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Synchronous Gastric Adenocarcinoma and Pancreatic Ductal Adenocarcinoma: A Case Report

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1. Abstract

Pancreatic ductal adenocarcinoma is the most common type of pancreatic cancer, often diagnosed at advanced stages with a very poor prognosis. While the synchronous occurrence of pancreatic and gastric cancer is rare, we present a case of an elderly male who underwent investigation for a 3-kilogram weight loss in a month and was incidentally found to have synchronous tumors of the pancreas and stomach. The patient underwent subtotal gastrectomy, distal pancreatectomy, and splenectomy. The pathology reports revealed adenocarcinoma in both the gastric and pancreatic tumors, with different immunohistochemical staining results. The patient received adjuvant chemotherapy of gemcitabine and TS-1(tegafur/gimeracil/oteracil) after the surgery. Through this case and a review of the literature, we discuss the diagnosis and differential diagnosis of double cancer of the stomach and pancreas, emphasizing the rarity of the synchronous occurrence of these tumors.

2. Introduction

As the prognosis for cancer patients continues to improve, the incidence of second or multiple primary tumors is on the rise [1]. Recently, there has been an increasing number of reported cases of synchronous cancers. Gastric and pancreatic cancers are respectively the second and fifth most common cancers of the gastrointestinal tract [2]. However, synchronous occurrence of gastric and pancreatic ductal adenocarcinoma is rare, with limited cases reported. In this article, we present a case of synchronous gastric and pancreatic ductal adenocarcinoma and review the literature on synchronous gastric and pancreatic cancers.

3. Case Report

We report a case of a 70-year-old male who presented with a 3-kilogram weight loss in one month and an elevated CA 19-9 level of 147.0U/ml. The patient denied nausea, vomiting, poor appetite, abdominal bloating, abdominal pain, change in bowel habits, yellowish skin or tea-colored urine recently. Notably, two of the patient's brothers had a past history of pancreatic cancer.

Abdominal computed tomography (CT) revealed an ill-defined hypodense lesion measuring 20x19x15mm in the pancreatic body that abutted the lesser curvature of the gastric body and pancreatic duct dilatation (Figure 1). The esophagogastroduodenoscopy (EGD) revealed circumferential wall thickening with nodularity change and lumen narrowing of the distal antrum. Endoscopic biopsy of the prepylorus revealed well-differentiated adenocarcinoma. Subsequent whole body positron emission tomography (PET) scan revealed focal increased FDG uptake in the pancreatic body corresponding to the hypodense lesion seen in the CT images. No evidence of abnormally increased FDG uptake was found elsewhere in the body.

The laparoscopic surgery was converted to a laparotomy because of adhesion of the pancreatic tumor to the stomach, which may have indicated tumor invasion. The adhesion between the pancreatic tu-

mor and the stomach was divided carefully, and subsequently, the patient underwent subtotal gastrectomy, distal pancreatectomy, and splenectomy (Figure 2).

The resected stomach lesion was 7.0x5.0x1.5 cm and was located in the low body and antrum with infiltrating type tumor configuration, classified as Borrmann's type IV. The pathology of the resected stomach specimen revealed a well to poorly differentiated adenocarcinoma (pathological stage IIIA, pT4aN2M0) that had invaded the serosa and perigastric tissue. Perineural and lymphovascular invasion were observed, and six regional nodes were positive with cancer cells in all forty-three harvested nodes. The resection margin was free of tumor cells, and there was no tumor cell infiltration into the omentum.

The resected pancreas lesion was 2.5x1.5x1.3 cm and was located in the body of the pancreas. The pathology was consistent with moderate differentiated pancreatic ductal adenocarcinoma (pathological stage IB, pT2N0M0). There was no peri-pancreatic tumor invasion, and no lymphovascular or perineural invasion was observed. Regional nodes were negative in all six harvested nodes, and the resection margin was free of tumor cells.

The immunohistochemistry results showed positive staining for S100 calcium-binding protein P (S100P), caudal-related homeobox transcription factor 2 (CDX-2), and carcinoembryonic antigen (CEA) in both the gastric and pancreatic tumors. The insulin-like growth factor 2 mRNA-binding protein 3 (IMP-3) staining was positive for the gastric tumor but negative for the pancreatic tumor (Figure 3 and 4). Adjuvant chemotherapy with gemcitabine and TS-1 was initiated after the surgery.

