Hepato-cellular carcinoma (cont.)

**Diagnosis**
- Sonography of abdomen
- Spiral CT –abdomen( using lipiodol) or use poppy seed oil
- CT-abdomen using Gadolinium

**Staging & clinical assessment depend on**
- General assessment
- Childs classification
- Size & site of the tumor
- E.g. if cirrhotic liver better to do liver transplantation than extensive resection of the liver, but if not cirrhotic better to do resection.

**Treatment**
- if no cirrhosis  do resection
- if Child A type ( good liver function, no portal hypertension ) also do resection
- if resection not possible because of bad liver function & meets Milan criteria do transplantation

**Milan criteria**
- One metas. But less than 5 cm
- 2 or 3 metas. all less than 3 cm
- No vascular invasion
- No extrahepatic metas.

**Cholangio-carcinoma (bile ducts tumor)**
- 2nd most common primary cancer within liver
- Common in elderly, but in young age also occur in primary sclerosing cholangitis.
- Common site is at confluence of right and left bile duct.
- Common in areas where bile stones and infection is common.
- Histologically is two types:
  1. Fibrous type at confluence of Rt. & Lt. bile ducts, causing tight stricture at confluence
  2. Polypoidal type at distal common bile duct, causing luminal obstruction.
- 10% of patients with primary sclerosing cholangitis develop cholangiocarcinoma, and usually multicenter.
- Other predisposing factors as anatomical abnormalities as intrahepatic cystic changes & choledochal cyst.
- Infestation of biliary tract with Chlonorchis Sinensis is another predisposing factors.
- Its adenocarcinoma which may arise from:
  1. Intrahepatic bile ducts, called peripheral type, less common type and represent itself as a mass in liver or at edges of the liver.
  2. Extrahepatic bile ducts, called central type, which may arise from proximal bile duct called Klatskins tumor, or from distal bile duct, which represent itself as obstructive jaundice.
- Both invade perineural plains and a long lymphatic channels.
- Peritoneal seeding is unusual.
- Liver biopsy cannot differentiate primary from secondary metastasis adenocarcinoma in liver, so you must always exclude other primary tumor in the body.
Clinical features

1. Mass in liver with pain
2. Obstructive jaundice painless
3. Symptoms of chronic liver disease as in primary sclerosing cholangitis.

Diagnosis

- Ultrasound of the abdomen
  - Dilatation of the intrahepatic bile ducts and not extrahepatic is suspicious
  - Liver SOL
- Cholangiography shows hilar stricture, filling defect in CBD.
- Brush cytology
- Angiography for invasion of portal vein and hepatic artery.
- Spiral CT-abdomen filling defect
- Liver biopsy

Treatment

1. Supportive measures for chronic liver disease.
2. Vit. K injection
3. Surgery
   - No liver transplantation
   - Radical resection of the liver parenchyma
   - Transhepatic portal vein embolization if residual liver is inadequate
4. There is a rule for neoadjuvant chemo-radiotherapy in loco regional spread.

Gall bladder cancer

- Unknown etiology
- Rare, aggressive, poor prognosis
- Is associated invariably with the gall stones.
- 90% of the patient diagnosed when they have cholelithiasis
- Porcelain gall bladder pre-cancerous condition.

Clinical features

1. When extend to liver and obstruct bile ducts it cause pain and obstructive jaundice.
2. Patient has symptoms of gall stones and the cancer is incidentally found.

Diagnosis

1. Sonography of abdomen
2. Spiral CT-abdomen
3. Angiography
4. Cholangiography
5. Selective hepatic angiography
6. Staging laparoscopy

Treatment

- If involving only mucosa, it has good prognosis
- If involving serosa, it has bad prognosis
- Only hope is radical surgery
- Right lobe resection
- Hilar lymphadenopathy
- Chemotherapy
- Obstructive jaundice... biliary endoprosthesis
- 5 years survival is about 5%, median survival is 12 months
Gall bladder & Bile ducts

