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Case Report

Synchronous pancreatic adenocarcinoma and intrahepatic cholangiocarcinoma arising in the context of intraductal papillary neoplasms

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ABSTRACT

Field cancerization theory provides rationale for the development of multiple pancreatic ductal and biliary lesions in a single patient through the development and accumulation of multiple genetic changes. Genetic alterations result in the development of precursor lesions including intraductal papillary mucinous neoplasms of the pancreas (IPMN), intraductal papillary neoplasm of the bile duct (IPN-B), and their malignant counterparts, pancreatic adenocarcinoma and cholangiocarcinoma. Although IPMN are frequently encountered, IPN-B are uncommon and the synchronous occurrence of both lesions is rare. We present a case of synchronous pancreatic adenocarcinoma and intrahepatic cholangiocarcinoma with histopathologic evidence of underlying precursor lesions, IPMN-P and IPN-B.

1. Clinical and laboratory assessment

A 70-year-old male presented to a gastroenterologist with a 1-month history of gas discomfort in the mid-abdominal region, flatulence, and eructation. Past medical history was significant for a recent diagnosis of type-2 diabetes. Past surgical history was positive for partial thyroidectomy more than 20 years prior. Social history revealed the patient to be a former smoker with a 50-pack-year smoking history. Physical examination was unremarkable. A borderline low hemoglobin level (13.5 g/dl) was identified. The remainder of the complete blood count and basic metabolic panel were normal. Notably, the following laboratory values were elevated (reference range): carcinoembryonic antigen, 3.5 ng/ml (0–3); chromogranin A, 183 ng/ml (0–95); C-reactive protein, 29.4 mg/l (0–5); erythrocyte sedimentation rate, 40 mm/h (0–15); gamma GT, 180 U/l (10–54); and CA, 19–9622.8 U/ml (0–35).

2. Imaging assessment

An abdominal ultrasound performed at an outside institution was significant for a hypoechoic mass in region of pancreatic head measuring 3.7 cm × 3.1 cm × 3.0 cm. A hypoechoic mass within the right lobe of the liver measuring 4.5 cm × 4.9 cm × 4.8 cm was also seen. The lesions in the pancreas and liver were concerning for malignancy and the patient was referred for further characterization with cross-sectional imaging.

The MRI examination was performed on a 1.5-T Siemens Avanto (Siemens Healthcare, Malvern, PA, USA) using a torso phased array coil. The imaging protocol included coronal and axial HASTE, GRE T1-weighted in and out of phase imaging, and precontrast and postcontrast enhanced T1 WI using axial breath-hold 3D T1 fat-suppressed spoiled gradient recalled echo sequence (VIBE). Postcontrast was performed following the dynamic intravenous injection of 20 ml gadolinium-based contrast agent using 4 time points: late arterial, portal venous, equilibrium, and late venous phases (5 min following contrast injection). Gadoversetamide 20 ml was administered (Optimark, Mallinkrodt Pharmaceuticals, Dublin, Ireland). Fluoroscopic bolus tracking technique was used.

A 2.3 cm × 2.8 cm T2 hyperintense, T1 hypointense mass was identified in the head of the pancreas. There was distention of the pancreatic duct in the body and tail of pancreas, measuring up to 6 mm. The common bile duct was not dilated. The remaining pancreas was atrophic. The pancreatic mass was noted to abut the portal vein at its origin and less than 180° of abutment of the superior mesenteric vein. There was no evidence of portal vein, superior mesenteric vein, or splenic vein thrombosis. The fat plane surrounding the SMA was preserved. The pancreatic mass demonstrated hypovascular enhancement following contrast administration, with mild peripheral enhancement noted on late venous phase images (Fig. 1).

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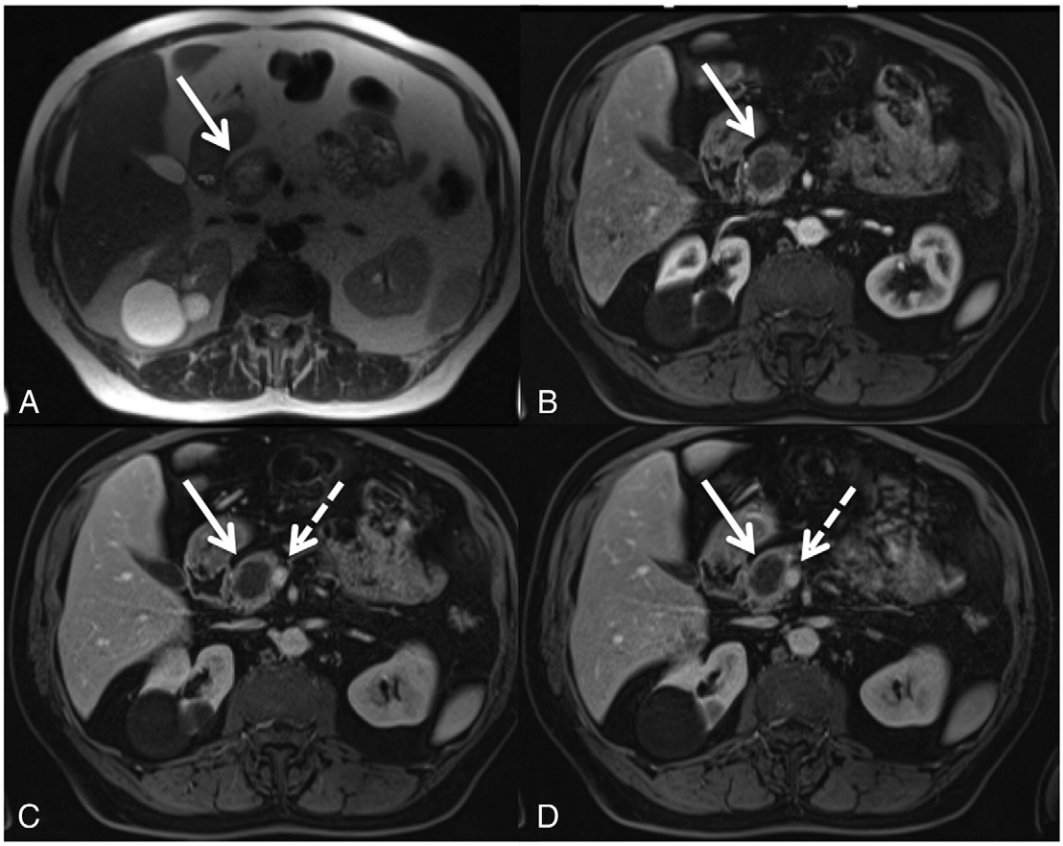

