# The effect of hyperthyroidism on renal function

**M.Ziya MOCAN, Berrin ÇETİNARSLAN, Cihangir EREM, Münir TELATAR**

Dept. of Internal Medicine, Medical School of Karadeniz Technical University, Trabzon, TURKEY

*We have studied the effect of hyperthyroidism on renal functions in 35 patients. BUN, plasma creatinine, urinary urea nitrogen, urinary creatinine, BUN/creatinine, urinary urea/plasma urea, urinary cretainine/plasma creatinine ratios, creatine clearance levels were statistically higher in hyperthyroid patients before the treatment and decreased to normal levels following the treatment when the patient were in normothyroid state (p<0.05). In hyperthyroid, patients the mean serum triiodothyronine (T3) levels were 334.71 ±66.8 ng/ml and serum thyroxine (14) levels were 18.2±5.9 \xg/dl and decreased to 143.8±30.4 ng/ml and 7.5±3.2 \\g/dl respectively, following the treatment. These results indicate that renal functions have been affected temporarily in the hyperthyroid state and the functions have reversed after the control of the hyperfunction of the gland. [Turk J Med Res 1995; 13(1): 28-30]*

Key Words: Hyperthyroidism, Kidney, Thyroid hormones

Thyroid hormones (Thyroxine, T4: triiodithyronine T3) have potent influence on renal function. They are known to have a number of systemic, and hemodinamic effects including a nonspesific enhancement of GFR (1). Thyroid hormones protect against nephrotoxic acute renal failure (ARF) (2). Sympathetic activity was also increased in thyrotoxicosis and plasma renin activity (PRA) is elevated related to this (3). Marked increase in cardiac output was also established in hyperthyroidism (4). We have studied the changes of renal function during hyperthyroidism and evaluated these functions after the control of the hyperthyroid status.

## MATERIALS AND METHODS

Thirty five patients (28F, 7M) with hyperthyroidism of Graves disease and 40 euthyroid normal subjects were examined in the present study. The diagnosis of hyperthyroidism was made by clinical findings and laboratory data. The mean serum $T_4$ levels were 18.2±5.9 ug/dl, and $T_3$ levels were 334.7±66.8 ng/ml before treatment. Renal functions were performed during this period and propylthiourasil 300 mg/d has been started for the patients with high sympatomimetic activity. The mean serum $T_4$ levels were decreased to 7.5±3.3 mg/dl and $T_3$ levels decreased to 143.8±30.4 ng/ml in the posttreatment period.

Serum $T_4$ and $T_3$ assays were measured by a radioimmunoassay method using commercial kits supplied by Amersham. Student's t test has been used for statistical analysis. Two way variance analysis and Duncan test has been performed and "p value" <0.05 was considered as statistically significant. Forty euthyroid subjects (25F, 15M) were taken into the study as controls. Their $T_4$ levels were at 6.5±1.3 mg/dl and $T_3$ levels were at 98±5 ng/ml (Table 1).

## RESULTS

As in Table 2, BUN, urinary UN, urinary creatinine levels were significantly higher (p<0.05) in the pretreatment period compared to the posttreatment period and the controls. Serum BUN/creatinine, urinary urea/plasma urea and urinary creatinine/plasma creatinine ratios were also significantly higher in the hyperthyroid state (p<0.05). Endogenous creatinine clearance values were significantly higher in the hyperthyroid state (p<0.05). Fractional sodium excretion was lower in the pretreatment patients (0.74±0.1) compared to the postreatment patients (0.96±0.3). This is also statistically significant (p<0.05).

