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GASTROINTESTINAL AND HEPATIC MANIFESTATIONS OF SYSTEMIC LUPUS ERYTHEMATOSUS – A NARRATIVE REVIEW

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ABSTRACT

Introduction and purpose: Systemic lupus erythematosus is a chronic, multifaceted autoimmune disorder. Throughout its course, it affects multiple organ systems, such as the skin, joints, kidneys, cardiovascular system, and central nervous system, among others. Less frequently observed clinical presentations include gastrointestinal involvement and hepatic symptoms. The objective of this review is to present and familiarise readers with these atypical manifestations, which are mostly not included in the diagnostic criteria for lupus, and which can pose a significant diagnostic challenge.
Description of the state of knowledge: Gastrointestinal (GI) and hepatic symptoms among patients with lupus may be related to the disease itself, but can also result from the side effects of treatment, viral infections, or emerge as a consequence of the overlap of two diseases. The gastrointestinal tract may exhibit symptoms of the disease at practically any point along its course, from the oral cavity to the anus. The most common GI manifestations include conditions such as oral ulcers, dysphagia, lupus enteritis, protein-losing enteropathy, intestinal pseudo-obstruction, and pancreatitis. Rare, but clinically significant presentations of lupus involving the liver, which are important due to their chronic health consequences, include autoimmune hepatitis and lupus hepatitis.
Conclusions: Patients with systemic lupus erythematosus who present with gastrointestinal or hepatic symptoms require prompt diagnosis, as these conditions, if not properly managed, contribute to increased mortality. It is also possible for these manifestations to appear as the initial signs of SLE. Therefore, familiarity with their characteristics is crucial to avoid delays in diagnosis and in the implementation of appropriate treatment.

KEYWORDS

SLE, Systemic Lupus Erythematosus, Hepatic, Gastric, Gastrointestinal, Dysphagia

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Introduction

Systemic lupus erythematosus (SLE) is a complex autoimmune disorder of unknown etiology, leading to persistent inflammation and dysfunction of multiple organ systems. SLE arises from complex interactions between genetic susceptibility and environmental influences. The pathogenesis of the disease is driven by the production of antiphospholipid and antinuclear autoantibodies, including those highly specific for systemic lupus erythematosus, such as anti–double-stranded DNA and anti-Smith antibodies. Potentially almost any organ can be affected by lupus. The most common clinical manifestations are observed primarily in the skin, kidneys, joints, muscles, heamatological, cardiovascular or central nervous system. Treatment involves the use of non-steroidal anti-inflammatory drugs, hydroxychloroquine, corticosteroids, and either immunosuppressants or immunomodulators (Gajewski Piotr, 2024a; Kaul et al., 2016).

SLE can also present the gastrointestinal involvement – virtually each part of the tract, from the mouth to the anus, can be influenced. When gastrointestinal symptoms occur as early signs of lupus, the diagnosis may be challenging and subsequently postponed. They can be provoked by the disease per se, drug-induced effects, bacterial or viral infections (Alves et al., 2016). According to the literature, the prevalence of GI complaints among patients with SLE ranges from 42,5% to even more than 50% (Alharbi, 2022; Fawzy et al., 2016). Another manifestation of this disorder is the involvement of the liver. There are several causes of hepatic abnormalities, such as lupus hepatitis, hepatotoxicity of the drugs used for SLE therapy, autoimmune hepatitis (AIH), viral hepatitis or fatty liver. The distinction between primary and non-primary involvement of the liver in lupus is paramount for the diagnosis of lupus hepatitis, which occurs only after the exclusion of non-primary causes and other etiologies. Secondary manifestations are more prevalent (Tahernia et al., 2017). Data on the prevalence of hepatic dysfunctions varies across sources and includes values from 15% to 60% (Adiga & Nugent, 2017; Brewer & Kamen, 2018; González-Regueiro et al., 2020; Kheyami et al., 2024). SLE activity

concerning liver and gastrointestinal system is comparatively rare, however these manifestations can cause serious, life-threatening consequences if not treated promptly. Patients with hepatic and GI symptoms associated with lupus have higher likelihood and risk of mortality (Raymond et al., 2021).

