

World Journal of *Gastrointestinal Surgery*

World J Gastrointest Surg 2023 September 27; 15(9): 1841-2097



Contents

Monthly Volume 15 Number 9 September 27, 2023

REVIEW

- 1841 Indocyanine green dye and its application in gastrointestinal surgery: The future is bright green
Lim ZY, Mohan S, Balasubramaniam S, Ahmed S, Siew CCH, Shelat VG
- 1858 Hepatic ischemia-reperfusion syndrome and its effect on the cardiovascular system: The role of treprostinil, a synthetic prostacyclin analog
Mouratidou C, Pavlidis ET, Katsanos G, Kotoulas SC, Mouloudi E, Tsoulfas G, Galanis IN, Pavlidis TE

MINIREVIEWS

- 1871 Advances and challenges of gastrostomy insertion in children
Bitar R, Azaz A, Rawat D, Hobeldin M, Miqdady M, Abdelsalam S
- 1879 Surgical decompression for the management of abdominal compartment syndrome with severe acute pancreatitis: A narrative review
Nasa P, Chanchalani G, Juneja D, Malbrain ML

ORIGINAL ARTICLE

Retrospective Cohort Study

- 1892 Excision of malignant and pre-malignant rectal lesions by transanal endoscopic microsurgery in patients under 50 years of age
Shilo Yaacobi D, Berger Y, Shaltiel T, Bekhor EY, Khalifa M, Issa N
- 1901 Safety and feasibility of modified duct-to-mucosa pancreaticojejunostomy during pancreatoduodenectomy: A retrospective cohort study
Sun Y, Yu XF, Yao H, Xu S, Ma YQ, Chai C

Retrospective Study

- 1910 Application of early enteral nutrition nursing based on enhanced recovery after surgery theory in patients with digestive surgery
Shao YR, Ke X, Luo LH, Xu JD, Xu LQ
- 1919 Autologous bone marrow infusion *via* portal vein combined with splenectomy for decompensated liver cirrhosis: A retrospective study
Liu BC, Cheng MR, Lang L, Li L, Si YH, Li AJ, Xu Q, Zhang H
- 1932 Application of multidisciplinary collaborative nursing with family care for enhanced recovery after surgery in children with inguinal hernia
Wang XM, Hou Q
- 1941 Preoperative and postoperative complications as risk factors for delayed gastric emptying following pancreaticoduodenectomy: A single-center retrospective study
Xie FL, Ren LJ, Xu WD, Xu TL, Ge XQ, Li W, Ge XM, Zhou WK, Li K, Zhang YH, Wang Z

- 1950** Efficacy of ileus tube combined with meglumine diatrizoate in treating postoperative inflammatory bowel obstruction after surgery
Yang W, Pu J
- 1959** Effect of internet multiple linkage mode-based extended care combined with in-hospital comfort care on colorectal cancer patients undergoing colostomy
Xu L, Zhou MZ
- 1969** Short- and long-term results of open *vs* laparoscopic multisegmental resection and anastomosis for synchronous colorectal cancer located in separate segments
Quan JC, Zhou XJ, Mei SW, Liu JG, Qiu WL, Zhang JZ, Li B, Li YG, Wang XS, Chang H, Tang JQ
- 1978** Prediction model of stress ulcer after laparoscopic surgery for colorectal cancer established by machine learning algorithm
Yu DM, Wu CX, Sun JY, Xue H, Yuwen Z, Feng JX
- 1986** Effect of two surgical approaches on the lung function and prognosis of patients with combined esophago-gastric cancer
Sun CB, Han XQ, Wang H, Zhang YX, Wang MC, Liu YN
- 1995** Clinical significance of serum oxidative stress and serum uric acid levels before surgery for hepatitis B-related liver cancer
Hou JX, Wang YB, Wu J, Ding GS, Wu Y, Wei LH, Wang F, Zhang ZM
- 2003** Multifactor analysis of the technique in total laparoscopic gastric cancer
Shi JK, Wang B, Zhang XS, Lv P, Chen YL, Ren SY
- 2012** Value of enhanced computed tomography in differentiating small mesenchymal tumours of the gastrointestinal from smooth muscle tumours
Nie WJ, Jing Z, Hua M
- 2021** Risk factors for myocardial injury during living donor liver transplantation in pediatric patients with biliary atresia
Wu YL, Li TY, Gong XY, Che L, Sheng MW, Yu WL, Weng YQ

Observational Study

- 2032** Comparative detection of syndecan-2 methylation in preoperative and postoperative stool DNA in patients with colorectal cancer
Song JH, Oh TJ, An S, Lee KH, Kim JY, Kim JS

