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# Reversible Liver Dysfunction Secondary to Thyrotoxicosis: A Case Report

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**Case Report**

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## Abstract

# Background:

Thyroid hepatopathy refers to liver dysfunction associated with thyroid disease, particularly hyperthyroidism. Liver dysfunction is a recognized but often underappreciated manifestation of severe thyrotoxicosis.

# Case Presentation:

A 28-year-old woman presented with a two-month history of progressive palpitations, heat intolerance, unintentional weight loss, and fatigue. Examination revealed bilateral exophthalmos, fine tremor of the hands, and a diffuse, large, non-tender goiter without bruit. Laboratory evaluation on initial presentation demonstrated severe thyrotoxicosis with suppressed thyroid-stimulating hormone (< 0.01 mIU/L), markedly elevated free triiodothyronine (> 50 pmol/L) and free thyroxine (> 100 pmol/L), and positive anti-thyroid peroxidase antibodies. Liver function tests showed significant transaminitis (AST 210 U/L, ALT 388 U/L) and elevated total bilirubin (8.6 mg/dL). Viral and autoimmune causes of liver disease were excluded. Thyroid ultrasound revealed a diffusely enlarged, hypervascular gland. The patient was treated with carbimazole, resulting in progressive normalization of thyroid function and complete resolution of liver enzyme abnormalities.

# Conclusion:

Severe thyrotoxicosis can present with marked and reversible liver dysfunction, mimicking primary hepatic disease. Early recognition of this association is essential to avoid unnecessary investigations and ensure timely management.

## Introduction

Liver dysfunction associated with hyperthyroidism, often referred to as thyroid hepatopathy, encompasses a spectrum ranging from mild biochemical abnormalities to clinically significant hepatic injury. In untreated patients with hyperthyroidism, abnormalities in liver function tests are common, reported in 45%–90% of patients (1–3). However, more severe manifestations such marked transaminitis and hyperbilirubinemia are less common and may mimic primary hepatic disease. When symptomatic hepatitis develops, a more severe disease known as thyrotoxic hepatitis occurs, but biochemical normalization is usually achieved after euthyroid status is reached (1, 4).

In cases of thyrotoxic hepatitis, a correct diagnosis can require extensive and potentially unnecessary investigations, particularly in resource-limited settings. We present a case of severe thyrotoxicosis complicated by significant and reversible liver dysfunction.

## Case Presentation

A 28-year-old female with no known illness presented with a two-month history of progressive palpitations, heat intolerance, unintentional weight loss despite preserved appetite, and generalized fatigue. She denied alcohol use, hepatotoxic medications, or herbal supplement intake. On examination, she had bilateral exophthalmos, and a fine resting tremor of the hands. Neck examination revealed a diffuse, large, non-tender goiter without an audible bruit (Fig. 1). Laboratory investigations demonstrated severe thyrotoxicosis with suppressed thyroid-stimulating hormone (TSH) < 0.01 mIU/L, and an elevated free triiodothyronine (Free T3) > 50 pmol/L, and high free thyroxine (Free T4) > 100 pmol/L. Anti-thyroid peroxidase antibodies were elevated (Anti-TPO: 53.59 IU/mL). Liver function tests revealed elevated transaminases, with alanine aminotransferase (ALT) of 388 U/L and aspartate aminotransferase (AST) of 210 U/L, along with significantly elevated total bilirubin of 8.4 mg/dL. Serologic testing for viral hepatitis (HBV, HCV, and HAV) were negative. Autoimmune screening, including antinuclear antibody (ANA), was also negative, as shown in Table 1. Thyroid ultrasound showed a diffusely enlarged, heterogeneous, and hyper vascular thyroid gland, consistent with diffuse thyroid disease, as shown in Fig. 2. Abdominal ultrasound demonstrated no evidence of hepatomegaly, biliary dilatation, focal liver lesions, or ascites. The patient was started on carbimazole 15 mg three times daily, along with Propranolol 40mg three times daily. On follow up, there was a progressive normalization of thyroid function tests accompanied by the resolution of liver enzyme abnormalities. On her 3-month follow up visit, her medications frequency was adjusted.

Table 1
Serial laboratory investigations.

