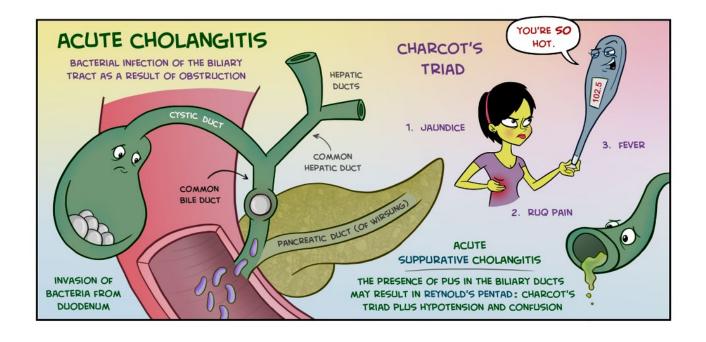
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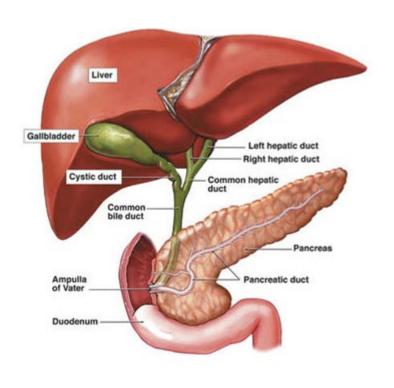
Benign Liver Lesions

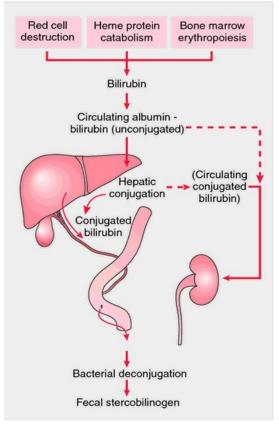
Incidence in autopsy series is 8% Cavernous haemangiomas may be enormous Clinically they are reddish purple hypervascular lesions Lesions are normally separated from normal liver by ring of fibrous tissue On ultrasound they are typically hyperechoic. Serum AFP normal. Ulver cell adenoma Liver cell Lesions are usually solitary They are usually sharply demarcated from normal liver although they usually lack a fibrous capsule On ultrasound the appearances are of mixed exholty and heterogeneous texture. On CT most lesions are hypodense when imaged prior to administration of IV contrast agents In patients with haemorrhage or symptoms removal of the adenoma may be required Congential and benign, usually present in infants. May compress normal liver hamartomas Liver abscess Billary sepsis is a major predisposing factor Structures drained by the portal venous system form the second largest source Common symptoms include fever, right upper quadrant pain. Jaundice may be seen in 50% Ultrasound will usually show a fluid filled cavity, hyperechoic walls may be seen in chronic abscesses Amoebic Between 75 and 90% lesions occur in the right lobe Presenting complaints typically include fever and right upper quadrant pain Ultrasongraphy will usually show a fluid filled structure with poorly defined boundaries Aspiration yield sterile odourless fluid which has an anchovy paste consistency Treatment is with metronidazole Hydatid cysts Seen in cases of topeworm parasite Echinococcus granulosus infection The cyst has no epithelial lining Cysts are commonly unilocular and may grow to 20cm in size. The cyst wall is thick and has an external laminated hilar membrane and an internal enucleated germinal layer These cysts are allergens which precipitate a type 4 hypersensitivity In biliary ruputure there may be the classical triad of: Biliary colic, Jaundice, and Urticaria Typically presents with malaise and right upper quadrant pain. Secondary bacterial infection occurs in 10%. Liver function tests are usually abnormal and eosinop		Benign Liver Lesions
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Amoebic abscess Liver abscess is the most common extra intestinal manifestation of amoebiasis		Ultrasound will usually show a fluid filled cavity, hyperechoic walls may be seen in chronic
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Liver function tests usually normal	Cystadenoma	
		Usually solitary multiloculated lesions
Ultrasonography typically shows a large anechoic, fluid filled area with irregular margins. Internal		Liver function tests usually normal
		Ultrasonography typically shows a large anechoic, fluid filled area with irregular margins. Internal
echos may result from septa		echos may result from septa
Surgical resection is indicated in all cases		Surgical resection is indicated in all cases



