

Original Research Article

Association of Subclinical Hypothyroidism and Kidney DiseaseNita Garg¹, Nil Kamal Kumar², Ishita Gupta³, Gazala A. Tahir³¹Professor & Head, Department of Biochemistry, SGRRIM&HS, Patel Nagar, Dehradun.²Associate Professor, Department of Surgery, SGRRIM&HS, Patel Nagar, Dehradun³MD Residents, Department of Biochemistry, SGRRIM&HS, Dehradun.***Corresponding author**

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Abstract: The aim of our study was to determine the relationship between renal function and subclinical hypothyroidism. A hospital based cross sectional study was conducted on patients attending the Out patient departments of Surgery, Medicine and Gynaec of SGRR Medical College, Dehradun (U.K.) during a period of 8 months form Jan 2016 to Aug 2016. A total No. of 114 cases (28M+86F) and 90 controls (40M and 50F) in the age group 20-60yrs were selected randomly for this study. Exclusion criteria was age less than 20 and more than 60 yrs, history of Chronic Glomerulonephritis, Nephrotic syndrome or advanced CKD, proterinuria or haematuria. All blood samples were collected in fasting state and Serum T₃, T₄, and TSH along with Blood Urea and Serum Creatinine were estimated on a fully automated machine 5600 of orthodiagnostics. Serum Sodium was determined on Electrolyte analyzer. Blood urea and serum creatinine values were slightly high in Subclinical hypothyroid females as compared to Subclinical hypothyroid males and much higher when compared with normal males. On the contrary, Sr. Sodium levels in Subclinical hypothyroid males and females were found to be lower than normal males and females. Serum Sodium levels were found to be slightly low in females with Subclinical hypothyroidism as compared to subclinical hypothyroid males.

Keywords: Subclinical hypothyroidism, chronic kidney disease

INTRODUCTION:

Subclinical hypothyroidism, defined as elevated serum TSH but normal free thyroxine, is common in older adults; researchers have found that the prevalence of Subclinical hypothyroidism was 15% in community-dwelling older adults [1]. Recently, Gopinath *et al.*; found that increased Sr. TSH was associated with increased prevalence of chronic kidney disease among older adults, independent of age, diabetes and hypertension [2]. Conversely, Subclinical hypothyroidism is frequently observed in Chronic Kidney Disease patients [3]. Chronic Kidney Disease is usually a progressive irreversible condition that is the 8th leading cause of death in United States [4]. Risk factors for Chronic Kidney Disease include Diabetes, Hypertension, hyperlipidaemia and thyroid disorders. T3 and T4 play a critical role in cell differentiation during development and help maintain metabolic homeostasis in the adults [5]. Patients with hypothyroidism, both overt and subclinical are characterized by a decreased GFR and renal plasma flow resulting in increased Sr. Creatinine [6]. Some clinical studies have shown that after L-Thyroxine

treatment for hypothyroidism Sr. Creatinine is rapidly restored to normal levels [7]. Spin *et al.*; reported that thyroid hormone replacement therapy preserved renal function in Chronic Kidney Disease patients with Subclinical hypothyroidism [8]. Data regarding this is scarce, so we focused on the clinically relevant interactions between thyroid function and renal function.

MATERIALS AND METHODS:

A hospital based cross sectional study was conducted on patients attending the Out Patients Department of Medicine, Surgery and Gynecology of SGRRIM&HS Dehradun (U.K.), during a period of 8months from Jan 2016 to Aug 2016. 114 Cases (28M+86F) and 90 Controls (40M+50F) in the age group 20-60 yrs were selected randomly. Exclusion criteria was Chronic Glomerulonephritis, Nephrotic Syndrome or advanced CKD, proteinuria or haematuria and age less than 20 yrs and more than 60 yrs. All the subjects both cases and controls underwent a biochemical analysis for Sr. T3, Sr. T4, Sr. TSH, Sr. Urea, Sr. Creatinine and Sr. Sodium. Samples were

collected in fasting state and Sr. T3, Sr. T4 and Sr. TSH were estimated using ELISA Competitive immunoassay method as described by Sterling L [9] and Sr. Urea and Sr. Creatinine were estimated by enzymatic method on a fully automated machine 5600 of Orthodiagnositics. Sr. Sodium was determined on an Electrolyte Analyzer.

