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Association Between Thyroid Disorders And Cardiac Troponins

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Article Details

Key words: Thyroid dysfunction, Subclinical hypothyroidism cardiac troponins, myocardial injury, TSH, T3, T4.

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ABSTRACT

Background: Thyroid dysfunction has been linked to various cardiovascular abnormalities, including changes in cardiac biomarkers. Cardiac troponins, which indicate myocardial injury, have been studied for their potential association with thyroid disorders, but findings remain inconclusive. Objective: To determine the association between thyroid Disorders and cardiac troponins. To investigate the impact of thyroid disorders on cardiac troponin levels and assess their potential role in diagnosing cardiac conditions. Methodology: Data were collected from Galaxy Diagnostic Laboratory and Al Zahid Laboratory, including 30 participants (24 males, 6 females). Thyroid function tests (TSH, T3, and T4) were analyzed, and cardiac troponin I levels were measured to assess myocardial injury. The results were categorized based on thyroid hormone levels and examined for potential correlations. Results: Out of 30 participants, 26 (86.7%) had normal thyroid function, while 4 (13.3%) had elevated TSH levels, indicative of hypothyroidism. Among those with hypothyroidism, 3 were female. Despite abnormal thyroid function in some individuals, all participants tested negative for troponin I, indicating no direct myocardial injury. The mean T3 and T4 levels remained within normal limits, while TSH was significantly elevated in hypothyroid individuals. Conclusion: This study suggests that thyroid dysfunction, particularly subclinical hypothyroidism, does not directly lead to myocardial injury detectable by troponin I. Although thyroid disorders are associated with long-term cardiovascular risks, they do not independently cause acute myocardial injury. Routine thyroid function assessment may be valuable in cardiovascular risk evaluation, particularly in women and individuals with thyroid abnormalities. Further research with larger sample sizes and high-sensitivity troponin assays is recommended to explore subtle cardiovascular effects.

INTRODUCTION

Thyroid disorders are widespread and can affect 10-15% of the adult population. [1] Both subclinical hypothyroidism (SCH) and subclinical hyperthyroidism (S Hyper) are linked to an increased risk of cardiovascular morbidity and death. [2-3] The prevalence of both SCH and S Hyper is determined by age, gender, iodine status, and smoking behavior. [4] Furthermore, multiple observational studies have confirmed that SCH and the occasional circulating triiodothyronine (T3) syndrome (LT3S) are related with worse outcomes in individuals with acute cardiac problems. [5-8] However, the prevalence and predictors of thyroid dysfunction in patients with acute myocardial infarction (AMI) remain unknown. Cardiovascular diseases including AMI remain a leading cause of mortality and morbidity worldwide.[9] Thyroid function tests are frequently requested both in community-living individuals and in hospitalised patients.[10] This understanding may help to prevent inappropriate diagnoses being made and unnecessary treatments being initiated.[11]

Thyroid disorder syndromes (hyper and hypothyroidism) are regarded to have an effect on the cardiovascular system in a number of approaches.[12] At the same time as the intense consequences of thyroid dysfunction at the cardiovascular device are more readily detectable – specially with hyperthyroidism – the evidence at the long time consequences of thyroid disorder on the heart and at the cardiovascular outcomes is much less clean. this is particularly real of the mild or subclinical styles of hypo and hyperthyroidism.[13] Excess thyroid hormone reasons tachycardia, palpitations, with some degree of exercising impairment and a widened pulse pressure. those modifications arise independently of the motive of the hyperthyroidism four. Subclinical hypothyroidism, or increased thyrotropin (TSH) levels with regular tiers of free thyroxine (FT4), is a common sickness affecting approximately 10% of the adult populace.[14] Inadequate serum thyroid hormone produces impairment of cardiac characteristic and might bring about bradycardia, endothelial dysfunction, multiplied intima-media thickness, diastolic dysfunction, elevated vascular resistance, and pericardial effusion.[15]

Troponin is a regulatory protein complicated placed on the skinny filament of striated muscle tissues. It includes 3 subunits: C, I ,and T. Troponins I and T have 3 isoforms, every managed through distinct genes; 1 is cardiac and the opposite 2 are gradual skeletal and rapid skeletal isoforms. Cardiac troponin T(cTnT) and cardiac troponin I (cTnI) have end up the

biomarkers of preference for the diagnosis of acute myocardial infarction (AMI), excellent ceding the creatine kinase myocardial band (CK-MB).

