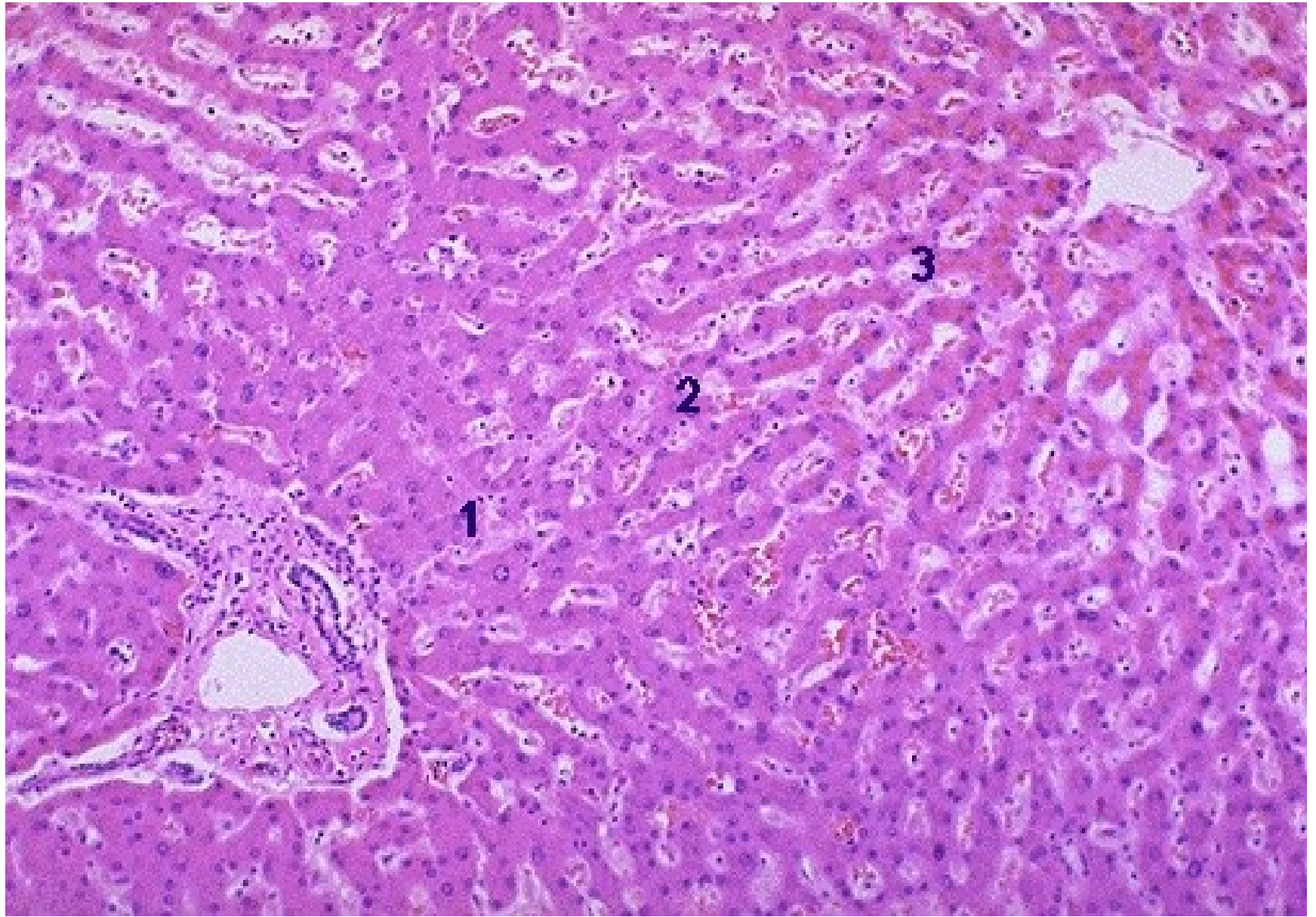
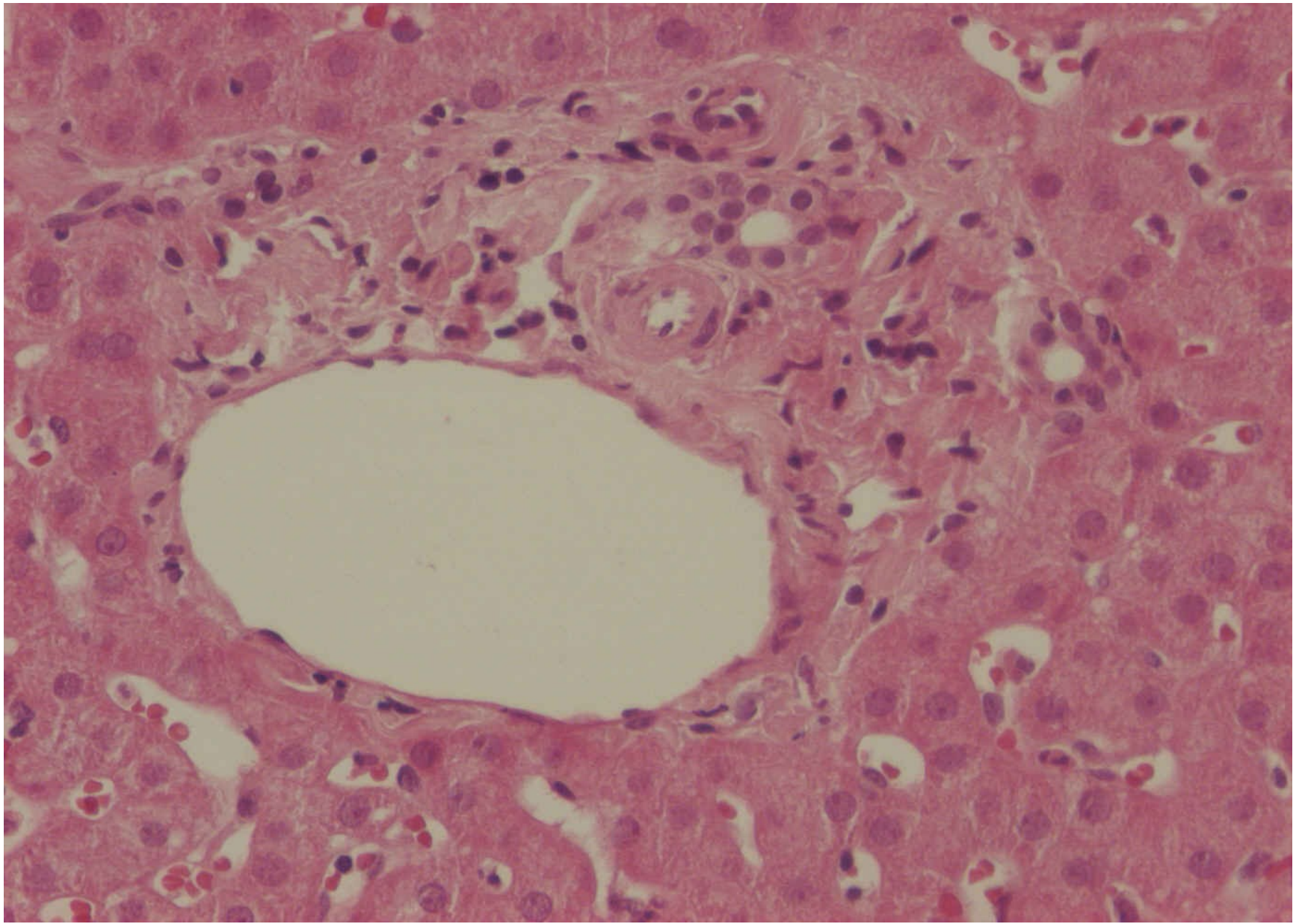
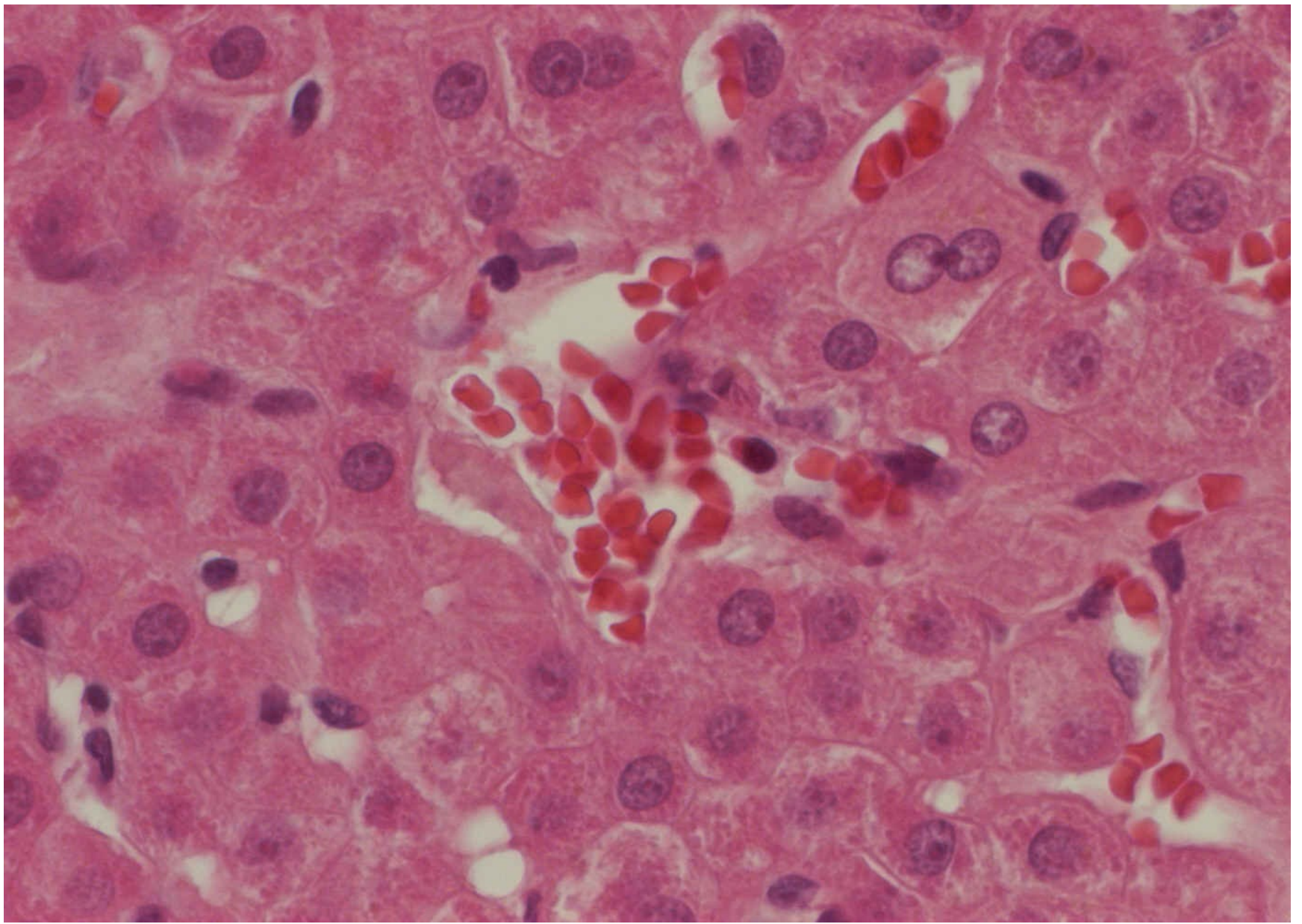

