# Lost in Passaro's triangle: the value of DOTATATE PET-CT

Authors:
Dr Zhiyuan Lin (Radiology Registrar)[1]
Dr Carla De Sa Goncalves (Consultant Radiologist in GI/HPB)[1]
Dr Cherry Sit (Consultant Radiologist in GI/HPB and Nuclear Medicine)[1]

1. Guy's and St Thomas' NHS Foundation Trust

# Initial presentation

A 55 yo female presented to ED over 20 times in 2 years with the same recurrent symptoms of intractable non-bloody/bilious vomiting, non-bloody diarrhoea and generalised abdominal pain

**PMHx/PSHx:**
- Uterine fibroids
- Diverticular disease
- Glaucoma
- Cholecystectomy 30 years ago

**DHx:**
- Lansoprazole
- Timolol/Travoprost eyedrops

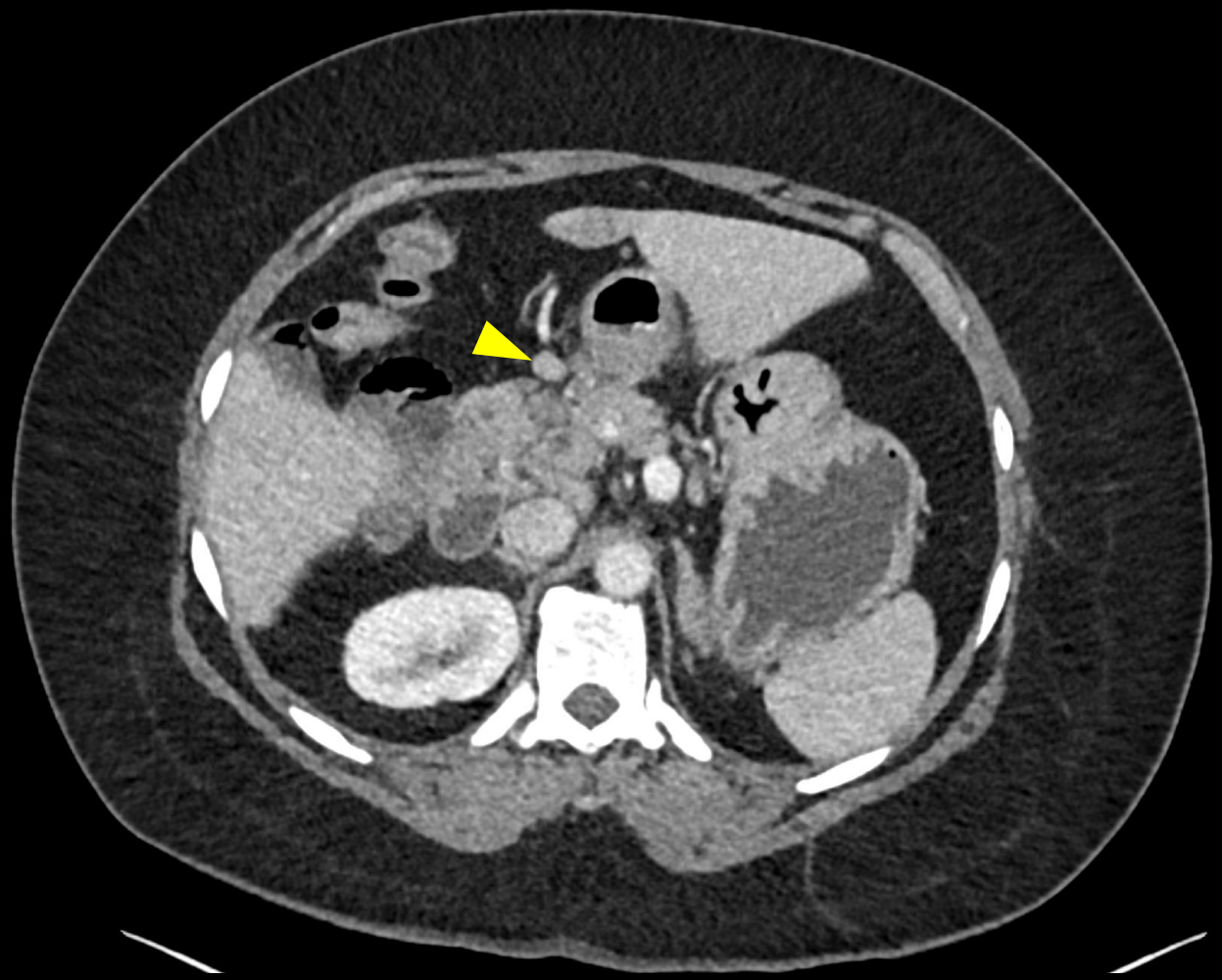
**FHx:**
- No known family history of cancer

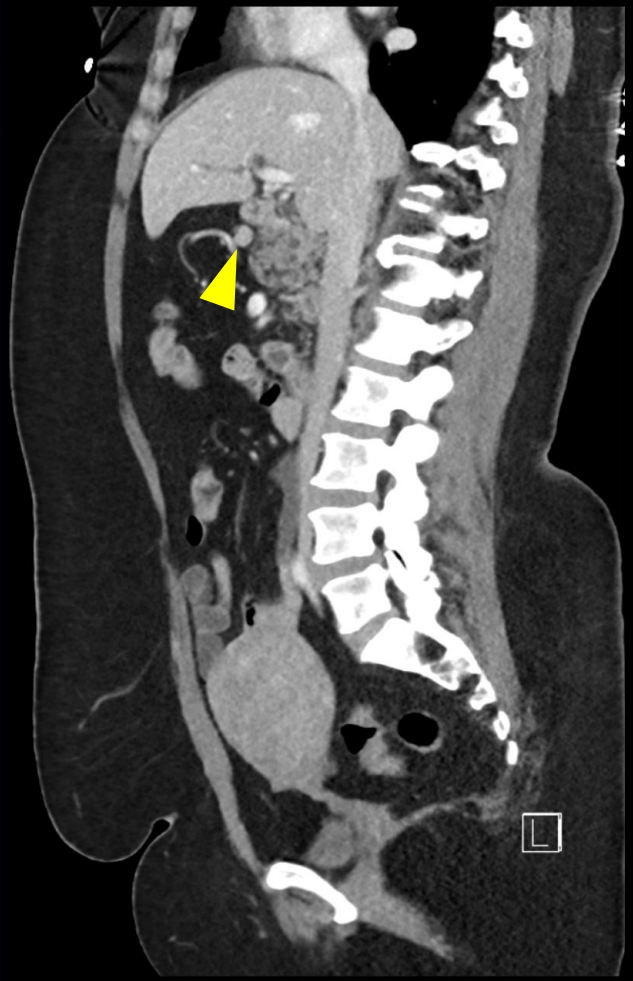
- **Physical examination** was unremarkable
- **Routine biochemical tests** were unremarkable
- **H. pylori testing** repeatedly came back negative
- **Immunology:** Coeliac antibodies and anti-gastric parietal cell antibodies were negative
- **Stool tests:** Faecal calprotectin, faecal elastase and stool cultures were all normal
- **Gut hormone profile:** a borderline elevated gastrin level (220 pmol/L) was dismissed due to concurrent PPI use. Other gut hormone levels and Chromogranin A levels were within normal range
- **Urinary 5-HIAA testing** was negative for carcinoid syndrome

# More negative tests

- **Abdominal ultrasound** only revealed mild hepatic steatosis
- **2x OGD** demonstrated erosive antral gastritis and duodenitis. Multiple biopsies were taken but histology remained negative for dysplasia or neuroendocrine cell proliferation
- **Colonoscopy** was normal
- **Video capsule endoscopy** was normal
- **MRCP** found no choledocholithiasis
- **SeHCAT studies** found no bile salt malabsorption
- **Nuclear medicine gastric emptying tests** excluded gastroparesis

