ISOLATED GASTRIC CROHN’S DISEASE: A RARE PRESENTATION OF A COMMON DISEASE

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ABSTRACT
Crohn’s disease is usually associated with intestinal disease when it affects the stomach. It is the most common cause of granulomatous gastritis; however, isolated gastric Crohn’s disease is an uncommon finding. This is a case report of a 26-year-old woman who presented with intermittent epigastric pain for 2 years and severe weight loss during the course of her illness. Paraclinical investigations ruled out the possibility of malignancy; further investigations revealed a short segment of focal thickening in the gastric antrum. However, small and large bowels were normal on abdominal computed tomography and severe form of hemorrhagic gastritis on upper gastrointestinal endoscopy. Multiple gastric biopsies obtained from the stomach were consistent with the diagnosis of Crohn’s disease. Treatment with prednisolone and azathioprine was effective in controlling the patient’s symptoms; however, these recurred 3 weeks after she interrupted therapy. Treatment was resumed with the same drugs, and she was symptom-free over the 18 months during which she was followed up. This case demonstrates the role of gastric biopsy as an important investigation in establishing the diagnosis of this rare presentation. It also reflects the dramatic response of gastric Crohn’s disease to the treatment with prednisolone and azathioprine.

Keywords: Crohn’s disease, Granulomatous gastritis, Gastric cancer, Prednisolone, Azathioprine.

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INTRODUCTION

Granulomatous gastritis (GG) is a rare entity of chronic gastritis and it represents 0.027-0.35% of gastric biopsies in the largest series reported by Shapiro et al. [1,2]. Although rare [3], Crohn’s disease (CD) was found to be the most common cause of GG [1,4]. Other less common causes for GG are sarcoidosis, gastrointestinal tuberculosis and reaction to malignancy [1,4-6]. In a few cases, *Helicobacter pylori* (*H. pylori*) was reported to be associated with GG, which resolved completely after eradication of *H. pylori* [4,7]. This is a case study of a young female patient who had severe upper gastrointestinal symptoms suggestive of possible malignancy, thus after investigation, diagnosed as a case of GG.

CASE PRESENTATION

A 26-year-old Saudi female was referred to the gastrointestinal clinic for suspected gastric malignancy. She presented with a 2-year history of intermittent epigastric pain that was colicky in nature, and aggravated by eating; for which improved spontaneously. The pain was of moderate intensity and did not affect her usual daily activities. Hence, it was associated with early post-prandial vomiting and anorexia. The vomitus was non-bloody and non-projectile, and did not ease the abdominal pain. Her history revealed a noticeable weight loss of about 34 kg within 2 years, and an undocumented intermittent low-grade fever of similar duration. There was no history of dysphagia, odynophagia, mouth ulcers, heartburn, early satiety, change in bowel habits, stool or urine color, jaundice, bone pain, convulsions, or focal neurological deficits. She had no other known medical illness and her surgical history was remarkable for adenoidectomy. She had received omeprazole 40 mg per day for two months prior to her consultation at our clinics without significant improvement of her symptoms. She had a history of smoking, and there was no previous exposure to tuberculosis (TB). Her family history was unremarkable.

On initial assessment, the patient was conscious, alert and oriented to time, place and person, and her vital signs were normal. She was pale and ill-looking. She had a weight of 44 kg, height 160 cm and body mass index of 17.2 kg/m². Head, ear, eye, nose, throat, and chest examination were normal. On abdominal examination, there was moderate epigastric and supra-umbilical tenderness, but no guarding or organomegaly. She did not have signs of thyrotoxicosis.

A provisional diagnosis was made for gastric malignancy. Other possibilities were gastric outlet obstruction from peptic stricture or thyrotoxicosis. Anorexia nervosa was thought of, but it was less likely.

Her initial laboratory examinations showed mild anemia and slightly low serum albumin (Table 1). Alpha-feto protein, carcinogenic embryonic antigen and cancer antigen19-9 were all normal. Abdominal computed tomography (CT) showed a short segment of focal thickening in the antrum, small and large bowels; abdominal lymph nodes and other abdominal organs were normal (Figure 1). An upper gastrointestinal endoscopy showed grade 2 gastroesophageal reflux disease (GERD), severe hemorrhagic gastritis of the body and antrum, as well as a small duodenal ulcer. Multiple gastric biopsies were obtained for histopathological analysis.

She was continued on omeprazole 40 mg per oral once daily. Histopathology results showed increased mononuclear inflammatory cells with many neutrophils and eosinophils. Cryptitis and crypt abscesses were present. Well-defined non-caseating granulomas composed of epithelioid histiocytes and multinucleated Langerhans giant cells were also seen. There was no evidence of malignancy; no acid fast bacilli, nor fungi were identified (Figure 2).

At this point, the differential diagnoses included gastric CD, tuberculosis and sarcoidosis. A repeat upper gastrointestinal endoscopy and gastric biopsy showed the same findings. Tuberculin test was performed, and it was negative (at 24 and at 48 hr). Based on these findings, CD was thought to be the cause of GG in the patient.

Table 1. Baseline laboratory results of the patient.

