

Hepatoma Presenting as Extrahepatic Biliary Obstruction Due to Hemobilia

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A case of hepatoma presenting as extrahepatic biliary obstruction due to hemobilia is reported. The patient, a 49-year-old woman, developed jaundice of the obstructive type after a history of B-viral hepatitis. On laparotomy, the liver revealed macronodular cirrhosis without any noticeable mass. A 4-cm sized friable tissue and blood clots were identified within the distended left hepatic duct. Pathologic examination of this tissue confirmed the diagnosis of hepatocellular carcinoma extended in the hepatic duct.

Key Words: Hepatocellular carcinoma, obstructive jaundice, extrahepatic biliary obstruction

Hepatocellular carcinoma is known to be one of the most common cancers in men of Korea (Lee *et al.* 1952; Lee and Lee 1961). The tumor usually presents as a painful and enlarging mass in the right upper quadrant of the abdomen (Ihde *et al.* 1974). Ascites, portal vein thrombosis, occlusion of hepatic veins, and hemorrhage from esophageal varices are other common features. However, it is rare for jaundice to occur as an initial presenting finding. At present, about thirty cases have been reported in the English literature (Afroudakis *et al.* 1978; van Sonnenberg and Ferrucci 1979; Jurco and Kim 1980; Sarma *et al.* 1987) and none in Korea.

In hepatoma, jaundice is usually caused by the infiltrating tumor or underlying cirrhosis. And less commonly, direct extension of the tumor into the major hepatic ducts may bring about jaundice in a later stage. We report a case of hepatocellular carcinoma causing obstructive jaundice as an initial presenting feature.

CASE REPORT

A 49-year-old woman was admitted with a 10-day history of epigastric pain, fever and chilling sensation. She had suffered from duodenal ulcer for one year,

which was intermittently treated with medications. Six months ago, she presented with jaundice, and was managed under the impression of hepatitis in a private clinic.

On admission she was mildly febrile (37.2°C). The sclera was slightly icteric. The bowel sound was hypoactive and direct tenderness was noted in the epigastrium. Total bilirubin was 5.0mg/100ml with direct 3.2mg/100ml, SGOT 98 units, SGPT 56 units, serum alkaline phosphatase 421 MIU, and prothrombin time was 13.2 sec (86.3%). The urine gave a positive test for urobilinogen. Serum HBs Ag, HBe Ag, anti-HBc Ab IgG and HBV DNA were positive, but anti-HBs Ab, anti-HBe Ab and anti-HBc Ab IgM were negative. Serum alpha-fetoprotein was 1779ng/ml.

Abdominal ultrasound examination revealed multiple echogenic materials without shadowing associated with dilatation of the peripheral duct. DISIDA scan demonstrated a well-visualized liver and gastrointestinal activity, but the gallbladder was not visualized. Abdominal C-T scan showed ill defined nodular increased density with partial central low density in the dilated hepatic duct, which is suggestive of intrahepatic stone (Fig. 1a). ERCP revealed non-visualization of the common hepatic duct and left hepatic duct, suggestive of choledocholithiasis or Klatskin tumor (Fig. 2a).

On the 14th hospital day, an explorative laparotomy was performed, and a 4-cm sized, irregular, reddish-tan, muddy, friable tissue admixed with blood clots was found at about 4 cm from the distended left hepatic duct. Grossly, the liver showed macronodular cirrhosis without any palpable mass.

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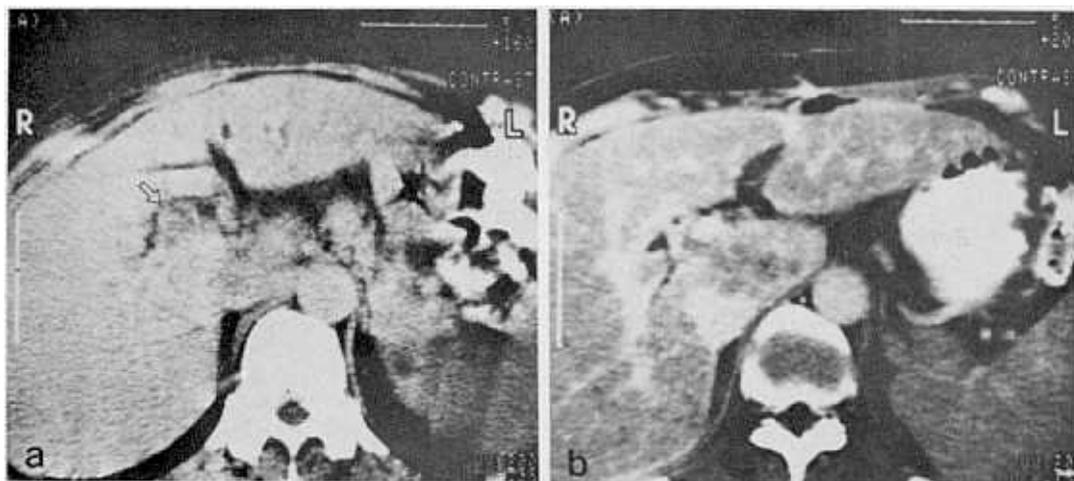


