LETTERS TO THE EDITOR

June 24, 1990

Dear Editor:

Drs. Lohmuller, Pemberton, Dozois, Ilstrup, and Van Heerdon in their article "Pouchitis and Extraintestinal Manifestations of Inflammatory Bowel Disease After Ileal Pouch–Anal Anastomosis" discuss the relationship between pouchitis, extraintestinal manifestations, indeterminate colitis, and Crohn's disease.[1] They do not mention the autoimmune response mechanism and immunosupressive therapy that may be a common factor. Present[2] assumes a 5% to 10% failure with the present surgery and suggests a medical trial before extensive surgery. His indications for the use of immunosurpressive therapy include (1) failure to respond to steroids and sulfasalazine; (2) steroid toxicity and continuous steroids for Crohn's disease; (3) patients with proctosigmoiditis who have not responded to oral and topical therapy; and (4) patients with left-sided or universal disease who are continually active and have not had disease long enough to be at risk for carcinoma of the colon. He has found 6-MP and azathioprine to be effective in 60% to 70% of patients and toxicity infrequent and reversible and no definite increase in superinfections or neoplasms in long-term use.

Cyclosporin is under investigation and does seem to show promise for inflammatory bowel disease (IBD) as it does for organ transplant immunosupressive therapy.[3] According to Riskin et al.,[4] synthetic carbohydrates may represent an important new class of drugs for the treatment of inflamatory, autoimmune diseases. Effectiveness of these drugs should lend credence to the theory that the nature of IBD is autoimmune and add another dimension, besides surgery, to its treatment.

References

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2. Present DH. 6-mercaptopurine and other immunosuppressive agents in the treatment of Crohn's disease and ulcerative colitis. Gastroenterol Clin NA 1989; 18:57–71.
3. Lichtiger S, Present DH. Cyclosporin A in the treatment of severe, refractory ulcerative colitis. Gastroenterology 1989; 96:A301.
4. Riskin WG, Gillings DB, Scarlett JA. Amiprilose hydrochloride for rheumatoid arthritis. Am JI Int Med 1989; 111:455–464.

LAWRENCE BRASLOW, M.D.
Riverside, California

August 28, 1990

Dear Editor:

In response to Dr. Lawrence Braslow's comments regarding our article, we would point out that the purpose of our study was not to investigate the etiology of ulcerative colitis nor its medical management but rather to study the relationship we have seen between pouchitis and extraintestinal manifestations of ulcerative colitis after ileal pouch–anal anastomosis. Be that as it may, at the conclusion of our discussion we do, in fact, suggest that the pathophysiologic mechanisms involved in pouchitis may be similar to those involved in chronic ulcerative colitis. Of course it is highly speculative whether this is an autoimmune process or some other mechanism.

Although Dr. Braslow's comments are appreciated, we cannot speak to them because they involve a subject far broader than our study.

JOHN H. PEMBERTON, M.D.
JOSEPH L. LOHMULLER, M.D.
Rochester, Minnesota

September 10, 1990

Dear Editor:

I read with interest the paper by Dr. Klein and colleagues entitled "Current Management of the Budd-Chiari Syndrome" in the August 1990 issue of *Annals of Surgery.*

In their discussion they did not mention the nonsurgical treatment of Budd-Chiari syndrome. The authors listed several causes of hepatic venous outflow occlusion, including membranous obstruction of the suprahepatic inferior vena cava. The latter, which is more common in the Orient, is suited ideally for nonoperative treatment by percutaneous balloon dilation. My colleagues from China and I have now successfully treated six such patients, three of whom had previous surgical procedures. Improvement was dramatic and long lasting.

Therefore, in the current management of the Budd-Chiari syndrome, one form, specifically membranous obstruction of inferior vena cava, which can be accurately diagnosed by angiography, could and should be treated nonsurgically by percutaneous balloon dilation. The latter approach is an effective and safe alternative to surgery.

TSUNG O. CHENG, M.D.
Washington, D.C.

October 2, 1990

Dear Editor:

Obstruction of hepatic venous drainage can result from disease at several anatomic locations including (1) nonthrombotic veno-occlusive disease originating in the terminal hepatic venules, (2) thrombotic occlusion of the hepatic veins or suprahepatic vena cava, and (3) membranous obstruction of the suprahepatic inferior vena cava. It has been common practice to group this diverse array of disorders together as the Budd-Chiari syndrome. Much of the confusion and debate concerning the optimal treatment for these patients could be avoided if such practice was abandoned.

I would agree with Dr. Cheng that invasive radiologic techniques may be appropriate treatment for selected patients who develop isolated membranous obstruction of the suprahepatic vena cava, and the successful outcome of such strategy has been published previously.[1,2] Percutaneous laser-assisted angioplasty has also been used in similar cases.[3,4] Membranous vena caval obstruction, although a common etiology of the Budd-Chiari syndrome worldwide, is rare in the United States except in those areas with a large population of Oriental immigrants.[5] Our discussion concerning the surgical management of patients with the Budd-Chiari syndrome was directed toward patients with