Amiodarone induced Graves’ thyrotoxicosis in a patient with resistance to thyroid hormone

M Nouvirt, R Coulson, P Owen, M Page

Royal Glamorgan Hospital, Llantrisant, Wales

Introduction
We present a patient with resistance to thyroid hormone (RTH) in whom administration of amiodarone induced hyperthyroidism in the presence of co-existing latent Graves’ disease.

Case
A 43 year old with RTH was reviewed with routine TFTs showing FT4 45.1pmol/l and TSH 0.93 mU/l. Nine months earlier he had been initiated on amiodarone prior to his fourth DC cardioversion for atrial fibrillation (AF) and had subsequently remained in sinus rhythm. Within 4 weeks his FT4 was >100pmol/l, T3 22.5pmol/l with TSH 0.01mU/l and was clinically thyrotoxic. He was initiated on Carbimazole 40mg and prednisolone 40mg and in discussion with Cardiology his amiodarone was stopped. One month later he remained biochemically thyrotoxic and complained of eye symptoms. TSH receptor antibodies were positive (10.2). The dose of carbimazole was increased and steroids withdrawn. A titration regime of Carbimazole therapy was given over the next six months. After discontinuation he remained clinically and biochemically euthyroid, FT4 32.4 pmol/l and TSH 1.28 mU/l (consistent with previous TFTs) with no recurrence of AF. Three months later he presented with hyperthyroid symptoms again and TFTs showed FT4 36.1 pmol/l and TSH 0.11mU/l and he has been restarted on Carbimazole prior to ablative treatment.

Discussion
This man had RTH and latent Graves’ disease. He presented with thyrotoxicosis after treatment with amiodarone. We suggest the iodine load of amiodarone led to an amiodarone induced thyrotoxicosis type 1 in the presence of Graves’ disease. The co-existence of these diagnoses presents a challenge to clinical management. Monitoring of anti-thyroid therapy is reliant on TSH levels and as with relapsed Graves’ disease definitive treatment needs to be carefully planned in view of his cardiovascular problems and mild thyroid eye disease.