Pathology of the Liver



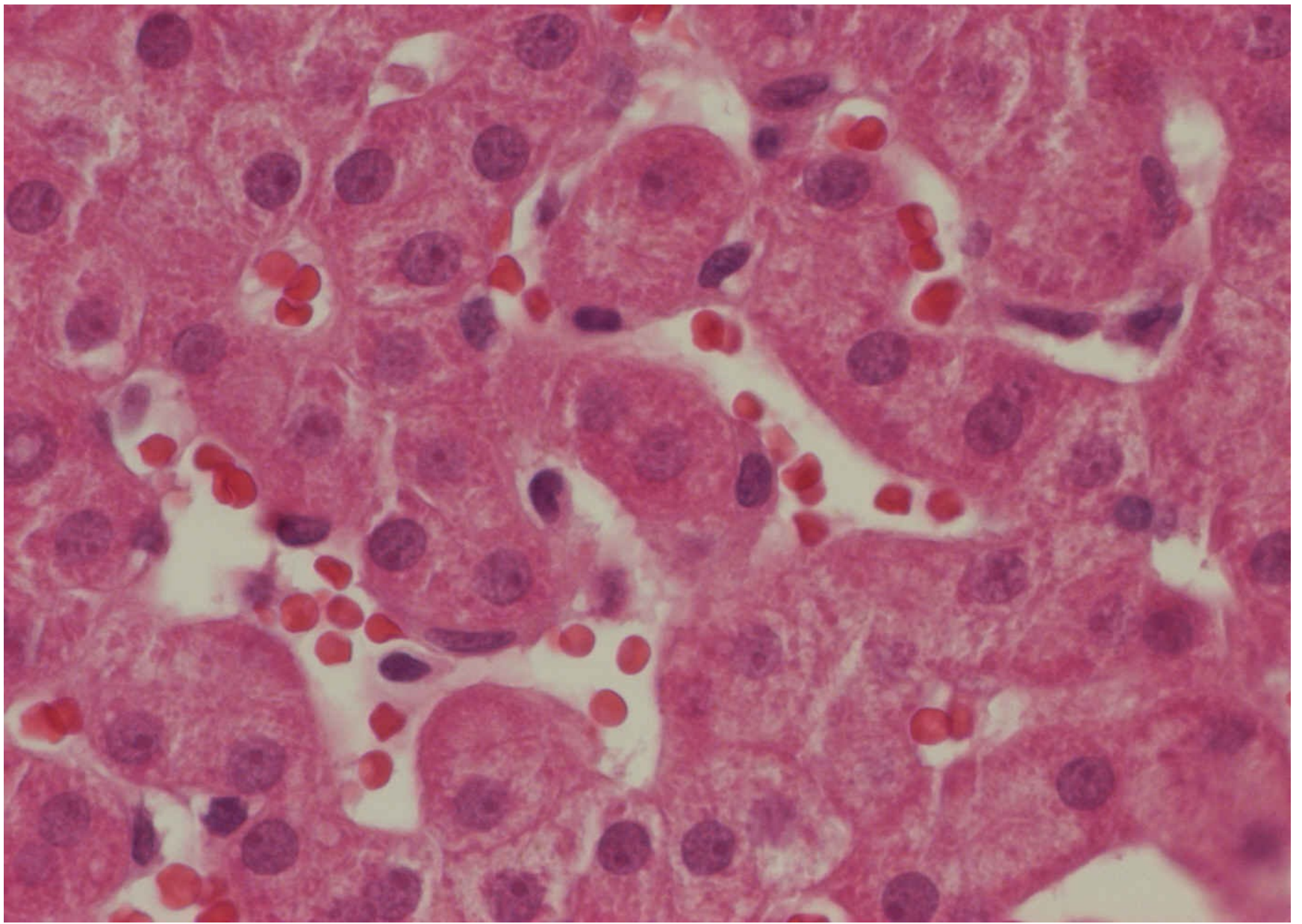
Liver – normal histology



Portal tract – normal histology



Area around a central vein – normal



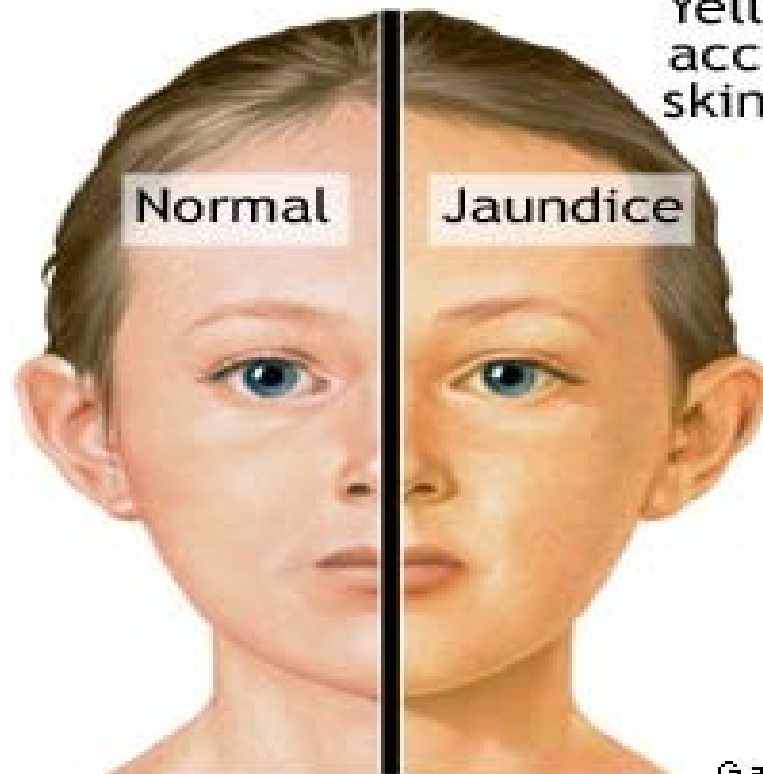
Liver parenchyma – norma

Pathology of the Liver

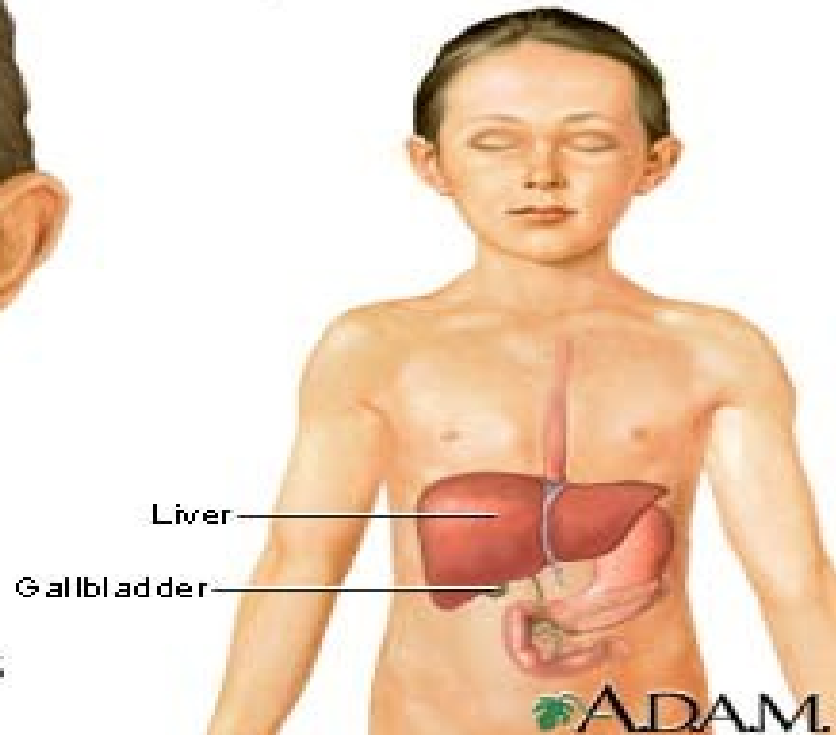
jaundice

- icterus – yellow discoloration of the skin and sclerae due to retention of pigmented bilirubin
 - bilirubin – the end product of hemoglobin degradation
 - icterus prehepatal, hepatal and posthepatal
-

Yellowing is associated with the accumulation of bilirubin in the skin, most often caused by liver and gallbladder disorders



Jaundice is a symptom where the skin and eyes become yellow



Pathology of the Liver

prehepatal icterus (hemolytic icterus)

- Excess offer of unconjugated bilirubin within hemolytic diseases or inherited impaired bilirubin conjugation (Crigler-Najjar syndrome, Gilbert syndrome etc.)
 - Clinical symptoms – increased level of unconjugated bilirubin in serum, urine is of a normal colour, stool may be hypercholic
-

Pathology of the Liver

hepatal icterus (hepatotoxic icterus)

- Damage of hepatocytes (hepatotoxic drugs, viral hepatitis...)
 - Clinical symptoms – increased level of either conjugated or unconjugated bilirubin in serum, urine is of dark colour, stool is of normal colour.
-

Pathology of the Liver

posthepatal icterus (obstructive icterus)

- Impaired bile flow into the gut (inherited atresia of bile ducts, choledocholithiasis, tumours of the head of pancreas!!, scar stricture of the choledochus...)
 - Clinical symptoms – increased level of particularly conjugated bilirubin, increased level of ALP and GMT, urine is of dark colour, stool is colourless
 - Icterus of obstructive type is accompanied with severe pruritus !!
-

Pathology of the Liver

viral hepatitis

- Infectious diseases caused by hepatotropic viruses (particularly viruses of hepatitis A-G, in our condition most commonly hepatitis viruses A, B, C, CMV a EBV)
-

Pathology of the Liver

viral hepatitis – clinical course

- Prodromal state
 - Flue symptoms (fatigue, increased temperature), GIT symptoms (lack of appetite, nausea...)
 - Stade of own illness
 - Icterus of variable intensity, enlargement of the liver...
 - Elevation of ALT, AST, bilirubin...
 - Recovery stade
-

Pathology of the Liver

viral hepatitis – clinical forms

- Inaparent form
 - Abortive form
 - Anicteric form
 - Icteric form
 - Cholesteatic form
 - Fulminant form
-

