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CASE REPORT

Jaundice Revealing a Descending Goiter

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Abstract

Hepatic involvement, though often asymptomatic, is frequently observed in cases of hyperthyroidism. However, jaundice can occasionally present as an unusual mode of discovery for this condition. We report the case of a 53-year-old patient who sought medical attention at the internal medicine department of the AbassNdao Hospital Center in Dakar (Senegal) for an isolated, non-fluctuating jaundice, without associated pruritus, in a context of cachexia.

Biological investigations revealed elevated serum levels of alanine aminotransferase (ALT: 58 IU/L, 1.4 times the normal), aspartate aminotransferase (AST: 98 IU/L, 2.6 times the normal), alkaline phosphatase (602 IU/L, 2.2 times the normal), gamma-glutamyl transferase (102 IU/L, 3 times the normal), and bilirubin (total: 203.3 mg/L, 20.3 times the normal; conjugated: 148.4 mg/L, 29.6 times the normal).

Complementary biological and radiological examinations excluded various hypotheses: biliary tract obstruction, viral, toxic, or autoimmune hepatitis, primary biliary cholangitis, and primary or secondary hepatic neoplasia. Thoraco-abdominopelvic computed tomography (CT-TAP) revealed a heterogeneous, plunging goiter. Cervical ultrasound confirmed a diffuse, hypervascular, plunging goiter, suggestive of Graves' disease, along with colloid cysts on the left side.

Hormonal analysis showed an elevated free T4 level (43 pmol/L, normal range: 10-25 pmol/L) and a suppressed TSH level (0.017 µIU/mL, normal range: 0.15-4.9 µIU/mL). Anti-TSH receptor antibodies were positive (3.304 IU/L, normal: <1.5 IU/L). These findings led to the diagnosis of hepatic involvement secondary to Graves' disease without palpable goiter or ocular manifestations.

The patient's clinical and biological condition improved under treatment with Benzylthiouracil (Basdene: 250 mg/day). Thus, in cases of unexplained isolated jaundice, screening for clinical and biological signs of hyperthyroidism could uncover unexpected diagnoses.

1. Introduction

The liver plays a key role in the metabolism of thyroid hormones, while these hormones also contribute to the maintenance of normal liver function. Therefore, itis not surprising that liver disorders, although often a symptomatic, are frequently observed in patients with thyroid diseases. This association was first reported by Habershonin 1874 (1). In hyperthyroidism, nonspecific biological abnormalities, such as an increase in liver enzymes and total bilirubin, mayoccur (2). Additionally, atypical clinical forms of Graves' disease, the main etiology of hyperthyroidism, have been described, including rare hepatic manifestations (3). We present here a new case of Graves' disease revealed by cholestatic jaundice, without associated pruritus.

2. Observation

Mr. O.S., 53 yearsold, consulted in December 2022 for skin and mucosal jaundice that had been progressing

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for about two months, without associated pruritus, accompanied by a weightloss estimated at 40% over six months and non-selective anorexia. The history revealed the use of phytotherapy, of unknown nature and dose, initiated after the appearance of jaundice and continued for about five weeks. This phytotherapy had been stopped a month before the consultation due to inefficacy, without any reported aggravation of symptoms. No significant medical history was identified, particularly no known liver disease or medication use. The patient did not consume alcohol and wasa former smoker (15 pack-years), having stopped smoking upon the onset of symptoms.

On clinical examination, distinct conjunctival jaundice was observed, with no scratching lesions or reported pruritus. A sensitive hepatomegaly was present, with a smooth loweredge, a regular surface, and a hepatic span measured at 15 cm. The heart rate was 126 beats per minute. The rest of the clinical examination was normal: no goiter, exophthalmos, splenomegaly, or signs of heart failure.

Biological tests revealed marked cholestasis, with alkaline phosphatases at 602 IU/L (2.2 N), gamma-glutamyl transferase at 3 N, total bilirubin at 20.03 N with a mixed predominance (conjugated bilirubin at 29.6 N), and cytolysis (AST at 2.6 N; ALT at 1.4 N). Prothrombin time was at 82.4%, and albumin levels were low at 24.5 g/L. Viral serologies for hepatitis B and C were negative, as were auto-antibody tests (antinuclear antibodies, anti-mitochondrial, anti-smooth muscle, and anti-LKM1).

Imaging tests showed: a normal abdominal ultrasound; a CT-TAP scan revealing no biliary tract or gallbladder pathology, but showing a heterogeneous descending goiter; a Bili-MRI showing no dilatation of intra- or extra-hepatic bile ducts; a Fibroscan indicating moderate fibrosis (F2) without steatosis (S0).

The thyroid function tests revealed biological hyperthyroidism with an elevated free T4 at 43.8 pmol/L (1.72 N) and a collapsed TSHus at 0.017 µIU/mL (norm: 0.15-4.9 µIU/mL).

Anti-TSH receptor antibodies were positive at 3.304 IU/L (2 N). A thyroid ultrasound confirmed a hypervascular descending goiter, with colloidalcysts in the left lobe, suggesting a diagnosis of Graves' disease.

