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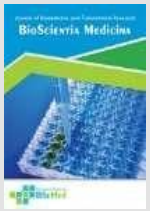

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A Rare Case of Systemic Lupus Erythematosus with Concomitant Inflammatory Bowel Disease: A Diagnostic and Therapeutic Challenge

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A B S T R A C T

Background: Systemic lupus erythematosus (SLE) and inflammatory bowel disease (IBD) are chronic conditions with complex pathophysiologies. While both diseases can affect the gastrointestinal tract, their co-occurrence is rare and presents unique diagnostic and therapeutic challenges. This case report describes a patient with SLE who developed IBD, highlighting the complexities of managing such cases. **Case presentation:** A 27-year-old female with a history of SLE presented with hematochezia, abdominal pain, and weight loss. A colonoscopy revealed findings consistent with IBD. The patient's SLE was well-controlled on immunosuppressive therapy, but the addition of IBD required careful medication adjustments to manage both conditions effectively. **Conclusion:** The coexistence of SLE and IBD is an uncommon but significant clinical scenario. This case underscores the importance of a thorough evaluation of IBD in SLE patients presenting with gastrointestinal symptoms. Furthermore, it emphasizes the need for a multidisciplinary approach to optimize treatment strategies and improve patient outcomes.

1. Introduction

Systemic lupus erythematosus (SLE) and inflammatory bowel disease (IBD) are chronic diseases that present unique challenges in both diagnosis and treatment. SLE is a complex and heterogeneous autoimmune disease characterized by widespread inflammation, multi-organ involvement, and a diverse range of clinical manifestations. IBD, encompassing ulcerative colitis (UC) and Crohn's disease (CD), is characterized by chronic inflammation of the gastrointestinal tract. While both SLE and IBD can affect the gastrointestinal system, their co-occurrence is rare, making diagnosis and treatment even more complicated.[1-3]

SLE is a complex and heterogeneous autoimmune disease characterized by widespread inflammation, multi-organ involvement, and a diverse range of clinical manifestations. The disease is characterized by the production of autoantibodies, primarily antinuclear antibodies (ANAs), that attack the body's own tissues and organs. SLE can affect any part of the body, but it most commonly involves the skin, joints, kidneys, and central nervous system. The clinical manifestations of SLE are highly variable, ranging from mild symptoms like skin rashes and joint pain to severe complications like renal failure and neurological deficits. The pathogenesis of SLE is complex and not fully understood, but it is thought to

involve a combination of genetic, environmental, and hormonal factors. Genetic susceptibility plays a significant role, with multiple genes contributing to the risk of developing SLE. Environmental factors, such as infections, ultraviolet radiation, and certain medications, can trigger or exacerbate the disease. Hormonal factors, particularly estrogen, are also thought to play a role, as SLE is more common in women of childbearing age.[4-6]

IBD, encompassing ulcerative colitis (UC) and Crohn's disease (CD), is characterized by chronic inflammation of the gastrointestinal tract. UC primarily affects the colon and rectum, while CD can affect any part of the digestive tract from the mouth to the anus. The clinical presentation of IBD typically includes abdominal pain, diarrhea, and weight loss, but can also involve extra-intestinal manifestations like arthritis and skin lesions. The pathogenesis of IBD is also complex and not fully understood, but it is thought to involve an inappropriate immune response to the gut microbiota in genetically susceptible individuals. Environmental factors, such as diet, smoking, and infections, can also contribute to the development and progression of IBD. The co-occurrence of SLE and IBD is rare, with an estimated prevalence of 0.4% for UC in SLE patients. The diagnostic process can be challenging, as gastrointestinal symptoms may overlap between the two conditions. Moreover, the management of co-existing SLE and IBD requires a delicate balance of immunosuppressive therapies to control both diseases without exacerbating either one.[7-10] This case report presents a rare instance of a young woman with established SLE who developed IBD.

