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CASE REPORT

Intestinal obstruction caused by extramedullary hematopoiesis and ascites in primary myelofibrosis

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Abstract

Primary myelofibrosis (PMF) is a clonal hematopoietic stem cell disorder. It is characterized by bone marrow fibrosis, extramedullary hematopoiesis with hepatosplenomegaly and leukoerythroblastosis in the peripheral blood. The main clinical manifestations of PMF are anemia, bleeding, hepatosplenomegaly, fatigue, and fever. Here we report a rare case of PMF with anemia, small bowel obstruction and ascites due to extramedullary hematopoiesis and portal hypertension. The diagnosis was difficult to establish before surgery and the differential diagnosis is discussed.

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Key words: Primary myelofibrosis; Intestinal obstruction; Ascites; Extramedullary hematopoiesis

Core tip: Bowel obstruction caused by extramedullary hematopoiesis and ascites due to portal hypertension are uncommon symptoms in primary myelofibrosis. Physicians should bear in mind that these rare manifestations can occur at the same time in a single patient.

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INTRODUCTION

Primary myelofibrosis (PMF) is a clonal hematopoietic stem cell disorder characterized by bone marrow fibrosis, extramedullary hematopoiesis with hepatosplenomegaly and leukoerythroblastosis in the peripheral blood[1]. The clinical manifestations of PMF include severe anemia which is caused by ineffective erythropoiesis, bleeding, marked hepatosplenomegaly due to extramedullary hematopoiesis, hyperuricemia and constitutional symptoms such as cachexia, fatigue, and fever. Ascites may occur in PMF due to portal hypertension[2,3]. As extramedullary hematopoiesis can occur anywhere, the clinical manifestations can be diverse. Extramedullary hematopoiesis mimicking acute appendicitis and intestinal obstruction, rectal stenosis, gastric outlet obstruction and bladder outlet obstruction due to extramedullary hematopoiesis have been reported[4-8]. However, intestinal obstruction and ascites

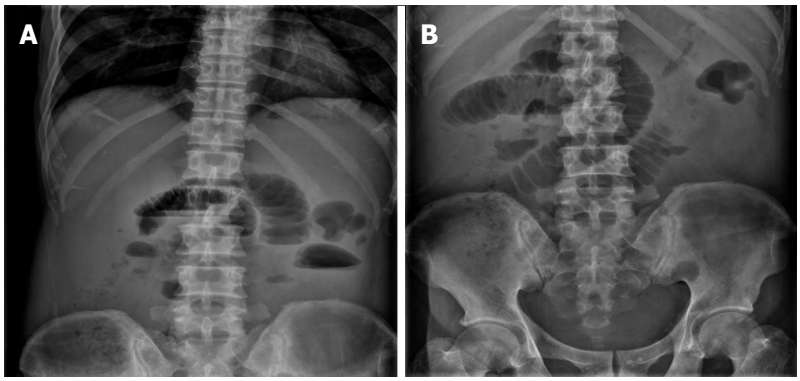

Figure 1 Plain abdominal radiography revealed incomplete small intestinal obstruction. A: The flat film of the abdomen in the standing position showed dilated small bowel loops with air-fluid levels; B: The flat film of the abdomen in the horizontal position showed dilated small bowel.

occurring at the same time in a patient with PMF has not been reported to date.

CASE REPORT

A 61-year-old man attended the Emergency Department of our hospital in October 2013 with complaints of significant weight loss, fatigue and anemia since May 2012. He underwent bone marrow biopsy on November 2012 and the results confirmed the diagnosis of PMF. In June 2013, the patient complained of vomiting, abdominal pain and abdominal distension with passage of flatus. He was then admitted to the Department of Hematology, where he underwent plain abdominal radiography which revealed incomplete small intestinal obstruction (Figure 1); computerized tomography (CT) scan and ultrasound B scan revealed hepatosplenomegaly and ascites, but no mass was found. Anal double-balloon enteroscopic examination was unremarkable, while peroral double-balloon enteroscopic examination was refused by the patient. After fasting for five days, the abdominal symptoms were relieved and the patient was discharged. The patient returned to our hospital with the chief complaints of vomiting, abdominal pain and abdominal distension, but with passage of flatus for a week. On physical examination, his vital signs were normal, however, pallor with ascites, hepatosplenomegaly and hyperactive bowel sounds with no palpable abdominal mass were observed.

