ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 17, Issue, 08, pp.34331-34333, August, 2025
DOI: https://doi.org/10.24941/ijcr.49353.08.2025

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

RESEARCH ARTICLE

COEXISTENCE OF AUTOIMMUNE THYROIDITIS AND IGA NEPHROPATHY IN A YOUNG ADULT MALE: EXPANDING THE SPECTRUM OF THYROID-RELATED RENAL INVOLVEMENT

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ARTICLE INFO

Article History:
Received 11th May, 2025
Received in revised form
24st June, 2025
Accepted 19th July, 2025
Published online 30th August, 2025

Keywords:
Autoimmune thyroiditis, Hashimoto's thyroiditis, IgA nephropathy, hematuria, rare association.

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ABSTRACT

Background*:* Autoimmune thyroiditis (Hashimoto's thyroiditis) is the most common cause of hypothyroidism in iodine-sufficient areas and may coexist with other autoimmune disorders due to shared genetic and immune mechanisms[1,2,3,4]. Renal involvement is uncommon but can include membranous nephropathy, minimal change disease, and rarely IgA nephropathy (IgAN)[5,6,7]. IgAN is the most frequent primary glomerulonephritis worldwide, characterized by mesangial IgA deposition, presenting with hematuria, proteinuria, and variable renal dysfunction[8,9,10]. Its association with autoimmune thyroiditis is rarely reported[11,12,13,14] and may involve shared autoimmune pathways or antigenic cross-reactivity[15]. Awareness of this overlap can aid early diagnosis and management. ***Case Presentation:*** A 26-year-old male presented with progressive shortness of breath and gross hematuria for 15 days. He had a prior diagnosis of autoimmune thyroiditis made 8 months earlier, for which he was on regular thyroxine therapy. Physical examination revealed pallor, bilateral pedal edema, and mild hypertension. Laboratory investigations showed hematuria, proteinuria, elevated serum creatinine, and normal complement levels. Thyroid function tests were consistent with treated hypothyroidism. Renal biopsy demonstrated mesangial proliferation with dominant IgA deposition on immunofluorescence, confirming IgA nephropathy[4]. ***Conclusion:*** The coexistence of autoimmune thyroiditis and IgA nephropathy is rare but clinically significant, suggesting a possible shared autoimmune pathogenesis[5,6]. Early recognition is important for appropriate management and monitoring of renal function in patients with autoimmune thyroid disease.

Citation: Dr. Mallinath S M, Dr. Jeetram Jat, Dr. R. L. Meena, Dr. Ashok Choudhary, Dr Rajveer Bunker and Dr. Shailendra Gupta. 2025. "Coexistence of Autoimmune Thyroiditis and IgA Nephropathy in a Young Adult Male: Expanding the Spectrum of Thyroid-Related Renal Involvement". *International Journal of Current Research, 17, (08), 34331-34333.*

INTRODUCTION

Autoimmune thyroiditis (Hashimoto's thyroiditis) is the most common cause of hypothyroidism in iodine-sufficient regions[7]. It is characterized by gradual thyroid destruction mediated by autoantibodies against thyroid peroxidase and thyroglobulin[8]. While it often coexists with autoimmune conditions like type 1 diabetes, celiac disease, and pernicious anemia[9], renal involvement is unusual. IgA nephropathy (IgAN) is the most common primary glomerulonephritis worldwide, characterized by mesangial IgA deposition[10,11]. Although its pathogenesis involves immune dysregulation and abnormal IgA glycosylation, an association with autoimmune thyroiditis is rare[1,2,12]. We present a rare case of IgA nephropathy in a young male with autoimmune thyroiditis, highlighting the clinical implications and possible pathophysiological links.

Case Presentation: A 26-year-old male presented in medicine OPD with shortness of breath which was insidious in onset and gradually progressive and not associated with any postural change and diurnal variationthere was no associated chest pain also. Patient also complains of gross hematuria for 15 days which was not associated with pain abdomen and burning micturation which was observed by patient himself. There was no history of fever, rash, joint pains, sore throat, or recent upper respiratory tract infections, purpura.

Past History: Eight months earlier, he had been diagnosed with autoimmune thyroiditis based on elevated anti-thyroid peroxidase antibodies, a hypoechoic heterogeneous thyroid on ultrasound, and elevated TSH levels. He was started on levothyroxine 75 µg/day with good compliance.

