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CASE REPORT

Recurrent acute pancreatitis as an initial presentation of primary hyperparathyroidism: A case report

Masood M Karim, Hira Raza, Om Parkash

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Abstract

BACKGROUND

With 4.9 to 35 instances per 100000 cases, hyperparathyroidism is one of the rarest causes of acute pancreatitis. The major cause of primary hyperparathyroidism is a parathyroid adenoma, which can manifest clinically in various ways.

CASE SUMMARY

We discuss the unusual case of a 13-year-old boy with recurrent pancreatitis as the initial presentation of primary hyperparathyroidism. The cause of his recurrent pancreatitis remained unknown, and the patient had multiple admissions with acute pancreatitis over 3 years. His diagnosis was delayed due to the initial normal levels of parathyroid hormone, which were later reported elevated in a subsequent episode where ultrasound neck and thyroid scintigraphy revealed a parathyroid adenoma as the underlying cause. After the diagnosis was made, he underwent surgical resection of the adenoma.

CONCLUSION

This case study stresses the importance of considering uncommon causes for recurrent pancreatitis.

Key Words: Recurrent pancreatitis; Primary hyperparathyroidism; Normal serum parathyroid hormone; Parathyroid adenoma; Case report

Core Tip: Primary hyperparathyroidism (PHPT) should be considered as a potential cause when investigating cases of acute pancreatitis, especially in pediatric patients, even if blood parathyroid hormone levels initially appear normal. For an accurate diagnosis, PHPT may need a comprehensive investigation, including imaging techniques such as neck ultrasonography and scintigraphy. PHPT may sometimes only present as pancreatitis. To prevent recurrent episodes of pancreatitis and the difficulties that go along with it, early detection and treatment of PHPT are essential.

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INTRODUCTION

Acute pancreatitis is a condition characterized by inflammation of the pancreas. It is a widespread condition that accounts for a significant portion of admissions to the gastrointestinal department. The worldwide incidence of acute pancreatitis is 4.9 to 35 per 100000 in the United States, which is increasing worldwide[1]. Gallstones and alcohol are responsible for more than half of the cases of acute pancreatitis in adults[2]. This is not the same for children and adolescents. The main culprits for acute pancreatitis in the pediatric population include abdominal trauma accounting for 13%-33% of cases[3]. Other causes include hereditary and infectious etiology.

Rarely, acute pancreatitis can manifest as hypercalcemia caused by primary hyperparathyroidism (PHPT). In 80%-85% of cases, benign parathyroid adenoma is the most frequent cause of PHPT[4]. Clinical presentation varies between individuals. Children and adolescents almost always have an unusual presentation[5]. In 1.5%-13% of those with PHPT they will develop acute pancreatitis. PHPT is a rare condition in children, with a prevalence of 2-5 per 100000 and an estimated incidence of 1 in 200000-300000[6].

We present a young boy with PHPT due to parathyroid adenoma who had multiple episodes of acute pancreatitis. The cause remained a query until the diagnosis was made. After treatment for parathyroid adenoma, no episodes of pancreatitis occurred, and the patient's symptoms resolved.

This case study emphasizes the value of thorough follow-up to reveal the underlying issues that can be resolved.

CASE PRESENTATION

Chief complaints

Generalized abdominal pain and vomiting for 2 days.

History of present illness

According to the patient and his parents, he was in his usual state of health 2 days previously when he developed abdominal pain in the epigastric region, of severe intensity, sudden onset, radiating to the back, and multiple episodes of vomiting associated with decreased appetite and nausea. The patient denied any prior similar episodes.

History of past illness

His past medical history was unremarkable.

Personal and family history

Personal and family history was unremarkable, with no family history of hereditary diseases.

Physical examination

On arrival, the patient was stable. On examination, sluggish stomach sounds, slight right upper quadrant, and epigastric discomfort were documented. Other systemic examinations were unremarkable.

Laboratory examinations

Laboratory workup was conducted including liver function tests, complete blood count, serum electrolytes, serum amylase, serum lipase, and lactate dehydrogenase (LDH). Serum amylase and lipase levels were found to be elevated at 361 IU/L and 447 U/L, respectively. Among others, serum calcium (12.5 mg/dL) and serum LDH (265 IU/L) were significant.

Imaging examinations

Ultrasound (US) of the abdomen was performed which showed the head of the pancreas with the suggestion of a streak of peripancreatic fluid, mild to moderate ascites, and mild left-sided pleural effusion. US findings were consistent with

acute pancreatitis (Figure 1). In 2022, the patient was admitted again with similar complaints, a CT scan showed acute pancreatitis with early chronic changes (Figure 2).

