Acute liver failure

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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)

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