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Sclerosing mesenteritis involving the pancreas: A mimicker of pancreatic cancer

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Abstract

Sclerosing mesenteritis (SM), also known as mesenteric lipodystrophy, rarely involves the parenchyma of the pancreas. When sclerosing mesenteritis does involve the pancreas, it can mimic pancreatic carcinoma both clinically and radiographically with pain, obstructive jaundice, a mass lesion and even the appearance of vascular invasion. We report 6 patients with sclerosing mesenteritis involving the pancreas (mean age 43.2 years, 5 female), and review their clinical presentation, radiographic findings, pathology, and outcome. Five of these 6 patients were originally thought to have a primary pancreatic neoplasm. Initial presenting clinical information was available for each patient: all 6 reported abdominal or epigastric pain, 3 reported weight loss, and 2 reported one or more of the following: back pain, fever, abdominal bloating/distention, nausea with/without vomiting, and anorexia. The lesions formed masses with an infiltrative pattern, and all had three key histologic features: fibrosis, chronic inflammation, and fat necrosis—without a known etiology. The inflammatory infiltrate was composed of a mixture of lymphocytes, plasma cells, and scattered eosinophils.

Of the five patients with post-treatment clinical information available, four had at least a partial response to treatment with steroids, tamoxifen, azathioprine, resection, or a combination of these, and 1 did not respond. A dramatic response to immunosuppressive therapy is illustrated by the case of a 46-year-old woman who presented with the presumptive diagnosis of an unresectable pancreatic cancer.

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Distinguishing sclerosing mesenteritis from pancreatic carcinoma is crucial to appropriate management, as patients with sclerosing mesenteritis may benefit from immunosuppressive therapy.

Keywords

Sclerosing mesenteritis; mesenteric lipodystrophy; retractile mesenteritis; pancreas; pancreatic mass

INTRODUCTION

Carcinoma of the pancreas is one of the deadliest of all of the solid malignancies, with an estimated 42,470 new cases in The United States in 2009 (10). Clinical symptoms of pancreatic adenocarcinoma are often nonspecific, and include epigastric pain, unexplained weight loss, jaundice and loss of appetite. Imaging typically reveals a mass, detected by computerized tomography (CT), magnetic resonance imaging (MRI), and/or endoscopic ultrasound (EUS). Depending on the location of the mass within the pancreas, there may be additional radiologic findings such as pancreatic duct and/or common bile duct dilatation, or arterial or venous encasement.

A number of entities can mimic the symptoms and imaging findings of pancreatic cancer. For example, patients with autoimmune pancreatitis are often misdiagnosed as having a pancreatic cancer given the overlapping imaging findings and clinical presentations (1). Peripancreatic masses such as lymphoma and nodal metastases to the peripancreatic region can mimic tumors of the head of the pancreas as well.

Sclerosing mesenteritis is a rare mass-forming lesion characterized by fibrosis, chronic inflammation, and fat necrosis. While sclerosing mesenteritis most often affects the small bowel mesentery, when it involves the pancreatic parenchyma it can mimic pancreatic carcinoma both clinically and radiologically (3, 6). Sclerosing mesenteritis needs to be distinguished from carcinoma, since treatment and prognosis of these two diseases are vastly different.

We report a well-characterized series of patients with sclerosing mesenteritis involving the pancreatic parenchyma. These cases mimicked a primary pancreatic carcinoma both clinically and radiologically.

METHODS

This study was approved by the Johns Hopkins University Institutional Review Board. Institutional pathology and radiology databases were searched for cases of sclerosing mesenteritis and these cases were then queried further to identify the cases of sclerosing mesenteritis that involved the pancreatic parenchyma. All available gross and microscopic pathology was reviewed, as were all available clinical records. Review of the clinical records included an analysis of presenting signs and symptoms, imaging results, initial clinical diagnosis, treatment and response to treatment. Available computed tomography (CT) scans and reports were reviewed to confirm the involvement of the pancreas and to define the spectrum of radiographic appearances of these lesions.

Histology was evaluated using previously published diagnostic criteria (5). Specifically, three key elements were required for the diagnosis of sclerosing mesenteritis: fibrosis, chronic inflammation, and fat necrosis in the absence of a known etiology. Seventeen cases

were identified on initial review. Two cases were excluded from the study after re-review of the histologic material, as were three patients with a history of abdominal radiation. Five cases were excluded due to lack of well-documented pancreatic involvement. One case was excluded because the histologic diagnosis was not established. This left six cases, four from the case files of The Johns Hopkins Hospital, and two from the Johns Hopkins referral consult service. Statistical data were calculated using Microsoft Office Excel 2007 (Microsoft Corporation, Redmond, WA).

