

# Laparoendoscopic Diagnosis of Eosinophilic Enteritis

Heron E. Rodriguez, MD, Risal S. Djohan, MD, William J. Cahill, MD,  
Mark M. Connolly, MD, Francis J. Podbielski, MD

## ABSTRACT

As the popularity of laparoscopic cholecystectomy continues to grow, evaluation of patients with documented cholelithiasis and concomitant vague abdominal complaints becomes less rigorous. We present the case of a patient with chronic cholecystitis documented by history and ultrasonography, incidentally noted on laboratory examination to have peripheral blood eosinophilia. At the time of laparoscopy, an inflamed segment of jejunum was discovered. Limited laparotomy and wedge biopsy revealed active eosinophilic enteritis.

**Key Words:** Laparoscopy, Eosinophilic enteritis.

## INTRODUCTION

First described by Kaijser in 1937,<sup>1</sup> eosinophilic enteritis is an uncommon gastrointestinal disorder characterized by symptoms of abdominal pain and partial obstruction. Laparotomy and biopsy reveals pathologic findings of eosinophilic infiltration of bowel wall in the absence of parasitic or extraintestinal disease. With less than 300 cases reported in the literature<sup>2</sup> the true prevalence of this condition is unknown. The diagnosis is established when histologic samples are obtained during endoscopy or laparotomy. The utility of laparoscopy makes it an ideal tool for evaluation of bowel obstructions of uncertain etiology. A paucity of cases appears in the laparoendoscopic literature describing serosal eosinophilic jejunitis diagnosed during diagnostic laparoscopy.<sup>3</sup>

This patient's presentation with colicky abdominal pain and emesis while suggestive of acute cholecystitis, is suspicious in the absence of liver enzyme abnormalities and poorly localized pain. Leukocytosis with eosinophilia was present on routine laboratory examination raising the possibility of eosinophilic enteritis in the differential diagnosis of this patient's distention and pain. Computed tomography showed a segment of proximal small bowel with a thickened wall. Exploratory laparoscopy revealed a loop of inflamed, thickened jejunum and the presence of turbid peritoneal fluid. Delivery of the loop of bowel and biopsy yielded the pathologic diagnosis.

## CASE REPORT

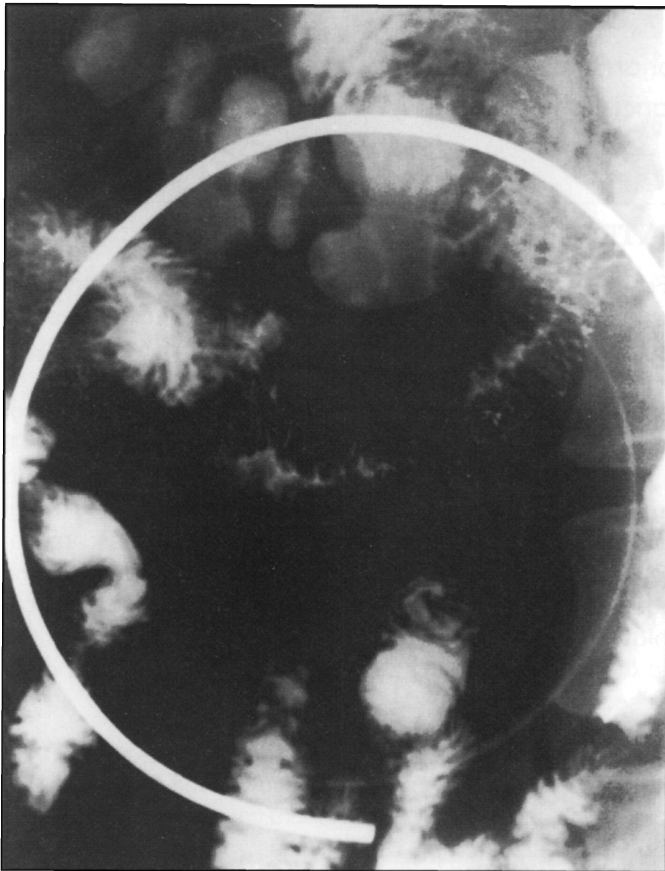
A 54-year-old woman presented with chief complaints of colicky epigastric pain and emesis of two weeks duration, exacerbated by eating. She is a retired nurse who emigrated from the Philippines eight years ago with no history of recent foreign travel or known infectious disease contacts. Her past medical history included only hypertension.

