Clinical presentation, radiological features, and endoscopic management of mediastinal pseudocysts: experience of a decade

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A pancreatic pseudocyst is a collection of pancreatic secretions enclosed in fibrous tissue layer without a lining of epithelium. The usual location is in the peripancreatic region, with reported atypical locations in the spleen, liver, mediastinum, pelvis, and kidney depending on the path taken by the activated pancreatic enzymes.[1-5] Pseudocysts in these atypical locations present a unique and difficult diagnostic and therapeutic challenge.

Mediastinal pseudocysts are rare and have been described in the literature previously as case reports and small case series. They can occur at any age ranging from 7 months to 73 years.[6] A mediastinal pseudocyst is caused by the rupture of the pancreatic duct posteriorly into the retroperitoneal space with entry of the pancreatic fluid into the mediastinum, usually through the esophageal or aortic hiatus.[6] The pancreatic fluid can also track into the mediastinum through the foramen of Morgagni or the inferior vena cava hiatus or by direct penetration of the diaphragm.[6,7] Mediastinal pseudocysts may rupture into the pleural space producing pleural effusion or may extend further into the neck.[3,6,7] Surgery has been the traditional approach for management of mediastinal pseudocysts.[6] Literature supporting the endoscopic drainage of mediastinal pseudocysts is scant.[3,5,7-9]

We previously reported cases of successful resolution of pancreatic pseudocysts at mediastinal as well as various other atypical locations by endoscopic transpapillary drainage, and these cases are also included here.[2-5,9] In this case study, we describe the clinical and radiological characteristics of mediastinal pseudocysts in 12 patients (the cases of 4 of these patients were published previously as case reports or their data included in a previously published article)[3,5,9] as well as our experience with endoscopic drainage and clinical outcome in these patients.

PATIENTS AND METHODS

We performed a retrospective analysis of patients with mediastinal pseudocysts seen at our institution over the past 10 years. Clinical records were reviewed to identify patient symptoms and imaging findings. All patients were referred to us for endoscopic drainage and were treated by attempted endoscopic transpapillary drainage. All patients were symptomatic, had mediastinal pseudocysts with a well-formed wall, as documented on contrast-enhanced CT (CECT), and had documented persistence of their pseudocyst for 6 weeks or more. We also noted imaging findings if patients underwent magnetic resonance imaging (MRI) and EUS. The latter has been available at our institution for the past 4 years. All the patients with mediastinal pseudocysts seen during that time frame underwent EUS examination with a radial scanning echoendoscope (EG-3670 URK radial echoendoscope; Pentax Inc, Tokyo, Japan) at 7.5 MHz. Patients with pancreatic mass; pregnancy; age younger than 18 years; or the presence of congestive cardiac failure, chronic renal failure, or compromised pulmonary status were excluded. Patients unable to provide informed consent were also excluded. All patients provided procedure informed consent at the time of their endoscopic treatment. More recently, our institutional ethics committee granted approval to conduct a retrospective study of patient records.

Intravenous ciprofloxacin was administered for antibiotic prophylaxis. Endoscopic retrograde pancreatography was performed by using a standard technique with a TJF-145 or TJF-160 side-viewing duodenoscope (Olympus Optical Co Ltd, Tokyo, Japan) with the patient under conscious sedation by using intravenous midazolam. Hyoscine butyl bromide was used to inhibit duodenal peristalsis. Pancreatic duct (PD) disruption was defined by free extravasation of contrast outside the PD system as seen on fluoroscopy after retrograde contrast injection of the main PD or dorsal duct (in patients with pancreatic divisum). PD disruption was defined as complete when the main duct upstream from the site of disruption was not visualized on fluoroscopy and as partial when the main duct was visualized upstream from the site of disruption. After confirm-

Abbreviations: CECT, contrast-enhanced CT; MRI, magnetic resonance imaging; NPD, nasopancreatic drain; PD, pancreatic duct.

