Intraluminal Tumor of the Common Bile Duct as a Metastasis of Renal Cell Carcinoma

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A 57-year-old woman was admitted for evaluation of liver dysfunction. A physical examination revealed jaundice and a left abdominal mass, which was diagnosed as being a large renal tumor. Cholangiography showed a smooth filling defect 1 cm in diameter at the common bile duct. Left nephrectomy, and resection of the common bile duct were performed. The pathological diagnosis was metastasis of the common bile duct wall resulting from renal cell carcinoma. Metastatic common bile duct tumors are extremely rare. However, it is important to consider that this is one of the causes of obstructive jaundice.

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Key words: metastatic tumor, obstructive jaundice, choledocholithiasis

Introduction

Obstructive jaundice due to a metastatic tumor occurs predominantly as a consequence of a lymphogenous metastasis, which results from extrinsic compressions of the bile duct. Among the underlying primary tumors, gastrointestinal carcinomas predominate, in particular those tumors originating in the stomach and pancreas (1). However, more rarely encountered neoplasms such as melanoma have sometimes been reported as intraluminal metastasis of the bile duct, indicating hematogenous metastasis (2–4).

We report herein a patient with intraluminal common bile duct metastasis due to renal cell carcinoma and review the cases of metastatic biliary tumor in the literature.

Case Report

A 57-year-old woman was admitted for evaluation of jaundice. Two weeks before admission, she experienced general fatigue and brown urine. There was no history of drug ingestion, transfusion, or needle exposure. She had no previous complaint or the suggestion of hepatobiliary disease. A physical examination revealed that the patient was jaundiced and afebrile. Her body weight was 77 kg and her height was 150 cm. Her abdomen was distended and an elastic hard mass about 10 cm in diameter was palpable on the left side of the abdomen which

was slightly tender. On admission, her hemoglobin level was 13.1 g/dl. Her leukocyte count was $5,700 \text{ cells/mm}^3$ and the platelet count was $17.7 \times 10^4 \text{ cells/mm}^3$. The total serum bilirubin value was 3.3 mg/dl (direct bilirubin was 2.1 mg/dl), alkaline phosphatase was markedly elevated to 1,443 IU/l (normal 105–290 IU/l), and γ -glutamyl transpeptidase was also elevated to 1,942.9 IU/l. The serum aspartate aminotransferase and alanine aminotransferase were 352 IU/l and 441 IU/l, respectively. The serum amylase value, serum electrolytic concentration and renal function including 24-hour creatinine clearance rate were normal. Serological studies for hepatitis B and C viruses were negative and some tumor markers (CEA, CA19-9, AFP) were also negative. An ultrasonography and computed tomography (CT) revealed a remarkably swollen gall bladder and a slightly dilated upper portion of the common bile duct (CBD). Furthermore, a giant tumor on the upper pole of the left kidney was also revealed. This tumor was $10 \times 12 \text{ cm}$ and was easily viewed on the enhanced CT image (Fig. 1). The pancreas was almost normal, however its tail had shifted to the cranial side due to the tumor. Endoscopic retrograde cholangiopancreatography (ERCP) showed a normal pancreatic duct image and a smooth filling defect (1 cm in diameter) at the lower end of the CBD, mimicking a common bile duct stone.

As the serum bilirubin level gradually increased up to 15.8 mg/dl, percutaneous transhepatic gall bladder drainage (PTGBD) was performed. Cholangiography through the drainage catheter

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also revealed the smooth filling defect, which was not moved by compression (Fig. 2). A left renal angiogram demonstrated a considerable hypervascular lesion on the upper pole of the

Figure 1. Computed tomography demonstrates a well enhanced mass about 10 cm in diameter at the left kidney (arrow) and a remarkably swollen gallbladder.

Figure 2. Cholangiography through the drainage catheter reveals the smooth filling defect 1 cm in diameter at the lower end of the common bile duct.

kidney, suggesting renal cell carcinoma. On the other side, a celiac arteriogram revealed a hypervascular lesion which was supplied by the gastroduodenal artery and located exactly on the obstructive site of the CBD (Fig. 3). At that time, we diagnosed that the patient was suffering from left renal cell carcinoma, however, the cause of the CBD obstruction was unknown. From a cholangiogram, the cause of the smooth filling defect seemed to be a stone or extrinsic compression by a regional lymph node or another benign tumor. However, an angiogram suggested that there was a hypervascular neoplasm at that obstructive lesion, therefore, the possibility of a metastatic renal cell carcinoma was considered.

Surgical findings showed an intraluminal polypoid tumor at the distal CBD without regional lymph node enlargement. Left nephrectomy, resection of the CBD including the gall bladder and choledochojejunostomy were performed. The pathological diagnosis of the renal tumor was renal cell carcinoma of the clear cell type with an alveolar structure. No invasion of the renal veins or lymph nodes was detected.

The gross appearance of the resected CBD demonstrated a solitary tumor 1 × 0.7 cm in diameter at the lower site of CBD, which had a reddish smooth surface and was well defined from the surrounding mucosa, protruding into the lumen (Fig. 4a). A lower power microscopic view revealed that the tumor was covered with a degenerated and eroded mucosal layer and localized mainly in the submucosa of the CBD (Fig. 4b). Histologically, comparison of these specimens with original nephrectomy specimens confirmed a similar morphological appearance consistent with the diagnosis of metastatic renal cell carcinoma (Fig. 5). The patient remains well 5 months later, with no evidence of recurrence.