2. Case Presentation

This report details the case of a 27-year-old female who presented with a primary complaint of hematochezia. The patient, with an established history of systemic lupus erythematosus (SLE), exhibited alarming gastrointestinal symptoms, prompting a thorough investigation to determine the underlying cause and implement appropriate management strategies. The patient's chief complaint was hematochezia, characterized by the passage of fresh blood mixed with mucus in her stools. This had occurred ten times since the previous day, indicating a significant and acute issue. Accompanying the hematochezia was abdominal pain, primarily localized to the lower left quadrant, which worsened during defecation. These symptoms are suggestive of lower gastrointestinal tract involvement, possibly related to inflammation or an anatomical abnormality. Further investigation revealed a history of intermittent hematochezia over the past month, suggesting a chronic condition with acute exacerbations. In addition, the patient reported experiencing generalized muscle weakness, to the extent that she was unable to lift both arms and experienced frequent falls while walking. This weakness, coupled with the hematochezia, raises concerns about potential anemia and its impact on her overall functional capacity. The patient had a significant past medical history, including a recent diagnosis of SLE in December 2023. SLE is a chronic autoimmune disease that can affect multiple organ systems, and its presence adds another layer of complexity to the case. The patient's SLE manifestations included a malar rash, joint pain, and hair loss, indicating systemic involvement. Notably, she also experienced significant weight loss of 18 kg over six months, which could be attributed to a combination of her underlying SLE, potential gastrointestinal issues, and decreased appetite. In addition to SLE, the patient was diagnosed with Diabetes Mellitus and Hypertension one month prior to her SLE diagnosis. These comorbidities further complicate her case, as they require careful consideration when selecting treatment options and managing potential drug interactions. The patient's medication list included Methylprednisolone, a corticosteroid used to manage inflammation in SLE; Lacosib (etoricoxib), a non-steroidal anti-inflammatory drug (NSAID) for pain management; Lansoprazole, a proton pump inhibitor to reduce gastric acid production; Amlodipine, an antihypertensive medication; and Imuran (azathioprine), an

immunosuppressant commonly used in SLE. The use of multiple medications, especially corticosteroids and NSAIDs, can increase the risk of gastrointestinal complications, including bleeding and ulceration.

Upon physical examination, the patient appeared weak and pale, corroborating the reported symptoms and suggesting possible anemia due to blood loss from the hematochezia. The patient's vital signs revealed an elevated blood pressure (BP) of 155/108 mmHg and a rapid heart rate (HR) of 120 bpm. These findings could be indicative of pain, anxiety, dehydration due to blood loss, or potential complications related to her underlying conditions. Her temperature was within the normal range at 36°C, ruling out an active infection. The respiratory rate (RR) was slightly elevated at 20 breaths/min, and oxygen saturation (O_2 Sat) was normal at 98%. Examination of the head revealed anemic conjunctiva, further supporting the possibility of anemia. Chest examination was unremarkable, indicating no apparent respiratory or cardiovascular involvement. Abdominal examination revealed a red rash and tenderness in the lower left quadrant. The rash could be related to her SLE, while the tenderness is consistent with her reported abdominal pain and suggests localized inflammation or irritation in the lower gastrointestinal tract. Hematological investigations revealed leukocytosis (high white blood cell count) at 11,900/µl, which could indicate an inflammatory process or infection. More importantly, the patient had anemia, evidenced by a low red blood cell count (2.44 x 10^6/L), low hemoglobin (7.1 g/dL), and low hematocrit (20.8%). These findings are consistent with the suspected blood loss from hematochezia and explain the patient's weakness and pale appearance. The platelet count was within the normal range (203 x 10^3/µL). Biochemical investigations revealed normal albumin levels (3.8 g/dL), indicating no significant protein loss or malnutrition. Liver function tests (SGPT and SGOT) were also within the normal range, suggesting no liver involvement. Renal function was normal, with urea at 11 mg/dL and creatinine at 0.5 mg/dL, ruling out any kidney-related complications. An abdominal X-ray showed meteorism (distension of the abdomen with gas) but no evidence of opaque stones, masses, or ileus (intestinal obstruction). This ruled out some potential causes of abdominal pain and distension but did not provide specific information about the source of the patient's hematochezia. A colonoscopy was performed to visualize the colon and rectum directly. This revealed grade 1 internal hemorrhoids in the anus, which could contribute to minor bleeding but are unlikely to explain the significant hematochezia. More importantly, the colonoscopy showed hyperemia (increased blood flow), multiple erosions, and active bleeding in the rectum, sigmoid colon, and descending colon. These findings are highly suggestive of Inflammatory Bowel Disease (IBD), specifically Ulcerative Colitis, given the continuous inflammation pattern observed in the affected areas. Based on the comprehensive evaluation, including the patient's clinical presentation, laboratory findings, and imaging studies, the following diagnoses were made; Systemic Lupus Erythematosus (SLE): This diagnosis was already established, and the current presentation may represent an SLE flare or an unrelated condition; Hematochezia due to Inflammatory Bowel Disease (IBD): The colonoscopy findings strongly support this diagnosis, suggesting that the patient's hematochezia and other gastrointestinal symptoms are primarily due to IBD; Grade II Hemorrhoids: This is an incidental finding and likely not contributing significantly to the patient's primary complaints. This case represents a rare and challenging scenario of a young woman with established SLE who developed IBD. The co-occurrence of these two conditions necessitates a multidisciplinary approach to management, balancing the need to control both diseases without exacerbating either one (Table 1).

