none of the patients had increased levels of free T3. Mechanisms underlying the euthyroid sick syndrome are likely to be related to hormone changes in concentration, distribution, production, clearance, affinity to carrier proteins and response to target organs [(21)]. Some theories have been proposed to justify the "euthyroid sick syndrome", such as decrease in the extra thyroidal conversion of T4 to T3 secondary to lower extracellular clearance of T4 or reduced 5'deiodinase enzyme activity. Other mechanisms may be involved: reduced thyrotropin secretion, with decreased T3 and T4; thyroxine-binding globulin, albumin and the affinity of both to thyroid hormones may be reduced, impairing 5' monodeiodinase's action and T4 and T3 uptake, as well as these post-receptors action[(22)]. In contrast with our study ***Saurabh et al.*** [(23)] reported that euthyroid sick syndrome is the increase in free T4 level. Many theories explain increase free T4 in non thyroidal illness as T4-binding prealbumin (TBPA), and albumin are reduced during non thyroidal illness, there is studies postulate the existence of a binding inhibitor that could explain the observed alterations in free T4 fraction. In this study, we evaluate our patients during inhospital stay and after discharge for major cardiovascular adverse event (MCAE) and mortality during period of 6 month. It was found that 53 patients developed MCAE (27.0%) and 15 patient deceased (7.6%) as whole. However, there was a significant increase of MCAE and mortality (p value <0.0002) in acute coronary syndrome patients with thyroid dysfunction and ESS in comparison to euthyroid patients. These results were in consistent with ***Pimentel et al.*** [(24)] who studied 70 patients with both ST elevation and non-ST elevation myocardial infarction (NSTEMI). They found that in-hospital mortality of the euthyroid sick group was significantly higher than euthyroid subjects. Also, **Kazim *et al.*** [(16)] reported that thyroid dysfunction, particularly sick euthyroid syndrome, was found to be related to inhospital and long term mortality in patients with STEMI undergoing primary percutaneous intervention. ***Lazzeri et al.*** [(25)] found that the failure of intervention was also higher in patients with sick euthyroid syndrome on 641 STEMI patients. Moreover, ***Molinaro et al.*** [(26)] found that cardiac mortality was higher in the group with subclinical hypothyroidism and sick euthyroid syndrome in their study which was conducted on 1026 patient with acute cardiac disease for 3 month duration. Thyroid hormone (TH), apart from its "classical" actions on cardiac contractility and heart rhythm, appears to regulate various intracellular signaling pathways related to response to stress and cardiac remodeling. It affects cardiac remodeling by limiting reperfusion injury, and at later states, by inducing distinct changes in cardiac chamber geometry in a time dependent manner [(27)]. In the other hand, ***Bayrak et al.*** [(28)] noted no relationship between thyroid hormone levels and sudden cardiac death and major cardiovascular disorders at 3 and 6 months follow-up and this difference because the type of thyroid dysfunction in their study was mainly subclinical hypothyroidism and less frequent ESS. As regard to the type of cardiac insult the present study showed a significant increase in MCAE in thyroid dysfunction and also ESS patients in STEMI group (p<0.001) while there was no significant increase of MCAE in NSTEMI and unstable angina [(22)]. In contrast to our result, ***Rodrigo et al.*** [(29)] reported no significance difference between two groups as regard prognosis. Also, ***Adawiyah et al.*** [(19)] reported that ESS in patients with ACS is associated with increased cardiovascular mortality and morbidity and affects UA, NSTEMI and STEMI equally. This difference may be due to their small sample size which was done on 85 ACS patients and most of their patients had more killips class III and IV during hospital stay. In our study we found that thyroid dysfunction in acute coronary syndrome increases thee relative risk of occurrence of shock, arrhythmia and reinfarction by 6.04, 2.05 and 1.67 fold respectively than euthyroid patients. Similar result was reported by ***Adawiyah et al.*** [(19)] who found that ESS increase incidence of arrhythmia and re admission by 15. 6% and 22.2% than euthyroid patient which was 5% and 2% respectively. Our study found that thyroid dysfunction in STEMI group increase relative risk of arrhythmia, reinfarction by 2.25 and 2.4 fold respectively than euthyroid patients while it increases the arrhythmia by 1.5 fold with no impact on reinfarction in NSTEMI and unstable angina group which is consistent with ***Wartofsky et al.*** [(22)] who reported that ESS had no significant increase of morbidity in NSTEMI and UA. Also Thyroid dysfunction in STEMI group increase relative risk of Shock by 8.3 fold than euthyroid patients in comparison to 1.4 fold in NSTEMI and unstable angina group. This was in same line with ***Shilpa and Prashant*** [(30)] who reported decreased left ventricular ejection fraction (LVEF) significantly more in patients who had reduction of serum T3 (p<0.001). ***Pantose et al.*** [(31)] found significant correlation between total T3 and EF% (r=0.56, p=0.0004). ***Adawiyah et al.*** [(19)] noted significant difference in killips classification on day-1 between ESS and non ESS group (p=0.030). In their study, more patients admitted with killips class III and IV (cardiogenic shock) developed ESS and they concluded that thyroid hormones are important for the systolic as well as diastolic functions of the heart. When the thyroid hormone system is down-regulated in AMI, intracellular calcium handling is affected in a way that may contribute to myocardial stunning and reperfusion injury due to calcium overload. Furthermore, there is increased systemic vascular resistance leading to increased cardiac workload due to this down-regulation. If the heart is unable to cope with this, cardiac output and consequently LVEF is reduced. As regard mortality we found statistically significant increase in mortality among thyroid dysfunction patients and ESS (p <0.0003) and (p <0001) respectively in STEMI group as compared to NSTEMI and unstable angina group and thyroid dysfunction in STEMI group increase relative risk of mortality by 9.1 fold than euthyroid patients in comparison to 1.4 fold in NSTEMI and unstable angina group. This was in line with ***Wartofsky et al.*** [(22)] who reported that the importance of recognizing the "Euthyroid Sick Syndrome" in coronary heart disease patients, suggesting an association with poorer prognosis in patients with ST elevated myocardial infarction in form of increased mortality than those with NSTEMI and unstable angina. In contrast to these result, ***Adawiyah et al.*** [(19)] reported no significant difference between STEMI and NSTEMI and UA regarding mortality. This difference may be due to their patient had more killips class III and IV during hospital stay. A forward stepwise multivariate logistic regression analysis was conducted to determine the independent predictors of morbidity in ACS. It was found that hyperglycemia followed by euthyroid sick syndrome (ESS) and increase APPACHE II score > 14 are independent predictor of morbidity (odds