Physical examination demonstrated mild tenderness to deep palpation in the epigastric area without rebound or guarding. Laboratory analysis showed a leukocyte count of 16,000 with marked eosinophilia (14%). The serum chemistry and liver enzymes were within normal limits. Blood serology for giardia lamblia was negative, as were stool cultures. Chest and abdominal roentgenograms were

---

Columbus Hospital/Catholic Health Partners  
Department of Surgery  
Chicago, Illinois, USA

Address reprint request to: Francis J. Podbielski, MD, C.D.N. Surgical Associates,  
2515 N. Clark St., Suite 903, Chicago, Illinois 60614-2720, USA. Telephone: (773)  
472-3427, Fax: (708) 396-9869, E-mail: Fjpmteu@aol.com



**Figure 1. Upper gastrointestinal barium contrast study demonstrating extrinsic luminal narrowing of the proximal jejunum.**

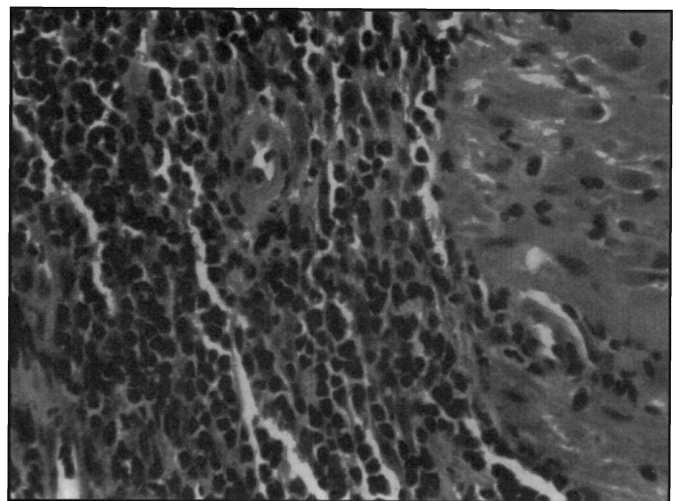


**Figure 2. Computed tomogram of the abdomen with oral and intravenous contrast showing a thickened loop of proximal small bowel.**

unremarkable. A barium contrast study of the small bowel suggested a segment of narrowing in the proximal jejunum (**Figure 1**). Abdominal ultrasonography showed a thickened gallbladder and cholelithiasis. 99m-Technetium-iminodiacetic acid (HIDA) scan revealed normal visualization of the biliary system and gallbladder. Esophagogastroduodenoscopy showed normal esophageal and gastric mucosa. Computed tomography of the abdomen with oral and intravenous contrast demonstrated marked thickening of a loop of proximal small bowel (**Figure 2**).

Video laparoscopy was performed under general anesthesia with an infraumbilical and three additional access ports. Upon entry to the abdomen, turbid peritoneal fluid and a grossly dilated loop of jejunum were evident. After performing an uneventful cholecystectomy and intraoperative cholangiogram, the transverse subxyphoid port site was extended to three centimeters in length and the questionable loop of jejunum externalized. A wedge resection was performed. On gross inspection the seromuscular layer was thick and erythematous, but the mucosa appeared normal. After repair of the enterotomy the bowel was returned to the abdomen and the wounds closed in layers.

Pathologic examination of the peritoneal fluid revealed a red blood cell count of 280,000/mm<sup>3</sup> and a white blood cell count of 22,000/mm<sup>3</sup>, with eosinophilia. Dense eosinophilic infiltration of the bowel serosa with normal histological mucosa was seen (**Figure 3**). The gallbladder showed signs of chronic cholecystitis without evidence of eosinophilic infiltration.



**Figure 3. Photomicrograph (Hematoxylin and Eosin stain, magnification 320X) of biopsied jejunal wall with a dense subserosal infiltration of eosinophils and a normal muscularis.**

The patient's hospital course was uneventful. She was discharged on postoperative day three and at four months follow-up remains asymptomatic.

