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An Autopsy Case of Mesenteric Panniculitis with Massive Pleural Effusions

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Patient: Female, 81
Final Diagnosis: Suspicious of mesenteric panniculitis
Symptoms: Dyspnea • edema
Medication: —
Clinical Procedure: Diuretics
Specialty: Gastroenterology and Hepatology

Objective: Rare disease

Background: Mesenteric panniculitis (MP) is an idiopathic chronic inflammatory condition of the mesentery. The main symptoms include abdominal pain, abdominal distention, weight loss, fever, nausea, and vomiting. The patients also present with chylous ascites in 14% of the cases and chylous pleural effusion (CPE) in very rare occasions. Despite the previous view of excellent prognosis of MP, two recent papers reported several fatal cases. However, there are still only a few autopsy case reports that describe the macroscopic and histological details of MP cases.

Case Report: The patient was an 81-year-old Japanese woman. She complained of edema of her lower legs and face, general fatigue, and dyspnea. She was overweight and had type 2 diabetes (T2D). Computerized tomography (CT) demonstrated massive bilateral pleural effusions, with mild pericardial effusion and mild ascites. There was no pulmonary, cardiac or hepatic condition to explain the effusions. However, MP was suspected based on her CT. She gradually deteriorated into respiratory failure. The autopsy revealed CPEs (left 1,300 mL, right 1,400 mL) and MP in the mesentery of the small intestine. Neither neoplasia nor inflammatory conditions other than MP were detected.

Conclusions: In rare occasions, patients with MP present with CPE or chylothorax. We thought that a possible mechanism of the CPEs was a diaphragmatic defect. We suspected that being overweight and T2D had an etiological relationship with MP in our patient's case. Adipose tissue of the mesentery is the main focus of MP. We believed that MP would be the best umbrella term of the many synonyms.

MeSH Keywords: Autopsy • Mesentery • Obesity • Panniculitis • Pleural Effusion

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Background

Mesenteric panniculitis (MP) is a rare or underdiagnosed inflammatory condition of unknown etiology in the adipose tissue of the mesentery [1–3]. MP or sclerosing mesenteritis is most often used as the umbrella term for many synonyms [4–10].

The main symptoms include abdominal pain (68–78%), nausea and vomiting (6–32%), abdominal distention (9–26%), fever (6–26%), diarrhea (7–25%), and constipation (6–15%) [3,11,12]. Some of the case reports on MP patients have reported no associated symptoms [5,11,12]. In rare occasions, a patient may present with ascites, usually of the chylous type [1,11]. The report of MP with massive ascites is rare [13–18]. The report of MP with massive chylous pleural effusion (CPE) or chylothorax is extremely rare [19,20].

Physical examination, when positive, may reveal abdominal tenderness (24–38%) and mass (15–34%) [3,11,12]. Elevation of C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) is frequently observed, with other abnormalities infrequent on blood tests [11,12]. More than half of the cases of MP were suggestive of the disease on computerized tomography (CT) [11], but a histological diagnosis was required in most of the cases [12].

Following diagnosis of MP, the chief management strategies for the patient are "watchful waiting", medical treatment, and surgical resection [12]. Complications of the disease include bowel obstruction and perforation, ischemia, ileus, peritonitis, sepsis, and therapy-related complications [11,12].

Generally, MP is considered to have an excellent prognosis [3,4]. However, a recent systemic review and the largest case series have reported some cases that had poor prognosis [11,12]. The mortality rate due to complications of MP was reported to be 6.3% [12]. There are only a few autopsy case reports of MP available in the English literature, mainly because the absolute number of fatal cases is very small [1,21–23].

Case Report

An 81-year-old female patient was admitted to our hospital due to a complaint of edema in the lower legs and face, general fatigue, and dyspnea. Her medical history included appendectomy in childhood, right shoulder fracture in her sixties, type 2 diabetes (T2D) for which she had been taking medication for more than ten years.

Upon physical examination, pitting type of edema in the lower extremities and the face was marked. Her body mass index (BMI) was 29.3 kg/m^2 (her height was 150.4 cm and her body weight was 66.3 kg); her body temperature was 37.2°C, blood pressure was 140/62 mm Hg, and pulse rate was 92 beats/minute. Here SpO2 was 92% in room air. The lower eyelids were not anemic and the conjunctivas bulbi were not icteric. She had no peripheral lymphadenopathies. Bilateral rales were audible and there was no heart murmur. The abdomen was flat and soft, and no masses were found.