Biliary Disease

	Dilially	Disease
Diagnosis	Typical features	Pathogenesis
Gallstones	Typically history of biliary colic or episodes of chlolecystitis. Obstructive type history and test results.	Usually small calibre gallstones which can pass through the cystic duct. In Mirizzi syndrome the stone may compress the bile duct directly- one of the rare times that cholecystitis may present with jaundice
Cholangitis	Usually obstructive and will have Charcot's triad of symptoms (pain, fever, jaundice)	Ascending infection of the bile ducts usually by <i>E. coli</i> and by definition occurring in a pool of stagnant bile.
Pancreatic cancer	Typically painless jaundice with palpable gallbladder (Courvoisier's Law)	Direct occlusion of distal bile duct or pancreatic duct by tumour. Sometimes nodal disease at the portal hepatis may be the culprit in which case the bile duct may be of normal calibre.
TPN (total parenteral nutrition) associated jaundice	Usually follows long term use and is usually painless with non-obstructive features	Often due to hepatic dysfunction and fatty liver which may occur with long term TPN usage.
Bile duct injury	Depending upon the type of injury may be of sudden or gradual onset and is usually of obstructive type	Often due to a difficult laparoscopic? cholecystectomy when anatomy in Calot's triangle is not appreciated. In the worst scenario the bile duct is excised and jaundice develops rapidly post operatively. More insidious is that of bile duct stenosis which may be caused by clips or diathermy injury.
Cholangiocarcinoma	Gradual onset obstructive pattern	Direct occlusion by disease and also extrinsic compression by nodal disease at the porta hepatis.
Septic surgical patient	Usually hepatic features	Combination of impaired biliary excretion and drugs such as ciprofloxacin which may cause cholestasis.
Metastatic disease	Mixed hepatic and post hepatic	Combination of liver synthetic failure (late) and extrinsic compression by nodal disease and anatomical compression of intra hepatic structures (earlier)



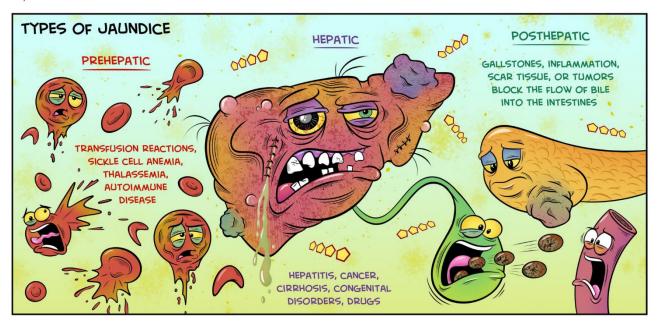


Surgical Jaundice

Jaundice can present in a manner of different surgical situations. As with all types of jaundice a careful history and examination will often give clues as to the most likely underlying cause. Liver function tests whilst conveying little in the way of information about liver synthetic function, will often facilitate classification as to whether the jaundice is pre hepatic, hepatic or post hepatic. The typical LFT patterns are given below:

Location	Bilirubin	ALT/ AST	Alkaline phosphatase
Pre hepatic	Normal or high	Normal	Normal
Hepatic	High	Elevated (often very high)	Elevated but seldom to very high levels
Post hepatic	High-very high	Moderate elevation	High- very high

In post-hepatic jaundice the stools are often of pale colour and this feature should be specifically addressed in the history.



Modes of presentation

These are addressed in the table (see previous page)

Diagnosis

An ultrasound of the liver and biliary tree is the most commonly used first line test. This will establish bile duct calibre, often ascertain the presence of gallstones, may visualise pancreatic masses and other lesions. The most important clinical question is essentially the extent of biliary dilatation and its distribution.

Where pancreatic neoplasia is suspected, the next test should be a pancreatic protocol CT scan. With liver tumours and cholangiocarcinoma an MRI/ MRCP is often the preferred option. PET scans may be used to stage a number of malignancies but do not routinely form part of first line testing.

Where MRCP fails to give adequate information an ERCP may be necessary. In many cases this may form part of patient management. It is however, invasive and certainly not without risk and highly operator dependent.

Management

Clearly this will depend to an extent upon the underlying cause but relief of jaundice is important, even if surgery forms part of the planned treatment. Patients with unrelieved jaundice have a much higher incidence of septic complications, bleeding and death.

