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Early Intervention and Resolution of Pediatric Intestinal Pseudo-Obstruction in Systemic Lupus Erythematosus: A Pediatric Case Report

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Pediatric intestinal pseudo-obstruction (PIPO) is a rare condition characterized by severe dysmotility. PIPO presents with signs and symptoms of bowel obstruction in the absence of anatomic blockage and can result from damage to the enteric nervous system, smooth muscle, and/or interstitial cells of Cajal. While the course is variable, fulminant cases result in long-term dependence on parenteral nutrition (PN) support, making chronic dysmotility the second most common cause of intestinal failure (IF) after short bowel syndrome (1).

Chronic intestinal pseudo-obstruction (CIPO) has been reported in the adult literature as an under-recognized manifestation of systemic lupus erythematosus (SLE). The pediatric incidence is not known (2–5). Early identification is essential since early intervention may improve prognosis. The window to treat early signs of dysmotility and prevent PIPO has not been reported.

We report a case of PIPO as the initial presentation of SLE in a previously healthy child who exhibited dramatic recovery with plasmapheresis, rituximab, and cyclophosphamide. To our knowledge, this is the youngest reported case of dysmotility secondary to SLE. This case highlights the importance of early recognition and treatment of PIPO to prevent IF and reduce disease-related morbidity.

CASE PRESENTATION

A 7-year-old Pacific Islander female presented with abdominal pain, fatigue, constipation, ageusia, anorexia, and nonbloody, nonbilious emesis. Figure 1 summarizes her clinical course. The onset was insidious, developing over weeks, and preceded by intermittent low-grade fevers and sore throat. Physical examination was remarkable for hypoactive bowel sounds and hepatomegaly without distension, ascites, or splenomegaly. She had lost 8 kg at presentation (weight z-score 1.07 before illness and nadir weight z-score of −2.27).

Laboratory analysis was significant for elevated aspartate and alanine transaminase (29–108 units/L and 39–220 units/L, respectively), hypoalbuminemia (3.4 g/dL) without cholestasis, liver synthetic dysfunction, or coagulopathy. She had a normocytic anemia (hemoglobin 10.6 g/dL), leukopenia (2.8 K/μL), and lymphopenia (1300/mm^2). Erythrocyte sedimentation rate (14–27 mm/hr) and immunoglobulin G level were elevated (1430 mg/dL). In the evaluation for autoimmune hepatitis, she had a positive antinuclear antibody (>1:2560) and negative anti-liver-kidney microsomal and antismooth muscle antibodies.

Postviral dysmotility was considered because Epstein Barr Virus serologies were suggestive of remote infection without reactivation. A diagnosis of SLE was established with positive antinuclear antibody, anti-Smith (92 U/ml), antiribonucleoprotein (>100 U/mL), hypocomplementemia (C3 71 mg/dL and C4 10 mg/dL), hematologic abnormalities, a small pericardial effusion on echocardiogram, positive anticardiolipin antibodies (33 IgG Phospholipid Units), antineuronal cell antibody (>400 Units), and elevated rheumatoid factor (>100 IU/mL). A paraneoplastic evaluation and anti-dsDNA antibodies were negative.

Abdominal ultrasound, elastography, and chest computed tomography were normal. Abdominal radiographs showed air fluid levels without mechanical obstruction. Magnetic resonance imaging with angiography of the abdomen showed large stool burden in the colon and gas paucity. Upper gastrointestinal fluoroscopy wasn't performed as she was unable to swallow contrast. Upper endoscopy showed scant antral erythema that was *Helicobacter pylori* negative.

She developed intractable bilious emesis and ileus, becoming dependent on PN. PIPO secondary to SLE was suspected. Pulse therapy with methylprednisolone 30 mg/kg/d was administered for 3 days, followed by maintenance dosing of 1 mg/kg/d twice daily. Erythromycin and amoxicillin/clavulanic acid were initiated as promotility agents. Despite this, her IF progressed.

Her severe, refractory symptoms prompted escalation of therapy, targeting clearance of circulating antibodies and immune complexes, and suppression of antibody production, to prevent ongoing end-organ damage. She received rituximab 500 mg/m^2 on day 25 and therapeutic plasma exchange (TPE) on day 26. She developed bowel sounds 1 day after TPE and 2 days later had a bowel movement for the first time in 16 days.

