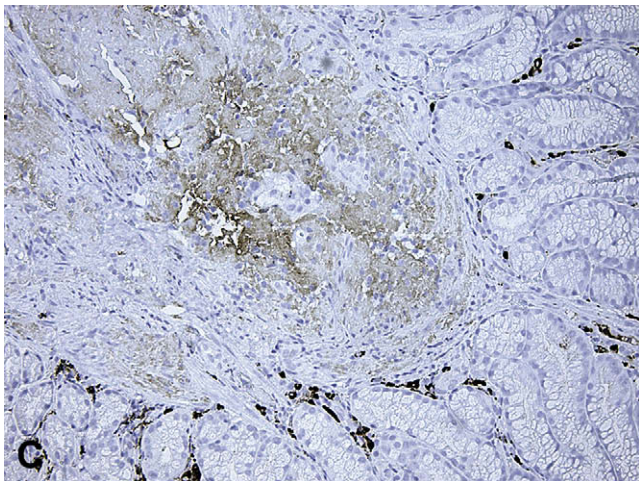
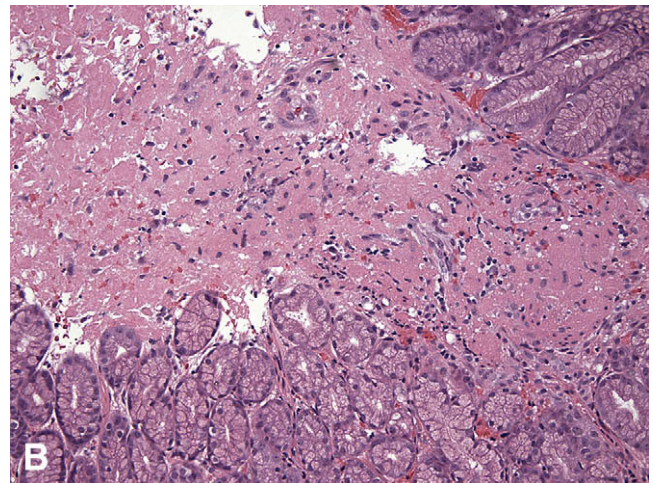
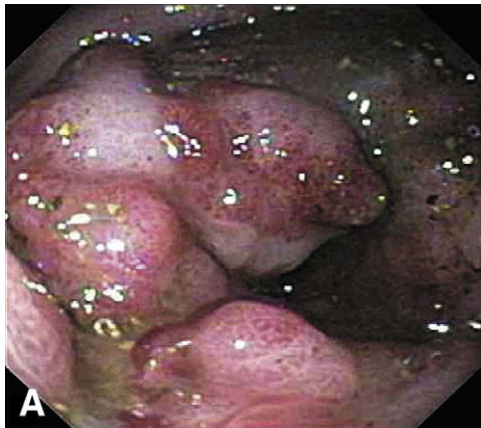


Commentary

First described by Dittrich in 1847, gastroduodenal fistulas are rare and are usually acquired as a complication of gastric ulcer disease: the ulcer penetrates into the duodenum, scarring occurs, and the ulcer tract epithelializes. Fortunately, these fistulas most often are clinically innocuous, although, in the presence of renal failure and with the continuous exposure to prednisone and NSAIDs, the previously benign nature of this patient's ulcer diathesis was seriously challenged. As for the observation that his double pylorus developed a third lumen, I am reminded of the French philosopher Henri Bergson who said, "life does not proceed by the association and addition of elements, but by dissociation and division." Here, we have an association blended with either division or addition; the association of ulcer diathesis with NSAIDs and pyloric lumina, which either were divided further by the scarring of kissing ulcers or added to by formation of yet another gastroduodenal fistula. Regardless of mechanism, if life is to proceed, get the patient off the injurious agents, if possible, and keep him on an acid-suppression regimen.