Received: Dec. 15, 1994 Accepted: Dec. 29, 1994

Correspondence Cihangir EREM
Dept. of Internal Medicine
Medical School of Karadeniz Technical University,
Trabzon, TURKEY

**Table 1**. Patients with hyper and euthyroid status

| | Ages | Hyperthyroid Patients | Euthyroid Controls | Total |
|---|---|---|---|---|
| Female | 20-49 | 16(%57.14) | 16(%64) | 32 |
| | 50-76 | 12 (%42.86) | 9 (%36) | 21 |
| Male | 20-49 | 3 (%42.85) | 8 (%53.33) | 11 |
| | 50-76 | 4(%57.14) | 7 (%46.66) | 11 |
| Total | | 35 | 40 | 75 |

Also animal studies exhibit an impaired tubular reabsorption of sodium in thyroid hormone deficiency (13). Our findings also support these authors by the way that hyperthyroid patients showed decreased fractional Na+ extraction, compared to controls.

Our results indicated that the changes in renal function in hyperthyroid patients are reversible and non-organic in the origin.

**Table 2.** Biochemical profile of the patients with hyperthyroidism and controls

| | Pre Treatment | Post Treatment | Controls |
|---|---|---|---|
| BUN (mg/dl) | 19.28±2.82 | 13.97±2.42 | 14.21+2.63 |
| Serum creatinine (mg/dl) | 0.62+0.05 | 0.8±0.07 | 0.75±0.07 |
| Urinary UN (mg/dl) | 132.5±122.3 | 695±61.3 | 802±54.9 |
| Urinary Creatinine (mg/dl) | 84.6±12.4 | 68.6+9.7 | 74.1±12.7 |
| Serum BUN/Creatinine | 30.90±12.4 | 17.11 ±2.86 | 18.40i3.42 |
| Urinary/Plasma Urea | 69.77±8.93 | 50.85+8.62 | 52.41 ±7.63 |
| Urinary/Plasma Creatinine | 178.82± 14.21 | 85.21±8.19 | 98.71+8.77 |
| Creatinine Clearence (ml/mm) | 144.45+10.99 | 114.59+10.27 | 123.68±9.67 |
| Fractional Sodium Excretion | 0.74±0.10 | 0.96+0.34 | 1.01+0.08 |

## DISCUSSION

Thyroid hormones have a potent influence on renal function (11). Our data clearly indicate that in untreated hyperthyroid patients BUN/serum creatinine ratio increases. Urinary protein was absent in all patients during the study, so no parenchymal disease was considered. Serum creatinine levels decreased and BUN levels increased. These results are consistent with the results of Aizawa et al (4). So, we can accept hyperthyroidism as a pre-renal failure for high BUN/creatinin ratio index (5,6). Protein catabolism is increased, so negative nitrogen balance is the results. Thus BUN levels and urinary BUN are increased in hyperthyroidism (7,8). Cardiac output is increased in hyperthyroidism (9) which is due to the stimulation of cell membrane Ca++-ATPase activity by thyroid hormones in myocardial sarcolemma membrane (10). Thus pre-renal cardiovascular factors were ruled out as the cause of increased BUN/creatinine ratio. Table 3 shows the thyroid hormone levels and the ages of the patients. In the older age group (50-76 years) both in the pretreatment and in the posttreatment patients the thyroid hormone levels were less than the younger group (20-46 years).

The effects of thyroid hormones are largely confined to proximal tubules (2,11) Thyroid-deficient animals exhibit a number of renal functional defects such as decreased renal plasma flow and glomerular filtration rate (GFR) (12). Our findings also support these authors in the way that hyperthyroid patients showed increased GFR.

