

# The Efficacy and Tolerability of AST-120 (Spherical Carbon Adsorbent) in Active Pouchitis

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**OBJECTIVES:** Although a majority of patients with pouchitis respond favorably to antibiotic therapy, many relapse frequently, and nonabsorbable and non-antibiotic-based agents are desirable for reducing bacterial resistance and the systemic adverse effects associated with long-term antibiotic exposure. AST-120 (a spherical carbon adsorbent) comprises highly adsorptive, porous carbon microspheres with the ability to adsorb small-molecular-weight toxins, inflammatory mediators, and harmful bile acids. The aim of this pilot trial was to evaluate the efficacy and tolerability of AST-120 in the treatment of active pouchitis.

**METHODS:** Eligible patients were recruited from two subspecialty pouchitis clinics. Inclusion criteria were (i) ileal pouch-anal anastomosis performed for ulcerative colitis; (ii) active pouchitis with Pouchitis Disease Activity Index (PDAI) scores  $\geq 7$ ; and (iii) discontinuation of antibiotic therapy for at least 2 weeks. Exclusion criteria included Crohn's disease of the pouch, isolated cuffitis, pouch strictures, abscess, and sinuses. All eligible patients received AST-120 in 2-g sachets (oral) open label, thrice a day for 4 weeks. The primary efficacy end point was remission as defined by a PDAI score of  $< 7$  points; the main secondary end point was clinical response, defined by a reduction of the PDAI score of  $\geq 3$  points.

**RESULTS:** Nineteen of 20 patients completed the trial. Eleven patients (55.0%) had a clinical response to the therapy and 10 patients (50.0%) entered remission. Median reduction in the PDAI symptom, endoscopy, and histology subscores, and PDAI total scores after 4 weeks were  $-2$  ( $P=0.002$ ),  $-2$  ( $P=0.003$ ),  $0$  ( $P=0.32$ ), and  $-4$  ( $P=0.001$ ) points, respectively. The agent was well tolerated; one patient experienced transient mild elevation of alkaline phosphatase of uncertain significance and one patient experienced an upper respiratory infection after taking one dose of AST-120 and was excluded from the final analysis for the calculation of pre- and post-trial PDAI scores.

**CONCLUSIONS:** AST-120 seems to be effective and well tolerated in treating patients with active pouchitis. A randomized, placebo-controlled trial is warranted for assessing the long-term efficacy and safety of AST-120 in the disease.

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## INTRODUCTION

Restorative proctocolectomy with ileal pouch-anal anastomosis (IPAA) is the preferred surgical treatment for patients with ulcerative colitis who require colectomy. Pouchitis, a non-specific inflammatory condition of the pouch reservoir, is the

most common long-term complication of IPAA (1–5). The etiology of pouchitis is not well understood, but likely involves alterations in luminal bacteria (e.g., bacterial overgrowth) and subsequent dysregulation of inflammatory responses in genetically susceptible patients (1,3,6). The efficacy of antibiotics and

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probiotics in treating pouchitis provides additional evidence supporting the role of bacterial alterations in the pathophysiology of pouchitis (1,7). In addition, bacterial toxins (6,8–10), hydrogen sulfide (11), and metabolic products of bile acids (12–20) may contribute to pathogenesis and symptom development.

Antibiotic therapy has been the mainstream medical treatment for pouchitis. Although a majority of patients with pouchitis respond favorably to the therapy, there are concerns regarding bacterial resistance, adverse effects, and disease relapse (3,5,21–23). Some patients develop a refractory course after long-term antibiotic use. In fact, pouchitis can be classified into antibiotic-responsive, antibiotic-dependent, and antibiotic-refractory types (2,7). For these reasons, efforts have been made to identify non-antibiotic-based agents, such as budesonide enemas (24), alicaforsen enemas, an anti-sense inhibitor of intercellular adhesion molecule-1 (25), and probiotics (26). These agents have been evaluated in open-label trials with some success.

AST-120 (a spherical carbon adsorbent) is an agent that is comprised of highly adsorptive, porous, spherical carbon particles of ~0.2–0.4 mm diameter with high adsorption ability and large surface area (1,600 m<sup>2</sup>/g). The agent has been used in Japan since 1991 for the treatment of patients with chronic kidney disease (27), as well as for fistulizing Crohn's disease (28). The clinical utility of AST-120 is thought to reside in its ability to adsorb small-molecular-weight toxins (29,30), inflammatory mediators, and harmful bile acid products from the gastrointestinal tract, preventing local toxicity and their systemic absorption. AST-120 has a selective adsorption profile for certain acidic and basic organic compounds, and has a lower adsorptive capacity than activated charcoal for digestive enzymes. The aim of our trial was to evaluate the efficacy and safety of AST-120 in a 4-week, open-label trial in patients with active pouchitis.