Results of the blood test are shown in Table 1 and were positive for a mild increase in blood urea nitrogen, creatinine, hemoglobin A1c, and blood glucose, with a mild decrease in total protein and albumin. CRP was within normal range. There were no abnormal findings on tumor markers and autoantibodies examined. The urine test revealed pH of 7.506, urine specific gravity of 1.026, urine protein 3+, occult urine blood ±, negative white blood cell, urine protein to creatinine ratio of 1.18 g/g·Cre, and urine protein excretion in 24 hour collections of 434 mg/day.

The chest x-ray revealed cardiothoracic ratio of 58.7%, decreased translucency of bilateral lower lung fields, and blunting of costophrenic angles. The CT demonstrated massive bilateral pleural effusions, small amount of pericardial effusion and ascites, and "misty mesentery" with lymph node swelling of less than 1 cm in diameter in the small bowel mesentery (Figure 1A, 1B). However, no tumors were found in the visceral organs.

Thoracocentesis was performed and showed a pleural effusion that was yellowish in color and slightly turbid. The pleural culture was negative. The pleural cytology was also negative. The chemical analyses are shown in Table 1. The effusion was considered as exudative because of the high ratio of pleural to serum protein (0.75), and as chylous because of the high triglycerides (167 mg/dL) [24,25].

The electrocardiogram showed no abnormal findings. The ultrasonic cardiography revealed normal left ventricular wall motion, mild thickening of the interventricular septal wall, mild sclerosis of the aortic valve, and mitral annular calcification. The calculated aortic valve area was 1.1 cm^2 and the maximum upstroke of the trans-aortic valve velocity was 3.2 m/second. And mean pressure gradient of the aortic valve was 14 mm Hg.

Treatment with furosemide of 20 mg IV twice daily was initially administered and later treatment with furosemide of 20 mg IV three times daily and tolvaptan of 15 mg orally once daily was initiated to manage her edema and effusions. The effusions were not ameliorated by treatment with diuretics, and her dyspnea persisted. Chest tubes were inserted and 300 mL of effusions were drained every day. Chemical pleurodesis was considered as a treatment against the effusions but it could not be implemented due to an incidence where the trocar dropped

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Table 1. Blood test and pleural effusion analysis.

Blood test		
Na 133 mEq/L	K 4.4 mEq/L	CL 96 mEq/L
BUN 30.0 mg/dL	Cre 1.05	eGFR 38 mL/min
TP 6.0 g/dL	Alb 3.2 g/dL	CRP 0.14 mg/dL
HbA1c 6.4%	BG 170 mg/dL	TG 182 mg/dL
T-C 149 U/mL	LDL-C 14 U/mL	HDL-C 61 U/mL
TSH 3.39 μIU/mL	F-T3 1.78 pg/mL	F-T4 1.32 ng/mL
LDH 220U/L	TB 0.9 mg/dL	AST 25U/L
ALT 14U/L	ALP 161 U/L	γ-GT 22U/L
WBC 7100/mm^3	Neu 65.5%	Ba 0.3%
Eo 0.3%	Ly 26.0%	Mo 7.9%
RBC 391×10^4/mm^3	Hb 12.0 g/dL	Ht 34.7%
Plt 24.3×10^4/mm^3	PT-INR 0.97	PT-% >100
PT 11.8s	APTT 28.8	D-dimer 6.2 μg/mL
Pleural effusion analysis		
SG 1.032	pH 7.7	Protein 4.5 g/dL
Sugar 215 mg/dL	TG 167 mg/dL	HA 2 μg/ml
ADA: 7.6 U/L		