# 1 CT-AP (portovenous phase)

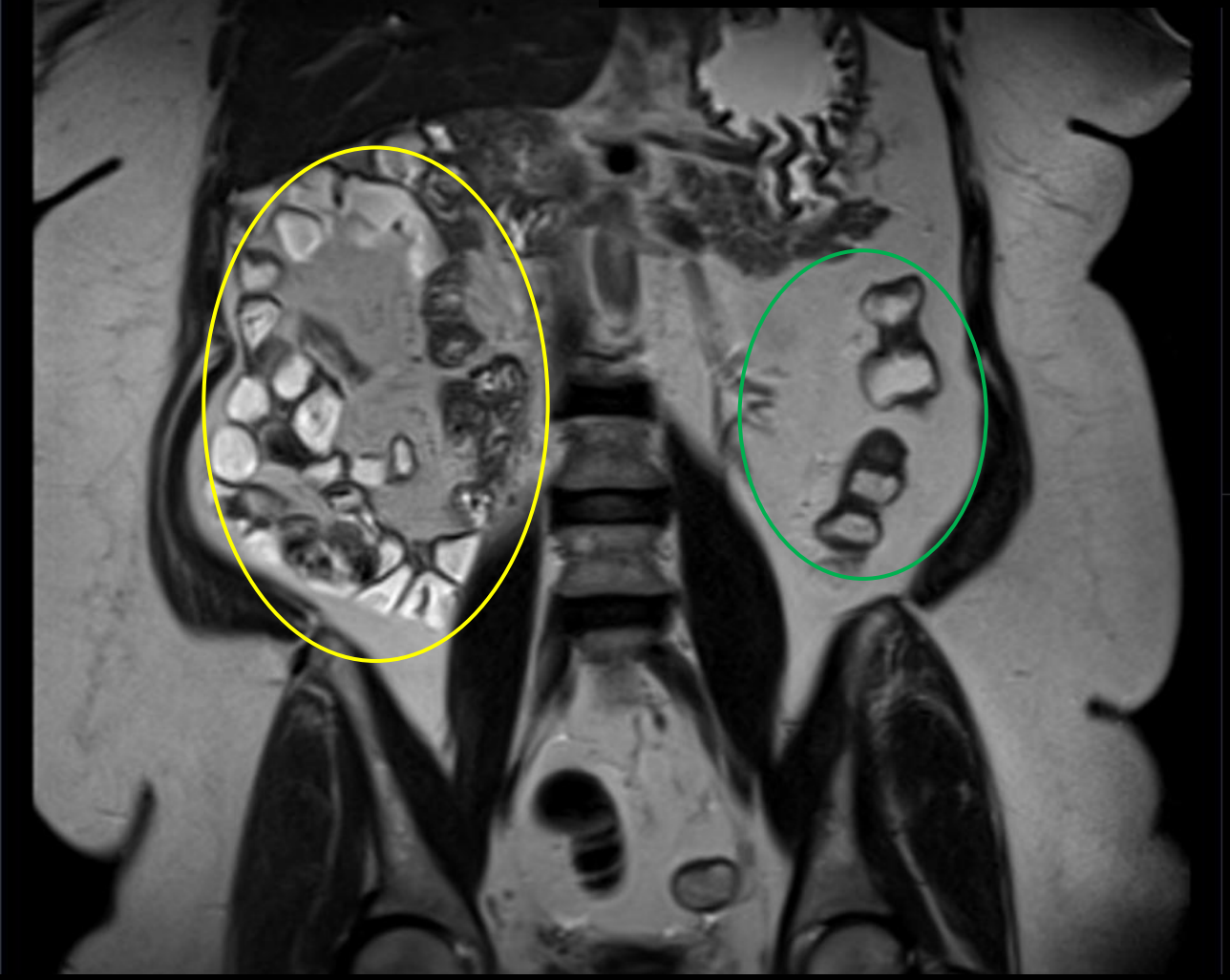


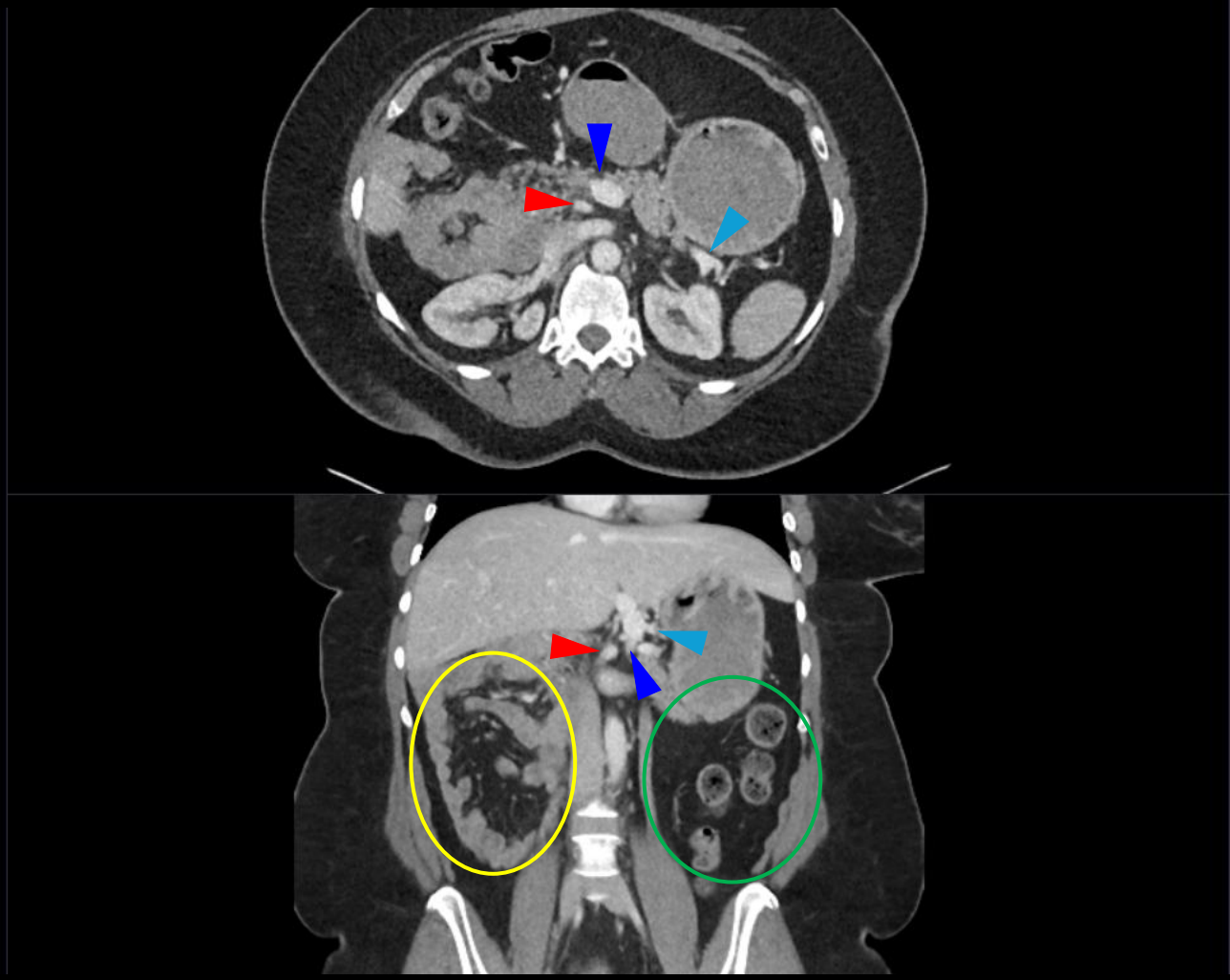




The first CT-AP demonstrated no acute abnormalities. In retrospect one of the peripancreatic nodes in Passaro's triangle (yellow arrowhead), which was not size significant, correlated to a DOTATATE avid node on subsequent PET-CT.

