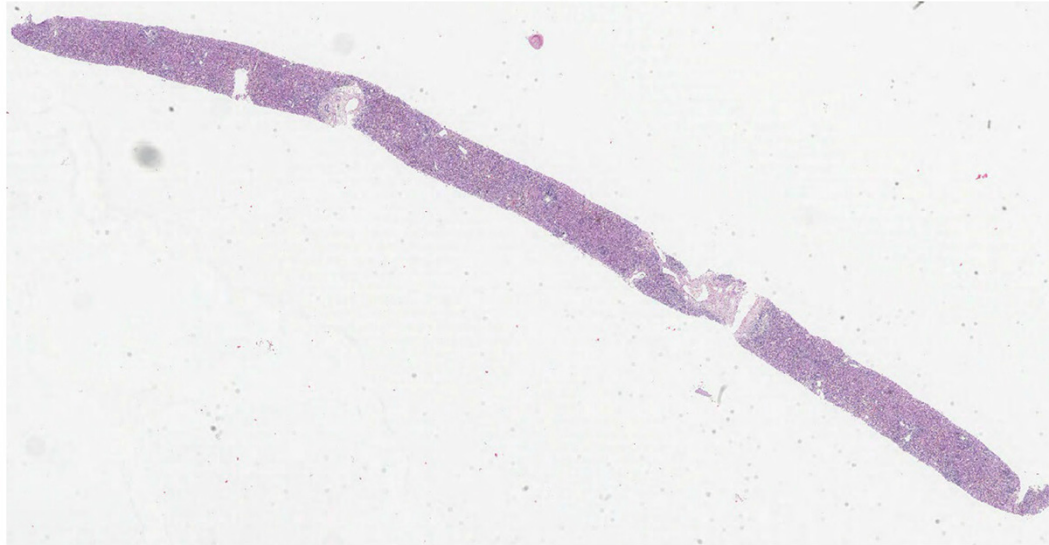
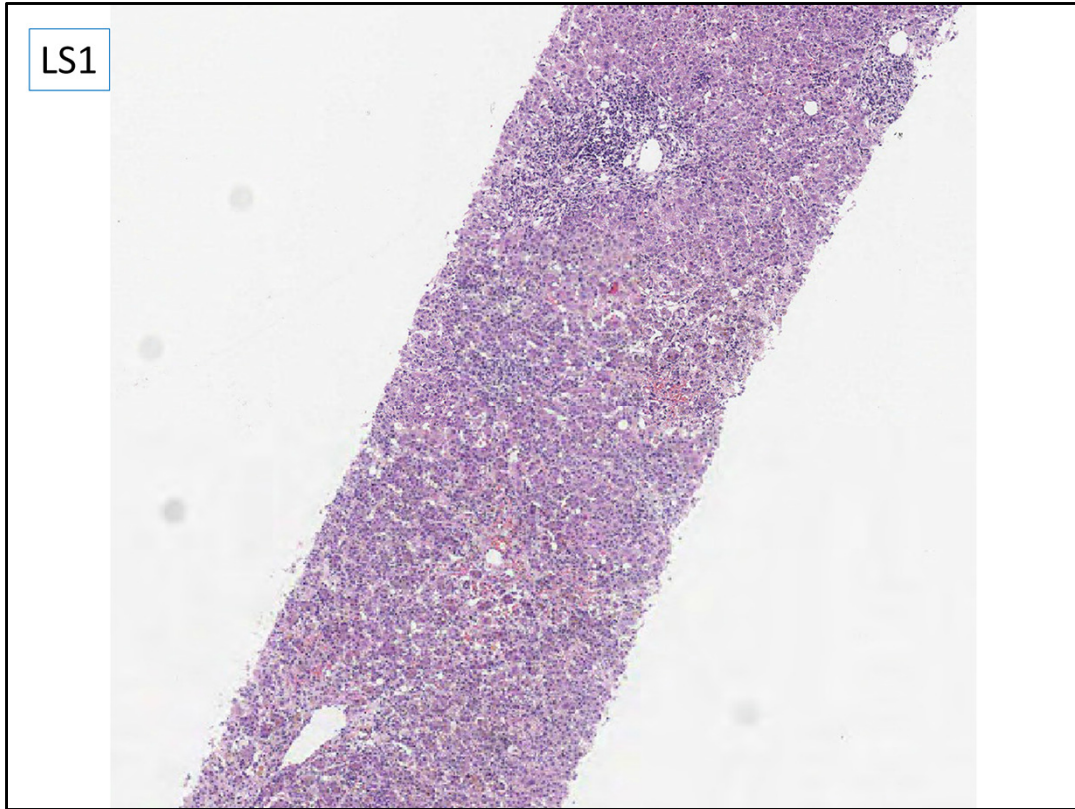