Na – sodium; K – potassium; CL – chloride; BUN – blood urea nitrogen; Cre – creatinine; eGFR – estimated glomerular filtration rate; TP – total protein; Alb – albumin; CRP – c-reactive protein; HbA1c – hemoglobin A1c; BG – blood glucose; TG – triglyceride; T-C – total cholesterol; LDL-C – low density lipoprotein cholesterol; HDL-C – high density lipoprotein cholesterol; TSH – thyroid-stimulating hormone; F-T3 – free triiodothyronine; F-T4 – free thyroxine; LDH – lactate dehydrogenase; TB – total bilirubin; AST – aspartate transaminase; ALT – alanine transaminase; ALP – alkaline phosphatase; γ-GTP – γ-glutamyltransferase; WBC – white blood cell; Neu – neutrophil; Ba – basophil; Eo – eosinophil; Ly – lymphocyte; Mo – monocyte; RBC – red blood cell; Hb – hemoglobin; Ht – hematocrit; Plt – platelet; PT-INR – prothrombin time-international normalized ratio; PT – prothrombin time; APTT – activated partial thromboplastin time; SG – specific gravity; pH – potential of hydrogen; HA – hyaluronic acid; ADA – adenosine deaminase.

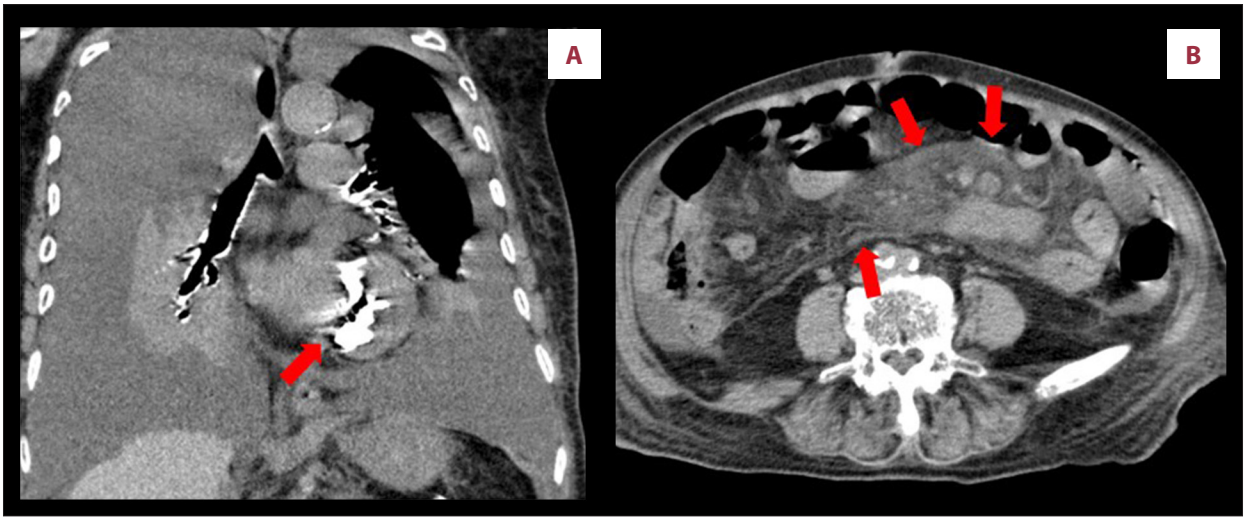

Figure 1. (**A**) Massive bilateral pleural effusions and mitral annular calcification (arrow) on CT. (**B**) Misty mesentery with pseudocapsule (arrows) and sub-centimeter lymph nodes on CT.

