Hepatocellular Carcinoma

- Most frequent primary visceral malignancy in world.
- Elderly patient with history of cirrhosis, ascites, weight loss, RUQ pain, and ↑ α-fetoprotein (AFP)
- Complications
  - Spontaneous rupture and massive hemoperitoneum
  - IVC invasion, possible tumor embolism to lungs
  - Metastases (adrenals and lungs most common)
- Goal is to diagnose tumors < 3 cm in diameter that are limited to liverProperly performed and interpreted CT or MR can accomplish this goal
- **Use LI-RADS classification system**
  - Provides standardized criteria for interpreting findings of CT & MR examinations in patients with chronic liver injury
- Accurately report vascular invasion and number and size of lesions

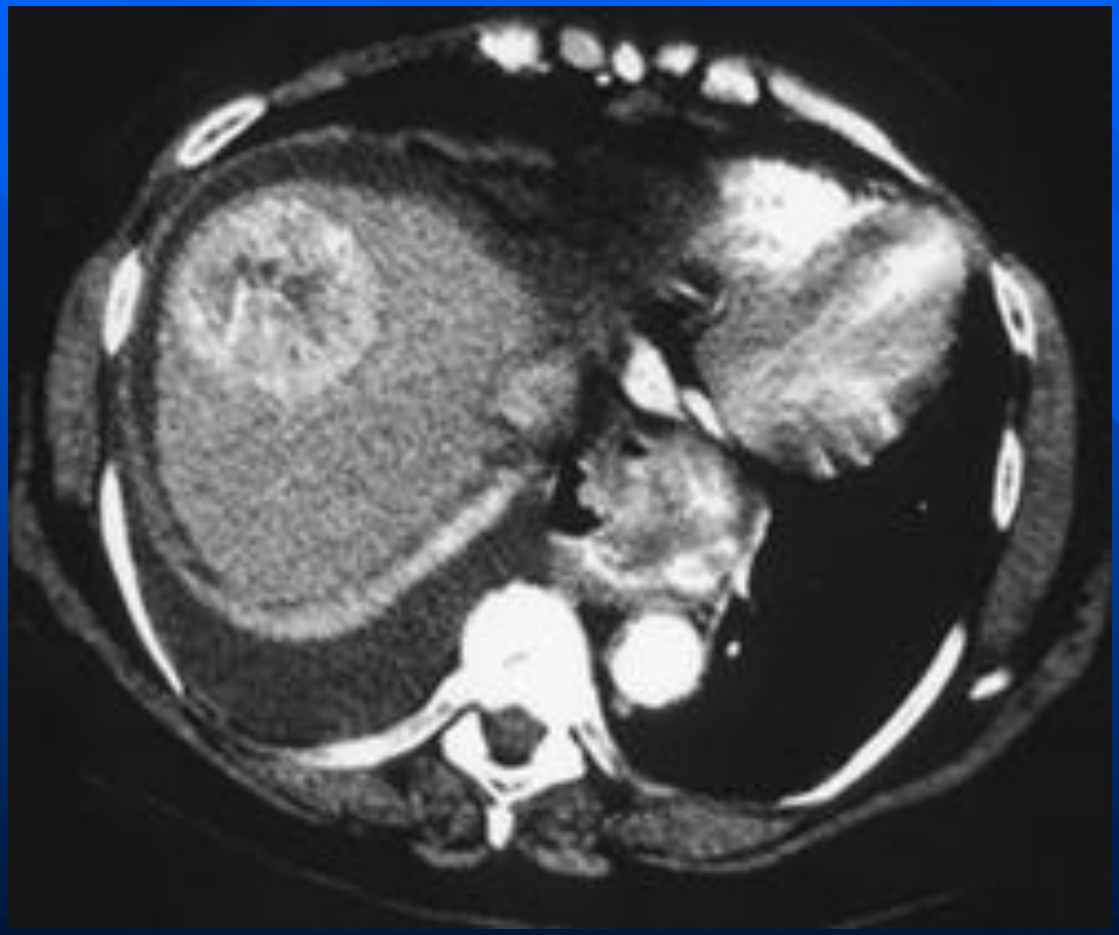
Etiology

- Cirrhosis (60-90%): Due to chronic viral hepatitis (HBV, HCV) or alcoholism
  - Even patients without cirrhosis usually have chronic liver injury (e.g., by hepatitis B)
- Nonalcoholic steatohepatitis (NASH)
  - Increasingly common cause for cirrhosis and HCC
- Carcinogens
  - Aflatoxins, siderosis, Thorotrast, androgens
- α-1-antitrypsin deficiency, hereditary hemochromatosis, Wilson disease, tyrosinosis
- **Genetics**
  - HBV DNA integrated into host's genomic DNA in tumor cells

