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Research Article

Study the Level of Certain Biochemical Parameters in Iraqi Patients with Thyroid Disorder

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ABSTRACT

Aim: This study aimed to evaluate changes in serum levels of some elements (phosphorus and chloride ions) and relationship with hypothyroidism, hyperthyroidism; and euthyroidism as a control groups. Method: Analysis of serum phosphorus and chloride were done by enzymatic methods using AGAPPE kit. Result: Patients with hyperthyroidism and hypothyroidism showed highly significant increase in serum phosphorus levels ($p<0.001$). In case of hypo- and hyperthyroidism significant difference could not be obtained among euthyroidism and patients ($p>0.05$) in chloride level. While a positive correlation with thyroid stimulating hormone (TSH), serum phosphorous and chloride in case of hypothyroidism. In hyperthyroid patients, the correlation was negative for phosphorous and positive for chloride, also none of correlations were statistically significant ($p>0.05$). Conclusion: Thyroid patients should be regularly checked for serum electrolytes. Early discovery and management can prevent the further complications and will be useful during the controlling of thyroid patients.

Keywords: hypothyroidism, hyperthyroidism, euthyroidism, serum phosphorus, serum chloride.

INTRODUCTION.

Thyroid hormones (TH) are essential moderate forms of electrolyte disorders which may not cause any symptoms. Such disorders can go undetected until they are discovered during a routine blood test. Symptoms usually start to appear once a particular disorder becomes more severe. Thyroid hormone differently from most hormones which commonly are specialized molecules able to influence on other cells, tissues and systems'. Dysfunction of thyroid which prevalent in the population affects in many organs including male and female gonads and interferes with human reproductive physiology and metabolic functions such as regulation of lipid, carbohydrate, protein, and mineral metabolisms of water and electrolytes[1]. Clinical abnormalities in thyroid function are estimated to affect >5% of individuals during their life time[2]. Early diagnosis and management of thyroid disease are crucial, since it is associated with increased morbidity and mortality, especially in the elderly. Electrolytes ionized molecules found through the blood, tissue and all of the body and important for body presses, acid –base balance, blood clotting and muscle contraction[3,4]. One of the electrolytes was inorganic phosphorus which critical for numerous normal physiologic functions including skeletal development, mineral metabolism, energy transfer through mitochondrial metabolism, cell membrane phospholipid content and function, cell signaling, and even platelet aggregation. Total adult body stores of phosphorus is approximately 700 g, of which 85% is contained in bone in the form of hydroxyapatite $[(Ca)_{10}(PO4)_6(OH)_2]$ of the remaining, 14% is intracellular, and only 1% is extracellular. Of this extracellular phosphorus, 70% is organic and contained within phospholipids, and 30% is inorganic, 15% is protein bound, and the remaining 85% is either complexed with sodium, magnesium, or calcium or circulates as the free monohydrogen or dihydrogen forms. It is this latter 0.15% of total body phosphorus (15% of extracellular phosphorus) that is freely circulating a measured[5]. Thyroid hormones effects on osteoblasts from nuclear receptors to stimulate osteoclastic bone resorption[6]. Hyperthyroidism has been reported as one of the major causes of secondary osteoporosis[7]. Thyroid hormones generally stimulate bone resorption directly by increasing ionized serum calcium and phosphorus levels and suppressing parathyroid hormone (PTH). Thyroid hormones are essential for normal growth and maturation of skeletal system and dysfunction of thyroid hormone was associated with disturbances of phosphorous homeostasis[8].

Chloride ion other electrolyte necessary for maintaining the proper balance of body fluids. Hyperchloremia occurs when there is too much chloride in the body. While Hypochloremia develops when there is too little chloride in the body[9].

Patients and Methods

Sera of samples were collected from patient and control groups from those conducted in the department of diagnostic and research unit for hormones analysis and evaluation at Al-Kadhimiya teaching hospital Baghdad City on Iraq. The study involved patient suffering from

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Table 1: Distribution of studied groups according to age/year.

