The Bloody Pancreas: MDCT and MRI Features of Hypervascular and Hemorrhagic Pancreatic Conditions

V. Anik Sahni1
Koenraad J. Mortelé

OBJECTIVE. The purpose of this study was to illustrate the varied MDCT and MRI appearances of hypervascular and hemorrhagic pancreatic conditions and their mimics.

CONCLUSION. Pancreatic hypervascular conditions are easily detected at multiphasic contrast-enhanced MDCT and MRI. Hemorrhagic pancreatic abnormalities are best depicted with unenhanced CT and, especially, fat-suppressed T1-weighted MRI. Familiarity with the spectrum of possible underlying causes and the imaging features and conditions that can act as mimics assists radiologists in making an accurate presumptive diagnosis.

Hypervascular and hemorrhagic pancreatic abnormalities are a diverse group of conditions. They have a wide spectrum of causes that include neoplastic, vascular, inflammatory, traumatic, and congenital conditions. Knowledge of the range of pathologic findings is helpful in limiting the differential diagnosis and facilitates an accurate presumptive diagnosis, resulting in optimal patient care. Several entities can mimic pancreatic hypervascular and hemorrhagic lesions, and an appreciation of these conditions is crucial to prevent unnecessary intervention.

Imaging Techniques

Accurate diagnosis of hypervascular and hemorrhagic pancreatic abnormalities is contingent on optimized imaging technique. Unenhanced MDCT and MRI, especially MRI, which provides superior soft-tissue contrast, are particularly useful in the detection of hemorrhage. Fat-suppressed T1-weighted MRI is highly sensitive to the paramagnetic effect of methemoglobin and thus to the presence of hemorrhage. T2-weighted imaging also is susceptible to the paramagnetic effects of blood and can be used in conjunction with T1-weighted imaging for further characterization. Gradient-recalled echo (GRE) sequences are more sensitive to the magnetic susceptibility effects of hemorrhage than are spin-echo sequences [1].

Both contrast-enhanced MDCT and MRI can be used to assess the vascularity of pancreatic lesions. A multiphasic approach is ideal for precise characterization. Imaging can be performed with a combination of phases depending on the indication. These phases include the early arterial phase (20–30 seconds after IV contrast administration), the pancreatic parenchymal phase (35–50 seconds after IV contrast administration), and the portal venous phase (60–70 seconds after IV contrast administration). Most nonvascular hypervascular abnormalities are best depicted in the pancreatic parenchymal phase. Acquiring images at the optimal time is critical and can be achieved by administering a test bolus of contrast material or by using bolus-tracking techniques [2].

Contrast-enhanced MDCT should be performed with 120–150 mL of iodinated contrast material administered IV at a rapid rate (4–5 mL/s). Thin-section reconstruction (0.625–1.25 mm) of the axial data should be performed with multiplanar reconstructions [3]. MRI can be performed at a field strength of 1.5 or 3 T with a phased-array torso coil. T1-weighted imaging is performed with a high-resolution breath-hold spoiled GRE dual-echo in-phase and out-of-phase sequence. T2-weighted imaging can be performed in the axial and coronal planes with a single-shot fast spin-echo sequence or a fast recovery fast spin-echo sequence. Gadolinium is administered IV in a bolus of 0.2 mL/kg at 2 mL/s followed by a saline flush. Breath-hold serial axial T1-weighted 3D spoiled GRE fat-suppressed images with slice interpolation are acquired before contrast administration and in at least three phases after IV contrast administration.

Keywords: hemorrhage, MDCT, MRI, neoplasms, pancreas, pancreatitis

DOI:10.2214/AJR.08.1602

Received July 28, 2008; accepted after revision October 27, 2008.

1Both authors: Department of Radiology, Brigham and Women’s Hospital, Harvard Medical School, 75 Francis St., Boston, MA 02115. Address correspondence to K. J. Mortelé (kmortele@partners.org).

AJR 2009; 192:923–935
0361–803X/09/1924–923
© American Roentgen Ray Society
 Imaging Appearances of Hemorrhage

Acute hemorrhage typically has high attenuation on unenhanced MDCT scans. The hematoma ages, its attenuation value decreases. The appearance of blood on MR images is highly variable depending on the age of the hematoma and the imaging sequence used [1]. With time, hemoglobin evolves through several forms. Each type of hemoglobin has a specific imaging characteristic. These appearances have been primarily described with regard to intracranial hemorrhage [1] and may not extrapolate directly to intraabdominal hemorrhage. Subacute hemorrhage is typically hyperintense on T1-weighted images and hypointense on T2-weighted images. Because of the hemosiderin content, chronic hematoma can be hypointense on both T1- and T2-weighted images.

