ptosis; and diplopia. An episode affected either or both eyes and lasted one to three weeks. The patient was completely normal between attacks. She had had a partial thyroidectomy when aged 19 years for hyperthyroidism with exophthalmos. There had been complete recovery, and no thyroid medication was given. A sister aged 50 years had also had hyperthyroidism. There were no recent symptoms of thyroid, muscular, or neurological disease. Throughout two and a half years of observation by us the patient remained clinically euthyroid. Changes comprising eye pain, swelling of eye muscles, redness and oedema of conjunctivae, and severe ophthalmoplegia affected one or both eyes to differing extents in different episodes. The most usual restriction of eye movement was upwards, but sometimes lateral or medial deviation was the more severe abnormality. During a representative period without treatment consecutive attacks came at intervals of 29, 32, 18, 8, 3, 23, 55, 21, 10, 13, 17, and 25 days. They were not associated with menstruation or changes in body weight. There was no myasthenia, and Tensilon testing was normal on three occasions. There was no exophthalmos, maximum Hurtle reading at 100 mm separation being 16 mm.

Serum thyroxine (T4) and triiodothyronine (T3) have been normal on nearly all occasions (figure). Serum thyroid stimulating hormone (TSH) was

or improvement over months or years.¹ In our patient eye symptoms fluctuated relatively rapidly, with complete remissions between attacks, but over the four years before prednisone was started the severity had not changed. We believe that we have witnessed an unrecorded part of the range of dysthyroid eye disease.

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Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0HS

W F KELLY, MB, MRCP, honorary senior registrar

G F JOPLIN, PHD, FRCP, senior lecturer and honorary consultant physician

Measurement in case of intermittent ophthalmoplegia. Normal ranges are hatched. Arrows show peak serum TSH response (normal < 25 mU/l) after TRH 200 µg intravenously.

Conversion: SI to traditional units—T4: 1 nmol/l ≈ 0.78 ng/ml. T3: 1 nmol/l ≈ 0.65 ng/ml.

usually at or slightly above the upper limit of normal, both basally and after thyrotrophin releasing hormone (TRH) 200 µg intravenously. Free thyroxine index, neck uptake of technetium, and T3 suppression were all normal. Long-acting thyroid stimulator (LATs) and LATs-protector were not detected. Thyroid antibodies were positive (tanned red cells titre 640, cytoplasm haemagglutination titre 6400). Serum complement, full blood count, and ESR were always normal and there were no circulating immune complexes. Radiology of the optic foramina, pituitary fossa, and orbital veins was normal. An EMI scan showed slightly enlarged eye muscles. In May 1977 prednisone 10 mg/day was started empirically. There was a reduction in severity but not frequency of symptoms. We have titrated the dose of prednisone against the severity of symptoms and found 10 mg/day to be the lowest reasonably effective dose.

Comment

The history, family history, and antibody studies all implicate the thyroid gland. Alternative diagnoses such as myasthenia gravis, orbital tumour, multiple sclerosis, and ethmoiditis are now excluded. Ophthalmoplegia and other eye signs occur in association with both hyper- and hypo-thyroidism,¹⁻³ but the pathogenesis remains enigmatic. An association with abnormal thyroid antibodies directed against orbital tissues has been proposed.⁴ In hyperthyroidism either LATs or LATs-protector are detectable in 90% of patients.⁵ The natural time course of dysthyroid eye disease is of gradual worsening

Hashimoto's thyroiditis presenting as T3 toxicosis

In the early phases of Hashimoto's thyroiditis the patient may have the clinical features of hyperthyroidism,¹ usually associated with increased secretion of thyroxine. We describe here a case of Hashimoto's thyroiditis presenting with hyperthyroidism associated with increased secretion of triiodothyronine rather than thyroxine, and this particular patient shows elegantly the progression from T3 toxicosis to hypothyroidism.

