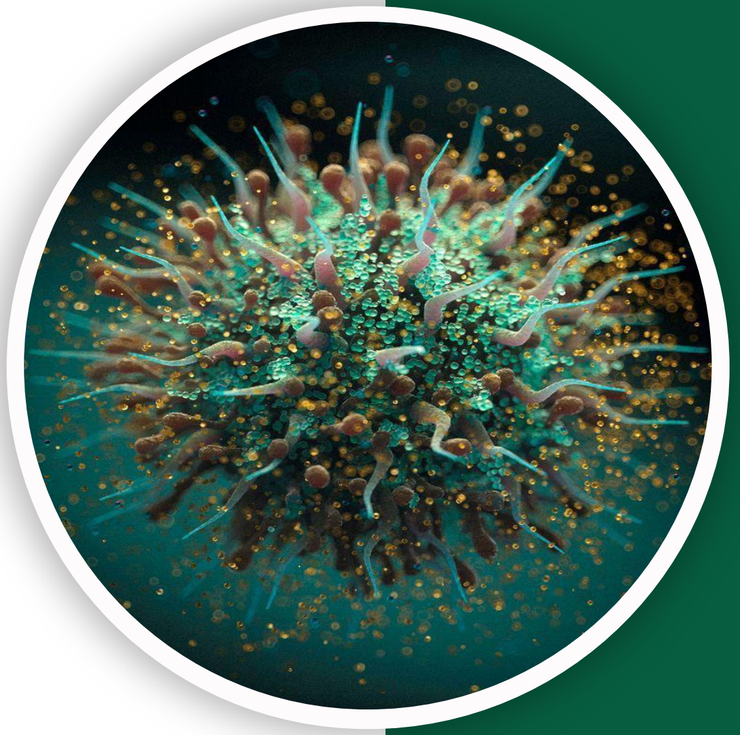
بسم الله الرحمـٰن الرحيم

(وَفَوْقَ كُلِّ ذِي عِلْمٍ عَلِيمٌ)

GIS Pathology | FINAL 1

# Liver



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Reviewed by : Rawan Okour

# Liver


- We are going to study the normal liver , whether it’s gross or microscopic  appearance and the functions.
- We will discuss different diseases , which they are related to diagnosis and the manifestation (presentation) of the patient is related to loss of these functions.
- Some of these might be  primary of manifestation of liver function , however liver is responsible for other functions so the patient can be present with other manifestation away from the organ itself, so keep in mind that these manifestation can vary in severity of which can be different. Sometimes the manifestation can be non-specific. Always keep in mind the possibility of these manifestations related to liver disease .
- It's very important when discussing diseases or changes that occur in the liver to already know what is the normal liver so you can differentiate the changes.

- The doctor means that liver diseases can cause different manifestations depending on which liver function is affected.
- Some symptoms are directly related to the liver itself, while others appear in different parts of the body because the liver has many important functions.
- The severity of symptoms can vary, and sometimes the manifestations are non-specific, such as fatigue, nausea, or weight loss.
- So, when evaluating a patient, always consider that different symptoms may be caused by liver disease.

# Liver

- **Function:** These functions are very important and liver disease means that these functions are lost.

1 **Metabolic :** Glucose metabolism
2 **Synthetic :** Albumin, clotting factors ,hormones, enzymes.

**3-Detoxification** of different chemicals that either produced in the liver or any chemical produced in the body : **Drug** ( that's why liver can get injury) , **hormones , NH3**

**4 Storage :** Glycogen, TG(triglyceride), Fe, Cu, vit (vitamin A).

**5 Excretory :** **Bile** production (it's a liquid that contain many different types of enzymes, fat and minerals). The bile will be excreted outside to gallbladder and then to the GI tract in order to prevent the effect of these enzymes and toxic substances to affect tissue.

"preventing toxic substances from affecting tissues" means that the liver sends these waste materials into bile so they can leave the body through the intestine instead of accumulating in tissues and causing damage.

- **Net weight :1400 – 1600gm (2.5% of body weight)**

- **Blood supply:**

**1)Portal vein: 60 - 70% ( main source of blood)**

It is important because liver should deal with all the substances that are collected from different organs to be metabolized and to be executed.

**2) Hepatic artery : 30 – 40%**

- **Microstructure: (** its important because it is designed to serve liver function )

- **1) Hexagonal** (in shape) **2) lobules** (the functional unit) **→6 acini**

Each angle contains portal tract ( contain a branch of portal vein , hepatic artery and a bile duct) , the flow of blood is from portal area to central vein.

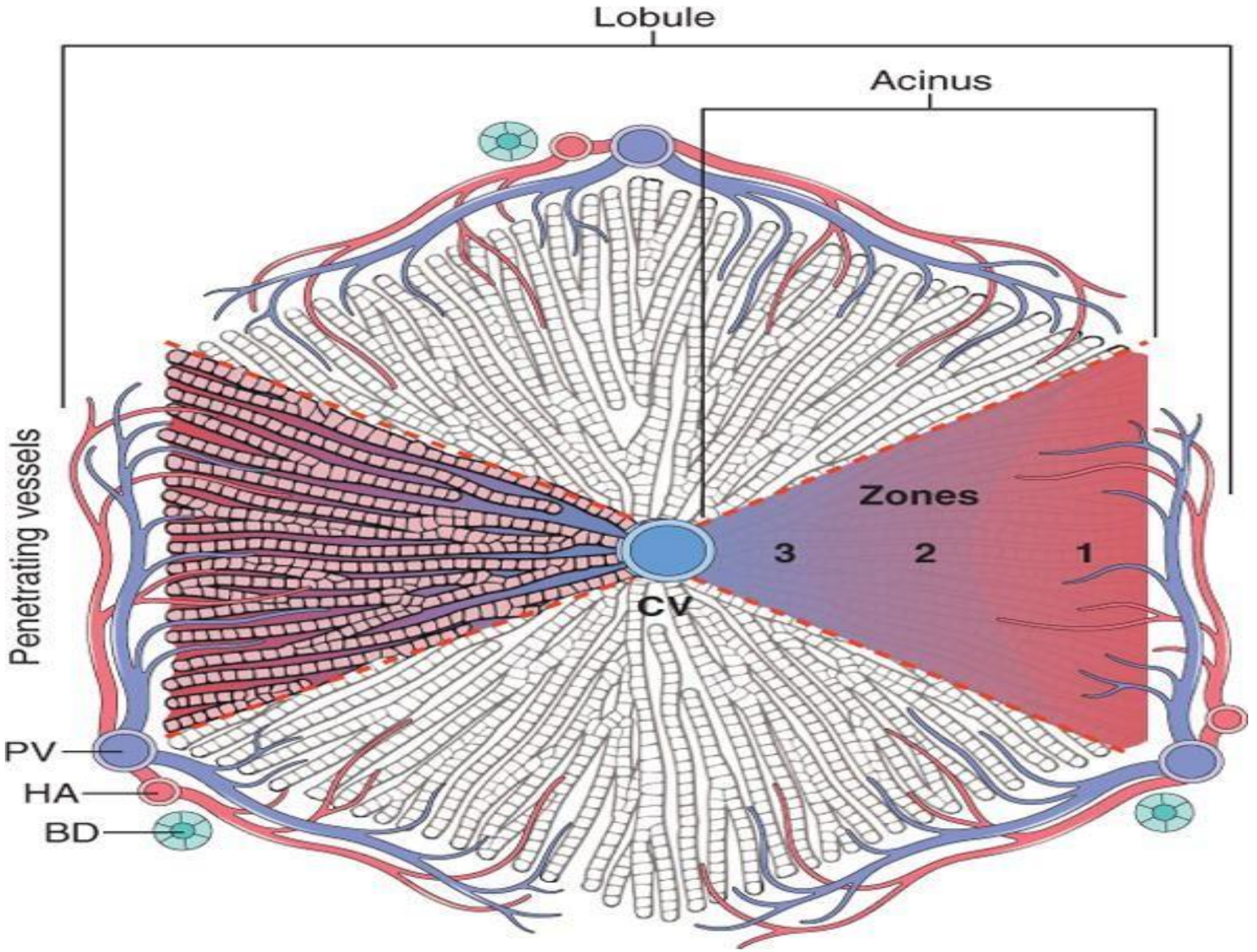
The parenchyma ( liver tissue) in between is divided into three zones. Zone one is the periportal area. The parenchyma surrounding the portal area. Zone three is around the central vein. The importance of this zoning process is that different diseases tend to start in zone one or zone three, the progression (severity) of disease all zones will be affected.