| Lab Test | Normal Values | At Presentation | Day 21 | Day 45 | 3 Month |
|---|---|---|---|---|---|
| Thyroid Function test | | | | | |
| *fT4* | 12–22 pmol/l | > 100 | 45.4 | 25 | 19 |
| *fT3* | 3.1–6.8 pmol/l | > 50 | 16.3 | 12 | 5.5 |
| *TSH* | 0.4–4.2 µU/mL | < 0.01 | < 0.01 | < 0.01 | 1.3 |
| *Anti-TPO* | 0–34 IU/ML | 53.59 | - | - | - |
| Liver Function Test | | | | | |
| *ALP* | 40–112 U/L | 179 | 176 | 170 | 141 |
| *AST* | 0–40 U/L | 210 | 90 | 39 | 29 |
| *ALT* | 0–40 U/L | 388 | 50 | 25 | 23 |
| *Total Bilirubin* | 0.20–1.10 mg/dl | 8.6 | 4.28 | 1.0 | 0.9 |
| *Direct Bilirubin* | 1.02–1.46 mg/dl | 3.8 | 1.1 | 0.3 | 0.3 |
| *Albumin* | 3.5–5.5 g/dl | 3.6 | 3.6 | 4.0 | 4.0 |
| Hematology | | | | | |
| *WBC* | 4–10 $10^{12}$/l | 5.18 | - | - | 4.8 |
| *Hgb* | 11–16 g/dl | 12.2 | - | - | 12 |
| *Plt* | 100–450 $10^{9}$/l | 227 | - | - | 315 |
| Autoimmune | | | | | |
| *ANA* | 0–20 AU/ML | 1 | - | - | - |
| Viral Screening | | | | | |
| *HAV antibody* | Negative | Negative | - | - | - |

| Lab Test | Normal Values | At Presentation | Day 21 | Day 45 | 3 Month |
|---|---|---|---|---|---|
| *HBV surface antigen* | Negative | Negative | - | - | - |
| *HBV core antigen* | Negative | Negative | - | - | - |
| *HCV antibody* | Negative | | - | - | - |
| *HIV antigen/antibody* | Negative | Negative | - | - | - |
| Medication | - | CMZ + Propranolol | CMZ + Propranolol | CMZ + Propranolol | CMZ + Propranolol |

*fT4: free T4; TSH: thyroid-stimulating hormone; Anti-TPO: ALP: alkaline phosphatase; ALT: alanine transaminase; ALP: alkaline phosphatase; Hb: hemoglobin; WBC: white blood count; Plt: Platelets; ANC: absolute neutrophil count; ANA: antinuclear antibodies; HAV: hepatitis A virus; HBV: hepatitis B virus; HCV: hepatitis C virus; HIV: human immunodeficiency virus; CMZ: carbimazole

# Discussion

This case highlights a significant yet reversible form of liver dysfunction associated with severe thyrotoxicosis. Although mild abnormalities in liver biochemistry are relatively common in hyperthyroid patients, marked transaminitis and hyperbilirubinemia remain uncommon and can create substantial diagnostic uncertainty, particularly in settings where infectious and primary hepatobiliary diseases are more prevalent (5, 6).

The relationship between thyroid disease and hepatic dysfunction is complex and multifactorial (1). Thyroid hormones exert significant effects on hepatic metabolism, oxygen consumption, lipid regulation, and mitochondrial activity. In hyperthyroidism, the hypermetabolic state leads to increased hepatic oxygen demand without a proportional increase in hepatic blood flow, resulting in relative centrilobular hypoxia and hepatocellular injury. This mechanism is believed to contribute significantly to elevations in aminotransferases observed in thyrotoxic patients. In severe disease, hepatic ischemia secondary to increased metabolic demands may produce substantial hepatocellular damage, even in the absence of overt cardiovascular compromise (7, 8).

Several mechanisms have been proposed to explain liver injury in thyrotoxicosis, including direct hepatocyte toxicity from excess thyroid hormone, increased oxidative stress, and relative hepatic hypoxia caused by increased metabolic demand and reduced effective hepatic perfusion (2, 5). Autoimmune mechanisms may also contribute, particularly in Graves' disease, while complications such as heart failure or atrial fibrillation can further worsen hepatic dysfunction (4, 5). An important diagnostic challenge in such cases is distinguishing thyrotoxic hepatopathy from alternative causes of liver injury. Viral hepatitis, autoimmune hepatitis, biliary obstruction, drug-induced liver injury, Wilson disease, and

alcoholic liver disease must all be considered, particularly in younger patients presenting with jaundice and elevated transaminases (2, 5, 9). In regions such as Somalia, where infectious causes of hepatitis remain common, clinicians may understandably prioritize infectious etiologies (10).

In our patient, the lack of heart failure or hemodynamic compromise suggests that the hepatic dysfunction was most likely related to direct metabolic and hypoxic effects of severe thyrotoxicosis. The liver injury exhibited a predominantly hepatocellular pattern, characterized by marked elevations in ALT and AST along with hyperbilirubinemia, closely resembling acute hepatitis. Alternative causes of liver disease, including viral and autoimmune etiologies, were excluded through comprehensive evaluation, and the complete resolution of liver abnormalities following treatment of hyperthyroidism further supports thyrotoxicosis as the underlying cause of hepatic injury.

The temporal relationship between treatment initiation and biochemical recovery further supports the diagnosis of thyroid hepatopathy. Restoration of euthyroidism is generally associated with normalization of liver function tests, often within weeks to months (11). Our patient demonstrated progressive improvement in both thyroid and liver parameters after initiation of carbimazole therapy, consistent with previous reports describing reversibility of hepatic dysfunction once hyperthyroidism is adequately controlled (5, 7, 12, 13).

