Refractory Sclerosing Mesenteritis Involving the Small Intestinal Mesentery: A Case Report and Literature Review

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Abstract

A 28-year-old Japanese man presented with upper abdominal pain. Computed tomography (CT) revealed a soft tissue mass in the small bowel mesentery. We diagnosed the patient with sclerosing mesenteritis according to the histological findings of small bowel mesentery. Although he was treated with prednisolone, colchicine and azathioprine, neither his symptoms nor CT findings improved. This case is rare in that the disease was refractory. The characteristics of Japanese patients with sclerosing mesenteritis involving small bowel mesentery are not well understood. We herein describe the details of such patients based on a literature review including 32 recently reported Japanese cases.

Key words: sclerosing mesenteritis, mesenteric panniculitis, small bowel, Japanese

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Introduction

Sclerosing mesenteritis is a rare idiopathic disorder that affects the adipose tissue of the mesentery with varying degrees of fat necrosis, inflammation and fibrosis (1). Although the natural history of this disease is not well understood, many patients exhibit a good response to treatment and with a good prognosis. The disease usually involves the small bowel mesentery in Western patients (2), in contrast to the large bowel mesentery in Japanese patients (3, 4). In particular, the sigmoid mesentery is most frequently involved among the large bowel mesentery in Japanese patients (3, 4). Therefore, the major portion of the mesentery involved in sclerosing mesenteritis appears to differ between races. However, the reasons for this difference have not been clarified. The characteristics of Japanese patients with sclerosing mesenteritis involving the small bowel mesentery are

also not well understood.

In this report, we present a rare Japanese case of refractory sclerosing mesenteritis involving the small bowel mesentery. We describe the details of our patient and provide a literature review including recent Japanese case reports in order to compare the various presentations, etiologies, diagnoses and treatments for sclerosing mesenteritis.

Case Report

A 28-year-old Japanese man was admitted to our hospital with a three-month history of upper abdominal pain. The upper abdominal pain had been increasing gradually since onset, and severe pain persisted throughout almost the entire day of admission. The patient exhibited no changes in bowel habits or body weight loss. He had no past history of any diseases, including abdominal surgery or trauma, and no history of medication use. He also had no history of smoking

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Table 1. Laboratory Data on the Admission

WBC	7,300 / μ L	AST	22 IU/L	PT	105.1 %
Seg	81 %	ALT	30 IU/L	APTT	26.6 sec
Eosino	1 %	ALP	198 IU/L	IgG	378 mg/dL
Baso	0 %	TP	5.4 g/dL	IgA	153 mg/dL
Lymph	9 %	Alb	3.4 g/dL	IgM	109 mg/dL
Mono	9 %	BUN	11 mg/dL	IgG4	6.5 mg/dL
RBC	491 $\times 10^4/\mu$ L	Cr	0.9 mg/dL	IgE	85.2 IU/mL
Hb	15.8 g/dL	Na	142 mEq/L	sIL-2R	177 U/mL
Ht	45.4 %	K	3.7 mEq/L	ANA	negative
Plt	26.4 $\times 10^4/\mu$ L	Cl	105 mEq/L		
		CRP	0.1 mg/dL		

WBC: white blood cell, Seg: segmented neutrophils, Eosino: eosinophils, Baso: basophil, Lymph: lymphocytes, Mono: monocytes, RBC: red blood cell, Hb: hemoglobin, Ht: hematocrit, Plt: platelets, AST: aspartate aminotransferase, ALT: alanine aminotransferase, ALP: alkaline phosphatase, TP: total protein, Alb: albumin, BUN: blood urea nitrogen, Cr: creatinine, Na: sodium, K: potassium, Cl: chloride, CRP: C-reactive protein, PT: prothrombin time, APTT: activated partial thromboplastin time, IgG: immunoglobulin G, IgA: immunoglobulin A, IgM: immunoglobulin M, IgE: immunoglobulin E, IgG4: immunoglobulin G4, sIL-2R: soluble interleukin-2 receptor, ANA: anti-nuclear antibody

Figure 1. Abdominal dual-phase computed tomography (CT) revealed a soft tissue mass in the small bowel mesentery (arrows).

or drinking and no family history of autoimmune disease or any form of malignancy.

On admission, the patient was 170 cm tall and weighed 65 kg. His vital signs were stable, with no fever. A physical examination revealed abdominal tenderness, a palpable mass in the upper abdomen and slight pitting edema of the lower extremities.

The laboratory findings (Table 1) indicated low levels of total protein, albumin and IgG. A blood cell count and the C-reactive protein, IgE and IgG4 levels were normal. Anti-nuclear antigens were negative.