Laboratory blood examinations showed the following indices (normal range in parentheses): hemoglobin, 59 g/L (120-140 g/L); peripheral white cell count, 5.39 × 10^9/L (5 × 10^9/L-10 × 10^9/L); neutrophils, 72.2% (40%-60%); peripheral red cell count, 2.47 × 10^{12}/L (4.0 × 10^{12}/L-4.5 × 10^{12}/L); platelet count, 210 × 10^9/L (100 × 10^9/L-300 × 10^9/L); peripheral eosinophil count, 0.02 × 10^9/L (0.02 × 10^9/L-0.52 × 10^9/L); C-reactive protein, 20.5 mg/L (0-6.0 mg/L); erythrocyte sedimentation rate, 31 mm/h (0-20 mm/h); albumin, 31.8 g/L (36-51 g/L); total immunoglobulin, 29.8 g/L (25-35 g/L); total bilirubin, 4.3 μmol/L (4-23.9 μmol/L); alkaline phosphatase, 57 U/L (35-125 U/L); c-glutamyl transpeptidase, 18 U/L (7-50 U/L); aspartate aminotransferase, 11 U/L (14-40 U/L); alanine aminotransferase, 5 U/L (5-35 U/L); creatinine, 192 μmol/L (31.8-91.0 μmol/L); blood urine nitrogen, 4.71 g/L (2.4-8.2 g/L); uric acid, 1108 μmol/L (90-420 μmol/L); prothrombin time, 14.3 s (11.0-14.5 s). Hepatitis B and C markers were negative. Serum tumor marker, cancer antigen 125 (CA125) was 321.2 U/mL (0-35 U/mL); alpha-fetoprotein (AFP), carcinoembryonic antigen (CEA) and cancer antigen 19-9 (CA 19-9) were not elevated. Tuberculosis (TB)-related antibodies were not found in the blood, and the TB-purified protein derivative (PPD) skin test was negative. Antinuclear antibodies (ANA), antineutrophil cytoplasmic antibody (ANCA) and rheumatoid factor (RF) were not found in the blood. Routine urine and stool tests did not reveal any RBCs or proteins on the first day of hospitalization. Routine ascites test: color, yellow; Rivalta test, negative; red cell count, 280 × 10^6/L; red cell count, 66 × 10^6/L; lymphocytes, 70%; granulocytes, 30%; and no obvious eosinophilic granulocytes. The total protein, albumin, glucose, lactic dehydrogenase (LDH) and adenosine deaminase (ADA) levels in the abdominal fluid were 30.9 g/L, 18 g/L, 4.87 mmol/L, 95 U/L and 3.0 U/L, respectively. The serum-ascites albumin gradient (SAAG) = 31.8 g/L - 18 g/L = 13.8 g/L >11 g/L. No tumor cells were found in the ascites fluid following two cytopathology tests.

Abdominal CT scanning was repeated and revealed hepatosplenomegaly, huge ascites and thickened ileum wall with obvious enhancement in the arterial phase causing obstruction (Figure 2). However, it was difficult to determine whether the intestinal lesion was malignant or an inflammatory lesion, as there was an obvious enhancement in the arterial phase and ascites simultaneously.

During the first ten days of hospitalization, the patient received a nasogastric tube along with blood transfusion, albumin infusion, an intravenous proton pump inhibitor (pantoprazole 40 mg, twice daily), antibiotics and an intravenous diuretic (furosemide 20 mg, once or twice daily). Ascites reduced considerably, blood hemoglobin increased from 59 g/L to 89 g/L, blood creatinine decreased from 192 μmol/L to 116 μmol/L, and serum albumin increased from 31.8 g/L to 37 g/L, but there was no relief of abdominal symptoms.

