Gastrointestinal manifestations of systemic lupus erythematosus

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INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic inflammatory disease of unknown cause that can affect the skin, joints, kidneys, lungs, nervous system, serous membranes, and/or other organs of the body.

The gastrointestinal manifestations of SLE in adults will be reviewed here. An overview of other clinical manifestations as well as the treatment of SLE in adults are presented separately. (See "Overview of the management and prognosis of systemic lupus erythematosus in adults" and "Clinical manifestations and diagnosis of systemic lupus erythematosus in adults", section on 'Clinical manifestations'.)

EPIDEMIOLOGY

Gastrointestinal involvement is common in patients with systemic lupus erythematosus (SLE), and up to 40 percent of patients have gastrointestinal issues during their lifetime [1-5]. These manifestations may be due to manifestations of active disease like vasculitis, side-effects of the medications used to treat SLE, infection, or other intercurrent processes (eg, uremia).

ESOPHAGEAL INVOLVEMENT

Dysphagia is the most frequent gastrointestinal complaint in patients with systemic lupus erythematosus (SLE) and may occur in association with retrosternal chest pain, heartburn, regurgitation, or odynophagia. These symptoms may be due to an underlying esophageal motility disorder, concomitant gastroesophageal reflux disease, or other causes of esophagitis such as medication-induced esophagitis or Candida esophagitis.

Approximately 20 to 70 percent of patients with SLE have an underlying esophageal motility disorder [6-8]. Esophageal motility disorders do not appear to be associated with disease activity, duration, and treatment of SLE [9]. Although esophageal motility disorders have been associated with the Raynaud phenomenon and the presence of antiribonucleoprotein antibodies, it is unclear if this association is due to the presence of other connective tissue disorders [5]. The mechanism by which SLE leads to an esophageal motility disorder is not known. It may result from an inflammatory reaction in the esophageal muscles, or by ischemic or vasculitic changes to Auerbach's plexus [10,11]. (See "Distal esophageal spasm and hypercontractile esophagus".)

Patients with lupus who are being treated with immunosuppression are at increased risk of infectious esophagitis (eg, *Candida*, cytomegalovirus, herpes simplex). In addition, esophagitis may result from pill-induced esophageal mucosal injury. Other rare causes of esophagitis in patients with lupus include vasculitis-induced ischemia, which in rare cases can result in esophageal perforation [12]. (See "Pill esophagitis" and "Esophageal candidiasis in adults" and "Herpes simplex virus infection of the esophagus" and "Epidemiology, clinical manifestations, and treatment of cytomegalovirus infection in immunocompetent adults", section on 'Gastrointestinal manifestations' and "Overview of esophageal injury due to blunt or penetrating trauma in adults".)

GASTRIC INVOLVEMENT

Peptic ulcer disease — Patients with systemic lupus erythematosus (SLE) may present with epigastric pain or food-provoked epigastric discomfort and fullness, early satiety, and nausea, or they may be asymptomatic until they present with gastrointestinal bleeding, gastric outlet obstruction, penetration, fistulization, or perforation. (See "Peptic ulcer disease: Clinical manifestations and diagnosis", section on 'Clinical manifestations'.)

It is not known whether SLE confers an increased risk of peptic ulcer disease that is separate from other known risks such as nonsteroidal antiinflammatory drugs (NSAIDs) and *Helicobacter pylori* infection [7,13]. Although glucocorticoids alone are not associated with an increase in the

risk for peptic ulcer, they exacerbate NSAID-induced ulceration. (See "Peptic ulcer disease: Epidemiology, etiology, and pathogenesis", section on 'Etiology'.)

Prior to treatment with NSAIDs, testing for *H. pylori* may be considered in patients with SLE and particularly in patients expected to be taking NSAIDs continuously for several weeks. In addition, a proton pump inhibitor should be coadministered in patients who require an NSAID and are at high or moderate risk for gastrointestinal toxicity.

