

LIVER TUMORS

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Classification of Hepatocellular Nodules

- Regenerative nodules
 - Monoacinar regenerative nodule
 - Multiacinar (large) regenerative nodule
 - Lobar or segmental hyperplasia
 - Cirrhotic nodule
 - Focal nodular hyperplasia

Classification of Hepatocellular Nodules

- Dysplastic or neoplastic lesions
 - Hepatocellular adenoma
 - Dysplastic nodule
 - Hepatocellular carcinoma

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Nodular Regenerative Hyperplasia

- Usually results from obstruction of hepatic or portal veins
 - Budd-Chiari, leukemia, polycythemia, etc.
- Patients have abnormal LFTs but not cirrhosis

NRH – CT findings

- Multiple (2-20), small (1-4 cm)
- Homogeneous enhancement, +/- ring

NRH – Problem Solving

- MR – same features as CT
 - usually hyperintense on T1, hypo on T2
- Unlike HCC, may take up and retain Gd-BOPTA (= benign)
- Recognize associated clinical and liver abnormalities
- May have to biopsy

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Lobar or Segmental Hyperplasia

- Budd-Chiari syndrome and primary sclerosing cholangitis
- Enlargement of a lobe or large portion of a lobe
- Caudate lobe usually involved
- Atrophy, necrosis or fibrosis of other lobes
- Also referred to as the *atrophy-hypertrophy complex*

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Cirrhotic Nodules – Classification

- Based on
 - 1) hyperplastic or dysplastic
 - 2) nature of surrounding liver
- All are due to local proliferation of hepatocytes + stroma in response to injury (ischemic or toxic)
- May be indistinguishable histologically
 - imaging plays a key role

Regenerating Nodules

- Usually too small to detect by imaging
 - May be surrounded by fibrotic septa
 - May contain iron, copper
- Siderotic nodules
 - Hyperdense on NCCT, disappear on HAP & PVP
 - Hypointense on T2 MR, “bloom” on GRE
- Larger or vascular/enhancing RN
 - Can not be distinguished from dysplastic nodule or HCC

Dysplastic Nodules

- “Adenomatous hyperplasia” (old term)
- Are premalignant
- Rarely diagnosed by US or CT
- MR – iso to hyperintense on T1
 - Hypo on T2 (opposite of HCC)
 - Should not enhance much on HAP
- Diagnosed correctly 5 – 15% of cases

Krinsky et al. Radiology 2001; 219: 445-454

Dodd et al. AJR 1999; 173: 1185 - 1192

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Focal Nodular Hyperplasia (FNH)

- Present in 2-5% of population (F >> M), incidental finding, rarely symptomatic, may simulate tumor
- Size 2-10 cm
- Number: solitary (77%) to numerous (brain neoplasms + AVMD)
- Path: normal hyperplastic liver tissue with central scar; other vascular lesions (adenoma, hemangioma)

FNH – CT Criteria

- Homogeneous on all phases
- Almost isodense with liver (noncon., PVP, delayed)
- Uniformly and very hypervascular on HAP (like vessels)
- Non-encapsulated, fuzzy margin, no Ca⁺⁺, no capsule (pseudocapsule in fatty liver)
- Small scar, 65% of “big” FNH, 35% of small (< 3cm)

FNH – Problem Solving

- Radionuclide scanning TcSC (uptake in 50-60%) or HIDA (80%)
- MR same features as CT with Gd-DTPA; very bright on enhanced T1WI
- Hepatobiliary agents show uptake in FNH and prolonged excretion, mangafodipir (MnDP), Gd-BOPTA

FNH vs. Fibrolamellar HCC

- FL-HCC typically solitary, large tumor (13 cm)
- Typical patient: young to middle age, no predisposition