- Gall bladder is 7-12 cm, capacity 50 cc
- Fundus, body, neck, infundibulum
- Muscle fibers are crisscross manner
- Mucosa sink in to m. coat, crypts of Luscka
- Cystic duct
  - 3cm, lumen 2-3 mm
  - Mucosa spiral folds valve of Heister
  - Sphinicter structure, sphincter Lutkins
  - It join CHD 80% superaduodenal
  - or join it retroduodenal or retropancreatic part of CBD
  - or join rt. Hepatic duct or right hepatic sectorial duct
- CHD <2.5 cm, formed by rt & lt bile duct union
- CBD 7.5 cm, has 4 parts
  - Supraduodenal
  - Retroduodenal
  - Infraduodenal, groove or tunnel post. Surface of pancreas
  - Intraduodenal, via duod. wall, surrounded by sphincter of Oddi, terminate at summit of papilla of Vater.
- Arterial supply from rt. Hepatic a. passes behind CHD or arise from gastroduodenal a.
- Cystic artery may pass in front of cystic duct
- Lymphatic
  - Subserosal and submucosal passes to cystic node of Land lie in the fork of CHD & Cystic duct, from there to hilar of liver or caeliac LN.
  - Subserosal passes also to subcapsular lymph channels then to liver.

Gall bladder function

1. Reservoir for bile (97% water, 1-2% is bile salts, 1% fatty acids, cholesterol, pigments)
2. Concentration for the bile
3. Mucus secretion about 20 cc per day.

Investigations

1. Plain radiography, 105 radiopaque stones, calcification of gall bladder, limy bile
3. IV cholangiography
4. Sonography
5. Radioisotope scanning with Te99 (when biliary enteric anastomosis is inadequate, it shows stenosis or obstruction
6. CT- abdomen
7. MRCP
8. ERCP (visualize ducts, cytology, stenting, stone removal, micro-calcification)
9. PTC by Chiba or Okuda needle external drainage. Stent indwelling, choledochoscopy
10. Perioperative cholangiography
11. Operative biliary endoscopy, choledochoscopy
**Congenital abnormalities of gall bladder**

- Hepatic diverticulum from ventral wall of foregut, elongate into stalk form choledochus.
- Lateral bud form cystic duct and gall bladder
  1. Absence of gall bladder, so not visualized, it is not always pathological
  2. Phrygian cap, 2-6 % of cholecystograms
  3. Floating gall bladder, may undergo torsion, cause mucocele of gall bladder with severe abdominal pain, which necessitate cholecystectomy.
  4. Double gall bladder, rarely, one of them is intrahepatic
  5. Absence of cystic duct, usually pathological duo to stone which ulcerate the cystic duct and open to CBD.
  6. Low insertion of cystic duct, may open to CBD near ampulla of Vater.
  7. Extrahepatic biliary atresia
     - 1:14000 live birth, M= F
     - Duct destroyed by inflammatory process.
     - Associated with cardiac lesion, polysplenia, situs inversus, absence vena cava, preduodenal portal vein.
     - Types:
       - Type 1 atresia restricted to CBD
       - Type 2 atresia restricted to CHD
       - Type 3 atresia restricted to Rt. & Lt. hepatic duct.
     - Clinical features
       - Jaundice at birth, depend at the end of first week.
       - Meconium little bile stained, later pale stool & dark urine.
       - Prolong steatorrhea......osteomalacia ( biliary rickets )
       - Severe pruritis
       - Clubbing & skin xanthomas ( duo to increase cholesterol level)
     - DD
       1. Alpha 1 antitrypsin deficiency
       2. Intravenous feeding cholestasis
       3. Choledochal cyst
       4. Inspissitated bile syndrome
       5. Neonatal hepatitis
     - Treatment
       - Surgical
       - If patent proximal bile duct 10%, do Roux en Y anastomosis
       - If type 2 & 3 do Kasai procedure ( R-y hepato-jejunostomy)
  8. Congenital dilatation of intrahepatic ducts (Carolis disease)
     - Non familial, appear in childhood or early adult life
     - Multiple irregular sacular dilatation of intra-hepatic ducts separated by normal or stenotic segments.
     - Cause biliary stasis....stone formation....cholangitis
     - Associated with congenital hepatic fibrosis, medullary sponge kidneys, cholangiocarcinoma
     - Treatment
       - Antibiotic
       - Calculus removal
       - Lobectomy if limited to one lobe of the liver
9. Choledochal cyst

- Specific weakness in a part of whole of the wall of CBD
- Pt. has anomalous biliary pancreatic junction, long common channel, may cause pancreatitis.
- It is premalignant condition
- Clinical features:
  - At any age may presents as jaundice attacks, cholangitis, and abdominal pain.
  - Some patient has swelling at upper abdomen.
- Diagnosis
  - Sonography of abdomen
  - MRCP
- Treatment:
  - Surgery excision of the cyst & doing R-Y loop jejunostomy

Gallstones (cholelithiasis)

- Most common pathology, commonest surgery in Western countries.