RESULTS:

A total No. of 114 cases (28M & 86F) and 90 controls (40M & 50F) in the age group 20-60 yrs were included in the study and assayed for Sr. T3, T4, TSH, Urea, Creatinine and Sodium. Blood Urea levels were found to be high in both Subclinical hypothyroid males ($59.1 \pm 15.23 \pm 2.93$ mg/dl) and females ($62.85 \pm 11.95 \pm 1.29$ mg/dl) as compared to normal males ($35.8 \pm 5.67 \pm 1.56$ mg/dl) and normal females ($32.4 \pm 2.86 \pm 1.45$ mg/dl) respectively. Blood urea values were slightly high in Subclinical hypothyroid females as

compared to Subclinical hypothyroid males. Similarly, Sr. Creatinine was found to be slightly high in Subclinical hypothyroid males ($2.5 \pm 0.50 \pm 0.10$ mg/dl) than Subclinical hypothyroid females ($2.08 \pm 0.79 \pm 0.09$ mg/dl) and much higher when compared with normal males ($0.91 \pm 0.13 \pm 0.10$ mg/dl) and normal females ($0.75 \pm 1.45 \pm 0.08$ mg/dl). On the contrary, Sr. Sodium levels in Subclinical hypothyroid women ($132.42 \pm 3.40 \pm 0.37$ mg/dl) were found to be lower than normal women ($142.4 \pm 2.34 \pm 0.28$ mg/dl). Similarly Sr. Sodium levels in Subclinical hypothyroid males were also ($134.5 \pm 4.48 \pm 0.86$ mg/dl) was also found to be lower than normal males ($140.2 \pm 2.41 \pm 0.65$ mg/dl). Sr. Sodium levels were found to be slightly low in females with Subclinical hypothyroid as compared to subclinical hypothyroid males. These observations are tabulated in Table (i) and (ii) and depicted graphically in fig (i) and (ii).

Table 1:

Parameter	Normal Males (40)	Males with SCH (28)
TSH	$3.2 \pm 1.86 \pm 0.65$	$6.2 \pm 2.94 \pm 0.52$
T3	$5.30 \pm 0.85 \pm 0.21$	$5.14 \pm 1.0 \pm 0.2$
T4	$12.96 \pm 2.40 \pm 0.6$	$13.69 \pm 4.29 \pm 0.8$
Urea	$35.8 \pm 5.67 \pm 1.56$	$59.1 \pm 15.23 \pm 2.93$
Creatinine	$0.91 \pm 0.13 \pm 0.10$	$2.5 \pm 0.50 \pm 0.10$
Serum Sodium	$140.2 \pm 2.41 \pm 0.65$	$134.5 \pm 4.48 \pm 0.86$

Table 2:

Parameter	Normal females (50)	females with SCH (86)
TSH	$3.8 \pm 1.2 \pm 0.43$	$6.64 \pm 3.76 \pm 0.41$
T3	$4.8 \pm 0.51 \pm 0.08$	$4.98 \pm 1.46 \pm 0.16$
T4	$13.24 \pm 3.36 \pm 0.56$	$11.77 \pm 3.51 \pm 0.4$
Urea	$32.4 \pm 2.86 \pm 1.45$	$62.85 \pm 11.95 \pm 1.29$
Creatinine	$0.75 \pm 1.45 \pm 0.08$	$2.08 \pm 0.79 \pm 0.09$
Serum Sodium	$142.4 \pm 2.34 \pm 0.28$	$132.42 \pm 3.40 \pm 0.37$

