Surgical conditions of liver

Somkit Mingphruekedi, M.D.
Division of HPB Surgery, Department of Surgery
Ramathibodi Hospital
Anatomy

- Hepatic Artery
- IVC
- Splenic Vein
- Portal Vein
- Gallbladder
- CBD
- SMV
Surgical Anatomy
The Brisbane 2000 terminology of hepatic anatomy and resections

HPB. 2000;2:333-339
Liver Functions

Nutrition/Metabolic
- stores glycogen (glucose chains)
- releases glucose when if no insulin
- absorbs fats, fat soluble vitamins
- manufactures cholesterol

Bile Salts
- lipids derived from cholesterol
- dissolves dietary fats (detergent)

Bilirubin
- breakdown product of haemoglobin
Liver Functions

Clotting Factors
- manufactures most clotting factors

Immune function
- Kupfer cells engulf antigens (bacteria)

Detoxification
- drug excretion (sometimes activation)
- alcohol breakdown

Manufactures Proteins
- albumin
- binding proteins
Symptoms

Early disease
• asymptomatic
• fatigue, malaise
• anorexia, nausea
• jaundice
• pruritis
• easy bruising and bleeding
• abdominal pain

Cholestatic patients
• fatigue, malaise
• anorexia, nausea
• jaundice
• +++ pruritis
• +++ grey or clay-coloured stools
Disease Progression

**Acute Liver Failure**
- <6 weeks duration
- Jaundice
- Encephalopathy
- Cerebral edema
- Acute renal failure
- Acidosis
- Hypoglycaemia
- MOF

**Chronic Liver Disease**
- >6 months

Cirrhosis leading to
- Recurrent decompensation
  - Ascites
  - Portal Hypertension (variceal bleeding)
  - Encephalopathy
- Low albumin/Malnutrition
- Hepatorenal syndrome
- Hyponatraemia
- Hepatoma
Cirrhosis
Causes of Chronic Liver disease

- **Viral**
  - Hepatitis B
  - Hepatitis C

- **Autoimmune Hepatitis**

- **Metabolic**
  - NASH
  - Amyloid

- **Alcoholic Cirrhosis**

- **Inherited**
  - Haemochromatosis
  - Wilsons Disease
  - \(\alpha-1\) Antitrypsin Deficiency

- **Biliary Disease**
  - PBC
  - PSC
  - Secondary sclerosing cholangitis
  - Caroli’s syndrome
Signs of Chronic Liver Disease

- None
- Asterixis/Flap
- Relative hypotension
- Oedema
- Jaundice/No jaundice
- Large/Small liver
- Splenomegaly
- Gynecomastia
- Testicular atrophy-loss of secondary sexual characteristics
Child-Pugh score

• The Child-Pugh score is used to assess the prognosis of chronic liver disease, mainly cirrhosis. To determine treatment required and the necessity of liver transplantation.

• The score employs five clinical measures of liver disease. Each measure is scored 1-3, with 3 indicating most severe derangement.
### Assessment of hepatic reserve

**Child – Pugh scoring system**

<table>
<thead>
<tr>
<th>Points</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ascites</td>
<td>None</td>
<td>Small or diuretic controlled</td>
<td>Tense</td>
</tr>
<tr>
<td>Encephalopathy</td>
<td>Absent</td>
<td>State I–II</td>
<td>State III–IV</td>
</tr>
<tr>
<td>Albumin (g/L)</td>
<td>&gt; 3.5</td>
<td>2.8–3.5</td>
<td>&lt; 2.8</td>
</tr>
<tr>
<td>Bilirubin (mg/dL)</td>
<td>&lt; 2</td>
<td>2–3</td>
<td>&gt; 3</td>
</tr>
<tr>
<td>PT(sec above control)</td>
<td>&lt; 4</td>
<td>4–6</td>
<td>&gt; 6</td>
</tr>
<tr>
<td>INR</td>
<td>&lt; 1.7</td>
<td>1.7–2.3</td>
<td>&gt; 2.3</td>
</tr>
</tbody>
</table>

( Class A, 5–6 total points; Class B, 7–9 total points; Class C, 10–15 total points )
Decompensation in Cirrhosis

Development of:

• Ascites
• Hepatic Encephalopathy
• Portal hypertension (variceal haemorrhage)
Development of Ascites

50% of compensated cirrhotics develop ascites over 10 yrs

50% of cirrhotics with ascites will die within 2 yrs
Portal Circulation
Esophageal varices
Management of Bleeding Varices

- Prevention - Propranolol, Band Ligation
- Resuscitation
- Endoscopy - Band Ligation, Sclerotherapy
- Pharmacotherapy - Octreotide, Terlipressin, Propranolol
- Balloon Tamponade
- Transjugular Intrahepatic Portosystemic Shunt (TIPS)
Variceal Ligation
Variceal Band Ligation
Sengstaken-Blakemore Tube
TRANSJUGULAR INTRAHEPATIC PORTAL SYSTEMIC SHUNT

- Inferior vena cava
- Shunt
- Hepatic vein
- Esophagus
- Liver
- Stomach
- Varices
- Spleen
- Portal vein
- Splenic vein
- Superior mesenteric vein
- Coronary vein
Common Liver Lesions

