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Hyperplastic Polyposis following Treatment of Gastric Vascular Ectasia: A Case Report and Review of Clinical Correlates

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Key Words
Hyperplastic polyps · Gastric vascular ectasia · Pathogenesis · Transplantation

Abstract
The etiology of gastric antral vascular ectasia (GAVE) and hyperplastic polyps (HP) is not fully understood, but there is no known overlap. We report a case of gastroduodenal HP arising in a patient treated for GAVE.

Case Report

A hospitalized 55-year-old male was evaluated for intermittent melena and transfusion dependent anemia 4 months post cardiac transplantation for ischemic cardiomyopathy. His comorbidities included graft failure requiring a biventricular assist device with anticoagulation, hemodialysis dependent renal failure, and cytomegalovirus viremia. His medications included cyclosporine, prednisone, warfarin, and pantoprazole. Liver disease was suggested by a nodular liver and mild splenomegaly on imaging, with intact synthetic function and negative serologic testing. On endoscopy GAVE was treated with gold probe cautery (GPC) (fig. 1). Duodenal biopsies taken for an appearance of duodenitis showed hemosiderin laden macrophages and were negative for cytomegalovirus. Colonoscopy was normal. Serial endoscopies for persistent symptoms over the following 12 months revealed antral arteriovenous malformations (AVM) treated with GPC, congestive gastropathy, but also evolving mucosal nodularity in the duodenum, and finally antral and duodenal polyps (fig. 2). The largest polyp (antral, 1.5 cm) was removed and confirmed as hyperplastic (fig. 3). The patient expired later due to unrelated infectious complications.

Discussion

Despite unclear etiologic overlap, this and previous cases [1, 2] may suggest a possible connection between GAVE and HP.

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In this case, conditions that may be associated with GAVE include possible cirrhosis, transplantation, cardiac and renal failure. Portal hypertension can be present in 31% of patients with GAVE, with diffuse antral rather than typical watermelon pattern [3]. It may be mediated by factors other than simple vascular congestion, such as altered antral pyloric motility [4, 5]. Bone marrow transplantation is associated with GAVE through unclear mechanisms [6], but there is no known association with solid organ transplantation. Congestive heart failure has been associated with congestive gastroduodenopathy and vascular ectasia [7], and the use of a ventricular assist device has been associated with AVM [8]. Vascular ectasia has also been associated with renal failure and hemodialysis [9].

HP account for more than 50% of gastric polyps, are considered benign, and may present with anemia or mechanical obstruction. Though their malignant potential is low, it is present, particularly with polyps >5 mm [10]. There is evidence supporting clonality and neoplastic potential of gastric HP [11]. The etiology of gastric HP is not known, but there is an association with atrophic gastritis, *Helicobacter pylori* infection, and elevated gastrin levels [12]. Gastric HP have been reported with proton pump inhibitor therapy [13]. Duodenal HP may represent hyperplasia of Brunner's glands. Gastroduodenal mucosal hyperplasia has been reported in patients with chronic renal failure [14] and hemodialysis [15], possibly due to a trophic effect of hypergastrinemia and hyperpepsinogenemia. HP have also been reported post solid organ transplantation [16], possibly in association with immunosuppression and CMV infection [17], which has been associated with foveolar gastric hyperplasia [18].

Vascular ectasia and HP can in this case be explained independently, with renal disease as the only potential common etiologic factor. Nevertheless HP have been reported following treatment of GAVE [1, 2], and gastric ulcers [19] without underlying renal disease. Based on the temporal relationship and current data, a multifactorial process may be at play. Trophic and genetic factors may result in hyperplastic changes, particularly in new mucosa at healing sites of treated GAVE. Such an association may have clinical relevance in therapeutic endoscopy in patients with chronic renal and liver disease and in transplant recipients.

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Fig. 1. **a** Gastric antrum with vascular ectasia prior to endoscopic treatment. **b** Duodenum with punctuate erythema and possible duodenitis on initial endoscopy.

