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STUDY OF THYROID FUNCTION IN PATIENTS ADMITTED IN INTENSIVE CARE UNIT

Dr. Appalanaidu Rongali[1], Dr. P. Sai Sekhar[2], Dr. K Govardhan Reddy

[1]Assistant Professor, Department of General Medicine,NRI Institute of Medical Sciences, Sangivalasa, Visakapatnam, Andhra Pradesh, India.
[2]Associate Professor, Department of General Medicine,NRI Institute of Medical Sciences, Sangivalasa, Visakapatnam, Andhra Pradesh, India.
3rd Year Post Graduate, Department of General Medicine,NRI Institute of Medical Sciences, Sangivalasa, Visakapatnam, Andhra Pradesh, India.

Corresponding author: Dr. P. Sai Sekhar

INTRODUCTION

Circulating hormone levels are usually changes in critical illness.[1] These changes are correlate with intensive care unit (ICU) patients morbidity and the outcomes.[2] Thyroid hormones have important role in body growth as required for metabolism and the immune system. Thyroid dysfunction is correlates with the mortality of patients in the intensive care unit.[3] Alterations in thyroid hormone levels are known as "euthyroid sick syndrome" or "nonthyroidal illness syndrome" (NTIS), defined by low serum levels of free and total triiodothyronine (T3) and high levels of reverse T3 (rT3) along with normal or low levels of thyroxine (T4) and thyroid-stimulating hormone (TSH). [4],[5]

Few studies showed relation between nonthyroidal illness syndrome (NTIS) and adverse outcomes in patients with severe infection [6], major lung failure [7] and mechanical ventilation[8]. Thyroid hormones as predictors of adverse outcomes in intensive care unit patients has not been concluded and the results of the previous studies have not been consistent. Some studies stated that nonsurvivors have lower triiodothyronine (T3) levels compared to survivors.[8] Low T3 is an principal marker of mortality in severe ill patients. T4 and TSH did not vary between survivors and nonsurvivors.[9] whereas other researchers showed that there was no association. [10] It is indeterminate whether thyroid hormone as indicators can influence intensive care unit mortality. Therefore this study undertook the medical intensive care unit (ICU) patients to find out the independent markers of intensive care unit mortality owing to thyroid hormone levels (TT3, TT4, TSH,) and to determine the ability of thyroid hormone level to find out intensive care unit mortality.

MATERIALS AND METHODS

This study was done in NRI Institute of Medical Sciences (NRIIMS) and Research Foundation which is a tertiary care, teaching hospital in South India. 100 patients admitted and treated as in-patients in the intensive care unit (ICU) of NRIIMS were included for this cross-sectional study.

ISSN:0975-3583,0976-2833 VOL13,ISSUE05,2022

Inclusion Criteria

Patients of age above 18yrs, both sexes, admitted in intensive care units (ICU) with following diseases were included

Septicemia
Acute renal failure
Respiratory failure
Congestive cardiac failure
Diabetic ketoacidosis
Stroke

Exclusion Criteria

Known cases of thyroid disorders, such as hyperthyroidism, hypothyroidism and thyroid tumors
Thyroid nodule on physical examination at the time of admission to the ICU
Women conceived in the previous 6 months
Patients on hormonal therapy
Patients receiving massive blood transfusion or having steroid or dopamine therapy and drugs known to interfere with thyroid hormone metabolism, e.g. rifampicin, ketoconazole, antiepileptic's.

Study Design

Patients fulfilling inclusion and exclusion criteria were taken into study. A detailed clinical examination was done and all the patients were managed applicable to their primary condition, and were segregated into two groups.

Group 1 – survivors (discharged from the hospital) and Group 2 – nonsurvivors (patients who were died due to illness in the hospital). Relevant haematological and radiological examination were done. Fasting venous blood samples were collected from all ICU subjects and were subdued for hormone analyses. Total T3, total T4, and TSH levels were tested in these samples. Chemiluminescence assay was done to estimate the hormonal levels. The normal values for thyroid hormones are taken as 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 was considered to be abnormal (low or elevated).

