

Acute pancreatitis and hyperparathyroidism: A case series

Sudipta Dhar Chowdhury · Reuben Thomas Kurien · Sandip Pal · Veena Jeyaraj ·
Anjilivelil Joseph Joseph · Amit Kumar Dutta · Anuradha Chandramohan ·
Deepak Abraham · Joby Augustine · Julie Hephzibah · Ebby George Simon

Received: 26 May 2013 / Accepted: 15 October 2013 / Published online: 14 January 2014
© Indian Society of Gastroenterology 2014

Abstract Primary hyperparathyroidism is a rare cause of acute pancreatitis. Five consecutive patients with acute or recurrent acute pancreatitis and primary hyperparathyroidism were included. All patients had elevated serum calcium on admission and high levels of circulating parathyroid hormone. Both ultrasonography and Sestamibi scan was used to localize parathyroid adenoma. Except for one, all patients underwent parathyroidectomy and postoperative histology was consistent with parathyroid adenoma. One patient died while on treatment. Metabolic causes of acute pancreatitis, though uncommon, are important as early recognition helps management and prevents recurrence.

Keywords Hypercalcemia · Hyperparathyroidism · Pancreatitis

S. D. Chowdhury (✉) · R. T. Kurien · S. Pal · A. J. Joseph ·
A. K. Dutta · J. Augustine · E. G. Simon
Department of Gastrointestinal Sciences, Christian Medical College,
Vellore 632 004, India
e-mail: sudipto.d.c@gmail.com

V. Jeyaraj
Department of Pathology, Christian Medical College,
Vellore 632 004, India

A. Chandramohan
Department of Radiology, Christian Medical College,
Vellore 632 004, India

D. Abraham
Department of Endocrine Surgery, Christian Medical College,
Vellore 632 004, India

J. Hephzibah
Department of Nuclear Medicine, Christian Medical College,
Vellore 632 004, India

Background

The association of primary hyperparathyroidism (PHPT) and acute pancreatitis is controversial [1]. While hospital-based cohort studies have reported a high incidence of acute pancreatitis in patients with PHPT, community studies of patients with PHPT found that the estimated rate of development of acute pancreatitis is similar in patients with PHPT and age and gender matched controls [2–4].

Cases

In this series, we present five cases of acute pancreatitis (Table 1). Of five patients, three had prior history of acute pancreatitis. None of them consumed alcohol or had gallstone disease. Evaluation for a metabolic cause revealed normal triglycerides but high serum calcium levels in all patients. Further evaluation revealed elevated intact parathyroid hormone in all. One patient died after she developed multiorgan failure. Ultrasound neck (Fig. 1) and parathyroid scintigraphy (Fig. 2) was done in four patients and all had a lesion in thyroid gland which was suspicious of a parathyroid adenoma. The patients subsequently underwent successful removal of the lesions. Histological examination confirmed parathyroid adenoma in all four patients (Fig. 3). The patients have since been on follow up for 1 year and are doing well.

Discussion

The mechanism by which PHPT leads to acute pancreatitis is unclear. Carnaille et al. suggested that elevated serum calcium level is responsible for the development of pancreatitis in patients with PHPT [5]. Elevated serum calcium levels lead

Table 1 Cases of primary hyperparathyroidism with acute pancreatitis

	Age (years)/sex	Pain duration (days)	Past history of pancreatitis	Alcohol	Amylase (U/L)	Lipase (U/L)	S. calcium (mg/dL)	S. PTH (pg/mL)	Ultrasound neck	Nuclear scan	Surgical histology	Follow up
Case 1	41/female	20	Absent	No	1,216	1,616	18	1,166	NA	NA	NA	Expired
Case 2	20/male	7	Present	No	660	960	13.8	186.1	Hypoechoic lesion posterior-inferior to left lower lobe of thyroid	Tracer accumulation in left lower pole of thyroid manage selected	Adenoma	Doing well
Case 3	28/male	4	Absent	No	353	282	11.5	218.9	Hypoechoic lesion posterior to left lobe of thyroid	Tracer accumulation in left lower pole of thyroid	Adenoma	Doing well
Case 4	40/female	3	Present	No	2,709	1,871	11.6	386.3	Hypoechoic lesion posterior to right lobe of thyroid	Tracer accumulation inferior to lower pole of right lobe of thyroid	Adenoma	Doing well
Case 5	17/male	1	Present	No	280	254	11.3	210	Well-defined hypoechoic nodule inferior to the left thyroid lobe	Normal	Adenoma	Doing well

