Elmer Press

Case Report

J Med Cases • 2013;4(5):314-315

Gastroparesis in Systemic Lupus Erythematosus: A Case Report

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Abstract

Gastrointestinal symptoms in systemic lupus erythematosus (SLE) are varied and can involve any part of the gastrointestinal tract. Although esophageal dysmotility and intestinal pseudo-obstruction have been described, the occurrence of gastroparesis in SLE patients has yet to be the subject of a medical report. We present a case of a 29-year-old woman with SLE whose predominant symptom is persistent nausea and vomiting with no evidence of mechanical obstruction.

Keywords: Systemic lupus erythematosus; Gastrointestinal manifestation; Gastroparesis

Introduction

Systemic lupus erythematosus (SLE) can involve any part of the gastrointestinal tract and hepatobiliary system [1]. Gastrointestinal symptoms are relatively common and can be the initial manifestation of SLE. Oral lesions, mesenteric vasculitis, gastritis, pancreatitis, inflammatory bowel disease, protein-losing enteropathy, intestinal pseudo-obstruction, and esophageal dysmotility have been described [1-3]. Gastroparesis, literally "paralyzed stomach", is characterized by delayed gastric emptying in the absence of mechanical obstruction [4, 5]. It has yet to be presented as a gastrointestinal manifestation of SLE.

Gastroparesis is a chronic failure of gastric motor function that causes anorexia, early satiety, nausea, persistent postprandial fullness, and vomiting [5]. It occurs most commonly in an idiopathic, diabetic, or post-surgical context and is more prevalent in women in its idiopathic form [4, 5]. Less commonly, rheumatologic disorders, including SLE, are also associated with abnormalities of gastrointestinal motor activity [4]. We report a case of a woman with SLE whose dominant symptom was gastroparesis.

Case Report

A 29-year-old woman was diagnosed with systemic lupus erythematous (SLE) during an admission for nausea, vomiting, and generalized weakness. She tested positive for ANA and antibodies to double stranded DNA and Sm nuclear antigen. She also presented with acute renal failure and underwent a renal biopsy with the pathologic finding significant for diffuse membranous lupus nephritis. The patient was treated initially with oral agents including mycophenolate mofetil, azathioprine, and prednisone. Intravenous cyclophosphamide was added a few months later.

Subsequent to this the patient had two additional admissions with persistent nausea and vomiting which were attributed in part to mycophenolate mofetil and cyclophosphamide infusions. An esophagogastroduodenoscopy (EGD) was performed and revealed chronic gastritis without evidence of obstruction. A gastric emptying study (GES) was also completed. It was equivocal with 48.3% clearance of gastric content in 90 minutes (normal being 50% or greater). Mycophenolate mofetil and cyclophosphamide were discontinued upon discharge, and the patient was started on intravenous rituximab for four infusions, with partial and transient improvement. Azathioprine and prednisone were continued as maintenance therapy. Additional measures for relief of gastroparesis symptoms included dietary modification, pantoprazole, and metoclopramide prior to meals. A second GES revealed moderately delayed gastric emptying rate with 21% clearance in 90 minutes. Erythromycin was added, with partial subjective improvement.

The patient developed urinary tract and vaginal yeast infections on the erythromycin regimen, resulting in with-

Manuscript accepted for publication March 20, 2013

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doi: http://dx.doi.org/10.4021/jmc1180w

holding of the medication until her infections were cleared. She had a third GES while off erythromycin which revealed moderately severe delay in gastric emptying with 30% clearance. Erythromycin was restarted after GES and fluconazole was prescribed to treat the yeast infections.

On subsequent follow-ups the patient continued to complain of nausea and vomiting. During this time she had been self-tapering prednisone and was non-complaint with her medications, resulting in flare-ups of SLE with worsening urinary protein excretion and aggravation of gastroparesis symptoms. Rituximab was prescribed thus for two infusions. A fourth GES was obtained after the first rituximab infusion and revealed 30% gastric emptying rate. Urinary protein excretion normalized, however gastroparesis continued. GES was repeated after the second rituximab infusion and revealed 33.5% clearance. The patient had remained on the same dosage of metoclopramide throughout this time.

Discussion

Gastrointestinal symptoms are fairly common in SLE, occurring in up to two-thirds of patients at some point in their illness and might be the disease's initial manifestation. Gastroesophageal disease symptoms, including heartburn and dysphagia, occur in 13-50% of SLE patients [6], with abnormal manometry studies noted in 10-32% [7]. Thus far the mechanism causing esophageal dysmotility remains unexplained, although inflammatory reaction, esophageal muscle atrophy, and ischemic vasculitis have been proposed as etiologies. Intestinal dysmotility, presenting as intestinal pseudo-obstruction (IPO), has also been observed, with a preponderance of small bowel involvement. Sixty-three percent of IPO cases also have concurrent involvement of the bladder, and one-third has histologic features of interstitial cystitis, suggesting an autoimmune etiology with vasculitis leading to muscular damage and dysmotility [1, 7]. Interestingly, there is no mention in the literature of gastric dysmotility or gastroparesis in SLE, although esophageal and intestinal dysmotility suggest that gastroparesis is likely a part of the spectrum of the same process.

The symptoms of gastroparesis commonly include nausea, vomiting, postprandial fullness, and may be associated with anorexia, abdominal discomfort, and weight loss. Gastric motor function is traditionally assessed with a radionuclide gastric emptying test, which is the gold standard. Delayed gastric emptying is considered the hallmark of gastroparesis, although symptoms might not always correlate with gastric emptying rate. Gastroparesis may be of diabetic, neurological, post-surgical, vascular, or pseudo-obstructive etiology [4]. Autoimmune diseases are also in the differential. Although treatment of the autoimmune disease might be expected to alleviate gastroparesis symptoms, gastroparesis does not respond well to the treatment of SLE in our patient. This suggests gastroparesis is either irreversible or slowly responsive to the usual medications used for SLE. Current treatment modalities for gastroparesis include dietary modifications such as small multiple meals, prokinetic agents such as metoclopramide and erythromycin, antiemetics, antidepressants, and pyloric injection with botulinum toxin and gastric electrical stimulator implantation in refractory cases [4, 8].

We report a case of a patient who presented with intractable nausea and vomiting on her initial diagnosis of SLE, and delay in gastric content clearance on multiple GES without evidence of mechanical obstruction. Although gastroparesis is not a typical feature of SLE, and coincidental idiopathic gastroparesis might be used as an explanation, this case raises the possibility of gastroparesis as a primary and severely symptomatic manifestation of SLE.

Disclosure

All authors declare there is no conflict of interest and no financial disclosures related to this case.

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