Neuroendocrine tumours of the gastro-entero-pancreatic system

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Learning objectives

- Review the spectrum of appearances of the gastro-entero-pancreatic neuroendocrine tumours.

- Review the WHO 2010 classification system for gastro-entero-pancreatic neuroendocrine tumours.

- Understand the clinical, pathological and laboratorial findings that together interfere in the prognosis.
First described in 1907 by Siegfried Oberndorfer, neuroendocrine neoplasms (NENs) are a group of heterogeneous tumours that can affect almost any location in the human body, although the majority of them occur in the gastro-entero-pancreatic (67%) and the bronchopulmonary (31%) systems. Although rare, some studies describe an increased incidence in the last years. These tumours can present sporadically or associated to familial syndromes, such as Multiple Endocrine Neoplasia (MEN) syndrome, von Hippel-Lindau syndrome, Neurofibromatosis or Tuberous Sclerosis.

Their definition comes from the fact that these neoplasms acquire neuroendocrine differentiation, expressing synaptophysin and chromogranin A, and producing hormones freed in the bloodstream.

The heterogeneity of NENs results from multiple variables, including, mainly, the location, the degree of differentiation and proliferation, but also the type of cell differentiation of the tumour that can present as a functional or non-functional NEN.

Carcinoid syndrome, mainly associated with NENs, happens only in 10% of cases and consists of colicky abdominal pain, diarrhea, bronchospasm, cutaneous flushing, sweating, and right-sided cardiac valve fibrosis. This syndrome is the result of serotonin released by NENs usually located in the small intestine, mainly when they present with local lymph nodes and hepatic metastases.

Several classification systems have been proposed, the last published by the World Health Organization (WHO) in 2010, in which a new nomenclature as well as a new classification for NENs is established. According to this, NENs should be classified based on two main principles: the grading system and the site-specific staging system, both of which have shown to be important prognostic factors.

WHO has made possible a linear and regular system of classification of NENs of the gastro-entero-pancreatic system, as well as reinforced the clinical and prognostic heterogeneity between these various NENs.

Based on this new classification, NENs are divided into two main categories: well-differentiated neuroendocrine tumours (G1 or G2), and poor-differentiated neuroendocrine carcinomas (G3 small-cell or large-cell), supported by the grading system, which is solely based on the proliferation rate of these tumours. (Table 1 on page 8).

In cases where the mitotic count differs from the Ki67 index, the higher one is used to classify the NENs.
The only definitive treatment is surgical excision, possible in less than 20% of the cases. Nevertheless, in many cases, patients survive years with chemotherapy, radionuclide treatments and other techniques of interventional radiology, due to tumours' indolent behaviour.

**Gastrointestinal Neuroendocrine Neoplasias**

Gastrointestinal NENs represent the bigger percentage of NENs and are the commonest neoplastic lesion of the small bowel. They originate from the multitude of endocrine cells that populate the mucosa and submucosa of the gastrointestinal tract and, since they have variable biological and hormonal functions, so these neoplasias will have different clinical syndromes and outcomes, mostly related to the site of origin. They are defined as functional or non-functional NENs, if a clinical syndrome related to hormone release is present or absent.

Oesophageal NETs are exceedingly rare and most of them are poor-differentiated G3 NECs. They are related with heavy smoke and Barret's oesophagus, many times associated with adenocarcinoma, and tend to have an aggressive behaviour, with mural infiltration and metastasis.

Gastric NENs are rare, representing only 1.8% of all the gastric malignancies and 2-3% of all gastrointestinal NENs. Most of them are non-functional, well-differentiated enterochromaffin-like (ECL) cell NETs, presenting in the gastric fundus and body, and are divided in three different groups.

Type I ECL-cell NET is the most common (74% of gastric NENs) and is related with chronic atrophic gastritis and hypergastrinemia as a result of achlorhydria. Most of the lesions are found during endoscopy.

Type II ECL-cell NET (6% of gastric NENs) is associated with Zollinger-Ellison syndrome in patients with MEN1. Their origin comes from two synergic factors: genetic susceptibility caused by MEN1 syndrome and ECL cell hyperplasia caused by hypergastrinemia, which is produced by a gastrinoma of the pancreas or the duodenum and result in clinical symptoms related with this hormone.