The management of this patient with co-existing SLE and IBD presented a significant therapeutic challenge, requiring a careful balance between controlling both diseases without exacerbating either one. The treatment strategy involved addressing the acute manifestations of IBD, continuing the management of SLE, and providing supportive care to

stabilize the patient's overall condition. Initially, the patient had been receiving treatment for her SLE, which included Methylprednisolone 2 x 4 mg, Lacosib (etoricoxib) 2 x 1 tablet, Lansoprazole 230 mg, Amlodipine 1 x 5 mg, and Imuran (azathioprine) 1 x 50 mg. However, upon hospitalization for the acute IBD exacerbation, her treatment regimen was modified to address the immediate concerns. During hospitalization, the patient was given intravenous fluids (0.9% NaCl) to maintain hydration and electrolyte balance, particularly in light of the fluid loss from diarrhea and hematochezia. Tranexamic acid 3 x 1,000 mg IV was administered to control bleeding, and the Methylprednisolone dose was increased to 2 x 62.5 mg IV to address the acute inflammation associated with IBD. Paracetamol 2 x 1,000 mg IV was provided for pain management, and Omeprazole 40 mg bolus + 8 mg/hour drip was used for gastric acid suppression to prevent gastrointestinal complications. Ondansetron 2 x 8 mg IV was given to manage nausea and vomiting, while Ceftriaxone 2 x 2 g IV was administered as a prophylactic antibiotic to prevent infections, considering the patient's immunocompromised state due to SLE and the use of immunosuppressive medications. The patient's SLE medications were also adjusted during hospitalization. Amlodipine 1 x 5 mg was continued for blood pressure control, and the Imuran dose was increased to 150 mg orally for its immunosuppressive effects on both SLE and IBD. Fluconazole 1 x 150 mg was added as an antifungal prophylaxis, considering the patient's increased risk of opportunistic infections due to immunosuppression. Sulfasalazine 2 x 500 mg, a medication commonly used in IBD, was introduced to help control intestinal inflammation. Additionally, Gitas Plus 3 x 1 tablet, a multivitamin supplement, was given to support overall health and address potential nutritional deficiencies. Importantly, the patient received three units of Packed Red Cells (PRC) to address the anemia caused by blood loss. This transfusion aimed to improve oxygen delivery to tissues and alleviate symptoms like weakness and fatigue. By day 4 of hospitalization, the patient's condition showed improvement, with no further reports of bloody stools. This indicated that the treatment regimen was effective in controlling the acute IBD flare. The therapy was continued with a focus on symptomatic treatment and efforts to improve her general condition, including nutritional support and physical therapy. Post-discharge, the patient required close follow-up to monitor both her SLE and IBD. This involved regular appointments with a multidisciplinary team, including a rheumatologist, gastroenterologist, and dietician, to ensure comprehensive care. Regular blood tests were scheduled to monitor disease activity and medication side effects, including complete blood count (CBC), inflammatory markers, and liver and kidney function tests. A repeat colonoscopy was planned after 3-6 months to assess the extent and severity of IBD and guide further treatment decisions. Patient education was also a crucial component of the follow-up care. The patient was counseled on lifestyle modifications, including dietary adjustments and stress management techniques, to help manage her conditions and prevent future flares. The importance of medication adherence and regular follow-up appointments was emphasized to ensure long-term disease control and improve her overall quality of life (Table 2).

3. Discussion

The diagnostic journey for a patient suspected of having both systemic lupus erythematosus (SLE) and inflammatory bowel disease (IBD) is fraught with complexities. This is largely due to the considerable overlap in the ways these two conditions manifest in the gastrointestinal (GI) tract. SLE, known for its multi-system involvement, can mimic IBD with its range of GI symptoms, leading to potential misdiagnosis or delayed treatment. This section delves into the intricate challenges faced when differentiating between these two conditions. Both SLE and IBD share a diverse array of GI symptoms, making it a herculean task to distinguish between them based solely on clinical presentation.

Table 1. Anamnesis, physical examination, laboratory, imaging, and diagnosis.

