I. Introduction

The thyroid is a small butterfly-shaped gland in the front of the neck below the larynx, or voice box. The thyroid gland makes two thyroid hormones, triiodothyronine (T₃) and, in much greater quantities, thyroxine (T₄). Thyroid hormones affect metabolism, brain development, breathing, heart and nervous system functions, blood cell formation, body temperature, muscle strength, bone health, skin dryness, menstrual cycles, weight, and cholesterol levels. The classically described cellular actions of thyroid hormones are mediated by nuclear thyroid hormone receptors that function to regulate the expression of specific cardiac genes such as plasma membrane sodium potassium ATPase and voltage-activated K₁ channel genes including Kv4.2, Kv4.3, and Kv1.5¹.

Sakaguchi and co-workers showed that T₃ caused a shortening of the action potential duration in guinea pig ventricular myocytes by increasing whole cell inward rectifier potassium current (I_K₁). Single I_K₁ channel recordings showed that T₃ increased the open probability mainly resulting from shortening of interburst duration without any changes in burst behavior. Neither the number of channels nor unit amplitude of single I_K₁ was changed by T₃. Sodium channel activity has also been shown to be regulated by T₃. Potential molecular mechanisms by which T₃ may elicit the observed rapid effects on ion flux may involve direct binding of T₃ to cardiac potassium channel gating activity of sinoatrial node cells and atrial muscle cells and altered showed an increased rate of diastolic depolarization². Thyroid hormones increase Na⁺-K⁺-ATPase activity in skeletal muscle. Thyroid hormones in the physiological range are the major endocrine factors controlling the content of Na⁺-K⁺ pumps in skeletal muscle.
Their marked stimulating effect on the synthesis of Na+-K+ pumps seems to be driven by an early increase in the passive leaks to Na+ and K+. This is important for whole body Na+-K+ homeostasis and contractile performance. Moreover, the decrease in Na+-K+ pump content in fasting or diabetes may be due to reduced thyroid status. Thyroid hormone production is regulated by another hormone called thyroid-stimulating hormone (TSH). TSH is made by the pituitary gland, a pea-sized gland located in the brain.

**Aims and objectives:**
- To study the role of thyroid hormones on serum electrolytes viz. potassium, sodium
- To study the serum electrolytes status in hyperthyroidism and hypothyroidism

**II. Material And Methods**

This study was carried out in the Department of Biochemistry, Shri B M Patil Medical College and Hospital, Bijapur. The study included 75 subjects with their informed consent which consists of control group of 25 normal, healthy individuals with no history of any thyroid disorder. Case group consists of 25 individuals with diagnosed hypothyroidism and 25 individuals diagnosed of hyperthyroidism. The following biochemical parameters will be estimated in both the groups.
1. Triiodothyronine (T3)
2. Thyroxine (T4)
3. TSH
4. Serum potassium
5. Serum sodium

**Statistical analysis**

The statistical analysis was performed using SPSS software 11 version. The descriptive results are expressed as mean and standard deviation. Significant difference between the patient and control groups observed is assessed by using the student t test. The p values are expressed along with mean values and standard deviation. The p values less than 0.05 were considered statistically significant. Collection of blood sample for analysis:

5ml of fasting venous blood sample was drawn from patients, into a sterile disposable syringe which was transferred into plain tubes. The sample is centrifuged at 3000 rotations per minute for 10 minutes and serum was collected. The serum was processed within one hour of collection.

**III. Results**

The present study was undertaken in the Department of Biochemistry, Shri B M Patil Medical College, Bijapur. The study includes 75 subjects with their informed consent which consists of 25 hypothyroid patients, 25 hyperthyroid patients and 25 age and sex matched healthy individuals were taken as controls. The study included the female patients aged between 25 to 50 years.

The biochemical parameter, serum sodium was measured to know the electrolyte status in patients and controls. The mean value of the parameter in both the groups are shown in table I and II, which do not show a significant difference between the patient and control group. The levels of serum sodium were within normal range in hypothyroid patients indicating no statistically significant difference between the patient group and control group whereas the p value for serum potassium in hyperthyroid patients and the control group was statistically significant (<0.001)

**IV. Discussion**

Thyroid dysfunction is common in adults and frequently has significant clinical consequences. Hypothyroidism and hyperthyroidism can be accurately diagnosed with laboratory tests.

In this study which includes 25 hypothyroid patients, 25 hyperthyroid patients and 25 age and sex matched controls, serum sodium and potassium were estimated to assess the electrolyte status in these patients as compared to that of controls.

The levels of serum sodium and serum potassium were within normal range in hypothyroid patients indicating no statistically significant difference between the patient group and control group. The levels of serum sodium was within normal range in hyperthyroid patients as that of control group. Hence there was no statistically significant difference between the patient and control group.
The levels serum potassium in hyperthyroid patients, there was significant decrease as compared to that of control group. There are various studies conducted which show decrease in serum potassium levels in hyperthyroid patients, hence hypokalemia.

Studies show that potassium becomes very deficient in the hyperthyroid state. It can become so deficient that hypokalemic paralysis results. This is a condition in which the whole body becomes rigid because of potassium deficiency. Potassium, sodium, and lithium are alkaline minerals which are involved in the cellular pumps which regulate the transport of water and nutrients through the cell walls.4

Our study showing decreased serum potassium levels is in accordance with study by Edmonds CJ which shows the patients with hyperthyroidism, the greatest muscular weakness was present in those with the greatest body potassium loss. Total body potassium changes were closely related to total plasma tri-iodothyronine concentrations but unrelated to the thyroxine levels5.

A study by Shishiba Y et al shows that potassium deficiency may sensitize NaK-ATPase to the effect of thyroid hormone showing that potassium deficiency alters the physiology of thyroid hormone activity6.

This study indicates that hyperthyroidism might lead to hypokalemia hence leading to muscular weakness and other complications. Thyrotoxic hypokalemic periodic paralysis is characterized by recurrent episodes of motor weakness of variable intensity associated with thyroid overactivity. It is usually associated with low plasma potassium levels and is often precipitated by physical activity or ingestion of carbohydrates.

Thyrotoxic periodic paralysis should be considered in the differential diagnosis of all acute episodes of motor paralysis in young patients. Determination of the plasma levels of potassium and thyroid hormones helps diagnosis. Early diagnosis is important so as to be able to establish antithyroid treatment and avoid further episodes of weakness.

References

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