gastroenteropancreatic neuroendocrine neoplasm: MDCT, MRI and MRI diffusion findings.

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Learning objectives

- To review the clinical features and histological classification of gastroenteropancreatic (GEP) neuroendocrine neoplasms.

- To illustrate the radiological characteristics of these tumors using MDCT, conventional MRI and MRI diffusion-weighted imaging.

Acronyms:

GEP: gastroenteropancreatic

GI: gastrointestinal.

MEN syndrome: multiple endocrine neoplasia syndrome.

NETs: neuroendocrine tumors

PNET: pancreatic neuroendocrine tumors

ZES: Zollinger- Ellison syndrome.
Background

NEUROENDOCRINE TUMORS (NETS) comprise a clinical and radiological heterogeneous group of neoplasms derived from peptide- and amine-producing cells of the neuroendocrine system. They are characterized histologically by the intracellular presence of markers of endocrine tissue, such as chromogranin A, synaptophysin, and neuron-specific enolase (FIGURE 1 background on page 7).

PRIMARY TUMOR SITE:

NETs can arise in almost any organ. Approximately two thirds are found in the gastrointestinal (GI) tract, and approximately one quarter occur in the lung, with the remainder arising in pancreas, thymus, adrenal glands (pheochromocytomas), thyroid, hepatobiliary tract, and ovary. Very rarely NETs may originate from the gallbladder, kidney, prostate, middle ear or testis among other organs.

The gastroenteropancreatic (GEP) NETs subgroup, which includes gastrointestinal and pancreatic endocrine tumors, accounts, for the majority of NETs.

In the GI tract, NETs account for approximately 1.5% of all gastrointestinal neoplasms. They can arise from esophagus to rectum. The appendix is the most common location (30-90% of all GI NETs), followed by the ileum (25-35%) and rectum (10%).

The term carcinoid tumor refers to neuroendocrine tumors that secrete serotonin (5-hydroxytryptamine). It remains in use as a synonym for well-differentiated NETs of the GI tract and lung.

For clinicians carcinoid syndrome refers to a serotonin-producing tumor with characteristic clinical manifestations that include cutaneous flushing and gut hypermotility with diarrhea. The classic carcinoid syndrome develops in less than 10% of patients with GI carcinoids and it is more likely to be present in the setting of metastases.

Pancreatic NETs (PNETS) are also denominated islet cell tumors as they originate from endocrine cells of the islets of Langerhans. They account for 3-1% of all NETs.

EPIDEMIOLOGY:

NETs are relatively uncommon, with an annual incidence of 2 cases per 100,000 persons in the general population and they account for 0.5% of all malignancies.
Excluding appendiceal tumors, their peak incidence is at the age of 65 years and they occur equally in men and women.

Appendiceal NETs are more frequently seen in young patients, particularly in women. This may be explained by the fact that these tumors are often incidental findings after acute appendicectomy, which is five times more often performed in women.

In USA,NETs occur more frequently in African Americans as compared with other ethnicities.

NETs usually present sporadically, but can be occasionally associated with genetic syndromes such as multiple endocrine neoplasia type 1 (MEN1) syndrome, von Hippel-Lindau (VHL) disease, neurofibromatosis type 1, and tuberous sclerosis (TABLE 1 on page 7).

**PATHOLOGIC CLASSIFICATION AND STAGING:**

Traditionally NETs have been classified according to their embryologic derivation as foregut, midgut, and hindgut tumors (TABLE 2 on page 8).

Later several histology-based classification systems, in which the tumor subtype is determined according to cellular differentiation, have been proposed for tumors of specific organs such as the lung, the pancreas, or the GI tract. Most of these classification systems have proven to correlate with patient survival and they are therefore useful for grading and to stratify prognostic subgroups of NETs. The more recently proposed classifications for thoracic and GEP NETs are summarized in TABLE 3 on page 9.

In general NETs are divided into **low, intermediate and high grade** according to their cellular **proliferative grade** (TABLE 4 on page 10) and into **well-differentiated** and **poorly differentiated** categories. Well-differentiated NETs include both low- and intermediate-grade groups whereas poorly differentiated NETs include high grade group.

Also recently, TNM staging systems for NETs of different anatomical sites have been published by the American Joint Committee on Cancer (AJCC) and the European Neuroendocrine Tumor Society (ENETS).

**CLINICAL FEATURES OF GEP NETS:**

All endocrine tumors are hormonally active to a variable degree, but they are typically divided into syndromic (functional, 85%) and nonsyndromic (nonfunctional, 15%) depending on their clinical and laboratory findings.
Patients with early stage GI NETs have nonspecific abdominal symptoms such as cramping and mild episodic diarrhea. More advanced disease usually presents with acute gastrointestinal obstruction from primary tumor or mesenteric fibrosis and carcinoid syndrome in the setting of liver metastases.

Presenting symptoms of nonfunctional pancreatic NETs are mainly related to mass effect or metastatic disease and include abdominal pain, weight loss, or jaundice. These tumors usually present an advanced stage when first diagnosed and have a poorer prognosis than functional neoplasms.

Functional pancreatic NETs usually manifest early owing to the symptoms of hormone overproduction. Tumors are named according to the predominant hormone they secrete. Insulinoma is the most common, followed by gastrinoma. Other types include glucagonoma, vipoma, and somatostatinoma. Main clinical features of functional pancreatic NETs are summarized in TABLE 5 on page 11.

IMAGING OF GEP NETS:

Imaging studies are needed for all phases of management of patients with GEP NETs, such as identification of primary tumor, staging, treatment planning and follow-up after surgical or medical treatment. Imaging techniques commonly used include contrast-enhanced MRI and CT, endoscopic and intraoperative ultrasound, PET and somatostatin receptor scintigraphy (Octreoscan).