The patient's condition and laboratory markers improved significantly following this approach. In total, she received seven sessions of TPE and 2 doses of rituximab for induction followed by a 6-month regimen of monthly intravenous cyclophosphamide 500 mg/m^2. Enteral feeds were initiated and PN weaned, and she was discharged on oral feeding. As an outpatient, she successfully weaned off corticosteroids, and is on mycophenolate mofetil as maintenance therapy. She is currently maintaining adequate growth on oral feeding.

DISCUSSION

This is the first report to demonstrate prevention of chronic IF in a child with PIPO caused by SLE using TPE and immunosuppression. In patients with PIPO on lifelong PN, a cause is not frequently identified. Although an association between SLE and CIPO has been established in adults, SLE-induced PIPO has rarely

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Figure 1. Patient's clinical course.

TABLE 1. Multisystem involvement in previously described patients with CIPO and SLE

Study ID	Age (y)/ gender	Systems involved	ANA	Anti-dsDNA antibody (IU/ml)	Anti-ENA antibody	Treatment	Outcomes
Yamazaki-Nakashimada et al (2)	10/M	Gastrointestinal Renal Hematologic Neurologic	1:160	−	−	Laparotomy, Aspirin, IVIG, MP, CTX	Clinical improvement
	15/F	Gastrointestinal Renal Hematologic Neurologic	1:160	−	Anti-SM Anti-RNP	Laparotomy, MP, CTX	Clinical improvement
Munyard and Jaswon (3)	15/F	Gastrointestinal Hematologic Cardiac	NR	220	NR	1. Laparotomy and Pred 2. MP 500 mg × 3 d and HCQ 200 mg BID 3. Oral steroids	Clinical improvement
Tanaka et al (4)	11/F	Gastrointestinal Hematologic Renal	1:60	−	−	1. Pred 60 mg 2. CTX 70 mg × 12 weeks 3. AZA 70 mg daily and Pred wean to 10 mg daily 4. MP 40 mg × 3 d 5. Pred 20 mg	Clinical improvement followed by relapse of abdominal pain, pollakiuria and thrombocytopenia when prednisone was decreased to 20 mg daily
Bader-Meunier et al (5)	2 pediatric patients	NR	NR	NR	NR	NR	NR
Perlemuter et al (6)	19/F	Gastrointestinal Urological	1:320	−	Anti-RNP	1. Pred 1 mg/kg/d 2. MP 1 g/d 3. Pred 1 mg/kg/d and Octreotide 50 μg/d	Complete remission at 48 mo
Perlemuter et al (6); Hill et al (7)	28/F	Gastrointestinal Musculoskeletal Cardiac Neurologic Urologic	1:4000	1000	Anti-SM	1. Pred10 mg/d and HCQ sulfate 200 mg/d 2. MP 1 g/d 3. Pred 1 mg/kg/d	Complete remission at 48 mo
	29/F	Gastrointestinal Musculoskeletal	1:100	−	Anti-RNP	1. MP 1 g/d 2. Pred 1 mg/kg/d and AZA 2 mg/kg/d	Complete remission at 48 mo
	34/F	Gastrointestinal Rheumatologic Neurologic Urologic	1:400	−	−	1. Pred 5 mg/d, Erythromycin 750 mg/d, Cisapride 100 mg/d After developing systemic disease: 1. MP 1 g/d 2. Pred 1 mg/kg/d and CTX	Death at 5 mo
	29/F	Gastrointestinal Musculoskeletal Cardiac Urologic	1:5000	80	Anti-RNP	Cisapride 30 mg/d and Pred 1 mg/kg/d	Complete remission at 60 mo
	38F	Gastrointestinal Musculoskeletal Renal	1:2560	57	Anti-RNP Anti-SM	1. Pred and HCQ 2. MP 500 mg 3. Pred 50 mg/d, HCQ 200 mg and CTX 1200 and 900 mg 4. Cyclosporin A, Azathioprine, and Pred 5. Metronidazole for *Clostridium difficile*	Had extensive ileocolic resection due to ischemia. Symptoms resolved at 3-mo follow-up
Pardos-Gea et al (8)	57/F	Gastrointestinal Musculoskeletal Renal	1:2560	1440	NR	1. MP 1 g/d × 3 d and CTX 1200 mg/d × 1 d 2. Pred 40 mg/d, prokinetic agents and antibiotics 3. CTX monthly and Pred 10 mg/d 4. MMF 1 g/d	Clinical remission at 2-y follow-up. Has persistent common biliary tract and pancreatic duct dilatation
Ceccato et al (9)	24/F	Gastrointestinal Musculoskeletal	+	−	Anti-Ro Anti-La Anti-SM	CTX	Clinical improvement

