CASE REPORT

A Diagnostic Journey

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Abstract

We describe the case of a patient who came with features suggestive of diabetic ketoacidosis. On further evaluation of DKA, we found that it was caused by acute pancreatitis. This acute pancreatitis was found to be caused by hypercalcemia, which was in turn due to primary hyperparathyroidism. Imaging studies done for hyperparathyroidism revealed a thyroid nodule which later turned out to be malignant. This patient was also incidentally found to have hypertrophic obstructive cardiomyopathy.

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Introduction

We describe the case of a patient whose disease required a diagnostic journey. The patient was a 55-year-old housewife from Mumbai, Maharashtra, India, with no history of any prior comorbidities but for a double J (DJ) stent placement for an obstructive right ureteric calculus done a week prior to admission on 13th June 2022. After her successful treatment for the calculus, she was discharged home only to get readmitted to another hospital on 20th June 2022 with complaints of abdominal pain and vomiting. An ultrasound (USG) abdomen was done, which was suggestive of acute pancreatitis, following which she was brought to the emergency room of Hinduja Hospital on 22nd June 2022 for further management.

Case Description

Clinical Features and Examination

On arrival to the emergency room, the patient was conscious, cooperative, and well oriented to time, place, and person.

Her pulse was 100/minute, regular. The blood pressure was 110/80 mm Hg in the right arm in a recumbent position.

Respiratory rate was 20/minute and oxygen saturation was 96% on room air.

Her blood glucose level was checked and it was 546 mg/dL!

General examination showed no pallor, icterus, clubbing, cyanosis, or edema. No lymph nodes were palpable in the neck. No nodules in the thyroid gland were palpable.

Abdominal examination revealed tenderness present diffusely over the entire abdomen, more marked in the epigastric region.

Examination of the cardiovascular system revealed audible first and second heart sounds. A systolic murmur was audible in the neo-aortic area. The character of the murmur could not be assessed due to tachycardia.

Examination of the respiratory and nervous systems did not reveal anything of significance.

Immediate Investigations and Management

A complete blood count, renal function test, and electrolytes were sent along with amylase, lipase, and hemoglobin A1C (Hba1c) level. Stat urine ketone dipstick was done, which showed 2+ (moderate) ketonuria.

Arterial blood gas analysis was also done, which showed a pH of 7.32 with a bicarbonate of 18 and a partial pressure of carbon dioxide of 30. The anion gap was only 12.

The patient was started on intravenous crystalloids. A bolus of 500 mL 0.9% normal saline was given, followed by crystalloids at 100 mL/hour overnight. She was also given intravenous potassium correction. Insulin therapy was started with a bolus of 0.1 units/kg, followed by infusion of 0.1 units/kg/hour. She responded to insulin well, and her blood sugars reduced to 200 mg/dL over the next several hours. Once the ketoacidosis had resolved and the patient started taking food orally, she was switched over to subcutaneous insulin after a brief overlap period.[1]

The complete blood counts showed a total white blood cell count of 22,000 with 90% neutrophils. HbA1c was 6.5%, indicating a relatively recent worsening of her blood sugars. The amylase and lipase, which were sent, turned out to be 400 and 260, respectively, supporting the diagnosis of pancreatitis.

It was determined that the diabetic ketoacidosis was likely to have been caused by acute pancreatitis. Since common causes of acute pancreatitis are gallstones, alcohol, hypertriglyceridemia, and hypercalcemia, relevant investigations were sent.[2]

The USG report available did not show any evidence of gallstones. The patient also did not have any history of recent or past alcohol intake. Her serum triglyceride levels were done, which were not elevated (121 mg/dL).

The calcium levels turned out to be 13.6 with albumin of 2.0, resulting in a corrected calcium level of 15.2! Thus, it was determined that acute pancreatitis was most probably caused by hypercalcemia. Hypercalcemia also explains the lower than expected anion gap in diabetic ketoacidosis.[3]

Common causes of hypercalcemia include parathormone (PTH) independent causes like iatrogenic hypercalcemia due to hypervitaminosis D, lytic lesions of the bone including multiple myeloma, and PTH dependent causes including hyperplasia or adenoma. Thus, we evaluated the patient for the same.

There was no history of recent vitamin D use. Serum 25 hydroxy vitamin D levels were normal. Serum protein electrophoresis was found to be normal. Serum phosphate levels were sent, which were low at 2.3, while PTH levels sent were found to be very high at 2065 pg/dL against the normal range of 10–55 pg/dL! Thus, the hypercalcemia was likely due to a primary parathyroid disease.[3]

In order to evaluate the parathyroid disease, USG of the neck was done, which showed a well-defined heterogeneously hypoechoic nodular lesion with few cystic spaces within, showing rich vascularity on Doppler suggestive of left parathyroid adenoma.