Prospective Study

- 2042** Preoperative prediction of microvascular invasion in hepatocellular carcinoma using ultrasound features including elasticity
Jiang D, Qian Y, Tan BB, Zhu XL, Dong H, Qian R
- 2052** Quantitative evaluation of colorectal tumour vasculature using contrast-enhanced ultrasound: Correlation with angiogenesis and prognostic significance
Li MH, Li WW, He L, Li JF, Zhang SY

CASE REPORT

- 2063** Laparoscopy-assisted gastrectomy for advanced gastric cancer patients with situs inversus totalis: Two case reports and review of literature
Liu HB, Cai XP, Lu Z, Xiong B, Peng CW
- 2074** Acute flare of systemic lupus erythematosus with extensive gastrointestinal involvement: A case report and review of literature
Huang H, Li P, Zhang D, Zhang MX, Yu K
- 2083** Surgical management of gallstone ileus after one anastomosis gastric bypass: A case report
El Feghali E, Akel R, Chamaa B, Kazan D, Chakhtoura G
- 2089** Dual transformation therapy for giant hepatocellular carcinoma: Two case reports and review of literature
Gao Q, Zhu GZ, Han CY, Ye XP, Huang HS, Mo ST, Peng T

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AIMS AND SCOPE

The primary aim of *World Journal of Gastrointestinal Surgery* (WJGS, *World J Gastrointest Surg*) is to provide scholars and readers from various fields of gastrointestinal surgery with a platform to publish high-quality basic and clinical research articles and communicate their research findings online.

WJGS mainly publishes articles reporting research results and findings obtained in the field of gastrointestinal surgery and covering a wide range of topics including biliary tract surgical procedures, biliopancreatic diversion, colectomy, esophagectomy, esophagostomy, pancreas transplantation, and pancreatectomy, etc.

INDEXING/ABSTRACTING

The WJGS is now abstracted and indexed in Science Citation Index Expanded (SCIE, also known as SciSearch®), Current Contents/Clinical Medicine, Journal Citation Reports/Science Edition, PubMed, PubMed Central, Reference Citation Analysis, China National Knowledge Infrastructure, China Science and Technology Journal Database, and Superstar Journals Database. The 2023 Edition of Journal Citation Reports® cites the 2022 impact factor (IF) for WJGS as 2.0; IF without journal self cites: 1.9; 5-year IF: 2.2; Journal Citation Indicator: 0.52; Ranking: 113 among 212 journals in surgery; Quartile category: Q3; Ranking: 81 among 93 journals in gastroenterology and hepatology; and Quartile category: Q4.

RESPONSIBLE EDITORS FOR THIS ISSUE

Production Editor: Rui-Rui Wu, Production Department Director: Xiang Li, Editorial Office Director: Jia-Ru Fan.

NAME OF JOURNAL

World Journal of Gastrointestinal Surgery

ISSN

ISSN 1948-9366 (online)

LAUNCH DATE

November 30, 2009

FREQUENCY

Monthly

EDITORS-IN-CHIEF

Peter Schemmer

EDITORIAL BOARD MEMBERS

<https://www.wjgnet.com/1948-9366/editorialboard.htm>

PUBLICATION DATE

September 27, 2023

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INSTRUCTIONS TO AUTHORS

<https://www.wjgnet.com/bpg/gerinfo/204>

GUIDELINES FOR ETHICS DOCUMENTS

<https://www.wjgnet.com/bpg/GerInfo/287>

GUIDELINES FOR NON-NATIVE SPEAKERS OF ENGLISH

<https://www.wjgnet.com/bpg/gerinfo/240>

PUBLICATION ETHICS

<https://www.wjgnet.com/bpg/GerInfo/288>

PUBLICATION MISCONDUCT

<https://www.wjgnet.com/bpg/gerinfo/208>

ARTICLE PROCESSING CHARGE

<https://www.wjgnet.com/bpg/gerinfo/242>

STEPS FOR SUBMITTING MANUSCRIPTS

<https://www.wjgnet.com/bpg/GerInfo/239>

ONLINE SUBMISSION

<https://www.f6publishing.com>



Acute flare of systemic lupus erythematosus with extensive gastrointestinal involvement: A case report and review of literature

Hua Huang, Ping Li, Dan Zhang, Ming-Xuan Zhang, Kai Yu

Specialty type: Gastroenterology and hepatology

Provenance and peer review: Unsolicited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's scientific quality classification

Grade A (Excellent): 0
Grade B (Very good): 0
Grade C (Good): C, C, C
Grade D (Fair): 0
Grade E (Poor): 0

P-Reviewer: Makovicky P, Slovakia; Yale SH, United States

Received: April 24, 2023

Peer-review started: April 24, 2023

First decision: June 12, 2023

Revised: July 9, 2023

Accepted: July 28, 2023

Article in press: July 28, 2023

Published online: September 27, 2023



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Abstract

BACKGROUND

Lupus mesenteric vasculitis (LMV) is a serious condition that may occur as an acute manifestation of gastrointestinal (GI) involvement and is not easily diagnosed by physicians. Delayed diagnosis and treatment of LMV may lead to rapid disease progression and can be life threatening.