NETs frequently overexpress somatostatin receptors which bind with high affinity various synthetic analogs of somatostatin such as octreotide. Octreoscan is a scintigraphy performed with 111In-labeled octreotide and was developed for NETs scanning. This imaging method has been reported to be useful for the detection and staging of NETs. It has the advantage of allowing total body scanning quickly at one time, and its use results in a change in management of a significant number of patients with NETs. Somatostatin receptor scintigraphy combined with CT detection (SPECT imaging) is more sensitive that conventional imaging for detection of both the primary NET (except nonmetastatic insulinomas) and metastases to liver, bone or other distant sites.

PROGNOSIS AND TREATMENT:

Most GEP-NETs are well differentiated and slow-growing. Even though their growth is slow in comparison with adenocarcinomas, it is generally recognized that, with the exception of 90% of insulinomas, almost all of them have long-term malignant potential and a substantial risk of relapse after surgery.

Predicting the clinical behaviour of well-differentiated NETs is difficult. In general, larger tumors tend to behave more aggressively, with invasion of local anatomic structures and
vessels and are more likely to metastasize. The presence or absence of metastases is the major predictor of survival.

However, even in the presence of metastatic disease, patients can survive for several years with current treatment strategies.

Management strategies include surgery of primary tumor (curative or cytoreductive), and medical treatment such as chemotherapy and biotherapy with somatostatin analogues and alpha-interferon to control tumor growth and symptoms of carcinoid syndrome. Therapy directed to liver metastases includes hepatic cytoreductive surgery, radiofrequency ablation, arterial embolization and transplantation in selected cases.
**FIGURE 1 background:** Photomicrographs of a well-differentiated NET of the pancreas showing reactivity with antibodies to chromogranin A (A) and synaptophysin (B).

**Fig. 0:** FIG 1 Backgr

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**Table 1: Heritable Tumor Syndromes Associated with Neuroendocrine Tumors**

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Associated NET Type</th>
<th>Frequency of PNETs presentation</th>
<th>Other tumors</th>
</tr>
</thead>
<tbody>
<tr>
<td>MEN (1) type 1</td>
<td>-Gastrinoma (majority duodenal) &gt; other PNET&lt;br&gt;-Less frequently gastric, lung and thymic carcinoids and paragangiomas</td>
<td>80-100% of patients</td>
<td>-Parathyroid -&gt; pituitary -&gt; adrenal -&gt; thyroid adenomas&lt;br&gt;-skin tumors</td>
</tr>
<tr>
<td>VHL disease (2)</td>
<td>Nonfunctional PNET</td>
<td>10-17% of patients</td>
<td>-Central nervous system hemangioblastomas&lt;br&gt;-retinal angiomas&lt;br&gt;-renal cell carcinomas, &lt;br&gt;-pheochromocytomas</td>
</tr>
<tr>
<td>Neurofibromatosis type 1</td>
<td>Duodenal Somatostatinoma&lt;br&gt;nonfunctional PNET, insulinoma, and gastrinoma</td>
<td>10% of patients</td>
<td>-plexiform neuromas&lt;br&gt;-Skin sebaceous adenoma&lt;br&gt;-Subependymal and cortical hamartoma&lt;br&gt;-Giant cell astrocytoma</td>
</tr>
<tr>
<td>tuberous sclerosis</td>
<td></td>
<td>occasionally</td>
<td></td>
</tr>
</tbody>
</table>

(1) Multiple Endocrine neoplasia syndrome<br>(2) von Hippel–Lindau disease

**Fig. 0: TABLE 1**

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<table>
<thead>
<tr>
<th>Foregut tumors</th>
<th>Midgut tumors (1)</th>
<th>Hindgut tumors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thymus</td>
<td>Jejunum</td>
<td>Transverse colon</td>
</tr>
<tr>
<td>Bronchopulmonary tract (2)</td>
<td>Ileum</td>
<td>Descending colon</td>
</tr>
<tr>
<td>Esophagus</td>
<td>Appendix (3)</td>
<td>Rectum</td>
</tr>
<tr>
<td>Stomach</td>
<td>Ascending colon</td>
<td>Ovary or testis</td>
</tr>
<tr>
<td>Duodenum</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pancreas</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hepatobiliary tract</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(1): account for 2/3 of NETs. (2) account for 1/4 of NETs. (3) most common GI NETs

**Fig. 0: TABLE 2**

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### Table 3: Systems of Nomenclature for Neuroendocrine Tumors

<table>
<thead>
<tr>
<th>Differentiation</th>
<th>Proliferative Grade (1)</th>
<th>Lung and Thymus (WHO) (2)</th>
<th>GEP-NETs (ENETS (3), WHO 2010)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Well differentiated</td>
<td>Low grade</td>
<td>Carcinoid tumor</td>
<td>Neuroendocrine tumor grade 1 (G1)</td>
</tr>
<tr>
<td></td>
<td>Intermediate grade</td>
<td>Atypical carcinoid tumor</td>
<td>Neuroendocrine tumor grade 2 (G2)</td>
</tr>
<tr>
<td>Poorly differentiated</td>
<td>High grade</td>
<td>Small cell carcinoma (4)</td>
<td>Neuroendocrine carcinoma grade 3 (G3), small cell carcinoma (4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Large cell neuroendocrine carcinoma (4)</td>
<td>Neuroendocrine carcinoma grade 3 (G3), large cell neuroendocrine carcinoma (4)</td>
</tr>
</tbody>
</table>

1. See table 4  
2. World Health Organization  
3. European Neuroendocrine Tumor Society  
4. Cell size and nuclear morphology are used to distinguish small cell carcinoma from large cell neuroendocrine carcinoma

**Fig. 0: TABLE 3**

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Table 4: Grading Systems for Neuroendocrine Tumors

