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LETTERS TO THE EDITOR
EDİTÖRE MEKTUP
A patient with gastric carcinoid tumor: Treatment and surveillance options

Gastrik karsinoid tümörlü bir hasta: Tedavi ve takip seçenekleri

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To the Editor,

Neoplastic differentiation of histamine secreting enterochromaffin-like (ECL) cells leads to carcinoid tumors which belong to the neuroendocrine tumor family. Foregut carcinoids comprise 20% of all carcinoid tumors and include thymic and lung carcinoids in addition to gastric and duodenal carcinoids (1). Within the GI tract, most carcinoid tumors occur in the small bowel, appendix and rectum; however, the frequency of gastric carcinoid tumors has increased markedly due to endoscopic screening. Gastric carcinoids account for 0.3% of gastric neoplasms (2). Type I (ECLoma) is associated with type A chronic atrophic gastritis, achlorhydria, and often pernicious anemia, and is considered to be the most benign. Type II carcinoids are associated with the Zollinger-Ellison syndrome in combination with multiple endocrine neoplasia type 1 (MEN 1). They are usually multiple and more malignant than type 1. Type III carcinoids are known as "sporadic" and not associated with hypergastrinemia and high malignant potential (3).

A 25-year-old female patient was admitted to our clinic with complaints of fatigue, weakness and irritability which had lasted for several years. She had already been advised to have an operation because of goiter. She was found to have megaloblastic anemia, but refused an endoscopic examination. Later, her symptoms subsided after taking vitamin B_{12} treatment. Six months after treatment, she accepted the endoscopic procedure. She had no history of diarrhea, palpitation, facial flushing, or weight loss and was not a vegetarian. There was no specific feature in any of her family members. Physical examination was normal. Laboratory findings and clinical course revealed in historical order the following: *One and half years previously:* Hct 18%, Hb 6.6 g/dl, MCV 106 fl, serum AST 56 U/L, LDH 3024 U/L, and vitamin B_{12} 120 pg/ml, and thyroid hormone levels were compatible with subclinic hypothyroidism. Anti-parietal cell antibody (APCA) was positive. Peripheral blood smear and bone marrow aspirates showed megaloblastic changes. Based on clinical diagnosis of pernicious anemia, 1000 µg of cyanocobalamin monthly injection was prescribed, and hemoglobin level was normalized. *One year previously:* There was no laboratory finding of anemia. Serum fasting gastrin level was 580 pg/ml (N:25-125). Gastroscopic examination also showed a polypoid lesion with a diameter of 0.8 cm on the lesser curvature of the proximal side of corpus. Endoscopic biopsy of the lesion revealed hyperplastic polyp. Thereafter, we carried out endoscopic polypectomy and the specimen evaluated histopathologically was compatible with well-differentiated carcinoid tumor (Figure 1a). The specimen showed positive staining for chromogranin-A (CgA) (Figure 1b) and synaptophysin by immunohistochemistry. We repeated the endoscopic surveillance and polypectomy sessions four times until there was no longer any tumor residue. Abdominal ultrasonography (USG),

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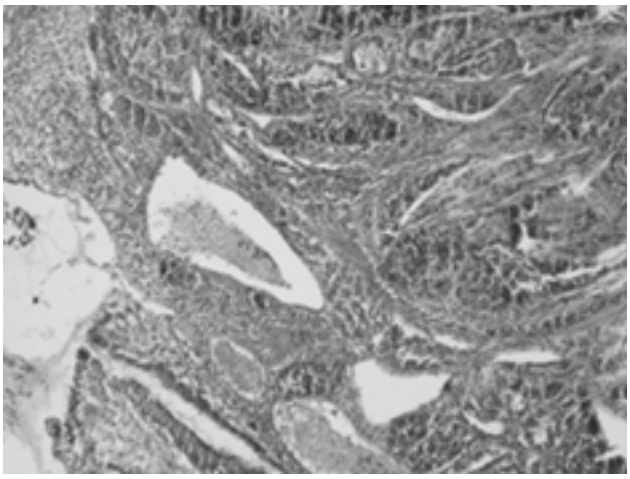

Figure 1: a) Carcinoid tumor (HE -x100)

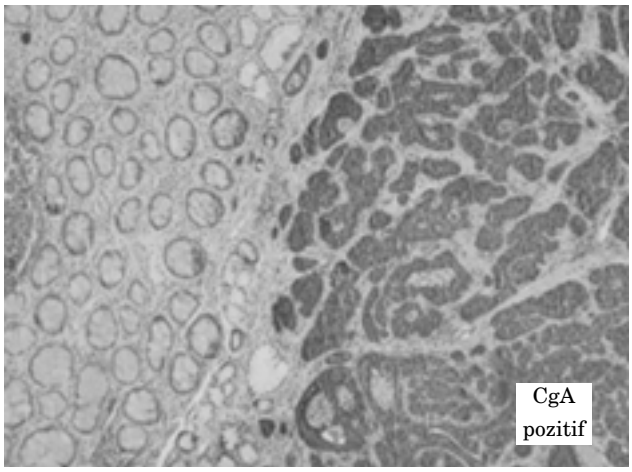

Figure 1: b) Tumor cells positive for Chromogranine A (x100)

computerized tomography (CT) and endosonography were all normal. We added thyroid hormone replacement to cyanocobalamin treatment. *Now:* Complete blood count parameters, serum thyroid hormones, thyrotropin, calcium, parathyroid hormone, calcitonin, insulin, corticotropin, prolactin, and growth hormone levels were all normal. APCA was positive but anti-intrinsic factor antibody negative. Serum CgA (Pasteur Cerba Lab) level was 610 µg/L (19.4-98.1). Serum β-hCG and urinary 5-HIAA levels in 24 hours were normal. We performed a deep endoscopic mucosal resection to the irregular polypectomy area and encountered the carcinoid tumor again only in one area, and some ECL cell hyperplasia. One month after this procedure, we obtained 20 biopsies from the same region and nearby. There was no tumor or *Helicobacter pylori,* but atrophy and inflammation were present.

Our patient was diagnosed as type I gastric carcinoid tumor. Superficial biopsy was defective for diagnosis. Polypectomy should always be kept in mind in all polypoid lesions in the stomach in both diagnosis and treatment. In a recent study, it was pointed out that plasma CgA was higher in those with type III than in those with type I carcinoids (4). Serum CgA level was still very high after the last endoscopic mucosal resection in our patient, and this condition may require a more frequent endoscopic or endosonographic follow-up program. In light of some studies (5), we also agree on endoscopic follow-up and endoscopic mucosal resection whenever it is necessary, as well as offering antrectomy option in the most radical situation. A result which warrants the likelihood of indication for total gastrectomy seems a remote probability for this patient. The carcinoid syndrome shortens life expectancy, as encountered in 4% of patients with carcinoid tumor (6). Finally, although the most effective treatment of type I gastric carcinoids is under discussion, limited surgery with endoscopic polypectomy and/or antrectomy, aimed at reducing the high serum gastrin, or observation are the preferred methods of treatment and should have been taken as proposal. On the other hand, all gastric polypoid lesions -especially in patients with pernicious anemia- should be removed for a correct diagnosis and also for treatment, because of probable neoplastic transformation potential.

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