Statistical analysis

Summary data were entered in MS - Excel and analysed in SPSS V22 software. Descriptive statistics, Mann-Whitney U- test, Logistic regression, Receiver operating characteristic curves (ROC) were applied. P values calculated for all statistical tests and a value of <0.05 was considered to be significant.

RESULTS

In this study, 100 patients were participated, most of the patients were in-between the age group of 51- 60 years, with minimum age of 19yrs, maximum age of 80yrs and a mean of 48.83 with standard deviation of 14.13. No significant relation was present between age and mortality (p value – 0.63). 31% of the patients had hypertension, out of which 28% were dead, 38% had diabetes out of which 46.4% of diabetics succumbed later.

ISSN:0975-3583,0976-2833 VOL13,ISSUE05,2022

There was no significant relation between systolic blood pressure (p value 0.98), diastolic blood pressure (p value 0.74), pulse rate (p value 0.12), respiratory rate (p value 0.06), hemoglobin (p value 0.23), total counts (p value 0.77), Random blood sugars (p value 0.95), serum urea (p value 0.06) and serum creatinine with respect to mortality (p value 0.3).

In our study, out of 100 patients 26 had abnormal Electrocardiogram (ECG) in which most of them had left ventricular hypertrophy changes (9 patients), abnormal q waves were present in 5 patients who have suffered from myocardial infarction in the past. Tall t- waves which suggest hyperkalemia were present in 4 patients, p pulmonale which suggests right atrial enlargement was seen in 5 patients, right ventricular hypertrophy (RVH) was present in two patients, p mitral which suggests left atrial enlargement was seen in one mitral stenosis patient.

Among all the patients, 31 had abnormal chest x rays in which consolidation patch present commonly in 15 patients, cardiomegaly present in 9 patients, cavity in 2 and emphysematous changes in 5 patients. Twenty-seven patients had abnormal ultrasonogram of abdomen of which renal parenchymal changes were most commonly observed in 11 patients. Out of the 11 abnormal CT scans, haemorrhage stroke was present in 7 patients and ischemic stroke in 4 patients.

Out of 100 critically ill patients, 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. A total of 28 patients (28%) had died, 8 patients with sepsis, 4 patients with acute renal failure, 6 patients with lung failure, 2 patients with diabetic keto acidosis, 5 patients with congestive cardiac failure with and 3 patients with stroke in our study. There was a significant relation between T3 and mortality with p value 0.0001 and no significant relation between T4 (p value 0.65), TSH and mortality (p value 0.16). There is significant relation between T3 and need for ventilation with p value 0.004 and duration of ventilation and mortality with p value 0.009.

DISCUSSION

The metabolic support of the severly ill patient is a relatively new target of activeresearchandlittleisyetknownabouttheeffectsofcriticalillnessonmetabolism. Thenonthyroidalillnesssyndrome consists of decreasedserumlevelsoftriiodothyronine(T3)withincreasedlevelsof reverse T3 and normal or low levels of thyroxine (T4) and thyroid-stimulatinghormone(TSH), also known asthelowT3syndromeoreuthyroid sick syndrome, which is present in patients withsystemicillnesses and mayaffect nearly 60to70%ofcriticallyillpatients.

In our study, 59 Patients (59%) had low T3 level, 41(41%) patients had normalT3, 31 patients (31%) had low T4, 69 patients (69%)had normal T4 level and TSH was low in 11 patients (11%), 76 patients (76%) hadnormalTSHand14patients(14%)slightlyhigh. Out of 17 patients with sepsis, 14 patients (82.35%) had low serum T3level, 11 (64%) patients had low T4 level andTSH is low in 7 (41%) patients.Sepsis patients have more decrease inTSHandT4 than critically ill patients, asobservationspresentinMonigetal.[11]

Inflammatory cytokines, such as IL1b, IL6, and TNF-a, can suppress the thyroid function through direct or indirect pathways.[12] Insepsis,pro-inflammatorycytokinesisraised more than other types of critical illness. Because of this,baselinevaluesofT4,T3,andTSH lower in septic patients than in critically ill non-septic patientsofsimilarseverity.[11]Furtherevaluation need for

ISSN:0975-3583,0976-2833 VOL13,ISSUE05,2022

theroleofthethyroidhormoneabnormalitiesaspredictorsofoutcomeofsepticpatientsin relation to knownriskprognosticscoringsystems.

