Neither residual adenocarcinoma nor lymph node metastasis was identified.

DISCLOSURE

All authors disclosed no financial relationships relevant to this publication.

Commentary

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I'm not sure what prompted this patient's endoscopy, but this is yet another case for the serendipitous EGD log book and a reminder of the perils of risky behavior. Although EBV is a highly prevalent, orally transmitted human herpes virus with well-established oncogenic potential, its role in gastric cancer would make a great trivia question. EBVaGC is thought to account for 10% of all nonendemic gastric carcinoma worldwide, and its tendency to remain confined to the epithelium renders its prognosis favorable. The endoscopic pearl here is its classic superficially depressed and ulcerated appearance and predominance in the proximal regions of the stomach (in contrast, for example, to the distal prevalence of *Helicobacter pylori*-associated gastric cancer). Although ESD is a competitive modality to formal resection in superficialappearing carcinoma, the presence of ulceration belies a more invasive process that, in my experience, frequently trumps an ultrasonographic appearance of a more superficial process. I should point out the hypoechoic EUS image seen here is a textbook example of submucosal invasion, and the lack of adjacent adenopathy suggests ESD as a reasonable next step.

A characteristic histologic feature of EBVaGC is its dense lymphoid infiltration. How EBV causes cancer is not fully understood, but the infection does interfere with cell cycle checkpoints that regulate apoptosis and cellular proliferation. This chronic inflammation–cancer paradigm is seen in a host of other GI malignancies, including those associated with Barrett's esophagus, *H pylori*, chronic viral hepatitis, and inflammatory bowel disease.

> David Robbins, MD, MSc Assistant Editor for Focal Points

Perivascular epithelioid cell tumor: an unusual pancreatic mass diagnosed by EUS-FNA

A 38-year-old woman was referred because of 2 months of epigastric pain. The results of physical examination and laboratory investigations were unremarkable. CT of the abdomen demonstrated a hypervascular pancreatic uncinate lesion. EUS confirmed an 18-mm, wellcircumscribed, hypoechoic, homogenous mass without vascular invasion (**A**). FNA cytology revealed scattered epithelioid and spindled-shaped cells, and periodic acid–Schiff (PAS) staining gave positive confirmation of a glycogen-rich cytoplasm (**B**, **C**, **D**, **E**). Immunocytochemistry was positive for melanoma-associated antigen (HMB-45) and smooth muscle actin (SMA) (**B**, **C**, **D**, **E**) but was negative for cytokeratin (CK), synaptophysin (SYN), chromogranin (CG), and S-100. The staining pattern was consistent with a perivascular epithelioid cell tumor (PEComa), which was confirmed upon pancreaticoduodenectomy (**F**, **G**, **H**).

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Commentary

Perivascular epithelioid cell tumors are poorly understood mesenchymal neoplasms whose cytologic signature is composed of distinctive perivascular epithelioid cells. I love any moniker that ends in "-oid" (or "-oma") because it's just



like saying "I have no idea who you are but you sure look familiar!" That kind of vague opener is on rare occasion a good one, but more often than not it just generates a blank stare.

The curious hallmark cell, with its clear acidophilic cytoplasm, has been identified in a hodgepodge of obscure entities such as angiomyolipomas, pulmonary clear cell sugar tumors (there's nothing sweet about them), and lymphangioleiomyomatosis. In 1996, Zamboni (no relation to the storied ice rink resurfacer) described the first pancreatic variant and coined the term "PEComa." Most of the hundred or so cases have originated in the uterus and behave in a benign fashion. These "-oids" are exceptionally rogue—they have no known normal cellular counterpart, and a precursor lesion has not been elucidated. PEComas make carcinoids look like the girl next door.

Further muddying the waters are the few reports of malignant PEComas, and distant metastases may present years after resection of the primary tumor. It is this potential that no doubt prompted radical surgery for the young woman described in this Focal Point. There has been an attempt to better characterize the potential for bad behavior, including a size of > 8.0 cm, a mitotic count of > 1 per 50 high-power fields, and the presence of necrosis, but the rarity of the dis-

ease has prevented the development of formal pathologic and behavioral grading criteria. Pancreatic PEComas are exceptionally rare, and this is the first (and probably last) report of a pancreatic PEComa diagnosed by EUS-FNA you are likely to encounter.

David Robbins, MD, MSc Assistant Editor for Focal Points

Non-lifting sign from cold biopsy of sessile serrated polyp



A 71-year-old woman presented for surveillance colonoscopy. Screening colonoscopy 3 months previously, at our institution by another endoscopist, had shown multiple small polyps, the largest of which was a 1-cm sessile serrated polyp. In addition, an area of thickened mucosa with ill-defined margins was identified in the ascending colon, which, by a single, standard cold forceps biopsy, was revealed to be a sessile serrated adenoma (SSA).

Surveillance colonoscopy demonstrated melanosis coli and a 2-cm \times 1-cm flat, pale polyp draped across a fold in the ascending colon with a central area of scarring (**A**). Injection-assisted polypectomy by use of a combination of methylene blue and normal saline solution failed to lift the polyp in the region of mucosal scarring (**B**), and en bloc removal of the polyp could not be performed (**C**). The residual polyp tissue adjacent to the scar was ablated with argon plasma coagulation (APC). Histopathologic examination confirmed SSA, and surveillance colonoscopy was recommended to be done in 3 months.

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Commentary

Classification of polyps continues to evolve as greater insight is brought to bear by new technologies. We now appreciate, for example, that the benign hyperplastic polyp of years ago is actually part of a larger family of lesions now expanded to include the traditional serrated adenoma and the sessile serrated adenoma (SSA) or polyp; serrated adenomas—especially