The patient was then referred to the Department of Gastrointestinal Surgery, where a laparotomy and partial

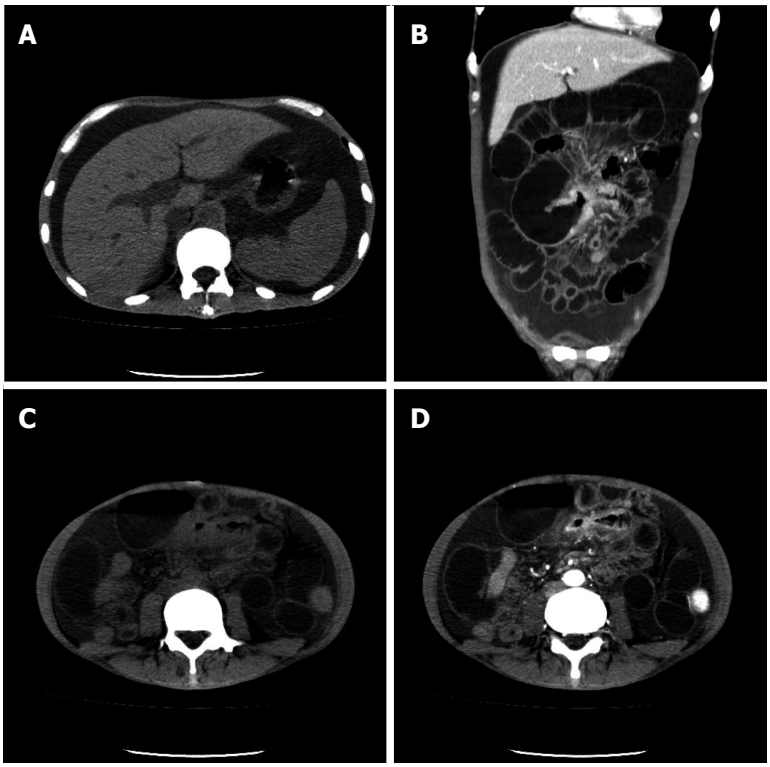

Figure 2 Computerized tomography images indicated hepatosplenomegaly and ascites (A) and the presence of thickened intestinal wall with obvious enhancement in the arterial phase and dilated small bowel (B-D). A: Plain computerized tomography (CT) scan; B: Coronal CT scan; C: Plain CT scan; D: The arterial phase.

enterectomy were performed. During surgery, an intestinal mass of approximately 5 cm × 3 cm × 3 cm and an obstruction in the ileum about 130 cm from the ileocecal valve were found, and intestinal adhesion forming a closed loop due to the mass was also observed. The resected ileum was 15 cm in length with a 5 cm × 3.5 cm yellow ulcerated mass in the center (Figure 3).

The pathological results were as follows: A gross view revealed a 5 cm × 3.5 cm brownish-yellow ulcerated mass in the 15 cm resected intestine. A microscopic view confirmed an ulcer in the resected specimen and there was significant hyperplasia of blood vessels in the deep layers of the intestine under the ulcer, along with hyperplasia of vascular endothelial cells. A large number of infiltrated inflammatory cells could be seen in the wall of the intestine; and a significant quantity of Megakaryocytes was observed around the serosal area along with an accumulation of immature myeloid cells and erythroid cells. Immunohistochemical (IHC) examination showed the following: CD61 (+), CD68 (-), MPO (+), CD34 (+), CD31 (+), CD11 (-), and Ki-67 (30%). An extramedullary hematopoietic mass of the small intestine with an ulcer and excessive vascular proliferation were confirmed pathologically (Figure 4).

Following surgery, the patient's abdominal symptoms and ascites completely resolved and he was discharged. Diuretics, testosterone undecanoate and thalidomide were prescribed in the outpatient department during a two-month follow-up and no abdominal symptoms were noted.

DISCUSSION

Extramedullary hematopoiesis occurs in conditions with an increased number of circulating myeloid progenitor cells, such as in PMF. The most common sites of extramedullary hematopoiesis are the spleen, liver, kidneys and the adrenal glands[9]. However, other organs are occasionally involved, such as the gastrointestinal tract[4-7], skin[10], joints[11,12], posterior mediastinum[13,14], the pericardium[15], and the brain[16-18]. A hematopoietic mass can cause symptoms resulting from stricture of hollow organs and compression of adjacent structures. In this patient, the hematopoietic mass was adherent to adjacent structures which also played an important role in causing symptoms. Intestinal obstruction, rectal stenosis, gastric outlet obstruction and bladder outlet obstruction due to extramedullary hematopoiesis have been reported[4-8], and progressive paraplegia may develop when extramedullary hematopoiesis occurs in the epidural space[17,18]. This patient suffered from a closed loop intestinal obstruction due to both intestinal stenosis and adhesion to the adjacent intestine caused by an extramedullary hematopoietic mass. As a closed loop was formed, it is reasonable that the ileac lesion could not be reached by a double-balloon enteroscopic examination. The lesion was identified on CT scan before surgery. In addition to gastrointestinal endoscopic examinations, CT scanning can also serve as an important tool in identifying gastrointestinal lesions. However, there was obvious enhancement in the arterial phase which was consistent with significant hyperplasia of blood vessels as confirmed by the pathological results in this patient, and it was difficult to determine whether the intestinal lesion was malignant on the CT scan.