## METHODS

### Patients

Eligible patients were recruited from the Pouchitis Clinic, the Digestive Disease Institute, Cleveland Clinic, Cleveland, OH and Inflammatory Bowel Disease Clinic, Division of Gastroenterology and Hepatology, Mayo Clinic, Rochester, MN. The trial was approved by the Institutional Review Boards at Cleveland Clinic and Mayo Clinic. Written informed consent was provided by all patients.

### Inclusion and exclusion criteria

The inclusion criteria were (i) active pouchitis (defined as having a Pouchitis Disease Activity Index (PDAI) (31) score  $\geq 7$  points) after IPAA for ulcerative colitis and having presented with primary symptoms, such as increased stool frequency and abdominal pain; (ii) 18–75 years of age; (iii) able to give informed consent; (iv) able and willing to comply with all trial procedures; and (v) females in post-menopausal stage, surgically incapable of bearing children, or practicing a reliable

single-barrier method of birth control (condom, intrauterine devices, spermicide and barrier, Depot). Partner/spouse sterility could also qualify at the investigator's discretion. Female patients with childbearing potential must have had a negative urine pregnancy test at baseline.

The exclusion criteria were (i) having been treated earlier with infliximab or any investigational immunomodulators; (ii) antibiotic use within 2 weeks before the entry of the trial; (iii) Crohn's disease of the pouch; (iv) active specific infection of the pouch: cytomegalovirus infection and *Clostridium difficile* infection; (v) history of non-inflammatory disease of the pouch: decreased pouch compliance, irritable pouch syndrome, afferent or efferent limb obstruction; (vi) isolated cuffitis; (vii) strictures of the pouch inlet or outlet; (viii) ileal pouch patients with familial adenomatous polyposis; (ix) history of lactose intolerance; (x) known celiac disease; (xi) primary sclerosing cholangitis with or without liver transplant and with or without ursodeoxycholic acid therapy; (xii) uncontrolled systemic diseases; (xiii) needing oral or topical steroid treatment or 5-aminosalicylate (5-ASA) agents; (xiv) other major physical or major psychiatric illness within the last 6 months that in the opinion of the investigator would affect the patient's ability to complete the trial; (xv) active use of cholestyramine, and non-steroidal anti-inflammatory drugs or aspirin; and (xvi) being pregnant, breast feeding, or planning to become pregnant during the trial.

### Trial design

This was an open-label pilot trial, in which all patients received AST-120 in 2-g sachets (oral) thrice a day for 4 weeks. All antibiotics, probiotics, and nutritional agents must have been discontinued for at least 2 weeks before trial entry. For each eligible patient, clinical evaluation with symptom assessment and physical examination, pouch endoscopy with biopsies, and laboratory tests (complete blood counts, basic metabolic panel, liver function panel, stool *C. difficile* toxins A and B, and urine pregnancy tests (for female patients)) were performed at the time of entry into the trial and at the end of the 4-week trial. Total PDAI scores and PDAI symptom (range from 0 to 6), endoscopy (range from 0 to 6), and histology (range from 0 to 6) subscores were documented at the time of entry into the trial and at the end of the trial. Blinded gastrointestinal pathologists at each institution graded inflammation of the pouch using PDAI histology subscores. Dysplasia, viral inclusion bodies, or granulomas, if present, were documented.

Patients were checked through telephone or e-mail on a weekly basis by the trial coordinators for symptom response, compliance, and development of adverse effects.

Any co-prescribed medicines must have been given at least 30 min before AST-120 administration. Non-narcotic anti-diarrheal agents could be co-prescribed or maintained during AST-120 treatment if the dose had been stabilized for 2 weeks at the time of entry into the trial. The utilization (dosage and duration) of anti-diarrheal drugs was recorded as a secondary end point.

### Efficacy end points

The primary efficacy end point was induction of remission as defined by a PDAI score of <7 points. The secondary efficacy end points were (i) response defined as a  $\geq 3$ -point reduction in the 18-point PDAI scoring system; and (ii) reduction in the PDAI symptom, endoscopy, and histology subscores.

