INTRODUCTION

Neuroendocrine tumors (NETs) are a heterogeneous group of rare cancers that derive from neuroendocrine cells and show distinct functional and biological behavior depending on the location, tumor size, and clinical symptoms (1).

NETs are classified histologically based on tumor differentiation. In general, neuroendocrine neoplasms are divided into well-differentiated and poorly differentiated categories. According to the World Health Organization (WHO) 2010 classification, there are three types of NETs: well-differentiated grade 1 (<2 mitoses / 10 high-power fields (hpf) AND <3% Ki67 index), intermediate-differentiated grade 2 (2-20 mitoses / 10 hpf OR 3%-20% Ki67 index) and poorly differentiated grade 3 neuroendocrine cancer (>20 mitoses / 10 hpf OR >20% Ki67 index) (2).

Carcinoid tumors are the most common NETs. Three types of gastric carcinoid tumors are described. Type I: associated with chronic atrophic gastritis, Type II: associated with Zollinger-Ellison syndrome, and Type III sporadic lesions. Gastric carcinoids associated with hypergastrinemia are relatively benign, and endoscopic resection is a feasible treatment modality for lesions that are 2 cm or smaller (3).

CASE REPORT

A 37-year-old man with complaints of epigastric pain and weight loss was admitted to the hospital. Upper gastrointestinal endoscopy revealed two subepithelial lesions (10 mm and 5 mm) at the greater curvature of the stomach (Figure 1). Narrow band imaging (NBI) method was used to determine borders of the lesion more precisely (Figure 2). Gastric atrophy was more marked in the body-fundus than in the antrum. Biopsy specimens from both lesions showed microlobular-trabecular cell clusters with no cellular polymorphism. No ex-
tragastric hormonal syndromes such as flushes or diarrhea were identified. Elevated serum gastrin level (2138 pg/ml) and atrophic gastritis suggested Type I NET. Endoscopic ultrasound showed a hypoechoic, homogeneous, 1 cm lesion originating from the submucosal layer with an intact muscularis propria layer. Cap-assisted endoscopic mucosal resection (EMR), an "inject, suck and cut" technique, was performed (Figures 3, 4). The mucosal defect was closed with 2 hemoclys. Pathologic examination of both resected specimens showed a histological architecture of microlobular-trabecular cell clusters in the mucosal layer with marked fundic gland atrophy. Endocrine cell micronests were observed in the mucosal layer, which was compatible with NET (Figure 5). Neither lymphatic nor vascular invasion was documented. Lesions were chromogranin A-positive and synaptophysin-positive (Figure 6). Ki-67 index was <2%. All resection margins were negative for neuroendocrine cells. Neither cellular polymorphism nor mitosis was observed; thus, the tumor was graded as G1. The patient recovered uneventfully; there was no lesion on the follow-up endoscopy three months later.

**DISCUSSION**

Standard treatment for NETs is surgical resection. Endoscopic treatment is an accepted treatment modality for small (≤ 20 mm) submucosal lesions. It has been proven that the small lesion limited in the submucosa with intact muscularis propria has a minimal risk for metastatic disease (4). Conventional snare excision, cap-assisted mucosectomy, submucosal dissection, and unroofing techniques may be preferred depending on the type and size of the lesion (5,6). Cap-assisted endoscopic resection technique is a valid, safe, effective treatment, and it allows total excision of the lesion; therefore, full pathologic assessment is possible to determine the malignant potential.

**REFERENCES**