Fig. 1. Axial MRI images from axial HASTE (A) and dynamic breath-hold VIBE images with fat saturation were obtained following the administration of intravenous contrast (20 ml gadoversetamide). The 2.8-cm pancreatic head mass (solid arrow) demonstrates T2 hyperintensity (A) and hypovascular enhancement on arterial (B) and portal venous phase (C) with minimal peripheral enhancement on late venous phase images (D). The mass demonstrates less than 180° of abutment of the patent superior mesenteric vein (dashed arrow). The superior mesenteric artery is patient and uninvolved.

A 5.0 cm × 4.2 cm T2 hyperintense, T1 hypointense mass with lobulated margins was identified in segment 6 of the liver (Fig. 2). There was mild biliary distention in segments 6 and 7 peripheral to the mass. No fat or hemorrhage was detected within the lesion. No capsular retraction was present. No satellite lesion was identified. The right posterior portal vein branch abutted the mass, but without evidence for invasion or tumor thrombus. The lesion demonstrated mild continuous peripheral enhancement on arterial phase images with progressive rim enhancement on equilibrium and late venous phase images. Enhancing peripancreatic lymph nodes measuring up to 11 mm were identified.

3. Pathologic assessment

Three weeks after the MRI examination, the patient underwent an upper endoscopy that confirmed the presence of a mass in the head of the pancreas. Fine-needle aspiration of this mass revealed adenocarcinoma. The patient also underwent CT-guided biopsy of the right hepatic mass that revealed poorly differentiated malignant neoplasm. Tumor cells were arranged in large groups and often showed marked nuclear pleomorphism and occasional tumor giant cells. Prominent cherry-red nucleoli were seen; the cytoplasm was amphophilic and dense. Degenerative changes and tumor necrosis were present. The uninvolved liver showed chronic portal inflammation, periportal fibrosis, and changes reflecting the vicinity of a space-occupying lesion. Immunostains were positive for CK7, CEA, p63, and CDX2 and negative for CK20, CD56, chromogranin, and S100. The immunohistochemical patterns combined with the morphological features were consistent with carcinoma with both glandular and squamous features.

Approximately 3 weeks following the EUS on CT-guided liver biopsy, the patient underwent successful pylorus—preserving total pancreatectomy, splenectomy, and resection of liver segments 6 and 7. Pathology showed a poorly differentiated pancreatic adenocarcinoma that extensively involved the pancreatic head, body, and tail (Fig. 3A). The main pancreatic duct also showed extensive carcinoma in situ with micropapillary features (Fig. 3B). Three of nine lymph nodes were positive for metastatic carcinoma. Pathology of the resected liver tumor demonstrated a 6.3-cm poorly differentiated adenocarcinoma with squamoid features (Fig. 4A). Focally, the tumor was papillary and glandular (Fig. 4B). Tumor extended along lymphovascular channels in portal tracts and invaded perineural spaces. Tumor cells with squamoid features and p63 reactivity were also observed lining a large bile duct with invasion into its surrounding stroma (Fig. 4C–E).

4. Discussion

We demonstrate here a case of synchronous pancreatic adenocarcinoma and intrahepatic cholangiocarcinoma (ICC) arising in the context of IPN-B and IPMN-P, respectively. We believe that this is the first case of such an entity described in the radiology literature. Pancreatic adenocarcinoma and ICC are both highly malignant tumors and, as we have shown in our case, may not present with symptoms early in the disease course. The pathologic finding of the presence of dilated ducts with micropapillary and squamoid features within the tumors indicates that each of the malignant tumors arose from underlying corresponding precursor lesions (IPMN-P and IPN-B). At initial histopathologic examination, pancreatic adenocarcinoma and ICC may have similar appearance [1], and immunohistochemical analysis was essential in this case in making the distinction between these solid neoplasms.

A relatively new thought emerging in the pathology and hepatology literature is the concept of biliary disease with a pancreatic counterpart