Fig. 1. The abdominal C-T scan shows ill-defined nodular increased density with partial central low density in the dilated hepatic duct (a). The follow-up C-T scan reveals a new ill-defined inhomogeneous density in the caudate lobe of the liver (b).

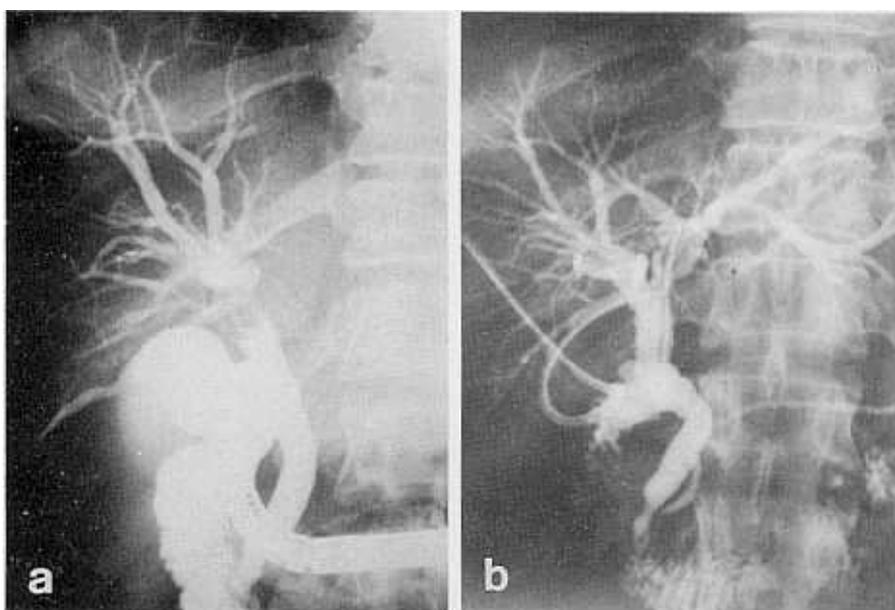


Fig. 2. The cholangiogram revealed an intraluminal filling defect of the common hepatic duct and left hepatic duct (a). The intraoperative T-tube cholangiogram revealed good filling of dye in both hepatic ducts (b).

The gallbladder was normal in appearance and no intraluminal stones were found. Tissue obtained from the left intrahepatic duct was sent for frozen section and the frozen diagnosis was malignancy. An intraoperative T-tube cholangiogram showed good filling of dye in both right and left hepatic ducts (Fig. 2b).

Sections of the tissue revealed a pink-gray granular tumor mass within the blood clots. Microscopically, a layer of the choledochal tall columnar epithelium and a sheet of large clusters of tumor cells, resembling polyhedral hepatocytes, were noted with large vesicular and hyperchromatic nuclei, prominent

