THE LIVER
Lobar and segmental anatomy
Vascular anatomy (dual blood supplies)
- Pyogenic liver abscess
- Amebic liver abscess
- Pyogenic liver abscess
- Amebic liver abscess
Liver invasion by bacteria:

- Ascending infection in the biliary tract
- Vascular seeding (portal or arterial)
- Direct invasion from a nearby source
- Traumatic implantation
### Etiology

#### Origins and causes of pyogenic liver abscess

<table>
<thead>
<tr>
<th>Cause</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Biliary tract</strong></td>
<td>Lithiasis</td>
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<tr>
<td></td>
<td>Cholangiocarcinoma</td>
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<td></td>
<td>Strictures</td>
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<td></td>
<td>Biliary-enteric anastomosis</td>
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<tr>
<td></td>
<td>Biliary procedures</td>
</tr>
<tr>
<td><strong>Portal vein</strong></td>
<td>Appendicitis</td>
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<tr>
<td></td>
<td>Diverticulitis</td>
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<tr>
<td></td>
<td>Crohn's disease</td>
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<tr>
<td><strong>Hepatic artery</strong></td>
<td>Bacterial endocarditis</td>
</tr>
<tr>
<td></td>
<td>Dental infection</td>
</tr>
<tr>
<td><strong>Direct extension</strong></td>
<td>Gall bladder empyema</td>
</tr>
<tr>
<td></td>
<td>Perforated peptic ulcer</td>
</tr>
<tr>
<td></td>
<td>Subphrenic abscess</td>
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<tr>
<td><strong>Trauma</strong></td>
<td>Abdominal trauma</td>
</tr>
<tr>
<td></td>
<td>Chemoembolization</td>
</tr>
<tr>
<td></td>
<td>Ethanol injection or radiofrequency ablation</td>
</tr>
<tr>
<td><strong>Cryptogenic</strong></td>
<td></td>
</tr>
</tbody>
</table>
Depended on cause of abscess

- **Biliary**
  - gram-negative aerobic bacilli and enterococci

- **Pelvic/intraperitoneal**
  - Mixed aerobic and anaerobic (*Bacteroides fragilis*)

- **Hematogenous/trauma**
  - Single (*Staph aureus, Strep*)
Clinical manifestation

Fever (90%)
RUQ, epigastric pain

Jaundice (25%)
Chills
Anorexia
Weight loss
Nausea, vomiting
Weakness, malaise
Clinical manifestation

↑ alkaline phosphatase (80%)
↑ bilirubin (20-50%)

leukocytosis (70-90%)

CXR
- R hemidiaphragm elevation
- R basilar infiltrate
- Unilateral pleural effusion
1. Imaging
   - Ultrasound: initial test
   - CT: If suspected intraabdominal pathology
   - MRI: no benefit over CT scan

2. Microbial cultures (aerobic, anaerobic)
   - Aspiration

3. Serology
   - R/O Amebic
1. Abscess management
2. Underlying cause management
1. Abscess management

(1) IV ATB

3rd gen cephalosporins + metronidazole

or ampicillin + gentamicin + metronidazole

x 10-14 days then

continue oral ATB until 4-6 weeks

(2) Drain abscess

- Percutaneous drainage**

- Percutaneous needle aspiration

- Surgical drainage
PCD success rate > PNA

But PNA may consider in unilocular abscess < 5 cm

Surgery
1. Tx primary pathologic process
2. Failed PCD
- Pyogenic liver abscess
- Amebic liver abscess
Entamoeba histolytica

Trophozoite

Cyst
Pathogenesis

1. Cysts

2. Cysts are consumed by man

3. In the small intestine, the cyst wall disintegrates and trophozoites are released

4. Invasive trophozoite

5. Invasion to mesenteric vessels

6. Into the portal circulation

7. Rupture of abscess may cause:
   - Empyema
   - Bronchohepatic fistulae
   - Pericarditis
   - Peritonitis

8. Hematogenous spread:
   - Lungs
   - Spleen
   - Brain
   - Kidneys
Clinical manifestation

Symptoms like pyogenic liver abscess but delay onset and less sepsis

Male : female = 10 : 1, Age < 50 yrs

1/3 diarrhea but 70% of pt : stool exam neg

CBC : leucocytosis without eosinophilia
LFT : elevate ALP, transaminases
Diagnosis

