Case Report

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An unusual cause of encephalopathy in compensated cirrhosis

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An unusual cause of encephalopathy with renal failure in a patient with treated HCV cirrhosis

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Abstract

A 63-year-old teetotaller male, previously treated for hepatitis C-related compensated cirrhosis, presented with acute-onset encephalopathy with no focal neurological deficit and stable vitals. Investigations revealed elevated serum creatinine (2.94 mg/dL), hypercalcemia, hypophosphatemia, and high serum PTH levels. He was diagnosed with right parathyroid adenoma (1.3×1.2×0.7 cm) with the help of a neck ultrasound. His encephalopathy and renal failure persisted despite adequate IV fluids, calcitonin, and bisphosphonates. Urgent hemi-parathyroidectomy was performed on day four, following which he recovered completely.

Keywords: Hepatic encephalopathy; hypercalcemic crisis; metabolic encephalopathy; parathyroid adenoma.

Introduction

Hypercalcemia in the background clinical history of liver disease has been reported on very few occasions like hepatocellular carcinoma or cholangiocarcinoma[1,2] with cirrhosis and hepatoma without cirrhosis.[3] The incidence of hypercalcemia in patients with liver disease without a liver tumor is extremely rare. Here, we present the case of a 63-year-old male with history of compensated chronic liver disease (CLD) who presented with encephalopathy and acute renal failure (ARF) secondary to hypercalcemia. Parathyroid adenoma was detected on ultrasonography of the neck. Early diagnosis and treatment in the form of hemi-parathyroidectomy achieved the timely clinical cure of the ARF and encephalopathy, which often mimic the hepatorenal syndrome (HRS) and hepatic encephalopathy, respectively, in the setting of CLD.

Case Report

A 63-year-old man teetotaler with a history of hepatitis-C-virus-related compensated cirrhosis treated 4 years ago and in virologic remission presented with progressively-worsening confusion and agitation for one day. On presentation, he was drowsy, poorly responding to verbal commands, and had no focal neurological deficit. There was no history of fever, headache, vomiting, constipation, or any drug intake. Physical examination revealed stable vital signs, with no clinical evidence of any hepatic decompensation in the form of jaundice, ascites, or upper gastrointestinal bleeding. He was started empirically on broad-spectrum antibiotics and anti-hepatic coma measures after sending blood and urine cultures. Biochemistry revealed a hemoglobin level of 11.4gm/dL, white cell count of 7280/mm^3, platelet count of 87.000/mm^3, INR of 1.8, serum creatinine level of 2.94 mg/dL (with the last known baseline of 0.8 mg/dL about seven days earlier), BUN level of 41 mg/dL, serum magnesium level of 1.8 mg/dL, serum bilirubin level of 1.2 mg/dl, serum ALT level of 25 U/L, serum AST level of 32 U/L, serum GGT level of 95 U/L, serum alkaline phosphatase level of 128 U/L, albumin level of 3.5 g/dL and alpha-fetoprotein level of 0.68 ng/ml. The total serum calcium level was 15.9 mg/dL (normal range 8.8–10.2) and ionized calcium level was 8.02 mg/dl (normal range 4.6–5.4) were high, there was hypophosphatemia (S.Po4 -1 mg/dL) with a high serum parathormone (PTH) level of 297 pg/ml. His 25-hydroxy- and 1, 25-dihydroxy Vitamin D levels were low, at <14 ng/mL and 13 pg/mL, respectively. His serum ammonia level was normal (41 umol/L). His electrocardiogram showed a QT interval of 360 mSec. Chest X-ray and urinalysis were normal. USG abdomen showed features suggestive of CLD with portal hypertension, with no evidence of free fluid or any space-occupying lesion. Thyroid ultrasound revealed a hypoechoic focal solid lesion measuring 1.3×1.2×0.7 cm, with lobulated margins adjacent to the lower pole of the right thyroid lobe (Fig. 1). Treatment for hypercalcemia was initiated with IV fluids, IV calcitonin (4 IU/Kg every 12 hours for 3 days), and IV bisphosphonates. He continued to be in encephalopathy with progressive non-oliguric renal failure despite adequate treatment. Urgent right inferior parathyroidectomy was performed on day 4 of admission in view of his non-responsive hypercalcemic crisis. Histopathology showed a parathyroid neoplasm composed of variable sizes of chief cells separated by dense, broad bands of fibrosis (Fig. 2a) with positive chromogranin A immunostaining (Fig. 2b) with no signs of vascular or perineural invasion. Soon after surgery, his serum calcium and parathyroid hormone levels returned to normal; and his

