Lesson of the Week

Acute adrenal crisis precipitated by thyroxine

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It is well recognised that in patients with pituitary insufficiency thyroid hormone replacement without concomitant adrenal steroid replacement may precipitate an acute adrenal crisis. It is less widely appreciated that similar problems may arise during treatment of primary myxoedema if the patient also has unrecognised or subclinical adrenocortical disease. We report on two patients with primary hypothyroidism in whom thyroxine replacement precipitated an acute Addisonian crisis.

Case reports

CASE 1

A 47 year old woman had presented elsewhere three years previously with a history of lethargy. No clinical or biochemical abnormality had been detected. Her symptoms persisted, and two years later a low serum thyroxine concentration and a raised serum thyroid stimulating hormone concentration were recorded. Plasma urea and electrolyte concentrations were normal. She was prescribed thyroxine 50 μg daily, increasing to 100 μg after two weeks, but felt ill and dizzy, lost weight, and stopped taking the thyroxine, attributing her symptoms to it. Over the next few months she was seen by several doctors; low serum thyroxine and raised serum thyroid stimulating hormone concentrations were found each time, and she was advised to resume taking thyroxine. Initially she took it only intermittently because it made her feel unwell within a few days, but eventually, because of repeated medical persuasion, she took it continuously for two months.

During this period she became progressively more unwell, lost 10 kg in weight, and was confined to bed, unable to cope with daily activities; menstruation, however, continued. She then stopped taking thyroxine and was referred to this hospital.

On examination she was thin and pale; there was no pigmentation of the cheek or skin. Secondary sexual characteristics were well preserved. Her blood pressure was 110/70 mm Hg both recumbent and standing. Systemic examination did not elicit any abnormality. Investigation showed: plasma urea concentration 8 mmol/l (48 mg/100 ml)/normal range 4-7 mmol/l; 24-42 mg/100 ml), sodium 137 mmol/l (138-145 mmol/l), potassium 4-4 mmol/l (3-6-4-8 mmol/l), and glucose 5-1 mmol/l (92 mg/100 ml). Blood was taken for estimation of serum concentrations of thyroxine and thyroid stimulating hormone, and an adrenal stimulation test was performed using tetraacacetin 0-25 mg intramuscularly.

During the next two weeks she felt increasingly unwell and restarted taking thyroxine 100 μg daily of her own accord. Two days later she was admitted to hospital as an emergency. Her pulse rate was 100 beats/min and her blood pressure 90/60 mm Hg. Plasma urea concentration had increased to 10-5 mmol/l (63 mg/100 ml) and potassium to 5-1 mmol/l while sodium

In the absence of such studies our policy in Oxford is to immunise preterm babies from 3 months postnatally. This policy is in line with the Red Book recommendations and also with the views of the BPA/ICNI liaison group; we recognise that it is based on limited evidence.—DAVID ISAACS

References

1 The Joint Committee on Vaccination and Immunisation for the Secretary of State for Social Services, the Secretary of State for Scotland, and the Secretary of State for Wales. Immunisation against infectious disease. London: HMSO, 1984.

An unfavourable response to treatment with thyroxine in cases of unequivocal hypothyroidism may reflect the coexistence of another, underlying condition.
A 65 year old man who had a wartime head wound that resulted in herniation in his left temporal lobe and left homonymous hemianopia had been having short (one to three minute) episodes accompanied by a loss of power in the right hand. What might be the cause of these episodes and what treatment is advised?

The episodes are likely to be either seizures or transient ischaemic attacks (which are difficult to differentiate clinically). Transient ischaemic attacks are defined as acute disturbances of focal neurological function of vascular origin lasting less than 24 hours. A useful diagnostic rule of thumb is to ask yourself whether, if the symptoms had persisted rather than resolved, you would have diagnosed a stroke. If the answer is yes, as I think would be the case here, the episodes may be regarded as transient ischaemic attacks.

Unfortunately, clinical medicine is not an exact subject and this diagnosis will be wrong in 1-5% of cases, the correct diagnosis being that of a focal seizure. This has led some to suggest that all patients suspected of having transient ischaemic attacks should have a computed tomography scan, a view with which I do not agree as it is totally impractical. Nevertheless, in this patient, who has suffered a penetrating head injury, the risk of epilepsy is quite high and it would be wise to arrange for an EEG and computed tomography scan to be performed. If either of these investigations support a diagnosis of epilepsy then anticonvulsant treatment should be started and the patient should be given the usual advice regarding driving, etc. If these investigations are unhelpful I would favour a diagnosis of transient ischaemic attacks. The management of these varies considerably depending as much on personal inclination as on facts. The major difficulty is that controlled studies have failed to provide definite evidence that ant platelet drugs, anti coagulants, or different forms of vascular surgery appreciably reduce the risk of stroke in patients who have suffered transient ischaemic attacks. Accordingly, in the man described I would treat any risk factors such as hypertension and until the results of the United Kingdom transient ischaemic attack aspirin study are available I would give him 300 mg of soluble aspirin daily. -N E F CARTLIDGE, consultant neurologist and senior lecturer in neurology, Newcastle upon Tyne.

What treatment or care is advised for a woman with an atomic bladder resulting from demyelinating disease?

An atomic, anticholinergic bladder is relatively uncommon in women with multiple sclerosis. Irritative bladder symptoms such as frequency, urgency, or urge incontinence are five times more common. If voiding of an anticholinergic bladder can be achieved by abdominal straining treatment is only indicated for secondary complications. Thus if there is no active infection and the bladder pressure is low a large residual urine is acceptable. When treatment is required, intermittent self catheterisation is the method of choice. Irritative symptoms may be helped at every stage by quite low doses of probanthine, dicyclomine, or imipramine, but oxybutynin may be necessary in severe spasticity. Severely disabled patients may require an indwelling urethral catheter despite the risks of recurrent infection, formation of stones, or damage to the urethra. A small catheter with a small balloon should be used to reduce the risks to a minimum. Chronic leakage around the catheter or detrusor hyper reflexia may respond to phenol injection of the pelvic nerve plexus behind the bladder base. The most practical alternative to a permanent urethral catheter is a suprapubic catheter with formal closure of the urethra. Urinary diversion into an ileal conduit requires emptying and manipulation by the patient who may lose finger control as the disease progresses. Spinal stimulation through epidural electrodes has not as yet realised its potential. -N E WATSON, consultant urologist, Preston.