

[2276] **Figure 1.** A nonexpansile patchy intramedullary signal abnormality from the level of C2-3 to the level of C6, slightly asymmetric to the right with no evidence of cord compression. Nonspecific however suspicious for inflammatory etiology such as transverse myelitis.

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Role of Oral Vancomycin in Inducing Remission for Biologic Experienced Ulcerative Colitis With Concomitant Primary Sclerosing Cholangitis and Liver Transplantation

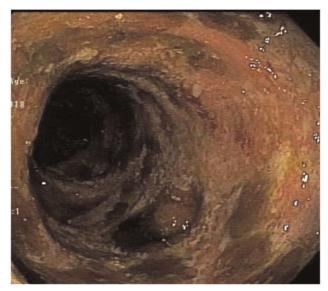
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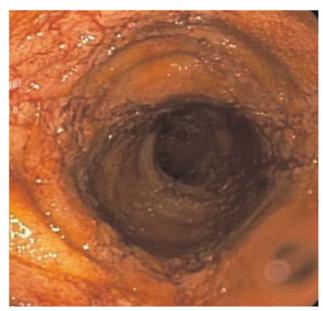
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INTRODUCTION: Ulcerative colitis associated with primary sclerosing cholangitis (UC-PSC) is distinct entity. One third of patients with UC may experience worsening colitis despite on immunosuppression after liver transplantation (LT) for PSC. We report a case of severe UC after LT for PSC refractory to Vedolizumab and Infliximab that responded to oral vancomycin (OV).

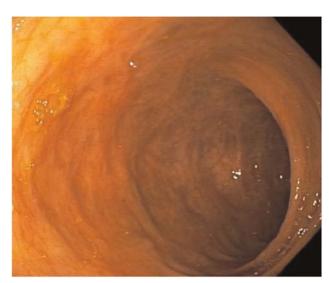
CASE DESCRIPTION/METHODS: A 51-year-old man with mild ulcerative pancolitis and PSC presented with worsening bloody diarrhea. His UC was diagnosed at age 12, is mild and responding to oral mesalamine. He had LT for PSC 7 months ago, with normal post-transplant course on mycophenolate mofetil (MMF) and tacrolimus. Stool testing for Clostridium difficile, ova/parasites, and cultures were negative. Colonoscopy showed severe active (Mayo score 3) ulcerative pancolitis (Figure 1). Pathology showed severe inflammation, crypt abscesses, and negative CMV. Prednisone showed improvement. MMF switched to delayed release MMF. Vedolizumab started with tapering steroids, however patient had recurrence of bloody diarrhea, led to increasing frequency of Vedolizumab at week 14 of treatment. Repeat colonoscopy at 20 weeks of initiating Vedolizumab showed



[2277] Figure 1. Severe (mayo 3) inflammation in the descending colon.



[2277] Figure 2. Severe (Mayo 3) colonic inflammation.



[2277] Figure 3. Endoscopic remission seen after initiation of Oral Vancomycin.

severe pancolitis (Mayo score 3) (Figure 2). Vedolizumab switched to Infliximab. Patient responded, hence prednisone was tapered off. However, had episodes of bloody diarrhea requiring 2 courses of prednisone, frequency of Infliximab was increased after 22 weeks of treatment initiation. After another flare requiring prednisone, infliximab levels checked were adequate at 19 mg/ml without antibodies. At this point, OV was started. In the following 3 months, had significant clinical improvement and did not require prednisone. Repeat colonoscopy showed resolution of inflammation-Mayo endoscopic score of 1 (Figure 3). Patient still on OV and reduced dose of Infliximab without flares.

DISCUSSION: UC-PSC has its unique pathophysiology and genetics, and therefore it responds differently than UC. They have gut microbiota less diverse compared to healthy controls, which could explain the refractory course and the utility of OV. OV has poor oral absorption, concentrates in the intestine. It acts as immunomodulator, by reducing cytokine release from T cells, and antimicrobial agent. In patients refractory to conventional therapies (5-ASA, steroids); azathioprine, anti-tumor necrosis factor antibodies, or Vedolizumab are recommended. In severe refractory colitis, surgery can be curative, which can be avoided by using OV.

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What Happens When SARS-COV2 Infection Overlaps With Administration of Anti-TNF Therapy in an IBD Patient?

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