Pathology of the Liver

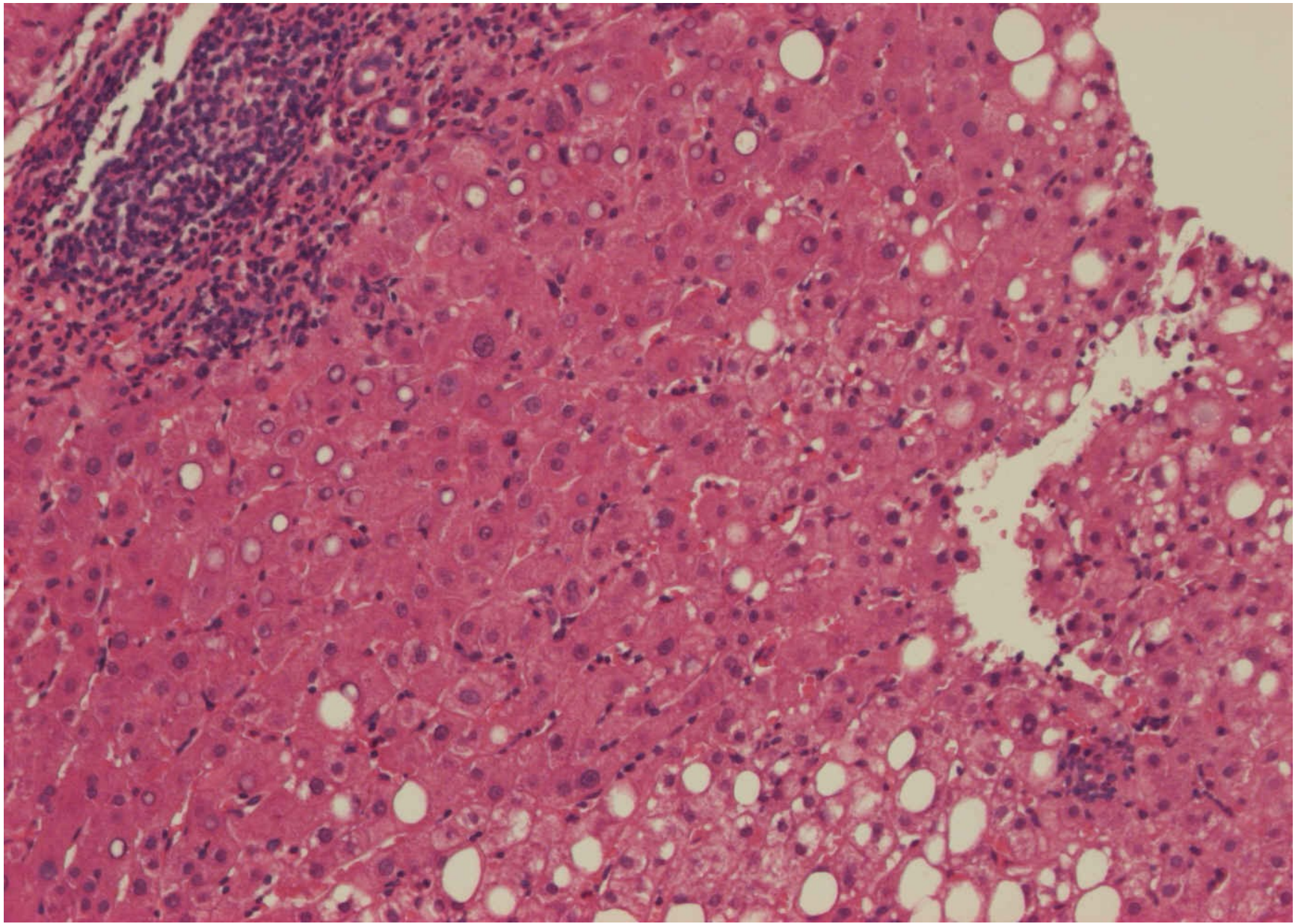
viral hepatitis - division

Type of the virus	Transmission	Incubation period	Chronic hepatitis	Vaccination
HAV	fecal-oral	15-55 days	ne	yes
HBV	parenteral	55-180 days	yes (5-10%)	yes
HCV	parenteral	14-90 days	yes (60-90%)	no
HDV	coinfection or superinfection with HBV	15-48 days	yes	no (HBV)
HEV	fecal-oral	20-50 days	no	no

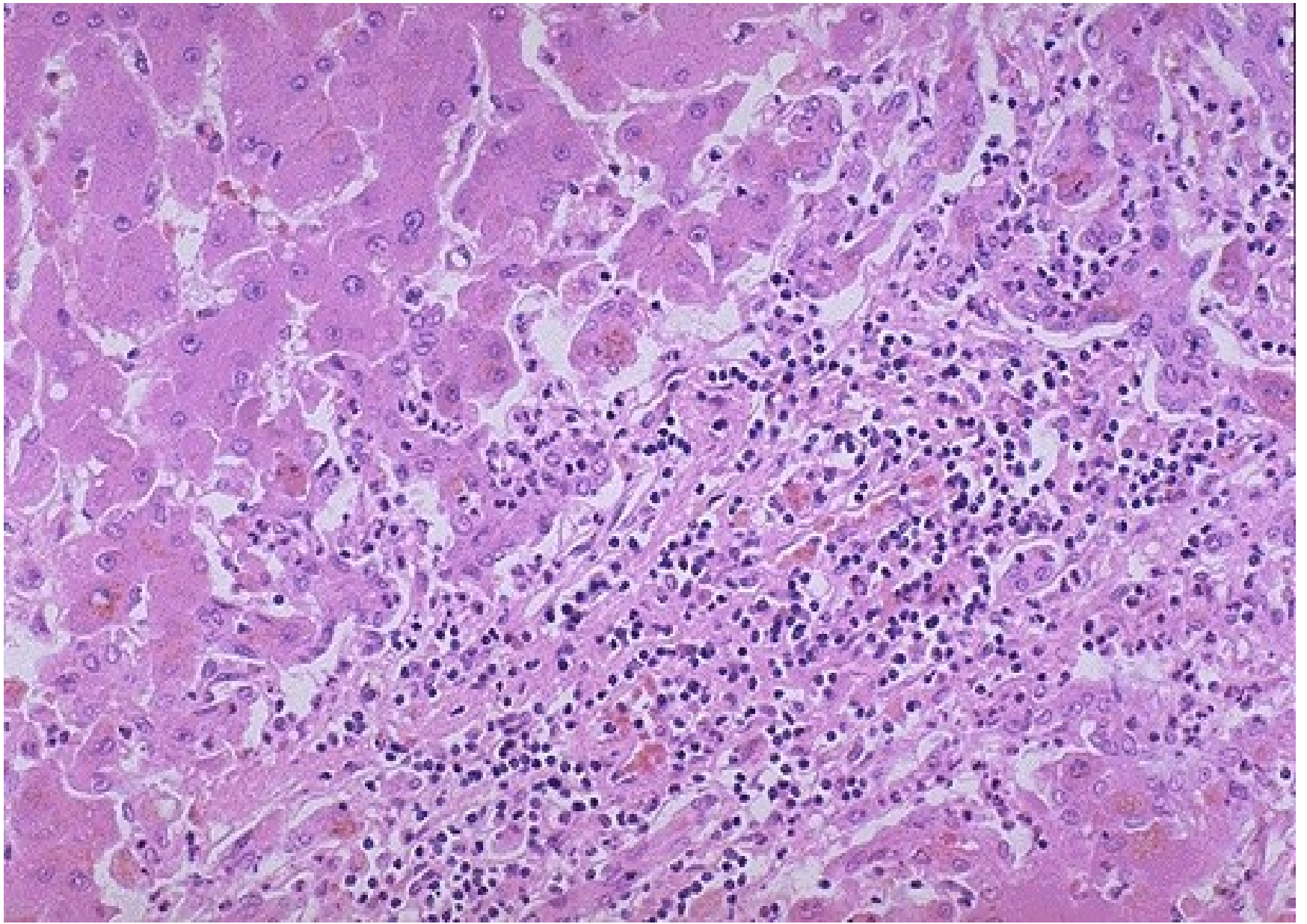
Pathology of the Liver

viral hepatitis – histology

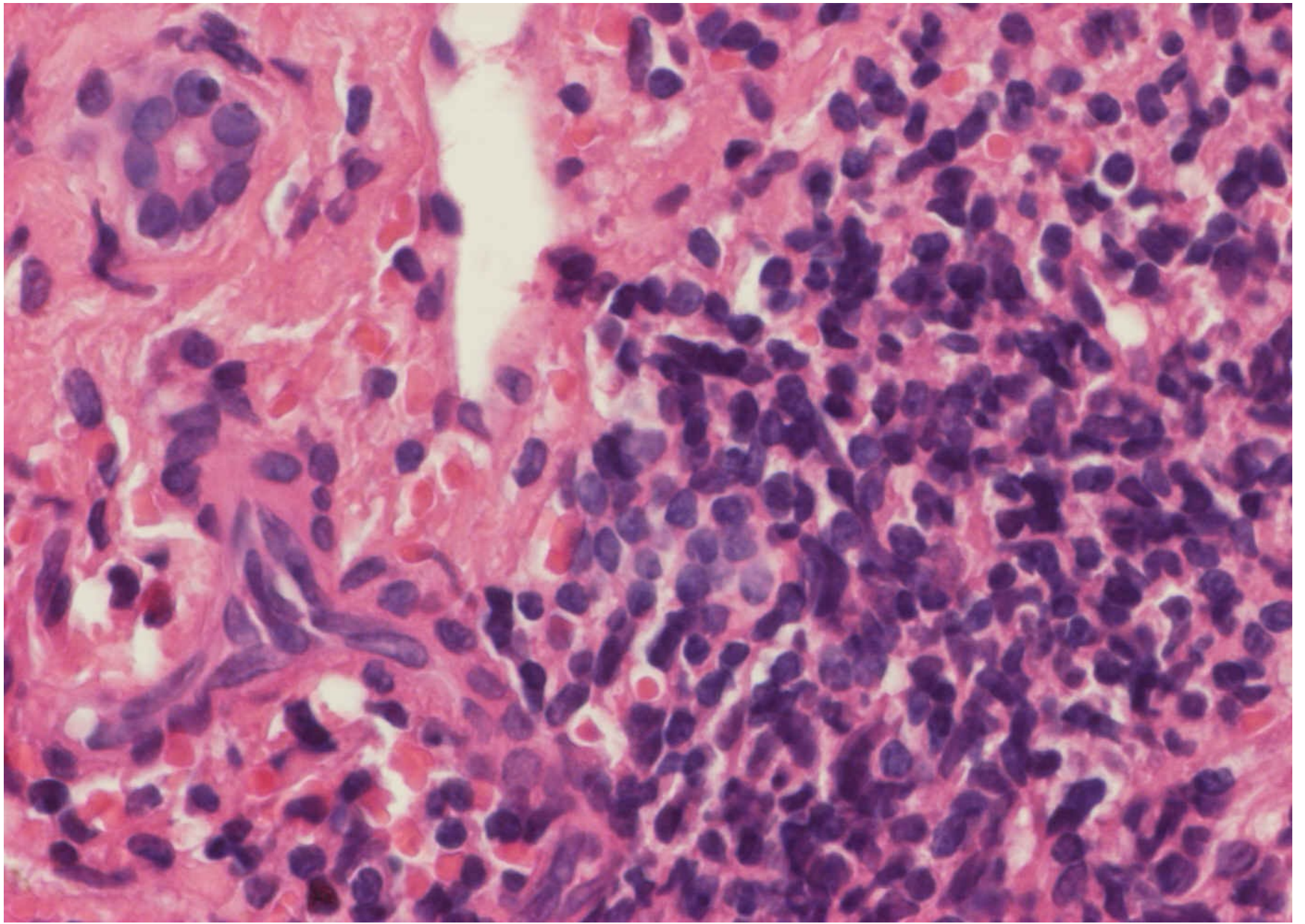
- acute hepatitis
 - necrosis of hepatocytes (focal, zonal, ev. bridging)
 - cholestasis in icteric form
 - portal tracts are infiltrated with a mixture of inflammatory cells
 - chronic hepatitis
 - minimal to mild form – inflammation limited to portal tracts (macrophages, lymphocytes, plasma cells), smoldering hepatocyte necrosis throughout the lobule.
 - moderate to severe active form – piecemeal necrosis, dispersed necrosis of hepatocytes, inflammatory infiltration of portal tracts, depositions of fibrous tissue (periportal and bridging necrosis)
-