Ascites are found in some PMF cases, the main cause of ascites is portal hypertension[2,3], peritoneal or other ectopic hematopoiesis can also be the main cause[19-22],

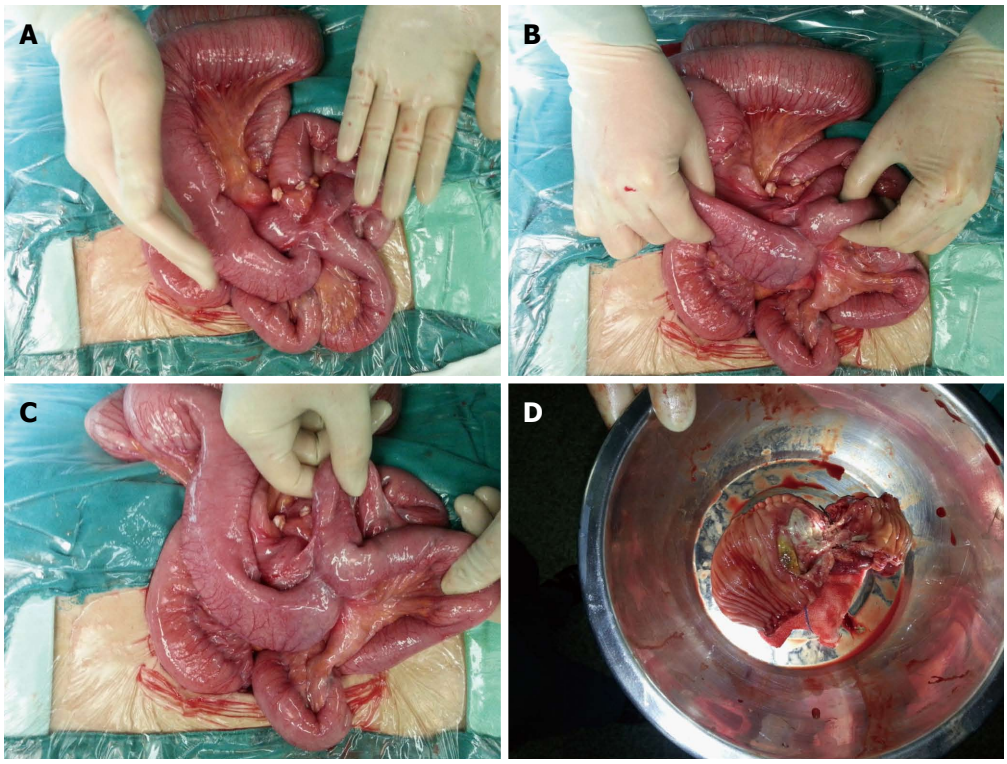

Figure 3 Exploratory laparotomy revealed intestinal obstruction caused by an intestinal mass approximately 5 cm × 3 cm × 3 cm and intestinal adhesions. A: The intestinal mass; B-C: Intestinal adhesions; D: The brownish-yellow mass with an ulcer.