SCH is biochemically characterized as a TSH level over the upper limit of the reference range with normal thyroid hormone levels.[16] SCH is linked to a higher risk of coronary heart disease (CHD) and death.[17] The occurrence of cardiovascular disorders increases with age. Simultaneously, TSH levels increase with aging. As a result, subclinical hypothyroidism (SCH) is a prevalent illness that has a significant impact on the cardiovascular system among the elderly. Because ECG has limited sensitivity and specificity in detecting acute myocardial infarction (AMI), the AMI criteria were developed in collaboration with the European Society of Cardiology (ESC) and the American College of Cardiology (ACC).

Therefore, a patient has to have at least of the following:

Regular symptoms, a characteristic elevation pattern in cardiac markers (eg, CK-MB isoenzymes), ideally serum troponins (cTnI or cTnT), or an average ECG trace with Q waves that indicate a prognosis of AMI. Four the existing study attempted to discover affiliation among TSH and cardiac markers in elderly subclinical hypothyroid sufferers offering with chest pain and myocardial infarction.

This study seeks to decorate our comprehension of the complete effects of thyroid dysfunction at the anatomy and function of the coronary heart by way of explaining the complex molecular mechanisms and systemic effects, Specially on the subject of the position of cardiac troponins as markers of myocardial injury. The purpose is to establish a foundation for informed medical remedy and destiny research efforts.

MATERIAL AND METHODS

STUDY DESIGN: Cross Sectional Study

STUDY SETTING: Data was collected from Galaxy diagnostic laboratory and Al zahid laboratory Lahore.

DURATION OF STUDY: The investigation was started after the approval of synopsis and the duration of time of the study was 6 months.

SAMPLE SIZE: 30 samples was taken for this diagnosis.

SAMPLING TECHNIQUE: A Stratified random sampling technique was employed for data collection.

SAMPLE SELECTION

INCLUSION CRITERIA

- Patients having age of 18 years and older.
- Patients with Confirmed diagnosis of hypothyroidism, hyperthyroidism, or autoimmune thyroiditis.
- Undergoing evaluation for cardiac health, including cardiac troponin testing.

EXCLUSION CRITERIA

- Pregnant or breastfeeding individuals.
- Current medications affecting troponin levels (e.g., chemotherapy agents).
- Thyroid surgery within the last 3 months.

EQUIPMENT

Automated laboratory analyzers used to measure cardiac biomarkers like troponin I (cTnI) and troponin T (cTnT) and also used to measure thyroid function testing.

SCANNING TECHNIQUE

Blood samples were collected via venipuncture and analyzed using chemiluminescent assay (CLIA) or radioimmunoassay (RIA) to detect and quantify hormone levels and cardiac biomarkers.

RESULTS

Among the 30 participants included in the study, 26 individuals (86.7%) exhibited normal thyroid function, whereas 4 participants (13.3%) were classified as hypothyroid based on their elevated TSH levels exceeding the normal reference range (0.17–5.0 mIU/L). The gender distribution showed that out of the 24 male participants, 23 (95.8%) had normal thyroid function, and only 1 male (4.2%) was classified as hypothyroid. Conversely, among the 6 female participants, 3 (50%) had normal thyroid function, while the remaining 3 (50%) were diagnosed with hypothyroidism. This indicates a higher proportion of hypothyroidism in females compared to males, suggesting a possible gender-related predisposition to thyroid dysfunction.