### Safety end points

The primary safety end point was any adverse event deemed as possibly, probably, or definitely related to treatment with the investigational product during 4 weeks of treatment. The secondary safety end points were abnormalities in clinical laboratory tests (complete blood count, basic metabolic panel, and liver function tests), worsening gastrointestinal symptoms (diarrhea, abdominal pain, urgency or bleeding), or new gastrointestinal and systemic symptoms (headache, nausea, vomiting, and constipation), and new abnormal physical examination findings, such as blood pressure, heart rate, respiration rate, and temperature, at the end of the trial.

### Treatment failure

Treatment failure and criteria for trial discontinuation were defined as (i) the need to prescribe antibiotics, anti-inflammatory agents and/or immunosuppressants, probiotics, nutritional agents (short-chain fatty acid enemas or suppositories, glutamine, and additional dietary fibers), and narcotic-based antidiarrheal agents as “rescue” medications; (ii) patient’s withdrawal from the trial; and (iii) no change or worsening of the PDAI score at the end of the trial.

### Statistical analyses

Treatment remission and response were analyzed by intention-to-treat design. Total PDAI scores and PDAI subscores were analyzed in those patients who completed the 4-week therapy. Wilcoxon rank sum tests were carried out to assess differences in the pre- and post-treatment PDAI total scores and subscores after 4 weeks of treatment. *P* values <0.05 were considered to be statistically significant.

## RESULTS

Eligible patients were recruited from the Pouchitis Clinic, Cleveland Clinic and Inflammatory Bowel Disease Clinic, Mayo Clinic. A total of 24 patients were screened at the Cleveland Clinic site and 4 had screening failures. A total of seven consecutive patients with antibiotic-refractory pouchitis offered to participate in the study at the Mayo Clinic. Three patients entered the study with data analyzed, two opted for other medical therapies, and two were screen failures.

At the time of screening, daily antibiotic use by the participants was as follows: none (*n*=3); rifaximin (*n*=6); metronidazole (*n*=3); tinidazole (*n*=6); ciprofloxacin (*n*=5); and itraconazole (*n*=1). Some of the patients had been on antibiotic combination therapy. Antibiotic use was discontinued for at least 2 weeks before entry into the study, as per the study protocol.

A total of 20 patients signed their consent for the trial and received AST-120 and all 20 patients were analyzed for clinical response and remission. A total of 19 patients were included in the final analysis for the calculation of pre- and post-trial PDAI scores. None of the patients had significant laboratory abnormalities at baseline. All the patients tested negative for *C. difficile* toxins A or B at the time of entry into the trial.

Demographic and clinical data of the 20 patients who were enrolled into the trial are listed in **Table 1**.

### Efficacy

Of the 20 patients who were enrolled, 19 completed the trial. One patient developed an upper respiratory infection on the second day and discontinued treatment on the same day. Eleven patients (55.0%) had a clinical response to the therapy and 10 patients (50.0%) entered remission. For the responders, improvement of symptoms typically occurred within 2 weeks after initiation of the therapy. Changes in the PDAI subscores and total scores in the 19 cases who completed 4 weeks of therapy are summarized in **Table 2**. There were statistically significant reductions in the PDAI symptom subscores (median reduction: 2.0 points, *P*=0.002), endoscopy subscores (median reduction: 2 points, *P*=0.003), and total scores (median reduction: 4 points, *P*=0.001). The PDAI histology subscore showed a median reduction of 0 point, which was not statistically significant (*P*=0.32) (**Figure 1**).

Of the 19 patients who completed the study, 1 patient (5.3%) had never had pouchitis before entry into the trial, 2 (10.5%) had antibiotic-responsive pouchitis, 13 had antibiotic-dependent pouchitis (68.4%), and 3 (15.8%) had antibiotic-refractory pouchitis. Of the 2 patients with antibiotic-responsive pouchitis, 2 had a clinical response and 1 had clinical remission; of the 13 patients with antibiotic-dependent pouchitis, 7 had a clinical response and 8 achieved clinical remission; and of the 3 patients with antibiotic-refractory pouchitis, 2 had a clinical response and 1 achieved remission.

Dysplasia, viral inclusion bodies, or granulomas were not found in biopsy specimens from pouch endoscopy before and after the trial.

