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Successful Long-Term Vancomycin Therapy in an Adult With Primary Sclerosing Cholangitis

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Abstract

Primary sclerosing cholangitis (PSC) is a chronic autoimmune liver disease characterized by progressive inflammation, fibrosis, and multifocal strictures of intrahepatic and extrahepatic bile ducts. PSC often accompanies inflammatory bowel disease and may progress to cirrhosis and the need for liver transplantation. Medical management is not consistently successful, and despite numerous reports of vancomycin therapy for PSC, no broad consensus for it, particularly in adults, has emerged. We present the case of an adult female PSC patient with sustained symptomatic and biochemical improvement while receiving oral vancomycin for more than a decade, during which a full-term pregnancy and elective cesarean delivery occurred. Emerging evidence suggests that immunomodulatory and microbiome-mediated effects of oral vancomycin contribute to sustained biochemical remission and disease stabilization in selected PSC patients. Unique in the duration of vancomycin treatment response with a successful pregnancy during therapy, this case adds to a growing body of evidence supporting oral vancomycin treatment for PSC patients.

Categories: Transplantation, Allergy/Immunology, Gastroenterology
Keywords: liver disease, oral vancomycin, primary sclerosing cholangitis, remission, ulcerative colitis

Introduction

Primary sclerosing cholangitis (PSC) is a well-defined, chronic autoimmune cholestatic liver disease characterized by progressive inflammation and fibrosis of the intrahepatic and extrahepatic bile ducts [1-4]. Over time, PSC leads to cirrhosis, portal hypertension, and even hepatic malignancy; it is strongly associated with inflammatory bowel disease (IBD), particularly ulcerative colitis. Wide-ranging symptoms include clinical features of IBD as well as fatigue, pruritus, abdominal pain, jaundice, and fever associated with intestinal or hepatic infection.

PSC diagnosis is often confirmed when magnetic resonance cholangiopancreatography (MRCP) shows multifocal strictures and segmental biliary duct dilation [5]. Laboratory findings are typically elevated enzymes including alkaline phosphatase, alanine aminotransferase (ALT), aspartate aminotransferase (AST), and gamma-glutamyl transferase; total and direct bilirubin may initially be normal but rise as PSC impairs bile flow. Liver biopsy is confirmatory when portal inflammation, fibrosis, and bile duct changes are evident. Patient care has typically been supportive, and no generally accepted medical treatment has demonstrated consistent long-term efficacy [3-4]. However, over the past three decades, oral vancomycin has been shown to reverse PSC and accompanying IBD in children and, more recently, in adults [6-10]. Yet because of safety concerns, scant evidence of long-term efficacy, and lack of placebo-controlled trials, no broad consensus to accept vancomycin PSC therapy has emerged. We report an adult PSC patient with severe symptoms and abnormal liver function who has had persistent PSC remission with over 12 years of oral vancomycin treatment. The patient, who signed an attestation and informed consent for publication, is co-author Elizabeth J. Peters, daughter of first author Thomas G. Peters. The study was deemed IRB exempt by Baptist Medical Center, Jacksonville, Florida. To avoid bias, clinical data were verified by source, date, and result prior to review in detail by each author.

Case Presentation

In December 2013, a 33-year-old woman was evaluated with endoscopic retrograde cholangiopancreatography (ERCP) for right upper quadrant pain and clinical jaundice. Findings included biopsy-confirmed acute hemorrhagic gastritis and primary common bile duct stones extracted by sphincterotomy. Laparoscopic cholecystectomy and cholangiography the following day showed free flow of contrast and no stones; pathology diagnosis was chronic cholecystitis. In March 2014, intermittent abdominal pain and persistently elevated liver enzyme and bilirubin levels prompted repeat ERCP disclosing irregular intrahepatic bile duct caliber with some pruning in the secondary and tertiary biliary radicals.