Abdominal dual-phase computed tomography (CT) disclosed a soft tissue mass in the small bowel mesentery (Fig. 1). Abdominal magnetic resonance imaging (MRI) also demonstrated a soft tissue mass in the small bowel mesentery, similar to the CT findings. The image of the pancreas was normal on both CT and MRI. Positron emission tomography (PET) combined with CT was negative. Gastro-duodenoscopy and total colonoscopy showed no abnormal findings. Radiography of the small intestine indicated slight edema in the jejunum. Per oral double-balloon enteroscopy (DBE) revealed mucosal edema, white villi, white nodules

Figure 2. Per oral double-balloon enteroscopy (DBE) showed mucosal edema, white villi, white nodules and white debris in the third portion of the duodenum and jejunum.

Figure 3. Microscopic findings of the biopsy specimens obtained from the jejunal mucosa revealed infiltration of chronic inflammatory cells and lymphangiectasia (arrows). Immunohistochemically, there were no IgG4-positive cells in the specimens. (A) Hematoxylin and Eosin staining, $\times 10$, (B) Immunohistochemical staining for D2-40, $\times 10$, (C) Immunohistochemical staining for IgG4, $\times 10$

and white debris in the third portion of the duodenum and jejunum (Fig. 2). A histological examination of the biopsy specimens obtained from the jejunal mucosa demonstrated infiltration of chronic inflammatory cells and lymphangiectasia (Fig. 3A, B). Immunohistochemically, there were no IgG4-positive cells in the specimens (Fig. 3C) and no evidence of malignancy, including carcinoma or lymphoma. Based on these findings, a diagnosis of sclerosing mesenteritis

was highly suspected. In order to obtain pathological confirmation and completely exclude the possibility of malignancy, we performed an open surgical biopsy under general anesthesia. The gross findings of laparotomy included diffuse mesenteric thickening that formed a mass lesion in the jejunal mesentery (Fig. 4A, B). Additionally, the portion of the jejunum affected by the mesenteric mass appeared edematous, and dilated lymphatic vessels were noted on the

Figure 4. (A) and (B): The gross findings during laparotomy revealed diffuse mesenteric thickening that formed a mass lesion in the mesentery of the oral jejunum. (C): The jejunum around the mesenteric mass appeared edematous. Dilated lymphatic vessels were observed on the surface of the jejunal serosa (arrows).

surface of the jejunal serosa (Fig. 4C). A histological examination of the thickened mesentery revealed fat necrosis with foamy macrophages and infiltration of chronic inflammatory cells (Fig. 5A, B). No apparent fibrosis was observed. Immunohistochemically, there were no IgG4-positive cells in the specimens (Fig. 5C). Therefore, we finally diagnosed the patient with sclerosing mesenteritis.

Treatment with 30 mg of prednisolone once daily was started; however, the patient's symptoms (upper abdominal pain and pretibial edema) did not improve. Therefore, the daily dose of prednisolone was gradually reduced every two weeks. A follow-up CT scan performed four weeks after the first administration of prednisolone revealed that the mass lesion in the mesentery had not changed. Although we additionally administered colchicine and azathioprine, the patient exhibited no response to these treatments. A follow-up CT scan performed approximately 1.5 years after symptom onset demonstrated that the mass lesion in the mesentery remained unchanged. The patient now receives follow-up visits and takes pain-relieving drugs.

Discussion

Sclerosing mesenteritis is a rare, idiopathic, benign, chronic fibrosing inflammatory disease that affects the adipose tissue of the mesentery (5, 6).

Since the disease was first reported in 1924, various cases have been reported in the literature under several terms: retractile mesenteritis, sclerosing mesenteritis, mesenteric panniculitis, mesenteric lipodystrophy and so on. In these re-

ports, the patients exhibited various pathological features, including fat necrosis, chronic inflammation and fibrosis. In recent years, these histological variants have been thought to be based on a single pathological entity characterized by nonspecific chronic inflammatory infiltration and fibrosis in mesenteric fat. Adipocyte necrosis (mesenteric lipodystrophy) is thought to progress to a chronic inflammatory state (mesenteric panniculitis) and finally to fibrosis (sclerosing mesenteritis). Therefore, sclerosing mesenteritis may be the best proposed umbrella term (1, 2). In the present case, fat necrosis and infiltration of chronic inflammatory cells were observed in the histological findings; however, no apparent fibrosis was detected. Therefore, in the narrow sense, the strict histological diagnosis was mesenteric panniculitis. However, as mentioned above, "sclerosing mesenteritis" is thought to be the best diagnosis to represent the patient's condition as an umbrella term.

