GI

PATHOLOGY

#1

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- **Function:**

Liver diseases are related to storage disorders where the stored material accumulate disturbing the normal function

1-Metabolic : Glucose
2-Synthetic : factors ….. Albumin, clotting
3-Detoxification : Drugs, hormones , NH3
4-Storage : vit Glycogen, TG, Fe, Cu,
5-Excretory : Bile

- Net wt. 1400 —16OOgm (2.5% of body wt)
- Blood supply:

Portal v : 60 —70%

Hepatic a : 30 0 40%

- Microstructure
- Hexagonal lobules —•6 acini
- Acinus is divided into 3 zones:

1-Zone 1

Periportal areas —closet to the vascular supply

2-Zone 3

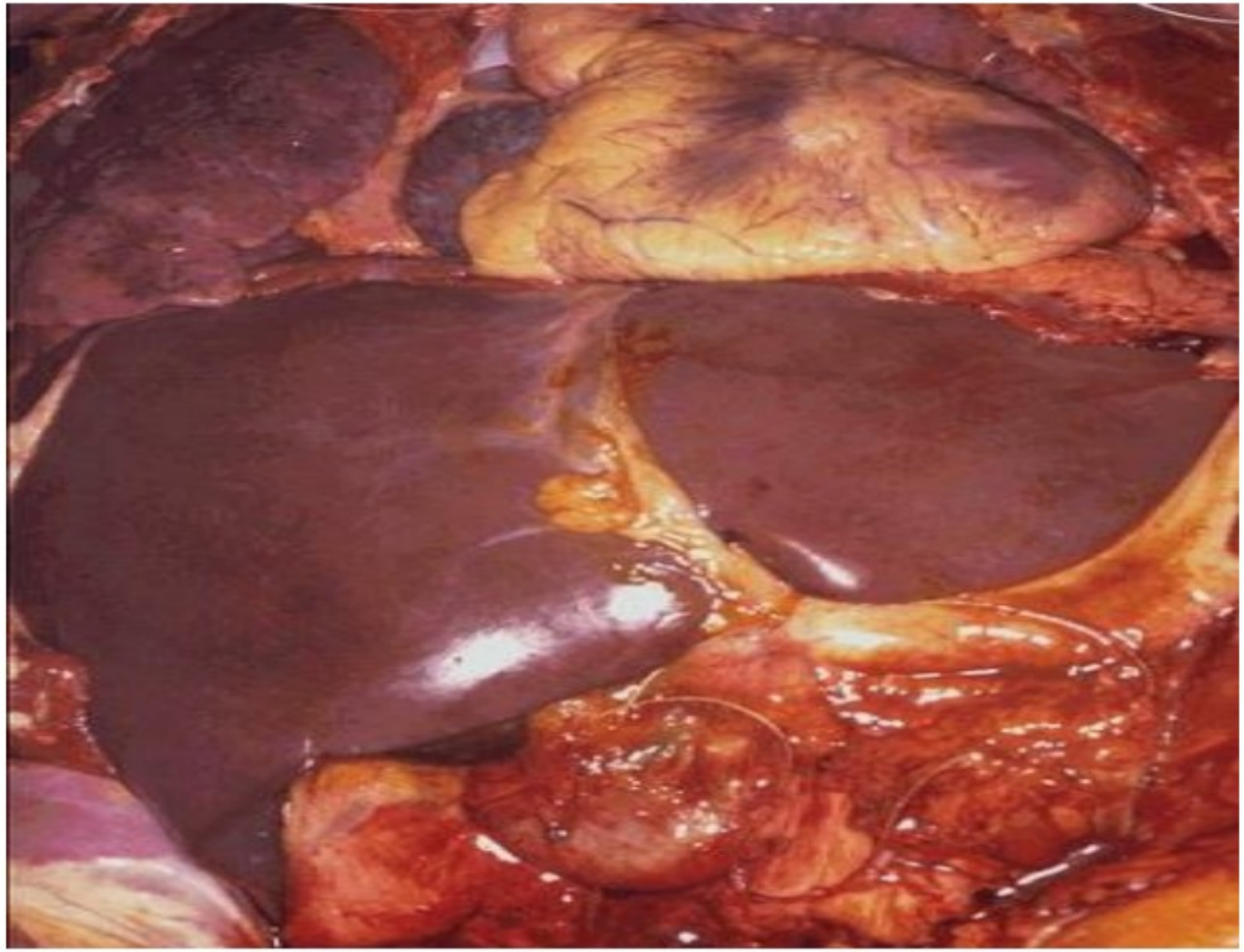
Pericentral area

3-Zone 2

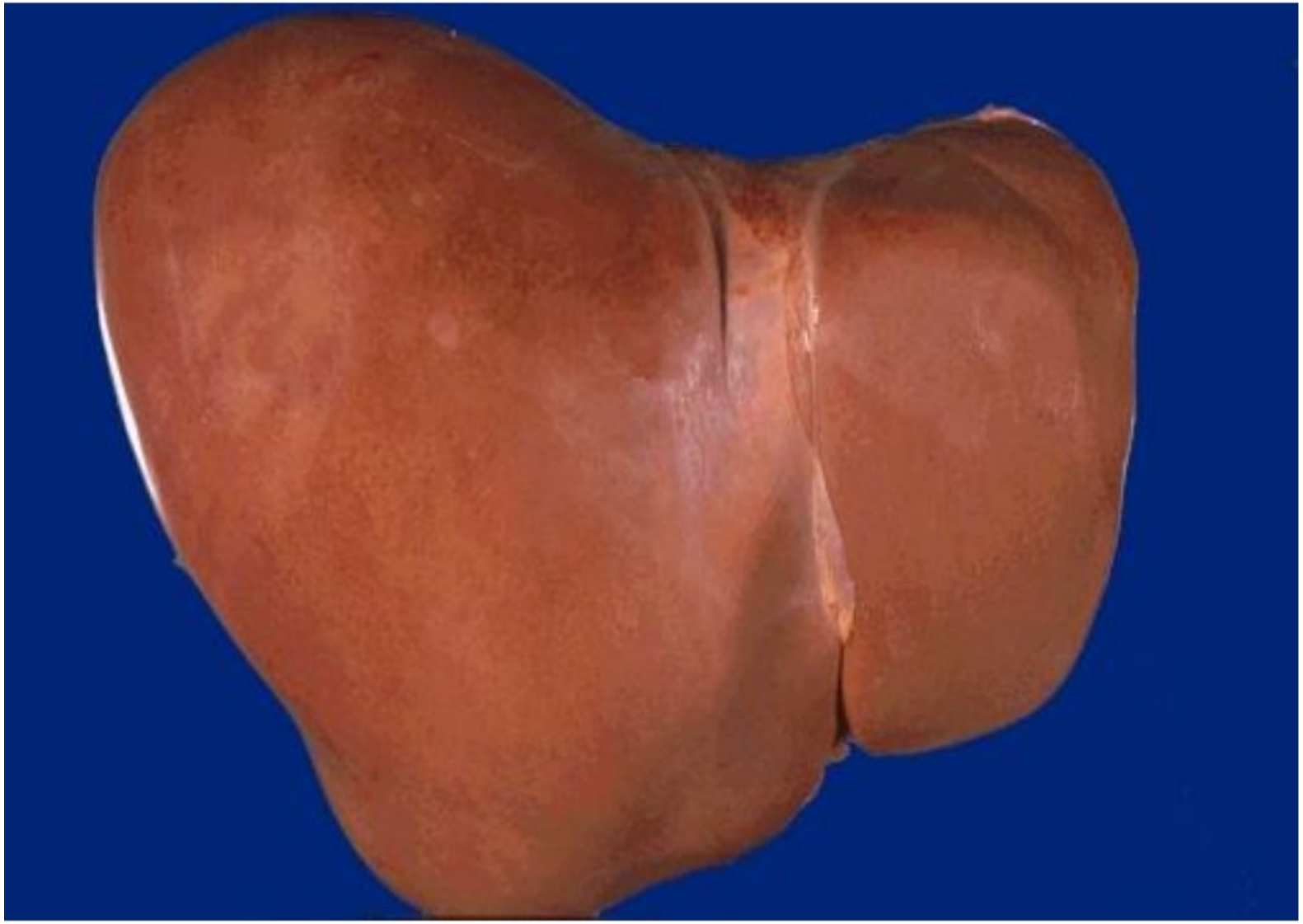
Inrermediate bet. Zone 1&2