## DISCUSSION

Eosinophilic enteritis most frequently involves the stomach and proximal small bowel. Lesions in the esophagus,<sup>4</sup> colon,<sup>5</sup> terminal ileum<sup>6</sup> and gallbladder<sup>7,8</sup> have also been reported. In 1970, Klein proposed a classification system based on the depth of histological infiltration with eosinophils.<sup>9</sup> Talley et al.<sup>10</sup> in their series of 50 patients identified mucosal infiltration in 23, muscle layer disease in 12, and subserosal involvement in only five cases. Ten patients had classic abdominal symptoms and peripheral blood eosinophilia, but no demonstrable gastrointestinal lesions. Hematologic studies show a range of blood eosinophilia from 8.5 to 80% of the total leukocyte count, with as many as one-fourth of patients having a normal eosinophil count. Fifty to 75% of patients with this disorder report a history of allergic hypersensitivity.

**Table 1.**

Clinical Manifestations of Eosinophilic Enteritis

Depth of Invasion	Symptoms
Mucosal	Post-prandial nausea and vomiting Weight Loss Malabsorption, iron deficiency anemia, steatorrhea Protein-losing enteropathy
Muscularis	Nausea and vomiting Abdominal distention Intermittent bowel obstruction
Subserosal	Abdominal pain and distention Ascites Pleural effusion

The presenting symptoms can vary depending on the segment of gastrointestinal tract involved as well as the depth of bowel wall eosinophilic infiltration (**Table 1**).<sup>11-13</sup> Patients with mucosal disease report abdominal pain, diarrhea, nausea and vomiting, and may manifest signs of intestinal malabsorption and protein losing enteropathy. The presentation of disease localized to the muscle layer disease is usually partial or complete obstruction. Symptoms again include abdominal pain and distention.

Serosal disease occurs infrequently with a total of 28 cases reported.<sup>14</sup> Abdominal pain and bloating are the prevailing complaints. Presentation with eosinophilic ascites, an elevated peripheral blood eosinophil count, and dramatic response to steroid treatment characterize this form of the disease. The eosinophil count of the ascitic fluid is usually elevated while abnormalities in the erythrocyte sedimentation rate and peripheral eosinophil count are variable.<sup>13</sup>

The radiographic presentation, depending on the segment involved, can suggest Crohn's disease, gastric outlet obstruction, or may simply show non-specific bowel wall thickening,<sup>15</sup> as illustrated in this case. The character of this patient's pain in the light of normal liver enzymes and a leukocytosis with eosinophilia prompted our investigation with computed tomography in an attempt to localize the affected segment of bowel.

A paucity of eosinophilic enteritis cases described during laparoscopy has been reported. Operative findings include ascites, and a hyperemic, thickened bowel with fibrinous exudate. The pathophysiology of this condition may be secondary to a derangement in the release of eosinophil major basic protein.<sup>16</sup> It remains unclear whether our patient's symptoms were attributable to her gallbladder disease or were a manifestation of ongoing serosal eosinophilic infiltration of her jejunum. Furthermore, although remote from the site of the gallbladder, it is possible that enteritis might have been a sequelae of chronic cholecystitis. Perhaps more aggressive investigation of local bowel abnormalities in acute cholecystitis will reveal some degree of eosinophilic infiltration.

The affected loop of jejunum appeared grossly abnormal through the videoscope; our impression may, however, have been biased by the preoperative radiographic findings. Eosinophilic gastroenteritis arose in our differential diagnosis only after a thorough hematologic evaluation. Whether or not the loop of jejunum in question would have appeared sufficiently abnormal as to prompt further investigation during a routine laparoscopy will remain in question.

The extension of the subxyphoid port site with extracorporeal wedge resection of the intestine added minimal morbidity and cost to our procedure. Given the number of laparoscopic procedures currently performed and the probable underdiagnosis of eosinophilic enteritis, we recommend patients undergoing laparoscopy who show evidence of peripheral blood eosinophilia, ascites, or ill-defined gastrointestinal symptoms undergo a thorough laparoscopic exploration of the abdomen. A sample of

intraperitoneal fluid can easily be obtained to rule out an infectious or neoplastic disease process, along with a minimally invasive biopsy or resection of bowel accomplished.

