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Serum Thyroid Hormones and Cortisol in Patients with Chronic Renal Failure

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ABSTRACT:

Background Thyroid hormones are necessary for growth and development of the kidney and for the maintenance of water and electrolyte homeostasis. The effects of hypothyroidism on the kidney are usually opposite to the effects of hyperthyroidism.

Patients and Methods: Serum samples were collected from 120 individuals, 50 of these individuals were normal and 70 chronic renal failure (CRF) patients that aged from (18-60) year with no personal history of Hypertensive, Thyroid diseases or Diabetes Mellitus. Serum samples were collected from renal failure patients that admitted to dialysis unit of Tikrit Teaching Hospital from August 2012 to January 2013.

The Results :Blood urea, S.Creatinine, S.TSH, S.Cortisol, Potassium and Chloride were increased in CRF, while T3, T4 and Sodium were decreased rather than control.

Conclusions: These findings suggest the presence of intrathyroidal and pituitary disturbances associated with uremia.

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Introduction

The kidney normally plays an important role in the metabolism, degradation, and excretion of several thyroid hormones. It is not surprising therefore that impairment in kidney function leads to disturbed thyroid physiology. All levels of the hypothalamic-pituitary-thyroid axis may be involved, including alterations in hormone production, distribution, and excretion. The kidney normally contributes to the clearance of iodide, primarily by glomerular filtration. The kidney normally contributes to the excretion of cortisol and its water soluble metabolites (1). As a result, the serum half-life of cortisol becomes prolonged in advanced renal failure. Hyperthyroidism results in increased renal blood flow (RBF) and glomerular filtration rate (GFR) (2). The effect of thyroid hormones on RBF and GFR occurs at multiple levels. The effects of hypothyroidism on the kidney are usually opposite to the effects of hyperthyroidism. Both normal and elevated levels of serum cortisol have been reported in this setting (3). The kidney is the primary organ responsible for potassium excretion, when potassium builds up in the bloodstream, it is called hyperkalemia. Hyperkalemia can cause abdominal cramping, tiredness, muscle weakness or paralysis. Severe hyperkalemia will slow down the cardiac impulses and can lead to cardiac arrest. Sodium plays a major role in fluid balance, neuromuscular function and acid-base balance. The kidneys either conserve or excrete sodium depending on the body's needs, if the kidneys are not able to excrete sodium, hypernatremia will occur. Hypernatremia can lead to disorientation, muscle twitching, increased blood pressure and weakness (4). The aim of this study is to evaluate serum Thyroid hormones, Cortisol, Blood urea, S.creatinine and S. electrolyte in patients with CRF.

Materials and Methods

Serum samples were collected from renal failure patients that admitted to dialysis unit of Tikrit Teaching Hospital from August 2012 to January 2013. Several investigations were measured for patients and controls like blood urea (by enzymatic method/ randox), s. creatinine (by kinetic method / biolabo), sodium, potassium, chloride (by electrolyte analyzer), T3, T4, TSH and cortisol (by Minividus). Serum samples were collected from 120 individuals, 50 of these individuals were normal and 70 renal failure patients that aged from (18-60) year with no personal history of hypertensive, thyroid diseases or diabetes mellitus.

Results

The Mean ±SD of blood urea for control and CRF patients were (32.2±4.0) and (173.32±56.37) respectively with highly significant (p≤ 0.05) difference between them. S.creatinine for control and CRF patients were (0.7±0.2) and (8.42±4.15) respectively with significantly increased (p≤ 0.05). T3 (0.81±0.47) and T4 (67.26±31.26) were significantly decreased in CRF patients compared with control (1.64 ±0.42) and (88.50±14.7) respectively, while S.TSH for CRF patients (8.0±16.27) was significantly increased rather than control (1.80±0.78). S. cortisol was increased in CRF patients (215.49±171.16) compared with control (171.2±60.4). S. electrolytes were disturbance when sodium (133.32±6.84) highly significant decreased (p≤ 0.01) rather than control (171.2±60.4), while potassium (4.56±0.93) and chloride (104.57±6.08) were slightly increased (p≤ 0.05) in CRF, compared with control (4.2±0.82) and (99.8±4.82) respectively (as shown in table 1 and fig. 1).

Statistical Analysis: The present results are analyzed by the following statistical method which includes: Statistical descriptive tables, relative frequencies (percent), arithmetic mean and standard deviation (SD). The suitable statistical tests are used as follows: T-test, Qi-square test.