THYROID FUNCTION TEST ANALYSIS

The mean T4 level among all participants remained within the normal range of 65–175 nmol/L. Individuals with normal thyroid function exhibited a relatively stable distribution of T4 values, while those classified as hypothyroid had some variation in their T4 levels. Similarly,

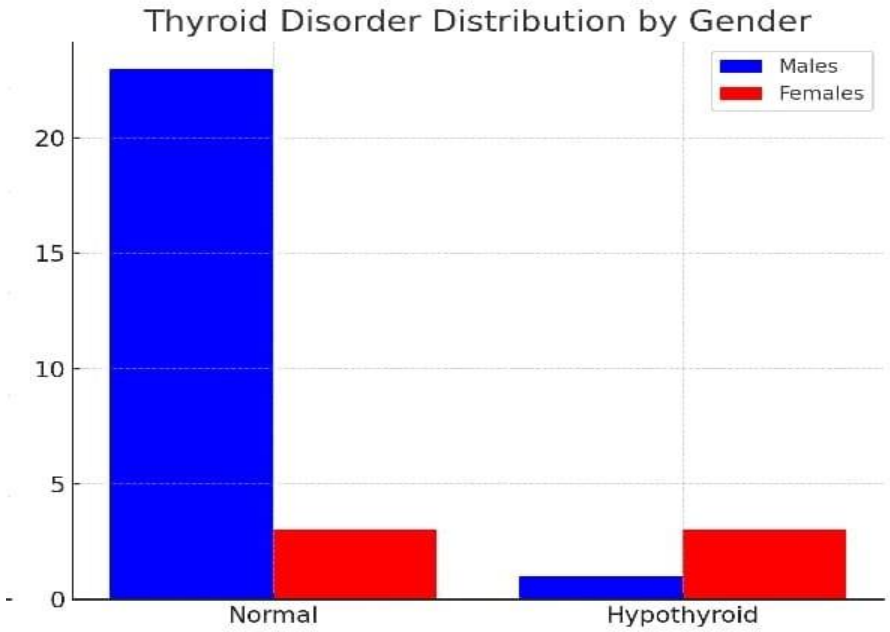
T3 levels remained within the normal reference range (0.9–3.0 nmol/L) across both normal and hypothyroid groups, with no significant deviations observed.

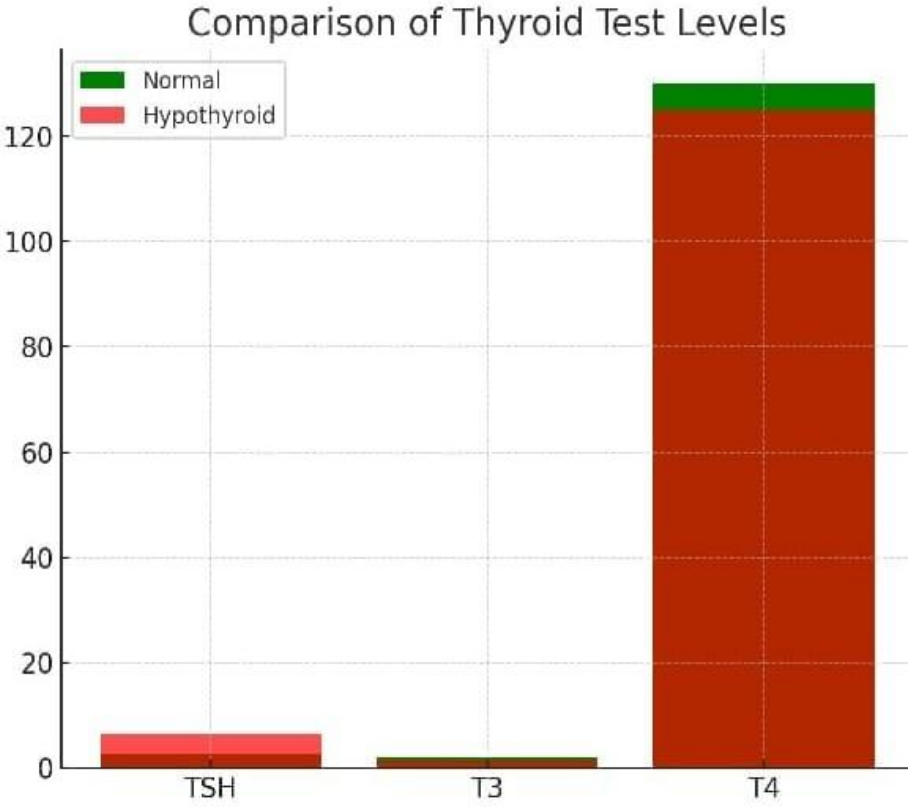
In contrast, TSH levels showed distinct differences between the two groups. Participants with normal thyroid function had TSH values within the reference range (0.17–5.0 mIU/L), whereas those classified as hypothyroid had TSH levels significantly elevated above 5.0 mIU/L, with values recorded at 8.3, 5.8, 6.1, and 5.9 mIU/L. This elevation in TSH levels confirms thyroid dysfunction in these individuals, aligning with the diagnostic criteria for hypothyroidism.