			Studied group			Total	Chi-Square Tests (P-value)
			Hyperthyroidism	Hypothyroidism	Euthyroidism		
Age/year	20-29	Count	8	10	6	24	P =0.211
		% of Total	5.0%	6.2%	3.8%	15.0%	No sign.
	30-39	Count	12	4	6	22	(P>0.05)
		% of Total	7.5%	2.5%	3.8%	13.8%	
	40-49	Count	8	18	8	34	
		% of Total	5.0%	11.2%	5.0%	21.2%	
	50-70	Count	32	28	20	80	
		% of Total	20.0%	17.5%	12.5%	50.0%	
Total		Count	60	60	40	160	
		% of Total	37.5%	37.5%	25.0%	100.0%	

Table 2 :Distribution of studied group according to gender.

			Studied .group			Total	Chi-Square Tests (P-value)
			Hyperthyroidism	Hypothyroidism	Euthyroidism		
gender	male	Count	8	12	6	26	P =0.594
		% of Total	5.0%	7.5%	3.8%	16.2%	No sign.
	female	Count	52	48	34	134	(P>0.05)
		% of Total	32.5%	30.0%	21.2%	83.8%	
Total		Count	60	60	40	160	
		% of Total	37.5%	37.5%	25.0%	100.0%	

Table 3: comparison of biochemical parameters between euthyroidism and hypothyroidism (mean± SD)

Test	Euthyroidism	Hypothyroidism	P value Sig.(2-tailed)
T3 (tri-iodothyronine) nmol/L	1.73±0.39	1.34±0.62	P= .000 Highly sign. (P<0.001)
T4(thyrxine) nmol/L	85.39±13.68	66.45±28.74	P= .001 Highly sign. (P<0.001)
TSH (thyroid stimulating hormone) µ/ml	2.13± 1.19	18.86± 19.44	P= .000 Highly sign. (P<0.001)
P (phosphorous) mg/dl	3.13±0.54	5.15±1.22	P= .000 Highly sign. (P<0.001
Cl (chloride) mEq/L	82.40±16.15	8.70± 17.19	P=.093 No sign. (P>0.05)

thyroid disorder, their age range of (20 – 70) years which divided into three groups: hypothyroidism (60), hyperthyroidism (60) and euthyroidism as controls group (40). 5ml of venous blood samples was drawing. The blood in plan tube was left for short time to allow blood clotted. Then clear serum sample was obtained by centrifugation at 3000 rpm for 5 min. Biochemical parameters including phosphorus and chloride ion were determined by enzymatic methods using AGAPPE kit while T3,T4 and TSH were determined by using enzyme linked fluorescent assay method (ELFA) by Mini Vidas instrument. Exclusion criteria of participation in this study were: pregnancy, lactation, renal failure, and patients using drugs affected the study. All patients were selected randomly, evaluated and selected by detailed medical history, physical and systemic examination. All statistical analysis was carried out by Pentium-4 computer using a statistical package program (SPSS 20).

RESULTS

The distribution of studied group according to age groups is listed in table (1). It was observed that all the age groups give no significant differences **(**P>0.05). In spite the largest group in age (40-49) years and (50-70) years. The data in table (2) showed no significant differences **(**P>0.05) among studied groups according to gender. In spite of predominance the percentage of female patients 134(83.3%) compared with male patients 26(16.2%).

Table 4: comparison of biochemical parameters between euthyroidism and hyperthyroidism (mean± SD)

Tests	Euthyroidism	Hyperthyroidism	P value Sig.(2-tailed)
T3 (tri-iodothyronine) nmol/L	1.73±0.39	2.37±1.41	P=.010 sign. (P<0.05)
T4(thyrxine) nmol/L	85.39±13.68	115.96±38.29	P= .000 Highly sign. (P<0.001)
TSH (thyroid stimulating hormone) µ/ml	2.13± 1.19	1.47±2.55	P= .047 sign. P<0.05)
P (phosphorous) mg/dl	3.13±0.54	5.15±1.22	P= .000 Highly sign. (P<0.001)
Cl (chloride) mEq/L	82.40±16.15	89.76±19.86	P= .064 No sign. (P>0.05)

Table 5 : Pearson's correlation coefficient (r) between various parameters and TSH.