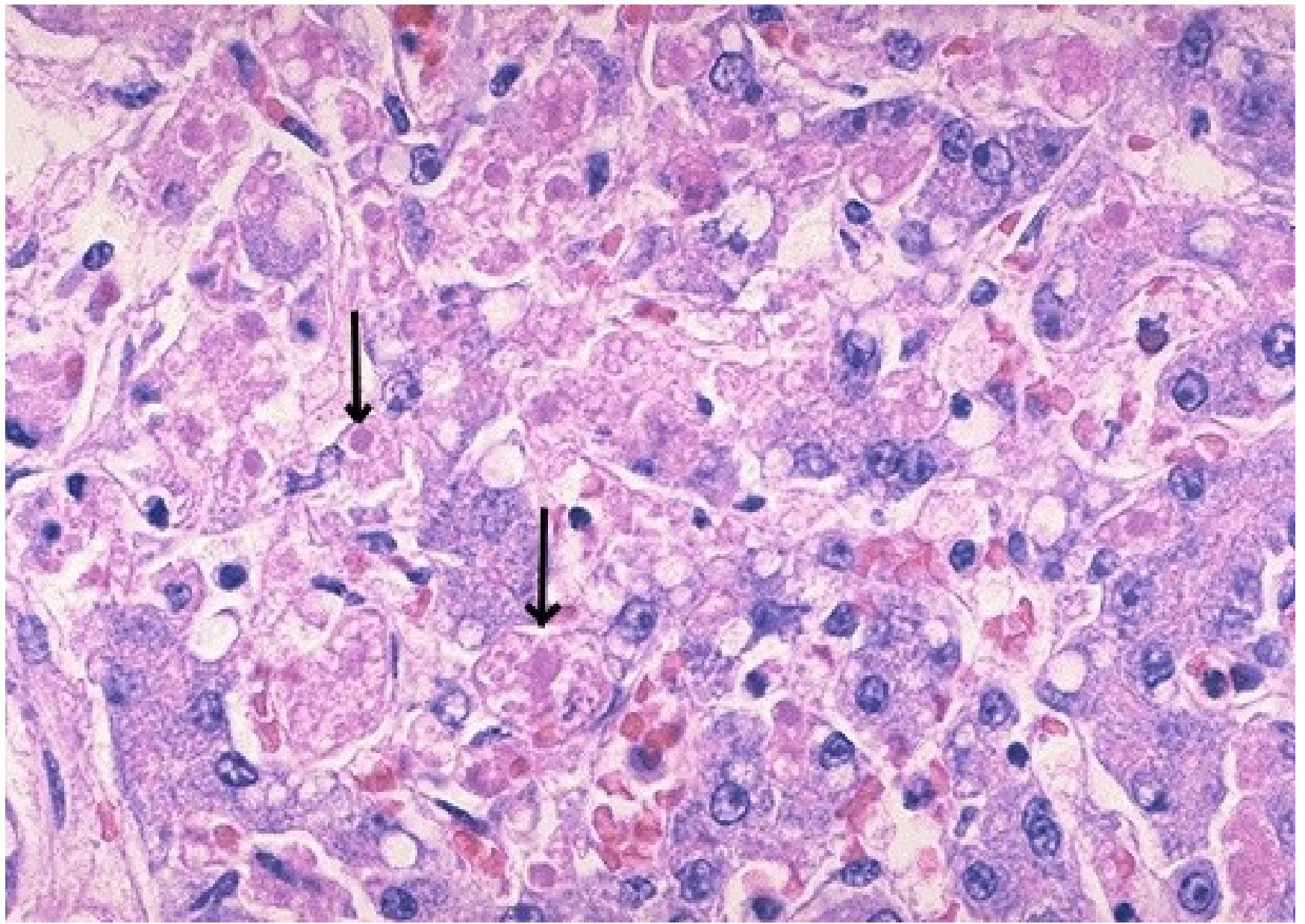
chronic viral hepatitis – piecemeal necrosis, dense inflammatory infiltrate



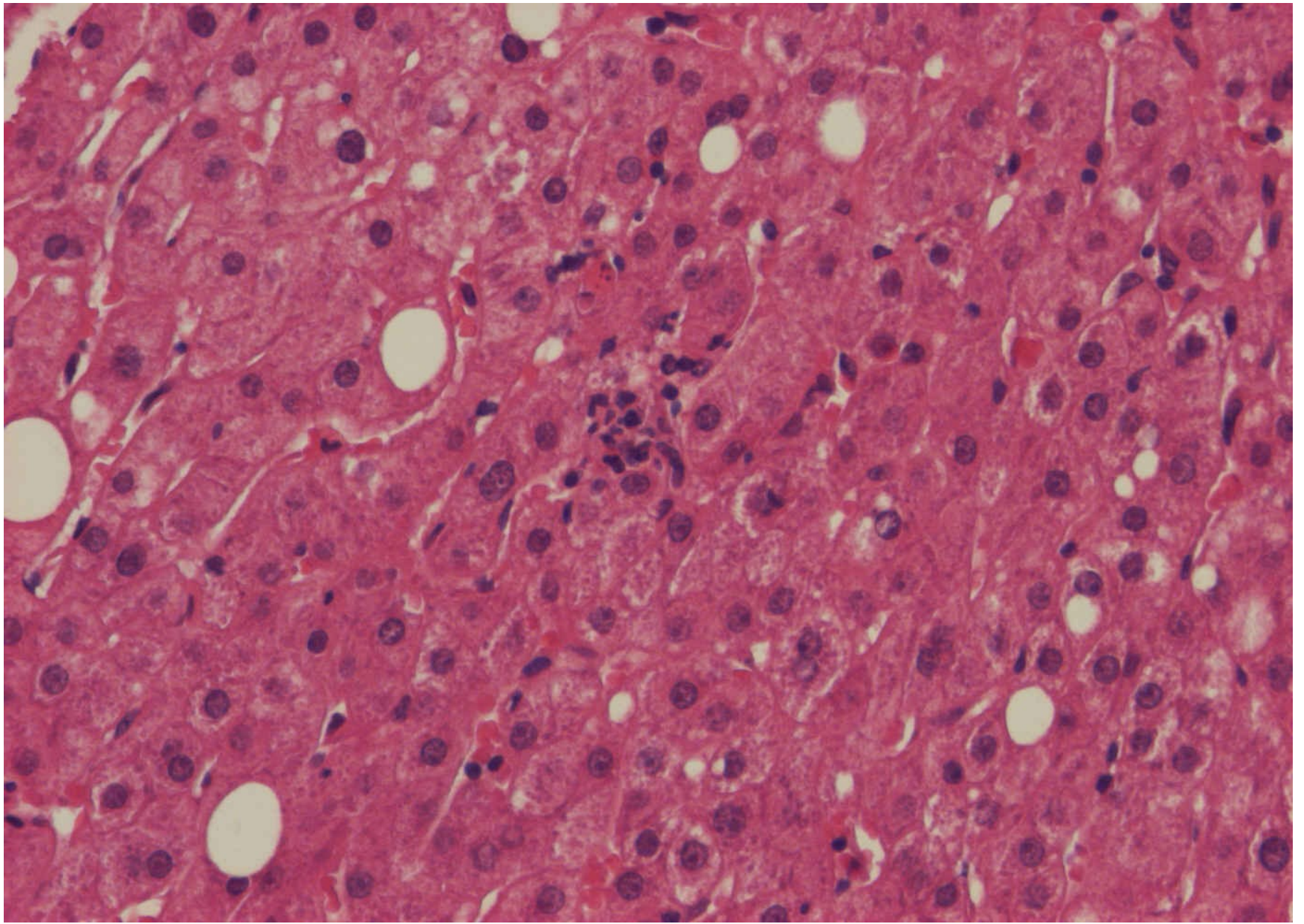
chronic viral hepatitis – piecemeal necrosis



chronic viral hepatitis – portal tract



chronic viral hepatitis – Councilman bodies



chronic viral hepatitis , active – neutrophils around necrotic hepatocyte

Pathology of the Liver

cirrhosis

- is a consequence of chronic liver disease characterized by replacement of liver tissue by fibrosis, scar tissue and regenerative nodules (lumps that occur as a result of a process in which damaged tissue is regenerated), leading to progressive loss of liver function.
-