1. circulating amebic antibody (sens&spec 95%)
   +

2. Imaging study: USG

CT scan when
No response to medication
Diagnosis uncertain
Suspicious complication
**Metronidazole** 750 mg po tid x 10 days

**Intraluminal amebicidal agents**
(Iodoquinol, paromomycin, diloxanide furoate)

PNA not indicate in every patients but selected in
1. abscess > 5 cm
2. abscess at Lt lobe liver
3. secondary bacterial infection
4. clinical not improve in 3-5 days after medication
5. amebic cytology inconclusive
   (cannot R/O pyogenic)
6. pregnancy
   (contraindication for metronidazole)
<table>
<thead>
<tr>
<th></th>
<th>PLA</th>
<th>ALA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age ( yr)</td>
<td>&gt; 50</td>
<td>20-40</td>
</tr>
<tr>
<td>M : F</td>
<td>2 : 1</td>
<td>10 : 1</td>
</tr>
<tr>
<td>Solitary</td>
<td>50 %</td>
<td>&gt; 80 %</td>
</tr>
<tr>
<td>Location</td>
<td>right lobe</td>
<td>right lobe</td>
</tr>
<tr>
<td>Cause</td>
<td>biliary tract infection</td>
<td>GI portal vein</td>
</tr>
<tr>
<td>Organism</td>
<td>bacteria</td>
<td>ameba</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>history, imaging aspiration, hemoculture</td>
<td>imaging, serology</td>
</tr>
<tr>
<td>Treatment</td>
<td>antibiotic + drainage</td>
<td>amebicidal</td>
</tr>
</tbody>
</table>
Benign liver tumors
<table>
<thead>
<tr>
<th>Tissue Type</th>
<th>Benign Liver Tumor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatocyte</td>
<td>hepatic adenoma</td>
</tr>
<tr>
<td></td>
<td>focal nodular hyperplasia</td>
</tr>
<tr>
<td>Mesenchymal tissue</td>
<td>hamartoma</td>
</tr>
<tr>
<td></td>
<td>hemangioma</td>
</tr>
<tr>
<td>Heterotopic tissue</td>
<td>adrenal rest</td>
</tr>
<tr>
<td></td>
<td>pancreatic rest</td>
</tr>
</tbody>
</table>
Cavernous hemangioma
The most common benign tumor of liver

Mean age 50 years; female predominant

“Giant Hemangioma” – size $\geq 10$ cm

Pathogenesis – not well understood
Hepatic Hemangioma

Arterial Phase  Portal Phase  Delayed Phase

ART  PORT  EQUIL
Asymptom

Symptom:
- Rupture
- Compression
- Kasabach Merritt syndrome
“Whatever the size, there is no treatment for asymptomatic hemangioma”

Rare growth and complication
Indications for surgical resection

- Severe abdominal pain
- Rapid enlargement of tumor
- Rupture or potential rupture
- Indeterminate diagnosis
- Complication

Surgery: enucleation, liver resection or liver transplantation
Focal nodular hyperplasia
Benign tumor like lesion

Hyperplasia/regeneration process

No malignant potential

Rarely complication
Asymptomatic pt: no Tx

**Indication for surgery**
- Indeterminate diagnosis
- Severe abdominal pain (R/O other causes)

Prefer resection > enucleation
Hepatic adenoma
Associated oral contraceptive use

Risk of hemorrhage or rupture ~20-40%

Increase risk of bleeding
  - Women taking oral contraceptives
  - During pregnancy
  - Tumor >4-5 cm
Risk of malignant transformation – 10%
Hepatic Adenoma

Arterial Phase  Portal Phase  Delayed Phase

Art  Port  Equil
**Treatment**

- **size < 4 cm**  
  - Cessation of pill + avoidance
  - Pregnancy

**indication for surgery**

- **size > 4 cm**
- Don’t shrink after cessation of pill
- Can’t stop pill
- Plan become pregnancy
- Rupture

**Procedure of choice = resection**
Simple hepatic cyst
Hepatic Cyst

Arterial Phase  Portal Phase  Delayed Phase
Asymptomatic : F.U.

