteria

- Patients using drugs like corticosteroids, amiodarone, and on thyroid oranti-thyroid drugs regularly.
- Patients receiving within previous 2 week any iodinated contrast agent.
- Patients with history of established diseases such as malignancy, chronic renal diseases, COPD and liver disease or psychiatric disease.
- Patients with active infection and any conditions that are known to affect profile of thyroid function.

- Patient with hypertension on beta-blocker and type I DM.
- Patients with CSA class I,II,III
- Patients with history of thyroid disease.

This is an observational study, 50 patients with UA were admitted to the CCU at AL-Diwaniyh Teaching Hospital during period from 1st of March 2017 to the 30 of November 2017.
10 patients were excluded for different causes(2 patients refused participation, 1 patients on B-blocker therapy,3 patients on amiodaron,2 patients with COPD, and 2 patients discharge on their responsibility).
40 patients were involved in this study with UA(18 patients had class I,12 patients with class II, and 10 with class III)

Methods
The consent of the patients and official requirement were taken, all patients were subjected to the following:
A) Full history: By applying specific personal questionnaires thorough history of the present illness and past history of previous hospital admission and any medical disorder with particular attention to HTN, DM, cardiovascular disease, dyslipidemia, thyroid disorders, previous history of cardiac attack and family history of similar conditions.

B) Full clinical examination:
C) Investigations:
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1. Essential investigations: CK-MB, cardiac biomarkers, CBC, LFT, RFT, lipid profile(non-fasting) and RBS.
2. Other investigations: ECG, Echo, other radiological investigations when needed during stay in CCU.
3. Specific investigations: include measurement of serum TSH, FT4, FT3 levels by ELISA kits after 48 hr from admission.

Statistical analysis
The collected data are included in a data-based system and analyzed by SPSS, version 23. Parametric-data were expressed as mean ± SD(analyzed statistically by using student t-test) such as age variable difference in group of clinical manifestation of thyroid dysfunction while non-parametric data were expressed in percentages (analyzed by using chi-square). P-value ≤ 0.05 was considered statistically significant.

Results
1. Distribution of patients with unstable angina according to the gender and age:
21(52.5%) out of 40 patients were males, 19(47.5%) were females, 16(40%) belonged to age group >60 years, 24(60%) belonged to age group 40-60 years . The age ranged between 40 years to 75 years of age. The mean age of the patients was 58.7±7.6 years, as shown in table1

Table 1. Distribution of patients with UA according to the gender and age

Variable	No.	%	40-60 yr		>60yr		Mean age
			No.	%	No.	%	
Male	21	52.5	16	40	5	12.5	58.7± 7.6
Female	19	47.5	8	20	11	27.5	
Total	40	100	24	60	16	40	

2. Distribution of comorbidities in patients with UA:

Tables2shows 19 patients were aknown case of HTN and 10 patients were diabetics, 5 patients with CSA and 15 patients with PVC, 3 patients withAF, and 2 patients withSVT.

Table 2. Distribution of comorbidities inpatients with UA

Variable		Positive		Negative		P-value
		No.	%	No.	%	0.6
HTN		19	47.5	21	52.5	
DM		10	25	30	75	
CSA		5	12.5	35	87.5	
Arrhythmias	AF	3	7.5	37	92.5	
	SVT	2	5	38	95	
	PVC	15	37.5	25	62.5	

3. Distribution of lipid profilein patients with UA according to age group:

In non-fasting state, in age group 40-60 there is 14 patients with high total cholesterol,16 patients with high TG,18 patients with high LDL,10 patients with high VLDL, and 6 patients withlow HDL whilein age group >60 years there is 10 patients with high total cholesterol,12 patients with high TG,8 patients with high LDL,7 patients with high VLDL, and 6 patients withlow HDL, (P-value=0.9), as shown in table 3.

Table 3. Distribution of lipid profilein patients with UA according to age group

Variable	Total	T. cholesterol		TG		LDL		VLDL		HDL		P-value
		N	High	N	High	N	High	N	High	N	Low	0.9
40-60y	24	10	14	8	16	6	18	14	10	18	6	
>60y	16	6	10	4	12	8	8	9	7	10	6	
Total	40	16	24	1	28	14	26	23	17	28	12	
N denote to normal												

4. Thyroid hormone profile:

Figure 1 showing 14 patients(35%) out of 40 patients had normal thyroid hormone profile and 26 patients (65%) had abnormal thyroid hormone profile.