- **Acinus is divided into 3 zones:**

1) zone 1 : periportal area – closet to the vascular supply
2) zone 3 : pericentral area
3) zone 2 : intermediate area between zone 1 and zone 3

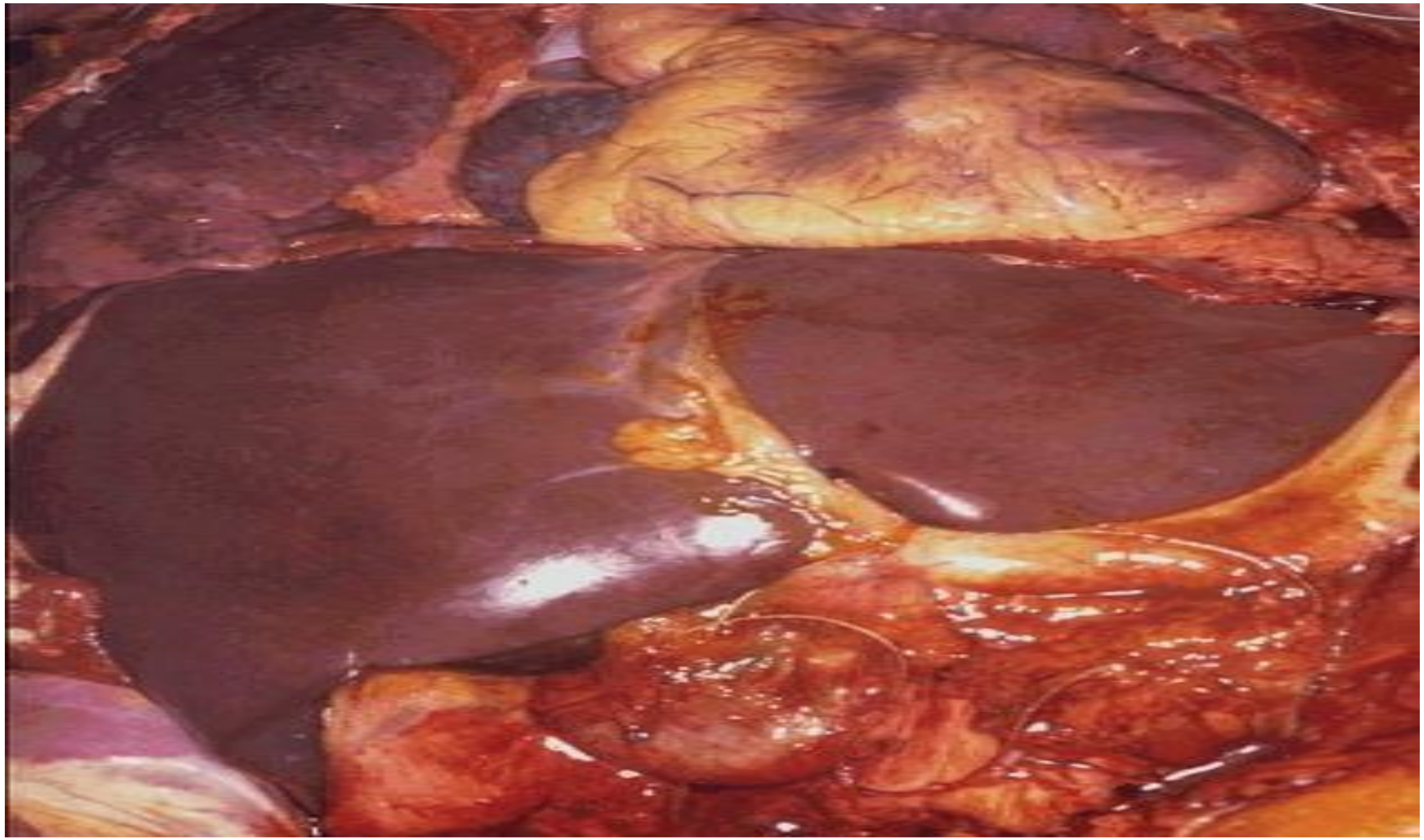
It can sometimes be useful, particularly in cases of overlapping histological changes, to identify the exact hepatic zone in which the disease process begins. Lesions that are predominantly centered around the central vein, corresponding to **zone 3**, are commonly associated with **ischemic injury**. In contrast, lesions involving **zone 1**, near the portal tract, are more suggestive of an **infectious or inflammatory process**.

Therefore, understanding the hepatic zones is important not only for interpreting the pattern of disease but also for accurate microscopic recognition and diagnosis.

