

# Cirrhosis

- Diffuse parenchymal injury, extensive fibrosis, and conversion of liver architecture into structurally abnormal nodules
- 3rd most common cause of mortality among middle-aged men in USA; 10th most common worldwide.
- Rule out other causes of nodular dysmorphic liver Imaging can strongly suggest cirrhosis
- Biopsy is necessary for establishing presence, etiology, and severity.
- Atrophy of right lobe and medial segment of the left lobe
- Hypertrophy of the caudate and lateral segment.

# General Features

## ■ Best diagnostic clue

- Nodular contour, widened fissures, and enlarged caudate lobe with ascites, splenomegaly, and varices

## ■ Size

- Moderate to advanced cirrhosis: Decreased size
  - » Earlier disease: May be enlarged
  - » Especially in primary biliary cirrhosis

## ■ Key concepts

- Common end response of liver to variety of insults and injuries
- Classification by **morphology** (not very useful)
  - » Micronodular (Laennec) cirrhosis
    - Usually due to alcoholism
  - » Macronodular (postnecrotic) cirrhosis
    - Usually viral hepatitis
- Classification by **etiology** and **severity** more useful

# Etiology

- Alcohol: No longer most common cause of cirrhosis in USA and Europe
- Etiology of cirrhosis in USA
  - » Hepatitis C (~ 25%)
  - » Alcoholic liver disease (~ 20%)
  - » "Cryptogenic" (believed to be due to nonalcoholic steatohepatitis [NASH] in most cases) (~ 20%)
  - » Hepatitis C and alcohol (~ 15%)
  - » Hepatitis B ( $\pm$  hepatitis D) (~ 15%)
  - » Miscellaneous (~ 5%)
    - Autoimmune hepatitis
    - Primary biliary cirrhosis
    - Secondary biliary cirrhosis (obstruction)
    - Primary sclerosing cholangitis
    - Hemochromatosis
    - Wilson disease
    - $\alpha$ -1 antitrypsin deficiency
    - Granulomatous disease (e.g., sarcoidosis)
    - Type IV glycogen storage disease
    - Drug-induced liver disease (e.g., methotrexate, amiodarone)
    - Venous outflow obstruction (Budd-Chiari and venoocclusive disease)
    - Chronic right-sided heart failure
    - Tricuspid insufficiency
- Due to combination of cell death, fibrosis, and regeneration
  - Deposition of extracellular matrix and fibrous tissue in parenchyma
  - Separation of hepatocytes into islands or nodules

# Staging, Grading, & Classification

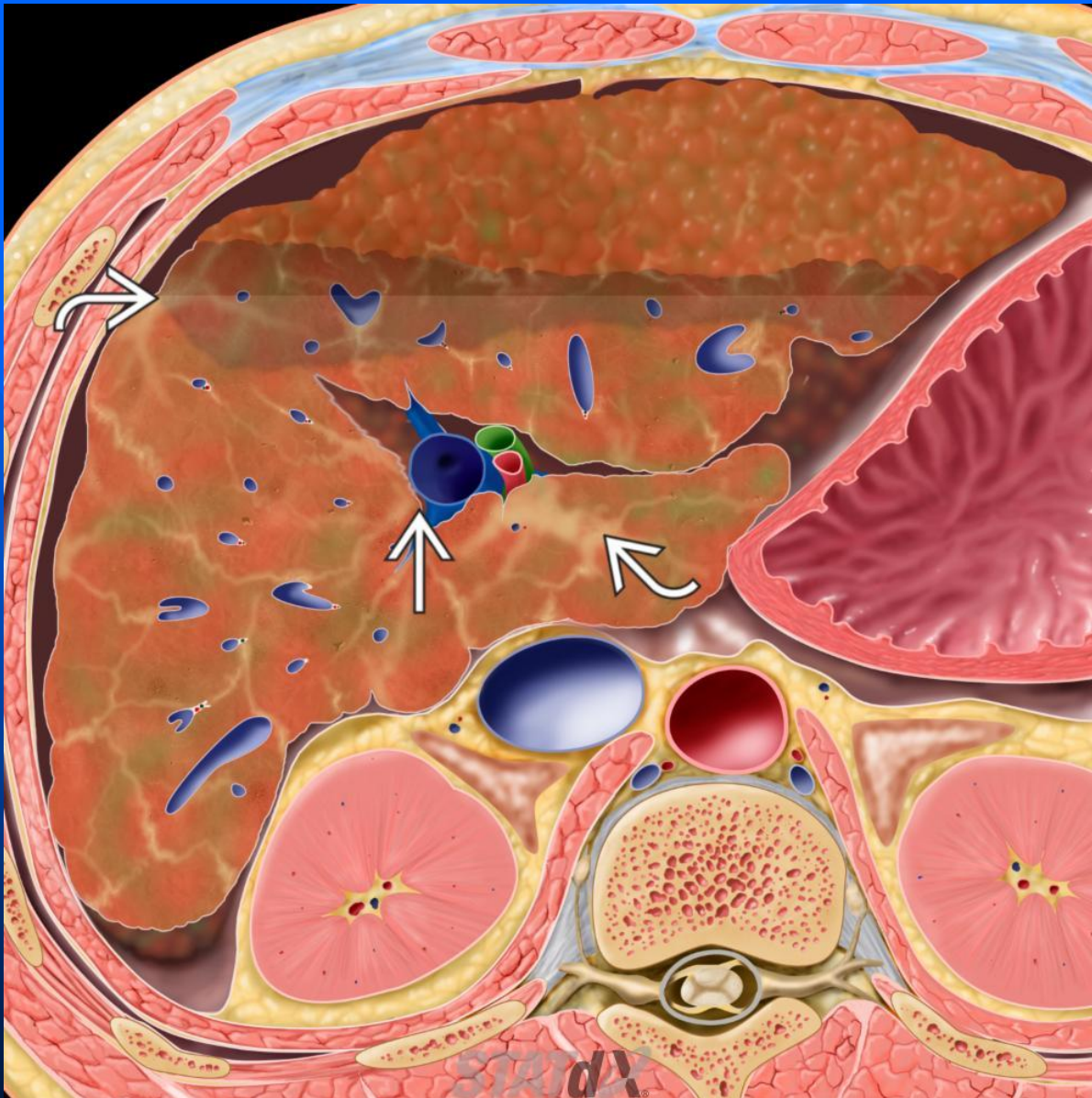
## ■ Grading of severity: **Child-Pugh classification**

