MRI of Benign Liver Lesions

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St James’s Leeds, Clinical Radiology
Benign Liver Lesions
Common problem
• Increasing application of imaging investigations covering the liver (US & CT)
• Increasing sensitivity of investigations for focal liver lesions
The problem.......
• At autopsy - 52% have benign focal liver lesions*

• Incidence of primary liver malignancies increasing in the UK (and most of Europe)

• 36% patients dying of cancer have liver metastases*

*J Am Coll Radiol 2010:7;754-773

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Prevalence Benign Liver Lesions

- Cysts: 2 – 7%
- Haemangiomas: 2 – 20%
- Focal Nodular Hyperplasia: 3 – 5%
- Adenomas: 0.03 – 0.04%
- Focal Fat (infiltration/sparing): 10 – 15%
  (pseudolesions)

*Patel DV et al BMJ 2012;344:e657*
Prevalence Benign Liver Lesions

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Most benign lesions do not cause symptoms

“Incidentaloma”

An incidentally discovered mass or lesion detected by imaging performed for some other reason

American Colllege of Radiology
Community vs HPB Sx population

• Incidentaloma’s 10% practice

• Hypervascular tumours (CT)
  – 81 lesions
  – 72 benign (24 H, 40 FNH, 8 HCA)
  – 9 cancers (5 HCC, 4 mets)

• Hypovascular tumours
  – 35 benign (18 cysts, 12 fatty sparing, 1 foetal lobulation, 4 solitary necrotic nodules
  – 5 cancers (5 cholangiocarcinomas)

*Koea JB HPB 2013:15:379-83*

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Key Determinants

• Why was the investigation being done? (Was the patient truly asymptomatic)
• Patients age
• ? Past history or current malignancy
• ? Risk factors or evidence of parenchymal liver disease (cirrhosis)
Imperative to make a confident diagnosis as quickly and with as few steps as possible

**Majority**
- Reassure → Discharge

**Minority**
- Treat

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Percutaneous Liver Biopsy

- Morbidity 3%
- Mortality 0.03%
- Sampling issues
  - Primary Hepatocellular tumours
  - Regional differences within the lesion
- Seeding
Post Liver Biopsy

Subcapsular Haematoma

Active Bleeding
Uncharacterised lesions

- ? CEUS
- MRI
Liver MRI Protocol

Pre – Contrast sequences

• FISP (GRASS/FFE/SARGE)

• In/Op Ph GRE T1

• HASTE (SS-FSE/FSE-ADA/FASE) alternatively FSE T2 with Long and Short TE some use FSE-STIR
In/Op T1

Chemical Shift Imaging

GRE T1

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2 Point Dixon

In Ph
Fat Only
Ie Water Sat

Op Ph
Water Only
Ie Fat Sat

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2 Point Dixon

In Ph

Op Ph

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2 Point Dixon

In Ph
Op Ph
Loss of signal
Fat & water same voxel

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Focal Fat

CT

In Ph T1

Op Ph T1

Still give contrast – could be Adenomatosis

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T2 – HASTE
(SS-FSE/FSE-ADA/FASE)

• This sequence is for characterisation not detection
T2 – HASTE
(SS-FSE/FSE-ADA/FASE)
Long TE T2FSE v HASTE for small lesions (SS-FSE, FSE-ADA, FASE)

TE 180msec  HASTE+FS
Contrast Agents

Non-specific Extracellular Contrast Agents
  – Gadolinium Chelates..........................T1

  – Versatile can be used to address most characterisation issues
  – Can be used with liver specific contrast agents
  – Contrast agent of choice to characterise lesions bright on T2
Extracellular Contrast Agents

Dynamic Gadolinium Enhanced T1

• 3D T1 GRE Fat Sat
  (VIBE, LAVA, FAME, THRIVE)
  – Pre
  – Arterial Phase (typically 12 sec)
  – Portal Venous Phase (40-50 sec)
  – Equilibrium Phase
  – (DWI)
  – Delayed (10 min) – not routine
Dynamic Imaging Options

