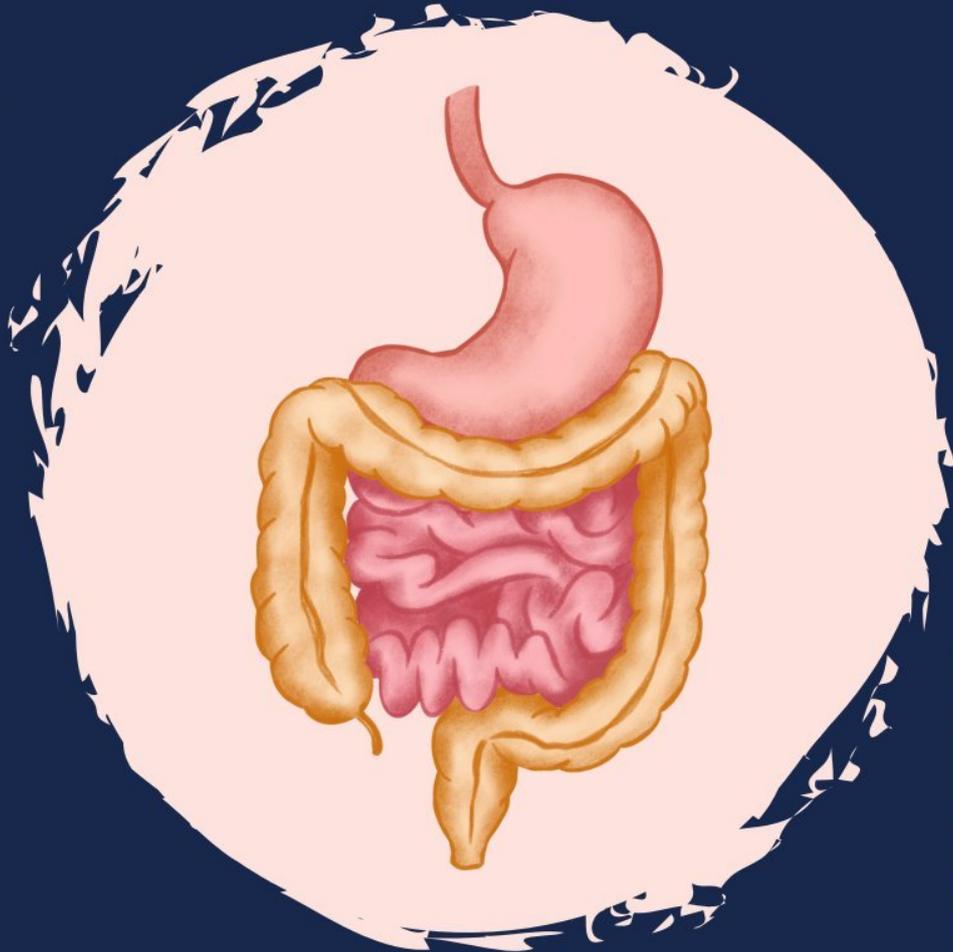


Level (3) - Semester (5)