Neoplastic Conditions

Endocrine Pancreatic Tumors

Endocrine pancreatic tumors originate from the islet cells of Langerhans that make up the endocrine pancreas. These tumors are rare with a reported incidence of five cases per million persons per year [4]. All endocrine tumors are hormonally active to a variable degree but are typically divided into syndromic (hyperfunctioning, 85%) and nonsyndromic (nonhyperfunctioning, 15%) depending on the clinical and biochemical manifestations [5]. Endocrine tumors can be further classified according to the cell type from which they originate. The cell type determines the predominant hormone secreted by the tumor. Examples of syndromic endocrine tumors in decreasing order of frequency are insulinoma (60%), gastrinoma (20%), glucagonoma (3%), VIPoma (2%), and somatostatinoma (less than 1%) [5]. Syndromic tumors tend to come to medical attention early because of the clinical manifestations of the hormone secreted and tend to be relatively small at diagnosis. Nonsyndromic tumors manifest later, at a larger size, and the clinical symptoms are usually related to mass effect [6]. The malignant potential of insulinoma is low, ranging from 6% to 10%. The other syndromic tumors, however, are more frequently malignant [6].

Imaging of endocrine pancreatic tumors can be challenging to radiologists. Insulinoma is located with an equal distribution in the head, body, and tail of the pancreas. Gastrinoma, however, is located in the gastrinoma triangle formed by the cystic duct confluence, the junction of the neck and body of the pancreas, and the junction of the second and third parts of the duodenum [7]. VIPoma can be extrapancreatic in 10–20% of cases, and somatostatinoma is duodenal in 50% of cases [6]. The size of the tumors is variable; for example, 90% of insulinomas are smaller than 2 cm in diameter, whereas glucagonoma usually is large, having a mean diameter of 6.5 cm [5]. Unenhanced cross-sectional imaging may provide useful information. Areas of necrosis, cystic change, or calcification can be found at MDCT. Insulinoma occasionally has high attenuation on unenhanced MDCT scans [8]. In addition, at MRI, the tumor is usually hypointense on T1-weighted images and hyperintense on T2-weighted images [7]. The key to diagnosis, however, lies in the rich vascular supply of the tumor. A vascular blush on contrast-enhanced MDCT scans or MR images is characteristic (Figs. 1 and 2).

The optimal phase of acquisition is still contentious. The aim of contrast-enhanced imaging is to maximize the contrast between the tumor and the pancreatic parenchyma. The contrast usually is greatest in the late arterial or pancreatic parenchymal phase [9]; however, the portal venous phase has been reported to be more reliable in certain cases [10]. Biphasic scanning therefore is advocated with images obtained in both the late arterial and portal venous phases.

Serous Microcystic Pancreatic Adenoma

Serous microcystic pancreatic adenoma, with the exception of rare cystic endocrine tumors, is the only hypervascular cystic pancreatic neoplasm, an important discriminatory feature for diagnosis. Intratumoral hemorrhage also has been reported to occur [11, 12]. Serous microcystic pancreatic adenoma is a benign neoplasm that occurs most commonly in women older than 60 years. It has a predilection for the pancreatic head but can be found in all parts of the pancreas [13]. An association exists with von Hippel–Lindau disease. The classic histopathologic signature is multiple cysts (more than six), which individually measure less than 2 cm in diameter. A macrocystic or oligocystic variant, however, is composed of fewer and larger cysts [12].

On images, serous microcystic pancreatic adenoma has a lobulated appearance. It has low attenuation on unenhanced MDCT scans and high signal intensity on T2-weighted MR images, reflecting the cystic composition. High signal intensity on fat-suppressed T1-weighted images represents hemorrhage. A stellate fibrous central scar may be present and can calcify. After contrast administration, there is early and vivid enhancement of the fibrous components, which may be persistent on delayed images (Figs. 3 and 4).