Examination and Investigations: On general examination, the patient appeared pale with bilateral pitting pedal edema. His blood pressure was mildly elevated at 148/92 mmHg, pulse rate 84 beats per minute, respiratory rate 18 per minute, and he

was afebrile. There was no goiter, lymphadenopathy, skin rash, or joint swelling. Cardiovascular and respiratory system examinations were unremarkable, and abdominal examination revealed no organomegaly or ascites. Neurological examination was normal. Laboratory evaluation revealed hemoglobin of 9.4 g/dL, total leukocyte count of 7,800/µL, and platelet count of 2.1 × 10^5/µL. Renal function tests showed blood urea of 56 mg/dL and serum creatinine of 1.8 mg/dL. Urinalysis demonstrated 3+ proteinuria with numerous red blood cells and the presence of red blood cell casts. A 24-hour urine collection showed protein excretion of 2.3 g/day. Antinuclear antibody and anti-dsDNA antibody tests were negative, and complement levels (C3 and C4) were within normal limits. Thyroid function testing showed a TSH of 4.2 mIU/L with normal free T4 levels, consistent with treated hypothyroidism. Anti-thyroid peroxidase antibody remained elevated.

Renal Biopsy

Gross

LM: - Received single linear core measuring 1.6cm in length.

IF: - Received single linear core measuring 0.9cm in length.

Light Microscopy Report: The kidney biopsy shows 21 glomeruli out of which 2 are globally sclerosed and one show segmental sclerosis. 2 Glomeruli shows a partial fibrocellular crescent. The viable glomeruli are enlarged with features of compensatory hypertrophy. There is increase in mesangial matrix and cellularity. Occasional glomeruli show features of segmental endocapillary proliferation. Basement membrane show focal thickening on silver methenamine stain. Tubules show protein reabsorption granules. Blood vessels show medial sclerosis. The interstitium show small collections of inflammatory cells. 15-20% shows chronic parenchymal damage on Masson trichrome

Immunofluorescence Report: The tissue sent for immunofluorescence shows 8 glomeruli, of which one is globally sclerosed. The viable glomeruli show coarse granular deposits of IgA(4+), IgG(2-3+) and C3(2+) in the mesangium. These deposits show greater intensity of staining for Lambda light chain(4+) over Kappa(0).

Impression: Findings are suggestive of IgA Nephropathy with mild chronic parenchymal damage.

MEST-C Score: - M1 E1 S1 T1 C1

Management and Outcome

The patient was started on:

- Angiotensin-converting enzyme inhibitor (Ramipril 2.5 mg OD)
- Oral corticosteroids (prednisolone 1 mg/kg/day)
- Salt and protein restriction
- Continued levothyroxine

After 3 months, proteinuria reduced to 0.8 g/day, creatinine improved to 1.4 mg/dL, and hematuria resolved. Blood pressure was controlled.

DISCUSSION

The association between autoimmune thyroiditis and IgA nephropathy is rare[1,2,3,14,15]. The underlying mechanisms may involve:

- Shared genetic predisposition — HLA-DR alleles and immune-regulatory gene polymorphisms[7,8].
- Autoimmune cross-reactivity — Chronic thyroid autoimmunity may induce abnormal IgA1 glycosylation, facilitating mesangial deposition[5,10].
- Immune complex deposition — Circulating immune complexes containing thyroid antigens might deposit in renal glomeruli[6,11].

Previous case reports[2,3,14] indicate most patients are young adults with microscopic or gross hematuria. Membranous nephropathy and minimal change disease are more frequently reported in autoimmune thyroiditis, making IgAN an unusual presentation[12]. In our case, the diagnosis was confirmed histologically with dominant mesangial IgA deposits. Early intervention with corticosteroids and RAAS blockade helped preserve renal function, consistent with KDIGO recommendations[13].

CONCLUSION

This case demonstrates the rare coexistence of autoimmune thyroiditis and IgA nephropathy in a young adult male, underscoring the importance of considering renal involvement in patients with autoimmune thyroid disease who develop urinary abnormalities. Although renal manifestations are uncommon, IgA nephropathy should be included in the differential diagnosis alongside other glomerulopathies. A shared autoimmune basis and possible cross-reactive mechanisms may underlie this association, highlighting the need for collaboration between endocrinology and nephrology teams. Routine urinalysis and renal function monitoring in such patients can enable early detection and timely management, potentially preventing long-term renal impairment. Further studies are warranted to clarify the immunopathogenic links between thyroid and renal autoimmunity, which could guide preventive and therapeutic strategies.

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