Acute Levothyroxine Poisoning in a Hypothyroid Patient with Total Thyroidectomy: A Case Report

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ABSTRACT

Levothyroxine intoxication is a rare clinical entity which is usually asymptomatic. However, severe symptoms such as respiratory failure, malignant hyperthermia, seizures, arrhythmia, and coma have been reported. In this case report, a patient who ingested a high dose (10 mg) of levothyroxine for suicide and was admitted to the intensive care unit is presented. Despite ingestion of a high dose of levothyroxine, thyrotoxicosis symptoms resolved without any major treatment and the patient was discharged from the ward on the 7th day

Keywords: Levothyroxine Overdose, Suicidal

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INTRODUCTION

Levothyroxine overdose or poisoning by accidental ingestion may be seen in the paediatric age group but is very rare in the adult population.[1,6] Overdose due to suicidal ideation may be the cause but the appearance of toxicity depends on the ingested dose, disease chronicity and amount of active hormone. Concomitant psychiatric illness and drug interaction with thyroid hormones should always be considered. The common clinical signs following thyroxine overdose can either be limited to tachycardia, agitation, nervousness, insomnia, anxiety, tremor,[2] or severe features, though less likely, like thyroid storm involving cardiac, neurological, respiratory and thermoregulatory centre.[3] Acute levothyroxine ingestion up to a dose of 4 mg/day is usually asymptomatic and well tolerated.[2] Symptoms of thyrotoxicosis are evident in chronic over-dosage cases which is also associated with higher morbidity and mortality.[4] In an established case of thyrotoxic crisis propranolol, glucocorticoids, thioamides, iodine compounds, bile acid sequestrants, and in some cases plasmapheresis is also opted[5] along with other supportive measures.

We report a case of acute levothyroxine ingestion of 10mg (10000 mcg) maximum dose reported in the literature.

CASE HISTORY

A 27-year-old married female was admitted to the Emergency Room after consuming 100 tablets of 100 mcg of Levothyroxine (total of 10 mg) with suicidal intention. She took the tablets with video selfie recording at about 10.05 am and the video recording was sent to her husband at 10.20 am. The husband brought her to the hospital at about 10.45 am, 40 minutes after consumption. She gave the history of total thyroidectomy for papillary carcinoma in 2015. Since then she was on replacement cum suppression therapy with 150 mcg of Levothyroxine with recent TSH being 0.05 Micron IU. No h/o any mental disorders in the past except for some marital issues and she had a verbal argument on that day with her husband following which she took the medication.

On arrival at the Emergency Department, she was alert, her GCS was 14/15 (E4M6V4). Vitals were stable with a pulse of 84/min, regular, BP- 120/90 mm of Hg, Respiratory rate of 20/min, a temperature of 98.2°F and SpO2 of 98% in room air; she was hemodynamically

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Table 1. Thyroid profile

Empty cell	6 hours	24 hours	48 hours	96 hours	15 days	25 days	Ref. range
Free T4	>6.99	8.0	10.3	3.03	1.53	0.67	1.2-4.1 pg./ml
Free T3	26	-	-	10.2	-	-	8.9-17.1 pg./ml
TSH (Thyroid stimulating hormone)	<0.22	<0.095	<0.015	0.19	0.031	1.58	0.3-4.5 pg./ml
TG (Thyroglobulin)		1.6 ng/ml					1.4-to 29.2 ng/ml
Anti TG		1.2 iu/ml					0.1to 10

stable, with no palpitations, toxic symptoms or thyroid storm. She was given an effective stomach wash, shifted to CCU and vital signs monitored. She received adequate fluids, Setalin, Propranolol, Carbimazole, Steroids and Clonazepam. ECG was normal with no QTc abnormality. Baseline blood chemistry including ABG was normal. Daily FT4, TSH, and Electrolyte tests were done for 5 days and after that advised every week for 4 weeks. Serial FT4 values are- > 6.99, 8, 10.3, 3.03 and TSH values are <0.22, <0.95, <0.015, 0.19. **(Table 1).** Her Thyroglobulin and Anti-Thyroglobulin antibodies are normal. She was advised to restart Levothyroxine after 20 days and once TSH was stabilized. During her stay for one week in hospital she was asymptomatic except for fever and loose stools on the 2nd day. She was given psychiatric consultation and counselling. She was discharged with advice to follow up weekly for 4 weeks.

DISCUSSION

The clinical reflection of massive L-T4 ingestion in adult patients can present with a wide range of symptoms. Intoxication symptoms can be minimal or may present with severe symptoms such as dysrhythmia, respiratory failure, myocardial infarction, hemiparesis, hyperthermia and coma.[8] Binimelis et al reported 6 cases of thyrotoxicosis after 7-10 mg of L-T4 ingestion. Five of these cases were comatose and one case presented with stupor. There was left ventricular failure in two cases and arrhythmia in three cases.[9] The biologically active part of thyroid hormones is T3. Symptoms in thyrotoxicosis become apparent as T4 is transformed into T3. Therefore, patients may be asymptomatic during the first 24 hours.[5] Also, symptoms may last or worsen for 11 days because of the long half-life (7 days) of levothyroxine.[8] Our case presented with no thyrotoxicosis. Symptoms can occur when ingested in massive doses.[8] Some drugs like Olanzapine and Citalopram hydrobromide have cardiotoxic and neurotoxic side effects;[10,11] Nygaard et al reported that there is no relation between the severity of symptoms and ingested dose of levothyroxine intoxication.[7] Although there is no consensus regarding treatment; first of all, patients should be closely monitored. Gastric lavage and active carbon application should be performed to prevent gastrointestinal absorption. Cholestyramine binds to thyroxin and increases elimination via decreasing systemic absorption. Beta-blockers (propranolol 1-2 mg/kg/d) decrease sympathetic hyperactivity and control tachycardia. Propylthiouracil (5-7 mg/kg/d) decreases transformation of fT4 to fT3. Glucocorticoids and sodium iodate also decrease the transformation of fT4 to fT3 and may be used in combination with beta-blockers in patients with severe symptoms.[11,12] Haemodialysis is minimally effective since fT3 and fT4 are highly bound to serum proteins.[12] Hemoperfusion and therapeutic plasma exchange decrease fT4 levels.[8,13]

CONCLUSION AND LEARNING POINTS

A case of acute massive Levothyroxine overdose is presented for its rarity.

1. A "thyrotoxic storm" is never an early feature of acute thyroxine ingestion, even if the dose is massive. This ingestion is not life-threatening.
2. Chronic thyroxine excess is more likely to cause clinically important thyrotoxicosis and is particularly concerning in the elderly and those with cardio-respiratory comorbidities.
3. most patients remain asymptomatic following thyroxine overdose
4. symptoms may occur if >10mg thyroxine is ingested
5. if symptoms do develop, they usually do not arise until after 48 hours post-ingestion, (although thyroxine is rapidly absorbed, it requires peripheral conversion into its more active form (T3 or tri-iodothyronine) and its clinical effects require changes in gene expression)
6. symptoms (when present) may last up to 3 weeks.
7. clinical manifestations of thyroxine overdose are largely sympathomimetic and result from adrenergic stimulation – they include: agitation, sweating, tachycardia, hypertension, headache, diarrhoea and vomiting.

END NOTE

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