Cholecystitis – 16/09/03

STRUCTURE & FUNCTION OF BILIARY SYSTEM

Gall Bladder: stores and concentrates the bile due to active mucosal reabsorption of H2O

Lipid passes into the duodenum → stimulates secretion of CCK → endocrine cells in duodenal wall → gall bladder contracts → releases bile which acts as emulsifying agent and facilitates breakdown and absorption of fats.

If there is a stone → fat metabolism decreases and you get steatorrheoea, fat soluble vitamins ADEK are not absorbed therefore you get inadequate prothrombin synthesis → defective clotting (SURGICAL PROBLEM). The way they handle this during surgery is to give IM Vit K (4-6hrs) prior to surgery depending on the Liver Fn tests.

PATHOPHYSIOLOGY OF GALL STONES

Western world: 75-90% of gall stones are mixed. They consist of organic material containing bacteria (sometimes) as their inner core, surrounded by cholesterol + bile pigment (calcium bilirubinate) + other calcium salts. 10% of stones are cholesterol pure stones. You can also get stones of calcium carbonate only.

Asia: mainly get gall stones composed of bile pigment alone.

HOW STONES ARE FORMED?

1. changes in [] of constituents of bile (MOST IMPORTANT)
2. biliary stasis
3. infection

- Bile salts are responsible for micelle formation and keeping cholesterol within them. In the event of excess cholesterol in the body → micelle formation cannot keep up → therefore you get very unstable micelles which collapse → gall stones form (VERY GREY AREA).
- Stasis due to obstruction (neck of gall bladder) or gall bladder contractility problems is a precipitating factor
- Infection of the gall bladder will damage the muscular layer → precipitating gall bladder contraction problems.

PROGRESSION OF GALL STONES

Biliary colic pain → obstruction of the gall bladder neck → persistent obstruction causes a chemical inflammation (acute cholecystitis) → gall bladder becomes distended due to mucous secretion (mucocele) → infection settles in due to stasis and the gall bladder further distends due to pus (empyema of gall bladder) → blocking of venous/arterial supply will cause atrophy and necrosis → fibrosis and diverticula occur → gall bladder eventually forms a fistula with another structure. Notice that pus has high osmolality → therefore there is more water reabsorption into the pus → causing increased distension.

OTHER PATHOLOGICAL MECHANISMS

1. Chronic inflammation independent of stones → "cholecystitis sans stones"
2. Biliary dyskinesia + cystic duct syndrome: typical symptoms of gall bladder disease but biliary manometry shows abnormally high pressures at the sphincter of ODDI (sphincterotomy / sphincteroplasty).

INVESTIGATIONS FOR GALL BLADDER DISEASE

Gall stones are more predisposed during pregnancy, fatty foods & females.

Liver Fn tests

Bilirubin, Total protein, Albumin, ALT, Gamma-GT [made by hepatocytes – any damage will cause release of these enzymes), ALP [made my epithelial cells of common bile duct – any stone scratching this surface causes release into blood).

Plasma amylase + lipase – if pancreatitis is occurring due to distal blockage, then you will have increase in such enzymes → autodigestion.

Ultrasound – good for jaundiced patients

- Gall bladder wall thickens (mucosal oedema and fibrosis → post inflammatory response)
- Dilatation of the common bile duct system – indicating distal duct obstruction (unreliable for identification of bile stones directly).

INVESTIGATION OF BILIARY DUCT SYSTEM

If the patient is not jaundiced:

- Preoperative cholangiography (at open laparotomy operation)
    - Dye is injected into the cystic duct and X rays are used to demonstrate duct morphology including: 1) thickness, 2) dilatation, 3) obstruction

If patient is jaundiced:

- Ultrasonography
    - Shows dilatation and thickness of the intra-extra hepatic ducts
- ERCP
    - Endoscopic route through sphincter of ODDI. Problems include: duodenal perforation, pancreatitis, and duct perforation.
    - You can release the stone immediately and relieve patient of jaundice.
- Percutaneous transhepatic cholangraphy
    - Insert dye into 1 extra hepatic duct and see the obstruction.

COMPLICATIONS OF GALL STONES (Tutorial: 25/09/03)

1. The complication of inflammation of the gall bladder (acute cholecystitis due to chemical inflammation) → mucocele → empyeme of the gall bladder → perforation has all been explained above.
2. Other complications include: acute pancreatitis (stone breaks off and gets stuck in the ampulla → causing back up of pancreatic juice → pancreatic autodigestion), jaundice

(obstructive), generalised peritonitis (gall bladder ruptures and contents spread within peritoneum), bowel obstruction (i.e.: gall blader forms fistula with duodenum → gall stones enter the small bowel and get stuck in the ileum → gall stone ileus).