Solid Pseudopapillary Tumor

Solid pseudopapillary tumor is a rare pancreatic cystic tumor that occurs almost exclusively in young women. It is usually benign or of low-grade malignancy and therefore has a slow growth rate [13]. Histologically, it is encapsulated [14]. In addition, intratumoral hemorrhage is common and has been reported in 74–98% of cases [14, 15]. These two features can be identified on images and help discriminate solid pseudopapillary tumor from other pancreatic neoplasms.

MDCT classically shows a well-encapsulated lesion with mixed solid and cystic components, which result from hemorrhagic degeneration (Fig. 5). Calcifications and solid areas may be seen peripherally. After contrast administration, the solid tissue and capsule become enhanced to a lesser degree than the surrounding pancreas [16]. MRI also shows a well-defined mass with a heterogeneous appearance on both T1- and T2-weighted images. Areas of hemorrhage are hyperintense on fat-suppressed T1-weighted images and hypointense on T2-weighted images (Fig. 6). Images show early peripheral enhancement with progressive heterogeneous incomplete fill-in in later phases [14].

Acinar Cell Carcinoma

Acinar cell carcinoma is a rare neoplasm that originates from the acinar elements of the exocrine pancreas. Histologically, hemorrhage often is present within the tumor [17]. Acinar cell carcinoma makes up only 1% of pancreatic exocrine tumors and has a slightly better prognosis than ductal adenocarcinoma [18]. The tumor cells can produce excessive pancreatic enzymes, in particular lipase, in 10% of patients, resulting in lipase hypersecretion syndrome, which manifests itself as subcutaneous fat necrosis, polyarthritis, and bone infarcts [5]. The MDCT and MRI appearance of acinar cell carcinoma is an exophytic, well-margined mass [18], which is usually solid when small but contains hemorrhagic, necrotic areas as it enlarges. Enhancement is generally homogeneous but less than that of the surrounding pancreatic parenchyma [18] (Fig. 7).

Metastatic Tumors

Metastatic lesions to the pancreas are rare and usually occur at an advanced stage. Most
Inflammatory and Vascular Conditions

Hemorrhagic Pancreatitis

Pancreatic necrosis occurs in approximately 20% of cases of acute pancreatitis [20]. This disorder can be associated with hemorrhagic changes affecting the pancreatic parenchyma and the extrapancreatic fatty tissue. Mortality in hemorrhagic pancreatitis is variable and reported to range from 33% to 100% [21]. Autodigestion by extravasated pancreatic enzymes results in venule and capillary disruption [22]. The bleeding that ensues is usually self-limited, but marked hemorrhage can occur in 2–5% of patients with acute pancreatitis [23]. High-attenuation material within the pancreatic bed on unenhanced CT scans and high signal intensity on fat-suppressed T1-weighted images correlate with the presence of blood products (Figs. 9 and 10). Pseudocysts may also exhibit hemorrhage [24] (Fig. 11).

Peripancreatic Pseudoaneurysm

Pseudoaneurysm formation occurs in as many as 10% of cases of pancreatitis [20]. The time interval is variable, ranging from days to years after the acute episode. The most common vessels affected are the splenic and gastroduodenal arteries. Early detection and management are paramount given the high mortality associated with rupture. Rupture can occur into the peritoneum, adjacent hollow organs, pseudocyst, or pancreatic duct (hemosuccus pancreaticus) [25]. Dedicated MDCT or MR angiography can elegantly depict the pseudoaneurysm as a well-delineated rounded structure originating from the donor artery [26]. High-attenuation or high-signal-intensity thrombus may be seen within the sac on unenhanced CT scans and fat-suppressed T1-weighted MR images. After contrast administration, the sac may fill with contrast material if it is not completely thrombosed (Fig. 12). If juxtapancreatic in location, a pseudoaneurysm can mimic a hypervascular lesion (Fig. 13). Conventional angiography remains the standard of reference and provides the capability of endovascular management.

Portomesenteric Venous Thrombosis

A range of conditions can result in thrombosis of the portomesenteric venous system. These conditions include abdominal infection, pancreatitis, neoplasms, hypercoagulable states, and cirrhosis with portal hypertension. Because of its proximity to the organ, acute thrombosis of the splenic vein or superior mesenteric vein in particular can mimic a hemorrhagic pancreatic lesion. Important diagnostic features that help in discrimination are usually the lack of enhancement and the tubular configuration of the thrombosed vessel (Figs. 14 and 15). Care should be taken, however, in the interpretation of portal venous thrombosis secondary to malignant neoplasms, such as hepatocellular carcinoma and cholangiocarcinoma. Unlike nonneoplastic (bland) thrombus, this tumor thrombus classically exhibits neovascularity and enhancement [27, 28]. Other features that help in differentiation of benign and malignant thrombus are direct invasion of the portal vein by the tumor and portal venous expansion of 23 mm or more [27].

