Chronic Diarrhea as the Presenting Complaint of Systemic Lupus Erythematosus in a Man

Ashwin Rajendiran¹, Stalin Viswanathan², Bhavith Remalayam¹, Vivekanandan Muthu² and Thomas Alexander³

Abstract

Systemic lupus can involve any part of the gastrointestinal (GI) tract. Diarrhea generally results from complications arising from infection, drugs or pancreatitis. We herein report the case of a 40-year-old hypertensive man with a psychotic disorder in whom the evaluation of chronic diarrhea revealed a diagnosis of systemic lupus erythematosus (SLE), diffuse proliferative glomerulonephritis and protein-losing enteropathy that required treatment with both steroids and mycophenolate mofetil. Over the following year, the patient developed atrial fibrillation, miliary tuberculosis and generalized clonic tonic seizures. He is currently under regular follow-up care and receives antiepileptics, antihypertensives, diltiazem, amiodarone and warfarin.

Key words: male SLE, gastrointestinal manifestations, diarrhea

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Introduction

The clinical presentation of systemic lupus erythematosus (SLE) in males varies among different populations. The gastrointestinal manifestations of SLE can involve any part of the gastrointestinal (GI) tract, with incidences ranging from 1.3% to 27.5% (1-3). Diarrhea occurring in patients with SLE generally results from parasitic or bacterial intestinal infections and can be caused by chronic pancreatitis or drugs such as azathioprine (4, 5). Rarer complications of SLE, including protein-losing enteropathy, lupus enteritis (mesenteric vasculitis), intestinal pseudo-obstruction and SLE-associated disorders such as eosinophilic enteritis and celiac disease, can also contribute to diarrhea (6). Diarrhea has been reported to be the initial complaint of SLE in mostly female patients. We herein report the case of a 40-year-old man with chronic diarrhea as the presenting complaint of SLE.

Case Report

The patient presented to the referring hospital due to non-bloody diarrhea lasting for three months with a low grade fever, vomiting and weight loss. Following 10 days of cefotaxime and metronidazole therapy, his symptoms improved. He was referred to our hospital two weeks later after his symptoms recurred. Six years previously, he had been noted to have hypertension and was prescribed metoprolol. Eight months previously, the patient had been diagnosed with paranoid schizophrenia and underwent one session of electroconvulsive therapy. His laboratory results were as follows: hemoglobin: 10.6 g, platelets: 185×10⁹/L, ESR: 89 mm/hr, urea: 7 mmol/L and creatinine: 90 μmol/L. Since then, he had been taking nitrazepam, oxcarbamazepine, risperidone and metoprolol regularly. He smoked -10 cigarettes/day and consumed -120 g of alcohol per week. On admission, he had severe pallor, crusted lips, retromolar mucosal erosions, bilateral pitting pedal edema, bilateral wheezes with infra-axillary and infra-scapular crackles and intention tremors.

The patient’s test results are listed in Table. His liver function and electrolyte results were normal. The 24-hour urinary protein level was 770 mg. Bacterial and fungal cultures of blood, urine and stools were sterile. Other test results showed fatty liver (ultrasonography), esophagitis and mild duodenal nodularity (esophagogastroduodenoscopy), an

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One year later, the patient presented with loss of appetite, fatigue and fever lasting 20 days with Medical Research Council (MRC) grade IV dyspnea. With a miliary pattern observed on chest radiography and the patient’s refusal to undergo high resolution chest CT, empirical antituberculous treatment was initiated and the symptoms began to resolve. The patient’s renal function and diarrhea remained stable. Three months later, he was readmitted after experiencing a generalized tonic clonic seizure that was treated with diazepam and phenytoin. Brain contrast CT and MRI did not reveal any abnormalities. Currently, the patient is taking diltiazem, risperidone, folic acid, amiodarone, telmisartan, carbamazepine and warfarin.

### Table. Laboratory Results for the Patient after Admission

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Normal</th>
<th>D1</th>
<th>D 10</th>
<th>D 15</th>
<th>Discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (g %)</td>
<td>12-14</td>
<td>5.5</td>
<td>7.9</td>
<td>7.1</td>
<td>9.3</td>
</tr>
<tr>
<td>WBC count × 10^9/L</td>
<td>4-10</td>
<td>5.1</td>
<td>8.2</td>
<td>2.7</td>
<td>11.7</td>
</tr>
<tr>
<td>Platelets × 10^9/L</td>
<td>150-450</td>
<td>150</td>
<td>140</td>
<td>150</td>
<td>142</td>
</tr>
<tr>
<td>Urea (μmol/L)</td>
<td>2.5-7</td>
<td>24.64</td>
<td>29.92</td>
<td>24.29</td>
<td>9.85</td>
</tr>
<tr>
<td>Creatinine (μmol/L)</td>
<td>44-80</td>
<td>308</td>
<td>325.6</td>
<td>237</td>
<td>90</td>
</tr>
<tr>
<td>Albumin (g/L)</td>
<td>40-50</td>
<td>20</td>
<td>26</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>ESR (mm/hr)</td>
<td>10-20</td>
<td>32</td>
<td>30</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urine RBCs/hpf</td>
<td>10 - 15</td>
<td>Anti-ds DNA &gt; 300</td>
<td>&lt;15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urine Pus cells/hpf</td>
<td>10 - 12</td>
<td>ANA</td>
<td>5.2</td>
<td>&lt;1.0</td>
<td></td>
</tr>
<tr>
<td>Stool ova/cyst</td>
<td>Negative</td>
<td>C3</td>
<td>0.76g/dL</td>
<td>0.83-1.77</td>
<td></td>
</tr>
<tr>
<td>Stool occult blood</td>
<td>Positive</td>
<td>C4</td>
<td>0.10g/dL</td>
<td>0.16-0.47</td>
<td></td>
</tr>
<tr>
<td>HIV</td>
<td>Negative</td>
<td>APL</td>
<td>Negative</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HBsAg</td>
<td>Negative</td>
<td>ACL</td>
<td>Negative</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AntiHCV</td>
<td>Negative</td>
<td>Leptospira IgM Negative</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


