It has long been known that most recurrent clinical episodes occur in the first three to four weeks, and it was believed, before the effects of spasm were appreciated and often without evidence of repeat lumbar puncture, that they were always due to fresh haemorrhage, sometimes perhaps small but regarded as a sign of increasing urgency; it seems, however, that rapidly recurring small haemorrhages, or leaks, are uncommon and would not produce cerebral signs. Very early operation to forestall recurrent haemorrhage has a higher mortality than later operations due partly to the greater risk of operation haemorrhage and partly to angiography. Conservative treatment, on the other hand, does not prevent deaths from recurrent haemorrhage. We believe that better overall results can be obtained by relying less on angiography and more on clinical observation and selection before investigation, in spite of possible fatal haemorrhage while waiting for spasm to subside. This entails recognizing the small minority of very severe cases, either with large haematoma requiring immediate aspiration or with signs of severe spasm necessitating delaying angiography up to even several weeks. Most patients not in coma who are improving and have moderate signs due either to medium-size haematoma or to less severe spasm that often cannot be differentiated clinically can have careful angiography after about a week before recurrent haemorrhage becomes more frequent. Perhaps the overriding consideration in the management of subarachnoid haemorrhage is referral immediately after the initial, usually mild haemorrhage.

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Hospital Topics

Thyroxine "Addicts"

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Summary

Four patients are described who were surreptitiously taking thyroxine-containing tablets. In two cases this caused considerable diagnostic problems. The diagnosis should be considered in patients with clinical features of hyperthyroidism (but without goitres or proptosis), who appear psychiatrically abnormal and whose thyroid function tests show conflicting results.

Introduction

Difficult diagnostic problems may be posed by patients who take unusual medications without telling their doctor. This report draws attention to one such instance—namely, the development of apparent thyrotoxicosis due to surreptitious self-administration of thyroxine. Four patients attended the Middlesex Hospital in the course of a 14-month period with clinical features suggestive of hyperthyroidism, and a combination of thyroid function tests indicated that all were taking exogenous thyroxine.

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Case Reports

Case 1.—A 51-year-old woman was referred to the outpatient clinic with a number of complaints, of which malaise and depression were the most prominent. There was a history of psychiatric disturbances for at least 15 years, unrelieved by electrical treatment. She had previously been investigated at at least two other London teaching hospitals and variously believed to have an overactive thyroid gland (unsubstantiated by tests) and hypopituitarism. She denied taking any medication apart from chlordiazepoxide (Librium). She was hyperkinetic and had a pulse rate of 120/min, but no goitre or eye signs of thyrotoxicosis were present. Thyroid function tests showed a grossly raised serum thyroxine level (269 ng/ml) and a paradoxically low thyroid uptake of radioiodine (3% at 24 hours) (see table). On her second visit she was confronted and eventually admitted to taking "antidepressant" tablets from a private doctor, who was treating her without the knowledge of her own general practitioner.

Case 2.—A 21-year-old student nurse presented with loss of weight and grossly hysterical behaviour. She was hyperkinetic, with a resting pulse rate of 100/min, but no goitre or eye signs or thyrotoxicosis were present. She denied taking any medication. Serum thyroxine was 176 ng/ml and there was virtually no thyroid uptake of radioiodine (4% at 24 hours). When confronted, she admitted to taking tablets, prescribed by the same doctor as in case 1, as a combined medication for slimming and depression. She later had a major psychiatric episode requiring inpatient treatment at a mental hospital.

Case 3.—A 25-year-old woman was referred by her general practitioner for investigation of weight loss. There was a long history of various social problems and abnormal eating habits, and at the age of 14 she had been thought to have anorexia nervosa, with refusal of all fattening foods and loss of 3 st. (19 kg) in weight. She had also been treated as an inpatient at a mental

hospital on another occasion, after admission on an order. She denied taking any tablets. She was hyperkinetic, with a resting pulse rate of 140/min and lid retraction and lag. The thyroid gland was impalpable. Serum thyroxine was 229 ng/ml and the ^{131}I uptake 3% at 24 hours. She was admitted to hospital. Although she persistently denied all medication, tablets were found in her bag. When confronted she said that these were combined slimming and pep pills belonging to her mother. Their ultimate source was never determined. but a limited analysis showed that they contained phenolphthalein and 0·1% iodine by weight.