Type III ECL-cell NETs represent sporadic tumours (13% of gastric NENs). They tend to be more aggressive and the clinical symptoms relate with this behaviour, with weight loss, bleeding and anorexia.

The other types of gastric NENs are very rare and may present as gastrinoma, serotonin-producing NET (associated with carcinoid syndrome), ACTH-producing NET or undifferentiated G3 NECs.
Small intestine NENs present differently depending on the segment of origin.

Duodenal NENs are uncommon, representing 6-8% of the gastrointestinal NENs. Most of them are G-cell NETs (62% of duodenal NENs) or D-cell NETs (21% of duodenal NENs).

G-cell NETs can be non-functional or functional, producing gastrin and manifesting as a Zollinger-Ellison syndrome, sometimes related to MEN1 syndrome. Sporadic gastrinomas occur in the gastrinoma triangle in 85% of cases.

D-cell NETs have a strong relation with Neurofibromatosis I and are present in 50% of the cases. They are typically localized in and around the ampulla of Vater and, although they usually produce somatostatin, the clinical manifestations are usually related to pancreatobiliary obstruction and not hormone-released symptoms.

Other types of NENs are very rare in the duodenum.

Jejunal and ileal NENs are the most common of the gastro-intestinal system and all of them are graded G1 or G2 well-differentiated NETs. Specially located in the distal ileum, they correspond to 44,7% of all gastro-intestinal NETs. They usually present as enterochromaffin-cell (EC-cell) NETs, producing serotonin and, when metastasized to local lymph nodes and the liver, give rise to the carcinoid syndrome.

They can also be differentiated in other cells, although rarely, mostly L-cell glucagon producing NETs or pancreatic polypeptide producing NETs.

Appendiceal NENs represent 19% of gastrointestinal NENs, usually have a benign course and are mostly encountered in appendix specimens from appendicectomy. As ileal NENs, they are usually EC-cell and L-cell NETs.

Colorectal NENs are common and represent 35% of all gastrointestinal NENs, most of them located in the rectum. EC-cell NEN is more frequent in the cecum and progressively becomes rarer along the large intestine, being substituted for L-cell NEN, which is commoner in the rectum.

Proximal colon NENs are quite similar with jejunoileal NENs and present with carcinoid syndrome when metastasised. On the contrary, rectum NENs aren't usually associated with hormone-related symptoms and 50% discovered with the medical examination. When symptomatic, they present with local symptoms such as pain, constipation and bleeding.

**Pancreatic Neuroendocrine Neoplasias**
Pancreatic neuroendocrine tumours (PNETs), a subgroup of the NENs, were formerly known as islet cell tumours. Corresponding only to 1-2% of all the pancreatic neoplasms, their incidence is approximately 1 per 100,000 individuals per year and can present intra or peri-pancreatically.

Although classified the same way as the other NENs as neuroendocrine tumours or carcinomas, 65-80% of them present with malignant behaviour, even though only 1-2% are classified as G3 neuroendocrine carcinomas.

A few PNETs are associated with familial syndromes, the most important being MEN1 in which 80%-100% present with a PNET, most of the times a gastrinoma.

PNETs can produce different clinical syndromes, as they can differentiate in a multitude of endocrine cells delivering a variety of hormones. According to this property, as all the other NENs, they are divided as functional or non-functional PNETs. Most of the times the clinical syndrome is associated with one single hormone but, rarely, PNETs hypersecrete more than one type and present synchronously or sequentially with different clinical syndromes. The most common of the functional PNETs are Insulinomas, followed by Gastrinomas, Glucagonomas, VIPomas, Somatostatinomas and other rare PNETs that may secrete hormones such as adrenocorticotropin hormone. Nonetheless, studies refer that 50-70% of PNETs are classified as non-functional, most of the times secreting peptides that don’t cause clinical manifestations but can otherwise be detected in the blood stream, such as chromogranin A, pancreatic polypeptide and neuron-specific enolase.

Radiology has a preponderant role in NENs, mainly because of its ability to detect, stage and treat these lesions.