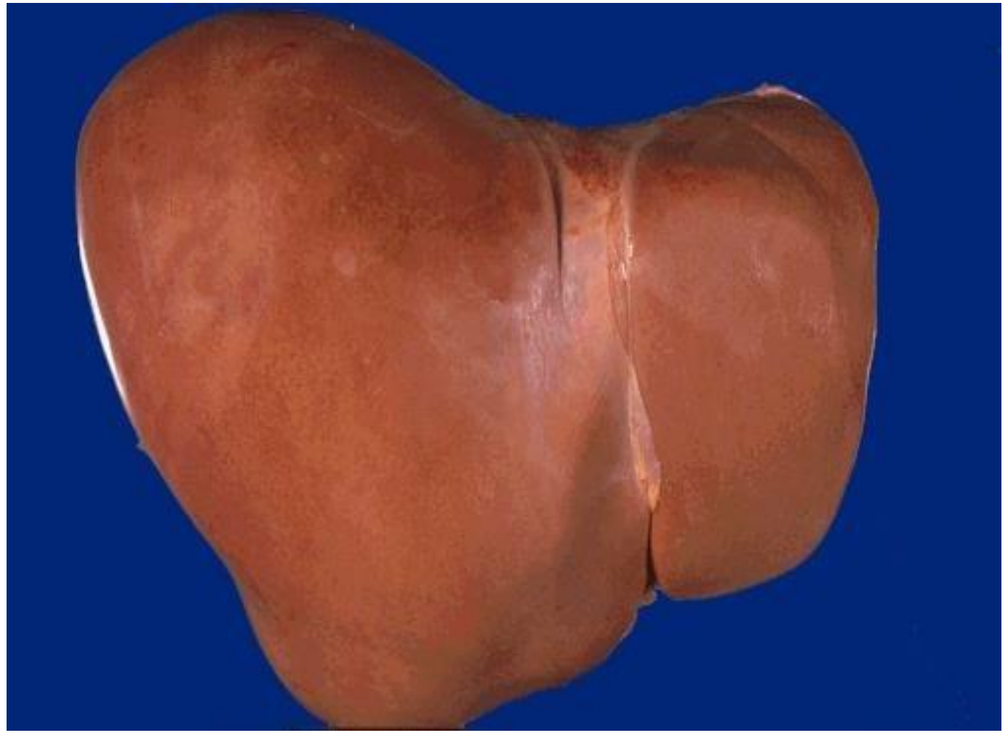


# Normal Liver

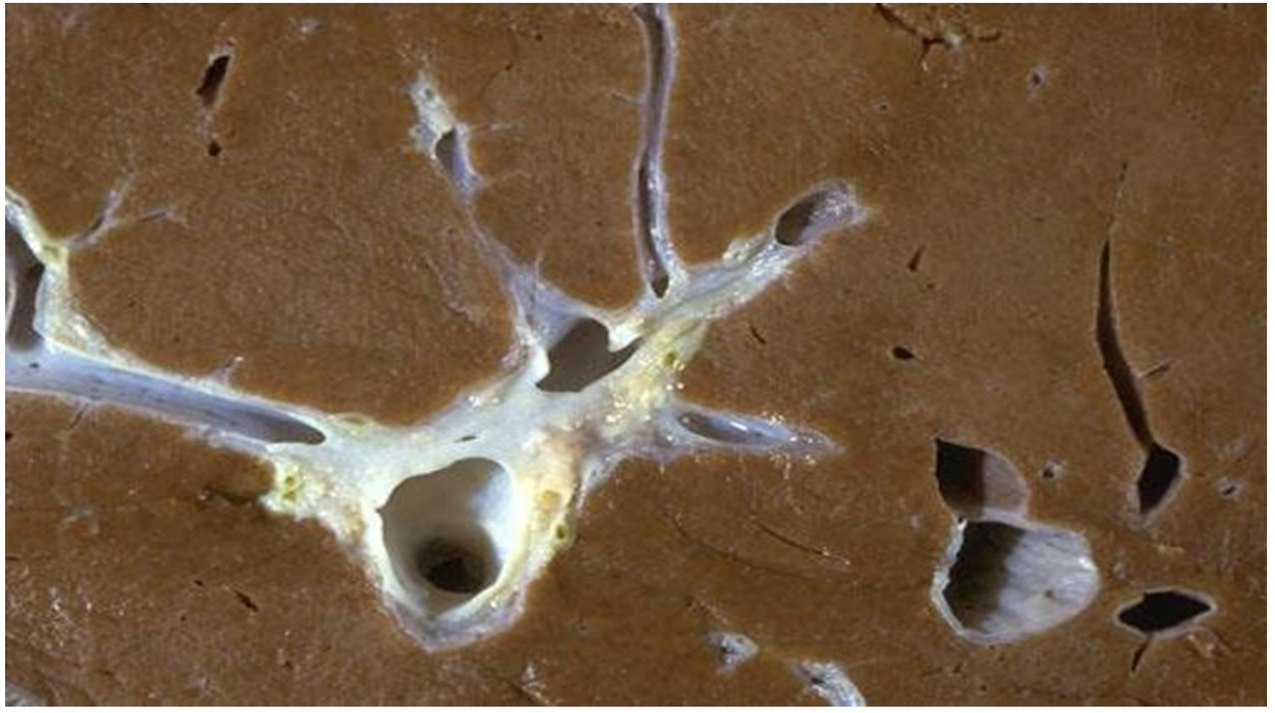
The **brown area** represents the **liver tissue**, which normally has a **smooth, shiny capsular surface** and a **soft, fleshy consistency**. In contrast, the **yellow area** represents the **surrounding adipose tissue**.



This specimen shows the liver after removal of the surrounding fat. During gross examination, several features should be assessed, including the **size**, **shape**, and **lobar anatomy** of the organ. The liver demonstrates a **homogeneous brown color** and a **smooth external surface**. These gross features are important to evaluate because deviations from the normal appearance may indicate underlying hepatic disease.



The hepatic parenchyma appears **homogeneous**, with no obvious variation in the texture or color of the tissue. In addition to the parenchyma, two main tubular structures can be identified: the **biliary tract**, which drains bile toward the gallbladder, and the **vascular structures**, which include the blood vessels supplying and draining the liver.
Together, the liver can be broadly assessed in terms of three main components: the **hepatic parenchyma**, the **biliary tract**, and the **vascular system**. Each of these components may be affected by different categories of disease. For example, diseases may primarily involve the bile ducts, producing **biliary diseases**, or they may affect the liver tissue itself, resulting in **parenchymal diseases**, which are the most common.



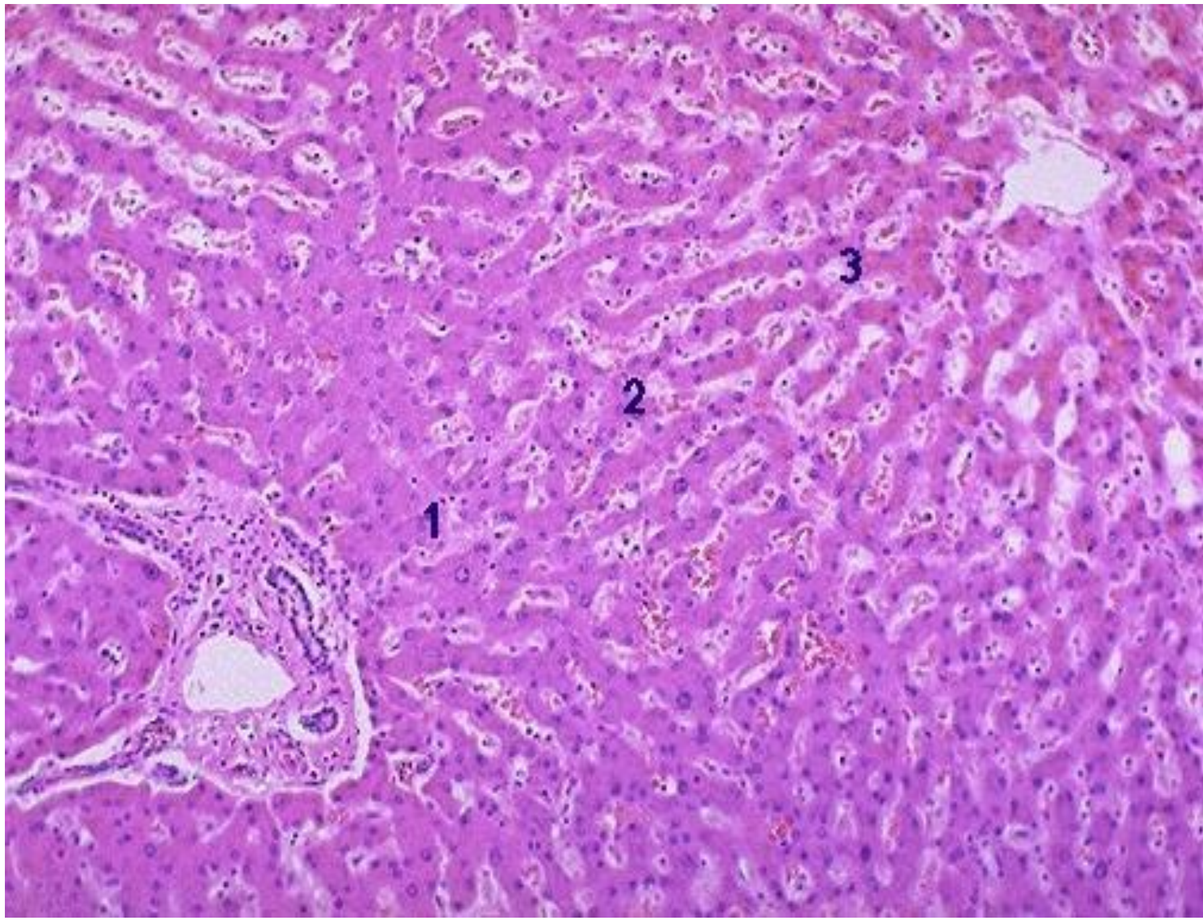
# Liver Function Tests and Their Clinical Significance

•Any alteration, deterioration, or impairment in hepatic function may result in liver dysfunction. This dysfunction can range from mild to severe and may be detected through laboratory investigations. For this reason, a group of blood tests, collectively known as **liver function tests (LFTs)**, is commonly performed in patients suspected of having liver disease.
•Liver function tests assess several important parameters, including the level of **bilirubin**, the activity of various **liver enzymes**, and the concentration of **albumin**, which is the major protein present in the blood. These tests are useful because their results can provide clues about the primary site of liver injury.
•For example, elevation of enzymes released from damaged hepatocytes, such as **ALT** and **AST**, suggests **hepatocellular or parenchymal liver disease**. In contrast, if the disease primarily involves the biliary system, there is usually a marked increase in **alkaline phosphatase (ALP)**, which is characteristically associated with cholestasis or biliary obstruction.
•Therefore, liver function tests are clinically valuable because they help identify whether the main abnormality is related to the hepatic parenchyma, the biliary system, or the liver's synthetic and excretory functions. Elevated bilirubin, for instance, indicates impaired bilirubin metabolism or excretion, reflecting hepatocellular dysfunction or biliary obstruction.

