Neuroendocrine/squamous gastric collision tumor: A rare entity

Esophagogastric junction cancer is the 4th most common cancer in humans. Gastric adenocarcinomas are the most common histopathological type of gastric tumors, with the others constituting 5% of tumors (1,2). Combined tumors can be synchronous, composite, or collision tumors. Collision tumors are non-intermingling contiguous tumors of two or more different histopathological types. Here we report a case of neuroendocrine/squamous collision tumor.

A 45-year-old man, with a history of subtotal gastrectomy for peptic ulcer twenty years ago, presented with upper gastrointestinal hemorrhage. Gastroscopy revealed a fragile mass surrounding the gastric cardia with normal appearance of the esophagus and the gastroenterostomy anastomosis. Endoscopic biopsy revealed squamous cell carcinoma (SCC). Thoracoabdominal computed tomography did not reveal any metastasis. Total gastrectomy, omentectomy, lymph node dissection with en bloc liver resection were performed.

Macroscopically, a stage-IIIA submucosal SCC (grade II, T4) extending from the esophagus to the gastric fundus was observed. A stage-IIA neuroendocrine carcinoma (grade IV, T2) was also observed to arise from the distal end of the squamous component (Figure 1). Surgical borders and the 19 dissected lymph nodes were tumor free. Octreotide scintigraphy revealed no metastases. Because high-grade neuroendocrine tumor has a poor prognosis and determines survival, we administered cisplatin+etoposide combination chemotherapy along with radiotherapy.

Gastric neuroendocrine neoplasms (NEN) constitute <1% of gastric tumors (2). Collision tumors are tumors that arise adjacent to each other. They originate from different cells but are not mixed at the cellular level. Most NECs are composite tumors with intermingling of different cell types (3).

The individual tumors comprising a collision tumor have different risk factors. Smoking may be the cause of the SCC in our patient. It was reported that enterochromaffin-like cell hyperplasia and subsequent gastric carcinoid development are caused by acid suppression due to long-term proton pump inhibitor (PPI) therapy (4). Because carcinogenesis is a long-term process, the effects of PPIs on carcinogenesis remain unclear (4). Although our patient had undergone gastric resection, he did not have a history of long-term PPI therapy.

The limitation of this report is our inability to perform a tumor-suppressor gene mutation analysis, which may offer an explanation for the co-occurrence of the two different tumor types. However, our case is noteworthy because neuroendocrine/squamous collision tumors have not been reported in the literature.

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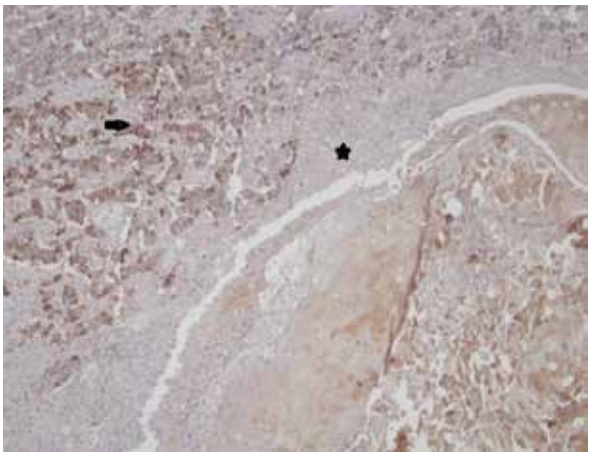

Figure 1. The neuroendocrine (arrow head), and the squamous (star) sides of tumor (chromogranine staining, X4)

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