ratio=12.426, 5.063 and 1.092) respectively. This is in agreement with ***Drazner et al., Adawiyah et al., Molinaro et al. and Saurabh et al.*** [32, 19, 26 and 23] who reported that ESS predicts the risk of MCAE than euthyroid patients. Also, we found that APACHE II score > 14 and the presence of ESS (p= 0.007) are most independent predictor of mortality in ACS in our study. This is in agreement with ***Drazner et al., Giorgio et al., Adawiyah et al., Lazzeri et al. and Molinaro et al.*** [32,33,19,25,26] who found that low-T3 syndrome is a strong predictor of death in cardiac patients and might be directly implicated in the poor prognosis of cardiac patients. The APACHE II score is the most commonly used predictor of mortality in intensive care patients. This score involves 12 routine physiological measurements, age and previous health status. It ranges from 0 to 71 points and correlates with the severity of illness [34]. However, this score does not consider hormonal responses to illness, particularly serum levels of cortisol and thyroid hormones, which have been shown to be highly associated with mortality in critically ill patients [35]. Therefore, we can consider that the most important predictor of mortality in ACS is the presence of ESS in those subjects.

6. Conclusion and Recommendations

We can conclude that the thyroid dysfunction in our cohort of ACS is highly prevalent as 23% of our patients experienced thyroid dysfunction and these dysfunction were reported in both STEMI and NSTEMI&UA subjects and the most frequent dysfunction was ESS and ESS was significantly associated with all cause morbidity and mortality but more significant in STEMI group than NSTEMI &UA group. We recommend:

1. Test for thyroid disorders in acute coronary syndrome can give predictor for risk of morbidity and mortality in those subjects.
2. The addition of thyroid dysfunction to APACHE II score for measurement of severity and predict mortality on those subjects of ACS.
3. There is a need for further studies designed to answer the question whether restoration of euthyroidism might influence morbidity and mortality or not?

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