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Biochemistry

EVALUATION OF THYROID DYSFUNCTION IN CHRONIC KIDNEY DISEASE

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ABSTRACT

Aim: The aim of our study was to estimate the levels of Serum T3, Serum T4 and Serum TSH to establish a correlation of these hormones in CKD patients.

Study Design: A hospital based cross sectional study was conducted on patients (both male & female) attending the Out Patient Department of Nephrology & Medicine and those admitted in SMI Hospital for a period of 4 months from January 2014 to April 2014. 54 cases (both male and female) above 15 years of age were selected randomly for the study. A control group of 50 subjects comprising of Medical staff / students who had no signs & symptoms of Renal disease were included in the study.
Exclusion criteria was age less than 15 years and those not willing to participate.

Methodology: 5ml blood was collected in sterile tubes from all the 104 subjects and was analyzed for serum T3, serum T4 and Serum TSH on a fully automated Biochemical Analyzer of Orthoclinical Diagnostics 5600.

Results: Serum T3 levels were found to be significantly low ($p<0.01$) in CKD Patients as compared to the control groups. Serum T4 levels are significantly low ($p<0.05$) and serum TSH is significantly elevated ($p<0.01$) in CKD patients when compared with control group.
The statistical analysis demonstrated that there is a nonsignificant ($p>0.05$) elevation of serum T4 levels in CKD female group when compared to male CKD group. Serum T3 and Serum TSH were nonsignificantly ($p>0.05$) lower in CKD female group than CKD male group.

KEYWORDS : CKD, Thyroid hormones.

Introduction: CKD is usually a progressive, irreversible condition and is the 8th leading cause of death in the United States[1], Risk factors for CKD include Diabetes, Hypertension, hyperlipidaemia and Thyroid disorders.

The pathophysiology of CKD involves 2 broad sets of mechanisms of damage:

1. Initiating mechanisms specific to the underlying etiology eg: immune complexes and mediators of inflammation in certain types of glomerulonephritis, or toxin exposure in certain diseases of the renal tubules & interstitium.
2. Set of progressive mechanisms involving hyperfiltration and hypertrophy of the remaining viable nephrons, that are consequences following long term reduction of renal mass, irrespective of underlying etiology.

Thyroid hormones affect renal clearance of water load by their effects on the GFR[2]. Thyroid hormones influence Na^+ reabsorption at the Proximal convoluted Tubule by increasing the activity of Na-K-ATPase[3] and tubular potassium permeability[4].

Thyroid dysfunction affects the Renal blood flow, GFR, tubular function, electrolyte homeostasis and kidney structure[5].

The interplay between thyroid and the kidney in each others functions is known for many years[6]. Thyroid dysfunction affects renal physiology and development, whereas kidney disease could result in thyroid dysfunction. Disorders of the thyroid and kidney may coexist with common etiological factors.

CKD affects the hypothalamus-pituitary-thyroid axis & the peripheral metabolism of thyroid hormones.

In view of the variability of thyroid function tests in patients with CKD in previous studies, it was decided to undertake a prospective clinical & Biochemical study of thyroid function to establish correlation between the two.

Material & Methodology: A hospital based cross sectional study was conducted on patients both male & female attending the OPD of Nephrology and Medicine and those admitted in SMI hospital for a period of 4 months from January 2014 to April 2014. 5ml fasting venous blood was collected in sterile tubes from all the 104 subjects and were analyzed for Serum T3, Serum T4 and Serum TSH using ELISA competitive immunoassay method as described by Sterling L[7] and Serum urea[8] and Serum creatinine[9] were estimated by enzymatic method on a fully automated analyzer 5600 of Orthoclinical diagnostics.

Results: Serum T3 levels were found to be significantly low ($p<0.01$) in CKD Patients as compared to the control groups. Serum T4 levels are significantly low ($p<0.05$) and serum TSH is significantly elevated ($p<0.01$) in CKD patients when compared with control group as shown in Table 1 and depicted graphically in fig 1.

Table 1: Mean values of Thyroid profile, serum urea and serum Creatinine in both test and control groups.

Parameter	Test group (n=54) Mean±SD	Control group (n=50) Mean±SD	t=value	p-value	Level of significant
T3	4.1±1.48	5.03±0.74	1.99	<0.0001	HS
T4	14.2±4.16	15.44±2.39	1.98	<0.038	S
TSH	4.08±3.7	2.23±0.96	2.00	<0.01	S
Urea	137.5±50.52	24.84±5.39	15.68	<0.0001	HS
Creatinine	6.3±2.74	0.825±0.13	14.12	<0.0001	HS

Figure 1: Mean values of Thyroid profile serum urea and serum Creatinine in both groups.

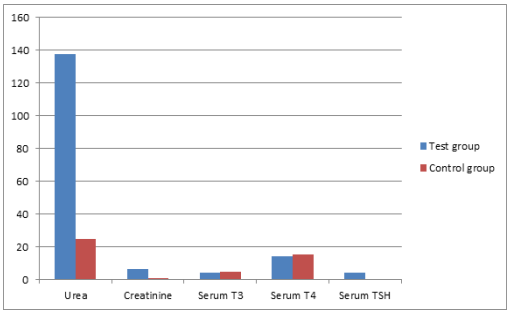
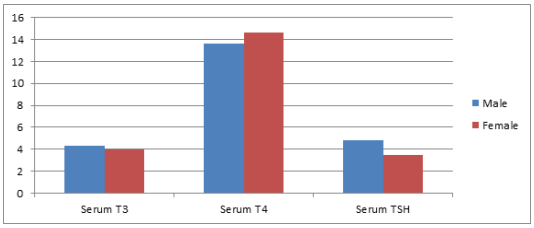


Table 2: Mean values of Thyroid profile in male and female groups.