(*Continued*)

TABLE 1. (*Continued*)

Study ID	Age (y)/ gender	Systems involved	ANA	Anti-dsDNA antibody (IU/ml)	Anti-ENA antibody	Treatment	Outcomes
Ceccato et al (9); Khairullah et al (10)	21/F	Gastrointestinal Musculoskeletal Renal	+	+	Anti-SM	CTX, Metoclopramide, and octreotide	Clinical improvement
	25/F	Gastrointestinal Musculoskeletal Renal	+	+	Anti-Ro Anti-La	CTX, MP, prokinetic agents	Clinical improvement
	49/F	Gastrointestinal Musculoskeletal	+	−	Anti-RNP	MP 1 g/d × 3 d	Clinical improvement
	42/F	Gastrointestinal Musculoskeletal renal	+	NR	Anti-SSA Anti-SSB	1. High-dose steroids 2. Steroid taper and HCQ 200 mg daily 3. Azathioprine 25 mg daily 4. Steroids (due to nonadherence)	Clinical improvement
Wang et al (11)	49/F	Gastrointestinal Renal	1:320	−	Anti-SSA Anti-SSB	MP × 5 d	Clinical improvement
Alexopoulou et al (12)	32/F	Gastrointestinal Musculoskeletal Hematologic Renal	>1:640	37	Anti-RNP	1. Pred 1 mg/kg/d and CTX 20 mg/kg monthly 2. Steroid taper	Clinical improvement
Chen et al (13)	47/F	Gastrointestinal Renal	1:2560	155.5	Anti-RNP Anti-SSA Anti-SSB	1. Pred 1 mg/kg and Hydrochloroquine 400 mg daily 2. Pred 15 mg daily	Clinical improvement
Chen et al (14)	24/F	Gastrointestinal Renal Hematologic	1:3200	NR	−	1. MP 500 mg daily × 2 d 2. MP 160 mg daily, Neostigmine, Octreotide 3. MP 200 mg and IVIG 0.4 g/kg × 7 d 4. MP 40 mg daily and CTX 600 mg monthly	Clinical improvement
Garcia Lopez et al (15)	27/F	Gastrointestinal Musculoskeletal Hematologic Renal Neurologic	−	29	Anti-SM Anti-Ro	1. MP 1000 mg/d × 5 d 2. Pred and erythromycin 3. IVIG 400 mg/kg/d × 5 d 4. MMF and Pred	Clinical improvement
Garcia Lopez et al (15); Kansal et al (16)	23/F	Gastrointestinal Hematologic Renal Neurologic	−	34	Anti-Ro	1. Exploratory laparotomy 2. MP 1000 mg/d × 3 d 3. IVIG 400 mg/kg/d 4. CTX monthly and Pred daily	Clinical improvement
	25/F	Gastrointestinal Musculoskeletal Hematologic Renal	−	40	−	1. MP 1000 mg/d × 3 d 2. Pred daily and CTX 1 g/m^2 monthly	Abdominal pain recurred after 2 mo and managed similarly
	37/F	Gastrointestinal Musculoskeletal Hematologic Renal	NR	NR	−	Pred 1 mg/kg daily	Experienced occasional GI symptoms and lost to follow-up
	27/F	Gastrointestinal Musculoskeletal Hematologic Neurologic	+	104.2	Anti-Ro	1. MP 500 mg × 3 d and CTX 500 mg biweekly 2. Pred 1 mg/kg/d and HCQ 200 mg daily	Clinical improvement
Khairullah et al (10)	42/F	Gastrointestinal Hematologic Renal	+	NR	Anti-SSA Anti-SSB	1. High-dose steroids 2. HCQ 200 mg daily 3. AZA 25 mg daily	Clinical improvement
Kim and Kim (17)	20/M	Gastrointestinal Renal	1:320	NR	Anti-SM Anti-Ro	1. Pred 30 mg/d × 3 d 2. Subtotal colectomy 3. Steroids and AZA	Clinical improvement
Leonardi et al (18)	51/F	Gastrointestinal Renal	NR	NR	NR	1. CTX 5 mg/kg and Rifaximin 600 mg daily 2. Octreotide 50 mg, Clarithromycin 500 mg, Rifaximin 1200 mg, and AZA 100 mg daily 3. MP 500 mg daily × 3 d 4. Steroid taper	Clinical improvement