TSH	Correlation coefficient (hypothyroidism)	Correlation coefficient (hyperthyroidism)	Correlation coefficient (euthyroidism)
P (phosphorous)	.050	-.031	-.005
Cl (chloride)	.108	.229	.092

Data revealed in table (3) clearly showed that mean concentration of T 3 and T4 in hypothyroidism decreased (1.34 ± 0.62) nmol/L and (66.45± 28.29) nmol/L respectively with a highly significant difference (P < 0.001) in compared with euthyroidism. Otherwise the mean concentration of TSH in case of hypothyroidism increased (18.86 + 19.44) µ/ml with a highly significant difference (P < 0.001) in compared with euthyroidism. Also highly significant increased (P < 0.001) in the mean concentration of phosphorus ion in hypothyroidism (5.15± 1.22) mg/dl in comparison with euthyroidism (3.13 ± 0.54)mg/dl , also there is no significant differences (p>0.05) between hypothyroidism and euthyroidism in mean concentration of chloride ion

The data in table (4) showed that the mean concentration of T 3 and T4 in hyperthyroidism were elevated (2.37 ± 1.41 nmol/L) and (115.96± 38.29 nmol/L) respectively with a highly significant difference (P < 0.001) in comparison with euthyroidism. Otherwise the mean concentration of TSH in hyperthyroidism was decreased (1.47± 2.55) µ/ml with a highly significant difference (P < 0.001) in compared with euthyroidism. Also highly significant increased (P < 0.001) in the mean concentration of phosphorus ion in hyperthyroidism (5.15± 1.22)mg/dl mg/dl in comparison with euthyroidism (3.13 ± 0.54)mg/dl ; also there were no significant differences (p>0.05) between hypothyroidism and euthyroidism in mean concentration of chloride ion.

Table (5) showed a positive correlation among TSH, phosphorous and chloride in hypothyroidism, while in hyperthyroidism and euthyroidism the TSH and phosphorous were negatively correlated, otherwise a positive correlation between TSH and chloride.

DISCUSSIONS

In this study 75% of the patients were suffering from hypo- and hyperthyroidism whereas 25.0% had euthyroidism. Hypothyroidism is one of the most prevalent endocrine diseases. It can lead to a variety of clinical situations, including congestive heart failure, electrolyte disturbances and coma[10]. Thyroid hormone is a central regulator of body hemodynamics, thermoregulation and metabolism. Therefore, it has an influence on renal hemodynamics, glomerular filtration and electrolyte handling[11].

In the present study there was highly significant elevated in phosphorous levels in patients with hyper- and hypothyroidism (p<0.001) whereas no such results was obtained for chloride level in hypo- and hyperthyroidism (p>0.05). Our finding was in accordance with Elhashimi *et al*[12]. This agrees with a study done by Suneel *et al*, who reported that there was a significant decrease of the mean phosphate in patients with hypothyroidism compared to control. This is mainly due to calcitonin which is regulates the over tubular reabsorption of phosphate from kidney. Phosphate levels are raised due to compensatory effect of calcitonin and parathyroid hormone which favors tubular excretion (by inhibiting tubular reabsorption)[13]. Hyperphosphatemia in hyperthyroidism has been explained on the basis of an enhanced tissue catabolism leading to an excess input of phosphorous to the plasma pool from bone and tissues and lower fractional clearance of phosphorous and increased renal tubular reabsorption of phosphorous. The changes in serum phosphorous are due to suppressed PTH levels as well as direct effects of thyroid hormones on tissue phosphate metabolism and renal phosphate handling. These effects lead to increased filtered load of phosphorous in patients with hyperthyroidism[14].

The present work confirms the correlation among levels of serum phosphorous and chloride with the TSH. In case of hypothyroidism serum phosphorous and chloride were positively correlated. Whereas in case of hyperthyroidism, a negative correlation between serum phosphorous and TSH were showed. In contrast a positive correlation between serum chloride and TSH were cleared. None of these correlations were statistically significant ($p>0.05$). This result agrees with Murgod *et al*[15]. Other study reported negative correlation of serum phosphorous with TSH. The study of Kavitha *et al* showed significant positive correlation ($p< 0.003$) with TSH in overt hypothyroidism[16].

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