Category	Subcategory	Findings
Anamnesis	Chief Complaint	A 27-year-old female with Hematochezia (fresh blood mixed with mucus in stools, 10 times since the previous day)
		Abdominal pain, primarily in the lower left quadrant, worsens during defecation
		Weakness, nausea, vomiting, loss of appetite
	History of Presenting Illness	Intermittent hematochezia for the past 6 months
		Generalized muscle weakness, inability to lift both arms, frequent falls while walking (December 2023)
		Malar rash, joint pain, hair loss, 18 kg weight loss over 6 months (December 2023)
	Past Medical History	SLE (diagnosed in December 2023)
		Diabetes Mellitus (diagnosed 1 month prior to SLE diagnosis)
		Hypertension (diagnosed 1 month prior to SLE diagnosis)
	Medications	Methylprednisolone 2 x 4 mg
		Lacosib (etoricoxib) 2 x 1 tab
		Lansoprazole 2 x 30 mg
		Amlodipine 1 x 5 mg
		Imuran (azathioprine) 1 x 50 mg
Physical examination	General Appearance	Weak, pale
	Vital Signs	BP: 155/108 mmHg
		HR: 120 bpm
		Temp: 36°C
		RR: 20 breaths/min
		O_2 Sat: 98%
	Head	Anemic conjunctiva
	Chest	Normal
	Abdomen	Red rash, tenderness in the lower left quadrant
Laboratory	Hematology	Leukocytes: 11,900/µL (High)
		Erythrocytes: 2.44 x 10^6/µL (Low)
		Hemoglobin: 7.1 g/dL (Low)
		Hematocrit: 20.8% (Low)
		Platelets: 203 x 10^3/µL (Normal)
	Biochemistry	Albumin: 3.8 g/dL (Normal)
		SGPT: 8 U/L (Normal)
		SGOT: 17 U/L (Normal)
		Urea: 11 mg/dL (Normal)
		Creatinine: 0.5 mg/dL (Normal)
Imaging	Abdominal X-ray	Meteorism with no evidence of opaque stones, masses, or ileus
	Colonoscopy	Grade II internal hemorrhoids in the anus
		Hyperemia, multiple erosions, and bleeding in the rectum, sigmoid colon, and descending colon
Diagnosis		Systemic Lupus Erythematosus (SLE)
		Hematochezia due to Inflammatory Bowel Disease (IBD)
		Grade II Hemorrhoids

Table 2. Treatment and follow-up.

Category	Subcategory	Findings
Treatment	Initial (for SLE)	Methylprednisolone 2 × 4 mg
		Lacosib (etoricoxib) 2 × 1 tab
		Lansoprazole 2 × 30 mg
		Amlodipine 1 × 5 mg
		Imuran (azathioprine) 1 × 50 mg
Treatment	During Hospitalization	0.9% NaCl at 20 drops per minute
		Tranexamic acid 3 × 1,000 mg IV
		Methylprednisolone 2 × 62.5 mg IV
		Paracetamol 2 × 1,000 mg IV
		Omeprazole 40 mg bolus + 8 mg/hour drip
		Ondansetron 2 × 8 mg IV
		Ceftriaxone 2 × 2 g IV
		Amlodipine 1 × 5 mg
		Imuran 1 × 50 mg orally
		Fluconazole 1 × 150 mg
		Sulfasalazine 2 × 500 mg
		Gitas Plus 3 × 1 tablet
		3 units of Packed Red Cells (PRC)
Follow-up	Day 4 of Hospitalization	No longer reported bloody stools
		Therapy continued with symptomatic treatment and efforts to improve the general condition
Follow-up	Post-discharge	Close follow-up to monitor both SLE and IBD
		Regular blood tests to monitor disease activity and medication side effects (e.g., CBC, inflammatory markers, liver and kidney function tests)
		Repeat colonoscopy to assess IBD extent and severity (e.g., after 3-6 months)
		Regular appointments with a multidisciplinary team (e.g., rheumatologist, gastroenterologist, dietician)
		Patient education on lifestyle modifications (e.g., diet, stress management)

A common complaint in both conditions is that abdominal pain can arise from different mechanisms. In SLE, it may be attributed to serositis (inflammation of the membranes lining the abdominal cavity), vasculitis (inflammation of blood vessels), or pancreatitis. Conversely, in IBD, abdominal pain typically stems from inflammation and ulceration of the intestinal lining. The variability in the character, location, and severity of abdominal pain in both conditions further complicates the diagnostic process. Nausea, vomiting, and diarrhea are non-specific symptoms that can manifest in both SLE and IBD, making it difficult to pinpoint the underlying cause. In SLE, they may be related to medication side effects, particularly from nonsteroidal anti-inflammatory drugs (NSAIDs) or immunosuppressants. In IBD,