(*Continued*)

TABLE 1. (*Continued*)

Study ID	Age (y)/ gender	Systems involved	ANA	Anti-dsDNA antibody (IU/ml)	Anti-ENA antibody	Treatment	Outcomes
Luman et al (19)	22/F	Gastrointestinal Hematologic Renal	1:640	+	NR	1. Laparotomy 2. Steroids and antituberculosis therapy 3. AZA 50 mg and Pred 12.5 mg daily	Chronic constipation requiring laxatives and rectal enemas
Maruoka et al (20)	23/F	Gastrointestinal Renal	1:160	NR	NR	1. Steroids, AZA and CTX 2. Tacrolimus	Clinical improvement
Mok et al (21)	36/F	Gastrointestinal Hematologic Renal	1:360	118	Anti-Ro	1. Laparotomy 2. MP 45 mg daily	Readmitted for fungal peritonitis and died from multiorgan failure
	21/F	Gastrointestinal Hematologic Renal	1:360	112	Anti-Ro	1. MP 40 mg 2. AZA 50 mg daily	Recurrence of steroid-responsive intestinal pseudo-obstruction
	42/F	Gastrointestinal Hematologic Renal Neurologic	1:360	116	Anti-Ro	1. Laparotomy 2. MP 1 g daily × 3 d 3. Pred 40 mg and CTX 100 mg 4. Cisapride 5 mg TID and AZA 75 mg daily	Recurrence of steroid-responsive intestinal pseudo-obstruction
	25/F	Gastrointestinal Musculoskeletal Hematologic	1:1080	137	Anti-Ro Anti-RNP	1. Laparotomy 2. Pred 40 mg daily	Clinical improvement with intermittent dysuria
	18/F	Gastrointestinal Hematologic Renal	1:160	400	NR	1. MP 80 mg daily	Clinical improvement with one recurrence of steroid-responsive intestinal pseudo-obstruction
	22/M	Gastrointestinal Musculoskeletal Hematologic Renal	1:360	NR	Anti-Ro	1. Pred 50 mg and HCQ 200 mg daily	Clinical improvement with one recurrence of steroid-responsive intestinal pseudo-obstruction
Narvaez et al (22)	44/F	Gastrointestinal Musculoskeletal Hematologic	+	1536	−	1. MP 1 mg/kg/d, Cisapride and erythryomycin × 6 d	Lost to follow-up
Nguyen et al (23)	35/F	Gastrointestinal Hematologic Renal	1:640	−	Anti-SM	1. High dose Pred and erythromycin 2. Pred 10 mg/d	Clinical improvement
Oh et al (24)	43/F	Gastrointestinal Hematologic	1:1280	NR	Anti-Ro Anti-La Anti-SM	1. Prednisolone 40 mg/d × 7 d 2. Pyridostigmine 60 mg/d	Later developed oral ulcers and arthralgia and diagnosed with SLE
Park et al (25)	46/F	Gastrointestinal Renal Neurologic				1. Tegaserod maleate, neostigmine, metoclopramide and erythromycin 2. CTX 3. MP and MMF	Persistent dysfunction of intestinal pseudo-obstruction, uterohydronephrosis and hepatobiliary dilatation
Shapeero et al (26)	21/M	Gastrointestinal Musculoskeletal	NR	NR	NR	1. ACTH	Clinical improvement
Shapeero et al (26); Wang et al (27)	23/F	Gastrointestinal Musculoskeletal Hematologic Renal	+	NR	NR	1. Steroids	Clinical improvement
	42/F	Gastrointestinal	1:400	+	NR	1. MP × 3 d 2. Pred 1.5 mg/kg/d and tapered off	Clinical improvement
Zhang et al (28)	31/F	Gastrointestinal Cardiac Serosal Renal	1:320	+	NR	1. Laparotomy 2. MP, IVIG, Imipenem-cilastatin	Clinical improvement
Zhang et al (29)	18/F	Gastrointestinal Hematologic Serosal Renal Mucocutaneous	1:320	+	Anti-RNP Anti-SSA Anti-SSB	1. MP 2 mg/kg/d × 3 d 2. Pred 1 mg/kg	Clinical improvement

(*Continued*)

TABLE 1. (*Continued*)