## 2 MRI small bowel

Further CTs also demonstrated intestinal non-rotation which confounded the diagnosis as it potentially explained the intermittent abdominal pain. MRI small bowel confirmed intestinal non-rotation but demonstrated no active inflammation.

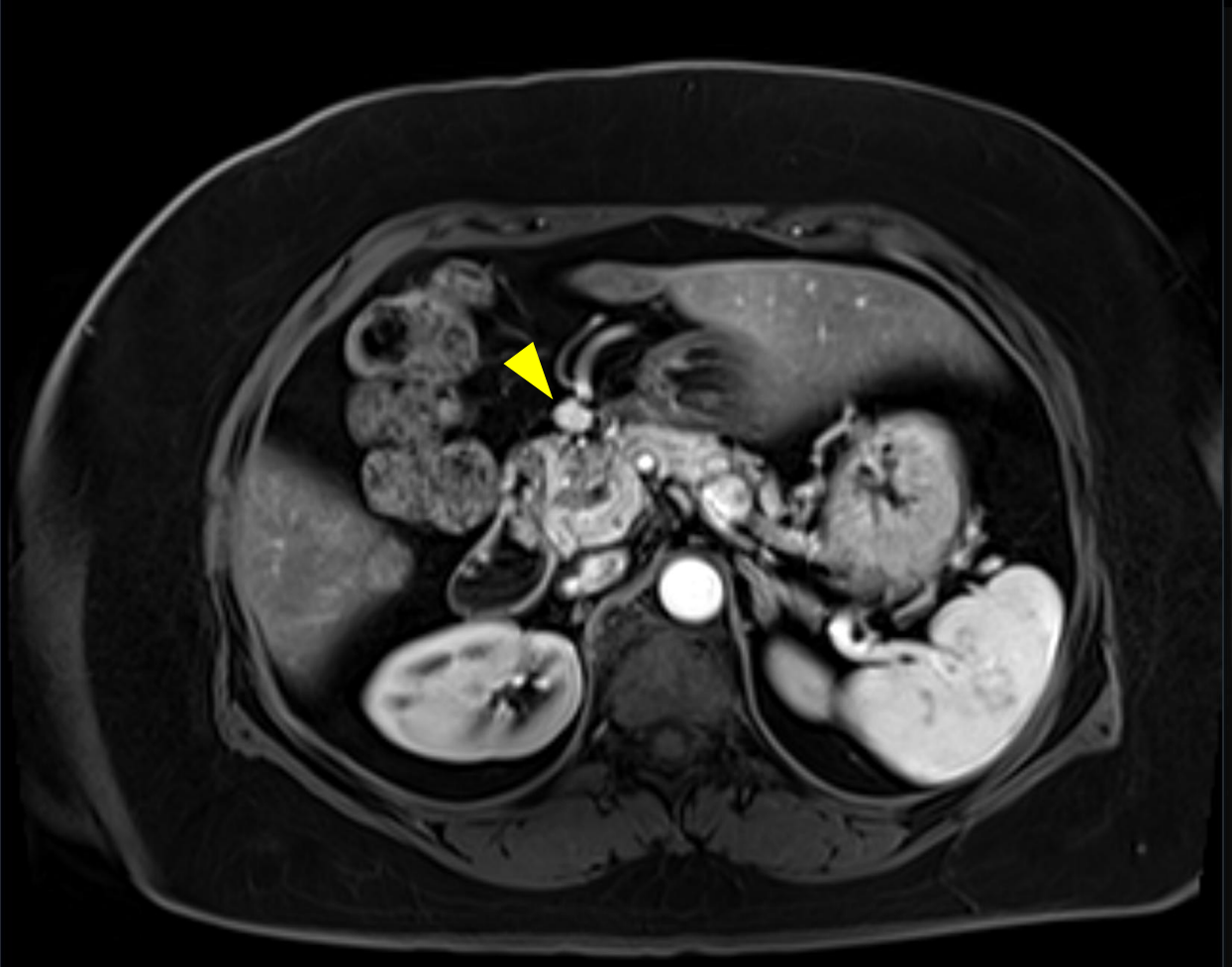




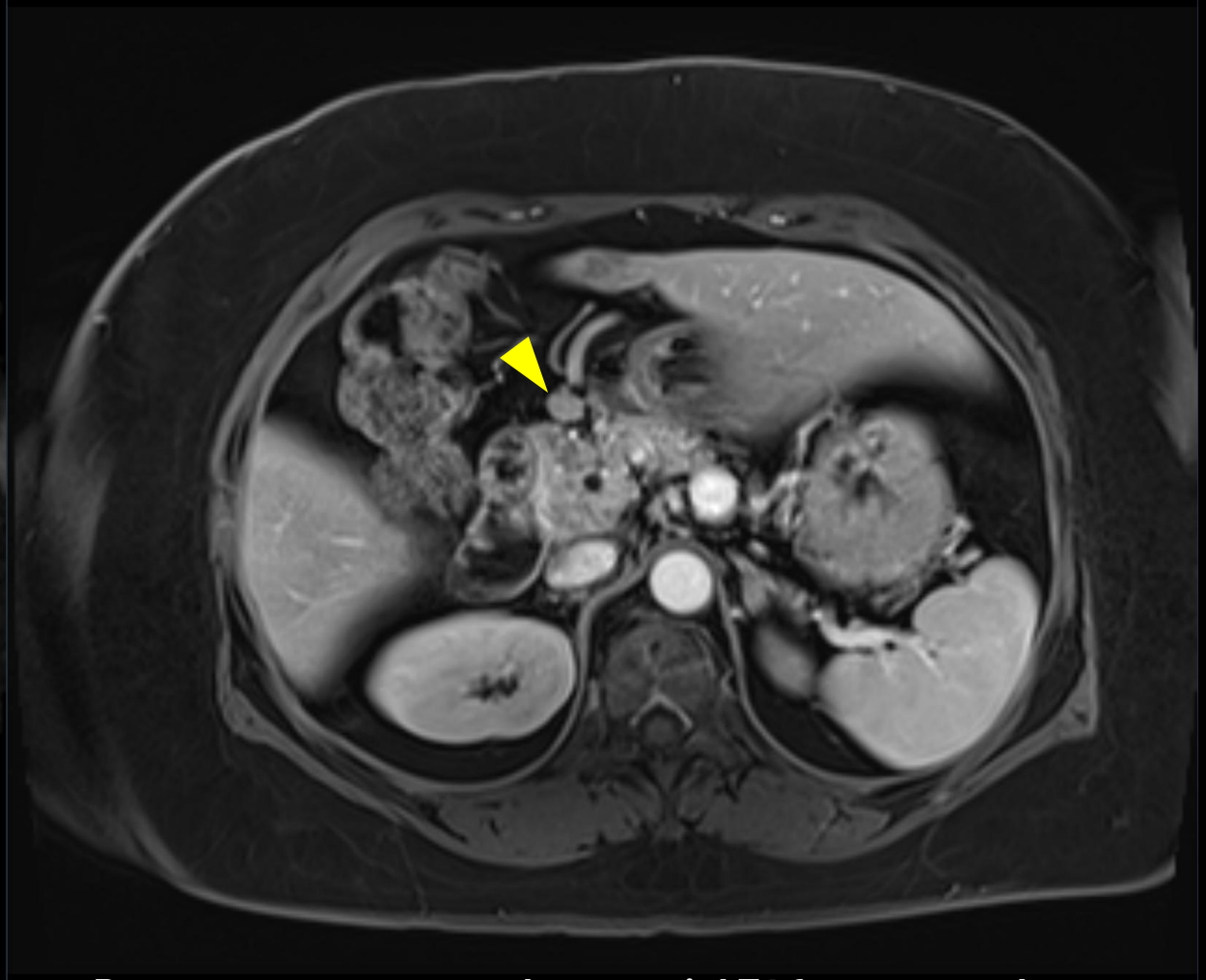
MRI and CT showing predominantly right-sided small bowel loops (yellow circle) and left sided large bowel loops (green circle). The relationship of the SMA (red arrowhead) and SMV (dark blue arrowhead) is inverted, with the SMA found to the right of the SMV. The splenic vein is also marked for reference (light blue arrowhead).

