Introduction

Thyroid storm or thyrotoxic crisis is an extreme situation in hyperthyroidism, characterised by the presence of life threatening clinical features like hyperthermia, tachycardia, severe agitation and altered mental status. Severe liver dysfunction in this clinical setting is quite rare. Jaundice in such patients is usually attributed to congestive heart failure. We report a 53 year old female patient with fever and jaundice as the presenting manifestation of thyroid storm which was a diagnostic dilemma and therapeutic challenge.

Case Report

A 53 year old female was admitted with a history of low grade fever for 1 month with jaundice, anorexia and frequent loose motions for two weeks. She also noticed tremulousness of hands and had significant weight loss (8 kg in the past four months). The patient had no history of any chronic disease or prolonged drug intake or blood transfusion or contact with tuberculosis or travelling.

The patient was severely emaciated with height of 162 cms and weighed 35 kgs (BMI=13.3 kg/m²). On initial examination, the patient was febrile (100° F) and was lethargic. Her level of consciousness on Glasgow Coma Scale was 15. There was icterus and fine tremors of hands. The blood pressure was 110/60 mm Hg and the pulse rate was 120/min (regular). Liver was not visibly enlarged it was soft and diffusely enlarged on palpation (grade 1 goitre). There were no signs of ophthalmopathy.

Investigations revealed:

- Hemoglobin 9.4 g/dl, TLC 10400/ cumm and platelet count 1.93 lac/cumm. Blood urea was 22 mg% and serum creatinine was 0.62 mg%. Liver function revealed serum bilirubin(total)-16.5 mg/dl and (direct)-10.9 mg/dl, SGPT-40 U/L, SGOT-67 U/L, serum protein(total) – 5.6 gm%, serum albumin – 2.1 gm%, ALP- 193 U/L, prothrombin time-32.5 sec. Screening for hepatitis A, B, C & E viruses were negative. Upper gastrointestinal endoscopy did not reveal any abnormality. USG thyroid showed a diffuse goiter with increased vascularity. The liver was of normal size and did not demonstrate distension of hepatic veins. Her CXR P/A view was normal and ECG showed sinus tachycardia. 2D Echo showed no significant abnormality. Autoimmune markers such as antinuclear antibody were negative. Thyroid hormone levels showed: - TSH<0.01uIU/L (N: 0.40 – 4.00 uIU/L), T3-2.66 ng/ml (N: 0.58 – 1.59 ng/ml), T4- 15.85 µg/ml (N: 4.87 – 11.72 µg/dl). Thyroid scan and auto antibody tests could not be performed because of her moribund condition. In view of clinical and biochemical evidence of thyrotoxicosis, the patient was treated with beta blockers (Propranolol 10 mg 8hrly), iv Hydrocortisone 100mg iv stat and then 50 mg 8 hrly, Tab Neomercazole 30 mg stat and then 10mg 8 hrly. After ½ hour of neomercazole, Lugol’s Iodine 8 drops in half glass water 8hrly was given (treatment with Lugol’s iodine was stopped after 7 days). On the ninth day of admission the patient got drowsy and her level of consciousness on the Glasgow Coma Scale decreased to 8. The patient was tachypneic and her temperature was 102° F. She worsened over the next two days and had persistent tachycardia (Pulse rate-120) with hypotension (Blood pressure- 80/60) and unfortunately expired due to cardiac arrest.

Discussion

Grave’s disease accounts for 60-80% cases of thyrotoxicosis. The prevalence varies among populations, depending mainly on iodine intake. The liver is the primary organ of thyroid hormone metabolism. Upto 85% of extrathyroidal deiodination of T4 to T3 and reverse T3 occurs in liver and plasma binding proteins of thyroid hormone are produced by liver. Mild liver abnormalities such as hypoalbuminemia and increased serum level of AST, ALT and ALP may be seen in 45% to 90% of patients with hyperthyroidism. Clinically, patients present with self limited hepatitis, with mild elevations in serum bilirubin is seen in upto 5% of patients with thyrotoxicosis.

Possible mechanisms responsible for liver abnormalities in thyrotoxicosis are venous congestion due to high output heart failure and/or relative hypoxia. Massive hepatomegaly and fulminant hepatic failure have been rarely described. Cholestatic hepatitis without heart failure is rare. Thyrotoxicosis may increase oxygen demand and utilization which cannot be totally compensated by hepatic blood flow. The pericentral areas of hepatic acini constitute the

Abstract

Thyrotoxic crisis is a life threatening medical condition that requires urgent diagnosis and treatment. Because of the wide variety of presenting symptoms, its diagnosis can be difficult in some cases and a high index of suspicion is required for diagnosis. We present a case of 53 year old patient who presented with fever, jaundice and passage of loose stools. Upon investigations other etiologies for jaundice and passage of loose stools. Upon investigations other etiologies for jaundice were ruled out and she was found to be in thyrotoxic crisis causing liver injury.

Jaundice Heralding the Onset of Thyrotoxic Crisis

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most zone susceptible to ischemia. The hyperbilirubinemia is not well understood and there are no available data supporting the direct toxic effects of thyroid hormones on the liver. Liver biopsy findings in patients with mild hyperthyroidism-related hepatic injury are non-specific. Biopsy specimens may reveal lobular inflammation with some infiltrate consisting of polymorphonuclear leukocytes, eosinophils and lymphocytes. In addition, some patients may exhibit focal or diffuse centrilobular necrosis and perivenular fibrosis.4

The reported patient presented with acute icteric hepatitis and thyroid storm, due to untreated prolonged hyperthyroidism. In the absence of viral hepatitis, drug-induced and autoimmune diseases, congestive heart failure or ischaemic hepatitis (in our patient ECG showed sinus tachycardia and 2D Echo had no significant abnormality), it was inferred that thyrotoxicosis had caused Acute Hepatitis. Serologic testing excluded the usual causes of viral hepatitis and ANA was also negative. Other serological markers for autoimmune hepatitis could not be done.

Although rare, but anti thyroid drugs such as propylthiouracil and methimazole (or its pro drug carbimazole) are known to cause hepatotoxicity. Histopathologically, propylthiouracil causes toxic hepatitis with necrosis and carbimazole causes cholestatic hepatitis without necrosis.5 The mean duration after initiation of treatment for symptoms to appear is 36 days for carbimazole and the hepatotoxicity is dose independent.5 But our patient had deranged liver function tests even before starting neomercazole and previously she was not on any other hepatotoxic drugs which rules out any etiology for drug induced hepatitis.

In conclusion, we have presented this case to increase the awareness of this treatable condition amongst the physicians. The diagnostic and therapeutic exercise in such a case can be quite challenging. Severe hepatitis may rarely occur as a presenting feature of thyrotoxicosis and therefore warrants the term “thyrotoxic hepatitis.” Thyroid function tests should be part of the investigation of unexplained hepatitis. The mechanism of this condition is not completely understood. Antithyroid drugs with or without corticosteroids are crucial in this life-threatening hepatitis.

References