### Adverse effects

None of the female patients tested positive for pregnancy before and after the trial. None of the patients, including non-responders, experienced worsening of any symptoms. The agent was well tolerated. One patient developed an upper respiratory infection after taking one dose of the agent and terminated the trial on the second day. One patient developed transient mild elevation of alkaline phosphatase. Subsequent liver function panels were normal at 1 and 3 months after the trial. One non-responder was found to have developed *C. difficile* infection at the 4-week follow-up visit, and was started on oral vancomycin at the end of the trial. None of the non-responders required rescue medicines.

**Table 1. Demographic and clinical data (n=20)**

Variable	Overall summary
Age (years)	44.0±11.1
Male gender	14 (70.0%)
Caucasian race	20 (100%)
Family history of Crohn's disease	1 (5.0%)
Family history of ulcerative colitis	2 (10.0%)
Family history of colon cancer	0
Family history of celiac disease	1 (5.0%)
Current smoker	2 (10.0%)
Ex-smoker	7 (35.0%)
Regular alcohol drinker	1 (5.0%)
Osteoporosis	2 (10.0%)
Renal stones	4 (20.0%)
Depression	3 (15.0%)
Coronary artery disease	2 (10.0%)
Hepatitis C	1 (5.0%)
Breast cancer	1 (5.0%)
Current weight loss >5%	2 (10.0%)
Pre-op diagnosis of ulcerative colitis	17 (85.0%)
Pre-op diagnosis of indeterminate colitis	3 (15.0%)
Duration of ulcerative/indeterminate colitis, yrs	15.1±7.7
Pancolitis	18 (90.0%)
Colectomy for refractory colitis	16 (80.0%)
Colectomy for dysplasia	4 (20.0%)
Duration of pouch, yrs	6.0±4.0
J pouch configuration	19 (95.0%)
2-stage pouch surgery	10 (50.0%)
<i>History of pouchitis</i>	
None	1 (5.0%)
Antibiotic-responsive pouchitis	2 (10.0%)
Antibiotic-dependent pouchitis	14 (70.0%)
Antibiotic-refractory pouchitis	3 (15.0%)
<i>Extraintestinal manifestations</i>	
Arthralgia	10 (50.0%)
Thromboembolic events	1 (5.0%)
<i>Concurrent meds at the entry of and during trial</i>	
None	5 (25.0%)
Antidiarrheal	8 (40.0%)
Topical narcotics	3 (15.0%)
Topical or oral 5-ASAs	1 (5.0%)
Antidepressants	4 (20.0%)

**Table 1. Continued**

Variable	Overall summary
Anti-anxiety	5 (15.0%)
Topical corticosteroid	1 (5.0%)
Oral budesonide	1 (5.0%)
Aspirin	2 (10.0%)
Statins	2 (10.0%)
Estrogens	1 (5.0%)

## DISCUSSION

Active pouchitis can usually be treated effectively with antibiotics, making these agents the mainstay of treatment. For acute pouchitis, the first-line therapy includes a 14-day course of metronidazole (15–20 mg/kg/day) or ciprofloxacin (1,000 mg/day) (32,33). A randomized trial of ciprofloxacin and metronidazole showed that patients treated with ciprofloxacin experienced significantly greater reductions in the PDAI scores and fewer adverse effects than those treated with metronidazole (32). A small randomized trial of oral rifaximin 1,200 mg/day vs. placebo showed a marginal therapeutic benefit for active pouchitis (34). A recent open-label trial of a highly concentrated probiotic preparation (VSL#3, 900 billions/sachet lyophilized viable bacteria) showed that a high dose (3,600 billion bacteria/day) for 4 weeks was effective in the treatment of mildly active pouchitis, with 16/23 patients (69%) being in remission after treatment (26).