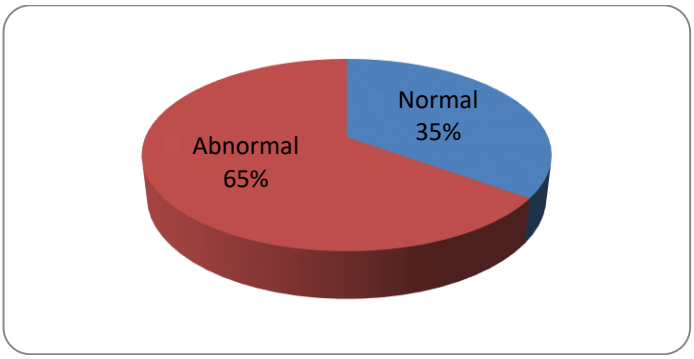

Figure 1. Distribution of thyroid hormone profilein patients with UA

5. Distribution of abnormal profile of thyroid hormone in patients with UA according to the gender and age groups:

13 male patients (50%) out of 26 patients had abnormal thyroid hormone profile and13 female patients (50%) had abnormal thyroid hormone profile. The difference with no statistical significance in prevalence of abnormal thyroid hormone profile considering the gender of the patients, (p- value =0.54), as shown in table 4.

14 patients (54%) were in age group > 60 years, 12 patients (46%) were in age group 40-60 years, with no significant difference (p- value = 0.7).

Table 4. Distribution of abnormal thyroid hormone profilein patients with UA according to the gender and age group

Variable	No.	%	P-value	>60 yr		40-60 yr		P-value
				No.	%	No.	%	
Male	13	50	0.54	8	30.8	5	19.2	0.7
Female	13	50		6	23	7	27	
Total	26	100		14	53.8	12	46.2	

6. Distribution of clinical types of thyroid hormone profilesin patients with UA according to gender:

11 patients (27.5%) out of 40 patients had SES, 6 patients (15%) had subclinical hyperthyroidism, 9 patients (22.5%) had subclinical hypothyroidismand 14 patients (35%) had normal thyroid profile, with no significant difference considering the gender (p-value=0.5). The results are shown in table 5and figure 2.

Table 5. Distribution of clinical types of thyroid hormone profilesin patients with UA according to gender

Variable	SES		Subclinical hyperthyroid		Subclinical hypothyroid		Normal		Total		P-value
	No.	%	No.	%	No.	%	No.	%	No.	%	0.5
Male	5	12.5	2	5	6	15	8	20	21	52.5	
Female	6	15	4	10	3	7.5	6	15	19	47.5	
Total	11	27.5	6	15	9	22.5	14	35	40	100	

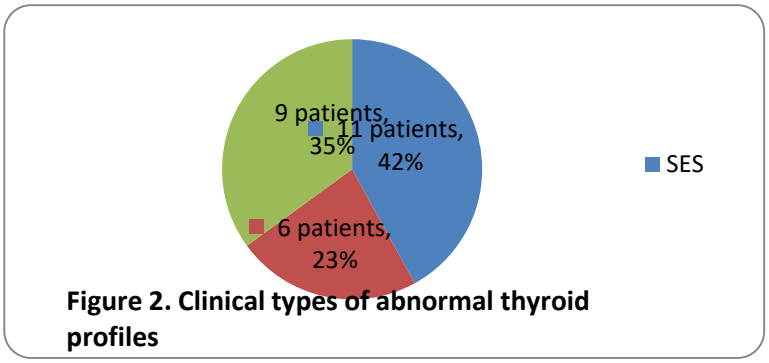

Figure 2. Clinical types of abnormal thyroid profiles

7. D istribution ofthyroid profilesin patients with UAaccording to age group:

In age group of 40-60 years, 5patients(12.5%)out of 24 patients (60%) had SES,4patients(10%)had subclinical hyperthyroidism and 3patients(7.5%)had subclinical hypothyroidism, while in age group> 60 years, 6patients(15%) out of 16 patients (40%)had SES,2patients(5%)had subclinical hyperthyroidism, and 6 patients(15%) had subclinical hypothyroidism,with significant difference between age groups,(p-value = 0.05%), as shown in table6.

Table 6 . Distribution of thyroid profilesin patients with UA according toagegroup

Variable	SES		Subclinical hyperthyroid		Subclinical hypothyroid		Normal		Total		P-value
	No.	%	No.	%	No.	%	No.	%	No.	%	0.05
40-60	5	12.5	4	10	3	7.5	12	30	24	60	
>60	6	15	2	5	6	15	2	5	16	40	
Total	11	27.5	6	15	9	22.5	14	35	40	100	

Discussion

Thyroid hormones have multi-systemic affection mainly on CVS, in-addition, any cardiovascular event can affect thyroid function state through manypathways[32]. A typical change of thyroid hormone metabolism in ill state especially in cardiac conditions characterized by low serum level of circulating T3 has been described in patients with AMI, heart failure and in ayoung and children after cardiopulmonary bypass [33]. This low-T3 syndrome has commonly been considered as aSES and describe asan adaptive compensatory response that decreases energy consumption in diseased conditions[34].

Our study shows changes in thyroid hormone profile were observed in 65% of the patients where 42% had SES, 35% had subclinical hypothyroidism, 23% had subclinical hyperthyroidism . In a study of 400 patients with ACS done by Qari FA, reported change in thyroid hormones profiles was 23.3% of patients [35]). Khalil OA et al. [36]) in their study of 196 patients with ACS reported thyroid dysfunction in 23% of patients [37].Mathur P et al. [38] in their study of 85 patients of ACS reported changes in thyroid hormone profile in 31.7% [38].