Case report

A woman aged 43 was referred to the endocrine clinic in July 1977 complaining of loss of 3.5 kg in weight and a fine tremor of her fingers that interfered with her work as an artist. She was taking no medication. Her mother had suffered from Graves's disease, but there was no other relevant family history. On examination she was fidgety and had lid-lag without exophthalmos and a fine tremor of the fingers. The thyroid gland was not enlarged. The serum thyroxine (T4) concentration was normal but the serum triiodothyronine (T3) concentration was raised (table). Thyroid microsomal antibodies were detected by immunofluorescence (+ ±) but no antibodies to thyroglobulin were detected by the immunofluorescent or tanned red cell techniques.

During the next seven months no treatment was given. The tremor and lid-lag persisted but the weight remained virtually constant. The serum concentration of T4 remained normal and that of T3 was persistently raised. The serum concentration of thyroid stimulating hormone (TSH) was low, and the thyrotrophin-releasing hormone (TRH) test showed a flat response (basal TSH < 1, at 20 min 1.4, and at 60 min < 1 mU/l). In April 1978 the patient said she felt distinctly better, with loss of her tremor and some weight gain.

Serial measurements of serum thyroxine (T4), triiodothyronine (T3), and thyroid stimulating hormone (TSH) concentrations in case of Hashimoto's thyroiditis presenting as T3 toxicosis

	T4 (nmol/l)	T3 (nmol/l)	TSH (mU/l)	Weight (kg)
July 1977	127	3.35	<1.0	58.00
Sept 1977	141	3.40		58.25
Oct 1977	121	3.55		58.65
Feb 1978	107	3.20		58.93
April 1978	61	1.30	1.3	59.70
May 1978	40	1.60	40.0	61.85
June 1978	37	1.60	50.0	62.70
Normal range	70-160	1.08-2.61	<1-3.5	

Conversion: SI to traditional units—Thyroxine: 1 nmol/l ≈ 0.08 µg/100 ml. Triiodothyronine: 1 nmol/l ≈ 0.06 ng/100 ml.

At this time the T4 concentration had fallen to slightly below normal and the T3 concentration was within the normal range for the first time, although the TRH test remained suppressed (basal TSH 1.3, at 20 min 1.2, and at 60 min 1.0 mU/l). A thyroid scan showed uptake confined largely to the left lobe of the gland. Thyroid microsomal and thyroglobulin antibodies were detected (+ + and + ± respectively). In May 1978 the patient had gained more weight and complained of tiredness. The concentration of T4 had fallen further while that of T3 remained normal, and the basal serum TSH was now raised. There was no change in the thyroid scan, which was thought to be consistent with extensive thyroiditis on the right side. A month later the hormone concentrations were unchanged.

Comment

Patients with Hashimoto's thyroiditis may be hyperthyroid, euthyroid, or hypothyroid depending on the duration of the disease and the stage at which they are first seen and assessed. For example, in a review of 217 patients² 77.9% were euthyroid, 18.4% were hypothyroid, and 3.7% were hyperthyroid. Hyperthyroidism due to T3 toxicosis seems to be unusual. We were unable to find another report of a convincing case of T3 toxicosis proceeding to hypothyroidism in the absence of treatment. Our patient, on no antithyroid medication, progressed in 11 months from a state of undoubted T3 toxicosis to hypothyroidism associated with the expected rise in serum TSH concentrations.

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Department of Endocrinology, Westminster Hospital, Dean Ryle Street, Horseferry Road, London SW1

R K MALLYA, MB, MRCP, medical registrar

A J ISAACS, BM, MRCP, senior medical registrar

RICHARD BAYLISS, MD, FRCP, consultant physician

Profound bradycardia in response to atrial fibrillation in the Wolff-Parkinson-White syndrome

In the Wolff-Parkinson-White (WPW) syndrome an anomalous pathway bypasses the physiological filtering characteristics of the atrioventricular node. Atrial fibrillation may therefore result in a rapid ventricular response that may cause syncope or sudden death. In the patient with WPW syndrome described here atrial fibrillation paradoxically resulted in syncope due to profound ventricular bradycardia.