The diagnosis and management of peptic ulcer disease is the same as that for patients without SLE, and is discussed separately. (See "NSAIDs (including aspirin): Pathogenesis and risk factors for gastroduodenal toxicity" and "Major side effects of systemic glucocorticoids", section on 'Gastrointestinal effects' and "Indications and diagnostic tests for Helicobacter pylori infection in adults" and "Peptic ulcer disease: Clinical manifestations and diagnosis" and "Peptic ulcer disease: Treatment and secondary prevention".)

SMALL BOWEL AND COLON INVOLVEMENT

Intestinal pseudo-obstruction — Intestinal pseudo-obstruction is a rare complication of systemic lupus erythematosus (SLE) and is characterized by signs and symptoms of a mechanical obstruction of the small or large bowel in the absence of an anatomic lesion obstructing the flow of intestinal contents. It usually occurs in patients with active SLE, but can also be the initial presentation, though this is rare [14]. Patients with intestinal pseudoobstruction present with abdominal pain, bloating, and distension. Symptoms may be acute, recurrent, or chronic. The pathogenesis of SLE-related intestinal pseudo-obstruction is unknown, but possible mechanisms include immune complex deposition in smooth muscle cells, or vasculitis leading to chronic ischemia and hypomotility [15,16]. (See "Chronic intestinal pseudo-obstruction: Etiology, clinical manifestations, and diagnosis", section on 'Clinical manifestations'.)

Management includes treatment of the underlying SLE with high-dose steroids alone or in combination with other immunosuppressive agents such as azathioprine or cyclophosphamide [14,17,18]. Other measures, such as nutritional support and prokinetic agents used in other causes of intestinal pseudo-obstruction, may also be required. (See "Chronic intestinal pseudo-obstruction: Management", section on 'Initial management'.)

Protein-losing enteropathy — Protein-losing enteropathy is rare in patients with SLE and has been described in a number of small series [17,19-21]. It typically occurs in young women and is characterized by the onset of profound edema and hypoalbuminemia in the absence of

nephrotic range proteinuria. Severe diarrhea is present in 50 percent of patients. Protein-losing enteropathy typically occurs in patients with clinically severe SLE with multi-organ involvement [22].

The diagnosis of SLE-related protein-losing enteropathy depends on evidence of enteral protein loss with the exclusion of other causes of hypoalbuminemia such as proteinuria, liver disease, decreased protein synthesis, or protein malnutrition [17,19]. The clinical manifestations and diagnosis of protein-losing enteropathy are discussed separately. (See "Protein-losing gastroenteropathy".)

Management of protein-losing enteropathy involves maintenance of the nutritional status and treatment of the underlying SLE. Lupus-related protein-losing enteropathy typically responds well to treatment with glucocorticoids, although immunosuppressive drugs including azathioprine and cyclophosphamide have also been used [17,19,20,23,24]. (See "Overview of the management and prognosis of systemic lupus erythematosus in adults", section on 'Approach to drug therapy'.)

HEPATIC INVOLVEMENT

Liver test abnormalities in patients with systemic lupus erythematosus (SLE) are relatively common, but clinically significant liver disease is rare [25-29]. Hepatic involvement in patients with SLE may be due to a wide range of factors including drug-induced damage, steatosis, viral hepatitis, vascular thrombosis, overlap with autoimmune hepatitis, or SLE itself [29]. The following studies are illustrative:

- In a study including 206 SLE patients, 124 had at least one abnormal liver chemistry and 43 met strict criteria for the existence of liver disease [25]. In most cases, a specific viral or drug etiology could not be implicated. Liver biopsy results from 33 patients with persistent liver chemistry abnormalities found a number of different diagnoses, including cirrhosis (in four patients), chronic active hepatitis; granulomatous hepatitis, cholestasis, centrilobular necrosis, chronic persistent hepatitis, primary biliary cholangitis (previously referred to as primary biliary cirrhosis), steatosis; fatty liver, and drug toxicity. Of nine patients who had serial liver biopsies, four showed progression and three died.