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TABLE 1. Profile of 12 patients with mediastinal pseudocysts

Age, y/Sex	Etiology	Acute/chronic	Disruption	NPD/stent	Size, cm	Abdominal pseudocyst	Dysphagia	Resolution (weeks)
28/M*	Idiopathic	Chronic	Single	5F NPD	2	Yes	No	4
50/M*	Alcohol	Chronic	Single	PES only	4	Yes	No	6
26/F*	Gallstones	Acute	Single	5F NPD	3	Yes	No	6
42/M*	Alcohol	Chronic	Single	5F stent	5	Yes	No	4
37/M	Alcohol	Chronic	Single	5F NPD	4	Yes	No	4
52/M	Alcohol	Chronic	Single	5F stent	6	Yes	Yes	4
30/F	Gallstone	Acute	Single	5F NPD	2.5	Yes	No	4
44/M	Alcohol	Chronic	Lost to follow-up	—	8	Yes	Yes	—
21/M	Idiopathic	Chronic	Single	5F stent	3	Yes	No	6
36/M	Alcohol	Chronic	Single	5F NPD	4	Yes	No	5
40/M	Alcohol	Chronic	Single	5F stent	2.5	Yes	No	4
28/M	Alcohol	Acute	Single	5F stent	3	Yes	No	6

NPD, Nasopancreatic drain; *M*, male; *PES*, pancreatic endoscopic sphincterotomy; *F*, female.
*Cases published earlier.

ing the ductal disruption, a 5F nasopancreatic drain (NPD) or stent was placed across the papilla into the PD by advancing it over a 0.025- or 0.035-inch hydrophilic guidewire (Jagwire, Microvasive Endoscopy; Boston Scientific, Natick, Mass). An attempt was made to place the NPD across the area of disruption.

After endoscopic retrograde pancreatography, patients with an NPD were instructed to empty the drainage bag and record the daily drain output. They were advised to seek medical attention if there was no drainage from the NPD over 24 hours or the color of the output changed to bilious, indicating displacement of the NPD into the duodenum. When a blockage was suspected (no recorded output for 24 hours), NPD was flushed with sterile saline solution and flow established by suction by using a disposable syringe. These patients were followed every 2 weeks by (1) clinical re-evaluation, (2) abdominal US (in patients with coexistent abdominal pseudocysts), and (3) a chest radiograph in patients with pleural effusion. CECT was repeated when there was complete clinical recovery along with complete resolution of pseudocysts as demonstrated on US of abdomen. When available, EUS was also repeated to document complete resolution.

Therapeutic success was defined as symptomatic improvement with radiological resolution of all pseudocysts on CECT, and therapeutic failure was defined as the persistence of a pseudocyst at 8 weeks after endoscopic therapy or the need for surgical or radiological intervention. After resolution, the stent/NPD was removed, and a repeat pancreatogram was obtained to document healing of ductal disruption.

RESULTS

Twelve patients with mediastinal pseudocysts (10 male patients, mean age ± SD 36.1 ± 9.8 years, age range 21-52 years) were referred to us for possible endoscopic drainage (Table 1). Nine patients had chronic pancreatitis, and 3 had pseudocysts as sequelae of acute pancreatitis. The majority of the patients (8/12, 67%) had alcoholic pancreatitis. Other etiologies included gallstones (n = 2) and idiopathic (n = 2). One of the patients with idiopathic chronic pancreatitis had complete pancreas divisum. All of the patients had abdominal pain on presentation, and 5 patients (42%) had shortness of breath. Only 2 patients (17%) had dysphagia because of compression of the esophagus by the pseudocyst. Both of these patients with dysphagia had a large mediastinal pseudocyst (6 cm and 8 cm, respectively). The size of mediastinal pseudocysts ranged from 2 to 8 cm (median 4 cm). All patients had a coexistent abdominal pseudocyst, and 11 patients (92%) had pleural effusion.

The mediastinal pseudocysts were well demonstrated on CECT (Fig. 1). MRCP was performed in 6 patients, and ductal communication was noted in 2 of these patients (33%). EUS was performed in 4 patients with good visualization of pseudocysts, and no necrotic debris was seen within the pseudocysts (Figs. 2 and 3). None of these patients received parenteral nutrition or octreotide or somatostatin.

One of the patients with alcohol-induced chronic pancreatitis refused treatment and was lost to follow-up. The