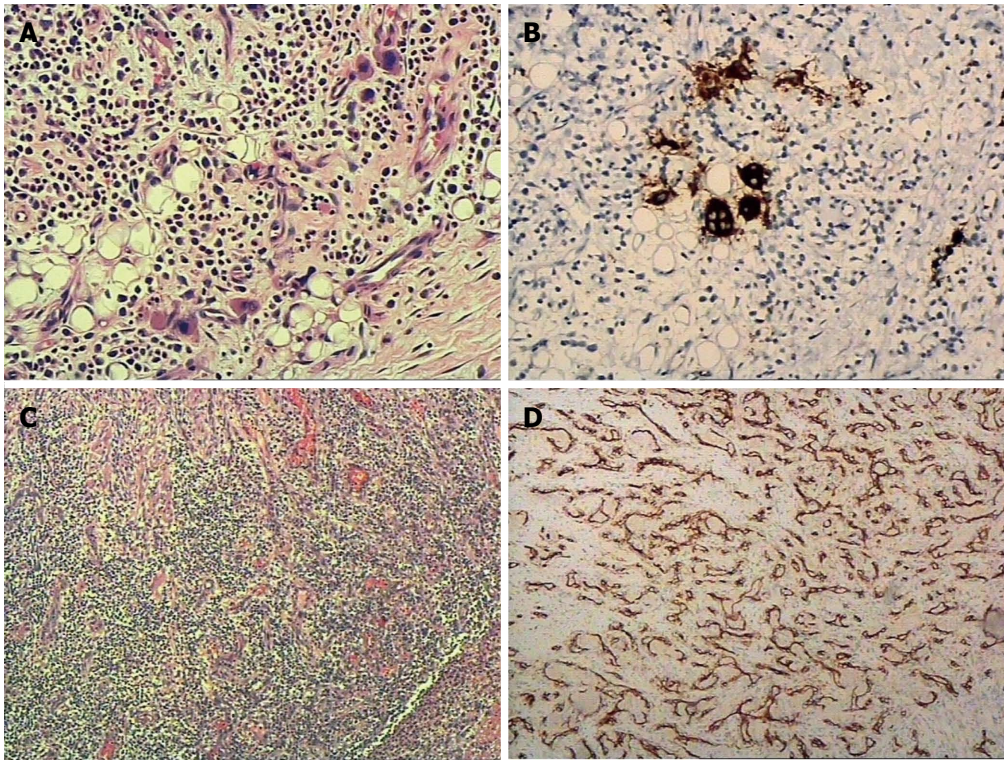

Figure 4 Extramedullary hematopoietic mass of the small intestine with ulcer and excessive vascular proliferation was confirmed by histopathology. A: A significant quantity of Megakaryocytes along with the accumulation of immature myeloid cells and erythroid cells were observed; B: Megakaryocytes were CD61 positive and CD68 negative (not shown); C: Significant hyperplasia of blood vessels and a large number of infiltrated inflammatory cells were seen; D: Blood vessels were confirmed by positive CD31 staining.

and hypoalbuminemia may play a role, as seen in this patient. Portal hypertension is due to two main mechanisms: firstly, increased blood flow through the massively enlarged spleen; secondly, functional intrahepatic obstruction caused by extramedullary hematopoiesis or periportal fibrosis in the liver[2,3,23,24]. Portal hypertension was proved indirectly by the high SAAG level which was higher than 11 g/L in this patient. The ascites, due to typical liquid leakage, reduced significantly following albumin infusion and intravenous diuretic before surgery, and were completely resolved by oral diuretics after surgery. Unfortunately, some patients with ascites are refractory to a sodium-restricted diet and high-dose diuretic treatment, TIPS may be a rescue therapy for refractory ascites secondary to portal hypertension, however, caution is necessary with respect to the presence and/or development of peritoneal or other ectopic hematopoiesis[3,19-22]. Ascites caused by peritoneal hematopoiesis have been reported to respond well to chemotherapy[19].

Intestinal obstruction and ascites occurring simultaneously in a patient is not rare, however, intestinal obstruction caused by extramedullary hematopoiesis and ascites occurring at the same time in a patient with PMF has, to our knowledge, not been reported. A series of differential diagnoses should be considered. Firstly, tuberculous peritonitis causing intestinal obstruction or intestinal tuberculosis causing intestinal obstruction with tuberculous peritonitis have been reported previously, and it is possible that abdominal tuberculosis occurs in PMF; however, there were no symptoms of tuberculosis, tuberculosis (TB)-related antibodies were not found in the blood, the TB-purified protein derivative (PPD) skin test was negative, and ascites was not an inflammatory exudate; thus, tuberculous peritonitis and intestinal tuberculosis were not diagnosed. Secondly, gastrointestinal carcinoma with intestinal stenosis causing metastatic ascites[25] or malignant ascites, such as peritoneal mesothelioma, causing intestinal obstruction[26-28] should also be considered. Fortunately, in this patient, blood CEA was normal, the ascites was due to typical liquid leakage, and no tumor cells were found in the ascites. These results revealed that the patient did not have a tumor. Thirdly, some autoimmune diseases such as systemic lupus erythematosus (SLE) can cause both intestinal obstruction and ascites[29,30]; however, in this patient, ANA, RF or ANCA were not found in the blood and the ascites was not an inflammatory exudate; thus, the diagnosis of autoimmune diseases was not considered. Fourthly, eosinophilic gastroenteritis presenting with intestinal obstruction and ascites has been reported[31,32]. Talley *et al*[33] identified three main diagnostic criteria for eosinophilic gastroenteritis: (1) the presence of gastrointestinal symptoms; (2) biopsies demonstrating eosinophilic infiltration of one or more areas of the gastrointestinal tract; and (3) no evidence of parasitic or extraintestinal disease; taking the normal level of eosinophilic granulocytes in the blood and ascites and a normal double-balloon enteroscopic examination into consideration, eosinophilic gastroenteritis was not a reasonable diagnosis. Lastly, bloody ascites caused by strangulation obstruction is a common clinical emergency, and was easily excluded in this patient.