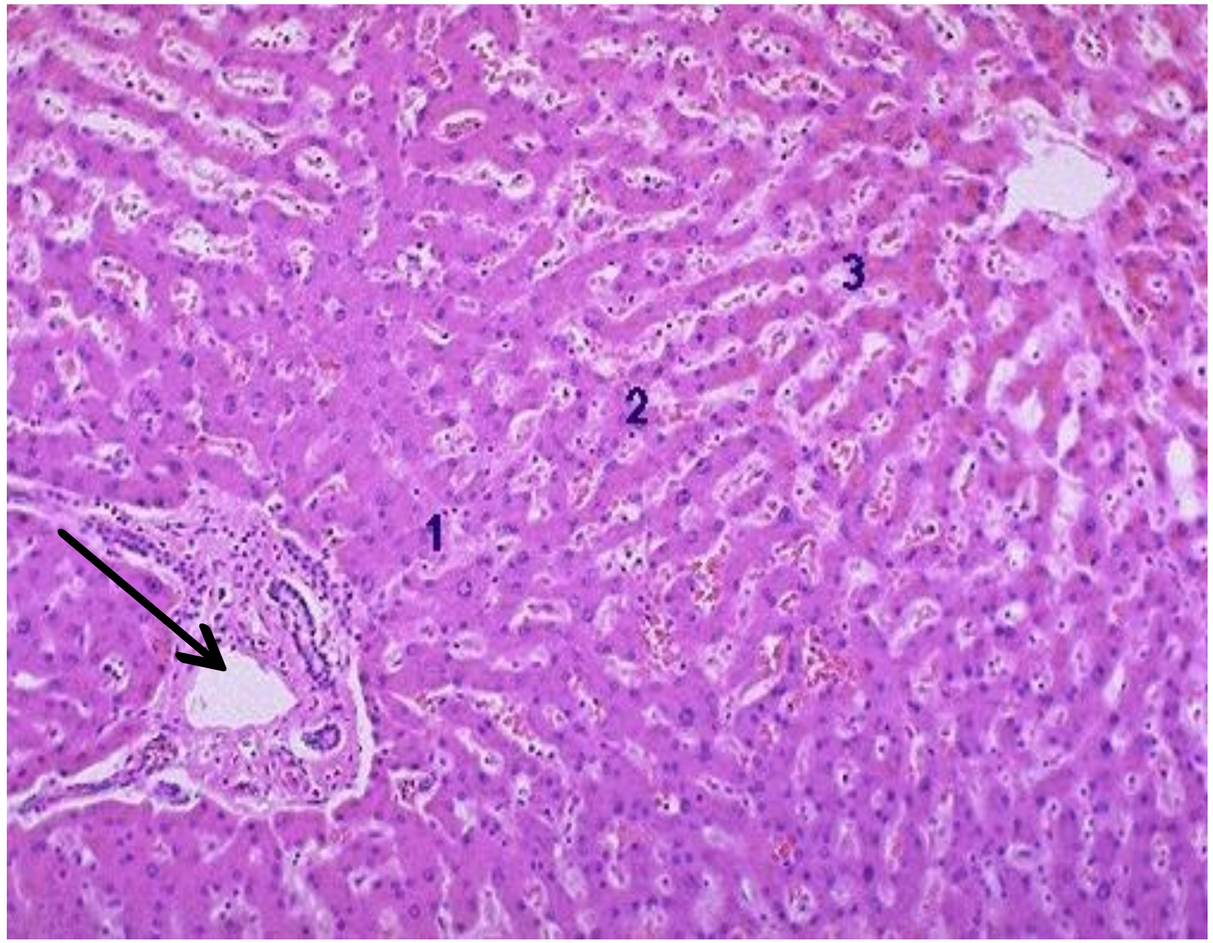
•This pic shows the **normal microscopic appearance of the liver**. The hepatic cells are well organized and arranged in plates or cords. The **hepatic parenchyma** appears organized within each hepatic lobule, and the hepatocyte plates are separated by vascular spaces called **sinusoids**.
•The thickness of the hepatocyte plates is important. In a normal liver, the plates are usually **one to two cells thick**. A mild variation may be acceptable, but a marked increase in thickness is considered abnormal and may suggest a pathological process.

- The spaces between the hepatocyte plates are called **hepatic sinusoids**. These are vascular channels lined by a very thin endothelial layer. This thin lining is important because it allows efficient exchange between the blood and hepatocytes. Substances absorbed from the gastrointestinal tract, including nutrients and potentially toxic products, reach the liver through the portal blood. The close relationship between the blood in the sinusoids and the hepatocytes allows the liver to metabolize, detoxify, store, or modify these substances effectively.

- Therefore, the thin sinusoidal barrier is essential for normal liver function. Any thickening, damage, or alteration of this barrier may interfere with the exchange between blood and hepatocytes, reducing the efficiency of hepatic function. This is why changes in sinusoidal architecture can be seen in many liver diseases.



- This pic shows the microscopic organization of the liver, including the hepatic zones and the portal area. On the left side of the image, the **portal tract** can be identified. The portal tract normally has three important structures: a branch of the **portal vein**, a branch of the **hepatic artery**, and a **bile duct**.
- The bile ducts are especially important to assess during microscopic examination because several liver diseases primarily affect the biliary system. Normally, each portal tract should contain approximately **one or two bile ducts**. Therefore, a marked decrease or absence of bile ducts may indicate a pathological process involving the biliary tree.
- The portal tract is also the only area in the normal liver where a small amount of **fibrous connective tissue** is normally present. This area appears relatively pale and hypocellular compared with the surrounding hepatic parenchyma. Because this background fibrosis is normally expected in the portal tract, it is not considered abnormal by itself. However, if the fibrous tissue becomes enlarged, extends beyond the portal tract, or bridges into the surrounding parenchyma, this suggests chronic liver injury and may indicate progression toward fibrosis or cirrhosis.




- In this image, the numbered areas represent different hepatic zones extending from the portal tract toward the central vein. Recognizing these zones is important because different patterns of liver injury may preferentially involve specific zones.

•On the other hand, the center of the hepatic lobule, or the hexagonal unit, is occupied by the **central vein**. Blood normally flows from the **portal vasculature** at the periphery of the lobule toward the **central vein**.

•The central veins drain into larger hepatic veins, which eventually empty into the **inferior vena cava**. Around the central vein, there is normally only a very thin layer of fibrous tissue. Any increase in this pericentral fibrous tissue may suggest a vascular-related liver disease **(or chronic injury affecting the central venous/outflow region)**.
•These features are important to recognize and evaluate during microscopic examination because they help in identifying the pattern of disease and reaching the correct diagnosis.

•The hepatocytes appear relatively uniform in size and shape. They contain abundant eosinophilic cytoplasm **(pink-staining cytoplasm due to the presence of proteins and numerous mitochondria)**, and their nuclei are usually centrally located. Any significant deviation from this normal morphology may indicate hepatocellular injury or an underlying pathological process.

The parenbchyma is organized into plates of hepatocytes

Hepatocytes are radially oriented around terminal hepatic vein ( central v.)

-Hepatocytes show only minimal variation in the overall size but nuclei may vary in size , number & ploidy esp. with advancing age

-Vascular sinusoids present bet. cords of hepatocytes

# Liver disease

- Liver injury can produce different morphological changes depending on the underlying disease. However, these changes may overlap and are often not specific to one particular condition.
- The injury may involve different components of the liver, including the hepatic parenchyma, bile ducts, or blood vessels. Therefore, to reach an accurate diagnosis, all available information must be considered. This includes the patient's clinical presentation, past medical history, laboratory results, radiologic findings, and finally, microscopic examination of the liver tissue.
- On liver examination, we try to identify the primary site of injury. If the main injury involves the hepatic parenchyma, it is considered a **parenchymal liver disease**. If the blood vessels are primarily involved, it is considered a **vascular liver disease**. If the bile ducts are affected, it is considered a **biliary disease**.
- It is important to remember that, in medicine, diagnosis should not depend on a single finding. Instead, the diagnosis is reached by collecting and correlating all possible clinical, laboratory, radiologic, and histologic data **(biopsy findings)** in order to understand the pattern of disease and reach the correct diagnosis.