RESULTS

Clinical Features

Of the six patients, 5 were women and 1 was a man (Table 1). The mean age at presentation was 43.2 years (range 16-64 years). Four patients identified themselves as White, one patient was of unknown heritage (this patient was adopted), and one as Hispanic. Information about the patients' initial presentation was available for each of the 6 patients. Each patient specifically reported abdominal/epigastric pain (100%), and one patient reported back pain or pain radiating to the back. Weight loss was reported by three patients and diarrhea was reported by one patient. Fever and nausea with or without vomiting were each reported by two patients. One patient reported fatigue and one sweats. When the duration of symptoms was recorded (N=4), the length of symptoms ranged from 2 months to 1 year. One patient had a past medical history of psoriasis, and another had hypothyroidism. No other significant medical problems were reported in the remaining 4 patients.

The range of differential diagnoses reported at the time of imaging was broad. In five cases (83.3%) the clinical diagnosis included a primary pancreatic neoplasm (including pancreatic cystadenocarcinoma, unspecified pancreatic neoplasm, pancreatic cystadenoma, pancreatic neuroendocrine tumor, pancreatic adenocarcinoma, and intraductal papillary mucinous neoplasm (IPMN)). The other clinical diagnoses included cystic lymphangioma or hemangioma (1 patient), Klatzkin tumor (1 patient), and lymphoma (1 patient).

Histologic Features

By definition, all cases contained three key histologic features: fibrosis, inflammation composed of lymphocytes with scattered eosinophils and plasma cells, and fat necrosis (Figure 1) (5) (17).

All available histology was re-reviewed for each case. Fibrosis was the predominant feature in each case, although the pattern of fibrosis varied. The main fibrotic component in five cases was composed of abundant loosely fibrotic tissue with edema and frequent plump, reactive-appearing fibroblasts. In one case, a hypocellular, dense, eosinophilic fibrosis predominated, with a mature glassy or hyaline appearance.

Lymphocytes comprised the predominant inflammatory cell type in each case examined. Several cases showed scattered patches of eosinophils, especially surrounding medium-sized and large vessels. Two of the 6 cases contained non-caseating granulomata; one of these contained granulomata in lymph nodes exclusively, while the other contained scattered granulomata throughout the mass of fibrous tissue. Special stains for acid-fast bacilli and fungi were performed in each of these cases, and all stains for microorganisms were negative. Granulomata are not typical histologic features of sclerosing mesenteritis, and their presence may represent a response to prior needle biopsies in these patients.

The interface between the lesions and the pancreatic parenchyma varied only slightly among the cases. Most cases showed an infiltrative pattern, with bands of fibrosis dissecting between and surrounding lobules of slightly atrophic appearing nodules of pancreatic

parenchyma. In some areas, wisps of fibrosis entered lobules to surround and separate individual acinar cells. The fibrosis appeared to be more dense directly surrounding the pancreatic lobules. Features of autoimmune pancreatitis, including a duct-centric mixed infiltrate and venulitis were not present in any of the cases.

Treatment and Clinical Follow-up

Follow-up information was available for 5 of the 6 patients, and follow-up ranged from three months to 7 years after diagnosis. Four of the five patients showed at least a partial response to therapy and one of the five patients showed no response (stable disease).

Three of the 6 patients were treated with surgical resection. Both of the two patients treated with surgery only (no concurrent medical interventions) showed response to therapy, with resolution of symptoms between 3 months (Patient 5) and 5 years (Patient 2). Patient 2 had a normal erythrocyte sedimentation rate (ESR) after 4-year follow-up. Carcinoembryonic antigen (CEA) and CA19-9 were within normal ranges for Patient 5 one year after the surgery. Two of the four patients who responded to therapy were treated with medical intervention alone: one with a combination of steroids and tamoxifen (Patient 3), one with high-dose steroids alone (Patient 6). Patient 6 had an elevated ESR (134-135 mm/hour; reference range: 4-25 mm/hour) at presentation, however, after a month-long treatment with high-dose steroids, his ESR dropped to 57 mm/hour. One patient was not treated with either surgical or medical intervention; this patient had persistent stable disease after 7 years (Patient 4). A normal CEA was detected at presentation. A few months after her first presentation, she had elevated C-reactive protein (CRP, 4.0 mg/dl; reference range: 0.0-0.5 mg/dl) and ESR (35 mm/hour). Three year later her CEA level was increased to 7.5 ng/ml (reference range: 0-3.0 ng/ml). Meanwhile, CT imaging revealed an anterior medistinal mass, which was proved by biopsy to be sclerosing medistinitis, a similar disease process to sclerosing mesenteritis. However, since then, her disease has been stable.