While previous cases of eosinophilic enteritis have been managed successfully with sodium cromoglycate,<sup>17,18</sup> the current recommended treatment is a short course of high dose prednisolone (40 milligrams per day for one week) followed by a maintenance regimen (5 to 10 milligrams per day). All patients in the study by Lee et al.<sup>14</sup> treated with this regimen had complete resolution of their symptoms and a return of their peripheral blood eosinophil count to normal within four weeks.

### References:

1. Kaijser R. Zur Kenntnis der allergischen Affektion der Verdauungskanal von Standpunkt des Chirurgen aus. *Arch Klin Chir.* 1937;188:36-64.
2. Talley NJ. Eosinophilic gastroenteritis. In Sleisenger MH, Fordtran JS, eds. *Gastrointestinal Disease: Pathophysiology, Diagnosis, Management.* Philadelphia: WB Saunders; 1993:1224-1232.
3. Solis-Herruzo JA, de Cuenca B, Munoz-Yague, MT. Laparoscopic findings in serosal eosinophilic gastroenteritis. Report of two cases. *Endoscopy.* 1988;20:152-153.
4. Riou PJ, Nicholson AG, Pastorino U. Esophageal rupture in a patient with idiopathic eosinophilic esophagitis. *Ann Thorac Surg.* 1996;62:1854-1856.
5. Naylor AR, Pollet JE. Eosinophilic colitis. *Dis Colon Rectum.* 1985;28:615-618.
6. Haberkern CM, Christie DL, Haas JE. Eosinophilic gastroenteritis presenting as ileocolitis. *Gastroenterology.* 1978;74:896-899.
7. Dabbs DJ. Eosinophilic and lymphoeosinophilic cholecystitis. *Am J Surg Pathol.* 1993;17:497-501.
8. Kerstein MD, Sheahan DG, Gudjonsson B, Lewis J. Eosinophilic cholecystitis. *Am J Gastroenterol.* 1976;66:349-352.
9. Klein NC, Hargrove RL, Sleisenger MH, Jeffries, GH. Eosinophilic gastroenteritis. *Medicine.* 1970;49:299-319.
10. Talley NJ, Shorter RG, Phillips SF, Zinmeister, AR. Eosinophilic gastroenteritis: a clinicopathological study of patients with disease of the mucosa, muscle layer, and subserosal tissues. *Gut.* 1990;31:54-58.
11. Kuri K, Lee M. Eosinophilic gastroenteritis manifesting with ascites. *South Med J.* 1994;87:956-957.
12. Cello JP. Eosinophilic gastroenteritis—A complex disease entity. *Am J Med.* 1979;67:1097-1104.
13. Fortman LM, Johanson JF, Baskin WN, Greenlaw, RL. Eosinophilic ascites without eosinophilia: a unique presentation of serosal eosinophilic gastroenteritis. *Am J Gastroenterol.* 1993;88:1280-1281.
14. Lee CM, Changchien CS, Chen PC, et al. Eosinophilic gastroenteritis: 10 years experience. *Am J Gastroenterol.* 1993;88:70-74.
15. Van Hoe L, Vanghillewe K, Baert AL, Ponette, E, Geboes, K, Stevens, E. CT findings in nonmucosal eosinophilic gastroenteritis. *J Comput Assist Tomogr.* 1994;18:818-820.
16. Torpier G, Colombel JF, Mathieu-Chandelier C, et al. Eosinophilic gastroenteritis: ultrastructural evidence for a selective release of eosinophil major basic protein. *Clin Exp Immunol.* 1988;74:404-408.
17. Heatley RV, Harris A, Atkinson M. Treatment of a patient with clinical features of both eosinophilic gastroenteritis and polyarteritis nodosa with oral sodium cromoglycate. *Dig Dis Sci.* 1980;25:470-472.
18. Moots RJ, Prouse R, Gumpel JM. Near fatal eosinophilic gastroenteritis responding to oral sodium cromoglycate. *Gut.* 1988;29:1282-1285.