Addison's disease and thyrotoxicosis presenting simultaneously

P. G. NEWRICK
M.B., M.R.C.P.

Department of Neurology, Frenchay Hospital, Bristol BS16 1LE

Summary
The simultaneous presentation of two endocrinological diseases is uncommon. A case is discussed in which Addison's disease and thyrotoxicosis were discovered in an individual following presentation as an emergency neurological problem.

KEY WORDS: vitiligo, autoimmunity.

Case report
A 65-year-old man was referred as an emergency with a history from his wife of a vaso-vagal episode complicated by short-lived twitching of one leg while recovering. Physical examination was unremarkable apart from extensive vitiligo. Blood pressure, serum electrolytes and skull X-ray were normal. He was discharged home but 2 weeks later re-admitted following a progressive severe deterioration with anorexia, nausea, vomiting and profound weakness. Examination showed a very ill man with the previously noted vitiligo of face, trunk and limbs but no palmar or buccal pigmentation. Blood pressure was 70/40 mmHg supine, and pulse 130/min. There were no abnormal abdominal or neurological signs.

A presumptive diagnosis of acute adrenal failure was made and the patient treated with intravenous normal saline and hydrocortisone. Serum electrolytes before treatment were: sodium 127 mmol/l, potassium 5-7 mmol/l, bicarbonate 26 mmol/l and urea 31-6 mmol/l. A short Synacthen test performed immediately before hydrocortisone replacement gave cortisol values as follows: zero min 84 nmol/l (normal 280–690), 30 min 82 nmol/l, 60 min 106 nmol/l. The following investigations were normal: electrocardiograph, chest X-ray, computed tomographic scan of head, abdominal X-ray, blood glucose, serum calcium, liver function tests and cardiac enzymes. A Mantoux test (1:1,000) was negative and acid fast bacilli absent from early morning urine on three occasions. An auto-antibody screen (including adrenal auto-antibodies) was negative.

The patient made a slow recovery over 5 days but needed an unusually large dose of hydrocortisone (80 mg daily) to maintain a blood pressure of 120/80 mmHg; in addition to fludrocortisone therapy. Despite symptomatic improvement a tachycardia of 120/min persisted. There were no other clinical signs or symptoms suggestive of hyperthyroidism but thyroid function tests showed: total thyroxine 206 nmol/l (normal 58–167), free thyroxine 45-3 nmol/l (normal 10–30), tri-iodothyronine 5-9 nmol/l (normal 0-8–3). A TRH test gave a flat response consistent with primary thyrotoxicosis, with TSH values at zero, 20 and 60 min all less than 1-5 mIU/l. Following 5 days of intramuscular Synacthen (1 mg daily) a further short Synacthen test was performed and gave results consistent with primary adrenal failure with cortisol values as follows: zero min 10 nmol/l, 30 min 49 nmol/l, 60 min 69 nmol/l. He was discharged on hydrocortisone, 60 mg mane and 20 mg nocte, fludrocortisone 50 μg and carbimazole 30 mg daily. Two weeks later he was in excellent health, with a pulse rate of 68/min. Over the next month it became possible to maintain him on a daily total of 30 mg hydrocortisone as he was rendered euthyroid. A technetium isotope thyroid scan was later performed with the patient off carbimazole for 14 days. This showed a diffuse increase in uptake but no focal abnormality. Repeat thyroid membrane auto-antibodies were again negative and serum B₁₂ levels were subnormal at 100 (normal 200–1000 ng/l).

Comment
Primary adrenal and thyroid disorders can both be manifestations of auto-immune endocrine disease. The association between primary adrenal failure and hypothyroidism is relatively common and occurs in 10% of Addisonian patients at some time (Ramsay, 1980). More recently transient hypothyroidism which clears following adequate treatment for Addison's has been reported (Barrett, Donald and Espiner, 1982). The link between adrenal failure and hyperthyroidism, however, is much rarer. It has been argued that any such link is fortuitous and depends
on the closer surveillance of Addisonian patients but Gastineau et al. (1964) found that as many as 16 (2.9%) of 538 Addisonian patients were thyrotoxic at some time. In 12 patients thyrotoxicosis was the first event, in three thyrotoxicosis followed Addison’s disease and in only one case did both diseases present together. Rupp and Paschkis (1957) in their search found nine cases of simultaneous presentation. The aetiology of such presentation is unclear but possibilities include genetic predisposition and simultaneous auto-immune attack on adrenal and thyroid (Gastineau et al., 1964). An auto-immune aetiology seems likely in this case although thyroid antibodies were not detected as is not uncommon at this stage. The absence of an adenoma on isotope scan, the presence of vitiligo and a low serum B12 (in the absence of any other obvious factors) are additional supportive evidence. The higher dose of hydrocortisone required by this patient initially was presumably due to an increased rate of degradation or increased requirements consequent upon a raised metabolic rate from hyperthyroidism (Hellman et al., 1961). It is suggested that any Addisonian patient with persistent tachycardia or high steroid requirement should have thyroid function investigated.

Acknowledgment

I thank Dr M.J. Campbell for permission to report his patient.

References


(Accepted 1 June 1983)
Addison's disease and thyrotoxicosis presenting simultaneously.

P. G. Newrick

Postgrad Med J 1984 60: 478-479
doi: 10.1136/pgmj.60.705.478

Updated information and services can be found at:
http://pmj.bmj.com/content/60/705/478

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/