Fibrolamellar HCC – CT Findings

- Heterogeneous enhancement on all phases (~100%, no hemorrhage or fat)
- Lobulated irregular contour, well defined (75%)
- Large central/eccentric scar (75%)
 - + Radiating fibrous septa, +/- delayed enhancement
- Central calcification (68%)
- Lymph node + other mets (65%)
- Vascular/biliary obstruction – common, invasion rare

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Adenoma

- Well defined, smooth, spherical
- >90% in young women (30-50) on OCP
 - rest in glycogen storage disease, anabolic steroids
- Often bleed (25%), rarely have malignant degeneration
 - should be resected
- 50% solitary
 - 50% multiple (rarely “adenomatosis”, dozens of lesions)
- Path
 - benign hepatocytes, few Kupffer cells, no bile ducts
 - excessive glycogen, lipids, hemorrhage, necrosis

Adenoma – CT Findings

- Usually heterogeneous due to fat (5-10% on CT, 50-75% on MR), necrosis, hemorrhage, calcifications (5%)
- Hypervascular
- Partial capsule (~ 25%)

Adenoma – Problem Solving

- Hard to distinguish from HCC
 - usually resect, especially if hemorrhage
- MR shows fat and blood better than CT

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Conventional HCC

- Solitary (1/2) or multiple (1/2)
- Variable size
- Heterogeneous
 - best detected on HAP or delayed
- Capsule, hemorrhage, fat – all < 10%
- HV or PV invasion/obstruction – very common
 - may be seen on CT only in large vessels
- Nodes – present in cardiophrenic and porta hepatis
- Typical patient:
 - 45 – 65 with chronic hep B or C, ETOH

HCC in Noncirrhotic Liver

- HCC typically large tumor (13 cm), solitary or dominant mass (82%)
- Symptomatic 87%
- No identifiable risk factors 62%
- Calcifications 28%, hemorrhage 26%, fat 10%
- Tumors heterogeneous 97%
- Hypoattenuating on NC 87%
- Hyperattenuating on HAP 97%
- Hypoattenuating on PVP 90%

Imaging Plays a Pivotal Role

- Diagnosing + distinguishing hyperplastic, dysplastic and malignant hepatocellular masses
- Accurate diagnosis requires:
 - Evaluation of lesion in multiphasic CT (+/- MR)
 - Evaluation of surrounding liver
 - e.g. cirrhosis, Budd-Chiari, hemochromatosis
 - Clinical information
 - e.g. glycogen storage disease, hepatitis B or C

LIVER TUMORS

- HEPATOCELLULAR ORIGIN
 - Focal nodular hyperplasia
 - Hepatocellular adenoma
 - Hepatocellular carcinoma
 - Fibrolamellar Carcinoma

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LIVER TUMORS

- NON-HEPATOCELLULAR ORIGIN
 - Hemangioma
 - Metastasis
 - Biliary microcystic hamartoma
 - Bile duct cyst (simple cyst)
 - Cystadenoma-adenocarcinoma
 - Cholangiocarcinoma
 - Epithelioid hemangioendothelioma
 - Angiosarcoma



HEMANGIOMA

Incidence & Clinical Presentation

- Most common tumor (1-20% gen.pop.)
- Uniform geographical distribution



HEMANGIOMA

- Women (5:1)
- Post menopausal
- Asymptomatic (85%)



HEMANGIOMA

- Multiple vascular channels
- Peripheral feeding vessels
- Areas of fibrosis, necrosis
- Normal liver contour



HEMANGIOMA

- Solitary (90-50%)
- Sharply circumscribed
- Subcapsular (normal liver contour)
- Cystic areas
- Occasionally Pedunculated



HEMANGIOMA

- Calcification (<10%)
- Coarse (fibrosis)
- Phleboliths



HEMANGIOMA

CT/MRI APPEARANCE

- 1) Single, small (< 3 cm)
- 2) Confined within normal liver contour
- 3) Peripheral globular filling, arterial density, slow flow
- 4) Homogeneously hyperintense in T2



