



Endovascular Embolization of a Pancreatic Pseudoaneurysm in an Adolescent Girl with Chronic Pancreatitis

Zehra Filiz Karaman¹ , Güven Kahrıman¹ , Derya Altay² , Muhammed Şamil Aydın¹ , Aytaç Doğan³

ABSTRACT

Background: Pancreatic pseudoaneurysm (PSA) is a rare, potentially fatal complication of pancreatitis that is commonly associated with a pseudocyst. There is no clear information on the incidence of PSA in children. Surgery and endovascular techniques are the primary treatment options for PSA.

Case Report: We describe the case of a 16-year-old girl with a pancreatic pseudoaneurysm in association with chronic pancreatitis. The diagnosis was made using ultrasonography and magnetic resonance imaging findings. The pseudoaneurysm was successfully treated by endovascular transcatheter embolization without intra- or post-procedural complications.

Conclusion: Early diagnosis and appropriate management are essential to prevent fatal outcomes because the timing of rupture is unpredictable. Endovascular transcatheter embolization of PSA is an invasive, safe, and effective procedure and should be the first choice in the management of PSA.

Keywords: Pancreatic pseudoaneurysm, pancreatitis, pseudocyst, endovascular embolization

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INTRODUCTION

Pancreatic pseudoaneurysm (PSA) is a potentially fatal complication of pancreatitis, which is encountered in approximately 2%–3% of cases. This complication is rare in children, and there is no clear information on the actual incidence of PSA. Pseudocyst is the most common cause of PSA. The formation of PSA exhibits a greater incidence in chronic pancreatitis (7%–10%) than in acute pancreatitis (1%–6%) (1).

CASE REPORT

A 16-year-old girl was admitted to the emergency department with symptoms of abdominal pain, vomiting, and melena. Physical examination revealed abdominal tenderness. A blood test was performed that revealed a normal lipase level of 72 U/L (reference 28–100 U/L) and a slightly higher-than-normal amylase level of 104 U/L (reference 13–60 U/L). The white blood cell count was 14.8 ($10^3/\mu\text{L}$) [reference 4.8–10.7 ($10^3/\mu\text{L}$)], and the C-reactive protein level was 30 mg/L (reference 0–5 mg/L). Other biochemical parameters showed normal levels.

Ultrasonography (US) revealed increased volume and heterogeneous echogenicity at the head and neck of the pancreas. A pulsatile shapeless vascular structure of 2.5-cm diameter was observed at the neck of the pancreas, which indicated the presence of a pseudoaneurysm. The remaining pancreas was thin with irregular dilatation of the pancreatic duct, indicating chronic pancreatitis. The patient had no known underlying cause for pancreatitis such as cholelithiasis, trauma, or metabolic or autoimmune diseases.

On magnetic resonance imaging (MRI), a 5×4 cm (in transverse plane), mass-like formation was detected involving the head and neck of the pancreas consistent with a pseudocyst (Fig. 1). At the center of this formation, a pseudoaneurysm was detected as a dilated shapeless vascular structure. The relationship between the pseudoaneurysm and the adjacent arteries could not be clearly determined by MRI or US.

The patient was urgently referred for Interventional Radiology for delineating the vascular relationships and for embolization of the pseudoaneurysm. Digital subtraction angiography revealed a pseudoaneurysm originating from the gastroduodenal artery, which was also connected with the superior mesenteric artery branches. The pseudoaneurysm was selectively microcatheterized and embolized using n-butyl cyanoacrylate glue that was available at the time of the procedure (Fig. 2, 3).

Follow-up Doppler US conducted 3 days after the endovascular intervention showed no vascularity within the pseudocyst.

