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ACUTE AND CHRONIC LIVER DISEASE Liver blood tests

ACUTE AND CHRONIC LIVER DISEASE Liver blood tests

The liver performs a myriad of biochemical, metabolic and immunological functions. Anhepatic humans survive for 24–48 hours. It is the only organ in the body that regenerates. Awareness of currently available liver blood tests and their significance is essential (Table 69.1). Main functions of the liver /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Maintaining core body temperature pH balance and correction of lactic acidosis Synthesis of clotting factors Glucose metabolism, glycolysis and gluconeogenesis Urea formation from protein catabolism Bilirubin formation from haemoglobin after breakdown of effete red cells in the spleen Drug and hormone metabolism and excretion Removal of gut endotoxins and foreign antigens Vitamin and mineral storage, including A, D, E, K and B12 Immunological function as part of the mononuclear phagocyte system Albumin production for transport of fatty acids, steroids and waste products Angiotensin synthesis Body temperature through heat production Cytochrome P450 detoxi /f_i es contaminants and pollutants, insecticides, food additives and alcohol

ANATOMY OF THE LIVER Embryology

ANATOMY OF THE LIVER Embryology

Liver development begins at 3–4 weeks’ gestation when a hepatic foregut diverticulum buds into the ventral wall of the primitive midgut. This diverticulum is the anlage for the liver, extrahepatic biliary ducts, gallbladder and ventral pancreas, which develop over the next week. The basement membrane surrounding the liver bud is then lost and cords of bipotential hepatoblasts invade the septum transversum and di ff erentiate into hepatocytes and cholangiocytes. Francis Glisson , 1597–1697, Regius Professor of Physic, Cambridge, UK, described the capsule of the liver and its blood supply in his book (1654).

The management of benign and cystic liver lesions • The management of intrahepatic cholangiocarcinoma • The management of hepatocellular carcinoma • The management of colorectal liver metastases •

Ablation for liver tumours

Ablation for liver tumours

Ablative therapies destroy tumour by the direct application of energy or toxic substances to discrete lesions. The basic tech - nique for RFA was described in 1891 by d’Arsonval, who first - demonstrated that heat was produced when radiofrequency waves passed through tissue. Ablation techniques now include RFA, micr owave ablation, cryoablation, laser ablation, irre - versible electroporation (IRE) and alcohol injection, all of which can be performed percutaneously , laparoscopically or at open surgery . There is wide variation in ov erall survival and local recurrence rates following ablation and surgery remains the gold standard treatment for resectable disease. Despite - these concerns, ablation still has a role as an adjunct to resec - tion and for combined approaches. Patients with small-volume resectable lesions who are not su ffi ciently fit to undergo liver resection should be considered for ablation, as should those with liver metastases where predicted FLR precludes resection. RF A and microwave ablation, which rely on the generation of heat, are the most widely used techniques. Increasing lesion - size exponentially increases impedance, limiting the size of the e ff ective ablation zone and increasing the risk of local recur - rence. Microwave ablation has been designed to overcome these issues with higher intratumoral temperatures, larger tumour ablation volumes and faster ablation times. Newer microwave tec hnologies include the use of cooled applicators and multiple antennae, such that tumours adjacent to large vascular structures can be e ff ectively treated. Local recurrence after RFA and microwave ablation is 5–15%.

Acute liver failure

Acute liver failure

Causes of acute liver failure Acute liver failure is the development of sudden, severe hepatic dysfunction from an acute insult associated with the onset of hepatic encephalopathy and coagulation abnormalities. The most widely accepted definition, from the American Associa tion for the Study of Liver Diseases, is: evidence of coagulation abnormality, usually an international normalized ratio above 1.5, and any degree of mental alteration (enceph alopathy) in a pa tient without pre-existing liver disease and with an illness of less than 26 /uni00A0 weeks' duration. Treatment of acute liver failure Acute liver failure is rare in the developed world, with an annual incidence of <10 cases per million and a current mortality of 30–40%. In the early stages, there may be no objective signs, but with severe dysfunction clinical jaundice may be associated with neurological signs of liver failure (hepatic encephalop athy), consisting of a liver flap, drowsiness, confusion and eventually coma. Liver transplantation is appropriate for some patients, although the short-term results are poor compared with Sir James Paget , 1814–1899, Surgeon, St Bartholomew's Hospital, London, UK. - - - transplantation for chronic liver disease and suitable donor livers are frequently not available in a suitable time frame owing to the precipitate deterioration.

assessment of liver function. Test Normal Signi /f_i cance range Bilirubin is synthesised in the liver 5–17 Bilirubin and excreted in bile. Increased /uni03BC mol/L levels may be associated (0.3–1.2 with increased haemoglobin mg/dL)

breakdown, hepatocellular dysfunction resulting in impaired bilirubin transport and excretion or mechanical biliary obstruction. In patients with known parenchymal liver disease, progressive elevation of bilirubin in the absence of a secondary complication suggests deterioration in liver function 30–140 The serum ALP is particularly Alkaline IU/L elevated with cholestatic liver phosphatase disease or biliary obstruction. It (ALP) is important to note that routine laboratory analysis of ALP is not isoform speci /f_i c and so

ALP from a skeletal source may also lead to elevation, particularly Paget’s disease and prostate cancer 5–40 IU/L Although signi /f_i cant liver injury Aspartate does occur in the presence of transaminase normal liver blood tests, levels (AST) of the transaminase (AST, AL T 5–40 IU/L Alanine and GGT) usually re /f_l ect acute transaminase hepatocellular damage and GGT is (ALT) a useful marker of alcohol intake 10–48 Gamma

IU/L glutamyl transpeptidase (GGT) The synthetic functions of the 35–50 Albumin liver are indicated by the ability to g/L synthesise proteins (albumin level) (3.5–5 and clotting factors (PT) and the g/dL) standard method of monitoring liver 60–85 Total protein function in patients with chronic g/L liver disease is serial measur ement (6–8.5 of bilirubin, albumin and PT g/dL) 12–16 s Prothrombin time (PT)

Ascites

Ascites

- Accumulation of ascites is a common feature of advanced liver disease irrespective of the aetiology . Development is usually insidious and fluid accumulation is associated with abdominal discomfort and a dragging sensation. CT will confirm the aeti - ology of the ascites and demonstrate the irregular, shrunken cirrhotic liver, associated portal hypertension and splenomeg - aly . Intravenous contrast will demonstrate abdominal varices - and assess patency of the portal vein. Portal vein occlusion is a common finding and in non-cirrhotic patients malignancy is usually r esponsible. The protein content and amylase levels will exclude pancrea tic ascites and determine the serum–ascites albumin gradient (SAAG), with a high gradient (>1.1 /uni00A0 g/dL) indicating portal hypertension. Cytology may confirm the presence of malignant cells, and microscopy and culture will Chapter 65). Summary box 69.11 Determining the cause of ascites /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Management of ascites in chronic liver disease The initial treatment is to restrict salt intake and commence diuretics (spironolactone or frusemide), together with advice on avoiding precipitating factors, including alcohol intake, infection and causes of hypoproteinaemia. Patients on diuret ics require regular biochemical monitoring. Summary box 69.12 Treatment of ascites in chronic liver disease /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Imaging, ultrasonography Aspiration or CT Culture and microscopy Irregular cirrhotic liver Protein content Portal vein patency Cytology Splenomegaly of Amylase level cirrhosis Salt restriction Peritoneovenous shunts Diuretics TIPSS Abdominal paracentesis Liver transplantation

Benign tumours

Benign tumours

A number of pathologies produce focal liver lesions, and the three most common benign hepatic tumours are haemangio - mas, focal nodular hyperplasia (FNH) and hepatic adenomas. These lesions are common and often discovered incidentally on cross-sectional imaging. The main clinical challenge is confirming the benign nature non-invasively where possib le. Hepatic adenoma Adenomas are benign liver tumours seen almost exclusively in women aged between 25 and 50 years. These well-defined and vascular lesions are classically associated with use of the oral found incidentally on imaging, although up to one-third may present with pain because of rupture or bleeding. Adenomas are recognised as having malignant potential, with up to 10% developing into HCC. The risk of rupture and malignancy means that surgical excision is generally recommended if >5 /uni00A0 cm in size, although some lesions may regress after discon tinuation of the oral contraceptive pill. Focal nodular hyperplasia FNH is an unusual but not uncommon benign condition of unknown aetiology , in which there is a focal overgrowth of functioning liver tissue supported by fibrous stroma. Patients are usually middle-aged women, and there is no association with underlying liver disease. Ultrasonography shows a solid tumour mass. Contrast CT or MRI may show central scarring and a hypervascular lesion. FNH contains both hepatocytes and Kup ff er cells. MRI using liver-specific contrast agents may be useful in determining the hepatocellular origin of FNH and allowing di ff erentiation of FNH from metastatic cancer. FNH does not have any malignant potential and, once the diagnosis is confirmed, does not require any treatment or follow-up. Haemangiomas These are the most common benign liver lesions, and the reported incidence has increased with the widespread availability of diagnostic ultrasonography . They consist of an abnormal plexus of vessels, and their nature is usually apparent on ultrasonography . If diagnostic uncertainty exists, CT scanning with delayed contrast enhancement shows the characteristic appearance of slow contrast enhancement owing to small vessel uptake in the haemangioma. Often, haemangiomas are multiple. Lesions found incidentally require radiological confirmation of their nature and no further treatment. Diagnosis is usually incidental, and surgical resection is only recommended if patients are significantly symptomatic or significant diagnostic uncertainty remains after multimodal imaging.

Biopsy of liver lesions

Biopsy of liver lesions

Liver biopsy is generally considered a safe procedure but is not without risk of mechanical complications, which although minor (pain and subcapsular or intrahepatic haematoma) occur in 6–25% of patients. Significant complications, including bile leak, sepsis, pancreatitis, local infections or pneumothorax, occur in 0.25% and haemobilia in 0.05% of cases. Mortality Summary box 69.14 Benign liver lesions /uni25CF /uni25CF /uni25CF /uni25CF rhage and varies from 0.1% to 0.3%. Lesions found within the liver that are likely to be malig - nant will need resection; if surgery is possible then FNA or true cut biopsy should not be performed. Biopsy of malignant liver lesions results in poorer long-term survival f ollowing resec - - tion and confers no diagnostic advantage over non-invasive imaging and tumour markers. Needle tract seeding also occurs (1000–100 /uni00A0 000 cells/tract) or peritoneal spread, particularly with HCC.

May present with symptoms or be found incidentally on imaging for another condition Signi /f_i cant symptoms (pain or pressure effects) justify surgery If imaging is equivocal biopsy may be necessary to exclude a primary or metastatic lesion If observation for 3–6 months demonstrates a stable lesion malignancy is unlikely

Blood loss and transfusion

Blood loss and transfusion

The reduction of blood loss during liver surgery has developed such that resection is often possible without blood transfusion. Better understanding of the segmental anatomy of the liver and patient selection, low central venous pressure anaesthesia and Masatoshi Makuuchi , b. 1946, surgeon, Koto Hospital, Tokyo, Japan. In 1990 described preoperative portal embolisation to increase the safety of major hepatectomy for hilar bile duct carcinoma. Jacques-Arsène d’Arsonval , 1851–1940, physician and physicist, Collège de France, Paris, France. better control of the coagulation cascade and significantly reduced bleeding in patients with liver disease and underlying coagulopathies. - The Pringle manoeuvre can r educe blood loss during parenchymal transection and improves the e ff ectiveness of other techniques used to treat oozing fr om the resected sur - face of the remnant liver. These include the topical applica - - tion of fibrin glue or fibrin-impregnated collagen fleece and non-contact electrosurgical technique such as argon plasma coagulation.