Pathology of the Liver

cirrhosis

- macroscopically – irregularly nodulated surface, thickened capsule, hard consistency
 - micronodular – diameter of nodules less or equal to 3mm
 - macronodular – diameter of nodules more than 3mm
 - mixed
-



micronodular liver cirrhosis



micronodular liver cirrhosis

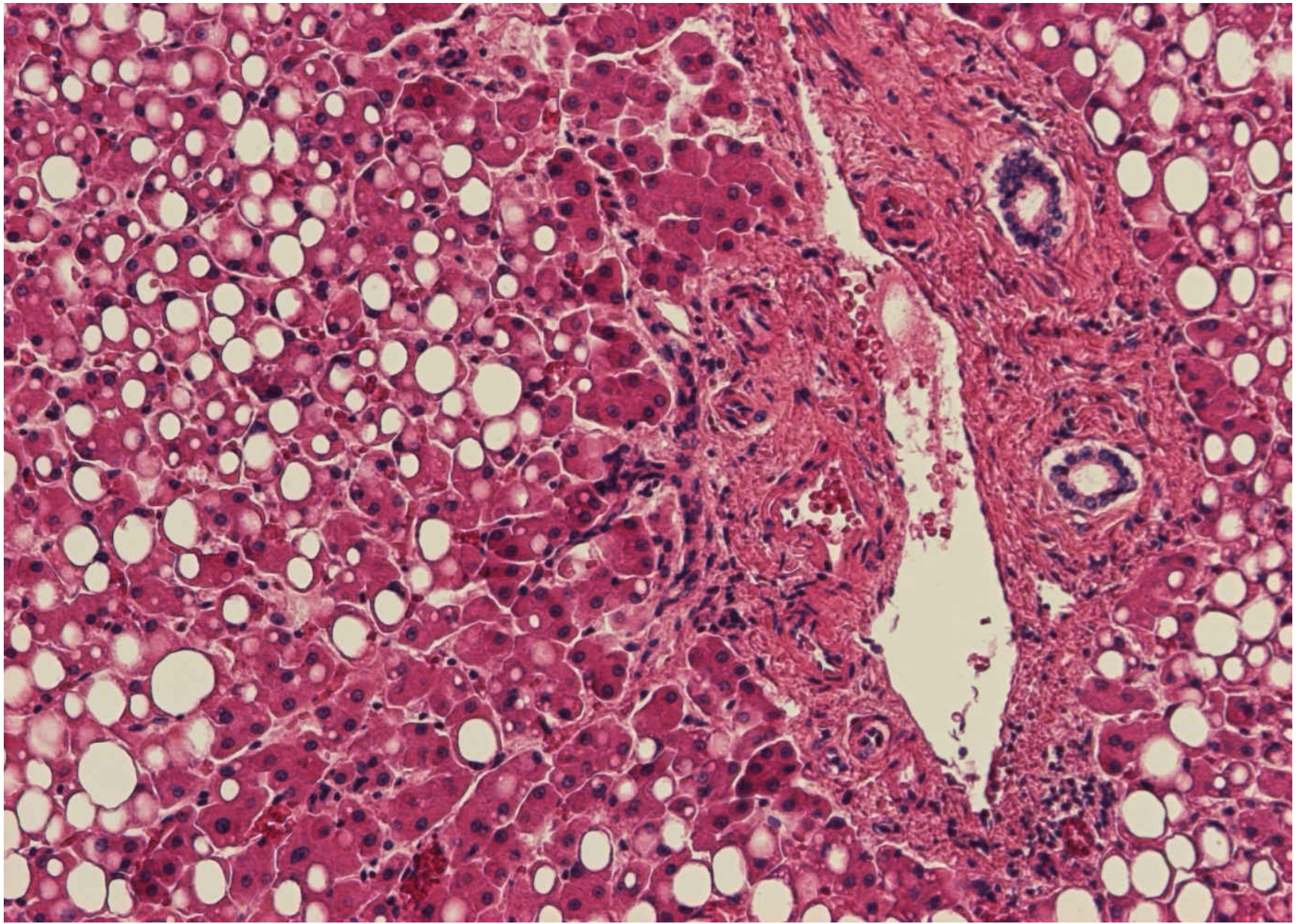
Pathology of the Liver

liver cirrhosis - etiology

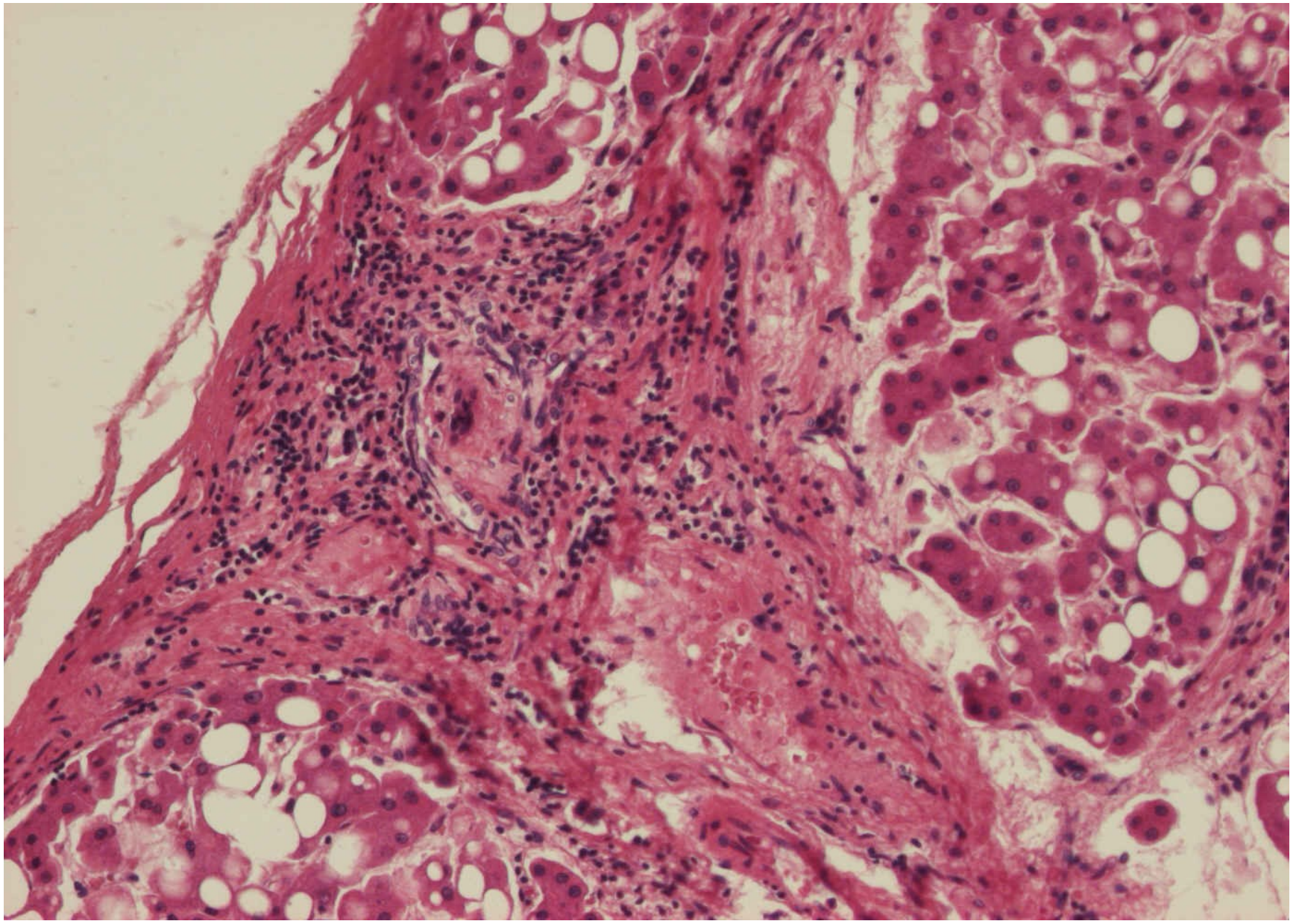
- posthepatitic cirrhosis
 - chronic hepatitis B, C, B+D
 - alcoholic liver disease
 - alcoholic steatosis
 - alcoholic steatohepatitis
 - alcoholic cirrhosis
-



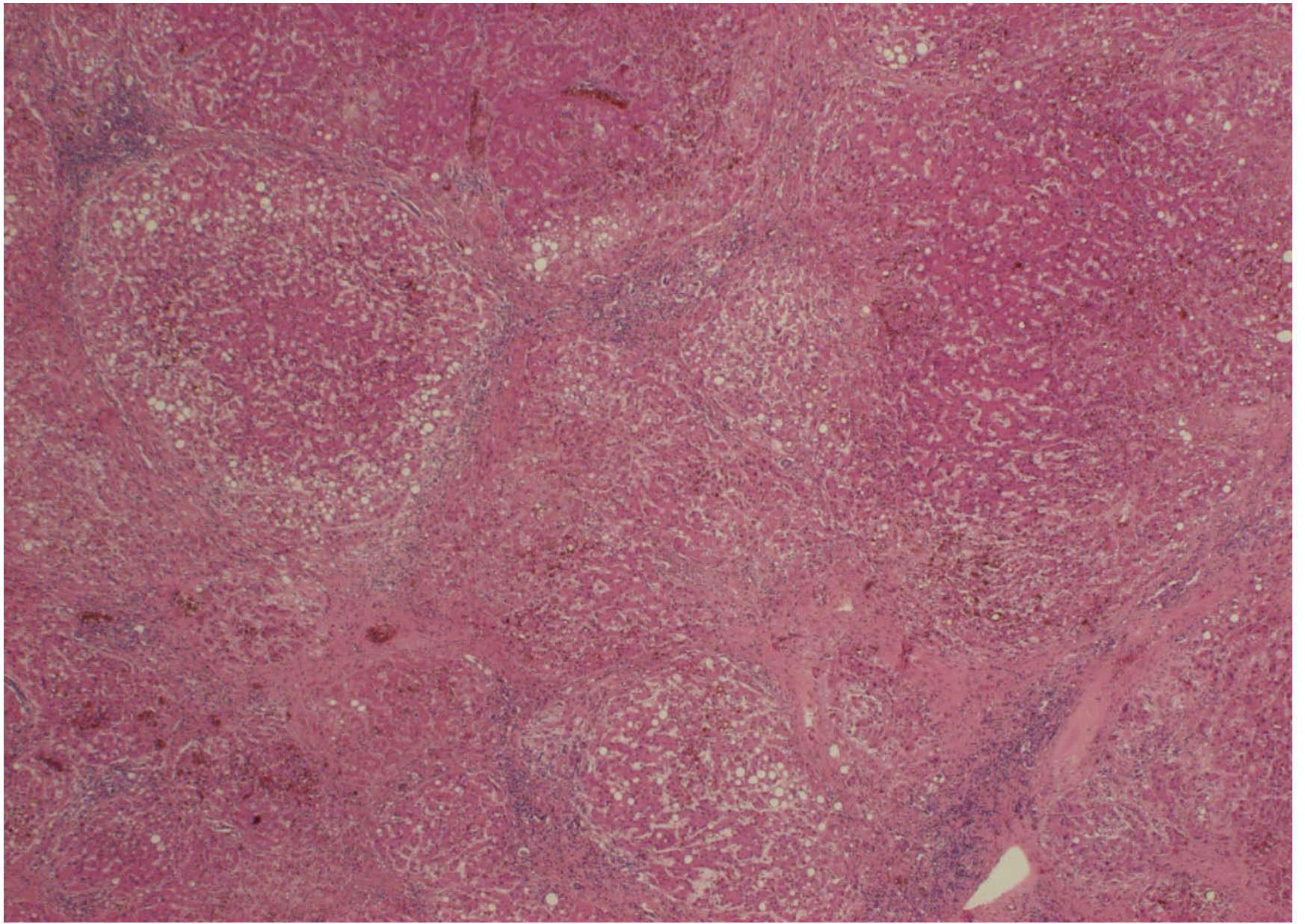
alcoholic liver disease – hepatic steatosis