CASE SUMMARY

A previously healthy 27-year-old woman presented with abdominal pain following a history of fatigue and consumption of cold water. Laboratory investigations, physical examinations, and enhanced abdominal computed tomography (CT) suggested systemic lupus erythematosus complicated by LMV. She received treatments, such as GI decompression, somatostatin, glucocorticoids, and immunosuppressants, and was evaluated using color ultrasonography. Twenty days later, the patient reported no stomach discomfort and was able to consume semi-liquid food. Laboratory investigations showed that inflammatory factors decreased to normal levels and complement levels increased slightly. One year after discharged, she recovered with methylprednisolone being tapered to 4 mg per day, mycophenolate mofetil to 0.75 g bid, and hydroxychloroquine to 0.2 g bid; however, only C3 complement level was slightly below the normal level.

CONCLUSION

Early diagnosis of LMV is essential for successful treatment; this depends on a combination of clinical manifestations, laboratory investigations, and imaging findings. Enhanced CT is preferred, but ultrasonography can be used for prompt screening and follow-up.

Key Words: Systemic lupus erythematosus; Gastrointestinal involvement; Lupus enteritis; Lupus mesenteric vasculitis; Ultrasonography; Computer tomography; Case report

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Core Tip: When a systemic lupus erythematosus patient presenting with gastrointestinal symptoms, lupus mesenteric vasculitis (LMV) must be considered despite being rare. Enhanced abdominal computed tomography revealing thickened jejunal wall (target sign) and engorgement of the mesenteric vessels (comb sign) were suggestive of mesenteric vasculitis and provide evidence for accurately treatment. Abdominal ultrasonography can be a reliable tool used for preliminary screening, therapeutic evaluation and follow-up for LMV.

Citation: Huang H, Li P, Zhang D, Zhang MX, Yu K. Acute flare of systemic lupus erythematosus with extensive gastrointestinal involvement: A case report and review of literature. *World J Gastrointest Surg* 2023; 15(9): 2074-2082

URL: <https://www.wjgnet.com/1948-9366/full/v15/i9/2074.htm>

DOI: <https://dx.doi.org/10.4240/wjgs.v15.i9.2074>

INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic multisystemic autoimmune disease of unknown etiology. Under the influence of genetic predisposition and environmental risk factors, alterations in the immune response include hyperactivation of T and B lymphocytes, loss of self-tolerance and formation of circulating pathogenic immune complexes and subsequent deposition in several organs, thereby causing damage. The incidence of SLE is approximately 30–70 cases per 100000 individuals in China[1]. Females of reproductive age are mostly affected.

Gastrointestinal (GI) symptoms are present in > 50% of SLE patients throughout the course of the disease[2]. Among the wide spectrum of SLE-associated GI complications, including abdominal serositis/peritonitis, protein-losing enteropathy, intestinal pseudo-obstruction, hepatic involvement, and pancreatitis, lupus mesenteric vasculitis (LMV) is rare, but one of the most serious complications of SLE with high mortality[3]. Early diagnosis of LMV is crucial for prompt treatment.

LMV or lupus enteritis (LE), was originally proposed by Hoffman and Katz[4] in 1980. The early clinical presentation of LE comprises abdominal pain, nausea, vomiting, or severe GI bleeding, which is unremarkable and non-specific and is easily mistaken for infectious gastroenteritis and medication-related adverse effects. The rarity of LE in SLE, which makes it unincorporated in any SLE classification criteria or weighted in the SLE Disease Activity Index (SLEDAI), makes early clinical suspicion difficult to elicit. In the British Isles Lupus Assessment Group disease activity index, LE is defined as either vasculitis or inflammation of the small or large bowel with supportive imaging and/or biopsy findings[5]. Since histological evidence is difficult to obtain, it is important to find a rapid, reliable, and safe diagnostic tool. This case review aimed to identify possible strategies for early diagnosis of LE among women of reproductive age.

CASE PRESENTATION

Chief complaints

A 27-year-old woman was referred to our department on account of abdominal pain that had persisted for six days.

History of present illness

The patient was initially transferred to our department after an enhanced abdominal computed tomography (CT) examination and GI decompression.

History of past illness

The patient had no photosensitivity, alopecia, oral ulcers, arthritis, or Raynaud's phenomenon, and was previously healthy.

Personal and family history

The patient had no family history of SLE.

