Case Report

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Unlocking the Conundrum: Shedding Light on Thyrotoxicosis Caused by Levothyroxine Overdose

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Abstract

Thyrotoxicosis secondary to levothyroxine overdose presents multifaceted clinical dilemmas influenced by dosage, patient age, and psychiatric factors. Our case, involving a 32-year-old female with chronic hypothyroidism ingested a substantial amount of levothyroxine, highlights the potential severity of such incidents. While accidental poisoning is more common in paediatric age group, our case underscores the significance of adult cases, particularly those with underlying psychiatric conditions. Prompt recognition and intervention are paramount, given the risk of severe manifestations like thyroid storm. Treatment strategies, including symptom management and hormone elimination, facilitated the patient's recovery, emphasizing the importance of comprehensive multidimensional care and heightened clinical vigilance. This case underscores the need for tailored management approaches and multidisciplinary collaboration to optimize outcomes in cases of levothyroxine intoxication induced thyrotoxicosis.

Keywords: Thyrotoxicosis; Levothyroxine; Poisoning; Clinical Vigilance; Hyperthyroidism.

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INTRODUCTION

Thyrotoxicosis resulting from levothyroxine overdose presents a complex clinical scenario, influenced by factors such as the dosage ingested, patient age, and underlying psychiatric conditions.[1] While levothyroxine (T4) overdose is relatively uncommon, its consequences can range from mild symptoms to life-threatening manifestations, particularly in cases of chronic overconsumption. Accidental ingestion, primarily observed in children, contrasts with intentional overdose, more prevalent among adults with psychiatric disorders and suicidal ideation.[2]

Clinically the spectrum of thyrotoxicosis secondary to levothyroxine encompasses a wide array of symptoms, from subtle signs of hyperthyroidism like tachycardia, agitation and insomnia, to severe outcomes including thyroid storm, characterized by multi-system involvement and high mortality rates.[3] Markedly, acute overdoses within a certain threshold may remain asymptomatic or present with mild effects, whereas chronic overuse often leads to more pronounced thyrotoxicosis and associated morbidity.[4]

Recognizing the intricacies of this condition is vital for healthcare professionals, as it can swiftly escalate into life-threatening complications. Therefore, adopting a comprehensive approach to both diagnosis and treatment is imperative, integrating pharmacological interventions and supportive measures. This strategy is indispensable for enhancing patient outcomes and reducing the likelihood of serious consequences.[5]

We present a case involving a young woman diagnosed with chronic hypothyroidism who ingested 25 tablets of Levothyroxine 100mcg.

CASE

A 32-year-old female presented to our Emergency Department with complaints of multiple episodes of vomiting fatigue, breathlessness, chest pain, and palpitations persisting since last two hours. On primary survey, her airway was patent, respiratory rate was 40 cpm with SpO2 was 98% and bilateral vesicular breath sounds on auscultation, pulse rate was 178 bpm regular and bounding with blood pressure of 170/80 mmHg with normal heart sounds, her Glasgow Coma Scale score was 15/15 blood glucose level was 189 mg/dl. She had a temperature of 102.1°F and dry mucosa.

Diagnostic evaluations revealed SVT on ECG, Oxygen by mask at 6L/min was connected, 18G IV cannula was secured in right upper limb and Inj Adenosine 6mg IV push was given and a repeat of 12mg was given to revert to sinus rhythm with a heart rate of 124bpm. (Fig. 1 & 2) 2D echocardiography indicated dilated cardiomyopathy with an ejection fraction of 40%. Arterial blood gas analysis showed high anion gap metabolic acidosis with compensation (pH of 7.25, PCO_2 of 23 mmHg, HCO_3 10 mEq/dL and electrolyte levels Na-138, K-3.6, Cl-98).

On Secondary survey she admitted to ingest around 25 tablets of Levothyroxine 100mcg 6hr prior to arrival to ED. She was diagnosed as hypothyroidism and is on Tab Levothyroxine 100 mcg since 2 years for same. Her last meal was 6 hr prior and she gives no history of any allergies.

A provisional diagnosis of hyperthyroidism secondary to tab Levothyroxine over dose was made and Burch-Wartofsky Point Scale score was found to be 75, suggesting thyroid storm with arrhythmia. Temperature control measures including cold intravenous fluids, ice packs, and cold saline through a Ryle's tube were employed.

100 g of charcoal and 4g of Cholestyramine suspension was instilled in Ryle's tube. Intravenous fluids 0.9% normal saline at 200 mL/hr was started via the peripheral cannula. She was started on oral Propranolol 60mg every 4th hourly, intravenous hydrocortisone 200mg iv followed by 100mg 8th hourly and Tab Proprylthiouracil 300mg 6th hourly for neat 24 hours then 100mg twice a day was started.

Thyroid function tests at ED revealed suppressed TSH and markedly elevated Free T3 and Free T4, and was repeated every 24hrs (Table 1). Patient was shifted to critical care unit and was managed, who remained stable throughout her hospital stay, with repeat echocardiography demonstrating systolic recovery. She was shifted to wards after 72 hrs and later discharged after 10 days with psychiatric consultation.