- A chart review of 40 SLE patients with liver enzyme abnormalities who were evaluated by a gastroenterologist found that end-stage liver disease SLE patients is rare [27]. The major diagnostic categories included primary biliary cholangitis (n = 3), drug-induced liver enzyme abnormalities (n = 4), autoimmune hepatitis (n = 6), nonalcoholic fatty liver disease

(n = 8), viral hepatitis (n = 8), and miscellaneous hepatic diseases (n = 11). After a median follow-up period of 44 months, the estimated five-year serious liver disease-free survival was 93 percent.

Hepatic complications attributable to antiphospholipid antibodies can include Budd-Chiari syndrome, hepatic venoocclusive disease, nodular regenerative hyperplasia, liver infarction, and transient elevation of hepatic enzymes resulting from multiple fibrin thrombi [30]. The diagnosis and clinical manifestations of antiphospholipid syndrome is presented separately. (See "Diagnosis of antiphospholipid syndrome" and "Clinical manifestations of antiphospholipid syndrome", section on 'Gastrointestinal involvement'.)

Autoimmune hepatitis — The term "lupoid hepatitis" was formerly used to describe autoimmune hepatitis because of some clinical and serologic similarities to SLE. This has since been referred to as Type I autoimmune hepatitis and is a subset of chronic active hepatitis characterized by circulating antibodies to nuclei (ANA) and/or smooth muscle (ASMA). Autoimmune liver disease is relatively uncommon in patients with SLE. As an example, in a retrospective review of 377 adult patients with SLE, only five (1.3 percent) had histologically confirmed liver damage with autoimmune features [31]. Another retrospective analysis of 147 SLE patients found that 4.7 percent of patients were diagnosed with autoimmune hepatitis [32]. The clinical manifestations and diagnosis of autoimmune hepatitis are discussed in detail elsewhere. (See "Overview of autoimmune hepatitis".)

Lupus hepatitis — A distinct clinical entity referred to as lupus hepatitis is thought to exist, and it is described as an insidious, rarely acute onset of transaminase elevations in patients with a diagnosis of SLE and who frequently have a positive test for ribosomal P antibody and biopsy findings of lymphocytic infiltration of periportal areas with isolated areas of necrosis [33,34]. There appears to be an association of antiribosomal P antibody and SLE-associated hepatitis, but there is no evidence to date that it plays a pathogenetic role in the development of hepatitis [35]. Elevations in transaminases are typically less than two- to threefold, but, in severe cases, significant elevations (eg, greater than 10-fold) may be observed [33]. The symptoms of lupus hepatitis are usually nonspecific, such as fatigue, malaise, and anorexia. Severe cases may lead to jaundice, hepatomegaly, or ascites, and may occur in the setting of other organ-threatening SLE manifestations (see "Clinical manifestations and diagnosis of systemic lupus erythematosus in adults", section on 'Clinical manifestations'). As with autoimmune hepatitis, lupus hepatitis typically responds to treatment with glucocorticoids.

PANCREATIC INVOLVEMENT

Acute pancreatitis — Acute pancreatitis occurs in 2 to 8 percent of patients with systemic lupus erythematosus (SLE) [2,36-38]. Patients with SLE may have pancreatitis from any cause. However, in the majority of cases, the development of acute pancreatitis occurs in the setting of active SLE [17]. The presentation of pancreatitis does not differ from patients without SLE, and includes acute onset of severe persistent epigastric pain often radiating to the back and a three times or greater elevation in serum amylase and lipase. However, elevated levels of serum amylase have also been detected in patients with SLE in the absence of pancreatitis [36]. (See "Clinical manifestations and diagnosis of acute pancreatitis", section on 'Clinical features'.)

The pathogenetic mechanism of pancreatitis related to SLE is unknown, but vascular damage from vasculitis or thrombosis (often in association with antiphospholipid antibodies) has been proposed [39]. Risk factors to the development of pancreatitis, as in patients without SLE, include gallstones, alcohol use, infection, and hypertriglyceridemia [37]. Azathioprine has been implicated as a cause of drug-induced pancreatitis. However, azathioprine-induced pancreatitis is rare, and other causes of acute pancreatitis should be considered when attempting to establish the etiology [36,38]. As an example, a study of 35 SLE patients with 49 episodes of acute pancreatitis, the majority of cases were idiopathic and there was no evidence to suggest that the use of glucocorticoids or azathioprine were potential causes [38]. (See "Etiology of acute pancreatitis".)