One month later, an ERCP at a tertiary medical center that treats all phases of PSC showed hepatic ductal irregularity. MRCP and magnetic resonance (MR) elastography demonstrated early PSC in sub-segmental

intrahepatic ducts. Calculated mean stiffness was 2.2 kPa, suggesting normal liver parenchyma. A liver biopsy performed in May 2014 revealed PSC; ulcerative colitis was diagnosed by mucosal abnormalities seen on colonoscopy and confirmed on biopsy. From January through June 2014, peak laboratory values were: AST 352 U/L; ALT 340 U/L; alkaline phosphatase 1044 U/L; total bilirubin 3.6 mg/dL; and direct bilirubin 2.3 mg/dL. Recommendations were ursodeoxycholic acid, 1000 mg at bedtime, and intermittent low-dose prednisone until that center listed the patient for a liver transplant.

Symptoms did not improve, and liver enzyme values fluctuated widely but were usually elevated; hepatic synthetic function was preserved. Under direction of personal community physicians, the patient started oral vancomycin, 500 mg three times daily in July 2014. Within five weeks, symptoms resolved. Elevated liver enzymes as well as total and direct bilirubin levels normalized by October 2014 (Figure *1*). Oral vancomycin and ursodeoxycholic acid were continued; prednisone was withdrawn. Table *1* depicts hepatic enzyme and bilirubin levels in the three months before and after oral vancomycin therapy was begun. Thereafter, serum bilirubin levels remained normal, while hepatic enzyme assays increased infrequently year-over-year from normal baseline to slightly elevated levels (Table *2*).

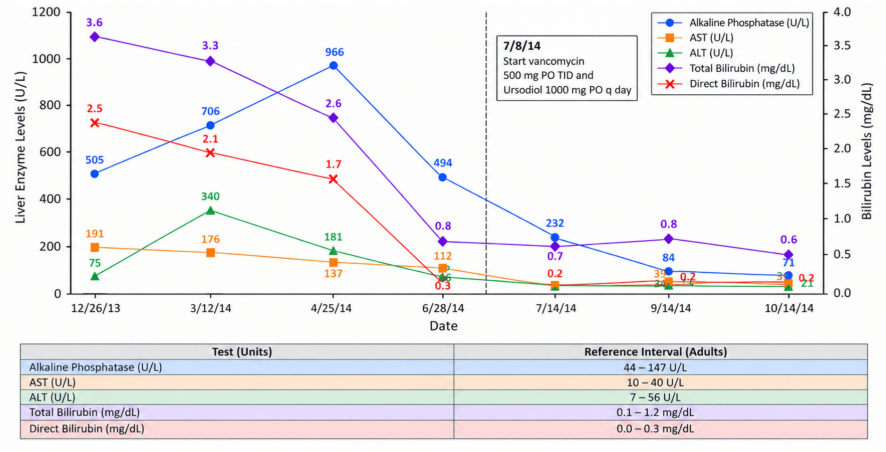


FIGURE 1: Liver enzyme and bilirubin trends before and after vancomycin initiation

Lab Assay	Before Vancomycin Average ± SD (Range)	After Vancomycin Average ± SD (Range)	Reference Value Interval
Alkaline phosphatase	750.9 ± 226.6 (386-1044)	125.5 ± 73.4 (71-232)	44-147 U/L
Aspartate aminotransferase	118.8 ± 85.8 (54-352)	31.8 ± 9.1 (23-41)	10-40 U/L
Alanine aminotransferase	133.2 ± 62.7 (56-253)	28.0 ± 4.8 (23-33)	7-56 U/L
Total bilirubin	1.43 ± 0.86 (0.5-3.1)	0.65 ± 0.13 (0.5-0.8)	0.1-1.2 mg/dL
Direct bilirubin	0.90 ± 0.75 (0.2-2.3)	0.20 ± 0.00 (0.2-0.2)	0.0-0.3 mg/dL

TABLE 1: Hepatic assays three months before and three months after vancomycin therapy

Year	Total Bilirubin, mg/dL (Ref: 0.1-1.2)	Direct Bilirubin, mg/dL (Ref: 0.0-0.3)	Alkaline Phosphatase, U/L (Ref: 44-147)	Aspartate Aminotransferase, U/L (Ref: 10-40)	Alanine Aminotransferase, U/L (Ref: 7-56)
2013	3.6	2.5	505	191	137
2014	3.1	2.3	1044	352	340
2015	1.0	0.2	202	34	40
2016	0.8	0.3	469	73	76
2017	1.0	0.3	216	94	90
2018	0.7	0.2	80	31	27
2019	0.9	-	75	30	27
2020	0.7	-	87	23	26
2021	0.7	-	84	28	23
2022	0.7	-	23	23	75
2023	0.6	-	91	22	21
2024	0.7	-	77	33	28
2025	0.4	-	89	22	19
2026	0.7	-	92	20	18

TABLE 2: Highest liver assay results by year 2013-2026.