- "Points" assigned for ascites, encephalopathy, ↑ serum bilirubin, ↓ serum albumin, ↑ prothrombin time
- Correlates well with survival (1 year)
  - » Child-Pugh A = 100%
  - » Child-Pugh B = 80%
  - » Child-Pugh C = 45%

## ■ **Model for End-Stage Liver Disease (MELD)**

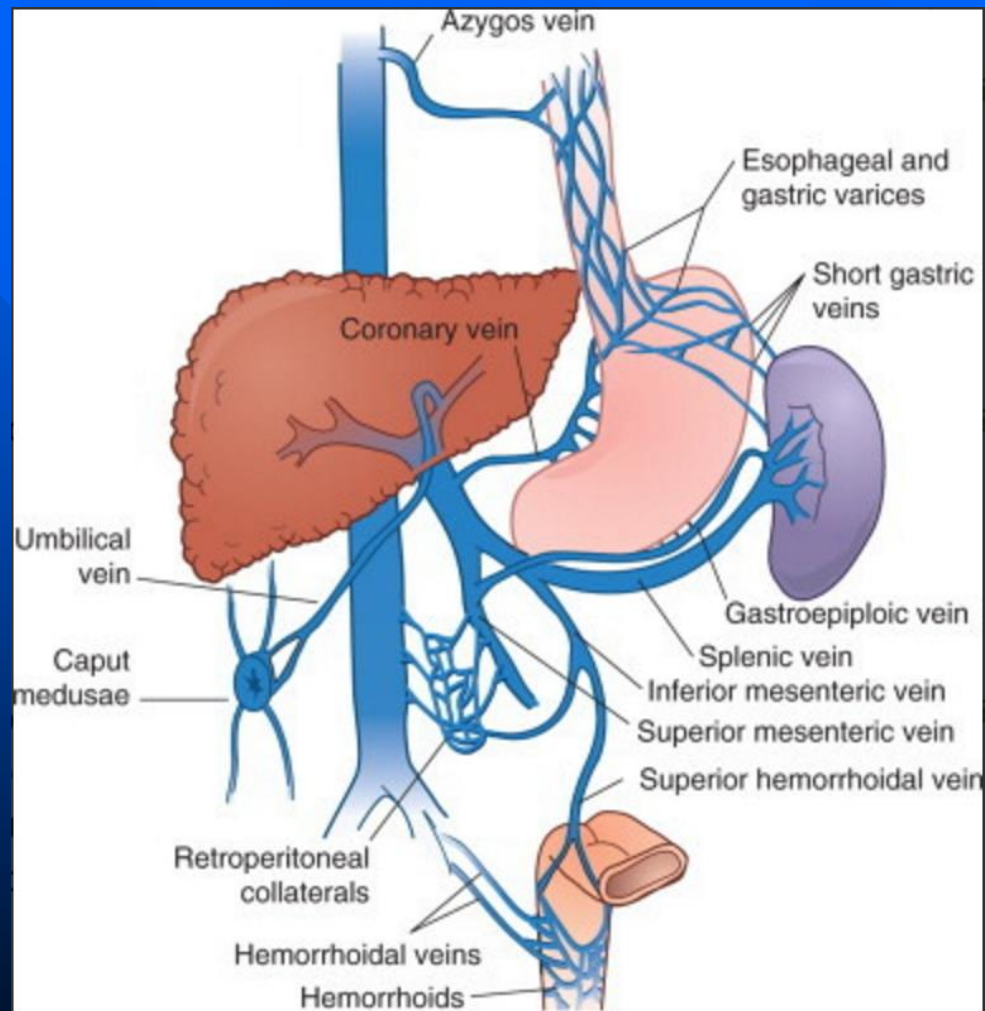
- Based on assessment of etiology of cirrhosis, serum creatinine and bilirubin level, and international normalized ratio (INR)

