Thyroid disorders and renal dysfunctions

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Abstract:
Thyroid hormone plays an important role in formation of proteins, metabolic reactions, development and synthesis of hormone. T3 (Triiodothyroine) and T4 (thyroxine) the two important hormones. It effects the kidney structures, renal hemodynamics, development of renal gland, GFR. The functions of nephron and H_2O and sodium homeostasis. Thyroid hormone causes intrinsic renal and prerenal effect which increases the renal blood flow and the glomerular filtration rate. Both hyper as well as hypothyroidism affects the kidney. Hyperthyroidism results into increased GFR and hypothyroidism results into reduced GFR. Disorders caused by thyroid functions can be glomerular injury and alteration of thyroid hormones in kidney diseases. Thyroid functions are affected by chronic kidney diseases in many ways such as lower circulating thyroid circulation and alteration of thyroid hormone metabolism.

Keywords: *Thyroid hormone, Disorder, Hyperthyroidism*

1. Introduction

The thyroid gland functions are most important to the body of human beings as it maintains majority of body physiological activities. The interrelation of thyroid and kidney is known for a long time. Thyroid dysfunctions can affect metabolism development physiology where as kidney diseases good a cause thyroid dysfunction. Thyroid hormone effects almost all the parts of the body but specially kidney. Thyroid gland secrets thyroxin which effects under the control of anterior pituitary hormone thyroid stimulating regulated by hypothalamic thyrotropin releasing hormone. Thyroxin is produced only by thyroid gland where as triiodothyroine be produced by deionization of T4. Treatment for 1 disease caused by thyroid gland in the Kidney may cure to other organs also. Renal functions disorder is visualised to coexist with the specific level of thyroid hormone present.

2. Thyroid hormone influencing renal development

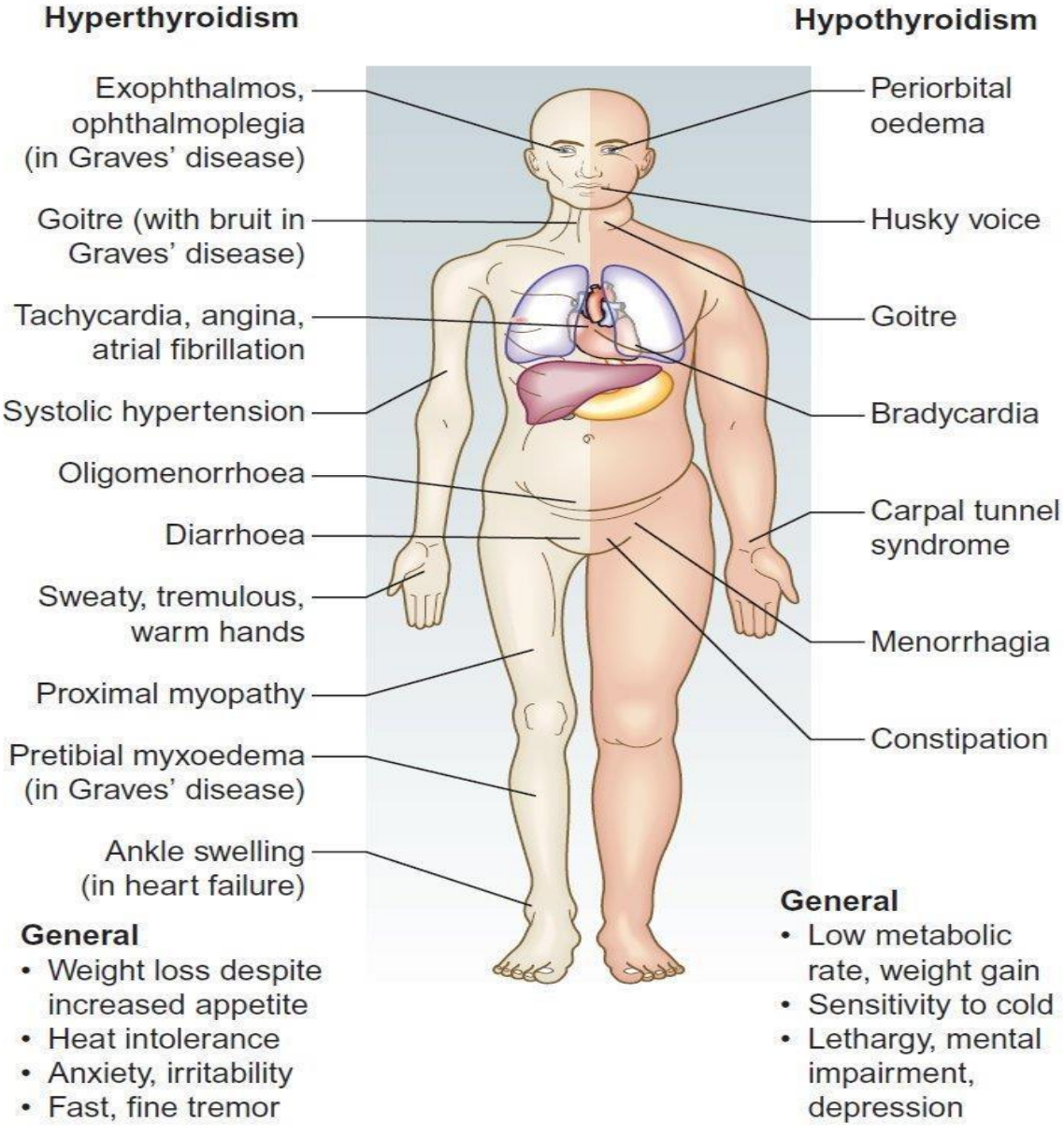
Protein manufacturing and cell growth is been affected by thyroid hormones. Level of thyroid hormone effects the functional renal mass, with hyperthyroidism increasing its ratio and hypothyroidism decreasing its ratio. Hypothyroidism results in the breakdown of proteins and eventual renal atrophy. For instance, children with hyperthyroidism have reduced renal mass and higher prevalence of renal and urological disorders including this plastic kidney, renal agencies, ectopic kidney, posterior urethral valves, and hypospadias. Mutation taking place in gene coding Pax8, a transcription factor necessary for development and function, as seen in some patients, acts as a connection between congenital hypothyroidism and renal dysmorphogenesis. As same direct activation of the renin-angiotension-aldosterone system by thyroid hormone can be independent of influencing on hemodynamics. Kobold et al have bespeak that renal hypertrophy seen in hypothyroid is blocked with losartan but not with nicardipine. The existing group has also defined that the promoter activities of the renin calori – 6 cells is stimulated by thyroid hormone with a thyroid hormone response element dependent mechanism increasing viewing of m r n a encoding renin. Where as some renin-angiotension – aldosterone system component serves directly or indirectly as a thyroid hormone -modified growth factor is not clear.