## 3 MRI pancreas with dynamic contrast

No focal gastric, pancreatic or duodenal lesions were identified on MRI pancreas with dynamic contrast.



**Post-contrast arterial phase, axial T1 fat saturated**
Peripancreatic lymph node which demonstrates arterial enhancement (yellow arrowhead)



**Post-contrast venous phase,  axial T1 fat saturated**
Peripancreatic lymph node washes out on the venous phase (yellow arrowhead)

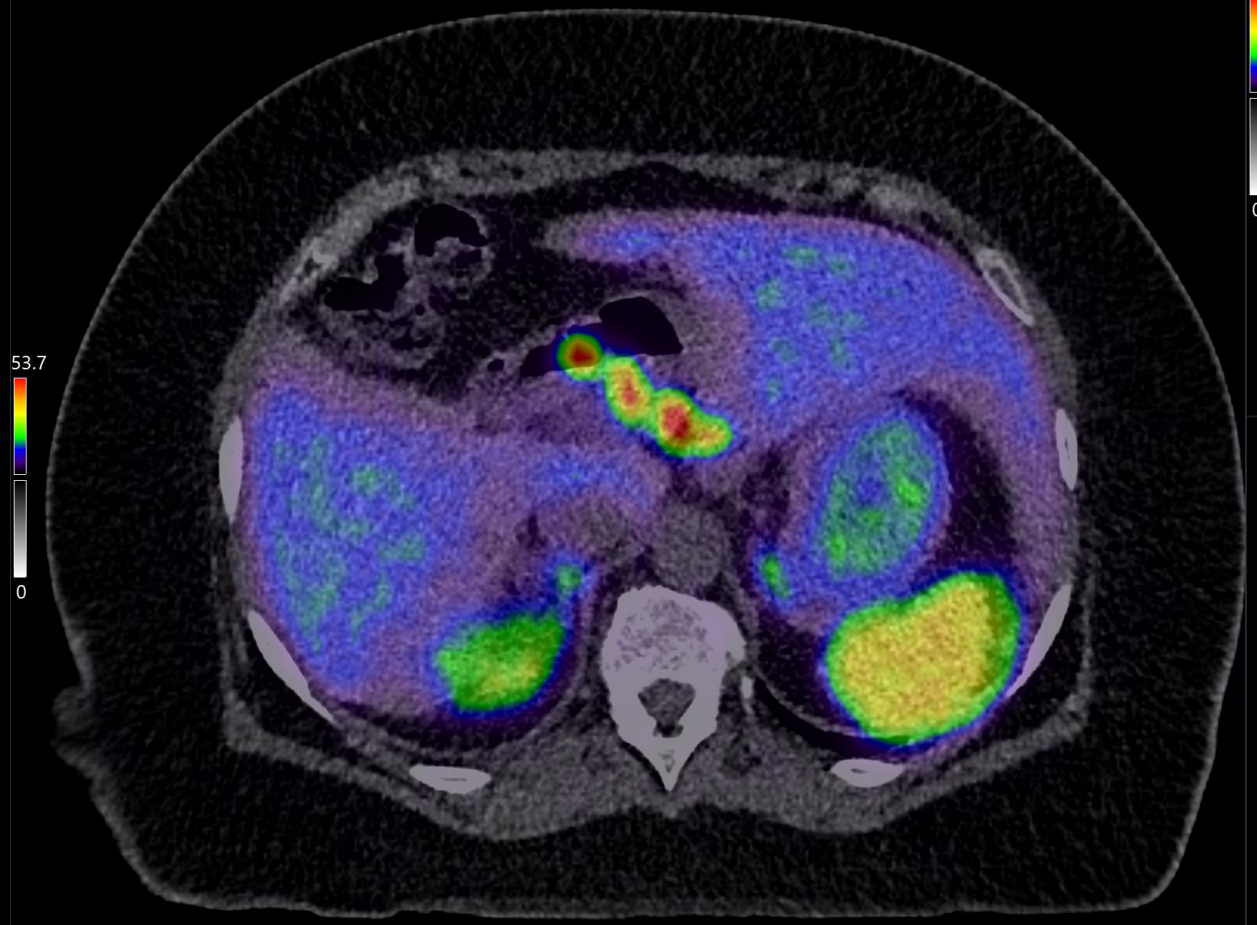
# DOTATATE PET-CT lights the way

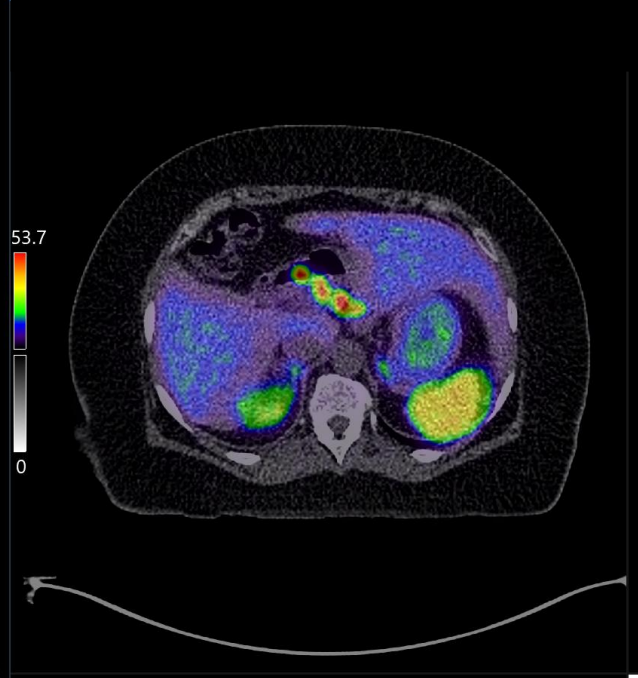
| **Gut hormone profile** | Dec 2024 (pmol/L) | July 2025 (pmol/L) | Normal range (pmol/L) |
|---|---|---|---|
| Glucagon | 39 | 29 | <50 |
| Gastrin | **220** | **1501** | <40 |
| Somatostatin | 23 | 14 | <150 |
| VIP | 12 | 11 | <30 |
| Pancreatic polypeptide | 30 | 155 | <300 |
| Chromogranin A | 55 | **64** | <60 |
| Chromogranin B | 98 | **233** | <150 |