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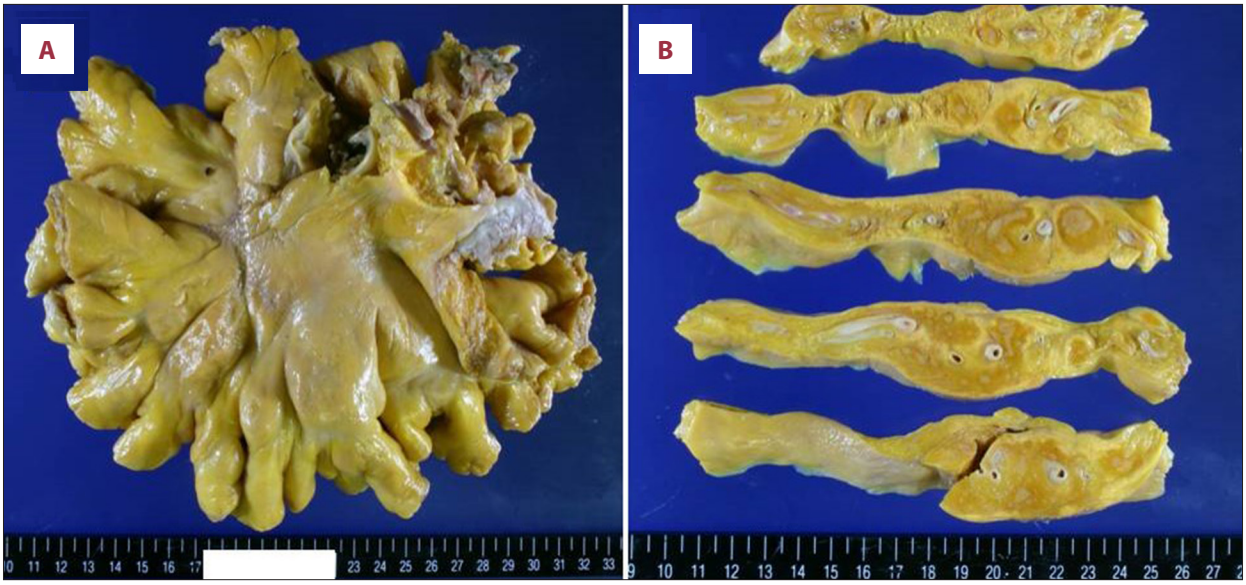

Figure 2. (**A**) A surface view of the mesentery with elastic hard consistency and no obvious findings of peritonitis and metastatic tumors. (**B**) Cut sections of the diffusely thickened mesentery with intermingling of white-yellow and brown-yellow areas.

off the thoracic wall. Despite the persistent effusions, she was discharged 36 days after hospitalization because of her stable physical condition and her preference for discharge.

However, she returned to the hospital three days after discharge due to dyspnea and anorexia. She was treated with 60 mg/day of azosemide, 10,000 IU/day of heparin, and therapeutic thoracocentesis. However, her pleural effusions and dyspnea gradually caused her condition to further deteriorate. She and her family decided to avoid further invasive procedures. She died of respiratory failure probably caused by massive CPEs.

Autopsy findings

Autopsy was performed to confirm the CT diagnosis and to elucidate the pathophysiology of the effusions.

Bilateral, white-yellow, turbid pleural effusions (left 1,300 mL, right 1,400 mL) were found on autopsy. However, pleuritis, pneumonia, and congestive edema were not demonstrated. Both lungs had the same weight of 200 g. There were no detected bilateral diaphragmatic defects. Pericardial effusion of 150 mL, and mild and chronic pericarditis were found. The heart weight was 350 g. Mild hypertrophy of the left ventricle with a wall thickness of 1.5 cm and moderate coronary sclerosis was revealed. No dilatation of the ventricles and no mural thrombosis were found. A calcified nodule 2.2×0.8 cm in size was observed at the mitral annulus. Histology of the heart did not show infarcts or cardiomyopathy.

Ascites of 200 mL was observed and peritonitis was not found. Diffuse thickening of the small bowel mesentery was demonstrated (Figure 2A). The size of the lesion was approximately 12×11×2.5 cm. There was focal and mild thickening of the peritoneum in the mesentery. No adhesion between the small bowel and the mesentery was found. The cut sections showed that patchy white-yellow areas intermingled with brown-yellow areas surrounding the mildly swollen mesenteric lymph nodes (Figure 2B). Histology of the white-yellow area revealed aggregates of foamy macrophages and moderate fibrosis of no specific pattern, as in storiform fibrosis (Figure 3A, 3B). Histology of the brown-yellow area demonstrated mild-to-moderate panniculitis, with mild-to-moderate infiltration of small lymphocytes and plasma cells, mild-to-moderate degeneration of adipocytes, and mild fibrosis (Figure 3C, 3D). Immunohistochemically, the small lymphocytes consisted of B and T lymphocytes. And IgG4 positive plasma cells focally increased. Neither necrotizing arteritis nor occlusive phlebitis was found. The mesenteric lymph nodes revealed mild reactive hyperplasia with no findings of malignant lymphoma and metastatic tumors.

The liver was atrophic and the weight was 620 g. Nevertheless, there were no findings of hepatitis, fibrosis, or cholestasis. The small and large bowels did not reveal any macroscopic or microscopic abnormalities. The pancreas showed moderate fatty infiltration and the weight was 140 g. Focal panniculitis-like inflammation in the peri-pancreatic adipose tissue were observed.

