

Acute Icteric Hepatitis as a Presentation of Grave's Thyrotoxicosis

Doraiswamy A¹ Henry A¹ Kamaruddin NA²

1 Department of Medicine, Hospital Melaka, Melaka, Malaysia

2 Endocrine unit, Department of medicine, National University Medical Centre, Kuala Lumpur, Malaysia



MINISTRY OF HEALTH
MALAYSIA

INTRODUCTION

Graves' thyrotoxicosis has been known to affect other organ systems in the body including the liver. However, severe hepatitis in this clinical context is quite rare. There are several postulated mechanisms, including liver abnormalities due to its association with autoimmunity, liver derangement from thyrotoxic heart failure and concomitant liver enzyme abnormalities in the setting of a hyperthyroid state.

CASE

We report a 55-year-old man who presented with marked weight loss and jaundice. In addition, he had a diffuse goiter (Figure 1) with fine tremors in the hands. Laboratory work-up revealed thyrotoxicosis with FT4 of 87.9 pmol/l and TSH of <0.01mIU/L. He had abnormal liver function with total bilirubin 258.3 umol/l, Direct Bilirubin 217 umol/l, Indirect Bilirubin 41.2 umol/l, ALP 306 U/L and ALT at 54 U/L. Serological tests excluded viral hepatitis. Ultrasound of the thyroid showed a diffuse goiter with increased vascularity (Figure 2). Ultrasound hepatobiliary system together with MRCP were normal. In view of the hepatitis, he was given a potassium iodide solution and dexamethasone (10mg daily) to prevent the peripheral conversion of T4 to T3. The jaundice and liver function test improved within a few days. Carbimazole 20mg daily was slowly commenced with the resulting drop of FT4 to 20.6 pmol/l. However the ALP increased to 359mmo/l requiring the reduction of carbimazole dose down to 10mg daily. Subsequent FT4 came down to 15 pmol/l (Table 1 shows thyroid and liver profile)and he successfully underwent an RAI therapy 2 months later, which was uneventful. He was noted to be hypothyroid 2 months after RAI. Serum bilirubin and ALT normalised as thyroid function improved.