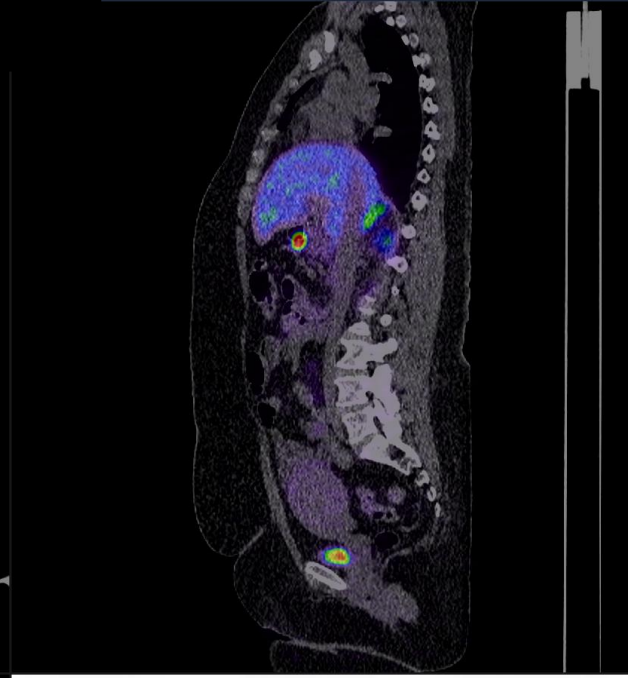
| **Bone profile** | July 2025 | Normal reference |
|---|---|---|
| Adjusted calcium | **2.79** | 2.20-2.60 mmol/L |
| Phosphate | 0.95 | 0.80-1.50 mmol/L |
| ALP | 114 | 30-130 U/L |
| Albumin | 41 | 35-50 g/L |
| PTH | **241** | 15 – 68 ng/L |
| Vitamin D | **44** | >50 nmol/L |

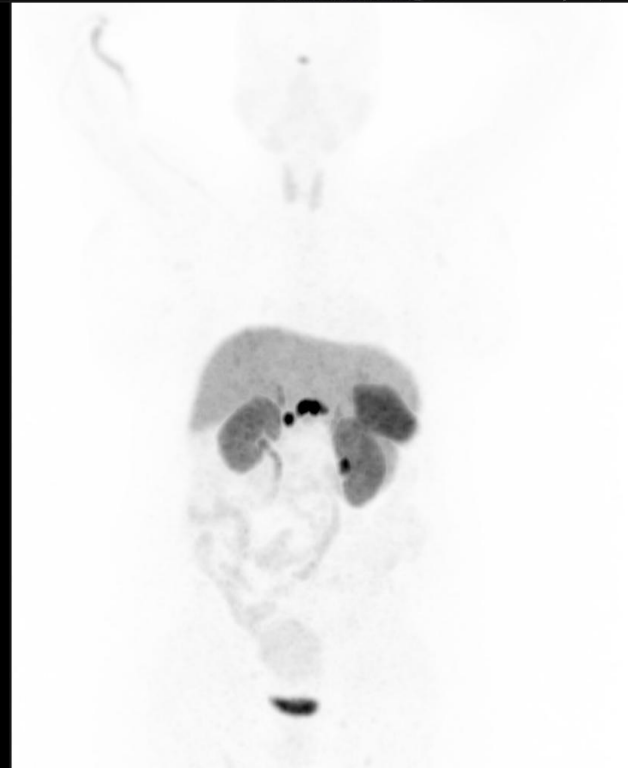
Repeat blood tests revealed a hypercalcaemia secondary to primary hyperparathyroidism and an US thyroid confirmed a left parathyroid mass. A repeat gut hormone profile was ordered and found significantly raised gastrin (1501 pmol/L) and chromogranin levels. This re-raised the suspicion for an occult gastrinoma and MEN-1. Following this [Ga-68]-DOTATATE PET-CT was requested by the MDT to hunt for an occult tumour.

