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CASE REPORT **OPEN ACCESS**

A CASE REPORT OF UNUSUAL PRESENTATION OF MESENTERIC PANNICULITIS

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ABSTRACT

Mesenteric panniculitis is a rare, benign and chronic fibrosing inflammatory disease that affects the adipose tissue of the mesentery of the small intestine and colon. The specific etiology of the disease is unknown. The diagnosis is suggested by computed tomography and is usually confirmed by surgical biopsies. Treatment is empirical and based on a few selected drugs. Surgical resection is sometimes attempted for definitive therapy, although the surgical approach is often limited. We report a 44-year-old male with a past medical history of hypertension, and alcohol abuse presented with complaints of nausea and abdominal pain. CT scan of the abdomen was done which showed fat stranding around mesentery, representing mesenteric panniculitis.

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INTRODUCTION

Mesenteric panniculitis is an acute benign fibrosing and inflammatory condition that involves the adipose tissue of the mesentery. It was first described by Jura in 1924 as "retractile mesenteritis" and further labeled as "mesenteric panniculitis" by Odgen later in the 1960s. Currently, it has several names: sclerosingmesenteritis, mesenteric lipodystrophy, mesenteric sclerosis, retractile mesenteritis, mesenteric Weber-Christian disease, liposcleroticmesenteritis, lipomatosis and lipogranuloma of the mesentery (Zissin, 2006). It can be categorized according to three pathological changes: chronic nonspecific inflammation, fat necrosis and fibrosis (Emory, 1997). This varied terminology has caused considerable confusion, but the condition can now be evaluated as a single disease with two pathological subgroups. If inflammation and fat necrosis predominate over fibrosis, the condition is known as mesenteric panniculitis, and when fibrosis and retraction predominate, the result is retractile mesenteritis. The overall presence of some degree of fibrosis makes the pathological term sclerosingmesenteritis more accurate in most cases (Vettoretto, 2007).

CASE REPORT

A 44-year-old male patient was admitted to our hospital with a 4day history of abdominal pain, moderate in intensity, which lasted for many hours, and was associated with nausea but not vomiting. He had no change in bowel habits and was passing flatus and stools.

His past medical history included hypertension for 10 years, dyslipidemia. He had no known allergies, no significant family history, and a review of his systems was unremarkable. Upon physical examination, the patient appeared well, in no acute distress and had stable vital signs.

The remainder of the examination was unremarkable, except for moderate tenderness upon superficial and deep palpation of the abdomen (right iliac & left iliac fossa with voluntary guarding. His laboratory profile was normal.

Initial workup revealed laboratory values as mentioned in Table 1.

Table 1. Initial lab values upon presentation

Test	Results	Reference value
Hemoglobin (g/dl)	17.3	13-17
Hematocrit (%)	53.0	39-49
White blood cells (10*3/uL)	7.73	3.60-9.50
Platelets (10*3/uL)	164	150-440
AST (U/L)	23.2	10-50
ALT (U/L)	33.8	6-40
Amylase(U/L)	85.1	28-100
Lipase(U/L)	87	

AST: Aspartate aminotransferase, ALT: Alanine aminotransferase

USG Whole abdomen on day of admission showed central IHBRD with altered echo texture of liver with B/L renal calculi, thickened/Echogenic mesentry (Figure 1).

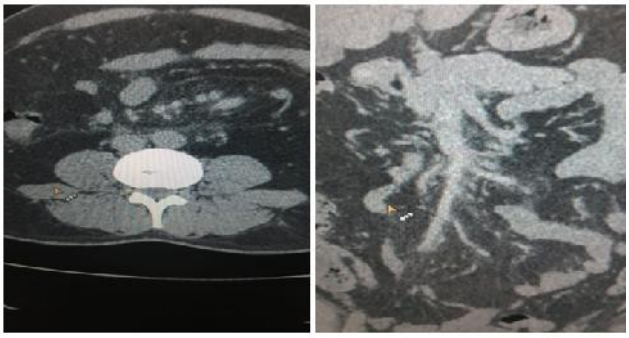
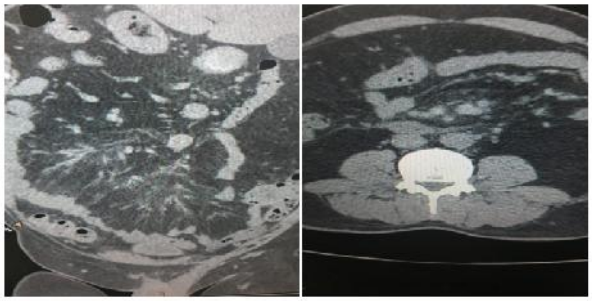