# PATHOLOGY

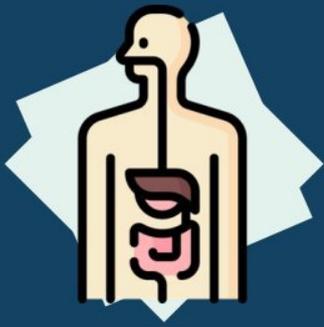


SCAN ME

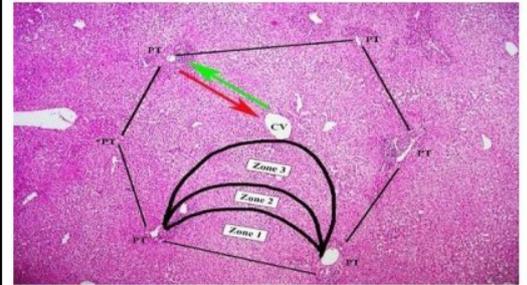
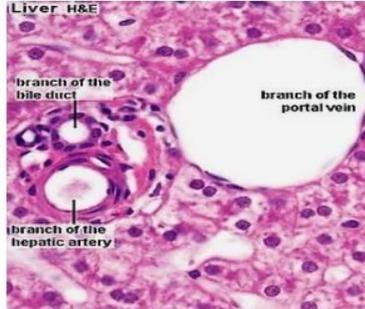
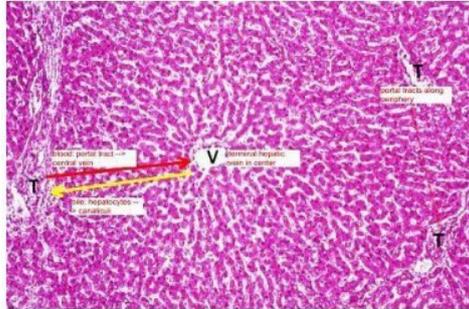
## GIT - Lecture (5)

Hepatitis (Acute & Chronic)

**DR M. YUSUF**



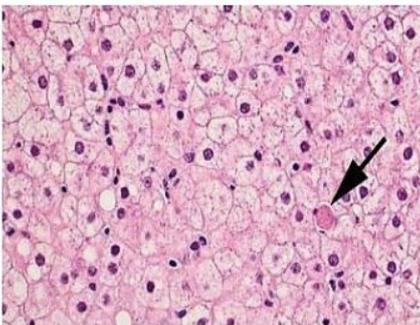
# HEPATITIS



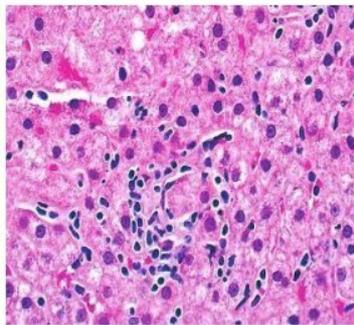
**Liver Histology**

## PATTERNS OF LIVER CELL INJURY

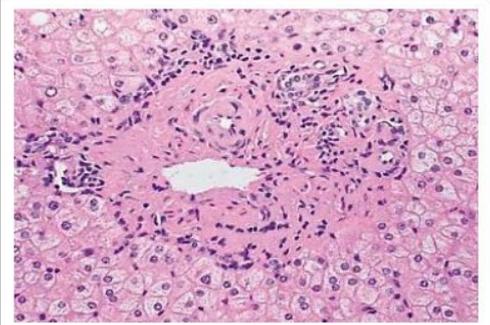
<b>Apoptosis</b>	<ul style="list-style-type: none"> <li>Single cell death</li> <li>Cytoplasm is granular &amp; dense pyknotic nucleus.</li> <li>Councilman body</li> </ul>
<b>Interface hepatitis</b>	<ul style="list-style-type: none"> <li>Apoptosis of peripheral hepatocytes</li> <li>Resulting in irregular limiting plate</li> </ul>
<b>Focal necrosis</b>	<ul style="list-style-type: none"> <li>Necrosis of small groups of hepatocytes within the lobule.</li> <li>With lymphocytes &amp; may be neutrophils</li> </ul>
<b>Confluent necrosis</b>	<ul style="list-style-type: none"> <li>Large groups of hepatocytes resulting in collapse of framework</li> </ul>



**Apoptosis of hepatocyte**



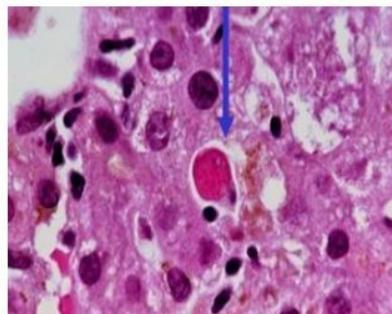
**Focal necrosis**



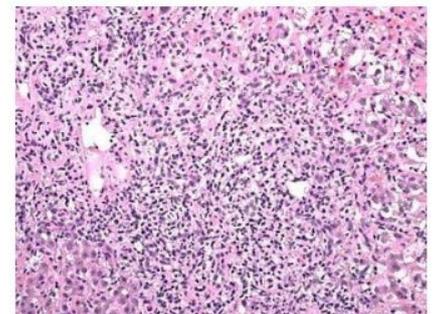
**Interface hepatitis**



**acute viral hepatitis with focal necrosis**



**Apoptosis (councilman bodies)**



**Confluent necrosis**





## ACUTE HEPATITIS

### Definition

- Acute parenchymal liver damage lasts **less** than 6 months.