The weight of the left and the right kidney was 150 g and 120 g, respectively. The finely granular surfaces suggested moderate arteriosclerotic atrophy. The cut sections showed mild atrophy and mild dilatation of the pelvis. The histology demonstrated moderate glomerular sclerosis and hyalinosis of the arterioles,

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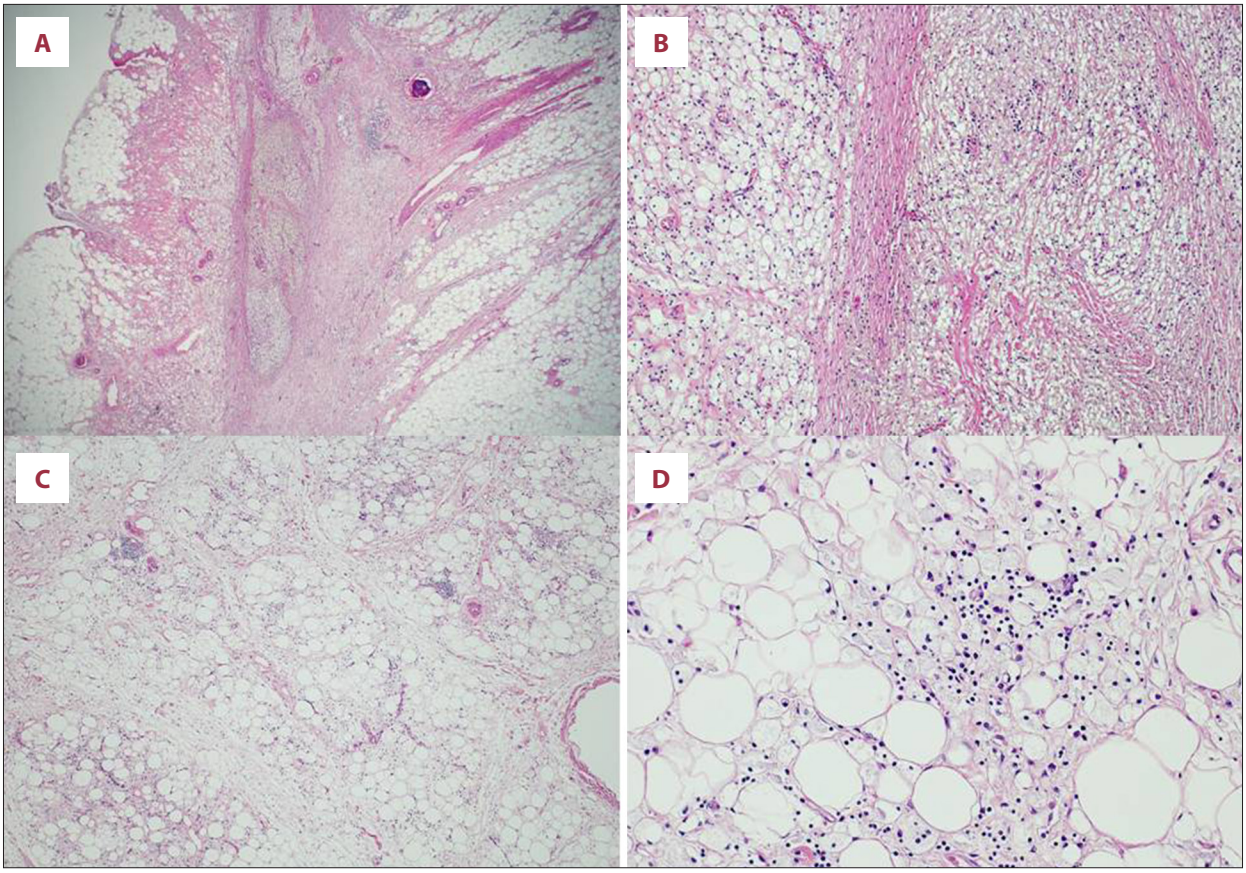

Figure 3. (**A**) A low power microscopic field from the white-yellow area of the mesentery (H&E 20×). (**B**) A magnified field of Figure 3A shows fibrosis and many foamy macrophages (H&E 100×). (**C**) A low power microscopic field from the brown-yellow area (H&E 40×) (**D**) A high power microscopic field from the brown-yellow area shows moderate infiltration of chronic inflammatory cells and degeneration of adipocytes (H&E 200×).

which was consistent with diabetic renal changes. Neither tubulointerstitial nephritis nor retroperitoneal fibrosis was found.