Screen for and address any clotting irregularities

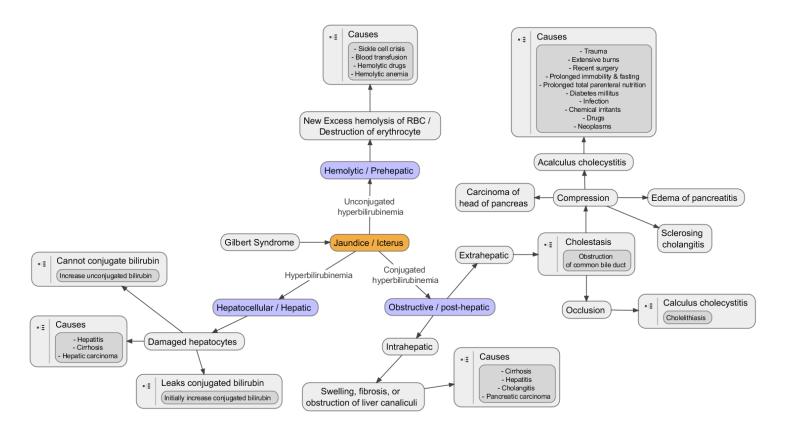
In patients with malignancy a stent will need to be inserted. These come in two main types; metal and plastic. Plastic stents are cheap and easy to replace and should be used if any surgical intervention (e.g. Whipples) is planned. However, they are prone to displacement and blockage. Metal stents are much more expensive and may compromise a surgical resection. However, they are far less prone to displacement and to a lesser extent blockage than their plastic counterparts.

If malignancy is in bile duct/ pancreatic head and stenting has been attempted and has failed, then an alternative strategy is to drain the biliary system percutaneously via a transhepatic route. It may also be possible to insert a stent in this way. One of the main problems with temporary PTC's is their propensity to displacement, which may result in a bile leak

In patients who have a bile duct injury surgery will be required to repair the defect. If the bile duct has been inadvertently excised then a hepatico-jejunostomy will need to be created (difficult!)

If gallstones are the culprit, then these may be removed by ERCP and a cholecystectomy performed. Where there is doubt about the efficacy of the ERCP an operative cholangiogram should be performed and bile duct exploration undertaken where stones remain. When the bile duct has been formally opened the options are between closure over a T tube, a choledochoduodenostomy or choledochojejunostomy.

Patients with cholangitis should receive high dose broad spectrum antibiotics via the intravenous route. Biliary decompression should follow soon afterwards, instrumenting the bile duct of these patients will often provoke a septic episode (but should be done anyway).



Gallstones

Up to 24% of women and 12% of men may have gallstones. Of these up to 30% may develop local infection and cholecystitis. In patients subjected to surgery 12% will have stones contained within the common bile duct. The majority of gallstones are of a mixed composition (50%) with pure cholesterol stones accounting for 20% of cases.

The aetiology of CBD stones differs in the world, in the West most CBD stones are the result of migration. In the East a far higher proportion arise in the CBD de novo.

The classical symptoms are of colicky right upper quadrant pain that occurs post prandially. The symptoms are usually worst following a fatty meal when cholecystokinin levels are highest and gallbladder contraction is maximal.

Investigation

In almost all suspected cases the standard diagnostic work up consists of abdominal ultrasound and liver function tests. Of patients who have stones within the bile duct, 60% will have at least one abnormal result on LFT's. Ultrasound is an important test, but is operator dependent and therefore may occasionally need to be repeated if a negative result is at odds with the clinical picture. Where stones are suspected in the bile duct, the options lie between magnetic resonance cholangiography and intraoperative imaging. The choice between these two options is determined by the skills and experience of the surgeon. The advantages of intra operative imaging are less useful in making therapeutic decisions if the operator is unhappy about proceeding the bile duct exploration, and in such circumstances pre operative MRCP is probably a better option.

Specific gallstone and gallbladder related disease

Disease	Features	Management
Biliary colic	Colicky abdominal pain, worse post prandially, worse after fatty foods	If imaging shows gallstones and history compatible then laparoscopic cholecystectomy
Acute cholecystitis	 Right upper quadrant pain Fever Murphys sign on examination Occasionally mildly deranged LFT's (especially if Mirizzi syndrome) 	Imaging (USS) and cholecystectomy (ideally within 48 hours of presentation) (2)
Gallbladder abscess	 Usually prodromal illness and right upper quadrant pain Swinging pyrexia Patient may be systemically unwell Generalised peritonism not present 	Imaging with USS +/- CT Scanning Ideally surgery, subtotal cholecystectomy may be needed if Calots triangle is hostile In unfit patients percutaneous drainage may be considered
Cholangitis	 Patient severely septic and unwell Jaundice Right upper quadrant pain 	 Fluid resuscitation Broad spectrum intravenous antibiotics Correct any coagulopathy Early ERCP
Gallstone ileus	 Patients may have a history of previous cholecystitis and known gallstones Small bowel obstruction (may be intermittent) 	Laparotomy and removal of gallstone from small bowel, the enterotomy must be made proximal to the site of obstruction and not at the site of obstruction. The fistula between the gallbladder and duodenum should not be interfered with.
Acalculous cholecystitis	 Patients with inter current illness (e.g. diabetes, organ failure) Patient of systemically unwell Gallbladder inflammation in absence of stones High fever 	If patient fit then cholecystectomy, if unfit then percutaneous cholecystostomy

Treatment

Patients with asymptomatic gallstones rarely develop symptoms related to them (less than 2% per year) and may therefore be managed expectantly. In almost all cases of symptomatic gallstones the treatment of choice is cholecystectomy performed via the laparoscopic route. In the very frail patient there is sometimes a role for selective use of ultrasound guided cholecystostomy.

