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THYROID STORM OR STORM BREWING? A CASE OF LEVOTHYROXINE OVERDOSE WITH DELAYED PRESENTATION

General Medicine

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ABSTRACT

Levothyroxine overdose in adults is a rare but potentially serious condition, often linked to intentional ingestion in individuals with psychiatric illness[1]. Clinical manifestations range from asymptomatic cases to thyrotoxicosis, characterized by tachycardia, palpitations, agitation, and, in severe cases, seizures, arrhythmias, or thyroid storm[12]. Symptoms may be delayed due to peripheral conversion of T4 to T3[1]. Systemic involvement includes cardiovascular, neurological, and hepatic effects[2]. Emergency management involves gastric decontamination with activated charcoal, beta-blockers like propranolol to control sympathetic activity and inhibit T4-to-T3 conversion, and corticosteroids or antithyroid drugs in severe cases[12]. Cholestyramine may enhance hormone clearance; extracorporeal removal is rarely effective[12]. Continuous monitoring of vitals and thyroid function is crucial for at least 72 hours, with follow-up over two weeks or more until normalization[12]. Psychiatric assessment is essential in intentional cases[3]. With prompt and appropriate care, prognosis is generally favorable despite high hormone levels[12].

KEYWORDS

Levothyroxine poisoning, Thyroid storm, Thyroid hormone overdose.

INTRODUCTION

Levothyroxine, a synthetic form of the thyroid hormone thyroxine (T4), is commonly prescribed for the treatment of hypothyroidism and certain types of goiters[1]. It functions as a metabolic regulator, primarily through conversion to its active form, triiodothyronine (T3), which influences gene expression and modulates protein synthesis across nearly all tissues[12]. Clinically, it supports growth, thermoregulation, cardiovascular output, gastrointestinal motility, and neurological function[2].

While therapeutic in appropriate doses, excessive intake of levothyroxine can lead to thyrotoxicosis, a potentially life-threatening condition[12]. The estimated toxic dose varies, but ingestion of more than 0.3–0.5 mg/kg is considered high risk in adults[2]. Lethal doses are rarely defined due to individual variability in hormone metabolism, but doses above 3–5 mg in adults may produce significant toxicity, particularly in the presence of comorbid conditions[23]. Overdose may occur accidentally, due to dosing errors, or intentionally, particularly in patients with underlying psychiatric disorders[3].

Toxicity arises from excessive circulating T4, which undergoes peripheral conversion to T3, leading to heightened sympathetic activity[1]. Importantly, due to this delayed conversion, the onset of clinical symptoms can be latent, typically emerging 24–48 hours post-ingestion[12]. This delayed presentation complicates early diagnosis and management[2].

Clinical features range from mild palpitations, anxiety, tremor, and tachycardia to severe systemic manifestations such as arrhythmias, hyperthermia, convulsions, respiratory failure, myocardial infarction, and thyroid storm[12]. The most life-threatening complications are cardiovascular—particularly atrial fibrillation and ventricular tachyarrhythmias—as well as CNS events like seizures or coma[12].

Initial emergency management includes gastrointestinal decontamination with activated charcoal if presented early, and symptomatic control using beta-blockers, particularly propranolol, which not only attenuates adrenergic symptoms but also inhibits peripheral T4-to-T3 conversion[12]. In more severe cases, corticosteroids (e.g., hydrocortisone or dexamethasone) and antithyroid agents such as propylthiouracil or carbimazole may be employed[12]. Cholestyramine, a bile acid sequestrant, can also aid in hormone elimination[2]. Extracorporeal removal strategies like hemodialysis are largely ineffective due to high protein binding of thyroid hormones, though therapeutic plasma exchange has been tried in select cases[4].

Given the delayed onset and prolonged half-life of T4 (approximately 7 days), patients should be monitored in-hospital for at least 72 hours, with outpatient follow-up continuing for up to two weeks or until normalization of thyroid function tests[12]. Continuous monitoring of cardiac and neurological status is crucial to prevent delayed complications[12].

CASE REPORT

A 26-year-old female presented to the emergency department approximately twelve hours after the intentional ingestion of 75 tablets of levothyroxine sodium which was prescribed to her mother , each containing 100 mcg, totaling 7.5 mg. She had no known comorbidities, psychiatric history, or prior thyroid disorders. On arrival, she had complaints of giddiness with no symptoms of palpitaion, sweating, tremors , tinnitus or anxiety

General Examination:

Patient was conscious, coherent, answering questions well, moderately built and nourished, no pallor, no icterus, no cyanosis, no clubbing, no generalized lymphadenopathy and no pedal edema.

Vitals blood pressure 110/70 mmHg, heart rate 126bpm, respiratory rate 18 breaths per minute, SpO_2 98% on room air, and afebrile. Arterial blood gas analysis revealed normal acid-base status.

