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CASE REPORTS

Myxedema as a Reversible Cause of Ischemic Hepatitis: Severe Hashimoto's Hypothyroidism Presenting with Profound Bradycardia, Hypotension and Marked Hypertransaminasemia.

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ABSTRACT

Background. Ischemic (hypoxic) hepatitis, the "shock liver" of the emergency setting, follows a fall in hepatic oxygen delivery, usually from cardiac, circulatory or respiratory failure, and produces a steep but short-lived rise in liver transaminases. Severe primary hypothyroidism is a classic yet easily forgotten cause of a reversible low-output circulation, and it can present first in the liver.

Case report. A 65-year-old man with no known thyroid disease presented to the emergency department with severe hypotension (60/40 mmHg) and bradycardia (45 beats/min) and marked hepatocellular injury (aspartate aminotransferase, AST, 1643 U/L; alanine aminotransferase, ALT, 819 U/L), with a minimally raised troponin and acute kidney injury. Lactate dehydrogenase was disproportionately elevated (LDH 1279 U/L; ALT-to-LDH ratio 0.7) and creatine kinase was markedly raised (CK 2946 U/L), while synthetic function was preserved. Viral serology was negative and the liver was structurally normal. As blood pressure recovered, the transaminases fell rapidly and the AST-to-ALT ratio reversed (from 2.0 to 0.3 by day 6). A coarse voice present for several weeks prompted thyroid testing, which showed TSH 80.371 mU/L, suppressed free hormones and anti-thyroid peroxidase antibodies of 848.77 IU/mL, establishing Hashimoto's thyroiditis. Both the circulation and the liver recovered with fluids and levothyroxine.

Conclusion. In an obtunded, bradycardic, hypotensive patient with marked transaminase elevation and no viral, autoimmune or toxic explanation, severe hypothyroidism should be considered early. The ALT-to-LDH ratio and the reversal of the AST-to-ALT ratio help identify an ischemic mechanism, a markedly raised creatine kinase signals a coexisting hypothyroid myopathy contributing to the AST, and a single thyroid-stimulating hormone can redirect the entire differential.

Keywords. myxedema; hashimoto; bradycardia; hypertransaminasemia; severe hypotension

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INTRODUCTION

When a previously well adult arrives in the emergency department obtunded and shocked, with transaminases in the thousands, the differential narrows fast, toward acute viral hepatitis, drug toxicity or ischemia. Endocrine disease seldom makes the first list. Yet severe hypothyroidism can produce exactly this picture, and it can do so quietly, without the jaundice, the alcohol history or the drug exposure that usually accompany a liver in trouble.

Ischemic hepatitis, also called hypoxic hepatitis or shock liver, is one of the commonest reasons for very high transaminases in hospitalized patients. It reflects a mismatch between the oxygen the liver receives and the oxygen it needs, most often in the setting of cardiac failure, circulatory shock or respiratory failure. (1,2)

The biochemical signature is distinctive: aspartate (AST) and alanine (ALT) aminotransferases climb steeply, frequently past 1000 U/L, then retreat almost as quickly once perfusion improves, while lactate dehydrogenase rises disproportionately. (1,3,5)

In the largest pooled series to date, the mean age was about 64 years and an acute cardiac event preceded roughly four of every five episodes, with in-hospital mortality near one-half; a documented fall in blood pressure was present in only about half of patients, so frank shock is not a prerequisite. (4)

Hypothyroidism enters this story through the heart. Thyroid hormone sets the resting heart rate, sustains myocardial contractility and helps regulate vascular tone. Remove it and the circulation becomes the opposite of the thyrotoxic state: slow, underfilled and poorly contractile, with reduced cardiac output and, in advanced disease, pericardial effusion and conduction block (6).

These changes reverse with thyroid hormone, which is precisely what makes the organ injury that accompanies them reversible as well. (6) The overlap of these two processes is reported only occasionally and is easily mistaken for primary liver disease. We describe a man whose severe Hashimoto's hypothyroidism declared itself as ischemic hepatitis, and in whom both the circulation and the liver recovered within days.

