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Original Research Article

Study of thyroid function in patients admitted in intensive care unit

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ABSTRACT

Background: The thyroid gland produces two related hormones, tetraiodothyronine (T4) and triiodothyronine (T3) play a critical role in cell differentiation during development and maintain thermogenic and metabolic homeostasis in the adult. Critically ill patients have been defined as those that by dysfunction or failure of one or more organ system depend on survival from advanced instruments monitoring and therapy. The objective was to study the thyroid dysfunction in critically ill patients admitted in intensive care units and its relation to the mortality and severity of disease.
Methods: This is a cross sectional study carried out in Dr. Pinnamaneni Siddhartha institute of medical sciences and research foundation, Chinoutpalli, Andhra Pradesh from 1st January 2022 to 30th September 2022 involving 100 patients. Patients of age above 18 years, both sexes, admitted to intensive care units with critical illness were analyzed and approved by institutional ethics committee of Dr. PSIMS and RF data were entered in MS-excel and analyzed in SPSS V22 software. Descriptive statistics, Mann-Whitney U test, logistic regression, ROC curves were applied. P values were reported for all statistical tests and a value of<0.05 was considered to be significant.
Results: Out of 100 critically ill patients out of which 17 patients had sepsis, 18 had acute renal failure, 19 patients had acute respiratory failure, 19 patients had diabetic ketoacidosis, 16 patients had congestive cardiac failure, and 11 patients had stroke and their correlation with t3 hormone decrement showed positive correlation.
Conclusions: Thyroid profile can be used in predicting the mortality in ICU patients.

Keywords: Tetraiodothyronine, Triiodothyronine, Critical illness

INTRODUCTION

During critical illness, changes in circulating hormone levels are a common phenomenon.[1] These alterations are correlated with the severity of morbidity and the outcomes of patients in ICU.[2] Thyroid hormones have a crucial role in adapting the metabolic functions during stress and critical illness. Patients who are critically ill may have profound changes in thyroid hormone metabolism. In the 20th century, researchers found that thyroid dysfunction is associated with the mortality of patients admitted to the ICU[3] These alterations in thyroid hormone levels are referred to as "euthyroid sick syndrome" or "nonthyroidal illness syndrome" (NTIS), which is characterized by low serum levels of free and total triiodothyronine (T3) and high levels of reverse T3 (rT3) accompanied by normal or low levels of thyroxine (T4) and thyroid-stimulating hormone (TSH).[4,5] Subsequent studies confirmed the association between NTIS and adverse outcomes in patients with sepsis, acute respiratory distress syndrome, respiratory failure and mechanical ventilation.[6-8] However, the performance of thyroid hormones as predictors of adverse outcomes in general ICU patients has been unimpressive until now. First, the results of previous studies were inconsistent. Researchers in some studies demonstrated that triiodothyronine (T3) levels in non-survivors were

significantly lower than those in survivors, low T3 is an important marker of mortality in critically ill patients.[8] T4 and TSH did not vary between survivors and nonsurvivors.[9] whereas other researchers showed that there was no association.[10]

Whether thyroid hormone indicators can predict ICU mortality is unclear. We therefore undertook study of medical ICU patients to detect the independent predictors of ICU mortality on the basis of thyroid hormone levels (TT3, TT4, TSH) and to evaluate the ability of thyroid hormone level to predict ICU mortality.

METHODS

Sample size

This is a cross sectional study carried out in Dr. Pinnamaneni Siddhartha institute of medical sciences and research foundation, Chinoutpalli, Andhra Pradesh which is a tertiary care, teaching hospital in south India from 1st January 2022 to 30th September 2022.

The sample size taken for this study was 100 patients. No specific sampling technique was used. Ethical issues were discussed and approved by the institutional ethics committee of Dr. PSIMS and RF on October 15, 2021. Signed informed consent was taken prior to the recruitment of subjects into the study and relevant details regarding the purpose, procedure to be carried out and potential hazards of the study were explained to the patient in their own language.