Kaptein et al studied thyroid hormone indices in acute renalfailure patients with and without severe illnesses.Critically ill patients with acute renal failure had lowertotal T4 and T3 levels and elevated rT3, which suggestsfailingkidneyorthemetabolicconsequencesofuremiaspecificallyaffectrT3metabolism.[13]

In our study, out of 18 acute renal failure patients,7 subjects had low serum T3 level, 5 Patients with low serum T4 and 18 had near normalrangeserumTSH.LowT3iscommonestabnormalitypresentinourstudyfollowedbyT4 levels in patients with acuterenalfailure.

Arelationshipbetweenserumtriiodothyronine(T3)andseveralmarkersofinflammation, nutrition, and endothelial activation in chronic renalfailure(CRF)hasbeendocumented.[14,15]LowT3levels due to inflammationcan become independent markers of cardiovascular disease mortality inbiochemically euthyroid patients with end-stage renal disease.

Some authors, but not all, have reported that both total and free T3 behave asmarkers of survival in uremic patients undergoing either hemodialysis (HD)[16] orperitonealdialysis(PD).[14]

Semple et al. measured serum TotalT3 and TotalT4 levels in marked COPD patients and concluded that hypoxemia causes a minor change in thehypothalamic–pituitary–thyroidaxisatthehypothalamic–pituitary(central)level.However,thestudygroupistoosmalltogeneralizethese findings.[17]

Banksetal.conducted a study on thyroidhormonesofCOPDpatients, they observed norelationbetweenthyroid hormones and pH, PaO2 or PaCO2 ,also seen an inverse correlationbetweenserum thyroxineandsteroids. None of the patients of our study under systemic steroids. Systemic steroids wereadded to therapy only in exacerbation period, but baseline blood samples weretakenbeforethetherapy.[18]

Wawrzynska et al, estimate the influence of severe respiratory failure (RF) on blood serum thyroid hormone concentration by conducting PO2, pH, PCO2 and TT3, TT4, FT3, rT3, FT4 tests in22Intensive Care Unit (ICU) patients.They concluded that increase of TT3 serum concentration associated with stable outcome of the of patients and lowest levels were seen in "antemortem"patients.ThisfactsuggeststheprognosticvalueofTT3,TT4concentrationmeasurementsinpatientswith RF.[19]

In our study 19 patients of acute respiratory failure, 11 patients (57.8%) had lowserum T3level,6 (31.5%)patients had low T4levelandTSH islow in(5%)patients. Our study was similar to previous which shows most of them had low T3levelsandT3hasprognosticvalueinrespiratoryfailurepatients.

For many years, nonthyroidal illness is thought as a transient adaptiveprocess, but there is increasing evidence that an induced hypothyroid-like statemay itself worsen the patient's clinical status in respiratory failure patients.Hypothyroidismleadstoventilatordependentrespiratoryfailure.[20] The mechanisms

ISSN:0975-3583,0976-2833 VOL13,ISSUE05,2022

postulated to be the cause of respiratory failureinhypothyroidismincludeimpairmentofthenormalventilatoryresponsestohypercapnia andhypoxia,diaphragmaticandskeletalmuscledysfunction, pleural effusions, and obstructive sleep apnea. In hypothyroidism, muscle biopsy specimens have shown type IIfiberatrophy
andupto50%lossoftotalmass.Thesefindingsseemtobearesultofincreasedmembranepermeabi lityanddecreasedadenosinetriphosphateformation,manifesting asariseincreatinekinase levels.[21]

Still it is indeterminatethatthelowT3staterepresentsonlyabiochemicalprognosticmarkerorwhetheritc ontributestothedevelopmentandprogressionofrespiratoryfailure.

