synaptophysin negative, suggestive of solid pseudopapillary tumor rather than a neuroendocrine tumor.

Laparotomy was done and the gall bladder was found to be absent. A pancreatic mass of size 5 × 5 cm was found in the head of pancreas (Figure 1B). The mass was enucleated and sent for histopathology. The report described a well circumscribed non-encapsulated neoplasm with extensive areas of fibrosis, necrosis, hemorrhage and inflammatory cell infiltrate. Few areas showed cystic dilated glandular spaces containing papillary processes lined by cuboidal and columnar epithelium containing oval nuclei with coarse chromatin (Figure 2A). Some areas showed solid groups of similar appearing cells (Figure 2B). Based on the above findings a diagnosis of solid pseudo papillary tumor was made. The post operative period was uneventful and the patient is doing fine after 3 months of enucleation.

Discussion

Frantz tumor or solid pseudo papillary tumor (solid cystic papillary tumor of pancreas) is a borderline tumor. It accounts for less than 1% of pancreatic tumors. Gall bladder is congenitally absent in 0.013 to 0.075% of general population. So we are uncertain whether the absent gall bladder in our patient is it an incidental finding or is associated with the pseudo papillary tumor. 15% of these patients present with metastasis. The treatment of choice is resection. Some surgeons prefer enucleation for small tumors. Given a 5-year survival greater than 95%, radical surgery is not recommended for this neoplasm. But metastatic disease may require complete surgical excision with metastatectomy.

Cholestatic hepatitis in a patient with Graves’ disease resolved with total thyroidectomy

Introduction

Hepatic dysfunction has been recognized as a rare complication of hyperthyroidism since Habershon first reported it in 1874. The use of the antithyroid medications propylthiouracil, methimazole, and carbimazole has been associated with toxic hepatitis and cholestatic jaundice. Therefore, avoidance or withdrawal of such drugs in patients with severe thyrotoxicosis could expose critically ill patients to the potentially lethal complications of hyperthyroidism. Clinically evident cholestasis secondary to hyperthyroidism is rare. The mechanism is not clear, but multiple factors are believed to play part in the pathogenesis, including heart failure, infection, and weight loss. Once hyperthyroidism is controlled, cholestasis will improve, as demonstrated in this report. Untreated hyperthyroidism alone can cause liver function test abnormalities, but jaundice is uncommon and usually occurs in cases of hyperthyroidism complicated by congestive heart failure.

References

failure and secondary hepatic dysfunction. Liver injury caused by hyperthyroidism is relatively common. Increases in levels of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were reported in 27% and 37% of hyperthyroid patients, respectively.

Case Report

A 43 year old African American male with history of Graves’ disease medically managed with propylthiouracil and potassium iodine for 3 months was referred to us. The patient also had cholestatic hepatitis confirmed with a previous liver biopsy. The patient was referred to our institution from an outside clinic for definitive treatment of Grave’s disease after failure of medical treatment. Initial symptoms of his disease included neck discomfort associated with difficulty in swallowing and progressed to include anxiety, difficulty in sleeping, 50 pounds weight loss over a two month period, and hyperreflexia. Prior to our initial clinic visit the patient was also undergoing treatment for generalized pruritus and papular excoriated lesions secondary to cholestatic hepatitis. Physical exam was significant for icterus, jaundice under his tongue, and thyromegaly with substernal extension.

Laboratory investigations revealed significant suppression of thyroid stimulating hormone (TSH) (<0.01 IU/L) and a high free serum T4 (3.39 ng/dl). Hepatic function tests revealed total bilirubin levels at 39 mg/dl, an abnormal alkaline phosphatase of 1256 U/L, an AST and ALT of 53 U/L and 30 U/L respectively, and an albumin of 3.6 g/dl. Coagulation studies were within normal limits. Thyroid uptake scan revealed evidence of diffuse symmetric uptake of radiiodine consistent with Graves’ disease.