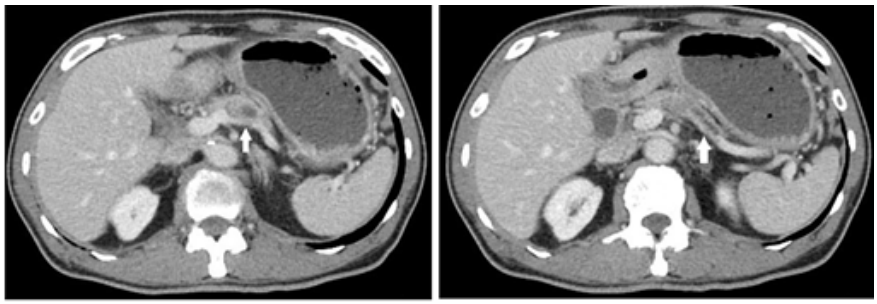

Figure 1: Contrast-enhanced abdominal CT (cross-section image) A: Ill-defined hypodense lesion in the pancreatic body (arrow); B: Dilatation of the pancreatic duct (arrow).

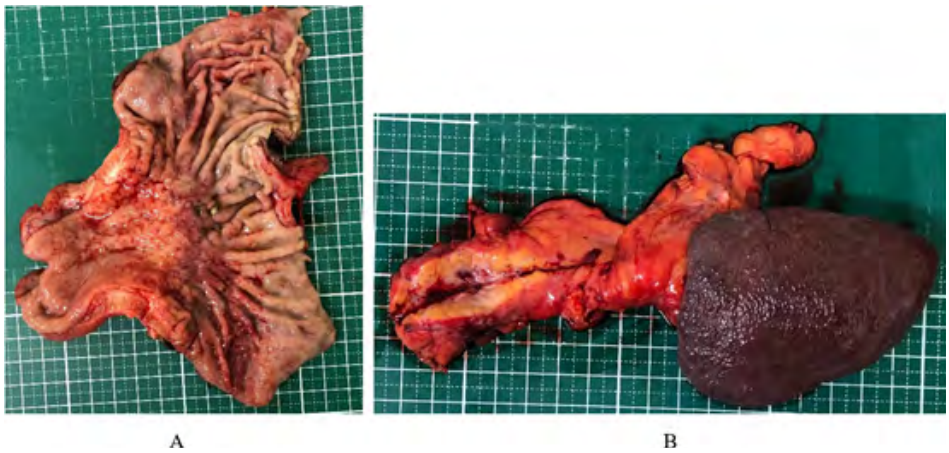

Figure 2: Resection specimen. A: resection specimen of gastric tumor; B: Resection specimen of pancreatic tumor and the spleen.

Table 1: Immunochemical stain result of the specimens of our case

IHC markers	Stomach	Pancreas
S100-P	positive	positive
CDX-2	focal positive	positive
CEA	focal positive	strong positive
IMP-3	positive	negative

