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Commentary

With the prevalence of approximately 2%, Meckel's diverticulum is the most common congenital anomaly of the GI tract. The majority of cases remain asymptomatic; however, Meckel's diverticulum is associated with a slight increased risk of malignancy. The most common malignant tumor within Meckel's diverticulum is neuroendocrine tumor (NET), followed by adenocarcinoma, GI stromal tumor, sarcoma, and lymphoma. It has been hypothesized that the incidence of Meckel's diverticulum-associated malignancy increases with age. This phenomenon has been seen proportionally higher in male patients. The most common symptoms of NETs within the Meckel's diverticulum include abdominal pain, diarrhea, weight loss, intermittent GI bleeding, and bowel obstruction, although the majority of cases are asymptomatic and are detected incidentally. NETs within a Meckel's diverticulum are associated with a high rate (24% to 75%) of metastasis (nodal and liver metastasis) at the time of diagnosis. This aggressive tumor behavior is more similar to small-bowel NETs as opposed to appendiceal NETs. Owing to a higher tendency for early metastasis in smal-bowel NETs, lymphadenectomy in addition to bowel resection is recommended. Late-onset metastasis after only bowel resection in NETs within Meckel's diverticulum has been reported. In cases without lymphadenectomy, perhaps close interval imaging surveillance should be considered. Small NETs, such as in this case, could be easily missed, and lifesaving treatment could be delayed inadvertently. This case is a great example of "dig deeper" in those small-bowel capsule endoscopy images!

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Severe cytomegalovirus gastritis due to pembrolizumab-induced agranulocytosis



A 60-year-old man with non-small-cell lung cancer received immunotherapy with triweekly pembrolizumab and experienced immune-related colitis after the first administration. Although the colitis improved after pembrolizumab was discontinued, immune-related agranulocytosis developed 4 months after initial treatment and manifested as epigastric pain. EGD showed irregularly shaped esophageal ulcers (A) and a large gastric ulcer with extensive disappearance of mucosa at the greater curvature of the middle third of the stomach (B). At the gastric angle and antrum, normal mucosa with a pseudopolyposislike appearance remained (C). Steroid treatment improved the immune-related agranulocytosis but not the ulcers. Cytomegalovirus gastritis was diagnosed by antigenemia assay, immunohistochemistry staining, and polymerase chain reaction test with the use of gastric tissue. He received antiviral therapy with ganciclovir, and 2 months later the esophageal ulcer and gastric ulcer had remarkably improved,

although scarred and deformed (\mathbf{D}) , and food intake was possible.

Cytomegalovirus gastritis with extensive mucosal shedding is rare. We believe the patient's serious immunosuppressed state resulting from pembrolizumab-induced agranulocytosis caused severe cytomegalovirus gastritis. Although immunerelated gastritis was also a differential diagnosis, the coexistence of esophageal ulcers and antiviral treatment course supported the diagnosis of cytomegalovirus gastritis in this case.

DISCLOSURE

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Commentary

In this case, the authors describe a severe case of gastritis and gastric ulceration secondary to cytomegalovirus (CMV) infection in a patient with solid organ malignancy treated with immune checkpoint inhibitors (ICIs). CMV co-infection is an exceedingly rare occurrence in patients treated with ICIs, with a low cumulative incidence rate of 0.3%. The mechanism by which CMV is exacerbated in the setting of therapy with ICIs has remained unclear at large, but potentially it is related to blocking of anti-inflammatory pathways. Blocking the PD-1/PD-L1 pathway may provoke latent CMV infection by enhancing CMV-specific T cell activity. CMV infection in patients treated with ICIs has been reported more commonly in the setting of steroids-refractory disease or after treatment with immunosuppressive agents. Pseudopolyposis or pseudotumor appearance as the result of CMV has been reported in immunosuppressed patients with HIV. This phenomenon is uncommon in patients treated with ICIs, as described in this case. This case highlights the importance of keeping a broad differential diagnosis in cases of steroid-refractory or immunotherapy-refractory ICI-related gastritis and colitis and also relapsed disease. Physicians should be vigilant about the diagnosis of CMV and proceed with extensive biopsy with dedicated CMV staining in high-risk patients. Early detection and treatment of CMV in these patients could perhaps be lifesaving and could limit the morbidity associated with CMV gastroenterocolitis.

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Heartburn from a neo-acid pocket formed by a laparoscopic gastric band





A 63-year-old woman with a laparoscopic gastric band presented for acid reflux and dysphagia. EGD revealed a gastroesophageal junction 34 cm from the incisors and an extrinsic impression of the laparoscopic band (**A**) at 37 cm. A barium esophagram showed evidence of a "pseudo hiatal hernia" (**B**, *yellow bracket*) between the lower esophageal sphincter (**B**, *red arrow*) and the gastric band (**B**, blue arrow). High-resolution manometry (**C**) showed a pattern of esophagogastric junction outflow obstruction (integrated relaxation pressure 34.5 mm Hg upright, 26.5 mm Hg su-

pine), likely an artifact from the laparoscopic band. There was also separation of the lower esophageal sphincter from a pressure zone, again resulting from the laparoscopic band, consistent with a "pseudo hiatal hernia." The patient subsequently underwent Bravo pH testing without proton pump inhibitor therapy, which revealed an acid exposure time of 27.1% (normal <4% to 6%), with the longest reflux episode lasting 177 minutes (**D**, *arrow*). The findings were suggestive of a "pocket" formed between the lower esophageal sphincter and the gastric band, where gastric acid can