Thyroid ultrasound revealed evidence of thyromegaly with diffuse increase vascularity consistent with Grave’s disease. Due to associated compressive symptoms, failure of medical treatment and progressive cholestatic hepatitis, it was determined the patient was an appropriate candidate for definitive surgical treatment of Grave’s disease. Total thyroidectomy with cervical approach for a substernal goiter was performed with no complications. His postoperative course was uneventful and the patient was discharged home the next day. At one month follow up the patient’s symptoms of hyperthyroidism and cholestatic hepatitis had resolved. Laboratory studies demonstrated significant improvement revealing total bilirubin 0.5 mg/dl, direct bilirubin 0.3 mg/dl, and alkaline phosphatase of 204 U/L. TSH was elevated at 21.9 and the patient was instituted thyroid hormone replacement by his treating endocrinologist.

Discussion

Features indicative of a role of the endocrine disease in the pathogenesis of the cholestatic syndrome were: the appearance of liver damage in conjunction with the clinical onset of thyroid hyperfunction and its disappearance with the amelioration of hyperthyroidism; the absence of congestive heart failure and of infectious, toxic or obstructive agents of liver damage; and the pathological and biochemical findings of intrahepatic cholestasis. Hyperthyroidism can be rarely complicated by a severe cholestatic syndrome that may dominate the clinical presentation and course.

The co-existence of liver disease and thyroid imbalance has been well documented in literature. The premise that excess thyroid hormone damages the hepatocyte is consistent with findings that liver enzymes are elevated in patients with inadequately controlled Grave’s disease and in cases of subacute thyroiditis. This is further supported by studies which demonstrated a protective role of hypothyroidism in cirrhotic liver disease. Although an association between the two entities has been known for more than a century, the exact mechanism by which thyroid hormone causes hepatocyte damage is unclear. Numerous explanations are plausible, including direct toxic effects of thyroid hormone on the hepatocytes; impaired distant organ function affecting hepatic function, and relative hepatic ischemia secondary to peripheral vasodilation. Thyroid hormone increases the activity of the Na+/K+ pump in tissues, resulting in an increase in metabolic requirements. It is likely that, without a concomitant increase in blood flow to the liver, this metabolic stimulation stresses the hepatocytes, thereby inducing damage. In this sense, the metabolic demand exceeds supply. In addition, the liver is responsible for clearing a significant proportion of circulating thyroid hormone, and chronic liver disease can be associated with clinical hyperthyroidism.

The hepatic injury associated with hyperthyroidism can vary in intensity from mild liver function test abnormalities to severe central hepatic ischemia. The frequency of jaundice varies from 5.3% in uncomplicated hyperthyroid patients to 79% if hyperthyroidism-induced congestive heart failure is present. In conclusion, Graves’ disease may manifest with marked intrahepatic cholestasis, and adequate surgical treatment of the thyrotoxicosis can lead to resolution of all
clinical and biochemical signs of cholestasis.

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References


Iatrogenic gastro-renal fistula

Introduction

Congenital pelviureteric junction obstruction can present clinically at any time of life. In neonates and infants it presents as a palpable flank mass. Older children and adults present with intermittent abdominal or flank pain, nausea, vomiting, hematuria or hypertension. Here we present a case of congenital pelviureteric junction obstruction misdiagnosed as pseudocyst of pancreas.

Case report

A 30 year old unmarried male was referred to us by a general surgeon with a diagnosis of recurrent pseudocyst of pancreas. The case had been operated by the same surgeon around 11 years ago. The patient was suffering from unrelieved left upper abdominal pain. On abdominal examination a left upper paramedian scar was noted. A 12×10 cm mass was palpable in the left lumbar and hypochondriac regions. The mass was bimanually palpable and ballotable, suggesting a left renal origin.

A prior ultrasound abdomen of the patient showed a pseudocyst of pancreas. Contrast enhanced computed tomography (CECT) of abdomen was performed with oral and intravenous contrast administration (Figure 1). It revealed gross left hydronephrosis with thinned out renal parenchyma. The pancreas and gastrointestinal tract were normal. In light of these findings a left pelviureteric junction obstruction was suspected. A technitium 99m diethyltriamine penta acetic acid (DTPA) renogram was performed which revealed hydronephrotic, nonfunctioning left kidney due to pelviureteric junction obstruction. The right kidney was normal. A decision was taken to perform a left simple nephrectomy.

Figure 1: CECT scan abdomen showing left gross hydronephrosis with thinned out parenchyma