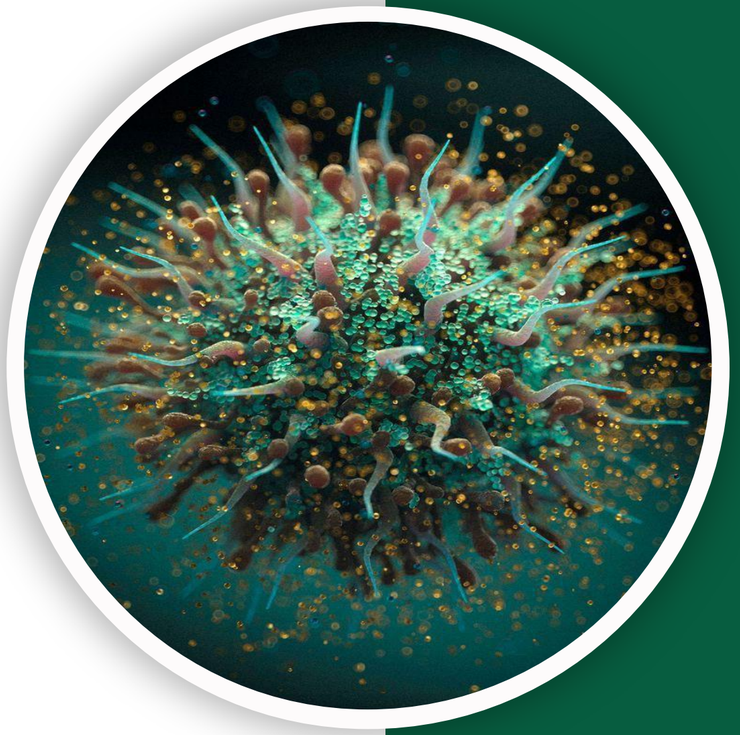
بسم الله الرحمـٰن الرحيم

(وَفَوْقَ كُلِّ ذِي عِلْمٍ عَلِيمٌ)

GIS Pathology | FINAL 6

Liver Tumors



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Liver tumors

❑ Benign

- **Most common is :**

➢ **cavernous hemagioma**

- **Usually < 2cm (small)**
- **Subcapsular**

➢ **Liver cell adenoma**

- The presentation is usually a :
  - **Young female**
  - With **History of oral contraceptive intake**
- **May rupture esp. during pregnancy causing severe intraperitoneal hemorrhage** particularly if enlarged due to hormonal effect.
- **Rarely may contain HCC**
- Can be **Misdignosed with HCC**

Liver tumors

Liver cell adenoma (Pathoma Book High-Yield Notes) :

A. Benign tumor of hepatocytes.
B. Associated with oral contraceptive use; regresses upon cessation of drug.
C. Risk of rupture and intraperitoneal bleeding, especially during pregnancy.
    1. Tumors are subcapsular and grow with exposure to estrogen.

Liver Nodules

- They are important because they can be misdiagnosed for Malignant tumors.

Focal noudular hyperplasia

- **Well demarcated hyperplastic hepatocytes with central scar.**
- **Non-cirrhotic liver** (not associated with Cirrhosis).
- **Not neoplasm but nodular regeneration.**
- It may be due to **Local vascular injury.**
- Usually the presentation is **Females of reproductive age.**
- **No risk of malignancy**
- **20% of cases have cavernous hemagnioma.**

Macroregenerative Nodules

- Simply a cirrhotic nodule that is larger than typical ones.
- **Cirrhotic liver**
- **Larger than cirrhotic nodules**
- **No atypical features,**
- **Reticulin** of the liver **is intact** indicating the preservation of the liver architecture.
- **No malignant potential**

Dysplastic nodules

This topic was not mentioned in the lecture

- Larger than 1 mm
- Cirrhotic liver
- Atypical features, pleomorphism and crowding
- High proliferative activity
- High or low dysplasia
- Precancerous (monoclonal, +ve gene mutations
- Types:

1. Small – cell dysplastic nodules
2. Large – cell dysplastic nodules

Hepatocellular carcinoma

- 5.4% of all cancers
- The Incidence can vary between countries:

<5/100000 population in N&S America
N& central Europe
Australia

15/100000 population in Mediterranean
36/100000 population in Korea, Taiwan
mozambique, china

- **Blacks > white**
- **M:F ratio :**
  3:1 in low incidence areas. >60yr
  8:1 in high incidence areas. 20-40yr

Predisposing Factors

1. **Hepatitis carrier state :**
   vertical transmission of HBV from mother to child during pregnancy is strongly associated with hepatitis carrier state **(increases the risk** of HCC development by **200X).**
   cirrhosis may be absent.
   young age group (20-40yr).