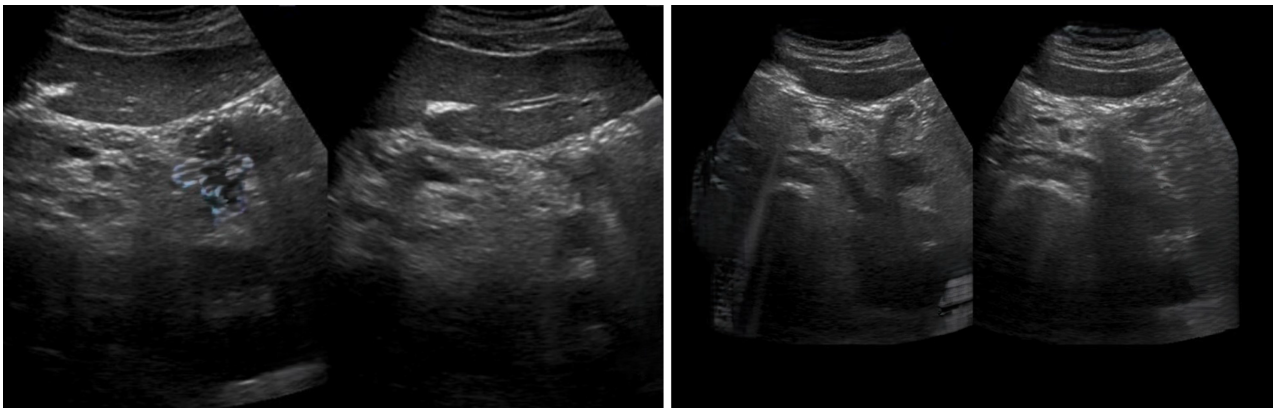

Figure 1 Abdominal ultrasound demonstrating swollen head of the pancreas with the suggestion of peripancreatic fluid.

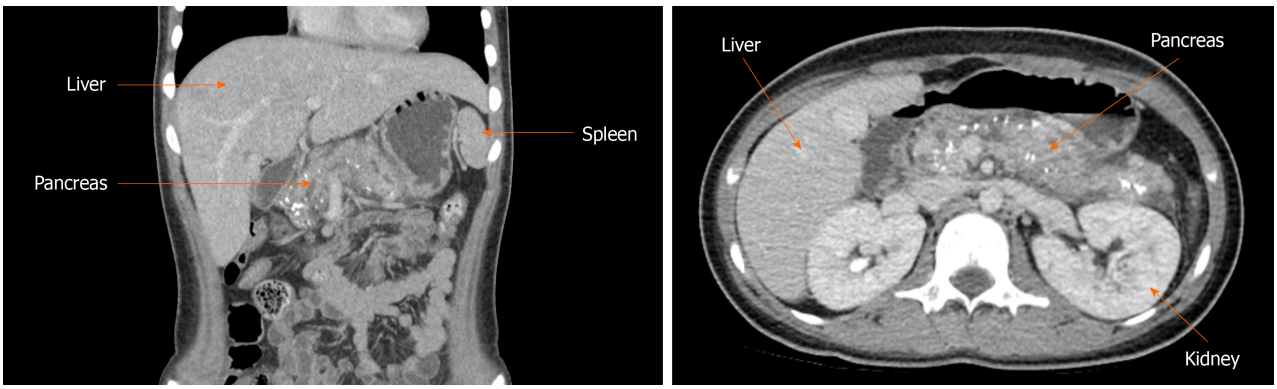

Figure 2 Abdominal computed tomography: Pancreas is bulky showing inhomogeneous enhancement, with patchy non-enhancing areas.
Internal calcifications are seen. Minimal surrounding inflammatory changes are also noted. The pancreatic duct is dilated.

The pediatric endocrinology team advised a neck US (Figure 3), and a scintigraphy scan (Figure 4). The US of the neck showed a parathyroid adenoma at the lower pole of the right thyroid lobe.

FINAL DIAGNOSIS

Parathyroid adenoma.

TREATMENT

Surgical removal of the parathyroid adenoma.

OUTCOME AND FOLLOW-UP

Following resection of the adenoma, serum calcium levels normalized, and the patient's symptoms resolved.

DISCUSSION

Acute pancreatitis as a complication of hypercalcemia due to PHPT has been defined in the literature. The mechanism is still unclear, but the theory is that raised serum calcium levels activate trypsinogen to trypsin, causing pancreatic damage

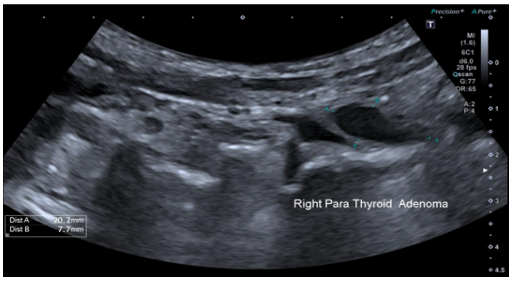

Figure 3 Ultrasound of the neck showed a large parathyroid adenoma at the inferior lobe pole of the right lobe of the thyroid medially, measuring 207 mm.