Methods used

Patients fulfilling inclusion and exclusion criteria are taken into study. All the patients had a detailed clinical examination and were managed appropriate to their primary condition. The patients were divided into two groups for comparison: Group 1-survivors (discharged from the hospital) and group 2-non survivors (patients succumbed to their illness inside the hospital). Fasting venous blood samples were collected immediately on admission to ICU from all patients and were subjected for hormone analyses. Samples were tested for total T3, total T4, and TSH. The hormone estimation was done by chemiluminescence assay. The normal reference range for thyroid hormones in our laboratory are TSH (0.3-6.02 µU/ml), T3 (0.5-2 ng/mL), T4 (4.4-12 µg/dL). Any deviation of the hormone results from the normal ranges is considered to be abnormal (low or elevated).

Statistical analysis

Summary data were entered in MS-excel and analyzed in SPSS V22 software. Descriptive statistics, Mann-Whitney u test, logistic regression, ROC curves were applied. P values were reported for all statistical tests and a value of <0.05 was considered to be significant.

Inclusion criteria

Patients of age above 18 years, both sexes, admitted to intensive care units with following diseases included septicemia, acute renal failure, respiratory failure, congestive cardiac failure, diabetic ketoacidosis and stroke.

Exclusion criteria

H/O any thyroid diseases, such as hyperthyroidism, hypothyroidism and thyroid tumors; thyroid nodule found by physical examination when admitted to ICU, pregnancy within previous 6 months, undergoing any hormonal therapy, patients receiving massive blood transfusion/ having steroid/ dopamine therapy and drugs known to interfere with thyroid hormone metabolism e.g. rifampicin, ketoconazole, antiepileptics were excluded.

RESULTS

Out of 100 subjects 58 are men (i.e., 58%) and 42 are women (i.e., 42%) of study population as shown in Figure 1.

In the study population, most of the patients are present in-between the age group of 51-60 years, the minimum age is 19 years and maximum age is 80 years as shown in the Table 1 with mean 48.83 and standard deviation 14.13. No significant relation present between age and mortality (p=0.63).

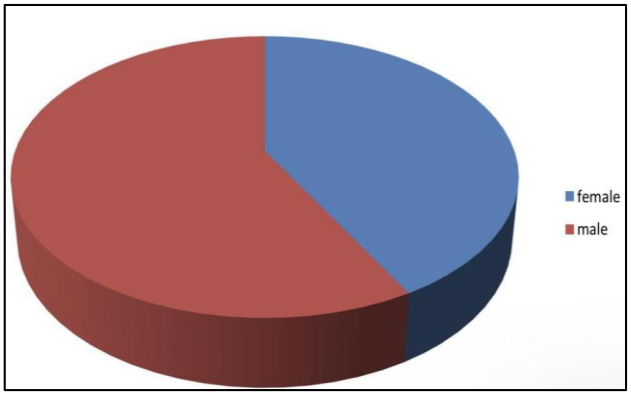

Figure 1: Sex distribution with men 58% and female 42%.

Table 1: Age distribution of patients studied with majority of patients between 51-60, (n=100).

Age (Years)	N	Percentage (%)
<30	12	12.0
31-40	21	21.0
41-50	20	20.0
51-60	26	26.0
61-70	16	16.0
71-80	5	5.0
Total	100	100

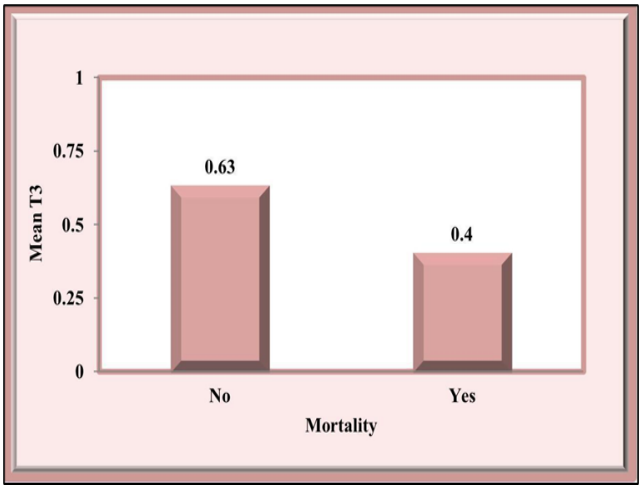

Figure 2: Relation between T3 and mortality.