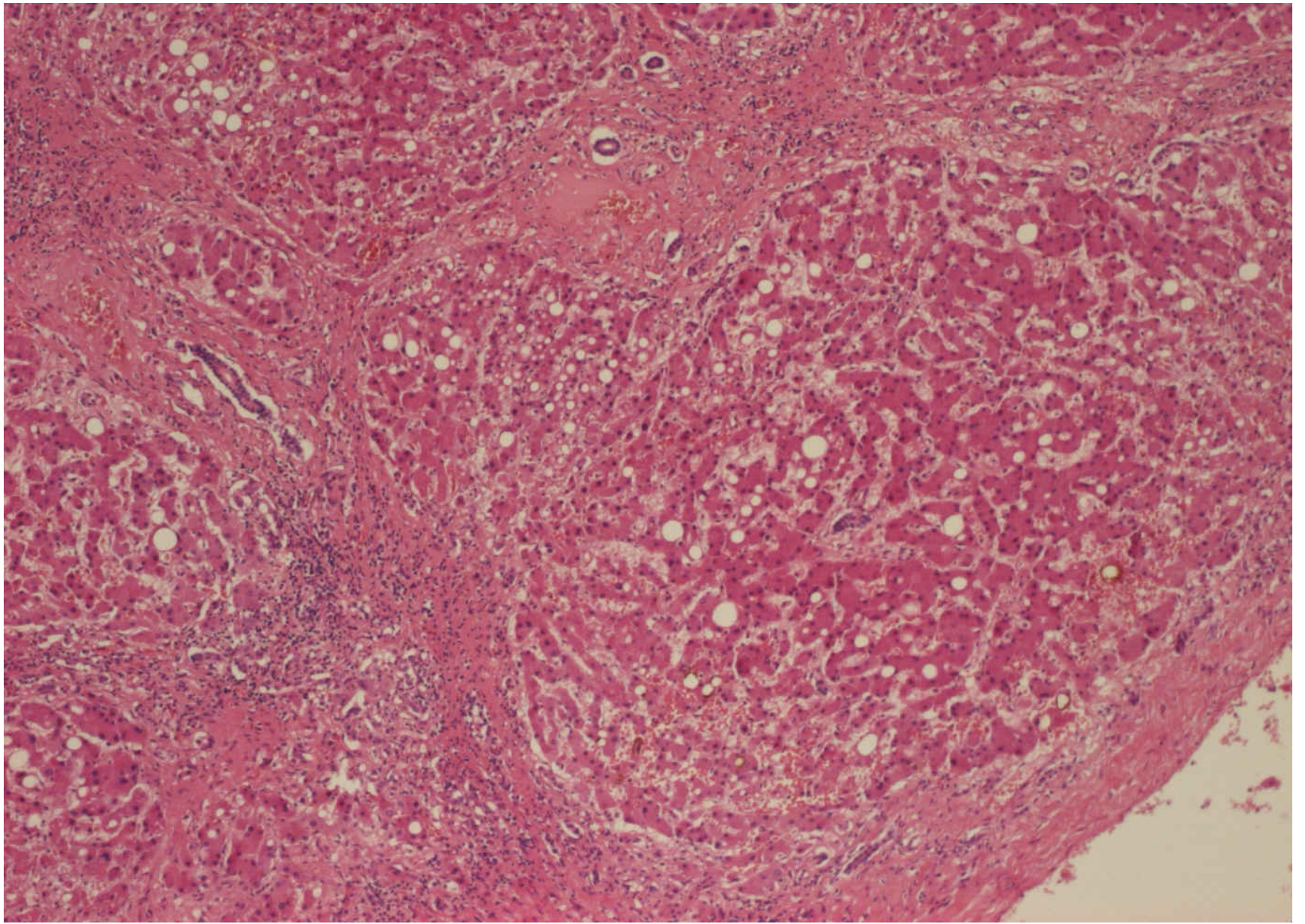
alcoholic liver disease – steatosis



alcoholic liver disease – steatohepatitis



alcoholic liver disease – micronodular cirrhosis



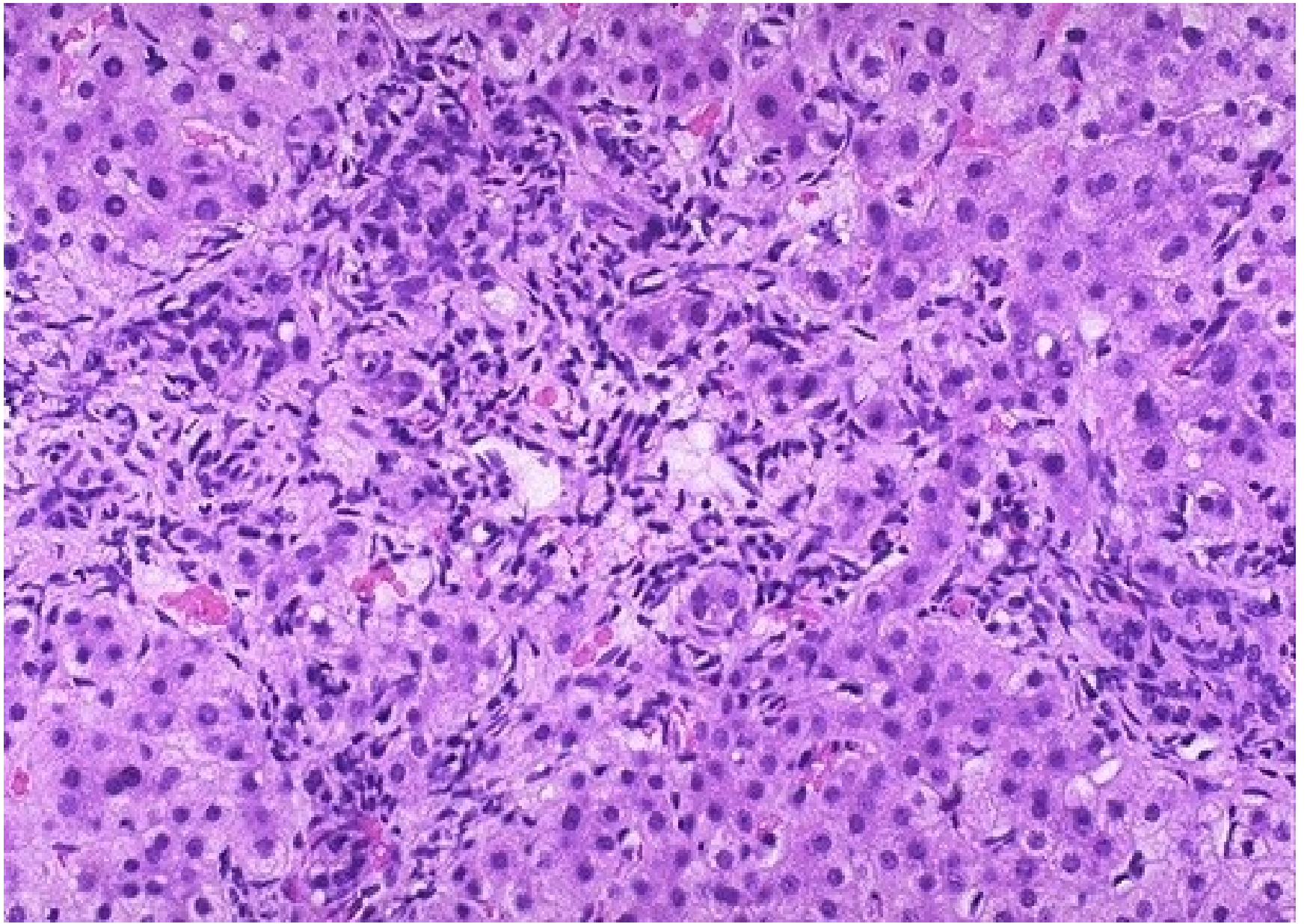
alcoholic hepatopathy – detail

Pathology of the Liver

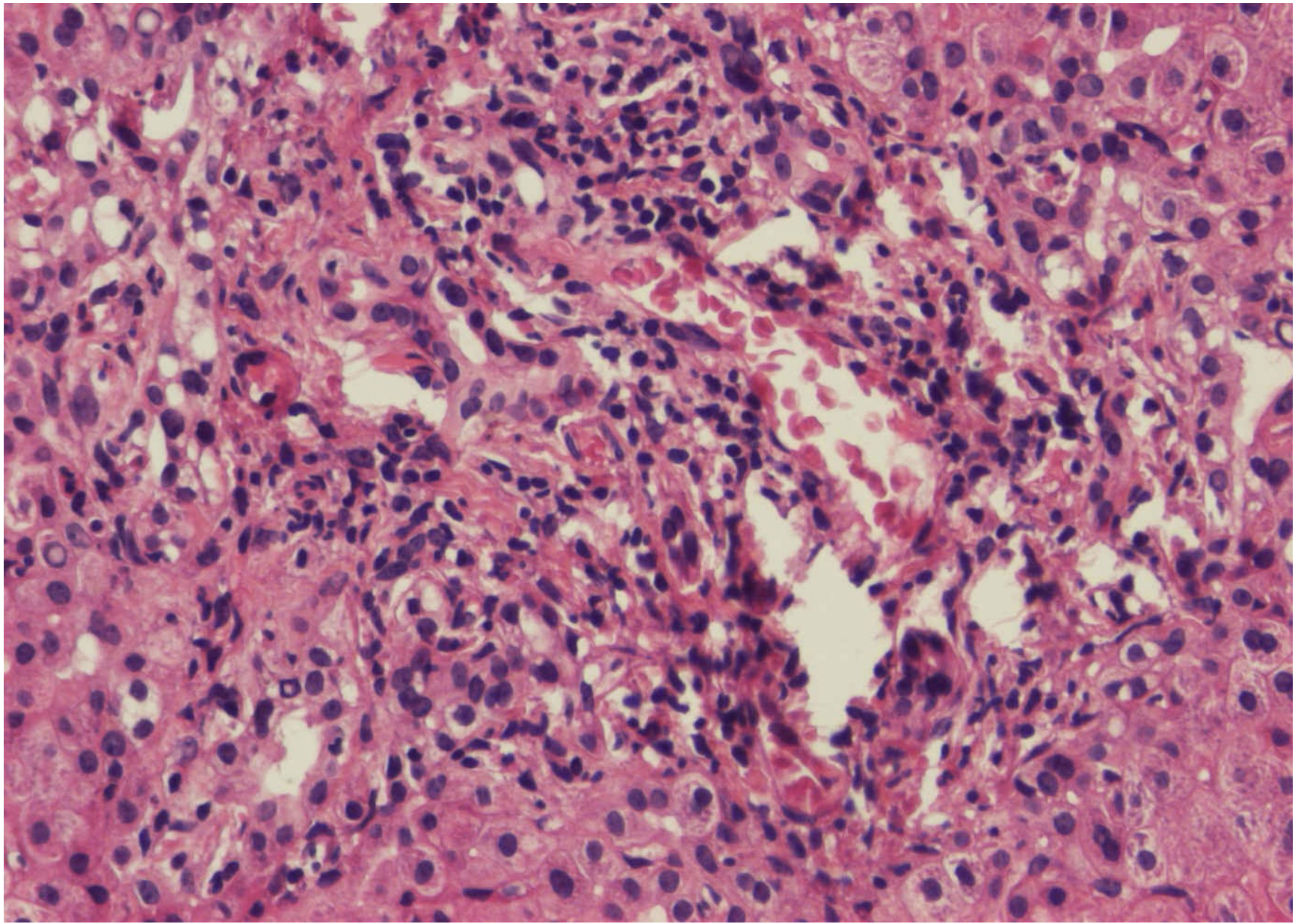
liver cirrhosis – etiology

■ Biliary cirrhosis

- primary biliary cirrhosis (PBC) – autoimmune disease, non-suppurative, granulomatous destruction of medium-sized intrahepatic bile ducts.
 - secondary biliary cirrhosis – prolonged obstruction to the extrahepatic biliary tree
-



