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Accidental Thyroxine Ingestion in a Toddler: A Case Report on Clinical Course and Management

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ABSTRACT

Accidental thyroxine ingestion in toddlers is a rare but concerning event due to its potential to cause transient hyperthyroxinemia. We report a case of a 1-year-old female who ingested an unknown quantity of thyroxine tablets but remained asymptomatic despite significantly elevated FT3 and FT4 levels. The child was closely monitored with serial thyroid function tests and managed conservatively without pharmacological intervention. Thyroid hormone levels gradually normalized over 11 days without complications. This case highlights the importance of differentiating biochemical abnormalities from true toxicity and reinforces the need for parental education on safe medication storage to prevent accidental pediatric exposures. Early recognition, timely evaluation and appropriate management can ensure a favorable outcome and prevent unnecessary interventions in such cases.

Keywords: Accidental ingestion, thyroxine toxicity, pediatric emergency.

INTRODUCTION

Accidental ingestion of medications by toddlers is a common concern in pediatric emergency medicine. Thyroxine toxicity in children is relatively rare, but it can cause significant physiological changes if not managed promptly. Ingestion of even a small amount of thyroxine can lead to hyperthyroxinemia, requiring careful monitoring and appropriate intervention [1]. The severity of symptoms depends on the dose ingested, metabolic status and individual physiological response. Given the increasing availability of thyroid hormone replacement therapy due to the high prevalence of hypothyroidism, unintentional pediatric exposures are becoming more frequently reported [2].

Clinical manifestations of thyroxine toxicity can range from mild symptoms such as irritability, restlessness and tachycardia to severe complications including hypertension, hyperthermia and cardiac arrhythmias [3]. The latent period before symptom onset can vary often leading to a diagnostic challenge in cases where the

ingestion is unwitnessed. Laboratory investigations typically reveal elevated free T4 and suppressed TSH levels, aiding in the confirmation of thyroxine overdose [4].

Management of thyroxine toxicity is largely supportive, focusing on symptomatic treatment and preventing further absorption. Activated charcoal may be considered if the ingestion is recent, while beta-blockers such as propranolol are often used to control adrenergic symptoms. In severe cases, corticosteroids and cholestyramine may be administered to enhance thyroxine metabolism and clearance [5, 6].

Proper parental awareness and medication storage practices are crucial to preventing such incidents. Child-resistant packaging, secure medication storage and educating caregivers about potential risks can significantly reduce the likelihood of accidental ingestion [7].

Case Presentation: A 1-year-old female toddler with normal developmental milestones was brought to the emergency department with a history of accidental ingestion of thyroxine tablets. The exact quantity ingested was unknown. The mother, a known case of hypothyroidism had been on Thyronorm 125 mcg daily for the past four years. The parents suspected ingestion and brought the child to the emergency department approximately four hours after the incident. On clinical examination the child appeared normal and was completely asymptomatic. There was no history of vomiting, abdominal pain, excessive sweating, ear discharge, rashes or urinary disturbances. Neurologically there was no evidence of altered consciousness, seizures or increased cardiac activity such as palpitations or tachycardia.

Diagnosis and Initial Workup: The child was admitted for close monitoring and underwent an initial laboratory evaluation.

Parameters	Result	Reference Range	Units	Methodology
FT3	14.06	2.0 - 4.4	pg/ml	ECLIA
FT4	>7.77	0.89 - 1.76	ng/dl	ECLIA
TSH	0.422	0.27 - 4.20	uIU/ml	ECLIA
SGOT	30	10 – 35	U/L	UV without P5P
SGPT	15	0 – 35	U/L	UV without P5P
Sodium	138	138 - 145	mEq/L	ISE Direct
Potassium	3.86	Neonates: 3.7 - 5.9 Children: 3.4 - 4.7 Adult: 3.5 - 5.0	mEq/L	ISE Direct
Chloride	105	95 - 107	mEq/L	ISE Direct
Bicarbonate	20.5	20 - 27	mEq/L	ISE Direct

Table 1: shows the initial lab test result on day 1

Electrolytes, liver function tests and other biochemical parameters were within normal limits. The child remained asymptomatic during hospitalization and was managed conservatively with supportive care, including close clinical observation, hydration and serial thyroid function monitoring.

Follow-up and Outcome: Thyroid function tests were repeated on the 6th day, showing a decline in hormone levels:

Parameters	Result	Reference Range	Units	Methodology
FT3	8.39	2.0 - 4.4	pg/ml	ECLIA
FT4	3.36	0.89 - 1.76	ng/dl	ECLIA
TSH	0.018	0.27 - 4.20	uIU/ml	ECLIA
SGOT	13	10 – 35	U/L	UV without P5P
SGPT	15	0 – 35	U/L	UV without P5P

Table 2: shows the 6th day thyroid profile result.

By the 11th day, the child's thyroid hormone levels had returned to normal indicating recovery without any long-term complications.

Parameters	Result	Reference Range	Units	Methodology
FT3	3.59	2.0 - 4.4	pg/ml	ECLIA
FT4	1.02	0.89 - 1.76	ng/dl	ECLIA
TSH	0.29	0.27 - 4.20	uIU/ml	ECLIA

Table 3: displays the 11th day thyroid profile result.

DISCUSSION

Thyroxine overdose can lead to thyrotoxicosis causing symptoms such as tachycardia, hyperthermia, agitation and gastrointestinal disturbances. However, in children accidental ingestion often results in transient hyperthyroxinemia without significant clinical symptoms due to the body's compensatory mechanisms [8]. The peripheral conversion of T4 to the more active T3 is regulated by deiodinases, which may slow down in response to excess thyroxine, limiting its immediate effects [9].

In this case despite elevated FT3 and FT4 levels the child remained asymptomatic likely due to delayed peripheral conversion of thyroxine and a gradual downregulation of thyroid hormone receptors. Additionally, the absence of significant adrenergic symptoms suggested an adaptive physiological response, which prevented the development of overt thyrotoxicosis. Close monitoring without aggressive intervention was sufficient for recovery, aligning with previous reports that mild to moderate accidental thyroxine ingestion in children often follows a benign course [10].

Management of thyroxine overdose is largely supportive, with a focus on symptomatic treatment. Beta-blockers such as propranolol are considered if significant tachycardia or hypertension develops but in asymptomatic cases observation and serial thyroid function tests are usually sufficient [11]. Activated charcoal is only effective if administered within a short window post-ingestion and more advanced therapies like cholestyramine or glucocorticoids are reserved for severe cases [12].

This case highlights the importance of differentiating between biochemical abnormalities and true clinical thyrotoxicosis in pediatric thyroxine ingestion. It also underscores the need for parental education on safe medication storage to prevent accidental exposure in young children.

CONCLUSION

Accidental thyroxine ingestion in toddlers can be alarming for parents but may not always result in clinical toxicity. This case highlights the importance of serial thyroid function monitoring and supportive management. While most cases resolve without intervention, recognizing potential complications such as significant tachycardia or hyperthermia is crucial for timely intervention. Pediatricians should educate caregivers about medication storage safety to prevent such incidents, emphasizing the need for child-resistant packaging and secure storage away from a child's reach. Timely intervention, parental reassurance and appropriate follow-up can ensure a favorable outcome without complications, reinforcing the importance of preventive strategies in pediatric medication safety.

Declaration by Authors

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