Case LS1 35F

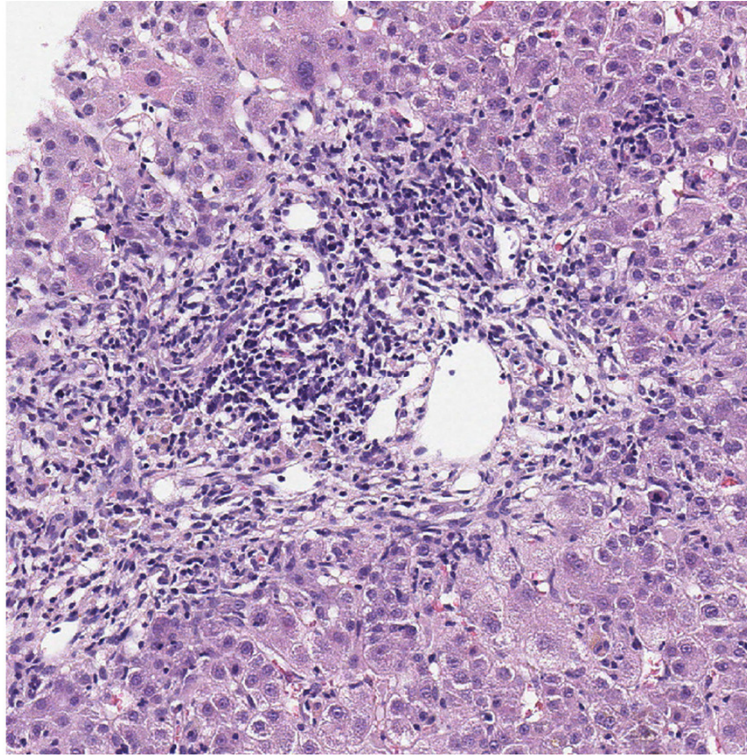
New onset severely deranged LFTs, no previous history. Admitted with severe jaundice and very deranged LFTs, ALT >1000
also retic, van Gieson, PASD





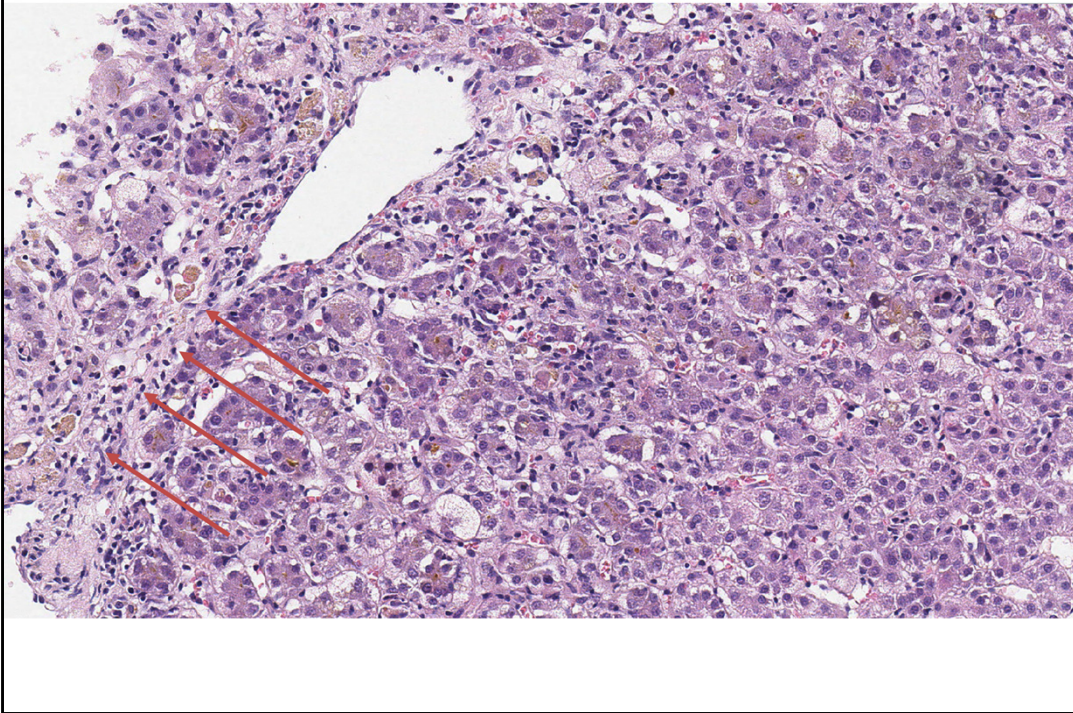
Low magnification – good core, vascular relationships preserved, lobular disarray