Peripancreatic Varices

There are multiple causes of portal hypertension. Regardless of the cause, multiple sites of portosystemic collateralization can decompress the increased portal pressure. In the region of the pancreatic head, portocaudodenal varices can develop and simulate a pancreatic hypervascular lesion (Fig. 16). Findings at MDCT or MRI performed with meticulous technique can lead to accurate diagnosis. Dedicated MDCT and MR angiography with 3D multiplanar reformations are useful for visualizing these vessels, which are characterized by a tubular and tortuous configuration. Enhancement is best in the portal venous rather than the arterial phase [29]. Identification of the other features of portal hypertension, such as splenomegaly and ascites, adds to diagnostic confidence.

Miscellaneous Conditions

Trauma

Pancreatic injury can result from blunt (27%) and penetrating (73%) trauma [30] (Fig. 17). In addition, iatrogenic causes such as biopsy and surgery can result in injury (Fig. 18). The primary method of evaluation is contrast-enhanced MDCT. A spectrum of imaging findings can occur that include pancreatic hematoma, laceration, transection, and diffuse gland enlargement. Extrapancreatic findings such as peripancreatic fat stranding, hematoma, and fluid also can be identified [31]. The integrity of the pancreatic duct can be assessed with secretin-enhanced MR cholangiopancreatography or ERCP. Secretin-enhanced MR cholangiopancreatography involves dynamic MR pancreatography after IV stimulation with human or porcine secretin (0.2 µg/kg body weight). The secretin stimulation leads to stimulation of the exocrine pancreatic gland. It also temporarily increases the tone of the sphincter of Oddi during the first minutes after injection, inhibiting release of fluid through the papilla of Vater. The tone then decreases [32]. Secretin therefore initially improves delineation of the pancreatic duct. Secretin-enhanced MR cholangiopancreatography can depict ductal disruption and active leaks. Unlike ERCP, it can depict the duct distal to the injury. This capability is useful for planning intervention. In addition, the technique is useful for evaluation of the pancreatic parenchyma and peripancreatic collections [33].

Accessory Spleen

Accessory spleen is common, being found at 10–30% of autopsies. The location of accessory spleen is variable, 21.8% being located near or within the pancreatic tail [34]. As a consequence, accessory spleen can be misdiagnosed as a hypervascular pancreatic mass. At CT or MRI, an accessory spleen should have the same attenuation or signal intensity as the main spleen. In addition, an accessory spleen becomes enhanced to the same degree as the main spleen with both techniques (Figs. 19 and 20). If CT and MRI findings continue to be equivocal, nuclear scintigraphy can be performed with 99mTc-labeled sulfur colloid or 99mTc-labeled heat-damaged RBCs. The successful diagnosis of intrapancreatic accessory spleen with MRI with reticuloendothelial system-specific contrast agents has been reported [35].

Pitfalls

Duodenal Diverticulum

Duodenal diverticula are located most frequently in the peripanillary region along the medial aspect of the second and third portions of the duodenum. When filled with fluid they can mimic cystic lesions of the pancreatic head [36]. They also can simulate hypervas-
cular pancreatic lesions and peripancreatic pseudoaneurysm if filled with oral contrast material (Fig. 21). Identifying gas within the diverticulum or persistent high density on delayed images can clarify the situation. Gas causes a susceptibility blooming artifact on spoiled GRE MR images that can be identified in the sequence with the longer TE in dual-echo chemical shift imaging.