This review aims to present the characteristics and diversity of gastrointestinal and hepatic symptoms in the course of systemic lupus erythematosus. Familiarity with these manifestations will aid in the timely diagnosis and initiation of appropriate treatment.

Materials and methods

We conducted a literature review from 2016 to 2025, focusing on the association between gastrointestinal and hepatic symptoms and systemic lupus erythematosus. Materials were retrieved from the PubMed database using selected keywords: lupus; SLE; systemic lupus erythematosus; hepatic; gastric; gastrointestinal; dysphagia; swallowing. The search yielded 1,206 results, of which 32 articles were included in the study after excluding those that did not meet the established criteria.

Description of the state of knowledge

Gastrointestinal system

Oral ulcers

Oral cavity lesions may occur with a frequency of approximately 7-52% (Alharbi, 2022; Halabi et al., 2021). It is important to note that they consitute the European League Against Rheumatism (EULAR)/American College of Rheumatology (ACR) dignostic citerion for SLE (Aringer et al., 2019). The hard palate and buccal mucosa are the most common locations for such lesions. The abnormalities may or may not cause pain, which can be probably attributed to distinct nature of alterations. Erythematous changes are typically painless, whereas discoid and ulcerative ones are more likely to be painful (Alharbi, 2022). Oral ulcers may be a consequence of SLE activity, however they may also result from the effects of rheumatological medication, such as NSAIDs, methotrexate, azathioprine, and corticosteroids (Halabi et al., 2021).

Esophageal manifestations

Dysphagia, heartburn and regurgitation are frequently reported complaints among patients with systemic lupus erythematosus. The incidence of dysphagia is estimated to be between 2% and 25% (Amos et al., 2016). The literature points to various reasons for this manifestation. One such condition is the aforementioned oral ulceration, which can affect swallowing. Gastroesophageal reflux disease (GERD), which occurs in 11%-50% of cases, may also play a role. Another cause of dysphagia is esophageal dysmotility, affecting 21%-72% of patients (Alharbi, 2022; Halabi et al., 2021). The pathophysiological basis of this phenomenon is not entirely explained. There are numerous factors that influence esophageal motility, such as muscle atrophy, ischaemic vasculitis, and inflammation in the esophageal muscles (Alharbi, 2022; Halabi et al., 2021). According to manometry studies, the upper third of the esophagus is the most hypokinetic part, while the lower esophageal sphincter demonstrates no alterations in function. Motility disorders can present with a wide range of manifestations, including esophageal epidermolysis bullosa acquisita, vagal nerve dysfunction, and esophageal spasms (Hegazy et al., 2023).

Lupus enteritis

Lupus enteritis (LE), also referred to as lupus mesenteric vasculitis or lupus arteritis, is a relatively rare manifestation of gastrointestinal involvement in SLE. This complication has been observed to affect between 0.2% and 5.8% of patients, with some sources reporting a higher incidence of up to 14% (Alves et al., 2016; Lee et al., 2016; Patnaik et al., 2024). In 2004 the definition of lupus enteritis appeared in the British Isles Lupus Assessment Group (BILAG) classification criteria. It is described as vasculitis or inflammation of the small intestine supported by imaging findings and/or biopsy, with emphasis on the wide spectrum of manifestations(Isenberg et al., 2005). The pathophysiological underpinnings of LE remain to be fully elucidated. The production of antiphospholipid antibodies, infections, NSAIDs, chemicals and herbal medicines are probable triggering factors (Chaparro et al., 2024; Muñoz-Urbano et al., 2024). They contribute to the development of inflammation within the wall of small blood vessels, which is associated with the deposition of immune complexes and the activation of the complement system. Often, multiple vascular areas are affected simultaneously (Chaparro et al., 2024). The clinical presentation of lupus enteritis is characterized by non-specific symptoms, which complicates early diagnosis. Furthermore, the use of glucocorticoids and immunosuppressants in affected patients may attenuate symptom severity and obscure the clinical picture of an acute abdomen. Common manifestations include sudden and severe abdominal pain, ascites, nausea, vomiting, fever, and diarrhea (Luís et al., 2019). Less frequent symptoms comprise anorexia, haematemesis,

