Several Forms of Renal Disease May Be Associated with Thyroid Autoimmune Disease


SUMMARY

BACKGROUND

The coexistence of Hashimoto’s thyroiditis and Graves’ disease (1) with other autoimmune diseases (AID) is well established and possibly more frequent in adolescent and young adults than in later life (2). Furthermore, most textbooks mention the association with adrenal insufficiency, disseminated lupus, psoriasis, type 1 diabetes and other AID. Occasionally, the coexistence of thyroid AID with renal disease has been reported. Yet all these associations are rare events, perhaps with the exception of the association of type 1 diabetes with thyroid autoimmune disease. The present article reports on an unusually large series of cases of renal involvement in Hashimoto’s thyroiditis.

METHODS AND RESULTS

From 2007 to 2011, 28 patients with Hashimoto’s disease were referred to the department of nephrology in an Ankara hospital because of unexplained hematuria, proteinuria, or renal impairment. Patients with a history of kidney stones, systemic vasculitis, connective-tissue disease, or diabetes mellitus were excluded from the present study. Renal biopsies were performed in 20 patients. In 8 patients, the renal abnormalities were transient and did not need to be biopsied. The mean (±SD) age of patients was 46±17 years; 18 women and 10 men were affected. The glomerular filtration rate (GFR) was below 60 ml/min/1.73 m² of body-surface area in 43% of the patients. Hematuria was found in 39% and proteinuria in 86%. Hypertension was present in 57% of patients. There was a significant difference in GFR between recent-onset Hashimoto’s thyroiditis (<12 months) and long-standing disease. In early Hashimoto’s thyroiditis, the mean GFR was 83 ml/min/1.73 m², in long-standing disease 55 ml/min/1.73 m². In 15 to 20% of the patients, renal biopsies showed either focal or membranous glomerulonephritis, immunoglobulin A nephritis, or minimal change disease. There was one case of amyloidosis. Most importantly, there was no correlation between renal function and thyroid antibodies (antithyroglobulin and or anti-TPO antibodies). Sixteen patients (57%) received either angiotensin-converting enzyme inhibitors or angiotensin receptor blockers. Four patients did not receive antihypertensive treatment because of hypotension or absence of proteinuria. Nevertheless, in 7 of 20 patients, immunosuppressive therapy had to be introduced. The success of treatment was variable but within the frame of treatment responses observed in similar cases of renal diseases without Hashimoto’s thyroiditis.

CONCLUSIONS

The study reported on 28 patients with concomitant Hashimoto’s thyroiditis and renal glomerular disease. There was no association between thyroid antibody titer and renal function. This is a novel and important finding because so far only sporadic isolated cases have been described. The response of the glomerular disease to therapeutic interventions did not differ from that in patients without Hashimoto’s thyroiditis. No data concerning a possible link between thyroid antibody immune complexes and glomerular disease are available. The authors do not comment on possible intrarenal immune complexes with antithyroglobulin or anti-TPO antigenicity. It is not clear whether they tested this possibility. Apparently, the treatments for the renal disease did not differ from those in patients without Hashimoto’s thyroiditis.

continued on next page
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ANALYSIS AND COMMENTARY

The association between Hashimoto’s thyroiditis and renal glomerular disease was first recognized a few years ago. It is, however, most difficult to obtain quantitative information about the frequency of this association. Even the present article leaves this question open, since the patients were probably referred from many centers.

The value of this article lies in the large number of cases and in the facts that these were not adolescents or young adults and that there was no correlation between antibody titer and renal involvement. The latter point has to be interpreted cautiously, since antibody titers can fluctuate greatly over time. The finding that long-standing hypothyroidism was associated with more severe renal involvement may hint at a possible causal relationship. Despite the relatively large number of cases, little can be said about the therapeutic response, particularly since the data relating to the response is difficult to understand.

The clinician must realize that a causal relationship between renal disease and Hashimoto’s thyroiditis is, at present, no more than an unproven hypothesis, since statistical proof is lacking. Nevertheless, it makes sense to follow the decrease of serum creatinine after starting thyroxine treatment in Hashimoto’s thyroiditis, and particularly in cases of coexisting hypertension, to look for any form of renal disease.

— Albert G. Burger, MD

REFERENCES