Study ID	Age (y)/ gender	Systems involved	ANA	Anti-dsDNA antibody (IU/ml)	Anti-ENA antibody	Treatment	Outcomes
Zhang et al (29)	48/F	Gastrointestinal Serosal Renal	1:3200	NR	Anti-Sm Anti-RNP Anti-SSA Anti-SSB	1. MP 1.5 mg/kg/d × 7 d 2. Prednisone 1 mg/kg/d	Clinical improvement

AZA = azathioprine; ANA = antinuclear antibody; CIPO = chronic intestinal pseudo-obstruction; CTX = cyclophosphamide; F = female; HCQ = hydroxychloroquine; M = male; MMF = mycophenolate mofetil; MP = methylprednisolone; NR = not reported; Pred = prednisone; SLE = systemic lupus erythematosus.

been described (Table 1). Thus, the assessment of systemic autoimmune disorders should be considered early in the evaluation of PIPO, since early recognition and treatment can rapidly reverse acute IF and prevent chronic disability as we demonstrate here (4–7,30).

The pathophysiology of PIPO in SLE is unknown. We hypothesize that SLE led to antibody-mediated PIPO in our patient. This could explain why she was refractory to corticosteroids, but demonstrated dramatic improvement with TPE. The combination of TPE, rituximab, and cyclophosphamide has not been described in SLE-associated dysmotility, but we utilized this approach given the severity of her presentation and narrow therapeutic window opportunity needed to intervene successfully.

Limitations to our report include that this is a single case study and our inability to complete motility studies. As such, our results may be not generalizable. Our patient may have improved without aggressive treatment, as steroids and one immunosuppressive agent has been effective in some adult and pediatric cases, but since she showed no functional bowel recovery on high dose steroids, we intensified her regimen with a goal of avoiding lifelong PN dependence. Despite these limitations, we hope this case will inform clinicians and researchers striving to prevent disability in this rare condition and guide future "bench-to-bedside" assessments.

CONCLUSION

PIPO is a severe condition that can result in lifelong dependence on PN. Although an etiology is not typically identified, we present a unique case of a pediatric patient with SLE-induced PIPO who showed rapid clinical improvement following treatment with TPE, rituximab, and cyclophosphamide. We suggest that the evaluation of patients with acute PIPO should include a workup for SLE and therapy directed against clearance of pathogenic antibodies and antibody mediated tissue injury could be considered if steroids are ineffective.