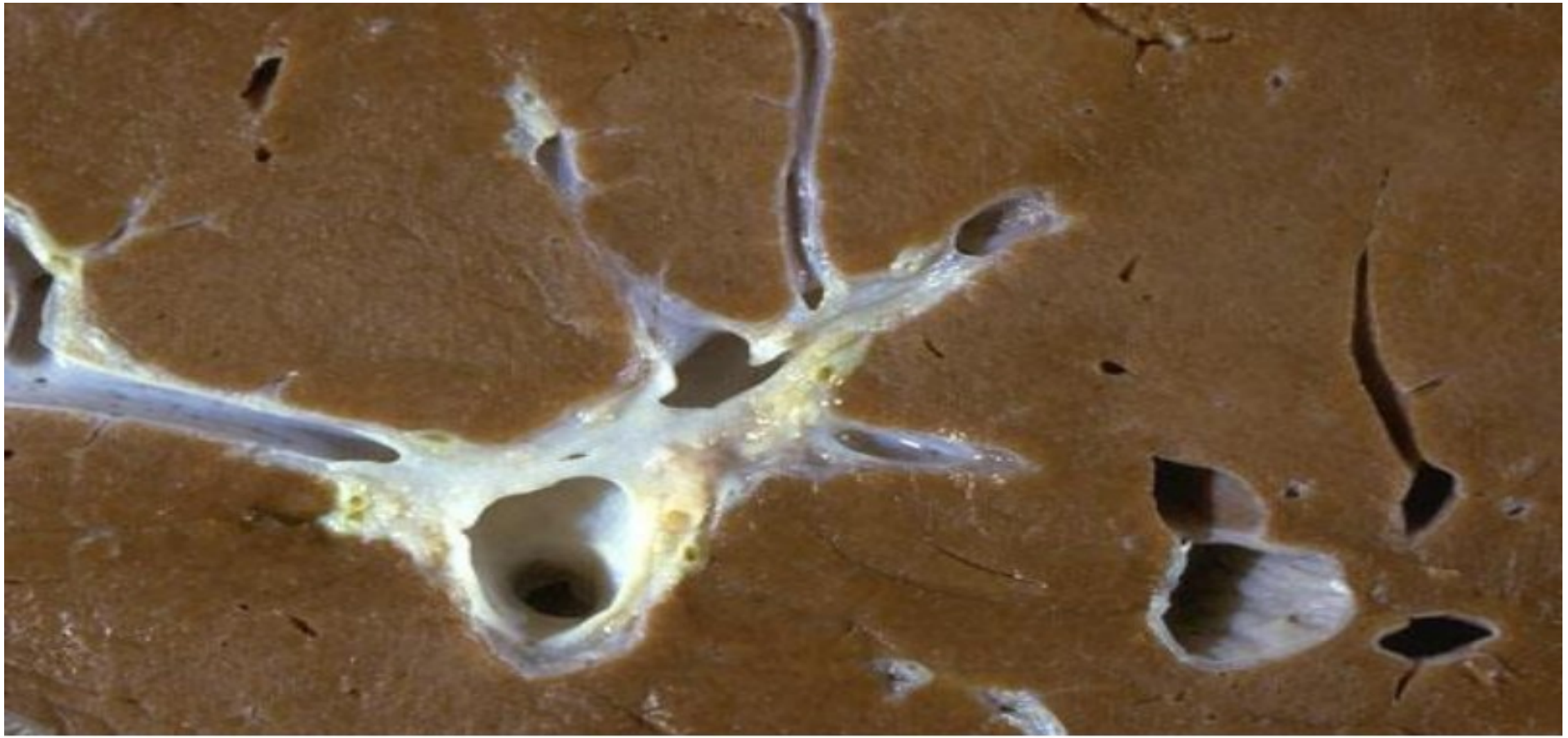
Normal Liver

Disease that may affect the liver :
1) primary: each componentof the liver can have specific type of disease (parynchema , vascular).
2)seconary : adiseaes of liver that results as a consequence of aprimary disease



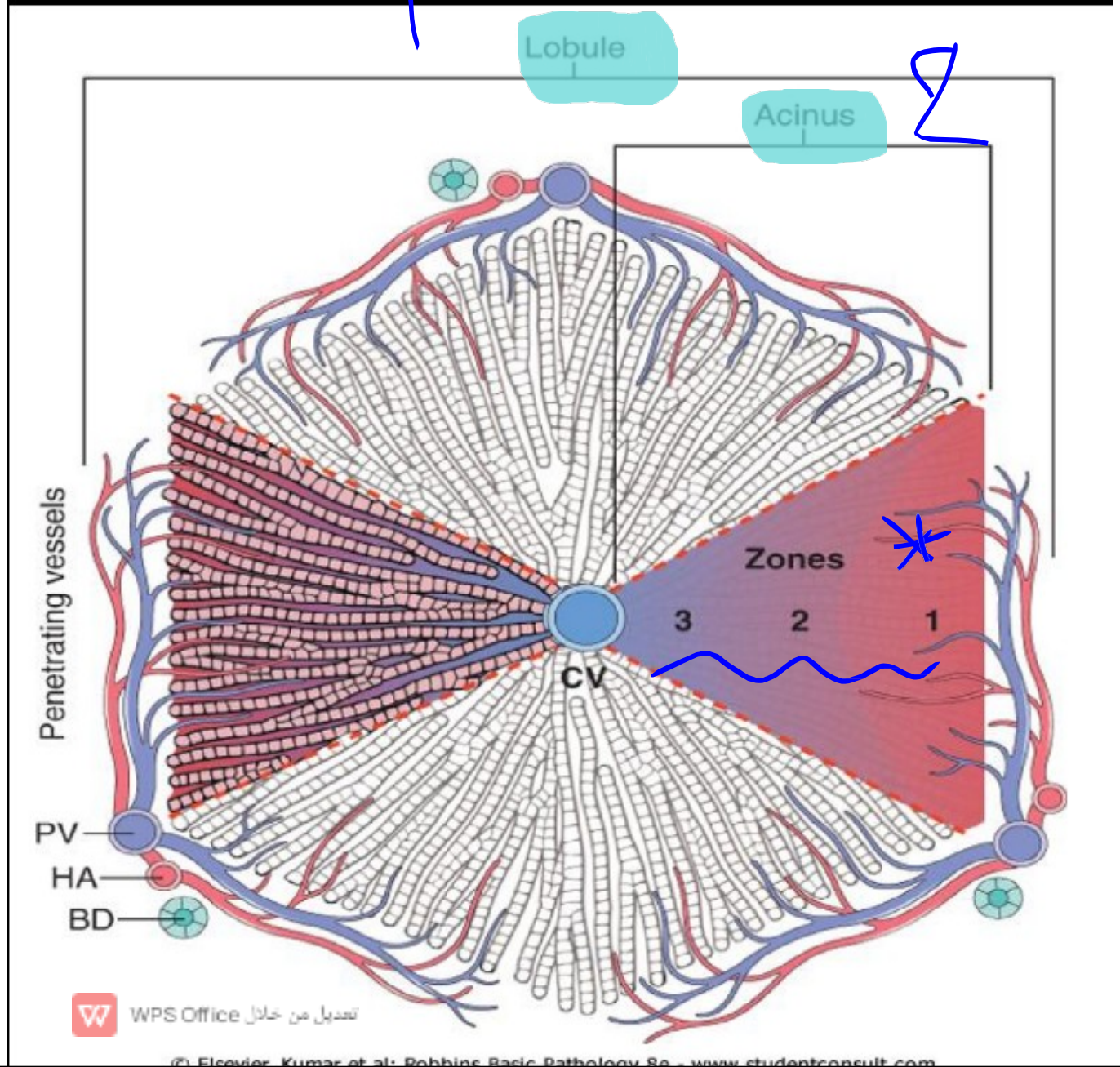
brown ,smooth and shiny (due to gisson capule) structure.. . macroscopic apperance