Figure 1. Image from CT scan of the abdomen performed with oral and intravenous contrast

The yellow arrows show multiple clustered nodes of the mesenteric fat representing mesenteric panniculitis.



The patient was started initially on conservative management. He was kept nothing-by-mouth and was started on intravenous fluids, intravenuous antibiotics and intravenous pain medications. On the second day after admission, all laboratory abnormalities were normal. normalized. The patient was still having abdominal pain, persistent nausea. Complete blood count, metabolic panel, and hepatic function remained unremarkable. Soadviced for CT contrast abdomen which revealed mesenteric panniculitis. His symptoms were believed to be secondary to mesenteric panniculitis seen on the CT scan.The patient was started on 40 mg of oral prednisone daily. After starting prednisone, his symptoms completely resolved within 36 hours. He started tolerating diet and was discharged home with a gastroenterology follow-up. He was prescribed prednisone oral taper over a period of two weeks and was discharged.

DISCUSSION

Mesenteric panniculitis is a rare inflammatory condition that is characterized by chronic and nonspecific inflammation of the adipose tissue of the intestinal mesentery. So far, 130 cases have been reported in the literature under several names: retractile mesenteritis, sclerosingmesenteritis, liposcleroticmesenteritis, isolated lipody-strophy of the mesentery, mesenteric lipomatosis, and lipogranuloma of the mesentery, and mesenteric manifestations of Weber-Christian disease (Grieser, 2008 and Gu, 2008). Most studies have indicated that the disease is more common in men, with a male/female ratio of 2-3:1 and several reports have indicated it to be more common in Caucasian men. Incidence increases with age, and pediatric cases are exceptional, probably because children have less mesenteric fat when compared to adults (Delgado, 2007). The pathogenic mechanism of mesenteric panniculitis seems to be a nonspecific response to a wide variety of stimuli. Although various causal factors have been identified, the precise etiology remains unknown. Emory et al (Emory, 1997) have reported a series in which 84% of patients had a history of abdominal trauma or surgery. Furthermore, the disease is related to other factors, such as mesenteric thrombosis, mesenteric arteriopathy, drugs, thermal or chemical injuries, vasculitis, avitaminosis, autoimmune disease, retained suture material, pancreatitis, bile or urine leakage, hypersensitivity reactions, and even bacterial infection (Delgado Plasencia, 2007 and Daskalogiannaki, 2000). Other factors, such as gallstones, coronary disease, cirrhosis, abdominal aortic aneurysm, peptic ulcer, or chylousascitis, have also been linked to this disease (Patel, 1999). More recent studies have shown a strong relationship between tobacco consumption and panniculitis (Daskalogiannaki, 2000). Retractile mesenteritis has been associated with a number of malignant diseases such as lymphoma, lung cancer, melanoma, colon cancer, renal cell cancer, myeloma, gastric carcinoma, chronic lymphocytic leukemia, Hodgkin's disease, large cell lymphoma (giant-cell carcinoma), carcinoid tumor, and thoracic mesothelioma (Emory, 1997; Delgado Plasencia, 2007; Cuff, 1998; McCrystal, 1998 and Shah, 1982). In over 90% of cases, mesenteric panniculitis involves the small-bowel mesentery, although it may sometimes involve the sigmoid mesentery (McCrystal, 1998). On rare occasions, it may involve the mesocolon, peripancreatic region, omentum, retroperitoneum or pelvis (Akram, 2007). The mean clinical progression is usually 6 months, ranging from 2 wk to 16 years. The disease is often asymptomatic. When present, clinical symptoms vary greatly, and may include anorexia, abdominal pain, abdominal fullness, nausea, pyrexia, and weight loss (Shah, 1982). On occasions, the disease may also present with merely a single or multiple palpable masses. Exceptionally, rectal bleeding, jaundice, gastric outlet obstruction, and even acute abdomen have been reported. Such a wide variety of manifestations means that a large number of illnesses must be considered for differential diagnosis; therefore, careful assessment by the treating physician is strongly advised. Histologically, the disease progresses in three stages.

