Case Report

Coexistent Ampullary Squamous Cell Carcinoma with Adenocarcinoma of the Pancreatic Duct

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ABSTRACT

Primary squamous cell carcinoma (SCC) of ampulla has seldom been reported. However, metastatic SCC to ampulla of Vater is well known. We report a case of primary SCC of ampulla of Vater coexistent with well-differentiated adenocarcinoma of the distal pancreatic duct. A 50-year-old female presented with evidence of obstructive jaundice. Endoscopic retrograde cholangio-pancreatography revealed bulging papilla with ulcero-infiltrative growth at the ampulla of Vater. An initial endoscopic biopsy of the ampullary mass showed a well-differentiated SCC. The patient underwent Whipple's operation. Thorough sampling of the dilated portion of the pancreatic duct showed presence of well-differentiated adenocarcinoma of the distal pancreatic duct. Immunohistochemical study with synaptophysin and chromogranin was done with negative result, ruling out neuroendocrine differentiation. Also, a detailed clinical, endoscopic and radiological examination was carried out, that excluded the presence of primary SCC elsewhere.

Key Words: Ampulla of Vater, coexistent tumor, ductal adenocarcinoma, squamous cell carcinoma

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Tumors of the ampulla of Vater are mostly adenocarcinomas and other histological types are less frequent.[1] Squamous cell carcinoma (SCC) of the ampulla is quite rare and most of the reported cases are of metastatic SCC from elsewhere.[2,3] Herein we report a case of coexistent SCC of the ampula of Vater with adenocarcinoma of the distal pancreatic duct.

CASE REPORT

A 50-year-old female presented with history of generalized body itching and jaundice since two months. History of dark-colored urine and decreased appetite was also elicited. On examination, the patient was cachectic and generalized icterus was noted. Laboratory investigations revealed deranged liver function tests. Total bilirubin was increased to 6.27 mg/dl- direct bilirubin - 5.15 mg/dl and indirect bilirubin - 1.12 mg/dl. Serum glutamic pyruvic transaminases (SGPT) and serum glutamic oxaloacetic transaminases (SGOT) levels were normal (29 IU/L and 32 IU/L respectively). Alkaline phosphatase was markedly raised (217 IU/L), indicating obstructive pathology. Total protein was within normal limits. Ultrasonography showed a marked dilatation of intra and common hepatic bile duct. Endoscopic retrograde cholangio-pancreatography (ERCP) showed a bulging papilla with ulcero-infiltrative growth at the ampulla on duodenoscopy, suggestive of ampullary carcinoma [Figure 1]. Papillectomy was done and a stent was placed in the common bile duct. Histopathological examination revealed well-differentiated SCC. Based on this, Whipple's procedure was performed and the specimen was sent for histological evaluation. On cutting open the duodenum, a stent was seen in the ampulla of Vater, extending in the dilated portion of the pancreatic duct. The cut surface of the ampullary region showed a gray-white, infiltrating tumor tissue adjacent to the stent [Figure 2]. Multiple sections from the ampullary end revealed intact peri- ampullary mucosa with islands of moderately differentiated SCC in the submucosa up to the muscularis propria [Figure 3a]. Sections from the pancreatic duct revealed well-differentiated adenocarcinoma in the distal segment of the duct. This carcinoma was seen infiltrating the surrounding periductal tissue, sparing the pancreas [Figure 3b]. Immunohistochemistry confirmed the

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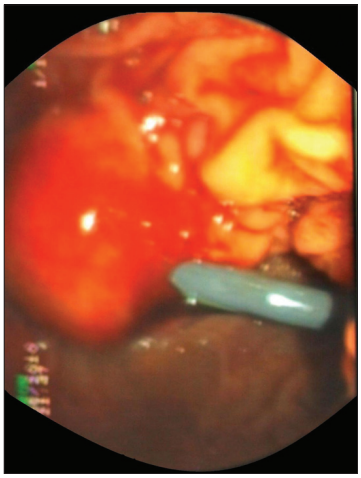