LS1

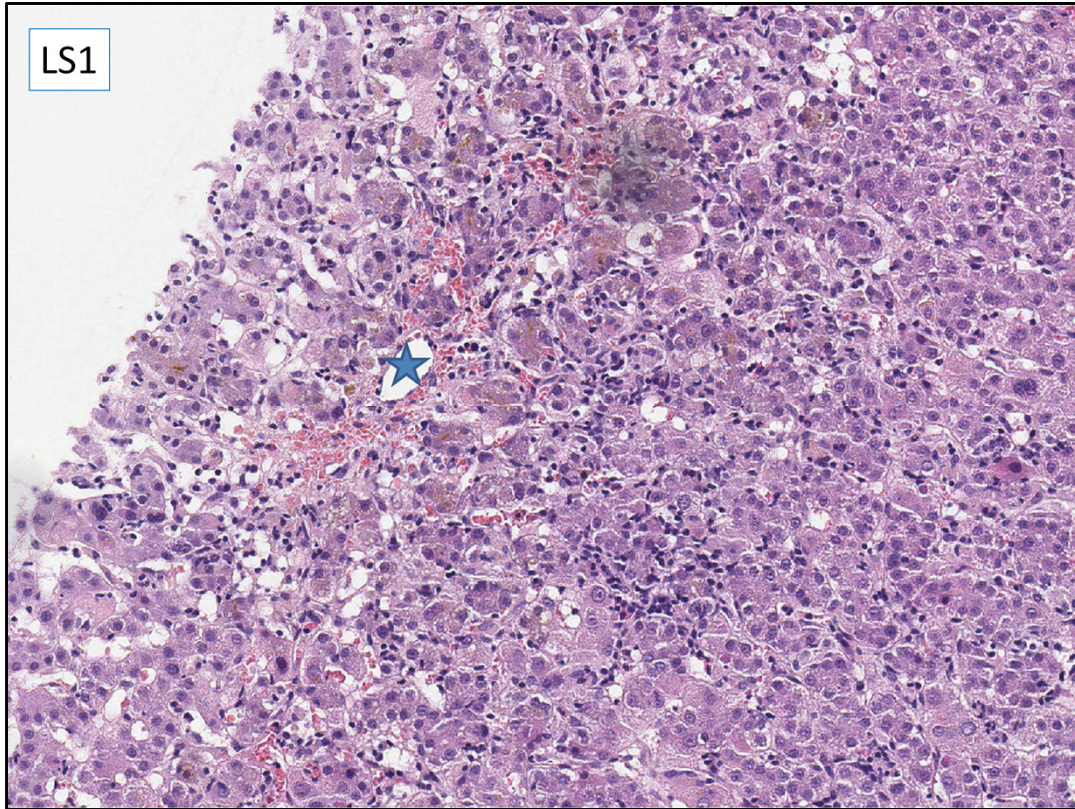


Portal areas – some crease in mononuclear cells, mild portal expansion

LS1

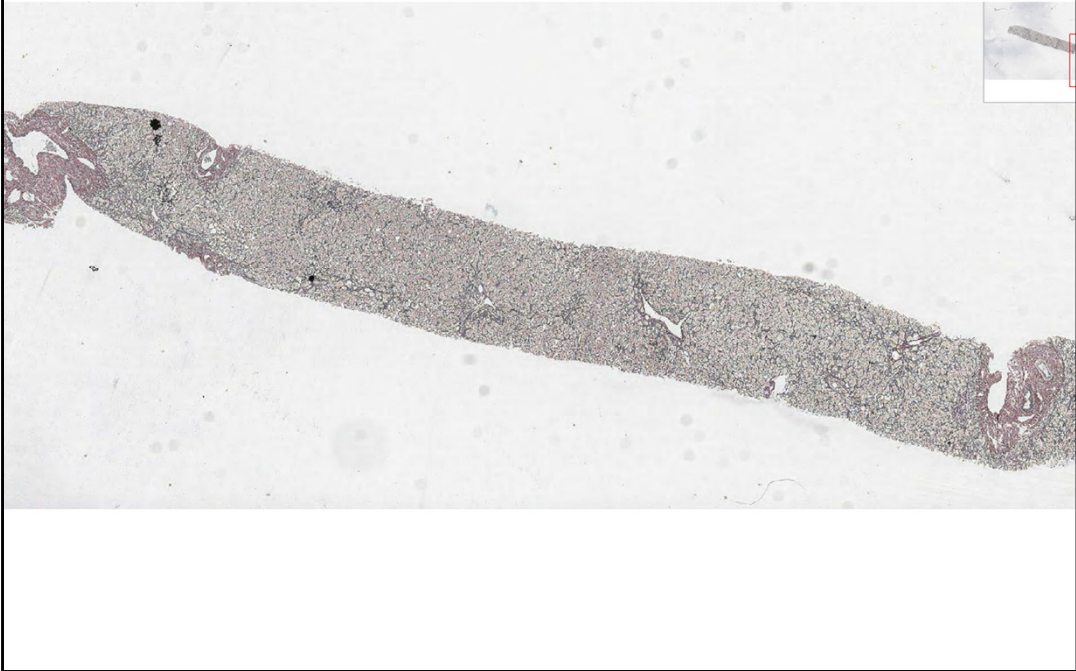


Perivenular area – marked lobular disarray, with variation in hepatocyte size, canalicular cholestasis, with re-arrangement of hepatocytes into rosettes. Sinusoidal inflammatory cells, some areas with loss 'drop-out of hepatocytes = bridging necrosis linking hepatic vein to portal tract (arrows)