Tahirovic et al studied concentrations of thyroid hormones in 62 type I diabeticchildren and adolescents. Patients with poor control (HbA1c greater than10%) and diabetic ketoacidosis had significantly lower T3 and higher rT3 levels (p lessthan0.001)comparedtothematchedcontrols.[22]

In this study of 19 patients of diabetic ketoacidosis, 8 patients (42.1%) had lowserum T3 level, 4 (21%) patients had low T4 level and TSH is low in (10%)patients.whichindicates mostofthe subjects hadlowT3levels.

ConstantinosPantos,AthanasiosDritsas,IordanisMourouzisetalstudiedThyroid hormone is a critical determinant of myocardial performance in patientswith heart failure: potential therapeutic implications. In this study, 37 patients withmean ejection fraction (EF%) of 26.2 were included. Myocardial performance wasassessed by echocardiography and cardiopulmonary exercise testing.

Total tri-iodothyronine (T3), thyroxine (T4), and TSH levels were measured in plasma analysis revealed that total T3 was an independent predictor of VO(oxygen consumption). Fluctuations in levels of thyroid hormone were closely correlated to myocardial functional status in patientswithheartfailure.[23]

In our study of 16 patients of congestive cardiac failure 8 patients (50%) had lowserum T3 level, 2 patients (12.5%) hadlow T4 level and TSH is normal in allpatients

Alevizaki*etal*.,conductedastudyon 737consecutivepatientswithacutefirsteverstroke whopresented within 24 h fromsymptomonsetand concludedthat ahigh proportion of these patients had low levels of T3 . The low-T3 syndrome is anindependent predictor of early and late survival in patients with acute stroke andpredictshandicapat1year.[24]

Apurva Pande et al conducted a study on patients of acute hemorrhagic stroke,highmortalityrateswereobservedinpatientswithalowT3andT4.Consequently,lowT3an dlowT4predictapooroutcomeinpatientsofhemorrhagicstroke.[25]

In our study of 11 patients of stroke 10 patients (90%) had low serum T3 level,1 (0.9%) patients hadlow T4 level and all patients had TSHnormal whichshowsmostofthemhadlowT3levels.

PrevalenceofThyroidhormones abnormalities

Our study showed low T3 (59%) is the commonest abnormality followed by lowT4(31%)andlowTSH(11%). Thiswascorresponding tothe data ofKumaretall

ISSN:0975-3583,0976-2833 VOL13,ISSUE05,2022

whichshowed low T3 (61%) is the commonestabnormalityfollowedbylow T4(14%)andlowTSH(7%).[9]

Table 7:Prevalence ofThyroidhormonesabnormalities

studies	T3%	T4%	TSH%
Ourstudy	59%	31%	11%
Kumaretal [9]	61%	14%	7%
Meyeretal [26]	57%	26%	-
Suvarnaetal [27]	80%	50%	6.7%

Table8:ComparisonofT3andmortalityofvariousstudies

Studies	MeanT3values		
	survivors	Nonsurvivors	Pvalue
Ourstudy	0.63	0.40	0.0001
Meyeretal	1.0	0.8	0.04
Godsmithetal	1.99	1.02	0.001
Suvarnaetal	1.17	0.53	0.0001
Kumaretal	0.66	0.49	0.0044

Our study showing the relationship between T3 and mortality was comparable to theseveralstudiesmentionedabove.

ISSN:0975-3583,0976-2833 VOL13,ISSUE05,2022

Table9:ComparisonofT4andmortalityofvariousstudies

studies	T4		
	survivors	**Non survivors**	**Pvalue**
Ourstudy	5.72	5.58	0.65
Kumaretal	7.5	6.8	0.5442
Meyeretal	15.3	11.9	0.02
Suvarnaetal	8.24	5.9	0.003

Ourstudydidn't showanysignificant relationbetweenT4 andmortality.

