Mimicking Gastric Natural Killer/T-Cell Lymphoma

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Question: A 71-year-old woman with hypertension and dyslipidemia was referred to our hospital because of a high level of carbohydrate antigen (CA) 19-9 of 85 U/mL. She was asymptomatic and had no findings on physical assessment or blood tests, except a high CA19-9 level. Computed tomography scan and colonoscopy showed no evidence of malignancy. Esophagogastroduodenal endoscopy found atrophic gastritis and reddish, flat, elevated lesions with erosion on the anterior wall of the lower gastric body (Figure A). This lesion was approximately 1 cm in diameter. Biopsy samples were obtained from this lesion. Pathologic examination revealed intermediate- to large-sized cells with irregular nuclei infiltrating the lamina propria. The cells had abundant clear or slightly eosinophilic cytoplasm. Immunochemical staining revealed CD3+; CD5−; CD20−; CD56+; and cytokeratin−. Epstein–Barr virus-encoded small RNA (EBER) in situ hybridization was negative (Figure B; original magnification, 400×; inset, 1000×). Polymerase chain reaction for T-cell receptor γ gene rearrangement was negative. F-18 fluorodeoxyglucose positron emission tomography showed no other lesions.

What is the most likely diagnosis?

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Conflicts of interest
The authors disclose no conflicts.

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This patient was diagnosed with lymphomatoid gastropathy, which can be initially misdiagnosed as extranodal natural killer (NK)/T-cell lymphoma, nasal type. However, no patients have progressive disease and spontaneous regression is observed without any treatment in most cases. Lymphomatoid gastropathy is also referred to as NK cell enteropathy. In this case, 6 months later, a repeat endoscopic examination found that this lesion had spontaneously regressed (Figure C). Although another new lesion was detected in the fornix after 6 months, this new lesion also regressed within a few months. After that, we performed endoscopic examinations every 6 months. The patient has shown no disease progression in 4 years.

Several characteristic features of lymphomatoid gastropathy are distinct from those of extranodal NK/T-cell lymphoma, nasal type. The clinical course of lymphomatoid gastropathy is not aggressive. The differential pathologic findings are: (1) EBER in situ hybridization is consistently negative (in contrast, EBER is positive in almost all cases of extranodal NK/T-cell lymphoma, nasal type), (2) eosinophilic cytoplasmic granules seen in atypical cells, and (3) angiodestructive (angiocentric) growth patterns or prominent apoptotic bodies lymphoma are not observed. Although this case was suspected to be NK/T-cell lymphoma from the result of immunostaining, EBER negativity and eosinophilic cytoplasmic granules led to the correct diagnosis.

The endoscopic characteristics of lymphomatoid gastropathy are unclear, but most of the lesions are <1 cm in diameter, and may variously include erosive, ulcerative, or elevated lesions. Almost all lesions characteristically have strong redness. Some cases have multiple lesions and others are single. This case was consistent with the appearance of lymphomatoid gastropathy.

Recognition of endoscopic and pathologic characteristics of this entity is critically important for endoscopists to prevent misdiagnosis and misinterpretation as an aggressive lymphoma, such as NK/T-cell lymphoma.

References