There was a significant relation between T3 and mortality with p=0.0001 in critically ill patients.

Figure 3: Relation between T4 and mortality.

There is no significant relation between T4 and mortality with p=0.65

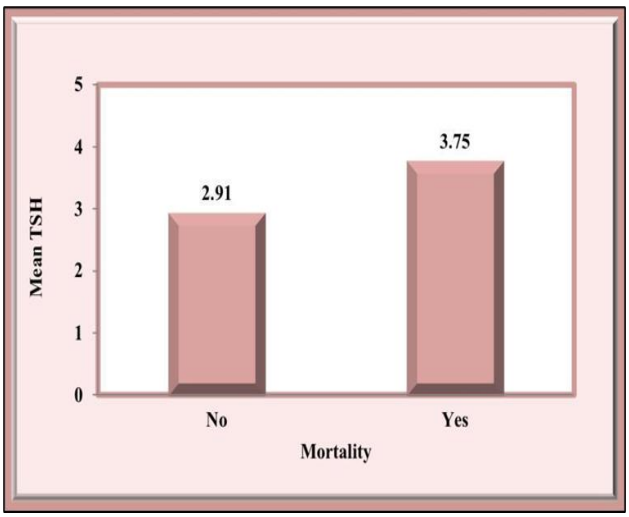

Figure 4: Relation between TSH and mortality.

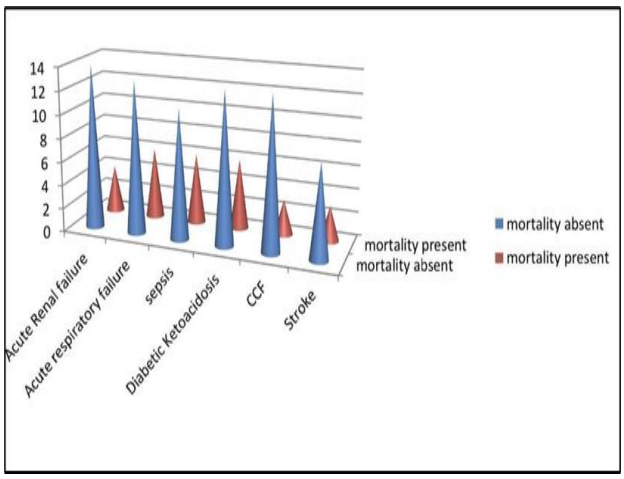

Figure 5: Relation between diagnosis and mortality.

There is no significant relation between TSH and mortality with p=0.16.

Table 2: Relation between T3 and need for ventilation.

Need	T3					P value
	N	Min	Max	Mean	SD	
No	41	0.16	1.75	0.67	0.43	0.004
Yes	59	0.10	1.75	0.49	0.47	
Total	100	0.10	1.75	0.56	0.46	

There is significant relation between T3 and need for ventilation with p=0.004 in critically ill patients.

Out of 100 critically ill patients out of which 17 patients had sepsis, 18 had acute renal failure, 19 patients had acute respiratory failure, 19 patients had diabetic ketoacidosis, 16 patients had congestive cardiac failure, and 11 patients had stroke.

Table 3: Relationship between diagnosis and mortality.

Diagnosis	Mortality				Total	
	No		Yes			
	N	%	N	%	N	%
Sepsis	9	12.5	8	28.5	17	17
Acute renal failure	14	19.4	4	14.3	18	18
Acute respiratory failure	13	18.1	6	21.4	19	19
Diabetic keto-acidosis	17	23.61	2	7.1	19	19
CCF	11	15.3	5	17.8	16	16
Stroke	8	11.1	3	10.7	11	11
Total	72	100	28	100	100	100

Out of 100 critically ill patients, 28 patients (28%) had

died, 8 patients with sepsis, 4 patients with acute renal failure, 6 patients with acute respiratory failure, 2 patients with DKA, 5 patients with CCF with and 3 patients with stroke had died in our study as shown in the Table 3.