Non-emergent

1. Liver cancers
   - Primary
   - Secondary
2. Benign liver tumors
   - Hemangiomas
   - Adenomas
   - Focal nodular hyperplasia
   - Simple cysts incl PCLD
   - Cystadenoma
   - Bile duct adenoma
   - Hamartomas

Emergent

1. Liver abscess
2. Cholangitis from cholangiocarcinoma
3. Ruptured liver tumors
Liver abscess

Pyogenic abscess

Amebic liver abscess
Pyogenic liver abscess

Clinical features

• Right upper quadrant pain and tenderness
• Nocturnal fevers and sweats
• Anorexia and weight loss
• Raised right hemidiaphragm in chest radiograph
• Raised white cell count and erythrocyte sedimentation rate with mild anemia
Etiology of pyogenic liver abscess

• Biliary tract
  Gall stones
  Cholangiocarcinoma
  Strictures

• Portal vein
  Appendicitis
  Diverticulitis
  Crohn's disease

• Hepatic artery
  Dental infection
  Bacterial endocarditis

• Direct extension of:
  Gall bladder empyema
  Perforated peptic ulcer
  Subphrenic abscess

• Trauma

• Iatrogenic
  Liver biopsy
  Blocked biliary stent

• Cryptogenic

• Secondary infection of liver cyst
Etiology of pyogenic liver abscess

Microbiology

Most patients presenting with pyogenic liver abscesses have a polymicrobial infection usually with Gram negative aerobic and anaerobic organisms.

Most organisms are of bowel origin, with *Escherichia coli*, *Klebsiella pneumoniae*, bacteroides, enterococci, anaerobic streptococci, and microaerophilic streptococci being most common.

Staphylococci, haemolytic streptococci, and *Streptococcus milleri* are usually present if the primary infection is bacterial endocarditis or dental sepsis.
Pyogenic liver abscess

**Treatment**

Antibiotics should include *penicillin, an aminoglycoside, and metronidazole*. In elderly people and those with impaired renal function a *third generation cephalosporin* should be used instead of an aminoglycoside. The regimen should be modified after culture has identified the infective organism.

Treatment is continued for two to four weeks

Antibiotics alone are effective in only a few patients, and most patients will require *percutaneous aspiration or catheter drainage* guided by ultrasonography or computed tomography.
Amebic liver abscess

*Entamoeba histolytica*

The abscess is usually solitary and affects the right lobe in 80% of cases.

The abscess contains sterile pus and reddish-brown ("anchovy pus") liquefied necrotic liver tissue. Amoebae are occasionally present at the periphery of the abscess.
Amebic liver abscess

Symptoms of amoebic liver abscess

• Pain
• Enlarged liver with maximal tenderness over abscess
• Intermittent fever
• Night sweats
• Weight loss
• Nausea
• Vomiting
• Cough
• Dyspnea
Amoebic liver abscess
Amebic liver abscess

Treatment

Ninety five per cent of uncomplicated amoebic abscesses resolve with metronidazole alone.

Percutaneous drainage

Surgical drainage
Ruptured Amebic liver abscess with Empyema Thoracis
Liver Tumors
Classification

Benign

• Hemangioma
• Focal nodular hyperplasia
• Adenoma
• Liver cysts

Malignant

1. Primary liver cancers
   • Hepatocellular carcinoma
   • Intrahepatic cholangiocarcinoma

2. Metastases
Hepatic Hemangioma

• The commonest liver tumor
• 5% of autopsies
• Usually single small
• Well demarcated capsule
• Usually asymptomatic
Hepatic Hemangioma

**Diagnosis**

- US: echogenic spot, well demarcated
- CT: venous enhancement from periphery to center
- MRI: hypointense in T1, hyperintense in T2
- No need for FNA

**Treatment**

- No need for treatment
Hemangioma
Giant Hepatic Hemangioma
Focal Nodular Hyperplasia (FNH)

Clinical Features

• Benign nodule formation of normal liver tissue
• Central stellate scar
• More common in young and middle age women
• No relation with sex hormones
• Usually asymptomatic
• May cause minimal pain
Focal Nodular Hyperplasia (FNH)

Diagnosis
• US: Nodule with varying echogenicity
• CT: Hypervascular mass with central scar
• MRI: iso or hypo intense
• FNA: Normal hepatocytes and Kupffer cells with central core.

Treatment
• No treatment necessary
• Pregnancy and hormones OK
Hepatic Adenoma

Clinical features

• Benign neoplasm composed of normal hepatocytes, no portal tract, central veins, or bile ducts

• More common in women

• Associated with contraceptive hormones

• Usually asymptomatic but may have RUQ pain

• May presents with rupture, hemorrhage, or malignant transformation (very rare)
Hepatic Adenoma

Diagnosis
- US: filling defect
- CT: Diffuse arterial enhancement
- MRI: hypo or hyper intense lesion
- FNA: may be needed

Treatment
- Stop hormones
- Observe every 6m for 2 y
- If no regression then surgical excision
Liver Cysts

- May be single or multiple
- May be part of polycystic kidney disease
- Patients often asymptomatic
- No specific management required
- Hydatid cyst
Malignant Liver Lesions

Hepatocellular Carcinoma
Cholangiocarcinoma
Liver Metastases