Thyroid hormone is also necessary in the development of tubular function in both the postnatal as well as prenatal periods. For example, in animals' thyroid hormone effects the maturation activity and the density of the Na+-Pi cotransporter, increases Na+-K+-ATPASE activity and place a crucial role in the ISO form switch from the neonatal Na+-H+ exchanger 8 to adult Na+-H+ exchanger 3.

3. Hyperthyroidism affecting renal function

RBF and GFR are increased by hyperthyroidism. RBF and GFR are affected at multiple levels by thyroid hormone. Along with the prerenal factors, thyroid hormone increases the cardiac output by positive effect on Chrono tropic and I know tropic as well as decreasing in systematic vascular resistance. RBF is indirectly contributed by this. And growing endothelial production of nitric oxide in the renal cortex and medulla by the induction of nitric oxide synthesis directly by the thyroid hormone and affecting indirectly by high arterial pressure associated endothelial sheer stress. This is accompanied by decreasing in renal vasoconstrictor endothelin. So, the net increase of RBF can be seen as in increased intrarenal and decreased vasoconstrictor.

It has been reported that the GFR has increased about 18 to 25% among the hyperthyroidism patients.



Features of hyper- and hypothyroidism.

Source : Macleods Clinical Examination 13th Ed (2013)

Due to increased RPF the GFR is to be increased also. The activity of renin– angiotension- aldosterone system also initiate to increase the GFR. There are multiple methods to stimulate RAAS by thyroid hormones. In this hyperthyroidism, and increased Beta adregenic receptors in the renal cortex out coming as increased stimulation of RAAS. Triiodine increases the gene expression of renin. The enzyme level is converted by serum angiotension plasma renin, angiotension by the increase in thyroid hormones. This increases RAAS activity.