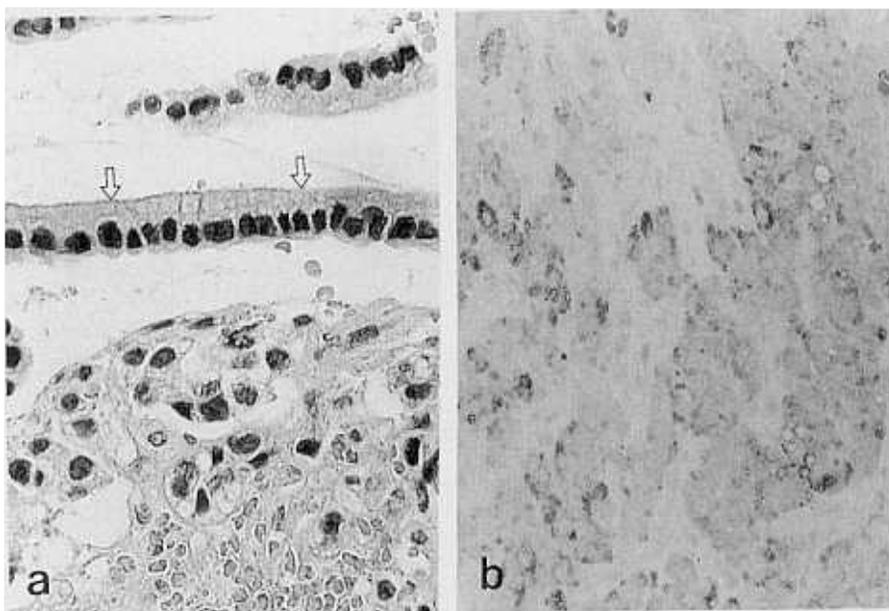


Fig. 3. The section of tissue taken from the left hepatic duct demonstrated a layer of choledochal epithelium (arrow) and a sheet of large atypical tumor cells resembling hepatocytes. The sinusoidal pattern is evident in the tumor (a: H-E, $\times 200$). The immunohistochemical stain for alpha-fetoprotein shows a positive reaction (b: PAP, $\times 200$).

nucleoli and abundant granular eosinophilic cytoplasm (Fig. 3a). In some areas, sinusoidal patterns were evident. Some were multinucleated and bizarre nuclear forms were also noted. The immunohistochemical stain demonstrated strong positive reaction for alpha-fetoprotein (Fig. 3b). The final diagnosis was hepatocellular carcinoma. A wedge biopsy of the liver revealed macronodular cirrhosis divided by broad fibrous septae with some remaining activity of piecemeal necrosis at the limiting plate, but no tumor cells were noted.

The postoperative course was uneventful and she was discharged on the 20th day. One month later, a follow-up abdominal C-T scan was performed in order to search for the primary lesion and revealed an ill-defined, small, inhomogeneous density at the caudate lobe of the liver (Fig. 1b). She received two cycles of chemotherapy (5-fluorouracil).

COMMENT

The clinical manifestations of hepatocellular carcinoma fall into at least eight different types; the frank type, the cirrhotic type, the occult type, the febrile type, the acute abdominal type, the metastatic type, the hepatitis type, and the cholestatic type, based on

symptoms, signs, and courses (Okuda 1976). Among them, the first two types are usually the common forms of presentation, while the cholestatic type is relatively rare.

Mechanisms of hepatoma-induced biliary obstruction include pedunculated tumor extension (Johns and Zimmerman 1961), obturating hemorrhagic clot and tumor debris (Fisher and Creed 1956; Johns and Zimmerman 1961; Gerson and Schinella 1969; Ischikawa *et al.* 1973; Brand *et al.* 1976), direct invasion of the intrahepatic biliary system by a tumor nodule, and metastatic lymph node compression of the major ducts in the porta hepatis (Mallory 1947). Obstructive jaundice occupies about 20% of clinical findings of hepatoma in Korea (Kim and Lee 1981) and is usually a late manifestation due to massive liver involvement by a tumor in association with underlying cirrhosis. Moreover, obstructive jaundice as an initial presenting symptom secondary to complete obstruction of the common duct by a tumor is rare. No such cases have been reported in Korea and only a few in English literature (Ihde *et al.* 1974; Lai *et al.* 1981). One of the characteristics of hepatoma is its propensity to undergo necrosis and degeneration, especially in a large tumor nodule. If this should occur in an area of a tumor contiguous with a bile duct, it is con-

ceivable that a large fragment of this tumor may become free and traverse the biliary system until it is lodged in the common bile duct (Gerson and Schinella 1969).

The finding in most cases undergoing exploration is a dilated duct filled with soft, fleshy material, so-called "chicken fat" (Edmondson and Steiner 1954). Interestingly, the frozen section report of the tumor usually comes as a surprise since few of the reported cases had a palpable tumor close to the surface of the liver (Sanford 1952; Edmondson and Steiner 1954; Gerson and Schinella 1969; Waldron et al. 1973; Brand et al. 1976; Wind and Futterman 1977; Afroudakis et al. 1978). We could not identify hepatoma in our patient by gross examination of the liver, but eventually we could identify a hepatic mass radiologically by the follow-up abdominal scan. From these findings, we assumed that hepatic duct invasion occurred from a small nodule contiguous with one of the major hepatic ducts and obstructive jaundice took place by continuous tumor cell growth and hemorrhage from the surrounding tissue.

The development of a cholestatic picture in a patient with underlying cirrhosis should alert the physician to the biliary tract complications of hepatocellular carcinoma (Afroudakis et al. 1978). The differential diagnosis of a biliary obstruction involving proximal main hepatic ducts should include rare hepatocellular carcinoma among other lesions such as adenocarcinoma of the hepatic duct junction (Klatskin tumor), metastatic tumors, and stones (Sarma et al. 1987), and infestation with *Clonorchis sinensis* (Gerson and Schinella 1969). It would appear worthwhile to submit any unexplained debris or hemobilia found in the common hepatic duct for histologic study since it may contain malignant cells.

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