Liver tumors: an overview

Munechika Enjoji

Department of Clinical Pharmacology, Faculty of Pharmaceutical Sciences, Fukuoka University, 8-19-1 Nanakuma, Johnan-ku, Fukuoka 814-0180, Japan (ME)

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Classification

WHO histological classification of the liver and intrahepatic bile ducts.

Epithelial tumors

Benign:
- Hepatocellular adenoma
- Focal nodular hyperplasia
- Intrahepatic bile duct adenoma
- Intrahepatic bile duct cystadenoma
- Biliary papillomatosis

Malignant:
- Hepatocellular carcinoma
- Intrahepatic cholangiocarcinoma
- Bile duct cystadenocarcinoma
- Combined hepatocellular and cholangiocarcinoma
- Hepatoblastoma
- Undifferentiated carcinoma

Non-epithelial tumors

Benign:
- Angiomyolipoma
- Lymphangioma and lymphangiomatosis
- Hemangioma
- Infantile hemangioendothelioma

Malignant:
- Epithelioid hemangioendothelioma
- Angiosarcoma
- Embryonal sarcoma
- Rhabdomyosarcoma
- Others

Miscellaneous tumors

- Solitary fibrous tumor
- Teratoma
- Yolk sac tumor
- Carcinosarcoma
- Rhabdoid tumor

Others

- Hematopoietic and lymphoid tumors
- Secondary tumors
- Epithelial abnormalities
  - Liver cell dysplasia
  - Dysplastic nodules
  - Bile duct abnormalities
- Miscellaneous lesions
  - Mesenchymal hamartoma
  - Nodular transformation
  - Inflammatory pseudotumor

Clinics and pathology

Disease

Benign tumors

Hepatic hemangioma: Hemangioma is the most common benign tumor of the liver and is more frequent in women. Many cases are discovered incidentally and the reported incidence is 2-20%. Tumor size may be associated with pregnancy and estrogen levels. Hepatic hemangioma is usually asymptomatic, and rarely grows or bleeds. Surgical excision is required in some symptomatic cases. No genetic alterations have been reported.

Focal nodular hyperplasia (FNH): FNH is the second most frequent benign tumor of the liver and occurs mainly in women (80-90%). In a hyperplastic lesion, all of the normal liver constituents are present but in an abnormally organized pattern. Underlying congenital arteriovenous malformation, oral contraceptives, and some medicines such as azathioprine are considered to be pathogenic factors. FNH has no malignant potential but a combination of various imaging techniques may be needed for correct diagnosis. FNHs exhibit few chromosomal abnormalities. Strong immunostaining of
Hepatocellular carcinoma is the most frequently observed and clinically important primary hepatic neoplasm. It occurs more commonly in men than women and its geographical distribution varies considerably. In areas with high incidence, chronic infection with HBV or HCV is a well-known underlying cause. A frequent association with chronic liver disease/cirrhosis has also been reported. The reported mutation rates of K-ras, which is converted to an active oncogene by point mutations, vary from 0-8% in Thai patients. Inactivation of p53 by mis-sense or non-sense mutations and by loss of chromosome 17p induces disruption of critical growth-regulating mechanisms and may have a crucial role in carcinogenesis. It has been reported that the p53 mutation and loss of chromosome 17p was present in 11-37% and 38% of intrahepatic CCs, respectively. Alterations of the tumor suppressor gene, p16INK4A, which is frequently seen in combined tumors similar to that in CCs, has been reported. No cytogenetic alterations have been reported.

Malignant tumors

Hepatocellular carcinoma (HCC): HCC is the most frequently observed and clinically important primary hepatic neoplasm. It occurs more commonly in men than women and its geographical distribution varies considerably. In areas with high incidence, chronic infection with HBV or HCV is a well-known underlying cause. A frequent association with chronic liver disease/cirrhosis has also been reported. The reported mutation rates of K-ras, which is converted to an active oncogene by point mutations, vary from 0-8% in Thai patients. Inactivation of p53 by mis-sense or non-sense mutations and by loss of chromosome 17p induces disruption of critical growth-regulating mechanisms and may have a crucial role in carcinogenesis. It has been reported that the p53 mutation and loss of chromosome 17p was present in 11-37% and 38% of intrahepatic CCs, respectively. Alterations of the tumor suppressor gene, p16INK4A, which is frequently seen in combined tumors similar to that in CCs, has been reported. No cytogenetic alterations have been reported.

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liver epithelium and displays various histological patterns. The incidence is twice as high in boys than girls. Key markers include elevation or non-decreasing expression of α-fetoprotein, hepatomegaly, and weight loss. It is strongly associated with familial adenomatous polyposis probably owing to altered expression of the adenomatous polyposis coli (APC) gene. The most common genetic aberrations are extra copies of chromosomes 1q, 2q, 4q, 7q, 8, 17q, and 20, and LOH for 11p. It has been reported that p53 mutations contribute to hepatoblastoma.

**Bile duct cystadenocarcinoma:** Hepatic cystadenocarcinomas, p53, c-erbB-2, and bcl-2 were contribute to hepatoblastoma. It has been reported that p53 mutations for 11p. It has been reported that p53 mutations contribute to hepatoblastoma. For curative resection, the prognosis is good. In an polyposis probably owing to altered expression of the the pathogenesis of combined hepatocellular-cholangiocarzinoma. Lab Invest. 1999 Apr;79(4):477-83

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