The management of SLE-related pancreatitis, as with other causes of acute pancreatitis, is with supportive care including pain control, intravenous fluids especially during the first 24 hours, correction of electrolyte and metabolic abnormalities, and restriction of oral intake. While a few studies have suggested that the use of glucocorticoids to treat acute pancreatitis in patients with SLE has been associated with a reduction in mortality, we limit the use of systemic glucocorticoids to patients with acute pancreatitis and clear evidence of active SLE elsewhere [17]. (See "Management of acute pancreatitis".)

OTHER

Mesenteric vasculitis/ischemia — Mesenteric vasculitis is a rare but life-threatening complication of systemic lupus erythematosus (SLE) that can present insidiously with postprandial abdominal pain, food aversion, weight loss, nausea, vomiting, and diarrhea due to chronic mesenteric ischemia [2,4,40]. Patients with mesenteric thrombosis and infarction can present with the picture of an acute abdomen due to infarction, perforation, and peritonitis. Proposed pathogenetic mechanisms include vasculitis secondary to immune complex deposition and thrombosis of the small intestinal vessels secondary to circulating

antiphospholipid antibodies [41]. (See "Overview of gastrointestinal manifestations of vasculitis", section on 'Systemic lupus erythematosus'.)

During the early stages, plain radiographic studies may reveal nondiagnostic findings such as segmental bowel dilatation, air-fluid levels, "thumbprinting" or narrowing of the lumen, and pseudoobstruction. Abdominal computed tomography (CT) or magnetic resonance (MR) angiography scan may reveal dilated bowel loops, focal or diffuse bowel wall thickening, abnormal wall enhancement, mesenteric edema, stenosis or engorgement of mesenteric vessels [42]. (See "Overview of gastrointestinal manifestations of vasculitis", section on 'Vascular imaging'.)

In addition to complete bowel rest, the initial medical treatment of mesenteric vasculitis in SLE patients without perforation includes high-dose glucocorticoids to achieve disease control [2,43,44]. For patients who do not respond to intravenous glucocorticoids or have recurrent symptoms, intravenous cyclophosphamide should be added.

Surgery is usually reserved for patients with clinical signs and symptoms of advanced ischemia (eg, peritonitis, sepsis, pneumatosis) or those who fail to respond promptly to medical therapy [2]. Additional details regarding the management of intestinal ischemia can be found elsewhere. (See "Overview of intestinal ischemia in adults".)

Peritonitis and ascites — Primary peritonitis secondary to SLE is rare but can develop rapidly during a lupus flare. Patients can present with abdominal pain suspicious for a surgical abdomen, although symptoms may be masked by concurrent immunosuppressive use. Chronic lupus peritonitis usually presents with a gradually developing ascites that is painless [7].

Ascites is otherwise rare in patients with SLE. Other possible causes of ascites related to SLE include congestive heart failure and hypoalbuminemia secondary to nephrotic syndrome or protein-losing enteropathy. Abdominal paracentesis should be performed in patients with ascites to determine the underlying cause. An approach to determining the underlying cause of ascites is discussed separately. (See "Evaluation of adults with ascites", section on 'Determining the cause of the ascites'.)

Acute or chronic peritonitis generally responds to treatment with glucocorticoids. Other agents, including chloroquine, azathioprine, and cyclophosphamide, have also been used to treat lupus peritonitis [45-47]. (See "Overview of the management and prognosis of systemic lupus erythematosus in adults", section on 'Definitions'.)

SUMMARY AND RECOMMENDATIONS

- **Epidemiology** – Gastrointestinal involvement is common in patients with systemic lupus erythematosus (SLE), and up to 40 percent of patients have gastrointestinal manifestations during their lifetime. Symptoms may be nonspecific and may be due to underlying SLE, side-effects of the medications used to treat SLE, infection, or other intercurrent processes. (See 'Epidemiology' above.)