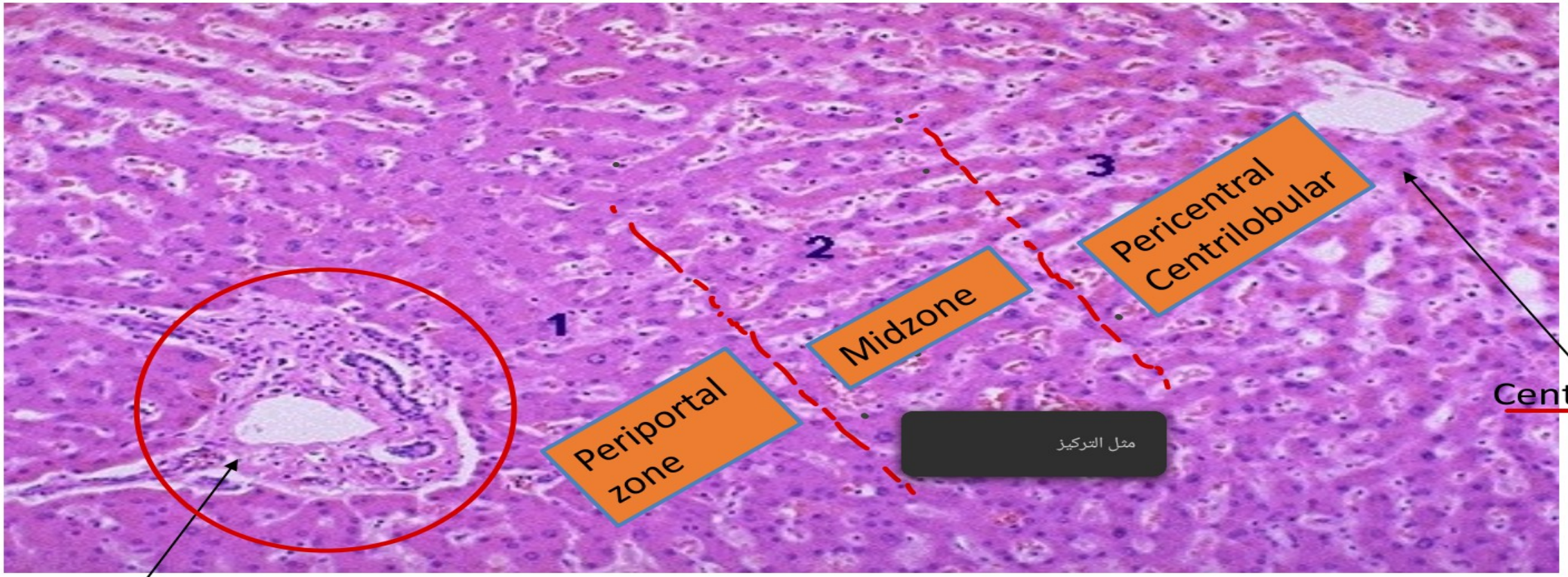


The liver can be divided into :

Zones are so important concept as each zone differ with respect to its metabolic activity .. so the susceptibility to certain forms of hepatic injury .. significant in early stage of the disease



This is a microscopic image of an acinus :-



(It is important to look at each zone as certain diseases occur in particular zones rather than the other. However, aggressive diseases affect all three zones)

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

*correct diagnosis: histo ,clinical manifestations then lab test.
*Laboratory evaluation of liver disease : measures hepatocyte integrity (AST ,ALT) .....Biliary excretory function (urine bilirubin , serum bile acids).

Hepatic injury

Usually it starts in the peripotal areas (zone1)

1-Inflammation (Hepatitis)

Can be: 1) chronic or acute 2) severe or mild ....depends on the cause and the extent of inflammation

2-Ballooning degeneration :

-irregularly clumped cytoplasm showing large, clear spaces.

-Substances may accumulate in viable hepatocytes, including fat, iron, copper, and retained biliary material

Indicates the chronicity of that disease

Hypoxia ↓O2... ↓ATP generation .... disturbances in the function of Na K pump .... ↑ Na inside hepatocyte ...osmotic pressure ...draws water toward the cell.

