of recurrence are 25% in subsequent offerings.

MITUL B KALATHIA[1]
YAGNESH POPAT[2]
JAGDISH PRASAD GOYAL[1]

Correspondence: Dr Jagdish Goyal
Department of Pediatrics[1],
All India Institute of Medical Sciences,
Rishikesh.
PDU Govt. Medical College, Rajkot
Om Baby care Hospital[2],
Email: jpgoyal@rediffmail.com

References

1. Johanson A, Blizzard R. A syndrome of congenital aplasia of the alae nasi, deafness, hypothyroidism, dwarfism, absent permanent teeth, and malabsorption. *J Pediatr*. 1971;**79**:982–7.
2. Almashraki N, Abdulnabee MZ, Sukalo M, Alrajoudi A, Sharafadeen I, Zenker M. Johanson-Blizzard syndrome. *World J Gastroenterol*. 2011;**17**:4247–50.
3. Ramos S, Ramos HF, Ramos RF, Peixoto CA, Ramos BF. Johanson-Blizzard syndrome. *Braz J Otorhinolaryngol*. 2010;**76**:794.
4. Kulkarni ML, Shetty SK, Kallambella KS, Kulkarni PM. Johanson—blizzard syndrome. *Indian J Pediatr*. 2004;**71**:1127–9.
5. Sudarshan R, Annigeri RG, Shettar SS. Johanson –Blizzard Syndrome: A rare case report. *Journal of Indian Academy of Oral Medicine and Radiology*. 2010;**22**:225–8.
6. Hoffman WH, Lee JR, Kovacs K, Chen H, Yaghmai F. Johanson-Blizzard syndrome:autopsy findings with special emphasis on hypopituitarism and review of the literature. *Pediatr Dev Pathol*. 2007;**10**:55–60.
7. Vanlieferinghen P, Gallot D, Francannet Ch, Meyer F, Dechelotte P. Prenatal ultrasonographic diagnosis of a recurrent case of Johanson-Blizzard syndrome. *Genet Couns*. 2003;**14**:105–7.

Endoscopic ultrasound detection of a parathyroid adenoma in a patient with chronic pancreatitis due to hyperparathyroidism

Introduction

A proportion of patients will have no identifiable cause of their chronic pancreatitis and are diagnosed with idiopathic chronic pancreatitis. In this report, we discuss a case of primary hyperparathyroidism presenting as chronic pancreatitis to the gastroenterologist.

Case report

A 27-year-old male was referred to a tertiary gastrointestinal hospital for the complaint of recurrent epigastric pain of 4 months duration. He had been evaluated at another hospital and was diagnosed with chronic pancreatitis based on a CT scan. He had elevated serum amylase and lipase at the time of initial presentation. The cause of his chronic pancreatitis was presumed idiopathic, as he was a teetotaller and did not have gallstones or hypertriglyceridemia.

We evaluated the pancreas and biliary tree with endoscopic ultrasound. A radial echoendoscope (GF-UE160, Olympus Optical Co, Ltd, Tokyo, Japan) was inserted with the patient under moderate sedation. The pancreas showed diffuse hyperechoic strands, echogenic foci, honeycombing and lobularity (**Figure 1**). The pancreatic duct was irregular, but not dilated. There were no pancreatic ductal stones or pseudocysts. The stack sign was observed from the duodenal bulb, suggesting that pancreas divisum was unlikely. Examination of the biliary tree and gallbladder did not reveal any microlithiasis. These features were consistent with a diagnosis of chronic pancreatitis.