Budd–Chiari syndrome

Budd–Chiari syndrome

The Budd–Chiari syndrome a ff ects 1/1 /uni00A0 000 /uni00A0 000 adults and is a collective term for conditions that impede hepatic venous outflow at any level from the small hepatic veins to the junction of the IVC with the right atrium. Cardiac and pericardial diseases and sinusoidal obstruction syndrome are excluded from this definition. It principally a ff ects young women, who present the classic triad of abdominal pain, ascites and hepato megaly . A hypercoagulable condition such as antithrombin 3, protein C or protein S deficiency is identified in 75% of patients, extrinsic compression in 25% and rarely congenital or acquired IVC webs. The liv er becomes acutely congested, with impaired liver function and portal hypertension; ascites and oesophageal var ices develop. Fulminant hepatic failure may result fr om acute thrombosis but in the majority of cases abdominal discomfort and ascites are the main presenting features. If chronic, the liver progresses to established cirrhosis. Colour and pulsed Doppler ultrasonography and CT scanning together with detailed hae matological studies will usually identify the cause. The diagno sis should be suspected in patients with ascites where a CT scan demonstrates a large, congested liver or cirrhosis with gross enlargement of the caudate lobe resulting from preservation and hypertr ophy of the segment due to direct venous drainage to the IVC. Further IVC compression or occlusion and portal George Budd , 1808–1882, Professor of Medicine, King’s College Hospital, London, UK. Hans Chiari , 1851–1916, Austrian pathologist, later Professor at the University of Strasbourg, France. Christian Johann Doppler , 1803–1853, Professor of Experimental Physics, Vienna, Austria, enunciated the ‘Doppler principle’ in 1842. Jacques Caroli , 1902–1979, Professor of Medicine, Hôpital St Antoine, Paris, France, described the disease in 1958. Jean-Martin Charcot , 1825–1893, French neurologist and Professor of Pathology , Hôpital Pitié-Salpêtrière, Paris, France. hypertrophy . Treatment of Budd–Chiari syndrome depends on the stage of disease at presenta tion and the specific findings in each patient. Fulminant liver failure , established cirrhosis and complications of portal hypertension may require liver trans - plantation. If the liver parenchyma is relatively normal TIPSS or a side-to-side portocaval shunt should be considered and IVC compression relieved by the insertion of a retrohepatic expandable metallic stent. W ith e ff ective Budd–Chiari syn - drome treatment prognosis depends on whether it is possible to treat the underlying pathology . Patients usually require lifelong anticoagulation.

Caroli’s disease

Caroli’s disease

- Caroli’s disease is a rare congenital dilatation of the intra- - hepatic biliary tree with an incidence of <1/100 /uni00A0 000. It is often complicated by intrahepatic stone formation and presentation is usually with abdominal pain or sepsis. Imaging is diagnostic with ultrasonography or CT demonstrating intrahepatic biliary lakes containing stones which predispose to potentially life-threatening biliary sepsis. Acute infective episodes are treated with antibiotics and obstructed and infected bile ducts may be drained radiologically , endoscopically or surgically . - Ductal dilatation is usually di ff use but if segmental may be treated by resection of the a ff ected part. Cholangiocarcinoma is more common than for other forms of biliary ectasia and treatment by liver transplantation is described.

Chronic liver disease

Chronic liver disease

Liver disease is the third leading cause of premature death in the UK, and since 1970 deaths have increased by 400%. Liver - disease is potentially preventable in 90% of cases, and 75% of patients present with late-stage disease. Lethargy and weakness are common features, irrespective of the aetiology , and often precede clinical jaundice. In advanced cirrhosis, glucuronyl conjugation of bilirubin and biliary excretion of conjugated Causes of acute liver failure /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Summary box 69.4 Supportive therapy for acute liver failure /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF bilirubin are impaired and jaundice develops. Progressive dete rioration in liver function is associated with a hyperdynamic circulation with a high cardiac output, large pulse volume, low blood pr essure and flushed warm extremities. Fever is common and may be related to underlying inflammation and cytokine release or bacterial infection due to innate immune dysfunc tion in acute and chronic liver disease. Skin changes include spider naevi (cutaneous vascular abnormalities that blanch on pressure), palmar erythema and white nails (leukonychia) and endocrine abnormalities produce hypogonadism and g ynaecomastia. Hepatic encephalopathy is responsible for the mental derangement with memory impairment, confusion, personality changes, altered sleep patterns and slow , slurred speech. The most useful clinical sign is a flapping tremor when SA Kinnier Wilson , 1878–1937, Professor of Neurology , King’s College, London, UK. Ralph Douglas Reye , 1912–1977, pathologist, Royal Alexandra Hospital for Children, Sydney , Australia. King's College selection criteria for liver transplantation in acute liver failure: paracetamol (acetaminophen) and non-paracetamol induced /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF the patient extends their arms while hyperextending the wrist joints. Ascites is a common late feature causing abdominal distension, detected clinically by the demonstration of a fluid thrill or shifting dullness. Protein catabolism produces sarco - penia and wasting, and bruising suggests a coagulopathy . - Summary box 69.6 Features of chronic liver disease /uni25CF /uni25CF - /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Viral hepatitis (hepatitis A, B, C, D, E) Drug reactions (halothane, isoniazid − rifampicin, antidepressants, non-steroidal anti-in /f_l ammatory drugs [NSAIDs], valproic acid) Paracetamol overdose Prescription medicines, including antibiotics, NSAIDs, anticonvulsants and statins Herbal supplements Mushroom poisoning Toxins, including carbon tetrachloride in refrigerants, solvents for industrial use and varnishes Shock and multiorgan failure Autoimmune disease Acute Budd − Chiari syndrome Rare metabolic disorders, including Wilson’s disease Cancer Fatty liver of pregnancy Heat stroke Reye’s syndrome in children following a viral infection including Chickenpox Severe acute respiratory syndrome coronavirus (SARS-CoV-2) can cause liver failure in up to 20% of patients with a severe episode Fluid balance and electrolytes Acid–base balance and blood glucose monitoring Nutrition Renal function (haemo /f_i ltration) Respiratory support (ventilation)

Monitoring and treatment of cerebral oedema Treat bacterial and fungal infection Extracorporeal liver support devices (principally as a bridge to transplantation) Paracetamol toxicity Non-paracetamol toxicity Criteria met if INR >6.5 (PT Criteria met if arterial pH <7.30

> “ 100 s) OR/AND OR/AND All three of the following Three out of /f_i ve of the present: following present: INR >6.5 (PT >100 s) Age less than 10 or greater Serum creatinine 3.4 than 40 mg/dL (301 µmol/L) Aetiology non-A, non-B Grade III or IV a hepatitis, idiosyncratic drug encephalopathy reactions b Additionally, Duration of jaundice Hyperlactaemia or before development of hyperphosphataemia are encephalopathy >7 days strong predictors of poor PT greater than 50 s prognosis for survival (approximate INR >3.5) without transplantation Serum bilirubin >18 mg/dL (300 µmol/L) INR, international normalised ratio; PT, prothrombin time. a Hepatic encephalopathy grades can be described as: Grade 1: inverted sleep pattern, agitation, forgetfulness, irritability, apraxia Grade 2: lethargy, time and/or place disorientation, personality change, ataxia Grade 3: somnolence to semistupor but responds to verbal stimuli, place disorientation, asterixis, hyperactive re /f_l exes Grade 4: coma b The addition of lactate or phosphate thresholds to the criteria may improve sensitivity and negative predictive value. Lethargy Portal hypertension Fever Ascites Jaundice Oesophageal varices Protein catabolism (wasting) Splenomegaly and hypersplenism Coagulopathy (bruising) Cutaneous Cardiac (hyperdynamic circulation) Spider naevi Neurological (hepatic Palmar erythema encephalopathy)

A number of parameters are required to accurately assess the degree of liver dysfunction, enable predictions about a patient’s ability to tolerate surgical or radiological procedures and assess the prognosis following transplantation. Two prog nostic models commonly used are the Child–Turcotte–Pugh (CTP) classification (Table 69.2) and the Model for End-Stage Liver Disease (MELD) score. The original Child classifica was developed to predict mortality following shunt surgery in patients with cirrhosis, with the CTP classification modified to predict mortality after any surgery . The MELD score was devised to predict the short-term prognosis following transjug ular intrahepatic portosystemic stent shunt (TIPSS) but has been adopted to prioritise patients on liver transplant waiting lists. In the MELD model survival probability is calculated based on the patient’s international normalised ratio (INR), serum bilirubin and creatinine. Operating in the presence of chronic liver disease Surgical and anaesthetic complications are increased in chronic liver disease, with the risk dependent on the magni tude of the procedure, degree of liver impairment and type of anaesthesia. Overall surgical mortality rates are increased by 10% in CTP-A disease, 30% in CTP-B and 75–80% in CTP-C (Table 69.2). MELD scores cor relate with operative mortality: 1% increase for each MELD point up to 20 and a further 2% for each point above 20, with rates considerably higher following emergency presentation.

TABLE 69.2 Child–Turcotte–Pugh (CTP) classi /f_i cation of hepatocellular function in cirrhosis. Points 1 point each 2 points each 3 points each Bilirubin (/uni03BC mol/L) <34 34–50

50 Albumin (g/L) 35 25–35 <25 Ascites None Easily controlled Poorly controlled Encephalopathy None Grade I or II Grade III or IV INR <1.7 1.7–2.2 2.2 CTP-A, 5 or 6 points; CTP-B, 7–9 points; CTP-C, 10–15 points. INR, international normalised ratio.

CHRONIC LIVER DISEASE

Several rare chronic liver conditions are important because they require a specific investigation plan and treatment and may imitate more common clinical conditions (Table 69.6

TABLE 69.6 Important chronic liver conditions. Condition Common presentations Primary sclerosing cholangitis Abnormal LFTs, pruritus or jaundice Primary biliary cirrhosis Malaise, lethargy, pruritus, abnormal LFTs Budd–Chiari syndrome Ascites, pain, abdominal distension Caroli’s disease Abdominal pain, sepsis, biliary obstruction Simple liver cysts Coincidental /f_i nding, pain, palpable mass Polycystic liver disease Hepatomegaly, pain LFT, liver function test.

Clinical signs

Clinical signs

Depending on the severity of liver dysfunction, the aetiology and acute or chronic development, symptoms vary and combi nations occur. The most common include jaundice, drowsiness, abdominal pain/swelling, nausea, tremors, vomiting, malaise, confusion and disorientation, bruising, peripheral oedema and foetor hepaticus (strong musty smell to the breath).

Colorectal liver metastases

Colorectal liver metastases

Worldwide colorectal cancer (CRC) is the third most common solid organ malignancy and the fourth most common cause of cancer-related deaths. Up to 70% of patients with CRC develop synchronous (15–25%) or metachronous (20–45%) liver metastases. Thirty years ago, metastatic CRC was asso - ciated with a 5-year survival of less than 3%; however, liver resection in selected patients with liver-only metastatic disease demonstrated 5-year survivals of 50%, so the potential benefits were recognised. Despite recent advances in chemotherapeutic agents, resection remains the only potentially curative option, but only 20% of patients will be candidates at presentation. A further 20–30% will become operable following chemotherapy , the 5-year overall survival rate following resection is 50% and tumour recurs in 65% with the recurrent disease limited to the liver in 40%. - Defining resectability for colorectal liver metastases t - Previously , patients with synchronous metastases, a rectal primary , multiple di ff use metastases, metastases larger than 5 /uni00A0 cm, disease-free intervals of less than 1 year from the diag - - nosis of primary disease or a high serum CEA were considered irresectable and suitable only for palliative treatment. Modern surgical techniques and chemotherapy regimes now mean that r esection with curative intent is defined as the ability to successfully remove all residual disease from the liver with clear surgical margins, leaving adequate disease-free viable liver. Technical contraindications to resection are related to the anatomical location of metastases, principally the involvement - of major vascular or biliary structures. An R0 resection with a negative surgical margin of 1 /uni00A0 cm - is considered the gold standard, but with e ff ective modern chemotherapy patients with an R1 resection (≥ 1-mm margin have similar survivals to those with R0 resections. An FLR of 25% of preoperative volume is considered su ffi cient to prevent postoperative hepatic failure (see Portal vein embolisation Historically , extrahepatic metastatic disease was considered a contraindication to liver surgery , but long-term survival is now possible in selected patients. Survival after lung resection for colorectal metastases is similar to that seen after liver resection (40–50%) with low morbidity and mortality . Staging and selection of patients for liver surgery A specialist multidisciplinary team should coordinate the staging of patients with colorectal liver metastases and the treatment plan. Routine staging involves triple-phase CT chest/abdomen/pelvis, contrast MRI scan of the liver, whole- body PET-CT to identify metastatic disease with/without laparoscopy and intraoperative ultrasonography . Chemotherapy for colorectal liver metastases Despite new chemotherapeutic agents and locoregional therapies (embolisation, percutaneous ablation, hepatic artery- directed infusion chemotherapy , internal radiation) the role of adjuvant and neoadjuvant chemotherapy remains unclear. The traditional approach was to resect an operable colonic primary followed by routine postoperative chemotherapy or holding chemotherapy ‘in reserve’ in case of metastases. Neoadjuvant chemotherapy is now recommended by some groups for the majority of patients even if the liver disease is resectable. The aim is to reduce lesion size and improve resectability while treating occult disease and revealing the tumour biology where progression despite chemotherapy signifies a poor prognosis. A major development with advanced disease was the recognition that a subgroup of patients may become resectable after systemic chemotherapy .