Physical examination

Upon examination, the abdomen was distended with no tenderness or rebound pain. No palpable masses or active bowel sounds were detected.

Laboratory examinations

Antinuclear antibody titer was 1:320, and anti-U1 ribonucleoprotein and anti-Sjogren's syndrome A antibodies were positive, whereas anti-double-stranded DNA (anti-dsDNA) antibody and anti-Smith antibody were negative. The levels of C3 and C4 complements decreased to 0.245 g/L (0.7–1.4 g/L) and 0.024 g/L (0.1–0.4 g/L), respectively. Immunoglobulin G (IgG) level was 10.45 g/L (7–16 g/L), and direct antiglobulin "Coombs" test results were positive. Routine blood tests showed a white blood cell count of $1.40 \times 10^{10}/L$ (3.5×10^9 – $9.5 \times 10^9/L$), platelet count of $2.01 \times 10^{11}/L$ (1.25×10^{11} – $3.50 \times 10^{11}/L$), and hemoglobin level of 114 g/L (115–150 g/L). Urine microscopy revealed a pathological tube-type 1 per low power field. Liver enzyme levels and renal function were normal. Moreover, serum albumin level was 33.2 g/L (40–55 g/L); erythrocyte sedimentation rate (ESR) was 3 mm/h (0–22 mm/h); C-reactive protein (CRP) level was 1.28 mg/L (< 10 mg/L); procalcitonin level was 0.293 ng/mL (0–0.046 ng/mL); blood potassium level was 2.9 mmol/L (3.5–5.3 mmol/L); cardiac enzyme levels were normal; and 24 h total urine protein level was 0.51 g/d (0.01–0.15 g/d).

Imaging examinations

Lung CT showed bilateral pleural effusion (Figure 1). Enhanced abdominal CT showed the typical "target and comb signs" performance (Figure 2). Further investigation revealed LMV and incomplete intestinal obstruction. Some segmental bowel walls of the transverse colon were thickened by approximately 5 mm, and the main color flow of the superior mesenteric artery was well filled without embolism (Figures 3 and 4).

FINAL DIAGNOSIS

According to the 2019 European League Against Rheumatism/American College of Rheumatology classification criteria for SLE, autoimmune hemolysis (4 points) + pleural or pericardial effusion (5 points) + low C3 and C4 (4 points) + proteinuria (4 points) = 17 points, which was > 10 points, and the patient met the diagnostic criteria; therefore, LMV and lupus nephritis were diagnosed. The SLEDAI-2K score was eight.

TREATMENT

After fasting, GI decompression, intravenous nutrition, methylprednisolone 80 mg per day intravenously, and immunoglobulin 20 g per day intravenously for five days, the symptoms were relieved, and the patient was able to tolerate semi-liquid food. Furthermore, the methylprednisolone dose was gradually decreased to 40 mg per day intravenously. Fourteen days later, the methylprednisolone dose was adjusted to 36 mg per day orally, and this dosage was maintained for approximately one month, even after the patient was discharged. Moreover, she was treated with cyclophosphamide 0.2 g qod intravenously twice with a cumulative dose of 0.4 g, which was switched to mycophenolate mofetil (MMF) 0.75 g bid orally. Owing to the GI inflammation, intravenous antibiotics were administered for 15 d. Somatostatin reduced the intestinal inflammation, exudation, and gastric acid secretion. Nadroparin calcium was administered to prevent micro- or small mesenteric thrombosis.

OUTCOME AND FOLLOW-UP

Twenty days after the treatment, the patient was able to eat semi-liquid food without stomach discomfort. Bowel sounds returned to normal. Laboratory investigations showed complement levels had increased. The IgG level was 15.6 g/L (7–16 g/L), and renal function, hepatic enzyme levels, and electrolyte levels were normal. Routine urine and stool test results were normal. Ten days after the treatment, ultrasonography was used to assess the patient's condition, which demonstrated that the local intestinal wall was approximately 0.35 cm at its thickest part (Figure 5), and that there was no effusion in the pleural, abdominal, or pelvic cavities. Most of the intestinal wall thickness decreased to a normal size. Finally, the intestinal lumen did not expand, and the multiserous cavity effusion disappeared. The abdominal signs and symptoms reduced, and the patient tolerated full feeding and was discharged.

The patient was follow-up at two-weekly intervals for the first three months and monthly thereafter. One year later, the patient was treated with methylprednisolone, which was tapered to 4 mg per day, MMF 0.75 g bid, and hydroxychloroquine 0.2 g bid. The C4 complement level was normalized, but the C3 complement level was 0.663 g/L (0.7–1.4 g/L), still slightly below the normal level. Table 1 shows the timeline from symptom onset to the last follow-up visit.