Discussion

MP is a chronic inflammatory process mainly involving fatty tissue of the mesentery. A previous report showed that around 40% of cases had no symptoms [5]. But two recent reports indicated that patients with MP, which was incidentally identified, were 10% of 92 cases and 1.6% of 192 cases, respectively [11,12]. MP is usually diagnosed during the fifth or sixth decade of life and it is twice as common in men as in women. Its main symptoms include abdominal pain, abdominal distention, weight loss, fever, nausea, and vomiting [3,11,12]. The laboratory tests often show elevation of CRP and ESR with infrequency of leukocytosis, anemia, and low level of total serum protein [12]. In more than half of the patients, CT findings were suggestive of MP, but in at least one fourth of cases, the radiologic or gross appearance was indistinguishable from a malignant process, especially lymphoma [11]. In one study, although CT scan was performed in the majority of patients, only 15.1% (n=26) were diagnosed based on the imaging studies; thus, histology remained the gold standard despite invasive testing [12].

Despite previous reports of excellent prognosis of MP [3,4], a systemic review reported that there was a total of 14 deaths (7.3% of 192 cases), 12 of which was secondary to MP-related complications [12]. The largest series reported poor prognosis in 20% of the cases and three deaths attributed to complications of the condition or its treatment [11]. On the other hand, there have been very few autopsy reports from the fatal cases probably due to the scarcity of such cases [1,21–23]. MP was found as an incidental finding at autopsy in one report [11].

The main symptom of our patient was dyspnea, which was caused by massive CPEs. The CT scan suggested MP in the small bowel mesentery. At autopsy, we identified MP in the mesentery. However, there were no specific lesions in any other organs which were likely to lead to the patient's effusions.

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Regarding ascites in MP, two English papers reported chylous ascites, the frequency of which was 14% (13/92) and 18.5% (5/27), respectively [1,11]. Although the amount was usually small [1], massive chylous ascites was reported in several cases, most of which were Japanese [13–18]. Mechanism of the chylous ascites was believed to be direct mechanical compression by the mesenteric mass encasing the bowel, blood vessels, and lymphatics that resulted in abdominal pain, bowel obstruction, ischemia, and chylous ascites [11]. Therefore, production of the ascites may be differently influenced by three types of mesenteric lesions: a single mass, multiple masses, and diffuse thickening [4,5]. MP is more frequent in the large bowel (64%) than in the small bowel mesentery (36%) in Japan [26]. And yet it is more common in the small bowel (69%) than in the large bowel mesentery (22.6%) in the Unites States [4]. The small bowel mesentery was involved in all the cases with massive chylous ascites [13–18]. Other types of ascites seem to be very rare without the complications, including bowel perforation, ischemic change, cardiogenic shock, and secondary infection [22,23,27–29].

Generally, CPE develops in a wide variety of conditions that bring about disruption of the large intrathoracic lymphatics due to traumatic injury or obstruction by benign or malignant causes [30]. It also presents itself in intra-abdominal conditions which include hepatitis, cirrhosis, primary sclerosing cholangitis, pancreatitis, malignancy of the bile duct or pancreas, and abdominal surgery [30]. To the best of our knowledge, there have been only two single-case reports of MP with massive CPE [19,20]. CPE was bilateral in one case and right in the other. As with our case, both of these cases had a small amount of ascites. It is hard to explain the exact mechanism of massive fluid accumulation almost exclusively in the pleural cavity in MP, mainly involving the mesenteric adipose tissue. However, it has been reported that massive hydrothorax happens through diaphragmatic defects in some cirrhotic patients (hepatic hydrothorax) [31,32] and in peritoneal dialytic patients with end-stage kidney diseases [33,34]. The negative intrathoracic pressure favors the transfer of fluid across the defect and hence patients usually have mild ascites [32]. The mechanism of the right CPE with MP was presumed to be a diaphragmatic defect [20], and hepatic hydrothorax is usually right-sided (65–87% of reported cases). However, it may be left-sided or bilateral [35]. As the defect can sometimes be microscopic in size [32], we could not identify it clinically and macroscopically at autopsy. Therefore, the mechanism in our case remains to be elucidated.