Although resectability rates after c hemotherapy for initially irresectable disease vary , when successful 5-year survivals of 35–50% are similar to disease resectable at presentation. Chemotherapy with 5-fluorouracil and folinic acid produces a response rate of approximately 30% but combination with oxaliplatin increases this to 50–60%. Combination chemotherapy with monoclonal antibodies that recognise vascular endothelial growth factor receptor (VEGFR) or epidermal growth factor receptor (EGFR) provide additional benefit (see also Chapters 12 and 77).

Complications of liver trauma

Complications of liver trauma

A subcapsular or intrahepatic haematoma requires no specific intervention and should be allowed to resolve spontaneously . Abscesses may form as a result of secondary infection of an area of parenchymal ischaemia and treatment is systemic antibiotics and US-guided aspiration once liquefaction has occurred. Bile collections require US-guided aspiration with/ without drain insertion and biliary fistulae are investigated by endoscopic or percutaneous cholangiography with/without stent insertion for biliary decompression. If a fistula persists liver resection may be required. Late vascular complications include hepatic artery aneu rysms and arteriovenous fistulae (hepatic artery to hepatic vein, César Roux , 1857–1934, Professor of Surgery and Gynaecology , Lausanne, Switzerland. Described the Roux-en-Y loop in 1908.). tension) and arteriobiliary fistulae indicated by haemobilia are treated by embolisation. Hepatic insu ffi ciency may occur following extensive liver trauma but usually recover s following supportive treatment if the blood supply and biliary drainage - to an adequate liver remnant are preserved (Figure 69.11). - Summary box 69.8 - Complications of liver trauma /uni25CF /uni25CF /uni25CF /uni25CF tic /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Unstable Liver Resuscitate trauma • Investigate • Peritoneal lavage • Ultrasound Stable • CT • Laparoscopy • Angiography Figure 69.11 Algorithm for management of liver trauma. CT, computed tomography. Intrahepatic haematoma Biliary strictures Liver abscess Intra-abdominal collections Bile collection Hepatic artery aneurysm Biliary /f_i stula Arteriovenous /f_i stulae Haemobilia Arteriobiliary /f_i stulae Ascites Liver failure

Cystic lesions

Cystic lesions

Simple liver cysts Simple cysts of the liver are usually asymptomatic and were thought to be uncommon before the routine use of ultra - sonography . The exact prevalence and incidence are not known but they are estimated to occur in 5% of the population with 10–15% presenting because of symptoms. Fortunately , their appearance on ultrasonography and MRI scanning generally allows them to be dismissed; confusion usually only occur s when CT scanning was the initial investigation. Cysts are more common in females (1.5:1) and are larger in patients over 50 years of age. The situation is di ff erent for very large symptomatic or complex cysts, which are 10 times more common in women, and huge cysts requiring treatment in men are rare. Most cysts only cause problems and require treatment when they increase in size or become complex or infected. Prior to treatment it is important to exclude other causes, particularly parasitic infections. Polycystic liver disease Multiple liver cysts are frequently associated with adult poly - cystic kidney disease (PKD) but may occur alone. PLD is a term for a heterogeneous group of patients; it is an inherited disorder estimated to a ff ect around 1 in 100 /uni00A0 000 people and 10% ha ve cerebral aneurysms. It is characterised by the progressive growth of cysts of various sizes that are widely and randomly distributed throughout the liver. In many patients the cysts are asymptomatic but when extensive they produce mechanical symptoms from stretching of the liver capsule, pressure e ff ects on the stomach when the left side is involved and gross abdominal distension related to the large increase in the size of the liver. Cysts can become infected, but this normally follows ill-advised aspiration of what is felt to be a symptomatic cyst. Biliary obstruction occurs as a result of distortion or compression and must be treated endoscopically as there is no surgical option. Surgical treatment is usually employed for bulk reduction or a mechanical problem and, when planning surgery , it is important to remember that bili - ary radicals and vessels run between cysts and are di ffi cult to di ff erentiate from the wall. Hepatic cystadenomas and cystadenocarcinomas The prevalence of hepatic cystadenomas is low , with fewer than 200 cases reported. Of the two types, cystadenomas with mesenchymal (ovarian-like) stroma occur only in females and men. Cystadenomas are often multilocular, septated, non calcified and surrounded by compressed liver tissue. Upper abdominal pain and an abdominal mass can occur, but patients are usually asymptomatic. Cystadenomas are slow growing but have malignant potential and must be distinguished from simple hepatic cysts , polycystic liver disease, hydatid cysts and complex non-neoplastic cystic lesions. Serum and cyst fluid CA 19-9 levels may be elevated in cases of cystadenoma with mesenchymal stroma, but serum carcinoembryonic antigen (CEA) is unhelpful although it may be raised in cyst fluid. Cyst fluid cytology is unreliable. Ultrasonography may demonstrate an anechoic mass with internal septations. CT scanning generally demonstrates the multilocular nature with internal septations. In the contrast phase, the wall of the cyst enhances and nodules may be seen. MRI also demonstrates septations and a hyperintense signal on T2-weighted images. The risk of recurrence and the potential for malignant transformation mandates complete anatomical resection. If the lesions are technically irresectable or cannot be separated from major venous or arterial structures liver transplantation may be required. Biliary

cystadenocarcinomas account for 0.4% of malignant epithelial hepatic lesions.

Diagnosis and grading of liver injury

Diagnosis and grading of liver injury

The liver is an extremely well-vascularised organ and blood loss is the major early complication following injury . A high index of suspicion is essential with any chest or upper abdominal stab wound, especially where significant blood loss is obvious. Severe crushing injuries to the lower chest or upper abdomen frequently result in rib fractures, haemothorax and splenic and/or liver injury . Focused assessment sonography in trauma (FAST) performed by an experienced operator will identify free intraperitoneal fluid. In haemodynamically unstable patients with penetrating wounds a laparotomy and/or thoracotomy is indicated once active resuscitation has commenced. Penetrat - ing injuries are frequently associated with massive, continued blood loss and coagulopathies; transfer to the operating resuscitation continues. In haemodynamically stable patients, urgent contrast-enhanced CT scan of the chest and abdomen is performed to look for parenchymal injury and concomitant damage to other thoracic or abdominal organs. The American Association for the Surgery of Trauma liver injury scale was revised in 2018 to incorporate vascular injury such as pseudoaneurysm and arteriovenous fistula. The guide lines recommend dual arterial/portal venous phase imaging (Table 69.5). /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Penetrating injuries Modern approaches to liver trauma are based on conserva - tive management where possible. The initial management is maintenance of airway patency , breathing and circulation (ABC), following the principles of advanced trauma life - support (ATLS). Peripheral venous access requires tw o large- bore cannulae and blood is sent for cross-match of 10 units of blood, full blood count, urea and electrolytes, liver function tests, clotting screen, glucose and amylase. Initial volume replacement should be with blood; arterial blood gases should be obtained; and the patient intubated and ventilated if gas exchange is inadequate. Intercostal chest drains are indicated if an associated pneumothorax or haemothorax is suspected. Once resuscitation has commenced, the patient should be transferred to the operating theatre, with further resuscitation performed on the operating table. The necessity for fresh-frozen plasma (FFP) and cryoprecipitate should be discussed with the blood transfusion service immediately the patient arrives in the hospital (often by activation of a major transfusion protocol), as these patients rapidly develop irreversible coagulopathies due to a lack of fibrinogen and clotting factors. Standard coagulation profiles are inadequate to evaluate this acute loss of clotting factors, and products should be given empirically aided by the results of thromboelastography if available (see Chapter 2). Blunt trauma Enhanced resuscitation, anaesthesia and intensive care have contributed to reduced mortality rates. Optimum results are obtained with specialist teams that include experienced liver surgeons, anaesthetists, endoscopists and interventional radiol - ogists (Figure 69.8). Initial resuscitation and management are as outlined for penetrating injuries. Unstable patients require immediate lap - arotomy , b ut the majority of haemodynamically stable patients should be managed non-surgically

(management depends on haemodynamic stability not the grade of injury). Haemodynamic instability and signs of generalised peri - tonitis mandate surgical intervention. Interventional radiology with embolisation for hepa tic arterial bleeding is safe and

TABLE 69.5 Grading of liver injuries according to American Association for the Surgery of Trauma. Grade 1 Haematoma: subcapsular, <10% surface area Laceration: capsular tear, <1 /uni00A0 cm parenchymal depth Grade 2 Haematoma: subcapsular, 10–50% surface area Haematoma: intraparenchymal, <10 /uni00A0 cm diameter Laceration: capsular tear 1–3 /uni00A0 cm parenchymal depth, <10 /uni00A0 cm length Grade 3 Haematoma: subcapsular, >50% surface area of ruptured subcapsular or parenchymal haematoma Haematoma: intraparenchymal, >10 /uni00A0 cm Laceration: capsular tear, >3 /uni00A0 cm parenchymal depth Vascular injury with active bleeding contained within liver parenchyma Grade 4 Laceration: parenchymal disruption involving 25–75% hepatic lobe or involves 1–3 Couinaud segments Vascular injury with active bleeding breaching the liver parenchyma into the peritoneum Grade 5 Laceration: parenchymal disruption involving >75% of hepatic lobe Vascular: juxtahepatic venous injuries (retrohepatic vena cava/central major hepatic veins) Additional points: • Advance one grade for multiple injuries up to grade III. • ‘Vascular injury’ (i.e. pseudoaneurysm or arteriovenous /f_i stula): appears as a focal collection of vascular contrast that decreases in attenuation on delayed images. • ‘Active bleeding’: focal or diffuse collection of vascular contrast that increases in size or attenuation on a delayed phase. (a) (b) Figure 69.8 Computed tomography scans demonstrating the signi /f_i cant differences between blunt (a) and penetrating (b) trauma (assault with a kitchen knife).

e ff ective in stable patients. If conservative management is successful patients are discharged after 8–10 days, advised to avoid abdominal trauma and rescanned after 6–8 weeks. If fever, bleeding or pain occurs prompt readmission is required.

Figure 69.9 Packing the liver to achieve haemostasis. The abdomen can then be closed and the patient transferred to critical care for stabilisa tion prior to relook laparotomy 24–48 hours later. (Adapted from Poston GJ, D’Angelica M, Adam R (eds). and pancreatic disorders . Boca Raton: CRC Press, 2010.)

Endoscopic retrograde cholangiopancreatography

Endoscopic retrograde cholangiopancreatography

- Endoscopic retrograde cholangiopancreatography (ERCP) is performed in patients with obstructive jaundice when a thera - peutic endoscopic procedure is indicated (see Chapter 9). It provides definitive views of Klatskin tumours (hilar cholangiocarcinoma) and facilitates staging by defining the extent of involvement of intrahepatic ducts. ERCP also provides clear images of the intrahepatic ducts in primary sclerosing cholangitis (PSC) and facilitates stenting and drain age of obstructed, infected segments. Endoscopic (peroral) cholangiography Cholangioscopy enables direct visualisation of the bile ducts, either operatively or endoscopically . The original ‘mother and baby’ ERCP cholangioscopy has been superseded by SpyGlass™ (Boston Scientific) cholangioscopy , which enables a single operator to examine the biliary mucosa. The inclusion of a working channel facilitates visualised biopsies and targeted therapy . Targeted biopsies improve diagnostic yields, with an overall accuracy of 85–95% in patients with indeterminate biliary strictures compared with 55–90% for brush cytology . Endoscopic ultrasonography Endoscopic ultrasonography (EUS) is predominantly used to evaluate the extrahepatic biliary tree and pancreas, but linear Summary box 69.7 Management of liver trauma /uni25CF nodes and the caudate lobe and assessment of the liver paren - chyma (Figure 69.7). Percutaneous transhepatic cholangiography Percutaneous transhepatic cholangiography (PTC) is indicated where endoscopic cholangiography has failed or is impossible because of anatomical constraints from previous surgery or malignant involvement of the duodenum. Tumour extent in patients with hilar tumours can be assessed and combined percutaneous/endoscopic or antegrade metal stent placement is facilitated. Laparoscopy and laparoscopic ultrasonography Laparoscopy is useful to stage primary hepatopancreatobiliary cancers. Unrecognised peritoneal metastases, superficial liver tumours and peritoneal disease can be identified and biopsied, avoiding an inappropriate laparotomy . Routine biopsy of resectable lesions is contraindicated to avoid tumour seeding. -

Figure 69.7 Endoscopic ultrasonography demonstrating

liver paren chyma and a caudate lobe metastasis being biopsied. Remember associated injuries At-risk groups Stabbing or gunshot to lower chest or upper abdomen Crush injury with multiple rib fractures High-speed road traf /f_i c accident Resuscitate Airway Breathing Circulation Assessment of the injury CT chest and abdomen with contrast Laparotomy if haemodynamically unstable Treatment Correct coagulopathy Suture lacerations Resect if major vascular injury Packing if diffuse parenchymal

injury

FURTHER READING

FURTHER READING

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Follow-up

Follow-up

Optimal follow-up remains controversial, and protocols vary . Close observation will identify patients who may benefit from further surgery and at least a yearly scan should be performed for the first 5 years. Tumour markers are measured if initially elevated and patients seen if symptoms develop. Recurrence within 12 months has a poor prognosis.