Table 3. Thyroid hormone levels of the hyperthyroid patients

| Ages | | T3 (range: 86-178 ng/ml) | T4 (range 4.5-11.5 mg/dl) |
|---|---|---|---|
| 20-49 | A | 410.2±72.4 | 19.3+6.1 |
| | B | 130.5±24.3 | 7.5+2.9 |
| 50-76 | A | 283.2±43.1 | 15.02±4.9 |
| | B | 123+26.9 | 7.2±2.8 |

A: Pre treatment
B: Post treatment

Hipertiroidinin böbrek fonksiyonu üzerine etkisi

*Hipertiroidinin böbrek fonksiyonları üzerine olan etkisi 35 hastada incelendi. Hipertiroidili hastalarda tedaviden önce BUN, plazma kreatinin, idrar üre nitrojen, idrar kreatinin, BUN/kreatinin, idrar üre/plazma üre. idrar kreatinin/plazma kreatinin oranları, kreatinin klirens seviyeleri istatistik olarak kontrol grubuna göre daha yüksekti (p<0.05) ve bu değerler tedaviden sonra normotiroid durumda normal seviyelere indi Fraksiyonel sodyum ekskresyonu hipertiroid dönemde istatistiksel olarak daha düşüktü (p<0.05). Hipertiroidili hastalarda ortalama serum triiyodotironin (T3) seviyeleri 334.71 ±66.8 ng/ml ve serum tiroksin (14) seviyeleri 18.2±5.9 \xg/dl idi ve tedaviden sonra bu değerler sırasıyla 143.8±30.4 ng/ml ve 7.5±3.2 vg/dl'ye*

*düştü. Bu sonuçlar böbrek fonksiyonlarının hipertiroid durumdan geçici olarak etkilendiğini ve fonksiyonların tiroid bez hiperfonksiyonu kontrol altına alındıktan sonra geri döndüğünü göstermektedir. (TurkJMedRes 1995; 13(1): 28-30]*

## REFERENCES


1. Bradley SE, Stephan F, Coelho JB et al. The thyroid and the kidney. Kidney Int 1974; 6:346-65.
2. Cronin RE, Brown DM, David M et al. Protection by thyroxine in nephrotoxic acute renal failure. Am J Physiol 1986; 251:408-16.
3. Kleven H, Brofn H, Zavaleta J. Plasma renin activity in hyper and hypothyroidism: Effects of adrenergic blocking agents. J Clin Endocrinol Metab 1972; 34:625-9.
4. Azzawa T, Hiromatsu K, Othsuka H. An elevation of BUN/creatinine ratio in patients with hyperthyroidism. Horm Metabol Res, Stuttgart-New York: Georg Thieme Verlag 1986;18:771-4.
5. Contiguglia SR, Mishell JL, Klein MH. Renal and urinary tract disorders. In: Friedman EH, ed. Problem oriented medical diagnosis. Boston: Little, Brown and Company, 1979:214-26.
6. Morgan DB, Carver ME, Payner RB. Plasma creatinine and urea: Creatinine ratio in patients with raised plasma urea. Brit Med J 1977; 2:929-32.
7. Spaulding SW, Lippes H. Hyperthyroidism: Causes, Clinical Features and Diagnosis. Med Clin North Am 1985; 69:937-50.
8. Loeb JN. Metabolic changes: Vitamin metabolism, renal function body water and electrolytes. In: Werner SC, Ingbar SH, eds. The Thyroid. Hagerstown: Harper and Row Publishers Inc. 1978:705-15.
9. Stead EA Jr. Studies of cardiac output and of blood flow and metabolism of splanhnic area, brain and kidney. Tr Assoc Amer Physicians 1950; 63:241-5.
10. Dillmann WH. Mechanism of Action of Thyroid Hormones Med Clin North Am 69:849-61.
11. Ford RV, Owens JC, Curd GW, Moyer JH, Spur CL. Kidney function in various thyroid states. J Clin Endocr Metab 1961; 21:548-53.
12. Montiel M, Ruiz M, Jimenez E, Morelli M. Anjiotens Converting Enzyme in Hyper- and Hypothyroid Rats. Horm Metabol Res 1987; 19:90-2.
13. Mackovic-Basic M, Salihagic A, Ries N, Sabolic I. Absence of increased electroneutral Na+-H+ exchange in renal cortical brush-border membranes from hyperthyroid rats. Biochem Pharmacol 1988; 37:1699-705.