Total Pancreatectomy for Metastatic Renal Cell Carcinoma with Marked Extension into the Main Pancreatic Duct

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Abstract

A 59-year-old man who had undergone left nephrectomy for renal cell carcinoma (RCC) 14 years previously was admitted for the treatment of obstructive jaundice. Imaging studies showed head-to-tail dilation of the main pancreatic duct (MPD) and a few ring-shaped enhanced nodules. Main duct-type intraductal papillary mucinous neoplasm was suspected and total pancreatectomy was performed. Pathologically, the entire length of MPD was filled with tumor. It consisted mainly of necrotic material, but included some clear cell carcinoma; the final diagnosis was metastatic RCC of the pancreas. This is an extremely rare case of pancreatic metastasis from RCC, with marked extension into MPD.

Key words: renal cell carcinoma, pancreatic metastasis, main pancreatic duct invasion

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Introduction

Metastatic carcinoma of the pancreas is uncommon—it accounts for 2% to 5% of pancreatic malignancies (1). Renal cell carcinoma (RCC), in addition to lung, colon, and breast carcinomas, is known to metastasize to the pancreas (2). Pancreatic metastases from RCC often cause non-destructive changes, such as pancreatic duct stenosis or compression. However, in the current case, metastatic lesions extensively invaded the main pancreatic duct (MPD), making it difficult to distinguish these lesions from an intraductal papillary mucinous neoplasm (IPMN). Herein, we describe an extremely rare case of pancreatic metastasis from RCC, with marked extension into MPD.

Case Report

A 59-year-old man was admitted to our hospital for the evaluation and treatment of obstructive jaundice, which had been noted on blood-chemistry analysis. Fourteen years previously, he had undergone a left nephrectomy for the treatment of RCC.

Early-phase computed tomography (CT) revealed dilation of MPD (Fig. 1a, b). Although MPD itself was not enhanced, tumor enhancement was observed along its wall. A few nodules in the body and tail of the pancreas showed ring-shaped enhancement (Fig. 1c, d). Tumor enhancement was prominent in the portal phase and persisted into the late phase. Fat-suppressed T1-weighted abdominal magnetic resonance imaging (MRI) showed isointensity from MPD to the pancreatic parenchyma; as a result, MPD could not be identified. On T2-weighted images, MPD was more hypointense than the gallbladder, suggesting that MPD was filled with necrotic material or a viscous substance. On magnetic resonance cholangiopancreatography (MRCP), MPD had a patchy appearance (Fig. 2). Neither dilation of the duodenal papillary orifice nor mucus secretion was observed on duodenoscopy, but the duodenal papilla was brownish, and a protruding mass covered by the hooding fold was observed.

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Figure 1. Contrast-enhanced abdominal CT. On axial and coronal abdominal CT scans during the early arterial phase, the main pancreatic duct was dilated from the pancreatic head to tail (arrows) (a, b), and nodules displaying ring-shaped contrast enhancement were observed (arrows) (c, d).

Figure 2. On MRCP, MPD appeared patchy (arrows).

Endoscopic retrograde cholangiography (ERC) revealed bile duct compression at the level of the duodenal papilla. Endoscopic retrograde pancreatography (ERP) revealed dilation of MPD, which was hypertranslucent from the pancreatic head to the body and irregular in the tail; nodular filling defects were also observed (Fig. 3c). Brush cytology during ERP revealed class IIIb atypia. Pancreatoscropy revealed whitish nodular elevations that filled the ductal lumen, but no salmon roe-like elevation, as is typically seen in main duct-type IPMN (MD-IPMN) (Fig. 3d). Thus, although some findings were inconsistent with MD-IPMN, they were also not consistent with a diagnosis of intraductal tubular tumor. In addition, despite the patient’s history of RCC, the tumor differed markedly from pancreatic metastasis of RCC, which is typically an encapsulated, well-demarcated, hypervascular tumor. The presence of these conflicting findings made it difficult to include other conditions in the differential diagnosis. Therefore, we suspected MD-IPMN with mural nodule in which the lesion was present throughout MPD, from head-to-tail, and in a branch duct of the tail; according to the MPD wall enhancement. Thus, a total pancreatectomy (TP) was thought to be necessary as a curative resection, and hence it was performed. Figure 4a shows a postoperative pancreatogram.

Pathologically, the gross inspection revealed tumor nodules that extended into the luminal duct throughout the entire length of MPD (Fig. 4b, c arrow) but not replacing the epithelium duct. The tumor connected to the MPD wall in only 1/4 to 1/5 circles (Fig. 5a), which contained nodular tumor components with extensive necrosis (Fig. 5b arrowhead). The viable carcinoma cells could be seen in the MPD wall (Fig. 5c) and branch wall (arrowhead) and also the adjacent parenchymal contained similar tumor nodules connected with the tumor part of MPD (Fig. 5d). In the surrounding pancreas, fibrosclerosis and the disappearance of parenchymal components were marked. In the head of the
Figure 3. Duodenoscopy revealed brownish papilla and a protruding mass (a). Pre-cutting exposed the mass, which was continuous with the pancreatic duct (b). ERC showed bile duct at the duodenal papilla was compressed (arrow), and revealed a dilated MPD that was hypertranslucent from the pancreatic head to the body and irregular from the pancreatic body to the tail, with nodular filling defects (c). Pancreatoscropy revealed whitish nodular elevations filling the ductal lumen (d).