However, this USG also incidentally revealed a well-defined hypoechoic taller than wider nodule in the right thyroid lobe measuring 1.6 × 1.3 cm, which was reported as thyroid imaging reporting and data system-4 (Fig. 1).[4]

A sestamibi parathyroid scan was then done, which confirmed increased uptake in

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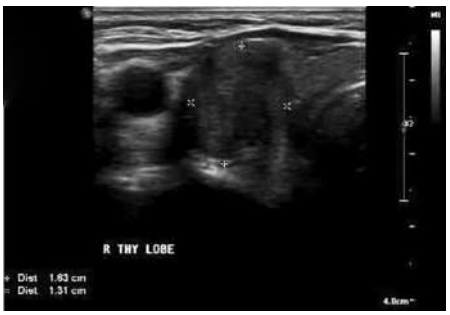

Fig. 1: Thyroid nodule on USG scan

the left lower parathyroid gland with delayed tracer retention (Fig. 2).

A two-dimensional echocardiography was done to evaluate the systolic murmur, which revealed severe concentric left ventricular hypertrophy asymmetric hypertrophic obstructive cardiomyopathy! The left ventricular outflow tract gradient was found to be 55 mm Hg. The left ventricle ejection fraction was 55%, and type I left ventricular diastolic dysfunction was noted.

Management

The patient was observed in the general wards for further management initially. Intravenous hydration was continued in view of hypercalcemia. She was also started on a furosemide infusion. However, on the 2nd day of admission, the patient developed drowsiness and was shifted to the intensive care unit (ICU). In the ICU, she was given an injection furosemide and intranasal calcitonin, after which she improved over time. She was also given a single dose of 5 mg of zoledronate.[3]

A referral was given to the oncosurgeon for surgical management.

Surgery was performed on 2nd of July 2022. Excision of the left inferior parathyroid adenoma was done, and the sample was sent for frozen section examination. On table, the right lobe of the thyroid was noted to have a 2 × 1.5 cm hard nodule adherent to the thyroid bed. Right hemithyroidectomy was done, and the sample was sent for the frozen section.

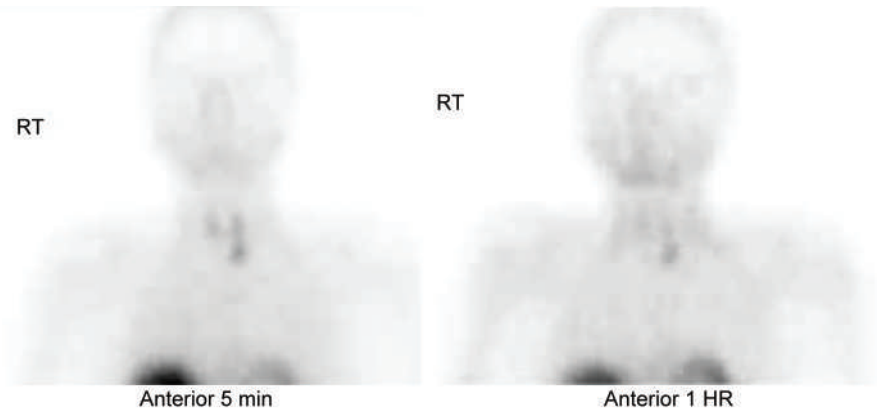

Fig. 2: Parathyroid scan

Frozen section samples revealed parathyroid adenoma and papillary carcinoma of the thyroid.[4]

Patient relatives were explained about the requirement for total thyroidectomy, and total thyroidectomy was done after getting their consent.

Intraoperative after the removal of the adenoma, it was found that the PTH levels had fallen to 112. The patient gradually improved and calcium levels started falling. By the next day of surgery, calcium levels had fallen to 8.6, and she was started on calcium and vitamin D supplements. She was discharged 2 days after surgery on subcutaneous insulin and advised to follow-up for further therapy of post thyroidectomy status and diabetes mellitus.[3]

A total of 10 days after surgery, the DJ stent was removed in the outside hospital. The patient followed up with our hospital for further management. Thyroglobulin levels were negative, and antithyroglobulin antibodies were negative. The patient was determined to have no residual thyroid tissue and was then started on thyroid replacement therapy.

Discussion and Comments

This patient came to the hospital with pain in the abdomen and was found to have diabetic ketoacidosis. It was found that diabetic ketoacidosis was caused by acute pancreatitis. Acute pancreatitis was caused by hypercalcemia. Hypercalcemia was caused by a parathyroid adenoma. The hypercalcemia also caused renal calculi, for which DJ stenting was required. She also had a papillary carcinoma of the thyroid detected incidentally on evaluation of the parathyroid gland, for which a total thyroidectomy was done. In addition to that, the patient was also incidentally found to have hypertrophic obstructive cardiomyopathy. When an abnormality is detected on investigation, it is important to search for the cause of this abnormality. Such an approach may reveal difficult to diagnose diseases and may help improve patient morbidity and mortality in the long term.

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