### CAUSES

<b>Viral hepatitis</b>	
<b>Infective</b>	<ol style="list-style-type: none"> <li>Hepatitis A, E (&gt;95% of viral cause) B, C.</li> <li>Herpes simplex</li> <li>Cytomegalovirus</li> <li>Epstein-Barr.</li> </ol>
<b>Non infective</b>	<ol style="list-style-type: none"> <li>Alcohol</li> <li><b>Toxins:</b> Amanita toxin in mushrooms - carbon tetrachloride</li> <li><b>Drugs:</b> Paracetamol</li> <li><b>Metabolic diseases:</b> Wilson's disease</li> </ol>

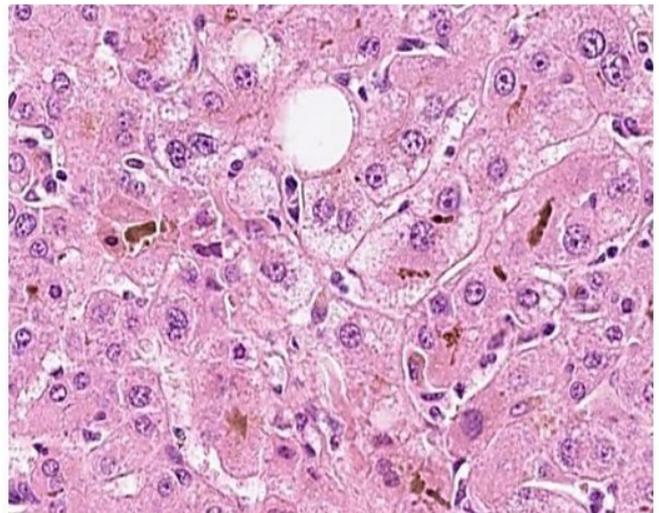
### PATHOGENESIS

#### ① Liver cell injury is caused by:

- Direct cytopathic effect of the virus.
- Cell mediated immune response (by cytotoxic T lymphocytes).

#### ② Systemic manifestations are caused by:

- Circulating immune complexes



Acute cholestasis in acute hepatitis





## PATHOLOGY

## ① CLASSIC ACUTE VIRAL HEPATITIS WITH FOCAL NECROSIS:

N/E	<ul style="list-style-type: none"> <li>▪ <b>Size:</b> The liver is slightly enlarged</li> <li>▪ <b>Colour:</b> Shows <b>red</b> (congested) areas &amp; <b>green</b> (cholestatic) areas</li> </ul>
M/E	<p>1) <b>Hepatocytes show:</b></p> <ol style="list-style-type: none"> <li>a. Ballooning degeneration.</li> <li>b. Feathery degeneration (foamy cytoplasm with droplets of bile).</li> <li>c. Fatty change (with HCV).</li> <li>d. Apoptosis and councilman bodies.</li> <li>e. Focal spotty necrosis.</li> <li>f. Many hepatocytes are normal.</li> </ol> <p>2) <b>Inflammatory cells (macrophages &amp; lymphocytes):</b></p> <ul style="list-style-type: none"> <li>• In portal tracts &amp; around the necrotic foci.</li> </ul> <p>3) <b>Acute cholestasis:</b></p> <ul style="list-style-type: none"> <li>• <b>Due to obstruction of bile canaliculi by:</b> <ul style="list-style-type: none"> <li>- Swollen hepatocytes</li> <li>- Cholangiolitis of bile canaliculi.</li> </ul> </li> </ul>