The potential benefits of an accurate diagnosis with successful treatment are illustrated by the case of Patient 3 (Table 1). This 46-year-old woman presented to our institution for evaluation of what was previously diagnosed radiographically as a locally advanced, unresectable adenocarcinoma of the head of the pancreas. The patient's family history was negative for malignancy, and her personal history included a remote non-diagnostic pancreatic biopsy with placement of a common bile duct stent. Her CA 19-9 was 80.6 U/ml (reference range: 0.0-30.0 U/ml). 3D CT scans with and without contrast performed at our institution revealed an ill-defined mass in the head of the pancreas with associated intrahepatic and extrahepatic bile duct dilatation. In addition, multiple enlarged lymph nodes were noted in the mesentery as well as free fluid that extended down to the pelvis. Circumferential soft tissue density was present around the celiac axis and superior mesenteric artery. The superior mesenteric vein was noted to be occluded, and there was a focal area of thrombus within the portal vein (Figure 2). Percutaneous and endoscopic biopsies revealed only fibrosis, focal fat necrosis and a mixed inflammatory cell infiltrate. Due to the strength of the clinical and radiologic findings suggesting a pancreatic malignancy, chemotherapy with Gemcitabine and concurrent radiation therapy were given. She received a single infusion of Gemcitabine and one fraction of radiation (1.8 Gy), after which the decision was made to re-evaluate her pancreatic process with an open biopsy. During this surgical procedure, a mass was noted at the base of the mesentery, consistent with the mass identified on CT scan. Multiple excisional biopsies of this mass were performed which again revealed only fibrosis, focal fat necrosis and a mixed inflammatory cell infiltrate. Numerous core biopsies of the firm pancreatic head and 13 biopsies with intraoperative frozen section consultation yielded the same results. The diagnosis of sclerosing mesenteritis was thus solidified.

A course of Prednisone with the addition of Tamoxifen was initiated. Within two months, the patient had complete resolution of her abdominal and back pain. A CT scan performed two months after the initiation of treatment showed a significant decrease in retroperitoneal root stranding and soft tissue density. Six months after treatment, the mass-like process that surrounded the pancreatic head and peripancreatic regions down into the mesentery was no longer apparent (Figure 3). Over a year after presentation at our institution, follow-up CT imaging showed stable disease. Her ESR was also normal. Interestingly, the patient noted on further history taking, an improvement in abdominal pain a year prior with the steroids taken for another indication. She did however require repeat stenting and bile duct dilation via ERCP. Most recently her stents were removed and she has since been asymptomatic (3 months) but continues on tamoxifen treatment.

DISCUSSION

In rare cases, patients believed to have incurable pancreatic cancer, may have sclerosing mesenteritis mimicking pancreatic malignancy. We present 6 cases of sclerosing mesenteritis involving the pancreas and identify a pattern of clinical, radiographic and histologic findings which can suggest the inclusion of the disease in the differential diagnosis.

In the past, sclerosing mesenteritis has also been known as mesenteric lipodystrophy, intestinal lipodystrophy, isolated lipodystrophy, mesenteric panniculitis, retractile mesenteritis, liposclerotic mesenteritis, sclerosing lipogranulomatosis, inflammatory pseudotumor, extrapleural solitary fibrous tumor, and xanthogranulomatous mesenteritis (3-6, 9, 12, 13, 16, 19, 20, 22, 23). Traditionally, the predominant histology dictated the nomenclature used, with cases that showed fibrosis as a predominant feature described as 'sclerosing mesenteritis,' a predominance of inflammation earning a designation of 'mesenteric panniculitis,' and a predominance of fat necrosis referred to as 'mesenteric lipodystrophy' (6). These entities are now believed to represent a spectrum of changes along the same pathologic process (6), and today, the term 'sclerosing mesenteritis' is used to encompass the entire spectrum of tumor-like lesions of the mesentery unified by the presence of fibrosis, chronic inflammation, and fat necrosis (6).