Table (1):- Several biochemical tests in control and CRF patients

Tests	Control	CRF patients
Number of subject	50	70
T3 ng/ml	1.64 ±0.42	0.81±0.47
T4 µg/dl	88.50±14.70	67.26±31.26
TSH µIU/ml	1.80±0.78	8.0±16.27
Cortisol ng/ml	171.2±60.4	215.49±171.16
Sodium mmol/L	145.4±8.23	133.32±6.84
Potassium mmol/L	4.2±0.82	4.56±0.93
Chloride mmol/L	99.8±4.82	104.57±6.08
Blood Urea mg/dl	32.2±4.0	173.32±56.37
Creatinine mg/dl	0.7±0.2	8.42±4.15

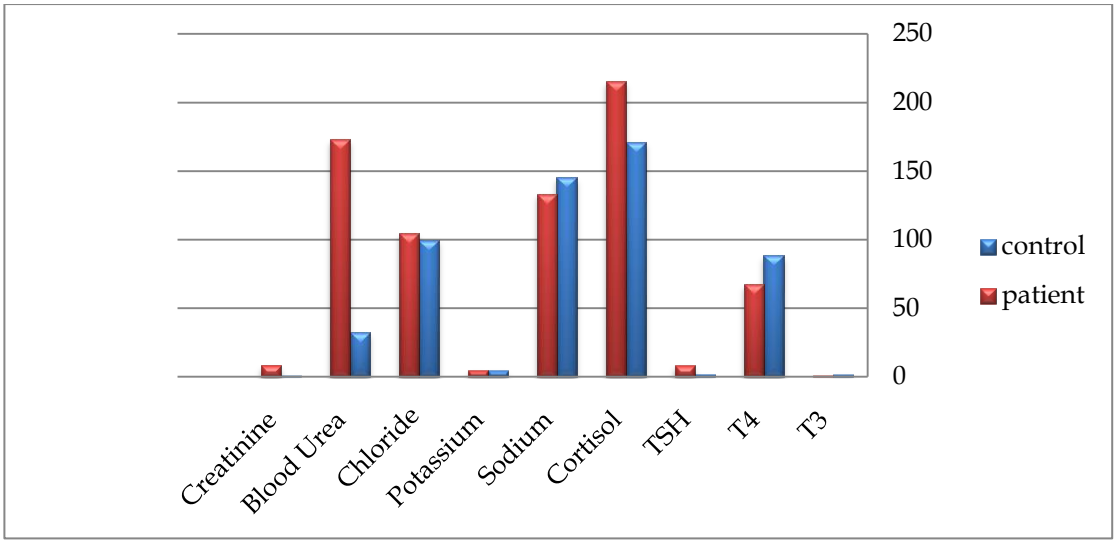

Fig. 1:- Several biochemical tests in control and CRF patients

Discussion:

Most patients with end-stage renal disease have decreased plasma levels of free triiodothyronine (T3), which reflect diminished conversion of thyroxine T4 to T3 in the periphery. This abnormality is not associated with increased conversion of T4 to the metabolically inactive reverse T3 (rT3), since plasma rT3 levels are typically normal [(1)]. This finding differentiates the uremic patient from patients with chronic illness in which the conversion of T4 to T3 is similarly reduced, but the generation of rT3 from T4 is enhanced. In addition to decreased production, low levels of total T3 also may reflect reduced protein binding [(2)]. Thus, iodide excretion is diminished in advanced renal failure, leading sequentially to an elevated plasma inorganic iodide concentration and an initial increment in thyroidal iodide uptake. The ensuing marked increase in the intra-thyroidal iodide pool results in diminished uptake of radiolabeled iodide by the thyroid in uremic patients [(5)]. Increases in total body inorganic iodide can potentially block thyroid hormone production (the Wolff-Chaikoff effect). Such a change may explain the slightly higher frequency of goiter and hypothyroidism in patients with chronic kidney disease [(6, 7)]. Chronic kidney disease has symptoms of subclinical hypothyroidism, and a reduction in the degree of thyroid hormone concentrations depends on intensification of kidney disease [(8, 9)]. Serum TSH concentrations are usually normal or elevated in CKD, but its response to its releasing hormone (TRH) is generally low [(10-14)]. These findings suggest the presence of intrathyroidal and

pituitary disturbances associated with uremia [13]. Also, both TSH circadian rhythm and TSH glycosylation are altered in CKD. The latter may compromise TSH bioactivity. The diagnosis of abnormal glucocorticoid metabolism can be challenging in the chronic renal failure patient. The kidney normally contributes to the excretion of cortisol and its water soluble metabolites. As a result, the serum half-life of cortisol becomes prolonged in advanced renal failure [15]. Both normal and elevated levels of serum cortisol have been reported in this setting [16, 17]. A decreased sodium concentration (hyponatremia) usually indicates that the extracellular fluid is being diluted. Hyponatremia may result from either excess of water in the system, or from the loss of sodium, chloride ions also enter the cells as countering to sodium. Because the intracellular concentration of potassium is much higher than in the ECF and plasma, a relatively minor movement of potassium between ECF and ICF may result in large changes in the serum potassium concentration, changes in serum potassium concentration are also associated with the acid-base disorders [18].

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