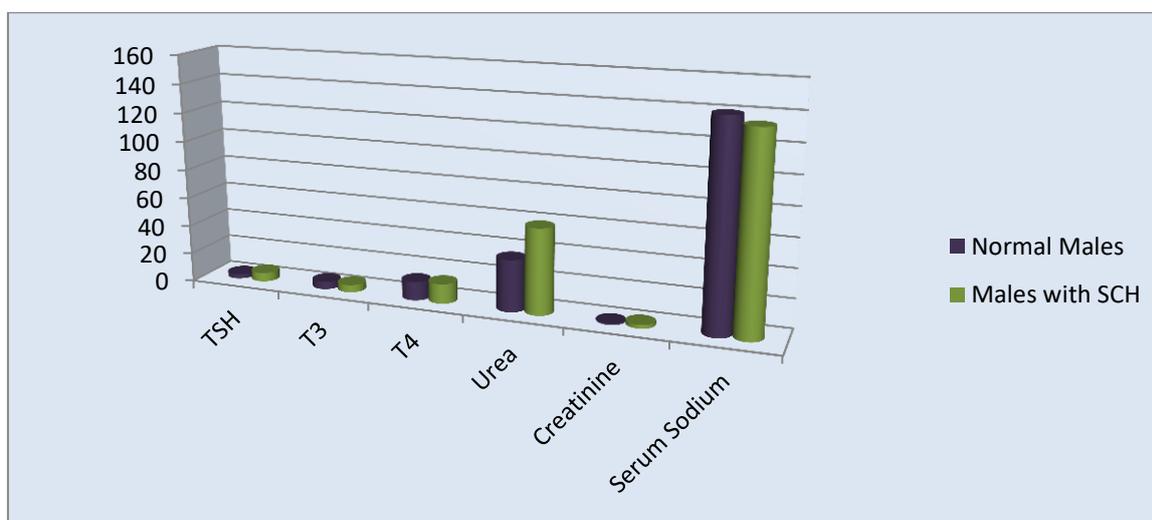


Fig 1:

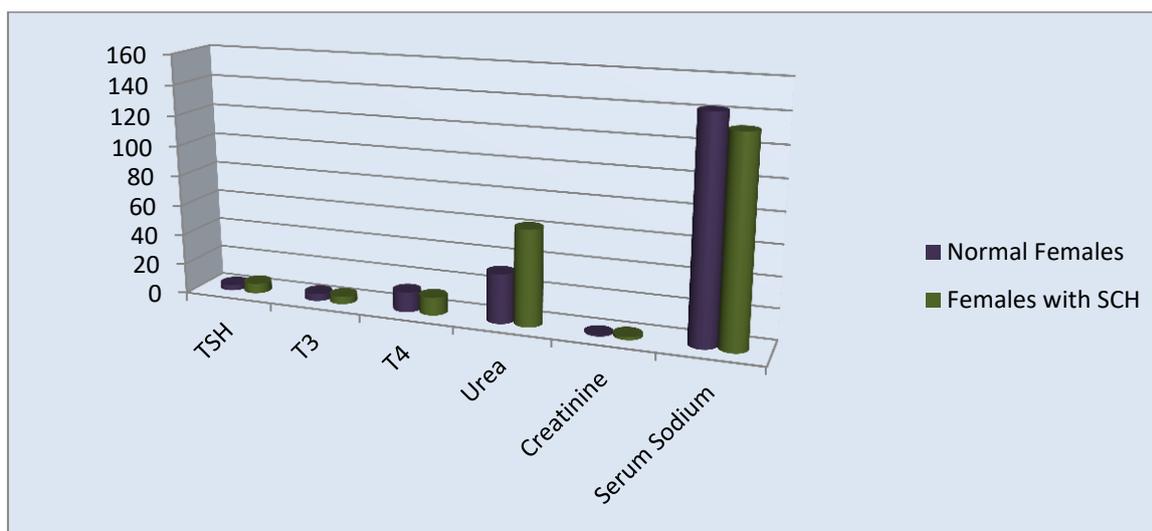


Fig 2:

DISCUSSION:

Thyroid hormones influence the function of all body organs and cells. The data presented here indicates that the biochemical markers are affected by alterations in the levels of thyroid hormones in the body. Thyroid dysfunction affects renal physiology and development where as kidney disease could result in thyroid dysfunction. Disorders of thyroid and kidney may coexist. Prevalence of subclinical hypothyroidism increases consistently with decrease in GFR [10]. Hypothyroidism results in reversible elevation in Sr. Creatinine due to reduction in GFR as well as possible myopathy.

Hypothyroidism also results in increased glomerular capillary permeability to proteins [11], thus the consequent proteinuria precedes resolution in GFR [12]. Thyroid hormones influence sodium reabsorption at Proximal Convolved Tubule primarily by increasing activity of Na/K ATPase and tubular potassium permeability [14]. Thyroid hormones also regulate the adrenergic receptors and dopaminergic activation of the renal tubular cells [15]. They have been shown to affect the rennin-angiotensin-aldosterone axis by adrenergic regulation [16], rennin release [17] and angiotensinase activity [19].

CONCLUSION:

We conclude that thyroid function and kidney disease coexist and increased TSH levels may be considered a risk factor for Chronic Kidney Disease progression in elderly patients.

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