Table 1: Thyroid function tests result

Time from ingestion (Hours)	TSH (µIU/mL)	fT4 (pg/mL)	fT3 (pg/mL)
Reference range	(0.3–4.5 µIU/ L)	(8.9–17.1 pg/ml)	(1.2–4.1 pg/ml)
6 Hours	<0.005	59.1	18.3
24 Hours	<0.005	42.4	14.1
48 Hours	<0.01	39.2	11.5
72 Hours	<0.01	28.5	7.6
12 0Hours	<0.01	17.8	3.1

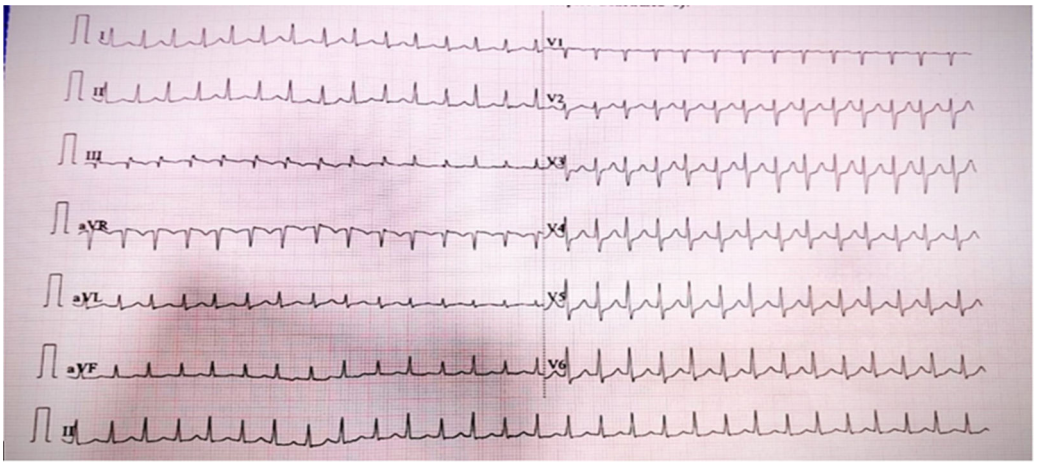

Fig. 1: ECG showing Supraventricular Tachycardia (SVT)

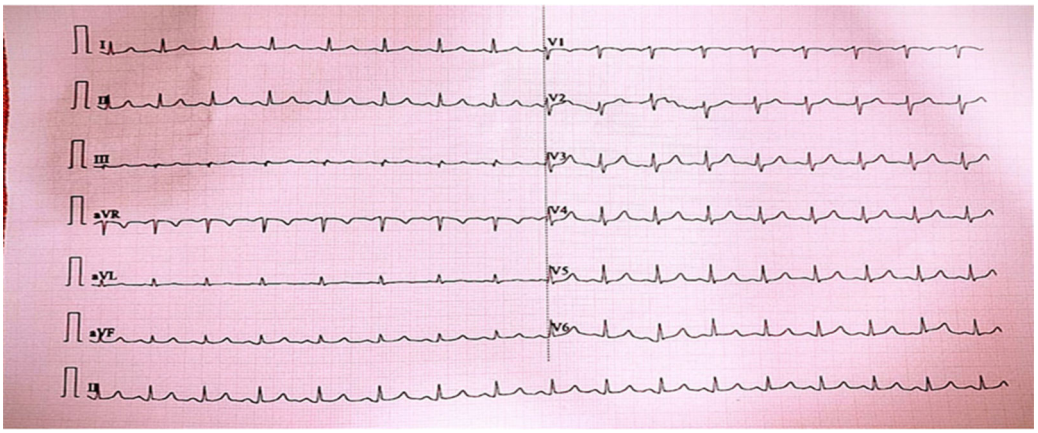

Fig. 2: Reverted to normal sinus rhythm

DISCUSSION

Accidental levothyroxine poisoning primarily affects pediatric populations, while intentional overdoses are more common in adults, often associated with psychiatric comorbidities.[6] However, case reports in adults remain scarce, highlighting the rarity and severity of such incidents.[7] Additionally, hypothyroidism's association with mood disorders underscores the complex interplay between thyroid function and mental health.[8]

Levothyroxine (T4) is converted to the biologically active triiodothyronine (T3) in vivo, exerting its effects and potentially leading to adverse outcomes when in excess. This process of deiodination usually takes 24–48 hours, explaining the delayed onset of symptoms and their potential persistence due to the hormone's prolonged half-life.[9] Consequently, an expected biochemical profile includes elevated total and free T3 and T4 levels, accompanied by a decrease in TSH, which may normalize over a week, as in our case. Notably, acute ingestion of levothyroxine up to 4 mg is often asymptomatic, although adverse features are more likely to occur at higher doses.[4,10]

In our case, patient presented with a arrhythmia which was successfully managed on arrival to emergency room. After which she remained relatively asymptomatic in critical care, likely attributed to the relatively low ingested dose (2.5 mg) and concurrent propranolol use, which potentially inhibited peripheral conversion of T4 to T3.[11]