these symptoms are often a direct consequence of intestinal inflammation and altered bowel motility. Differentiating between medication-induced GI side effects and IBD-related symptoms can be particularly challenging in patients already diagnosed with SLE. The presence of hematochezia, or fresh blood in stool, is often associated with IBD, especially ulcerative colitis. However, SLE can also manifest with hematochezia due to vasculitis affecting the GI tract. This overlapping symptom can lead to diagnostic confusion, as it may be initially attributed to SLE, potentially delaying the diagnosis and appropriate management of IBD. Given the significant overlap in GI symptoms, healthcare providers must maintain a high index of suspicion for IBD in SLE patients presenting with GI complaints. It is crucial to recognize the possibility of co-existing IBD in these patients and include it in the differential diagnosis, especially when symptoms are persistent, severe, or do not respond to standard SLE treatment. A thorough and detailed medical history, including a comprehensive review of GI symptoms, is paramount. The frequency, duration, and character of symptoms, as well as any associated factors such as food intake, medication use, or recent infections, should be meticulously documented. A comprehensive physical examination, with particular attention to the abdominal examination, should be performed to assess for tenderness, distension, or any palpable masses. When IBD is suspected in an SLE patient, a colonoscopy is often indispensable for direct visualization of the colon and rectum. This procedure allows for a detailed assessment of the intestinal mucosa, enabling the identification of characteristic features of IBD, such as erythema, edema, friability, ulcerations, and pseudopolyps. Biopsies can be obtained during the colonoscopy for histopathological examination, which can further aid in differentiating between SLE-related GI manifestations and IBD. In the case presented earlier, the patient's persistent hematochezia, abdominal pain, and weight loss, despite well-controlled SLE, prompted the decision to perform a colonoscopy. This proved instrumental in establishing the diagnosis of IBD, as it revealed classic endoscopic findings consistent with ulcerative colitis, including hyperemia, erosions, and bleeding in the rectum, sigmoid colon, and descending colon. Early diagnosis of IBD in SLE patients is of utmost importance to ensure prompt and appropriate management. Timely intervention can help prevent serious complications, such as toxic megacolon, intestinal perforation, or massive GI bleeding. Moreover, early treatment can improve symptoms, enhance quality of life, and potentially alter the disease course favorably. The management of co-existing SLE and IBD requires a delicate balancing act, aiming to control both conditions without exacerbating either one. Immunosuppressive therapy is often necessary for both diseases, but the choice of medications must be carefully considered to avoid potential drug interactions and minimize the risk of adverse events. Beyond colonoscopy, other diagnostic modalities can be helpful in evaluating SLE patients with suspected IBD. Blood tests, such as complete blood count, inflammatory markers (e.g., C-reactive protein, erythrocyte sedimentation rate), and fecal calprotectin, can aid in assessing disease activity and differentiating between SLE and IBD. In some cases, imaging studies, such as magnetic resonance enterography (MRE) or computed tomography enterography (CTE), may be used to evaluate the small bowel, which is not directly visualized during colonoscopy. These imaging modalities can help identify small bowel involvement in Crohn's disease or assess for complications such as abscesses or fistulas. Capsule endoscopy may be considered in specific situations to evaluate the small bowel when MRE or CTE are inconclusive or contraindicated. In some cases, SLE can present with predominantly GI symptoms, mimicking IBD. This can lead to diagnostic delays and misdiagnosis, as the initial focus may be on ruling out IBD rather than considering SLE as the primary cause. IBD, particularly Crohn's disease, can have extra-intestinal manifestations, such as arthritis, skin lesions, or uveitis, which can overlap with SLE symptoms. This overlap can further

complicate the diagnostic process. Certain medications, such as hydralazine or procainamide, can induce drug-induced lupus, which can mimic SLE and present with GI symptoms. Distinguishing between drug-induced lupus and SLE with co-existing IBD can be challenging. The diagnosis and management of co-existing SLE and IBD often necessitate a multidisciplinary approach involving collaboration between rheumatologists, gastroenterologists, and other healthcare professionals. This collaborative approach ensures comprehensive and holistic care that addresses the unique needs of each patient.[11,12]