DISCUSSION

LE, also known LMV[6], is rare and one of the most devastating complications of SLE. The incidence of LMV is estimated to range between 0.2%–9.7% among patients already diagnosed with SLE and between 13.0%–62.5% in patients with isolated LE as the initial presentation of SLE[7].

Table 1 Timeline from symptom onset to the last follow-up visit

	Time of admission (March 29, 2022)	Time of discharge (April 4, 2022)	Last follow-up (June 16, 2023)
WBC (3.5×10^9 – 9.5×10^9 /L)	6.4	4.3	6.2
Hemoglobin (115–150 g/L)	98	100	133
Platelet (1.25×10^{11} – 3.50×10^{11} /L)	212	226	224
CRP (0–6 mg/L)	0.4	1.28	0.2
ESR (0–20 mm/h)	3	11	6
IgG (7–16 g/L)	10.45	15.6	11.56
IgA (0.7–4 g/L)	0.81	1.02	0.78
IgM (0.4–2.3 g/L)	0.46	0.603	0.72
C3 (0.9–1.8 g/L)	0.245	0.449	0.663
C4 (0.1–0.4 g/L)	0.027	0.082	0.143
Serum albumin (40–55 g/L)	22.8	38.3	43.5
Alanine aminotransferase (7–40 U/L)	9.22	14	13.31
Aspartate aminotransferase (13–35 U/L)	20.86	9	22.37
Serum creatinine (41–73 μ mol/L)	73.2	42	52.2
Serum urea nitrogen (3.1–8 mmol/L)	8.24	3.84	3.74
Lung CT	Bilateral pleural effusion	Normal	Normal
Ultrasonography	Bowel walls were thickened by approximately 5 mm	Bowel walls were thickened by approximately 3.5 mm	Normal

WBC: White blood cell; CRP: C-reactive protein; ESR: Erythrocyte sedimentation rate; IgG: Immunoglobulin G; IgA: Immunoglobulin A; IgM: Immunoglobulin M; CT: Computed tomography.

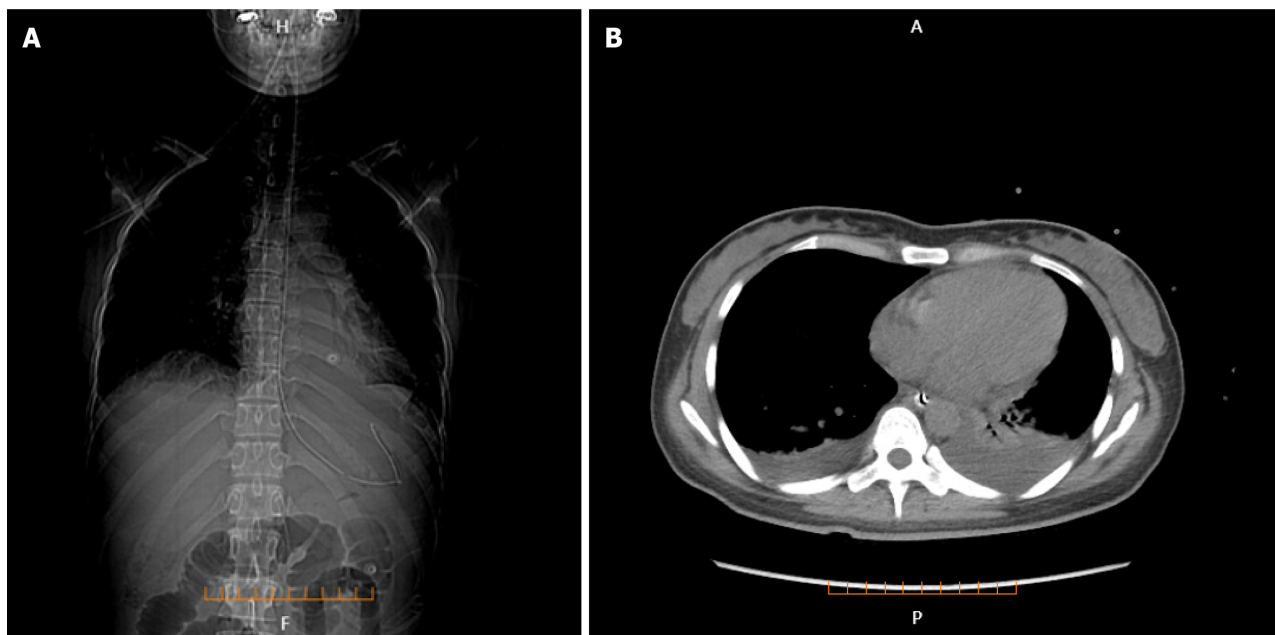
The pathophysiology of LMV is leukocytoclastic vasculitis secondary to the deposition of immune complexes in vascular walls, and antiphospholipid antibody-associated vasculopathy leading to thrombosis[8]. Ischemia causes the greatest damage to the mucosal mesenteric vessels, followed by the vasculature of the muscularis propria, submucosa, and plasma membrane[6]. Certain triggering factors (bacterial or viral infections, chemicals, *etc.*) can activate endothelial cells and expose them to cryptic antigens, resulting in the production of anti-endothelial cell antibodies, activation of complement, and deposition of immune complexes on inflamed blood vessel walls, which can lead to vascular damage and thrombosis[6]. The literature reported that vasculitis was histologically confirmed by biopsy samples of the affected intestinal segments[9]. The submucosa, filled with a mild diffuse inflammatory infiltrate of mononuclear cells, becomes edematous, and hemorrhage can be observed mostly in the muscular and subserosal layers; fibrinoid necrosis, leukocytoclasia on the vascular wall, and fibrin thrombus formation can be observed in the subserosal vessels.