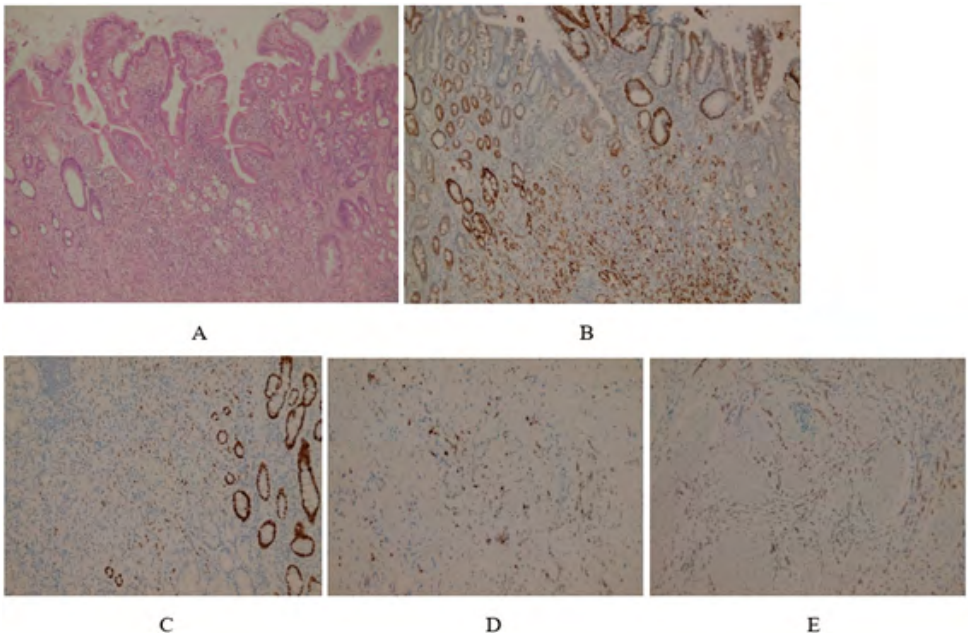

Figure 3: Microscopic examinations of stomach. A: Routine histology, stained using hematoxylin-eosin, shows gastric adenocarcinoma; B: Immunohistochemical staining of gastric tumor cells is positive for S100P; C: IHC staining of gastric tumor cells is focal positive for CDX-2; D: IHC staining of gastric tumor cells is focal positive for CEA; E: IHC staining of gastric tumor cells is positive for IMP-3.

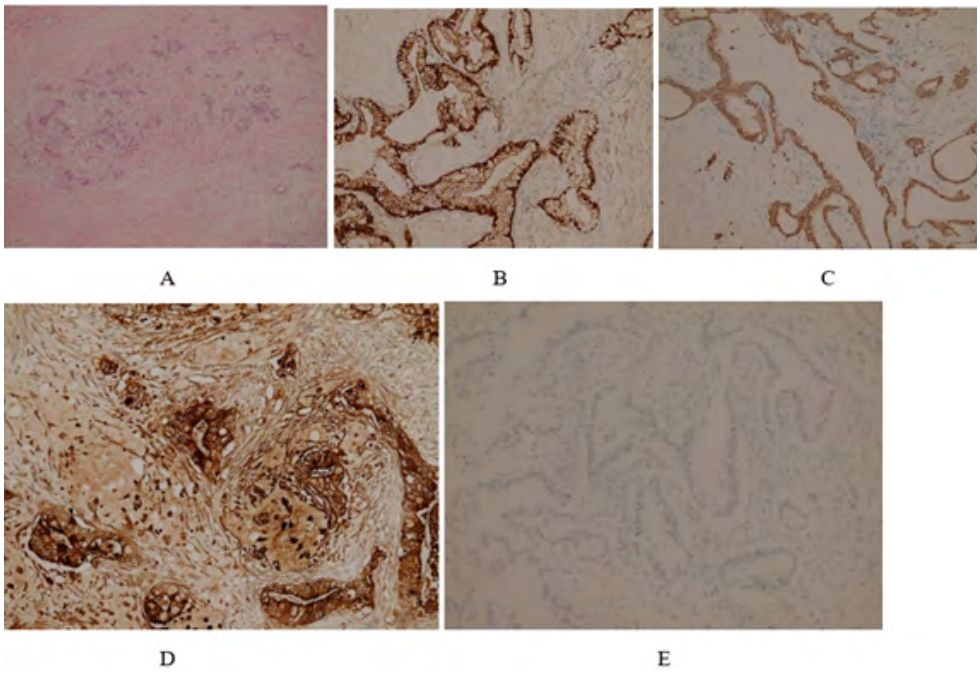

Figure 4: Microscopic examinations of pancreas A: Routine histology, stained using hematoxylin-eosin, shows pancreatic ductal adenocarcinoma; B: IHC staining of pancreatic tumor cells is positive for S100P; C: IHC staining of pancreatic tumor cells is positive for CDX-2; D: IHC staining of pancreatic tumor cells is strong positive for CEA; E: IHC staining of pancreatic tumor cells is totally negative for IMP-3.