Systemic examination: Cardiovascular and respiratory systems were normal except tachycardia. There was no rigidity of abdomen and there was no organomegaly. CNS examination was normal at the time of presentation. there were no signs of thyrotoxicosis.

Investigation

Following ingestion of 7.5 mg levothyroxine, the patient's TSH remained suppressed (<0.01 µIU/mL) until day 7, normalizing by day 15. FT4 declined from 28 to 6.3 ng/ml, and FT3 from 5.3 to 2.8 pg/mL over the same period. ECG showed sinus tachycardia initially, normalizing by day 5. Temperature peaked at 100.6°F on day 1 and returned to baseline by day 15. This pattern reflects delayed T3 conversion and gradual hormonal clearance, supporting conservative management with close clinical and biochemical monitoring.

Laboratory Findings

Table 1. Serial Monitoring Of Parameters

Days after ingestion	Day 1	Day 3	Day 5	Day 7	Day 15	Normal Range
TSH (µIU/mL)	<0.01	<0.01	<0.01	<0.01	0.035	0.4 to 4.0 mIU/L
FT4 (ng/ml)	28.2	20.7	16.9	10.5	6.3	0.93 to 1.7 ng/dL
FT3 (pg/dl)	5.31	4.92	3.2	2.8	1.9	1.4-4.2pg/ml
ECG	Tachy	Tachy	Tachy	NSR	NSR	
Temperature (F)	100.6	99.6	99.2	99	98.2	97.5°F to 98.6°F

Table 2 : Burch-Wartofsky Scoring System

Criteria	Points
Thermoregulatory dysfunction	
Temperature (°C)	
37.2–37.7	5
37.8–38.3	10
38.4–38.8	15
38.9–39.4	20
39.4–39.9	25
≥ 40.0	30
Cardiovascular	
Tachycardia (beats per minute)	
100–109	5
110–119	10
120–129	15
13–139	20
≥ 140	25
Atrial fibrillation	
Absent	0
Present	10
Congestive heart failure	
Absent	0
Mild	5
Moderate	10
Severe	20
Gastrointestinal-hepatic dysfunction	
Manifestation	
Absent	0
Moderate (diarrhea, abdominal pain, nausea/vomiting)	10
Severe (jaundice)	15
Central nervous system disturbance	
Manifestation	
Absent	0
Mild (agitation)	10
Moderate (delirium, psychosis, extreme lethargy)	20
Severe (seizure, come)	30
Precipitating event	
Status	
Absent	0
Present	10
Total score	
> 45	Thyroid storm
25–45	Impending storm
< 25	Storm unlikely

Following ingestion of 7.5 mg levothyroxine, the patient exhibited signs of acute thyrotoxicosis including sinus tachycardia, low-grade fever (100.6°F), suppressed TSH (<0.01 µIU/mL), and elevated FT4 and FT3 levels. Gastric lavage was promptly performed, followed by activated charcoal to reduce gastrointestinal absorption. Propranolol (20 mg orally every 6–8 hours) was initiated to control adrenergic symptoms and inhibit peripheral T4-to-T3 conversion. Hydrocortisone (100 mg IV every 8 hours) was administered to further block T3 conversion and support potential adrenal suppression. Propylthiouracil (200 mg orally every 6 hours) was added to inhibit thyroid hormone synthesis and peripheral conversion. Cholestyramine (4 g orally every 6 hours) was given to enhance fecal excretion of thyroid hormones via interruption of enterohepatic circulation. Continuous ECG and vitals monitoring was done, with normalization observed by day 15.

DISCUSSION

Levothyroxine (T4) overdose remains an uncommon but clinically important toxicological event, especially in adults where it is more likely to be intentional[1]. While accidental ingestion is more typical in children, adult overdoses—often linked to psychiatric illnesses or weight loss attempts—are rarely reported, which contributes to the limited literature guiding their clinical management[2]. Levothyroxine (T4) is a prohormone requiring peripheral conversion to triiodothyronine (T3) for biological activity[1]. This conversion, coupled with T4's long half-life (approximately 7 days), contributes to the delayed onset of symptoms—ranging from 12 hours up to 11 days post-ingestion[1]. Toxic manifestations vary from mild (anxiety, palpitations, tremor) to life-threatening complications such as thyroid storm, arrhythmias, hyperthermia, coma, and seizures[1].

Thyroid storm, the most severe form of thyrotoxicosis, can occur days after overdose and requires prompt diagnosis using criteria like the Burch-Wartofsky scoring system. A score above 45 is highly suggestive of thyroid storm, often necessitating intensive care support[4].