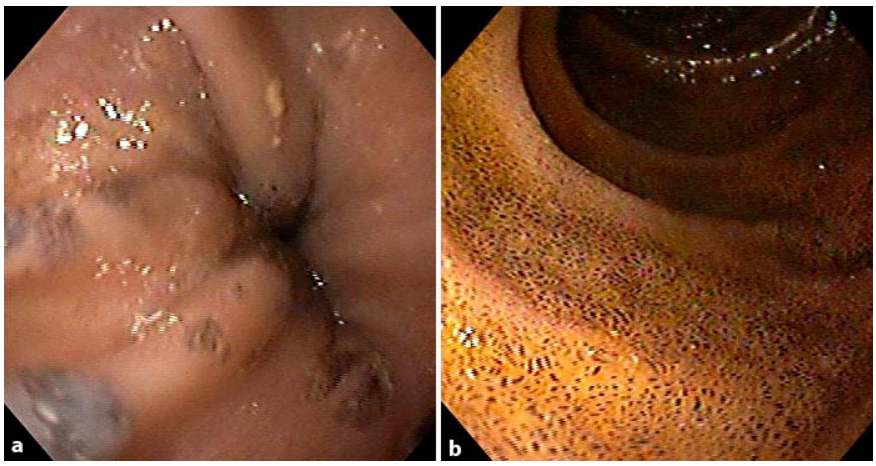

Fig. 2. **a** Gastric antral polyps 12 months after initial endoscopic treatment of GAVE. **b** Duodenal polyps 12 months after initial endoscopy.

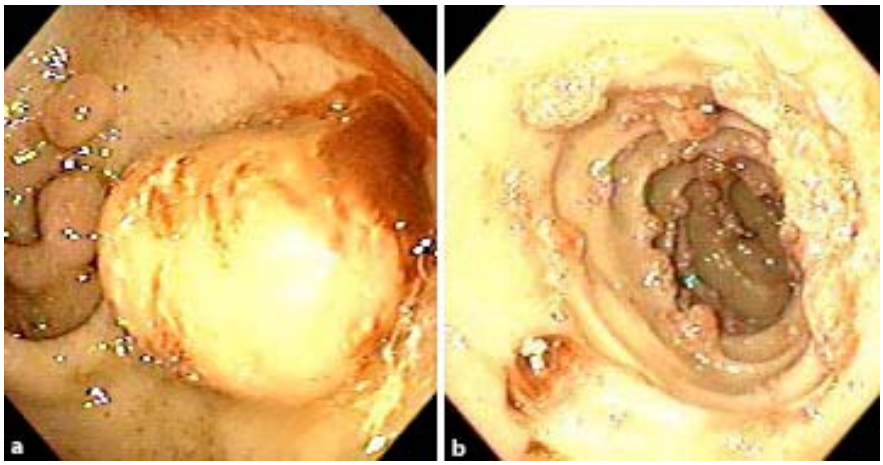

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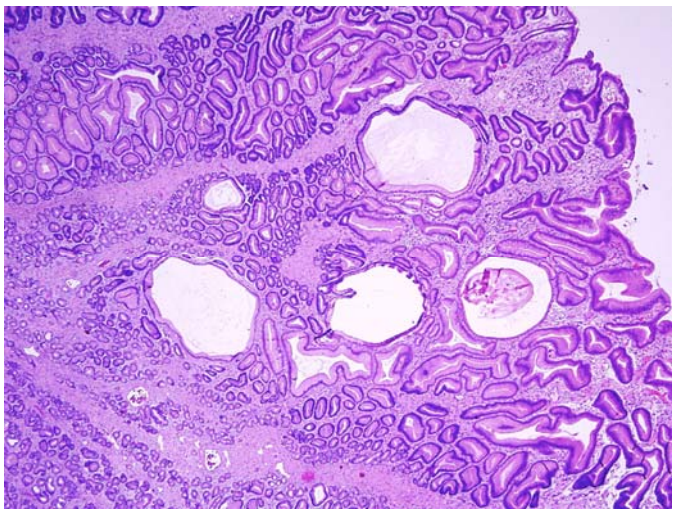
Fig. 3. Hyperplastic polyp with cystically dilated, branched foveolar epithelium and stromal edema. H&E stain, magnification ×40.



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