postprandial fullness, and melena (Muñoz-Urbano et al., 2024). The gold standard for diagnosing LE is computed tomography. However, the absence of characteristic images for this condition often results in delayed diagnoses and subsequent complications, including bowel infarction with bleeding, perforation and peritonitis (Patnaik et al., 2024). The mortality rate among patients diagnosed with lupus and experiencing acute abdominal pain is 11%(Koo et al., 2015). Engorgement of the mesenteric vessels, dilated intestines, thickening of the intestinal wall are usually observed findings in the CT scan of patients with lupus enteritis (Alharbi, 2022; Frittoli et al., 2021).

Intestinal pseudo-obstruction

Intestinal pseudo-obstruction (IPO) is defined as ineffective bowel motility that presents with symptoms analogous to mechanical intestinal obstruction, yet without an identifiable organic basis (Alharbi, 2022). The pathophysiology of IPO is thought to be associated with vasculitis in the visceral smooth muscle, resulting in muscle damage and impaired intestinal motility. In addition to muscular involvement, the autonomic nervous system may also be implicated, with its dysfunction contributing to and exacerbating hypomotility (Brewer & Kamen, 2018; Zhang et al., 2016). This complication may occur subsequent to the diagnosis of lupus, yet in patients exhibiting IPO, it frequently constitutes the primary manifestation of SLE. According to Zhang et al., 57.6% of patients presented IPO as the first sign of the disease, and 78% of them were misdiagnosed (Zhang et al., 2016). The clinical picture of intestinal pseudo-obstruction encompasses symptoms such as abdominal pain, distension, nausea, vomiting, and weight loss. While some patients present with diarrhea, others may experience obstipation accompanied by absent peristaltic sounds. The diagnosis is also made on the basis of imaging modalities, such as abdominal X-ray or CT scan, which reveal gaseous dilation of the intestinal loops with air-fluid levels and thickening of the bowel wall (Alharbi, 2022; Naeem et al., 2023). It is noteworthy that IPO is frequently correlated with complications involving the urinary tract and bile ducts. According to a systematic review conducted by Li et al., 64.44% of patients with systemic lupus erythematosus-associated intestinal pseudo-obstruction (SLE-IPO) also experienced ureterohydronephrosis, while hepatobiliary dilation was observed in 17.36% of cases (Li et al., 2017). Moreover, a retrospective case-control study by Zhang et al. demonstrated that in 58.9% of cases, intestinal pseudo-obstruction co-occurred with pyeloureterectasis or megacholedochus (Zhang et al., 2016).