CASE REPORT

A 65-year-old man was brought to the emergency department with progressive lethargy and reduced responsiveness. He had no known thyroid disease and no prior history of liver disease. Neither the patient nor his family reported alcohol excess, paracetamol or other drug ingestion, or use of herbal or traditional remedies. On arrival he was hypotensive at 60/40 mmHg and bradycardic at 45 beats per minute, and he was afebrile.

The skin was dry and coarse and the periorbital tissues puffy; the deep tendon reflexes relaxed slowly, giving the "hung-up" quality typical of hypothyroidism. As telling as these findings was what we did not see: no jaundice, no spider naevi or palmar erythema, no asterixis, and a soft, non-tender abdomen with no hepatomegaly. On examination the liver looked entirely innocent.

The electrocardiogram showed sinus bradycardia at 45 beats per minute.

The complete blood count was unremarkable at presentation. Initial biochemistry showed marked hepatocellular injury, with AST 1643 U/L and ALT 819 U/L, a minimally elevated high-sensitivity cardiac troponin (19.6 ng/L) and acute kidney injury (creatinine 1.38 mg/dL). Arterial hypotension persisted on the first day, with modest improvement.

A second panel the following day showed ALT 873 U/L, AST 1006 U/L, GGT 151 U/L, alkaline phosphatase 166 U/L, total protein 6.6 g/dL and albumin 4.4 g/dL, with a markedly elevated lactate dehydrogenase (LDH 1279 U/L) and creatine kinase (CK 2946 U/L). Troponin was 26.7 ng/L, creatinine 1.47 mg/dL and urea 57.1 mg/dL, and coagulation showed an INR of 1.32 (prothrombin activity 67%). An absolute neutrophilia was now present, and the ALT-to-LDH ratio was 0.7. Protein electrophoresis was normal. Serology for hepatitis A (anti-HAV IgM), hepatitis B surface antigen, hepatitis C antibody, cytomegalovirus (CMV IgM) and toxoplasma (IgM) was negative. Abdominal ultrasound demonstrated a structurally normal, non-enlarged liver with patent vessels and biliary tree and no ascites, excluding biliary obstruction and a Budd–Chiari-type vascular lesion.

A disproportionately raised LDH, an ALT-to-LDH ratio below 1.5 and negative viral serology together pointed away from viral hepatitis and toward an ischemic mechanism (5). The full laboratory trajectory is shown in Table 1 and Figure 1.

The magnitude of the aminotransferase rise initially suggested acute viral hepatitis, drug-induced or toxic liver injury, autoimmune hepatitis, or ischemic hepatitis. Viral serologies were negative, there was no history of hepatotoxic ingestion, and the liver was structurally normal with patent vasculature, making biliary and vascular causes unlikely.

The disproportionate LDH with a low ALT-to-LDH ratio, the preserved synthetic function and the rapid fall of the transaminases as the blood pressure recovered were most consistent with ischemic hepatitis.

By the third hospital day the patient regained a normal arterial pressure (110/70 mmHg) with intravenous fluids and supportive care. A sample on day 4 already showed a clear downward trend (ALT 563 U/L, AST 432 U/L, CK 1652 U/L, troponin 11 ng/L, creatinine 1.39 mg/dL). As his communication recovered, a coarse voice became apparent; he reported that the hoarseness and slower speech had been present for several weeks.

Thyroid function testing revealed profound primary hypothyroidism: TSH 80.371 mU/L, FT3 <0.95 pg/mL and FT4 <0.42 pg/dL, with anti-thyroid peroxidase

Table 1. Laboratory trajectory over the six-day admission.