# Liver biopsy

- Liver biopsy is not performed frequently in all cases of liver disease. However, it becomes necessary in certain conditions when a definite diagnosis cannot be reached by clinical evaluation, laboratory tests, or imaging alone.
- Some liver diseases may share similar features or show overlapping histologic changes. Therefore, biopsy can be important to differentiate between these conditions, especially when treatment depends on the exact diagnosis.
- In some cases, liver biopsy may also reveal a more severe or advanced pattern of injury than expected **(an exacerbated or progressive condition)**. This makes it helpful not only for diagnosis, but also for assessing the severity and stage of the disease.

# Hepatic injury

## 1 Inflammation (Hepatitis)

•Hepatitis is a **general term** and is not specific to one cause. Therefore, when hepatitis is identified, we need to determine the underlying cause.
•Hepatitis may be:
•**Acute hepatitis**, which is less common, or **chronic hepatitis**, which is more frequently encountered.
•It may be caused by different agents, including **viruses, bacteria, or parasites**.
•Microscopically, hepatitis is characterized by the presence of an **inflammatory infiltrate**. In the liver, inflammation usually starts in the **portal area**. Depending on the severity of the disease and the underlying cause, the inflammation may extend from the portal area into the hepatic parenchyma.
•When inflammation extends into the parenchyma, this indicates a more significant injury rather than a mild process. This pattern can help assess the severity of hepatitis, such as in cases of viral hepatitis.
•In addition, hepatocyte injury can vary in severity, ranging from **mild hepatocellular damage** to **severe hepatocyte injury**.

# 2. Ballooning degeneration :

- Mild hepatocellular injury may appear as **ballooning degeneration**, a reversible change in which hepatocytes become swollen due to intracellular water accumulation. The term *ballooning* describes the enlarged, pale appearance of the affected cells, caused by hydropic change and disruption of the normal cytoplasmic structure.

- **-irregularly clumped cytoplasm showing large, clear spaces.**
- **-Substances may accumulate in injured but viable hepatocytes, including fat, iron, copper, and retained biliary material.** Although some of these substances may normally be present in small amounts, their excessive accumulation indicates cellular dysfunction and may contribute to further injury.

- This occurs because abnormal accumulation can interfere with normal cellular metabolism, enzyme activity, mitochondrial function, and membrane integrity. As a result, cellular components may become damaged, leading to progression of disease.

- For example, iron is normally stored in the liver, but excessive hepatic iron deposition may cause **hemochromatosis**, which can lead to progressive liver injury, fibrosis, and eventually cirrhosis. Similarly, copper accumulation, as seen in Wilson disease, can damage hepatocytes and produce chronic liver disease.

- Bile may also accumulate within hepatocytes and canaliculi in conditions such as **cholestasis** or biliary obstruction. Bile accumulation is toxic mainly because bile salts have detergent-like effects that can injure cellular membranes. With persistent cholestasis, the hepatic parenchyma becomes affected by bile-induced toxicity, which may lead to hepatocyte injury, inflammation, fibrosis, and eventually cirrhosis.

# 3. Steatosis ( fatty change)

- This is another group of diseases. Steatosis or fatty infiltration is a descriptive term. It is not a diagnosis. Because it can be caused by many diseases. That's why when we have fatty infiltration, we must see what is the cause of fatty infiltration. If we know what are these causes. These patients should be followed up for the possibility of fatty infiltration.
- Initially, fatty infiltration is reversible. If we manage the underlying cause or exposure to a toxic substance. As in alcoholism, the fatty infiltration is reversible.
- However, with chronicity, this is not reversible. It can produce more chronic damage. Fibrosis is irreversible.
- So it is important now that these patients should be followed up.

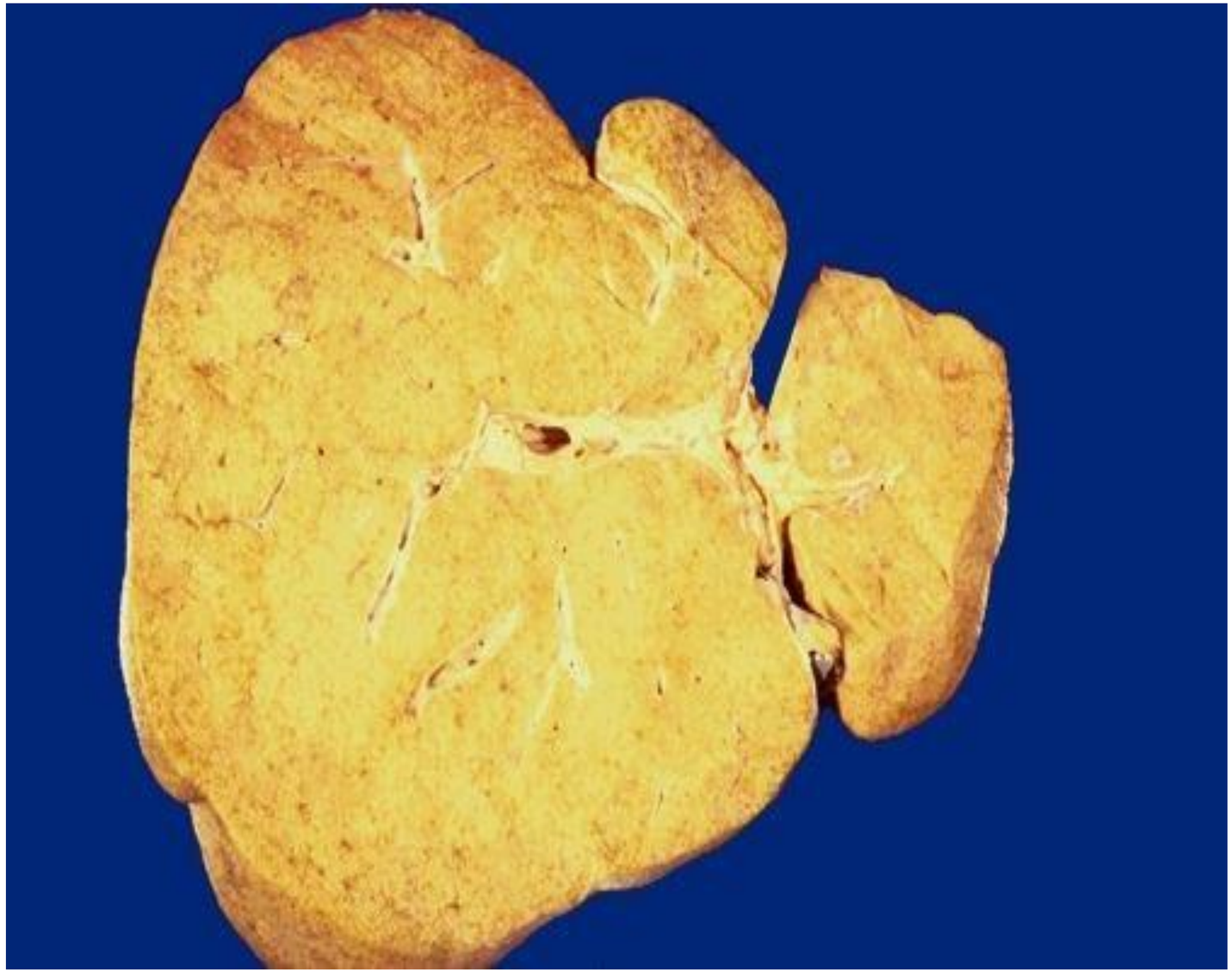
**Who can be affected?**

Microvesicular: ALD (alcoholic liver disease) ,Reye syndrome ( occur in children) , acute fatty change of pregnancy