Computed tomography (CT) is the imaging technique of choice to detect and stage most of the NENs. The staging CT protocol includes a non-contrast phase, an arterial/pancreatic parenquimal phase and a portal venous phase, and the use of low-density/negative oral contrast. For hepatic metastases, Magnetic Resonance (MR) has shown better sensibility. Ultrasound is of less use in NENs, having a variable sensitivity for detecting PNETs and being useful for guiding hepatic metastases biopsy.

Since these tumours have neuroendocrine differentiation, most of them will present somatostatin receptors on the surface of the cells. This way, nuclear medicine studies have a major role in detecting NENs, as well as their metastasis, using somatostatin analogs marked with radioisotopes. $^{111}$In-octreotide scintigraphy has an overall sensitivity of 80% in NENs with these two characteristics: well differentiated G1 or G2 and expression of somatostatin receptors type 2 and 5, for which Octreotide binds. This way, $^{111}$In-octreotide scintigraphy can also determines the efficacy of somatostatin-based
therapeutics for control of hormone-related symptoms. Positron emission tomography (PET) and, more recently PET-CT, with 2-[fluorine-18]fluoro-2-deoxy-D-glucose (FDG) can detect G3 NECs because of the high metabolic rate and, consequently, high FDG uptake of these neuroendocrine carcinomas.

As referred above, radiology has also a role in treatment of these neoplasias. Most of its work is centered in hepatic metastasis and nowadays there are multiple treatment options. Surgical removal of the primary lesions and metastases is still the first and only definitive treatment. However, few cases gather the appropriate conditions to apply surgical intervention. So, interventional radiology can control and, sometimes, help curing the disease. Transcatheter Arterial Embolization (TAE) and Transcatheter Arterial Chemoembolization (TACE) have shown promising results, and other techniques as Radiofrequency Ablation and Cryotherapy are increasingly being used, mostly for palliative purposes in the control of hormonal symptoms, but also for tumour bulk reduction and conversion to a resectable status.

Besides diagnostic purposes, $^{111}$In-octreotide scintigraphy can also access receptor status for radionuclide-based treatments, which can also delay the tumour growth and even ablate the lesions.
Table 1: Table 1: Relationship between the grading system, the proliferation rate and the nomenclature, according to WHO. HPF: High Power Field.

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Findings and procedure details

From a NETs pathologically confirmed database at our institution dated from January 2006 until June 2014, 145 cases were retrieved. From this pool, 30 cases of enterogastric NET’s and 14 cases of pancreatic NETs were gathered (Table 2 on page 12).

Multi-modality imaging findings were reviewed and the most illustrative cases selected.

Oesophageal NECs are indistinguishable with other oesophageal carcinomas. Most of the times, detection and biopsy are made by direct visualization with endoscopic procedures, and CT is used to stage these lesions. They usually present as heterogeneous masses or asymmetric circumferential thickness of oesophageal wall on distal segments, with variable contrast enhancement in arterial phase and signs of local invasion such as transmural penetration and infiltration of adjacent structures and local lymph nodes (Fig. 1 on page 12)

Gastric NENs present differently depending of the type of ECL-cell NET. As oesophageal lesions, most of them are found and biopsied by endoscopic procedures. Type I and type II present as multifocal smoothly marginated small lesions with less than 2 cm located mainly in the body and fundus. Type I rarely metastasise but type II can present with local lymph node and hepatic metastasis in 10-30%. When primary lesions have more than 1 cm, they are identified in CT studies, presenting as hypervascular mucosal or submucosal masses, better delineated in arterial phase images, sometimes accompanied with thickened gastric folds because of associated hypergastrinemia. However many times CT has little value in detecting the gastric lesions because of its low sensitivity when they are small (less than 1 cm), being mostly used to detect local and distant metastasis, which are also hypervascular. Type III ECL-cell NENs mostly present as a unique large mass (>2 cm) frequently with wall invasion, local lymph node and haematogenous spread (73% of the cases). Double-contrast barium studies are also useful in detecting gastric NENs (Fig. 2 on page 13, Fig. 3 on page 14).

Duodenal NETs can present as a polypoid intraluminal lesion or as an intramural mass. G-cell sporadic NETs tend to present as a single round and well-defined lesion, while G-cell MEN1 associated NETs show multiple lesions located in the proximal duodenum. D-cell NETs mostly present as single peri-ampullary masses. They all are hypervascular and show enhancement in arterial phases, with little washout in portal venous phases.