<table>
<thead>
<tr>
<th>Proliferative Grade (1)</th>
<th>Lung and Thymus (WHO)(2)</th>
<th>GEP-NETs (ENETS(3), WHO 2010)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low grade</td>
<td>&lt;2 mitoses / 10 hpf (1)</td>
<td>&lt;2 mitoses / 10 hpf AND &lt;3% Ki67 index (1)</td>
</tr>
<tr>
<td></td>
<td>AND no necrosis</td>
<td></td>
</tr>
<tr>
<td>Intermediate grade</td>
<td>2-10 mitoses / 10 hpf (1)</td>
<td>2-20 mitoses / 10 hpf OR 3-20% Ki67 index (1)</td>
</tr>
<tr>
<td></td>
<td>OR foci of necrosis</td>
<td></td>
</tr>
<tr>
<td>High grade</td>
<td>&gt;10 mitoses / 10 hpf (1)</td>
<td>&gt;20 mitoses / 10 hpf OR &gt;20% Ki67 index (1)</td>
</tr>
</tbody>
</table>

(1) assessed as the number of mitoses per unit area of tumor (expressed as mitoses per 10 high-power microscopic fields) or as the percentage of neoplastic cells immunolabeling for the proliferation marker Ki67.

(2) World Health Organization

(3) European Neuroendocrine Tumor Society

Fig. 0: TABLE 4

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Table 5: Clinical Presentation of Functional Pancreatic NETs

<table>
<thead>
<tr>
<th>Tumor</th>
<th>Symptoms or Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insulinoma</td>
<td>Whipple triade: fasting serum glucose &lt; 50 mg/Dl; Symptoms related to hypoglycemia (intermittent confusion, sweating, weakness, nausea); and symptom relief following glucose administration.</td>
</tr>
<tr>
<td>Gastrinoma</td>
<td>Hypergastrinemia: severe peptic disease and diarrhea (ZES(^{(1)})). Tumor may be sporadic or associated to MEN1</td>
</tr>
<tr>
<td>glucagonoma</td>
<td>Diabetes, dermatitis, glossitis, cachexia, deep venous thrombosis</td>
</tr>
<tr>
<td>Vipoma(^{(1)})</td>
<td>Watery diarrhea, hypokalemia and achlorhydia</td>
</tr>
<tr>
<td>somatostatinoma</td>
<td>Diabetes, diarrhea, cholelithiasis</td>
</tr>
</tbody>
</table>

\(^{(1)}\) Zollinger-Ellison syndrome: severe peptic disease caused by tumoral gastrine overproduction.

Fig. 0: TABLE 5

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Imaging findings OR Procedure details

In this study we retrospectively review the clinical manifestations and radiological findings of 15 cases of GEP neuroendocrine neoplasms (8 gastrointestinal and 7 pancreatic) reported at our institution during a 2-year period, between January 2008 and December 2010. We include 6 men and 9 women with an age range of 19 - 80 years (mean age: 54 years).

All our cases but 2 were nonfunctional lesions at presentation. 1 patient with a pancreatic insulinoma was diagnosed because of neurological symptoms of hypoglycemia (episodic confusion and aggressiveness). Another patient with an hereditary MEN1 syndrome presented with hypergastrinemia and peptic disease suspicious for gastrinoma. He had already been diagnosed of parathyroid and pituitary adenomas, carotid glomus and pulmonary carcinoid tumor.

Epidemiologic features, primary tumor site, clinical manifestations, diagnostic procedure and pathological findings are shown in TABLES 6A on page 20 and 6B on page 20.

Of 15 cases reviewed, 2 carcinoid tumors of the appendix were incidental surgical findings and had no radiological explorations.

In 1 patient with a cecal carcinoid and gastrointestinal obstruction, the tumor was also a surgical finding and we do not have radiological images of the primary tumor. Both MDCT and MRI scans were later performed in this patient to search for hepatic metastases.

In the remaining 12 cases, 1 pancreatic lesion was studied only with MRI, 4 pancreatic tumors were studied with both MDCT and MRI and 7 lesions (2 pancreatic and 5 gastrointestinal tumors) were studied with MDCT.

MDCT scans where performed in a 20-section multidetector CT scanner and included arterial phase (35 seconds delay) and venous phase (70 seconds delay) in all patients. MRI scans were performed in a 1,5 T system. They included axial T1-WI, axial T2-WI and 3D fat suppressed T1-WI dynamic contrast enhanced images of liver and pancreas (0s, 30s, 60s, 120s and 240s scan delay). Diffusion MR images (b factor = 600) were obtained in 3 patients with pancreatic NETs and in one patient with liver metastases of a cecal NET.

6 patients presented with focal pancreatic tumors of variable size and enhancement, 1 patient with diffuse pancreatic infiltration, and 3 patients with enhancing gastric or ileal mural lesions. 2 gastrointestinal lesions (gastric and rectal), smaller than 1 cm, were not visible on MDCT scans.
Liver metastases were demonstrated in 5 patients, bone metastases in 1 patient and lung metastases 1 patient.

Main radiological findings of patients of this study are summarized in TABLES 7A on page 21 and 7B on page 22.

A- GASTROINTESTINAL NETs (OR CARCINOID TUMORS):

Clinical and radiological manifestations of GI NETs vary depending on anatomical location of the primary tumor along the gastrointestinal tract.

Esophageal NETs:

They are extremely rare. Patients usually present with dysphagia. Tumors are usually located in the distal esophagus and manifest as a small polypoid or ulcerative tumor similar to esophageal carcinomas.

Gastric NETs:

In the pre-endoscopy era, gastric NETs comprised 2% of all carcinoids, but in more recent studies, 10% to 30% of all carcinoids are reported in the stomach. They comprise less than 1% of gastric neoplasms.

Three subtypes of gastric carcinoids have been described, each with different morphologic, clinical and prognostic characteristics:

- Type I: It is the most common subtype (70%-80%) and is associated with achlorhydric atrophic gastritis of the gastric body. Type I tumors are often detected incidentally at endoscopy performed because of dyspepsia. In CT they are seen as multiple small enhancing mucosal nodules (of 1 cm of size or smaller) within the gastric fundus and body. They are usually considered benign and treated with endoscopic resection (FIGURE 1 on page 23).