DISCUSSION

Critical illness is often associated with alterations in thyroid hormone concentrations in patients with no previous intrinsic thyroid disease. The metabolic support of the critically ill patient is a relatively new target of active research and little is as yet known about the effects of critical illness on metabolism. The nonthyroidal illness syndrome, also known as the low T3 syndrome or euthyroid sick syndrome, describes a condition characterized by abnormal thyroid function tests encountered in patients with acute or chronic systemic illnesses. This syndrome includes low serum levels of triiodothyronine (T3) and high levels of reverse T3, with normal or low levels of thyroxine (T4) and normal or low levels of thyroid-stimulating hormone (TSH). This condition may affect 60 to 70% of critically ill patients.

In this cross-sectional study, 100 critically ill patients admitted in intensive care unit of Dr. Psims are observed. In this study, most of the patients are present in-between the age group of 51- 60yrs, the minimum age is 19 years and maximum age is 80yrs with mean 48.83 and standard deviation 14.13. No significant relation present between age and mortality (p=0.63) Out of the 100 patient's male (58) are more than females (42).

Out of 100 critically ill patients out of which 17 patients had sepsis, 18 had acute renal failure, 19 patients had acute respiratory failure, 19 patients had diabetic ketoacidosis, 16 patients had congestive cardiac failure, and 11 patients had stroke.

T3, T4 and TSH analysis were done, 59 patients (59%) had low T3 level, 41 (41%) patients had normal T3, 31 patients (31%) had low T4, 69 patients (69%) had normal T4 level and TSH was low in 11 patients (11%), 76 patients (76%) had normal TSH and 14 patients (14%) slightly high.

Out of 100 critically ill patients, 28 patients (28%) had died, 8 patients with sepsis, 4 patients with acute renal failure, 6 patients with acute respiratory failure, 2 patients with DKA, 5 patients with CCF with and 3 patients with stroke had died in our study.

Angelousi et al reviewed a prospective Cohort design association between thyroid function test at baseline and the outcome of patients of sepsis or septic shock. Reported analysis of most of the relevant studies identified favor the concept that decreased thyroid function at baseline might be associated with a worse outcome of patients with sepsis or septic shock.[6]

In our study of 17 patients of sepsis, 14 patients (82.35%) had low serum T3 level, 11 (64%) patients had low T4 level and TSH is low in 7 (41%) patients. Compared to the other critically ill patients, sepsis patients have more decrease in TSH and T4. This is similar to the observations present in Monig et al.[11]

Several inflammatory cytokines, such as IL1b, IL6, and TNF-a, can suppress, via direct or indirect pathways, the thyroid function at different levels.[12,13] In sepsis, the increase in the production of pro-inflammatory cytokines is more pronounced than that in other types of critical illness.[14,15] In this respect, baseline levels of thyroid hormones, including T4, T3, and TSH, can be substantially lower in septic patients than in non-septic patients with critical illness of similar severity.[11] The role of the thyroid hormone abnormalities as predictors of outcome of septic patients on top of the known risk prognostic scoring systems warrants also further evaluation.

Kaptein et al study evaluated thyroid hormone indices of patients with acute renal failure without other systemic illnesses (n=12), as compared to patients with critical illnesses in the presence (n=16) and absence (n=6) of acute renal failure. Critically ill patients with acute renal failure differed in that they had lower total T4 and T3 levels and elevated T3 uptake ratio values. The group with critical illness alone differed only in that the total rT3 concentrations were elevated in all patients. The alterations of thyroid hormone indices in acute renal failure are similar to those of other nonthyroidal illnesses with the exception of the normal total rT3 levels. This suggests that the failing kidney or the metabolic consequences of uremia specifically affect rT3 metabolism.[16]

Out of 100 critically ill patients, 18 patients had acute renal failure out of which 7 had low serum T3 level, 5 patients' low serum T4 level and 18 had near normal range serum TSH. Low T3 is commonest abnormality present in our study followed by T4 in acute renal failure patients.