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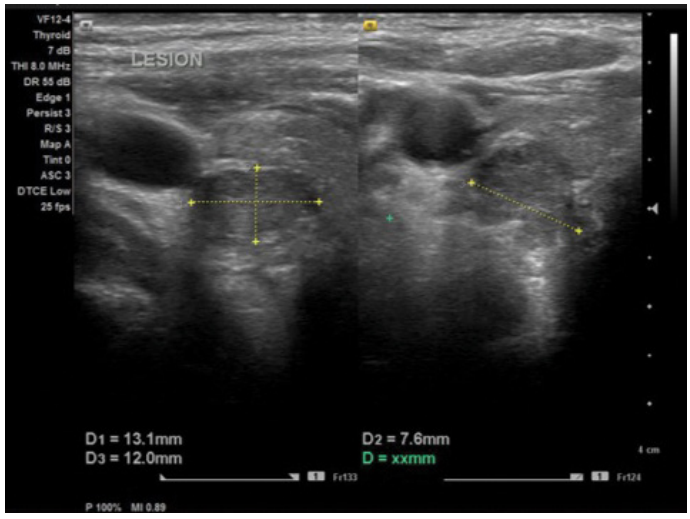

Figure 1. Thyroid ultrasound showing a hypoechoic focal solid lesion with lobulated margins measuring 1.3x1.2x0.7 cm.

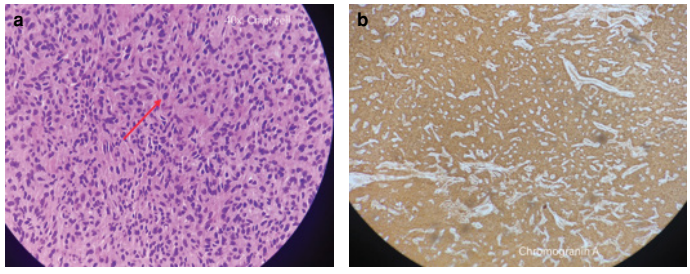

Figure 2. (a) Parathyroid histopathology showing a parathyroid neoplasm composed of irregularly-sized chief cells separated by dense, broad bands of fibrosis. **(b)** Immunostaining showing positive chromogranin A.

renal failure and encephalopathy resolved. He was discharged in a stable condition. He did not develop hungry bone syndrome in the postoperative period.

Discussion

The most common disorder of calcium metabolism associated with chronic liver disorder is hypocalcemia. Advanced CLD is not a known cause of hypercalcemia except in the setting of iatrogenic or hepatic neoplasia.[4–6] Hypercalcemic coma can be confused with hepatic encephalopathy, as was the case in our patient. Hypercalcemia-induced renal failure can also be misdiagnosed as HRS. Hypercalcemia is broadly classified as PTH-mediated and Non-PTH mediated hypercalcemia. Our patient had hypercalcemia of primary hyperparathyroidism due to parathyroid adenoma. Effective management of hypercalcemia can successfully reverse both of these complications. Treatment options for hypercalcemia include IV hydration with Saline, weight-based calcitonin doses, calcimimetic agents, bisphosphonates, dialysis, and management of the underlying pathology. The treatment modality is chosen on the basis of emergency of the treatment and underlying etiology of hypercalcemia. Our patient had a very good outcome, and his encephalopathy and renal failure resolved completely after parathyroidectomy. Hypercalcemia as an etiology of encephalopathy is less often considered in the differential diagnosis of hepatic encephalopathy and should be considered in patients with cirrhosis with non-resolving encephalopathy. Early diagnosis and aggressive management of hypercalcemia improve the outcome of such a patient with hypercalcemic crises.

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