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HETEROTOPIC PANCREAS REVEALED BY POST-TRAUMATIC PANCREATITIS

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We report the case of an 82-year-old female presenting with acute epigastric abdominal pain after traumatic blow on the epigastrum. High-resolution multimodal imaging comprising Ultrasound, CT and MR, correlation with laboratory blood analyses and a 6 months CT follow-up allowed us to make a definite diagnosis of traumatic heterotopic pancreatitis. This case emphasizes the relevance of a well-targeted high-definition ultrasound study in the following of the first imaging modality.

Key-words: Pancreatitis – Abdomen, injuries.

Heterotopic pancreas (HP) is characterized by pancreatic tissue found in ectopic locations at various sites of the body, most frequently in the gastrointestinal tract. This anatomic variation is quite frequently observed in postmortem examinations but is very rare and difficult to demonstrate by non-invasive imaging modalities. In this case, we would like to emphasize the relevance of a well-targeted high-definition ultrasound study to characterize non specific tissular abnormalities observed on the first imaging modality.

HP is most often asymptomatic but can present the same pathology than normotopic pancreas or lead to mechanical complication due to aberrant localization. Pancreas inflammation can be idiopathic but is most often cause by biliary or gallstones and alcohol. Less common causes are auto-immune pancreatitis, drug-induced pancreatitis, vasculitis, viral infections, hypertriglyceridemia or hypercalcemia, porphyrias and direct trauma of the gland (including post ERCP). The diagnostic criteria for pancreatitis combine characteristic abdominal pain with serum elevation of amylase and/or lipase, and characteristic findings of acute pancreatitis on CT scan.

Case report

An 82-year-old woman presented to the emergency room with a 12 hours history of increasing continuous epigastric abdominal pain. Symptoms began shortly after the patient received a blow on the epigastrum by falling on a small table.

Fig. 1. — Unenhanced abdominal CT. Axial (A,B), coronal (C) and sagital (D) views reveal an inflammatory round mass (white arrowhead) adjacent to the duodenojejunal flexure (asterisks), associated with focal bowel wall thickening, peripheral mesenteric fat infiltration and descending fluid collection along the proximal jejunum (black arrows). Pancreatic tissular lobules and fatty interstitium are well perceptible (white arrowhead), similar to normal pancreas (white arrows).

Nausea, vomiting or melena were absent and the patient was apyretic. At physical examination central abdominal soreness was found, but peristaltism was still present and defense, rebound or organomegaly

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were absent. Pertinent laboratory values included normal liver function tests and normal white blood cell count but elevated serum amylase (158 IU/l), lipase (370 IU/l), LDH (619 IU/l) and CRP (14.3 mg/dl) were found.

Unenhanced abdominal CT was performed on admission (Fig. 1) and revealed an inflammatory round mass snuggled up to the duodenojejunal flexure at the angle of Treitz. Peripheral mesenteric fat stranding, localized focal bowel wall thickening and ill-defined fluid collection along the proximal jejunum were associated. The round inflammatory mass had a lobulated appearance with fatty infiltration strongly resembling to elderly pancreatic tissue.

High resolution ultrasound study was secondarily performed with a linear probe (3-9 MHz) and clearly delineated a 2,5 cm round echogenic homogenous mass, surrounded by an arciform non-peristalting and thickened duodenojejunal loop (Fig. 2). A central Y-shaped ductal system connected by a single duct to the thickened bowel wall was clearly delineated within the mass confirming the presumed diagnosis of heterotopic pancreatitis.

Classic conservative treatment was proposed with imaging and biological follow-up. Spontaneous recovery was obtained.

Pancreatic serum tests reached a maximum level 3 days after admission (amylase 170 IU/l, lipase 564 IU/l) and regained normal levels 18 days later (amylase 95 IU/l, lipase 61 IU/l).