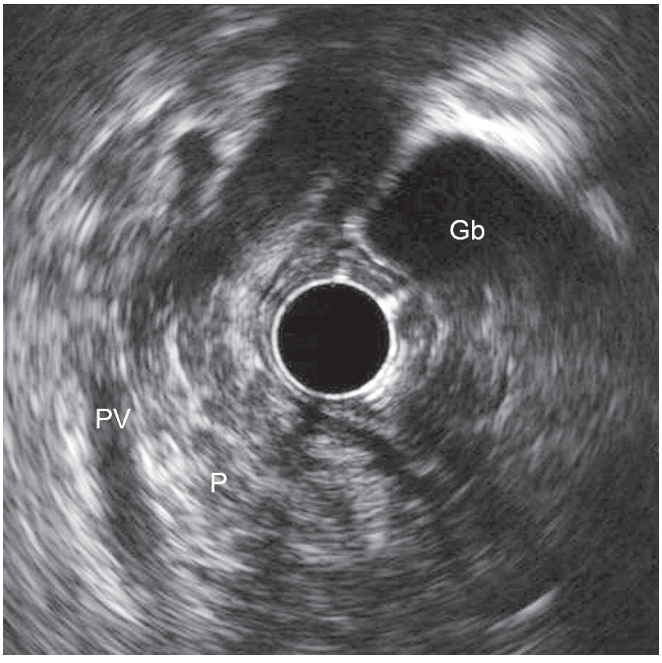

Figure 1: Radial EUS image from the duodenal bulb. The pancreatic head (P) shows lobularity and echogenic stranding suggestive of chronic pancreatitis. The portal vein (PV) and gallbladder (GB) are normal.

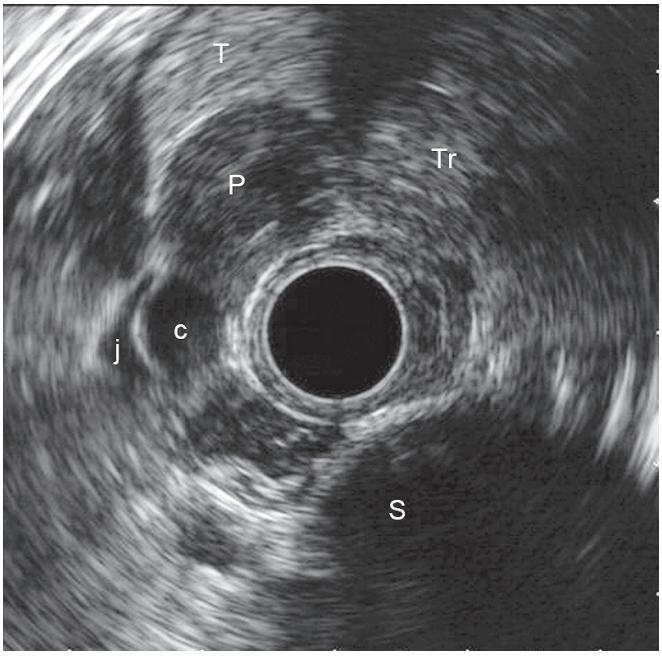

Figure 2: Radial EUS image from the upper esophagus. The parathyroid adenoma (P) is seen as a well-demarcated hypo-echoic lesion located within the right thyroid lobe (T). There is no invasion into the jugular vein (j) or internal carotid artery (c). Ultrasound views beyond the spine (S) and the trachea (Tr) are obscured due to calcification and air respectively.