3-Steatosis (fatty change) *REVERSIBLE initially*

microvesicular:ALD,Reye syndrome,acute fatty change of pregnancy

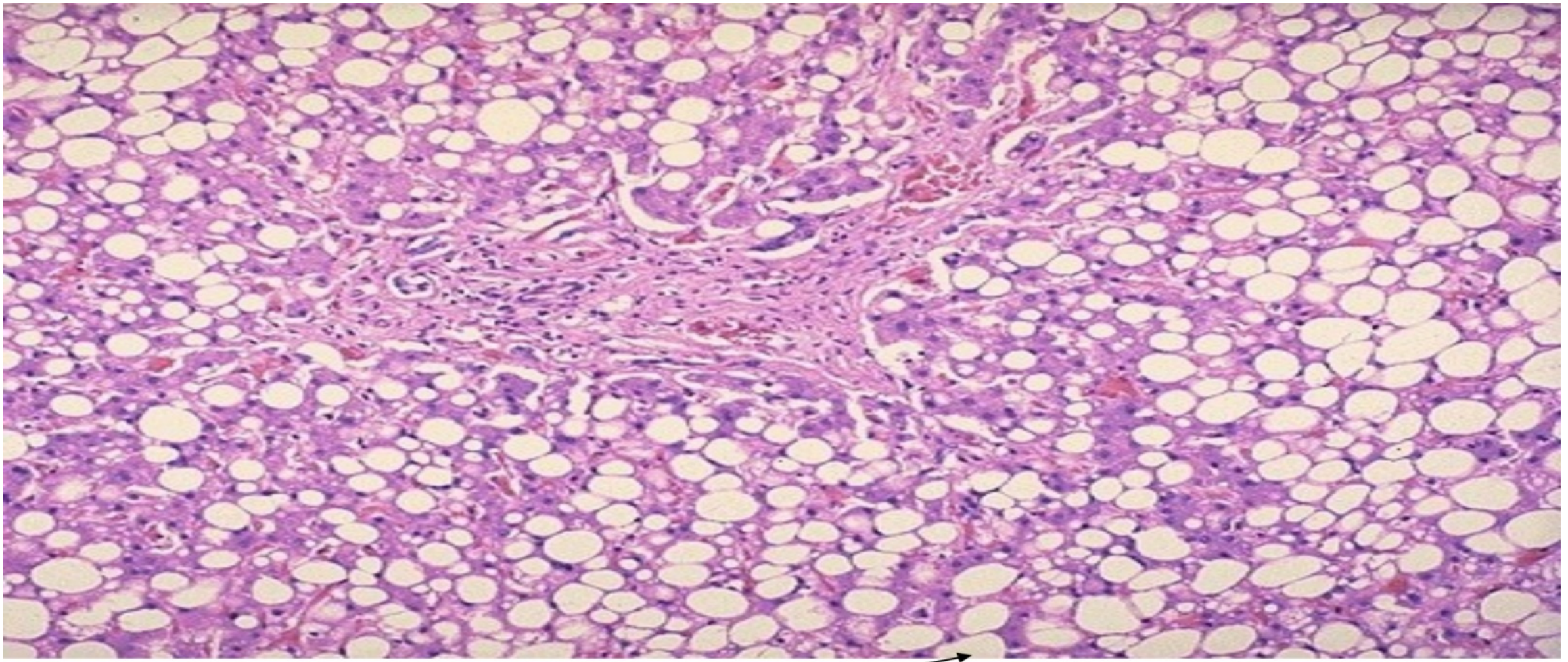
macrovesicular:DM,obese

The complication are similar

fatty change

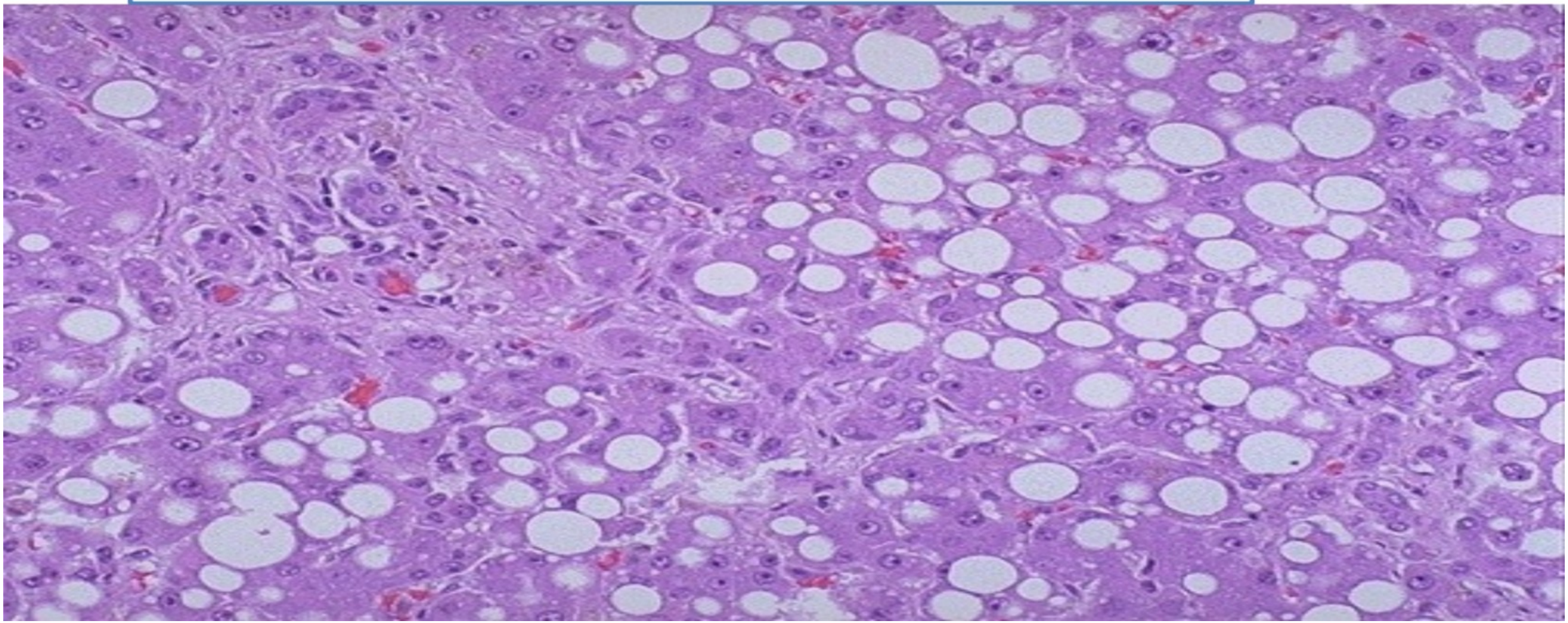
Yellow, greasy and enlarged organ (4-6)KG

—Microscopic image of fatty liver:—
-this is asevere form (you can see that atmost all hepatocytes has fat accumulation).



