Recurrence of Thyrotoxicosis after Correction of Uraemia in a Patient with Chronic Renal Failure

M.	Mohammad	Absy
Y.C.	Yin C.	Ng

Renal Unit, King Fahd Armed Forces Hospital, Jiddah, Saudi Arabia

Dr. Mohammad Absy, PO Box 926340, Amman (Jordan)

Dear Sir,

Chronic renal failure is associated with clinical euthyroid status in spite of reduced thyroid hormone levels (free T₄ and T₃). Thyrotropin (TSH) is normal or slightly raised, and the TSH response to TSH-releasing hormone (TRH) stimulation is impaired [1-4]. The occurrence of thyrotoxicosis in chronic renal failure is a rare event [4-7]. We describe a unique observation where the correction of uraemia was followed by clinical and biochemical recurrence of thyrotoxicosis in a patient with chronic renal failure and a history of hyperthyroidism.

An 83-year-old female was admitted with a history of recurrent urinary tract infection, proteinuria (less than 1 g/day), and progressive renal insufficiency over 11 years. She had a goitre since childhood. Three years prior to admission, she complained of palpitations and was found to be hypertensive, in atrial fibrillation, and thyroid function tests revealed free T₄ 26.5 pmol/l or 20.7 pg/ml (normal 11.0-23.0 pmol/l or 8.6-18.0 pg/ml), free T₃ 9.7 pmol/l or 6.2 pg/ml (normal 3.2-8.0 pmol/l or 2.1-5.2 pg/ml), TSH 0.13 mIU/l (normal 0.38-3.9 mIU/l), and colloid antibodies 1:320 (normal less than 1:100). Serum creatinine was 255 µmol/l (2.3 mg/dl). A thyroid sodium (^{99m}Tc) pertechnetate scan revealed an irregular pattern with multifocal areas of increased activity consistent with toxic multifocal goitre. She was not seen again for 1 year at which time she was still in atrial fibrillation. She was started on carbimazole (15 mg/d) and propranolol which was stopped 3 months later with the intention of treating her thyrotoxicosis with radio-iodine. Unfortunately, she did not appear for follow-up. On this admission her only medication was atenolol. Physical examination revealed an emaciated woman weighing 36.6 kg and having a regular pulse rate of 72/min (confirmed by ECG). There was an enlarged

multinodular thyroid gland. There was no lymph node enlargement, tremor, exophthalmos, or lid retraction. Blood analysis showed haemoglobin 5.2 g/dl (52 g/l), serum creatinine 858 µmol/l (9.8 mg/dl), urea 36.8 mmol/l (blood urea nitrogen 103 mg/dl), free T₄ 15.3 pmol/l (12.0 pg/l), free T₃ 3.5 pmol/l (2.2 pg/l), and TSH 0.06 mIU/l. A Tenckhoff peritoneal catheter was placed under local anaesthesia, and the patient was started on continuous ambulatory peritoneal dialysis 6 days after admission. Eleven days later, the laboratory findings were as follows: urea 10.3 mmol/l (blood urea nitrogen 28.8 mg/dl), creatinine 428 µmol/l (4.9 mg/dl), free T₄ 39.3 pmol/l (30.8 pg/ml), and TSH less than 0.06 mIU/l. A TRH stimulation test (TRH Roche) with 200 µg, was flat after 20 and 60 min. The results were

identical when 400 µg, TRH was used. Two weeks later, the patient became clinically thyrotoxic, having developed sinus

References

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- tachycardia and tremor, and was started again on carbimazole and propranolol.
- This elderly patient had a toxic multinodular goitre complicating a long-standing simple goitre. She was found previously thyrotoxic and was treated with carbimazole for a short period of time before being lost to follow up. It would seem that the worsening of her renal failure protected her from overt hyperthyroidism [1, 8], but when her uraemia was corrected with dialysis, clinical thyrotoxicosis became manifest, and the level of free T₄ was consistent with hyperthyroidism. These observations suggest a reciprocal relationship between thyroid hormone levels and uraemia and are consistent with a previous description of acute renal failure being associated with a decline in thyroid hormone levels in a thyrotoxic patient [8]. The mechanisms by which renal failure reduces free T₄ remain unknown.
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