Vancomycin started in July 2014. Direct bilirubin assay not obtained (2019-2026).

A positive pregnancy test in June 2015 raised issues in addition to the PSC and IBD. The last two of four prior pregnancies were associated with low birth weights and neonatal alloimmune thrombocytopenia. Therefore, at 20 weeks' gestation, treatment began with intravenous immunoglobulin (IVIG), 70 g weekly. Oral vancomycin was tapered to 250 mg twice daily. In February 2016, cesarean delivery of a normal full-term infant was followed by an uneventful recovery. Vancomycin therapy had been uninterrupted throughout the pregnancy, and except for the last month of gestation, liver enzyme assays were normal. Peak values at 36 weeks were as follows: AST 35 U/L, ALT 30 U/L, and alkaline phosphatase 469 U/L. Total bilirubin levels remained within 0.3 to 1.0 mg/dL during and after the pregnancy. The child, now aged 10, is healthy with no history of hematologic disorder.

Postpartum, symptoms of PSC and IBD were entirely absent. There were occasional elevated enzyme assays (Table *2*), and stool calprotectin levels were elevated in June 2016. Vancomycin dosing was increased from 250 mg to 500 mg twice daily, then thrice daily with no other medication changes. By December 2016, the AST, ALT, and alkaline phosphatase levels remained elevated, and the vancomycin dose was increased to 1000 mg three times daily (t.i.d.) followed by a three-week taper to 500 mg t.i.d. in early January 2017. Variable dosing of oral vancomycin with ursodeoxycholic acid, adjusted to 600 mg at bedtime, has continued since with no adverse effects. In February 2017, hepatic MR elastography disclosed no significant abnormalities, suspicious lesions, or evidence of liver fibrosis, with liver stiffness measurements of 2.5 kPa, which correlates with normal liver parenchyma. The PSC and ulcerative colitis are in persistent remission, and the patient continues full employment as a nurse along with duties of motherhood and routine community activities. She is asymptomatic, and laboratory measures remain normal on morning and evening doses of vancomycin 250 mg and 500 mg, respectively, plus ursodeoxycholic acid 600 mg at bedtime. Surveillance colonoscopies have shown normal mucosa and mild chronic active colitis on biopsy.

Discussion

The ideas that vancomycin may reverse signs, symptoms, and hepatic abnormalities of PSC were first published by Cox and Cox in 1998 [6]. They described three teenage PSC patients who were treated with vancomycin for complicated, severely symptomatic, and long-standing enteritis. In each case, PSC eased, and the authors speculated that vancomycin may "kill the enteric bacteria that produce ... biliary toxic inflammatory agents," thereby reducing gut-derived inflammatory injury to the biliary tree.

Building upon these observations, Davies and colleagues prospectively followed 14 children with PSC-IBD (mean age 12 ± 4 years, range 2-17 years) treated with oral vancomycin. Within one to two months, there was normalization of liver enzyme levels, improvement in erythrocyte sedimentation rates, and a reduction in serum autoantibody levels in 12 of the 14 patients [7]. Withdrawal of vancomycin was consistently followed by recurrence of clinical symptoms and worsening liver enzyme values, while re-institution of therapy resulted in renewed biochemical and clinical improvement. Four children with established cirrhosis demonstrated a less complete response than those with earlier-stage disease, suggesting that microbiome-directed therapy may be most effective before advanced hepatic fibrosis develops. Subsequent assessment of the same patient group disclosed previously unrecognized immunomodulatory effects of oral vancomycin, including enhanced regulatory T-cell activity, supporting the concept that its therapeutic benefit extends beyond antimicrobial activity alone [8]. In follow-up of these 14 patients, improved laboratory and imaging studies, symptom burden, and liver as well as intestinal biopsy findings pointed to vancomycin-mediated resolution of PSC and IBD.