Findings in the Four Thyroxine "Addicts"

Case No.	Age and Sex	Serum Thyroxine (ng/ml)	24-hour Uptake of Radioiodine	Source
1	51 F.	269	3%	Thyroxine/amphetamine tablets from private doctor. (Denied taking any tablets until confronted)
2	21 F.	176	4%	Same source as case 1. (Denied taking any tablets until confronted)
3	25 F.	229	3%	Never admitted to taking any medication but tablets found in handbag while an inpatient
4	47 F.	98*	6%	Admitted taking at least two different thyroxine-containing "slimming" drugs from doctors abroad

*Two weeks after stopping tablets.

Case 4.—A 47-year-old woman presented with obesity, malaise, and alternating energy and apathy. She had had a great many medical examinations in the past and admitted taking various "slimming" preparations obtained from doctors abroad. These included a diuretic, thyroid tablets U.S.P. 300 mg, one daily, and one or more tablets of a preparation (Tre-o-tabs) containing thyroid U.S.P. (60 mg, amphetamine 10 mg, and atropine 1/360 gr. (0·18 mg.). Two weeks after stopping the tablets serum thyroxine was 98 ng/ml, with a 24-hour ^{131}I uptake of 6%, which rose to 28% after administration of 10 I/U of thyrotropin.

Discussion

A patient taking excessive amounts of thyroid hormones and concealing this from her doctors can pose a difficult diagnostic problem. A combination of thyroid function tests is essential to make the diagnosis with confidence, but awareness of the possibility of self-medication is also important. In the four patients described here the characteristic combination of a high serum thyroxine with a very low neck uptake of radioiodine could have been due to only a few possibilities. Probably the most likely is thyrotoxicosis in combination with excessive iodine ingestion sufficient to "swamp" the thyroid uptake of radioiodine, which was found in about 4% of a recent series of thyrotoxic patients.[1] A history of iodine ingestion or recent exposure to *x*-rays with iodine-containing material, an appreciable discrepancy between the levels of serum thyroxine and protein-bound iodine (P.B.I.), or the presence of a greatly increased urinary iodide excretion should make the diagnosis clear. Measurement of the serum thyroxine level is preferable to measurement of the P.B.I. alone, as the combination of a raised P.B.I. and a low thyroid uptake of radioiodine is a frequent finding in euthyroid subjects who have received excess iodine from exogenous sources.[2 3]

A second and much less likely possibility is that of thyrotoxicosis arising in an aberrant thyroid gland. In this uncommon event the thyroid gland may be retrosternal or lingual, and its uptake of radioiodine may not be detected by counting over the neck alone. The diagnosis in these circumstances may be made by scanning for radioiodine over areas outside the neck. One further possibility should be considered. Acute thyroiditis (de Quervain's thyroiditis), by causing a damaged thyroid gland to release large amounts of thyroxine into the blood, may result in the combination of a raised serum thyroxine with a low thyroid uptake of radioiodine. This condition is usually associated with a tender swelling of the thyroid gland of relatively acute onset, so should not cause diagnostic difficulty. If the low neck uptake of radioiodine is not due to either excess circulating iodide, an aberrant thyroid gland or de Quervain's thyroiditis (or unlikely technical factors such as vomiting of the test dose of radioiodine or the development of a fault in the counting equipment) then the high serum thyroxine must be exogenous.

Self-medication with thyroxine has been reported previously from France[4] and the United States,[5 6] and most of the patients described have shown psychiatric abnormalities, often severe and requiring definitive treatment. Bricaire *et al.*,[4] when describing four such patients, reported that they often showed emotional immaturity and dependency, with a strong aggressive emotional claim on their mothers or a mother substitute. Of the 11 cases described in these three previous reports and the four cases described here all but one were women. The four patients in the present series all showed evidence of psychiatric abnormality, and two had required compulsory inpatient psychiatric treatment. Similar findings have been noted among purgative addicts and among surreptitious takers of other drugs, such as diuretics[7] or anticoagulants.[8]

Self-administration of thyroid hormones should be considered in patients with clinical features of hyperthyroidism (but without goitres or proptosis) who appear psychiatrically abnormal and whose thyroid function tests show conflicting results.

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