Analyte (units)	Day 1	Day 2	Day 4	Day 6	Reference (typical)*
AST (U/L)	1643	1006	432	54	<40
ALT (U/L)	819	873	563	168	<41
AST: ALT ratio	2.0	1.2	0.8	0.3	-
LDH (U/L)	-	1279	-	-	135-225
ALT: LDH ratio	-	0.7	-	-	<1.5 favours ischemia
Creatine kinase (U/L)	-	2946	1652	-	<170
Troponin (ng/L, hs)	19.6	26.7	11	-	assay-dependent
Creatinine (mg/dL)	1.38	1.47	1.39	1.07	0.7-1.3
Urea (mg/dL)	-	57.1	-	-	15-40
INR / PT (%)	-	1.32 / 67	-	-	~1.0 / >70
Total bilirubin (mg/dL)	-	-	-	0.47	<1.2
GGT / ALP (U/L)	-	151 / 166	-	-	<60 / 40-130
Albumin (g/dL)	-	4.4	-	-	3.5-5.0
Blood pressure (mmHg)	60/40	-	110/70 (D3)	-	-

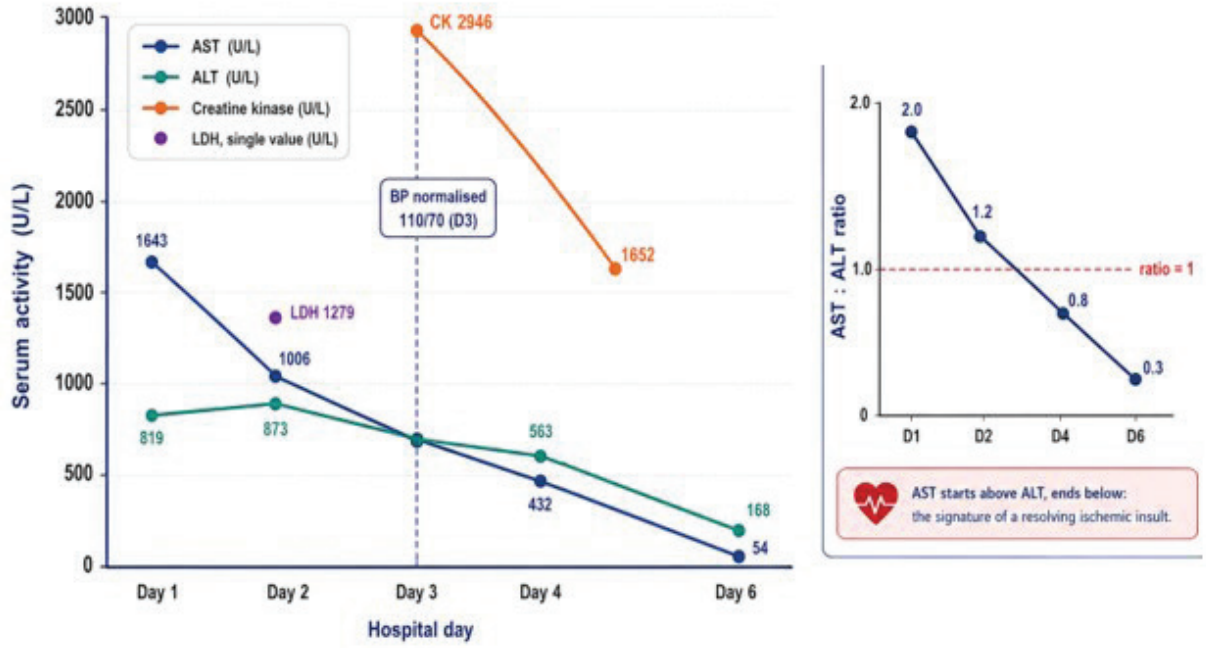

Figure 1. Laboratory and Hemodynamic trajectory over the six-day admission.