- Type II: They are the least common (5%-10%) and occur almost exclusively in MEN1 patients with a gastrinoma and associated Zollinger-Ellison syndrome (ZES). The tumors are multiple and usually small. However, in 10%-30% of cases they give metastases to regional lymph nodes and liver. On images these tumors present as multiple masses associated to gastric wall thickening. Treatment includes medical therapy with oral octreotide, which may induce gastric tumor regression.
- Type III: Relatively common (15%-25%). They are sporadic and occur in the absence of predisposing gastric pathologic conditions. They are generally solitary, larger than 2 cm and behave more aggressively than do other subtypes. Metastatic disease depends on tumor size. Cross-sectional imaging and octreotide scintigraphy are important for staging this subtype of gastric NETs. Treatment is similar to that for gastric adenocarcinoma (FIGURE 2 on page 24).

Duodenal NETs:

Carcinoid tumors of the duodenum account for approximately 2% of all gastrointestinal NETs. They are the third most common malignant tumors of the duodenum (11%) after adenocarcinoma (73%) and leiomyosarcoma (14%). Most are sporadic and small non-functioning tumor discovered incidentally during endoscopy. The majority of duodenal carcinoids (62%) are gastrinoma, and one-third of these are functioning tumors that manifest clinically as ZES. They can also be associated with MEN1 syndrome. Duodenal NETs are usually smaller than 1 cm and are located predominantly in the proximal duodenum. When associated with MEN1 they are usually multiple. Despite their small size, metastases are often found in the regional lymph nodes at the time of diagnosis (FIGURE 3A on page 25 and 3B on page 26).

Periampullary NETs can be in 30% of cases associated with type 1 neurofibromatosis.

On endoscopic ultrasonography images duodenal NETs are depicted as a small, polypoid, submucosal lesion. On CT they appear as focal mural or intraluminal duodenal masses, which may demonstrate early arterial phase enhancement.

Jejunal and ileal NETs:

Carcinoid tumor is the second most common malignant small-bowel neoplasm after adenocarcinoma. Most small-bowel NETs occur in the distal ileum and up to one third are multiple.

Most tumors are small and difficult or even impossible to detect on enteroclysis or cross-sectional images.

On MDCT small primary tumors can be detected as hyperenhancing nodules, especially if water is used as oral contrast agent and multiplanar image reformation is performed. More commonly CT detects a mesenteric desmoplastic reaction as an ill defined soft tissue mass that retraces ileal loops because of fibrosis. Even in very small primary tumors without direct tumoral invasion there is a mesenteric desmoplastic reaction due to ischemia and fibrosis caused by release of serotonin. Mesenteric mass presents calcification in approximately 70% of cases. Wall thickening of adjacent bowel loops due to ischemia can also be seen as a consequence of mesenteric vessels encasement (FIGURE 4 on page 27).
On MR images, primary tumors are also difficult to detect and it is more common that examination only reveals a mesenteric mass or hepatic metastases. Primary tumor may appear as well-defined nodular masses or regional bowel wall thickening, isointense on T1-WI, iso- or mild hyperintense on T2-WI and with intense homogeneous enhancement after contrast. Mesenteric metastases or desmoplastic reaction appear as a stellate soft tissue mass with intermediate or low signal intensity (SI) on T1 and T2-WI, with moderate to intense enhancement on delayed images. Peritoneal metastases present intermediate SI on T1-WI and intermediate-high SI on T2-WI. Identification of both mesenteric and peritoneal metastases is best on delayed fat-suppressed contrast enhanced T1-WI.

**Appendiceal NETs:**

Appendiceal carcinoid tumor is the most common GI NET and it is also the most common type of appendiceal tumor. Very often it is discovered incidentally at pathologic examination after appendectomy, as in the 2 patients we report.

Tumors are typically small (in 95% of cases < 2 cm) and not detected radiologically. If calcified, they can may mimic appendicoliths. Larger tumors may present as diffuse mural thickening or as a mesenteric soft-tissue mass.

Small (<1 cm) well-differentiated carcinoids confined to the tip of the appendix that are completely excised can be considered as cured. Larger tumors, tumors with invasion of mesoappendix or the base of the appendix, or with mesenteric nodal involvement, should undergo a right hemicolecction.

**Colonic NETs:**

They are very rare and occur more commonly in the right colon. They are usually poorly differentiated tumors and present as large lesions similar to colonic carcinoma. Most colonic NETs have already metastasized at the time of diagnosis, as the patient we report.

**Rectal NETs:**

Rectal NETs are much more common than colonic NETs, representing about 11% of all gastrointestinal carcinoids. Most of them (85%) are submucosal single tumors, smaller than 1 cm, detected incidentally at colonoscopy and still localized at the time of diagnosis, as the case of our study.

Imaging modalities for disease staging of larger tumors include CT, MRI and octreoscan.

Endoscopic US is ideally suited for evaluation of localized rectal NETs since it can accurately depict the depth of invasion, as well as perirectal nodal spread.
**B- PANCREATIC NETS (PNETs) OR ISLET CELL TUMORS:**

NETs of the pancreas (PNETs) have an estimated incidence of 1-1.5 per 100,000 individuals. They are slow-growing tumors that can be located within the gland or be peripancreatic or exophytic. Unlike adenocarcinomas, they rarely result in pancreatic ductal dilatation.

**Functional or syndromic tumors:**

In general, syndromic PNETs manifest at a relatively smaller size (less than 3 cm) than nonsyndromic PNETs owing to the symptoms produced by the associated hormone production. Insulinoma is the most common functional PNETs (50%) followed by gastrinoma (30%).

- Insulinomas are benign in 90% of cases, usually solitary (90%) and distributed uniformly throughout the pancreas (FIGURES 5A on page 28 and 5B on page 29). Treatment consists in surgical resection.