Hepatobiliary iminodiacetic acid

Hepatobiliary iminodiacetic acid

Hepatobiliary iminodiacetic acid (HIDA) labelled with tech - 99m netium-99m (Tc-HIDA) is concentrated by hepatocytes and excreted with bile, visualising intrahepatic uptake, the extrahepatic biliary system and the gallbladder. Under normal circumstances HIDA enters the duodenum within 30 minutes and scintigraphy produces an image together with an activity– time curve. HIDA scanning is useful because biliary excretion occurs despite hyperbilirubinaemia (<85 /uni00A0 µmol/L). It is most - commonly used to investigate biliary atresia, to investigate jaundice in liver transplant patients and to demonstrate patency of the biliary tract.

INFECTIVE CONDITIONS OF THE LIVER Ascending cholan

INFECTIVE CONDITIONS OF THE LIVER Ascending cholangitis

- Ascending cholangitis is a potentially life-threatening emergency associated with infection of the biliary tree and usually associated with obstruction. It presents with clinical jaundice, rigors and a tender right upper quadrant (Charcot’s triad). The most common bacteria linked to ascending /uni00A0 chol - angitis /uni00A0 are gram-negative bacilli: Escherichia coli (25–50%), - Klebsiella (15–20%) and Enterobacter (5–10%). The diagnosis is confirmed by the finding of dilated bile ducts on ultra - sonography , an obstructive picture of liver function tests and organisms identified from blood cultures. Delay in appropriate treatment may result in multiorgan failur e secondary to sepsis - and broad-spectrum antibiotics, rehydration, and endoscopic - or percutaneous transhepatic drainage are urgently required. Biliary stone disease is a common predisposing factor although strictures, pancreatitis, pancreatic tumours and parasites may also be responsible. If an obstructive cause is identified it must be urgently treated by ERCP , sphincterotomy (± stent) or percutaneous drainage. Microbial contamination of the liver leading to a liver abscess continues to occur at a fairly constant rate of approximately 1/5000 hospital admissions. The incidence of causative organ isms varies and reflects changes in aetiology and geographical distribution. Bacterial, parasitic and fungal organisms can cause liver abscess but, worldwide, bacteria remain the most common; although infection is usually polymicrobial Klebsiella, Escherichia coli and the Streptococcus milleri group are the usual organisms identified. There is an increased incidence in the elderly , those with diabetes and the immunosuppressed and presentation is usu ally with anorexia, fever, malaise and right upper quadrant discomfort. The overall mortality has declined because of improv ed imaging and e ff ective antimicrobial therapy and the outcome is incr easingly dependent on the underlying cause and the presence of comorbidities. Biliary tract pathology is the most common source (35%), followed by portal spread from the gastrointestinal tract, including diverticulitis and appendicitis (20%). Other unusual aetiologies include contiguous spread from subphrenic or intra-abdominal collections, bacteraemia secondary to trauma or infected cysts and necrotic tumours following chemother apy . The cause is not identified in 10% of cases and a number of liver abscesses become recurrent (12–38% depending on whether the responsible organism is identified and whether the patient has diabetes). The diagnosis is suggested by the finding of a multiloculated cystic mass on ultrasonography or CT scan (Figure 69.18) and is confirmed by aspiration. Treatment of liver abscesses initially requires identification of the source, if possible, aspiration of the lesion for micro biology and culture (r epeated aspirations may be required) and treatment with appropriate antibiotics. Simple cysts containing debris, hydatid cysts, necrotic tumours

and non-infected haematomas (after unrecognised or occasional trivial trauma) can all be mistaken for abscesses. Antibiotic treatment using Theodor Albrecht Edwin Klebs , 1834–1913, Professor of Bacteriology successively at Prague, Czechoslovakia, Zurich, Switzerland, and the Rush Medical College, Chicago, IL, USA. Metronidazole and clindamycin provide wide anaerobic coverage and excellent penetration into the abscess cavity . Third-generation cephalosporins and aminoglycosides are - very e ff ective against most Gram-negative organisms.

Figure 69.18 Liver abscess. Computed tomography scan showing an air– /f_l uid level and rim enhancement (open arrow). The second lesion seen is a haemangioma (closed arrow).

INVESTIGATING LIVER DISEASE Imaging modalities

INVESTIGATING LIVER DISEASE Imaging modalities

Major advances in surgical approaches to the liver required improvements in preoperative imaging. The choice of imaging modality is determined by the likely pathology and locally available equipment and expertise (Table 69.3). Charles Gardner Child , 1908–1991, surgeon, Michigan, USA. Child and colleagues from King’s College Hospital, London, UK, modified the scoring system by replacing nutritional status with prothrombin time or international nor malised ratio. - tion - Ultrasonography Ultrasonography (US) is traditionally the first imaging modality for the investigation of jaundice and right upper quadrant pain (Figure 69.3). Good quality US can characterise benign cystic lesions but is operator dependent; accurate characterisation of malignant pathologies is not possible. Body habitus and anatomical variations frequently prevent detailed examina - tion, with steatosis reducing liver penetration; US should not be used when malignancy is suspected. US can reliably identify biliary dilatation but not the aetiology , and ov erlying air-containing viscera often prevent complete examination. Contrast-enhanced ultrasonography (CEUS), introduced in the early 1990s, uses transient intravas - cular bubbles to di ff erentiate normal liver parenchyma from solid tumours. CEUS increases US sensitivity and specificity by assessing real-time lesion vascularity with an accuracy for focal liver lesions of 90%. Computed tomography Modern spiral computed tomography (CT) technology has increased the accuracy of diagnosis and staging of liver lesions, and contrast-enhanced CT is currently the most - widely used and best validated modality . Fine detail of liver lesions with resolutions of 6–8 /uni00A0 mm is possible, liver algorithms allow characterisation and density data identify cystic lesions (Figure 69.4a). The early arterial phase following intravenous contrast detects small hepatocellular carcinomas (HCCs) owing to their predominantly arterial blood supply . The venous phase demonstrates branches of the intrahepatic portal vein and the hepatic veins. Inflammatory liver lesions often exhibit rim enhancement with intravenous contrast, whereas haemangi - omas characteristically show late venous enhancement. CT is extremely accurate when assessing the stage and resectability of liver tumours apart from peritoneal metastases. Magnetic resonance imaging Magnetic resonance (MR) of the liver is superior to CT in char - acterising focal lesions (Figure 69.4b). The resolution for small metastases is superior and is further improved by liver-specific and Jeremiah G Turcotte first proposed the scoring system in 1964. In 1972, RN /uni00A0 Pugh -

Figure 69.3 Ultrasound scans of the liver. (a) Normal liver /uni00A0 and (b) surveillance for a patient with cirrhosis demonstrating hepatocellular carcinoma (arrow).

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Imaging modality Principal indication Ultrasonography Standard /f_i rst-line investigation CEUS 90% accurate for focal lesions Spiral CT Investigation of malignancy Cancer surveillance Anatomical planning for liver surgery MRI Alternative to spiral CT Characterisation of liver lesions Liver-speci /f_i c contrast agents are taken up by hepatocytes, which are absent in malignant lesions, which consequently contrast with the enhanced background liver MRCP First-line, non-invasive cholangiography Investigation and surveillance of parenchymal liver disease (sclerosing cholangitis, autoimmune cholangitis) for the development of malignancy ERCP Therapeutic procedure only Imaging the biliary tract when endoscopic intervention is required (stones, strictures, iatrogenic and traumatic injury) PTC Biliary tract imaging when ERCP not possible or failed EUS Generally for examination of the extrahepatic biliary tree and pancreas Caudate lobe, hilar nodes and liver parenchyma can be assessed Octreotide scanning Form of scintigraphy used to identify and localise NETs and carcinoid tumours Particularly useful to exclude metastatic disease HIDA scanning Determination of the patency of the intra- and extrahepatic biliary system and investigation of biliary atresia and jaundice following liver transplantation Angiography To detect vascular involvement by tumour Treatment of vascular pathology (pseudotumours, haemobilia, iatrogenic injuries, trauma) PET scanning To quantify tumour spread Differentiate benign and malignant pathologies Laparoscopy To detect peritoneal and serosal disease, assess the extent of tumours and spread ± laparoscopic Ultrasonography to determine the relationship of tumours to vascular structures and biopsy of liver tumours ultrasonography and super /f_i cial lesions CEUS, contrast-enhanced ultrasonography; CT, computed tomography; ERCP , endoscopic retrograde cholangiopancreatography; EUS, endoscopic ultrasonography; HIDA, hepatobiliary iminodiacetic acid; MRCP , magnetic resonance cholangiopancreatography; MRI, magnetic resonance imaging; NET, neuroendocrine tumour; PET, positron emission tomography; PTC, percutaneous transhepatic cholangiography. /uni00A0 (a) Figure 69.4 Computed tomography (CT) and magnetic resonance imaging (MRI) scans of the same patient following a road traf /f_i c accident and fall from a motorcycle. CT scan (a) was interpreted as a traumatic haematoma, but the MRI scan carcinoma. (b) (b) demonstrated an incidental hepatocellular

contrast agents, particularly in di ff erentiating between small HCCs and regenerative nodules. Magnetic resonance cholan giopancreatography (MRCP) provides excellent, non-invasive imaging of the intra- and extrahepatic biliary tract with an accuracy comparable to direct cholangiography . Positron emission tomography Positron emission tomography (PET) scanning is a functional test that demonstrates the metabolic activity of a tissue. A variety of tracers are available depending on the process being investigated. For the investigation of malignancy 18 F-2-fluoro-2-deoxy-d-glucose is commonly used, and detec tion depends on the avid uptake of glucose by malignant cells compared with benign or inflammatory tissue. Deoxyglucose is 18 labelled with the positron emitter fluorine-18 (F-FDG) which is administered prior to PET imaging. A three-dimensional image of the whole body is obtained, highlighting areas of increased glucose metabolism (Figure 69.5). A positive PET scan result does not always indicate malignant disease (inflam mation being the most common cause of a false-positive result), and conversely false-negative results occur. A critical mass is required for adequate uptake to be detectable, and resolution is similar to CT and MRI. PET scanning is particularly useful for the detection of metastatic disease and confirmation of lymph node involvement, serving as ‘its own control’ if some lesions prove to be FDG avid and some are

cold. Angiography Angiography is almost exclusively employed when therapeutic intervention is considered; occlusion of arteriovenous malfor mations, embolisation of bleeding sites in the liver and the treatment of liver tumours by transarterial chemoembolisation (TACE). Octreotide scan Octreotide scanning is a form of scintigraphy in which radio isotopes attached to drug carriers are taken up by specific tissues or processes (Figure 69.6). Octreotide is an octapeptide that pharmacologically mimics somatosta tin. When radiolabelled with indium-111 and administered intravenously it is taken Gerald Klatskin , 1910–1986, pathologist, Yale School of Medicine, New Haven, CT , USA. - - up by tumour cells containing somatostatin receptors; emitted gamma radiation is detected by a scintillation camera and a whole-body image constructed. It is particularly useful for the identification of carcinoid and neuroendocrine tumours (NETs) and metastatic disease, with a 75–100% sensitivity for - detecting pancreatic NETs.

(b) Figure 69.5 Computed tomography scan (a) and positron emission tomography scan (b) of a patient with a large colorectal metastasis and hilar lymph

adenopathy. Figure 69.6 Octreotide scan demonstrating liver metastases from a neuroendocrine tumour with additional metastatic disease in medias

tinal lymph nodes.