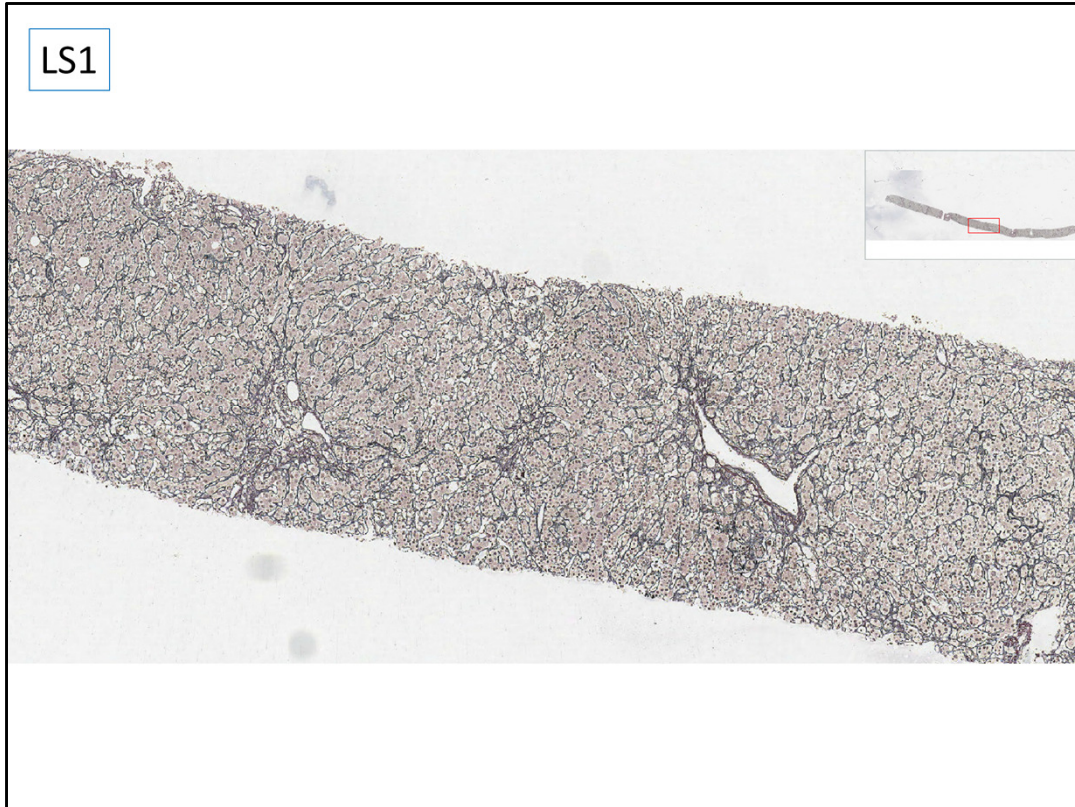


Also some congestion in perivenular areas of hepatocyte loss (star = hepatic vein), but when associated with lobular hepatitis this is not a sign of venous outflow obstruction.