Most studies from Western countries have indicated that sclerosing mesenteritis is more common in men than women, on the order of 2:1. Most patients are in the fifth to seventh decades of life, with a median age of 65 years (2). Pediatric cases are rare because children have lower amounts of mesenteric fat than adults (7). In Western patients, the disease usually involves the small bowel mesentery. Although some patients with involvement of the sigmoid mesentery have been reported (1, 8-10), such cases are very rare. On the other hand, in Japanese patients, the disease often involves the large bowel mesentery (3, 4). Mizutani et al. examined 109 Japanese patients and reported that only 36% had small bowel mesentery involvement, while 58%

Figure 5. The microscopic findings showed fat necrosis with foamy macrophages (A) and chronic inflammation (B). Immunohistochemically, there were no IgG4-positive cells (C). (A) Hematoxylin and Eosin (H&E) staining, $\times 2$, (B) H&E staining, $\times 10$, (C) Immunohistochemical staining for IgG4, $\times 10$

had large bowel mesentery involvement (11). The authors also found that the sigmoid mesentery was most frequently involved among the large bowel mesentery (11). Therefore, the portion of the mesentery most frequently involved in sclerosing mesenteritis appears to differ between races. Although some genetic factors may potentially be involved, the precise reasons underlying these differences have not been clarified.

The characteristics of Japanese patients with sclerosing mesenteritis involving the small bowel mesentery are not well understood. In order to elucidate the background factors and clinical courses of such patients, we reviewed recent Japanese cases. We conducted a comprehensive search of MEDLINE[®]/PubMed[®] and ICHUSHI[®] from 2000 to July 2013 using the term “sclerosing mesenteritis” and extracted 32 case reports of Japanese patients with sclerosing mesenteritis involving the small bowel mesentery. The demographic and clinical characteristics of the 33 patients, including the present patient, are summarized in Table 2. The average age at diagnosis was 61 ± 16.3 years (mean $\pm$ S.D.), and 25 of the 33 patients (76%) were men. Seven patients contracted the disease after abdominal surgery, while four patients also had chylous ascites and one patient also had protein-losing enteropathy (12). Although the evidence was insufficient for verification, our patient was also presumed to have, with a relatively high probability, protein-losing enteropathy, according to the serologic, endoscopic and histo-

logical findings. In addition, one patient had IgG4-related sclerosing mesenteritis (13). As for the diagnostic method used in each case, 12 patients were diagnosed using CT scans only, 11 patients were diagnosed using open surgical biopsies and 10 patients were diagnosed based on the pathological findings of the surgically removed involved mesentery. As for treatment, 21 of the 33 cases were managed with medical treatment only (Table 3). In the medical treatment group, steroids were administered in 12 of the 21 cases, and only one of these patients (our patient) was resistant to steroid therapy. Twelve of the 33 patients were treated surgically (Table 4). In four of these 12 cases, surgery was performed in order to make a histological diagnosis. In these four cases, it was necessary to rule out the presence of malignant tumors histologically because the involved mesentery formed mass lesions. Three cases underwent surgery due to the presence of ileus, three patients underwent surgery due to resistance to conservative therapy and two patients underwent surgery to control acute symptoms. As for the operative procedures, resection of the small intestine was performed in nine of the 12 surgical interventions; the operation was performed successfully in 11 cases. The clinical courses of the 11 successfully treated patients were good. However, intended resection of the involved lesion was impossible in one case, and the patient was reported to have died after the surgery. Overall, in 30 of the 33 cases (91%), the clinical course after medical treatment

Table 2. Summary of Recent Japanese Cases with Sclerosing Mesenteritis Involving Small Bowel Mesentery