Some authors, but not all, have reported that both total and free T3 behave as markers of survival in uremic patients undergoing either hemodialysis (HD) or peritoneal dialysis (PD).[17-19]

Semple et al. measured serum TT3 and TT4 levels in 16 stable patients with severe COPD and did not detect difference among hypercapnics, normocapnics and controls. The same investigators performed thyroid stimulation tests in 8 hypoxic, stable patients with severe COPD in a subsequent study and found that their basal thyroid hormone levels were normal, but 2 patients showed a delayed response to TRH. They concluded that hypoxemia causes a minor change in the hypothalamic-pituitary-thyroid axis at the hypothalamic-pituitary (central) level. However, the study group is too small to generalize these findings.[20]

Okutan et al evaluated the relation between thyroid hormones and pulmonary function in moderate-to-severe COPD. Thyroid hormone concentrations of COPD patients were within normal limits, but T3 was lower in COPD group than controls.[21]

In our study 19 patients of acute respiratory failure, 11 patients (57.8%) had low serum T3 level, 6 (31.5%) patients had low T4 level and TSH is low in (5%) patients. Our study was similar to previous which shows most of them had low T3 levels and T3 has prognostic value in respiratory failure patients.

For many years, nonthyroidal illness has been considered as a transient adaptive process, but there is increasing evidence that an induced hypothyroid-like state may in itself worsen the patient's clinical status in respiratory failure patients. Hypothyroidism is a known cause of ventilator dependent respiratory failure.[22,23] The mechanisms postulated to be the cause of respiratory failure in hypothyroidism include impairment of the normal ventilatory responses to hypercapnia and hypoxia, diaphragmatic and skeletal muscle dysfunction, pleural effusions, and obstructive sleep apnea.[22,24-28] Also a propensity for respiratory alkalosis that may persist even with appropriately decreased minute ventilation in mechanically ventilated patients has been described.[29] In hypothyroidism, muscle biopsy specimens have shown type II fiber atrophy and up to 50% loss of total mass.[30,31] These findings seem to be a result of increased membrane permeability and decreased adenosine triphosphate formation, manifesting as a rise in creatine kinase levels.[32] It is still unclear whether the low T3 state represents only a biochemical prognostic marker or whether it actually contributes to the development and progression of respiratory failure.

Hu et al showed euthyroid sick syndrome are associated with the severity of DKA.[33]

Joseph et al reported that, 21/110 (19.0%) patients had abnormal thyroid function at diagnosis of TIDM. Of these, 16 had normal thyroid function on reassessment after 45 (3-540) days. Abnormalities of thyroid function occurred more commonly in children with diabetic ketoacidosis (DKA) than those who did not have DKA (9/29, 31.0% vs. 12/81, 14.8%, p<0.025).[34]

In our study of 19 patients of diabetic ketoacidosis, 8 patients (42.1%) had low serum T3 level, 4 (21%) patients had low T4 level and TSH is low in (10%) patients which shows most of them had low T3 levels.

Daniel et al they conducted retrospective study and reported that abnormal thyroid function (TF) is associated with cardiac dysfunction and may result in decompensation in patients with pre-existing heart failure (HF). They reported that TF abnormalities are common in heart failure patients. The rate of HF decompensation is significantly lower in patients with normal TFTs than in those with unmeasured or abnormal TFTs.[35]

Others have found that patients with low T3 syndromes, but without overt cardiovascular disease, have an increased concentration of NT-proBNP, suggesting that low T3 levels may be a contributing factor in the development of cardiac dysfunction.[36] Researchers in clinical studies have also found that low T3 syndromes have a negative prognostic effect in patients with heart failure.[37] In our study of 16 patients of congestive cardiac failure 8 patients (50%) had low serum T3 level, 2 (12.5%) patients had low T4 level and TSH is normal in all patients which shows most of them had low T3 levels.