Managing patients with co-existing SLE and IBD is akin to navigating a complex therapeutic landscape, where decisions must carefully consider the intricate and often unpredictable interplay between these two conditions. Both SLE and IBD are chronic inflammatory diseases characterized by immune system dysregulation, and treatment often involves the use of immunosuppressive medications. However, the choice of these medications requires a delicate balancing act to avoid exacerbating either condition or increasing the risk of adverse events. Immunosuppressive medications are essential in managing both SLE and IBD, as they help to dampen the overactive immune response driving inflammation in both conditions. However, selecting the appropriate immunosuppressant is a critical step that requires careful consideration of several factors, including the severity of each disease, the presence of any comorbidities, and the potential for drug interactions. In the case presented earlier, the patient was already receiving azathioprine, an immunosuppressant commonly used in SLE that also exhibits some efficacy in IBD. Azathioprine, a purine analogue, works by inhibiting DNA synthesis, thereby suppressing immune cell proliferation. It is often employed as a steroid-sparing agent in SLE and can help maintain remission in IBD. However, to effectively manage the patient's IBD, the addition of sulfasalazine, another immunosuppressant specifically indicated for IBD, proved necessary. Sulfasalazine is a combination drug comprising sulfapyridine and 5-aminosalicylic acid (5-ASA). The 5-ASA component exerts anti-inflammatory effects in the gut, while the sulfapyridine component is believed to modulate the immune response. While the combination of azathioprine and sulfasalazine in this case successfully controlled both SLE and IBD without significant adverse effects, it is crucial to acknowledge that using multiple immunosuppressants raises concerns about potential side effects, including an increased risk of infections. This risk necessitates careful monitoring and proactive management to mitigate potential complications. Close monitoring for adverse events is paramount in patients with co-existing SLE and IBD receiving immunosuppressive therapy. Regular blood tests to assess blood counts, liver and kidney function, and inflammatory markers are indispensable to ensure the safety and efficacy of the treatment regimen. Immunosuppressants can suppress bone marrow function, potentially leading to a decrease in white blood cells (leukopenia), red blood cells (anemia), and platelets (thrombocytopenia). Leukopenia increases the risk of infections, anemia can cause fatigue and shortness of breath, and thrombocytopenia can lead to bleeding problems. Regular monitoring of blood counts can help detect these side effects early, allowing for timely dose adjustments or medication changes if necessary. Some immunosuppressants can cause liver or kidney damage. Regular monitoring of liver function tests (LFTs), such as AST and ALT, and kidney function tests, such as creatinine and blood urea nitrogen (BUN), can help identify any signs of toxicity, enabling prompt intervention to prevent further damage. Monitoring inflammatory markers, such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), can help assess disease activity and response to treatment. These markers can guide treatment decisions and help determine if medication adjustments are needed. Beyond immunosuppressants, other medications may be necessary to manage specific symptoms or complications of SLE and IBD. Corticosteroids, such

as prednisone or methylprednisolone, are potent anti-inflammatory drugs that can be used to control disease flares in both SLE and IBD. However, long-term use of corticosteroids is associated with significant side effects, including weight gain, osteoporosis, increased risk of infections, and adrenal suppression. Therefore, their use should be judiciously balanced against their potential benefits and risks, and efforts should be made to taper and discontinue them whenever possible. Biologics, such as tumor necrosis factor (TNF) inhibitors (e.g., infliximab, adalimumab), are a newer class of medications that target specific components of the immune system. They can be effective in both SLE and IBD but are also associated with an increased risk of infections, particularly opportunistic infections. Careful patient selection, pre-treatment screening for latent infections (e.g., tuberculosis), and ongoing monitoring are essential when using biologics. Depending on the specific manifestations of SLE and IBD, other medications may be used, such as antidiarrheals to control diarrhea, pain relievers to manage pain, or iron supplements to treat anemia. In some cases, medications may be needed to address extra-intestinal manifestations of SLE or IBD, such as skin involvement or joint pain. In addition to medication, non-pharmacological strategies play a vital role in managing co-existing SLE and IBD. These strategies empower patients to actively participate in their care and improve their overall well-being. Lifestyle modifications, such as stress management techniques, regular exercise, and smoking cessation, can help improve overall health and well-being. Stress can exacerbate both SLE and IBD, so managing stress through techniques like meditation, yoga, or deep breathing exercises can be beneficial. Regular exercise can help improve physical fitness, reduce inflammation, and enhance mood. Smoking cessation is crucial, as smoking can worsen both SLE and IBD. Dietary modifications, such as avoiding trigger foods and maintaining a balanced diet, can help manage gastrointestinal symptoms. Identifying and avoiding foods that trigger or worsen symptoms can be helpful. Maintaining a balanced diet that is rich in fruits, vegetables, and whole grains can help ensure adequate nutrition and support overall health. Patient education is crucial to empower patients to actively participate in their care and make informed decisions about their treatment options. Providing patients with information about their conditions, treatment options, and potential side effects can help them understand their disease and make informed decisions about their care. The management of co-existing SLE and IBD often necessitates a multidisciplinary approach involving collaboration between rheumatologists, gastroenterologists, and other healthcare professionals. This collaborative approach ensures comprehensive and holistic care that addresses the unique needs of each patient.[13-15]