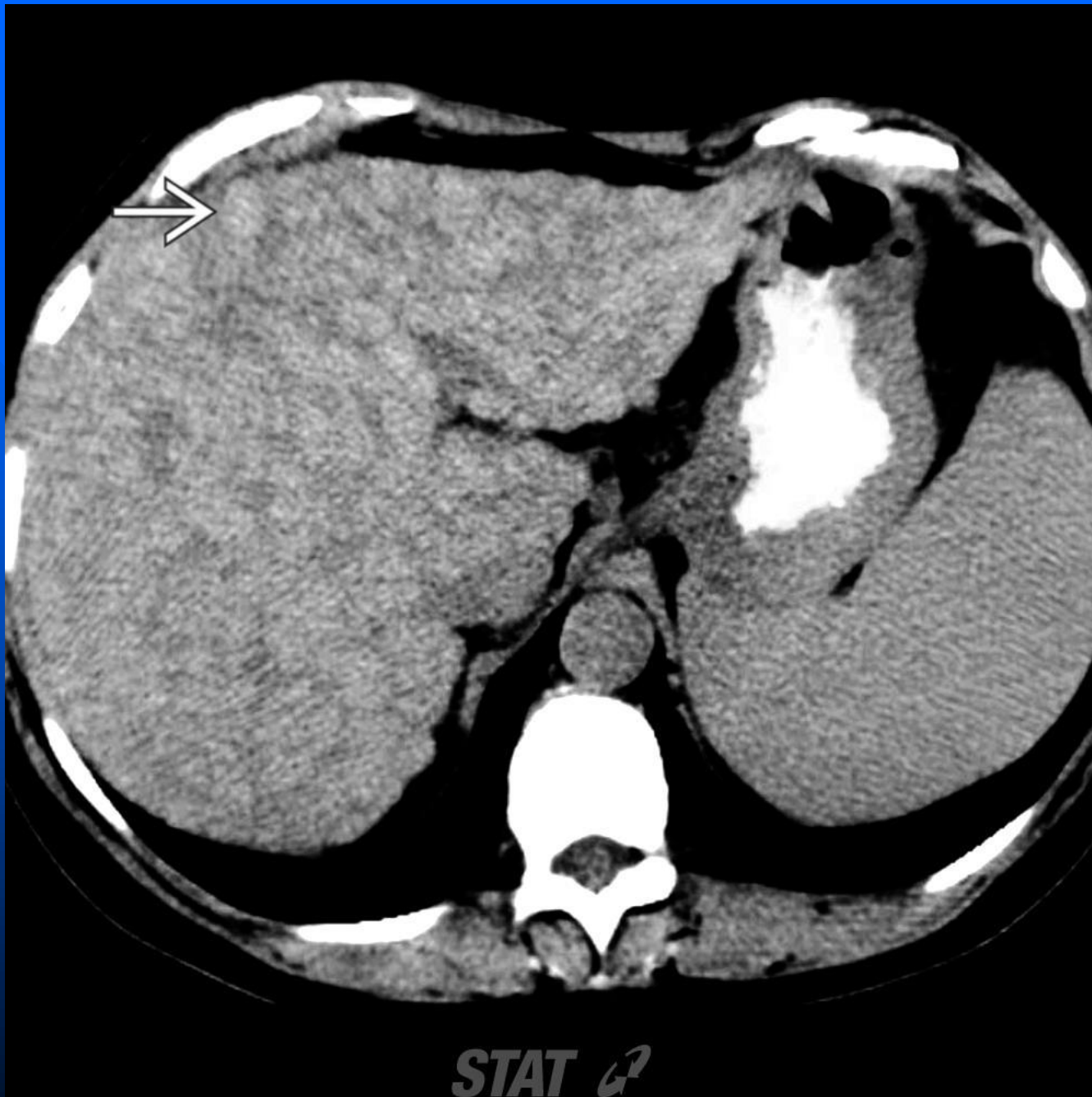




Graphic shows a cirrhotic liver with a nodular surface contour and an increase in the caudate to right lobe ratio, measured from the branch point of the right portal vein (white solid arrow) to the edges of the caudate and right lobes, respectively. Note the bands of fibrosis (white curved arrow) and ascites.

# Portal HTN



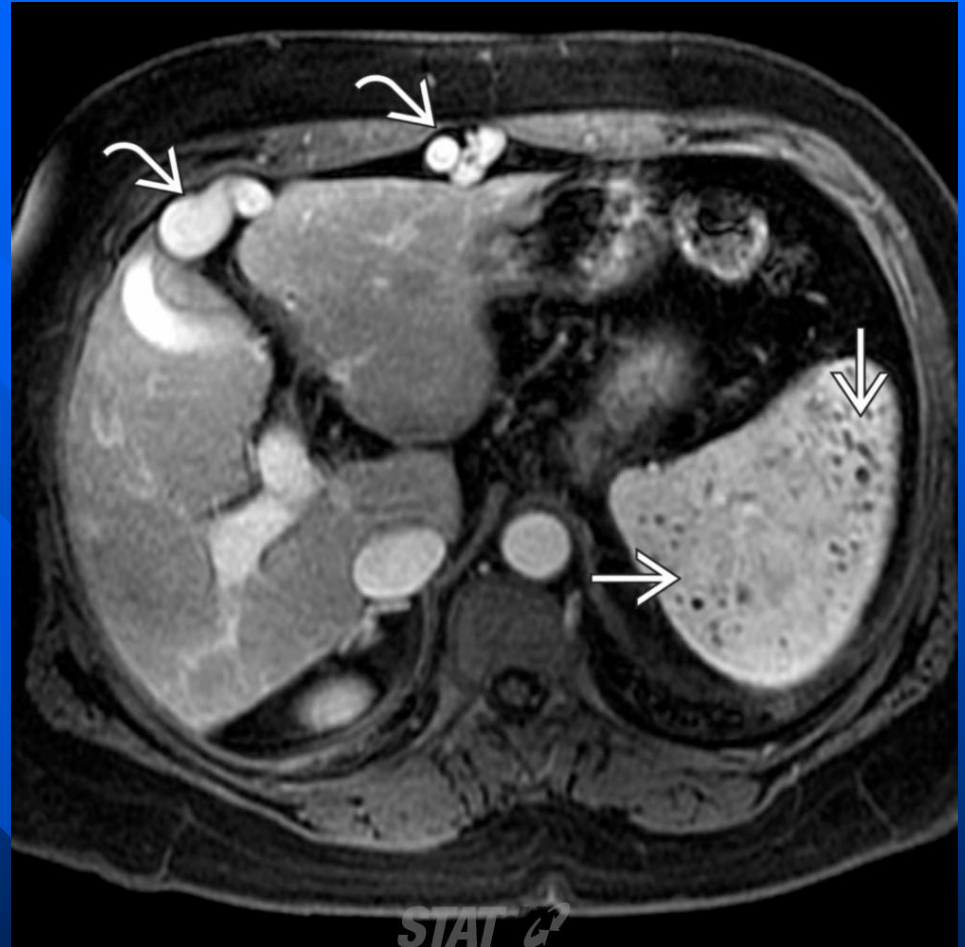


Axial NECT in this 50-year-old woman with primary biliary cirrhosis shows innumerable small hyperdense regenerative nodules (white solid arrow), surrounded by lace-like fibrosis.