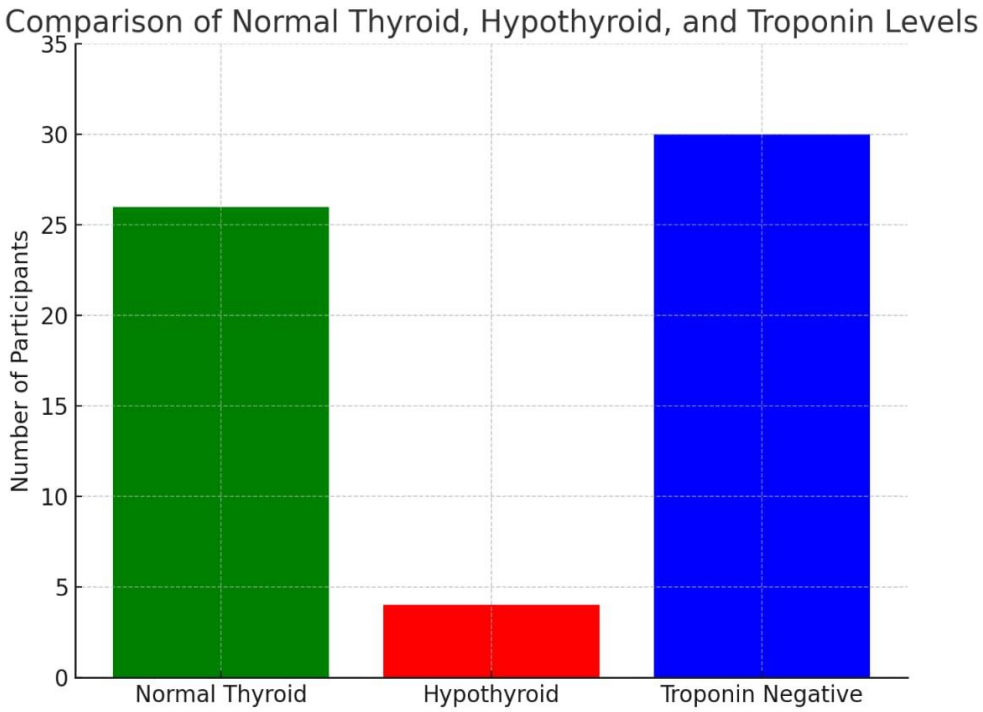
TROPONIN I LEVELS AND CARDIAC MARKER ASSESSMENT

Troponin I levels were negative in all participants, indicating an absence of detectable cardiac injury in both normal and hypothyroid groups. Despite variations in thyroid hormone levels, no direct correlation was observed between thyroid dysfunction and troponin levels. This suggests that thyroid dysfunction, particularly hypothyroidism in this cohort, did not contribute to significant myocardial stress or injury, as reflected by the undetectable troponin I levels.

