

Case Report

Acute thyroxine overdose: two cases of parasuicide

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Factitious hyperthyroidism is a well documented clinical entity where the signs and symptoms of hyperthyroidism are self-induced by the chronic ingestion of excessive amounts of thyroxine^{1, 2}. The acute ingestion of large amounts of thyroxine might be thought to carry a risk of acute thyrotoxicosis, but despite high plasma levels of thyroxine this may not be so. Previous reports suggest that such cases are largely asymptomatic³, but the majority of these have been in children⁴⁻⁷. There are few guidelines available for the specific management of acute thyroxine overdose in adults, where the amount ingested is usually relatively larger and treatment is more likely to be delayed. Two cases are presented where large amounts of thyroxine were taken in attempted suicide.

CASE 1. An attention seeking 14 year old girl was admitted to the metabolic unit about 15 hours following an overdose of about 100 mixed tablets, including ferrous sulphate, multivitamins and 0.1mg tablets of thyroxine. Over 3mg of thyroxine had been taken. She had vomited once nine hours after ingestion, and on admission complained of dizziness. No formal gut decontamination (emetics and/or lavage) was performed. She did not exhibit flushing, sweating, tremor or agitation and her temperature was normal. Her pulse was 80/min regular, blood pressure 110/80 mmHg. Repeat measurements of pulse and blood pressure were made at two hourly intervals for the next three days. The maximum pulse rate was 92/min. An electrocardiogram showed sinus rhythm. Her initial dizziness settled within a few hours. Serum free thyroxine (FT₄), total triiodothyronine (TT₃), and TSH were measured as shown (figure 1). She was discharged after three days and followed up for blood monitoring as an outpatient.

CASE 2. A clinically depressed 40 year old woman had been taking 0.2mg thyroxine daily for one year for primary hypothyroidism. She was admitted four hours after taking approximately ninety-five 0.1mg thyroxine tablets and a considerable amount of alcohol. Initially she was drowsy but rousable. Ipecacuanha was given and induced vomiting successfully. She had a fine tremor but there was no flushing, sweating, agitation or pyrexia. A regular tachycardia of 100 beats per min. was recorded which fell to an average of 78 beats per min after 18 hours.

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The initial blood pressure was 150/90 mmHg and fell to 130/80 mmHg after four hours. Serum free thyroxine and TSH levels were measured as shown (figure 1). She remained asymptomatic and after six days was transferred to a psychiatric unit for treatment of her depression, where further serum free thyroxine and TSH measurements were made.

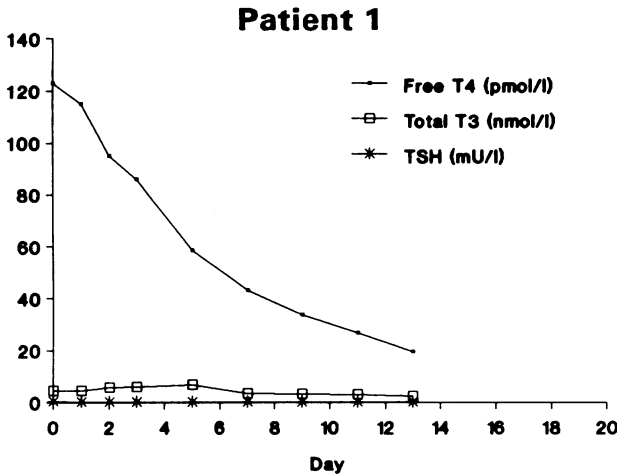
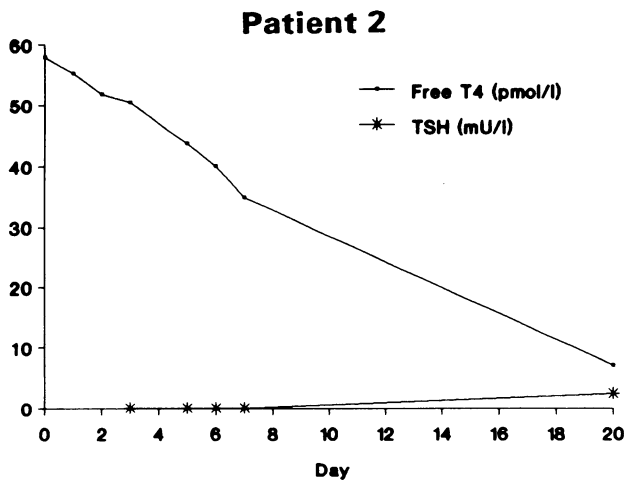


FIGURE 1. Serum free thyroxine (FT₄), total triiodothyronine (TT₃) and TSH levels in patients 1 & 2 following acute thyroxine overdose.