**Metastasis of Melanoma**

Metastasis to the pancreas from malignant melanoma has been found in as many as 37.5% of autopsy cases [37]. Metastatic lesions from melanoma are hyperintense on T1-weighted images and hypointense on T2-weighted images (Fig. 22). This manifestation is caused by the paramagnetic properties of melanin within the lesion, which shorten the T1 and T2 relaxation times [38]. The appearance on MRI is indistinguishable from that of a hemorrhagic lesion containing methemoglobin. This difference is important to appreciate, because metastasis to the pancreas from melanoma may be the only site of metastatic disease [39] and should be considered if a hyperintense lesion is identified on T1-weighted images. Subtraction imaging can be useful in identifying enhancement in lesions that are hyperintense on unenhanced T1-weighted images. The process involves subtracting T1-weighted unenhanced images from T1-weighted contrast-enhanced images. Note should be made of the amelanotic subtype of melanoma. The lesion can be isointense or hypointense on T1-weighted images owing to the reduced percentage of melanin-containing cells compared with conventional metastatic lesions from melanoma [40].

**Conclusion**

Pancreatic hypervascular conditions are common and easily detected on multiphasic contrast-enhanced MDCT and MR images. Unenhanced CT and, especially, fat-suppressed T1-weighted MRI, are valuable for detecting hemorrhage within pancreatic abnormalities. Familiarity with the spectrum of possible underlying causes and the imaging features and conditions that can act as mimics assists radiologists in making an accurate presumptive diagnosis in most cases.

**References**


34. Morte1e KJ, Morte1e B, Silverman SG. CT features of the accessory sphen. *AJR* 2004; 183:1653–1657

35. Boraschi P, Donati F, Volpi A, Campori G. Intra-


---

**Fig. 1**—55-year-old man with insulinoma. Coronal reformatted CT image obtained in late arterial phase shows homogeneous hypervascular mass (arrow) in mid pancreatic body. Proximal pancreatic duct dilatation (asterisk) is evident.

**Fig. 2**—65-year-old man with insulinoma. Axial T1-weighted fat-suppressed MR image obtained in late arterial phase shows heterogeneous hypervascular mass (white arrow) in pancreatic tail. Hepatic metastatic lesion (black arrow) is present in right lobe of liver. Right renal cyst (asterisk) is incidental finding.

**Fig. 3**—63-year-old woman with serous microcystic pancreatic adenoma. Coronal reformatted CT image obtained in late arterial phase shows lobulated well-circumscribed lesion (arrow) in uncinate process. Mass exhibits hyperenhancement of fibrous portions.
Fig. 4—55-year-old woman with serous microcystic pancreatic adenoma.
A, Axial T1-weighted fat-suppressed unenhanced MR image shows predominantly hypointense lobulated mass (arrow) in pancreatic tail. Areas of internal high signal intensity on T1-weighted images are in keeping with hemorrhage.
B, Coronal T2-weighted MR image shows lesion to be predominantly hyperintense (arrow), confirming its fluid nature.

Fig. 5—23-year-old woman with solid pseudopapillary tumor. Axial CT image obtained in portal venous phase shows large well-defined lesion originating from pancreatic body and tail. Lesion is predominantly cystic with high-attenuation areas (arrows) in keeping with internal hemorrhage.
Fig. 6—18-year-old woman with solid pseudopapillary tumor.  
A, Axial T1-weighted fat-suppressed unenhanced MR image shows large well-defined lesion (arrow) in pancreatic head. Peripheral areas of high T1 signal intensity are in keeping with internal hemorrhage.  
B, Axial T2-weighted MR image shows predominantly cystic lesion with hypointense capsule (arrow).

Fig. 7—64-year-old man with acinar cell carcinoma. Axial CT image obtained in portal venous phase shows large well-marginated mass (arrows) originating from pancreatic head. Lesion has undergone hemorrhagic necrosis in central aspect.

Fig. 8—72-year-old man with metastasis from renal cell carcinoma. Axial T1-weighted fat-suppressed MR image obtained in arterial phase shows multiple hypervascular masses (white arrows) in pancreas. Right adrenal metastasis (black arrow) is present.
Fig. 9—42-year-old man with hemorrhagic pancreatitis. Coronal reformatted CT image obtained in portal venous phase shows high-attenuation material (arrow) within pancreatic bed in keeping with hemorrhagic necrosis.

Fig. 10—62-year-old woman with hemorrhagic pancreatitis. Axial T1-weighted fat-suppressed unenhanced MR image shows large amount of inflammatory change in pancreatic bed. Scattered areas of high signal intensity (arrows) are consistent with hemorrhagic necrosis.

