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Endoscopic ultrasound appearance of pancreatic serotonin-staining neuroendocrine neoplasms

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ABSTRACT

Background/Objectives: The pancreatic localization of serotonin-staining neuroendocrine neoplasms is extremely rare. This is a retrospective study aimed at analyzing the endoscopic ultrasound appearance of pancreatic serotoninoma.
Methods: Between 2010 and 2016, all consecutive patients with histologically proven pancreatic serotoninoma who had undergone endoscopic ultrasound were enrolled.
Results: Eight patients (six F, median age 68.5 years) had a diagnosis of pancreatic serotoninoma and underwent endoscopic ultrasound examinations. Median diameter of the lesion was ten mm. The nodule echotexture was hypoechoic in seven out of eight cases. The most frequent localization was the pancreatic neck (four); in three cases, the tumor was located in the pancreatic head and in one in the body. In seven cases the tumor caused a main pancreatic duct dilation; in three cases also the secondary ducts were dilated. In one case a dilation of the common bile duct was observed. At contrast-enhanced endoscopic ultrasound no one showed the typical contrast-enhancement. Elastography (available in two patients) showed a rigid pattern of the lesion.
Conclusions: From this case series a specific endoscopic ultrasound appearance resulted for pancreatic serotoninoma, different from other types of pancreatic neuroendocrine neoplasm, but it is difficult to differentiate it from a pancreatic adenocarcinoma or an intraductal papillary mucinous neoplasm.
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1. Introduction

Pancreatic neuroendocrine neoplasms (pNENs) are relatively infrequent (1%–2% of all pancreatic neoplasms) [1].

Endoscopic ultrasound (EUS) is a key technique for their diagnosis, WHO grading and staging, which influence the prognosis and treatment of pNENs [2–10]. At EUS NENs generally have regular margins, are hyper-enhanced during the arterial phase and rarely show any infiltration of the pancreatic ductal system or peripancreatic vessels [11,12].

pNENs usually arise from islet cells. Otherwise, carcinoid tumors or serotoninomas, which arise from enterochromaffin cells, are rarely located in the in the pancreas. Pancreatic serotonin-staining neuroendocrine neoplasms (pSNENs) represent the 0.0057–1.4% of pNENs [13,14]. Following their first description by Peart and Porter in 1963 [15], less than 350 cases have been so far reported in literature: pSNENs differ from intestinal enterochromaffin cell tumors and from other pNENs arising from islet cells, as they produce an atypical carcinoid syndrome, (characterized by pain and less frequently by diarrhea and weight loss), had elevated urinary 5-Hydroxy-indolacetic-acid (5-HIAA) levels in 85% of the cases and, although immunocytochemical sensitivity for serotonin close to 100% [14,16,17], have a different immunohistochemical profile [18]. Moreover, a minority of pNENs has been recently described showing distinct clinicopathologic features: they are hypocellular and associated with dense stromal fibrosis, showing a typical trabecular architecture and serotonin immunoreactivity [19–21] and presenting with large pancreatic duct involvement [22].

The number of experiences with EUS in NENs remain limited. We therefore report on the results of a retrospective study of eight

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cases of pSNENs in order to describe their EUS and morphological features and to point out to the clinical implications raised by these rare tumors.

2. Methods

2.1. Subjects

We enrolled all the patients aged over 18 years old with histologically proven pSNEN who had undergone EUS between 2010 and 2016 at the Neuroendocrine Tumor Research Center of the Gastroenterology and Endoscopy Unit, Fondazione IRCCS Ca' Granda Ospedale Maggiore Policlinico of Milan (Italy), and Ospedale San Raffaele IRCCS of Università Vita-Salute of Milan.

2.2. Endoscopic ultrasound (EUS)

All the patients provided their written informed consent for EUS, contrast-enhanced EUS (CE-EUS) and endoscopic fine-needle aspiration (EUS-FNA). Moreover, all the patients underwent either CT or MRI scan.

EUS was performed under deep sedation induced by an intravenous injection of propofol. A linear scanning echoendoscope (EG-3870 UTK linear echoendoscope, Pentax Inc., Tokyo, Japan) equipped with an ultrasound processor (Arietta, HI VISION Preirus ultrasound platform, Hitachi Medical Corp., Tokyo, Japan) was used. The echoprobe was routinely covered with a water-filled balloon to allow for the adequate transmission of ultrasound and to improve image quality. Images of the whole pancreas were taken according to the standard method.

