Reumatologia 2019; 57, 5: 297–300
DOI: https://doi.org/10.5114/reum.2019.89524

Clinical case of mesenteric panniculitis

Iryna Kniazkova, Anna Korchevskaya, Maryna Bogun

Kharkiv National Medical University, Ukraine

Abstract

Mesenteric panniculitis (MP) is a relatively rare pathology characterized by idiopathic local chronic inflammation of adipose tissue, mainly affecting the mesentery of the small intestine. The purpose of the study is to describe a case of MP. A 60-year-old patient visited a rheumatologist due to the progressive deterioration of his condition for the last 4 months and revealed changes on the computed tomography of the abdominal cavity. Treatment protocol: methylprednisolone 8 mg/day, pantoprazole 40 mg/day; continue with antihypertensive drugs. On the background of treatment after 2 months, the temperature became normal, appetite improved, and weakness decreased, with no relapses of abdominal symptoms. Diagnosis of MP is a difficult task, which has to be solved by primary care specialists. This clinical case is an example of MP diagnosis and positive dynamics of the patient's clinical condition during therapy.

Key words: rheumatic diseases, computed tomography, mesenteric panniculitis.

Introduction

Mesenteric panniculitis (MP) is a relatively rare pathology characterized by idiopathic chronic local adipose tissue inflammation, mainly affecting the mesentery of the small intestine [1]. The prevalence of MP in the population ranges from 0.16–0.18% [2], up to 2.4–7.8% [3]; the disease is more common in males (2–3 : 1) [4] and usually occurs at age over 50 years [5]. The present article describes a case of symptomatic MP as a multidisciplinary problem of inflammatory disease.

Case report

A 62-year-old patient was referred to a rheumatologist with complaints such as increasing body temperature up to 37.1–37.3°C, gnawing pains in the epigastric region, worsening at night, weight loss of 15 kg for the last 4 months, lack of appetite, fatigue and general weakness.

From the anamnesis it is known that during the last 4 months the patient suffered from pain in the epigastric region, and lack of appetite occurred without any apparent cause. Due to these complaints esophagogastroduodenoscopy (EGDS) was performed and revealed signs of chronic gastritis and also epithelial cell damage and regeneration with minimal inflammatory changes described as gastropathy. In the histopathological results minimal polymorphonuclear leukocyte infiltration with mononuclear cell infiltration was found. *Helicobacter pylori* was excluded. There were no pathological findings in abdominal ultrasound examination.

The patient had been treated by a gastroenterologist with domperidone 10 mg twice daily and bismuth subcitrate 240 mg twice daily, but this treatment was inefficient and the symptoms did not improve. The patient started to take nimesulide on his own, which positively influenced the abdominal pain. Abdomen and pelvis computed tomography (CT) was performed and revealed signs of MP, so the patient was referred to a rheumatologist.

In the patient's medical history only common colds and childhood infectious diseases were reported. From 2006, arterial hypertension occurred and was treated with ramipril and nebivolol. In 2018, increased glycemic levels were observed and diabetes type 2 was diagnosed. However, in the treatment a diet without any hypoglycemic drugs was sufficient.

According to the patient's information, no previous history of tuberculosis, infections such as virus hepatitis (A, B and C) or any allergic reactions was noted.

Address for correspondence:
Iryna Kniazkova, Kharkiv National Medical University, 4 Nauky Ave, Kharkiv, Kharkiv Oblast, Ukraine, 61000, e-mail: iknyazkova@ukr.net
Submitted: 14.06.2019; **Accepted:** 30.09.2019

The physical examination revealed only pain in the epigastric area during abdominal palpation, temperature 37.0°C. There were no other evident abnormalities in the physical examination of the cardiovascular and musculoskeletal system, lungs or skin assessment.

The cardiac assessment using echocardiography did not reveal any significant disturbances; only due to arterial hypertension left ventricular hypertrophy was found, and the left ventricular ejection rate (LVEF) was 56%. Stool and diuresis were normal. No peripheral edema was observed. A blood test was performed and all results are presented in Table I.

In computed tomography (CT) imaging performed in spiral mode with 1 mm steps, an area of fatty tissue induration was found, enveloping the arterial trunk like a cuff and partially surrounding the upper mesenteric artery without any signs of narrowing of lumens, with approximate dimensions of 36 mm × 16 mm.