Jejuno-ileal NETs can present variably, mostly related with tumor size and the ability to invade and metastasise. These lesions can present as a polypoid intraluminal mass or as a segmental concentrically or asymmetrically mural thickness. Small, localized and
non-metastatic lesions most of the times can't be identified by CT and their detection depend mainly in \textsuperscript{111}In-octreotide scintigraphy. However, larger polypoid lesions can be identified as hypervascular masses. Mural thickening presentation results from infiltration and desmoplastic fibrotic reaction, which give rise to a rigid, stiff and distorted intestinal segment. When transmural invasion is present, CT-studies show an adjacent hypervascular soft tissue mass with radiating linear fibrotic bands that retracts the mesentery and the intestinal loops and can cause intestinal obstruction. This direct mesenteric invasion can also encircle and invade vascular structures and cause ischemic symptoms. More than 50\% of these NETs also presents with local mesenteric lymph nodes, liver metastasis or even peritoneal implants. Mesenteric lymph node metastasis can show multiple calcification patterns, are hypervascular and can be well defined or present with spiculated borders as a result of desmoplastic reaction. Liver metastases are also hypervascular (Fig. 4 on page 15, Fig. 5 on page 16, Fig. 6 on page 17).

MR can also be used to detect jeuno-ileal NETs. They present as hypointense masses in T1-weighted images and hyperintense in T2-weighted images, best defined on gadolinium-enhanced T1-enhanced images. Liver metastases show similar characteristics to the primary lesion, both in terms of enhancement and signal in T1 and T2-weighted images. When contrast administration is contraindicated, T2-weighted images have the best sensibility. With the use of hepato-specific contrast agents, liver metastases appear unenhanced due to the lack of hepatocytes (Fig. 7 on page 18).

Appendiceal NETs generally are not identified on imagiologic studies. Since these are mostly found in appendicectomy specimens, they are usually associated with inflammatory signs of appendicitis due to the obstruction caused by the tumour itself and are not well differentiated from the inflammatory reaction. Sometimes they are identified without appendicitis symptomatology, presenting the same way as jeuno-ileal NETs, as hypervascular polypoid lesions or mural thickening, less frequently with lymph node and hepatic metastases.

Colorectal NETs are radiologically indistinguishable from polypoid adenomas or adenocarcinomas. Colonic NETs usually present as intraluminal polypoid lesions, sometimes with unenhanced low-attenuation areas of necrosis. Rectal NETs are seen as small polypoid hypervasular lesions. Local infiltration and invasion of mesorectal space is seen less frequently (Fig. 8 on page 19, Fig. 9 on page 20).

Although some subtypes of PNETs demonstrate a local predilection (Gastrinomas and Somatostatinomas for the pancreatic head, Glucagonomas for the body and tail, and VIPomas for the pancreatic tail), they can present in any part of the pancreas. The size is variable and non-functioning PNETs generally arise bigger than the functioning ones,
mainly because of the diagnostic delay related to the lack of hormone-related clinical syndromes, presenting with mass effect symptoms.

Ultrasound evaluation shows a round or oval, homogeneous or heterogeneous, well-circumscribed hypoechoic mass, sometimes with calcifications and cystic changes. Liver metastases usually are hyperechoic but can manifest as hypoechoic masses or even with a target-like appearance.

Computed tomography (CT) is the imaging technique of choice to detect and stage PNETs. These tumours typically present as hyperattenuating masses in pancreatic parenchymal and portal venous phases, better delineated in the parenchymal one. There is a relation between size and heterogeneity of the tumour: the bigger the NEN the more heterogeneous with areas of necrosis or cystic change, calcifications and fibrosis. Arterial encasement of the celiac axis and the superior mesenteric artery has to be evaluated, as well as the venous invasion. Hepatic and lymph node metastasis are also hypervascular and better depicted in the arterial/pancreatic parenchymal phase (Fig. 10 on page 21, Fig. 11 on page 22, Fig. 12 on page 23, Fig. 14 on page 26).