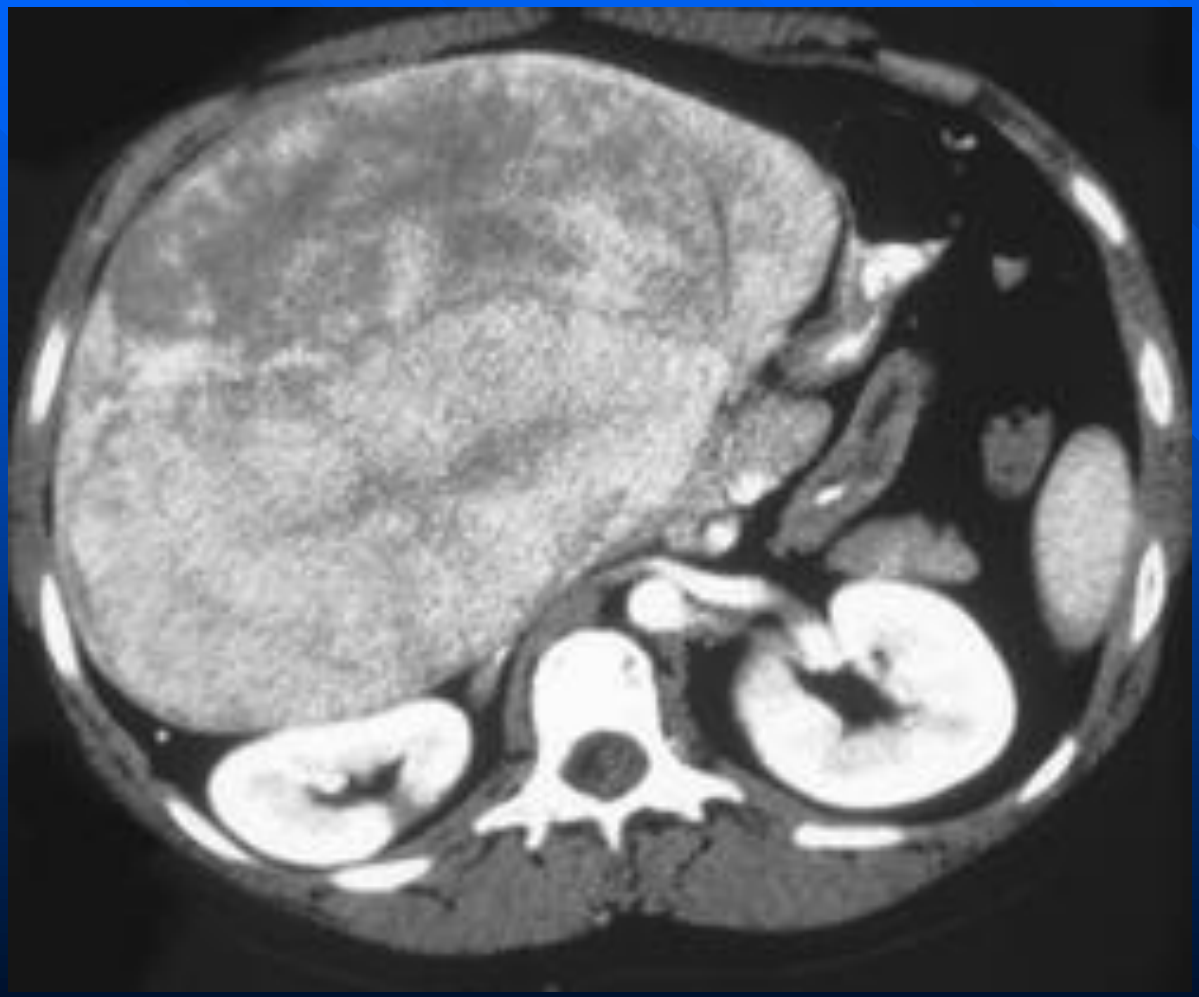
MRI

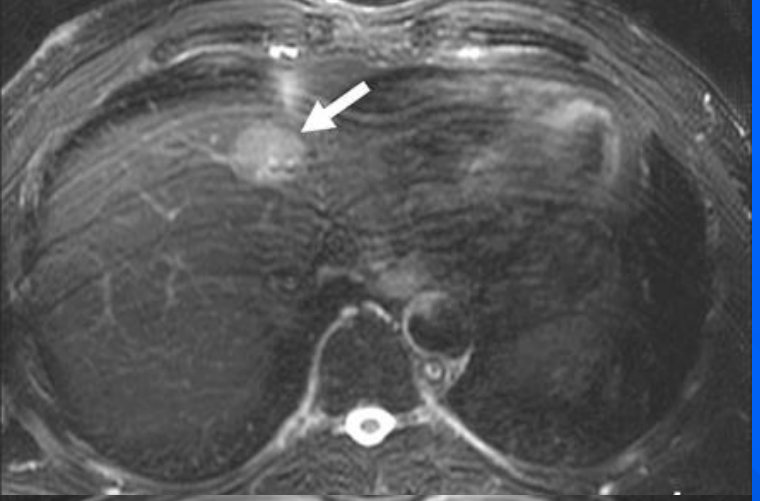
- Variable intensity depending on degree of fatty change, fibrosis, necrosis
- T1WI
  - HCC may be hypo-, iso-, or hyperintense to liver
  - Tumors with fat or hemorrhage are hyperintense
- T2WI
  - Usually hyperintense to liver
    - Regenerative nodules are hypointense on T2WI
  - HCC arising within dysplastic nodule
    - "Nodule within nodule" pattern
    - HCC appears as small focus of increased signal intensity within decreased signal intensity nodule
- T1 C+ (gadolinium)
  - Heterogeneously hyperintense, with washout on portal venous and delayed phase
  - Rapid central washout with residual capsular enhancement = HCC, **not** arterioportal shunt
- Hepatobiliary contrast agent (gadoxetate)
  - Trade names: Eovist or Primovist
  - On 20 minute delayed phase
    - Normal liver (and some portions of cirrhotic liver) enhance brightly
    - Most HCCs are seen as hypointense focal masses
    - Rare for well-differentiated HCC to show delayed persistent enhancement with gadoxetate
  - Increases sensitivity of MR in diagnosing small HCC
- Diffusion-weighted MR
  - Restricted diffusion within HCC often detected as bright signal in focal lesion
  - Adds sensitivity and specificity to MR detection of HCC