INVESTIGATIONS

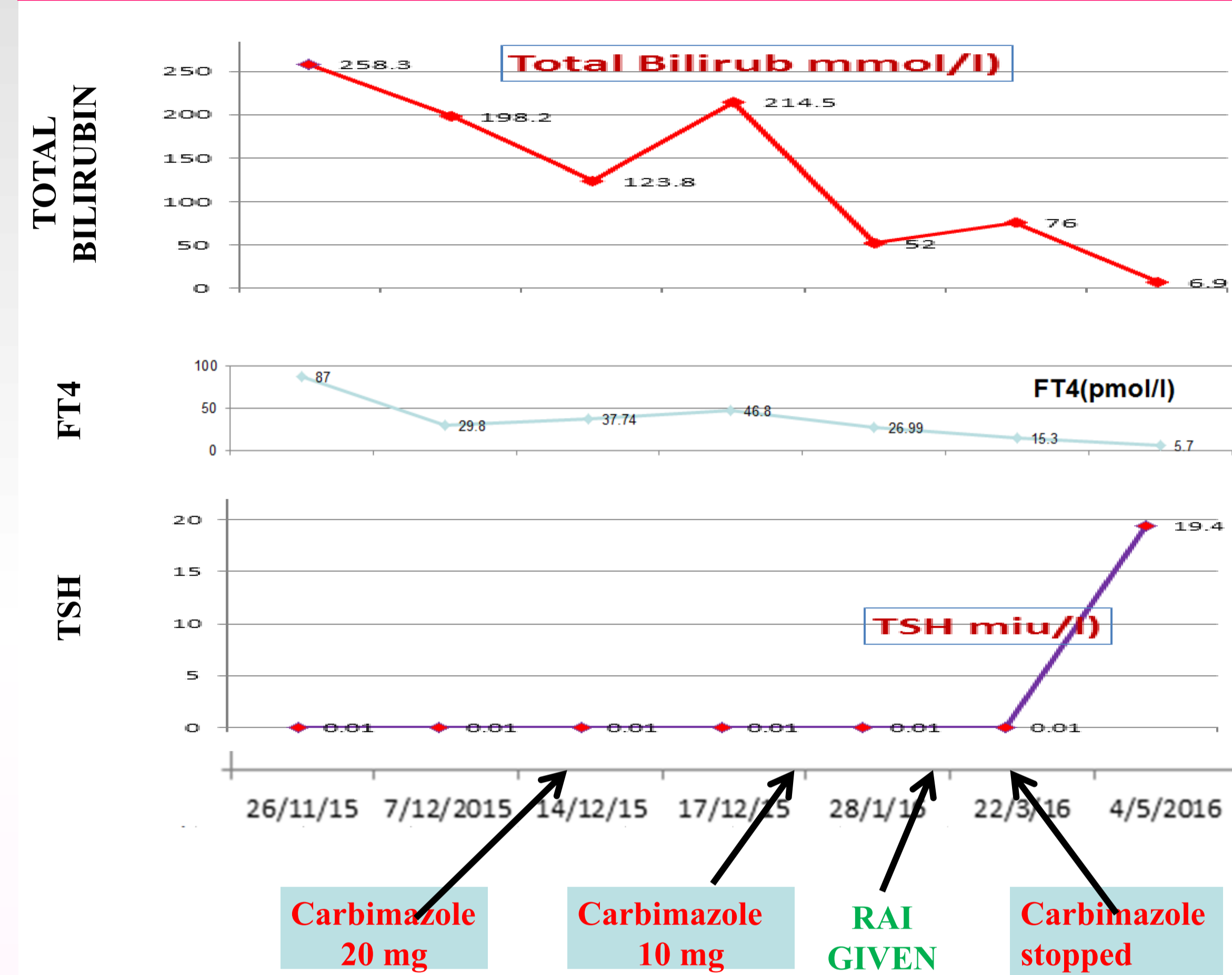


FIGURE 1- Showing diffuse goiter

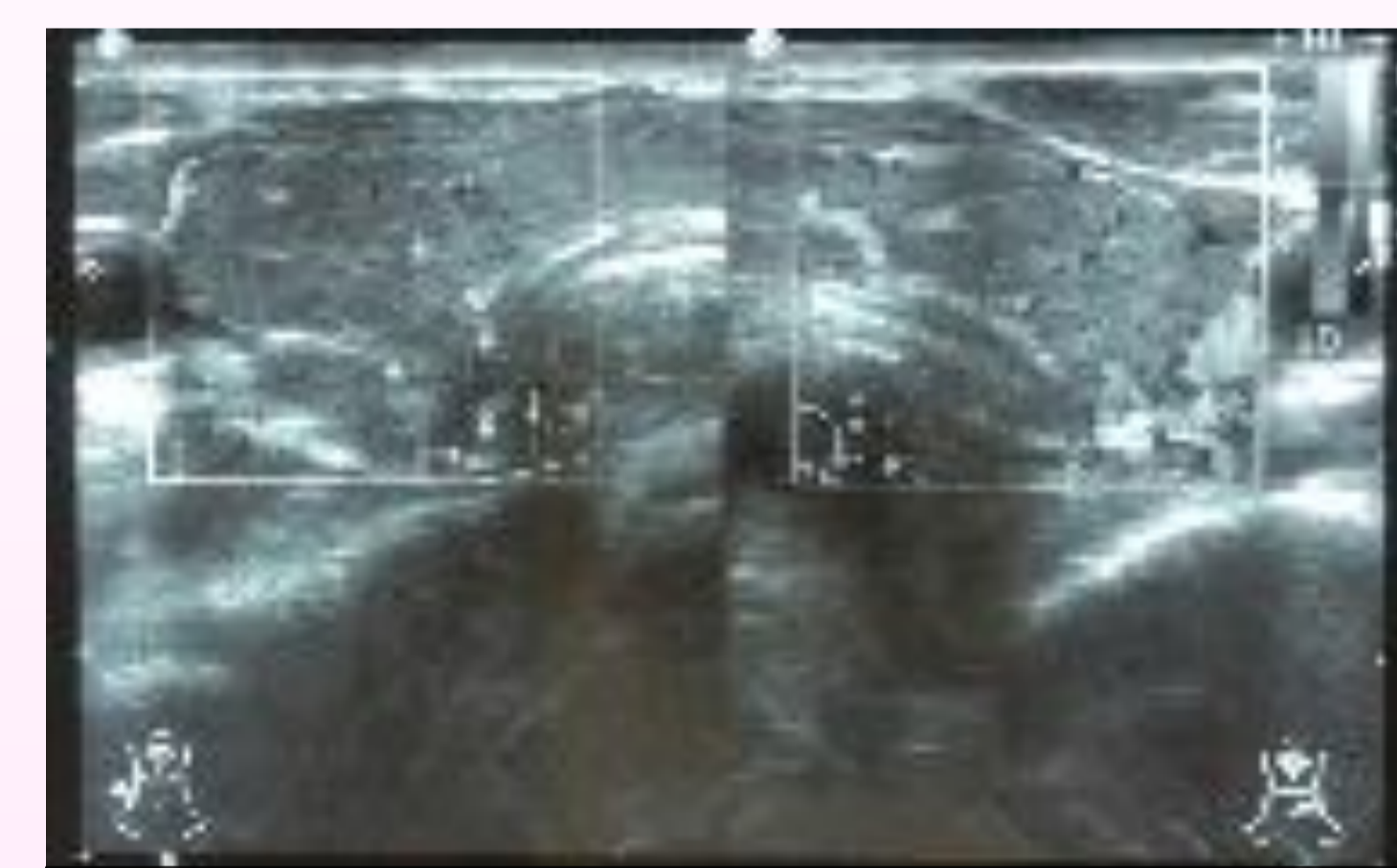


FIGURE 2- Ultrasound showing diffuse goiter with increased vascularity

DISCUSSION

We present a case of Grave's disease manifested by jaundice. Liver is the primary organ of thyroid hormone metabolism, on the other hand thyroid hormones have a major role in normal hepatic function. Mild liver abnormalities such as hypoalbuminemia and increased AST ALT and ALP may be seen in up to 45-90% of patients with hyperthyroidism. Clinically patients present with self limiting hepatitis, with mild elevations in serum bilirubin in up to 5% of patients with thyrotoxicosis. Jaundice in thyrotoxic patients may be due to heart failure or rarely cholestatic jaundice. Acute icteric hepatitis occurs in less than 1% of Graves' thyrotoxicosis, posing a management dilemma. This has to do with the close association between liver enzyme abnormalities and oral antithyroid medications. Propylthiouracil for example, may cause elevation in aminotransferase levels (28%) usually 2 months after administration of the drug, though this tends to be asymptomatic and transient despite the continuation of therapy (Tzemanakis et al). Carbimazole on the other hand may also result in fulminant hepatitis, usually 6 weeks after commencement of therapy. In our patient the elevated aminotransferase level preceded the administration of the drug which lead to rapid aggravation of the hepatocyte damage. In conclusion, it is apparent that the thyroid and liver are intertwined in many ways, a vigilant effort should be undertaken to diagnose the liver condition in patients with thyrotoxicosis so that appropriate therapy can be initiated.

References

1. Tzemanakis et al. *Ann of gastroenterol* 200, 13(1):54-57
2. Yao LDC et al, *Cholestatic jaundice in hyperthyroidism. Am J Med* 1989;86:619