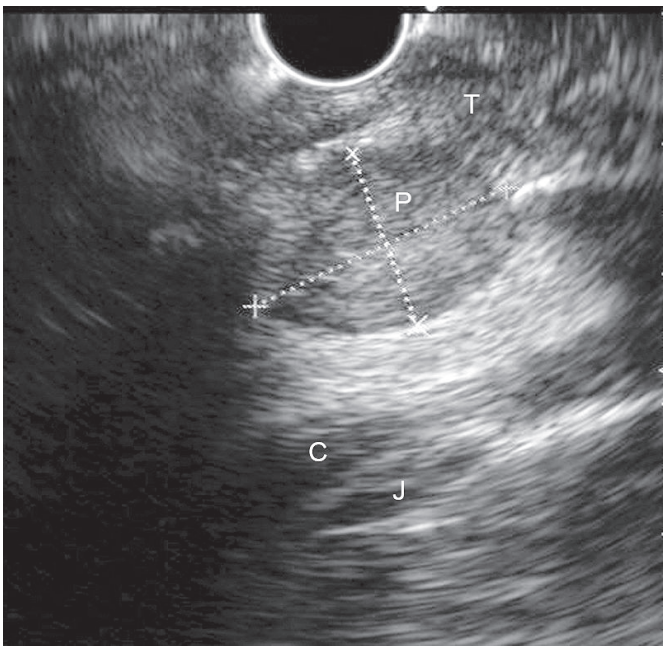

Figure 3: Linear EUS image from the upper esophagus. The parathyroid adenoma (P) is seen in sagittal view. The thyroid (T) is located to the right. Absence of vascular invasion into the carotid artery (c) and jugular vein (j) can be easily confirmed.

Upon withdrawal of the echoendoscope, a well-circumscribed, homogeneous, hypo-echoic mass with distinct margins was visualised from the proximal oesophagus. The lesion was identified as arising from within the right lobe of the thyroid gland. It measured 21 mm by 14 mm in diameter (**Figure 2**). This lesion was further investigated with a linear echoendoscope (GF-UCT180, Olympus Optical Co, Ltd, Tokyo, Japan). There were no features of vascular invasion or mediastinal lymphadenopathy (**Figure 3**).

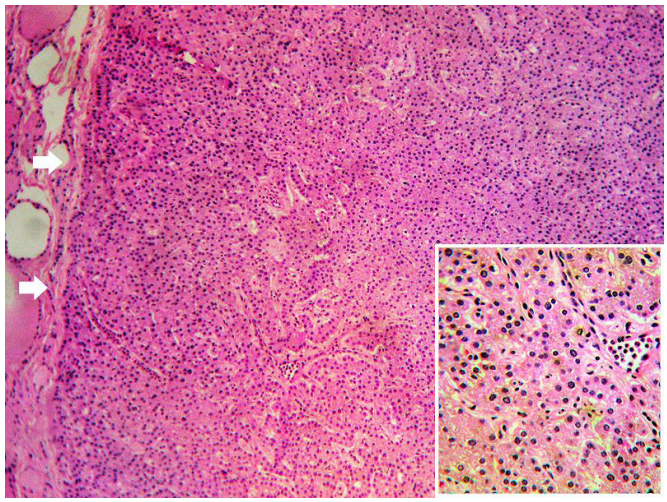

Figure 4: Photomicrography of the resected parathyroid adenoma (Hematoxylin and eosin, x100): the parathyroid adenoma is seen in the center surrounded by a fibrous capsule (arrows). Insert: (x400) epithelial cells are arranged in cords, in a micro-glandular pattern. No mitosis or nuclear atypia is present.

The lesion was suspected to be either a parathyroid or thyroid tumour. His serum calcium levels were marginally elevated at 12.8 mg/dL (normal 8-10 mg/L). In addition, his parathyroid hormone levels were elevated at 207.5 pg/mL (normal 10-55 pg/ml). A technetium-99m sestamibi scintigraphy scan confirmed the EUS diagnosis of a parathyroid tumour.

The patient underwent surgical excision of the lesion. The specimen was confirmed on histology to be a parathyroid adenoma (**Figure 4**). Post-resection, his calcium levels normalised. Further evaluation for tumours associated with the multiple endocrine neoplasia (MEN) syndrome was negative. The etiology of his chronic pancreatitis was due to hypercalcaemia from a parathyroid adenoma.

Discussion

Primary hyperparathyroidism and hypercalcaemia may present with gastrointestinal problems such as constipation, anorexia, nausea and abdominal pain. Hyperparathyroidism has also been associated with acute and chronic pancreatitis.