- **Dysphagia** – Dysphagia is the most frequent complaint in patients with SLE and may occur in association with retrosternal chest pain, heartburn, regurgitation or odynophagia. These symptoms may be due to an underlying esophageal motility disorder, concomitant gastroesophageal reflux disease, infectious (eg, *Candida*, cytomegalovirus, herpes simplex virus) or pill esophagitis. (See 'Esophageal involvement' above.)

- **Peptic ulcer disease** – Patients with SLE may present with symptoms of peptic ulcer disease, including epigastric pain or food-provoked epigastric discomfort and fullness, early satiety, and nausea, or may be asymptomatic until they present with ulcer complications. Prior to treatment with nonsteroidal antiinflammatory drugs (NSAIDs), testing for *Helicobacter pylori* should therefore be performed, particularly in patients expected to be taking NSAIDs continuously for several weeks. In addition, a proton pump inhibitor should be coadministered in patients who require an NSAID and are at high or moderate risk for gastrointestinal toxicity. (See 'Peptic ulcer disease' above.)

- **Pseudo-obstruction** – Intestinal pseudo-obstruction is characterized by signs and symptoms of a mechanical obstruction of the small or large bowel in the absence of an anatomic lesion obstructing the flow of intestinal contents. Patients present with abdominal pain, bloating, and distension. (See 'Intestinal pseudo-obstruction' above and "Chronic intestinal pseudo-obstruction: Etiology, clinical manifestations, and diagnosis".)

- **Protein-losing enteropathy** – Protein-losing enteropathy is characterized by the onset of profound edema and hypoalbuminemia in the absence of nephrotic range proteinuria. Severe diarrhea is present in 50 percent of patients. (See 'Protein-losing enteropathy' above and "Protein-losing gastroenteropathy".)

- **Pancreatitis** – Acute pancreatitis occurs in 2 to 8 percent of patients with SLE. Patients present with acute onset of severe persistent epigastric pain often radiating to the back, and an elevation in serum amylase and lipase. The management of SLE-related pancreatitis, as with other causes of acute pancreatitis, is with supportive care including pain control, intravenous fluids, correction of electrolyte and metabolic abnormalities, and restriction of oral intake. We limit the use of systemic glucocorticoids to patients with acute pancreatitis and clear evidence of active SLE elsewhere. (See 'Acute pancreatitis'

above and "Clinical manifestations and diagnosis of acute pancreatitis" and "Management of acute pancreatitis".)

- **Mesenteric vasculitis** – Mesenteric vasculitis is a life-threatening complication of SLE that can present insidiously with postprandial abdominal pain, food aversion, weight loss, nausea, vomiting, and diarrhea due to chronic mesenteric ischemia. Patients with mesenteric thrombosis and infarction can present with acute abdomen due to infarction, perforation, and peritonitis. The initial medical treatment of mesenteric vasculitis in SLE patients without perforation includes high-dose glucocorticoids to achieve disease control. For patients who do not respond to intravenous glucocorticoids or have recurrent symptoms, intravenous cyclophosphamide should be added. Surgery is usually reserved for patients with clinical signs and symptoms of advanced ischemia (eg, peritonitis, sepsis, pneumatosis) or those who fail to respond promptly to medical therapy. (See 'Mesenteric vasculitis/ischemia' above and "Overview of gastrointestinal manifestations of vasculitis".)

- **Peritonitis** – Primary peritonitis secondary to SLE is can develop rapidly during a lupus flare. Patients can present with abdominal pain suspicious for a surgical abdomen, although symptoms may be masked by concurrent immunosuppressive use. Chronic lupus peritonitis usually presents with a gradually developing ascites that is painless. Abdominal paracentesis should be performed in patients with ascites to determine the underlying cause. (See 'Peritonitis and ascites' above and "Evaluation of adults with ascites".)

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