There is a discrepancy with reference to the relation between T4 and mortality invariousstudies. This is due to patients taken in several studies are not in same stageofcriticalillness. Variations in thyroid hormones levels are decreased T3, increased T4, rT3 and normal TSH seen inthecriticalphaseof illness. Lower T3 and T4 levels along with low TSH seen in chronic stage of severe illness and also in centralhypothyroidism together with NTIS. During improvement the phase of critical illness, the thyroidal axisactivated as indicated by increase in serum TSH levels and after that will be normalization inT4concentration.[28]

Table10: ComparisonofTSHandmortalityofvarious studies

Studies	**TSH**		
	Survivors	**Non survivors**	**Pvalue**
Ourstudy	2.91	3.75	0.16
Suvarnaetal	1.8	3.2	0.77
Kumaretal	3.2	2.4	0.20
Belloetal [8]	1.20	1.10	0.264

Previous data from paediatric ICU patients from Mumbai showed low T3 in 80%,low T4 in 50%, and low TSH in 6.7% patients, and it was conducted in 30critically ill children and controls of less than 12 years age admitted in paediatricICU. Two samples were collected from all patients, first at admission and secondsample at the point of discharge from ICU or death. [9] It inferred that T3 and T4 levels were significantly lower in critically ill children andcombination of low T3 and T4 together increased the mortality

ISSN:0975-3583,0976-2833 VOL13,ISSUE05,2022

risk by 30 times.Our study didn't show this relationship and differs in the age of the studypopulation (adults), number of study samples,lackofthecontrolgroupfromthepreviousstudyexplainingthediscrepancyinobservedd ata.[27] There was also a significant relation between duration of ventilation and mortalitywithpvalue0.009whichis similartothestudyShehabiY etal.[29]

Bacakoglu, F et al showed low T3 and T4 levels increase the rates of invasivemechanical ventilation and mortality.[30]Our study also shows significant relationbetween need for ventilation and T3 values with p value 0.004. T3 can alsopredictthemortalitywithareaundercurve0.873.

underlying mechanism which defines the relation of lower T3 levelswith morbidity and mortality outcomes in ICU patients. It is inderminatewhetherthe role of thyroidhormones is there in theadaptive or the maladaptive response requiringtreatment.[28] Researchers assessing in supplemental therapy of NTIS have disconcordance results.[31] The change of T4 to T3 is accelerated by Inhibition of the enzyme 5'-deiodinase has been attributed forNTIS.[28] Many mechanisms responsible for inhibitionof5'-monodeiodination leads to the sub normal serum T3 concentrations in terminally illpatients. Inother studies have concluded that inflammatory cytokines(IFN-a, NF-B) are consistent withNTISinvitro.[32]

Out of the 100 patients, 11 have low TSH with low T4 and low T3. Which suggesttherewassuppressionof TSHcentralhypothyroidism.Recovery of critical illness patients, starts as hike in values of serum TSH, later normalizationinT4concentration were observed.

Table 11: Clinical studies considering the thyroid function serum parameters asstatisticallysignificant predictorsofpoorprognosis.

Reference	Issue	N = study people	Predictor
Scosciaet al [7]	Respiratoryfailure	32	T3
Borkowski et al [33]	Septic shock	20	TSH
Turemetal [34]	Criticalillness	206	T3
Schillingetal [35]	Complextrauma	20	T3
Wardlset al [36]	Criticalillness	42	T3,T4
Iervasigetal [37]	Heart failure	573	T3
Peeters et al [38]	Criticalillness	451	rT3, T3/rT3 ratio
Kumaretal [9]	Criticalillness	100	T3
Ourstudy	ICU criticallyillpatients	100	T3

ISSN:0975-3583,0976-2833 VOL13,ISSUE05,2022

LIMITATIONSOFTHISSTUDY

patients with unindentified thyroid disorders before ICUadmissioncan notberuled out in the presentstudy evenafter a thorough and careful examination ofthethyroidin the testedpatients whowereadmittedtothe ICU.