During the course of the procedure some surgeons will routinely perform either intra operative cholangiography or laparoscopic USS to either confirm anatomy or to exclude CBD stones. The latter may be more easily achieved by use of laparoscopic ultrasound. If stones are found then the options lie between early ERCP in the day or so following surgery or immediate surgical exploration of the bile duct. When performed via the trans cystic route this adds little in the way of morbidity and certainly results in faster recovery. Where transcystic exploration fails the alternative strategy is that of formal choledochotomy. The exploration of a small duct is challenging and ducts of less than 8mm should not be explored. Small stones that measure less than 5mm may be safely left and most will pass spontaneously.

Risks of ERCP

- Bleeding 0.9% (rises to 1.5% if sphincterotomy performed)
- Duodenal perforation 0.4%
- Cholangitis 1.1%
- Pancreatitis 1.5%

Notes and Mnemonics

Courvoisiers Law:

Obstructive jaundice in the presence of a palpable gallbladder is unlikely to be due to stones.

This is due to the fibrotic effect that stones have on the gallbladder. Like all these laws there are numerous exceptions and many cases will not present in the typical manner.

The development of jaundice in association with a smooth right upper quadrant mass is typical of distal biliary obstruction secondary to pancreatic malignancy

Mnemonic for the assessment of the severity of pancreatitis: PANCREAS

P a02 < 60 mmHg

A ge > 55 years

N eutrophils > 15 x 10/l

C alcium < 2 mmol/l

R aised urea > 16 mmol/l

E nzyme (lactate dehydrogenase) > 600 units/l

A lbumin < 32 g/l

S ugar (glucose) > 10 mmol/l

> 3 positive criteria indicates severe pancreatitis.

Acute early fluid collections are seen in 25% of patients with pancreatitis and require no specific treatment. Attempts at drainage may introduce infection and result in pancreatic abscess formation.

In **Mirizzi syndrome** the gallstone becomes impacted in Hartmans pouch. Episodes of recurrent inflammation occur and this causes compression of the bile duct. In severe cases this then progresses to fistulation. Surgery is extremely difficult as Calots triangle is often completely obliterated and the risks of causing injury to the CBD are high.

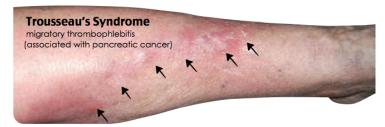


Pancreatic Cancer

- Adenocarcinoma (ductal epithelial origin)
- Risk factors: Smoking, diabetes, adenoma, familial adenomatous polyposis
- Mainly occur in the head of the pancreas (70%)
- Spread locally and metastasizes to the liver
- Carcinoma of the pancreas should be differentiated from other periampullary tumours with better prognosis

Clinical features

- Weight loss
- Painless jaundice
- Epigastric discomfort (pain usually due to invasion of the coeliac plexus is a late feature)
- Pancreatitis
- Trousseau's sign: migratory superficial thrombophlebitis

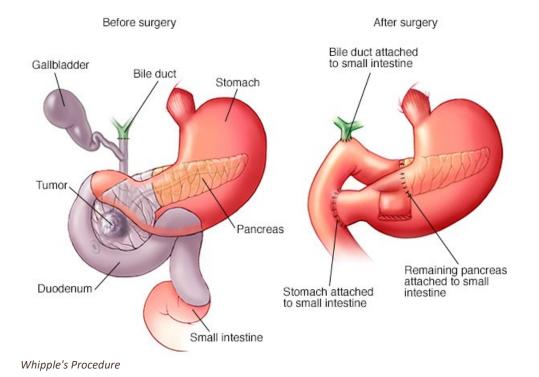


Investigations

- USS: May miss small lesions
- CT Scanning (pancreatic protocol). If unresectable on CT then no further staging needed
- PET/CT for those with operable disease on CT alone
- ERCP/ MRI for bile duct assessment
- Staging laparoscopy to exclude peritoneal disease

Management

- Head of pancreas: Whipple's resection (SE dumping and ulcers). Newer techniques include pylorus preservation and SMA/ SMV resection
- Carcinoma body and tail: poor prognosis, distal pancreatectomy, if operable
- Usually adjuvent chemotherapy for resectable disease
- ERCP and stent for jaundice and palliation
- Surgical bypass may be needed for duodenal obstruction



Management of Acute Pancreatitis in The UK

Diagnosis

- Traditionally hyperamylasaemia has been utilised with amylase being elevated three times the normal range.
- However, amylase may give both false positive and negative results.
- Serum lipase is both more sensitive and specific than serum amylase. It also has a longer half-life.
- Serum amylase levels do not correlate with disease severity.