Protein-losing enteropathy

Protein-losing enteropathy (PLE) is a disorder associated with significant protein loss through the gastrointestinal tract, resulting in substantial hypoalbuminemia. The diagnosis is made when other potential causes of hypoalbuminemia have been ruled out, including renal protein loss, liver dysfunction, impaired absorption, and low protein supply. In instances where the serum albumin level is below 3.0g/l, and protein excretion in the urine is within standard limits, PLE can be considered (Abu Jheasha et al., 2024; Williamson et al., 2024). According to a ten-year retrospective analysis conducted by Chinese researchers, the incidence of protein-losing enteropathy was reported to be 7.5% (Brewer & Kamen, 2018; Law et al., 2012). The majority of patients with PLE exhibit marked peripheral oedema, ascites, pleural effusion or, less frequently, pericardial effusion. These manifestations are causally linked to hypoalbuminemia. Other symptoms, although less prevalent, include diarrhoea, nausea and vomiting, fever, and abdominal pain (Li et al., 2017). The pathogenesis of protein-losing enteropathy is likely associated with increased intestinal wall permeability to proteins. This phenomenon may result from mesenteric or intestinal arterial vasculitis, vasodilation linked to cytokine-mediated mucosal injury, or intestinal lymphangiectasia (Brewer & Kamen, 2018). The fecal alpha-1-antitrypsin clearance test or 99mTc-Human Albumin Scintigraphy are the most commonly employed diagnostic methods to confirm the diagnosis of protein loss through the gastrointestinal tract (Law et al., 2012; Williamson et al., 2024).

Pancreatitis

Acute lupus pancreatitis (SLEAP) is an infrequent consequense of lupus. It is reported to occur in 0.7–4% of patients, although the actual prevalence may be higher, as some cases remain undiagnosed due to a subclinical presentation (Alves et al., 2016). This condition can lead to severe complications, contributing to a high mortality rate of 27%. The increased mortality is associated with central nervous system and cardiac involvement, low complement levels, hypocalcemia, and pancreatic sequelae (Halabi et al., 2021). Pancreatitis can be diagnosed at any stage of the disease, however it often develops within the first one to two years following the diagnosis of SLE. A systematic review by Li et al. reported that this manifestation was the initial symptom of lupus in 23.23% of patients (Frittoli et al., 2021; Li et al., 2017). Lupus-associated pancreatitis is diagnosed when the established criteria for acute pancreatitis are met, including characteristic clinical manifestations, imaging findings, as well as elevated serum amylase and lipase levels. Additionally, alternative

etiologies must be excluded (Williamson et al., 2024) (Gajewski Piotr, 2024b). The predominant symptom of SLE-related pancreatitis is acute abdominal pain, reported in over 90% of cases. Additionally, some patients present with nausea, vomiting, abdominal distension, reduced bowel sounds, fever, diarrhea, and abdominal tenderness (Alharbi, 2022; Li et al., 2017). Several theories have been proposed regarding the pathogenesis of SLE-associated acute pancreatitis (SLEAP). One hypothesis implicates vasculitis as a contributing factor, leading to pancreatic necrosis. Another proposed mechanism involves thrombosis, driven by the production of antiphospholipid antibodies, which results in arterial and arteriolar occlusion within the pancreas. Additionally, thickening of the inner layer of the pancreatic artery wall, along with immune complex deposition and complement activation, has been suggested as a potential pathophysiological mechanism. The literature also indicates a possible association between an increased risk of pancreatitis and the presence of anti-La antibodies. Furthermore, it is important to acknowledge that medications used in the management of SLE may also induce pancreatitis (Alharbi, 2022; Alves et al., 2016; Brewer & Kamen, 2018).

Liver

Hepatic manifestations in systemic lupus erythematosus (SLE) may arise from various causes, not solely from lupus itself. Potential contributing factors include lupus hepatitis, aitoimmune liver disease (AILD), and drug-induced liver disease related to medications used in lupus treatment (immune suppressants, hydroxychloroquine, and non-steroidal anti-inflammatory drugs). Other possible causes comprise viral hepatitis, primary biliary cirrhosis, and fatty liver triggered by corticosteroids. In the course of lupus, 25-59% of patients suffer from hypertransaminasemia – raised level of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) (Adiga & Nugent, 2017).

