Disconnected Pancreatic Duct Syndrome—An Often-Overlooked Complication of Acute Necrotizing Pancreatitis

Babu Peter Sathyanathan1  Suria Vennimalai1

1 Barnard Institute of Radiology, Madras Medical College, Chennai, Tamil Nadu, India

Indian J Radiol Imaging 2023;33:281–283.

We present here a case of disconnected pancreatic duct syndrome (DPDS), highlighting the useful role of magnetic resonance cholangiopancreatography (MRCP) in depicting this complication of acute necrotizing pancreatitis (ANP) and the necessity of heightened awareness of radiologists to pick up this diagnosis.

A 36-year-old male patient with a history of chronic abdominal pain secondary to alcoholic pancreatitis, complicated by walled-off pancreatic necrosis, underwent laparotomy with necrosectomy about 6 months ago. A percutaneous drain (PCD) tube was placed during surgery. The patient now presented with complaints of persistent drain output of about 100 mL/day for the past 6 months. At the time of presentation, the patient had no fever, vomiting, abdominal tenderness, or rigidity. The blood workup showed normal white cell count of 7.6 x 10^9/L (normal 4–11 x 10^9/L), normal serum amylase 52 U/L (normal 40–140 U/L), and mildly elevated lipase levels 86 U/L (normal 7–60 U/L). PCD output showed significantly elevated amylase and lipase levels of 29,090 and 37,670 U/L, respectively.

Initially an ultrasound scan of the abdomen was performed, which revealed multiple peripancreatic fluid collections. The patient was subjected to MRCP in a 3 Tesla magnetic resonance imaging scanner (Skyra, Siemens Healthineers, Erlangen, Germany). Standard institute protocol was followed and signal acquisition done using body coil. The sequences used include three-dimensional (3D) space T2 (repetition time, TR 2400 ms; time to echo, TE 698 ms), T2-weighted half-Fourier acquisition single-shot turbo spin-echo (HASTE) axial and coronal sequences (TR 1200 ms, TE 100 ms), true fast imaging with steady-state free precession, and Dixon sequences. HASTE sequence with fat suppression and thick collimation slab was obtained in the coronal plane. It revealed a mildly atrophic body and tail of pancreas (►Fig. 1). Main pancreatic duct (MPD) was visualized for a length of 1.8 cm from the ampulla. A focal discontinuity of MPD was seen in body of pancreas, measuring about 4.3 cm (►Fig. 2). MPD in distal body and tail of pancreas was irregularly dilated with multiple prominent side branches. PCD tube was noted with tip anterior to tail of pancreas (►Fig. 3), with peripancreatic fat stranding. These features were consistent with DPDS.

Endoscopic retrograde cholangiopancreatography (ERCP) and stenting were attempted twice, but failed. The patient was taken up for laparotomy, which revealed a plastered upper abdomen with dense adhesions. A fistulous tract was seen extending from PCD entry site to inferior surface of pancreas, near the tail region, traversing the transverse mesocolon. Adhesions were released, and distal pancreatecosplenectomy was done. The drain tube amylase dropped on postoperative day 10.

In DPDS, there is a circumferential disruption of the continuity of the pancreatic duct resulting from an area of cellular necrosis.1 This compromised ductal integrity leads to extraductal leakage of pancreatic secretions and destruction of viable pancreatic tissue surrounding the duct, and the viable upstream portion of the gland gives rise to the DPDS. The prevalence of DPDS is unknown but studies have shown that ANP can be complicated by DPDS in 16 to 44% of cases.2–4

The typical clinical presentation includes persistent external pancreatic fistula, recurrent pseudocyst, pancreatic ascites, region of walled off necrosis, or obstructive recurrent acute or chronic pancreatitis of the upstream pancreatic parenchyma.5 To confidently diagnose DPD, it is necessary...
to demonstrate all of the following features: (a) necrosis of at least 2 cm of pancreas, (b) viable pancreatic tissue upstream (i.e., toward the pancreatic tail) from the site of necrosis, and (c) extravasation of contrast material injected into the MPD at pancreatography.⁶

The imaging methods used include contrast-enhanced computed tomography (CECT), along with ERCP, MRCP, and endoscopic ultrasonography. To aid the diagnostic process, it is possible to measure amylase levels in the drain output in patients with percutaneously drained fluid collections. A drain amylase content greater than three times the serum amylase levels may raise the suspicion of a fistula.⁷

The reference method for confirming the diagnosis has been ERCP, but being an invasive procedure with a risk of complications, its indications are limited. Furthermore, it fails to show the upstream pancreatic duct and cannot differentiate between a high-grade stenosis and a disconnected duct.¹ In this respect, MRCP is far more appropriate.

MRCP done using secretin is an emerging imaging study of choice to diagnose a DPD, which shows a cutoff of the downstream pancreatic duct with enhancing upstream pancreatic parenchyma.⁸ However, secretin MRCP is not widely available and 3D MRCP is a useful technique to depict the DPD in most instances, as in our case. The main advantage of MRCP is that it can evaluate both the MPD and the pancreatic parenchyma at the same time in contrast to CECT combined with ERCP.

Most peripancreatic collections following acute pancreatitis are treated conservatively or with guided drainage. In the absence of infection, surgery is not usually contemplated. The most common reason for delayed or missed diagnosis is a lack of awareness and general unfamiliarity among treating physicians and radiologists. A timely and accurate diagnosis of pancreatic duct disruption and disconnection can prevent complications, particularly fistula formation and need of
unnecessary drainage procedures, thereby reducing the morbidity.\textsuperscript{9}

\textbf{Funding}
None.

\textbf{Conflict of Interest}
None declared.

\textbf{References}