4. Discussion

The incidence of second or multiple primary tumors has risen because of improved cancer patient survival rates. An increasing number of cases have been reported recently. A previous literature review showed that the prevalence of multiple primary cancers varied from 0.734% to 11.7% in cancer patients [3]. Among gastric cancer patients, the prevalence of second tumors ranges from 2.8% to 6.8% [4]. The incidence of gastric carcinoma associated with pancreatic carcinoma accounts for 3.8% of all cases of gastric carcinoma associated with carcinoma of other organs [1]. The frequency of pancreatic cancer with cancer of other organs ranges from 7.3% to 16.7% [1]. However, the association between gastric cancer and pancreatic cancer is uncommon. A previous literature review found that the most common primary tumors of pancreatic metastases were carcinomas of the lung, gastrointestinal tract, lymphoma, and kidney [5]. On the other hand, for gastric metastases were carcinomas of

the breast, lung, and melanoma [6]. Pancreatic metastasis of gastric cancer is extremely rare, with only 11 cases reported [7]. Similarly, gastric metastasis of pancreatic cancer is also very rare, with only 7 cases reported before [8].

To confirm the synchronous double cancer, it is important to review the patient's medical history to exclude the possibility of metastasis from a prior cancer. Additionally, imaging studies such as CT, magnetic resonance imaging (MRI), or endoscopic ultrasound (EUS) can be used to evaluate the second lesion and exclude the possibility of metastasis or local extension. Furthermore, endoscopic biopsy of the gastric lesion and EUS-guided fine needle biopsy (EUS-FNB) of the pancreatic lesion can aid in establishing a pre-operative diagnosis with the assistance of immunohistochemistry.

EUS-FNB of the pancreas is challenging because of its deep location and requires experienced endosonographers. Furthermore, there is a risk of needle tract seeding from the transgastric site, as reported in previous studies [9]. A literature review of 15 cases of needle tract seeding following EUS-FNB showed that 12 cases were caused by diagnostic EUS-FNB of pancreatic tumors located in the body and tail. However, no reports mentioned needle tract seeding after EUS-FNB for pancreatic head tumors, possibly because the transgastric puncture site of the needle tract was resected within the pancreaticoduodenectomy specimen [10].

Ductal adenocarcinoma is known to originate from the pancreatic duct, leading to the early phase dilatation of the pancreatic duct. On the other hand, pancreatic metastasis grows within the parenchyma and gradually expands, requiring more time to involve the pancreatic duct [7]. Among the 8 reported cases of solitary pancreatic metastasis of gastric cancer with recorded pancreatic duct conditions, none showed pancreatic duct dilatation [7]. Therefore, pancreatic duct dilatation may be a helpful clue in differentiating pancreatic metastasis from ductal adenocarcinoma.

In our case, histopathology of the two resected specimens revealed adenocarcinoma. We selected markers of S100P, CDX-2, CEA, and IMP-3 to distinguish the two adenocarcinomas. CDX-2 is positive in over 70% of diffuse-type gastric adenocarcinomas, while pancreatic ductal adenocarcinomas are positive for CEA [11]. S100P is commonly used to diagnose pancreatic adenocarcinoma in fine needle aspiration (FNA) specimens. Previous studies have shown that S100P and IMP-3 are immunohistochemical markers used to confirm the diagnosis of pancreatic ductal adenocarcinoma in both FNA and surgical specimens [12]. Although there are no guidelines for differentiating gastric and pancreatic adenocarcinomas from metastatic lesions, we used these immunohistochemical markers and found that both tumors were positive for S100P, CDX-2, and CEA, but had different results for IMP-3 (Table 1). Therefore, we concluded that the two cancers were not metastatic but synchronous primary tumors.

The limitation of our article is that we did not perform an EUS-FNB for preoperative diagnosis because of no experienced endosonographers available in our hospital. Instead, we arranged a whole-body PET scan, which revealed only a focal increased FDG uptake in the pancreatic body.

In conclusion, we employed immunochemical markers of S100P, CDX-2, CEA, and IMP-3 to distinguish the two adenocarcinomas. However, more research is needed to confirm the feasibility of these markers for accurately distinguishing between gastric and pancreatic adenocarcinoma and metastases. Furthermore, adequate preoperative evaluation for concomitant cancers, such as CT, MRI, and EUS-FNB, is crucial. Additionally, investigating the medical history of previous cancer is essential for accurate diagnosis. Preoperative evaluation of pancreatic duct dilatation may be helpful in distinguishing pancreatic metastasis from ductal adenocarcinoma.

5. Declaration of Patient Consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her names and initials will not be published and due efforts will be made to conceal her identity, but anonymity cannot be guaranteed.

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