- Of gastrinomas, 70-75% are sporadic and 20-25% occur as part of the MEN1 syndrome. They produce an increased gastrin secretion that results in ZES. In contrast to the other types of syndromic PNETs, gastrinomas are commonly extrapancreatic. Most are located in the "gastrinoma triangle" that is bounded by the cystic duct, second and third portions of the duodenum and neck of pancreas. Of sporadic gastrinomas, 60-50% occur in the pancreas, 35-40% in the duodenal wall and the remainder in the stomach and lymph nodes. However, more recent studies suggest that in more than 60% of patients with sporadic gastrinomas tumors are found in duodenum. In patients with MEN-1 syndrome, gastrinomas are almost invariably multiple and of duodenal in origin in 85% of cases (FIGURES 6A on page 30 and 6B on page 31).

- Other functional PNETs as glucagonomas, VIPomas and somatostatinomas manifest late in the disease course and are usually 3- 5 cm in size at the time of diagnosis (larger than insulinomas and gastrinomas). The majority are malignant and may present with lymph node and liver metastases.

Functional PNETs are typically hyperenhancing relative to the pancreas on at least one vascular phase in CT and MRI scans and are usually best seen during the arterial phase. However, some neoplasms will be hypoattenuating relative to pancreas on CT images. MDCT multiplanar reformatted images have improved the identification of small PNETs since they allow a better differentiation between tumors and vascular structures.

On MRI images functional PNETs show low SI on fat suppressed T1-WI, high SI on T2-WI and greater homogeneous or annular enhancement than pancreas on dynamic contrast
images. Glucagonomas, VIPomas and somatostatinomas show more heterogenous enhancement.

Functional PNETs localization may be difficult because of their small size and lack of mass effect and duct dilatation (especially insulinomas and gastrinomas). Although conventional imaging studies detect more than 70% of PNETs that are greater than 3 cm, they detect less than 50% of most PNETs that are less than 1 cm, therefore frequently missing small primary PNETs and small liver metastases. However, most insulinomas can be found during surgery by means of intraoperative ultrasound.

**Nonfunctional (NF-PNETs) or nonsyndromic tumors:**

Nonsyndromic PNETs tend to be larger than syndromic PNETs (>5 cm in 70%) and at an advanced stage when first diagnosed, with 60% to 85% of them having liver metastases in most series. They present clinically with symptoms due to the tumor per se, which include primarily abdominal pain, weight loss, or jaundice. In recent years, they are increasingly being discovered by chance on imaging studies being performed for nonspecific abdominal symptoms.

On CT and MRI scans they are more likely to be cystic or necrotic or to show calcifications than functional PNETs. Cross-sectional imaging typically demonstrates a relatively large pancreatic mass that is indistinguishable from other pancreatic tumors. On MRI they appear as a mass with low SI on T1-WI relative to pancreas, with foci of high SI necrosis and cystic degeneration on T2-WI and heterogenous enhancement (**FIGURES 7A on page 32, 7B on page 33, 7C on page 34 and 8 on page 35**). Less commonly they can present low SI on T2-WI because of fibrous tissue content (**FIGURES 9A on page 36, 9B on page 37, 9C on page 38 and 9D on page 39**). Enhancement, commonly hyperintense, may be observed on early or delayed images (**FIGURES 10 on page 40, 9C on page 38, 11A on page 41, 11B on page 42 and 11C on page 43**). They have a wide differential diagnosis that includes adenocarcinoma, mucinous cystic tumor, metastases, solid pseudopapillary tumor of the pancreas and lymphoma.

Diffusion weighted (DW) MR images have a limited value in PNETs. It has been reported that DW MRI may be useful in punctual cases to detect a lesion in patients with clinical suspicion of PNET and negative or inconclusive imaging findings on conventional MRI. In our 3 cases of PNETs with DW MRI, those images didn’t add any significant information for tumor detection due to the large size of the lesions. Apparent diffusion coefficient (ADC) values obtained in PNET and liver metastases of our patients are shown in **FIGURES 7C on page 34, 9D on page 39, 11C on page 43 AND 12A on page 44**. We have found apparent diffusion coefficient (ADC) values of the PNETs and liver metastases lower than those of pancreatic and hepatic parenchyma but the number of our cases is too much limited to obtain any significant result.
C- IMAGING OF METASTATIC DISEASE:

Approximately 20% of patients with NETs have metastatic disease at presentation, and quite often the primary tumor is not located at initial imaging.

Liver and lymph nodes metastases are common in GEP NETs, especially in jejuno-ileal and colonic carcinoid tumors. Occasionally they also metastasize to other organs such as pancreas, ovaries, bone, lung, pleural and mediastinum (FIGURE 8 on page 35).

Metastases to the liver are characteristically hypervascular, often enhancing avidly on arterial phase (FIGURE 6B on page 31, 9C on page 38 and 10 on page 40). However they may also present delayed hyperenhancement (FIGURE 6B on page 31, 9A on page 36, 9C on page 38, 12A on page 44 and 12B on page 45). At MRI imaging, they typically demonstrate low SI on T1-WI and high SI on T2-WI (FIGURE 6A on page 30, 9B on page 37, 11A on page 41 and 12A on page 44).