primary biliary cirrhosis – chronic fibroproductive non-purulent inflammation



primary biliary cirrhosis – chronic fibroproductive non-purulent inflammation

Pathology of the Liver

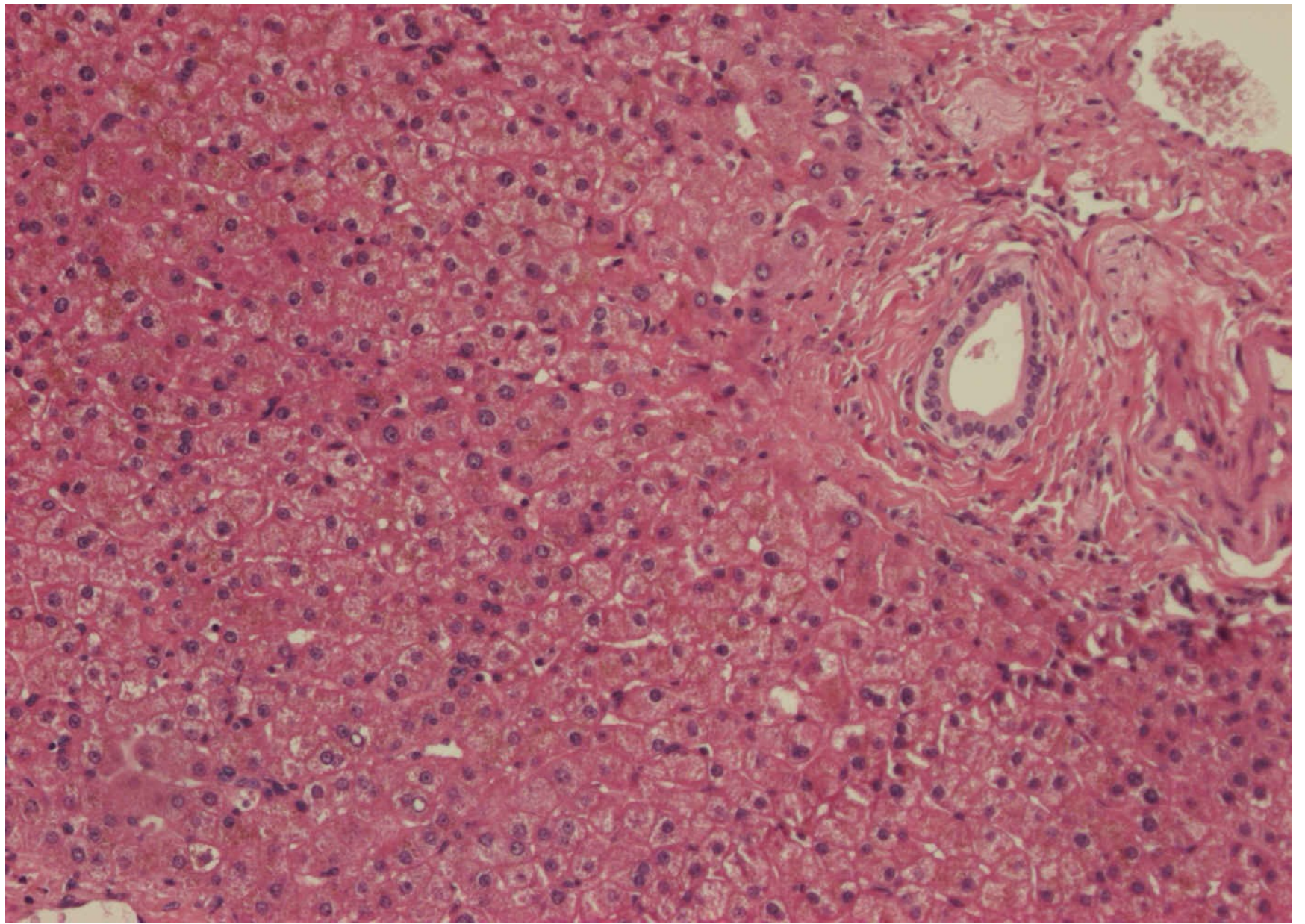
liver cirrhosis– etiology

- Metabolic diseases as causes of cirrhosis
 - Wilson disease – inherited failure of metabolism of copper (defective copper excretion into the bile) – accumulation of toxic levels of copper in the liver, brain and eye (Kayser-Fleischer rings)
 - hemochromatosis
 - α 1- Antitrypsin deficiency
 -
-