Alevizaki et al conducted a study on 737 consecutive patients with acute first ever stroke who presented within 24 h from symptom onset and concluded that a high proportion of these patients had low T3 values. The low-T3 syndrome is an independent predictor of early and late survival in patients with acute stroke, and predicts handicap at 1 year.[38]

In our study of 11 patients of stroke 10 patients (90%) had low serum T3 level, 1 (0.9%) patient had low T4 level and all patients had TSH normal which shows most of them had low T3 levels.

Prevalence of thyroid hormones abnormalities-our study showed low T3 (59%) is the commonest abnormality followed by low T4 (31%) and low TSH (11%). This was similar to the data of Kumar et al which showed low T3 (61%) is the commonest abnormality followed by low T4 (14%) and low TSH (7%).[9]

Table 4: Comparison of T3 and mortality of various studies.

Studies	Mean T3 values		
	Survivors	Non-survivors	P value
Our study	0.63	0.40	0.0001
Meyer et al[39]	1.0	0.8	0.04
Suvarna et al[40]	1.17	0.53	0.0001
Kumar et al[9]	0.66	0.49	0.0044

Kumar et al studied thyroid profile in 100 patients admitted to medical ICU showed mean T3 values of 0.66 (66ng/dl) in survivors and 0.49 (49 ng/dl) in non survivors with p=0.0044.[9]

Meyer et al studied the prognostic accuracy of thyroid hormone levels in 103 critically ill adult patients on admission and during follow up in a medical intensive care unit (ICU) Median T3 levels on admission to the ICU were lower in sepsis patients [0.9 nmol/l (IQR 0.6-1.1)] as compared to patients with SIRS and no infection [1.2 nmol/l (IQR 0.8-1.4), p=0.001].[39]

Suvarna et al studied case group consisted of 30 critically ill children mean serum T3 levels (ng/dl) in the cases

were significantly lower than that in controls. In the patients who survived T3 levels significantly improved in the second sample whereas failed to improve in the patients who expired.[40]

Our study showing the relationship between T3 and mortality was similar to the several studies mentioned above.

Table 5: Comparison of T4 and mortality of various studies.

Studies	Mean T4 values		
	Survivors	Non-survivors	P value
Our study	5.72	5.58	0.65
Kumar et al[9]	7.5	6.8	0.5442
Meyer et al[39]	15.3	11.9	0.02
Suvarna et al[40]	8.24	5.9	0.003

Our study didn't show any significant relation between T4 and mortality.

Kumar et al studied thyroid profile in 100 patients admitted to medical ICU showed mean T4 values of 7.5 µg /dl in survivors and 6.8µg/dl in non survivors with p-value 0.5442 [9] In the study of Suvarna et al mean serum T4 levels (µg/dl) in the cases were significantly lower than that in controls. At admission the mean serum T4 levels were not different in cases that subsequently survived or expired. However, in second sample, mean serum T4 levels in cases who expired were significantly lower than in those who survived.[40] There is a discrepancy with reference to the relation between T4 and mortality in various studies. This can be explained because the patients included may have been in different phases of critical illness in different studies. In the acute phase of critical illness, 68 the alterations in thyroid hormones present as decreased T3 and increased T4 and rT3, as well as normal TSH. In the chronic phase of critical illness, central hypothyroidism develops, and NTIS presents as decreased T3, decreased T4 and decreased TSH.[41] In the recovery phase of critical illness, the thyroidal axis begins with a rise in serum TSH, which is eventually followed by normalization in T4 concentration.[41]

Table 5: Comparison of TSH and mortality of various studies.