# 4 [Ga-68]-DOTATATE PET-CT



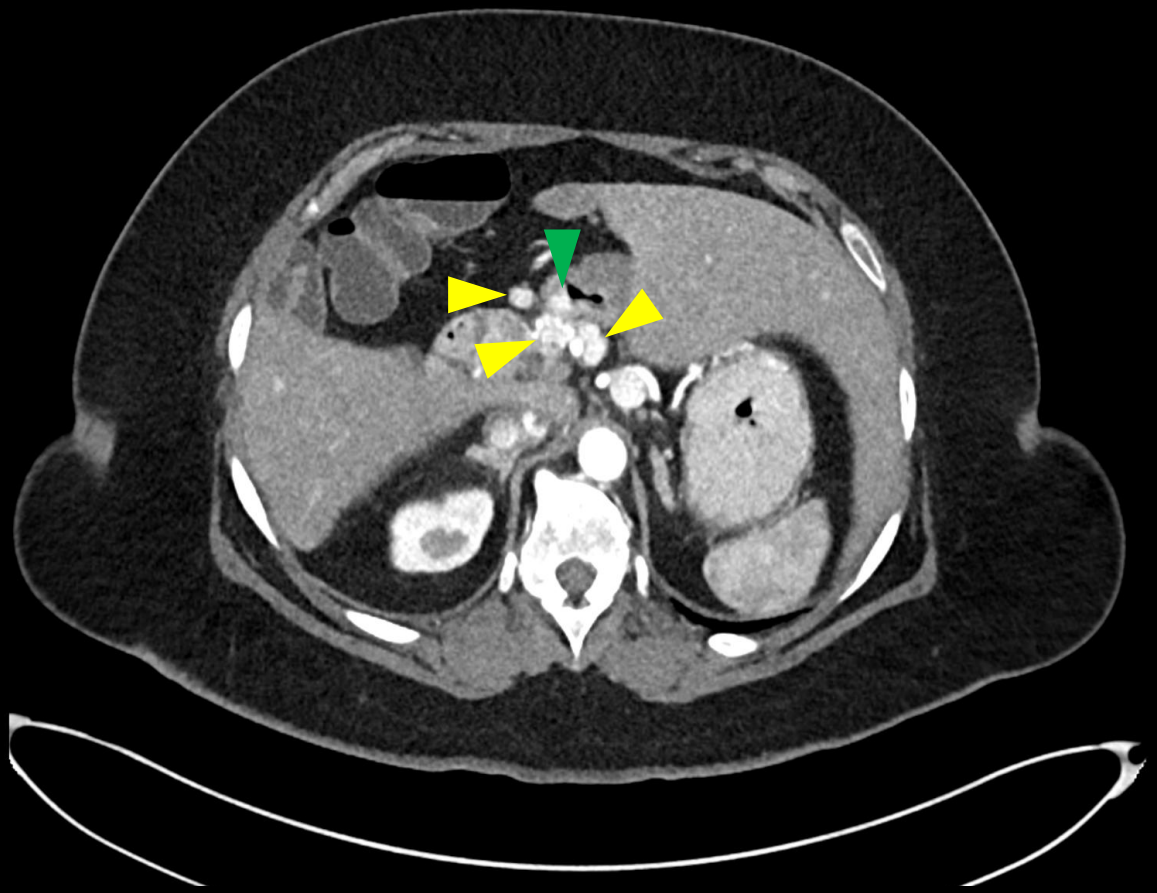





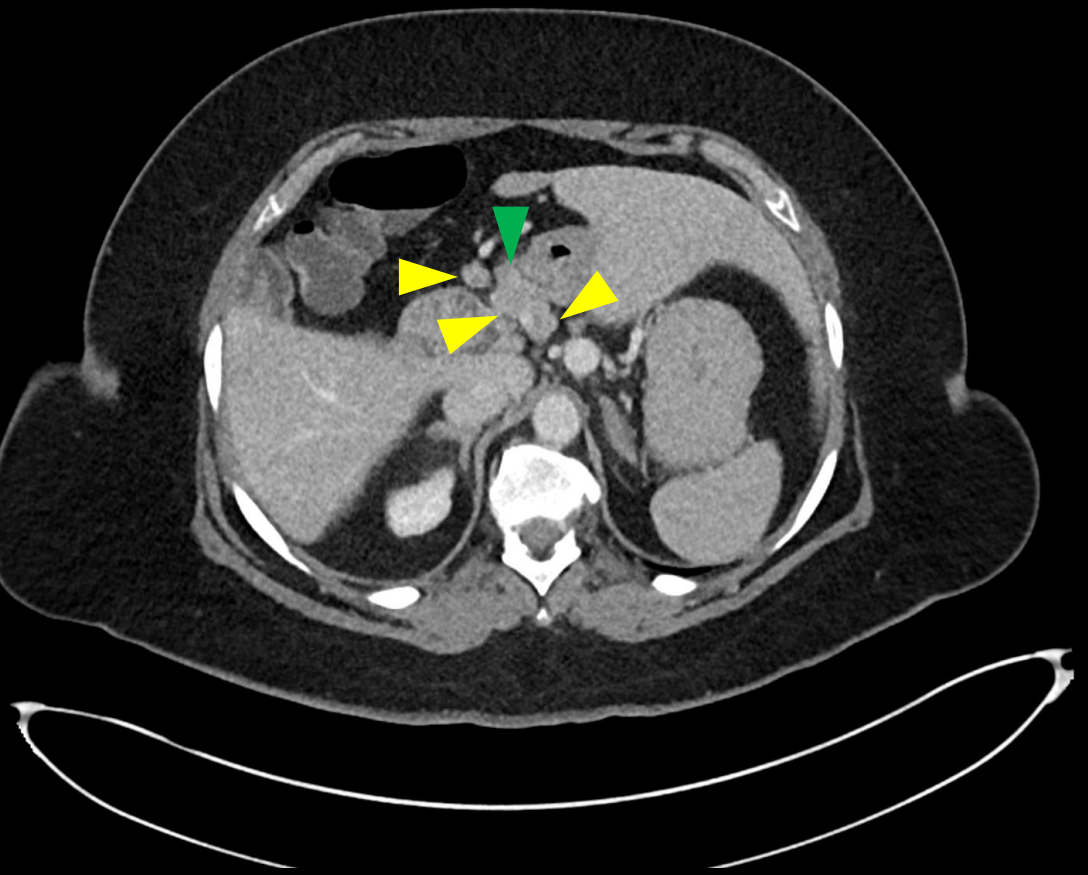
4 highly avid peripancreatic lymph nodes were seen on PET-CT. A gastric antral mucosal lesion was not DOTATATE avid and no avid primary pancreatic/duodenal lesion was identified.

# 5 Follow-up CT-AP (arterial & portovenous phases)

## Arterial phase



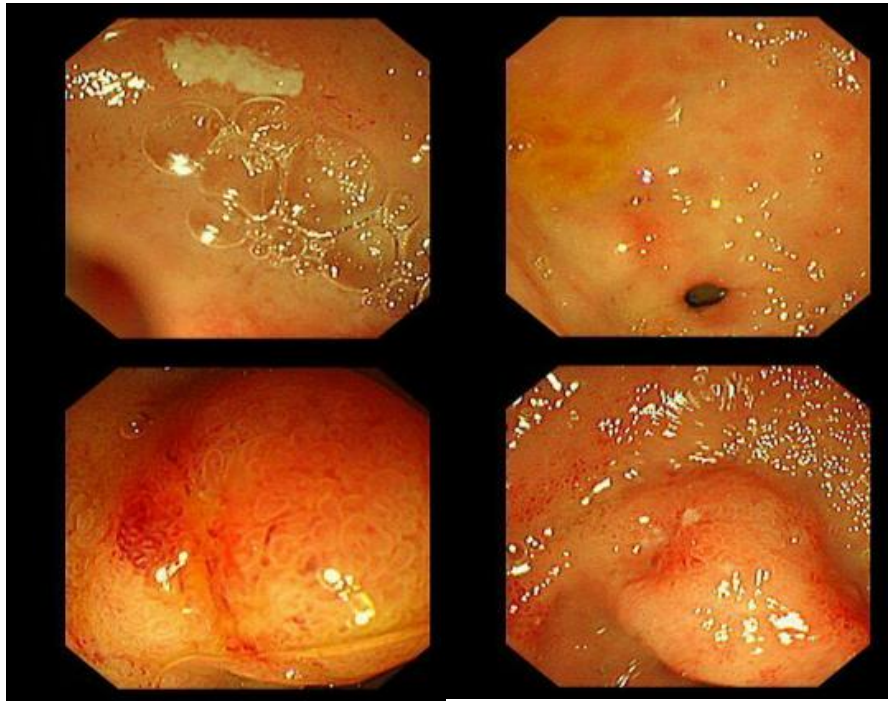
## Portovenous phase



Repeat CT-AP with dedicated arterial phase contrast did not identify any primary pancreatic or duodenal lesion. The peripancreatic nodes (yellow arrowhead) which correlated with sites of DOTATATE avidity demonstrated arterial enhancement with reduced conspicuity in the portovenous phase. A gastric antral mucosal lesion (green arrowhead) was also arterially enhancing, but not DOTATATE avid and when sampled on OGD, it was negative for neuroendocrine cell proliferation.

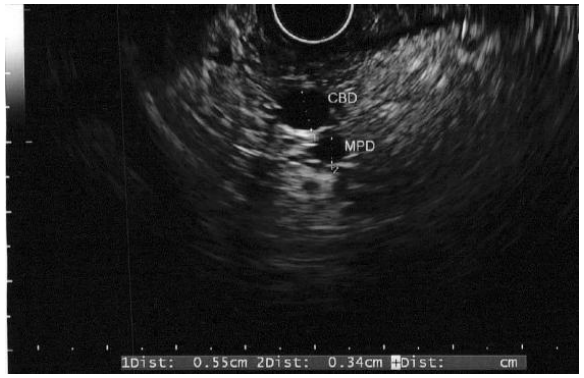
# Proving the diagnosis

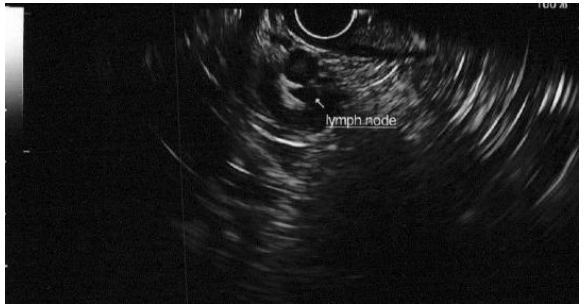
**Endoscopic findings:**
*"The antrum appeared atrophic but there was no mucosal mass. Erosive duodenitis in D1 with a few small ulcers. Mucosal biopsies taken."*

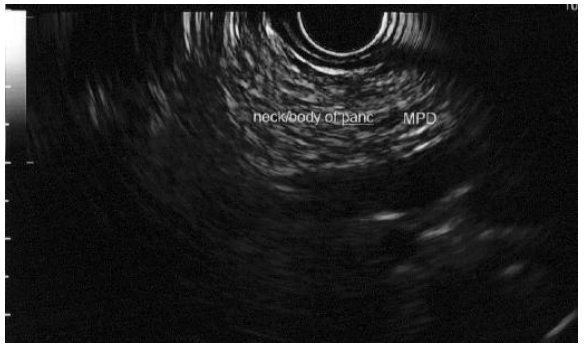


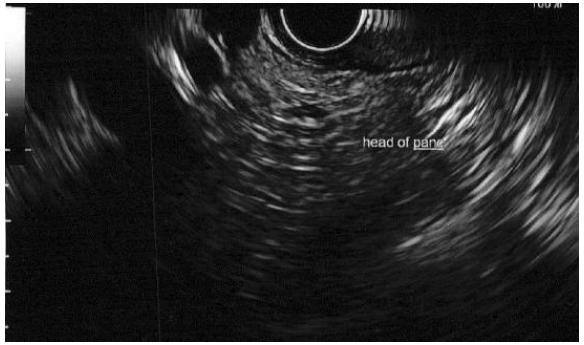
**EUS findings:**
*"Cluster of small peri-pancreatic lymph nodes best seen from D1, closed related to the superior mesenteric artery. Lymph node appearances were non-specific and measured about 20 mm.*

