$^{99m}$Tc-Hynic-Tyr$^3$-Octreotide Uptake in a Patient with Hypergastrinemia

Dr. Augusto Llamas-Olier and Dr. Emperatriz Angarita.
Nuclear medicine department. Instituto Nacional de Cancerologia.
Bogota, Colombia.
• 82 year old female
• Complaint: gastritis
• **Lab results:** hypergastrinemia (fasting serum gastrin, 1600 pg/ml).
• **Upper endoscopy** findings:
  • Micronodular mucosa in gastric fundus with multiple 1-mm lesions.
  • 3 lesions > 1 mm were resected.
• **Anatomopathology** results:
  • Diffuse chronic atrophic gastritis.
  • Focal intestinal metaplasia with no dysplasia.
  • Neuroendocrine micronodular hyperplasia
  • Type-1 neuroendocrine tumor suspected.
• The patient refused surgical antrectomy.
• A **somatostatin receptor scintigraphy** was ordered to identify a gastrinoma and to rule out a gastric carcinoid.
$^{99m}$Tc-HYNIC-TOC imaging. There is focal uptake in the gastric antrum.

CT slice provided for orientation purposes. Not the same patient.
• The stomach contains at least five types of endocrine cells. Each endocrine cell secretes a dominant chemical messenger (peptide or amine).

• Histamine-secreting enterochromaffin-like (ECL) cells are the most common gastric neuroendocrine cell type (up to 80% of mucosal neuroendocrine cells).

• G cells are mostly found in the gastric antrum, and gastrin functions as a classical hormone to activate ECL cells, which are predominately distributed in the gastric fundus.

• Gastrin produced by antral G cells results in activation of ECL cells with consequent histamine release activating parietal cell H2 receptors with production of hydrochloric acid.
Discussion

- ECL cell secretion is inhibited by somatostatin, which binds to a variety of somatostatin receptor subtypes expressed on the ECL cell membrane.

- Loss of parietal cells (e.g., in atrophic gastritis) or acid suppression can lead to increased gastrin secretion, proliferation of enterochromaffin-like cells, and neoplasia (i.e., Type-1 gastric carcinoids).
Teaching Points

- In type 1 gastric carcinoids, diminution of parietal cell function (e.g., autoimmune destruction in pernicious anaemia or atrophic gastritis) reduces luminal acidity, which stimulates gastrin secretion and prolonged hypergastrinemia, culminating in ECL cell proliferation through phases of hyperplasia, dysplasia, and neoplasia.

- Gastric carcinoids are of increasing clinical concern because they may develop in hypergastrinemic states. However, they are difficult to diagnose.

- Somatostatin receptor scintigraphy is a reasonably sensitive and highly specific imaging modality to localize gastric carcinoids in patients with hypergastrinemic states.
Teaching Points

• In the present case, no abnormal uptake was seen in the gastric fundus, therefore we can confidently rule out a gastric carcinoid.

• We can only speculate that the focal uptake in the gastric antrum was a false positive result induced by G cell hyperplasia.

• Antrectomy is no longer considered a useful treatment because the antrum only produces 60% of the body gastrin, whereas the duodenum can produce the remaining 40%.
References