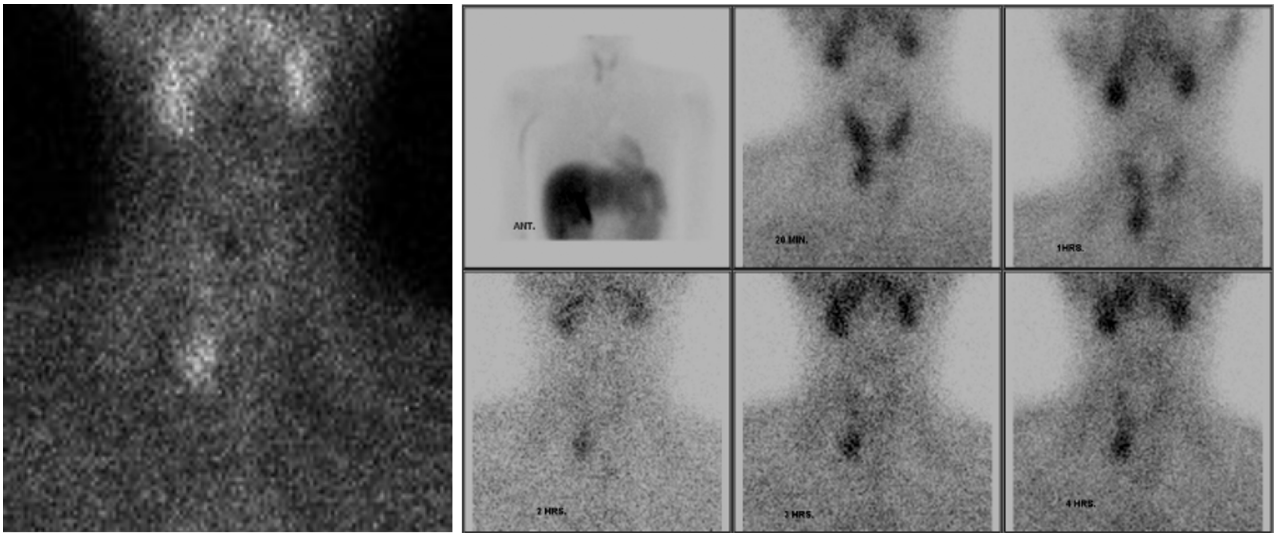

Figure 4 Parathyroid scintigraphy with sestaMIBI. Radionuclide parathyroid scintigraphy was carried out following the administration of 550 MBq of Tc-99m sestaMIBI by intravenous injection. Multiple static images were taken starting from the time of injection and continuing up to 4 hours later. An area of abnormal tracer deposition is observed in the lower pole of the right thyroid lobe.

and calcium deposits in the pancreatic duct, resulting in obstruction[7].

Lanitis *et al*[8] reported that the relationship between pancreatitis and PHPT is not incidental but a consequence of hypercalcemia. As early as the late 1950s, Cope *et al*[9] suggested acute pancreatitis as a sole presentation of PHPT and a clue to the diagnosis of PHPT. A review study in Vellore reported a causal relationship between acute pancreatitis and PHPT. This study also reported that patients with PHPT have a 28-fold higher risk of developing pancreatitis than patients with no evidence of PHPT[10]. A similar survey by Ramachandran *et al*[11] reported a linear relationship between acute pancreatitis and hypercalcemia. Pancreatitis is now an established complication of PHPT[12].

Diagnosing PHPT in children is difficult as it is uncommon in children, resulting in misdiagnosis or a delayed diagnosis. The diagnostic criteria for PHPT include hypercalcemia, hypophosphatemia, and elevated levels of serum parathyroid hormone. The delay in the diagnosis in our case can be attributed to the fact that the initial work-up showed normal levels of parathyroid hormone, lowering the suspicion of hyperparathyroidism as the cause of hypercalcemia. The literature has documented similar cases in which diagnosing the condition was challenging due to parathyroid hormone levels falling within the normal range[13]. A study conducted by Mischis-Troussard *et al*[14] evaluated serum PTH levels in patients with surgically proven cases of PHPT and reported that 7.4% of their patients had normal serum PTH levels pre-operatively. Another retrospective case-control study by Amin *et al*[15] reported that 7% of their patients had normal PTH levels.

Underdiagnosis and delayed diagnosis of PHTP primarily due to the normal levels of PTH result in an increased risk of developing complications.

After reviewing our experience, engaging in a comprehensive assessment is advisable to determine the underlying cause of undetermined pancreatitis. When encountering hypercalcemia accompanied by serum PTH within normal ranges, it is essential to include PHPT in the list of potential differential diagnoses.

CONCLUSION

Due to the rarity of PHPT, it poses a diagnostic challenge for physicians. PHPT should be included in the differential

diagnoses when evaluating the cause of undefined pancreatitis even with normal PTH levels. Pancreatitis can be a solo presentation, as seen in our case. High clinical suspicion can help diagnose and manage PHPT early to avoid its complications.

FOOTNOTES

Author contributions: Karim MM and Raza H contributed to manuscript writing and editing; Parkash O contributed to conceptualization and supervision; All authors have read and approved the manuscript.

Informed consent statement: Informed written consent was obtained from the patient for publication of this report and accompanying images.

Conflict-of-interest statement: We ensure that there are no conflicts of interest that would compromise the impartiality or authenticity of this case report per the finest ethical standards. We hereby declare that any financial ties, personal ties, or other circumstances have not influenced the material, analysis, or conclusions offered herein. To improve patient care and the medical profession as a whole, we are exclusively committed to the quest for accurate and relevant medical knowledge.

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