Tx when only symptomatic pt

aspiration only  recurrence 100 %

Choice of treatment

1. PAIR (percutaneous aspiration instillation and reaspiration)

2. Unroofing or fenestration

3. Cystectomy or hepatectomy: rarely required
Biliary cystadenoma
Biliary Cystadenoma

Arterial Phase  Portal Phase  Delayed Phase
multi-loculation, internal septation, calcification, papillary projection, thick nodular wall.
Cystadenoma : malignant 25 %

Tx of choice : resection
- Hepatocellular carcinoma
- Cholangiocarcinoma
- Metastatic liver tumor
Hepatocellular carcinoma
Risk factors

- Liver cirrhosis
- Hepatitis B, C
- Aflatoxin
- Alcohol
- Hemochromatosis
- Wilson’s disease
Clinical manifestation

- Symptom of cirrhosis
- Abdominal pain/mass, jaundice, fever
- Asymptom but incidental findings
Diagnosis
AASLD
(American Association for the Surgery of Liver Disease)

Diagnosis guided by the size of the lesion
Lesion < 1 cm in diameter
- Follow with US q 3 – 6 months x 2 yrs
  - Stable lesion → revert to routine surveillance
  - Enlarging lesion → proceed according to size

Lesion 1 – 2 cm in diameter
- 2 dynamic imaging studies
  - Typical HCC x 2 technique → Dx HCC
  - Others → liver biopsy
Lesion > 2 cm in diameter

- **One dynamic imaging**
- Typical features of HCC → Diagnosis
- AFP > 200 ng/mL → Diagnosis
- Atypical vascular pattern → Biopsy
Mass on surveillance ultrasound in a cirrhotic liver

- <1 cm
  - Repeat US at 3- to 4-month intervals
    - Coincidental typical vascular pattern on dynamic imaging
      - Stable over 18–24 months
        - Return to standard surveillance protocol (6–12 monthly)
      - Enlarging
        - Proceed according to lesion size
    - Typical vascular pattern with one technique
      - Biopsy
        - Diagnostic of HCC
          - Repeat biopsy or imaging follow-up
            - Change in size/profile
              - Repeat imaging and/or biopsy
                - Positive
                  - Treat as hepatocellular carcinoma
                - Negative
  - 1–2 cm
    - Two dynamic imaging studies
      - Atypical vascular pattern with both techniques
        - Biopsy
          - Nondiagnostic
            - Other diagnosis
              - Repeat biopsy or imaging follow-up
                - Change in size/profile
                  - Repeat imaging and/or biopsy
                    - Positive
                      - Treat as hepatocellular carcinoma
                    - Negative
      - Atypical vascular pattern
        - Biopsy
          - Other diagnosis
            - Repeat biopsy or imaging follow-up
              - Change in size/profile
                - Repeat imaging and/or biopsy
                  - Positive
                    - Treat as hepatocellular carcinoma
                  - Negative
            - Other diagnosis
              - Repeat biopsy or imaging follow-up
                - Change in size/profile
                  - Repeat imaging and/or biopsy
                    - Positive
                      - Treat as hepatocellular carcinoma
                    - Negative
    - Typical vascular pattern on dynamic imaging
      - Biopsy
        - Nondiagnostic
          - Other diagnosis
            - Repeat biopsy or imaging follow-up
              - Change in size/profile
                - Repeat imaging and/or biopsy
                  - Positive
                    - Treat as hepatocellular carcinoma
                  - Negative
          - Other diagnosis
            - Repeat biopsy or imaging follow-up
              - Change in size/profile
                - Repeat imaging and/or biopsy
                  - Positive
                    - Treat as hepatocellular carcinoma
                  - Negative
      - Other diagnosis
        - Repeat biopsy or imaging follow-up
          - Change in size/profile
            - Repeat imaging and/or biopsy
              - Positive
                - Treat as hepatocellular carcinoma
              - Negative


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Treatment
Treatment

The Barcelona-Clinic- Liver-Cancer (BCLC) staging system

Includes variables related to

- Tumor stage (Okuda)
- Liver functional status (Child’s pugh)
- Physical status (WHO)
<table>
<thead>
<tr>
<th>Stage</th>
<th>Tumor Size</th>
<th>Ascites</th>
<th>Albumin</th>
<th>Bilirubin</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>&gt; 50% (+)</td>
<td>( + )</td>
<td>( - )</td>
<td>&gt; 3 g/dl (+)</td>
</tr>
<tr>
<td></td>
<td>&lt; 50% (-)</td>
<td>( - )</td>
<td>( - )</td>
<td>&gt; 3 g/dl (-)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>( - )</td>
<td>&gt; 3 mg/dl (+)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&lt; 3 mg/dl (-)</td>
</tr>
<tr>
<td>II</td>
<td></td>
<td>1 or 2 (+)</td>
<td></td>
<td></td>
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<tr>
<td>III</td>
<td></td>
<td>3 or 4 (+)</td>
<td></td>
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</tr>
</tbody>
</table>
# Child’s pugh classification