LS1

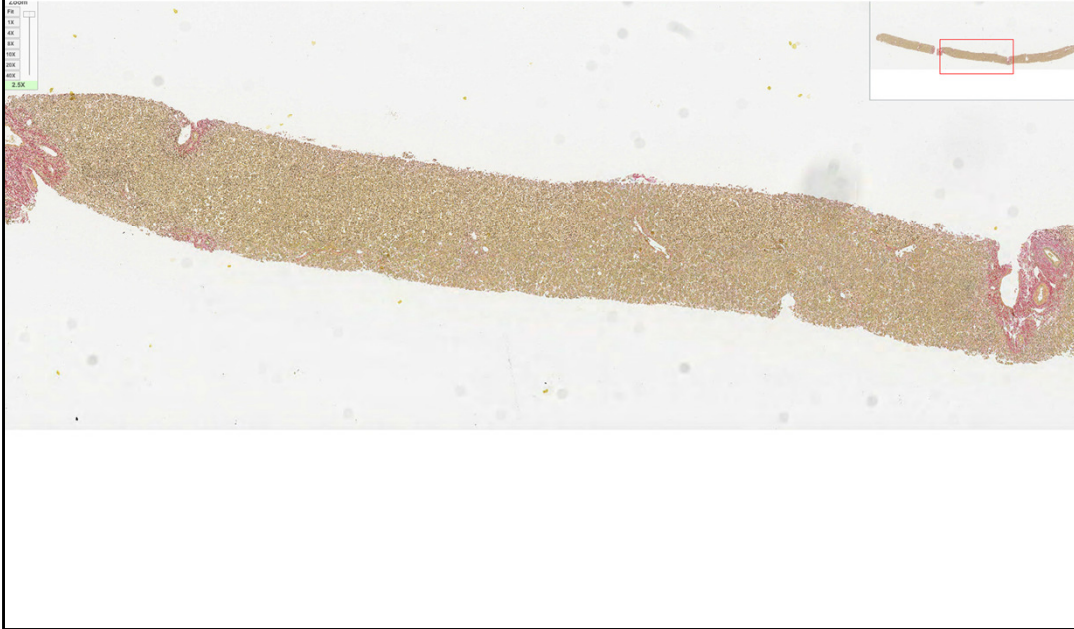


Reticulin stain. Low magnification



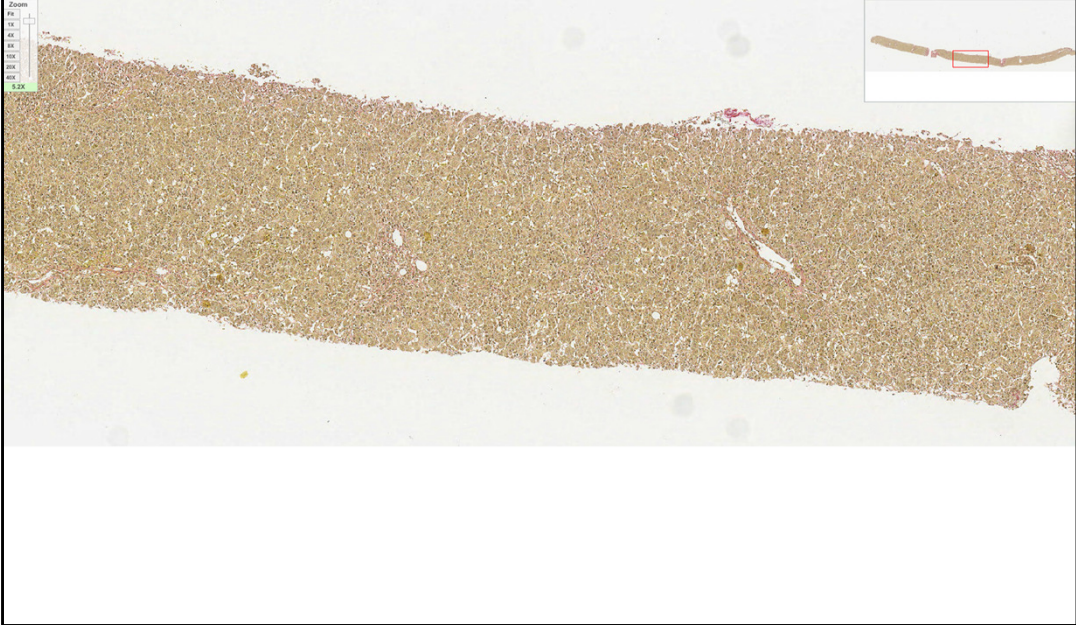
Reticulin – higher magnification – reticulin appears condensed around hepatic veins, and also shows interruption of liver cell plates due to hepatocyte necrosis.