Although these results were limited to pediatric patients, they established the plausibility that oral vancomycin therapy may be effective at all ages. A placebo-controlled trial of adults with PSC also demonstrated enzyme normalization and vancomycin-related relief of fatigue, pruritus, diarrhea, and anorexia [9]. In a multicenter vancomycin-treated PSC cohort of 59 children and adults followed for 0.2 to 14 (mean 2.7) years, over 80% experienced reduction in liver enzyme values; 95.3% reported symptomatic improvement of IBD-related diarrhea, and only one patient went on to liver transplantation eight years after initiating oral vancomycin therapy [10]. The worldwide Paediatric PSC Consortium reported 113 PSC-IBD children (mean age 12.7 years) who demonstrated vancomycin-associated clinical IBD remission [11]. Thus, there appears to be ample evidence that sustained oral vancomycin treatment is effective, safe, and well tolerated in children and adults with PSC and IBD. The inflammatory amelioration through immune dysregulation mutes the PSC and IBD responses, which result in the hallmarks of both diseases [12-14].

While there have been many vancomycin-treated PSC-IBD patients and calls for broader acceptance of this regimen, the general guidelines of PSC treatment still do not include vancomycin as first-line therapy [12]. The rationale for not issuing such a recommendation relates to few definitive trials and insufficient data on long-term results. Yet the American Association for the Study of Liver Diseases (AASLD) does recognize the utility of off-label vancomycin PSC therapy [13]. Hence, widely accepted PSC treatment guidelines, while acknowledging vancomycin efficacy, still affirm that evidence is not sufficient to recommend long-term treatment. A well-thought-out policy suggestion would treat PSC as a rare disease, allowing oral vancomycin therapy as an accepted adaptive treatment strategy, essentially bringing this widely applied treatment into the mainstream of PSC care [14].

Our case represents what is likely one of the longest, well-documented instances of successful PSC medical management using oral vancomycin. Notably, vancomycin treatment succeeded throughout a full-term, complex pregnancy managed with IVIG, a standard of care at that time [15]. The patient has had no side effects of treatment due, at least in part, to minimal systemic vancomycin absorption and its mode of action primarily within the gastrointestinal tract [16]. Other observational series and case reports affirm successful PSC care in patients with concurrent UC given vancomycin [17-18]. The sustained 13-year PSC remission experienced by our patient through a complex pregnancy and a very active adult life seems compelling and suggests that AASLD recommendations could be modified while still recognizing the long-term impacts of chronic vancomycin use. Although our single case cannot establish uniform efficacy, it meaningfully expands the long-term adult experience with microbiome-directed therapy and supports continued prospective investigation of oral vancomycin in adult patients with PSC.

Conclusions

Oral vancomycin therapy for PSC has been shown to avert microbiome-related causes of the disease through immunomodulation and other pathways. Such treatment in many cases leads to reversal and ultimately to clinical remission of progressive liver failure and its related IBD. Patients diagnosed with these disorders should be considered candidates for oral vancomycin therapy, as close monitoring and other supportive measures are considered.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Thomas G. Peters, Robbie Zeineddine, Kenneth Luberice, Elizabeth J. Peters, Michael A. Edwards

Acquisition, analysis, or interpretation of data: Thomas G. Peters, Robbie Zeineddine, Kenneth Luberice, Elizabeth J. Peters, Michael A. Edwards

Drafting of the manuscript: Thomas G. Peters, Robbie Zeineddine, Kenneth Luberice, Elizabeth J. Peters, Michael A. Edwards

Critical review of the manuscript for important intellectual content: Thomas G. Peters, Robbie Zeineddine, Kenneth Luberice, Elizabeth J. Peters, Michael A. Edwards

Supervision: Thomas G. Peters, Elizabeth J. Peters

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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