Lupus hepatitis

The BILAG-2004 glossary defines lupus hepatitis as elevated serum levels of AST and ALT without the presence of autoantibodies specific to autoimmune hepatitis. Given that hepatic manifestations in systemic lupus erythematosus may arise as either primary or secondary feature, a definitive diagnosis necessitates the exclusion of alternative causes of abnormal liver function tests (Isenberg et al., 2005; Williamson et al., 2024). This hepatic disorder has been reported to occur in patients with SLE at a frequency ranging from approximately 3% to 23% (Imran et al., 2021; Liu et al., 2015). The extant literature suggests that lupus hepatitis is more prevalent among patients with active lupus (11.8%) than among patients with low disease activity (3.2%) (Alves et al., 2016). A distinguishing feature of lupus hepatitis is the relatively frequent detection of anti-ribosomal P antibodies in the serum (González-Regueiro et al., 2020; Imran et al., 2021). Histopathologically, lupus hepatitis is associated with moderate periportal or lobular inflammation, characterized by lymphocytic, neutrophilic, and plasmacytic infiltration of the affected regions. Additional histopathological findings may include steatosis, mild cholestasis, focal necrosis, hepatocellular hydropic degeneration, and nodular cirrhosis. A hallmark feature that strongly supports the diagnosis and aids in distinguishing lupus hepatitis from other causes of hepatic dysfunction is the presence of complement component 1q (C1q) deposits on liver immunohistochemistry (Adiga & Nugent, 2017). Lupus hepatitis frequently manifests as a subclinical or sometimes asymptomatic condition with a mild course, and responds positively to medications employed in the treatment of lupus, particularly corticosteroids (González-Regueiro et al., 2020). The clinical course may, in some cases, be complicated by abdominal pain, hepatomegaly, jaundice, mild splenomegaly, signs of portal hypertension, and, less commonly, hepatic failure (Imran et al., 2021; Tkak et al., 2024).

Autoimmune hepatitis (AIH)

A rare coexistence of systemic lupus erythematosus and autoimmune hepatitis has been described in the literature and is termed the SLE-AIH overlap syndrome. Historically, the condition was designated as lupoid hepatitis (Alves et al., 2016). It has been demonstrated that there is an increased risk of developing the other disease in the course of both (Adiga & Nugent, 2017). This syndrome is reported to occur in approximately 1–2.6% of individuals diagnosed with autoimmune hepatitis and in 1.2–4.7% of those with lupus (Kheyami et al., 2024). The diagnostic criteria for autoimmune hepatitis include an elevated serum immunoglobulin G (IgG) level above the upper limit of normal, the presence of disease-specific autoantibodies (anti-smooth-muscle antibodies, anti-liver kidney microsomal type 1, ANA), characteristic histopathological features, and the exclusion of viral hepatitis as a causative agent (Hennes et al., 2008). Persistent transaminase elevation in individuals with lupus always warrants consideration of autoimmune hepatitis as a potential diagnosis. The differential diagnosis between AIH and lupus hepatitis is challenging due to the similarities in biochemical findings and clinical presentation. The histological hallmark serves as a useful tool for distinguishing between

these two conditions in lupus patients. The characteristic histopathological pattern of AIH includes lobular inflammation, rosetting of hepatocytes, emperipolesis, lymphoplasmacytic infiltration and fibrosis (Halabi et al., 2021). Autoimmune hepatitis is more frequently linked to an increased risk of progression to end-stage liver disease (Adiga & Nugent, 2017; Wang et al., 2024). The clinical presentation of the disease is heterogeneous, spanning from an asymptomatic course to non-specific manifestations including malaise, nausea, abdominal discomfort, anorexia, jaundice, and pruritus (Alves et al., 2016; Wang et al., 2024).

Conclusions

Gastrointestinal and hepatic manifestations in the course of lupus can range from mild, nearly asymptomatic to fulminant manner, leading to severe and life-threatening consequences. Therefore, familiarity with their characteristics is essential for the prompt diagnosis of these conditions and the initiation of appropriate treatment. It should also be noted that some presentations may not solely result from the direct effect of lupus on the organs, but may be related to the side effects of the treatment. The pathogenesis of many of these entities is not yet fully understood and requires further in-depth research to optimize treatment methods and reduce mortality rates.

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