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A case report of mesenteric panniculitis

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Abstract

A 65-year-old man had intermittent abdominal pain for the previous 2 years. This pain suddenly became worse with a fever and elevated inflammatory markers. We took a while to diagnose the patient with mesenteric panniculitis (MP). Although imaging findings suggested MP, we needed to rule out other diseases. Choosing a treatment for the patient also took some time and we finally used glucocorticoid to cure the patient.

Keywords

Mesentery, panniculitis, diagnosis, glucocorticoid treatment, computed tomography, methylprednisolone

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Introduction

Mesenteric panniculitis (MP) is a clinically rare mesenteric chronic inflammatory disease. The cause of MP is unknown and may be due to abdominal surgery, trauma, ischemia, drugs, allergies, or autoimmune disease.[1–4] This disease can occur in any age group, but is more common in 50- to 60-year-olds, and the male to female ratio is 1.5 to 1.8:1.0. Patients with MP have different clinical manifestations and some

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patients with MP may experience no discomfort. A few patients with MP present with an abdominal mass, peritonitis, peritoneal irritation, and ascites.[1–3] Diagnosis of MP is difficult and MP is easy to misdiagnose. Unfortunately, there is still no consistent diagnosis and no treatment guidelines. We experienced a case of MP, which was diagnosed as MP mainly on the basis of computed tomographic (CT) imaging features. We used methylprednisolone to finally cure the patient. We hope that understanding of the clinical manifestations and auxiliary examination of MP are strengthened by this case study. We also report processes for the diagnosis and treatment of MP and review the MP-related literature, especially regarding CT imaging features that contribute to diagnosis and treatment of this disease.

Case presentation

A 65-year-old man had intermittent abdominal pain without an obvious cause for 2 years, and this pain was aggravated for 4 days before admission. The pain was of a moderate degree and tolerable, and the patient experienced with dizziness without fever, nausea, vomiting, or diarrhoea. The patient was treated in a local hospital and diagnosed with acute cholecystitis. The patient experienced abdominal pain again 4 days before admission, and the pain was stronger than previously. The pain was accompanied by occasional vomiting and hiccups, and there was still no fever or diarrhoea. The abdomen was flat and soft. Additionally, the patient had myocardial disease and he underwent cardiac stent implantation 2 years previously. The patient presented with middle and upper abdominal tenderness, suspicious rebound tenderness, and Murphy's sign was negative. We did not detect an abnormal mass in the abdomen on admission. There were no abnormalities in the patient's vital signs. A haematological examination showed the following: white blood cell count, 16.67×10^9/L; neutrophils, 92.6%; neutrophil count, 15.44×10^9/L; urine, pH 5.0; urine specific gravity, > 1.030; urea level, 23.4 mmol/L; creatinine level, 169 μmol/L (blood urea nitrogen/creatine > 40); potassium level, 5.40 mmol/L; C-reactive protein level, 150,000 mg/L; total bilirubin level, 12.4 μmol/L; direct bilirubin level, 9.2 μmol/L; alanine aminotransferase level, 71 U/L; aspartate aminotransferase level, 52 U/L; gamma-glutamyl transferase level, 269/L; alkaline phosphatase level, 137 U/L; prothrombin time, 15.9 seconds; and activated partial thromboplastin time, 35.9 seconds. Myocardial enzymes, blood urease amylase, serum immune markers, and immune function indicators were relatively normal.

An abdominal X-ray showed incomplete intestinal obstruction on the day of admission (Figure 1 a). Three days after admission, chest, abdomen, and pelvic CT and mesenteric arteriovenous vascular CT showed pulmonary infection and mild expansion of the lower common bile duct (possible choledocholithiasis). The part of the mesentery in the middle part of the abdomen was swirled with mesangial thickening, and mesentery and adipose tissue encapsulated mesenteric vessels. Exudation surrounded this mesenteric soft tissue mass in the middle of the abdomen and there were scattered lymph nodes in this area. The pancreas was normal. Prostatic hyperplasia and a small amount of pelvic fluid were observed. There was no thrombosis in the superior mesenteric artery or vein and portal vein (Figure 1 b).

The patient had surgery for coronary heart disease 2 years previously. Combined with the patient's clinical symptoms and medical history, we ruled out a diagnosis of acute cholecystitis and acute pancreatitis. We considered the main diagnosis to be choledocholithiasis, biliary infection, pulmonary