## ② ACUTE VIRAL HEPATITIS WITH DIFFUSE NECROSIS (FULMINANT VIRAL HEPATITIS).

N/E	<ul style="list-style-type: none"> <li>▪ <b>Size:</b> The liver is small</li> <li>▪ <b>Colour:</b> Show <b>yellowish</b> areas admixed with foci of <b>hemorrhage</b></li> </ul>
M/E	<ul style="list-style-type: none"> <li>▪ <b>Extensive</b> hepatocytes <b>necrosis</b> with <b>few</b> surviving ones.</li> <li>▪ <b>Destroyed</b> reticulin framework.</li> <li>▪ <b>Moderate</b> inflammatory cellular infiltrate in the <b>portal tracts</b>.</li> </ul>

## FATE

## ① Typical (classical) cases:

- a) Complete recovery after 4 weeks to 4 months.
- b) Chronicity that may progress to cirrhosis
- c) Chronic carriers in whom the viral antigens are excess in hepatocytes and may reach the blood.

## ② Cases with fulminant hepatitis:

- a) **Death** rate 70-90 %.
- b) Post-necrotic scarring may result in:
  - Portal hypertension** - **Liver failure** - **Increased risk of hepatocellular carcinoma.**





Ballooning degeneration in acute hepatitis	Acute viral hepatitis with diffuse necrosis (fulminant viral hepatitis)

## CHRONIC HEPATITIS

### Definition

- **The continuity of hepatitis symptomatic or biochemical** (elevated enzymes or presence of viral antigens) **without steady improvement for more than 6 months.**

### CLINICAL COURSE

- ☑ Patient may show **spontaneous remission** course or may show **progressive liver cell failure**.

### MICROSCOPIC EXAM

N/E	<ul style="list-style-type: none"> <li>▪ <b>Mild hepatomegaly</b></li> </ul>
M/E	<ol style="list-style-type: none"> <li><b>1) Portal tract inflammation:</b> <ol style="list-style-type: none"> <li>Expansion of portal tracts by inflammatory cells <b>mainly</b> lymphocytes &amp; macrophages with <b>occasional</b> plasma cells.</li> <li>Lymphoid follicle formation (<b>with HCV</b>).</li> <li>Bile duct inflammation (<b>with HCV</b>).</li> </ol> </li> <li><b>2) Interface hepatitis</b></li> <li><b>3) Focal (spotty) necrosis</b></li> <li><b>4) Confluent necrosis connecting areas between adjacent lobules.</b> <ol style="list-style-type: none"> <li>Von Kupffer cell hyperplasia.</li> <li>Fatty change (<b>with HCV</b>).</li> <li>Hepatocytes dysplasia (<b>precancerous</b>)</li> </ol> </li> <li><b>5) Fibrosis:</b> Periportal fibrosis or bridging fibrosis.</li> <li><b>6) Cirrhosis</b></li> </ol>





### FINDINGS INDICATING THE ETIOLOGY

☑ **HBV:**

- **Ground glass hepatocytes:** HBV-infected hepatocytes are packed with spheres & tubules of HBs Ag producing a finely granular eosinophilic cytoplasm.

☑ **HCV:**

- a) Focal macrovesicular
- b) Steatosis (fatty change)
- c) Predominantly sinusoidal lymphocytic infiltrate, often with lymphoid follicles

☑ **Autoimmune hepatitis:**

- a) Portal plasma cell rich inflammation
- b) Interface hepatitis
- c) Hepatocyte rosettes

### PROGNOSIS OF CHRONIC HEPATITIS

☑ **Depends on:**

1) **Etiology:**

- a. Viral
- b. Autoimmune
- c. Drug induced

2) **Grade of activity by assessing 4 parameters:**

- **The degree of activity is graded as (mild – moderate – marked) according to the score of these parameters.**
  - a. Portal tract inflammation
  - b. Piece meal necrosis
  - c. Spotty necrosis
  - d. Bridging necrosis.

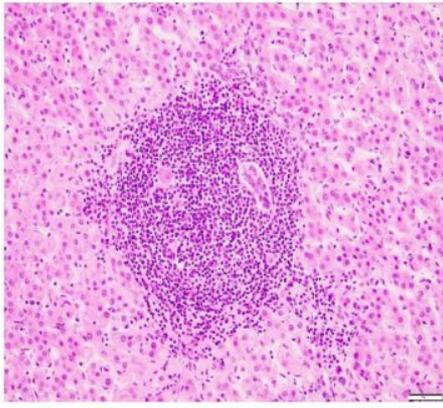
3) **Stage of fibrosis. by assessing 3 parameters:**

- **The degree of fibrosis is graded as (mild – moderate – marked)**
  - a. Portal fibrosis
  - b. Bridging fibrosis
  - c. Cirrhosis.

