T₃ toxicosis presented by depression in an elderly woman

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Summary
An elderly patient with thyrotoxicosis complicated by lack of classical clinical signs and normal conventional tests is described. The picture was compounded by development of apathy and depression. This is the second case report of apathetic T₃ thyrotoxicosis in the literature. Such a clinical entity can easily be overlooked, but represents one of the treatable disorders of 'old age depression'.

Introduction
Although a number of standard text books and reference sources indicate that thyrotoxicosis can be present with apathy, depression, and hypokinesis, the literature on apathetic T₃ thyrotoxicosis is limited to a single case report (Fairclough and Besser, 1979). The apathetic form of thyrotoxicosis, which is lacking classic clinical manifestations such as hyperkinesis, anxiety, tremor, but presenting with lethargy, mental confusion or depression, is even more difficult to diagnose when results of conventional thyroid tests are normal.

Report of a case
A 78-year-old white female was admitted to the Jewish Institute for Geriatric Care at Long Island-Hillside Medical Center, New York, in 1974. She entered for life-long nursing care with diagnoses of diabetes mellitus (20 years), ischaemic heart disease and myocardial infarction, thorazine-induced hepatotoxicity (for 19 years before admission), hysterectomy (4 years before admission), and cataract removal (2 years before admission). Two years before admission, the patient began having difficulty taking care of herself and encountered problems controlling the diabetes. Since the family could not support the patient in the community, she was committed to the Institute.

On admission the patient was alert, orientated, co-operative, and well nourished; BP was 110/64 mmHg, pulse was 84/min. She was afebrile. Pupils showed no abnormalities. No signs of ptosis or stare were noted. The thyroid was not palpable; there was no lymphadenopathy. The lungs were clear. Heart sounds were normal, rhythm regular, no murmur, and no gallop. The abdomen was soft with multiple surgical scars; no masses or organo-megaly were found. All peripheral pulses were palpable. No local neurological deficit was present.

Routine laboratory analyses were normal except for fasting blood sugar level which was 20-5 mmol/l.

The patient was receiving treatment with isophane insulin 26 u. and regular insulin 10 u. every morning, and digoxin 0-125 mg daily.

From 1974 to 1976 the clinical course was uneventful. In August 1976, carcinoma of the transverse colon was discovered and hemicolecctomy performed. Although the immediate postoperative course was uneventful, the general condition of the patient gradually deteriorated over the following months. The patient became progressively depressed and unmotivated. On examination by a psychiatrist at the beginning of 1977, the patient stated her desire to die, to be 'at peace'. The diagnosis of depression was made and treatment with psychotropic medication was considered by the psychiatrist. The treatment, however, was not given because of a sensitivity to thorazine recorded in past medical history. During the following months, uncontrolled diabetes necessitated an increased dose of insulin. Signs of heart failure appeared; the patient was treated with a diuretic and digoxin without significant improvement.

Physical examination at that time revealed no tachycardia, no thyrotoxic eye signs, no tremor of hands or tongue, no goitre or palpable nodules in the neck, and no hyperkinetic deep tendon reflexes. Nevertheless, in view of her progressive depression and uncontrolled diabetes, thyroid function tests were requested. Serum thyroxine (T₄) concentration (assessed by the method of Murphy-Pathee) was 170-3 nmol/l (normal, 58–187), T₃ resin uptake 34% (normal, 25–35%), serum triiodothyronine concentrations (radio immunoassay) was 384 (normal, 110–230), 24-hr radioactive iodine uptake was 41% (normal 25–35%). A thyroid scan showed a uniformly enlarged goitre. The diagnosis of T₃ thyrotoxicosis was made and treatment with methimazole was started (5 mg/8 hr).

With this treatment, heart failure and diabetes...
were controlled. The insulin dose was decreased to 25 u. isophane insulin/day. The patient became more alert and signs of depression improved. She was involved in social activity and discussions with other patients, and expressed her desire to meet friends and visitors, etc. After several weeks, the patient suddenly developed an acute myocardial infarct and died. Post-mortem findings revealed an acute transeptal and circumferential left ventricular wall infarct, artherosclerotic coronary vessel disease, and thyroid hyperplasia (weight 35 g).

Discussion

In 1931, Lahey called attention to a clinical variety of hyperthyroidism characterized by apathy, withdrawal, and depression rather than the more classical hyperdynamic state. He emphasized that this ‘apathetic’ hyperthyroidism primarily affects older patients. Its clinical picture lacks many of the usual features of thyrotoxicosis such as palpable goitre, exophthalmos, tachycardia, and smooth, warm skin. Although these patients do not appear extremely ill, the disease is far from mild. Under stress the patients may ‘quietly and peacefully sink into coma and die an absolutely relaxed death without activation’, unlike the classical thyrotoxic patient who may go into crisis (Lahey, 1931).

Further reports and a description of apathetic thyrotoxicosis as a distinctive clinical and laboratory entity have since appeared in the literature (Thomas, Mazzaferri and Skilman, 1970). In 1974, the first case of apathetic T₃ toxicosis was reported. This patient was regarded initially as suffering from ‘hypothyroidism’. Only an exaggerated response to a normal replacement dose of thyroxine led to the suspicion and diagnosis of thyrotoxicosis (Fairclough and Besser, 1974). That case and the present one emphasize the difficulty in diagnosing apathetic T₃ hyperthyroidism when the clinical picture and conventional thyroid function tests are deceptive. The low normal T₄ level, as in the present case, coupled with apathy may, in fact, incorrectly suggest hypothyroidism.

The virtual absence of reports on such cases may be a reflection of underdiagnosis rather than a true low prevalence, especially in elderly patients who often have depressive disorders and who have an increasing frequency of apathetic thyrotoxicosis (Davis, 1978). The need for the assessment of the precise incidence of T₃ toxicosis in the elderly and value of a routine assay of serum T₃ has recently been stressed in the Canadian literature (Walfish, 1977). In the present case, the increasing difficulty to control diabetus mellitus and congestive heart failure suggested the possibility of thyrotoxicosis. The high normal serum thyroxine and T₃ resin uptake levels prompted further investigation. The clinical course demonstrated that the patient’s depression was a clinical feature of the apathetic form of thyrotoxicosis and marked improvement of the patient’s mental status was observed with the treatment of thyrotoxicosis. The pathophysiological mechanism leading to a state of apathy and depression in thyrotoxicosis is not known.

Many factors are responsible for an increasing incidence of mental disorders in the elderly. Some of them are preventable or treatable (Butler and Lewis, 1973). The diagnosis of apathetic T₃ toxicosis is difficult in the elderly but it should be considered as one of the treatable causes of ‘old age depression’.

References

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