8 Look at the fat infiltration within the cytoplasm of the hepatocytes @ 'A'°SC*'''_“ ‘*

This is a higher magnification of the last slide :
it represents a macrovascular fatty change



Note:-
Microvascular fatty change may lead to macrovascular with more accumulation of fats

*The liver has a high capacity of regeneration therefore it's a result of severe injury.

*It indicates dead cells (nuclear changes: pyknosis (condesation of chromatin) ,and Karyolysis (fading of basophilia) And karyorrhexis (fragmentation)).

4-Necrosis

- Depending on the type:

Coagulative necrosis :around central v.

A wedge shaped infarct (ischemic injury) yellow

Councilman bodies Form during apoptosis

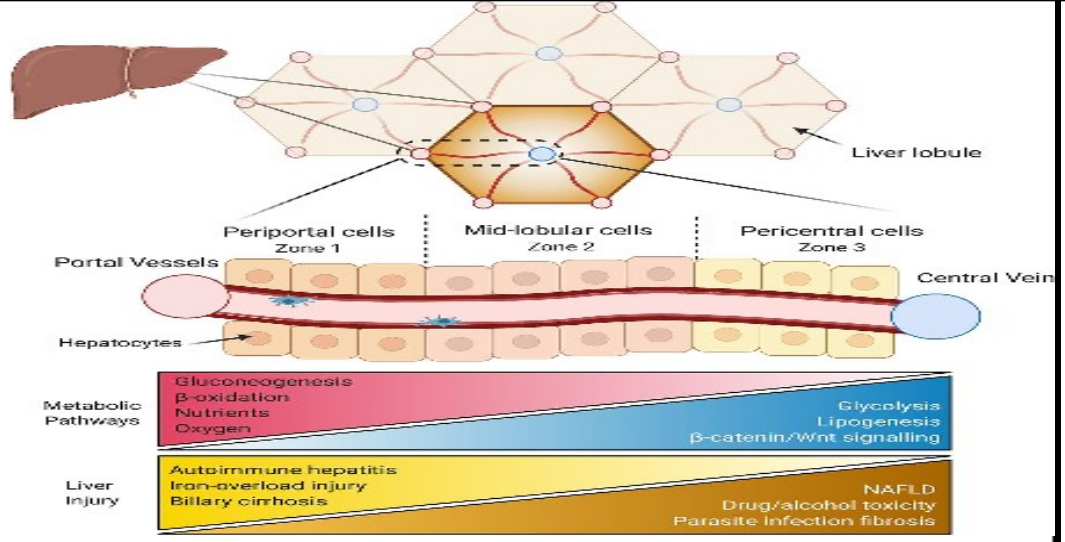
Lytic necrosis Focal liquifactive necrosis , leukocytes digest liquify the tissues .

Depending on the cause

Ischemic

Toxic Mostly they are consequences of infection

Coagulative preserves shape, lytic dose not



-depending on location

Centrilobular necrosis:

Mid zonal :

Periportal : interface hepatitis

Focal:

Piece meal necrosis

bridging necrosis

Individual,small group of cells

Bridging necrosis can be replaced by fibrosis (irreversible)