Even though statistically significant relation was obtained between T3 andmortality, statisticalanalysis cannot be done in each diseasebecauseofsmallsamplesize.

Confounding effects of some drugs like furosemide benzodiazepinesand dopamine commonly used in clinical practice may not be ruled out.

CONCLUSIONS

The evaluation of altered thyroid function parameters in systemic illness andstressremainsacomplexissueandpresentsmanydiagnosticproblemsbecause changes that occur atalllevelsof the hypothalamic-pituitary-thyroidaxis.

Uniquechangesinthyroidfunctionparametersareobservedinvariousrelevant clinical states, including starvation, fasting, cardiac disease, renaldisease,respiratoryfailure,diabetic ketoacidocis,sepsisandstroke

OurstudysuggestslowT3isthecommonestabnormalityseenintensive care unit admittedpatients

It also observed thatlowT3isanimportantmarkerofmortalityinintensive care unit admittedpatients

Thereisa significant relation betweenlowT3andneedforventilation

Whether alterations in thyroid parameters during critical illness representadaptivechangestoconserveenergyexpenditurebyreducingmetabolic activityisstill debatable.

It issuggestedthatinintensive care unit patientsT3levelsshouldbedoneandusedasa prognosticmarkerformortalityandneedforventilation

Furtherstudiesshouldaimwithlargenumberofpatientstoclearlyestablishthe strength of the above-mentioned association or even examine whether causal relationship between thyroid dysfunctionand adverse outcomeexists.

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ISSN:0975-3583,0976-2833 VOL13,ISSUE05,2022

Ageinyears	No.ofPatients(n=100)	%
<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	100.0

Table1: Agedistribution ofpatientsstudied

Variable	Mortality	N	Minimum	Maximum	Mean	SD	P-value
Age	No	72	19.0	80.0	49.13	14.38	0.63
	Yes	28	22.0	75.0	48.07	13.69	
	Total	100	19.0	80.0	48.83	14.13	

Table2:-Relationbetweenageandmortality

ISSN:0975-3583,0976-2833 VOL13,ISSUE05,2022

Diagnosis	Mortality				Total	
	No		Yes			
	Count	%	Count	%	Count	%
Sepsis	9	12.5%	8	28.5%	17	17.0%
Acute Renalfailure	14	19.4%	4	14.3%	18	18.0%
Acute respiratoryfailure	13	18.1%	6	21.4%	19	19.0%
DiabeticKetoacidosis	17	23.61%	2	7.1%	19	19.0%
CCF	11	15.3%	5	17.8%	16	16.0%
Stroke	8	11.1%	3	10.7%	11	11.0%
Total	72	100.0%	28	100.0%	100	100.0%

Table3:-Relationbetweendiagnosisandmortality

Variable	Mortality	N	Minimum	Maximum	Mean	SD	P-value
T3	No	72	.10	1.75	0.63	0.45	0.0001
	Yes	28	.10	1.70	0.40	0.46	
	Total	100	.10	1.75	0.56	0.46	

Table4:-Relation betweenT3 andmortality

ISSN:0975-3583,0976-2833 VOL13,ISSUE05,2022

Variable	Mortality	N	Minimum	Maximum	Mean	SD	P-value
T4	No	72	1.41	11.99	5.72	2.15	0.65
	Yes	28	1.41	11.74	5.58	2.89	
	Total	100	1.41	11.99	5.68	2.36	

Table5:-Relation betweenT4 andmortality

Variable	Mortality	N	Minimum	Maximum	Mean	SD	P-value
TSH	No	72	.10	8.90	2.91	2.18	0.16
	Yes	28	.10	8.30	3.75	2.44	
	Total	100	.10	8.90	3.14	2.28	

Table6:-Relationbetween TSHandmortalit