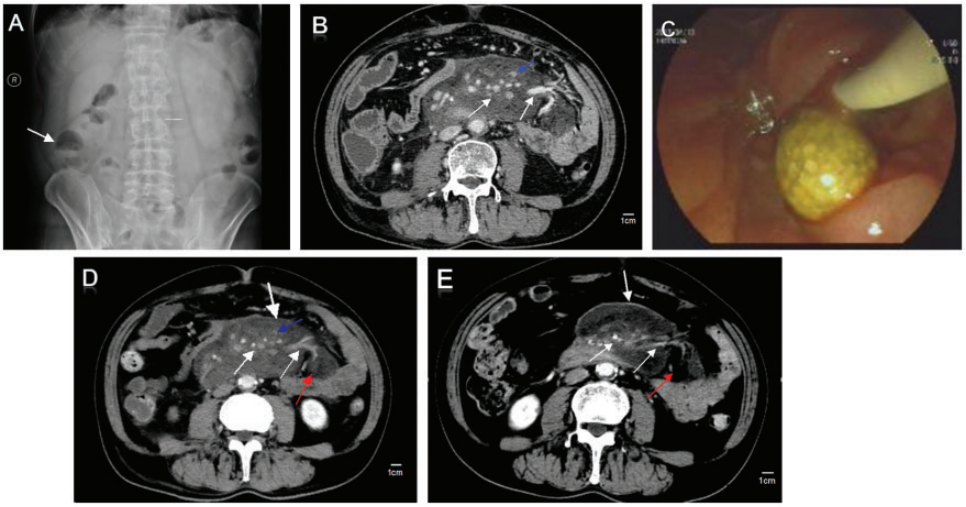

Figure 1. (a) X-ray of the abdomen while the patient was standing. An air-fluid level can be seen (white arrow). (b) Computed tomographic scan of the abdomen performed with intravenous contrast material. Part of the mesentery in the middle part of the abdomen is swirled with mesangial thickening, and mesentery and adipose tissue encapsulate mesenteric vessels. Exudation surrounds the mesenteric soft tissue mass in the middle of the abdomen (red arrow) and there are scattered lymph nodes in this mass (blue arrow). Branches of the superior mesenteric artery and tributaries of the superior mesenteric vein in the mesenteric soft tissue mass can be seen (the cavity of all of these vessels is not narrow) (thin white arrow). (c) Stones in the bile duct were taken out after performing endoscopic retrograde cholangiopancreatography. (d) Computed tomographic scan of the abdomen performed with intravenous contrast material, which was performed 4 weeks after the scan shown in panel B, shows a mesenteric soft tissue mass (11.6 × 6.8 cm). Part of the mesentery in the middle part of the abdomen is swirled with mesangial thickening and increased density of the mesentery. The mesentery and adipose tissue encapsulate mesenteric vessels. The surrounding exudation is more aggravated than previously observed (red arrow). There are scattered lymph nodes in the mesenteric soft tissue mass (blue arrow) and a pseudocapsule has formed around this mass (thick white arrow). Branches of the superior mesenteric artery and tributaries of the superior mesenteric vein in the mesenteric soft tissue mass can be seen (the cavity of all of these vessels is not narrow) (thin white arrow). (e) Computed tomographic scan of the abdomen performed with intravenous contrast material at discharge shows a reduction in size of the mesenteric soft tissue mass (6.8 × 5.0 cm). Exudation of the mesenteric soft tissue mass is greatly reduced (red arrows) and the pseudocapsule still remains (thick white arrow). Branches of the superior mesenteric artery and tributaries of the superior mesenteric vein in the mesenteric soft tissue mass can be seen (the cavity of all of these vessels is not narrow) (thin white arrow)

infection, and incomplete intestinal obstruction. Therefore, we did not allow the patient to eat, but he could drink a small amount of water. He was administered antibiotics, including third-generation cephalosporin, dilatants, and fluid infusions. We provided some medication for vasodilation and relieving coronary constriction to treat the patient's coronary heart disease. He was also treated with antispasmodics, laxatives, and other symptomatic treatments, including low-flow oxygen therapy (oxygen: 2 L/hour, 6–8 hours/day). On the 5th day after admission, two stones in the bile duct were removed after performing endoscopic retrograde cholangiopancreatography (Figure 1 c). His pneumonia greatly improved after administration of antibiotics. However,

the patient's abdominal pain was aggravated intermittently and accompanied by occasional vomiting and hiccups. Fever presented twice at this time, with the highest temperature reaching 38.5°C. Additionally, a physical examination showed that the abdomen had slightly expanded since admission, and the abdomen was slightly tense on palpation. The upper abdomen was tender with rebound tenderness. A CT scan of the abdominal cavity showed that the mesentery in the middle part of the abdomen was swirled with mesangial thickening and increased density of the mesentery. Mesentery and adipose tissue encapsulated mesenteric vessels, and the surrounding exudation was more aggravated than previously. The scattered lymph nodes were still in the mesenteric soft tissue mass and a "pseudocapsule" had formed around this mass.

A diagnosis of MP was then considered on day 9 after admission (Figure 1 d). In collaboration with our rheumatoid specialist, we started glucocorticoid treatment with methylprednisolone. We administered 80 mg of methylprednisolone per day and adjusted the dose to 40 mg after 3 days. His abdominal pain, vomiting, hiccups, and other symptoms were greatly relieved, and his diet and sleep considerably improved after 6 days on methylprednisolone. His blood picture returned to normal, and his liver and kidney function was normal. Abdominal physical signs were normal during a physical examination. We then adjusted the methylprednisolone dose to 20 mg per day and discontinued it after 1 month. An abdominal CT scan was reviewed at discharge 1 month after admission to hospital (Figure 1 e).