Clinical physicians should bear in mind that intestinal obstruction caused by extramedullary hematopoiesis and ascites due to portal hypertension can occur at the same time in PMF, although it is not very common. Ascites due to portal hypertension may be resolved by diuretics, as in this case. Intestinal obstruction caused by an extramedullary hematopoietic mass can be cured surgically by removing the mass.

COMMENTS

Case characteristics

A 61-year-old man with a history of primary myelofibrosis presented with intestinal obstruction and ascites.

Clinical diagnosis

Intestinal obstruction caused by extramedullary hematopoiesis and ascites due to portal hypertension in primary myelofibrosis.

Differential diagnosis

Abdominal tuberculosis, abdominal cancer, autoimmune disease, and eosinophilic gastroenteritis.

Laboratory diagnosis

Hemoglobin 59 g/L; tuberculosis-related antibodies negative; CEA negative; and SAAG 13 g/L.

Imaging diagnosis

Computerized tomography scan revealed hepatosplenomegaly, ascites and a thickened ileum wall with obvious enhancement in the arterial phase causing obstruction.

Pathological diagnosis

Extramedullary hematopoietic mass of the small intestine with ulcer and excessive vascular proliferation was confirmed by HE staining and immunohistochemistry.

Treatment

A partial enterectomy was performed and diuretics were prescribed.

Term explanation

Extramedullary hematopoiesis is a phenomenon in which hematopoietic cells are found in sites other than the bone marrow.

Experiences and lessons

Laparotomy should be performed in patients with small intestinal obstruction of unknown cause in primary myelofibrosis.

Peer review

This paper presents a rare case of both intestinal obstruction caused by extramedullary hematopoiesis and ascites due to portal hypertension in primary myelofibrosis.

REFERENCES

1 **Tefferi A**. Primary myelofibrosis: 2013 update on diagnosis, risk-stratification, and management. *Am J Hematol* 2013; **88**: 141-150 [PMID: 23349007 DOI: 10.1002/ajh.23384]

2 **Toros AB**, Gokcay S, Cetin G, Ar MC, Karagoz Y, Kesici B. Portal hypertension and myeloproliferative neoplasms: a relationship revealed. *ISRN Hematol* 2013; **2013**: 673781 [PMID: 24159391 DOI: 10.1155/2013/673781]

3 **Wiest R**, Strauch U, Wagner H, Strotzer M, Woenckhaus M, Schröder G, Schölmerich J, Lock G. A patient with myelofibrosis complicated by refractory ascites and portal hypertension: to tips or not to tips? A case report with discussion of the mechanism of ascites formation. *Scand J Gastroenterol* 2004; **39**: 389-394 [PMID: 15125474 DOI: 10.1080/00365520310007521]

4 **Elpek GO**, Bozova S, Erdoğan G, Temizkan K, Oğüş M. Extramedullary hematopoiesis mimicking acute appendicitis:

a rare complication of idiopathic myelofibrosis. *Virchows Arch* 2006; **449**: 258-261 [PMID: 16738896 DOI: 10.1007/s00428-006-0230-5]

5 **Solomon D**, Goodman H, Jacobs P. Case report: rectal stenosis due to extramedullary haemopoiesis--radiological features. *Clin Radiol* 1994; **49**: 726-728 [PMID: 7955840 DOI: 10.1016/S0009-9260(05)82672-8]

6 **Ismail SM**, Myers K. Infiltrative myeloid metaplasia: an unusual cause of gastric outlet obstruction. *J Clin Pathol* 1989; **42**: 1112-1113 [PMID: 2584415 DOI: 10.1136/jcp.42.10.1112-b]