Diffuse: Related to drug and toxin exposure

massive & submassive necrosis

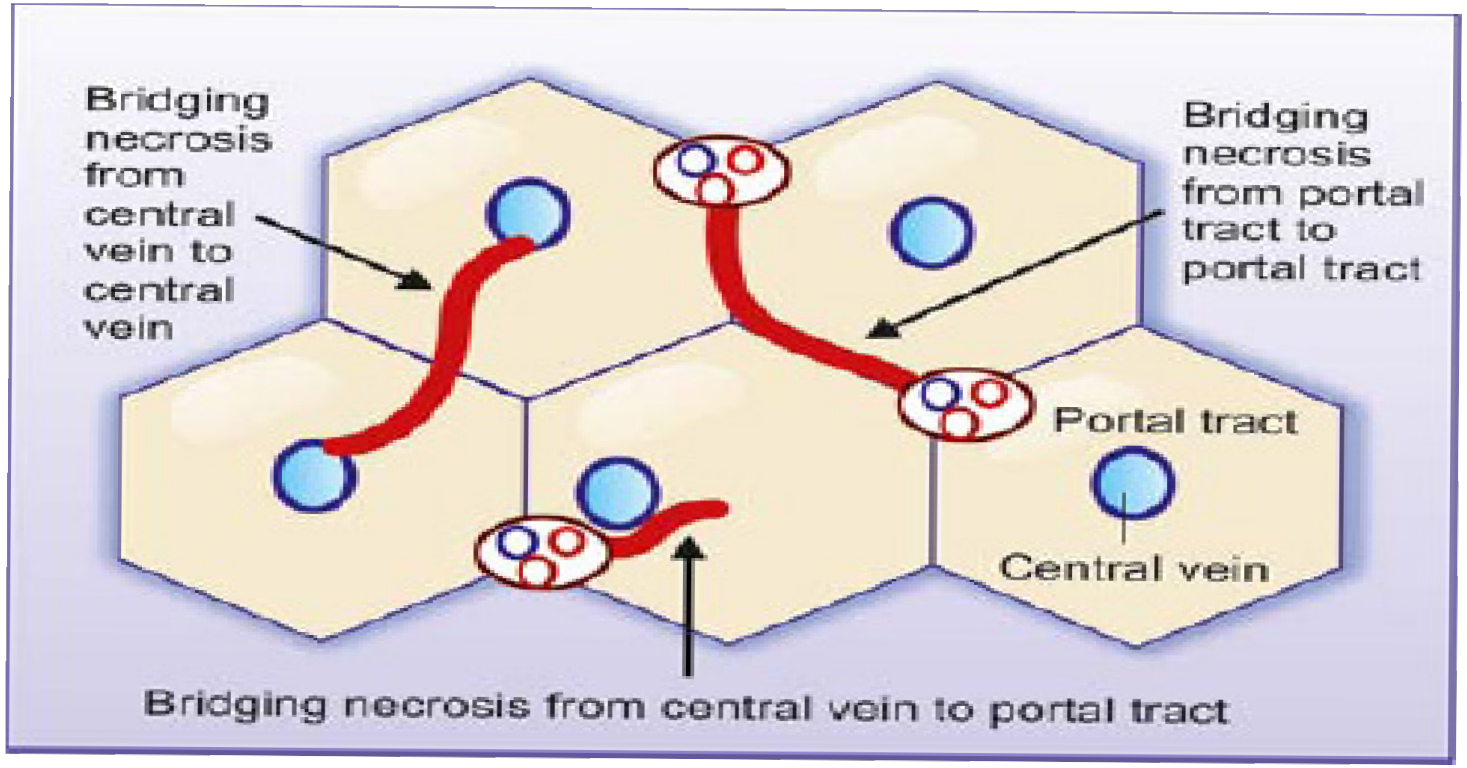
BRIDGING NECROSIS: involves vascular structur e ..centrilobular veins and liver portal tract... connect different lobule with each other