¹Department of Radiology, Erciyes University Faculty of Medicine, Kayseri, Turkey

²Department of Paediatric Gastroenterology, Erciyes University Faculty of Medicine, Kayseri, Turkey

³Department of Radiology, Oltu Public Hospital, Erzurum, Turkey

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Correspondence

Zehra Filiz Karaman,
Erciyes University Faculty
of Medicine, Department of
Radiology, Kayseri, Turkey
Phone: +90 352 207 66 66
e-mail:
dr.fkaraman@gmail.com

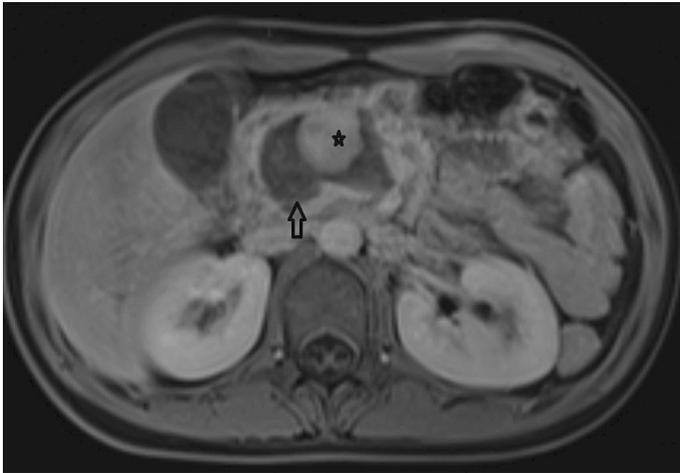


Figure 1. Contrast-enhanced T1-weighted axial MR image: The pseudoaneurysm is filled with contrast (star). The hypointense mass surrounding the pseudoaneurysm is believed to be a pseudocyst (arrow)

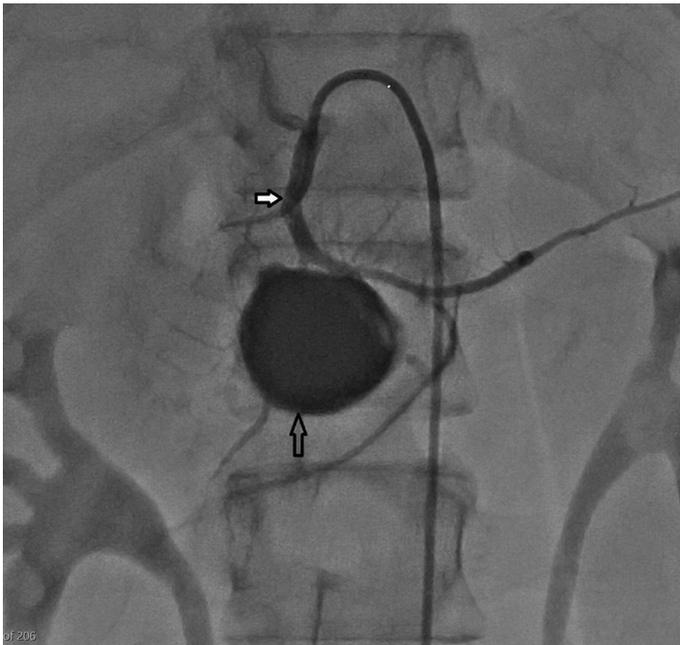


Figure 2. Digital subtraction angiogram: The pseudoaneurysm (open arrow) originating from the gastroduodenal artery (white arrow) is seen before embolization

DISCUSSION

The most commonly accepted pathophysiology involved in the formation of a PSA is arterial wall erosion by exocrine proteolytic enzymes of the pancreas (2). In the setting of pancreatitis, PSA occurs most commonly at the splenic artery. Gastroduodenal, pancreaticoduodenal, and superior mesenteric and common hepatic arteries are the other commonly involved arteries (3).