MR imaging was performed 10 days after admission. Axial T1-weighted series showed an area with a signal similar to that of the normotopic pancreas (Fig. 3). Unfortunately, this exam was of poor quality because of the difficulties of the patient to stay in apnea during the acquisitions. Moreover peripheral mesenteric fat infiltration and fluid

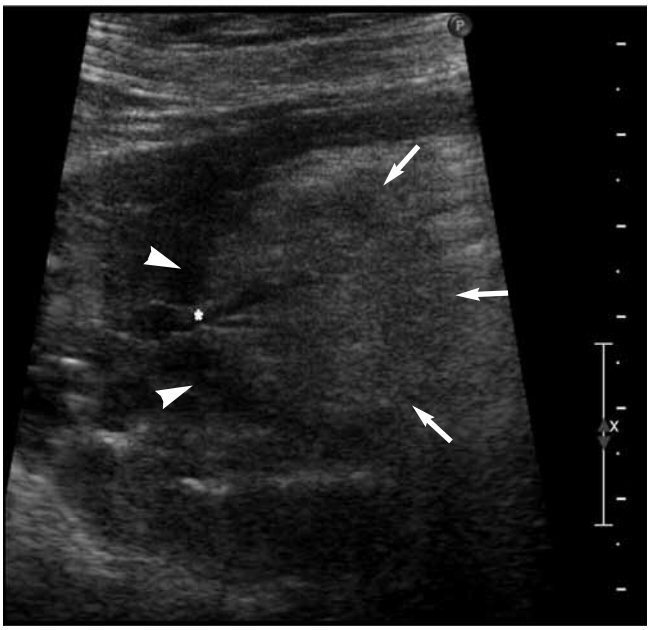

Fig. 2. — High-resolution ultrasound study reveals a 2.5 cm in diameter round mass (arrows) adjacent to a non-peristaltic thickened duodenojejunal flexure. A branched ductal system (asterisk) connected to the inferior wall of the duodenojejunal loop (white arrowheads) is well observed, confirming the diagnosis of ectopic pancreas.

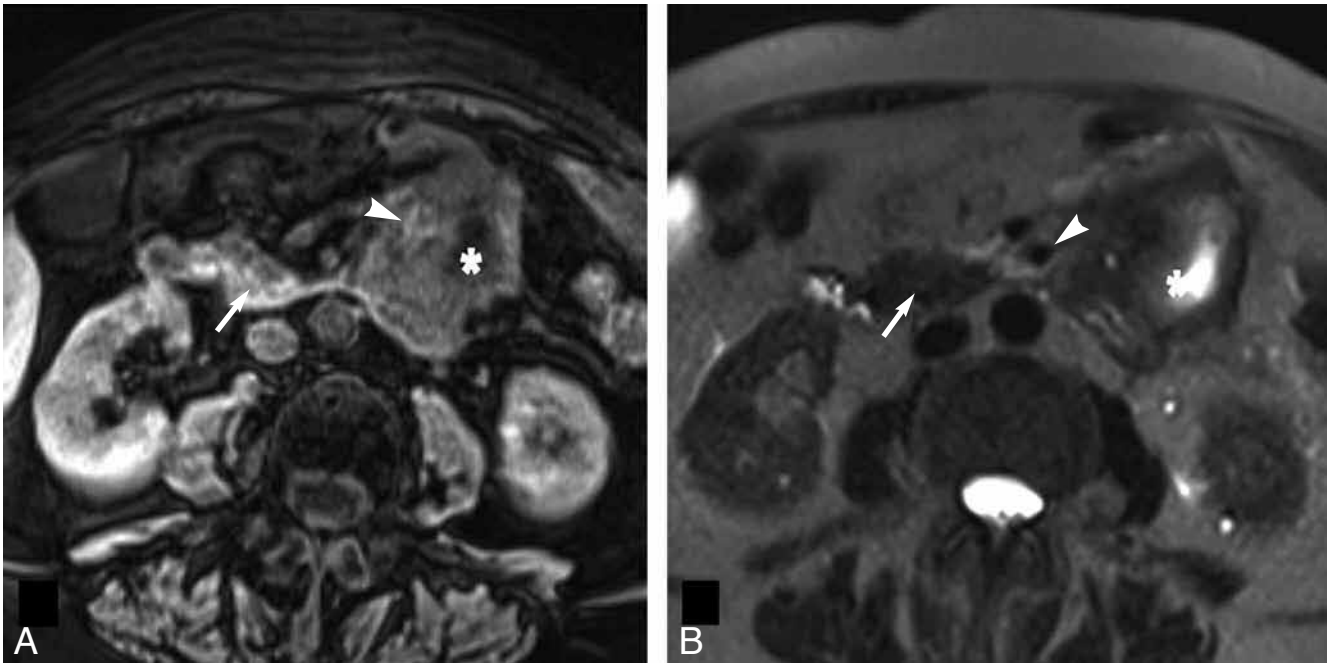

Fig. 3. — Axial MR series confirm the similar T1-weigthed (A) and T2-weighted (B) signal of normotopic pancreas (arrows) and ectopic pancreas (white arrowhead). Adjacent effusion is found (white asterisk).