Fig. 11—45-year-old man with pseudocyst. Axial unenhanced CT image shows large well-defined lesion (arrow) originating from pancreatic body. Lesion has high internal attenuation in keeping with hemorrhage.
MDCT and MRI of Bloody Pancreas

Fig. 12—39-year-old man with splenic artery pseudoaneurysm.
A–C, Axial (A), curved planar reformatted (B), and 3D reconstruction (C) CT images obtained in arterial phase show outpouching originating from posterior aspect of splenic artery (black arrow, A and B; arrow, C) that fills with IV contrast material in arterial phase. Multiple pseudocysts (white arrows, A) secondary to pancreatitis are present in upper abdomen. Splenic infarction (asterisk, A) is evident.

Fig. 13—44-year-old man with splenic artery aneurysm.
A and B, Axial T1-weighted fat-suppressed MR images obtained in arterial (A) and portal venous phases (B) shows apparent hypervascular lesion (arrow) in tail of pancreas.
C, Axial T2-weighted MR image shows flow void (arrow) in location of lesion, confirming its vascular origin.

Fig. 14—28-year-old woman with portomesenteric thrombosis.
A–C, Axial CT images obtained in unenhanced (A and B) and arterial (C) phases show thrombosis (arrow) of portal, superior mesenteric, and splenic veins. Thrombus in A and B has high attenuation, mimicking hemorrhagic pancreatic lesion.
Fig. 15—65-year-old man with thrombosis of superior mesenteric vein.  
A, Axial CT image obtained in arterial phase shows expanded superior mesenteric vein containing central high-attenuation thrombus (arrow). Lobulated contour to liver is caused by cirrhosis.  
B, Axial T1-weighted fat-suppressed unenhanced MR image shows area of high signal intensity (arrow) within vein in keeping with hemorrhagic thrombus.  
C, Axial T1-weighted fat-suppressed MR image obtained in late arterial phase shows peripheral flow around central thrombus (arrow).

Fig. 16—63-year-old man with peripancreatic varices. Axial CT image obtained in portal venous phase shows apparent well-circumscribed hypervascular mass (black arrow) in head of pancreas. Lesion is actually pancreaticoduodenal varix secondary to portal hypertension from liver cirrhosis. Irregular contour of liver and trace ascites (white arrow) is evident.

Fig. 17—27-year-old man with pancreatic trauma. Axial CT image obtained in portal venous phase shows pancreatic tail disruption after motor vehicle accident. High-attenuation hemorrhage is seen in pancreatic bed (short arrow) and retroperitoneum (long arrow).
Fig. 18—50-year-old woman with iatrogenic pancreatic trauma.
A, Unenhanced axial CT image shows biopsy needle with tip in mucinous cystic tumor (arrow) of pancreas.
B, Axial T1-weighted fat-suppressed unenhanced MR image obtained after biopsy shows high signal intensity in tumor (arrow) in keeping with hemorrhage. Intratumoral hemorrhage is not a feature of mucinous cystic tumors and in this case was secondary to biopsy.

Fig. 19—36-year-old woman with intrapancreatic accessory spleen.
A–C, Axial (A), curved planar reformatted (B), and coronal (C) CT images obtained in late arterial phase show well-circumscribed highly enhanced lesion (arrow) in tail of pancreas. Attenuation of lesion is identical to that of spleen.
**Fig. 20**—27-year-old woman with intrapancreatic accessory spleen.

A, Axial CT image obtained in arterial phase shows well-circumscribed highly enhanced lesion (arrow) in tail of pancreas. Attenuation and enhancement pattern of lesion are identical to those of spleen.

B, Axial T2-weighted MR image shows lesion is isointense to spleen.

**Fig. 21**—43-year-old man with duodenal diverticulum.

A, Axial CT image obtained in arterial phase shows well-circumscribed high-attenuation lesion (arrow) in region of pancreatic head.

B, Axial T1-weighted fat-suppressed unenhanced MR image shows signal void within lesion (arrow) due to presence of gas within duodenal diverticulum.
Fig. 22—49-year-old man with metastasis from malignant melanoma.

A. Axial T1-weighted fat-suppressed unenhanced MR image shows hyperintense mass (arrow) in pancreatic head. The hyperintensity is caused by melanin rather than methemoglobin.

B. Oblique coronal thick-slab MR cholangiopancreatography image shows biliary and pancreatic ductal dilatation (arrows) with abrupt termination caused by metastatic deposit in head of pancreas.