Incidence

- F: M is 3:1
- Both sex from childhood to centenarian may develop gallstones
- 1st relative of pt. with gallstones have twofold greater prevalence
- Rare in Africa, Southern India
- Female, forty, fat, fertile, flatulent

Classified according to chemical composition

1. Cholesterol
   - Pure rare, < 10 %, smooth single (solitary), <10% radio –opaque. The rest is radiolucent.
   - Mixed (bile pigment & calcium) multiple, irregular, faceted, hard, mulberry shaped & soft.
   - Whitish yellow......green......black in color.
   - Pure or mixed is duo to super-saturation of bile with cholesterol.
2. Pigmented stones
   - Common in Far East
   - Contain less than 20% cholesterol, dark color duo to calcium bilirubin
   - Black or brown in color
   - Black
     - Duo to super-saturation of calcium bilirubinate, carbonate, phosphate
     - Small ,brittle, speculated,
   - Brown
     - Usually secondary to bacterial infection which cause stasis, or by parasite infestation in Asian, or in Western by stricture.
     - < 1cm,brunish,soft,mushy form in gall bladder or bile ducts
   - If gas in stone, which appear as tri- or bi-radiate dark shadow called (Mercedes Benz, or Seagull)
Conditions predispose to gallstones

1. Obesity
2. Pregnancy
3. Dietary factors
4. Cohn’s disease
5. Terminal ileum resection
6. Gastric surgery
7. Hemolysis (Sickle cell anemia, thalassemia, hereditary spherocytosis)

Causal factors in gallstones formation:

It is multi-factorial, may be: 1-metabolic, 2-infective, 3-bile stasis

1- Metabolic

- More cholesterol (insoluble in water), form crystals called super saturated or Lithogenic bile.
- Bile cholesterol increase with age in female, pill, obesity, clofibrate in treatment of hyperlipoproteinemia.
- Decrease bile salt by estrogen & factors which interrupt intrahepatic circulation as in ileal disease, by passes, cholestryamine therapy.

2- Infection

- Unclear, often it is sterile bile
- Radiolucent center, mucus plug around bacteria.
- H. pylori antigen is isolated from gall bladder has stone

3- Bile stasis

- Contractibility of gall bladder decrease by estrogen, truncal vagotomy, parenteral nutrition, lack good oral intake cause decrease cholecystokinine secretion.

Effects and complications of gallstones

- In the gall bladder:
  - Silent stones
  - Chronic cholecystitis
  - Acute cholecystitis
  - Gangrene
  - Perforation
  - Empyema
  - Mucocele
  - Carcinoma
- In the bile ducts:
  - Obstructive jaundice
  - Cholangitis
  - Acute pancreatitis
- In the intestine:
  - Acute intestinal obstruction (‘gallstone ileus’)
Clinical presentation of the gallstones

1- May be asymptomatic 85-95%.
  
  - 17% at death has gall stones in UK, so more presence of gallstones in not an indication for surgical approach.
  - 3% of asymptomatic annually become symptomatic and develop biliary colic.
  - Once become symptomatic pt. get bouts of recurrent biliary colic.
  - 3-5 % of symptomatic cases get complications.
  - Over 20 years period, 2/3 of asymptomatic pt. remains symptoms free.
  - Because few pt. get complications without previous biliary symptoms, prophylactic cholecystectomy in rarely indicated.
  - Saints triad (hiatus hernia, diverticulaouse of colon, gallstones)

2- Acute cholecystitis
  
  - 90-95% is duo to gallstones
  - Obstruction of cystic duct cause inflammation of gall bladder.
  - Episodes of rt. Hypochondrial pain radiate to the shoulder and back. May radiate to chest or left hypochondrial quadrant
  - Continue from minutes to hours
  - Frequently start at night , awake the patient
  - Dyspeptic symptoms
  - No pain but pt is going to eat and drink ,so more pain
  - Recurrent in weeks and normal for months
  - If infection occur pt. get continuous pain, unwell, pyrexia, nausea, vomiting
  - Inflammation resolve if stone slipped back to gall bladder from cystic duct.

