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Original Article

Gastrointestinal Manifestation and Esophagogastroduodenoscopy Findings in Systemic Lupus Erythematous Patients.

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Abstract

Background: Systemic lupus erythematosus (SLE), an autoimmune illness, with severe inflammatory signs. Skin, neurological, central nervous system and hematological involvement are common issues**.**

Objectives: This study aimed to assess gastrointestinal (GIT) proof and superior endoscopic outcomes in SLE cases.

Methods: According to the 1997 American College of Rheumatology (ACR) revised categorization tests, 40 SLE cases, aged 18 and older, of either gender, and accompanied by two people of the same gender, participated in a cross-localized ER study. Laboratory investigations, including thorough ancestry pictures, liver and kidney function tests, lipid sketches, cells with hemoglobin sedimentation rate, C-reactive protein, complements, antagonistic-dsDNA, and a full excretion study, were performed on all patients. Comprehensive abdominal ultrasonography was performed on all cases, and esophagogastroduodenoscopy (EGD) was performed on selected prisoners.

Result: Regarding the dispassionate symptoms of GIT proofs, 8 (42.11%) inmates believed that things would go badly, 7 (36.84%) were gaunt, 6 (31.58%) inmates experienced abdominal pain, 3 (15.79%) experienced bloating, 3 (15.79%) experienced loose bowels, 3 (15.79%) experienced nausea or disgorging, 3 (15.79%) experienced burden loss, and 1 (5.26%) experienced constipation

Conclusions: The doctors see the GIT symptoms of SLE since early detection and the right

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Introduction

An autoimmune disease with basic inflammatory symptoms is SLE. In addition to hematological abnormalities, skin, renal, and primary nervous system problems are frequently seen. [1]

A common complaint among 40–60% of SLE detainees is GIT engrossment. In 8–10% of patients, GI signs that are clinically recognized have been interpreted [2]. In a similar vein, autopsy reports show 60–70% of patients have GIT difficulties, indicating that subclinical or hidden issue is widespread. [2, 3]

The majority of GIT proofs are always modest .[4] William Osler was the first to describe how the gastrointestinal issues of SLE can resemble an intestinal ailment and obscure the various ways that SLE is influenced by problems in 1895 .[5]

Oral ulcers, false stomach blockage, protein-defeated enteropathy, liver damage, autoimmune pancreatitis, lupus enteritis (LEn), and other complications are some of the ways that SLE-induced damage to the digestive system might appear. [6, 7]

A significant section of the GI region may experience a variety of symptoms as a result of GIT issues. Gauntness, nausea, or disgorging may occur in as many as 50% of patients. [8, 9] With the exception of early detection and appropriate action, vasculitis and thrombosis allow the arrangement of fatal symptoms that are superior to blood shortage, perforation, and barrier. [10].

We suggested evaluating EGD findings and GIT symptoms in SLE patients.

Patients and Methods

From March 2023 to September 2023, 40 individuals with identified SLE who were receiving treatment for medical issues at Sohag University Hospitals' gastroenterology and rheumatic hospitals participated in this cross-localized study.

Inclusion criteria

According to the 1997 American College of Rheumatology (ACR) revised categorization tests, we included SLE patients who were at least eighteen years old, regardless of their current age, of either common or accompanying gender. [11, 12]

Exclusion criteria

Patients who have diabetes mellitus and other vascular diseases, as well as those who have SLE, are associated with GIT.

The following procedures put all inmates at risk: dispassionate tests, lab studies, complete ancestry picture (red body fluid level, total leucocyte count (TLC), platelet count), liver (alanine transaminases (ALT), aspartate transaminase (AST), complements (C3 and C4), antagonistic-dsDNA, and complete urine reasoning for proteinuria (by dipstick form), hematuria (> 5 RBCs), pyuria (>5 WBCs) above capacity field, and spot urine for protein to creatinine percentage. [13]

The SLE Disease Activity Index (SLEDAI) was used to evaluate the afflicted project. SLEDAI score was predetermined; sufferers with a score of 6 or more were classified as having an active illness, while those with a score of less than 6 were considered to have an inactive illness. [14, 15]

Esophagogo-gastroduodenoscopy (EGD) and abdominal ultrasonography were performed on patients with documented above-GI symptoms, such as dysphagia, disgorging, or epigastric discomfort.