Intrahepatic cholangiocarcinoma

Intrahepatic cholangiocarcinoma

Cholangiocarcinomas develop in the bile duct and demonstrate considerable variation in origin, behaviour and pathophysiol ogy . Twenty per cent of cholangiocarcinomas are intrahepatic (ICC), representing 5–30% of primary liver tumours, which is second only to HCC. The y are rare in the West with an incidence of 0.5–2/100 /uni00A0 000, although the incidence is increasing and is significantly higher in South East Asia, where Clonorchis sinensis infection is endemic, and in Thailand the rate is 60/100 /uni00A0 000. There are three morphological subtypes – infiltrating periductal (Figure 69.23a), mass-forming (Figure 69.23b) and intraductal – and ICC is now considered to have multiple cellular origins. Ultrasonography will confirm intrahepatic biliary obstruction and MRCP is prefer red for diagnosis. ERCP will delineate tumour extent and with brush TM cytology or SpyGlass can achieve pathological confirmation of malignancy . ICC was originally staged with HCC but is now classified separately . Prognostic pathological features include vascular invasion, tumour multiplicity , local extension, periductal infil tration and lymph node metastasis. CT scanning is the best staging modality to identify distant metastases and confirm tha the lesion is primary and not metastatic. Angiography is some times required preoperatively to assess vascular involvement. The sensitivity and specificity of PET-CT for diagnosis of cholangiocarcinoma varies by location, being higher for intra hepatic (>90%) than for extrahepatic (60%) tumour s, although detection rates for distant metastases approach 100%. ICC is an aggressive tumour and, even when confined to the liver, only 30% of patients are suitable for resection at the time of presentation. If surgical resection is considered, biopsy should be av oided; in borderline cases diagnostic laparoscopy and intraoperative ultrasonography will exclude additional hepatic or peritoneal disease. Lymph node status (porta hepa tis, common hepatic artery and the gastroduodenal ligament) remains an important prognostic factor and should be sam pled. One-year survival rates have improved to 25%, although Vincenzo Mazzaferro , b. 1957, surgeon, Istituto Nazionale dei Tumori, Milan, Italy . - - - 5-year survival of 3% remains unchanged. Unfortunately , conventional chemotherapy o ff ers limited survival benefit for unresectable or metastatic disease. -

Figure 69.23 Hilar cholangiocarcinoma (Klatskin

tumour) demonstrat

ing tight stricture and intrahepatic dilatation of intrahepatic ducts due to in /f_i ltrating tumour (a) and an apparent space-occupying lesion due to the mass-forming variety (b) .

Introduction

INTRODUCTION

The liver is a highly complex organ found only in vertebrates that is responsible for over 500 individual functions. It is located in the right upper quadrant, protected by the ribs, and weighs on average 1.5 /uni00A0 kg (970–1860 /uni00A0 g). It is wedge shaped in both the coronal and axial planes and is divided by the middle hepatic vein into two lobes, with the larger right lobe generally representing 60% by volume. The parenchyma is covered by a thin capsule (Glisson’s capsule) and visceral peritoneum apart from the posterior surface, the ‘bare area’. Surgery for hepatic disease evolved slowly because of the complexity of hepatic function and anatomy . Remarkable progress has been made since the first formal resection in 1952 with the advent of cross-sectional imaging, liver transection technology and low central venous pressure anaesthesia. Progress continues with the incorporation of laparoscopic and robotic surgery and training techniques, including virtual reality .

LIVER TRAUMA

LIVER TRAUMA

Liver injury due to blunt or penetrating abdominal trauma is second in frequency only to that of the spleen. Blunt injury produces contusion, laceration and avulsion, often associated with splenic, mesenteric or renal injuries. Penetrating injuries, including stab and gunshot wounds, are often associated with chest or pericardial involvement. Blunt injuries are more common and have a higher mortality (Table 69.4).

TABLE 69.4 Mortality from liver trauma. Type of injury Mortality Blunt abdominal trauma Overall mortality 10–30% Severe and high-velocity injuries Up to 60% Injury to main hepatic veins or 50–100% retrohepatic inferior vena cava Penetrating injuries 12–20% 20–40% Penetrating injuries with associated duodenal, pancreatic or chest involvement; multiple stab wounds

LIVER TUMOURS

LIVER TUMOURS

Liver resection continues to evolve, and the safety has been established with a mortality of 1–2% and a 5-year survival following resection of colorectal metastases of 50%. Early surgical approaches involved a formal left or right hepatectomy and the presence of bilobar disease or more than three or four metastases were considered inoperable. Advances in surgery and anaesthesia, including combinations of staged procedures, portal vein embolisation (PVE), ablation and local resections, increased the number of potentially curative procedures. Concurrent progress in oncology has increased the ability of chemotherapy to ‘downstage’ disease su ffi ciently to operable lesions that would have been formerly considered inoperable.

Learning objectives

Learning objectives

To understand: The anatomy of the liver • The signs of acute and chronic liver disease • The investigation of liver disease • The management of liver trauma • The management of liver infections •

Ligaments and peritoneal reflections

Ligaments and peritoneal reflections

The liver is covered by visceral peritoneum (serosa), with a layer of connective tissue, the Glisson capsule, underneath. At the porta hepatis, the capsule envelops and travels along the portal tracts (triads) into the liver, carrying branches of the hepatic artery , portal vein and bile ducts. The liver is fixed in the right upper quadrant by the hepatic veins and ligaments formed from the peritoneal reflections. Division of the left triangular ligament on the superior surface of the left lobe mobilises the liver from the diaphragm, exposing the left lateral wall of the inferior vena cava (IVC). The right triangu - lar ligament similarly fixes the right lobe to the undersurface of the right hemidiaphragm, and division mobilises the liver su ffi ciently to allow it to be rotated to the left. Another major supporting structure is the falciform ligament (the remnant of the umbilical vein), which runs cephalad fr om the umbilicus, enters the liver at the interlobar fissure and passes anteriorly on the surface of the liver, attaching it to the anterior abdominal wall. Dividing the cephalad leaves of the falciform ligament exposes the suprahepatic IVC within a thin fibrous sheath. The final peritoneal reflection is the lesser omentum between the stomach and the liver, which contains the hilar structures in its right free edge.

Liver transplantation

Liver transplantation

Liver transplantation is the only therapy that treats portal hypertension and the underlying liver disease and may ulti mately be required in patents with variceal bleeding. Previous surgical shunts increase the complexity and morbidity of orthotopic liver transplantation and TIPSS should be the preferred management (see Chapter 89). Mitsuo Sugiura , 1925–1988, surgeon, University of Juntendo, Tokyo, Japan. (d) Summary box 69.10 - Management of bleeding oesophageal varices /uni25CF). /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

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(c) or splenorenal (d) anastomoses. Blood transfusion Correct coagulopathy Oesophageal balloon tamponade (Sengstaken–Blakemore or Minnesota tube) Drug therapy (terlipressin) Endoscopic sclerotherapy or banding Assess portal vein patency (Doppler ultrasonography or CT) TIPSS Surgery Portosystemic shunts Splenectomy and gastric devascularisation Sugiura procedure

Liver-first approach

Liver-first approach

The traditional surgical strategy for resectable synchronous colorectal liver metastases (CRLMs) is colonic resection followed by chemotherapy and a delayed liver resection. This may allow progression of the liver disease and render the CRLM unresectable, which is a particular concern in patients who develop postoperative complications following their colon resection that delay or prevent chemotherapy . The liver-first approach or reverse strategy is a downstaging regimen consist - ing of systemic chemotherapy , chemoradiotherapy and/or biological agents, followed by resection of the CRLM prior to removal of the colonic primary . T he approach is valuable in the subset of patients in whom failure to respond to systemic treatment potentially renders CRLMs non-resectable.

Long-term problems following liver trauma and thei

Long-term problems following liver trauma and their management

Late complications are rare, but biliary strictures occur many years after liver trauma and treatment depends on the extent and site of stricturing. A segmental or lobar stricture with atrophy of the corresponding area of liver parenchyma and compensatory contralateral hypertrophy is treated expectantly . A dominant extrahepatic bile duct stricture associated with obstructive jaundice should be treated endoscopically but may require surgical correction with a Roux-en-Y hepatodochoje - - junostomy .

Surgery Becomes Uneventful unstable • Manage complications Remains • Haemorrhage stable • Reactionary and secondary haemorrhage • Biliary • Vascular • Sepsis • Other associated injuries

Portal hypertension is most commonly due to liver cirrhosis, although it also occurs with extrahepatic portal vein occlusion, intrahepatic veno-occlusive disease and occlusion of the main hepatic veins (Budd–Chiari syndrome). The condition is common in clinical practice and portal hypertension represents a significant clinical challenge, with patients who have often been ill for long periods repeatedly presenting as emergencies. Many symptoms are intractable, surgery is technically di ffi cult and procedures and timing must be chosen with extreme care. Portal hypertension per se produces no symptoms and is generally diagnosed following presentation with decompen sated chronic liver disease causing encephalopathy , ascites or variceal bleeding (Figure 69.12). Surgical involvement occurs in four situations: 1 ascites; 2 oesophageal varices; 3 portosystemic shunting for problems not managed by other methods; 4 left-sided portal hypertension and hypersplenism.

Malignant liver tumours

Malignant liver tumours

Neuroendocrine/carcinoid tumours Carcinoids are the most common NETs a ff ecting men and women equally . The overall incidence is steadily increasing, estimated at 1.5–1.9 clinical cases per 100 /uni00A0 000 population, although in autopsy series this reaches 650/100 /uni00A0 000 popu lation. Primary carcinoid tumours in the liver are rare and hepatic lesions are almost invariably metastases from small bowel or colon. Approximately 20% of small intestine carci noids dev elop metastases and 30% of these patients develop carcinoid syndrome. If the primary has been resected, liver metastases can be observed unless carcinoid syndrome develops. Hepatic carci noid disease tha t is resectable should be treated but this must be pr eceded by a thin-slice CT scan and laparoscopy with intraoperative ultrasonography if doubt remains to exclude small-volume disease. Carcinoid syndrome is a di ffi cult clinical prob lem, especially when pharmacological approaches are ine ff ective; in these patients, debulking should be considered. Some patients will develop a small number of lesions of similar sizes that can often be resected with curative intent. A significant proportion will develop recurrence(s), but this may take 18–36 months and further surgery is often possible. When lesions are numerous and of di ff erent generations (sizes) there are no surgical options and ablative, transarterial embolisation or targeted treatment with somatostatin analogues should be considered. Hepatocellular carcinoma HCC is a malignant tumour arising from hepatocytes and is the most common primary liver cancer. There is a steadily rising global burden. In 2016 there were 1 million incident the fifth most common cause of cancer in men and the seventh - in women, representing a third of all cancer-related deaths. HCC is the leading cause of death in patients with cir rhosis, a ff ecting three times more men than women. There is wide variation in geographical incidence. More than 80% of cases occur in Asia and sub-Saharan Africa, with an incidence of 99/100 /uni00A0 000 compared with 5/100 /uni00A0 000 in Europe. Geographical variation reflects the incidence of aetiological factor s. Chronic hepatitis B virus (HBV) infection accounts for >50% of cases worldwide and HBV vaccination programmes reduce the incidence in high-risk areas. Hepatitis C virus (HCV) increases the risk of HCC 17-fold by promot - ing end-stage liver disease. Aflatoxin contamination of rice in some parts of the world is probably responsible for seasonal variations. Lifetime alcohol exposure remains an intractable risk factor and correlates with the incidence of HCC. Obesity and diabetes mellitus are additional independent risk factors. Cancer-related causes are fatal in 60% of patients and 40% die of underlying parenchymal disease. HCC is typically diag - nosed at a late stage and pr ognosis even in developed countries is limited with median survivals following diagnosis of 6–20 months and overall survival of less than 50% at 2 years and 10% at 5 years, with worse r esults in developing countries. Staging of hepatocellular carcinoma Clinical staging systems for HCC are designed to guide management. The Barcelona Clinic Liver Group (BCLC) staging system, initially designed to define both prognosis and optimal treatment for patients with HCC, is the most commonly used (Figure 69.22). As patients with HCC usually have underlying liver disease that has a marked impact on - prognosis, the BCLC system was designed to reflect underlying liver function and performance status together with

tumour characteristics. Underlying liver function is assessed using the - CTP system. Treatment of hepatocellular carcinoma Surgical resection for hepatocellular carcinoma - Only 20–40% of patients with HCC are candidates for surgery , but with surveillance programmes in at-risk patients, improved imaging and advances in perioperative management resection is increasingly possible. Selecting suitable patients remains controversial and although tumour size, vascular invasion and multifocal disease are poor prognostic indicators they are not absolute contraindications. Multinodular lesions may repre - sent multiple discrete lesions occurring independently against a background of procarcinogenic parenchymal damage or aggressive tumour biology with intrahepatic metastases. Oncological contraindica tions include extrahepatic metastasis, multiple/bilobar tumours, main bile duct involvement and tumour thrombus in the main portal vein/vena cava. Preoperative evaluation of patients with hepatocellular carcinoma Achieving good outcomes for patients undergoing surgical resection requires accurate assessment of tumour stage, comorbidities and liver function. This is particularly important when planning larger resections, where the function of the FLR becomes critical. Postoperative morbidity and mortality increase with higher CTP scores, and major liver resection is usually only possible in patients with CTP-A disease. Minor liver resection may be considered in those with CTP-B disease but remains a high-risk procedure, and CTP-C patients are not candidates for liver resection. If inadequate FLR is the only contraindication preoperative radiological PVE should be performed. Preoperative imaging for hepatocellular carcinoma Imaging is a critical part of the preoperative assessment of HCC and accurate tumour staging and anatomical assessment is essential to determine technical and oncological resectabil ity and exclude metastatic disease. Triple-phase CT chest/ abdomen/pelvis and MRI of the liver is the standard of care, although MRI and CT have limited sensitivity and specificity for lesions <1 /uni00A0 cm (improved with liver-specific contrast agents). FDG-PET does not appear to confer any benefit over standard imaging. Surgical principles for hepatocellular carcinoma Surgical resection is a compromise requiring resection of the tumour while preserving su ffi cient functional parenchyma. HCC spreads within the liver by direct invasion of portal and hepatic venous systems. Anatomical resections that include removal of the entire venous drainage of a tumour, including occult micrometastases, is the optimal approach. There are clear long-term survival benefits for anatomical versus non- anatomical resections, with anatomical resection now consid - ered the standard of care when underlying liver function allows. Improvements in patient selection and surgical technique have reduced the 30-day mortality to <5%. Disease recurrence after resection - Intrahepatic recurrence occurs in 80% of cases within 5 /uni00A0 years and neoadjuvant or adjuvant options do not reduce the risk. Intrahepatic recurrence is thought to result from missed micrometastases or the development of new lesions and the most e ff ective approach to reducing intrahepatic recurrence is liver transplantation.