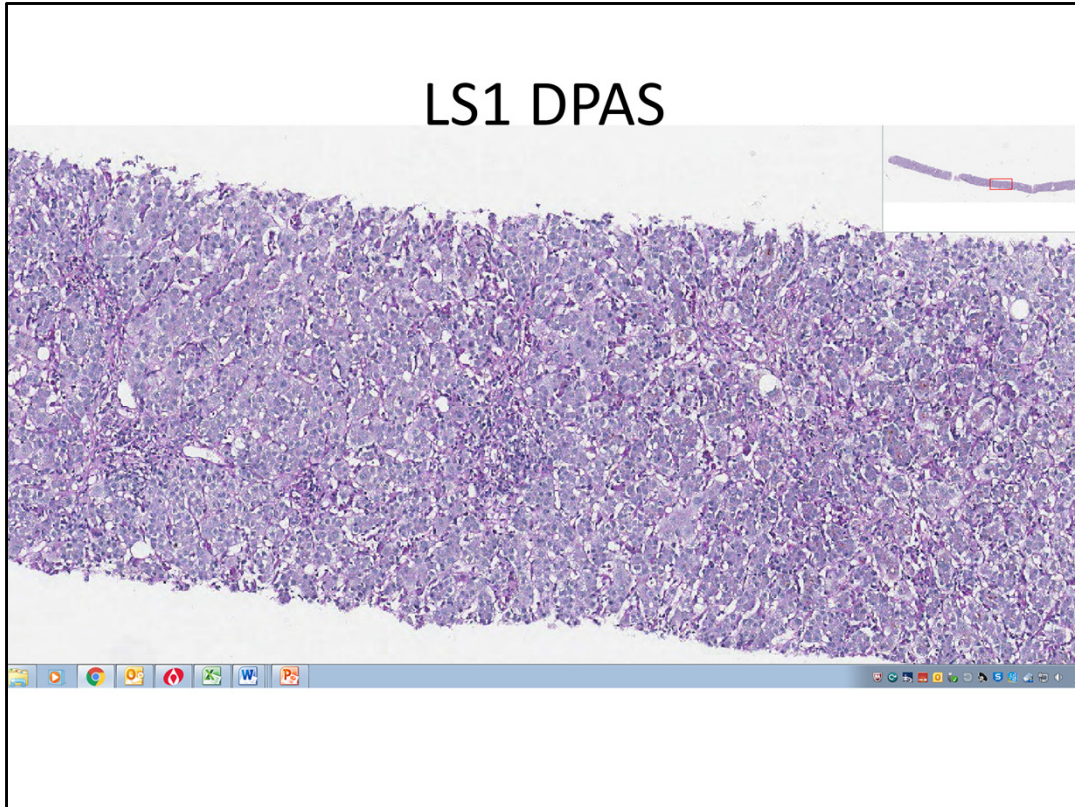
LS1



Van Gieson –no increased collagen to suggest chronic liver disease.

LS1





DPAS stain – highlights lobular disarray and shows some Kupffer cell activity – better seen at higher magnification.

Case LS1 35F

New onset severely deranged LFTs, no previous history. Admitted with severe jaundice and very deranged LFTs, ALT >1000 also retic, van Gieson, PASD

A	Chronic active hepatitis
B	Acute hepatitis
C	Sclerosing cholangitis
D	Hepatitis with bridging fibrosis
E	Autoimmune hepatitis

Correct answer: B. This is acute hepatitis with cholestasis. The very high ALT >1000 is seen in acute hepatitis, or in liver necrosis (paracetamol, ischaemia). The pattern of acute hepatitis can be seen in drug induced liver injury, recent onset autoimmune hepatitis and recent viral hepatitis, including hepatitis E. The presence of cholestasis should prompt a careful enquiry for history of drugs – cholestatic hepatitis is the commonest pattern of clinically important drug induced liver injury. It is less common in the acute presentation of autoimmune hepatitis, but can occur.

Comments on other options:

A Chronic active hepatitis

this is old terminology from the 1960's, when chronic hepatitis was classified according to morphological pattern into chronic persistent and chronic active hepatitis. Later, the primary classification of chronic hepatitis became based on aetiology – viral (hepatitis B, C) autoimmune, drugs. 'Chronic active hepatitis' is ambiguous – it is unclear whether this is a non-specific term for chronic liver disease, or implies autoimmune chronic hepatitis, and so is best avoided.

C Sclerosing cholangitis

there is bilirubinostasis, and a duct at one end of the biopsy had a suggestion of periductal fibrosis. However, the dominant pattern is of lobular disarray (acute hepatitis), and in view of the absence of portal expansion or fibrosis or ductopenia there is no corroborative histological support for an additional diagnosis of an underlying chronic biliary disease. In general, if there is a suspicion of biliary disease, (raised alkaline phosphatase and look closely for periportal copper associated protein

and/or periportal hepatocytes positive for keratin 7 (called 'intermediate hepatobiliary cells') If present the clinicians should consider further investigations for biliary disease.

D Hepatitis with bridging fibrosis – there is focal bridging between portal areas and hepatic veins, but this is due to hepatocyte loss in the context of acute hepatitis (bridging necrosis). There is no bridging fibrosis on the van Gieson stain.

E Autoimmune hepatitis

this could be in the differential, but requires clinical information to make the diagnosis. This is a clinico-pathological diagnosis, depending on the context of raised IgG and appropriate autoantibodies (usually anti-nuclear, anti-smooth muscle, or liver-kidney microsomal antibodies), together with high ALT and histological evidence of hepatitis. The characteristics of plasma cell rich inflammatory infiltrate with prominent interface hepatitis is not consistently present at presentation.