The initial presenting signs and symptoms of LE are heterogeneous and commonplace and are usually mistaken for GI infections, peptic ulcers, lithiasis cholecystitis, or side effects of medications. However, the most common symptom in more than 90% of patients is abdominal pain of varying intensity caused by intestinal ischemia secondary to vasculitis [10]. Signs and symptoms of impaired GI motility and peritonitis are also common in patients with SLE. Diarrhea was frequent, with a frequency of 43.5%–64.7% in different studies[11]. Vomiting was found among 39.5%–72.2% of Asian patients[12]. Ascites was also frequently reported in the literature (27.9%–94.1%). This was also observed in our patient. GI bleeding is a rare but serious presentation that may be due to intestinal ischemia and intestinal necrosis. If not recognized in time, it may lead to perforation with a mortality rate of up to 50% [13]. The ileum and jejunum were the most frequently involved intestinal sites (> 80%) reported in the literature[14].

Timely and accurate diagnosis of LMV is a great challenge due to the lack of knowledge about LMV. Therefore, it is necessary to explore the factors associated with LMV.

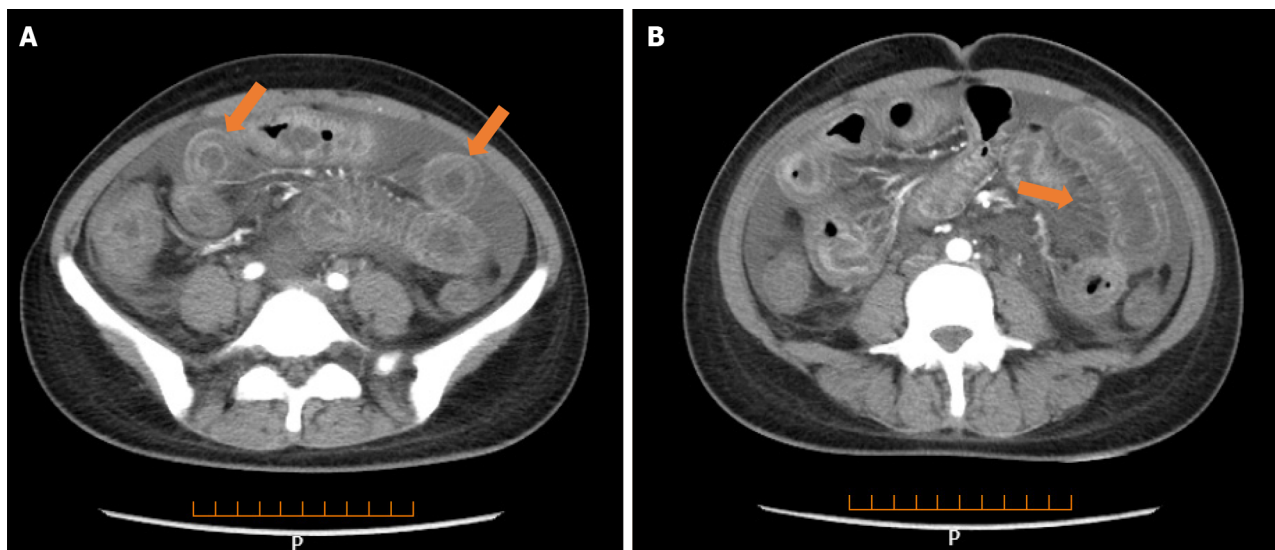
Several studies have found that patients accompanied by oral ulcers or lupus urinary tract involvement have a greater risk of encountering LMV[15–17]. Therefore, clinicians should suspect LMV when clinical laboratory results suggest lupus involvement of oral ulcers and the urinary system.