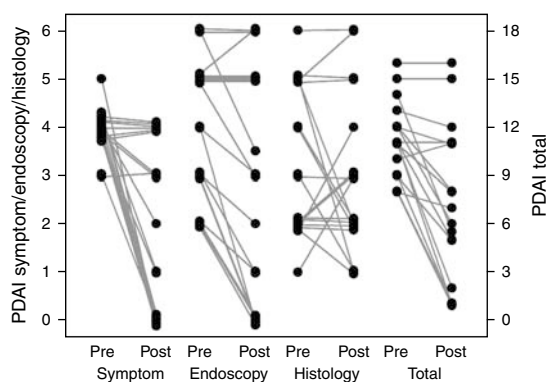
This open-labeled study showed that AST-120 may be safe and effective in treating active pouchitis. There are several strengths of this study. First, we applied an agent with an accepted track record of long-term tolerability and safety. AST-120 has been prescribed for treatment of chronic renal failure in Japan for almost two decades. Second, extensive and/or prolonged use of antibiotics in a pouchitis population seems to result in an increasing number of patients with chronic antibiotic-refractory pouchitis, which requires more aggressive antibiotic therapy (such as a combination of two antibiotics) (35,36), 5-ASA agents (35), corticosteroids (37), immunomodulators, and even biological agents (38). Finally, extensive use of antibiotics may have resulted in a high prevalence of *C. difficile* infection (up to 18% of patients in a recent study) (10). We recently reported a case with fulminant pouchitis from *C. difficile* infection with a fatal outcome (Shen B, et al. *Nat Rev Gastroenterol Hepatol*, unpublished data). Chronic pouchitis is a leading cause of pouch failure. There is an urgent need of non-antibiotic-based, safe, and efficacious agents for treating pouchitis. The efficacy seen in this open-label trial forms the basis for future randomized, placebo-controlled trials, which would have a potentially great impact on clinical management of pouchitis.

Relapse of pouchitis is common. Remission maintenance for this relapsing pouchitis often requires long-term antibiotic therapy (1–3,32,35,39) or probiotic therapy (38,40,41). As

**Table 2.** Treatment outcome (*n*=19 analyzable patients)

	Before treatment	After treatment	Reduction in score	<i>P</i> value
PDAI symptom score (0–6)	4 (4, 4)	2 (0, 4)	–2 (–4, 0)	0.002
PDAI endoscopy score (0–6)	4 (3, 5)	3 (0, 5)	–2 (–4, 0)	0.003
PDAI histology score (0–6)	3 (2, 5)	2 (2, 3.5)	0 (–1.5, 0.5)	0.32
Total PDAI score (0–18)	11 (9.5, 12.5)	7 (3.5, 11)	–4 (–7, –0.5)	0.001

PDAI, Pouchitis Disease Active Index.  
Values reported in median (interquartile range).

**Figure 1.** Plot of Pouchitis Disease Activity Index (PDAI) total and sub-scores before and after therapy.

long-term or frequent antibiotic therapy can be associated with antibiotic resistance and increased adverse effects with prolonged administration, safe maintenance of remission in antibiotic-dependent pouchitis can be a challenging task (1). Our trial did not address the question of how to maintain remission once it is induced. A follow-up trial is warranted to evaluate whether AST-120 is efficacious in maintaining remission.

The therapeutic benefits of antibiotic therapy are often correlated with the restoration of bacterial composition and quantity of the pouch (6,8). The mechanism of action of AST-120 in pouchitis is not known. We speculate that the agent may help neutralize bacterial toxins, similar to those found in *C. difficile*-associated pouchitis (9,10), or help in adsorption of abnormal products of bile acid metabolism. An increased microbial load in the pouch may lead to an abnormal metabolism of bile acids, which can exacerbate mucosal inflammation. For example, an increased bacterial load can cause an increased deconjugation of bile acids. Anaerobic bacteria may also modify conjugated (primary) bile through 7- $\alpha$  dehydroxylation of cholic acid and chenodeoxycholic acid, to produce secondary bile acids deoxycholic acid and lithocholic acid, respectively. The production of unconjugated and secondary bile acids is greater in patients with IPAA than in patients with end ileostomies (12). In addition, reabsorption of bile acids, for unknown mechanisms, is impaired in patients with IPAA (13–15). The possible etiologies for impaired bile acid reabsorption include inflammation of the

pouch (16), reduced mucosal surface area of the terminal ileum after the construction of IPAA, or deconjugation of bile acids as a result of the increased microbial load or reduced expression of bile-acid-transporting proteins (17). The accumulation of unabsorbed bile acids can exert adverse effects on the pouch mucosa. Secondary bile acids, through their detergent effect, can directly injure the ileal pouch mucosa, resulting in villous atrophy and infiltration of chronic inflammatory cells of the lamina propria (17). Secondary bile acids may also cause diarrhea through an osmotic effect (18). In an *in vitro* experiment, an ileal pouch dialysate containing unabsorbed bile acids was shown to be cytotoxic to the intestinal epithelial cell lines, which can be inhibited by administration of cholestyramine (19). A randomized, cross-over, placebo-controlled clinical trial of dietary inulin (a prebiotic agent) has shown that the agent reduced endoscopic and histological inflammation of the pouch corresponding to diminished concentrations of secondary bile acids in feces (42). As AST-120 is known to avidly bind bile acids, we speculate that its interference with bile acids mediated its beneficial effects in pouchitis (Ocera Therapeutics, data on file). In addition, an *in vitro* study showed that AST-120 may neutralize hydrogen sulfate production by bacteria (personal communication with Dr Michael Levit, University of Minnesota).