### Discussion

Studies of SLE have shown that men comprise 4-22% of cases (4). The male/female ratio was reported to be 11.4:1 in a Chinese study (7). Western studies have shown either no differences or conflicting organ involvement between the sexes, except in cases of renal disease, which is more common in men (7). Pande et al., in a study of 39 male SLE patients from India, noted that the prevalence of arthritis, mucosal lesions and alopecia was high, whereas that of hematological abnormalities, psychosis, diffuse proliferative nephritis and gastrointestinal involvement were low (4). The latter three complications comprised the initial manifestations in our patient. In a study of the largest cohort of male SLE patients, no GI manifestations or psychosis were observed at the onset of the disease (8). Mucosal symptoms, including ulcers, anorexia and abdominal pain, are common, while diarrhea, fecal incontinence, perforation and hemorrhage are rare (1, 2, 9). Other very uncommon abdominal manifestations in SLE patients include protein-losing enteropathy (PLE), lupus enteritis, esoinophilic enteritis and intestinal pseudo-obstruction (IPO) (3). Drugs and infection contribute most to the development of GI disease, in contrast to renal involvement, which is typically caused by disease activity (6). Among Indian males with SLE, GI manifestations occur at an incidence of 15% (4). The Asian population has a higher prevalence (18%) of GI manifestations compared to populations on other continents (2). Hepatosplenomegaly is the only GI manifestation that is more common in men (7) and that has been observed in the Israeli population. GI complications develop more commonly in SLE patients with hematological or renal manifestations or hypocomplementemia and in those who are positive for Raynaud’s phenomenon (9).

Diarrhea as a manifestation of SLE was observed in 37% to 45% of cohorts in three large studies (2). Forty-six cases (six men) of SLE with PLE have been reported. PLE was reported to be the initial presentation in 30 of the 46 patients: 17 had diarrhea and 24 had either ascites or pleural or pericardial effusion (2). Only one case involving a 24-year-old man who presented with PLE and serosal effusion on initial presentation has been reported. That patient also had associated primary sclerosing cholangitis. The exact pathophysiologic mechanisms of PLE have not been elucidated; however, causes of PLE have been attributed to vasculitis, complement deposition, chronic infection and inflammatory bowel diseases that disrupt lymphatic vessels. Patients with PLE present with severe pitting edema, diarrhea, hypoalbuminemia and hypocomplementemia. Patients with PLE have increased stool antitrypsin levels and high stool losses of radiolabeled albumin (10). Tc-99 albumin scintigraphy, the gold standard for diagnosing PLE was not performed in this case due to unavailability. Since the results of colonoscopy, ultrasonogram and abdominal CT were normal in this patient, diagnoses of IPO and lupus enteritis (mesen-
teric vasculitis) can be ruled out. Other causes of diarrhea, including pancreatitis, eosinophilic enteritis, pneumatosi
cystoides intestinalis and celiac disease were unlikely given
that radiology and intestinal biopsies were noncontributory. The
c results of the intestinal biopsies performed in our pa-
tient were suggestive of lymphocytic colitis, NSAID use or
autoimmune disease (11). Infection (bacterial, viral and
parasitic) and inflammatory bowel disease are common
causes of chronic diarrhea in India, and awareness of this
complaint as an initial manifestation of SLE is low. Lupus
nephritis- and malnutrition (poor intake)-related hypoalbumi-
nemia could also have contributed to the pedal edema ob-
served in our patient. Sixty percent of patients with PLE
generally improve with steroid therapy (6). Others require
additional immunosuppressants such as azathioprine and
cyclophosphamide. Our patient received mycophenolate
mofetil to treat severe renal disease, which also resulted in
the remission of diarrhea.

In conclusion, GI manifestations in patients with SLE are
generally mild and easily overlooked. Features of PLE such
as pedal edema, ascites, pleural effusion, dyslipidemia and
hypocomplementemia can mimic lupus nephritis if not asso-
ciated with diarrhea. Chronic diarrhea due to PLE as the
presenting manifestation of SLE is very rare in men, and a
diagnosis of SLE should be kept in mind when other sys-
temic manifestations are absent.

The authors state that they have no Conflict of Interest (COI).

References
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festations in lupus patients in Asia: lupus enteritis, intestinal