Metastases to bone account for 7%-15% of all carcinoid metastases. They may present as sclerotic bone lesions on CT images. On MRI lesions may be identical to that of bone metastases from other primary tumors (FIGURE 9D on page 39). If sclerotic, they may show low SI on T2-WI.
### Table 6A: Epidemiologic and clinical features of GI NETs cases

<table>
<thead>
<tr>
<th>Case N°</th>
<th>Sexe (1)</th>
<th>Age (years)</th>
<th>Tumor primary site</th>
<th>Clinical presentation</th>
<th>Diagnostic procedure</th>
<th>Pathologic findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>19</td>
<td>Appendix</td>
<td>Acute appendicitis</td>
<td>Surgery</td>
<td>Well-differentiated NET</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>23</td>
<td>Appendix</td>
<td>Acute appendicitis</td>
<td>Surgery</td>
<td>Well-differentiated NET</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>80</td>
<td>Stomach</td>
<td>Anemia, atrophic gastritis</td>
<td>Surgery</td>
<td>Well-differentiated NET, Multifocal.</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>53</td>
<td>Stomach</td>
<td>Anemia, Endoscopic finding.</td>
<td>Endoscopic biopsy</td>
<td>Well-differentiated NET</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>58</td>
<td>Stomach</td>
<td>Gastritis.</td>
<td>Endoscopic biopsy</td>
<td>Well-differentiated NET, &lt;1 cm.</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>65</td>
<td>Jejunum-proximal ileum</td>
<td>Acute gastrointestinal obstruction</td>
<td>Surgery</td>
<td>Well-differentiated NET, Mesenteric implants.</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>79</td>
<td>Cecum</td>
<td>Acute gastrointestinal obstruction</td>
<td>Surgery</td>
<td>Well-differentiated NET</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>62</td>
<td>Rectum</td>
<td>Abdominal pain, Endoscopic finding</td>
<td>Endoscopic biopsy</td>
<td>Well-differentiated NET, &lt;1 cm.</td>
</tr>
</tbody>
</table>

(1) M: male. F: female

---

**Fig. 0: TABLE 6A**

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Table 6B: Epidemiologic and clinical features of pancreatic NETs cases

<table>
<thead>
<tr>
<th>Case N°</th>
<th>Sexe (1)</th>
<th>Age (years)</th>
<th>Tumor primary site</th>
<th>Clinical presentation</th>
<th>Diagnostic procedure</th>
<th>Pathologic findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>M</td>
<td>34</td>
<td>Pancreas: insulinoma</td>
<td>Hypoglycemia</td>
<td>Surgery</td>
<td>Well-differentiated NET</td>
</tr>
<tr>
<td>11</td>
<td>M</td>
<td>58</td>
<td>Pancreas</td>
<td>Abdominal pain. Weight loss</td>
<td>Needle biopsy</td>
<td>Poorly-differentiated NET</td>
</tr>
<tr>
<td>12</td>
<td>F</td>
<td>58</td>
<td>Pancreas</td>
<td>Abdominal pain.</td>
<td>Needle biopsy</td>
<td>Well-differentiated NET, Intermediate grade</td>
</tr>
<tr>
<td>13</td>
<td>M</td>
<td>37</td>
<td>Pancreas</td>
<td>Abdominal pain. Weight loss</td>
<td>Needle biopsy</td>
<td>Poorly-differentiated NET, Large cell neuroendocrine carcinoma</td>
</tr>
<tr>
<td>14</td>
<td>F</td>
<td>67</td>
<td>Pancreas</td>
<td>Jaundice (obstructive)</td>
<td>Needle biopsy</td>
<td>NET. Grade not specified</td>
</tr>
<tr>
<td>15</td>
<td>F</td>
<td>58</td>
<td>Pancreas</td>
<td>Abdominal pain. Weight loss. Diabetes</td>
<td>Surgery</td>
<td>Well-differentiated NET</td>
</tr>
</tbody>
</table>

(1) M: male. F: female

Fig. 0: TABLE 6B

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### Table 7A: Radiological findings in GI NETs cases

<table>
<thead>
<tr>
<th>Case N°</th>
<th>Tumor primary site</th>
<th>MDCT</th>
<th>MRI</th>
<th>Distant metastases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Appendix</td>
<td>------</td>
<td>------</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>Appendix</td>
<td>------</td>
<td>------</td>
<td>No</td>
</tr>
<tr>
<td>3</td>
<td>Stomach</td>
<td>2 cm polypoid hyperenhancing mass in gastric body</td>
<td>------</td>
<td>No</td>
</tr>
<tr>
<td>4</td>
<td>Stomach</td>
<td>2 cm polypoid hyperenhancing mass in antrum</td>
<td>------</td>
<td>No</td>
</tr>
<tr>
<td>5</td>
<td>Stomach</td>
<td>Primary tumor &lt; 1 cm, not visible on CT images.</td>
<td>------</td>
<td>No</td>
</tr>
<tr>
<td>6</td>
<td>Jejunum-proximal ileum</td>
<td>Hyperenhancing mural tumor. Mesenteric implants.</td>
<td>------</td>
<td>No</td>
</tr>
<tr>
<td>7</td>
<td>Cecum</td>
<td>------</td>
<td>Primary tumor &lt; 1 cm, not visible on images.</td>
<td>Liver</td>
</tr>
<tr>
<td>8</td>
<td>Rectum</td>
<td>Primary tumor &lt; 1 cm, not visible on images.</td>
<td>Primary tumor &lt; 1 cm, not visible on images.</td>
<td>No</td>
</tr>
</tbody>
</table>

**Fig. 0:** TABLE 7A

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# Table 7B: Radiological findings in pancreatic NETs cases