• Contrast timing
  – Fixed Timing
  – Test bolus
    • time to peak aortic enhancement
    • Delay time
t(peak) + duration Gd inj – t(k)
  – Bolus tracking
Dynamic Imaging Options

• Contrast timing

  – Test bolus
    • time to peak aortic enhancement
    • Delay time
t (peak) + duration Gd inj – t (k)

  – Bolus tracking
Bolus Tracking

Threshold triggered with manual failsafe

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Haemangioma

HASTE  Art Ph  PV Ph  Equ Ph

3D GRE T1 FS Gad

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Haemangiomas

- Most Radiologists Familiar With Typical Features
- Commonest non-cystic benign liver lesion
- Often multiple
- Often found with other benign lesions
- More common in females
Central Scar - MR

HASTE  Op Ph T1  PV Ph
“Flash Filling” Early Sustained Enhancement

HASTE  Art Ph  PV Ph  10min

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Sclerotic Haemangioma

HASTE  Art Ph  PV Ph  10 min

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Haemangioma

FSE T2  Art Ph  PV Ph  Eq Ph  10 Min

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Peri-lesional shunting

Art Ph  PV Ph  Equ Ph

Gad

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High T2 lesions

HASTE    GRE T1

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Cyst & Colorectal Metastases

Colorectal - Vascular & Necrotic High Signal T2

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• Simple Cysts
  – Hepatic (Bile Duct) Cysts
  – Polycystic Liver Disease
  – Biliary Hamartomas
  – Caroli’s Disease
  – Parabiliary Cyst

• Complex Cysts
  – Foregut Ciliated Cysts
  – Hydatid
  – Biliary Cystadenoma/ Cystadenocarcinoma
  – Pyogenic Abscesses
  – Mesenchymal Hamartomas
  – Embryonal Sarcomas
  – Cystic Metastases
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  – Embryonal Sarcomas
  – Cystic Metastases
Biliary Cystadenoma

HASTE  OpPh T1  Gd

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Haemorrhage into simple cyst

HASTE

In Ph GRE T1
Ciliated Hepatic Foregut Cyst

HASTE  In Ph GRE T1

Beware segment 4

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Complexity of cystic lesions underestimated by CT

PV MDCT

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Biliary Cystadenoma

US

PV MDCT

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Biliary Cystadenoma

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Typically incidental finding in a young female

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MR Contrast Agent

Liver Specific Agent

• Hepatocyte
  – Gd - BOPTA
  – Gadoxetic Acid (Gd – EOB)

• Best option for lesions not bright on T2
Hepatocyte Specific Contrast Agents

Gadoxetic acid (Gd-EOB, Primovist)

- Bi-phasic imaging properties,
  1. ECF – perfusion – behaves like “regular Gad”
  2. Liver Specific Properties
    - Hepatocyte uptake (via MOAT – Oatp-8,1,B1, B3)
    - Excretion into bile (via MRP2)
    - Excretion into sinusoidal space (via MRP3)
- 50% taken up via liver – excreted in bile
- Optimum imaging at 20 mins
  (in non-cirrhotic can image at 10 mins)
Liver Specific Contrast Agents

3D T1 GRE Fat Sat
(VIBE, LAVA, FAME, THRIVE
– Pre
– Arterial Phase (typically 12 sec)
– Portal Venous Phase (40-50 sec)
– “Equilibrium” Phase
– (DWI)
– Hepato-biliary Phase
  10 - 20 min Gadoxetic Acid (Gd-EOB)
  1-3 hrs Gadobenate (Gd BOPTA)
Gadoxetic Acid