Parameters	Sex (Mean±SD)		Test values		Level of significant
	Male (24)	Female (30)	t-value	p-value	
Serum T3	4.28±1.01	3.95±1.04	2.03	0.45	NS
Serum T4	13.61±3.97	14.66±4.31	2.01	0.35	NS
TSH	4.79±6.70	3.52±2.22	2.05	0.38	NS

Figure 2: Mean values of Thyroid profile in male and female groups.



The statistical analysis demonstrated that these is nonsignificant ($p>0.05$) elevation of serum T4 levels in CKD female group when compared to male CKD group. Serum T3 and Serum TSH were nonsignificantly ($p>0.05$) lower in CKD female group than CKD male group.

Discussion: CKD is an internationally recognized public health problem affecting 5-10% of the world population[10]. CKD affects the hypothalamus-pituitary-thyroid axis and the peripheral metabolism of thyroid hormone. Low T3 is the most common laboratory finding & Subclinical hypothyroidism is most common thyroid disorder found in CKD patients[5]. Low T3 in CKD may be due to iodothyronine deiodinase which is affected by fasting chronic metabolic acidosis and protein malnutrition as seen in CKD, it may also be due to decreased peripheral conversion of T4 to T3 due to decreased clearance of the inflammatory cytokines like TNFα and IL1[11].

In our study we found low Serum T3 and Serum T4 levels and a slightly raised Serum TSH level as compared to the control group. These findings are in accordance with a study conducted by G. Avasthi et al in the year 2001[12].

A study was conducted by J.Horacek et al in the year 2012[13], they found low T3 and T4 hormone levels, the spearman correlation analysis revealed significant inverse correlation between TSH and thyroid hormones.

In another study conducted by Reuka Pangaluri et al in October 2013[14], showed that TSH was significantly increased in End stage Renal disease.

Conclusion: Chronic kidney disease encompasses a spectrum of different pathophysiological processes associated with abnormal kidney function and a progressive decline in glomerular filtration rate. Despite extensive studies, thyroid status in uremia is still inconclusive. T3 levels in our study are markedly decreased in comparison to control group, T4 levels are also decreased but to a lesser extent & TSH levels are markedly increased in CKD patients in comparison to control group. So we suggest early detection of Thyroid profile status in CKD patients can help in decreasing morbidity and mortality.

References:

1. "Centres for disease control & prevention", leading causes of death, 2009, http//www.cdc.gov/nchs/favats/htm.
2. Emmanouel DS, Lindheimer MD, Katz AI. Mechanism of compared water excretion is hypothyroid rat. J Clin Invest. 1974;54:926-34.
3. Lin HM, Tang MJ. Thyroid hormones upregulates Na, K+ ATPase alpha, & beta mRNA in primary cultures of proximal tubule cells life sci 1997;60:375-82[pubmed].
4. Katz AI, Lundheumer MD. Renal sodium & potassium activated adenosine triphosphatase & sodium reabsorption in hypothyroid rat. J. Clin. Invert. 1973;52:796-804 [pubmed].
5. Basu G, Mohapatra A. Interactions between thyroid disorders & kidney diseases. Indian J Endocrinol metab. 2012 Mar-Apr; 16(2): 204-13.
6. Kaptein EM. Thyroid function in renal failure Nephrol 1986;50:64-72 [pubmed].
7. Sterling L. Diagnosis and treatment of Thyroid disease: Level and CRC Press; 1975: pg 9-51.
8. Lo JC, Chertow GM et al. Increased prevalence of subclinical and clinical hypothyroidism in persons with chronic kidney disease. Kideny Int. 2005;67:1047-52[pubmed]
9. Wheatley T, Edwards OM. Mild hypothyroidism and oedema; Evidence for increased capillary permeability to protein. Clin Endocrinol (oxf) 1983;18:627-35 [pubmed].
10. Eknoyan G, Lanuere N, Barsoum R. The burden of kidney disease; improving global outcomes kidney Int. 2004;66:1310-14.
11. Lim VS, Fang VS, Katz AI, Refetoff S. "Thyroid Dysfunction in chronic renal failure". A study of the pituitary thyroid axis and peripheral turnover kinetics of thyroxine and triodothyronine. Journal of Clinical Investigation 1997:Vol 60(3): 522-34.
12. Avasthi G, Malhotra S, Narang APS, sengupta S. study of thyroid function in patients of chronic renal failure. Indian J Nephrol. 2001;11:165-69.
13. Horacek J, Sulkova DS, Kubisova M, Safranek R, Malisova E, Kalousova M, Svilias I, Maly J, Sobotka L, Zak P. Thyroid Hormone Abnormalities in hemolyzed patients: Low Triodothyronine as well as high Reverse Triiodothyronine are associated with increased mortality. Physiological Research. 2012;61:495-501.
14. Pangaluri R, Seban AS, William E, Padmanaban study of thyroid dysfunction & Insulin Resistance in hemodialysis patients. International Journal of Research in Pharmaceutical & Biomedical Sciences. ISSN 229-3701.