Figure 1: Photograph of the ERCP showing a stent placed in the bulging ampulla

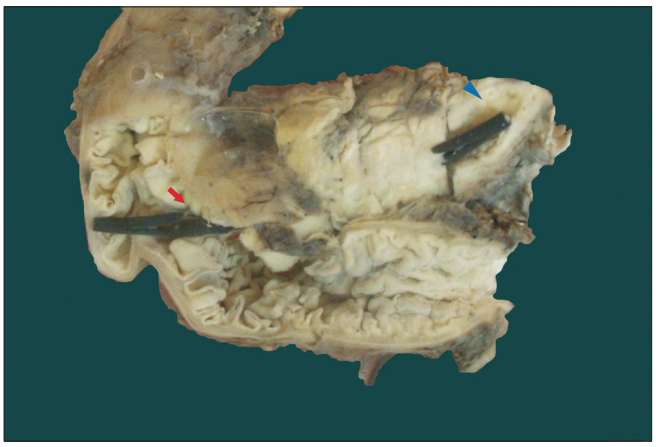

Figure 2: Photograph of the gross specimen, showing the stent on the duodenal side (arrow) and at the cut end of resection (arrowhead), with the surrounding gray-white tumor tissue

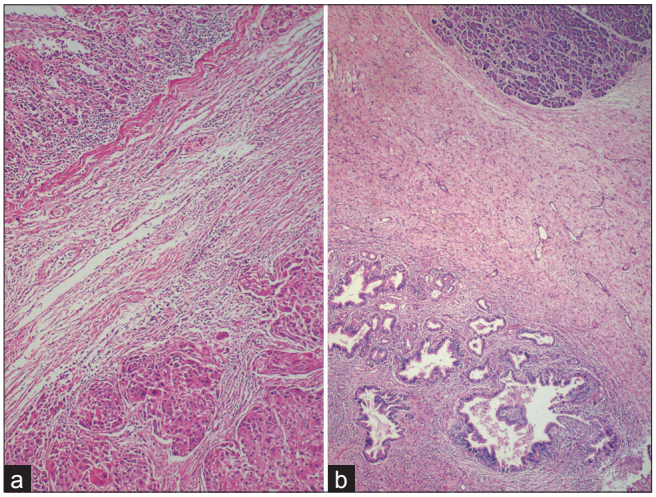

Figure 3: (a) Photomicrograph showing the ampullary squamous cell carcinoma covered by duodenal mucosa (H and E, ×100). (b) Ductal adenocarcinoma in the distal part of pancreatic duct (H and E, ×100)

above findings. Pan CK (Dako; clone:AE1/AE3) was positive in both types of tumor cells. Tumor cells in the ampullary region were negative for synaptophysin (Dako;clone:SY38) and chromogranin (Dako;polyclonal), ruling out neuroendocrine origin. Taking all these findings into consideration, a diagnosis of coexistent ampullary SCC and ductal adenocarcinoma was offered. Considering the rarity of the occurrence of SCC at the ampulla of Vater, detailed clinical, endoscopic and radiological examination of the patient was carried out, but it did not reveal any other primary focus of the disease. The patient is being followed up for the last eight months and has shown no signs of recurrence.

DISCUSSION

Carcinoma of the ampulla of Vater is a relatively rare neoplasm, comprising 15-37% of surgically resected pancreatoduodenal tumors and 0.2% of routine autopsy cases.[4] Other than adenocarcinoma, the other primary tumors reported at the ampulla are neuroendocrine carcimoma[5] and signet cell carcinoma,[6] etc. Until now, the reported cases of ampullary SCC have been those of metastases from other sites like the larynx, esophagus etc.[2,3] Reviewing the literature, we encountered two cases of primary SCC of the ampulla,[7] though, to the best of our knowledge this is the first report of coexistent ampullary primary SCC with pancreatic duct adenocarcinoma. The patient was extensively investigated to rule out the presence of a primary elsewhere. Histologically, the diagnosis of primary SCC of the ampulla of Vater should be differentiated from a neuroendocrine tumor. To rule out the presence of a neuroendocrine component in the tumor, immunohistochemical staining for synaptophysin and chromogranin was performed, which was negative. The possibility of adenosquamous carcinoma was ruled out as in this case, the squamous component was restricted to the ampulla and the adenocarcinoma to the distal pancreatic duct, with no intermingling of the two components.

The origin of SCC in the ampulla of Vater remains unelucidated. Squamous metaplasia of the pancreatic ductal epithelium after chronic inflammation has been hypothesized as one of the possible oncogenic mechanisms for the development of primary SCC of pancreatic duct.[8] It makes one wonder if a similar pathogenetic mechanism plays its role in ampullary squamous cell carcinoma.

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