UnE  Art Ph  PV Ph  “Equ”  HB Ph 10min Hyperintense

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Gadoxetic Acid

Focal Nodular Hyperplasia

UnE  Art Ph  PV Ph  “Equ”  HB Ph

10min Hyperintense

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BOPTA

Art Ph  PV Ph  Equ Ph  HB Ph
Focal Nodular Hyperplasia  2 Hr  Isointense

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Focal Nodular Hyperplasia

- mean size 4cm (range 1-11cm)
- multiple lesions 20%
- Associated with other benign lesions
  - Haemangiomas – 20-23%
  - Adenomas – 3.6%
- Rarely reported in the elderly
- Men encountered later & less typical
MR – typical features

- Iso- or hypointensity T1
- Iso – or slightly hyperintense T2
- Homogenous SI
- Hypervascularity – arterial phase
- Central hyperintense scar T2 which accumulates Gd on T1 delayed phase
- No capsule
- Uptake of Gadoxetic Acid/BOPTA
Focal Nodular Hyperplasia

- Many Lesions - no scar
- Unusual features
- Fat detectable on Chemical Shift Imaging
- Occasionally hypointense T2, hyperintense T1
- Non-enhancement of scar (low signal - T2)
- Brighter than background liver after Art Ph
- Pseudo-capsule (ring enhancement)
- Lack of hypervascularity described

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Delayed Enhancing Scar FNH

HASTE
Art Ph
10 min Delay
Conventional Gadolinium Chelates

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Focal Nodular Hyperplasia

T2  Op Ph  In Ph

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Focal Nodular Hyperplasia

Art Ph  PV Ph  HB Ph

Gadoxetic Acid

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Focal Nodular Hyperplasia

Art Ph  PV Ph  Delayed

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Differentiation of FNH and Adenoma with BOPTA/Gadoxetic Acid

1-3 hours after contrast:

124/128 (97%) of FNH hyper- or isointense
107/107 (100%) of adenomas hypointense
(Malignant lesion also hypointense)

_Grazioli et al. Radiology 2005; 236: 166-177_

90% FNH uptake Gadoxetic Acid 20 min

_Zech et al. Investig Radiol 2008;43:504-11_
FNH - Large Scar

Art Ph  PV Ph  Gadoxetic Acid 10min
Differential Diagnosis

Op Ph T1

T2

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Hepatocellular Adenoma

Art Ph
PV
HB Ph

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Hepatocellular Adenoma

Op Ph T1

T2

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Hepatocellular Adenoma

Subtypes

• Steatotic (HNF1A- mutated) (H-HCA)
• β-Catenin mutated (b-HCA)
• Inflammatory (IHCA)
  – Telangiectatic FNH/Adenoma
  – 10% IHCA also β-Catenin mutated
• Unclassified

*Bioulac-Sage P et al Semin Liv Dis 2011;31: 91*

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Steatotic (HNF1A- mutated)
Steatotic (HNF1A- mutated)
Inflammatory

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T2

In Ph T1

DWI
“Atoll” Sign

van Aalten S M et al.
Radiology 2011;261:172-181

HASTE
In Ph T1

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Inflammatory HCA

Unenhanced  Art Ph  PV Phase  10 min

Gadoxetic Acid

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Inflammatory HCA

Art Ph  PV Ph  Equ Ph

Gadolinium

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Inflammatory Adenoma

T2  Op Ph T1  In Ph T1

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Inflammatory Adenoma

Pre Art Ph PV Ph HPB Ph

T1 Fat Sat EOB - Gd

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• In most cases can make a confident diagnosis
• Small group indeterminate
  – Watch
  – Biopsy
  – Resect
• Choice based on:
  – Relative uncertainty
  – Resectability of the lesion
    location, fitness, patient preference……..
Conclusions

• A non-invasive diagnosis can be made in most cases using MRI
• Conventional Gadolinium Chelates are often the agent of choice especially if high signal on T2 or unsure in the non-cirrhotic liver
• Use EOB – Gd or BOPTA if likely to be a hepatocellular lesion
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