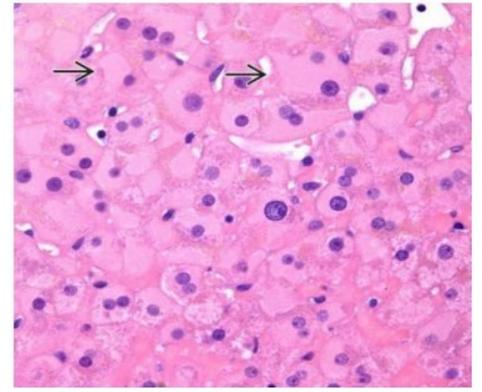
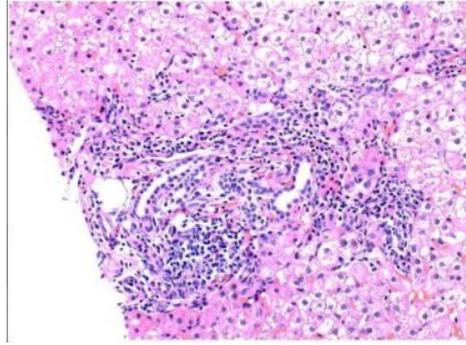
### COMPLICATIONS OF CHRONIC HEPATITIS

- ① Post hepatic cirrhosis.
- ② Liver cell failure.
- ③ Hepatocellular carcinoma.

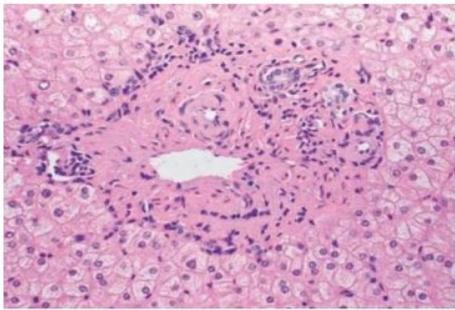




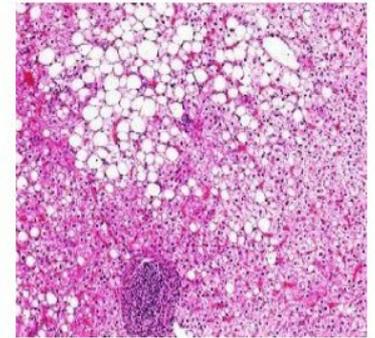
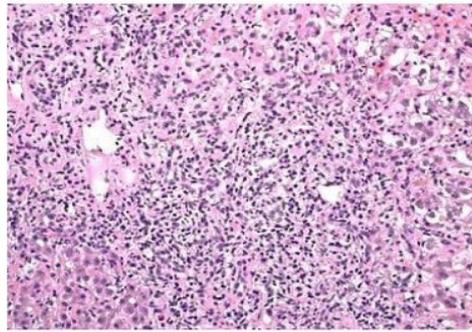
Portal tract inflammation in chronic hepatitis



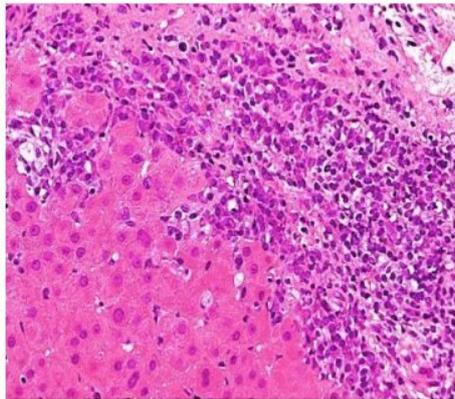
Ground glass hepatocytes in HBV



Interface hepatitis and confluent necrosis in chronic hepatitis



HCV chronic hepatitis



Autoimmune hepatitis

