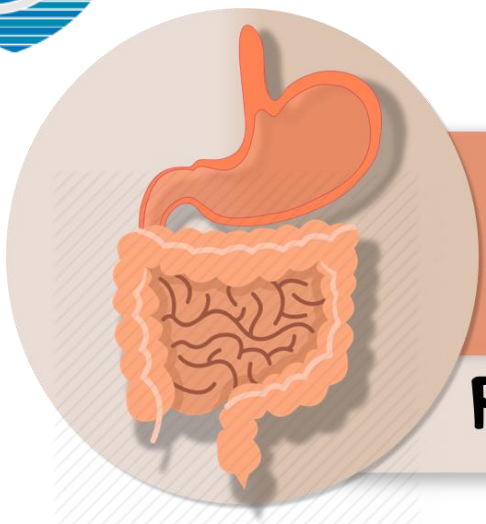

GI system

Pathology

◉ Sheet ○ Slide

Number:	5
Done by:	Hanan Jamal
Corrected by:	-
Doctor:	Maha Shomaf

PELIOSIS HEPATIS

It is a vascular condition characterised by sinusoidal dilatation.
Causes:

1. Anabolic steroids: Peliosis hepatis is caused by certain drugs but most commonly it is caused by steroids overdose (particularly in men for body building).
2. Oral contraceptives: in females.
3. Danazol.

Pathogenesis: unknown
It is an asymptomatic disease however in such patients the condition is constant due to complications which are intra abdominal hemorrhage and liver failure.
It is a reversible process once the underlying cause is treated or stopped.

LIVER TUMORS:

Liver is one of the most common sites to have secondaries (metastasized cancer from the GIT as primary site precisely the colon).
Benign: the most common benign tumor of the liver is cavernous hemangioma.
*cavernous:large dilated blood vessels.
Usually <2cm
Subcapsular: present on the surface of the liver.
The blood vessels are filled with blood and distended therefore may rupture; it might be spontaneous, or for example during biopsy or fine needle aspiration that might cause rupture leading to intraperitoneal hemorrhage.
Liver cell adenoma: is another type of benign tumor if the liver associated with the hepatocytes.
*usually when we have adenomas in the liver, they occur due to hormonal uptake.
Therefore patients are usually young females (Hx of oral contraceptive intake).
Males might also have it but it would be due to steroid uptake since this tumor is hormone dependent.
In this type of tumor, the adenoma may rupture especially during pregnancy causing severe intraperitoneal hemorrhage.
Rarely may contain hepatocellular carcinoma (HCC).

Misdiagnosis of HCC because some malignant tumors could be well differentiated resembling benign tumors.

LIVER NODULES:

Focal nodular hyperplasia.
* If a patient's radiograph shows a nodule, it is significant to know the nature of the nodule since it might be secondary, primary, or just a mass mimicking tumors.
Well demarcated hyperplastic hepatocytes with central scar
Non-cirrhotic liver: those nodules are similar to those of cirrhosis but they are localized and not diffused like in cirrhosis.
Not neoplasm but nodular regeneration (the rest of the liver is normal)
Local vascular injury (vascular ischemia for example).
Female of reproductive age
No risk of malignancy
20% of cases have cavernous hemangioma

MACROREGENERATIVE NODULES:

Cirrhotic liver: the continuous degeneration- regeneration can create certain nodules that become prominent.
Larger than cirrhotic nodules
No atypical features under the microscope
Reticulin is intact
No malignant potential

DYSPLASTIC NODULES

*usually it is difficult to diagnosis, hard to differentiate it from HCC/liver cancer)
Larger than 1 mm
Cirrhotic liver
Atypical features; pleomorphism and crowding
High proliferative activity
High or low dysplasia
precancerous (monoclonal which is an important feature of malignancy, +ve gene mutations seen in malignancies)
Types: small cell dysplastic nodules, large cell dysplastic nodules

HEPATOCELLULAR CARCINOMA:

• 5.4% of all cancers
• Incidence:
<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 increases the risk 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
3-Cirrhosis
In western countries cirrhosis is
present in 85-90% of cases
>60yr
HCV & alcoholism
4. Aflatoxins (Toxin produced by fungi in hot environments which usually is associated with gene mutation without cirrhosis; occurs in early age).
5. Hereditary tyrosinemia (in 40% of
cases)
6. Hereditary hemochromatosis

Pathogenesis

1. Repeated cycles of cell death & regeneration
HBC, HCV, gene mutations, Genomic instability (due to presence of a foreign genome within the DNA which causes instability)

*genomic instability is associated with high risk in developing mutation in other genes such as oncogenes and tumor suppressor genes.
2. Viral integration
HBV DNA integration which leads to clonal expansion (encoding proteins such as enzymes, receptors, growth factor leading to malignancies).
3. HBV DNA integration which leads to genomic instability not limited to integration site.
4. HBV
X-protein which leads to transactivation of viral & cellular promoters,
Activation of oncogenes,
Inhibition of apoptosis
5. Aflatoxins (fungus Aspergillus flavus)
mutation of p53
6. Cirrhosis
HCV
Alcohol
Hemochromatosis
Tyrosinemia (40% of pts. Develop HCC despite adequate dietary control)

Morphology

1. HCC: of hepatocyte origin
2. Collagen carcinoma (CC)which is an adenoma: epithelium of the bile duct origin
3. Mixed: both HCC and CC
• Unifocal : single tumor
• Multfiocal: multiple tumor
*The single tumor in the liver are usually primary and the multiple tumors are usually metastatic, however this is not a certain information.
• Diffusely infiltrative
Vascular invasion is common in all types (snake like fashion).
• differentiation varies between well differentiated and poorly differentiated
*Well differentiated might be difficult to diagnosis especially in cirrhosis background since it is disarranged.
well ----Anaplastic
• Fibrolamellar carcinoma
20-40 yr. M=F
No relation to HBV or cirrhosis
better prognosis

single hard scirrhous tumor
• Cholangiocarcinoma are desmoplastic (very hard due to large amount of fibrous tissue)
*pancreatic carcinomas are also desmoplastic.

Clinical presentation

abd. Pain, malaise, wt. loss
increase α-feto protein in 60 – 75% of patients.
*α-feto protein is a protein produced by the liver which is the tumor antigen.
• α-feto protein increases also with:
1-yolk sac tumor (germ cell) -> occurs in children
* If α-feto protein is high in adults and indicates malignancy then it would be HCC but never 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.
*A test can be performed to see the levels of α-fetoprotein, if it was in thousands then this indicates that its due to a tumor, however if its in hundreds then its by other causes.
Prognosis
• Death within 7 -10 months
• Causes:
1-Cachexia
2-GI bleeding
3-Liver failure
4-Tumor rupture and hemorrhage