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A CASE OF AUTOIMMUNE CKD WITH AUTOIMMUNE HYPOTHYROIDISM (HASHIMOTO'S)

General Medicine

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ABSTRACT

Autoimmune thyroiditis (AIT) is generally associated with hypothyroidism. It affects ~2% of the female population and 0.2% of the male population. The evidence of thyroid function- and thyroid autoantibody-unrelated microproteinuria in almost half of patients with AIT and sometimes heavy proteinuria as in the nephrotic syndrome point to a link of AIT with renal disease. The most common renal diseases observed in AIT are membranous nephropathy, membranoproliferative glomerulonephritis, minimal change disease, IgA nephropathy, focal segmental glomerulosclerosis, antineutrophil cytoplasmic autoantibody (ANCA) vasculitis, and amyloidosis. Here I present a case of a young female who had an autoimmune involvement of thyroid and kidney and responded to treatment.

KEYWORDS

INTRODUCTION:-

Hashimoto's thyroiditis is the leading form of autoimmune thyroiditis (AIT), which is the most prevalent autoimmune disorder and the most common cause of hypothyroidism, excluding iodine insufficiency. It affects ~2% of the female population and 0.2% of the male population. This condition is well known to be associated with other autoimmune diseases, the most common of which are chronic autoimmune gastritis, vitiligo, rheumatoid arthritis, polymyalgia rheumatica, celiac disease, type 1 diabetes, Sjögren's syndrome, systemic lupus erythematosus (SLE), multiple sclerosis, and sarcoidosis. Also glomerular disease may be related to autoimmune disease with several mechanisms. Thyroid hormone influence on kidney is mediated by its effect on the cardiovascular system and, consequently, by its effect on renal blood flow. Hypothyroidism initially decreases peripheral vascular resistance and blood pressure and subsequently activates the renin–angiotensin–aldosterone system, which increases tubular sodium reabsorption.[1]

Case Report:-

This patient was a married female, aged 24 years admitted with chief complaints of-
Generalized weakness for the last 15 days.
Generalized bodyaches for the last 15 days.

The patient was alright 15 days before admission when she started experiencing generalized weakness which progressed over the next few days. Then the patient complained of a lassitude, fatigue. Patient started experiencing generalised bodyaches 15 days before admission and had been present since then.

There was no history of fever, oliguria, polyuria, anuria. There was no history of vomiting. There was no history of altered sensorium, joint pains, any preferential weakness. The past historyshowed no previous history of similar complaints. There was no history of diabetes mellitus, hypertension, coronary artery disease, tuberculosis. Treatment history showed nothing significant. The family history showed no history of diabetes mellitus, hypertension, coronary artery disease, tuberculosis. The personal history showed that the patient was vegetarian, had no addictions, bowel and bladder were normal.

On general physical examination, the patient was conscious, cooperative, sitting comfortably in the bed. Patient was afebrile, the pulse rate was 82/min, regular, other parameters normal. The respiratory rate was 18/min, thoracoabdominal. The BP was 170/90 mmHg, right arm, supine position. Pallor was present, no icterus, cyanosis, clubbing, lymphadenopathy, edema. JVP was not raised. Systemic examination was normal.

On investigation:-

Hb:-7.0 gm%
TLC :- 6250/mm3, Polys – 68%
ANA +ve Bl Urea :- 136 mg%, Sr Creat :- 5.5 mg%
Sr Uric Acid :- 7.9 mg% RBS:- 114 mg%
Sr. Calcium :- 6.9 mg% Sr Na+:- 138 mEq/l
Sr. K+ :- 3.8 mEq/l
ESR :- 90 mm I hr ECG :-normal, CXR: normal
Ft3:- :- 2.80 LFT: normal
Ft4:- 0.86 Urine culture: normal
Sr TSH :- 22.5 Urine Exam: Protein 2+

The patient was started on treatment :-
Tab Dytor, Tab Folic Acid, Tab Kal4Me, Tab alphaD3, Tab Livogen, Cap Becosule, Tab Amlong, Tab Rabicip, Tab Febustat, Tab Thyronorm, Inj Monocef,

Tab Wysolone.
The patient responded to the treatment and the proteinuria, the symptoms of the patient, the RFT, TFT, all improved over time. After discharge from the hospital, the patient has been on regular follow up and has been keeping well.

DISCUSSION:-

Renin gene expression is also regulated by circulating levels of free triiodothyronine (FT3) and free thyroxine (FT4) through beta-adrenergic activation; accordingly, the reduced sensitivity to beta-adrenergic stimulus occurring in hypothyroidism can cooperate with other hemodynamic abnormalities decreasing renin release. The resulting negative inotropic effect on the heart, as well as the altered equilibrium between the reduced expression of vasodilators such as vascular endothelial growth factor or insulin-like growth factor-1, can lead to further renal vasoconstriction. Other consequences of thyroid hormone deficiency include lower secretion of atrial natriuretic factor and erythropoietin, therefore reducing further blood volume. Glomerular filtration rate (GFR) can thus decrease by up to 40%, with subsequent elevation of serum creatinine, both indices being directly proportional to circulating TSH levels (and therefore directly proportional to the extent of thyroid failure) independent of other confounding factors such as age, sex, body mass index, or comorbidities. Thyroid hormone replacement in patients with overt or subclinical hypothyroidism restores renal function.[2]

Kidney can be the victim of autoimmune processes through several mechanisms. Autoantibodies can damage glomeruli either targeting specific antigens as in membranous glomerulonephritis and in anti-glomerular basement membrane (GBM) nephropathy, or being trapped through the filtration barrier as in antineutrophil cytoplasmic autoantibody (ANCA) vasculitis or IgA nephropathy. Pathophysiology of renal impairment in the course of SLE is characterized by both events, because anti-DNA antibodies are located in capillary membranes and mesangial areas of glomeruli and because they cross-react with α-actinin and glycosaminoglycans on mesangial cells. All these immune complexes alter the structure of basement membrane, podocyte function, and activate the classical pathway of complement system, which exacerbate the inflammatory process due to chemotactic factors C3a and C5a. In addition, terminal pathway of complement worsens cell damage because of the cytolytic effect of C5b-9 complex. Finally, immune complexes stimulate infiltration of innate and specific immune cells, such as neutrophils, macrophages, natural killer (NK)

cells, and T lymphocytes, which express receptors for constant fraction (FcR). Natural killer cells have also a role in the pathogenesis of kidney damage as they produce interferon γ (IFNγ) and activate peripheral macrophages first and, then, resident glomerular cells that are responsible of chronic processes.[3]

CONCLUSION:-

Autoimmune process in the body tends to involve multiple organs. One should look for clinical evidence of autoimmune damage to the organs so that it can be treated at an early stage and end stage damage to organs can be prevented.

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