On examination
  
  - Tenderness at rt. Hypochondrial area, Murphy sign +
  - Mass in hypochondrium due to omentum wall off gall bladder.
  - Mild to moderate leucocytosis 12000-15000, more than 20000 indicate complicated cholecystitis as perforation, gangrene, cholangitis

DD
  
  1. Appendicitis
  2. Perforated PU
  3. Acute pancreatitis
  4. Pyelonephritis
  5. MI
  6. Rt. Lower lobe basal pneumonia
  7. Herpes Zoster
  8. Hepatitis

Diagnosis:
  
  - U/S of abdomen
  - Serum amylase to exclude pancreatitis
  - LFT, if jaundice or not
  - CXR
  - ECG
  - Renal (GUE)
Treatment:

1. Conservative treatment followed by cholecystectomy
   - NGT
   - IVF
   - AB
   - Analgesic
   - If responding remove NGT and start fluid diet
   - Operation done
     - Next list or
     - Interval cholecystectomy 6-10 weeks

2. Continue conservative treatment is abandoned if pain and tenderness increases. Do per-cutaneous drainage under guide of sonography and then subsequent cholecystectomy.

3. No conservative treatment advised when:
   - Uncertain about diagnosis
   - Possibility of high retrocecal appendicitis & perforated PU cannot be excluded. by laparoscopic or open operation

4. Routine early operation within 48 hours
   - Symptoms persist post cholecystectomy in 15% i.e has the same preoperative symptoms, this called post cholecystectomy syndrome. Exclude:
     1. Stone in CBD
     2. Stone in stump of cystic duct
     3. Stricture of bile duct due to damage
   - Do MRCP...ERCP to remove the stones

Mucocele of gallbladder

- Due to neck obstruction of gall bladder by a stone & content remain sterile or by pressure from outside by cholangiocarcinoma.
- Gall bladder may be palpable
- If secondary bacterial infection occur it cause empyema of gall bladder.
- Needs drainage then cholecystectomy.

Acalculus cholecystitis

- Acute or chronic may occur without stones with same clinical presentation.
- Some patient has nonspecific inflammation & some has cholecystosis
- Acute one seen especially in patients with major surgery, burn, trauma
- Diagnosis:
  - Mostly diagnosis missed and mortality is 20 %
  1. Oral cholecystography in chronic cases
  2. Radio isotop scanning with acute disease
  3. Cholesterol crystal in duodenal aspirate

The cholecytoses

- Not uncommon affect gall bladder
- Has chronic inflammation
1- **Cholesterosis (Strawberry gall bladder)**
   - Yellow speeks (submucosal aggregation of cholesterol crystals & cholesterol esterase correspond to seeds)
   - May be associated with cholecystitis

2- **Cholesterol polyposis of gall bladder**
   - Cholecystography shows negative shadow in functioning gall bladder
   - U/S of abdomen shows polyp, it is cholesterol polyposis with adenomatous changes
   - Do surgery if the size of the polyp become larger by U/S follow up

3- **Cholecystitis glandularis proliferation (polyp, adenomatosis & intramural diverticulosis)**
   - Polyp of mucus membrane is fleshy and granulomatous
   - All layers thickened
   - Intral mixed stones
   - Intramural later extramural abscess
   - If symptomatic do cholecystectomy

4- **Diverticulum of gall bladder**
   - Manifestation as black pigmented stones impacted in out-pouching of lacuna of Luscka
   - By cholecystogram seen
   - Treatment is by cholecystectomy

**Typhoid gall bladder (Typhoid Mary)**
- Pass Salmonella typhi in feces and urine
- Chronic carrier and pass bacteria to bile
- May form gallstones
- Treatment is antibiotic + cholecystectomy

**Parasite infestation of biliary tree:**

1- **Biliary ascariasis**
   - Cause biliary pancreatitis
   - Its complication stricture, suppurative cholangitis, liver abscess, empyema of gall bladder
   - Uncomplicated analgesia to relax sphincter of Oddi + antihelmink drugs
   - Operation ,ERCP to remove them

2- **Clonorchiasis (Asiatic cholangiohepatitis)**
   - Endemic in Far East
   - Fluk 25 mm long and 5mm wide
   - It inhabitate bile ducts
   - Cause fibrous thickening of bile ducts may be :-
     - Asymptomatic
     - Symptomatic, cholangitis, biliary pancreatitis, colick, stones, cholangitis, cirrhosis, bile duct carcinoma.
   - Treatment:
     - Choledochtomy with t-tube
     - Choledocho-duodeostomy

3- **Hydatid Cyst**
   - Large cyst obstruct hepatic duct
   - Rupture...obstructive jaundice or cholangitis
   - Needs surgical intervention