Ethical considerations:

Similar to the Declaration of Helsinki, this task was finished, and all parties provided their signed approval.

and dossier confidentiality was assured. The Scientific Research Ethical Committee of Sohag University's Faculty of Medicine certified the study contract.

Statistical analysis:

SPSS v28 was used to do statistical reasoning (IBM Inc., Armonk, NY, USA). Utilizing the uneven Student's t-test, quantitative variables were assigned as mean and predictable difference (SD), which separated the middle group from two points two groups together. Commonness and part (%) were assigned to the qualitative variables, which were then analyzed using the Chi-square test or, if applicable, Fisher's exact test. A two-tailed P profit of less than 0.05 was considered statistically significant.

Results:

40 cases with SLE were included in this study; their mean age was 31.7 ± 7.68 years, and 31

(77.5%) of them were women and 9 (22.5%) were men. Of the purposeful subjects, 21 (52.5%) did not display GIT manifestation, whereas 19 (47.5%) did. Table 1 demonstrates that there were little differences between the deliberate groups in terms of the dispassionate dossier (event of symptoms, medications, and SLEDAI score) and the guideline features (age and sexuality).

Table 1: Baseline characteristics and clinical data of the studied groups

		Total (n=40)	SLE with GIT manifestation (n=19)	SLE without GIT manifestation (n=21)	P value
Age (years)		31.7 ± 7.68	33.6 ± 7.75	30 ± 7.36	0.132
Sex	**Male**	9 (22.5%)	5 (26.32%)	4 (19.05%)	0.712
	Female	31 (77.5%)	14 (73.68%)	17 (80.95%)	
Duration of symptoms (years)			6.5 ± 3.01	6.95 ± 3.63	0.654
Medications					
NSAIDs		7 (17.5%)	3 (15.79%)	4 (19.05%)	0.990
Corticosteroids		9 (22.5%)	5 (26.32%)	4 (19.05%)	
Hydroxychloroquine		6 (15%)	3 (15.79%)	3 (14.29%)	
Azathioprine		8 (20%)	4 (21.05%)	4 (19.05%)	
Methotrexate		5 (12.5%)	2 (10.53%)	3 (14.29%)	
Mycophenolate mofetil & cyclophosphamide		5 (12.5%)	2 (10.53%)	3 (14.29%)	
SLEDAI score		8.43 ± 3.85	8.84 ± 4.68	8.05 ± 2.97	0.521

SLEDAI: SLE ailment activity index, NSAIDS: nonsteroidal antagonistic-threatening medications, GIT: gastrointestinal lot, and mean ± SD or repetitiveness (%) are the data provided.

Hb was significantly lower in SLE cases with GIT exhibition compared to SLE patients without GIT exhibition, according to the lab tests (P<0.001). Compared to SLE patients without GIT manifestation, CRP was considerably higher in SLE cases with GIT evidence (P=0.002). There were slight differences in other laboratory tests between the two groups. **Table 2**