Trajectory of serum AST, ALT, creatine kinase and lactate dehydrogenase across the admission, with the AST-to-ALT ratio shown in the inset. AST peaks highest and falls fastest, and the AST-to-ALT ratio crosses from 2.0 on admission to 0.3 by day 6. The disproportionately raised LDH (ALT-to-LDH ratio 0.7) and the markedly elevated, falling CK are shown on the same axis. Aminotransferase recovery follows restoration of blood pressure (dashed line, day 3). Day 5 was not sampled.

antibodies of 848.77 IU/mL. The endocrinology service diagnosed myxedema due to Hashimoto's thyroiditis in a patient with no previously recognized thyroid disease, and levothyroxine replacement was initiated with cautious dosing appropriate to his age and cardiac status.

Because thyroid hormone replacement can precipitate an adrenal crisis in a patient with unrecognized adrenal insufficiency, attention to the adrenal axis is advised before and during replacement in severe hypothyroidism.

By day 6 the AST had fallen to 54 U/L and the ALT to 168 U/L, with a normal bilirubin (0.47 mg/dL) and a creatinine of 1.07 mg/dL; heart rate and blood pressure were normal and his mental state had returned to baseline. He was discharged with endocrinology follow-up and serial monitoring of thyroid function and liver enzymes.

The AST-to-ALT ratio falls progressively and reverses (AST above ALT on admission, well below it by day 6), the lactate dehydrogenase is disproportionately raised with an ALT-to-LDH ratio below 1.5, and the creatine kinase is markedly elevated, indicating a coexisting hypothyroid myopathy. Hepatic recovery parallels restoration of blood pressure.

DISCUSSION

Three things made the diagnosis. Viral causes were excluded and the liver was structurally normal with patent vessels; the enzymes fell over days rather than weeks; and the recovery of the transaminases tracked the recovery of the blood pressure. Together they point away from a primary hepatitis and toward ischemic injury driven by the low-output circulation of severe hypothyroidism.

The enzyme trajectory is the most telling clue, and Table 1 and Figure 1 capture it. Hypoxic hepatitis produces a large but short-lived rise in transaminases through injury to the hepatocytes around the central vein, the centrilobular zone that lies furthest from the incoming oxygen supply and suffers first when perfusion drops. (1)

One of its more specific features is a reversal of the AST-to-ALT ratio during recovery (1), and that is exactly what we observed: AST was twice the ALT on admission (1643 versus 819 U/L), drew level by the second day, and had fallen to roughly a third of the ALT by day 6 (54 versus 168 U/L).

The reason is kinetic. AST clears from the blood faster than ALT, so after a single ischemic hit it rises higher and then falls away sooner; viral and autoimmune hepatitis behave differently, with enzymes that stay elevated for weeks.

The lactate dehydrogenase added a second, independent line of evidence. LDH was markedly elevated at 1279 U/L, and the ALT-to-LDH ratio was 0.7. In the work of Cassidy and Reynolds, the mean ALT-to-LDH ratio was 4.65 in acute viral hepatitis but only 0.87 in ischemic hepatitis, and a cut-off of 1.5 separated the two with a sensitivity of 94% and a specificity of 84%. (5)

A ratio of 0.7, sitting almost exactly on their ischemic mean, is therefore strong biochemical support for a hypoxic rather than a viral cause. The preserved synthetic function fits the same picture: ischemic hepatitis characteristically spares the bilirubin and produces only modest coagulopathy, and here the bilirubin was normal, the albumin was 4.4 g/dL and the INR rose only to 1.32. (3)

The minimally elevated troponin and the transient acute kidney injury, both of which recovered, mark a myocardium and a kidney briefly short of flow rather than a primary cardiac or renal event. (2,4)

The patient's age sat squarely within the reported range, where the mean is about 64 years. (4) Where did the low output come from? From the thyroid. Hormone deficiency slows the heart, weakens contraction and raises systemic vascular resistance, and severe disease can add a pericardial effusion and delayed conduction (6).