<table>
<thead>
<tr>
<th>Clinical or laboratory feature</th>
<th>1 point</th>
<th>2 points</th>
<th>3 points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Encephalopathy (grade)</td>
<td>0 (absent)</td>
<td>1 – 2</td>
<td>3 – 4</td>
</tr>
<tr>
<td>Ascites</td>
<td>Absent</td>
<td>Slight</td>
<td>Poorly</td>
</tr>
<tr>
<td>Bilirubin (mg/dl)</td>
<td>&lt; 2.0</td>
<td>2.0 – 3.0</td>
<td>&gt; 3.0</td>
</tr>
<tr>
<td>Albumin (g/dl)</td>
<td>&gt; 3.5</td>
<td>2.8 – 3.5</td>
<td>&lt; 2.8</td>
</tr>
<tr>
<td>INR</td>
<td>&lt; 1.7</td>
<td>1.7 – 2.2</td>
<td>&gt; 2.3</td>
</tr>
</tbody>
</table>

Each feature is assigned 1, 2, or 3 points.
Class A: 5 – 6 points; Class B: 7 – 9 points; Class C: 10 – 15 points
<table>
<thead>
<tr>
<th>Stage 0</th>
<th>Fully active, normal life, no symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1</td>
<td>Minor symptoms, able to do light activity</td>
</tr>
</tbody>
</table>
| Stage 2 | Capable of self-care but unable to carry out work activities  
Up for more than 50% waking hours |
| Stage 3 | Limited self care capacity  
Confined to bed or chair > 50% waking hours |
| Stage 4 | Completely disabled  
Confined to bed or chair |
Curative treatment

1. Resection
2. Liver transplantation
3. Radiofrequency ablation
Cholangiocarcinoma
Risk factors

- Primary sclerosing cholangitis
- Choledochal cyst
- Hepatolithiasis
- Liver flukes
- Prior biliary-enteric anastomosis
- Toxic substances: nitric oxide
  : nitrosamine
  : thorotrast
- Congenital hepatic fibrosis
Clinical manifestation

- Fever, anorexia, weight loss, abdominal mass

- “cholestatic jaundice”

- Progressive jaundice, dark urine, pale stool, pruritus
Diagnosis

LFT: direct hyperbilirubinemia, elevated ALP

USG: intrahepatic mass, bile duct dilatation

↓

CT scan or MRI
+ tumor marker CA 19-9
Treatment

Mainstay: Liver resection

Other modalities for noncurative treatment but poor outcome
Gallstone diseases
Gallstone disease

Female : male = 3:1

**Risk factors**

<table>
<thead>
<tr>
<th>Factor</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pregnancy</td>
<td>Terminal ileal resection</td>
</tr>
<tr>
<td>Obesity</td>
<td>Gastric surgery</td>
</tr>
<tr>
<td>Crohn's disease</td>
<td>Hemolytic anemia</td>
</tr>
</tbody>
</table>
Bile = bile salts, phospholipids, cholesterol

Gallstones due to imbalance rendering cholesterol & calcium salts insoluble

Pathogenesis involves 3 stages:
  1. cholesterol supersaturation in bile
  2. crystal nucleation
  3. stone growth
Asymptomatic gallstone
3% risk of developing symptoms/ year

2/3 will remain symptom free at 20 years

No require treatment
Study from Cochrane review 2007

No RCT/meta-analysis about treatment of asymptomatic gallstone
Some indication for prophylactic cholecystectomy

1. Gallstone > 3 cm
2. Calcified (porcelain) gallbladder
3. Gallbladder polyp > 1 cm
4. Splenectomy in hemolytic anemia patient
5. Bariatric surgery
6. Long term TPN
7. Transplant patient needed
8. No access to medical care
Chronic cholecystitis
Recurrent attack of biliary colic from recurrent cystic obstruction (from stone)