Thyrotoxicosis, characterized by excess circulating thyroid hormone, manifests with diverse symptoms, ranging from fever and tachycardia to life-threatening complications such as cardiac arrhythmias and altered mental status. While Graves' disease and multinodular goiter are

common etiologies, exogenous thyroid hormone ingestion can also precipitate thyrotoxicosis, albeit rarely reported in adults.[4]

Prompt diagnosis and treatment of thyroid storm, a severe manifestation of thyrotoxicosis, are crucial for patient outcomes. The Burch-Wartofsky scoring system aids in assessment, guiding therapeutic interventions to mitigate morbidity and mortality.[12]

Treatment strategies for levothyroxine intoxication focus on symptom management, hemodynamic stabilization, and enhancing hormone elimination. While gastric lavage, charcoal administration, and beta-blockers aim to reduce absorption and sympathetic hyperactivity, propylthiouracil inhibits thyroid hormone conversion. Glucocorticoids may be used in combination with beta-blockers in severe cases, while therapeutic plasma exchange may be considered to lower hormone levels. Hemodialysis is rarely useful in such cases.[2-4,13]

Despite the rarity and potential severity of levothyroxine intoxication, effective management facilitated the patient's recovery, underscoring the importance of heightened clinical awareness and comprehensive management strategies to optimize outcomes and prevent life-threatening complications.

CONCLUSION

The case of thyrotoxicosis secondary to levothyroxine ingestion underscores the complex interplay between thyroid function and medication interactions. Accidental poisoning with levothyroxine, though more prevalent in pediatric populations, poses significant risks in adults, particularly those with underlying mental health disorders or suicidal tendency. Despite the rarity of adult cases, the potential for severe manifestations such as thyroid storm necessitates prompt recognition and intervention.

The delayed onset of symptoms and prolonged half-life of levothyroxine highlight the importance of close monitoring and prolonged hospitalization for affected individuals. Management strategies, including symptom control, hemodynamic stabilization, and hormone elimination, are crucial in mitigating morbidity and mortality associated with thyrotoxicosis.

Furthermore, the case underscores the need for comprehensive psychiatric evaluation and medication review in patients presenting with thyroid dysfunction, as concurrent psychotropic medications may exacerbate symptoms or interfere with treatment efficacy. Overall, effective management and multidisciplinary care are essential in optimizing outcomes and preventing life-threatening complications in cases of levothyroxine intoxication-induced thyrotoxicosis.

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REFERENCES

1. Savran Y, Mengi T, Keskinkılıç M. A severe case of levothyroxine intoxication successfully treated in intensive care unit. Journal of Acute Disease. 2018;7(4):175-178.
2. Korkmaz HA, Dizdarer C, Hazan F, Karaarslan U. Attempted suicide with levothyroxine in an adolescent girl. Journal of Pediatric Endocrinology and Metabolism. 2013;26(12): 129–131.
3. Hays H, Jolliff HA, Casavant MJ. Thyrotoxicosis after a massive levothyroxine ingestion in a 3-Year-Old patient. Pediatric Emergency Care. 2013;29:1217–1219.
4. Brandt F, Green A, Hegedüs L, Brix TH. A critical review and meta-analysis of the association between overt hyperthyroidism and mortality. European Journal of Endocrinology. 2011;165: 491–497.
5. Wong O, Wong A, Greene SL, Graudins A. Prolonged coma resulting from massive levothyroxine overdose and the utility of N-terminal prohormone brain natriuretic peptide (NT-proBNP). Clinical Toxicology. 2018;57:415–417.
6. Litovitz TL, White JD. Levothyroxine ingestions in children: an analysis of 78 cases. American Journal of Emergency Medicine. 1985;3(4): 297–300.
7. Kraai EP, Seifert SA. Citalopram overdose: A Fatal Case. Journal of Medical Toxicology. 2015;11(2): 232-236.
8. Khemka D, Ali JA, Koch C. Primary Hypothyroidism Associated with Acute Mania: Case Series and Literature Review. Experimental and Clinical Endocrinology & Diabetes. 2011;119(8): 513–517.
9. Shilo L, Kovatz S, Hadari R, Weiss E, et al. Massive thyroid hormone overdose: kinetics, clinical manifestations and management. Israel Medical Association Journal. 2002;4(4): 298–299.

10. Binimelis J, Bassas L, Marruecos L, Rodríguez JMTI, Domingo M, Madoz P, et al. Massive thyroxine intoxication: evaluation of plasma extraction. Intensive Care Medicine, 1987;13(1): 33–38.
11. Schottstaedt ES, Smoller M. "Thyroid storm" produced by acute thyroid hormone poisoning. Annals of Internal Medicine. 1966;64: 847-849.
12. Burch HB, Wartofsky L. Life-threatening thyrotoxicosis. Thyroid storm. Endocrinology and Metabolism Clinics of North America. 1993;22(2): 263-277.
13. Idrose AM. Acute and emergency care for thyrotoxicosis and thyroid storm. Acute Med Surg. 2015;12(3): 147-157.