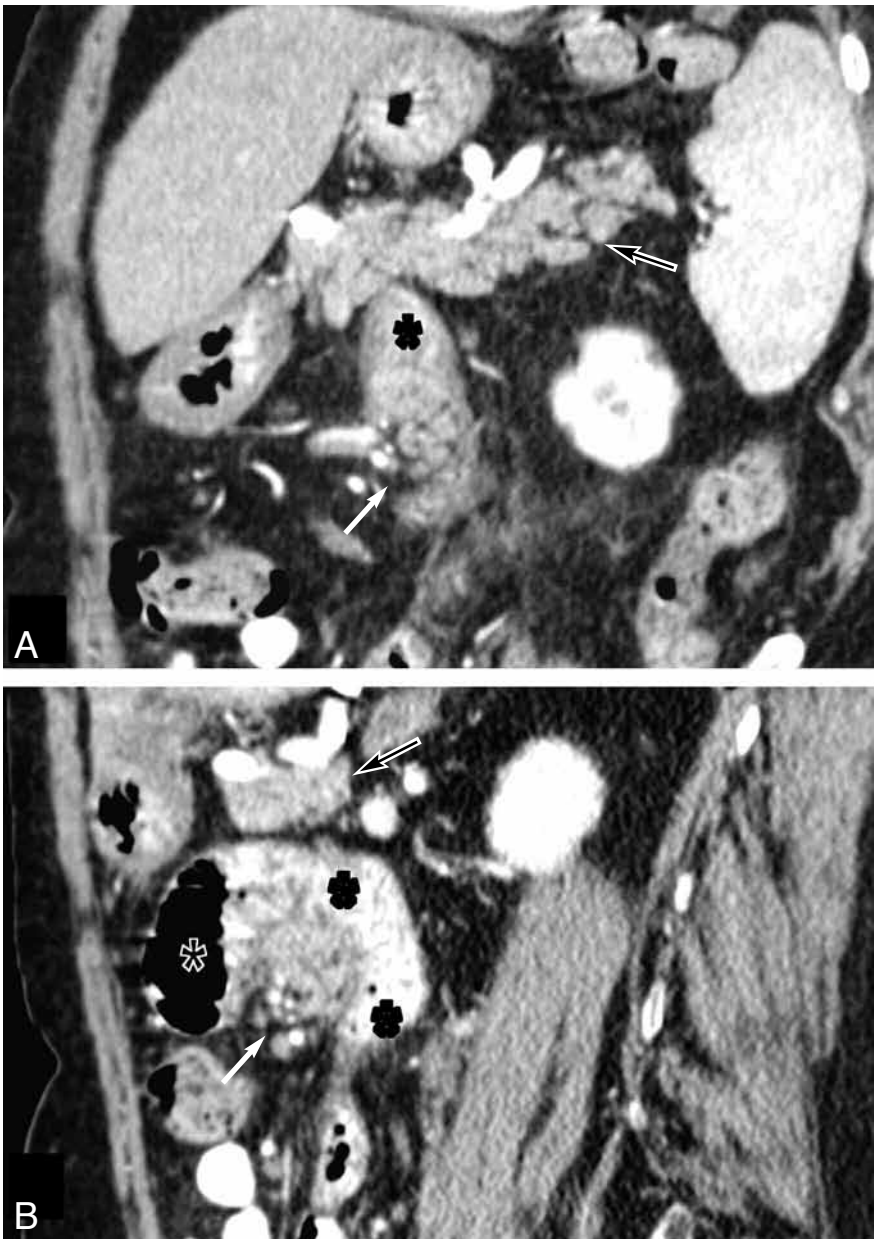

Fig. 4. — Contrast enhanced CT obtained 6 months after admission demonstrates spontaneous recovery, with disappearance of fluid collections. On MPR coronal oblique (A) and sagittal (B) view, ectopic pancreatic lobulation and enhancement (white arrows) is well visible and appears similar to normotopic pancreas (white arrow). HP is adjacent to the duodenojejunal flexure (black asterisks).

collections disturbed the precise visualization of the ectopic pancreas. The normotopic pancreas had a normal appearance.