Treatment with methimazole and propranolol was initiated. The clinical progression showed significant improvement, with a progressive regression of jaundice and aweight gain of 7 kg in one month. Biological tests performed 30 daysafter the start of treatment showed a decrease in free T4 to 38.27 pmol/L, TSHus to 0.11 µIU/mL, alkaline phosphatases at 396 IU/L (1.01 N), gamma-glutamyl transferases at 88 IU/L (1.4 N), and normalization of transaminases (AST at 28 IU/L; ALT at 24 IU/L). Total bilirubin had decreased to 12.8 mg/L (1.2 N), with conjugated bilirubin at 9.2 mg/L (1.8 N).

Table 1. *Comparative Table of Clinical-Biological Parameters*

ASAT in UI/L	ALAT in UI/L	TSHus in uUI/mL	T4L in Pmol/L	PAL in UI/L	GGT in UI/L	Total Bilirubin	Conjugated Bilirubin	Weight	Jaundice
At the time of diagnosis	98	58	0.017	43	602	102	203.3	148.4	59
One month after the start of treatment	28	24	0.11	38.27	396	88	12.8	9.2	66

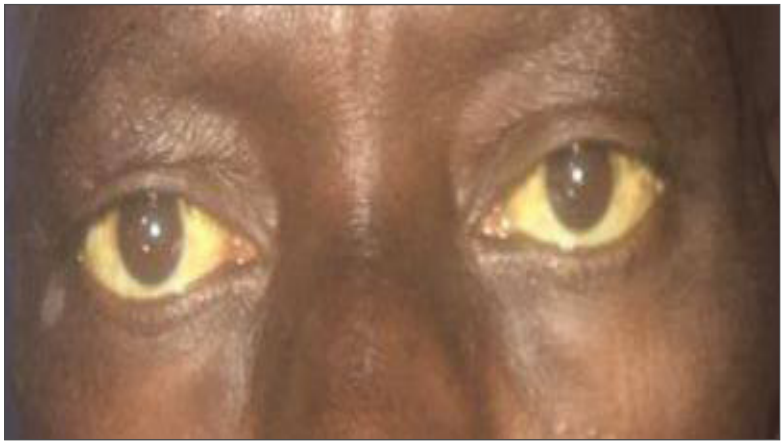

Image 1. *Appearance of the eyesbefore the start of treatment*

Image 2. *Appearance of the eyes one monthafter the start of treatment*

3. Discussion

Hepatic manifestations are among the common clinical presentations of hyperthyroidism. The interactions between the liver and the thyroid are numerous and complex. The liver plays a crucial role in the metabolism of thyroid hormones (4), whilethese hormones are essential for maintaining normal liver function and participate in the metabolism of various substances, including bilirubin and bile acids (4). According to literature data, liver damage is reported in 37 to 78% of cases (5), although it is often a symptomatic. In the absence of cardiac decompensation, jaundice is observed in about 5 to 11% of cases (6,7).

Biologically, the most frequent abnormality is an increase in serum alkaline phosphatase activity, found in 64% to 70% of cases (8,9). This increase is generally due to an elevation in the bone fraction of the enzyme (9). Gamma-glutamyl transferase (GGT) levels are elevated in 17% of cases (9), while alanine aminotransferases (ALT) are elevated in 28% to 37% of cases (9,10). Hepatic injury can be associated with an acute thyroidstorm, hyperthyroidism with cardiac decompensation, concomitant liver disease, or the toxicity of synthetic antithyroid treatments.

In this case, the patient had Graves' disease complicated by liver involvement, without signs of associated cardiac dysfunction. The hepaticorigin was attributed to thyrotoxicosis, after excluding causes such as drugtoxicity, biliary tract obstruction, viral hepatitis B or C, autoimmune hepatitis, or primary biliary cholangitis. Further more, no signs suggested an acute thyroidstorm. The clinical improvement observed under treatment with synthetic antithyroid agents strengthens the hypothesis of liver damage related to thyrotoxicosis.

Hepatic injury secondary to isolated thyrotoxicosis, in the absence of cardiac complications, was first documented in 1874 (3). Sincethen, several similar cases have been reported in the literature (11,12). However,theexactmechanismsinvolvedremainpoorly understood. The hypothesis of a direct toxic effect of thyroid hormones has not yet been demonstrated (13). On the other hand, the hypermetabolic state characteristic of hyperthyroidism could increase the liver'soxygen requirements (14). This imbalance between increased oxygen consumption and insufficient hepatic perfusion could induce tissue hypoxia, responsible for the biological abnormalities and cholestasis (15).

Liver biopsies performed in these patients show non specific abnormalities, such as intracellular cholestasis, predominant in the centrolobular areas, a lymphocytic infiltrate in the portal spaces, hepatocyte vacuolization, Kupffercell hyperplasia, and fibrosis around the suprahepatic veins (7).

4. Conclusion

Hepatic enzyme abnormalities are common in hyperthyroidism, but they most often present a symptomatically. However, the literature reports a few isolated cases of Graves' disease initially manifesting as jaundice. This observation emphasizes the need to systematically explore clinical and biological signs of hyperthyroidism in the case of unexplained jaundice.

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