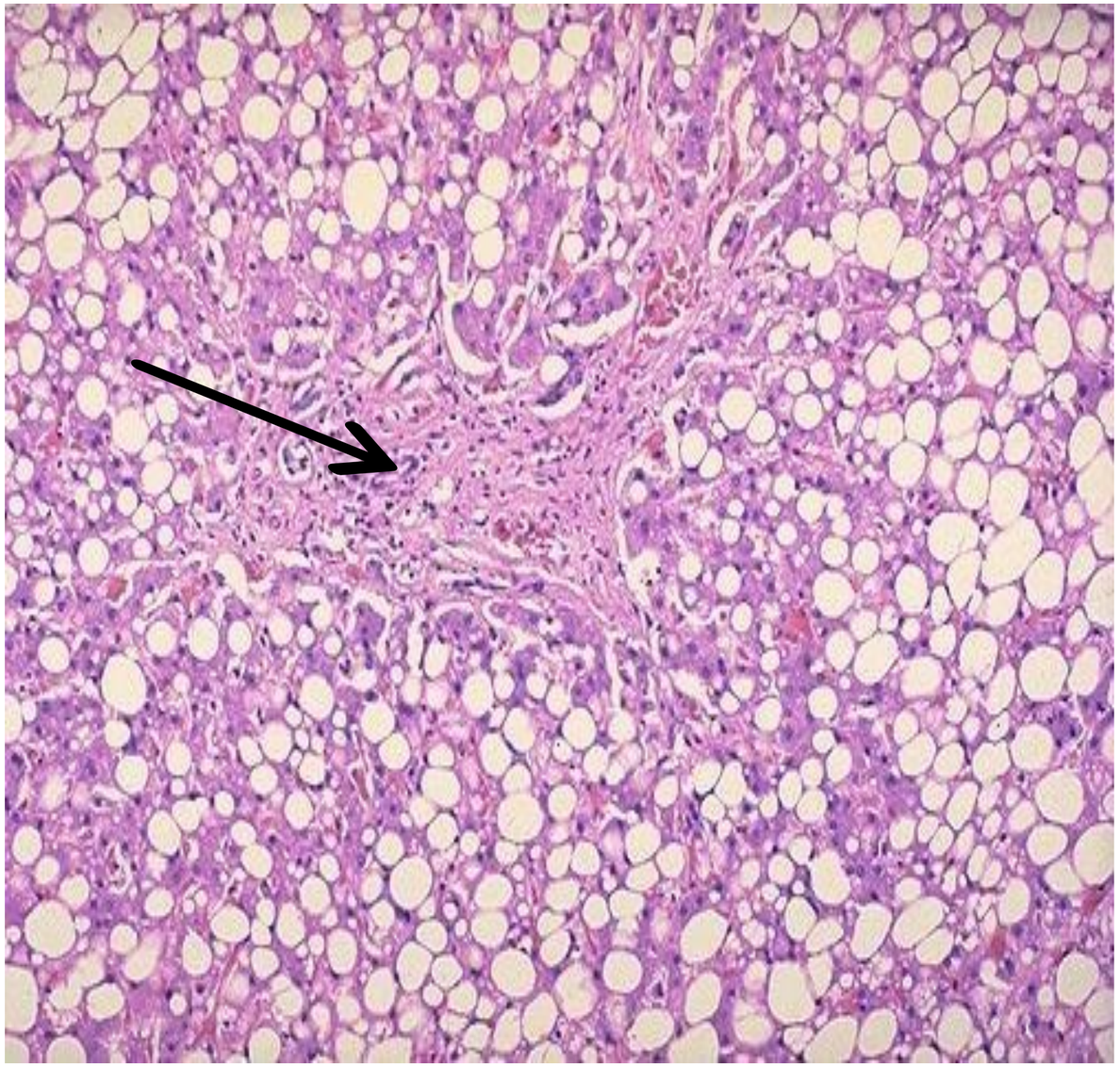
Macrovesicular: DM, obese

# fatty change

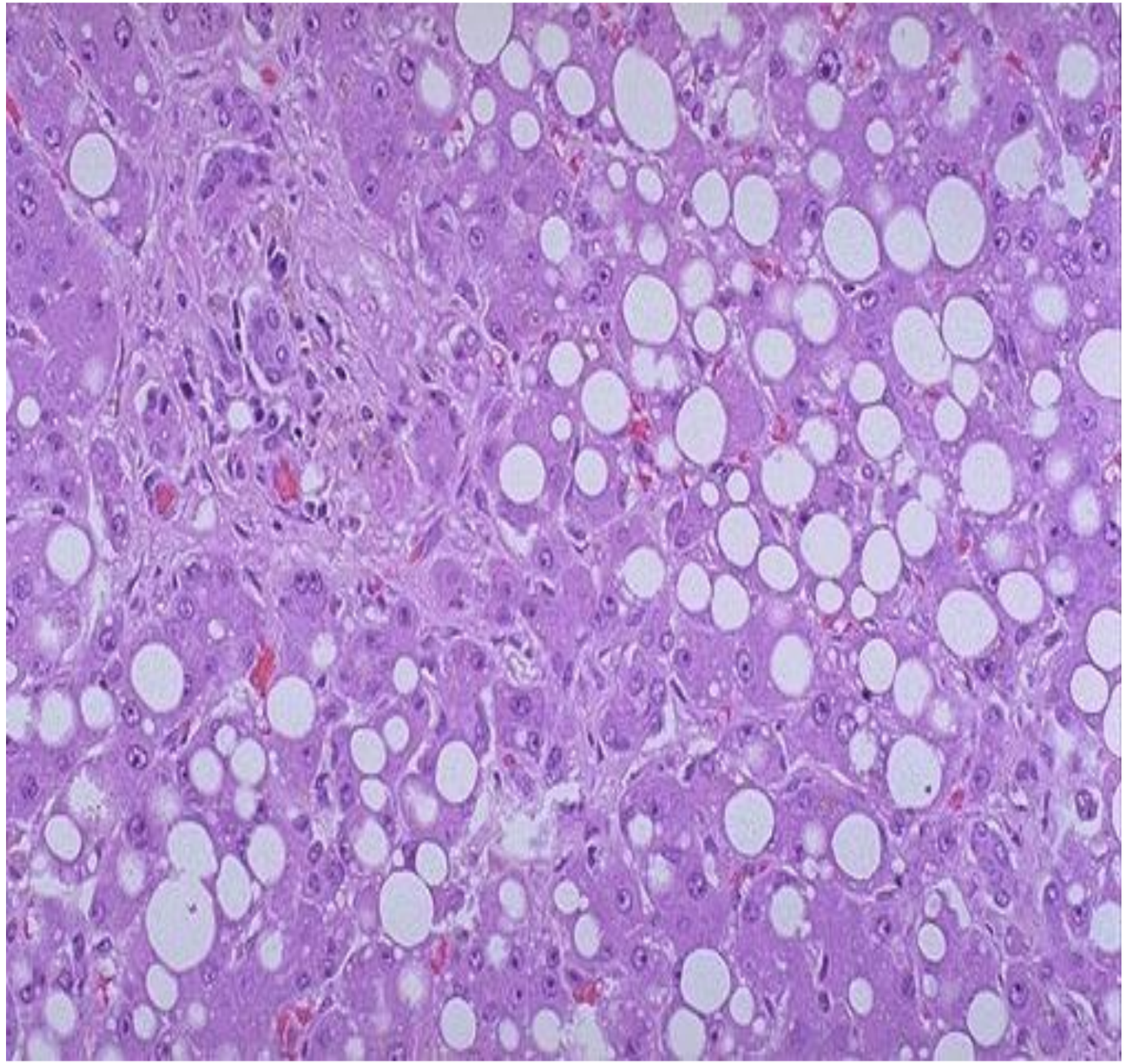
- This is the appearance of the liver with severe fatty infiltration.
- You can see that the color has changed. Now it is yellow because fat is yellow in color.
- It is greasy in consistency.
- This is severe. The whole liver is yellow.