Table 2: Laboratory investigations of the studied groups

	Total (n=40)	SLE with GIT manifestation (n=19)	SLE without GIT manifestation (n=21)	P value
Hb (g/dL)	11.09± 0.93	10.47 ± 0.65	11.66 ± 0.77	**<0.001***
PLT (*10^9/L)	198.68±30.58	197.37 ± 28.54	199.86 ± 32.97	0.801
TLC (*10^9/L)	6.92 ± 0.91	6.85 ± 0.88	6.98 ± 0.95	0.648
Total cholesterol (mg/dL)	159.03±17.73	159.95 ± 20.16	158.19 ± 15.67	0.759
ALT (U/L)	34.45 ± 9.28	32.47 ± 9.16	36.24 ± 9.25	0.204
AST (U/L)	30.98 ± 6.25	31.37 ± 6.72	30.62 ± 5.94	0.710
Serum creatinine (mg/dL)	0.86 ± 0.25	0.79 ± 0.27	0.92 ± 0.23	0.106
Urea (mg/dL)	43.25 ± 11.84	45.42 ± 13.31	41.29 ± 10.27	0.276
ESR (mm/hr.)	92.83 ± 24.09	94.26 ± 25.1	91.52 ± 23.68	0.724
CRP (mg/dL)	15.04 ± 2.69	16.39 ± 2.85	13.82 ± 1.87	**0.002***
C3 (g/L)	85.35 ± 46.91	82.89 ± 48.71	87.57 ± 46.31	0.757
C4 (g/L)	23.75 ± 11.48	21.11 ± 11.13	26.14 ± 11.52	0.169
Anti-dsDNA (IU/mL)	85.75 ± 39.78	81.05 ± 35.32	90.0 ± 43.86	0.485

Information provided as mean ± SD, Hb: red bodily fluid, GIT: gastrointestinal tract, SLE: systemic lupus erythematosus, ALT (alanine aminotransferase), AST (aspartate aminotransferase), ESR (blood corpuscle sedimentation rate), PLT (platelets), TLC (total blood corpuscle count), Double-abandoned DNA, or anti-dsDNA, is statistically significant when the P value is less than 0.05.

Table 3 shows that the number of patients with definite giardia in their seat study was significantly higher in the SLE accompanying GIT proof group than in the SLE outside GIT exhibition group (P<0.001). This indicates that the seat analysis was notably different between the two groups under research. Proteinuria, hematuria, and pyuria were somewhat different in the midst of two points for the purposeful groups in terms of excretion analysis.

Table 3: Stool and urine analysis of the studied groups

		Total (n=40)	SLE with GIT manifestation (n=19)	SLE without GIT manifestation (n=21)	P value
Stool analysis (Giardia)	**Positive**	20 (50.0%)	15 (78.95%)	5 (23.81%)	**0.001***
	Negative	20 (50.0%)	4 (21.05%)	16 (76.19%)	
Urine analysis	**Proteinuria**	8 (20.0%)	5 (26.32%)	3 (14.29%)	0.442
	Hematuria	3 (7.5%)	2 (10.53%)	1 (4.76%)	0.596
	Pyuria	5 (12.5%)	3 (15.79%)	2 (9.52%)	0.654

Data are provided as follows: *: statistically significant as P profit <0.05, SLE: systemic lupus erythematosus, and GIT: gastrointestinal tract.

With regard to the abdominal ultrasonography, there was very little difference between the two points of the intended groups. **Table 4**

Table 4: Abdominal ultrasonography of the studied groups

	Total (n=40)	SLE with GIT manifestation (n=19)	SLE without GIT manifestation (n=21)	P value
Splenomegaly	9 (22.5%)	5 (26.32%)	4 (19.05%)	0.911
Hepatomegaly	6 (15%)	3 (15.79%)	3 (14.29%)	
Ascites	5 (12.5%)	3 (15.79%)	2 (9.52%)	
No findings	18 (45%)	8 (42.11%)	10 (47.62%)	

Data are provided as follows: *: statistically significant as P profit <0.05, SLE: systemic lupus erythematosus, and GIT: gastrointestinal tract

Table 5 demonstrates the detached signs of GIT exhibitions. Eight (42.11%) patients experienced dyspepsia, seven (36.84%) experienced anorexia, six (31.58%) experienced intestinal pain, three (15.79%) inmates experienced bloating, three (15.79%) experienced diarrhea, three (15.79%) experienced revulsion or disgorging, three (15.79%) experienced pressure loss, and one (5.26%) patient experienced muscle spasm.