Etiology of MP is still unknown. A few reports have shown a high association of malignant neoplasia with MP, which has thus been considered a neoplastic syndrome [36–38]. Nevertheless, other articles have suggested that the prevalence of malignancy in MP was not higher than that in the general population [2,12]. We did not observe any neoplasia in our case at autopsy. Patients with MP have been reported to often have a past history of abdominal surgery or trauma [11,12]. Our patient had a history of appendectomy in her childhood. In a recent review, the theory of abnormal post-surgical healing and ischemia to the mesentery, as a source of MP, seemed plausible [12]. However, Emory et al. showed that only four of 84 cases with MP had a history of trauma and surgery [4]. A matched case-control study observed no significant difference in the rate of previous surgery between the two groups [2]. A small number of previous cases with MP shared some features with IgG4-related disease (IgG4-RD), including increase of IgG4-positive plasma cells and multifocal fibrosis [11,39–41]. Histology of our case revealed focal and moderate increase of IgG4-positive plasma cells and did not show occlusive phlebitis or storiform fibrosis. These findings and the absence of multifocal fibrosis were inconsistent with those of IgG4-RD. A recent paper suggested that most cases of MP are more likely to be an IgG4-RD mimic and that IgG4-RD seemingly seldom, if ever, affects this anatomic site [42].

Our patient was overweight and had T2D. Her BMI was recalculated to be 27.9 by subtracting the weight of the effusions from the body weight. In the available articles, only a few reports have put a focus of their studies on the relationship between MP and T2D or obesity. Pereira et al. reported that five cases of T2D were found on CT to have MP [43]. They hypothesized that changes in the mesenteric adipose tissue in inflammatory states (MP) might be similar to T2D because inflammation has been recognized to play a role in pathophysiology of T2D. Unlu et al. noted that people with idiopathic misty mesentery had high BMIs: 67.5% were classified as obese and 17.5% as overweight [44]. Misty mesentery is also considered to be an important CT sign indicating early stage of MP. And they suggested that there might be an association between obesity and misty mesenteric appearance on CT. According to comprehensive reviews, obesity and T2D are associated with low-grade chronic inflammatory processes [45–48]. In addition, human mesenteric adipose tissue is considered as a critical player in insulin resistance of T2D and metabolic syndrome [49]. These evidences may reinforce a close relationship between MP and obesity as well as T2D. However, a matched case-control study failed to demonstrate a significant association between MP and T2D [2]. Further study is needed to establish the association.

Fundamental histology of the mesentery consists of the surface mesothelium, connective lattice, and adipocyte population housed in interstices of the lattice [50]. Based on the definition of the mesentery, the term mesenteritis should properly mean that it is an inflammatory process involving the peritoneum, connective tissue including fibrous tissue, vessels, nerves, and adipose tissue. However, according to four case series studies and two reviews, ascites was not described as a

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major sign except for the small percentage of cases with chylous ascites [1,3–5,11,12]. Ogden et al. reported that they found no instances of adhesions binding of peritoneum-to-peritoneum or peritoneum-to-bowel or to other viscera; the peritoneum may hardly ever be involved in the condition. Phlebitis seldom if ever happens in ordinary cases of MP, but may in those with suspected IgG4-RD [42]. An emerging concept of the possible relationship between MP and obesity and/or T2D suggests that MP may be an inflammation exclusively involving the mesenteric adipose tissue [43,44]. Hence, we believe that mesenteric panniculitis would be the best umbrella term to encompass a spectrum of rare idiopathic diseases characterized by chronic and fibrotic inflammatory changes in adipose tissue of the mesentery.

Conclusions

We reported a rare autopsy case of MP with massive CPEs. We thought a possible mechanism of the fluid accumulation was a diaphragmatic defect. But it remains to be clarified. We could not identify any certain etiology for MP in our patient. It has recently been suggested that obesity and T2D have an association with MP. We suspected our patient's overweight condition and/or T2D as the potential etiology.

The adipose tissue of the mesentery is the main focus of MP. And the other components of the mesentery are rarely involved in MP. We thought that MP would be the best umbrella term for the synonyms in this case report.

Conflict of interest

None.

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