Laboratory reference ranges

Free T4: 10.5 – 25.5 pmol/l
 Total T3: 0.3 – 1.5 nmol/l
 TSH: 0.1 – 4.5 mU/l

DISCUSSION

Most of the reported cases of acute l-thyroxine ingestion have been in children. Although serious sequelae are generally rare, management is still justifiably aggressive when the child is even vaguely symptomatic. A combination of gut decontamination, antithyroid medication and beta blockade are standard measures^{6,7,8}. There are no guidelines for the management of adult cases where the amount consumed is usually relatively larger, the presentation more likely to

be delayed, and the metabolism of the drug slower. Both of our patients had minimal intervention and yet, despite very high levels of serum thyroxine, they were essentially asymptomatic.

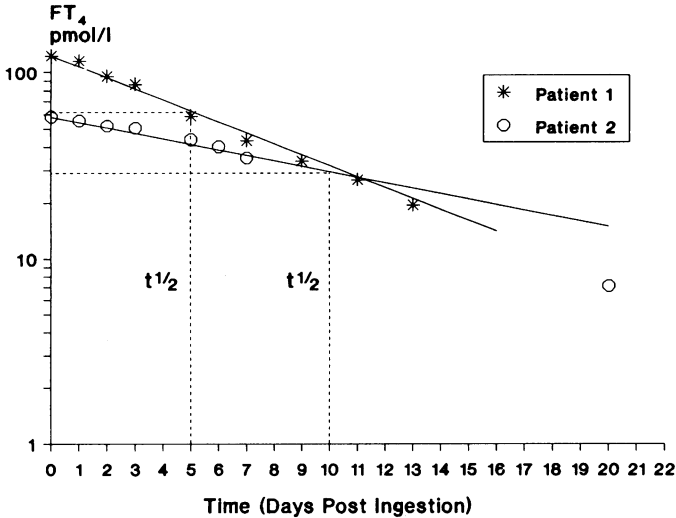


FIGURE 2. Decrease in serum free T₄ following acute thyroxine overdose: (Log scale to allow calculation of T_{1/2} (half life of thyroxine))

Estimated T_{1/2} patient 1: 5 days
 Estimated T_{1/2} patient 2: 10 days

such large doses of T₄ there is greater production of the biologically inactive reverse T₃ (rT₃) which tends to maintain a euthyroid state. This pathway is enhanced by both exogenous and endogenous steroids and in any extrathyroid illness. This should decrease the risk of thyroid storm after T₄ overdose,^{5, 6, 13, 14, 15} and down-regulation of the cell nuclear triiodothyronine receptors might also contribute⁶.

Thyroid storm following a massive ingestion of the hormone in children is thought to reflect high serum free T₄, and free T₃ levels, combined with hepatic and renal impairment. Tissue levels of thyroid hormone would appear to be more significant in adults, as a storm or crisis may develop as late as five days after the insult when serum levels are much reduced¹⁰. A thyroid crisis is characterised by a rising temperature which may rapidly reach a lethal level, tachycardia progressing to atrial flutter or fibrillation, a widened pulse pressure, extreme agitation, flushing, sweating and diarrhoea. The patient can become comatose in under 36 hours if the cause is not identified at a very early stage^{1, 17, 18}.

Only four other cases of acute thyroxine ingestion in adults have been published^{10, 13, 14, 15}. All had formal gut decontamination and all required propranolol for the treatment of tachycardia. None developed a thyroid storm despite T₄ levels ranging from three to 16 times the average level. The cases presented here required no symptomatic treatment at all, despite levels that were seven times higher than normal. As thyroid storm carries a mortality of up to 75%,^{1, 18} prevention is the key to management: although we did not encounter any problems with an expectant approach, these cases would seem to be unusual.

Calculation of the serum thyroxine half lives compared well with standard values of 6-7 days^{9, 10} (Fig 2). The longer estimate in case 2 may be explained in terms of age-related decreased tissue turnover. Serum total T₃ measurement in case 1 illustrates the slow conversion of T₄ to T₃ in the periphery, the peak level of T₃ being reached in five days. Serum TSH levels are predictably suppressed.

Paediatric studies suggest that when the body is presented with

In conclusion, the risk of acute thyroxine overdose in adults is slight, but the patient should be closely monitored for at least five days as an inpatient. Prophylactic propranolol may be of benefit if not otherwise contraindicated. Although the initial measurement of serum thyroxine levels may give a rough indication of the absolute quantity of hormone consumed, our results, and those in the literature suggest that further measurements in adults give a poor prediction of clinical outcome.

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