EGD found that 6 (31.58%) of the detainees had sane endoscopy, 3 (15.79%) had gastric and stomach ulcers, 3 (15.79%) had gastritis, 5 (26.32%) had erosive esophagitis, and 2 (10.53%) had esophagiti

Table 5: Clinical data of SLE with GIT manifestation group

		SLE with GIT manifestation (n=19)
Clinical symptoms of GIT	**Dyspepsia**	8 (42.11%)
	Anorexia	7 (36.84%)
	Abdominal pain	6 (31.58%)
	Bloating	3 (15.79%)
	Diarrhea	3 (15.79%)
	Nausea/ Vomiting	3 (15.79%)
	Weight loss	3 (15.79%)
	Constipation	1 (5.26%)
EGD findings	**Normal endoscopy**	6 (31.58%)
	Gastric and duodenal ulcer	3 (15.79%)
	Gastritis	3 (15.79%)
	Erosive esophagitis	5 (26.32%)
	Esophagitis	2 (10.53%)

Data are provided as follows: *: statistically significant as P value <0.05, SLE: systemic lupus erythematosus, and GIT: gastrointestinal tract.

demonstrates that skilled was a pointless link between SLEDAI and GIT symptom (Table 6)

Table 6: Relationship between SLEDAI scores of SLE and GIT symptoms in the group exhibiting GIT

	SLEDAI scores	P value
Dyspepsia	9.8 ± 4.1	0.188
Anorexia	9.0 ± 4.0	0.522
Abdominal pain	8.0 ± 4.1	0.889
Bloating	6.3 ± 5.7	0.572
Diarrhea	12.3 ± 0.57	0.051
Nausea/ Vomiting	8.7 ± 5.1	0.878
Weight loss	5.0 ± 2.6	0.102
Constipation	10.0 ± 0.0	0.656

Data are provided as follows: *: statistically significant as P profit <0.05, SLE: systemic lupus erythematosus, and GIT: gastrointestinal tract.

Discussion

Systemic Lupus Erythematosus is a chronic, complicated, autoimmune disease that has no known origin and is accompanied with a variety of symptoms. (16)

With an annual incidence of 60 cases per million and a prevalence of 500 cases per million, it is the most severe autoimmune illness. The 20–40 age bracket with the same status and a 9:1 female to male ratio is where SLE is most acknowledged. Some people's schemes may be impacted. (17)

In terms of GIT proof, we find that 8 (42.11%) subjects believed that things would go wrong, 7 (36.84%) had eating disorders, 6 (31.58%) had intestinal pain, 3 (15.79%) had bloating, 3 (15.79%) had dysentery, 3 (15.79%) had stomach sickness or vomiting, 3 (15.79%) had burden deficit, and 1 (5.26%) had muscle spasm.

Patients with SLE are considered to have gastrointestinal symptoms. Over half of the ruling class is brought on by bacterial and fervid contaminations as well as antagonistic reactions to drugs. Although less frequent than lupus nephritis, gastrointestinal problems associated with SLE are clinically significant because, if left untreated, the majority of cases can be growth-threatening. (18)

In numerous earlier investigations, the prevalence of gastrointestinal symptoms in patients with SLE ranged from 15% to 75%. (19)

According to our research, 47.5% of patients with SLE had gastrointestinal symptoms. 30–50% of SLE patients have gastrointestinal disorders such as gauntness, nausea, disgorging, dysphagia, hematemesis, postprandial breadth, loose bowels, and melena. [20]. Drug side effects, SLE's vasculopathy, stress-related mucosal disease (gastritis), or any coexisting illness can all cause gastrointestinal symptoms. (21, 22)

Ulutaş et al. (23)

According to a case study, patients with intrinsic lupus erythematosus guide the gastrointestinal system and erect. These patients also experienced muscle spasm episodes, stomach pain, diarrhea, and disgorging. Such syndromes can be caused by any medication used in conjunction with active lupus, including NSAIDs, corticosteroids, hydroxychloroquine, azathioprine, methotrexate, mycophenolate mofetil, and cyclophosphamide.