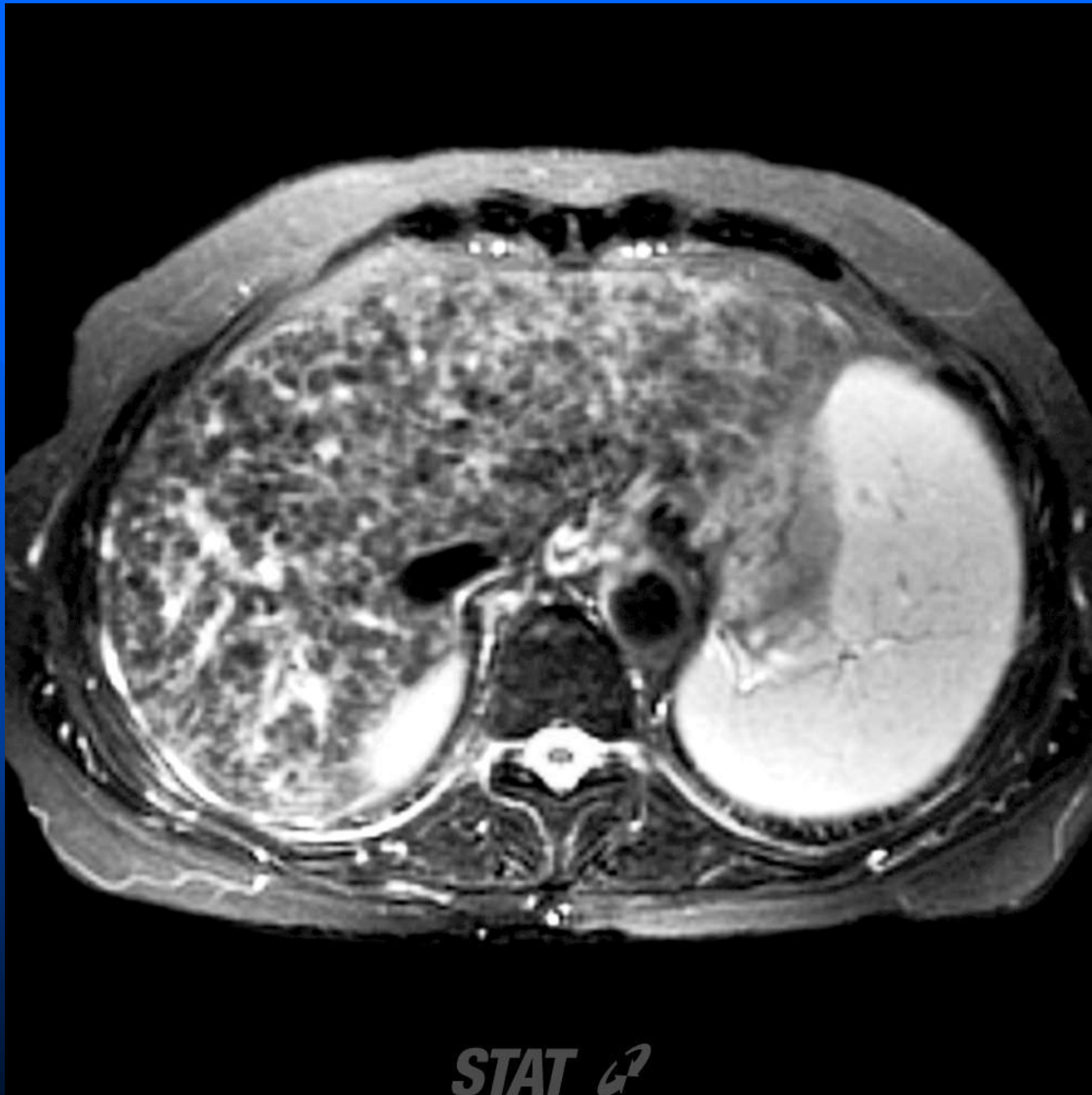
## ■ Gamna Gandy nodules

- Also known as splenic siderotic nodules and fibrosiderotic nodules
- Are small focal deposits of iron and calcium within fibrous tissue and elastic fibers in spleen resulting in tiny nodules of less than one millimeter in size.