Case report

A 41-year-old chef who was an extremely fit rugby player complained of several syncopal spells, frequent dizziness, and a six-year history of occasional bouts of palpitations. On examination no physical abnormalities were detected except for a resting bradycardia. His chest radiograph and blood values were normal. The resting 12-lead electrocardiogram (ECG) showed atrial bradycardia (35 beats/min) with abnormal P-wave morphology and a P-R interval of 0.16 second. The QRS complexes were broad (0.17 second) with initial slurred delta waves, positive in leads I and V6 and negative in lead V1, typical of WPW syndrome type B. A stress test showed excellent exercise tolerance and an appropriate increase in atrial rate. A 24-hour taped ECG showed a basic heart rate that varied from 35 to 120 beats/min and, in addition, pauses in the basic rhythm of up to 2.5 seconds long.

An atrial pacing study was performed which showed abnormal sinus node function with unduly long sinus node suppression (2.6 seconds) after rapid atrial pacing and impaired atrioventricular conduction resulting in 2:1 atrioventricular block at an atrial rate of 100 beats/min. A formal study of the conducting capacity of the anomalous bypass tract, responsible for the WPW syndrome, showed that this tissue was not capable of conducting at intervals shorter than 0.53 second. The induction of atrial fibrillation by rapid atrial

pacing resulted in the dramatic response (see figure) of extreme ventricular bradycardia with R-R intervals of up to 11 seconds in duration and uniformly pre-excited QRS complexes, indicating conduction to the ventricles by the bypass tract. This bradycardia was associated with symptoms identical with those for which the patient had been admitted. In view of this response to atrial fibrillation, impaired sinoatrial function, and atrioventricular conduction, a ventricular demand pacemaker was implanted. During follow-up of almost four months there have been no further episodes of near-syncope or blackouts.

Comment

A substantial proportion of patients with the WPW syndrome suffer spontaneous paroxysms of atrial fibrillation.¹ If the anomalous pathway which links atrium to ventricle can conduct atrial impulses at a high frequency then the ventricles may be stimulated too rapidly to maintain an adequate cardiac output. This is a mechanism commonly invoked to explain episodes of ventricular fibrillation, syncopal spells

Profound ventricular bradycardia with pre-excited QRS complexes during atrial flutter-fibrillation. HRAE, DCSE, PCSE, and HBE are electrograms recorded from the high right atrium, the distal and proximal coronary sinus, and His-bundle regions. I, AVF, V1, and V6 are simultaneous surface ECG leads.

and sudden death in these patients.² If the bypass tract cannot sustain high-frequency conduction atrial fibrillation is thought unlikely to provoke serious ventricular arrhythmias.³

In this patient reduced sinoatrial automaticity and atrioventricular nodal conduction might be ascribed to increased vagal tone in a physically fit individual⁴ and would not in itself indicate the need for cardiac pacing. In the presence of atrial fibrillation profound ventricular bradycardia sufficient to induce syncope cannot be wholly related to the autonomic state of a physically fit individual but must imply widespread conduction disease and the failure of subsidiary pacemaker mechanisms. The low ventricular rate is all the more remarkable in a patient with two atrioventricular conduction pathways but might be explained by repetitive and incomplete conduction into these pathways.⁵

The treatment of WPW syndrome and atrial fibrillation may require antiarrhythmic drug suppression of atrioventricular conduction in both the normal and abnormal pathways to prevent symptoms related to a rapid ventricular response. In this case such treatment might have proved disastrous. Simple atrial pacing techniques allowed an improved assessment of this patient's problem, showed an unsuspected syncopal mechanism, and suggested that the correct treatment should be ventricular pacing rather than antiarrhythmic drugs.

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⁵ Castellanos, A, et al, *British Heart Journal*, 1973, **35**, 811.

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Department of Cardiology, St Bartholomew's Hospital, London EC1

A J CAMM, MB, MRCP, lecturer in cardiology

D E WARD, MB, MRCP, registrar in cardiology

R A J SPURRELL, MD, FACC, consultant in charge