Case No.	Year	Reference	Age	Sex	Diagnostic Method	Main Treatment	Other Information
1	2000	[41]	48	M	open biopsy	gamma globulin	acute onset
2	2001	[42]	53	M	CT	conservative	
3	2001		50	M	CT	steroid	post-operated case
4	2002	[43]	63	M	open biopsy	duodeno-jejunostomy	
5	2002		31	F	CT	conservative	
6	2003	[31]	65	M	CT	steroid	post-operated case
7	2003	[44]	33	M	open biopsy	steroid	
8	2003	[45]	89	M	open biopsy	steroid	
9	2003		41	F	open biopsy	conservative	
10	2003	[11]	73	M	resection of the involved mesentery	partial resection of the small bowel	
11	2004		69	M	CT	steroid	post-operated case
12	2004		67	F	CT	conservative	
13	2004	[46]	77	F	open biopsy	steroid	accompanied by chylous ascites
14	2005		72	M	open biopsy	jejunal-transverse colostomy, steroid	post-operated case
15	2005	[47]	74	F	CT	conservative	
16	2006		79	M	CT	steroid	
17	2006		77	F	resection of the involved mesentery	duodeno-jejunostomy, partial resection of the small bowel	post-operated case
18	2006	[3]	37	M	resection of the involved mesentery	adhesiotomy	
19	2006		62	M	open biopsy	steroid	accompanied by sarcoidosis
20	2007		39	M	open biopsy	gamma globulin	
21	2007	[4]	79	M	CT	steroid	accompanied by chylous ascites
22	2007	[48]	79	F	resection of the involved mesentery	partial resection of the small bowel	
23	2007		56	M	resection of the involved mesentery	ileocectomy	post-operated case
24	2008	[49]	58	M	resection of the involved mesentery	partial resection of the ileum +steroid	post-operated case
25	2008	[50]	75	M	resection of the involved mesentery	partial resection of the ileum	
26	2009	[51]	51	M	CT	conservative	
27	2011	[12]	77	M	CT	conservative	accompanied by protein-losing enteropathy
28	2011	[13]	82	M	resection of the involved mesentery	ileocectomy	IgG4-related sclerosing mesenteritis
29	2011		62	M	resection of the involved mesentery	partial resection of the ileum	
30	2011	[52]	60	M	open biopsy	steroid	accompanied by chylous ascites
31	2012	[53]	59	F	resection of the involved mesentery	partial resection of the jejunum	caused by aberrant pancreas in the mesenterium
32	2012	[54]	52	M	CT	steroid	onset under peritoneal dialysis
33		Our case	28	M	open biopsy	steroid, colchicine and azathioprine	possibly accompanied by protein-losing enteropathy

or surgical intervention was adequate to improve the patient's condition. However, three patients, including ours, were resistant to therapy, two of whom died (not including our patient). According to this review, Japanese patients with sclerosing mesenteritis involving the small bowel mesentery generally have characteristics similar to those of Western patients. The present patient appears to be rare in that the disease was refractory.

The precise etiology of sclerosing mesenteritis remains unknown. However, the disease appears to be caused by a nonspecific response to a wide variety of stimuli, including abdominal surgery or trauma (1, 2, 14), autoimmunity (15-20), malignancy (1, 21, 22), ischemic injury and infection (23). Other factors have also been reported, such as chronic inflammatory conditions, collagen vascular diseases (2) and IgG-4 related disease (24). In the present case, none of the associated conditions described above were identified.

A variety of clinical manifestations of sclerosing mesenteritis have been described, and several asymptomatic cases

have been reported (1, 2, 21). Many of these cases were diagnosed incidentally when the patients underwent radiologic procedures for other indications (21). As for symptomatic cases, the most common presenting manifestations are abdominal pain, nausea, vomiting, altered bowel habits, anorexia, body weight loss and palpable abdominal masses (1, 2, 14). In the present case, both upper abdominal pain and a palpable abdominal mass were observed, and the area of abdominal pain completely accorded with the location of the palpable mass. There were no abnormalities, such as gastroduodenal ulcers, pancreatitis or gallbladder stones, that may have caused abdominal pain. Therefore, the patient's abdominal pain was strongly believed to have been caused by the sclerosing mesenteritis itself. Because the symptoms of sclerosing mesenteritis are nonspecific and varied, it is important to consider the possibility of this condition when examining patients with such symptoms.

Laboratory findings are not crucial for diagnosing sclerosing mesenteritis. Although an increased erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) level may be

Table 3. Summary of the Cases that Received Medical Treatment Only

Case No.	Age	Sex	Medical Treatment	Treatment response
1	48	M	gamma globulin	Responsive
2	53	M	conservative	Responsive
3	50	M	steroid	Responsive
5	31	F	conservative	Responsive
6	65	M	steroid	Responsive
7	33	M	steroid	Responsive
8	89	M	steroid	Responsive
9	41	F	conservative	Responsive
11	69	M	steroid	Responsive
12	67	F	conservative	Responsive
13	77	F	steroid	Responsive
15	74	F	conservative	Responsive
16	79	M	steroid	Responsive
19	62	M	steroid	Responsive
20	39	M	gamma globulin	Responsive
21	79	M	steroid	Responsive
26	51	M	conservative	Responsive
27	77	M	conservative	None (Died)
30	60	M	steroid	Responsive
32	52	M	steroid	Responsive
33	28	M	steroid , colchicine and azathioprine	None (Alive)

The case numbers are same as those in Table 2.

found in a minor proportion of patients, the laboratory findings in many patients are normal. In the present case, low levels of total protein, albumin and IgG were found, and hypoproteinemia likely induced the bilateral pretibial edema observed in our patient. Based on the endoscopic and histological findings of the jejunal mucosa, the above conditions were presumably caused by protein loss from the dilated lymphatic vessels of the jejunum. We speculate that the mesenteric mass disturbed the lymph flow in the ducts, inducing leakage of lymphatic fluid from the dilated lymphatic vessels as a secondary change.