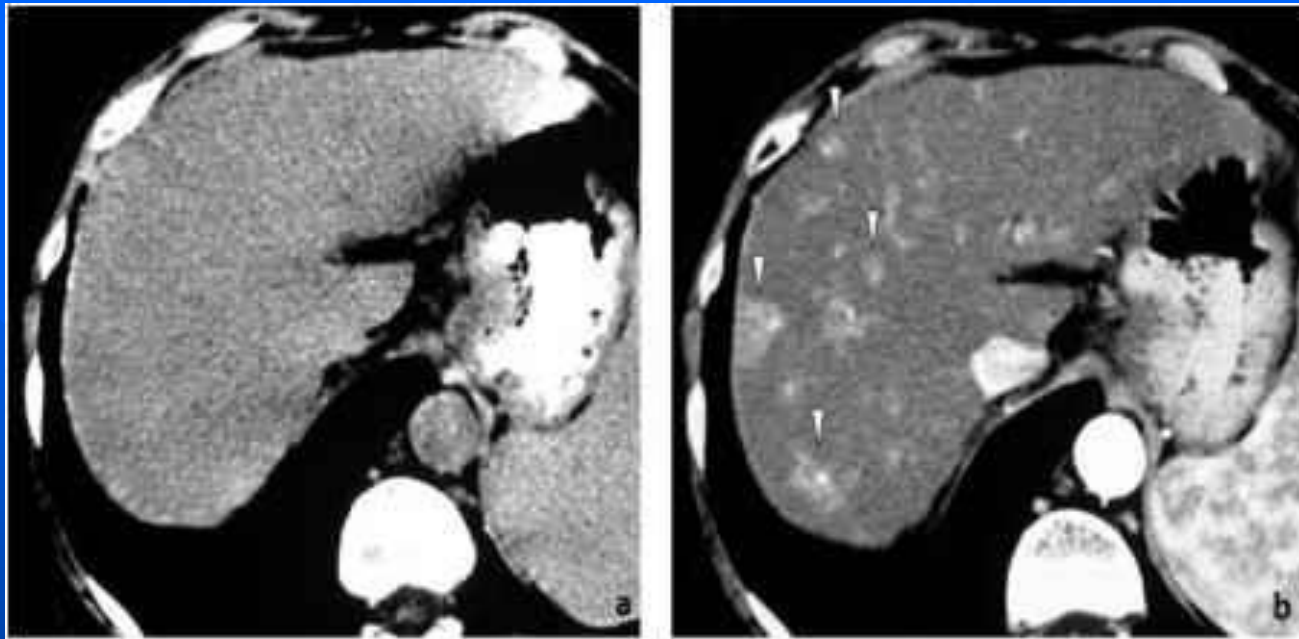


MR shows obvious signs of cirrhosis with widening of the fissures, right lobe atrophy, and large varices (white curved arrow). Within the spleen are innumerable small lesions that are especially evident as hypointense foci on this GRE T1WI contrast-enhanced image (white solid arrow), representing Gamna-Gandy bodies.