The co-occurrence of SLE and IBD, while a rare phenomenon, compels us to delve deeper into the potential pathophysiological connections between these two seemingly distinct conditions. Both diseases are characterized by complex immune dysregulation, with a multitude of genetic and environmental factors contributing to their development. While the exact relationship between SLE and IBD remains elusive, emerging evidence suggests that shared genetic susceptibility, common immune pathways, and environmental triggers may play a significant role in their co-occurrence. Genome-wide association studies (GWAS) have identified several genetic variants associated with an increased risk of both SLE and IBD, pointing towards a shared genetic predisposition. These genetic variants often involve genes that regulate immune responses, such as those involved in antigen presentation, T cell activation, and cytokine production. Variations in the Human Leukocyte Antigen (HLA) genes, which play a crucial role in immune regulation by presenting antigens to T cells, have been strongly linked to both SLE and IBD. Specific HLA alleles have been associated with an increased risk of both conditions, suggesting that these alleles may confer susceptibility to immune dysregulation that can manifest as either SLE or IBD. Genes involved in maintaining the integrity of the

intestinal barrier, such as NOD2 and ATG16L1, have also been implicated in both SLE and IBD. These genes play a crucial role in preventing the entry of harmful bacteria and antigens from the gut into the bloodstream, and their dysfunction can lead to increased intestinal permeability and immune activation, contributing to the development of both conditions. Both SLE and IBD involve dysregulation of the immune system, with an imbalance of pro-inflammatory and anti-inflammatory cytokines contributing to chronic inflammation. In SLE, the immune system mistakenly attacks the body's own tissues and organs, leading to widespread inflammation and damage. In IBD, the immune system mounts an inappropriate response to the gut microbiota, resulting in chronic inflammation of the digestive tract. The innate immune system is the body's first line of defense against infection. It involves cells such as macrophages, neutrophils, and dendritic cells that recognize and eliminate pathogens through pattern recognition receptors (PRRs). In both SLE and IBD, the innate immune system is dysregulated, leading to excessive inflammation. This dysregulation may involve increased activation of PRRs, leading to the production of pro-inflammatory cytokines and chemokines, and impaired clearance of apoptotic cells, which can further activate the immune system. The adaptive immune system is responsible for recognizing and remembering specific pathogens. It involves cells such as T cells and B cells that produce antibodies and cytokines to fight infection. In both SLE and IBD, the adaptive immune system is also dysregulated, leading to autoimmunity and chronic inflammation. This dysregulation may involve an imbalance of T helper cell subsets (Th1, Th2, Th17), with increased activity of pro-inflammatory Th1 and Th17 cells and decreased activity of regulatory T cells (Tregs) that normally suppress immune responses. Cytokines are signaling molecules that regulate immune responses. In both SLE and IBD, there is an imbalance of pro-inflammatory and anti-inflammatory cytokines, leading to excessive inflammation. For example, TNF-alpha, a potent pro-inflammatory cytokine, is elevated in both SLE and IBD and plays a crucial role in driving inflammation in both conditions. Other cytokines implicated in both conditions include interleukin-6 (IL-6), interleukin-17 (IL-17), and interferon-gamma (IFN-gamma). While genetic susceptibility lays the foundation for the development of both SLE and IBD, environmental factors are thought to be crucial triggers that initiate or exacerbate disease activity. Infections, such as Epstein-Barr virus (EBV) and cytomegalovirus (CMV), have been linked to both SLE and IBD. These infections may trigger the onset of disease by activating the immune system or by molecular mimicry, where viral antigens resemble self-antigens, leading to autoimmune responses. Smoking is a well-established risk factor for both SLE and IBD. It can trigger the onset of disease and worsen disease activity by promoting inflammation, impairing immune regulation, and disrupting the intestinal barrier. Diet may also play a role in the development of both SLE and IBD. Certain foods, such as red meat and processed foods, have been linked to an increased risk of both conditions. These foods may promote inflammation and alter the gut microbiome, contributing to disease development. UV radiation is a known trigger for SLE flares and may also play a role in IBD pathogenesis. UV radiation can induce apoptosis (programmed cell death) of skin cells, releasing self-antigens that can activate the immune system. Psychological stress can exacerbate both SLE and IBD by activating the hypothalamic-pituitary-adrenal (HPA) axis and increasing the production of cortisol, a stress hormone that can modulate immune responses. The gut microbiome, the vast community of bacteria that reside in the digestive tract, is increasingly recognized as an important player in immune regulation and overall health. Alterations in the gut microbiome, known as dysbiosis, have been implicated in both SLE and IBD. The gut microbiome influences the development and function of immune cells, such as T cells and B cells. Dysbiosis can lead to an imbalance of T helper cell subsets and impaired regulatory T cell function, contributing to autoimmunity and inflammation. Dysbiosis can

disrupt the integrity of the intestinal barrier, allowing bacteria and antigens to enter the bloodstream and activate the immune system. The gut microbiome produces various metabolites, such as short-chain fatty acids (SCFAs), that can influence immune responses. Dysbiosis can alter the production of these metabolites, potentially contributing to inflammation. Dysbiosis can lead to an overgrowth of harmful bacteria that can activate the immune system and trigger inflammation. Dysbiosis can disrupt the integrity of the intestinal barrier, allowing bacteria and antigens to enter the bloodstream and activate the immune system. SCFAs, such as butyrate, have anti-inflammatory effects in the gut. Dysbiosis can reduce the production of SCFAs, contributing to inflammation.[16-18]