Table I. Blood test results

Blood test	Laboratory values
Erythrocytes	5.85×10^{12}/l
Hb	16.8 g/dl
Hematocrit	47.7%
Mean cell volume	81.5 fl
Mean corpuscular hemoglobin concentration	28.7 g/dl
Mean cell hemoglobin	35.2 pg/cell
Platelets	246×10^{9}/l
ESR	7 mm/h
Leukocytes	7.19×10^{9}/l
Eosinophils	3.5%
Basophils	0.5%
Lymphocytes	26%
Monocytes	12%
Glucose	6.45 mmol/l
CRP	15,5 mg/l
Urea	7.6 mmol/l
Creatinine	0.083 µmol/l
Uric acid	2.06 mg/dl
Amylase	48 U/ l
Total protein	68 g/l
Total bilirubin	12.7 µmol/l
Conjugated bilirubin	4.2 µmol/l
Aspartate aminotransferase	40 U/l
Alanine aminotransferase	31 U/l

Hb – hemoglobin, ESR – erythrocyte sedimentation rate, CRP – C-reactive protein

At this level, a single celiac lymph node with a maximum size of 17 mm × 9 mm is observed. The stomach was rather stretched by the fluid, but there were no signs of pathological thickening of the walls. The fat tissue surrounding the loops of the small intestine – visualized in a collapsed state – was not changed. The colon was inflated with gas, the walls of the loops were not thickened, paracolitic fatty tissue was not changed, and nor were the liver and intrahepatic ducts.

The gallbladder is not enlarged in size, with homogeneous contents. X-ray positive calculi were not found, the gallbladder walls were not changed. The common bile duct was not dilated. The portal vein was not dilated. The lymph nodes of the porta hepatis were not swollen. The pancreas was not enlarged, the head 26 mm, body 10 mm, tail 8 mm. The gland structure was homogeneous, and the parenchyma at the level of the body and tail was atrophic. The pancreatic duct is not dilated. Parapancreatic fiber is not changed. The area of the major duodenal papilla is without changes. The spleen is not enlarged, uniform structure, normal density. The adrenal glands are typically located, of uniform structure. The kidneys are typically located, of normal size and shape. The parenchyma of the kidneys is not thinned. Cortico-medullary differentiation of the parenchyma is retained, its contrasting without peculiar properties. The hollow system of the kidneys is not dilated. The abdominal aorta and iliac arteries are atherosclerotically altered. Retroperitoneal lymph nodes are not enlarged. The prostate gland is 36 mm × 44 mm in size, with homogeneous structure, of normal density. Surrounding fiber is not changed. Seminal vesicles are without peculiar properties. The bladder is of normal size and shape. X-ray positive calculi in the lumen are not defined. The walls of the bladder are not thickened; surrounding fiber is not changed. Pelvic lymph nodes and inguinal lymph nodes are not enlarged. Conclusion: CT signs of mesenteric panniculitis.

Taking into account the data listed, the clinical diagnosis of mesenteric panniculitis was established based on the imaging picture and clinical signs. The disease activity was determined taking into account clinical and laboratory parameters. In the clinical case presented, the prevalence of moderately severe clinical symptoms in the clinical picture with the absence of any significant changes in laboratory tests made it possible to determine the activity of the 1st degree.

Due to MP diagnosis therapy with methylprednisolone 8 mg per day and pantoprazole 40 mg/day was administered, and the patient also continued antihypertensive drugs. Rheumatologist, endocrinologist and cardiologist supervision was recommended at the place of the patient's residence.

On the background of conducted therapy after 2 months of treatment the temperature returned to the normal range, appetite improved, and weakness decreased. The main symptom, epigastric pain, disappeared.

Discussion

In most cases the course of MP is asymptomatic. In some cases non-specific symptoms such as abdominal pain, loss of appetite, nausea, flatulence, weight loss, constipation or diarrhea, a palpable mass in the abdominal cavity, and less often rectal bleeding, intestinal obstruction and general symptoms (fever, general weakness), are observed [1, 2]. In addition, clinical findings may not differ from the symptoms of the concomitant disease and can be suggestive for gastric ulcers, inflammatory bowel disease (IBS), or even a neoplastic process. In clinical studies, such comorbidities as arterial hypertension, diabetes mellitus, inflammatory rheumatic diseases and malignant neoplasms may coexist with MP [4, 6].

Mesenteric panniculitis symptoms may also be a link with previous trauma, surgery or infection. It has been noted that progression of MP clinical symptoms ranges from 2 weeks to 16 years after the triggering factor [1].

Pathomorphology

According to the histological changes in the case of MP, there are 3 stages of the disease [7]:

- Stage 1 – mesenteric lipodystrophy, during which a layer of foamy macrophages replaces the adipose tissue of the mesentery. Acute inflammatory signs are minimal or absent; clinical symptoms are absent and the prognosis is favorable.
- Stage 2 – mesenteric panniculitis – an infiltrate is detected, consisting of plasma cells and polymorphonuclear leukocytes, a large number of lipid-laden macrophages present among fat cells. The most common symptoms include fever, abdominal pain and general weakness.
- Stage 3 – the last stage is sclerosing mesenteritis, which is characterized by collagen deposition, fibrosis and inflammation. Collagen deposition leads to cicatrization and mesenteric retraction, which in turn leads to obstructive symptoms.