Radiographic findings are essential for diagnostically evaluating sclerosing mesenteritis, with abdominal CT being the most sensitive imaging modality for diagnosis. The CT findings of the disease are varied, depending on the predominant tissue component (inflammatory phase or fibrotic phase) (2, 21). The most common finding is the presence of a soft tissue mass in the mesentery. Mass lesions are visualized as more heterogeneous in cases involving the inflammatory phase and more homogeneous in those involving the fibrotic phase. Calcification and cystic changes may also be seen, likely due to a necrotic process.

Additionally, the following two characteristic appearances have been proposed to be specific findings of sclerosing mesenteritis: 1) a "fat ring sign" and 2) the presence of a "tumoral pseudocapsule" (15, 25-27). The "fat ring sign" represents the preservation of fat nearest the mesenteric vessels (26, 28), while the presence of a "tumoral pseudocapsule" represents a band of soft tissue separating the uninvolved mesentery from the inflamed fat (21, 25). In the present case, a heterogeneous mass was found at the root of the small bowel mesentery and a "tumoral pseudocapsule" was detected, indicating that the patient had the typical CT findings of sclerosing mesenteritis.

The final and definitive diagnosis of sclerosing mesenteritis can be established based on a histologic examination of the mesentery. Histologically, the disease appears to progress in three consecutive stages: from mesenteric lipodystrophy to mesenteric panniculitis to retractile mesenteritis. Therefore, the histologic findings may reveal fat necrosis, chronic inflammation, fibrosis or a combination of any two or all three of these conditions (1, 2, 29). In the present case, a histological examination of the thickened mesentery revealed infiltration of lymphocytes with adipocyte necrosis, indicating that the disease had progressed to the stage of mesenteric lipodystrophy or mesenteric panniculitis without the development of fibrosis.

There is no consensus regarding medical therapy for sclerosing mesenteritis. Asymptomatic cases can be managed without medication. As for symptomatic patients, the treatment is usually empiric and individualized. Some symptomatic patients have been reported to improve following treatment with corticosteroids (2, 30, 31). In addition to corticosteroids, colchicine (32, 33), azathioprine (34, 35), cyclophosphamide (36), thalidomide (37) and tamoxifen (2, 38, 39) have been reported to be beneficial. However, the regimen for managing sclerosing mesenteritis has not been established because the use of the above medications has only been reported in small case series. Performing surgical resection of the lesion is quite difficult due to the presence of associated vascular compromise and the extent of disease. Therefore, surgery should be attempted only in patients with severe complications, such as intestinal obstruction or perforation (40). In the present case, neither the patient's symptoms nor radiographic findings improved after treatment with corticosteroids, colchicine and azathioprine. However, surgical resection was not performed because the small bowel mesentery of the jejunum was shown to be widely involved on the CT scan and gross findings during laparotomy. If we had performed surgical resection of the involved lesion, quite a long segment of the jejunum would have needed to be removed. Such an operation could, with high probability, result in short bowel syndrome. Therefore, we did not perform short bowel resection. Fortunately, the patient's symptoms were relatively mild, and he continues to be observed at our hospital as an outpatient while receiving pain control.

In conclusion, we herein presented a rare Japanese case of refractory sclerosing mesenteritis involving the small bowel mesentery and reviewed the literature, including 32 recent Japanese case reports. According to our review, Japanese patients with sclerosing mesenteritis involving the small bowel mesentery generally have characteristics similar to those of Western patients. The present case appears to be rare in that the disease was refractory and possibly accompanied by protein-losing enteropathy. The etiology of sclerosing mesenteritis is not well understood, nor is the reason why the major portion of the mesentery frequently involved in such patients differs between races. Furthermore, differences between the small bowel mesentery type and large bowel mes-

Table 4. Summary of the Cases Treated Surgically

Case No.	Age	Sex	Main reasons for the operation	Operative procedure	Clinical courses after the operation
4	63	M	To control the acute symptoms	duodeno-jejunostomy	Good
10	73	M	Resistant to the conservative therapy	partial resection of the small bowel	Good
14	72	M	ileus	jejunal-tranverse colostomy	Died
17	77	F	Resistant to the conservative therapy	duodeno-jejunostomy, partial resection of the small bowel	Good
18	37	M	ileus	adhesiotomy	Good
22	79	F	To make a histological diagnosis	partial resection of the small bowel	Good
23	56	M	Resistant to the conservative therapy	ileocectomy	Good
24	58	M	ileus	partial resection of the ileum	Good
25	75	M	To make a histological diagnosis	partial resection of the ileum	Good
28	82	M	To make a histological diagnosis	ileocectomy	Good
29	62	M	To make a histological diagnosis	partial resection of the ileum	Good
31	59	F	To control the acute symptoms	partial resection of the the jejunum	Good

The case numbers are same as those in Table 2.

enter type have not been clarified, and there is no standardized treatment for the disease. In particular, there is little information regarding Japanese cases involving the small bowel mesentery. The accumulation of additional case studies is required to understand this disease.