Stage 0 PST 0, Child–Pugh A Early stage (A) Very early stage (0) Single or 3 nodules <3cm Single

<2cm Carcinoma in situ PST 0 3 nodules <3cm Single Portal pressure and/or bilirubin Increased Associated diseases Normal No Liver transplantation Resection RF/PEI (DDLT/LDLT) Curative treatment (30–40%) Median OS >60 months; 5-year survival: 40–70% Figure 69.22 The Barcelona Clinic Liver Group staging system for the management of hepatocellular carcinoma (HCC). Patients with asymp tomatic early tumours (stage 0–A) are candidates for curative therapies (resection,

transplantation or local ablation). Asymptomatic patients with multinodular HCC (stage B) are suitable for chemoembolisation (TACE), whereas patients with advanced symptomatic tumours and/or an invasive tumoral pattern (stage C) are candidates for sorafenib. End-stage disease (stage D) includes patients with grim prognosis who should be treated by best supportive care. DDLT, deceased donor liver transplantation; LDLT PEI, percutaneous ethanol injection; PST, ECOG performance status; RF

, radiofrequency ablation; SD, standard deviation; TACE, transcatheter arterial chemoembolisation. (Reproduced with permission from Villanueva A. Medical therapies for hepatocellular carcinoma: a critical view of the evidence. Nat Rev Gastroenterol Hepatol 2013; 10 : 34–42.) Stage A–C Stage D PST 0–2, Child–Pugh A–B PST >2, Child–Pugh C Intermediate stage (B) Advanced stage (C) Terminal stage (D) Multinodular Portal invasion PST 0 N1, M1, PST 1–2 Yes TACE Sorafenib Best

supportive care Target: 10% Target: 40% Target: 20% OS: <3 months OS: 11 months OS: 20 months (SD 6–14) (SD 14–45)

, living donor liver transplantation; OS, overall survival;

Liver transplantation that definitively treats the tumour and underlying cirrhosis represents an attractive option, but organ shortages mandate careful selection of patients and early experience with transplantation was disappointing. Trans plantation for HCC, first described by Mazzaferro in 1996 for patients with tumours ≤ 5 /uni00A0 cm or up to three nodules ≤ 3 /uni00A0 cm, achieved 4-year overall survivals of 75% and recurrence-free surviv als of 83%. These inclusion criteria were adopted as the Milan criteria, with angioinvasion and extrahepatic involve ment as additional exclusion criteria, and are now universally accepted. Liver transplantation criteria, however, continue to evolv e and ‘expanded’ criteria remain debated. Locoregional therapies such as ablation may downstage HCC from beyond to within the Milan criteria and following a period of obser vation these patients may be considered as candidates for transplantation.

Management of variceal bleeding

Management of variceal bleeding

Resuscitation Varices are ubiquitous in patients with portal hypertension irrespective of the aetiology and usually present with an acute, and significant mortality . The lower oesophagus is the most common site and the diagnosis should be suspected in a patient known to have cirrhosis, but confirmation of the source is required f ollowing initial resuscitation. Variceal haemorrhage is a medical emergency and failure to control variceal bleeding with current medical management occurs in 10–20% of cases. Patients with massive haemorrhage should be admitted to an intensive therapy unit, venous access obtained through two - Summary box 69.9 Causes of portal hypertension /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Upper GI Overt blood bleeding from varices If severe and associated with variceal Hypersplenism Development of Portal varices hypertension CT/endoscopy Refractory ascites transplant candidate Figure 69.12 Management of complications of portal hypertension. CT, computed tomography; GI, gastrointestinal; TIPSS, transjugular intrahepatic portosystemic stent shunt. Pre-sinusoidal Extrahepatic: portal vein thrombosis, splenic vein thrombosis (pancreatitis, pancreatic tumour), myelo /f_i brosis, arterioportal shunt, tropical splenomegaly Intrahepatic: schistosomiasis, congenital hepatic /f_i brosis and portal in /f_i ltration (sarcoidosis), drugs and toxins, veno-occlusive disease Sinusoidal Cirrhosis Post-sinusoidal Hepatic vein occlusion (Budd–Chiari syndrome), veno- occlusive disease, congestive cardiac failure Endoscopic Anaemia surveillance Endoscopic treatment loss Tamponade +/– endoscopic TIPSS treatment Massive bleeding Shunt or Recurrent devascularisation Liver transplant bleeding may need splenectomy (portal vein thrombosis may result) Treat only if signs Endoscopic of bleeding surveillance Peritoneovenous shunt or TIPSS if

large-bore peripheral cannulae and resuscitation commenced, ideally with blood. Liver function tests will reveal underlying liver disease and a coagulation profile will identify any coagu lopathy . Hypervolaemia may increase portal pressure and exac erbate bleeding. Ten milligrams of vitamin K are administered intravenously but a coagulopathy requires FFP and activation of a major transfusion protocol. Thrombocytopenia secondary to hypersplenism is trea ted if the platelet count is <50 /uni00A0×/uni00A0 10 Treatment protocols include the use of splanchnic vasocon strictors, such as terlipressin, octreotide and somatostatin, and prophylactic antibiotics. When bleeding continues treatment options are sclerotherapy , banding, balloon tamponade and TIPSS. The use of oesophageal balloons should be avoided, w hich is usually possible when experienced endoscopists are available. As soon as the patient is haemodynamically stable the diagnosis should be confirmed endoscopically as 30% will have a non-variceal source of bleeding. Variceal bleeding is often associated with hepatic encephalopathy and endotra cheal intubation may be required prior to

endoscopy to protect the airway and prevent aspiration (Figure 69.13). Robert William Sengstaken Sr , 1923–1978, neurosurgeon, Garden City , New Y ork, NY , USA. Arthur Blakemore , 1897–1970, surgeon, the Columbia College of Physicians and Surgeons, New Y ork, NY , USA Balloon tamponade and self-expanding stents Balloon tamponade is e ff ective for massive or refractory vari - - ceal bleeding but is only recommended as a ‘bridge’ to defin - - itive treatment. If the rate of blood loss prohibits endoscopic evaluation, a Sengstaken–Blakemore tube (originally described in 1950) or a Minnesota tube (addition of an oesophageal aspi - ration port) can be inserted to provide temporary haemostasis 9 /L. (Figure 69.14). - Once inserted, the gastric balloon is inflated with 300 /uni00A0 mL of air and r etracted to the gastric fundus and the oesophago - gastric varices tamponaded by inflation of the oesophageal balloon to 60 /uni00A0 mmHg. T he two remaining channels allow gas - tric and oesophageal aspiration, and the position of the tube is confirmed radiologically . A strict protocol for the management of balloon tamponade is important to av oid complications par - ticularly oesophageal pressure necrosis. Recently , self-expanding cov ered metal oesophageal stents - have also been employed for the emergency treatment of oesophageal varices and r esults are equivalent to balloon tam - ponade unless the bleeding site is intragastric.

Urgent Suspected Endoscopy resuscitation variceal Airway bleeding protection Antibiotics Terlipressin Shunt surgery or devascularisation Consider transplantation Figure 69.13 The management of variceal bleeding. TIPSS, transjugular intrahepatic portosystemic stent shunt. Oesophageal Sclerotherapy Bleeding Eradication varices or banding control programme Fundal Cyanoacrylate Bleeding varices (thrombin) continues Balloon tamponade Repeat Bleeding sclerotherapy controlled Signi /f_i cant Continued TIPSS bleeding bleeding continues

Endoscopic treatment The two most commonly used endoscopic techniques are endoscopic band ligation to the base of the varix and injection of a sclerosant into or around the varix. Following resuscita tion, endoscopy is performed in a head-down position with good suction available. A double-channel endoscope with a bridge is essential to facilitate suction during injection and provide manoeuvrability of the needle, and power washers dramatically improv e visualisation. Some time should be spent assessing the bleeding, confirming it is variceal and obtaining a stable position. When the bleeding varix or varices are identified only the source should be treated. Sclerotherapy or banding both achieve e ff ective control with banding reducing rebleeding; a single treatment is usually su ffi cient. Transjugular intrahepatic portosystemic stent shunts The emergency management of variceal haemorrhage is extremely di ffi cult when pharmacological and endoscopic therapies have failed. Treatment of these patients now relies on TIPSS, a radiological procedure first described in 1969 but not widely available until the development of endovascular stents in 1985. TIPSS has replaced surgical portocaval shunt and is now accepted as the preferred method for treating refractory portal hypertension. A TIPSS is inserted under local anaes thetic, analgesia and sedation using fluoroscopic guidance and ultrasonography . Via the internal jugular vein, superior vena cava and hepatic vein, a guidewire is inserted through - the hepatic parenchyma into a branch of the portal vein. The tract is dilated; a metallic stent is then inserted and expanded, forming a portovenous channel (Figure 69.15). A satisfactory drop in portal venous pressure is usually associated with good control of the variceal haemorrhage. The main early compli - cation is perforation of the liver capsule, with potentially fatal intraperitoneal haemorrhage. TIPSS occlusion may produce further variceal haemorrhage and occurs more

commonly in patients with well-compensa ted liver disease and good synthetic function. The incidence of post-TIPSS encephalopathy is comparable to that following surgical shunts (40%) and due to portal blood avoiding hepatic detoxification, if severe, flow is reduced by inserting a smaller stent. The main contraindica - tion to TIPSS is portal vein occlusion, and long-term stenosis occurs in 50% of patients at 1 year. Surgical shunts The increasing availability of liver transplantation and TIPSS has greatly reduced the indications for surgical portosystemic shunts, which, because of their high morbidity and mortality , are now rarely considered for variceal haemorrhage. The - current indication is the failure of medical management in non-cirrhotic patients with extrahepatic portal vein occlusion. Surgical shunts e ff ectively prevent rebleeding from oesophageal

Oesophageal aspiration channel Oesophageal balloon: 40 mmHg Gastric balloon: at least 300 mL of air Gastric aspiration channel Figure 69.14 Oesophageal and gastric balloon tamponade with a Sengstaken–Blakemore or Minnesota tube. The tube must be care fully managed. Figure 69.15 An angiogram following insertion of a transjugular intra- hepatic portosystemic stent shunt (TIPSS)

(open arrow). Contrast in

the portal vein /f_l ows through the metallic stent and outlines the right hepatic vein. Pressure measurements are taken from within the portal vein before and after insertion. Solid arrows indicate coils placed at the site of previous embolisation.

(c) or gastric varices by reducing portal pressure and are divided into selective, splenorenal and non-selective portocaval. Selec tive shunts attempt to preserve hepatoportal blood flow while decompressing the left side of the portal circulation, which is responsible for oesophageal and gastric varices (Figure 69.16 Selective shunts have a low er incidence of encephalopathy but there is no evidence that prophylactic shunting is beneficial. Recurrent or refractory variceal bleeding Sugiura procedure The Sugiura procedure for oesophageal varices combines splenectomy with oesophagogastric devascularisation, permanently interrupting the intraoesophageal portacaval shunt while preserving perioesophageal varices. The surgery is performed on the stomach wall and all venous tributaries are divided as for highly selective vagotomy except on both the lesser and greater curves. The upper half of the stomach and 8–10 /uni00A0 cm of oesophagus are cleared (less than originally described but avoiding entering the chest). After devasculari sation with careful preservation of the collateral channels and the vagus, a large oesophageal stapler is introduced into the lower oesophagus, which is transected just above the cardia.

