

to suffer from progressive epigastric fullness and postprandial vomiting 5 days before the final admission on 6 May. After a nasogastric tube was inserted, about 1100 mL of dark greenish fluid with food material was drained. KUB revealed severe gastric distension. Abdominal sonography at the final admission showed an ill-defined mosaic tumor at the hilar region of the liver with daughter tumorous nodules, and dilatation of the intrahepatic duct and common bile duct. An ulcerative tumor was noted over the duodenal bulb by endoscopic examination, and direct invasion of the liver tumor to the duodenal bulb was suspected. The upper GI series showed derranged mucosa at the antrum and duodenal bulb. Although a semi-liquid diet was given, abdominal distension and vomiting persisted. So TPN was administered from 9 May. Unfortunately, intermittent low-grade fever was noted from June 3 and could not be controlled even with third-generation antibiotics. Radiotherapy and chemotherapy with FT-207 supplement were applied from 17 June. In spite of aggressive treatment, her condition deteriorated. Blood pressure drop and loss of consciousness occurred on 23 June. She expired on 24 June 1998. An autopsy was performed

DIFFERENTIAL DIAGNOSIS AND DISCUSSION

From a practical standpoint, clinical conditions that cause jaundice can be classified as isolated disorders of bilirubin metabolism, liver disease, or obstruction of the bile ducts. Isolated disorders of bilirubin metabolism include increased bilirubin production (e.g., hemolysis, blood transfusion, resorption of hematomas, and ineffective erythropoiesis) and decreased hepatic bilirubin clearance (such as decreased uptake or conjugation of bilirubin and decreased canalicular secretion of bilirubin). Liver diseases include acute or chronic hepatocellular injuries caused by viral hepatitis, hepatotoxins, drugs, ischemia, and metabolic or inherited disorders. Hepatic disorders with prominent cholestasis include diffuse infiltrative disorders (e.g., granulomatous disease), infiltrative malignancies, inflammation of intrahepatic bile ductules and/or portal tracts, and other miscellaneous conditions including benign recurrent cholestasis, drugs, systemic bacterial infection, uncommon presentations of viral or alco-

holic liver diseases, or inherited disorders.¹

According to the symptoms, signs, and the radiological examination of the patient, the jaundice was mainly due to obstruction of the bile ducts. Cholelithiasis from cholesterol or pigmented gallstones is one of the possible causes. However, infectious diseases or neoplasms of the bile ducts, for example, cholangiocarcinoma, should be excluded first. Besides, extrinsic compression of the biliary tree resulting from neoplasm or chronic pancreatitis should be considered, too.

Viewed clinically, cholestasis may result from conditions affecting the large bile ducts (extrahepatic cholestasis) or from disorders of the small bile ducts, canaliculi, or hepatocytes (intrahepatic cholestasis). Typically one would expect such a patient with obstructive jaundice to show (1) predominantly conjugated hyperbilirubinemia, (2) minimal biochemical changes of parenchymal liver damage, and (3) a moderate to a marked increase in the serum alkaline phosphatase level (usually 3 or 4 times normal [or greater than 250 IU/L]). An elevated alkaline phosphatase level in a patient with jaundice or liver disease, in the absence of other disorders such as bone disease, is most suggestive of interference with bile secretion or an infiltrative process in the liver. In conclusion, when the major detectable alterations of liver function tests include (1) conjugated hyperbilirubinemia and (2) moderate to marked elevation of the serum alkaline phosphatase level, the term obstructive or cholestatic jaundice may be appropriate.

Neoplasms are the most common and important causes of biliary obstruction. Primary malignancies of the liver, bile ducts, gallbladder, ampulla of Vater, and pancreas account for most diagnoses, and patients with these cancers most typically present with jaundice caused by bile duct obstruction. A metastatic tumor from any source to the lymph nodes in the porta hepatis can also cause extrinsic compression of the proximal common bile duct, and this is a common cause of cholestasis in patients with cancers of the breast, lung, colon, or stomach. Not all cholestasis caused by malignancies is extrahepatic. Diffuse infiltration of malignant cells along hepatic sinusoids with consequent cholestasis may also occur, especially in small cell carcinoma of the lung and malignant lymphoma.

Judging from the clinical pictures, image studies,