Aggravate by fatty meal/supine position when sleep

Diagnosis: US
Gallstone
Treatment

Procedure of choice:

Laparoscopic cholecystectomy
Acute cholecystitis
- Persistent cystic duct obstruction leads to GB distension, wall inflammation & edema

- Can lead to: empyema, gangrene, rupture
Epigastrium/RUQ pain >24hr

Radiate to interscapular or Rt scapular area

Anorexia, nausea/vomiting and fever

Palpable/tender or even visible RUQ mass

Positive Murphy's sign
( inspiratory arrest with deep palpation in the right subcostal area)
Lab:
Moderate leucocytosis
(WBC 12,000-15,000 cells/mm³)

If WBC > 20,000 cells/mm³: complicated cholecystitis

Mild abnormal LFT, jaundice no more than 4 mg/dl
Diagnosis

Study of choice = US

Gallstones, sonographic Murphy’s sign
gallbladder wall thickening, distended
gallbladder, pericholecystic fluid
Treatment

- NPO
- IV fluid resuscitation
- IV ATB cover gram neg aerobe/anaerobe (3rd generation cephalosporins with good anaerobic coverage or 2nd generation cephalosporins plus metronidazole)
Every patient must have surgery except medically unfit patients.

**Timing of surgery**

Early VS. delayed cholecystectomy

(early = Sx in 3 days,
  delayed = Sx in 6-10 wks after disease)
If patients present within 3 days of onset prefer early cholecystectomy

If patients present > 3 days of onset prefer delayed cholecystectomy except in unresponse to ATB or complicated cholecystitis

Medically unfit patients consider cholecystostomy and delayed cholecystectomy if patients recover and fit for surgery
Choledocholithiasis
6-12% of gallstone patients

Classified into

(1) Primary CBD stone
(2) Secondary CBD stone
Clinical manifestation

Obstructive jaundice, cholangitis, gallstone pancreatitis
<table>
<thead>
<tr>
<th><strong>Diagnosis</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>LFT</strong> : direct hyperbilirubinemia, elevated ALP</td>
</tr>
<tr>
<td><strong>US</strong> : CBD obstruction, proximal BD dilatation</td>
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<tr>
<td></td>
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<tr>
<td>------</td>
</tr>
<tr>
<td>TUS</td>
</tr>
<tr>
<td>CT</td>
</tr>
<tr>
<td>ERCP</td>
</tr>
<tr>
<td>MRCP</td>
</tr>
<tr>
<td>EUS</td>
</tr>
<tr>
<td>IOC</td>
</tr>
<tr>
<td>Clinical presentation</td>
</tr>
<tr>
<td>-----------------------</td>
</tr>
<tr>
<td>Ultrasound</td>
</tr>
<tr>
<td>Serum biochemistries</td>
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</table>

- **Risk of CBD stones**: 93%, 32%, 4%, 1%
Cholangitis
Pathogenesis

Bacteribilia + intraductal hypertension
Microbiology

*Escherichia coli*
*Klebsiella pneumoniae*
*Streptococcus faecalis*
*Bacteroides fragilis*
1. Fever with chills
2. RUQ pain
3. Jaundice
4. Charcot’s triad

5% of patients = acute toxic cholangitis
(Charcot’s triad + hypotension + mental status change = Reynold’s pentad)
Diagnosis

CBC: leucocytosis

LFT: hyperbilirubinemia, elevation of ALP and transaminases

Initial imaging: US

Definite diagnosis: ERC or PTC

If suspected cancer: CT or MRI
Treatment

- NPO
- IV fluid resuscitation
- IV ATB
- ERCP with drainage or PTBD

ATC: emergency biliary drainage
Malignant neoplasm
- Gallbladder carcinoma
- Extrahepatic cholangiocarcinoma
Gallbladder carcinoma
Incidence
- 5th common GI cancer
- elderly ( > 70 yrs)
- F : M = 2-3 : 1
- 70-90 % +ve GS
- < 0.5% GS +ve GB cancer
- adeno. CA. 90%
Risk factors

1) large GS > 3 cm
2) calcified GB wall ( porcelain GB )
3) choledochal cyst
4) chronic inflammatory state: typhoid, H.pylori
5) GB polyp > 1 cm
6) anomalous pancreaticobiliary duct junction ( APBDJ )
7) carcinogen: azotoluene, nitrosamine
Porcelain gallbladder
Choledochal cyst
Clinical manifestation