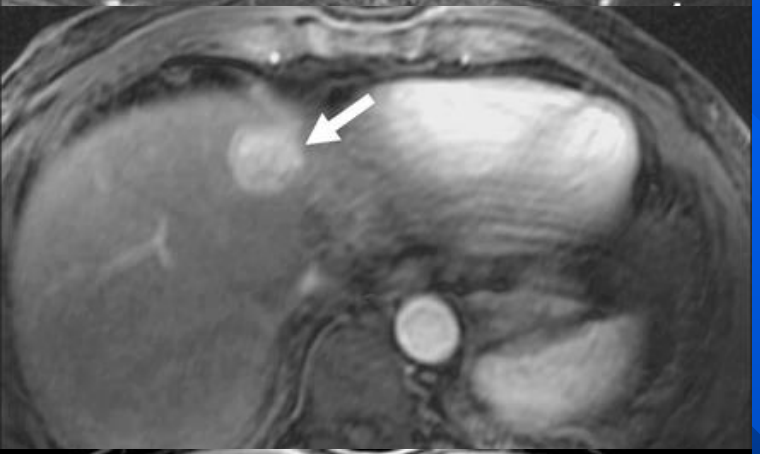
Hepatoma

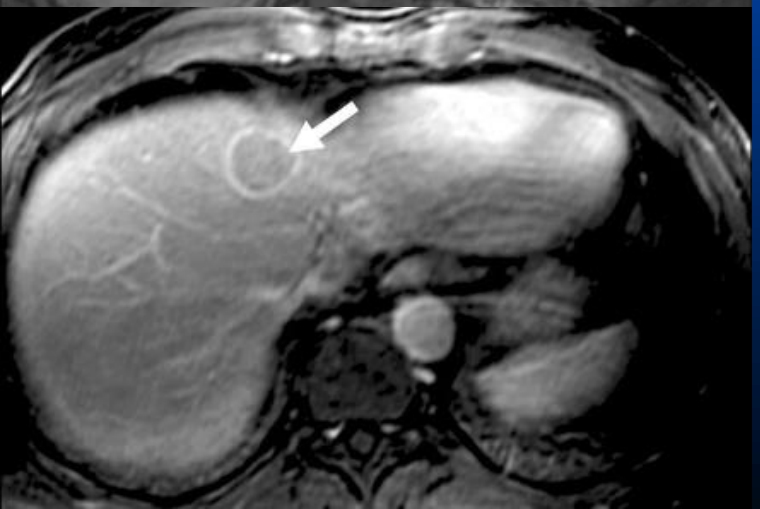


Hepatoma









Hepatoma

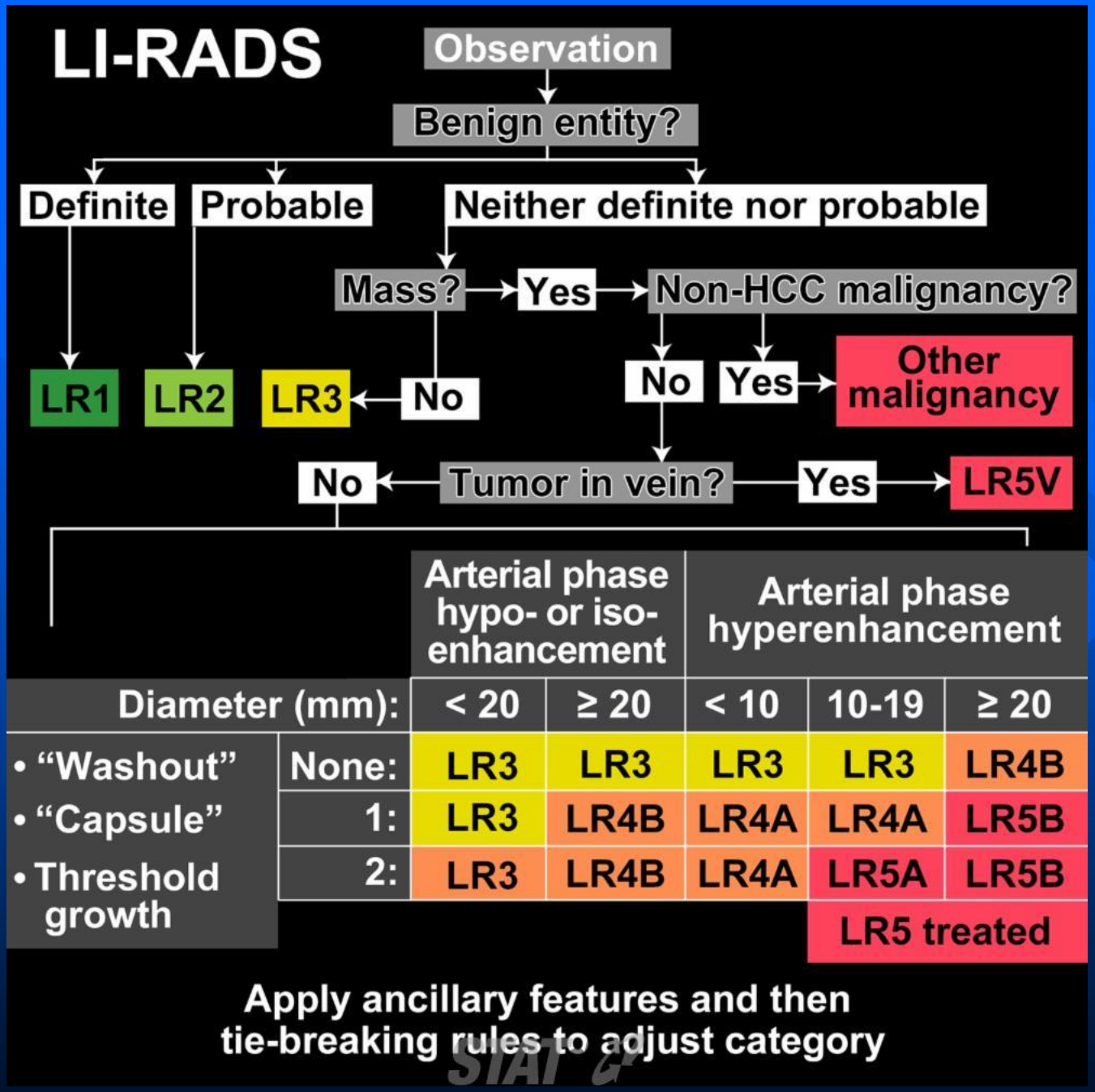
T2W FSE showing a hypointense Capsule due to fibrosis

T1W FSE post gad early arterial Showing diffuse enhancement Simulates a FNH

GRE post gad portal venous Phase showing vivid Enhancement of the surrounding Capsule

HCC / Dyplastic nodules

- **Dyplastic nodules** are dark on T2 iso to increased on T1
- **HCC** is bright on T2, will show early ehnhancement with rapid wash out with peripheral rim of enhancement on delayed images
- **Greater than 3 cm will invade portal**



The Liver Imaging Reporting and Data System (LI-RADS) is endorsed by the ACR for categorization of focal nodular lesions found on CT or MR in the cirrhotic liver. It is designed to standardize the interpreting and reporting of findings so that these are more uniform, accurate, and useful to referring physicians.