Abdominal CT with intravenous contrast agent injection was performed 6 months later. Fat infiltration and fluid collections had completely disappeared and the presumed heterotopic pancreas actually showed an homogenous enhancement pattern similar to that of normotopic pancreatic tissue (Fig. 4). Pancreatic lobules and fatty interstitium were better characterized. There was no expansive process.

The definite diagnosis of acute post-traumatic heterotopic pancreatitis was finally made on the basis of multimodality high-quality imaging, biological and clinical follow-up.

Discussion

Anatomical congenital pancreatic abnormalities are classified as: pancreas divisum, annular pancreas, agenesis of the dorsal pancreatic bud and ectopic pancreatic tissue.

Heterotopic pancreas (HP) is defined as aberrant but well-developed pancreatic tissue lacking anatomic and vascular continuity with the main body of the pancreas. HP incidence ranges from 1% to 14% in literature (1). The most frequent localizations are the stomach, duodenum, jejunum and ileum (including Meckel's or other diverticula). Less common sites include the liver, spleen, esophagus, biliary tract, fallopian tubes, mesentery and omentum, mediastinum or even umbilicus (1, 2). At least, heterotopic pancreatic tissue is frequently observed in gastric duplication cysts (3).

HP can have a submucosal localization (75%), or can be present within the muscularis propria or the serosal surface of the GI tract (4).

Variable amounts of pancreatic acinar and islet tissue are seen. The heterogeneity of these microscopic features is codified by the Heinrich classification. Class I lesions contain pancreatic acini, islets, and ducts; class II lesions contain acini and ducts but no islets; and class III lesions are composed of ducts alone.

The proposed pathogeneses are transplantation of pancreatic cells to adjacent structures during embryonic development or metaplasia of multipotent endodermal cells.

HP can be seen at any age, but because of its slow growing it is most often observed in adults (5). Moreover, in most cases HP remains asymptomatic and is an incidental finding. Symptomatic HP is usually found in the stomach or duodenum, with complaints of epigastralgia mimicking peptic disease (1).

Potential complications of HP are mass effect causing bowel intussusception or obstruction, acute pancreatitis, and less frequently bleeding, cystic degeneration or malignancy of the exocrine or endocrine ectopic tissue.

The need for treatment depends on symptoms and definite diagnosis, excluding particularly a malignant process (6). In our case, conservative treatment was privileged, like for classical mild entopic pancreatitis, after confirmation of clinical, biological and imaging recovery and owing to the patient old age. However, some investigators recommend surgical treatment (7-9), especially if diagnosis remains unclear.

Incidental finding of HP does not require any operation (8).

Heterotopic pancreatitis can lead to hemorrhage, necrosis, bowel perforation and acute or chronic inflammation, although being usually only as a microscopic finding. Late complications like pseudocyst formation have been reported (10). During acute HP inflammation, the elevation of serum amylase and lipase levels remain rather limited due to the small volume of pancreatic tissue in the heterotopic pancreas. In some systemic cause of pancreatitis, like drug-induced or autoimmune pancreatitis, simultaneous inflammation of the normotopic and ectopic pancreas can be observed (11).

In our patient, typical CT appearance of elderly pancreatic tissue with lobulation and fatty infiltration was observed within the normotopic and the heterotopic pancreas, facilitating their characterization. The US demonstration of a central ductal system within the HP tissue was also of most importance to establish the correct diagnosis. Therefore we would like to put the emphasis on the relevance of a well-targeted high-definition ultrasound study in the following of the first imaging modality.

In optimal conditions with cooperative patient, MRI exam could demonstrate the presence of a central ductal system, with T2-weigthed and cholangio-MR sequences (6). This result could equally be obtained with endoscopic ultrasound (EUS) if HP is localized in the stomach or the duodenum, especially if abdominal US exam is difficult (obesity…) (6). At least, some authors reported the role of barium X-ray series to demonstrate nonspecific fold thickening with the characteristic appearance of a centrally umbilicated nodule in the gastric mucosa within the gastric heterotopic pancreatic rest (6).

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