The contrast-enhancement examination was performed by *i. v.* injection of a bolus of 4.8 ml of a second-generation contrast medium (SonoVue, Bracco Spa, Milan, Italy), containing microbubbles of sulphur hexafluoride gas, through a three-route catheter from a 20-gauge needle in an antecubital vein. This was followed by a washing with 10 ml of saline solution, in order to allow for contrast-specific imaging at low insonation power (mean mechanical index <0.1). The contrastographic behavior was evaluated continuously for 3 min after contrast injection. For each lesion, contrast fill-in and wash-out were continuously assessed throughout the enhancement phases, comparing the lesion enhancement with the surrounding pancreatic parenchyma.

Whenever possible, a qualitative real-time tissue elastography was performed using a visual scale to evaluate the focal lesions: in the colour map the hardest tissue was displayed as blue and the softest tissue as red. The region of interest was manually selected so that the lesion is centred; large vessels and areas at depth with no B-mode signal area were excluded. For calculation of strain ratio, two different areas were selected, areas A (lesion) and B (pancreatic tissue outside the tumor). Strain ratio was automatically calculated as the quotient B/A.

EUS-FNA was performed using a system of 22- or 25-gauge needles (Expect; Boston Scientific, Boston, MA or Echotip; Wilson-Cook Medical Inc., Winston-Salem, NC) together with a linear scanning echoendoscope. The *trans*-duodenal or *trans*-gastric approaches were chosen for lesions in the pancreatic head, body and tail, respectively. The samples were immediately reviewed by on-site cytopathologists to ensure the adequacy of the specimens.

2.3. Histology

The slides were immediately evaluated for adequacy and preliminary interpretation. The aspirated material was totally expelled onto slides for conventional smears and prepared as previously described [23]. It was sprayed onto the slides by air using a syringe and adequacy was assessed for cell samples stained with the Diff-Quick technique. The cytotechnician reviewed the smears immediately on site to ensure the specimen was adequate and then the same slides were reviewed by a single blinded cytopathologist with experience in pancreatic FNA interpretation to give the final diagnosis. Cell-block material was obtained by a separate pass in each patient and fixed in 10%-buffered neutral formalin. The number of passes to undertake to obtain satisfactory specimens was documented in each case.

The tumors were classified according to the WHO classification [2]. Tumor stage and grade were assessed according to the criteria proposed by the European NeuroEndocrine Tumor Society [2,24]. All the lesions were pathologically characterized as NENs by positive immunohistochemical staining for chromogranin A and/or synaptophysin. The proliferation index was evaluated using MIB-1 antibody (Dako, clone MIB-1, mouse monoclonal, 1:100 dilution).

The pSNENs were identified and selected using immunohistochemistry. Only the tumors showing a strong immunoreactivity for

Table 1
Clinical and biochemical characteristics of patients with pancreatic serotonin-staining neuroendocrine neoplasms.

#	Gender	Age (years)	Histology ([F1]CgA/Synaptophysin/Serotonin)	Ki-67 (%)	[F2]WHO 2010 grading	Clinical picture	[F3]TNM and Stage	Biochemistry	Clinical diagnosis of[F4]IPMN
1	M	61	+/+/+	5	[F5]G2	Carcinoid syndrome	T3N1M1, stage IV	[F6]5HIAA = 126 (mg/24 h) *CgA = 2570 (ng/ml)	–
2	F	68	+/+/+	5	[F5]G2	Abdominal pain	T1NXM1, stage IV	[F7]NA	–
3	F	69	+/+/+	10	[F5]G2	Acute pancreatitis	T1N0M0, stage I	[F7]NA	–
4	F	69	+/+/+	1	[F5]G1	Abdominal pain	T1N0M0, stage I	[F7]NA	Mixed[F4]IPMN
5	M	77	+/+/+	2	[F5]G1	Acute pancreatitis	T1N0M0, stage I	[F7]NA	Main duct[F4]IPMN
6	F	72	+/+/+	2	[F5]G1	Asymptomatic	T2N0M0, stage IIa	[F7]NA	–
7	F	56	+/+/+	2	[F5]G1	Asymptomatic	T2N0M0, stage IIa	[F7]NA	–
8	F	60	+/+/+	1	[F5]G1	Acute pancreatitis	T1N0M0, stage I	[F7]NA	Mixed[F4]IPMN

[F1]CgA: Chromogranin A; [F2]WHO: World Health Organization; [F3]TNM: tumor, nodes, metastases; [F4]IPMN: intraductal papillary mucinous neoplasm; |[F5]G: grading; [F6]5HIAA: 5-Hydroxy-indolacetic-acid; [F7]NA: not available.

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