Studies	TSH		
	Survivors	Non-survivors	P value
Our study	2.91	3.75	0.16
Kumar et al[9]	1.8	3.2	0.77
Meyer et al[39]	3.2	2.4	0.20
Suvarna et al[40]	1.20	1.10	0.264

Previous data from pediatric ICU patients from Mumbai showed low T3 in 80%, low T4 in 50%, and low TSH in 6.7% patients.[40] and it was conducted in 30 critically ill children and controls of less than 12 years age admitted in pediatric ICU. Two samples were collected from all patients, first at admission and second sample at the time of discharge from ICU or death. This study showed that mean T3 and T4 levels were significantly lower in critically ill children than controls. The combination of low T3 and T4 together increased the mortality risk by 30 times. Our study didn't show this relationship. Our study differs in the age of the study population (adults), number of study samples (single sample at admission only), and lack of the control group from the previous study explaining the discrepancy in observed data.[40]

The pathophysiological mechanism underlying the association of lower T3 levels with worse outcomes in ICU patients has yet to be fully defined. It is still unclear whether the alteration in thyroid hormone levels during critically illness is the adaptive physiological response to stress or the maladaptive response requiring treatment.[41] Inhibition of the enzyme 5'-deiodinase, which catalyzes the conversion of T4 to T3, has been considered a possible mechanism responsible for NTIS.[41] Several mechanisms can contribute to the inhibition of 5'- mono-deiodination and thus to the low serum T3 concentrations in critically ill patients: cytokines (such as TNF, IFN-a, NF-B and IL-6), some drugs (amiodarone and high doses of propranolol) and free (non-esterified) fatty acids.

Out of the 100 patients 11 have low TSH with low T4 and low T3. Which suggest there was suppression of TSH central hypothyroidism. In the acute phase of critical illness, the alterations in thyroid hormones present as decreased T3 and increased T4 and rT3, as well as normal TSH. In the chronic phase of critical illness, central hypothyroidism develops, and NTIS presents as decreased T3, decreased T4 and decreased TSH.[41] In the recovery phase of critical illness, the thyroidal axis begins with a rise in serum TSH, which is eventually followed by normalization in T4 concentration.

The conclusions drawn regarding the predictive value of thyroid hormones may be different in other studies because the patients included may have been in different phases of critical illness. Future studies are needed to explore further underlying mechanisms.

Limitations

The inclusion of some patients with undetected thyroid disease before ICU admission may not be ruled out in the present study, even though we tested patients by palpation of the thyroid carefully when they were admitted to the ICU to exclude those with thyroid nodules. Our sample size was small Even though statistically significant relation was obtained between T3 and mortality, statistical analysis cannot be done in each individual disease because of small sample size of each disease although we excluded patients undergoing any replacement therapy except insulin use, as well as those

taking oral amiodarone, it is clear that many other drugs (for example, propranolol, barbiturates, benzodiazepines, furosemide and dopamine) may have interfered with thyroid function, it is difficult to adjust for these potential confounders in clinical practice because so many drugs are involved and some increase and others decrease thyroid hormone levels. However, blood samples were obtained from patients at the time they were admitted to the ICU. Before we obtained blood samples, most of the patients had not been given these drugs.

CONCLUSION

Our study showed low T3 (59%) is the commonest abnormality in ICU admitted patients. Triiodothyronine (T3) is the biologically active thyroid hormone and its low serum levels in critical illness reflect altered thyroid homeostasis and a mechanism of adaptation. There is a significant relation present between T3 and mortality (p=0.0001) and need for ventilation (p=0.004). Serum T4 in non-thyroid illness can be reduced within 24 to 48hrs. The initial decline is predominantly due to decreased binding to carrier proteins, such as thyroid hormone binding globulin (TBG). Our study didn't show any significant relation between T4 and mortality. There is a discrepancy with reference to the relation between T4 and mortality in various studies. TSH levels are commonly within the normal range and only in prolonged illness may be low. Our study didn't show any significant relation between TSH and mortality. The patients included may have been in different phases of critical illness. In the acute phase of critical illness, the alterations in thyroid hormones present as decreased T3 and increased T4 and rT3, as well as normal TSH. In the chronic phase of critical illness, central hypothyroidism develops, and NTIS presents as decreased T3, decreased T4 and decreased TSH In the recovery phase of critical illness, the thyroidal axis begins with a rise in serum TSH, which is eventually followed by normalization in T4 concentration.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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