Despite numerous reports of patients with both pancreatitis and primary hyperparathyroidism, the association between

these 2 diseases still remains somewhat controversial. This is due to the fact that not all patients with primary hyperparathyroidism will develop pancreatitis. The incidence of acute pancreatitis in patients with primary hyperparathyroidism has been reported to be between 1.5% and 12% only.[1-3] One large retrospective analysis revealed that primary hyperparathyroidism was associated with a 28-fold increased risk of developing pancreatitis as compared to patients without hyperparathyroidism.[4] Examination of the parathyroid and thyroid glands are not part of the routine EUS evaluation. However, a parathyroid tumour may be detected at EUS and this has been described in a case series by Catargi B et al.[5] Typical imaging characteristics of a parathyroid adenoma on ultrasound are a hypo-echoic structure, with regular borders and homogeneous echotexture. Features suggestive of a carcinoma based transcutaneous ultrasound images are an enlarged lesion with irregular borders, and a heterogeneous echotexture. In normal patients, the parathyroids are usually not visualised.

The sensitivity of EUS as compared to transcutaneous ultrasound and scintigraphy scan is inadequately studied. Neither has the learning curve to achieve competency and the inter-operator variability been evaluated. Therefore, EUS cannot substitute existing imaging modalities as a screening tool if a patient is suspected to have a parathyroid adenoma.

ERIC WEE[1],
RAJESH GUPTA[2],
SEKARAN ANURADHA[2],
DUVURU NAGESHWAR REDDY[2],
SUNDEEP LAKHTAKIA[2].

Correspondence: Dr. Eric Wee
Khoo Teck Puat Hospital[1], Singapore.
Asian Institute of Gastroenterology[2], Hyderabad, India.
Email: wee1976@gmail.com

References

1. Cope O, Culver PJ, Mixter CG Jr, Nardi GL. Pancreatitis, a diagnostic clue to hyperparathyroidism. *Ann Surg*. 1957;**145**:857–63.
2. Bess MA, Edis AJ, van Heerden JA. Hyperparathyroidism and pancreatitis. Chance or a causal association? *JAMA*. 1980;**243**:246–7.
3. Jacob JJ, John M, Thomas N, Chacko A, Cherian R, Selvan B, et al. Does hyperparathyroidism cause pancreatitis? A South Indian experience and a review of published work. *ANZ J Surg*. 2006;**76**:740–4.
4. Ward JB, Petersen OH, Jenkins SA, Sutton R. Is an elevated concentration of acinar cytosolic free ionised calcium the trigger for acute pancreatitis? *Lancet*. 1995;**346**:1016–9.
5. Catargi B, Raymond JM, Lafarge-Gense V, Leccia F, Roger P, Tabarin A. Localization of parathyroid tumors using endoscopic ultrasonography in primary hyperparathyroidism. *J Endocrinol Invest*. 1999;**22**:688–92.

Xanthelasma of the stomach- A rare pseudotumor

Introduction

Xanthelasmas (xanthomas) are non-neoplastic lesions composed of fat-laden histiocytes commonly seen in the dermis or subcutis. Their occurrence in the stomach is uncommon and can rarely present as gastric polyps mimicking other benign and malignant pathologies(1).Herein, we report an unusual case of xanthelasma of stomach presenting as gastric polyp and illustrate the clinical, endoscopic and histopathological findings. Moreover, we review the literature in order to clarify the clinical and pathological features of this rare pseudoneoplasm.

Case report

A 57-year-old female presented in the gastroenterology outdoor clinic with chief complaints of abdominal pain, dyspepsia and occasional episodes of nausea and vomiting of six months duration. There was no past history of weight loss, diabetes mellitus or any previous surgery. Her general physical examination was unremarkable. No xanthelasma nodules on skin, eyelids or tendons were seen. Routine hematological investigations and biochemical findings including fasting blood glucose, lipid profile and thyroid function tests were within normal limits. Upper gastrointestinal endoscopy done revealed a small 10 mm x 10 mm polypoidal lesion in the stomach cardia which was removed endoscopically using biopsy forceps. (**Figure 1**).The rest of the stomach, esophagus and duodenum were unremarkable.

Results

Histopathological examination of the lesion revealed dense aggregates of foamy histiocytes widening the lamina propria