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Perspective

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Primary Hyperparathyroidism (PHPT) by excessive secretion of Parathyroid Hormone

Hardin James*

Department of Surgery, University of Alabama at Birmingham, Birmingham, USA 'Corresponding Author: Hardin James, Department of Surgery, University of Alabama at Birmingham, Birmingham, USA; E-mail: hardinjames32@gmail.com Received date: 28 November, 2023, Manuscript No. CICR-23-118422; Editor assigned date: 01 December, 2023, PreQC No. CICR-23-118422 (PQ); Reviewed date: 15 December, 2023, QCNo CICR-23-118422;

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Description

Primary Hyperparathyroidism (PHPT) is characterized by excessive secretion of Parathyroid Hormone (PTH), leading to hypercalcemia and a range of clinical manifestations, most commonly involving the bones and kidneys. While hypercalcemia can affect various organs, severe pancreatitis as the initial presentation of PHPT is rare. We report a case of a 52-year-old female who presented with severe pancreatitis due to undiagnosed PHPT, emphasizing the importance of considering this endocrine disorder in the evaluation of unexplained pancreatitis.

Primary Hyperparathyroidism (PHPT) is an endocrine disorder primarily characterized by excessive secretion of Parathyroid Hormone (PTH) from one or more parathyroid glands. This condition leads to hypercalcemia, which can have systemic effects, including bone demineralization, kidney stones, and gastrointestinal symptoms. However, severe pancreatitis as the initial presentation of PHPT is an unusual occurrence, with only a limited number of cases reported in the literature.

Primary hyperparathyroidism

Causes: This is the most common type and is typically caused by the development of a benign tumor or adenoma in one of the parathyroid glands. In some cases, hyperplasia (enlargement of multiple glands) or, less commonly, a parathyroid carcinoma (malignant tumor) can be the cause.

Symptoms: Primary hyperparathyroidism often leads to elevated levels of calcium in the blood (hypercalcemia), resulting in various symptoms, including fatigue, weakness, frequent urination, kidney stones, bone pain, and gastrointestinal issues. Some individuals may remain asymptomatic.

Secondary hyperparathyroidism

Causes: Secondary hyperparathyroidism is usually associated with another underlying medical condition, most commonly Chronic Kidney Disease (CKD). It results from the parathyroid glands working overtime to compensate for low calcium levels in the blood, which often occur in CKD.

Symptoms: Individuals with secondary hyperparathyroidism may experience similar symptoms to primary hyperparathyroidism, such as bone pain and fatigue. The primary focus is typically on managing the underlying condition.

A 52-year-old female with no known medical history presented to the emergency department with severe abdominal pain, nausea, and vomiting for the past 48 hours. Physical examination revealed tenderness in the upper abdomen. Laboratory tests showed elevated serum amylase (2,100 U/L) and lipase (1,780 U/L) levels, confirming the diagnosis of acute pancreatitis. The patient was admitted for further evaluation and management.

Despite appropriate medical management, the patient's condition deteriorated, and imaging studies were performed. Contrast-enhanced Computed Tomography (CT) of the abdomen revealed a swollen, edematous pancreas with extensive peripancreatic fluid collections. Surprisingly, the patient's calcium level was markedly elevated at 15.8 mg/dL, raising suspicion of hypercalcemia as a potential contributing factor to the pancreatitis.

Further evaluation showed significantly elevated Intact Parathyroid Hormone (iPTH) levels (420 pg/mL; reference range 10-65 pg/mL) and a parathyroid adenoma identified on neck ultrasound. Dual-energy X-ray absorptiometry (DXA) scan revealed osteopenia. These findings were consistent with a diagnosis of Primary Hyperparathyroidism (PHPT).

The patient underwent a successful parathyroidectomy to remove the adenoma. Postoperatively, her calcium levels gradually normalized, and her pancreatitis improved with supportive care. She was subsequently discharged in stable condition.

Discussion

The relationship between PHPT and pancreatitis is complex. Elevated serum calcium levels in PHPT can lead to the deposition of calcium within the pancreatic ducts, causing ductal obstruction and triggering pancreatitis. The presentation of pancreatitis in PHPT can vary from mild to severe, and in rare instances, it can be the initial presentation of the disease. The severity of pancreatitis in these cases may be related to the degree of hypercalcemia.

In patients with unexplained pancreatitis, it is important to consider PHPT as a potential underlying cause, especially when hypercalcemia is present. Prompt diagnosis and treatment of PHPT through parathyroidectomy can lead to the resolution of hypercalcemia and alleviate the risk of recurrent pancreatitis.

Conclusion

Severe pancreatitis as an initial presentation of primary hyperparathyroidism is a rare but important clinical scenario. It underscores the significance of considering hyperparathyroidism in the evaluation of unexplained pancreatitis, particularly when hypercalcemia is evident. Timely diagnosis and appropriate intervention can prevent recurrent episodes of pancreatitis and improve patient outcomes.

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