• This figure shows the **microscopic appearance of hepatic steatosis**.
•The central area represents **fibrosis within the portal region**, while the surrounding tissue represents the **hepatic parenchyma**. Compared with normal liver parenchyma, the hepatocytes show numerous **clear cytoplasmic vacuoles**, which represent accumulated fat droplets.
•These lipid droplets may be **small or large**. In chronic or more severe cases, smaller fat droplets may coalesce to form larger vacuoles, causing marked expansion of the hepatocytes. As a result, the affected hepatocytes appear swollen, and the normal architecture of the liver parenchyma becomes distorted.
•Overall, the image demonstrates **severe fatty infiltration**, with widespread involvement of hepatocytes by intracellular lipid accumulation. Persistent severe steatosis may progress to steatohepatitis, fibrosis, and eventually cirrhosis



- This is a higher magnification of the liver.
- You can see that the fatty globules are present.
- It looks empty because during the preparation of the sections for the tissue, the fat dissolves. All reagents dissolve fat.
- The site of fat appears empty.
- In reality, there are no empty spaces in the body or tissues.



# 4. Necrosis

- Necrosis is not only to be documented whether present or not. It is also to evaluate the extent and the site of necrosis.
- Necrosis means the death of a group of cells or it can be an individual cell death ( apoptosis) . Both can occur in the liver.
- Depending on the different criteria, necrosis can be divided into different types. Why? It can be helpful in the diagnosis.

- **Depending on the type:**

1) Coagulative necrosis :around central vein Councilman bodies

Like ischemia so it is a vascular problem, It can be an obstruction or congestion or tumor pressure on the central vein. All the parenchyma surrounding the central vein will suffer from pressure and hypoxia.

2) Lytic (liquefactive) necrosis Next slide to understand more

- **2) Lytic (liquefactive) necrosis**
- **Lytic (liquefactive) necrosis** in the liver usually happens with **infections**, especially:
  - bacterial infections
  - parasites like amoeba → can produce amoebic cysts and cause liver abscesses.
- **Councilman bodies** are:
  - **apoptotic hepatocytes** (dead liver cells)
  - small, shrunken, very pink (eosinophilic) cells with tiny/dark nuclei
  - scattered in the liver parenchyma.
  - They indicate **liver cell injury**, especially in:

    -Chronic toxic injury

    -Prolonged exposure to damaging agents like drugs .

**Depending on the cause:**

Toxic or ischemic injury to the liver can cause coagulative necrosis of hepatocytes.

1)Ischemic

2)Toxic (high doses of drugs, chemicals, poison like mushroom poison)

**Depending on location:** we can evaluate the severity of the injury and the outcome of the injury.

1) Centrilobular necrosis: around the central vein

Mid zonal :

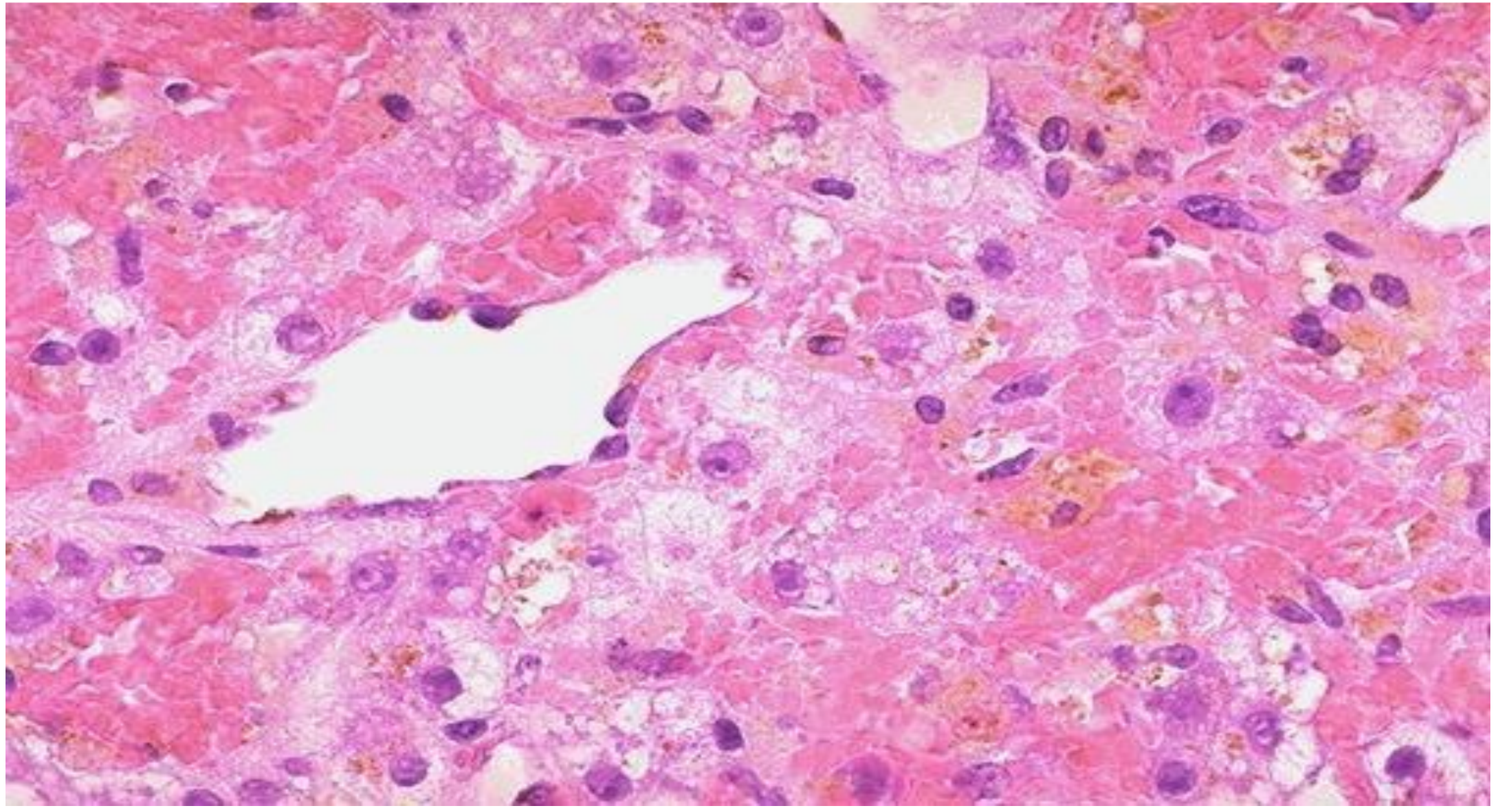
2) Periportal = interface hepatitis Focal = Piece meal necrosis

- Necrosis may become more severe and appear as **bridging necrosis**. This means that areas of hepatocyte death extend between important vascular or portal structures, such as from a **central vein to a portal tract**, from **central vein to central vein**, or from **portal tract to portal tract**.

- This finding is especially important in liver diseases that have the potential to become chronic, because necrotic areas usually heal by the formation of **fibrous scar tissue**. When fibrosis develops, it may connect two adjacent structures together, forming fibrous bridges.
- With continued injury, these fibrous septa can surround groups of regenerating hepatocytes, leading to the formation of nodules. This process is a key step in the pathogenesis of **cirrhosis**, which represents the end-stage outcome of many chronic liver diseases.
- Therefore, evaluating the extent and pattern of necrosis is clinically important. It helps predict whether the liver disease may progress to chronic hepatitis, fibrosis, and eventually cirrhosis. In general, the greater the degree of bridging necrosis, the higher the risk of progression to irreversible architectural distortion of the liver.

- 3) Diffuse: due to drug injury and exposure to toxin and this can cause liver failure

  massive & submassive necrosis ( types of diffuse)



# 5. Regeneration

- In the liver, it is privileged by having a very wide range of regeneration. Regenerative capacity is very high.
- That's why death of hepatocyte is followed by stimulation of regenerative cells to divide in order to compensate for the loss.
- This is an evidenced in disease process by increase in the mitotic activity in the cells. That's why liver failure is very difficult to reach because even a small number of cells can keep the function of the liver and can regenerate.
- That's why liver failure comes after consumption or death of most of the hepatocytes. Otherwise, the function is preserved

-evidenced by increased mitosis or cell cycle markers.

