Imaging features of intrapancreatic accessory spleen

L A SPENCER, MD, D L SPIZARNY, MD and T R WILLIAMS, MD

Henry Ford Hospital, Detroit, MI, USA

ABSTRACT. Although accessory spleens are commonly identified on CT, intrapancreatic accessory spleen (IPAS) is often not recognised or is mistaken for other pancreatic lesions. Currently, with improved cross-sectional techniques and spatial resolution, IPAS is more detectable. We report the imaging features and work-up for the differentiation between IPAS and other pancreatic lesions. An index case of a suspected pancreatic tail islet cell tumour, subsequently confirmed as IPAS, led to inquiries into the incidence of IPAS and the means of preventing unnecessary surgery. For 2 years, we searched for IPAS during our daily interpretations and compared these cases with those taken from our institution's database to determine the distinguishing characteristics. Three proven cases of IPAS, which mimicked pancreatic tail lesions on CT, are presented. Nine patients with suspected IPAS, based on imaging characteristics and stability, are also described. All cases of IPAS are well defined, 1–3 cm in size, follow the density and intensity of the spleen on CT and MRI, and accumulate technetium-99m ($^{99}\text{Tc}^{\text{m}}$) sulphur colloid and $^{99}\text{Tc}^{\text{m}}$ heat damaged red blood cell scintigraphy (in contrast to other lesions). In conclusion, radiologists should be aware that a subtle pancreatic tail lesion could be an IPAS. A high index of suspicion will lead to correlative imaging. A combination of CT, MRI and nuclear medicine examinations can confirm the diagnosis and prevent unnecessary surgery.

Received 14 January 2009
Revised 29 April 2009
Accepted 1 May 2009

DOI: 10.1259/bjr/20308976

© 2010 The British Institute of
Radiology

Accessory spleen is a congenital anomaly found in approximately 10% of the population, with only one out of six cases occurring in the pancreatic tail [1, 2]. In a 1959 autopsy study of 3000 patients, an intrapancreatic accessory spleen (IPAS) was found in 17% of patients who were identified with accessory spleens [1]. IPAS is benign, asymptomatic, stable over years of follow-up imaging and often mimics other enhancing pancreatic lesions. Currently, with improved cross-sectional techniques, better spatial resolution and dynamic contrast imaging, IPAS is more readily detected but can be mistaken for other pancreatic lesions. Our goal is to report the imaging findings of IPAS and to discuss the imaging work-up needed to improve the differentiation of IPAS from other pancreatic lesions in order to prevent unnecessary biopsies or surgical resection.

Methods and materials

Our index patient was a 47-year-old Caucasian female who presented with abdominal pain, nausea, vomiting and diarrhoea 1 month after trauma. CT reported a pancreatic tail lesion suspicious for islet cell tumour. Surgical excision confirmed IPAS.

During routine daily interpretation, we identified 11 additional patients with suspected IPAS over the next 2 years (from September 2006 to August 2008). Patient ages ranged from 47–87 years. Four patients were male and seven were female. Two of the male patients were proven to have IPAS by nuclear medicine sulfur colloid scintigraphy. The remaining nine patients had no other

imaging performed other than follow-up CT, but were suspected of having IPAS based on imaging characteristics and stability.

Three additional patients were gathered from our institution's database of teaching files for the purpose of comparison. Two patients were diagnosed with renal cell carcinoma metastases and one with surgically proven islet cell tumour.

Imaging studies used to confirm the aetiology of the identified pancreatic tail lesions included CT, MRI and nuclear medicine (including sulphur colloid (SC), heat damaged red blood cell (HDRBC) and Octreotide scintigraphy).

Results

We report three cases of IPAS that radiographically mimicked pancreatic tail masses on CT: one surgically proven IPAS case and two imaging-proven IPAS cases. We also report on 11 additional patients with suspected IPAS based on imaging characteristics and 1–6 years of stability on follow-up imaging.

Our index patient presented with abdominal pain, nausea, vomiting and diarrhoea 1 month after pelvic trauma. CT was reported to show a mass in the pancreatic tail (Figure 1a–c). IPAS was not considered in the initial differential diagnosis. A distal pancreatectomy was performed and the mass was confirmed as IPAS. This case demonstrates (i) improved lesion detection, (ii) the misinterpretation of a benign lesion for a surgical lesion and (iii) an unnecessary surgical intervention.

