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Kunal Modi
Residents, Department of General
Surgery, CUSMCH, Gujarat, India

Digvijay Sindhav
Residents, Department of General
Surgery, CUSMCH, Gujarat, India

Abhi Dalsania
Assistant Professor, Department of
General Surgery, CUSMCH,
Gujarat, India

Primary hyperparathyroidism presenting as recurrent acute pancreatitis: A case report

Kunal Modi, Digvijay Sindhav and Abhi Dalsania

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Abstract

The association between pancreatitis and primary hyperparathyroidism (PHPT) is controversial. We report a 22-year-old female who presented with three episodes of acute pancreatitis. Primary hyperparathyroidism was diagnosed after the third episode of pancreatitis. He had no additional risk factors for pancreatitis. After successful parathyroid surgery, there has been no recurrence of abdominal pain and his serum calcium is within the normal range.

Keywords: Pancreatitis necrosis, hyperparathyroidism, necrosectomy, percutaneous drainage

Introduction

Primary hyperparathyroidism (PHPT) syndrome is an endocrine disorder, characterized by excessive secretion of parathyroid hormone from one or more parathyroid glands. The elevation of PTH usually leads to hypercalcemia and hypophosphatemia; patients may present with classic skeletal disease, recurrent nephrolithiasis, or be asymptomatic, detected on routine biochemical screening.

Hypercalcemia is considered to be a rare cause of pancreatitis but the true cause and effect relationship between PHPT and pancreatitis remains controversial. PHPT has been associated with different types of pancreatitis. Despite its rarity, a cause and effect relationship is still suggested by the fact that parathyroidectomy seems to prevent recurrence of pancreatitis.

Some patients suffer from 2 or more attacks of pancreatitis before a diagnosis of PHPT is made. We present a patient who had repeated admissions for acute pancreatitis, 3 episodes over a period of 6 months. PHPT was diagnosed after the third episode.

Case report

A 22-year-old female was admitted in our hospital with 3rd episodes of acute pancreatitis for last 6 months. She gave no history of indigestion, vomiting, change of bowel habit or change in color of his stool or urine. Her family history was normal. She remained completely symptom free in between the episodes. During the first 2 episodes of pancreatitis, the serum calcium was not estimated. We found elevated serum calcium level and parathyroid levels on 3rd episode. Blood reports of 3rd episodes were Haemoglobin 11.8 gm/dl, ESR 75 mm in 1st hour, Total count of WBC 16000/mm³, Platelet count 350000/mm³, HbA1C 8.1%, Creatinine 0.89 mg/dl, ALT (SGPT) 54 U/L, S. Lipase 2270 U/L, F. Triglycerides 125 mg/dl, S. Albumin 30 gm/L, S. Calcium 10.2, phosphate 2.0 mg/dL, mg/dl, S. Alkaline Phosphatase 179 U/L, S. Parathyroid Hormone (PTH) 233.4 pg/ml. USG of thyroid and 99 mTc-sestamibi scintigraphy of parathyroid glands revealed right sided parathyroid adenoma. Ct abdomen (figure: 1) shows p/o sequelae of necrotizing pancreatitis with partly walled off necrosis.

Fig 1: Ct abdomen of acute pancreatitis

Corresponding Author:
Abhi Dalsania
Assistant Professor, Department of
General Surgery, CUSMCH,
Gujarat, India

Discussion

The occurrence of pancreatitis secondary to PHPT is not so rare, with a prevalence of 3.6% (1.5 to 15.3%). Pancreatitis occurs at an advanced stage of parathyroid disease. The occurrence mechanism of pancreatitis during PHPT remains controversial but may be related to hypercalcemia, high calcium level in more than 1.3 times normal was associated with a risk of occurrence of an AP. Serum calcium is generally higher in patients with pancreatitis during PHPT than those who have only PHPT. Hypercalcemia would act by several mechanisms: increased level of calcium in pancreatic juice at the origin of activation of trypsinogen to trypsin; activation of pancreatic enzymes through the lysosomal system and hydrolases; calcium precipitation and formation of protein plugs responsible for upstream pancreatitis.

A genetic risk factor has also been found. Indeed, mutation of SPINK1 gene (Serine Protease Inhibitor Kazal type I) and CFTR gene (Cystic Fibrosis Transmembrane Conductance Regulator) was found more often in patients with PHPT who developed an AP. Thus, viewing these different mechanisms, the association of PHPT and pancreatitis can take many forms. The diagnosis of AP is classic in front of abdominal pain, with the dosage of pancreatic enzymes and imaging.

PHPT has been associated with different types of pancreatitis, such as acute, subacute or chronic calcifying pancreatitis. A study involving 83 cases of pancreatitis combined with PHPT found that about 70% of the patients suffered from acute relapsing or chronic pancreatitis. Some patients suffer from 2 or more attacks of pancreatitis before a diagnosis of PHPT is made. In our patient there was a delay of 06 months before the diagnosis of PHPT was established. Serum calcium estimation after the first episode of pancreatitis would have eliminated this delay. It is important to estimate serum calcium after an episode of unexplained pancreatitis. This will minimize the delay before the diagnosis of PHPT is made.

Conclusion

Acute pancreatitis is one of the symptoms of primary hyperparathyroidism caused by a parathyroid adenoma. Hypercalcemia and hyperparathyroidism should be a high index of suspicion in all fresh cases of acute pancreatitis even though primary hyperparathyroidism is a rare cause. Any oversight will result in diagnostic delays. Complementary explorations such as serum calcium and intact parathyroid hormone levels, and imaging techniques such as cervical ultrasounds, computed tomography and scintigraphy using 99 mTc-Sestamibi, should be ordered which will lead to confirm clinical suspicion.

Declarations

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Conflict of interest: None.

Ethical approval: Taken.

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