This trial had several limitations, including the open-label design. The designed dosing of the trial was “empirical” and the ultimate dosage for effective therapy would need a dose-ranging trial. As the trial involved two of the largest pouchitis practices in the United States, the Cleveland Clinic and Mayo Clinic, there might have been referral bias. Until now, there have been no completely objective markers for diagnosing pouchitis and for monitoring treatment response. Fecal lactoferrin has been proposed in the diagnosis and differential diagnosis of pouchitis (43). A longitudinal study on the diagnostic and monitoring value of fecal lactoferrin is currently underway. Among the other limitations of the study was the use of PDAI instrument as a measurement of efficacy of the treatment. Although the PDAI instrument has some pitfalls and has not been validated systemically, it has been the most commonly used research instrument for prospective trials in pouchitis in the literature. There are no other established diagnostic instruments for pouchitis available that are prospectively validated. In our earlier study, we found that PDAI symptom, endoscopy,

and histology subscores were often not correlated (44). Of the three subscores, endoscopic evaluation is the most powerful tool for the diagnosis and differential diagnosis of pouchitis (43). The study also showed that histology evaluation alone is not accurate enough for the distinction between pouchitis and other inflammatory and functional conditions of the pouch (43). We have emphasized in our earlier studies and review articles that histological evaluation has a limited value in grading pouch inflammation, which was one of reasons why the modified PDAI score (eliminating the histology subscore from the PDAI) was proposed (45). The main role of histological evaluation is to identify granulomas (for Crohn's disease), pyloric gland metaplasia (as a marker for chronic mucosal injury), dysplasia, viral inclusion bodies (for cytomegalovirus infection), and ischemic changes. Therefore, we believe that endoscopic evaluation may provide the most objective and accurate judgment for treatment response among the three subscores of the PDAI. However, we acknowledge that in an open-label study, subjective outcome measures are more subject to bias, and thus there is a need for randomized controlled trials.

In conclusion, AST-120 was well tolerated, and seemed to be efficacious in the induction of remission or response in patients with active pouchitis. A randomized, placebo-controlled trial is warranted.

#### CONFLICT OF INTEREST

**Guarantor of the article:** Bo Shen, MD.

**Specific author contributions:** Concept, study design, patient recruitment, clinical and endoscopic evaluation, and paper preparation: Bo Shen; concept, study design, patient recruitment, clinical and endoscopic evaluation, and paper review: Darrell S. Pardi; histology evaluation: Ana E. Bennett; patient recruitment, follow-up, and data entry: Elaine Queener; patient recruitment, follow-up, and data analysis: Patricia Kammer; statistical analysis: Jefferey P. Hammel; concept and data monitoring: Caroline LaPlaca; study design and paper review: M. Scott Harris.

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**Potential competing interests:** In addition to receiving the research grant, Bo Shen has served as a consultant for Ocera Therapeutics, Inc., San Diego, CA. The Cleveland Clinic and Mayo Clinic maintain policies requiring that certain disclosures of financial interests accompany papers submitted for publication. These financial interests with companies must be disclosed by coauthors from Cleveland Clinic and Mayo Clinic whose research is sponsored by the companies or whose products (or direct and primary competitor's products) are discussed in the paper. In accordance with this policy, we are disclosing that we have received within the last year or will receive in the coming year the following forms of support from the companies listed. Bo Shen, MD: honoraria, UCB, Centocor, Salix, Abbott; research grant: Ocera; Darrell Pardi: research grant, Ocera; Elaine Queener, LPN: research support, Ocera; Patricia Kammer: research support, Ocera.

## Study Highlights

### WHAT IS CURRENT KNOWLEDGE

- ✓ Pouchitis is the most common long-term complication of restorative proctocolectomy.
- ✓ The majority of patients with pouchitis respond favorably to antibiotic therapy.
- ✓ Frequent or long-term antibiotic use can be associated with bacterial resistance and adverse effects.
- ✓ Non-antibiotic-based agents for treatment of pouchitis are needed.

### WHAT IS NEW HERE

- ✓ AST-120 (a spherical carbon adsorbent) was well tolerated and seemed to be efficacious in induction of remission of pouchitis.

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