Two patients had IPAS proven by imaging. Multi-phase CT and MRI demonstrated 1–2 cm hypervascular

Address correspondence to: Leslie Spencer, 803 Royal Avenue, Royal Oak, MI 48073, USA. E-mail: spencer_la@hotmail.com

Figure 1. (a) Arterial, (b) portal venous and (c) delayed CT images demonstrate a 2–3 cm pancreatic tail mass (arrows) that matches the density of the spleen on all phases.

pancreatic tail lesions that matched the density and intensity of the spleen on all phases of imaging. Further work-up with nuclear medicine (SC scintigraphy) confirmed the lesions to be IPAS (Figures 2a–e). These cases demonstrate how consideration of IPAS in the differential diagnoses resulted in the proper imaging studies being performed and the correct diagnosis being obtained through non-invasive means, thereby avoiding surgical intervention.

Nine additional patients had suspected IPAS based on their CT features and stability. All cases demonstrated a 1–3 cm hypervascular pancreatic tail mass that matched the density of the spleen on CT. Follow-up imaging by CT in all seven patients demonstrated 1–6 years of stability in the imaging appearance and size of the pancreatic tail lesions.

The Henry Ford database of teaching files was searched for cases of other hypervascular pancreatic tail lesions in order to characterise the features distinguishing these from our cases of IPAS. On multiphase CT, a pathologically proven islet cell tumour appeared as a 1 cm hypervascular pancreatic tail mass (Figure 3a).

Octreotide and HDRBC scintigraphy demonstrated no radiotracer uptake in the region of the pancreatic tail lesion, making islet cell tumour and IPAS, respectively, less likely. Laboratory values were normal. A distal pancreatectomy was performed, which confirmed the presence of an islet cell tumour.

Two patients with renal cell carcinoma metastases demonstrated larger and more heterogeneous hypervascular pancreatic tail lesions (Figure 3b), as well as other findings suggestive of metastatic disease on follow-up imaging, including increasing size, multiplicity and other sites of metastatic disease.

Discussion

IPAS has been documented in only 10 previous case reports [2–10], but is actually much more common according to the pathological literature, occurring in up to 17% of patients with accessory spleens. We believe that IPAS is becoming more conspicuous owing to multiphase CT and MRI with better contrast and spatial

Figure 2. (a) Pre-gadolinium and multiphase post-gadolinium (b–d) MR images demonstrate that the pancreatic tail mass (arrows) matches the signal intensity of the spleen on all phases. (e) Sulphur colloid coronal SPECT (single photon emission CT) imaging demonstrates radiotracer uptake in the pancreatic tail mass, confirming intrapancreatic accessory spleen.

resolution. Now that we have seen several cases of IPAS and are aware of this entity and its imaging characteristics on multiple imaging modalities, we believe we are better able to recognise these sometimes subtle lesions. This awareness, coupled with better imaging, may help

to diminish the disparity between the radiological and pathological literature.

On CT and MRI, IPAS is most often a small (1–3 cm), well-defined lesion that matches the density and intensity of the spleen on all contrast phases and remains

Figure 3. (a) Arterial phase CT of islet cell tumour (arrow). (b) Arterial phase CT Renal cell carcinoma metastasis (arrow).

stable over consecutive imaging. However, IPAS can be mistaken for other enhancing pancreatic tail lesions, including neuroendocrine tumours and metastatic disease (Table 1). There are multiple imaging modalities available to distinguish IPAS from other hypervascular pancreatic tail lesions, including CT, MRI and nuclear medicine.

MRI with ferumoxides shows preferential uptake in hepatic and splenic tissues given their rich reticuloendothelial composition, thereby allowing the differentiation of IPAS from other pancreatic tail lesions (Figure 4).

Nuclear medicine — technetium-99m ($^{99}\text{Tc}^{\text{m}}$)-SC and $^{99}\text{Tc}^{\text{m}}$ -HDRBC scintigraphy — can be utilised as a confirmatory modality when IPAS is suspected. Splenic tissue traps up to 90% of the injected HDRBC, thereby making $^{99}\text{Tc}^{\text{m}}$ -HDRBC scintigraphy a very sensitive and specific test. However, $^{99}\text{Tc}^{\text{m}}$ -HDRBC scintigraphy is more time-consuming than $^{99}\text{Tc}^{\text{m}}$ -SC scintigraphy and requires direct handling of blood products.

Neuroendocrine tumours, including islet cell tumours, are often small hypervascular pancreatic lesions. The presence of a functional neuroendocrine tumour can be diagnosed from the serum hormone levels; however, up to 30% of neuroendocrine tumours may be non-functional [11]. Neuroendocrine tumours contain somatostatin receptors and therefore can be confirmed with octreotide scintigraphy (Octreoscan). Octreoscan has a

70–95% sensitivity for detecting neuroendocrine tumours. Splenic tissue can also show uptake on Octreoscan, probably owing to the somatostatin receptors on lymphocytes, thereby leading to occasional false-positive results in the setting of IPAS [7]. The differentiation of neuroendocrine tumours from IPAS is best made with $^{99}\text{Tc}^{\text{m}}$ -HDRBC or $^{99}\text{Tc}^{\text{m}}$ -SC scintigraphy.