REFERENCES

1. Namjoshi SS, Muradian S, Bechtold H, et al. Nutrition deficiencies in children with intestinal failure receiving chronic parenteral nutrition. *JPEN J Parenter Enteral Nutr.* 2018;42:427–435.
2. Yamazaki-Nakashimada MA, Rodriguez-Jurado R, Ortega-Salgado A, et al. Intestinal pseudoobstruction associated with eosinophilic enteritis as the initial presentation of systemic lupus erythematosus in children. *J Pediatr Gastroenterol Nutr.* 2009;48:482–486.
3. Munyard P, Jaswon M. Systemic lupus erythematosus presenting as intestinal pseudo-obstruction. *J R Soc Med.* 1997;90:48–49.
4. Tanaka H, Waga S, Tateyama T, et al. Interstitial cystitis and ileus in pediatric-onset systemic lupus erythematosus. *Pediatr Nephrol Berl Ger.* 2000;14:859–861.
5. Bader-Meunier B, Armengaud JB, Haddad E, et al. Initial presentation of childhood-onset systemic lupus erythematosus: a French multicenter study. *J Pediatr.* 2005;146:648–653.
6. Perlemuter G, Chaussade S, Wechsler B, et al. Chronic intestinal pseudo-obstruction in systemic lupus erythematosus. *Gut*. 1998;43:117–122.
7. Hill PA, Dwyer KM, Power DA. Chronic intestinal pseudo-obstruction in systemic lupus erythematosus due to intestinal smooth muscle myopathy. *Lupus*. 2000;9:458–463.
8. Pardos-Gea J, Ordi-Ros J, Selva A, et al. Chronic intestinal pseudo-obstruction associated with biliary tract dilatation in a patient with systemic lupus erythematosus. *Lupus*. 2005;14:328–330.
9. Ceccato F, Salas A, Góngora V, et al. Chronic intestinal pseudo-obstruction in patients with systemic lupus erythematosus: report of four cases. *Clin Rheumatol*. 2008;27:399–402.
10. Khairullah S, Jasmin R, Yahya F, et al. Chronic intestinal pseudo-obstruction: a rare first manifestation of systemic lupus erythematosus. *Lupus*. 2013;22:957–960.
11. Wang R, Zheng B, Wang B, et al. A report of chronic intestinal pseudo-obstruction related to systemic lupus erythematosus. *Open Med*. 2018;13:562–564.
12. Alexopoulou A, Andrianakos A, Dourakis SP. Intestinal pseudo-obstruction and ureterohydronephrosis as the presenting manifestations of relapse in a lupus patient. *Lupus*. 2004;13:954–956.
13. Chen Y-Y, Yen H-H, Hsu Y-T. Intestinal pseudo-obstruction as the initial presentation of systemic lupus erythematosus: the need for enteroscopic evaluation. *Gastrointest Endosc*. 2005;62:984–987.
14. Chen YQ, Xue Q, Wang NS. Visceral muscle dysmotility syndrome in systemic lupus erythematosus: case report and review of the literature [published online ahead of print March 2012]. *Rheumatol Int*.
15. García López CA, Laredo-Sánchez F, Malagón-Rangel J, et al. Intestinal pseudo-obstruction in patients with systemic lupus erythematosus: a real diagnostic challenge. *World J Gastroenterol*. 2014;20:11443–11450.
16. Kansal A, Jain A, Thenozhi S, Agarwal V. Intestinal pseudo-obstruction associated with biliary tract dilatation in a patient with systemic lupus erythematosus. *Lupus*. 2013;22:87–91.
17. Kim J, Kim N. Intestinal pseudo-obstruction: initial manifestation of systemic lupus erythematosus. *J Neurogastroenterol Motil*. 2011;17:423–424.
18. Leonardi G, de Bortoli N, Bellini M, et al. Intestinal pseudo-obstruction in inactive systemic lupus erythematosus: an unusual finding. *World J Gastrointest Pharmacol Ther*. 2010;1:135–136.
19. Luman W, Chua KB, Cheong WK, et al. Gastrointestinal manifestations of systemic lupus erythematosus. *Singapore Med J*. 2001;42:380–384.
20. Maruoka H, Honda S, Takeo M, et al. Tacrolimus treatment for refractory lupus cystitis. *Mod Rheumatol*. 2006;16:264–266.
21. Mok M, Wong RWS, Lau CS. Intestinal pseudo-obstruction in systemic lupus erythematosus: an uncommon but important clinical manifestation. *Lupus*. 2000;9:11–18.
22. Narváez J, Pérez-Vega C, Castro-Bohorquez FJ, et al. Intestinal pseudo-obstruction in systemic lupus erythematosus. *Scand J Rheumatol*. 2003;32:191–195.
23. Nguyen H, Khanna N. Intestinal pseudo-obstruction as a presenting manifestation of systemic lupus erythematosus: case report and review of the literature. *South Med J*. 2004;97:186–189.
24. Oh DJ, Yang JN, Lim YJ, et al. Intestinal pseudo-obstruction as an initial manifestation of systemic lupus erythematosus. *Intest Res*. 2015;13:282–286.

25. Park F-D, Lee J-K, Madduri G-D, et al. Generalized megaviscera of lupus: refractory intestinal pseudo-obstruction, ureterohydronephrosis and megacholedochus. *World J Gastroenterol*. 2009;15:3555–3559.
26. Shapeero LG, Myers A, Oberkircher PE, et al. Acute reversible lupus vasculitis of the gastrointestinal tract. *Radiology*. 1974;112:569–574.
27. Wang J, Liu G, Liu T, et al.. Intestinal pseudo-obstruction in systemic lupus erythematosus: a case report and review of the literature. *Medicine (Baltimore)*. 2014;93:e248.
28. Zhang F-J, Zhang J, Zhou L-P, et al. Intestinal pseudo-obstruction as the initial manifestation of systemic lupus erythematosus. *Am J Emerg Med*. 2019;37:176.e1–176.e2.
29. Zhang J, Fang M, Wang Y, et al. Intestinal pseudo-obstruction syndrome in systemic lupus erythematosus. *Lupus*. 2011;20:1324–1328.
30. Richer O, Ulinski T, Lemelle I, et al. Abdominal manifestations in childhood-onset systemic lupus erythematosus. *Ann Rheum Dis*. 2007;66:174–178.