The diagnosis is made when one of the three main pathological signs has been detected: fibrosis, chronic inflammation or fatty infiltration of the mesentery. In most cases, all three components are present to different extents [1]. The views on the advisability of biopsy of the mesentery of the small intestine are disputed, not only due to the complicated approach, but also due to the development of adverse reactions after this invasive procedure.

Laboratory findings are usually within the normal range. Slight increases of leukocyte count, erythrocyte sedimentation rate, C-reactive protein, and anemia are possible [1].

Diagnostic imaging

Imaging diagnostics such as CT and ultrasonography (USG) play a key role in MP diagnosis and monitoring. Classical radiography examination has no diagnostic value in this case.

Ultrasonography allows one to distinguish unchanged mesenteric fat from the inflamed one; the latter is characterized by homogeneous echogenicity. It is possible to identify additional formations (without clear borders) of various sizes with the structure resembling thickened adipose tissue. The mesentery of the small intestine is "hypertrophied" with the presence of enlarged lymph nodes and dilated vessels in its mass [8].

The diagnosis of mesenteric panniculitis is made after CT or magnetic resonance (MRI) examination. Moreover, for the diagnosis of MP the changes in adipose abdominal tissue in CT are sufficient for diagnosis (specificity) [6]. The computed tomography image reflects main pathological components such as inflammation, fat necrosis or fibrosis. The inflammatory component is manifested by an increase in the density of the mesentery, fat proliferation and activity of small nodes [9].

Among the most frequently detected CT signs of MP the following should be mentioned: an increase in the density of adipose tissue of the mesentery (the symptom of "misty mesentery"); soft tissue formation in the root of the mesentery; the "fat ring" sign, based on the maintenance of normal densitometric values of fat near the mesenteric vessels; enlarged lymph nodes in the mesentery; and a pseudocapsule, appearing as a layer of soft tissue, which separates the unaffected mesentery from the inflamed fat [3].

If the discovered MP remains asymptomatic, no treatment is required. In the described patient abdominal pain and some general symptoms encouraged us to start treatment with glucocorticoids and a proton pump inhibitor with good effects.

In clinical practice and in the literature other anti-inflammatory, immunosuppressive and immunomodulatory drugs are used such as thalidomide, cyclophosphamide, progesterone, colchicine, azathioprine, tamoxifen, antibiotics and emetine, or even radiotherapy. In severe cases or as a diagnostic procedure in differentiation from other causes of abdominal symptoms such as perforation or bowel obstruction surgical procedures may be necessary [10].

Conclusions

In summary, it is necessary to emphasize that MP is a multidisciplinary problem and may occur in the clinical practice of various specialists (rheumatologists, general practitioners, surgeons, gynecologists, etc.). The variety of clinical manifestations suggests that for differential diagnosis it is necessary to take into account a great number of diseases, which requires a thorough survey and comprehensive clinical, laboratory and instrumental examination of a patient to verify the diagnosis and timely decide to observe or administer proper therapy.

The authors declare no conflict of interest.

References

1. Issa I, Baydoun H. Mesenteric panniculitis: Various presentations and treatment regimens. World J Gastroenterol 2009; 15: 3827-3830.
2. Sahin A, Artas H, Eroglu Y, et al. An Overlooked Potentially Treatable Disorder: Idiopathic Mesenteric Panniculitis. Med Princ Pract 2018; 26: 567-572.
3. Coulier B. Mesenteric panniculitis. Part 2: prevalence and natural course: MDCT prospective study. JBR-BTR 2011; 94: 241-246.
4. Gögebakan Ö, Osterhoff MA, Albrecht T. Mesenteric Panniculitis (MP): A Frequent Coincidental CT Finding of Debatable Clinical Significance. Rofo 2018; 190: 1044-1052.
5. Sharma P, Yadav S, Needham CM, Feuerstadt P. Sclerosing mesenteritis: a systematic review of 192 cases. Clin J Gastroenterol 2017; 10: 103-111.
6. Al-Omari MH, Qararha K, Garaleh M, et al. Mesenteric panniculitis: comparison of computed tomography findings in patients with and without malignancy. Clin Exp Gastroenterol 2018; 12: 1-8.
7. Delgado Plasencia L, Rodríguez Ballester L, López-Tomassetti Fernández EM, et al. Mesenteric panniculitis: experience in our center. Rev Esp Enferm Dig 2007; 99: 291-297.
8. Rosón N, Garriga V, Cuadrado M, et al. Sonographic findings of mesenteric panniculitis: correlation with CT and literature review. J Clin Ultrasound 2006; 34: 169-176.
9. Horton KM, Lawler LP, Fishman EK. CT findings in sclerosing mesenteritis (panniculitis) spectrum of disease. Radiographics 2003; 23: 1561-1567.
10. Kaya C, Bozkurt E, Yazıcı P, et al. Approach to the diagnosis and treatment of mesenteric panniculitis from the surgical point of view. Turk J Surg 2018; 34: 121-124.