The result is a hypodynamic circulation, the mirror image of thyrotoxicosis, and when it is severe enough it can lower hepatic oxygen delivery below what the centrilobular hepatocytes will tolerate. Because this cardiac depression lifts once thyroid hormone is replaced, (6) the hemodynamics and the liver enzymes recovered in step, exactly the pattern seen here.

We did not obtain an echocardiogram to image the low-output state, which is a limitation; the documented hypotension, bradycardia and the parallel biochemical recovery nonetheless make the mechanism clear.

The creatine kinase deserves particular comment, because in this patient it does real diagnostic work. AST is not liver-specific; skeletal and cardiac muscle carry it too, and severe hypothyroidism characteristically produces a myopathy that leaks both creatine kinase and AST. (7,8)

The CK here was markedly elevated, at 2946 U/L, and fell to 1652 U/L as the patient recovered, documenting a genuine hypothyroid myopathy. Part of the strikingly high AST, and the 2:1 AST-to-ALT ratio on admission, can therefore be attributed to muscle rather than liver.

The ALT settles what remains, since a value above 800 U/L cannot be explained by muscle injury alone and confirms genuine hepatocellular damage. The case thus carries two parallel consequences of thyroid hormone deficiency at once: an ischemic hepatitis from the low-output circulation, and a hypothyroid myopathy contributing to the aminotransferases.

Ischemia may not be the whole story. Thyroid hormone deficiency can depress hepatocyte metabolism and reduce hepatic blood flow in its own right, and longstanding hypothyroidism is linked epidemiologically to non-alcoholic fatty liver disease. (7,9)

Neither mechanism plausibly accounts for transaminases this high on its own, but either could have compounded the ischemic injury. The timing is the tie-breaker: enzymes that began falling as the blood pressure stabilized, and kept falling after levothyroxine, describe a perfusion-dependent process rather than a smouldering hepatitis.

What may be most instructive is how quietly the endocrine disorder presented. Biochemically the liver looked as though it were failing, yet none of the usual external signs of liver disease were present: no jaundice, no stigmata of chronicity. The clue that unlocked the diagnosis was not hepatic at all but a hoarse voice the patient had carried for weeks, set against a heart rate of 45 and a blood pressure of 60/40.

The liver was simply the loudest sign of a disease that was, in every other respect, hiding. The practical message for the emergency and acute-care setting is straightforward: thyroid function testing is cheap and quick and belongs early in the work-up of unexplained marked hepatocellular injury, particularly in an older patient who is bradycardic, hypotensive, hypothermic or confused, and the lactate dehydrogenase and creatine kinase are worth sending alongside it.

This report has limitations. An echocardiogram was not performed to document the low-output state, and the

adrenal axis was not formally characterized. As a single observation, the case is illustrative rather than conclusive; its value lies in the coherence of the biochemical story and the speed of reversal.

CONCLUSION

Severe Hashimoto's hypothyroidism can declare itself first in the liver. In this patient a slow, underfilled circulation produced marked but fully reversible ischemic hepatitis, supported by a disproportionately raised LDH and a low ALT-to-LDH ratio, alongside a hypothyroid myopathy that accounted for part of the aminotransferase rise; both resolved with fluids and levothyroxine while the endocrine cause stayed otherwise silent. When the hepatic picture refuses to fit a conventional diagnosis, and especially when the heart rate and blood pressure are low, a single thyroid-stimulating hormone may clarify matters faster than any further liver-directed test.

Editorial Statements

Ethics Approval and Consent to Participate
This study involved the analysis of data from human participants. The research protocol was reviewed and approved by the Institutional Ethics Committee and was conducted in accordance with institutional regulations, the principles of the Declaration of Helsinki, and internationally accepted standards for biomedical research ethics.

Data Availability Statement
The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Author Contributions (CRediT): Conceptualization & Methodology: AHL; Data Curation, Investigation and Writing – Original Draft: IT, EV: Formal Analysis: ET: Writing – Review & Editing: ARr: Supervision: AHL. All authors have read and approved the final version of the manuscript.

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