The first stage is mesenteric lipodystrophy, in which a layer of foamy macrophages replaces mesenteric fat. Acute inflammatory signs are minimal or non-existent; the disease tends to be clinically asymptomatic and prognosis is good. In the second stage, termed mesenteric panniculitis, histology reveals an infiltrate made up of plasma cells and a few polymorphonuclear leukocytes, foreign-body giant cells, and foamy macrophages. Most common symptoms include fever, abdominal pain, and malaise. The final stage is retractile mesenteritis, which shows collagen deposition, fibrosis, and inflammation. Collagen deposition leads to scarring and retraction of the mesentery, which in turn, leads to the formation of abdominal masses and obstructive symptoms. The exact diagnosis is often difficult and is usually made by finding one of three major pathological features: fibrosis, chronic inflammation, or fatty infiltration of the mesentery. To some extent, all three components are present in most cases (Seo, 2001). Blood tests tend to be within the normal range. Neutrophilia, increased erythrocyte sedimentation rate or anemia have been reported occasionally in the retractile mesenteritisstage.

Some reports even go as far as stating that few or none of the patients with mesenteric panniculitis can be diagnosed correctly before operation (Grieser, 2008 and Ege, 2002). However, with the advent of imaging technology like high-resolution CT or magnetic resonance imaging, distinguishing mesenteric panniculitis from other mesenteric diseases with similar imaging features such as carcinomatosis, carcinoid tumor, lymphoma, desmoid tumor, and mesenteric edema seems possible and feasible (Pickhardt, 2005 and Horton, 2003). The imaging appearance of mesenteric panniculitis varies depending on the predominant tissue component (fat necrosis, inflammation, or fibrosis) (Koornstra, 1997). It is visualized usually as a heterogeneous mass with a large fat component and interposed linear bands with soft tissue density in cases of mesenteric panniculitis, or as a homogeneous mass of soft tissue density in cases of retractile mesenteritis. Colonoscopy is usually unrevealing, since mesenteric panniculitis is extrinsic to the bowel. Paracentesis that reveals inflammatory cell populations without mitotic figures can also aid diagnosis. Mesenteric panniculitis resolves spontaneously in most cases, however, palpable masses may often be found between 2 and 11 years after diagnosis, especially in patients with associated comorbidity (Delgado Plasencia, 2007). In such cases, several types of treatment have been proposed but no consensus has been established. In general, treatment has been reserved for symptomatic cases. Incidental masses may be observed and left untreated.

Therapy is individualized on a case by case basis. Treatment may be attempted with a variety of drugs including steroids, thalidomide, cyclophosphamide, progesterone, colchicine, azathioprine, tamoxifen, antibiotics and emetine, or radiotherapy, with different degrees of success (Parra-Davila, 1998; Mazure1998 and Miyake, 2003). Surgery may be attempted if medical therapy fails or in the presence of life-threatening complications such as bowel obstruction or perforation (Gu, 2008).

CONCLUSION

In conclusion, mesenteric panniculitis is a rare clinical entity that occurs independently or in association with other disorders. Diagnosis of this nonspecific, benign inflammatory disease is a challenge to gastroenterologists, radiologists, surgeons and pathologists. CT features of the disease, usually highly suggestive, have recently been delineated clearly. Open biopsy seems rarely necessary. There is no standardized treatment, and it may consist of anti-inflammatory or immunosuppressive agents. We recommend resection only when the advanced inflammatory changes become irreversible or in cases of bowel obstruction. Overall prognosis is usually good and recurrence seems to be rare. The purpose of this case was to create more awareness about the condition, as improved understanding can help in the timely and better recognition of this condition and can help develop less invasive diagnostic methods and improve its treatment to prevent future complications.

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