Magnetic Resonance (MR) imaging has similar sensitivity as CT in evaluating PNETs. PNETs present as hypointense masses on T1 and hyperintense masses on T2-weighted images. After contrast administration, PNETs show vigorous enhancement on arterial phase, which may be homogeneous, heterogeneous or ring-like. Liver metastases show similar characteristics to the primary lesion (Fig. 12 on page 23, Fig. 13 on page 24, Fig. 14 on page 26, Fig. 15 on page 27).
Table 2: Table 2: Statistical resume of cases reviewed. a: lymphomatous or haematogenous metastatic disease; b: imagiologically or pathologically confirmed.

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Fig. 1: Figure 1: Oesophageal NEC G3. Computed tomographic (CT) scan in the axial (a), coronal (b) and sagittal (c) planes in the arterial phase showing an intraluminal hypoenhancing mass in the lower oesophagus (arrows) with peripheral enhancement. Positron emission tomography CT (PET-CT) of the same lesion (d) showing intense FDG captation, revealing a metabolically active lesion and suggestive of a G3 NEN.

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**Fig. 2:** Figure 2: Gastric NEC G3. Computed tomographic (CT) scan in the axial (a, b and d) and sagittal (c) planes in non-contrast phase (a) and late arterial phase (b, c and d) showing an irregular and heterogeneous thickening along the lesser curvature (asterisk) that reveals discrete peripheral enhancement in the late arterial phase and areas of cystic/necrotic change. Note the lymphadenopathy (arrow) located in the hepatic hilum.
Fig. 3: Gastric NEC G3. Computed tomographic (CT) scan in the axial (a, b and c) and coronal planes (d) in late arterial phase (from a to c sectional craniocaudal images) showing a regular focal thickening of the great curvature (black arrow) with implants in the gastrosplenic ligament (white arrow) and hypodense hepatic metastases.

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Fig. 4: Ileal NET G1. Computed tomographic (CT) scan in the axial (a) and coronal (b) planes in portal venous phase revealing a heterogeneous mesenteric mass with radiating fibrotic bands (asterisk) and an adjacent lymph node (arrow). No mural lesion was detected. 111In-octreotide scintigraphy (c) revealing the same lesion.

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Fig. 5: Figure 5: Ileal NET G1. Computed tomographic (CT) scan in the axial (a) and coronal (b) planes in the arterial phase showing a lobulated homogeneous hyperenhancing mesenteric mass (asterisk) just below the inferior renal poles. Note the hepatic metastases in (b).

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**Fig. 6:** Figure 6: NET G1 located in the ileocecal valve. Computed tomographic (CT) scan in the axial (a and b), coronal (c) and sagittal (d) planes in non-contrast (a) and late arterial phases (b, c and d) showing an irregular mural thickening in the ileocecal valve (asterisk) associated with an adjacent mesenteric mass (arrow), both hypervascular.

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**Fig. 7:** Ileal NET G1. Computed tomography (CT) scan in the axial plane in late arterial phase and Magnetic Resonance (MR) scan in the axial (b) and coronal (c) planes, T1-weighted with fat suppression and administration of gadolinium in the arterial phase. This patient had an ileal segmentectomy but persisted with a mesenteric mass (arrow). Image (a) reveals a mesenteric mass with discrete fibrotic bands and peri-lesional lymph nodes. Images (b) and (c) reveal a hypointense mesenteric lesion with peripheral and heterogeneous contrast enhancement.

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Fig. 8: Figure 8: Rectal NEC G3. Computed tomographic (CT) scan in the axial (a) and coronal (b) planes in arterial phase revealing a hypervascular lobulated mass in the rectum (asterisk). Note the lymph node metastasis in the mesorectum (white arrow in b). Endoscopic ultrasound revealing the NEC (c). Magnetic resonance (MR) scan in the axial (d) and coronal (e) planes, in T2-weighted image, after radiotherapy, showing a band of residual fibrosis as a hyposignal border and complete resolution of the NEC in the lateral wall of the rectum (black arrow).

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**Fig. 9:** Figure 9: Rectal NEC G3. Magnetic Resonance (MR) scan in the axial (a) and coronal (b) planes, in T2-weighted image showing a parietal lesion in the lateral wall of the rectum delineated by intraluminal gel, without clear mesorectum infiltration (asterisk).