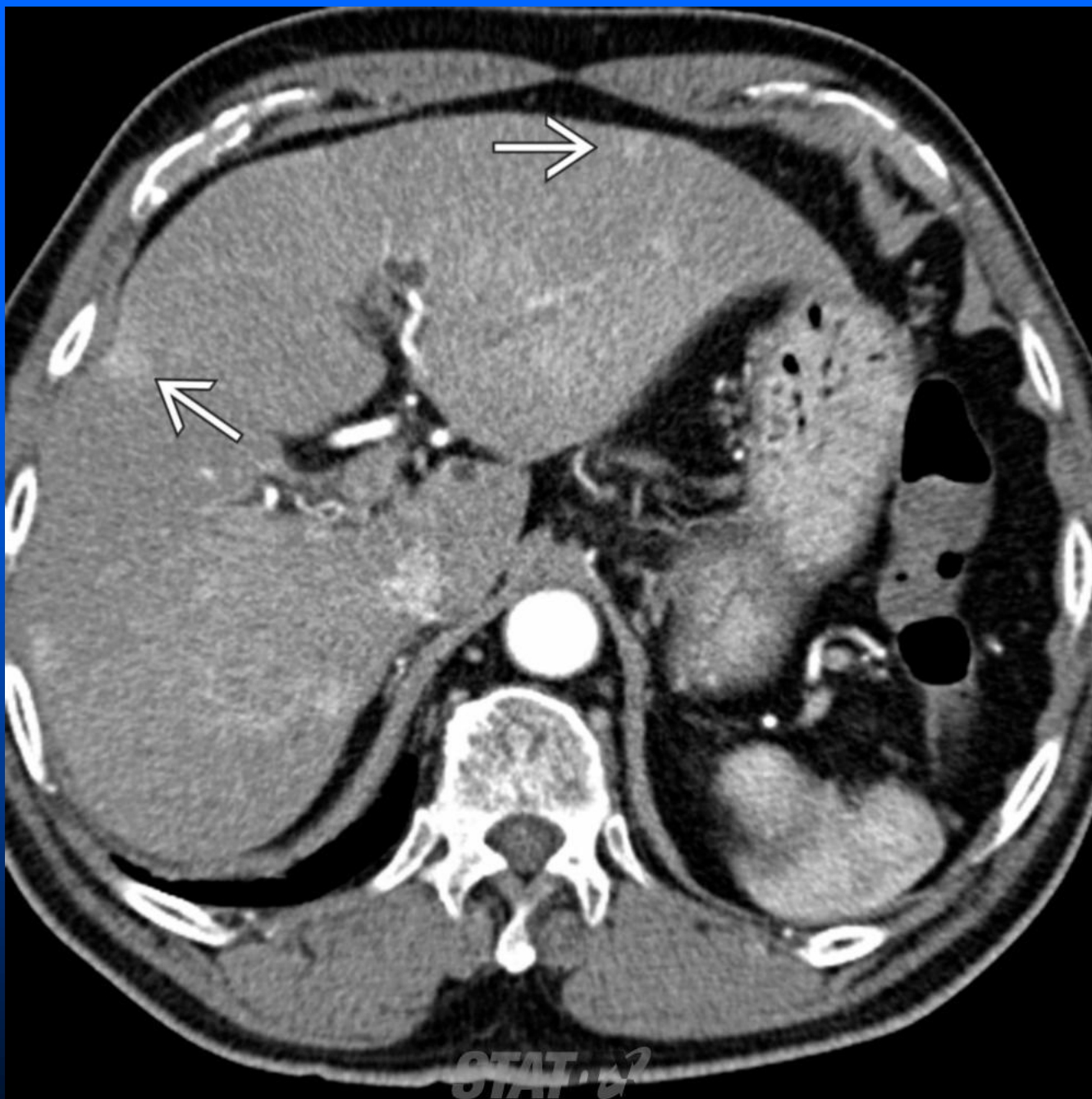




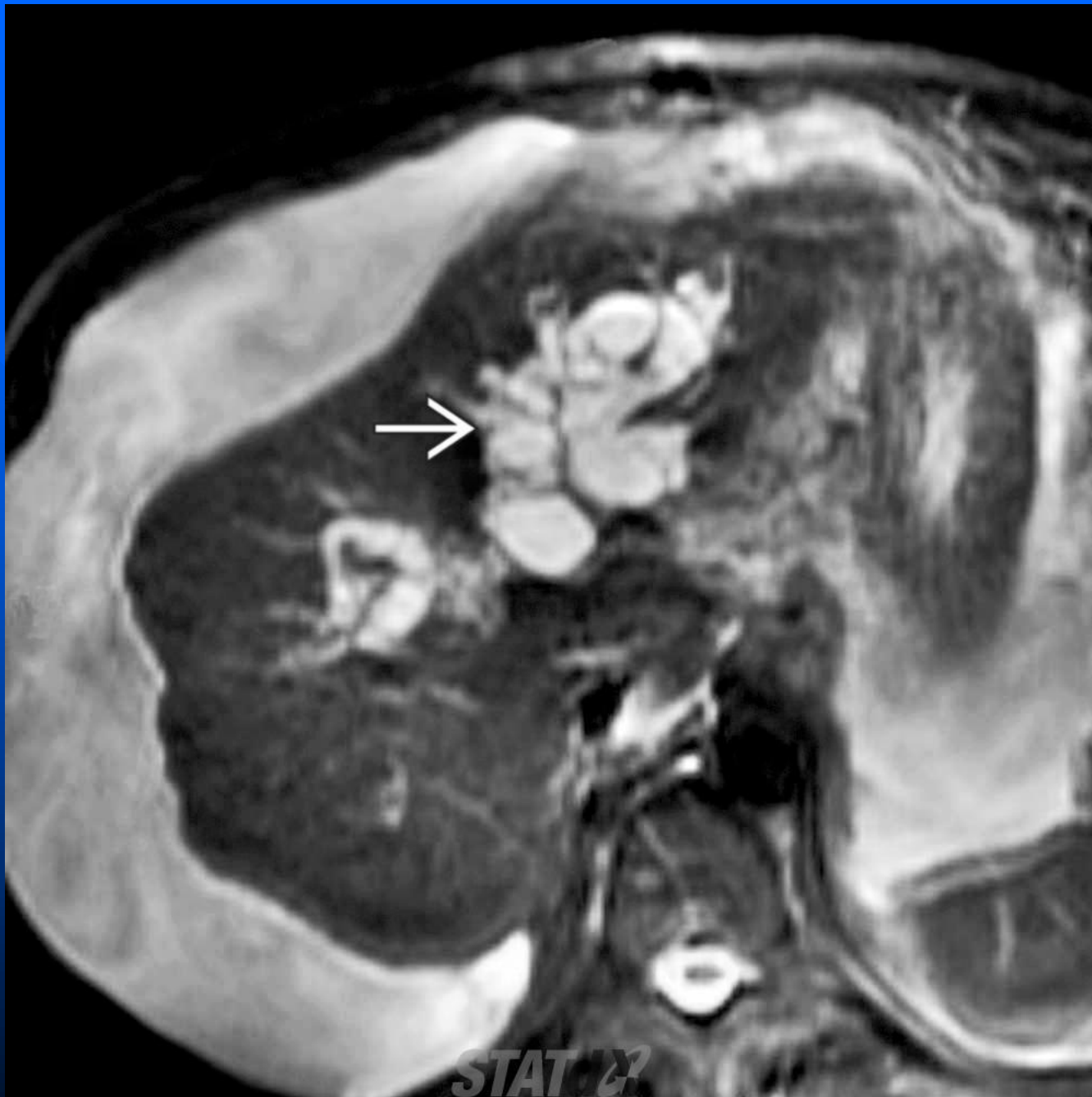
Axial T2WI MR shows innumerable subcentimeter hypointense nodules that are typical of cirrhotic regenerative nodules.