PTH parathyroid hormone, *NA* not available

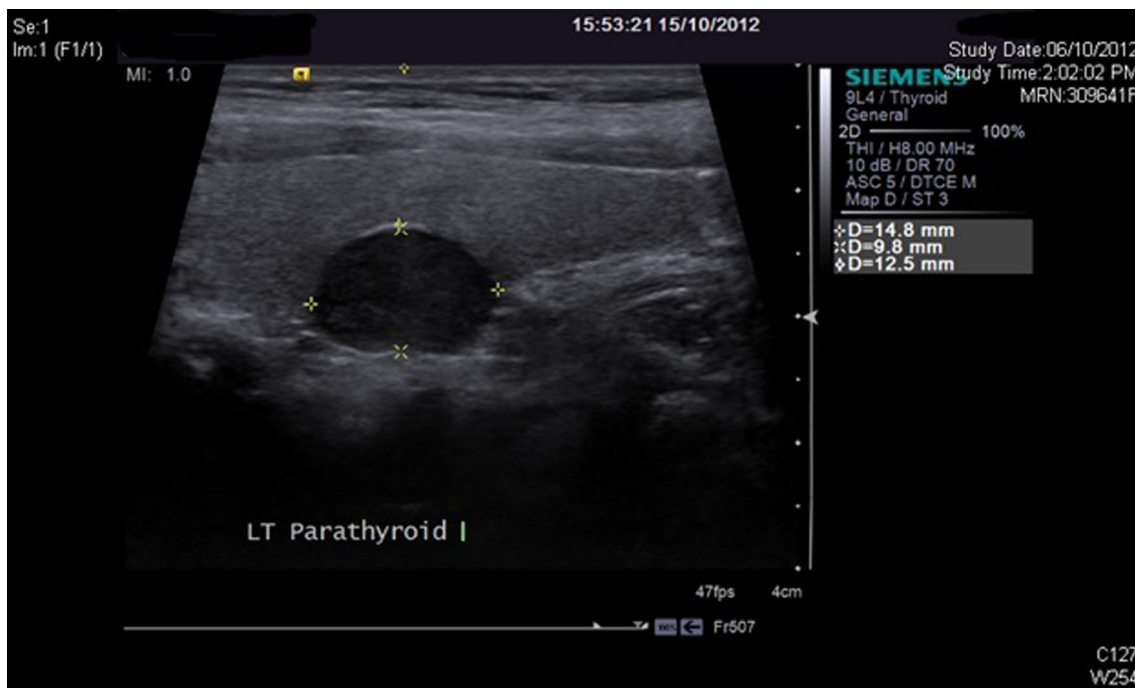


Fig. 1 Ultrasonography of neck showed a 14.8 mm × 12.5 mm × 9.8 mm hypoechoic solid lesion posterior to left lobe of thyroid

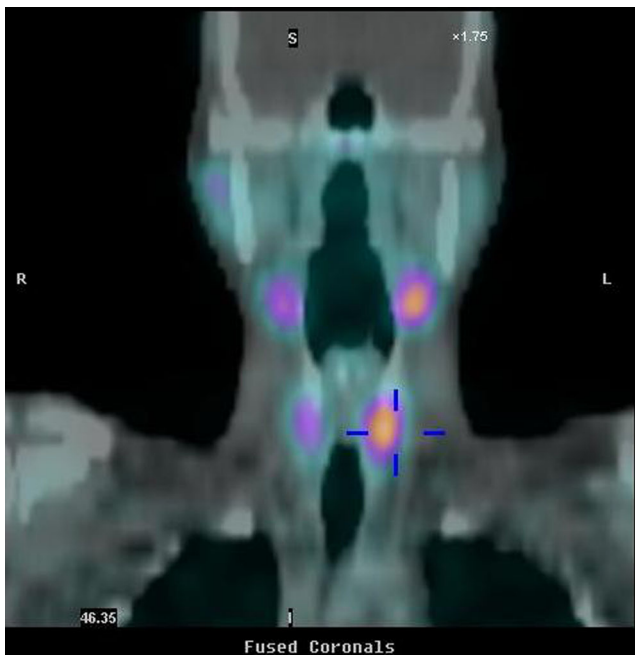


Fig. 2 Sestamibi scan: persistent tracer accumulation is seen in the left lower pole of thyroid in the delayed image at 90 min after complete tracer washout from thyroid gland

to premature activation of pancreatic protease leading to acute pancreatitis [1].