Figure 69.16 Surgical shunts for portal hypertension involve shunting portal blood into the systemic veins. This commonly involves a side-to side portocaval anastomosis (a) or end-to-side portocaval (b) , mesocaval ‘H graft’

Methods of parenchymal transection

Methods of parenchymal transection

An array of techniques and technologies have been developed to aid parenchymal dissection by facilitating identification of vascular and biliary structures to enable accurate diathermy , ligation or clipping. They also allow safe resection with adequate clearance of centrally placed tumours near the confluence - of the hepatic veins and the IVC or the inflow sheaths. Safe transection with minimal blood loss and an adequate tumour clearance can be achieved using a crushing clamp, cavitating ultrasonic suction and aspiration (CUSA), harmonic scalpel or radiofrequency ablation (RFA) and is a matter of personal preference with no evidence that any method is superior (Figure 69.20). Hepatic veins and the Glissonian sheath are now routinely stapled with an endoscopic vascular stapler. The parenchyma is divided after diathermy of the liver capsule along the plane of demarcation 5 /uni00A0 mm into the devas cularised liver. As the parenchyma is divided, v essels and bile ducts are diathermised, clipped or ligated depending on their size. The hepatic veins can be divided outside the liver at the time of mobilisa tion or parenchymal dissection continued until they are encountered, when they are ligated or stapled then divided.

Figure 69.20 Hepatectomy post resection. Cut surface of the residual liver following a right hepatectomy in which segments V–VIII have been removed. On the lower edge, the portal vein and bile duct can be seen.

Microscopic anatomy and structure

Microscopic anatomy and structure

The liver comprises approximately 100 /uni00A0 000 hexagonal func tional units known as lobules with a central vein surrounded Summary box 69.1 Liver anatomy /uni25CF /uni25CF /uni25CF /uni25CF Karl Wilhelm von Kup ff er , 1829–1902, Professor of Anatomy at Kiel (1869), Königsberg (1875) and Munich (1880), Germany , described these ‘stellate cells’ in 1880. by six hepatic portal veins and six hepatic arteries. These vessels are connected by capillary-like tubes called sinusoids, which extend to meet the central vein. Lobules are separated by hepatic sinusoids, which are large-diameter capillaries lined by endothelial cells between rows of plates or cords of hepato - cytes. Each sinusoid contains Kup ff er cells, a type of macro - phage that captures and breaks down e ff ete red blood cells, and hepatocytes, which are cuboidal epithelial cells making up the majority of cells in the liver. Hepatocytes perform most liver functions, including metabolism, storage, digestion and bile pr oduction. Tiny bile canaliculi run parallel to the sinusoids on the contralateral side to the hepatocytes and drain bile in the opposite direction to the blood flow via the bile duct tributaries within the portal tracts. -

There are two anatomical lobes with a separate blood supply, bile duct and venous drainage There is a dual blood supply; 80% portal vein and 20% hepatic artery The liver regenerates to 90–100% of its previous volume following resection Resection is based on anatomical lines to preserve maximal functioning liver and blood supply Portal vein Left hepatic artery Hepatic artery proper Left gastric artery Splenic artery Abdominal aorta Superior mesenteric artery

Mobilisation of the liver

Mobilisation of the liver

Incision A roof top incision is performed 2–3 /uni00A0 cm below the costal margin (Figure 69.19) with a vertical extension (Mercedes-Benz) if required. Fixed retraction under the ribs provides adequate access and thoracoabdominal incisions are no longer required. If doubt exists about operability a small right subcostal inci sion is used initially , and a thorough examination performed, including the caudate lobe. Intraoperative ultrasonography (IUS) is the standard of care for hepatobiliary surgery and is used with bimanual palpation to assess the e xtent of the tumour(s). IUS detects only an additional 10% compared with palpation alone. The hepatic pedicle Hilar dissection Having determined that tumour does not directly involve the hilar structures a standard cholecystectomy is performed. The CBD is identified in the free edge of the lesser omen tum, facilitated by following the cystic duct to its junction, dissected free and slung. The tissue in the right free border The Mercedes-Benz sign takes its name from the insignia displayed on the bonnet of a Mercedes-Benz car. of the hepatoduodenal ligament is dissected and removed by ligation and division to avoid lymphatic leaks and the portal vein identified and slung. Developing the plane anterior to the vein allows the bile duct and artery to be mobilised forwards and the bifurcation of the vein to be identified (the branch to the side to be retained must be clearly identified). At this point anterior tissue (the hilar plate) should be freed from the base of the liver, lowering the structures that bifurcate. The artery - and duct are separated at the bifurcation and slung just below it. The vascular anatomy is then confirmed, and the possibility of a replaced right hepatic artery arising from the superior mesenteric artery and lying posterior to the bile duct (25% of people) and an accessory left hepatic artery from the left gastric artery in the lesser omentum (25% of people) considered. Hilar arterial and biliary anatomy in the hepatoduodenal ligament and at the hilum is so variable that careful dissection is required even if the pattern appears to be one of the recognised variants. A standard approach is important in the event of unexpected intraoperative findings. The approach to the hilum - allows di ff erent conditions and pathologies to be approached with confidence, including formal resections (metastases and primary liver tumours), hilar cholangiocarcinoma, bile duct injuries and penetrating trauma.

Figure 69.19 Access for liver surgery. Rooftop incision with optional vertical extension.

Non-alcoholic steatohepatitis, non - alcoholic fat

Non-alcoholic steatohepatitis, non - alcoholic fatty liver disease and chemotherapy-associated hepatitis

Non-alcoholic steatohepatitis (NASH) is the inflammatory subtype of non-alcoholic fatty liver disease (NAFLD) and is associated with disease progression, the development of cirrhosis and frequently the need for liver transplantation. NAFLD is now recognised as the most prevalent chronic liver disease worldwide and this is expected to increase 60% by 2030. Presently the prevalence of NAFLD is 25% and NASH 1.5–6%, with an estimated 20% of patients with NASH devel oping cirrhosis. Some drugs may induce hepatotoxic lesions, such as ste atosis or steatohepatitis found in NAFLD. Among these drugs there are some antitumoral molecules, such as methotrexate, 5-fluorouracil, irinotecan, tamoxifen and l-asparaginase. The hepatotoxic phenotype developed from treatment with such drugs is known as chemotherapy-associated, chemotherapy- induced acute steatohepatitis or chemotherapy-associated hepatitis (CASH). The parenchymal consequences of CASH are important surgically and must be considered when predict ing future liver remnant (FLR) function.

Parasitic diseases of the liver

Parasitic diseases of the liver

- The liver is frequently a ff ected by parasitic infections, which, owing to the worldwide prevalence of these organisms, are responsible for considerable morbidity and mortality (see Chapter 6). Hydatid disease Human echinococcosis (hydatidosis, hydatid disease) is a para - sitic disease caused by the larval stages of cestodes (tapeworms) - of the genus Echinococcus . Medical treatment and diagnosis are discussed in Chapter 6 . Surgical intervention is occasionally required when medical management fails, and options range from liver resection or local excision of the cysts to deroofing with evacuation of the contents. Contamination of the perito - neal cavity at the time of surgery with active hydatid daughters should be avoided by continuing drug therapy with albenda - zole and adding preoperative praziquantel. This should be combined with packing of the peritoneal cavity with 20% hypertonic saline-soaked packs and instilling 20% hypertonic saline into the cyst before it is opened. A biliary communi - cation should be actively sought and sutur ed. Infection and Summary box 69.13 Infections of the liver /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Pyogenic liver abscesses 1/5000 admissions Worldwide billions of people have parasitic infections Parasitic infections cause live abscess and biliary tract damage Biliary tract involvement predisposes to cholangiocarcinoma Parasitic infections mimic pyogenic abscesses Obstructive jaundice from calci /f_i ed /f_l ukes or involvement of the biliary tract

greater omentum (an omentoplasty). Calcified cysts may be dead; however, if doubt exists as to whether a suspected cyst is active, it can be followed on ultrasonography as active cysts gradually enlarge and become more superficial.

Primary biliary cirrhosis

Primary biliary cirrhosis

As with PSC, patients with primary biliary cirrhosis often pres - ent insidiously with malaise, lethargy and pruritus or abnormal liver function tests prior to becoming clinically jaundice. The condition is largely confined to females and the diagnosis is suggested by circulating anti-smooth muscle antibodies with/ - without liver biopsy . As the condition progresses liver function deteriorates, and portal h ypertension, ascites and variceal tation when a normal lifestyle is not possible.

Primary sclerosing cholangitis

Primary sclerosing cholangitis

PSC is a chronic cholestatic liver disease of unknown aetiology , although a genetic predisposition is likely owing to its asso ciation with ulcerative colitis. It produces di ff use, progressive inflammation and fibrosis with structuring of the intra- and extrahepatic biliary tree and mainly a ff ects young men in their thirties. The exact worldwide prevalence is unclear, but it appears to a ff ect 1.5/100 /uni00A0 000 men and 0.5/100 /uni00A0 000 women. - In patients with PSC and ulcerative colitis, the condition usually progresses even following colectomy . The diagnosis is principally based on the finding of irregular, narrow ed bile ducts at cholangiography involving both the intra- and extra - hepatic biliary tree (Figure 69.17), but if the radiological appearances are equivocal a liver biopsy is required. There is no specific treatment and patients usually progress inexora - bly with progressive cholestasis and fatal liver failure. Isolated areas of intrahepatic sclerosing cholangitis can occasionally be resected but di ff use disease usually requires liver transplanta - tion. There is a strong predisposition to cholangiocar cinoma and gallbladder cancer, which should be considered when a new or dominant stricture is demonstrated on cholangiogra - phy or when gallbladder ‘polyps’ are identified. T he di ffi culty in the clinical setting is distinguishing scleros - ing cholangitis from a malignant process, particularly multi-). focal cholangiocarcinoma. Imaging cannot reliably di ff erentiate between inflammatory and malignant strictures and rarely demonstrates a mass lesion even in patients with advanced c holangiocarcinoma. Diagnosis often requires biliary brush TM cytology or direct endoscopic inspection (SpyGlass). Serum cancer antigen (CA) 19-9 levels may be increased but the sensitivity of CA 19-9 in detecting cholangiocarcinoma in PSC is only 60%. Temporary relief of obstructive jaundice owing to a dominant bile duct stricture can be achieved by biliary stenting, although there is considerable risk of cholangitis. Patients with good liver function, no dominant strictures and negative biliary cytology are monitored for disease progression. Liver transplantation produces excellent results if performed before the development of malignancy .

Figure 69.17 Typical appearance of primary sclerosing cholangitis with a ‘beaded’ appearance of the intrahepatic ducts and diffuse widespread strictures. The intrahepatic ducts usually do not dilate owing to the pathological process involving the whole of the biliary tract.

Re-do surgery

Re-do surgery

Close follow-up identifies recurrent isolated liver metastases and if CT and PET exclude additional disease repeat resection is appropriate when possible. The operative approach must take into account the consequences of previous surgery and hypertrophy following a major resection. Left lobe resections may produce a more inferiorly based and medially shifted portal triad, making the origin of the right hepatic pedicle deeper and more medial than expected, and a right hepatectomy will often rotate the hilum more anteriorly . Non-colorectal, non-neuroendocrine metastases Although metastases from non-CRCs do not spread via the portal circulation and are rarely confined to the liver, with the low mortality associated with liver resection palliative or poten - tially curative surgery for metastases from renal, breast, gastric and lung metastases together with deposits from melanoma, sarcoma and a range of rarer tumours is reported. Management of metastatic gastrointestinal stromal cell tumours Gastrointestinal stromal tumours (GISTs) are non-epithelial tumours originating in interstitial Cajal cells of the autonomic nervous system, which metastasise in 20–25% of patients. Management has changed with the e ff ective chemotherapy The primary bowel tumour should be removed if possible and the liver assessed to identify potentially resectable disease. If metastases respond to postoperative imatinib, surveillance is recommended; however, when metastases escape imati nib control debulking has no role and surgical resection is performed only if extirpation of all disease is possible.