- On the pre-contrast CT scan (a) no focal lesions are visible.
- During the arterial phase (b) after contrast medium administration numerous, hyperdense areas (arrowheads) of variable size are appreciable. In the portal venous phase (c) these areas demonstrate rapid contrast medium wash-out resulting in isodensity.
- Unlike HCC, these lesions are not hypo-dense and there is no indication of a pseudocapsule on the portal venous phase scan



Axial arterial phase CECT shows arterioportal (AP) shunts as multiple, small, wedge-shaped, peripheral hypervascular lesions (white solid arrow).



Axial T2WI FS MR shows a small cirrhotic liver and ascites. Also shown are water-intensity lesions in the portal triads (white solid arrow) that do not arborize (branch) as bile ducts and are spherical in shape, representing peribiliary cysts.

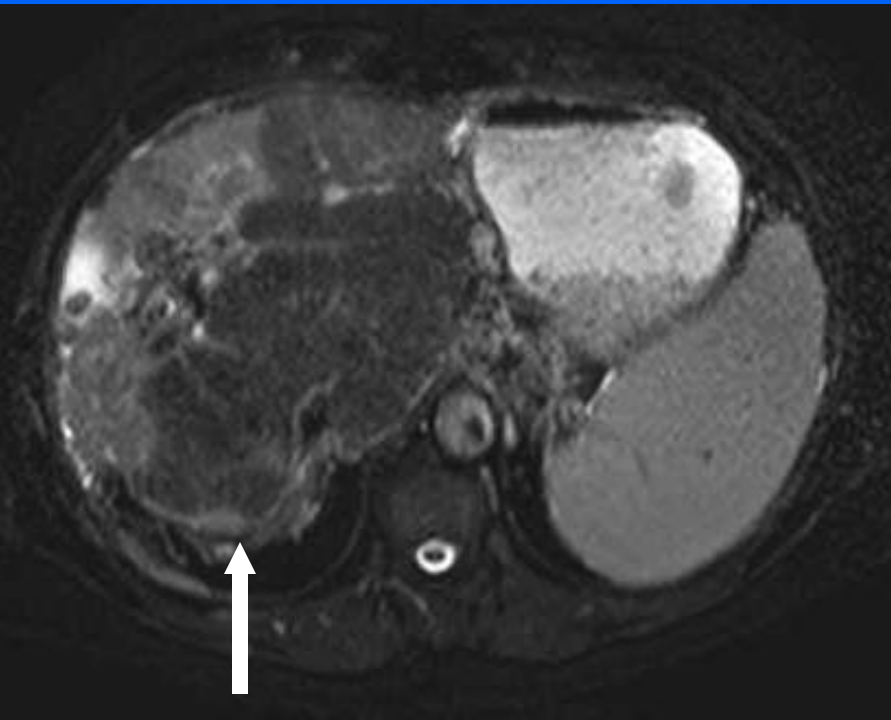


# Cirrhosis

- Typical Corkscrew appearance of the hepatic arteries
- May be normal until late
- Flow is increased in hepatic artery
- Trans jug biopsy – use R hepatic vein



# Cirrhosis With regenerating Nodule in Caudate



T2

T1 Pre