2. **>85% of cases of HCC occur in countries with high rates of chronic HBV infection** (Endemic areas have higher incidence of HCC).

3. Cirrhosis

In western countries cirrhosis is present in almost all cases (85-90% of cases) :

>60yr

Associated with HCV & alcoholism.

4. Aflatoxins

5. Hereditary tyrosinemia (in 40% of cases)

6. Hereditary hemochromatosis

Pathogenesis

1. Repeated cycles of cell death & regeneration
   Caused by : HBC, HCV → will results in gene mutations, Genomic instability.

2. Viral integration:
   HBV DNA intergration which leads to clonal expansion.

3. HBV DNA intergration which leads to genomic instability
   not limited to integration site but also to other areas where it can affect tumors suppressor genes or oncogenes.

4. HBV :
   Viral DNA integration results in the formation of → X-protein which leads to transactivation of viral & cellular promoters, which results in :
   a) Activation of oncogenes.
   b) Inhibition of apoptosis.

5. Aflatoxins (fungus Aspirgillus flavus) → mutation of p53 (tumor suppressor gene).

6. Cirrhosis, may be caused by :

   HCV

   Alcohol Hemochromatosis

   Tyrosinemia (40% of pts. Develop HCC despite adequate dietary control).

Morphology

1. HCC (**Hepatocelluar carcinoma,** from hepatic origin)
2. CC (Cholangiocarcinoma, from endothelium of the duct vessels).
3. Mixed

- Unifocal
- Multfiocal
- Diffusely infiltrative

- Vascular invasion is common in all types.
- **Regarding grade it varies :**
  - Well ---- Anaplastic

Hepatocellular Carcinoma Pathoma High-Yield Notes:

A. Malignant tumor of hepatocytes.

B. Risk factors include:

1. Chronic hepatitis (e.g., HBV and HCV)
2. Cirrhosis (e.g., alcohol, nonalcoholic fatty liver disease, hemochromatosis,Wilson disease, and A1AT deficiency)
3. Aflatoxins derived from Aspergillus (induce p53 mutations).

C. Increased risk for Budd-Chiari syndrome

1. Liver infarction secondary to hepatic vein obstruction
2. Presents with painful hepatomegaly and ascites

D. **Tumors are often detected late because symptoms are masked by cirrhosis; poor prognosis.**

E. **Serum tumor marker is alpha-fetoprotein.**

Fibrolamellar carcinoma

- 20-40 yr. M=F
- No relation to HBV or cirrhosis
- Better prognosis **(because of larg amounts of fibrosis that slightly prevent local invasive).**
- single hard scirrhous tumor **(because of fibrosis).**
- Cholangiocarcinoma are desmoplastic

Metastasis

Vascular – lungs, bones, adrenals, brain, in 50% of cholagiocarcinoma

- C/P **(Clinical Presentation)**
  abd. Pain, malaise, wt. loss **(unintended)**
  increase α-feto protein in 60 – 75% of pts.

- α-feto protein increases also with:

  1-yolk sac tumor

  2- cirrhosis,

  3-massive liver necrosis,

  4-chronic hepatitis,

  5-normal pregnancy,

  6-fetal distress or death

  7- fetal neural tube defect.

Prognosis

- Death within 7 -10 months
- **Causes:**
  1. Cachexia **(sever physical wasting and malnutrition)**
  2. GI bleeding
  3. Liver failure
  4. Tumor rupture and hemorrhage

THE END

رسالة من الفريق العلمي:

For any feedback, scan the code or click on it.

Corrections from previous versions:

Versions	Slide # and Place of Error	Before Correction	After Correction
V0 → V1			
V1 → V2			