Sclerosing mesenteritis most commonly involves the small bowel mesentery (18). It has also been described in the peripancreatic region, omentum, retroperitoneum, mesocolon, and pelvis (2, 6, 9, 14, 19, 21). Depending where it is located, sclerosing mesenteritis can mimic a neoplasm. For example, if the process occurs in the root of the mesentery, the CT appearance can be identical to carcinoid tumor or lymphoma. Cystic components are also commonly seen, probably a results of lymphatic obstruction or necrosis, and when present these changes can mimic a cystic neoplasm. In these cases, the characteristic CT finding of sclerosing mesenteritis, the "fat ring sign" (the preservation of the fat nearest the mesenteric vessels) and the presence of a tumoral pseudocapsule, can help suggest the correct diagnosis (9, 21).

Sclerosing mesenteritis can also be difficult to diagnose when biopsied. Since the key histologic findings of sclerosing mesenteritis (fibrosis, chronic inflammation, and fat necrosis) can be present to varying degrees in association with other lesions (including neoplastic lesions), it is difficult to establish the diagnosis of sclerosing mesenteritis definitively with limited material, especially in the face of clinical and radiologic findings that are worrisome for a neoplasm. For example, in a retrospective review by Emory *et al*, 12 of 84 cases of sclerosing mesenteritis evaluated had been originally histologically misclassified as sarcomas (6). Recently, Weaver and colleagues have suggested the use of MDM2 immunohistochemistry for differentiating sclerosing mesenteritis from well-

differentiated liposarcoma. They found that a lack of MDM2 immunoexpression essentially rules out an inflammatory well-differentiated liposarcoma (24).

It can be difficult to separate sclerosing mesenteritis from a paucicellular, markedly desmoplastic pancreatic adenocarcinoma, and small biopsies may miss a small carcinoma. For these reasons, multiple biopsies and careful correlation of the histopathology with the patients' clinical findings is recommended.

A literature search revealed only a few reports of sclerosing mesenteritis involving the pancreatic parenchyma (20, 21, 24). Phillips *et al* reported two patients who presented with abdominal pain and weight loss who subsequently underwent resections due to the clinical and radiologic suspicion of a pancreatic malignancy (19). Both were ultimately diagnosed with sclerosing mesenteritis. These two cases highlight a persistent problem in the diagnosis of sclerosing mesenteritis involving the pancreas. The histopathology is often non-diagnostic. In Phillip *et al*'s series, conclusive diagnoses of sclerosing mesenteritis were made only after re-review of the pathology after repeated biopsies were deemed inconclusive.

When sclerosing mesenteritis involves the pancreas it can clinically closely mimic a pancreatic malignancy. All 6 patients in our series of patients with sclerosing mesenteritis involving the pancreas reported epigastric pain, with or without radiation to the back, and 3 reported unexplained weight loss: symptoms that are typical of ductal carcinoma of the pancreas (the most common type of pancreatic neoplasm). Despite the clinical similarities, the treatment of sclerosing mesenteritis differs greatly from that of pancreatic neoplasms.

There is no current consensus as to the appropriate treatment for patients with sclerosing mesenteritis, and many clinicians develop a therapeutic approach based on a patient's clinical scenario, with a frequent focus on the inflammatory aspects of the disease process (8, 11, 13, 15, 20). Some investigators report spontaneous resolution of disease, and others describe resection as curative (and laparotomy is clearly indicated in the case of sclerosing mesenteritis-associated nonresolving bowel obstruction) (5, 13, 15, 18, 22). In the absence of spontaneous resolution, several different therapeutic approaches have found some measure of success, although no therapies are uniformly efficacious (21). Some investigators report a response with a combination of corticosteroids and colchicine (7). In a study of 92 patients (including 23 prospective evaluations) by Akram *et al*, 44 (48%) of patients received some treatment (3). Medical therapy was given to 32 patients, including tamoxifen, prednisone, budesonide, colchicines, azathioprine, methotrexate, and thalidomide (3). Of these 32 patients, 13 (41%) showed improvement (assessed by both symptoms and computed tomography findings), 11 (34%) had persistent symptoms, 6 (19%) had progression of their disease, and 2 (6%) were lost to follow-up (3).

The natural history of sclerosing mesenteritis varies: while the prognosis is usually regarded as favorable, the disease can be debilitating and ultimately fatal (3). Some fortunate patients experience prolonged survival with an asymptomatic course (5, 15). Durst *et al* reported a series of 68 patients who had a 97% recovery rate (5). Emory *et al*, however, reported three deaths in their review of 84 patients (each due to postoperative complications) (6). Several investigators mark an association between sclerosing mesenteritis and malignant lymphoma, reporting up to a 15% rate of developing malignant lymphoma after a sclerosing mesenteritis diagnosis (6, 15). Some have suggested that this relationship stems from the possibility of confusing a lymphoma-associated fibrosis with sclerosing mesenteritis at diagnosis, which is especially dangerous following a fine needle aspiration (as opposed to a core biopsy) or in cases where adequate sampling is difficult (6).