Fawzy et al. (8) conducted a case-control study on GI symptoms in SLE patients and found that the following were the most common symptoms: 6% of patients had acute intestinal pain (due to pleurisy and peritonitis); 23.5% had wordy intestinal pain; 29% had epigastric pain; 23.5% had epigastric pain with disgorging; 6% had epigastric pain with persistent constipation; 6% had persistent muscle spasms; and 6% had wordy abdominal pain with draining per rectum

Mehta et al. (12) 254 (11.5%) of the 2210 cases of systemic lupus erythematosus with SLE that were investigated for gastrointestinal proofs in the INSPIRE registry had GI proofs, and 39 patients also had one GI characteristic. Lupus enteritis (35, 13.8%), lupus pancreatitis (32, 12.6%), lupus hepatitis (19, 7.5%), lupus peritonitis (6, 2.3%), stomach obstruction, and lupus cholecystitis (3, 1.2%) with malabsorption and protein losing enteropathy were the most common conditions (193,76%).

With respect to giardia contamination, we found that the number of patients with specific giardia in their seat rationale was significantly higher in the group with SLE and GIT exhibition than in the group without GIT proof (P<0.001). Effective invulnerable defenses must function luminally

since Giardia infections are contained to the lumen. In order to regulate Giardia contaminations, both of the invulnerable arrangement's weapons seem to mimic one another. It is currently unclear exactly how the invulnerable technique interacts with Giardia trophozoites, however it seems to be primarily mediated by IgM, IgG, and IgA differentiating antibodies. Neutrophils, macrophages, complement, and the T-container subset all contribute once more. [24, 25]

According to a previous study, 10% of SLE patients are asymptomatic, and the disease Giardia is more common in SLE patients than in healthy controls. Giardia plague was more common in patients with GI symptoms compared to those without GI syndromes, with a P-value of 0.009. Giardia disease was more common in SLE prisoners, which was explained by the immune-suppressive effects of the drugs and the vulnerability to the disease.

We point out that there was a slight difference in the SLEDAI scores between the two groups (8.84 ± 4.68 vs. 8.05 ± 2.97, P=0.521).

This came in line with Fawzy et al. [8] He stated that patients with GI symptoms and those without GI symptoms did not significantly differ in their SLEDAI scores. On the other hand, patients with GI symptoms had a higher SLEDAI score, with a mean of 14.1 ± 4.7.

However, Mehta et al. [12] demonstrate that the understanding group's SLEDAI was much higher than the control group's.

Results from EGD were prevalent in 18.1% of patients, with 9.09% having stomach ulcers, 54.5% having gastritis, 9.09% having esophagitis, and 9.09% having both esophagitis and stomach abscess. The appearance of persistent instigative containers, especially the lymphocytes, was the most recurrent similarity in the pathology of the stomach, stomach and abdomen, and colon. Additionally, colonic biopsies showed edema of the covering layer and the combination of accompanying lymphoplasmacytic containers.

They further demonstrate that 45.4% of SLE prisoners with GI disorders had H. pylori.[8] Seropositive results for H. pylori have been found in the neighborhood of ANA, antagonistic dsDNA, and antagonistic-Ro antibodies, and H. pylori has been linked to a variety of autoimmune illnesses. [26]. Sawalha et al. [27] indicated that immunor-egulatory events greater than H. pylori seropositivity were in reverse order and associated with the risk of SLE, or that H. pylori infection was a plausible protective factor against the development of SLE.

In the current study, we found there was an insignificant relation between GIT symptoms and SLEDAI. This came in line with Soltani et al. [28] They did not detect a significant correlation between the incidence of GIT syndromes and SLEDAI scores. The study focused on gastrointestinal symptoms and upper endoscopy evaluations in basic lupus erythematous in 130 participants with SLE.

Our study was constrained by its small sample size, single-center design, and lack of a control group.

Conclusion

The doctors acknowledge the potential for GI symptoms of SLE because early detection and effective therapy can affect the prognosis for patients. In our investigation, EGD evaluations failed to discriminate symptoms of SLE. In order to investigate the relationship between chronic mesenteric blood shortage and SLE, a larger, multi-center clinical investigation is desired. This study will act in two ways and color-systematize the Doppler tests of the stomach artery and the superior mesenteric channel.

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