HEMANGIOMA - CT FINDINGS

- Low attenuation
- Well defined
- Multiple
- Normal contour
- Early, globular peripheral enhancement
- Isodense with aorta
- Slow centrepetal



HEMANGIOMA - Calcification

- Up to 20 % of cases
- Large fibrotic areas



HEMANGIOMA

Rapid Enhancement

- In small, capillary hemangiomas
- Uniform, early enhancement



HEMANGIOMA

MRI FINDINGS

T1: low SI

T2: - markedly high SI
- homogeneous
- no halo



LIPOMATOUS TUMORS

- Lipoma
- Angiomyolipoma
- Myelolipoma
- Angiomyolipoma



BILIARY MICROCYSTIC HAMARTOMA: (VON MEYENBURG COMPLEX)

- Primitive and aberrant bile ductules
- Cystic dilatation
- Multiple, < 1cm.
- Spectrum of adult fibropolycystic diseases



BILIARY CYSTADENOMA

- Continual with cystadenocarcinoma
- Women 45 - 55 years
- Symptomatic (RUQ pain, mass)



INTRAHEPATIC CHOLANGIOCARCINOMA

- 2nd most common primary liver malignancy
 - 10% - 20% of primary malignancies
 - Only 10% intrahepatic
- M > F (1.6:1)
- Age: 7th decade
 - Older than HCC



I-CAC

Pathology/Microscopy

Adenocarcinoma: mucin rich, no bile
Abundant fibrous stroma
Calcification



I-CAC

Pathology/Microscopy

Extension into:

- surrounding structures
- biliary tree

No tumor thrombus



I-CAC

Pathology/Gross

Large (5-20 cm),
Solid (Hemorrhage/necrosis rare)
Fibrous
No capsule, Local extension
Satellite nodules (20%)
NO cirrhosis



I-CAC

Pathogenesis/Associations

- Thorotrast
- Hepatolithiasis (5% - 20%)
- Clonorchis sinensis
- Sclerosing Cholangitis
- Caroli Disease
- Congenital Hepatic Fibrosis



I-CAC CT

- Hypodense, homogeneous, irregular borders
- Calcification
- Variable enhancement, peripheral
 - Central fibrosis, delayed enhancement
 - Vascular encasement, no invasion
- Extrahepatic extension



I-CAC MRI

- Homogenous
- T2 hyperintense, except for
 - Central hypointense fibrosis
- Gd-DTPA: peripheral and delayed enhancement



ANGIOSARCOMA

Clinical Findings

- Most common sarcoma of liver
- M > F (4:1)
- Non specific symptoms
- Risk Factors:
 - ▶ exposure--Thorotrast (10%), vinyl chloride, & arsenic
 - ▶ hemochromatosis
 - ▶ neurofibromatosis



ANGIOSARCOMA

Pathologic Findings

Foci of malignant vascular cells

Grows along vascular spaces

Displaces Thorotrast granules peripherally



ANGIOSARCOMA

Pathologic Findings

- Multinodular (70%)
- Solitary
- Thorotrast
 - peripherally displaced
 - reticulated surface fibrosis



EPITHELIOD HEMANGIOENDOTHELIOMA

- F > M (2:1); middle age
- Association with OC or vinyl chloride (?)
- Nonspecific symptoms or asymptomatic (20%)
- Slow, peripheral (subcapsular) growth
- Hypertrophy of uninvolved liver



METASTASIS

- TYPICAL CT/MRI APPEARANCE
 - 1) Multiple and variable
 - 2) Hypodense on NCCT
 - 3) Hypovascular (PVP), thin vascular rim
 - 4) Hypervascular (HAP)
 - 5) Relaxation times between liver and cyst
 - 6) Six patterns: Doughnut, Target, Amorphous, Halo, Lightbulb



Surgical vs. Nonsurgical Lesions

- Cyst
- Focal Fat
- Flow Phenomenon
- Abscess
- Hemangioma
- FNH
- Unresectable metastasis