Recurrent or refractory abscesses

Recurrent or refractory abscesses

Recurrent abscesses usually occur when the initial lesion was large, abscesses were multiple or there is continued commu - nication with the biliary tract. It can be di ffi cult to confirm whether a liver abscess is recurrent or new , but it is important - as treatment di ff ers. Recurrent lesions which wer e aspirated and treated with antibiotics can be re-aspirated, but a drain is often required. Refractory lesions should have microbiology repeated and a drain inserted, and recurrences are rare if left until resolution is complete. If unsuccessful occasionally surgery is required, and laparoscopy which allows a full examination of the peritoneal cavity (especially valuable when the source has not been identified) has replaced laparotomy .

Resection options

Resection options

Segmental resections Hepatic resection traditionally involved the formal removal of the right (segments V–VIII) or left (segments II–IV with/ without I) hemiliver to ensure the largest possible clearance. Although anatomical resection remains the treatment of choice for patients with HCC, a parenchyma-sparing non- anatomical approach involving multiple segmentectomies and/ or metastectomies is now the standard of care for colorectal liver metastasis (Figure 69.21). Staged procedures Extensive resection of the liver in two stages was first described in 1965. The aim is to ‘clear’ one lobe of the liver of all known disease followed 4–6 weeks later by a formal major resection to clear all residual disease. Staged resections are usually only possible if the left side can be cleared first by local resections, something that is usually not possible on the right because of the need to remove as little normal parenchyma as possible to avoid stimulating too much regeneration. The stimulus for regeneration following resection of too much liver may accel erate growth of the tumour, which despite chemotherapy may become inoperable. ‘ ALPPS’ stands for Associating Liver Partition and Portal vein Ligation for Staged hepatectomy and was first described in 2011. It is the most recent modification of techniques developed to facilitate two-stage hepatectomies for resection of widespread or extensive liver tumours and employs the remarkable capacity of the liver to regenerate. ALPPS involves two stages. Initially the right portal vein is ligated and, depending on the distribu - tion of the tumour within the liver, transection is performed as for a formal hemihepatectomy or left lateral segmentectomy (in situ splitting). In contrast to a classical hepatectomy , the liver containing the tumour(s) is left in situ and remains vascularised by the right hepatic artery and the biliary and systemic venous drainage, represented by the right bile duct and hepatic veins, preserved. The second stage of the procedure is performed 1–2 weeks after the first stage following CT demonstration of adequate hypertrophy; the involved liver is resected after division of the right hepatic artery , bile duct and hepatic vein. Initially ALPPS was associated with significant morbidity and mortality but modifications of the technique, particularly a reduction in the amount of liver transected, improved results. Portal vein embolisation Preoperative PVE induces hypertrophy of one side of the liver prior to a planned resection of the other side. The procedure - -

(a) (b) Figure 69.21 (a, b) Segmental resection. Removal of a primary liver tumour by resection of liver segment VI in a patient with well- compensated liver cirrhosis.

was first described by Makuuchi in 1984 before an extended hepatectomy for bile duct carcinoma. Several techniques for PVE have been reported, including intraoperative ligation, transileocolic PVE and the percutaneous transhepatic ipsilat eral or contralateral PVE techniques. The underlying principle is to block the portal venous blood flow to the liver segments that require resection. This induces atrophy of the ipsilateral liver segments and compensator y hypertrophy of the contra lateral liver segments, resulting in an increase in the size of the proposed FLR. In addition to the di ff erent techniques, di ff erent embolisation materials are used, including polyvinyl alcohol

particles, coils, gelatin sponge, n -butyl cyanoacrylate and lipiodol, or fibrin glue. Indications for PVE depend on the ratio of the proposed FLR to total estimated liver volume (TELV) and the condition of the liver. There is no clear consensus about the volume required for adequate postresec tion liver function but an FLR/TELV ratio of at least 25% is recommended with a normal liver and 40% in the presence of cirrhosis. Patients who have received extensive chemotherapy with an FLR/TEL V ratio less than 30% should also receive PVE prior to resection. Laparoscopic liver resections The development of laparoscopic liver resection (LLR) follow ing its first performance in the USA in 1991 for a benign tumour on the edge of the liver has been one of the most impressive in the field of hepatobiliary surgery . Technical innov ation has made LLR a safe and e ff ective procedure with significantly improved postoperative recovery . The potential advantages of LLR mean that it is gradually replacing conventional open liver resection. Indications have expanded from local resec tions to include major liver resection, isolated resection of the caudate lobe, living donor liver resection and ALPPS. For some procedures such as laparoscopic local resection and left lateral segmentectomy LLR is the appr oach of choice and formal hepatectomies are now routinely performed laparoscopically in high-volume centres. Robotic surgery With the safe and e ff ective development of laparoscopic liver surgery , robotic surgery , which obviates some of the technical issues, was welcomed and the first robotic liver resection was performed in 2007 for a 2.4-cm HCC. Indications for robotic surgery will expand but are presently limited by cost, time constraints, the lengthy learning curve, the lack of haptic feedback and the availability of dedicated instruments for parenchymal transection (see Chapter 10).

Segmental anatomy

Segmental anatomy

The liver is divided into functional right and left ‘units’ along the line between the gallbladder fossa and the middle hepatic vein (Cantlie’s line). Understanding the internal anatomy of the liver facilitated safe liver surgery and Couinaud, a French anatomist, described the liver as being divided into eight segments (Figure 69.1). Each segment can be considered a functional unit supplied by a branch of the hepatic artery , portal vein and bile duct, and drained by a hepatic vein tributary; this concept facilitates ‘anatomical’ liver resection. Liver segments V–VIII to the right of Cantlie’s line are supplied by the right hepatic artery and the right branch of the portal vein and biliary drainage is via the right hepatic duct. To the left of Cantlie’s line segments, I–IV are supplied by the left hepatic artery and left portal vein and drain via the left hepatic duct. Resections of individual segments, the whole of the left or the right hemiliver or combinations are possible.

Right hepatic artery Cystic artery Gastroduodenal artery Figure 69.2 Anatomy of the liver hilum.

Surgical approaches to liver trauma

Surgical approaches to liver trauma

When a laparotomy is indicated, especially when CT scanning is not possible, a ‘rooftop’ incision (see Figure 69.19 midline extension to the xiphisternum and retraction of the costal margins gives excellent access to the liver and spleen. If a midline incision is made initially a transverse right lateral extension will improve access. Required operative techniques include resectional debridement, hepatotomy with direct suture ligation and perihepatic packing. Anatomical resection, hepatic artery ligation and bypass techniques are possible following transfer of patients to tertiary hepatobiliary centres. Major complications include recurrent haemorrhage, sepsis and bile leak. Packing or manual pressure intended to compress the parenchyma without causing caval compression is the initial aim (Figure 69.9); if additional intra-abdominal bleeding is found the source needs to be identifi ed. Care should be taken to av oid overzealous packing, which may produce pressure necrosis of the liver parenchyma or abdominal compartment syndrome. Packing is e ff ective for the majority of liver injuries if the liver is packed against the natural contour of the diaphragm. If control is not achieved James Hogarth Pringle , 1863–1941, surgeon, The Royal Infi rmary , Glasgow , UK. a Pringle manoeuvre should be performed (Figure 69.10). Large /uni00A0 abdominal packs should be used to ease their removal, and the abdomen closed to facilitate compression. Continued bleeding implies damage to the hepatic veins and/or the IVC but exploration of a liver laceration should only be attempted if control is not possible. If insu ffi cient facilities or assistance are available and packing controls the situation, the abdomen should be closed and the patient transferred to) with a tertiary centre.

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Surgical management of hepatobiliary Figure 69.10 The Pringle manoeuvre.

haematomas and di ff use capsular lacerations (Figure 69.8a Suturing is ine ff ective, and perihepatic packing is frequently the only option. Necrotic tissue should be removed but poorly perfused but viable liver left in situ . If packing is necessary , this should be removed after 48–72 hours; usually , no further inter vention is required. Antibiotic cover is advisable and full rever sal of any coagulopathy essential. If a major vascular injury (hepatic vein or vena cava, grade V or VI) is suspected then packing and referral to a specialist centre should be consid ered as venovenous bypass is often required. Following transfer, a further laparotomy is performed, the liver fully mobilised and, after a Pringle manoeuvre and IVC occlusion above the renal veins and at the level of the diaphragm using atrauma vascular clamps (with/without venovenous bypass), caval or hepatic vein damage is repaired. Warm ischaemia of the liver is tolerated for up to 45 minutes.

Surgical approaches to resection of liver tumours

Surgical approaches to resection of liver tumours

Parenchyma-preserving resections that achieve adequate oncological clearance have emerged from an understanding of oncological principles and the impact of chemotherapy on hepatic function. Such resections preserve functioning liver volume, improving postoperative recovery , reducing morbidity and facilitating re-do surgery for recurrent metastases. Limited extrahepatic disease is also no longer an absolute contraindi cation and pulmonary and adrenal metastases and contiguous portal vein lymph nodes are increasingly resected.

Synchronous colon and liver resection

Synchronous colon and liver resection

Synchronous resectable liver metastases are frequently identified at the time of diagnosis. Treatment options include sequential, delayed and simultaneous resection strategies. Resection of the colonic primary followed by chemotherapy , restaging and resection of the liver disease, if appropriate, is the standard approach. It is occasionally possible with low-volume, accessible liver disease to perform synchronous Santiago Ramón y Cajal , 1852–1934, neurophysiologist and director Instituto Nacional de Hygiene, Madrid, Spain. both procedures represent a major undertaking per se.).

The blood supply to the liver

The blood supply to the liver

The liver is composed of eight segments (Figure 69.1), each supplied by terminal branches of the portal vein (80% of the blood flow) and hepatic artery (20%) and drained by bile ducts and hepatic veins. The shape of the segments varies among individuals, but the configuration remains relatively constant. Anatomia hepatis The arterial blood supply is variable in origin and course but in most individuals is derived from the coeliac trunk, which usually divides into left gastric, common hepatic and splenic arteries. After supplying the gastroduodenal artery , the hepatic artery branches at a variable level to produce the right and left hepatic arteries, the larger right branch supplying the right lobe. The right lobe may be partly or completely supplied by a right hepatic artery arising directly from the superior mesen teric artery running to the liver on the posterior wall of the bile duct after passing behind the uncinate process and head of the pancreas. Similarly , the left lobe artery may be augmented or replaced by a branch of the left gastric artery running in the lesser omentum from the lesser curv e of the stomach.

VIII VII II IV I III V VI Figure 69.1 The functional division of the liver and of the liver segments according to Couinaud’s nomenclature. (b) /uni00A0 In the ex vivo position.

The hilum of the liver

The hilum of the liver

The porta hepatis is a pronounced transverse fissure on the visceral surface of the liver running between the cephalad end of the fissure for the ligamentum teres and the gallbladder fossa. The neurovascular structures and lymphatics running in the right free edge of the lesser omentum (the hepatoduodenal ligament) enter at this point and the right and left hepatic ducts emerge. There are numerous variations of the hilar structures which are important in the planning and performance of operations on the liver (Figure 69.2). In the most common arrangement, the bile duct runs in the free edge of the hepatoduodenal ligament with the hepatic artery medially and the portal vein posteriorly , each dividing into two branches at the hilum. The right and left hepatic ducts arise from the hepatic parenchyma and form the common hepatic duct. The cystic duct draining the gallbladder enters the ligament at a variable level, joining the common hepatic duct to form the common bile duct (CBD). The right hepatic A hilum is a depression or fissure where nerves, vessels or ducts enter a bodily organ. Sir James Cantlie , 1851–1926, Scottish-born physician who cofounded the Hong Kong College of Medicine for Chinese (now Hong Kong University School of Medicine). artery crosses the bile duct anteriorly or posteriorly before giv - ing rise to the cystic artery , and multiple branches predomi - nantly from the right hepatic artery supply the bile duct. The portal vein is formed by the confluence of the splenic and supe - rior mesenteric veins behind the neck of the pancreas, with the left branch having a longer (approximately 2 /uni00A0 cm) extrahe patic course. The portal vein often has two large branches to the right lobe, w hich are usually outside the liver for a short length, - before giving a left portal vein branch that runs behind the left hepatic duct.

II VIII VII IV I III V VI (a) As seen in the patient.

The venous drainage

The venous drainage

The IVC occupies a groove on the posterior surface of the liver that drains into it via three large veins immediately below the diaphragm. The suprahepatic IVC immediately traverses the diaphragm to enter the right atrium, but below the liver there is a short clear segment above the insertion of the renal veins. A variable number of short inferior hepatic veins pass directly from the liver to the anterior wall of the IVC. The right hepatic vein can be exposed fully outside the liver parenchyma, but the middle and left veins usually terminate in a short common trunk before entering the IVC. The right adrenal gland is adjacent to the retrohepatic IVC and drains into it, usually by a single vein.