CONCLUSION

Sclerosing mesenteritis provides diagnostic challenges for the clinician, the radiologist, and the pathologist. While sclerosing mesenteritis rarely affects the pancreas, when it does it can mimic a primary pancreatic neoplasm both clinically and radiologically. More than threequarters of the cases we present mimicked a primary pancreatic cancer clinically and radiologically. The correct identification of this lesion is crucial to appropriate patient management. As illustrated by Patient 3 (Table 1), some patients can benefit greatly from immunosuppressive therapy.

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Figure 1.

Sclerosing mesenteritis, fibrotic and inflammatory components. **(A)** Fibrosis with a mixed chronic inflammatory component dissects around a pancreatic lobule with an infiltrating pattern, disrupting lobular architecture. **(B)** In this field, intact pancreatic parenchyma is surrounded by fibrous bands containing a sparse mixed chronic inflammatory cell infiltrate. **(C)** Two cases showed a dense, refractile, hyaline-like fibrosis such as that pictured here. **(D)** This high-power image shows the plump, reactive-appearing fibroblasts with open chromatin and large nuclei in association with lymphocytes, few plasma cells, and scattered eosinophils.

Figure 2.

Computed tomography of Patient 3 before treatment. **(A)** Contrast enhanced axial CT demonstrates a soft tissue mass (white arrow) anterior to the SMA (white arrowhead). A stent (black arrowhead) is present in the dilated common bile duct (CD). **(B)** Contrast enhanced axial CT demonstrates adenopathy (arrows) in the root of the mesentery. **(C)** Coronal volume rendered contrast enhanced CT images shows infiltrating soft tissue (white arrows) in the porta and surrounding the SMA (white arrowhead). There is extension of the soft tissue into the portal confluence (black arrow) and occlusion of the SMV. A stent (black arrowhead) is present in the dilated common bile duct (CD).

Figure 3.

Computed tomography of Patient 3 following treatment. **(A)** Contrast enhanced axial CT demonstrates a marked decrease soft tissue mass (white arrow) anterior to the SMA (white arrowhead). It has almost completely resolved. **(B)** Contrast enhanced axial CT demonstrates resolution of the previously noted adenopathy in the root of the mesentery. **(C)** Coronal volume rendered contrast enhanced CT images almost complete resolution of the previously noted infiltrating soft tissue. There is still narrowing at the portal venous confluence (arrow) compatible with stenosis but no residual mass. A wall stent (black arrowhead) is present in the common duct but the previously noted dilatation has resolved.

Table 1

Clinical Findings

Case	Age, Sex Race/Heritage	Presenting signs/symptoms	Clinical/ Radiographic impression	Treatment/follow-up
1	16 F White	Fever, severe abdominal pain, nausea, and vomiting	Stromal tumor (suggested at intraoperative frozen section evaluation)	• Resection
2	26 F Unknown (adopted)	Painful abdominal cramping, diarrhea	Retroperitoneal sarcoma vs. pancreatic cystadenocarcinoma	<ul style="list-style-type: none"> • Partial resection • Resolution of mass by CT 5 years later
3	46 F Hispanic	40-pound weight loss over several months, abdominal pain, and back pain	Locally advanced unresectable adenocarcinoma of the head of the pancreas	<ul style="list-style-type: none"> • Prednisone and Tamoxifen • Resolution of mass by CT 6 months later • Residual bile duct obstruction requiring repeat stent placement
4	48 F White	Intermittent epigastric pain accompanied by nausea, weight loss of 20 pounds over three months	Cystadenocarcinoma, cystadenoma, lymphoma, or neuroendocrine tumor of the pancreas causing compression of the common bile duct	• Persistence of mass by CT 7 years later, no evidence of new abdominal lesions
5	59 F White	Fatigue, vague abdominal pain, 20 pound weight loss over one year	Intraductal papillary mucinous neoplasm (IPMN) (7.0 cm)	<ul style="list-style-type: none"> • Resection of a 7.0 cm mass • Resolution of symptoms at 3 months
6	64 M White	Two month history of febrile illness, sweats, and painful abdominal mass	Mesenteric panniculitis with/without pancreatitis	<ul style="list-style-type: none"> • Intravenous high-dose steroids • Patient became afebrile within 24 hours • Size of the abdominal mass decreased over the course of days
7	78 M White	<i>Not available</i>	Pancreatic neoplasm	<i>Not available</i>

CT= computerized tomography