*Normal pancreas with normal main pancreatic duct throughout, measuring 3mm. Normal left lobe of the liver. Normal CBD measuring 5 mm. Left kidney and left adrenal normal. Normal coeliac axis. Gallbladder not visualised."*

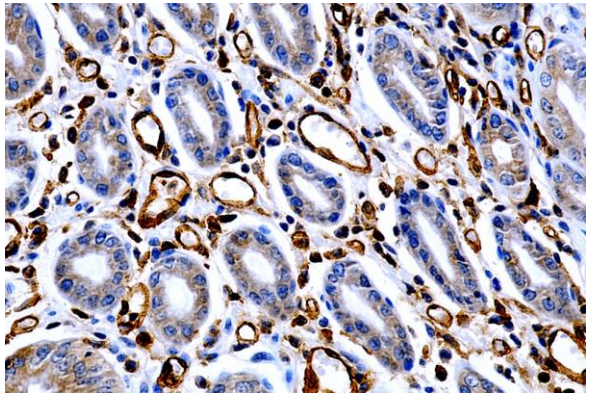


# Proving the diagnosis

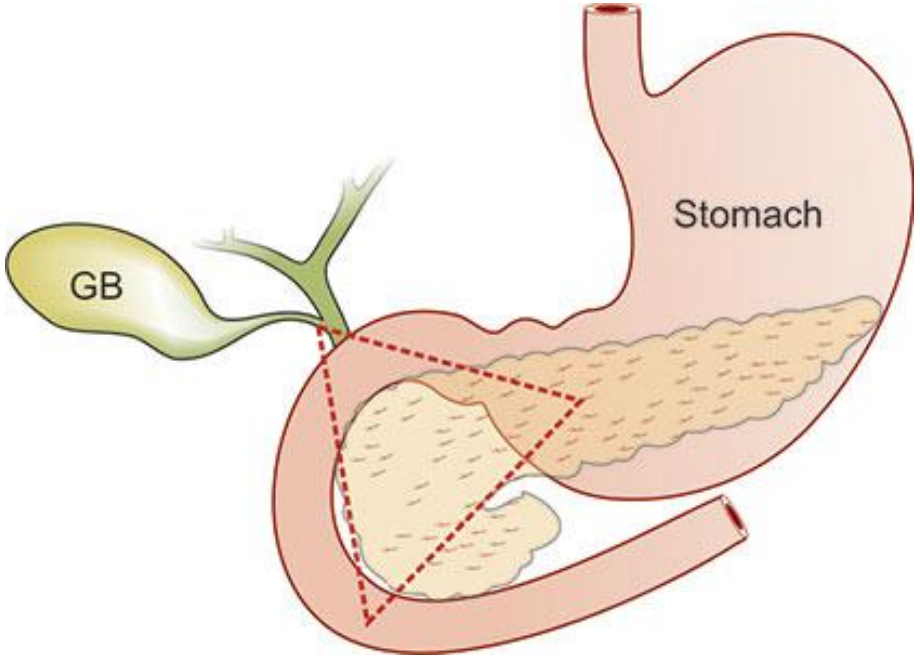


- **Histology from EUS-guided biopsy of these peripancreatic nodes ultimately confirmed well-differentiated gastrinoma**. No primary pancreatic/duodenal lesion was found on multiple CTs, MRIs and PET-CT, thus the patient was suggested to have primary lymph node gastrinoma.
- **The patient was treated for Zollinger-Ellison syndrome with Lanreotide** and high dose PPIs which was well tolerated and significantly improved her symptoms, leading to normalised gastrin levels and no further hospitalisations since they were started.
- **Extensive genetic panel testing for MEN-1 pathogenic variants was negative**. This does NOT preclude a genetic diagnosis. Germline pathogenic variants in the MEN1 gene are found in 87% of familial cases and only 45% of sporadic cases [1]. The likelihood of finding a mutation is correlated with the number of MEN1-related tumours (mutation detection rate of 79%, 37% and 15% in patients with three, two and one main MEN1-related tumours) and increased in with positive family history [1].

# Gastrinomas, Zollinger-Ellison Syndrome & Multiple Endocrine Neoplasia 1

- Gastrinomas are neuroendocrine tumours that ectopically secrete gastrin, leading to a clinical syndrome of refractory peptic ulcers first described by Zollinger & Ellison in 1955 [2].
- The incidence of gastrinomas is estimated to be between 0.5-4 new cases per year per million people [3]. Most gastrinomas (>60%) are malignant and have metastasized at the time of diagnosis [4].
- 20-30% of cases Zollinger-Ellison syndrome occur as part of MEN-1 whilst the majority are sporadic [5]. MEN-1 is an autosomal dominant condition due to mutation in the MEN1 gene characterised by multiple neuroendocrine tumours of the pancreas, parathyroid gland and pituitary gland [6].
- 85-90% of gastrinomas arise within Passaro's triangle (a.k.a. gastrinoma triangle) either as a pancreatic or duodenal lesion [7]. More rarely they can arise within the liver, ovaries or in lymph nodes [4].

# Passaro's triangle



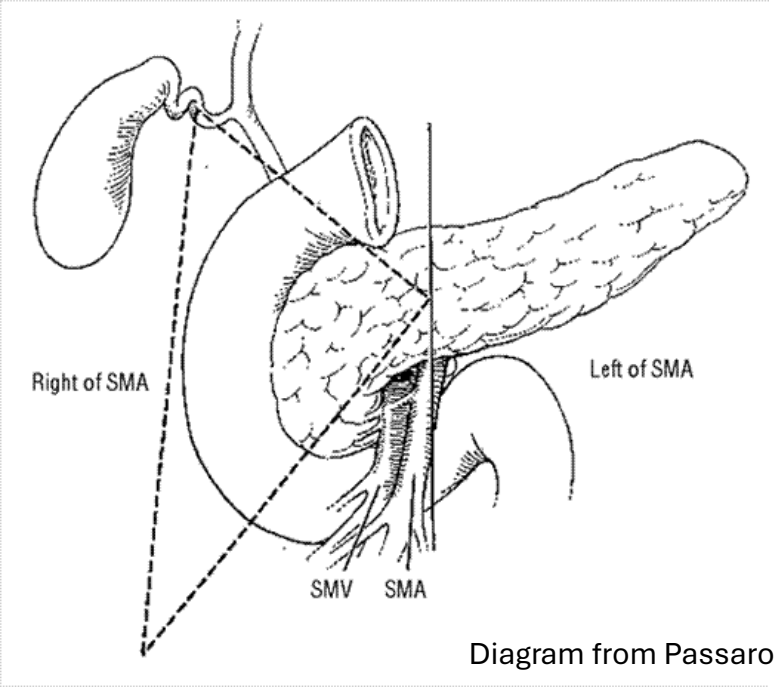




Diagram from Passaro et al. 1998 [8]

Passaro's triangle lies to the right of the superior mesenteric artery. 85-90% of gastrinomas are found within this triangle. It is defined by:

- Δ Confluence of the cystic duct and common bile duct superiorly
- Δ Junction of the D2 and D3 segments of the duodenum inferiorly
- Δ Junction of the neck and body of the pancreas medially