Management of hyperthyroidism-associated liver dysfunction presents an additional challenge because antithyroid medications themselves may cause hepatotoxicity (14). Carbimazole and methimazole are more commonly associated with cholestatic liver injury, whereas propylthiouracil has been linked to severe hepatocellular injury and fulminant hepatic failure (15). This creates a therapeutic dilemma when significant baseline liver dysfunction is already present. Despite concerns regarding antithyroid drug hepatotoxicity, current evidence suggests that untreated thyrotoxicosis itself is often the primary cause of hepatic dysfunction, and successful treatment generally results in biochemical improvement (1, 7, 12, 15). In our patient, carbimazole was administered safely with subsequent normalization of liver enzymes, nevertheless, careful monitoring of hepatic parameters after initiation of therapy remains essential.

This case highlights the importance of considering thyrotoxicosis in patients with unexplained hepatitis, particularly in resource-limited settings where extensive hepatology investigations may be costly or inaccessible. Recognition of clinical features such as weight loss, heat intolerance, tremor, goiter, and ophthalmopathy can facilitate early diagnosis, avoid unnecessary investigations, and allow timely treatment before serious complications develop. Reports of severe thyrotoxic hepatitis from sub-Saharan Africa remain limited in the literature. This case contributes valuable data from Somalia and highlights the need for greater awareness of thyroid hepatopathy in regions where hepatic infections are endemic and endocrine disorders may be underrecognized, as improved clinician awareness may enhance diagnostic accuracy and facilitate timely management.

In conclusion, severe thyrotoxicosis should be recognized as a potentially reversible cause of significant liver dysfunction. Thyroid hepatopathy may present with marked transaminitis and hyperbilirubinemia, closely mimicking primary hepatic disease. Careful clinical assessment and exclusion of other etiologies

are essential. Prompt treatment of the underlying hyperthyroidism can result in complete biochemical recovery, as demonstrated in this case.

## Abbreviations

ALT
Alanine aminotransferase
ALP
Alkaline Phosphatase
ANA
Antinuclear Antibody
AST
Aspartate aminotransferase
Anti
TPO–Anti–thyroid peroxidase
CMZ
Carbimazole
HAV
Hepatitis A virus
HBV
Hepatitis B virus
HCV
Hepatitis C virus
Hgb
Hemoglobin
HIV
Human Immunodeficiency Virus
fT3
Free Triiodothyronine
fT4
Free Thyroxine
TSH
Thyroid Stimulating Hormone
Plt
Platelet
WBC
White Blood Count

## Declarations

### Ethical Approval and Consent to Participate

Ethical approval was not required from the study site, Hodan Hospital in Mogadishu, Somalia, for publication of a single-patient case report. However, written informed consent for this case publication was obtained from the patient.

**Consent for publication**

Written informed consent was obtained from the patient publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

**Conflict of Interest**

The authors declare that they have no competing interests.

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# Author Contribution

Y.B and A.H.D. contributed to conceptualization, visualization, and writing the original draft of the manuscript. A.M.A. contributed to manuscript review and editing. A.R. prepared the figures and clinical images. H.E. contributed to manuscript review and editing and provided supervision. All authors read and approved the final manuscript.

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# Availability of data and materials

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

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# Figures



**Figure 1**

Thyroid Goiter.

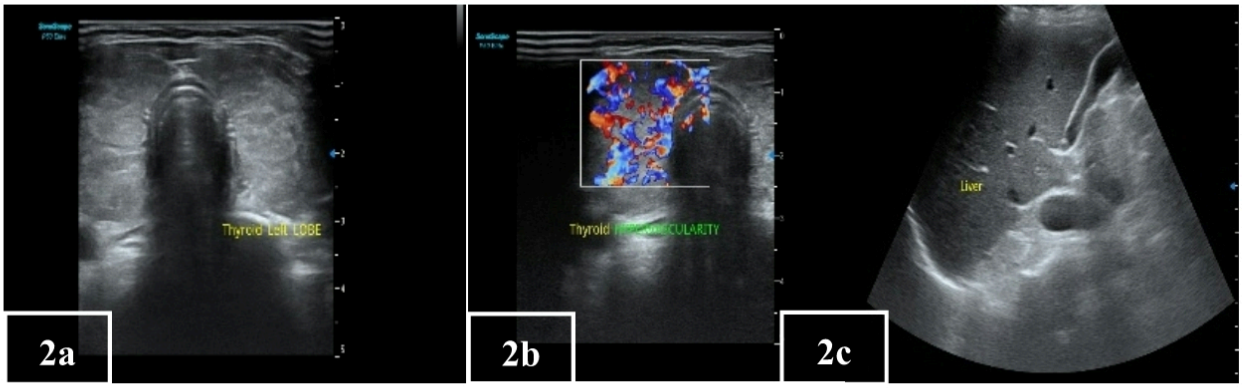



**Figure 2**

2a: Demonstrates transverse ultrasound scan of the thyroid using high frequency linear group showing diffuse heterogeneous texture of the thyroid. 2b: Shows Doppler interrogation of the thyroid showing moderate hypervascularity. 2c: Demonstrates longitudinal scan of the liver using convex probe showing homogeneous texture of the liver.