Previous studies have shown that there is no significant difference in SLEDAI scores in patients with SLE presenting with abdominal pain, regardless of the presence or absence of LE[14]. In patients with LMV, there was no difference in SLEDAI scores for serious adverse events, such as intestinal hemorrhage, infarction, perforation, or death due to serious complications, suggesting that SLE activity does not correlate with the severity of LMV[9]. Although SLEDAI was higher



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Figure 1 Lung computed tomography. A: Bilateral pleural effusion and the position of gastrointestinal decompression tube (coronal plane); B: Bilateral pleural effusion (axial plane).



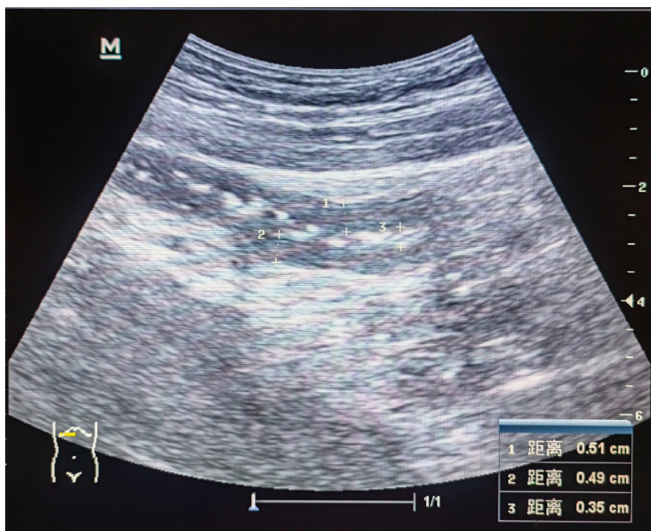
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Figure 2 Full enhanced abdominal computed tomography. A: Thickened bowel loops (target sign); B: Engorgement and increased visibility of the mesenteric vessels (comb sign).

at baseline in LE patients than in non-LE patients, it did not predict the development of LE[18].

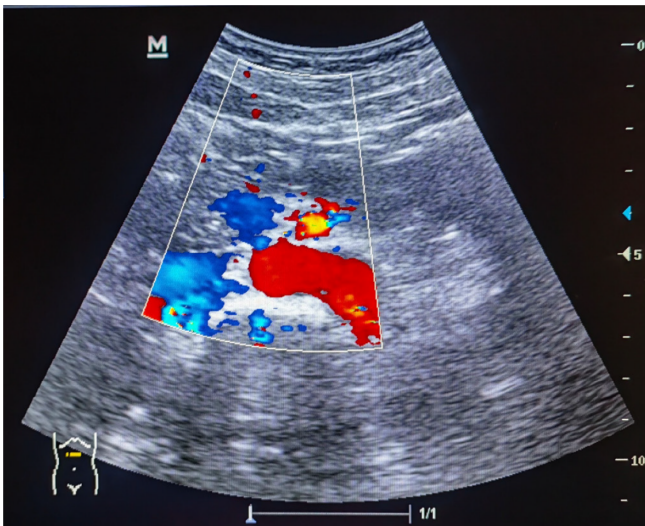
Laboratory tests are of value in the diagnosis of LE. However, CRP is usually normal and complement levels are decreased[7]. In seropositive cases, antibodies were more reliable, with increased titers of antinuclear, anti-dsDNA, and anti-Smith antibodies, which were approximately 92%, 80%, and 20%, respectively[19]. These autoimmune and inflammatory markers, while informative in diagnosis, are not essential. LE can occur without clinically active lupus[7]. Other researchers did not find laboratory parameters, including complement, ESR, CRP, lupus-related antibodies, and antiphospholipid antibodies to be correlated with the risk of LE[8,14].

Multiple methods, including ultrasonography, CT, magnetic resonance imaging, and GI endoscopy, have been used to evaluate LMV. However, CT is fast and non-invasive and can be used to perform image reconstruction and post-processing, to observe the LMV in multiple directions, and to observe lesions of the mesentery and blood vessels. Enhanced abdominal CT is more sensitive in detecting intestinal abnormalities, as it can reveal thickened and swollen bowel walls that contain enhanced mucosal, edematous submucosal, and enhanced serosal layers. Normal bowel wall thickness visualized by CT was < 3 mm; 3–5 mm thick bowel walls were considered mildly thickened, 6–7 mm



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Figure 3 Ultrasonography of the transverse colon showing thickened segmental bowel walls of approximately 5 mm.