Lawrence J. Brandt, MD
Associate Editor for Focal Points

Regression of a localized gastric amyloid mass in a patient treated for multiple myeloma



An 82-year-old man with a 4-year history of multiple myeloma (κ light chains), neuropathy, and macroglossia was admitted with vomiting and hematemesis. EGD

revealed gastric outlet obstruction with a copious amount of retained coffee-ground-stained fluid. After aspirating the fluid, a large circumferential antral mass

(A), which was responsible for the obstruction, was identified. The endoscope was passed with difficulty into the duodenum, and the lesion was considered most likely to be a gastric carcinoma. Histology revealed amorphous pink material consistent with amyloid, confirmed by a Congo red stain (B, orig. mag. $\times 100$). An immunohistochemical stain for kappa light chain stained the amyloid deposits and highlighted a predominance of kappa light chain-expressing plasma cells (C, orig. mag. $\times 100$). Several days later, a repeat EGD failed to reveal carcinoma, and biopsy specimens showed chronic inflammation, granulation tissue, and amyloid. In situ hybridization demonstrated a mild predominance of kappa light chain messenger RNA-expressing plasma cells (kappa:lambda = 5:1) in the lamina propria. The patient could tolerate liquids and was discharged on Alkeran (GlaxoSmithKline, Research Triangle Park, NC), Decadron (Merk & Co, Whitehouse Station, NJ), zoledronic acid and lenalidomide (a thalidomide analogue with immunomodulatory,

anti-angiogenic, and anti-neoplastic properties). Three months later, the patient decided to stop all medications except lenalidomide. A repeated EGD performed 6 months after the initial endoscopy showed complete regression of the antral mass (D), and random biopsy specimens revealed no evidence of amyloid deposition.

DISCLOSURE

All authors disclosed no financial relationships relevant to this publication.

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doi:10.1016/j.gie.2008.10.050

Commentary

First described by Rokitansky in 1842, amyloidosis today is known to be a systemic disease caused by the extracellular deposition of complex insoluble proteinaceous material produced in a variety of diseases and involving one or multiple organs. GI disease occurs in perhaps 10% of cases, of which 1% has symptomatic gastric involvement. Gastric symptoms are nonspecific and include nausea, vomiting, abdominal pain, and GI bleeding, usually accompanied by weight loss. Regardless of the GI organ, involvement may be mainly vascular with ischemic consequences, mucosal with malabsorptive sequelae, or muscular with resulting dysmotility. Amyloid, along with syphilis, lupus, and cytomegalovirus infection is one of the great imitators, and, when it involves the stomach, may present as gastroparesis, linitis plastica, ulcer, tumor, or gastric outlet obstruction. The disease can only be diagnosed with a biopsy specimen with Congo red staining to demonstrate amyloid protein or immunostaining to reveal its fibrillar protein component. Unfortunately, there is no specific therapy for the disease, and median survival is only about a year. The take-home message here is to be aware of gastric amyloid as a possibility whenever you see what seems to be a familiar disease pattern, especially when a patient has chronic inflammatory disease, multiple myeloma, monoclonal gammopathy, or a polyneuropathy, or is on hemodialysis. Also, anytime a big tongue, especially when associated with temporomandibular arthritis, prevents the endoscope from being easily advanced into the posterior pharynx, think of Rokitansky.

Lawrence J. Brandt, MD
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Gallstones mimicking malignancy

A 41-year-old woman underwent a laparoscopic cholecystectomy in October 2000 for gallstone disease. At the time of the operation, the gallbladder was removed easily, but a few stones were dropped into the peritoneal cavity. She was discharged and told that these would not cause any major concern. Eight years later, the patient was referred for evaluation of diffuse abdominal pain. Basic laboratory test results were normal, but a contrast CT scan of the abdomen revealed nodular peritoneal lesions (A, B). The possibility of these being caused by malignancy was discussed with the patient. Results of tests for tumor markers (CA-125, CA 19-9, and alpha-fetoprotein) were nor-

mal. The patient underwent a diagnostic laparoscopy, which revealed nodules scattered over the parietal peritoneum with omental adhesions (C). The liver, stomach, small bowel, uterus, and ovaries looked normal. Further examination of the nodules revealed them to be encapsulated gallstones, embedded in the omentum and pelvis (D). All visible nodules were extracted laparoscopically. Histopathologic examination revealed multiple yellow, faceted stones up to 1 cm in diameter and attached to fibrocollagenous connective tissue that was lined in places with reactive mesothelium, patchy inflammation, and fibrin deposition (E). The patient has been asymptomatic since the operation.