<table>
<thead>
<tr>
<th>Case N°</th>
<th>Tumor primary site</th>
<th>MDCT</th>
<th>MRI</th>
<th>Metastases</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>Pancreas: insulinoma</td>
<td>Solitary almost isoattenuating mass in proximal tail.</td>
<td>Solitary hypervascular mass in proximal tail.</td>
<td>Liver</td>
</tr>
<tr>
<td>10</td>
<td>Pancreas/duodenum: gastrinomas. MEN1</td>
<td>------</td>
<td>Multiple hypervascular masses in duodenum and proximal tail.</td>
<td>Liver</td>
</tr>
<tr>
<td>11</td>
<td>Pancreas</td>
<td>Huge heterogeneous retroperitoneal mass infiltrating pancreatic tail, spleen, left adrenal and kidney.</td>
<td>Huge heterogeneous retroperitoneal mass infiltrating pancreatic tail, spleen, left adrenal and kidney.</td>
<td>Liver</td>
</tr>
<tr>
<td>12</td>
<td>Pancreas</td>
<td>Hypoattenuating mass in pancreatic tail.</td>
<td>Heterogeneous mass in pancreatic tail.</td>
<td>Liver and bone</td>
</tr>
<tr>
<td>13</td>
<td>Pancreas</td>
<td>Huge retroperitoneal mass infiltrating pancreatic tail and celiac trunk.</td>
<td>------</td>
<td>Pulmonary, hilar and left supravacular nodes.</td>
</tr>
<tr>
<td>14</td>
<td>Pancreas</td>
<td>Hyperenhancing pancreatic head mass with bile duct obstruction</td>
<td>------</td>
<td>Hepatic hilar node. Liver.</td>
</tr>
<tr>
<td>15</td>
<td>Pancreas</td>
<td>Diffuse pancreas thickening</td>
<td>Diffuse pancreas thickening.</td>
<td>Liver</td>
</tr>
</tbody>
</table>

**Fig. 0: TABLE 7B**

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**FIGURE 1:** A 80 year old woman (case N° 3) with atrophic gastritis and gastric carcinoid type I. Contrast-enhanced MDCT. (A) arterial phase. (B) Venous phase. Enhancing polipoid mass (arrow) in the posterior wall of the gastric body. Patient was treated with partial gastrectomy because of adenocarcinoma suspicion.

**Fig. 0:** FIGURE 1

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**FIGURE 2:** A 53 year old woman with a gastric carcinoid type III in antrum (case N° 4). Axial contrast-enhanced MDTC images. (A) arterial phase and (B) venous phase. Enhancing tumor (arrow) in anterior wall of the gastric antrum. (C) Tumor presents as a gastric submucosal lesion on endoscopy.

**Fig. 0:** FIGURE 2

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FIGURE 3A: A 54 year old woman with MEN 1 syndrome and small gastrinomas in the pancreas and duodenum. Pancreatic and duodenal tumors were confirmed by surgical extirpation (Case Nº 10). Axial fat-suppressed T1-weighted MR images without (A) and with (B) gadolinium showing a small subserosal gastrinoma in the wall of the third portion of the duodenum (arrows). (d: Duodenum.  ivc: inferior vena cava)

Fig. 0: FIGURE 3A

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**FIGURE 3B:** Same patient with MEN 1 syndrome than in figure 3A. (A) Axial and sagittal oblique reformatted contrast-enhanced MDCT images of the neck. Hyperenhancing mass at the right carotid bifurcation corresponding to a paraganglioma. (B) Coronal FSE T2-weighted MR image detected a carcinoid tumor in the left lung.

**Fig. 0: FIGURE 3B**

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**FIGURE 4:** A 65 year old woman (Case N° 6) with a carcinoid tumor in the ileum and mechanical small bowel obstruction. Contrast-enhanced MDCT images. Axial (A) and coronal oblique reformatted (B and C) images demonstrate dilated fluid-filled small bowel loops, a smooth enhancing mass in the ileum (black arrows) and infiltration of mesenteric fat (white arrows).

**Fig. 0:** FIGURE 4

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FIGURE 5A: A 34 year old man with an insulinoma in the tail of the pancreas (case № 9). (1) Axial contrast-enhanced MDCT images in arterial (1A) and venous phase (1B). The tumor (arrow) shows slight enhancement as compared to the pancreas. (2) Axial MR images: On T1-WI (2A) the tumor (arrow) appears as a low SI bulge in the contour of the pancreas. On FSE T2-WI (2B) tumor is isointense to the pancreatic parenchyma.

Fig. 0: FIGURE 5A

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FIGURE 5B: A 34 year old man with an insulinoma in the tail of the pancreas (case N° 9).
Dynamic contrast-enhanced fat-suppressed gradient-echo T1-WI images, obtained before (A) and 20 (B), 60 (C), 120 (D) and 240 seconds (E) after intravenous bolus injection of gadolinium. The tumor shows low SI on unenhanced images, slight enhancement in portal phase and isointensity to the pancreas in arterial and delayed phases.

Fig. 0: FIGURE 5B

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**FIGURE 6A:** Same patient than in FIGURE 3. A 54 year old woman with MEN 1 syndrome and small gastrinomas in the pancreatic tail and duodenum (Case N° 10). (A) : On T1-weighted MR images this small gastrinoma (arrow) shows low SI as compared with the pancreatic parenchyma. The lesion is isointense to the pancreas on T2-WI (B) and slightly hyperintense on fat-suppressed T2-WI (C) (D) Fat-suppressed T2-weighted image showing two other small PNETs in the tail of the pancreas.

M: liver metastases

**Fig. 0: FIGURE 6A**

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FIGURE 6B: Gastrinoma and liver metastases (case N°10). Dynamic contrast-enhanced gradient-echo fat-suppressed T1-WI images. Scan delay on figures. Gastrinoma (arrow) shows avid enhancement in arterial phase. Metastases (☆) presents in anterior portion important enhancement in arterial and portal phases. Enhancement is predominant in delayed phases in the posterior portion, suggesting fibrosis (★).

**Fig. 0: FIGURE 6B**

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**Figure 7A.** 58 years old man with a large poorly-differentiated PNET (Case n° 11). (A) Axial fat-suppressed T1-weighted precontrast MR image demonstrates a large retroperitoneal tumor compressing the body and tail of the pancreas (p). The lesion presents low SI relative to the pancreas. (B) Arterial postcontrast image shows how the mass encases the aorta and the celiac trunk. (C) On delayed postcontrast image the lesion presents heterogeneous enhancement.