We obtained the patient's consent for publication. The study was approved by the Ethical Committee of the Affiliated Hospital of QingHai University on 6 June, 2018 (Grant No: P-SL-2018005).

Discussion

MP is also known as sclerosing mesenteric inflammation, mesenteric lipodystrophy, intestinal fat metabolism disorder, and mesenteric Weber–Christian disease.[1–3,5,6] MP was originally described by Jura in 1927 as a clinically rare mesenteric chronic inflammatory disease. Patients with MP have different clinical manifestations, such as bloating, nausea, vomiting, fever, weight loss, and loss of appetite. The cause of MP is unknown. The association of MP and Henoch–Schönlein purpura and MP concomitant with cancer have been reported.[5,7] In our case, we speculated that the patient's physical weakness and pneumonia were the cause of MP after common causes were ruled out. To date, there is no consistent diagnostic standard for MP. Previous data have shown an increase in inflammatory-related indicators, such as white blood cells, C-reactive protein levels, and the erythrocyte sedimentation rate, in laboratory tests,[4] but specific laboratory test results have not yet been determined. Most patients present with no abnormalities in biochemical tests, urine routines, and stool routines.[1,2,4,8,9]

Contrast-enhanced CT scans appear to be important for diagnosing MP, while MP is usually found by B-ultrasound. B-ultrasound can show mesenteric space occupation, but it cannot identify its nature. CT diagnosis of MP is firmly established and based on five well-recognized pathognomonic features comprising the following: (1) a well-defined mass effect; (2) mesenteric fat tissue of higher inhomogeneous attenuation than that of adjacent intraabdominal fat; (3) small soft tissue nodules; (4) a halo sign; and (5) a pseudocapsule.[1–8,10] Our patient's CT scan of the abdomen, which was performed with intravenous contrast material, showed the following. A mesenteric soft tissue mass (11.6×6.8 cm) was observed. Part of the

mesentery in the middle part of the abdomen was swirled with mesangial thickening and increased density of the mesentery. Mesentery and adipose tissue encapsulated mesenteric vessels. There was surrounding exudation of the mesenteric soft tissue mass in the middle of the abdomen, scattered lymph nodes in this mass, and a pseudocapsule had formed around this mass. Furthermore, branches of the superior mesenteric artery and tributaries of the superior mesenteric vein in the mesenteric soft tissue mass could be seen (the cavity of all of these vessels was not narrow) (Figure 1 d). His presentation matched typical CT findings of MP and MP was highly suspected. Magnetic resonance imaging of MP is not specific, but it is useful for visualizing fat, soft tissue components, and vascular involvement. On the basis of CT scans, the patient was highly suspected of having MP. Magnetic resonance imaging could not be performed because the patient had a stent. However, the patient's imaging findings were not specific for MP. For diagnosis of MP, pathophysiology and specific markers are required. If pathological tissue is available, pathology is the gold standard for diagnosis of this disease, but typical cases can be directly diagnosed without pathological biopsy.[2,8] MP is currently widely believed to be a benign, self-limiting inflammatory lesion, and it has a good prognosis, few recurrences after healing, and few serious complications.[1–6,10] There is no specific clinical treatment for MP. Surgical treatment for MP is not recommended because this disease causes extensive or localized mesenteric inflammatory changes, and locations close to large blood vessels are prone to recurrence after local ablation. Intestinal resection, removal of necrosis, and intestinal adhesion lysis can be considered for the tumour or corresponding lesion and intestinal obstruction, peritonitis, and other serious complications. According to the specific conditions of patients with MP, conservative treatments, such as anti-infective and/or immunosuppressive treatments, glucocorticoids, nonsteroidal anti-inflammatory drugs, colchicine, progesterone, and cyclophosphamide, can be used, but reports on their efficacy are inconsistent.[1–3,5] In our case, the patient was treated with methylprednisolone alone. The symptoms were obviously relieved, which is consistent with most case reports as follows. Coulier et al.[5] reported that steroids resulted in rapid improvement in joint symptoms, abdominal pain, and inflammatory syndrome of a patient who was diagnosed with MP and had an acute gastrointestinal attack of adult-onset Henoch–Schönlein purpura. Sousa et al.[11] reported that they used prednisolone in the first month, and replaced it with azathioprine after 12 months of therapy to cure a 74-year-old woman with MP. However, long-term follow-up of MP is required to observe posttreatment outcomes.[2]

We consider that understanding of clinical manifestations and auxiliary examinations of MP, especially CT imaging features, which can further clarify diagnosis and treatment of this disease, has been improved by our findings. Our findings will hopefully contribute to formation of a consistent diagnosis and treatment guidelines for MP.

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Authorship

MEZ and LQZ were involved in managing the patient and wrote the manuscript. LR, ZWL, XLX, and HJW were involved in managing the patient and follow-up. ZXW, HLL, and YYB processed data and images, and were responsible for the accuracy of the data and images. HNF and CRYD edited and reviewed the manuscript.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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