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Figure 4 Ultrasonography of the superior mesenteric artery showing the main color flow of the superior mesenteric artery was well-filled without embolism.

moderately thickened, and > 8 mm markedly thickened[20]. This categorization is important in judging the occurrence of LE[9,21]. There are three typical abdominal CT findings in patients with LE, and these can appear alone or concurrently: Bowel wall thickening (> 3.0 mm), which leads to separation of the mucosa and muscle layers and “target sign” appearance; mesenteric vasodilation with “comb sign” appearance; and increased attenuation of mesenteric fat[15,19,22-25]. The co-occurrence of “target sign” and “comb sign” is particularly specific to LE, and can be used to establish a diagnosis. Unfortunately, these imaging manifestations are not uncommon in inflammatory bowel disease, intestinal ischemia, and mesenteric vein thrombosis.

Magnetic resonance enterography can also be used to diagnose LE. The main advantages of magnetic resonance enterography are that it allows for a holistic assessment of the bowel, both intestinal and extra-intestinal, as well as a high degree of safety as the examination can be repeated in a short period of time[26]. However, the slightly longer scan time, the radiologist's manipulation, and the patient's tolerance of the contrast agent may limit magnetic resonance enterography.

Ultrasonography is likely to be the most readily available imaging method, and a valuable alternative to CT when the latter is not available or is contraindicated. Being a safe and more accessible technique, ultrasonography was the first imaging examination when our patient presented to the emergency room. The study[27] have described patients with LE who had characteristic intestinal wall edema and ascites based on ultrasonography. Ultrasonography revealed thickening of the intestinal wall, in which the submucosal edema of the Kerckring fold resembled an accordion[28], dilation of intestinal segments, increased reflectivity of mesenteric fat, and mild ascites. CT confirmed these features, suggesting that



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Figure 5 Ultrasonography showing the local intestinal wall with approximately 0.35 cm at its thickest part.

both methods have similar sensitivities for LE diagnosis. Regarding specificity, ultrasonography has similar limitations with CT. If the patient is a young woman of childbearing age, ultrasonography is likely to be the best screening examination for suspected LMV diagnosis and follow-up[14]. Therefore, ultrasonography is a reliable and accessible tool.

Although the gold standard for diagnosing LE is pathology, the positive rate is low. Therefore, endoscopy is not used as a routine diagnostic tool. Only a few cases present intestinal edema, congestion and ischemia with or without ulceration or necrosis[7].

Due to the lack of prospective randomized controlled clinical trials, there are currently no available guidelines or recommendations for LE treatment.

The prognosis is good when the diagnosis is made early, and high-dose steroids are started in a timely manner; however, patients may occasionally experience recurrence. Glucocorticoids are the primary medications used to treat LE. Cyclophosphamide is one of the most commonly used immunosuppressants used for SLE treatment in China, and its combination with corticosteroids results in a good response[29]. Cyclophosphamide, whether intravenous pulse or not, has been useful in preventing relapses of lupus flares[2]. However, considering its lower reproductive toxicity, we switched to MMF treatment, which is preferred in young men and women due to the high risk of testicular and ovarian failure following cyclophosphamide[30]. Although rituximab, an anti-CD20 antibody, showed promising potential in observational studies, the LUNAR randomized controlled trials did not show that rituximab combined with MMF had additional benefits during remission compared with MMF alone[31].

LMV causes inflammation in the intestinal wall, which enhances its permeability. Dysregulation of the intestinal microbiota can lead to intestinal infection. Antibiotics were administered to control the risk of infection. Octreotide treatment may be effective, owing to its immunomodulatory effects, regulation of intestinal microvasculature blood flow, and amelioration of lymphatic dilatation[32]. Considering the aforementioned effects of somatostatin, we administered it to the patient and good outcomes were achieved.

CONCLUSION

Despite being rare, LMV must always be considered in any SLE patient presenting with GI symptoms. The diagnosis of LMV requires a combination of history and clinical and immunological indicators. Moreover, enhanced CT examination can reveal the extent and scope of GI lesions involved in SLE and provide evidence for timely clinical diagnosis and treatment. For pregnant women, or physicians in primary hospitals, ultrasonography can be an alternative method for preliminary screening and follow-up.

ACKNOWLEDGEMENTS

We are grateful to the patient for permitting us to publish this study.

FOOTNOTES

Author contributions: Huang H and Li P contributed to manuscript drafting; Zhang MX was the physician responsible for the patient's

diagnosis and treatment in the Rheumatology and Immunology Department; Zhang D was the patient's nutritionist and provided nutritional guidance; Yu K was the patient's admitting physician in the Gastroenterology Department; All authors were responsible for the revision of the manuscript and final approval for submission.

Informed consent statement: Consent was obtained from the patient, both verbally and in written form and has been attached to this submission.

Conflict-of-interest statement: The authors declare no conflicts of interest.

CARE Checklist (2016) statement: The authors have read the CARE Checklist (2016), and the manuscript was prepared and revised according to the CARE Checklist (2016).

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S-Editor: Lin C

L-Editor: A

P-Editor: Guo X

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