Discussion

Renal cell carcinoma (RCC) sometimes metastasizes to the lung, liver or bone in a hematogenous metastatic form. How-

Figure 3. Celiac arteriogram shows a hypervascular lesion (arrow) which is supplied by the gastroduodenal artery and is located precisely on the obstructive site of the common bile duct.

Figure 4. A) The gross appearance of resected CBD demonstrates a solitary tumor 1 × 0.7 cm in diameter at the lower site of CBD. The tumor has a hemispherical shape, protruding into the lumen and is well defined from the surrounding mucosa. B) Lower power microscopic view reveals that the tumor is located mainly in the submucosa of the CBD (HE stain, ×10). CBD: common bile duct.

ever, metastasis to the pancreatobiliary system from RCC is extremely rare. Some authors have reported that the percentage of metastasis to the pancreas from far advanced RCC is 2.0% (5) or 2.8% (6). However, with regard to CBD metastasis from RCC, only five case reports have been listed on the MEDLINE data base from 1966 to 1995 (7–11). Of these cases, three were an ampullary mass and the others (10, 11) were intraluminal CBD metastasis. All five cases of metastatic CBD tumor from RCC were located at a distal site of the CBD, including Vater's ampulla. These locations are different from the preferred site of the lymphogenous metastasis, which is in the neighborhood of the hepatic hilus.

ERCP and/or cholangiography through the drainage catheter have been of diagnostic benefit in the evaluation of patients with obstructive jaundice. In most instances, these cholangiographies will allow differentiation of choledocholithiasis from

Figure 5. Histological view: A) Renal cell carcinoma; Alveolar structure separated by a thin-walled vascular stroma is shown. The tumor cell has clear cytoplasm with a small nucleus (HE stain, ×100). B) Common bile duct tumor; Architecture and tumor features are essentially the same as those of the renal cell carcinoma (HE stain, ×100).

both primary and metastatic diseases of the biliary tract. The most common pattern found is that of CBD stones; these are typically visualized as radiolucent and smooth defects within the CBD. Moreover, the primary biliary duct carcinoma presents the appearance of a polypoid ductal wall change or an abrupt convex filling defect. Such change often permits differentiation from metastatic obstructive jaundice, which narrows irregularly without intraluminal filling defects because of extrinsic compression of the CBD. In the present case however, the cholangiogram showed a smooth filling defect within the CBD, and thus we could not conclude that the cause of the obstruction was a metastatic tumor of the CBD.

Metastatic obstructive jaundice may result from extrinsic compression of the bile duct, for example direct involvement of the extending tumor from adjacent organs or neighboring lymph nodes. A small number of gall bladder or CBD metastasis from melanoma have been reported. Clinically, they have been presented as typical cholecystitis/cholelithiasis or choledocholithiasis (2–4). Especially, metastatic melanoma of the gall

bladder often begins as submucosal nodules that eventually become pedunculated, and some may even resemble primary malignant tumors of the gall bladder (12). The present case resembles a metastatic tumor from melanoma, which was polypoid-shaped and protruding into the lumen of the CBD. The same metastatic patterns, which are thought to be hematogenous metastasizations, have been reported in neoplasms other than melanoma. Three cases of colon carcinomas metastasizing to the lumen of the CBD have been reported (13–15). In particular, Dyess et al (15) reported a tumor that was “mimicking choledocholithiasis”. Some breast carcinomas metastasizing to the CBD have been reported (1, 16), however, these reported obstructive jaundice cases were thought to be the result of involvement of the extending adjacent tumor.

The majority of renal cell carcinomas are hypervascular tumors (17). In the same way, metastatic tumors from RCC are hypervascular on angiography and are distinguished from primary carcinoma of the pancreas except for non-functional islet cell tumors or cystic tumors (18). From the view of the cholangiogram, we were unaware of an intraductal metastatic tumor at the CBD. However, an angiogram revealed that the tumor had hypervascularity and retrospectively this hypervascularity was the key to the diagnosis of the metastatic tumor from RCC.

With regard to RCC, metastatic tumors are sometimes revealed more than 5 years after the initial excision (19, 20). Venu et al (9) and Robertson and Gertler (7) reported a metastatic ampullary tumor from RCC 11 years and 12 years after nephrectomy, respectively; Letessier et al (11) reported CBD metastasis 14 years after nephrectomy. It is important that long-term follow-up is done after excision of the primary RCC and that any hypervascular tumors developing after nephrectomy for RCC, even if more than 10 years after nephrectomy, should be considered to be metastatic tumors from RCC. In the present case however, RCC and its metastatic tumor were found at the same time. Because of the slow growing nature of the RCC, we did not discover the RCC in the early stage, so eventually obstructive jaundice, which was the metastatic form was the first sign of the RCC.

As far as the treatment is concerned, O’dea et al (20) demonstrated that surgical resection of the metastatic lesion seem to offer the best results in patients who presented a solitary metastatic lesion after nephrectomy, however, the prognosis was uniformly poor. Kauffmann et al (10) treated the intracholedocal metastatic tumor of RCC by intraluminal irradiation with radioiridium in order to maintain the patency of the CBD by controlling tumor proliferation within the duct, which resulted in a favorable outcome for 18 months. In any case, surgical resection of the metastatic lesion seems to be marginally better when possible, however, with regard to CBD metas-

tasis, it is debatable whether or not radical resection such as pancreaticoduodenectomy should be performed.

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