Measure	TSH (Normal)	TSH (Hypothyroid)	T3 (Normal)	T3 (Hypothyroid)	T4 (Normal)	T4 (Hypothyroid)
Mean	2.76	6.53	1.83	1.88	123.25	120.75
Median	2.9	6	1.8	1.95	122	122
Range	0.6 - 4.3	5.8 - 8.3	1.0 - 2.8	1.4 - 2.2	88.7 - 157.0	95.0 - 144.0
Standard Deviation	0.97	1.03	0.53	0.29	20.33	20.35


Thyroid Disorder Distribution by Gender
Males
Females
20
15
10
5
0
Normal
Hypothyroid


Comparison of Thyroid Test Levels
Normal
Hypothyroid
120
100
80
60
40
20
0
TSH
T3
T4



DISCUSSION

Thyroid dysfunction is widely recognized as a contributing factor to cardiovascular disease due to its impact on metabolism, endothelial function, and vascular resistance. Both hypothyroidism and hyperthyroidism have been associated with changes in blood pressure, heart rate, and lipid metabolism, which can predispose individuals to cardiovascular complications (Biondi & Cooper, 2019). One of the most widely used cardiac biomarkers, cardiac troponin I (cTnI), is highly specific for myocardial injury. However, its relationship with thyroid dysfunction remains an area of ongoing investigation. While some studies suggest that thyroid disorders may contribute to cardiac stress, others argue that troponin levels remain unaffected unless there is concurrent myocardial injury. [18]

This study examined the association between thyroid dysfunction and cardiac troponins, particularly in individuals with abnormal thyroid-stimulating hormone (TSH) levels. While some participants had elevated TSH levels consistent with hypothyroidism, cardiac troponin I levels remained negative in all cases. These findings suggest that mild or subclinical thyroid

dysfunction does not directly cause myocardial injury, reinforcing the specificity of troponin I as a biomarker for acute cardiac events. Thyroid hormones play a crucial role in maintaining cardiovascular health by regulating heart rate, cardiac output, and vascular tone. Hypothyroidism has been linked to increased systemic vascular resistance, impaired endothelial function, and atherosclerosis, all of which contribute to a higher risk of cardiovascular disease (Peeters et al., 2020). Subclinical hypothyroidism (SCH) has also been associated with an increased likelihood of coronary artery disease (CAD) and heart failure, although its direct impact on cardiac biomarkers remains debated . [19]

A study by Hak et al. (2000) found that SCH is an independent risk factor for atherosclerosis and myocardial infarction. However, they noted that patients with SCH do not typically show elevated troponin levels unless they have existing cardiovascular disease. This aligns with the findings of the present study, where no significant cardiac troponin elevations were observed despite the presence of thyroid dysfunction. Conversely, hyperthyroidism is known to cause tachycardia, increased cardiac output, and atrial fibrillation, leading to a higher incidence of heart failure and ischemic events. [20]

Some studies have reported conflicting findings. Kaur et al. (2024) found that elderly patients with SCH may exhibit borderline elevations in high-sensitivity troponin I (hs-TnI), even without acute coronary syndrome (ACS). Wiersinga (2018) suggested that in some patients with severe hypothyroidism, chronic low-level myocardial injury might contribute to mildly elevated troponin levels. However, this is typically observed in critically ill patients rather than in a general population. The lack of elevated troponins in this study suggests that the severity of thyroid dysfunction may determine its impact on cardiac biomarkers. While subclinical hypothyroidism can contribute to cardiovascular stress, it does not necessarily cause myocardial injury detectable by troponin I. [21]

The present study found a higher prevalence of hypothyroidism among females, which is consistent with prior research. Vanderpump (2011) reported that women are five to eight times more likely than men to develop thyroid disorders, primarily due to hormonal fluctuations and a greater likelihood of autoimmune thyroid disease (e.g., Hashimoto's thyroiditis). Additionally, Hak et al. (2000) found that women with SCH are more prone to developing cardiovascular disease compared to men with similar thyroid function. These

findings highlight the need for gender-specific screening and monitoring strategies in populations at risk for thyroid-related cardiovascular conditions. Cardiac troponins are the gold standard for diagnosing myocardial infarction (MI), as they indicate myocardial cell damage. [22]

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