4. Hyperthyroidism influencing renal function

In hyperthyroidism RBF is reduced by decreasing cardiac output and increasing peripheral vascular resistance, reduced renal response to vasodilators, intra renal Vasoconstrictor, and a reduced expression of renal such as growing factor of vascular endothelial and insulin. Glomerular structure shows changes such as Glomerular basement membrane thickening and mistake Matrix expansion, may also contribute to the reduced RBF. About 54 to 56% of adult with hyperthyroidism have reduced GFR. The loss of GFR is been seen by the decreased sensitivity to beta androgenic stimulus and decrease renin release among with decreased angiotension second and impaired RAAS activity. The absorption of sodium chloride and water is reduced by proximal tubule. So the reabsorption of chloride is reduced by the increasing out distil chloride delivery and triggering the macula densa mediated Tubular Glomerular feedback which reduces the RAAS activity.
Consequently, the GFR falls.

5. Thyroid dysfunction and GFR

The increased level of serum creatinine with hyperthyroidism is seen in human in many of the cases. The necessity of knowing the effect of thyroid dysfunctions on a renal function is been focused by recent studies indicating sub clinical and clinical hyperthyroidism common in all the patient with estimated GFR <60 ml/min per 1.73m, asking the question that whether hyperthyroidism contributing low GFR in some of the individuals.The level of serum creatinine excesseding of 6 mg/dl have been acollate to hyperthyroidism, with the patient explaining having ESRD, 1.5-2.5mg/dl is the level of creatinine reported in most of the cases. The promotion of level of serum creatinine can be found within 2 weeks of insignificant hyperthyroidism. These get normalised quickly with thyroid hormone interchanging after short period of hyperthyroidism, but slowly and incomplete recoverment have been noted with more prolonged period of severe hyperthyroidism. The studies have explained a decrease serum creatinine in settling the hyperthyroidism, which is reversible upon the treatment in human and animals. The kidney functions using the creatinine based estimating equations extended the changes which reflect in the true gfr as opposite to the alterations in creatinine metabolism or tubeless equation or to and myopathy has been undefined.

6. Glomerular disease- Thyroid diseases in patients

The cases are reported in animals as well as Children's and adults of the reversible proteinuria and biopsy provin GN associated with both hyper and hyperthyroidism most commonly found in the interrelation to auto immune thyroiditis. Where as the renal histopathology has given membranous nephropathy minimal changes membrane O proliferative GN and nephropathy Iga. There is no direct link between autoimmune thyroid and glomerular disease the process of immune mediation is affected by both comma the reports of thyroid peroxides and thyroglobulin deposited in the Kidney. So far after the therapy of hyperthyroidism the disease of glomerular disease has been described especially anti neutrophil cytoplasmic antibody positive cresentic GN after the therapy with the membraneous nephropathy after first treatment.

7. Thyroid functions influencing patience with kidney diseases

Kidney plays a crucial role in clearing of thyroid stimulating hormone, Iodine thyrotropin releasing hormone. Mostly patients are your thyroid, and the normal TSS and free thyroxin level. The patience affected with Aki and cdk have changed in thyroid function testing consistence with youth thyroid sick syndrome, low thyroxine, T3 and TSH concentration. The patients with renal failure and EU thyroid 6

syndrome have increased rivers T3 level. The decrease level of free T3 is seen in ESRD patients. The patients suffering with cdk and esrd are due to the alterations in the peripheral mono D iodination of thyroxin and reduced level of Plasma protein binded with thyroxine comma the thyroxin binding of Plasma protein and the presence of inhabiters metabolic acid and influencing of medications etc. T4 binding to plasma protein and transiently evaluate free tea for level is inhabited by heparin and furiousamide drugs. It is most commonly seen in the of cdk that thyroid gland enlargement thyroid noodles and thyroid carcinoma is present.

8. Conclusion

The renal development kidney structure GFR enal hemodynamics the function of many transports system along with the Sodium Potassium nephron and water homeostasis is been affected by the thyroid hormone. Kidney functions shows the effect of both hyper and hyperthyroidism by direct renal effect or systematic hemodynamic, cardiovascular effects metabolic rates etc. There for the renal manage recitation of the thyroid disorders are not clinically significant with those of hyperthyroidism and amendable with the treatment. Patience out of hyperthyroidism have important reduction in the GFR which is undetermined elevations in serum creatinine. But not always serum Creatinine and sea levels are accurate indicators of GFR in all the patients the accuracy of serum creatinine is based on the equation in the patience with the hyperthyroidism. Sweet concludes that the patients with nephrotic syndrome as well as having an acute or chronic kidney disease and alterations in the thyroid gland and their structures can have impact in the functions of thyroid status. It has been seen that in hyperthyroidism the transport function is reduced and in hyperthyroidism it is increased or the thyroid replacement with the exception of a AQP has the opposite pattern.

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