The role of MRI in Imaging Pancreatic Neuroendocrine Tumours

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Learning objectives
Describe clinicoradiological features of primary pancreatic neuroendocrine tumours (PNETs).
Highlight the role of MRI in distinguishing PNETs from adenocarcinoma.
Illustrate how MRI might aid in the diagnosis and prognostic stratification of PNETs.

Background
Pancreatic neuroendocrine tumours, first described in 1902 by Nicholls, comprise 1-2% of all pancreatic neoplasms. Together with gastrointestinal carcinoid they form the bulk of gastrointestinal neuroendocrine tumours. PNETs demonstrate no significant gender or age predilection and comprise diverse clinical features. The majority of PNETs are sporadic however 1-2% are associated with familial syndromes such as multiple endocrine neoplasia type 1, neurofibromatosis type 1, tuberous sclerosis and von Hippel Lindau syndrome.

All PNETs demonstrate some functionality in terms of hormone production however not all are manifestly clinically leading to their classification into syndromic and non-syndromic. The main syndromic PNETs include insulinomas, glucagonomas, VIPomas, gastrinomas, ACTHomas and somatostatinomas. PNETs are classified into well-differentiated and poorly differentiated. All have variable malignant potential. WHO classification utilizes tumour size, cell proliferation, mitotic rate and evidence of invasion. CT has been considered the first line modality, however with recent improvements in technology, MRI is showing promising results in terms of tumour detection, grading and monitoring purposes.

Detection
The detection of syndromic PNETs is usually challenging due to the fact that such tumours are often small in size (<1 cm) despite their overt clinical manifestations. In contrast, non-syndromic PNETs are usually larger in size (approx. 5-6 cm), having presented late from symptoms of mass effect or metastatic disease, however these are usually detected incidentally. On MRI PNETs are hypointense relative to native pancreatic tissue on T1 and show mild to moderate T2 hyperintensity. They usually demonstrate avid enhancement which is either homogenous, heterogeneous or ring-like.

With regards to PNET localisation MRI has showed a sensitivity of 85-100% and specificity of 75-100%, compared to CT which has a sensitivity of 63-82% and specificity of 83-100%. The detection rate of MRI is between 50-94% whilst that of CT ranges from 39-94%. 3,4,5

Differentiation from adenocarcinoma
Pancreatic adenocarcinomas are usually hypovascular and do not take up contrast. Additionally certain DWI parameters (such as perfusion fraction and incoherent microcirculation) are notably higher in PNETs as compared to pancreatic adenocarcinomas. 6

Grading
MRI has a potential to help in pre-operative grading given that G1 tumours demonstrate significantly higher mean ADC values and ADC ratios when compared to G2 and G3 tumours according to a recent study. G1 tumours also have increased uptake of intravenous gadoteric acid in view of being more vascular than G2 and G3 tumours. 8

Monitoring
Following any form of treatment such as surgery, ablative procedures or systemic therapy, follow-up imaging is recommended as per RECIST criteria. In young patients where multiple scans will be necessary MRI is preferred as it does not confer any ionising radiation. 9

Fig 1.1 35-year-old male with recurrent hypoglycaemic episodes. Diffusion weighted imaging formed as part of an MRI Pancreas (b=0 (A), 50 (B), 400 (C), 800 (D)) shows a 15 mm focus of restricted diffusion (arrow) in the pancreatic head. The lesion demonstrates intrinsic T2 hypointensity (A & B). This later was proved as an insulinoma.

Fig 1.2 This focus of restricted diffusion corresponds to a nodule showing T1 hypointensity (arrow A) and T2 hyperintensity (arrow B).

Fig 2.1: 65-year-old male. MRI demonstrates an incidental faintly hypointense T1 lesion pre-contrast (arrow A), with moderate arterial enhancement (B) which persists in portovenous (C) and delayed (D) phases. This lesion was shown to be a non-syndromic PNET.

Fig 2.2 Contrast enhanced CT demonstrates arterial enhancement of the lesion.

References