Primary malignancies that commonly metastasise to the pancreas include lung, breast, gastrointestinal and renal lesions, melanoma, lymphoma and osteosarcoma [12]. The majority of metastatic lesions are larger, more heterogeneous, and well-defined solitary masses. Imaging features that favour metastatic disease include multiplicity, hypervascularity, features consistent with the primary tumour and an enlarging mass on follow-up imaging [12]. Clinical history of a known malignancy also helps in diagnosing a pancreatic lesion as metastatic disease. Renal cell carcinoma is the most common malignancy to metastasise to the pancreas, often presenting as a small, well-defined, hypervascular mass.

IPAS is becoming a more easily detectable lesion with the ever-improving imaging capabilities and increasing awareness of the anomaly. However, we believe that we are still seeing only a fraction of what is reported in the pathological literature. Radiologists should know that a subtle enhancing pancreatic tail mass on CT could be an IPAS. A high index of suspicion will lead to correlative

Table 1. Differential diagnoses: imaging features

	IPAS	Neuroendocrine tumour	Metastasis
CT	Well-defined. Matches density of spleen on all phases Homogeneous enhancement	Hypervascular Does not match density of spleen	Multiple Heterogeneous May be hypervascular Variable T_1 , T_2 and enhancement
MRI	Matches intensity of spleen on all sequences (low T_1 , high T_2 , homogeneous enhancement)	Low T_1 , high T_2 Ring-like or homogeneous enhancement	
Nuclear medicine	Uptake on SC and HDRBC scans. Occasional false-positive on Octreoscan	Uptake on Octreoscan (70–95% sensitivity)	No uptake on SC, HDRBC or octreotide scintigraphy

IPAS, intrapancreatic accessory spleen; SC, sulphur colloid; HDRBC, heat damaged red blood cell.

Figure 4. MRI pre and post ferridex demonstrates signal loss in the pancreatic tail mass, confirming intrapancreatic accessory spleen.

Figure 5. Imaging work-up of intra-pancreatic accessory spleen.

imaging (Figure 5). A combination of CT, MRI and nuclear medicine examinations can confirm the diagnosis of IPAS and prevent unnecessary surgical resection.

References

1. Halpert B, Gyorkey F. Lesions observed in accessory spleens of 311 patients. *Am J Clin Pathol* 1959;32:165–8.
2. Lauffer JM, Baer HU, Maurer CA, Wagner M, Zimmermann A, Buchler MW. Intrapaneatic accessory spleen. A rare cause of a pancreatic mass. *Int J Pancreatol* 1999;25:65–8.
3. Tozbikian G, Bloomston M, Stevens R, Ellison EC, Frankel WL. Accessory spleen presenting as a mass in the tail of the pancreas. *Ann Diagn Pathol* 2007;11:277–81.
4. Uchiyama S, Chijiwa K, Hiyoshi M, Ohuchida J, Imamura N, Nagano M, et al. Intrapaneatic accessory spleen mimicking endocrine tumor of the pancreas: case report and review of the literature. *J Gastrointest Surg* 2008;12:1471–3.
5. Churei H, Inoue H, Nakajo M. Intrapaneatic accessory spleen: case report. *Abdom Imaging* 1998;23:191–3.
6. Ota T, Tei M, Yoshioka A, Mizuno M, Watanabe S, Seki M, et al. Intrapaneatic accessory spleen diagnosed by technetium-99m heat-damaged red blood cell SPECT. *J Nucl Med* 1997;38:494–5.
7. Brasca LE, Zanello A, De Gaspari A, De Cobelli F, Zerbi A, Fazio F, et al. Intrapaneatic accessory spleen mimicking a neuroendocrine tumor: magnetic resonance findings and possible diagnostic role of different nuclear medicine tests. *Eur Radiol* 2004;14:1322–3.
8. Harris GN, Kase DJ, Bradnock H, McKinley MJ. Accessory spleen causing a mass in the tail of the pancreas: MR imaging findings. *AJR Am J Roentgenol* 1994;163:1120–1.
9. Sica GT, Reed MF. Case 27: intrapancreatic accessory spleen. *Radiology* 2000;217:134–7.
10. Hayward I, Mindelzun RE, Jeffrey RB. Intrapaneatic accessory spleen mimicking pancreatic mass on CT. *J Comput Assist Tomogr* 1992;16:984–5.
11. Meyer-Rochow G, Gifford A, Samra J, Sywak M. Intrapaneatic splenunculus. *Am J Surg* 2007;194:75–6.
12. To'o KJ, Raman SS, Yu NC, Kim YJ, Crawford T, Kadell BM, et al. Pancreatic and peripaneatic diseases mimicking primary pancreatic neoplasia. *Radiographics* 2005;25:949–65.