Color Doppler US reveals a mass of mixed echotexture with vascular flow within it. Color Doppler has the advantages of no exposure to ionizing radiation and the lack of need for contrast medium (4). Moreover, the literature reports that contrast-enhanced US (CEUS) application adds a diagnostic value in providing a real-time

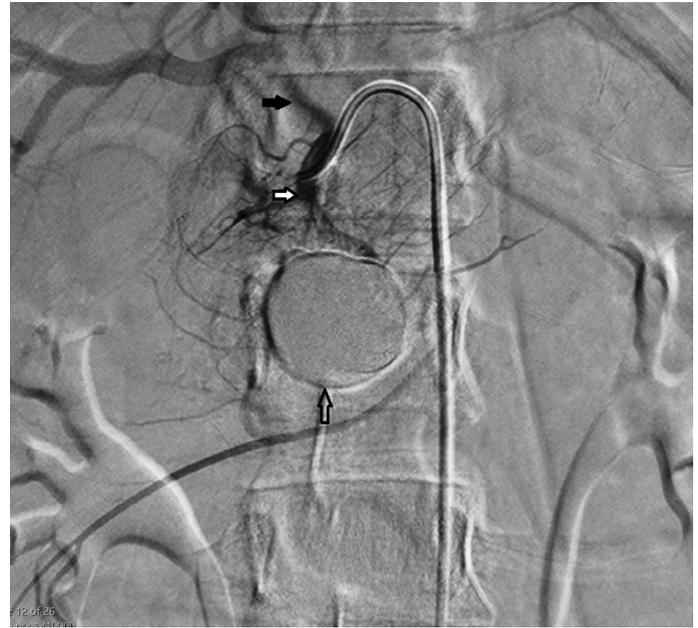


Figure 3. Digital subtraction angiogram after embolization: Cessation of the contrast filling in the PSA (open arrow) after embolization. The gastroduodenal artery (white arrow) and the hepatic proper artery (black arrow) are seen

evaluation of vascular and extravascular findings. CEUS can exclude an active extravasation in the context of a hematoma (5). Computed tomography (CT) angiography is a successful imaging modality for diagnosing and determining the origin of PSA. Digital subtraction angiography can be used to confirm the diagnosis, determine the location of the PSA and collateral vessels, and achieve the endovascular treatment. Moreover, angiography may detect the smaller PSAs that cannot be detected by US and CT (6).

Patients with PSA generally present with abdominal pain and bleeding (2), as in our case. Bleeding is caused by the erosion of the adjacent bowel or direct rupture into the retroperitoneum. A bleeding PSA has a mortality rate of up to 40% depending on the clinical conditions. Irrespective of the presence of symptoms or its size, early diagnosis and appropriate management are essential for preventing fatal outcomes because of the unpredictability of the timing of rupture (6).

Surgery and endovascular techniques are the primary treatment options for PSA. Endovascular treatment has several advantages over surgical repair, which has mortality rates of up to 37% (7). Endovascular therapy allows determining the exact localization of the PSA and evaluating the collateral vessels. In case rebleeding occurs, the procedure can be repeated. This approach represents an important option because of the much less invasiveness of the treatment, as the majority of cases are chronically ill with poor general condition and comorbidities (8, 9).

Balachandra et al. (3). reported that patients undergoing surgery have significantly higher mortality rates than those undergoing transcatheter arterial embolization (surgery alone has a mortality rate of 32%, and embolization alone has a mortality rate of 13%), which is a powerful tool for the control of hemorrhage, thereby avoiding the need for urgent high-risk surgery.

Rebleeding is a potential problem encountered during endovascular management. Kalva et al. (10) reported a rebleeding rate of 17% (4 in 23 patients) within 30 days after embolization. The overall mortality rate within 30 days was 9% in their study.

Hematoma formation, dissection, embolism, end vessel infarction, and coil migration are the primary complications of endovascular treatment (2). In our case, we preferred using n-butyl cyanoacrylate glue for embolization because of its availability. Metallic coils or stents, covered stents, detachable balloons, or gel foam are the other materials used for endovascular treatment (9). No rebleeding or other complications were encountered in the endovascular treatment or in the follow-up of our case.

In conclusion, early diagnosis and appropriate management of PSA are essential for preventing fatal outcomes. Endovascular transcatheter embolization of PSA is a less invasive, safe, effective, and repeatable procedure with a high success rate and should be considered as the first choice in the management of PSA.

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