<table>
<thead>
<tr>
<th>Investigations</th>
<th>Results</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>9</td>
<td>12-15</td>
</tr>
<tr>
<td>White blood cell (x10^9)K/µL</td>
<td>3.6</td>
<td>4.5-11.5</td>
</tr>
<tr>
<td>Platelets (x10^9)K/µL</td>
<td>184</td>
<td>150-400</td>
</tr>
<tr>
<td>Prothrombin time (s)</td>
<td>13.1</td>
<td>11-14</td>
</tr>
<tr>
<td>Sodium (mmol/L)</td>
<td>138</td>
<td>136-145</td>
</tr>
<tr>
<td>Potassium (mmol/L)</td>
<td>3.1</td>
<td>3.5-5.1</td>
</tr>
<tr>
<td>Creatinine (µmol/L)</td>
<td>70</td>
<td>53-115</td>
</tr>
<tr>
<td>Calcium (mmol/L)</td>
<td>2.01</td>
<td>2.12-2.52</td>
</tr>
<tr>
<td>Albumin (g/L)</td>
<td>30</td>
<td>34-50</td>
</tr>
</tbody>
</table>

Figure 1. Abdominal computed tomography scan showing a short segment of focal thickening in the antrum (Arrow).
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She was started on prednisolone 30 mg per oral once daily. After one week on prednisolone, the vomiting and abdominal pain improved dramatically and she had gained 3 kg. After the second week of treatment, she was completely asymptomatic and her weight increased to 50 kg from her baseline of 44 kg. At the end of the third week, she weighed 53.7 kg; prednisolone was tapered gradually, but her symptoms recurred when it was reduced to 5 mg per day. The dose was then increased to 30 mg per day and azathioprine 50 mg per day was added to her treatment and gradually increased to 100 mg per day. The patient recovered completely but was lost to follow-up. She presented again for consultation when her symptoms recurred 3 weeks after she stopped treatment. The previous treatment regimen was resumed, with the dose of prednisolone gradually reduced when she improved until cessation of the drug. She was, however, maintained on azathioprine 75 mg daily and had no recurrence over the next 18 months.

DISCUSSION

This report describes the case of isolated gastric CD. CD rarely affects the stomach[5,7], and when it does, it is usually associated with intestinal disease. Cases of isolated gastric CD occur less commonly[7,8]. Nevertheless, the symptoms as well as the endoscopic findings in patients with gastric CD are non-specific[9]. Some patients may have severe symptoms in the presence of gastric outlet obstruction[10] as was the case in our patient. She may have had incomplete gastric outlet obstruction as evidenced by the narrowing of the pyloric canal on abdominal CT examination. Furthermore, the histological features were closely similar to those of gastric tuberculosis (TB) and sarcoidosis[4,5,9]; posing a challenge in disease management.

Misdiagnosis of TB as CD will result in patients being treated with steroids, and hence lead to severe progression of the disease and deterioration of their condition[9]. Severe weight loss was observed in this case, and raised the possibility of malignancy, which is another probable cause of GG[5]. A negative tuberculosis skin test made the possibility of gastric tuberculosis less likely. Gastric sarcoidosis was another diagnosis considered; however, the histological features in this patient were more of CD than gastric sarcoidosis. In areas with high or intermediate prevalence of tuberculosis, like Saudi Arabia, differentiating tuberculosis from CD is sometimes challenging due to similarities in the endoscopic, histological and radiological features[9,11].

The basic treatment of gastric CD involves the use of steroids and immunomodulators like azathioprine[12,13]. Treated patients are expected to have complete response to immune modulation with prednisolone and azathioprine[12,13], similar to our patient. However, as she had relapse on two occasions when prednisolone was reduced or stopped, continuation of a maintenance dose of azathioprine was recommended. The management plan must be individualized for each case and situation is based on certain collaborators, including disease location, severity, and extent. In addition, the presence of disease complications require proper treatment. After the initiation of the management plan, the course of therapy may be tailored according to diverse aspects, such as patient response and tolerance to the intervention. The dose suggested in clinical trials for prednisone was 0.5-0.75 mg/kg (or 40 mg) daily with a success in inducing remission of 50-70%[14-16]. Higher response rates were achieved with higher doses of prednisone (1 mg/kg) or methyl prednisolone[17,18]. With azathioprine; however, the dose evaluated in clinical trials was 2-3 mg/kg daily[19]. Other available options for treating CD is using the monoclonal antibody directed against TNF-α, infliximab, but it is usually reserved for cases that have not responded to the previous mentioned modes of therapy. Certainly, many surgical interventions like resection of the involved bowel segment, stricturoplasty or abscess drainage may play a role in treating complications the patient possibly develops within the course of severe diseases, or in case of medically refractory diseases. Another indication to be considered is if the patient developed intolerable side effects to the medications[20-22].

Finally, though GG is rare, it should be considered in patients non-responsive to acid suppression therapy. Gastric biopsy is of great value in establishing the diagnosis, as well as other immunological and endoscopic procedures will aide in differentiating CD from other causes of GG.
REFERENCES