The authors state that they have no Conflict of Interest (COI).

References

- Emory TS, Monihan JM, Carr NJ, Sobin LH. Sclerosing mesenteritis, mesenteric panniculitis and mesenteric lipodystrophy: a single entity? *Am J Surg Pathol* **21**: 392-398, 1997.
- Akram S, Pardi DS, Schaffner JA, Smyrk TC. Sclerosing mesenteritis: clinical features, treatment, and outcome in ninety-two patients. *Clin Gastroenterol Hepatol* **5**: 589-596, 2007.
- Asada Y, Kanda K, Ozeki K, Tanaka T, Mizuta Y, Kohno S. Two cases of mesenteric panniculitis. *Nihon Shokakibyo Gakkai Zasshi (Journal of Japanese Society of Gastroenterology)* **103**: 1372-1376, 2006 (in Japanese, Abstract in English).
- Nishiya D, Mikami T, Fukuda S, et al. A case of suspected mesenteric panniculitis with a large amount of chylous ascites. *Nihon Shokakibyo Gakkai Zasshi (Journal of Japanese Society of Gastroenterology)* **104**: 1212-1217, 2007 (in Japanese, Abstract in English).
- Nicholson JA, Smith D, Diab M, Scott MH. Mesenteric panniculitis in Merseyside: a case series and a review of the literature. *Ann R Coll Surg Engl* **92**: W31-W34, 2010.
- Zafar AM, Rauf MA, Chawla T, Khanda G. Mesenteric panniculitis with pedal edema in a 33-year-old Pakistani man: a case report and literature review. *J Med Case Rep* **2**: 365, 2008.
- Delgado Plasencia L, Rodriguez Ballester L, Lopez-Tomassetti Fernandez EM, Hernandez Morales A, Carrillo Pallares A, Hernandez Siverio N. Mesenteric panniculitis: experience in our center. *Rev Esp Enferm Dig* **99**: 291-297, 2007.
- Levy AD, Rimola J, Mehrotra AK, Sobin LH. From the archives of the AFIP: benign fibrous tumors and tumorlike lesions of the mesentery: radiologic-pathologic correlation. *Radiographics* **26**: 245-264, 2006.
- Han SY, Koehler RE, Keller FS, Ho KJ, Zornes SL. Retractable mesenteritis involving the colon: pathologic and radiologic correlation (case report). *AJR Am J Roentgenol* **147**: 268-270, 1986.
- Popkharitov AI, Chomov GN. Mesenteric panniculitis of the sigmoid colon: a case report and review of the literature. *J Med Case Rep* **1**: 108, 2007.
- Mizutani K, Konishi J, Hayashida R, Nishimura Y, Yoshimori K, Shirouzu K. A case of mesenteric panniculitis associated with large amounts of chylous ascites. *Nihon Rinsho Gekagakkai Zasshi (J Jpn Surg Assoc)* **64**: 902-906, 2003 (in Japanese, Abstract in English).
- Kida T, Suzuki K, Matsuyama T, et al. Sclerosing mesenteritis presenting as protein-losing enteropathy: a fatal case. *Intern Med* **50**: 2845-2849, 2011.
- Nomura Y, Naito Y, Eriguchi N, et al. A case of IgG4-related sclerosing mesenteritis. *Pathol Res Pract* **207**: 518-521, 2011.
- Durst AL, Freund H, Rosenmann E, Birnbaum D. Mesenteric panniculitis: review of the literature and presentation of cases. *Surgery* **81**: 203-211, 1977.
- Horton KM, Lawler LP, Fishman EK. CT findings in sclerosing mesenteritis (panniculitis): spectrum of disease. *Radiographics* **23**: 1561-1567, 2003.
- Papadaki HA, Kouroumalis EA, Stefanaki K, et al. Retractable mesenteritis presenting as fever of unknown origin and autoimmune haemolytic anaemia. *Digestion* **61**: 145-148, 2000.
- Vidarsson B, Matthiasson P, Agnarsson BA, Onundarson PT. Mesenteric panniculitis presenting with autoimmune haemolytic anaemia. *Acta Haematol* **107**: 35-37, 2002.
- Vernace MA, Bellucci AG, Mossey RT, et al. Minimal change nephropathy associated with sclerosing mesenteritis. *Nephron* **73**: 473-476, 1996.
- Dor AM, Kohler JL, Aubrespy P, Scheiner C, Pizzi M, Lebreuil G. Mesenteric panniculitis, an unusual initial stage of acute lupus erythematosus in a ten-year old girl. *Arch Anat Cytol Pathol* **30**: 121-124, 1982 (in French, Abstract in English).
- Sauvaget F, Piette JC, Galewski N, et al. Relapsing polychondritis and mesenteric panniculitis: apropos of 2 cases. *Rev Med Interne* **14**: 253-256, 1993.
- Daskalogiannaki M, Voloudaki A, Prassopoulos P, et al. CT evaluation of mesenteric panniculitis: prevalence and associated diseases. *AJR Am J Roentgenol* **174**: 427-431, 2000.
- Goh J, Otridge B, Brady H, Breatnach E, Dervan P, MacMathuna P. Aggressive multiple myeloma presenting as mesenteric panniculitis. *Am J Gastroenterol* **96**: 238-241, 2001.
- Issa I, Baydoun H. Mesenteric panniculitis: various presentations and treatment regimens. *World J Gastroenterol* **15**: 3827-3830, 2009.
- Chen TS, Montgomery EA. Are tumefactive lesions classified as sclerosing mesenteritis a subset of IgG4-related sclerosing disorders? *J Clin Pathol* **61**: 1093-1097, 2008.
- Sabate JM, Torrubia S, Maideu J, Franquet T, Monill JM, Perez C. Sclerosing mesenteritis: imaging findings in 17 patients. *AJR Am J Roentgenol* **172**: 625-629, 1999.