7 **MacKinnon S**, McNicol AM, Lee FD, McDonald GA. Myelofibrosis complicated by intestinal extramedullary haemopoiesis and acute small bowel obstruction. *J Clin Pathol* 1986; **39**: 677-679 [PMID: 3722421 DOI: 10.1136/jcp.39.6.677]

8 **Humphrey PA**, Vollmer RT. Extramedullary hematopoiesis in the prostate. *Am J Surg Pathol* 1991; **15**: 486-490 [PMID: 2035742 DOI: 10.1097/00000478-199105000-00009]

9 **Banerji JS**, Kumar RM, Devasia A. Extramedullary hematopoiesis in the adrenal: Case report and review of literature. *Can Urol Assoc J* 2013; **7**: E436-E438 [PMID: 23826059 DOI: 10.5489/cuaj.1389]

10 **Miyata T**, Masuzawa M, Katsuoka K, Higashihara M. Cutaneous extramedullary hematopoiesis in a patient with idiopathic myelofibrosis. *J Dermatol* 2008; **35**: 456-461 [PMID: 18705835 DOI: 10.1111/j.1346-8138.2008.00502.x]

11 **Heinicke MH**, Zarrabi MH, Gorevic PD. Arthritis due to synovial involvement by extramedullary haematopoiesis in myelofibrosis with myeloid metaplasia. *Ann Rheum Dis* 1983; **42**: 196-200 [PMID: 6847265 DOI: 10.1136/ard.42.2.196]

12 **Alvarez-Argüelles Cabrera H**, Carrasco Juan JL, García Castro MC, González Gaitano M, Bonilla Arjona A, Díaz-Flores L. Synovial tumefactive extramedullary hematopoiesis associated to polycythemia vera. *Virchows Arch* 2007; **450**: 109-113 [PMID: 17109152 DOI: 10.1007/s00428-006-0325-z]

13 **Yeom SY**, Lim JH, Han KN, Kang CH, Park IK, Kim YT. Extramedullary hematopoiesis at the posterior mediastinum in patient with hereditary spherocytosis: a case report. *Korean J Thorac Cardiovasc Surg* 2013; **46**: 156-158 [PMID: 23614106 DOI: 10.5090/kjtcs.2013.46.2.156]

14 **Baikoussis NG**, Beis JP, Verra C, Siminelakis SN. A mass in the posterior mediastinum; extramedullary haemopoietic tissue. *Eur Rev Med Pharmacol Sci* 2012; **16**: 691-694 [PMID: 22774413]

15 **Toms DR**, Cannick L, Stuart RK, Jenrette JM, Terwiliger L. Helical tomotherapy for extramedullary hematopoiesis involving the pericardium in a patient with chronic myeloid leukemia. *Jpn J Radiol* 2010; **28**: 476-478 [PMID: 20661700 DOI: 10.1007/s11604-010-0452-y]

16 **Singer A**, Quencer R. Intracranial Extramedullary Hematopoiesis: A Rare Cause of Headaches. *J Neuroimaging* 2013; Epub ahead of print [PMID: 23621819 DOI: 10.1111/jon.12029]

17 **Alam MR**, Habib MS, Dhakal GP, Khan MR, Rahim MA, Chowdhury AJ, Mahmud TK. Extramedullary hematopoiesis and paraplegia in a patient with hemoglobin e-Beta thalassemia. *Mymensingh Med J* 2010; **19**: 452-457 [PMID: 20639844]

18 **Piccaluga PP**, Finelli C, Vigna E, Agostinelli C, Bacci F, Paolini S, Papayannidis C, Laterza C, Martinelli G, Pileri SA, Baccarani M. Paraplegia due to a paravertebral extramedullary haemopoiesis in a patient with polycythaemia vera. *J Clin Pathol* 2007; **60**: 581-582 [PMID: 17513521 DOI: 10.1136/jcp.2006.039149]

19 **Hung SC**, Huang ML, Liu SM, Hsu HC. Massive ascites caused by peritoneal extramedullary hematopoiesis as the initial manifestation of myelofibrosis. *Am J Med Sci* 1999; **318**: 198-200 [PMID: 10487412 DOI: 10.1097/00000441-199909000-00017]