Differential causes of hyperamylasaemia

- Acute pancreatitis
- Pancreatic pseudocyst
- Mesenteric infarct
- Perforated viscus
- Acute cholecystitis
- Diabetic ketoacidosis

Assessment of severity

- Glasgow, Ranson scoring systems and APACHE II
- Biochemical scoring e.g. using CRP

Features that may predict a severe attack within 48 hours of admission to hospital

Initial assessment	Clinical impression of severity
	Body mass index >30
	Pleural effusion
	• APACHE score >8
24 hours after admission	Clinical impression of severity
	APACHE II >8
	Glasgow score of 3 or more
	Persisting multiple organ failure
	• CRP>150
48 hours after admission	• Glasgow Score of >3
	• CRP >150
	Persisting or progressive organ failure

Management

Nutrition

- There is reasonable evidence to suggest that the use of enteral nutrition does not worsen the outcome in pancreatitis
- Most trials to date were underpowered to demonstrate a conclusive benefit.
- The rationale behind feeding is that it helps to prevent bacterial translocation from the gut, thereby contributing to the development of infected pancreatic necrosis.

Use of antibiotic therapy

- Many UK surgeons administer antibiotics to patients with acute pancreatitis. However, there is very little evidence to support this practice.
- A recent Cochrane review highlights the potential benefits of administering Imipenem to patients with established pancreatic necrosis in the hope of averting the progression to infection.
- There are concerns that the administration of antibiotics in mild attacks of pancreatitis will not affect outcome and may contribute to antibiotic resistance and increase the risks of antibiotic associated diarrhoea.

Surgery

- Patients with acute pancreatitis due to gallstones should undergo early cholecystectomy.
- Patients with obstructed biliary system due to stones should undergo early ERCP.
- Patients with extensive necrosis where infection is suspected should usually undergo FNA for culture.
- Patients with **infected necrosis** should undergo either radiological **drainage** or **surgical necrosectomy**. The choice of procedure depends upon local expertise.



Pancreatitis: Sequelae

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Peripancreatic fluid collections	 Occur in 25% cases Located in or near the pancreas and lack a wall of granulation or fibrous tissue May resolve or develop into pseudocysts or abscesses Since most resolve aspiration and drainage is best avoided as it may precipitate infection
Pseudocysts	 In acute pancreatitis result from organisation of peripancreatic fluid collection. They may or may not communicate with the ductal system. The collection is walled by fibrous or granulation tissue and typically occurs 4 weeks or more after an attack of acute pancreatitis Most are retrogastric 75% are associated with persistent mild elevation of amylase Investigation is with CT, ERCP and MRI or Endoscopic USS Symptomatic cases may be observed for 12 weeks as up to 50% resolve Treatment is either with endoscopic or surgical cystogastrostomy or aspiration
Pancreatic necrosis	 Pancreatic necrosis may involve both the pancreatic parenchyma and surrounding fat Complications are directly linked to extent of parenchymal necrosis and extent of necrosis overall Early necrosectomy is associated with a high mortality rate (and should be avoided unless compelling indications for surgery exist) Sterile necrosis should be managed conservatively (at least initially) Some centres will perform fine needle aspiration sampling of necrotic tissue if infection is suspected. False negatives may occur. The extent of sepsis and organ dysfunction may be a better guide to surgery
Pancreatic abscess	 Intra-abdominal collection of pus associated with pancreas but in the absence of necrosis Typically occur as a result of infected pseudocyst They are usually managed by placement of percutaneous drains
Haemorrhage	 Infected necrosis may involve vascular structures with resultant haemorrhage that may occur de novo or as a result of surgical necrosectomy. When retroperitoneal haemorrhage occurs Grey Turners sign may be identified

Acute pancreatitis is known to precipitate **ARDS**. ARDS is characterised by bilateral pulmonary infiltrates and hypoxaemia. Note that pulmonary oedema is excluded by the CVP reading < 18mmHg.