26. Valls C. Fat-ring sign in sclerosing mesenteritis. *AJR Am J Roentgenol* **174**: 259-260, 2000.
27. Okino Y, Kiyosue H, Mori H, et al. Root of the small-bowel mesentery: correlative anatomy and CT features of pathologic conditions. *Radiographics* **21**: 1475-1490, 2001.
28. Wat SY, Harish S, Winterbottom A, Choudhary AK, Freeman AH. The CT appearances of sclerosing mesenteritis and associated diseases. *Clin Radiol* **61**: 652-658, 2006.
29. Vettoretto N, Diana DR, Poiatti R, Matteucci A, Chioda C, Giovannetti M. Occasional finding of mesenteric lipodystrophy during laparoscopy: a difficult diagnosis. *World J Gastroenterol* **13**: 5394-5396, 2007.
30. Kikiros CS, Edis AJ. Mesenteric panniculitis resulting in bowel obstruction: response to steroids. *Aust N Z J Surg* **59**: 287-290, 1989.
31. Miyake H, Sano T, Kamiya J, et al. Successful steroid therapy for postoperative mesenteric panniculitis. *Surgery* **133**: 118-119, 2003.
32. Iwanicki-Caron I, Savoye G, Legros JR, Savoye-Collet C, Herve S, Lerebours E. Successful management of symptoms of steroid-dependent mesenteric panniculitis with colchicine. *Dig Dis Sci* **51**: 1245-1249, 2006.
33. Genereau T, Bellin MF, Wechsler B, et al. Demonstration of efficacy of combining corticosteroids and colchicine in two patients with idiopathic sclerosing mesenteritis. *Dig Dis Sci* **41**: 684-688, 1996.
34. Tytgat GN, Roozendaal K, Winter W, Esseveld MR. Successful treatment of a patient with retractile mesenteritis with prednisone and azathioprine. *Gastroenterology* **79**: 352-356, 1980.
35. Bala A, Coderre SP, Johnson DR, Nayak V. Treatment of sclerosing mesenteritis with corticosteroids and azathioprine. *Can J Gastroenterol* **15**: 533-535, 2001.
36. Bush RW, Hammar SP Jr, Rudolph RH. Sclerosing mesenteritis. Response to cyclophosphamide. *Arch Intern Med* **146**: 503-505, 1986.
37. Ginsburg PM, Ehrenpreis ED. A pilot study of thalidomide for patients with symptomatic mesenteric panniculitis. *Aliment Pharmacol Ther* **16**: 2115-2122, 2002.
38. Mazure R, Fernandez Marty P, Niveloni S, et al. Successful treatment of retractile mesenteritis with oral progesterone. *Gastroenterology* **114**: 1313-1317, 1998.
39. Venkataramani A, Behling CA, Lyche KD. Sclerosing mesenteritis: an unusual cause of abdominal pain in an HIV-positive patient. *Am J Gastroenterol* **92**: 1059-1060, 1997.
40. Parra-Davila E, McKenney MG, Sleeman D, et al. Mesenteric panniculitis: case report and literature review. *Am Surg* **64**: 768-771, 1998.
41. Kato N, Suzuki H, Tanaka J, et al. A case of mesenteric panniculitis with the onset of acute peritonitis, successful treatment with immunoglobulin. *Nihon Shokakigeka Gakkai Zasshi (Jpn Gastroenterol Surg)* **33**: 1525-1528, 2000 (in Japanese, Abstract in English).
42. Hyodo T, Kouno Y, Shibamoto K, et al. A case of mesenteric panniculitis that CT diagnosis was useful. *Rinsho Hoshasen (Jpn J Clin Radiol)* **46**: 825-829, 2001 (in Japanese, Abstract in English).