Thyroxine overdose leads to a hypermetabolic state resembling thyrotoxicosis[2]. However, due to the pharmacokinetics of levothyroxine, including its slow gastrointestinal absorption, high protein binding (>99%), and delayed peripheral conversion to T3—the biologically active form—initial presentations may be deceptively asymptomatic[1]. Tmax of levothyroxine is typically 5–6 hours, with a half-life of 7 days for T4 and up to 6 days for T3 in overdose states[1]. This explains why symptoms may be delayed and persist for days to weeks[2].

Clinically, patients may exhibit signs of sympathetic overactivity including palpitations, tremors, anxiety, tachycardia, and insomnia[1]. Severe systemic effects—such as atrial fibrillation, ventricular arrhythmias, hyperpyrexia, myocardial infarction, seizures, or thyroid storm—have been described, particularly in cases where T3 peaks rapidly or in those with pre-existing cardiac vulnerabilities[2]. In a study by Binimelis et al., ingestion of 7–12 mg of levothyroxine resulted in coma and arrhythmia in several adult patients, highlighting the potential lethality even in the absence of proportional dosing[2].

Management is tiered based on symptom severity[2]. In asymptomatic or mildly symptomatic patients, conservative monitoring and supportive care may suffice[2]. Early decontamination with activated charcoal is effective within one hour of ingestion, especially in cases of large overdoses (>5 mg in adults)[1]. Gastric lavage is reserved for massive ingestions identified shortly after ingestion[1].

Beta-blockers, particularly propranolol, are the first-line pharmacologic agents for symptomatic relief, also reducing peripheral T4-to-T3 conversion[1]. Corticosteroids like hydrocortisone or dexamethasone offer dual benefit—suppressing conversion and addressing potential adrenal insufficiency[1]. Propylthiouracil or carbimazole may be considered, particularly in suspected thyroid storm or in cases where endogenous T4 production may contribute to symptoms[2].

In severe or refractory cases, adjunctive therapies like cholestyramine (to interrupt enterohepatic circulation) or sodium ipodate (to inhibit monodeiodinase enzymes) may be used[1]. Extracorporeal clearance techniques such as hemoperfusion and plasmapheresis are infrequently employed but may be beneficial due to the high protein binding of T4, especially when thyroid storm is established and unresponsive to standard treatment[4].

Recent studies emphasize that there is no consistent correlation between ingested dose and clinical severity, suggesting that individual metabolic capacity, pre-existing comorbidities, and co-ingestion of other substances play critical roles[2]. Furthermore, a significant proportion of symptoms may appear late, even in initially asymptomatic patients[1]. Therefore, observation for at least 48–72 hours post-ingestion is recommended, along with serial monitoring of thyroid hormone levels and vitals[1].

Similarly, Binimelis et al. documented six adults with massive ingestion (up to 1200 mg), most of whom developed coma, arrhythmias, or left ventricular failure several days after ingestion [5] .The delayed peripheral conversion of T4 to T3 drives symptom onset, which typically peaks between days 3 and 10.Kulig et al. described a 2-year-old boy with seizures and signs of hyperthyroidism after ingesting 18 mg of levothyroxine, with symptoms peaking around day seven post-ingestion [6] . Golightly et al. noted that in 41 pediatric cases, only minor symptoms like tachycardia and irritability occurred, and all resolved without treatment. [6]

Given the onset of sinus tachycardia, biochemical thyrotoxicosis, and low-grade fever, propranolol was promptly initiated to attenuate beta-adrenergic symptoms and inhibit peripheral T4-to-T3 conversion. Concurrently, hydrocortisone was administered to suppress further T3 formation and provide adrenal support. Propylthiouracil was included to block endogenous T4 synthesis and reduce active hormone generation. Cholestyramine was used to enhance gastrointestinal clearance of circulating hormones. Hemodialysis was avoided due to its ineffectiveness with highly protein-bound thyroid hormones; extracorporeal measures were reserved as contingency. The patient was observed in-hospital for 7 days, with serial thyroid function tests guiding de-escalation of therapy. This case reinforces the need for aggressive early intervention in moderate overdoses, even in initially stable patients, and supports individualized management based on biochemical trends and clinical progression.

CONCLUSION

Levothyroxine overdose, though often benign, can result in delayed yet clinically significant thyrotoxic manifestations due to peripheral T4-to-T3 conversion. Our case highlights the importance of early recognition, timely decontamination, and proactive pharmacological intervention—including propranolol, hydrocortisone, propylthiouracil, and cholestyramine. Despite a substantial ingested dose, favorable outcomes are achievable with close inpatient monitoring, serial thyroid function assessment, and individualized

therapy. This case underscores the need for vigilance even in the absence of initial symptoms and the value of a structured yet flexible management approach in acute thyroid hormone toxicity.

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