Ischemic Colitis Probably Induced by Refractory Constipation after Voglibose Administration in a Patient with Total Gastrectomy

A side effect of α-glucosidase inhibitor (α-GI), fermentation of unabsorbed carbohydrates by intestinal bacteria, induces flatulence, abdominal distention, and constipation which in rare cases leads to ileus (1, 2). We report here a case of ischemic colitis (IC) which was probably induced by refractory constipation after α-GI administration.

A 73-year-old woman was admitted to our hospital in late February 1999 because of abdominal pain and melena. She was diagnosed with type 2 diabetes in 1979 and was treated with glibenclamide for several years. She underwent total gastrectomy for gastric cancer in October 1987, and was maintained on insulin therapy from 1988 up to the time of her admission. Her postprandial hyperglycemia was sustained from the initiation of the insulin therapy, when her HbA_{1c} level was measured at 11.8%, and a supplementary regimen of α-GI voglibose (0.6 mg/day) was started in July 1998. After the start of voglibose administration, abdominal distention and constipation appeared and gradually progressed. From the middle of February 1999, her constipation grew progressively more severe. At admission, bowel sounds were normal, but there was tenderness in the left flank region. She had mild diabetic retinopathy and advanced diabetic neuropathy, and her glomerular filtration rate was 38.5 ml/min. Postprandial plasma glucose concentration was 24.8 mmol/l, and the HbA_{1c} level was 11.4%. Colonoscopy revealed intraluminal bleeding, mucosal edema, erythema, and longitudinal ulcerations in the descending colon. A diagnosis of IC was made, voglibose was ceased, and she was treated with intestinal rest and intravenous fluids. Endoscopic findings and abdominal distention and pain were improved on the 10th day.

Habu et al reported chronic constipation and previous abdominal surgery as two factors strongly associated with IC (3). They postulated that these factors may promote the development of IC by increasing the intraluminal pressure and interfering with the colonic blood supply. Longo et al reported that among 47 patients with IC, 11 patients (17%) had diabetes mellitus (4). Although the strong association between IC and diabetes mellitus remains unclear, hypercoagulability in the diabetic state, chronic constipation due to diabetic gastroenteropathy, and inadequate tissue perfusion due to diabetic microangiopathy have all been identified as possible mechanisms. Interestingly, the development of IC after α-GI administration in this case suggests that α-GI may increase the intraluminal pressure through excessive gas formation and constipation, thereby reducing the colonic blood supply. Therefore, caution should be exercised in administering α-GI to diabetic patients with previous abdominal surgery, relatively advanced age, and autonomic neuropathy.

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