43. Hiraguri M, Koike S, Kubota K, et al. A case of gastric cancer occurred obstruction of the afferent loop after a gastrectomy with mesenteric panniculitis. *Nihon Rinsho Gekagakkai Zasshi (J Jpn Surg Assoc)* **63**: 2449-2452, 2002 (in Japanese, Abstract in English).
44. Mizunuma K, Haruta Y, Shinhara R, et al. Low-dose steroid medication is effective for mesenteric panniculitis: A case report. *Nihon Shokakigeka Gakkai Zasshi (Jpn Gastroenterol Surg)* **36**: 1598-1602, 2003 (in Japanese, Abstract in English).
45. Uji Y, Nakae A, Mstui S, Shimode Y, Akami T, Tanaka T. A case of mesenteric panniculitis of the small intestine. *Nihon Rinsho Gekagakkai Zasshi (J Jpn Surg Assoc)* **64**: 2486-2489, 2003 (in Japanese, Abstract in English).
46. Yoshimoto Y, Shimizu R, Saeki T, et al. A case of recurrent mesenteric panniculitis with large amount of chylous ascites. *Nihon Shokakigeka Gakkai Zasshi (Jpn Gastroenterol Surg)* **37**: 697-701, 2004 (in Japanese, Abstract in English).
47. Hagiwara K, Hashizume M, Tsunoda H, Yoshida N, Kawaguchi T. A case of mesenteric panniculitis of the jejunum diagnosed and improved without operation. *Nihon Rinsho Gekagakkai Zasshi (J Jpn Surg Assoc)* **66**: 1194-1198, 2005 (in Japanese, Abstract in English).
48. Shimoji K, Oshiro J, Kinjo O, Kuniyoshi Y. A case of mesenteric panniculitis. *Nihon Rinsho Gekagakkai Zasshi (J Jpn Surg Assoc)* **68**: 452-456, 2007 (in Japanese, Abstract in English).
49. Ueda S, Minari Y, Ienaga T. Intestinal obstruction caused by severe omento-mesenteric panniculitis following rectal cancer surgery. *Nihon Rinsho Gekagakkai Zasshi (J Jpn Surg Assoc)* **69**: 2413-2417, 2008 (in Japanese, Abstract in English).
50. Kojima Y, Matsumoto H, Mikami Y, et al. A case of preoperatively undiagnosed retractile mesenteritis. *Nihon Shokakigeka Gakkai Zasshi (Jpn Gastroenterol Surg)* **41**: 318-323, 2008 (in Japanese, Abstract in English).
51. Shimoji K. A conservative case of mesenteric panniculitis. *Nihon Hukubu Kyukyu Igakukai Zasshi (J Abdom Emerg Med)* **29**: 763-765, 2009 (in Japanese, Abstract in English).
52. Kubo S, Idani H, Asami S, et al. A case of mesenteric panniculitis with massive chylous ascites. *Nihon Rinsho Gekagakkai Zasshi (J Jpn Surg Assoc)* **72**: 801-805, 2011 (in Japanese, Abstract in English).
53. Iwasita S, Igarashi T, Matsushima S, et al. Panniculitis and fat necrosis derived from the ectopic pancreas of the jejunal mesentery. *Rinsho Hoshasen (Jpn J Clin Radiol)* **57**: 700-703, 2012 (in Japanese, Abstract in English).
54. Araki T, Murakami M, Sato M, Kanbe K, Tamura K, Miura H. Obstruction of dialysate drainage due to mesenteric panniculitis in a patient on peritoneal dialysis. *Nihon Toseki Igakukai Zasshi (J Jpn Soc Dial Ther)* **45**: 261-266, 2012 (in Japanese, Abstract in English).