Discussion

The pancreas is a rare site of metastasis—it accounts for fewer than 5% of pancreatic tumors removed in a large series (1, 3, 4). In an autopsy series, the frequency of metastases to the pancreas was 1.3% to 1.9% (5). In a review of 109 patients with metastatic RCC of the pancreas, Thomson and Heffess (6) reported that the patients’ mean age was 62.2 years, the mean interval between nephrectomy and metastasis manifestation was 8.4 years, the metastases were solitary in 71% of cases, and metastases to the pancreatic head accounted for 41% of cases.

Metastatic RCC of the pancreas is often encapsulated, well demarcated on imaging studies, and its hypervascularity is demonstrated by strong enhancement on contrast-enhanced CT. In an analysis of 15 cases of metastatic RCC of the pancreas, contrast-enhanced CT and MRI revealed that tumors smaller than 15 to 20 mm were homogeneously enhanced, whereas larger lesions displayed rim enhancement associated with decreased blood flow and necrosis in the tumor (7). Similarly, in the present case, metastatic nodules measuring approximately 15 mm in the pancreas displayed rim enhancement on contrast-enhanced CT. However, because these imaging features are also observed in pancreatic endocrine tumors, and because cell density and the degree of growth of small blood vessels varies in individuals, it is difficult to differentiate these tumors on the basis of contrast enhancement alone. The differential diagnosis for metastatic RCC also includes serous cyst adenoma and clear cell carcinoma (6). On ERP investigation of metastatic RCC of the
Figure 4. Postoperative pancreatogram (a). The MPD was dilated and irregular, nodular filling defects were also observed. The tumor extended into and filled MPD (arrow) (b, c). The surrounding pancreatic parenchyma contained nodular tumor component (arrowhead) (b).

Figure 5. Pathologically, the tumor extended into the luminal MPD connecting to the MPD wall in only 1/4 to 1/5 circles (a). Most of all in MPD mass were necrosis (b). The clear cell carcinoma cells were seen in the MPD wall (c) and in the branches duct (arrowheads). Adjacent parenchymal contained nodules connected with the part of MPD tumor (d).
In the head of the pancreas, clear cell carcinoma progression was noted in branches duct of the remaining pancreas as well (a, b). The tumor tissue obtained during nephrectomy also displayed features of clear cell carcinoma (c).

This is a schema according to the pathological and imaging findings.

pancreas, Tada et al observed MPD disruption, stenosis, and compression in 81% of cases (8). Because metastatic RCC of the pancreas typically displays expansive growth, it frequently leads to nondestructive changes. However, we believe that the present case is the first report of metastatic RCC that extended within and obstructed the MPD. Our search of the literature revealed only 2 other cases of pancreatic RCC metastases that extended into MPD (9, 10). In our case, pathologically a few nodular tumors lacking pancreatic duct wall components were observed in the parenchyma of the tail, and these corresponded to the ring-shaped tumor enhancements that were observed in the tail on CT image. These components partly connected with MPD tumor, thus it was considered that the parenchymal tumors invaded MPD this part. The tumor spread throughout MPD mostly in an extension form rather than an invasion form. Moreover, the tumor component in MPD was mostly necrotic; tumor cells were observed only near the site of connection between the tumor and the mucosa of the MPD. Thus, we speculated that RCC was metastasized hematogenously to the pancreatic tail parenchyma at first, then invaded MPD, and extended in the luminal duct space of MPD toward the duodenum and pancreatic tail without replacing the MPD epithelium. Hence the tumor component in MPD caused lack of tumor blood flow; eventually tumor necrosis occurred. This is why most of MPD did not display enhancement on contrast CT except for the MPD wall. This condition may mimic MD-IPMN findings. We misdiagnosed these findings as MD-IPMN with patchy mural nodule in MPD wall from the head to tail. Thus, we performed TP. However, retrospectively, considering that: 1) intense tumor enhancement was observed along MPD wall on enhanced CT, 2) that MPD was much more hypointense than would be the case for conventional IPMN on T2 MRI, 3) that the pancreatoscopic features were different from those of IPMN, and 4) that the patient had a history of surgery for RCC, we should have included metastatic RCC of the pancreas in the differential diagnosis.

When it is possible, aggressive pancreatic resection for...
metastatic RCC is believed to improve the prognosis (3, 4, 11-13): the 5-year survival rates of patients undergo ing pancreatic resection for solitary or multiple metastases are 75% and 64%, respectively (11). Based on these findings, we believe that although the preoperative diagnosis was incorrect, total pancreatectomy was nevertheless indicated in this patient. At the time of this writing—20 months after total pancreatectomy—the patient has shown no evidence of recurrence in other organs. Because RCC metastasis occurred as long as 32.7 years after nephrectomy (11, 14), long-term follow-up will be required.

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References