Autoimmune Chronic Thyroiditis on Chronic Hemodialysis

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Dear Sir,

Abnormalities in the thyroid function test are common in chronic renal failure: serum total thyroxine (T₄) and triiodothyronine (T₃) may be low, there is often a reduction in T₄ binding by serum proteins, and the thy-roidal response to TSH may be blunted [1, 2]. Whilst goiter and exophthalmos are well recognized in chronic renal failure [2, 3] populations, thyrotoxicosis is extremely rare with only 3 cases described [4–6]. We report a case of thyrotoxicosis due to Hashimoto's disease (autoimmune thyroiditis). We think that this is the first report of this clinical association.

A 63-year-old female was admitted for dialysis due to chronic interstitial nephropathy in October 1983. In October 1985, the dialysis treatment was complicated by paroxysmal supraventricular tachycardia which persisted for several hours after completion of the treatment. One of the patient's daughters suffered of thyroidectomy due to Graves' disease in 1987. In June 1988, she developed emotional instability, insomnia, heat intolerance, and weight loss.

Physical examination on admission revealed distress and muscle weakness. Her pulse rate was regular at 100/min. Mild proptosis and lid lag were present. The thyroid gland was moderately enlarged without nodules. No other signs of hyperthyroidism were present.

Laboratory data obtained on admission included: normal electrolytes: calcium 10.4, phosphorus 3.9 mg/100 ml; cholesterol 94, triglycerides 66 mg/100 ml, thyroid hormones: serum T₃ 314 ng/100 ml; serum T₄ 14.9 µg/100 ml, and 0.01 µU/ml; thyroglobulin: 4.6 ng/ml; antithyroglobulin antibodies: 1,397 UI/ml; antimicro-somal antibodies: 8,403 UI/ml. Thyroid ultrasound examination showed an adenomatous multinodular goiter. ¹³¹I gammagraphic examination revealed a diffusely hyperplastic thyroid gland with increased accumulation in the left lobe. Thyroid biopsy: coloidal goiter with oxyphilic cells (Hürthle cells) and lymphocytes (fig. 1). Fig. 1. Chronic lymphocytic thyroiditis. Lymphocytic cells around a group of oncocytes. Diff. Quick. × 40.

The patient was initially treated with propranolol (20 mg q.i.d., discontinued lately due to auriculoventricular block), diazepam and methimazole (40 mg/day). This further drug was

slowly tapered once an euthyroid state was reached. In January 1989, she developed bradypsychia and progressive increasing of weight with the following laboratory data: T₃ 0.51 ng/ml; T₄ 1.10 µg/ml, and TSH 0.02 µU/ml. Methimazole is reduced to 5 mg/day. In March 1989, she started again with emotional instability and weight loss. Her thyroid studies at this time were: T₃ 1.35 ng/ml; T₄ 8.70 µg/ml, and TSH 1.28 µU/ml. The dosage of methimazole was increased to 20 mg/day with improvement of her clinical and biochemical state. In July 1989, the patient developed mild leukopenia (white cell count 3,200/ml). Methimazole was discontinued for 1 month without improvement of the white cell count (3,600/ml). She became hyperthyroid again. Methimazole (35 mg/day), atenolol and diaze-

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epam were started one more time, until she became euthyroid. Then, methimazole was tapered again to 20 mg/day. The patient's euthyroid clinical and biochemical status has remained stable since then without changes.

We have been able to find only 3 previous case reports of thyrotoxicosis in patients with chronic uremia [4–6]; no one of them was due to autoimmune thyroiditis. The patient we described had immunologic and pathologic evidence supporting a diagnosis of Hashimoto's disease. As in the case described by Foley and Hammer [6] the diagnosis of hyperthyroidism was delayed some months in spite of the common signs and symptoms of that disease; anyway, they can easily be ascribed to symptoms and signs commonly found in the dialysis population. The familial antecedents were the key suggesting the diagnosis of hyperthyroidism in this patient.

The elective treatment of hyperthyroidism in chronic renal failure could be controversial. Since methimazole clearance is unaltered in renal failure [7], administration of this drug might be preferable to propylthiouracil in this circumstance. However, it has been suggested that propylthiouracil could be safely administered to thyro-toxic patients receiving hemodialysis, although it would be advisable to administer the drug after dialysis to get higher and more predictable serum levels [8–10]. Both drugs have been useful and safe when used in the previous cases of hyperthyroidism in hemodialysis [4–6]: Other cases of hyperthyroidism in hemodialysis have been successfully treated with radioactive thyroid ablation; nevertheless, the natural history of autoimmune thyroiditis and the good control of the disease with drugs has dissuaded us from this form of therapy.

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