The successful management of patients with co-existing SLE and IBD necessitates a multidisciplinary approach, involving close collaboration between various healthcare professionals. This collaborative approach ensures comprehensive and holistic care that addresses the unique needs of each patient, optimizing treatment outcomes and improving their overall quality of life. Rheumatologists are specialists in the diagnosis and management of autoimmune diseases, including SLE. They possess expertise in immunosuppressive therapies, disease monitoring, and managing SLE-related complications. Gastroenterologists specialize in the diagnosis and management of digestive system disorders, including IBD. They are skilled in performing endoscopic procedures, interpreting biopsies, and prescribing appropriate medications for IBD. Effective collaboration between the rheumatologist and gastroenterologist is crucial to ensure that treatment decisions are made in the best interest of the patient. Both SLE and IBD often require immunosuppressive therapy, and close collaboration between specialists helps to select the most appropriate medications and dosages, minimizing the risk of drug interactions and adverse events. Regular monitoring of disease activity in both SLE and IBD is crucial to assess treatment response and adjust medications as needed. Collaboration between specialists ensures that both conditions are closely monitored and managed effectively. Patients with co-existing SLE and IBD are at increased risk of complications, such as infections, anemia, and malnutrition. Collaboration between specialists helps to identify and manage these complications promptly. Dietary modifications can significantly impact the management of both SLE and IBD. A dietician can provide personalized dietary guidance to help patients manage gastrointestinal symptoms, ensure adequate nutrition, and address any dietary restrictions or deficiencies. Living with chronic illnesses like SLE and IBD can take a toll on mental health. Mental health professionals, such as psychologists or psychiatrists, can provide support and coping strategies to address anxiety, depression, and other emotional challenges. Nurses with expertise in rheumatology or gastroenterology can provide valuable education and support to patients, helping them understand their conditions, manage their medications, and navigate the healthcare system. Pharmacists can assist with medication management, ensuring patients understand their medications, potential side effects, and drug interactions. They can also help with medication adherence and access to affordable medications. Physical therapy can be beneficial for patients with SLE-related joint pain or muscle weakness. It can also help improve overall fitness and well-being. Occupational therapists can help patients with SLE and IBD adapt to their conditions and maintain independence in their daily activities. They can provide strategies for managing fatigue, pain, and other symptoms that may interfere with daily tasks. By coordinating care and addressing all aspects of the patient's health, a multidisciplinary team can improve disease control, reduce complications, and enhance quality of life. Patients feel more supported and empowered when they have access to a team of healthcare professionals working together to address their needs. A multidisciplinary approach can help reduce healthcare costs by preventing complications, reducing hospitalizations, and improving medication adherence. By coordinating

care and sharing information, a multidisciplinary team can improve efficiency and reduce duplication of services.[19,20]

4. Conclusion

This case report presents a rare instance of a young woman with established SLE who subsequently developed IBD, underscoring the complex interplay between these two conditions. The patient's initial presentation with hematochezia, abdominal pain, and weight loss prompted a thorough investigation, leading to the diagnosis of IBD. The diagnostic process was challenging due to the overlapping nature of gastrointestinal symptoms in both SLE and IBD. Colonoscopy played a crucial role in establishing the diagnosis, revealing characteristic endoscopic findings of IBD. The management of this patient with co-existing SLE and IBD presented a therapeutic challenge, requiring a careful balance between controlling both conditions without exacerbating either one. The treatment strategy involved addressing the acute manifestations of IBD while continuing SLE management and providing supportive care. Immunosuppressive therapy was adjusted to control both diseases, with close monitoring for potential adverse events. This case highlights the importance of a thorough evaluation for IBD in SLE patients presenting with gastrointestinal symptoms. It also emphasizes the need for a multidisciplinary approach to optimize treatment strategies and improve patient outcomes. Further research is needed to understand the underlying pathophysiological mechanisms linking SLE and IBD, which may lead to the development of targeted therapies for improved disease management.

5. References

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