Similar cholecystitis or cholelithiasis

May asymptom

25-50% : jaundice

50% of patients cannot Dx GB CA before surgery
Diagnosis

Initial test : US

Imaging before Tx : MRCP
  CT

Tumor marker : CA 19-9

Preop tissue Dx : not necessary except in advance CA before chemo
Gallbladder carcinoma
Treatment

Tumor not beyond muscularis propria

\[ T_x : \text{Simple cholecystectomy} \]
Treatment

Tumor beyond muscularis propria but not beyond serosa

\[ T_x : \text{Extended cholecystectomy} \]
(simple cholecystectomy + segment IVb,5 resection + regional lymphadenectomy)
Tumor perforate serosa, invade liver or adjacent organs

\[
T_x : \text{Extended RT. Hepatectomy (IV-VIII)}
\]
Treatment

Metastatic diseases

\[ T_x : \text{palliation for pain or jaundice (ERCP/PTBD)} \]
Cholangiocarcinoma
Classified into

1) **Hilar**: Klatskin’s tumor, 2/3
2) **Middle**
3) **Lower**
Clinical manifestation

Cholestatic jaundice (painless)

Clinical cholangitis

Anorexia, weight loss
Initial test: US

Imaging before Tx: MRCP
CT + ERCP/PTC

Tumor marker: CA 19-9

Preop tissue Dx: not necessary except in advance CA before chemo
Treatment

Hilar lesion: Bismuth-Corlette

Type I

Type II

Type IIIa

Type IIIb

Type IV
<table>
<thead>
<tr>
<th>Type</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I</td>
<td>bile duct resection ± segment 1</td>
</tr>
<tr>
<td>Type II</td>
<td>bile duct resection + segment 1</td>
</tr>
<tr>
<td>Type IIIa</td>
<td>Rt. hepatectomy</td>
</tr>
<tr>
<td>Type IIIb</td>
<td>Lt. hepatectomy</td>
</tr>
<tr>
<td>Type IV</td>
<td>palliation</td>
</tr>
</tbody>
</table>
Treatment

Middle lesion: major common bile duct resection

Distal lesion: Whipple operation
Approach to jaundice
Jaundice

Medical jaundice  Surgical jaundice
Symptoms/signs of surgical jaundice

Jaundice with dark urine, pale stool and pruritus

Abdominal pain

History of previous biliary tract surgery

Abdominal mass/RUQ mass/Courvoisier’s law

LFT : direct hyperbilirubinemia, elevated ALP
UA   : Bile+, urobilinogen -
Suspicion surgical jaundice ➔

US then

ERCP/PTC
Patient with jaundice

Stabilize serious signs and symptoms

History
- Abdominal pain, fever, chills
- Prior abdominal surgery
- Older age

Physical
- High fever
- RUQ abdominal tenderness
- Palpable mass
- Evidence of prior abd surgery

History
- Viral prodrome
- Alcohol/ILDU
- H/O transfusion
- Hepatotoxin exposure
- Known hepatitis exposure
- Pregnancy
- Malignancy

Physical
- Hepatomegaly
- Ascites
- Asterixis
- Encephalopathy
- Spider angiomata
- Caput medusa
- Gynecomastia
- Testicular atrophy
- Excoriations

Laboratory evaluation

Direct bili > indirect bili
- ± ↑ AST/ALT
- ↑↑ Alk phos
- ± ↑ Amylase/PT/PTT

Suggests obstructive process

Direct bili > indirect bili
- ↑↑ AST/ALT
- Mild ↑ alk phos
- Nl amylase: nL / ↑ PT/PTT

Suggests hepatocellular/cholestatic process (including fulminant hepatic failure)

Indirect bili > direct bili
- Normal liver function tests
- Abnormal hemogram

Suggests hematologic process

Reassess and treat signs and symptoms

Radiographic evaluation
- Ultrasound or CT
- Direct bile duct visualization
- ERCP/surgical
- GI and surgical consultations

Suggests obstructive process

Observation
- GI consultation
- Remove toxins
- Viral markers

Suggests hepatocellular/cholestatic process (including fulminant hepatic failure)

Type and cross
- Hematologic consultation

Suggests hematologic process

Reassess and treat signs and symptoms
THANK YOU