# 3rd Med Obstructive Jaundice

Adrian P. Ireland

#### Academic RCSI Department of Surgery, Beaumont Hospital

#### **Overview**

Obstructive Jaundice is very interesting

- Not uncommon in hospital to have a jaundiced patient
- Many different causes and various workups
- Common question for examiners to ask students

#### What is jaundice?



French term (Yellow)
Bilirubin more than 40 micromoles per litre
Stain concept, affinity for connective tissue

## **Bilirubin Metabolism**

- 1. Source of bilirubin
- 2. Liver metabolism
- 3. What happens in the gut
- 4. Enterohepatic circulation of Urobilinogen
- 5. Urobilinogen in urine



## Source of Bilirubin

- 85% from old RBC rest from non haem proteins
- Hb is degraded to Haem and Globin
- Iron is extracted from Haem
- Rest is converted to bilirubin (porphorin metabolism)
- Bilirubin travels to liver
   bound to albumin



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## Journey through the liver

- Bilirubin taken up
- Conjugated to form water soluble conjugate
- Conjugate secreted into bile



## In the gut

- Bilirubin diglucuronide may be
  - Deconjugated by beta glucuronidase enzyme (eg. E. coli) then becomes insoluble and predisposed to pigment stones.
  - Metabolised by bacteria to urobilinogen which is partially reabsorbed (remainder makes the stool brown)

#### Enterohepatic circulation of Urobilinogen

- Most is taken up by the liver and re excreted
- If it builds up in the blood then it may be filtered by the glomerulus and be detectable in urine

## Urobilinogen in the Urine

- Conjugated bilirubin is reaching the gut (ie Bile duct not blocked)
- The liver cell is not behaving well in that it is permitting urobilinogen to increase in the blood

## Bile salt metabolism

- Bile salts have nothing to do with bilirubin metabolism
- Bile salts are Bile acids
- 98% of bile is water of the rest
  - 80% is bile salts
  - 15% is phospholipid
  - 5% is cholesterol



#### Bile salts metabolism

- 1. Source of bile salts
- 2. Bile salts in the small bowel
- 3. Bile salts in the large bowel

Bile salts may be carcinogenic in the stomach and esophagus



#### Source of bile salts

- Made in the liver from cholesterol
- Have hydroxyl groups
- Conjugated by liver to make soluble with;
  - Glycine 75%
  - Taurine 25%
- These are the primary bile acids/salts;
  - Cholic acid (glycine or taurine conjugate)
  - Chenodeoxycholic acid (glycine or taurine conjugate)
- The primary bile acids/salts are released into the intestine in bile

#### Bile salts in the small bowel

- The primary bile salts are reabsorbed in the terminal ileum, (enterohepatic circulation)
- In all there is about 3-5 g of bile salts in a person
- These are turned over 6 times a day
- In the presence of ileal disease, too much bile salt reaches the colon and causes diarrhoea
- A small amount of bile salt enters the colon

#### Bile salts in the large bowel

- Bacterial metabolism of the primary bile salts occurs in the colon;
  - Cholic acid Deoxycholic acid
  - Chenodeoxycholic acid Lithocolic acid
- Deoxycholic acid is reabsorbed and has an enterohepatic circulation

## Some causes of jaundice

#### Anatomical classification

- Haemolysis
- Displacement from albumin
- Liver disease (Cell)
  - Hepatitis, Cirrhosis
  - Deficient enzymes
  - Primary and metastatic liver tumors
- Blocked bile ducts;
  - Intra-hepatic, Extra-hepatic

Aetiological classification

- Benign
- Malignant

## What is obstructive jaundice?



## Main causes of obstructive jaundice

#### Bile duct

- In the lumen of the common bile duct (gallstones, parasites)
- In the wall of the duct (choledochal cyst, sclerosing cholangigis, cholangiocarcinoma)
- Pressing in on the bile duct (Mirrizi, pancreatitis, pancreatic cancer, malignant nodes)

#### Ampulla

- Periampullary carcinoma
- Tumor invading the ampulla

## Consequence of obstructive jaundice

- Malabsorbtion
  - Fat (steatorrhoea) Fat soluble vitamins (DEKA)
- Jaundice Bilirubin, No bilirubin metabolites in stool –
   Pale
- Itch Bile salts
- Sepsis, cholangitis, Charcots triad
- ? Renal failure (Hepato-renal syndrome)
- Bleeding High INR

## Clinical features of cholangits, Charcot's triad

## 1. Jaundice

- 2. Intermittent chills / fever or rigors
- 3. Abdominal pain

Charcot's triad indicats cholangitis, this causes severe sepsis and may result in liver abscess formation

## Courvoisier's law

If in the presence of jaundice the gallbladder is palpable, then a stone is not a likely cause



## **Clinical** approach

## History

- Clinical examination
- Investigations /Management

#### History

- Pale stools, Dark Urine
- Itch ?
- Pain or not
- Intermittent, or progressive and unrelenting
- Drugs, operations (anaesthetic gas)
- Blood transfusion, innoculations
- Occupation and hobbies, (rats and leptospirosis)
- Alcohol intake
- Family History (Gilbert's disease)

## Examination

- Sclera, good light, mucous membranes
- Scratch marks
- Pigmentation
- Stigmata of chronic liver disease, gynacomastia, portal hypertension
- Ascitis
- Hepatomegaly, Palpable gall bladder
- Splenomegally

#### Investigations used for the biliary tract

#### Urinalysis

- Blood tests (Blood count, Liver blood tests,
   Fractionanted bilirubin)
- Ultrasound (Abdominal ultrasound, Endoscopic ultrasound)
- X-ray (Plain abdominal
  film, Oral cholecystogram,
  Intravenous cholangiogram,
  Percutaneous transhepatic
  cholangiogram, CT)
- HIDA scan, with CCK
- Endoscopic retrograde cholangio-pancreatography
- Magnetic resonance cholangio-pancreatography

## **Duct evaluation – Methods**

- 1. Infusion cholangiogram
- 2. MRCP
- 3. Endoscopic Ultrasound
- 4. ERCP
- 5. Trans hepatic cholangiogram
- 6. Per operative cholangiogram
- 7. Exploration

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## Algorithm



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### Common bile duct stone

There is no significant advantage to patients treated by preoperative sphincterotomy as opposed to open cholecystectomy and exploration of the common bile duct alone

Neoptolemos JP et al. A prospective randomised study of pre-operative endoscopic sphincterotomy versus surgery alone for common bile duct stones. **Br Med J** 1987; 294:470–4

## Triple bypass



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Hepatico-jejeunostomy Roux-en-Y