**Fig. 0: FIGURE 7A**

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Figure 7B. 58 years old man with a large poorly-differentiated PNET (Case nº 11). (A) and (B) Axial and coronal FSE T2-weighted MR images. (C) Coronal fat-suppressed T1-weighted postcontrast MR image. The tumor shows on T2-WI very hyperintense areas without enhancement on postcontrast T1-WI corresponding to cystic degeneration or necrosis. (c: hepatic cyst)

**Fig. 0: FIGURE 7B**

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Figure 7C. 58 years old man with a large poorly-differentiated PNET (Case nº 11). (A) Diffusion weighted (b = 600) axial MR image shows high signal intensity of the tumor and the small hepatic cyst (c). (B) Graphic representation of apparent diffusion coefficient (ADC) values. Solid tumor presents restricted diffusion as compared to pancreatic parenchyma. (1) Pancreas ADC value: $1.8 \times 10^{-3}$ mm$^2$/s. (2) Tumor ADC value: $0.99 \times 10^{-3}$ mm$^2$/s.

Fig. 0: FIGURE 7C

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FIGURE 8: A 37 years old man (Case N° 13) with a poorly-differentiated PNET in the body and tail of the pancreas and pulmonary metastases. (A and B) Axial contrast-enhanced MDCT images on venous phase demonstrate the iso-hypodense primary tumor (star). The mass encases the splenic vessels and infiltrates the left crura. (C) Pulmonary hilar nodes (arrows) and multiple lung metastases.

Fig. 0: FIGURE 8

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FIGURE 9A: A 58 years old woman (Case Nº 12) with a well-differentiated PNET in the pancreatic tail and liver metastases. Axial contrast-enhanced MDCT images. (A) arterial phase. (B) venous phase. Tumor (pc) presents as a well delimitated homogeneous mass with slight enhancement on venous phase. Liver metastases (Lm) shows homogeneous hypodensity to the liver in both vascular phases.

Fig. 0: FIGURE 9A

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**FIGURE 9B**: Case Nº 12. Axial MR images. (A) T1-WI. (B) fat-suppressed T1-WI. (C) fat-suppressed T2-WI. (D) T2-WI. On T2-WI pancreatic tumor (pc) presents low SI areas suggesting fibrosis. Multiple liver metastases are demonstrated as low SI lesions on T1-WI and high SI lesions on T2-WI. The large metastases of IV liver segment is heterogenous, showing higher central hyperintensity on T2-WI.

**Fig. 0: FIGURE 9B**

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**Figure 9C.** Case N° 12. Dynamic contrast-enhanced gradient-echo fat-suppressed T1-WI images. Scan delay on figures. The primary tumor and metastases follow different patterns of enhancement. Primary tumor presents higher enhancement on delayed images whereas small metastases show early enhancement and become isointense to the liver on following vascular phases. The larger liver lesion presents peripheral early enhancement and delayed central enhancement.
Figure 9D. Case Nº 12. (A) Diffusion weighted (b = 600) axial MR image shows high signal intensity of PNET (pc), liver (arrows) and bone metastases (arrow head).
(B and C) Graphic representation of apparent diffusion coefficient (ADC) values. Pancreas ADC value: 2,44 x10^-3 mm^2/s. (2) Tumor: 1,28 x10^-3 mm^2/s. Metastases: 1,28 and 2,09 x10^-3 mm^2/s.

Fig. 0: FIGURE 9D

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FIGURE 10: A 67 years old woman (Case N° 14) with common bile duct obstruction due to a pancreatic head PNET. (A and B) Axial arterial contrast-enhanced MDCT images show the primary tumor (pc) and multiple liver metastases with peripheral early enhancement. (C) On venous phase liver metastases (arrows) present rapid contrast wash-out. Sc: pancreatic pseudocyst.

Fig. 0: FIGURE 10

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FIGURE 11A: A 58 years old woman (Case N° 15) with a well-differentiated PNET and liver metastasis. (A) and (B) Axial arterial and delayed contrast-enhanced MDCT images. PNET (short arrows) presents as a diffuse homogeneous thickening of head, body and tail of the pancreas. Both primary tumor and liver metastasis (long arrows) are isodense to the liver parenchyma. (C) and (D): axial T1-weighted and T2-weighted MR images. Both lesions present low SI on T1-WI and high SI on T2-WI.

Fig. 0: FIGURE 11A

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FIGURE 11B: A 58 years old woman (Case Nº 15) with a well-differentiated PNET and liver metastasis. Dynamic contrast-enhanced gradient-echo fat-suppressed T1-WI images. Scan delay on figures. PNET (pc) and liver metastases (arrows on A) Both primary tumor and liver metastasis show early enhancement and progressive wash-up.

Fig. 0: FIGURE 11B

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**Fig. 0: FIGURE 11C**

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**FIGURE 12A:** Liver metastases in a 79 years old man (Case N° 7) with a cecal carcinoid. (A and B) Axial arterial and venous contrast-enhanced MDCT images. (C) detail of B. Metastases shows slight hypodensity to the liver parenchyma. (C and D) axial T1-weighted and T2-weighted MR images. Lesion demonstrates low SI on T1-WI and very high SI on T2-WI. (F) Diffusion weighed (b=600) axial MR image presenting hyperintense metastases. ADC value: 1,54 x10^{-3} mm²/s.

**Fig. 0:** FIGURE 12A

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FIGURE 12B: Liver metastases in a 79 years old man (Case Nº 7) with a cecal carcinoid. Dynamic contrast-enhanced gradient-echo T1-WI images. Scan delay on figures. Lesion shows progressive enhancement on venous and delayed phases.

**Fig. 0:** FIGURE 12B

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Conclusion

GEP neuroendocrine neoplasms are uncommon tumors with variable appearances on CT and MR images. They should be considered in patients with typical clinical syndromes or characteristics imaging features such as avidly enhancing primary tumors in GI tract and pancreas or mesenteric stellated masses. GE NETs liver metastases commonly present high SI on T2-WI and hyperenhancement on early or delayed contrast-enhanced images.
References