20 **Oren I**, Goldman A, Haddad N, Azzam Z, Krivoy N, Alroy G. Ascites and pleural effusion secondary to extramedullary hematopoiesis. *Am J Med Sci* 1999; **318**: 286-288 [PMID: 10522557 DOI: 10.1097/00000441-199910000-00009]

21 **Yotsumoto M**, Ishida F, Ito T, Ueno M, Kitano K, Kiyosawa K. Idiopathic myelofibrosis with refractory massive ascites. *Intern Med* 2003; **42**: 525-528 [PMID: 12857054 DOI: 10.2169/internalmedicine.42.525]

22 **Holden C**, Hennessy O, Lee WK. Diffuse mesenteric extramedullary hematopoiesis with ascites: sonography, CT, and MRI findings. *AJR Am J Roentgenol* 2006; **186**: 507-509 [PMID: 16423960]

23 **Abu-Hilal M**, Tawaker J. Portal hypertension secondary to myelofibrosis with myeloid metaplasia: a study of 13 cases. *World J Gastroenterol* 2009; **15**: 3128-3133 [PMID: 19575492 DOI: 10.3748/wjg.15.3128]

24 **Alvarez-Larrán A**, Abraldes JG, Cervantes F, Hernández-Guerra M, Vizzutti F, Miquel R, Gilabert R, Giusti M, Garcia-Pagan JC, Bosch J. Portal hypertension secondary to myelofibrosis: a study of three cases. *Am J Gastroenterol* 2005; **100**: 2355-2358 [PMID: 16181389 DOI: 10.1111/j.1572-0241.2005.50374.x]

25 **Amikura K**, Sakamoto H, Yatsuoka T, Kawashima Y, Nishimura Y, Tanaka Y. Surgical management for a malignant bowel obstruction with recurrent gastrointestinal carcinoma. *J Surg Oncol* 2010; **101**: 228-232 [PMID: 20039277 DOI: 10.1002/jso.21463]

26 **Naraynsingh V**, Ramdass MJ, Lum CL. Malignant peritoneal mesothelioma presenting as recurrent adhesion obstruction in general surgery: a case report. *J Med Case Rep* 2011; **5**: 420 [PMID: 21878098 DOI: 10.1186/1752-1947-5-420]

27 **Griniatsos J**, Sougioultzis S, Dimitriou N, Vamvakopoulou V, Alexandrou P, Kyriakou V, Tzioufas A, Papalambros E, Tzivras M. Diffuse malignant peritoneal mesothelioma presenting as intestinal obstruction. *South Med J* 2009; **102**: 1061-1064 [PMID: 19738519 DOI: 10.1097/SMJ.0b013e3181b671ef]

28 **Blair SL**, Chu DZ, Schwarz RE. Outcome of palliative operations for malignant bowel obstruction in patients with peritoneal carcinomatosis from nongynecological cancer. *Ann Surg Oncol* 2001; **8**: 632-637 [PMID: 11569777]

29 **Lin YJ**, Chen PC, Chen HA. Mesenteric vasculitis causing ileocecal intussusception as the initial presentation of systemic lupus erythematosus: a case report. *Clin Rheumatol* 2013; **32** Suppl 1: S37-S40 [PMID: 20238134 DOI: 10.1007/s10067-010-1421-7]

30 **Ceccato F**, Salas A, Góngora V, Ruta S, Roverano S, Marcos JC, Garcìa M, Paira S. Chronic intestinal pseudo-obstruction in patients with systemic lupus erythematosus: report of four cases. *Clin Rheumatol* 2008; **27**: 399-402 [PMID: 17938989]

31 **Lim KC**, Tan HK, Rajnakova A, Venkatesh SK. Eosinophilic gastroenteritis presenting with duodenal obstruction and ascites. *Ann Acad Med Singapore* 2011; **40**: 379-381 [PMID: 22065005]

32 **Yun MY**, Cho YU, Park IS, Choi SK, Kim SJ, Shin SH, Kim KR. Eosinophilic gastroenteritis presenting as small bowel obstruction: a case report and review of the literature. *World J Gastroenterol* 2007; **13**: 1758-1760 [PMID: 17461485]

33 **Talley NJ**, Shorter RG, Phillips SF, Zinsmeister AR. Eosinophilic gastroenteritis: a clinicopathological study of patients with disease of the mucosa, muscle layer, and subserosal tissues. *Gut* 1990; **31**: 54-58 [PMID: 2318432]

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