Chronic liver disease

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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 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 /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.

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