Estimation of serum calcium holds the key to early diagnosis and treatment. Identifying hypercalcemia during an episode of acute pancreatitis may pose a problem as calcium levels may be low during the attack [6]. Therefore, it is important to recheck calcium levels after resolution of acute pancreatitis especially in patients with idiopathic recurrent acute pancreatitis. PHPT and malignancy account for approximately 90 % of cases of hypercalcemia [7]. Elevated intact parathyroid hormone helps distinguish the two. Radionuclide scintigraphy and ultrasonography are the most common preoperative tests used for localization of parathyroid glands. In our series, parathyroid scintigraphy failed to detect parathyroid adenoma in a patient which was subsequently detected by ultrasonography; (Table 1). Haber et al. suggested a strategy in which either one of these methods is used first; the alternate method is used if the first evaluation is negative [8]. The treatment for hypercalcemia includes hydration with saline, forced diuresis, bisphosphonates, calcitonin, oral phosphates, glucocorticoids, and dialysis. The underlying primary hyperparathyroidism can be cured by surgery [9]. In patients with acute pancreatitis, surgery should be performed early so as to prevent recurrent episodes of acute pancreatitis.

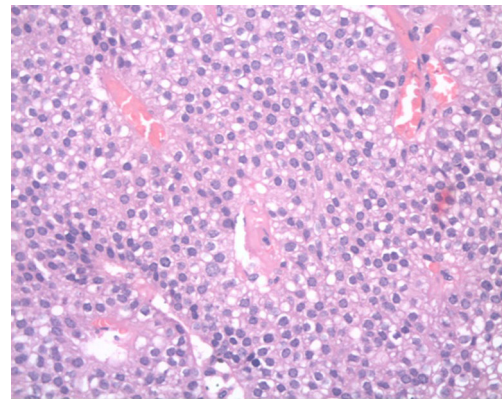


Fig. 3 Parathyroid adenoma—histology: ($\times 40$) nests and sheets of polygonal cells with clear to eosinophilic cytoplasm, minimal nuclear pleomorphism, and dispersed chromatin with interspersed delicate vascular channels

While alcohol and gallstones are the more common causes of acute pancreatitis. Metabolic causes like hypercalcemia and hypertriglyceridemia do account for a few. Early recognition and treatment probably hold key to successful management, as associated pancreatitis if severe can have fatal consequences.

Funding Nil.

References

1. Bai HX, Giefer M, Patel M, Orabi AI, Husain SZ. The association of primary hyperparathyroidism with pancreatitis. *J Clin Gastroenterol*. 2012;46:656–61.
2. Shepherd JJ. Hyperparathyroidism presenting as pancreatitis or complicated by postoperative pancreatitis. *Aust N Z J Surg*. 1996;66:85–7.
3. Jacob JJ, John M, Thomas N, et al. Does hyperparathyroidism cause pancreatitis? A South Indian experience and a review of published work. *ANZ J Surg*. 2006;76:740–4.
4. Khoo TK, Vege SS, Abu-Lebdeh HS, Ryu E, Nadeem S, Wermers RA. Acute pancreatitis in primary hyperparathyroidism: a population-based study. *J Clin Endocrinol Metab*. 2009;94:2115–8.
5. Carnaille B, Oudar C, Pattou F, Combemale F, Rocha J, Proye C. Pancreatitis and primary hyperparathyroidism: forty cases. *Aust N Z J Surg*. 1998;68:117–9.
6. Ammori BJ, Barclay GR, Larvin M, McMahon MJ. Hypocalcemia in patients with acute pancreatitis: a putative role for systemic endotoxin exposure. *Pancreas*. 2003;26:213–7.
7. Lafferty FW. Differential diagnosis of hypercalcemia. *J Bone Miner Res*. 1991;6 Suppl 2:S51–59. discussion S61.
8. Haber RS, Kim CK, Inabnet WB. Ultrasonography for preoperative localization of enlarged parathyroid glands in primary hyperparathyroidism: comparison with (99 m)Technetium sestamibi scintigraphy. *Clin Endocrinol (Oxf)*. 2002;57:241–9.
9. Suliburk JW, Perrier ND. Primary hyperparathyroidism. *Oncologist*. 2007;12:644–53.