The prevalence of thyroid dysfunction is higher in our study in compares with previous studies because of different sample size , different inclusion criteria and the patients in previous studies within healthy programs and examination was done annually and also related to other factors in our country (Iraq) that may affect thyroid hormones production,distribution,clearance like accidental radiation exposure in the environment,by exposed to radiation

contaminated air,food,water and increasing major stressful condition,smoking,iodine status[39],also some patients denying certain drugs and herbal that affect thyroid hormones.But in contrast to these studies, Adawiyah J et al [40] reported a prevalence of 53% of thyroid hormone dysfunction in their study among 85 patients[40], where the result is closest to theresult of our study because of small number of patients that included.

There was no difference with statistical significance in prevalence of abnormal thyroid hormone profile regarding gender of the patients (p =0.54) although number of males in our data more than females,there is slight increase in thyroid dysfunction in females as compare to males but non-significant.

SES was observed in (42%) of patientswith abnormal thyroid hormone profile in our study. Khalil OA et al. [36] in their study reported in 68.9% cases while Adawiyah J et al. [40] found 43.5% of cases in the study [35,40]. Our findings are likely in accordance with findings of Adawiyah J et al [40]because of small number of patients that included also this high prevalence may related to the factors that seem to be associated with development of SES include older age,lower body mass index,bad nutrition,DM,smoking. But, Lower prevalence of SES have been reported in studies of other authors as 21.67% by Potdar S et al. [41], 18.6% by Pimentel RC et al. [42].because large number of patients that included in their studies and different underline factors of participants.

In our study, (35%) of patients with abnormal thyroid profile had subclinical hypothyroidism. In compares to our study, Khalil OA noted 24.5% of patients had subclinical hypothyroidism[36]. this difference related to cut point of TSH for subclinical hypothyroidism in our study was >5mU/L while in other study groups,the cut point of TSH was >10mU/L . In our study,(23%) of patients with abnormal thyroid profile had subclinical hyperthyroidism. In compares to our study, Khalil OA et al reported 6.6% had subclinical hyperthyroidism [36].this difference in above values may related to age of patients in our study middle and old age, as we know subclinical disturbances of thyroid function are more frequent than overt diseases in general population, especially in elderly people.Also some patients neglected annual examination this can make it more difficult to diagnose thyroid disease in an older patients in our country(Iraq).

In our study, there is statistical significant differences of thyroid dysfunction between age groups>60years and 40-60years . In study of 100 patient of ACS by Vigay K S Satyanp,Kohli S C. , was reported higher prevalence of abnormal thyroid hormones in age group >60 years [43].because of many factors ,first the thyroid disease increase with age and more likely remain undiagnosed because presentation of patients with nonspecific symptomslike (mild cognitive impairment ,constipation,diarrhea,anemia,fatigue,sweating,..) as compared with thyroid disease in age group 40-60, that patients present with symptoms of overt diseases,also the aging is associated with a number of physiological changes that can affect result of thyroid function test,inaddition to, the presence of chronic non thyroidal illness and use of drugs that interfere with thyroid function test is common in elderly people[44] that some patients denying during history taken .

In our study no mortality was reported in patients with UA during hospitalization, this finding correlated with Bayrak etal[45] that noted no relationship between thyroid hormones levels and sudden cardiac death and major CVD at 3 and 6 months after follow-up,alsoWartofsky et al.[46] who reported that SES had no significant increase of morbidity in UA. On other hand, Adawiyah J et al [40]. Who found that SES increase cardiovascular mortality and morbidity,this difference is due to most of their patients included had more klips class III and IV during hospitalization period in CCU[40].

Conclusion

UAhas considerable effect on thyroid gland function with consequences effect in form of morbidity and affecting ventricular function, heart rate and rhythm. This study depicts changes in thyroid hormone profile were observed in most patients with UA. No significant difference considering genderwas noted in abnormal thyroid hormone profile. There is statistical significance of thyroid dysfunction in age groups> 60 years.

Limitations

A. Small size sample is a major limitation of our study.
B. Also economic-related limitations which affect sample size and single reading of hormones.
C. Another important factors including short duration of follow up in CCU because many patient discharge on their responsibility.
D. Study of thyroid hormone profile in patients with UA in different earlier studies does not match with inclusion criteria that used in our study and the results are not strictly comparable.

Recommendation

A. Larger studies with uniform inclusion criteria are required.
B. Thyroid function test recommended for elderly patients with UA presented with significant clinical symptoms of thyroid disease to decrease risk of morbidity .
C. Close and regular follow up of patients with UAthat presented with subclinical thyroid disorder every 3-6 month with clinical and laboratory checking and treated accordingly.

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