Features occasionally present in PHP and PPHP

- endocrinopathies: diabetes mellitus, hypothyroidism
- delayed puberty: hypomenorrhoea, diabetes insipidus, low 17-ketosteroid excretion
- genu valgum
- radius curvus
- blue sclerae
- gonadal dysgensis

Box 2

of parathyroid hormone), and renal resistance with conserved skeletal receptivity. Thus a spectrum of disease can be envisioned.[6] A working classification of the different forms of PHP is given in the table.

PHP and PPHP are treated by vitamin D and calcium supplementation. Early treatment may correct altered calcium and phosphorus levels and diminish the skeletal effects of osteitis fibrosa.

Final diagnosis

Pseudohypoparathyroidism with Albright's hereditary osteodystrophy.

Keywords: Albright's hereditary osteodystrophy; hypocalcaemia; parathyroid hormone; pseudohypoparathyroidism.

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An overweight woman with galactorrhoea

WS Chow, KSL Lam

A 32-year-old Filipino maid was referred to our Endocrine Clinic in Queen Mary Hospital because of galactorrhoea and hyperprolactinaemia. She had regular menstruation, but had developed persistent galactorrhoea after her last delivery 10 years ago. Medical consultation was sought because of increased galactorrhoea in recent months.

From September 1995 to April 1996, her body weight decreased from 55 kg to 49.5 kg while she put herself on Triac (3,5,3'-triiodothyroacetic acid) 1.05 mg daily. She stopped the drug one month before her first visit to the Endocrine Clinic. Physical examination confirmed the presence of galactorrhoea, but visual field and fundoscopy were normal. Her pulse was 90 beats/min and hands were warm. There was no palpable goitre or delayed relaxation of tendon reflexes.

In May 1996, investigations revealed hyperprolactinaemia with prolactin levels of 1610 and 1440 mIU/l (normal range, <500 mIU/l) on two occasions. Thyroid function tests showed low free thyroxine (3 pmol/l; normal 10–19 pmol/l) and thyroid-stimulating hormone (TSH) (<0.03 mIU/l; normal 0.35–5.5 mIU/l) levels. Other anterior pituitary hormones were normal.

A thyrotropin-releasing hormone (TRH) test was done in August 1996, four months after drug cessation (table). By this time, free thyroxine and TSH had increased to 9 pmol/l and 1.9 mIU/l, respectively, and her prolactin level had decreased to 840 mIU/l, although galactorrhoea was still present. Magnetic resonance imaging of the pituitary revealed no abnormality.

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Table TRH test (August 96)

	0 min	*20 min*	*60 min*
TSH (mIU/l)	1.9	16	12
Free T4 (pmol/l)	9.0	9.0	9.0

Questions

1 What are the common causes of hyperprolactinaemia?
2 How do you differentiate between primary and secondary hypothyroidism?
3 What was the underlying diagnosis?

Answers

QUESTION 1

The causes of hyperprolactinaemia are shown in the box.

QUESTION 2

In the presence of a low thyroxine level, raised TSH suggests primary hypothyroidism. On the other hand, a low or normal TSH level will suggest secondary hypothyroidism which can be confirmed with the TRH test. In patients with TSH or TRH deficiency, the TSH response to TRH will be subnormal, delayed, or both.

QUESTION 3

This patient had long-standing idiopathic hyperprolactinaemia aggravated by secondary hypothyroidism induced by Triac.

Causes of hyperprolactinaemia

Physiological
- pregnancy
- lactation

Pathological
- prolactinoma
- lesions (eg, tumours) involving the hypothalamus or pituitary stalk
- drugs, eg, oral contraceptives, major tranquillisers, α-methyldopa, reserpine, tricyclic antidepressants, cimetidine and verapamil
- idiopathic

Other
- hypothyroidism
- renal failure
- cirrhosis
- chest injury

Box

Discussion

The metabolism of triiodothyronine (T3) occurs by deiodination, conjugation, and side-chain alteration. Alteration of the alanine side-chain results in the production of 3,5,3'-triiodothyroacetic acid (Triac). Normal circulating concentrations of Triac are around 8.7 ng/100 ml.[1] On the basis of the free hormone concentration, Triac is more potent than T3 or thyroxine in suppressing basal and TRH-stimulated TSH release in animal studies.[2] Although numerous studies have shown that thyroid hormones act by altering transcriptional activity, the initial decrease in TSH secretion in response to Triac is independent of changes in TSH mRNA transcripts.[3] Triac probably decreases TSH secretion by influencing the secretory mechanism rather than via actions on gene transcription.

Triac has been demonstrated to have a preferential action on TSH suppression, the resultant low TSH returning to baseline levels three weeks after stopping the drug.[4] In this patient, both basal and TRH-stimulated TSH levels had returned to normal four months after drug cessation. Bracco *et al* suggested that Triac is sufficient by itself to maintain a normal metabolic rate while fully suppressing TSH secretion and endogenous thyroxine secretion. Owing to this preferential action, Triac has been used as a TSH suppressor in the treatment of thyroxine resistance syndrome and thyroid malignancy after operation.

Although the response of prolactin to TRH declines significantly after Triac treatment, the basal level has been found to be significantly elevated compared to controls.[5] . Whether or not this is related to an increase in TRH secretion secondary to the suppressed TSH levels is not known.

In conclusion, self-medication of Triac for weight reduction, which is not uncommon, should be excluded in overweight patients with biochemical secondary hypothyroidism.

Final diagnosis

Long-standing idiopathic hyperprolactinaemia aggravated by secondary hypothyroidism induced by Triac (3,5,3'-triiodothyroacetic acid).

Keywords: Triac; thyroxine; thyroid-stimulating hormone; hyperprolactinaemia

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