-the cells of the canal of Hering are the progenitor for hepatocytes( they get stimulated by growth factor due to severe loss of cells) & bile duct cells (oval cells ).

That’s why liver transplantation is possible

## 6. Fibrosis:

*-portal or periportal fibrosis*

-pericentral- around the central vein

-*pericellular fibrosis* or fibrous tissue may be deposited directly within the sinusoids around single or multiple hepatocytes

-bridging fibrosis

## 7. Cirrhosis ( irreversible)

Micronodular & Macronodular

## 8. Ductular proliferation

# Further explanation

**Fibrosis** represents an important stage in chronic liver injury and is considered a marker of **irreversibility**. Once fibrous tissue is deposited in the liver, it may persist and alter the normal hepatic architecture. Therefore, fibrosis is one of the key histologic features used to assess the chronicity and severity of liver disease.

Fibrosis may result from many different causes of liver injury. Evaluating the amount and distribution of fibrous tissue is clinically important because it helps determine the **stage of disease**, estimate the risk of progression, and assess the patient's response to treatment.

Fibrosis may appear in different patterns:

**Portal or periportal fibrosis** begins in or around the portal tracts. It may appear as expansion or thickening of the portal areas and can extend gradually into the surrounding hepatic parenchyma..

**Pericentral fibrosis** occurs around the central vein. It may develop in conditions associated with ischemia, toxic injury, or chronic tissue damage. Increased fibrous tissue around the central vein can disturb normal blood flow and contribute to hepatocellular dysfunction

**Perisinusoidal or pericellular fibrosis** refers to deposition of fibrous tissue within the sinusoids or around individual hepatocytes. This is especially important because it can thicken the normally thin space between hepatocytes and sinusoids. As a result, the exchange of substances between blood and hepatocytes becomes impaired, affecting both uptake of materials from the blood and secretion of hepatic products back into the circulation.

**Bridging fibrosis** occurs when fibrous septa connect two vascular or portal structures, such as portal tract to portal tract, portal tract to central vein, or central vein to central vein. This pattern is clinically significant because it indicates advanced disease and suggests progression toward **cirrhosis**, in which fibrous bands surround regenerating hepatocyte nodules and permanently distort the liver architecture.

# Hepatic Failure

Patient can present for the first time with hepatic failure with all the manifestation.

-It results when the hepatic functional capacity is almost totally lost ( 80 - 90%)

-**Causes:**

1) Massive hepatic necrosis ( acute hepatic failure = no specific symptoms)

- Fulminant viral hepatitis (suddenly can be presented by serious liver failure and extensive necrosis of the liver)
- Drugs & chemicals

  Acetominophen (precursor of paracetamol, suicidal attempt can cause liver failure, which means it's does dependent)

  Halothane ( not dose dependent, can cause liver failure at first dose)

  anti TB drugs

  CCL4(Carbon tetrachloride) poisoning

  Mushroom poisoning

2) Chronic liver disease ( more common , especially in patient with cirrhosis)

- Hepatitis is a classical inflammatory condition , in certain condition it can be associated with severe necrosis enough to cause liver failure, like hepatitis B, C,D and E.
- IF WE EXCLUDE THIS, WE SHOULD THINK OF DRUGS AND CHEMICALS
- A general role is to think of drugs if there is a liver disease, so we should always take drugs history.

There is certain hepatic failure not associated with hapatic necrosis but it's only loss of function of hepatocytes.

# 3-Hepatic dysfunction without overt cirrhosis (RARE)

-Reye's syndrome( finding in liver examination can be very minimal ( like minimal microvesicular steaosis) and mist. This is caused by malfunction occurs in children after upper respiratory tract infection.

-Tetracyline (antibiotic) toxicity **due to malfunction**

-Acute fatty liver of pregnancy (suddenly)

## Clinical features ( loss of function is represented by :)

1 Jaundice

2 Hypoalbuminemia ⟶ edema

3Hyperammonemia

4Fetor hepaticus (musty or sweet & sour)

4 Palmar erythema hyperestrogenemia

5 Spider angiomas

6 Hypogonadism & gynecomastia

# Clinical features

**1. Jaundice:**
Jaundice occurs when the liver is unable to properly excrete bilirubin into bile. As bilirubin accumulates in the blood, it becomes deposited in tissues, producing yellow discoloration of the **skin** and **sclera**.

**2. Hypoalbuminemia:**
Albumin is the major plasma protein responsible for maintaining oncotic pressure within blood vessels. In chronic liver disease, loss or dysfunction of hepatocytes reduces albumin synthesis. As a result, plasma oncotic pressure decreases, leading to fluid leakage into tissues and the development of **edema** and sometimes **ascites**.

**3. Hyperammonemia:**
The liver normally converts toxic ammonia into urea through the urea cycle. In liver failure, this detoxification process is impaired, causing ammonia to accumulate in the blood. Ammonia is toxic to the brain and may lead to neurological manifestations, including confusion, behavioral changes, and hepatic encephalopathy.

**4. Fetor hepaticus:**
Fetor hepaticus is a characteristic musty or sweet-sour odor of the breath in patients with severe liver disease. It results from volatile substances produced by intestinal bacteria during amino acid metabolism, which escape hepatic detoxification and reach the systemic circulation.

**5. Palmar erythema:**
Palmar erythema refers to reddish or dark pink discoloration of the palms. It occurs due to vasodilatation, commonly related to increased circulating estrogen levels because the diseased liver cannot metabolize estrogen efficiently.

**6. Spider angiomas:**
Spider angiomas are small vascular lesions with a central arteriole and radiating capillaries. They also result from hyperestrogenemia and are commonly seen in chronic liver disease.

**7. Hypogonadism and gynecomastia:**
In males, impaired hepatic metabolism of estrogen may cause hormonal imbalance. This can lead to **hypogonadism**, with reduced testicular function, and **gynecomastia**, which is enlargement of breast tissue.

Consequences:

1. Multiple organ failure: kidneys (because ammonia affects all mucosal sites)& lung
2. Coagulopathy → bleeding. In addition to vascular changes in the liver, it's also in the body because of the production of coagulative factors in the liver. That is why patients with cirrhosis when they bleed, they can develop acute liver failure ( especially upper GI bleeding ).

def. factors II, VII, IX, X

3. Hepatic encephalopathy due to effect of ammonia

↓ level of consciousness ( sensitivity)

Rigidity

Hyperreflexia ( developed at very late stages of cirrhosis)

EEG changes

Seizures

Asterixis

## 4-Hepatorenal syndrome

These patients are different from patients with organ failure, because organ failure is due to toxic substance

Renal failure in patients with severe liver disease with no morphologic or functional causes for renal failure

This means that if liver is corrected, kidneys will be corrected

# Massive hepatic necrosis

-Fulminant hepatic failure from the onset of symptoms to hepatic encephalopathy (within 2-3 weeks).

Sub fulminant ( within 3 months).

**Causes:**

1 Viral hepatitis 50 – 65% ( B, B-D, A,C hepatitis) ( especially in our region)

2 Drugs & chemicals 20 – 30%

3 Heat stroke

4 Hepatic vein obstruction

5 Wilson disease (due to increase copper in liver)

6 Acute fatty liver of pregnancy

7 Massive malignant infiltration

8 Reactivation of chronic HBV hepatitis on HDV superimposed infection (carriers of hepatitis B ( patients with no symptoms) however if they get a superimposed infection by hepatitis D , their condition will suddenly get worse and become with severe hepatitis with liver failure

9 Autoimmune hepatitis (rarely to have liver failure and hepatic necrosis)

# Additional Resources:

# رسالة من الفريق العلمي:

## Reference Used:

1. Robbins & Kumar Basic Pathology, 11th Edition.(Chapter 14: Liver and Gallbladder.)


اللهم صلِّ و سلم على نبينا محمد,
صلاة تخرجني بها من ظلمات الوهم
و تكرمني بنور الفهم و توضح لي ما أُشكل
عليّ حتى افهم 3<

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Corrections from previous versions:

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