central -central

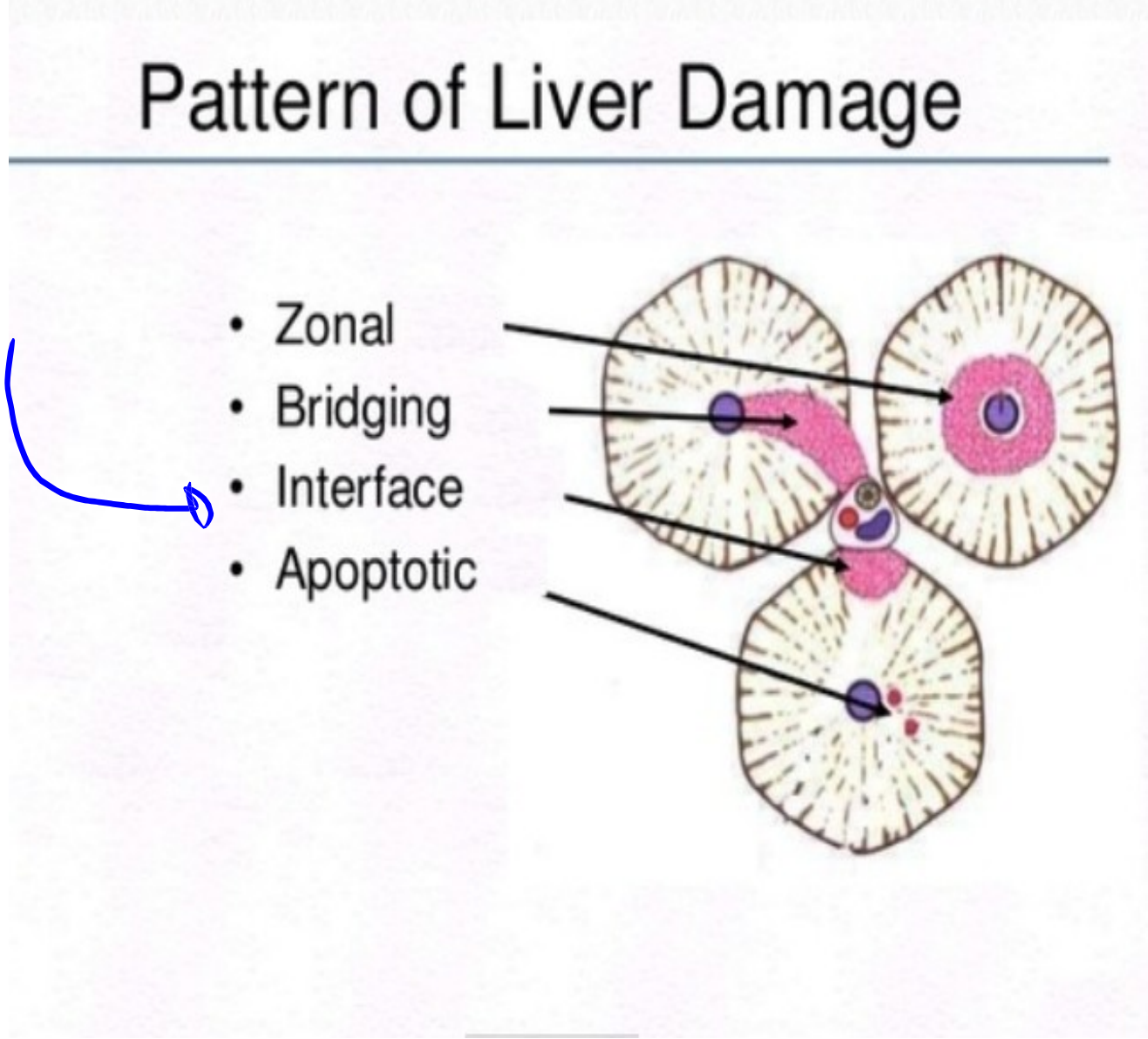
Portal -portal

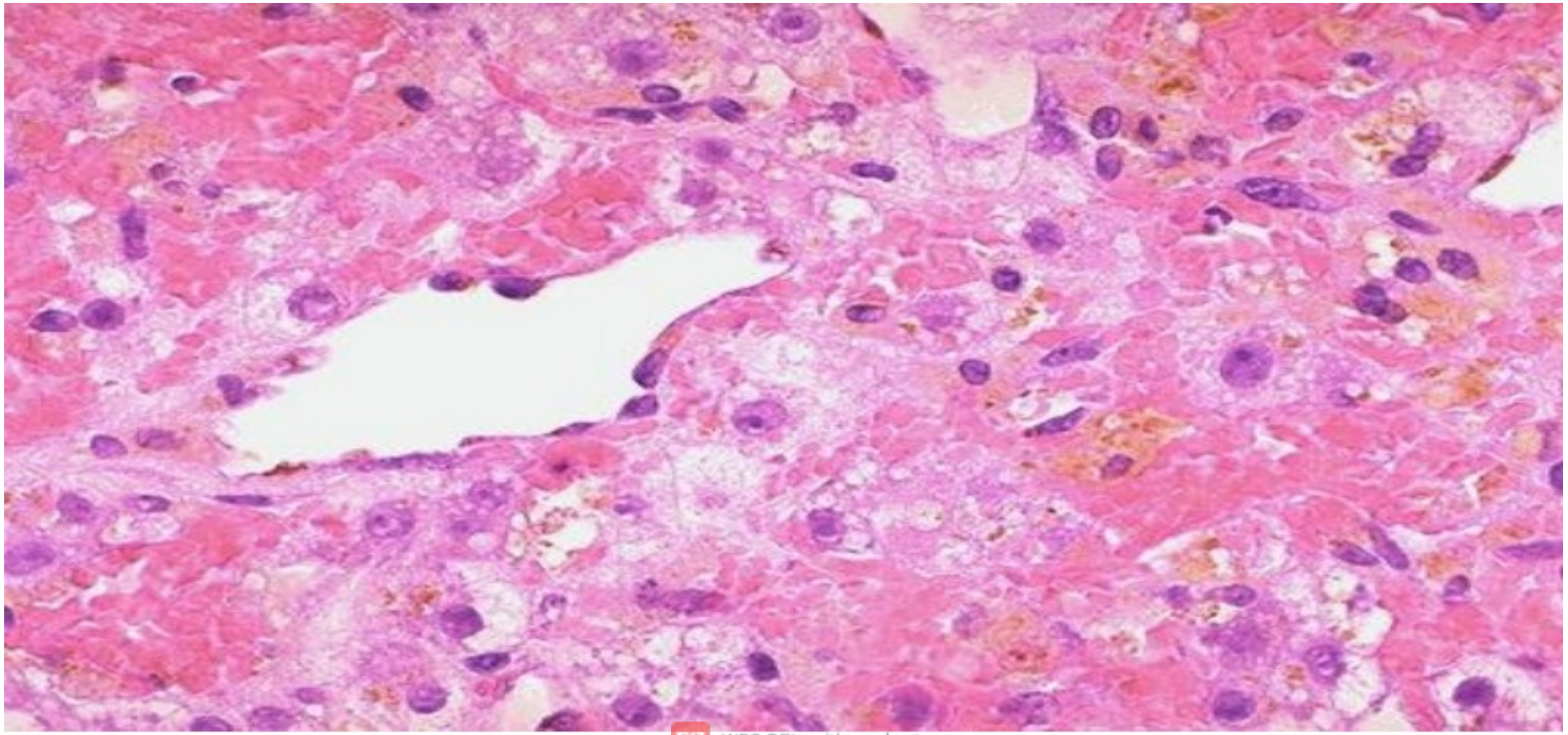
Central -portal

So extention into parynchema means the disease is now going to chronicity .



Interface hepatitis .. inflammation..inflamatory cells extend through limiting plate between portal tract and liver parynchema causing death of indiviual or group of cells .





Compensatory hyperplasia

5-Regeneration

-evidenced by increased mitosis or cell cycle markers.

-the cells of the canal of Hering are the progenitor for hepatocytes & bile duct cells (oval cells).

Again it has a high capacity of regeneration therefore more than (90-95)% of hepatocyte should be lost in order to lose its function

6-Fibrosis

-poNal or peripoNal íibrosis

-pericentral- around the central vein.

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

-bridging fibrosis

bridging fibrosis

7-Cirrhosis

micronodular

Macronodular

8- □uctular proliferation

V1