# Embryological origins

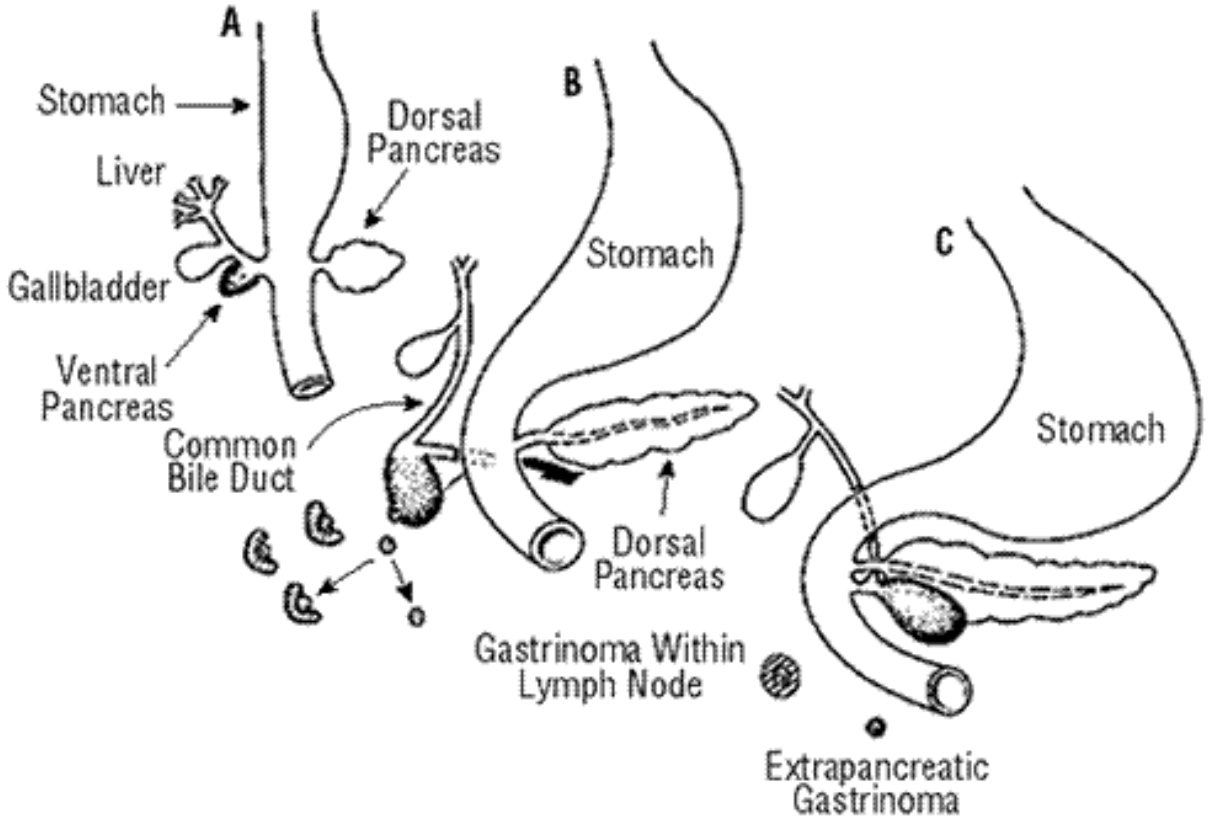



Diagram from Passaro et al. 1998 [8]

- Embryologically, gastrinomas arise from pluripotent stem cells of ventral pancreatic bud origin that become dispersed and incorporated into the duodenal wall during dorsal rotation [8].
- The ventral pancreatic bud predominantly gives rise to gastrinomas, PPomas and somatostatinomas of which 71-80% lie within Passaro's triangle [8].
- The dorsal pancreatic bud predominantly gives rise to glucagonomas and insulinomas, of which 74-77% lie outside of this triangle [8].

# Primary lymph node gastrinoma

- Primary lymph node localisation of gastrinoma has been reported in several case reports/series but remains a debated entity as to whether these cases represent metastatic disease from an occult primary tumour (microgastrinoma) or de novo occurrence of a gastrinoma in a lymph node [4,9,10].
- Some studies report that primary lymph node gastrinomas account for up to 10% of sporadic gastrinomas with one series reporting 18 patients who had only lymph node gastrinomas resected who remained disease free for a mean follow-up of 10 years [9,10].
- One theory that could explain the existence of lymph node gastrinoma localisation fits with the current evidence for the embryological origin of gastrinomas from the ventral bud of the pancreas. As the ventral bud rotates dorsally, some pluripotent stem cells migrate/diffuse towards the peripancreatic lymph node structures where they become trapped and proliferate [8,10].
- Regardless, the gold standard for localisation of functional pancreatic neuroendocrine tumours such as gastrinomas remains functional somatostatin receptor selective imaging of which DOTATATE PET-CT is the most sensitive [11].

# Learning points

- Whilst PPIs can falsely elevate gastrin levels, Zollinger-Ellison syndrome must be considered in all patients with intractable gastritis symptoms, refractory to PPIs. Most gastrinomas (60%) are malignant, so early diagnosis can allow for surgical intervention with curative intent.
- Most gastrinomas (85-90%) lie within Passaro's triangle, right of the SMA, and are extrapancreatic. In rare cases they might only be present in peripancreatic lymph nodes. Careful inspection of this region is needed to avoid missing lesions.
- Arterial phase CT imaging can help identify pancreatic neuroendocrine tumours like gastrinomas which enhance avidly in this phase; they typically appear isodense in the portovenous phase and can thus be missed if only portovenous phase imaging is acquired.
- [Ga-68]-DOTATATE PET-CT is the most sensitive imaging modality to identify pancreatic neuroendocrine tumours. Involvement of nuclear medicine is helpful to differentiate functional masses from non-functional as well as finding occult gastrinomas.

# References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# Synopsis

A 55 year old female with a previous cholecystectomy presented to A&E over 20 times in two years with recurrent abdominal pain, non-bilious/bloody vomiting and non-bloody diarrhoea.

Physical examination, routine biochemical tests, coeliac antibody testing, faecal calprotectin, faecal elastase and stool cultures were all normal. A borderline elevated gastrin level (220 pmol/L) was dismissed due to concurrent PPI use. Urinary 5-HIAA testing did not support carcinoid syndrome. Abdominal ultrasound only revealed mild hepatic steatosis.

OGD demonstrated erosive antral gastritis and duodenitis but histology was negative for dysplasia and neuroendocrine cell proliferation. Tests for H. pylori and anti-gastric parietal cell antibodies were also negative. Colonoscopy was normal and MRCP did not find choledocholithiasis. SeHCAT studies and gastric emptying tests ruled out bile salt malabsorption and gastroparesis.

Multiple portovenous phase CTs demonstrated intestinal non-rotation but no other abnormality was identified. MRI small bowel confirmed intestinal non-rotation but demonstrated no active inflammation and video capsule endoscopy was unremarkable. No primary pancreatic/duodenal lesions were identified on MRI pancreas with dynamic contrast.

Repeat blood tests revealed a hypercalcaemia secondary to primary hyperparathyroidism but also significantly raised gastrin (1501 pmol/L) and chromogranin levels. Following this [Ga-68]-DOTATATE PET-CT was performed which identified 4 avid peripancreatic lymph nodes in Passaro's triangle. These nodes demonstrated early enhancement on arterial phase CT, further supporting the suspicion of NET.

EUS-guided biopsy of these peripancreatic nodes ultimately confirmed well-differentiated gastrinoma. Zollinger-Ellison syndrome was diagnosed and the patient was started on somatostatin analogues which significantly improved her symptoms. MEN-1 genetic testing was negative although this does not preclude a diagnosis.