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**Fig. 10:** Figure 10: Pancreatic NET G1. Computed tomographic (CT) scan in the axial plane (a and b), in non-contrast (a) and pancreatic parenchymal (b) phases, showing a rounded mass in the pancreatic body with intense peripheral enhancement in this phase (arrow). This patient had no metastases.

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Fig. 11: Figure 11: Pancreatic NET G1. Computed tomographic (CT) scan in the axial plane (a, b and c) in non-contrast (a), pancreatic parenchymal (b) and portal venous (c) phases revealing a lobulated lesion in the pancreatic tail with a small focus of calcification, hyperenhancing in the parenchymal phase and discrete washout in the portal venous phase (arrow). 111In-octreotide scintigraphy (d) revealing the same lesion in the pancreatic tail.

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**Fig. 12:** Figure 12: Pancreatic NET G1. Computed tomographic (CT) scan in the axial (a) and coronal (b) planes in the parenchymal pancreatic phase revealing an irregular and heterogeneous mass in the pancreatic tail (white asterisk), with invasion of the homolateral kidney and in contact with abdominal aorta, hepatomegaly due to metastases, retroperitoneal lymph nodes and pelvic implants (arrows). Magnetic resonance (MR) scan in the axial plane (c) in T1-weighted fat suppression with hepatospecific contrast administration, in the beginning of the disease, showing a pancreatic tail mass with hyposignal (asterisk) and some small hepatic lesions, also with hyposignal (arrow).

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Fig. 13: Figure 13: Pancreatic NET G2. Computed tomographic (CT) scan in the axial (a and b) and coronal (c) planes in non-contrast (a) and pancreatic parenchymal (b and c) phases revealing a heterogeneous lesion in the pancreatic tail (arrow), with calcification focuses and discrete cystic/necrotic changes, hyperenhancing in the parenchymal phase. Note the hepatic metastases, some hypervascular (asterisk) in the parenchymal phase and others with cystic degeneration. Magnetic resonance (MR) scan in the axial plane (d, e and f) in T2-weighted with fat suppression (d), T1-weighted fat suppression with no gadolinium (e) and T1-weighted fat suppression with gadolinium in the arterial phase showing a heterogeneous lesion in the pancreatic tail with calcifications (arrow), with hypersignal in T2-weighted (d), hyposignal in T1 weighted with no contrast (e) and enhancing peripherally after gadolinium administration (f).
Fig. 14: Pancreatic NEN G unknown. Computed tomographic (CT) scan in the axial (a and b) and coronal (c) planes, in non-contrast (a) and pancreatic parenchymal (b and c) phases revealing an heterogeneous lobulated mass in the pancreatic tail (arrow) in contact with the homolateral kidney, with gross calcifications and cystic changes, and revealing peripheral arterial enhancement. There are also multiple hypervascular hepatic metastases with cystic degeneration (asterisks). Magnetic resonance (MR) scan in the axial plane (d), in T2-weighted with fat suppression (e) revealing a heterogeneous pancreatic lesion with gross calcification (bands of hyposignal), cystic degeneration and areas of hypersignal, as well as hepatic metastases with cystic degeneration.
**Fig. 15:** Figure 15: Pancreatic NET G1. Magnetic resonance (MR) scan in the axial plane (a and b), in T1-weighted image (a) and T1-weighted with fat suppression and gadolinium administration in the arterial phase, showing a large lobulated lesion in the pancreatic tail with hyposignal in (a) and contrast enhancement in (b).
**Fig. 16:** Figure 16: NEN with G unknown of undetermined origin (pancreatic / gastric). Magnetic resonance (MR) scan in the axial (a, b and d) and coronal plane (c), in T1-weighted image (a), T2-weighted with fat suppression image (b) and T2-weighted image (c and d) revealing a mass in the gastrohepatic ligament (asterisk with a celiac lymph node arrow), both showing hyposignal in T1 and hypersignal in T2. Note the hepatic metastasis in (d) with hypersignal in T2 located in the segment V.

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Conclusion

Having such a variable clinical presentation and originating in any part of digestive system, gastro-entero-pancreatic NENs require a multidisciplinary approach in which the radiology has a preponderant role in detecting, guiding the biopsy, treating and following-up the course of these tumours. CT is the imaging technique of choice to detect and stage NENs, although Nuclear Medicine studies also have a major role.
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