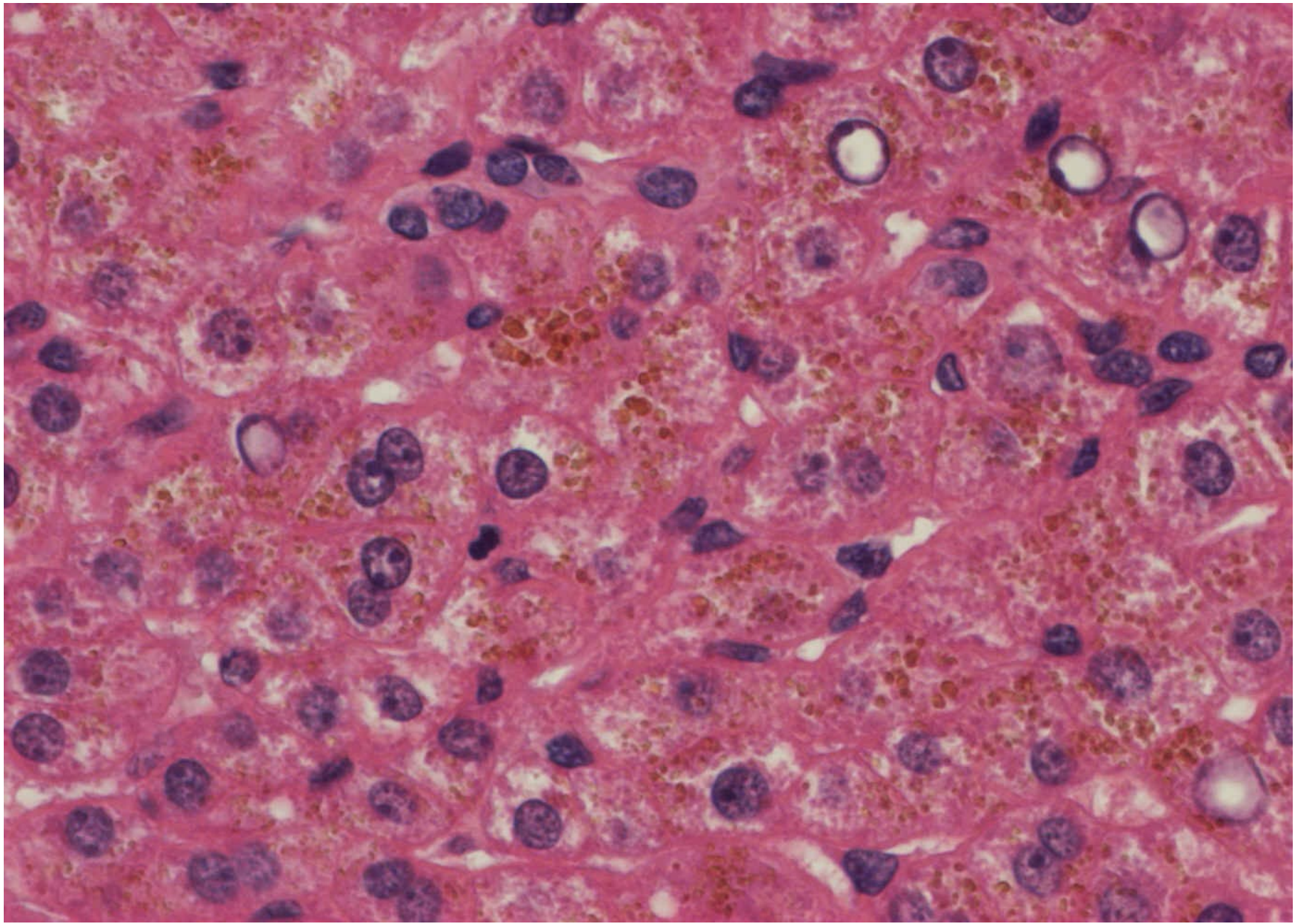
Pathology of the Liver

hemochromatosis

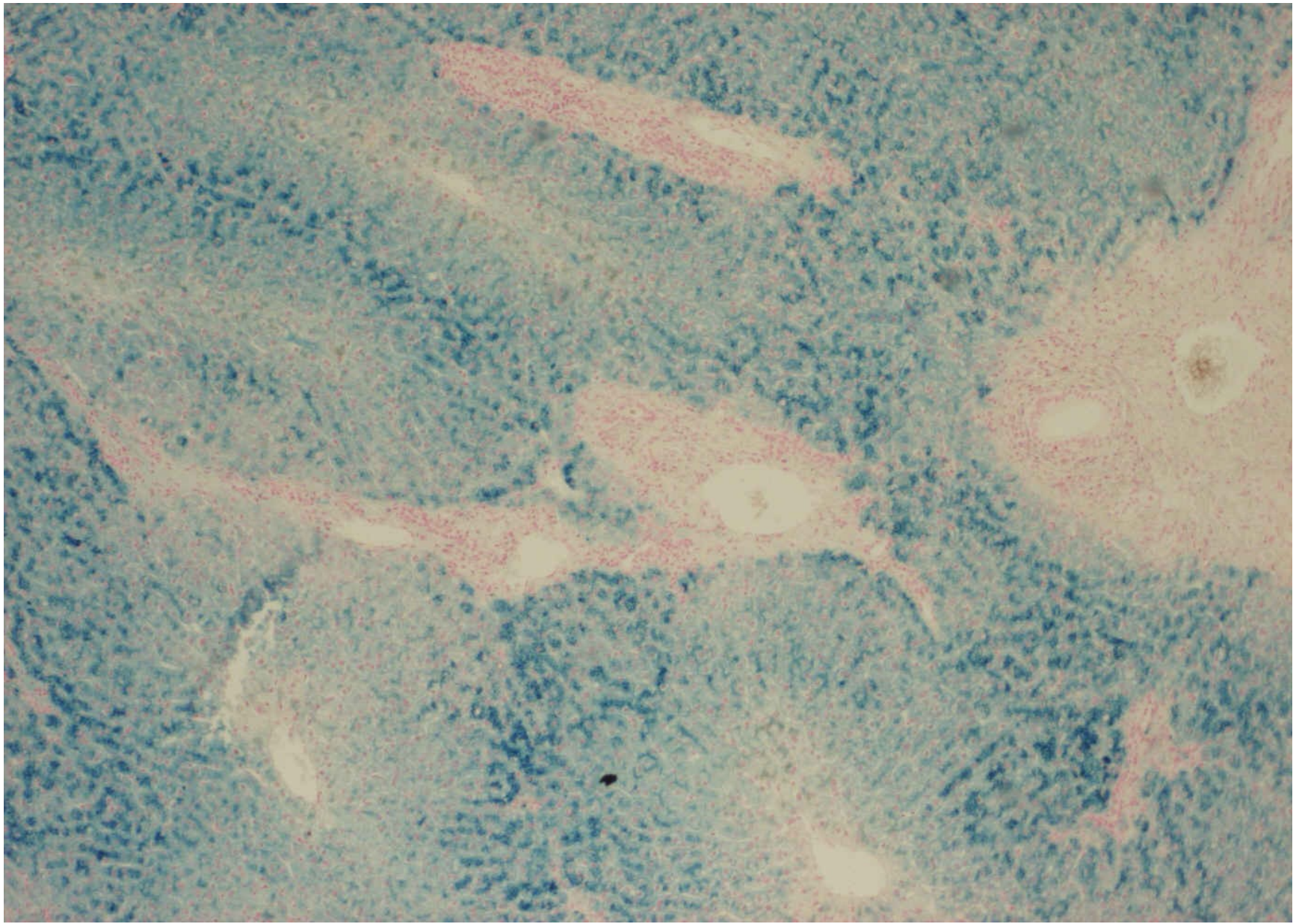
- excessive accumulation of body iron (liver, pancreas) – genetic defect, AR heritable disorder or secondary hemochromatosis
- clinically
 - micronodular cirrhosis (all patients), diabetes mellitus (75-80%), skin pigmentation (75-80%)
- pathogenesis
 - increased intestinal absorption of iron, excessive iron is directly toxic to host tissues (lipid peroxidation, stimulation of collagen formation, interactions with DNA)



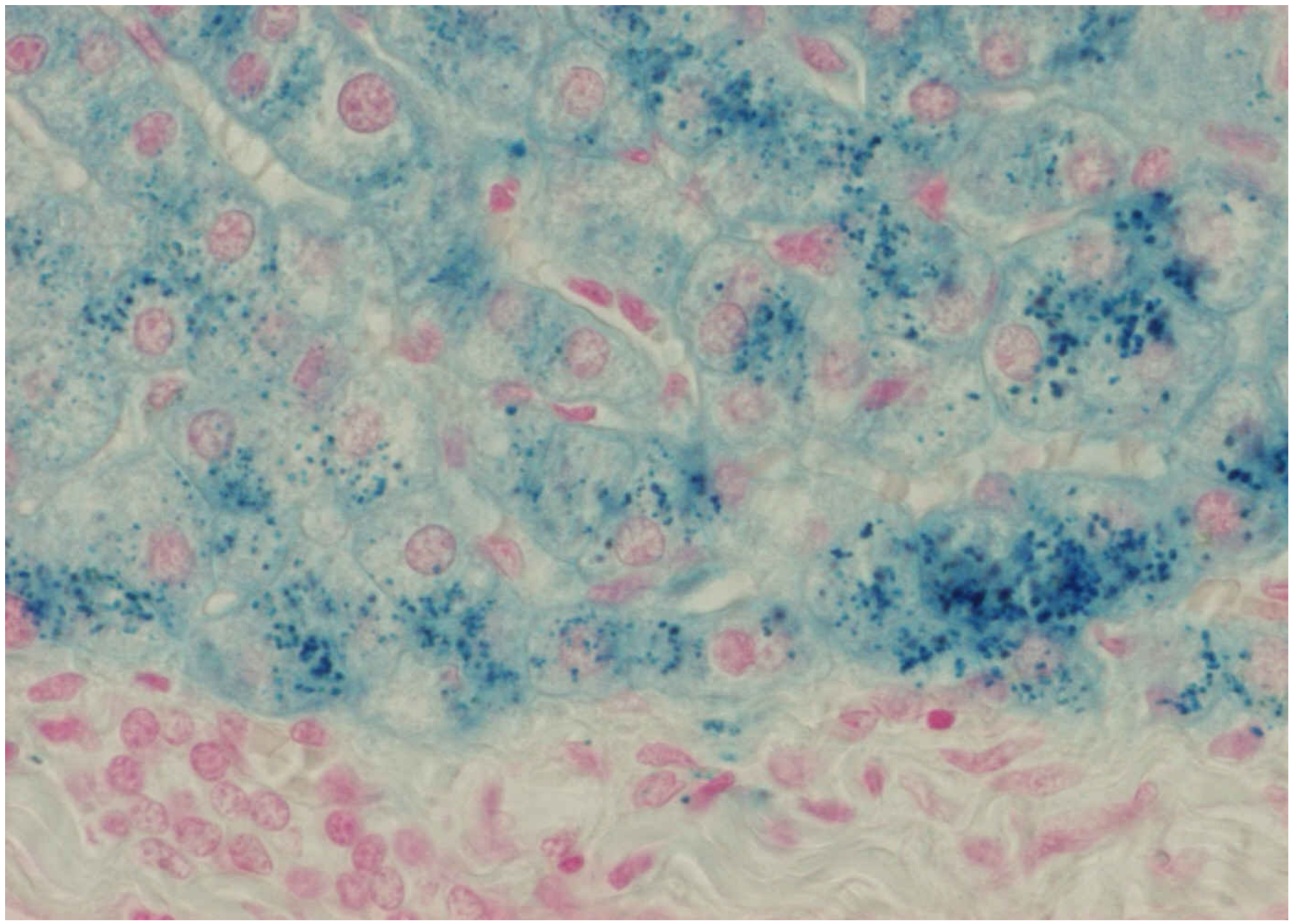
hemochromatosis



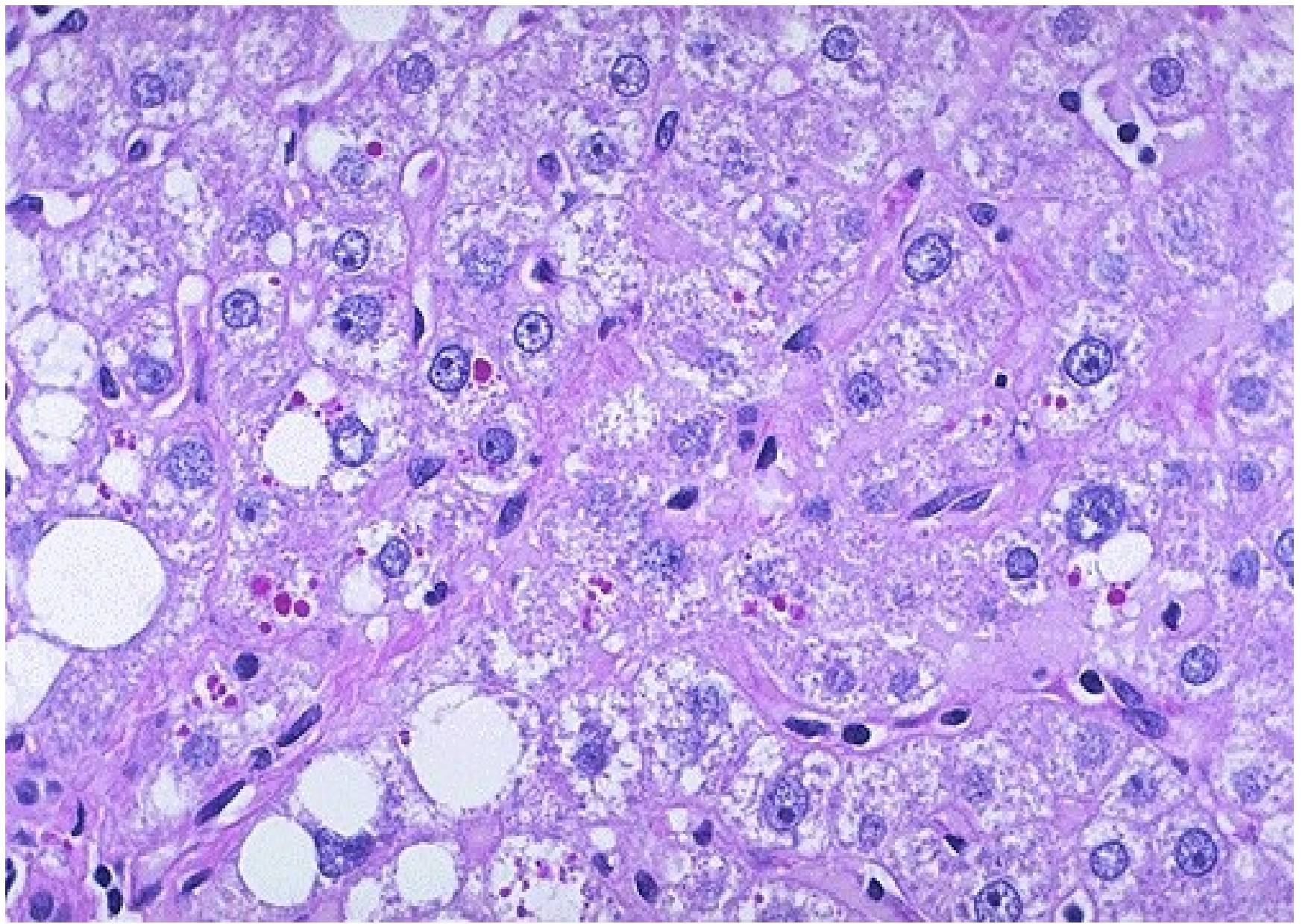
hemochromatosis – accumulation of hemosiderin intracellularly – detail



hemochromatosis - which staining by?



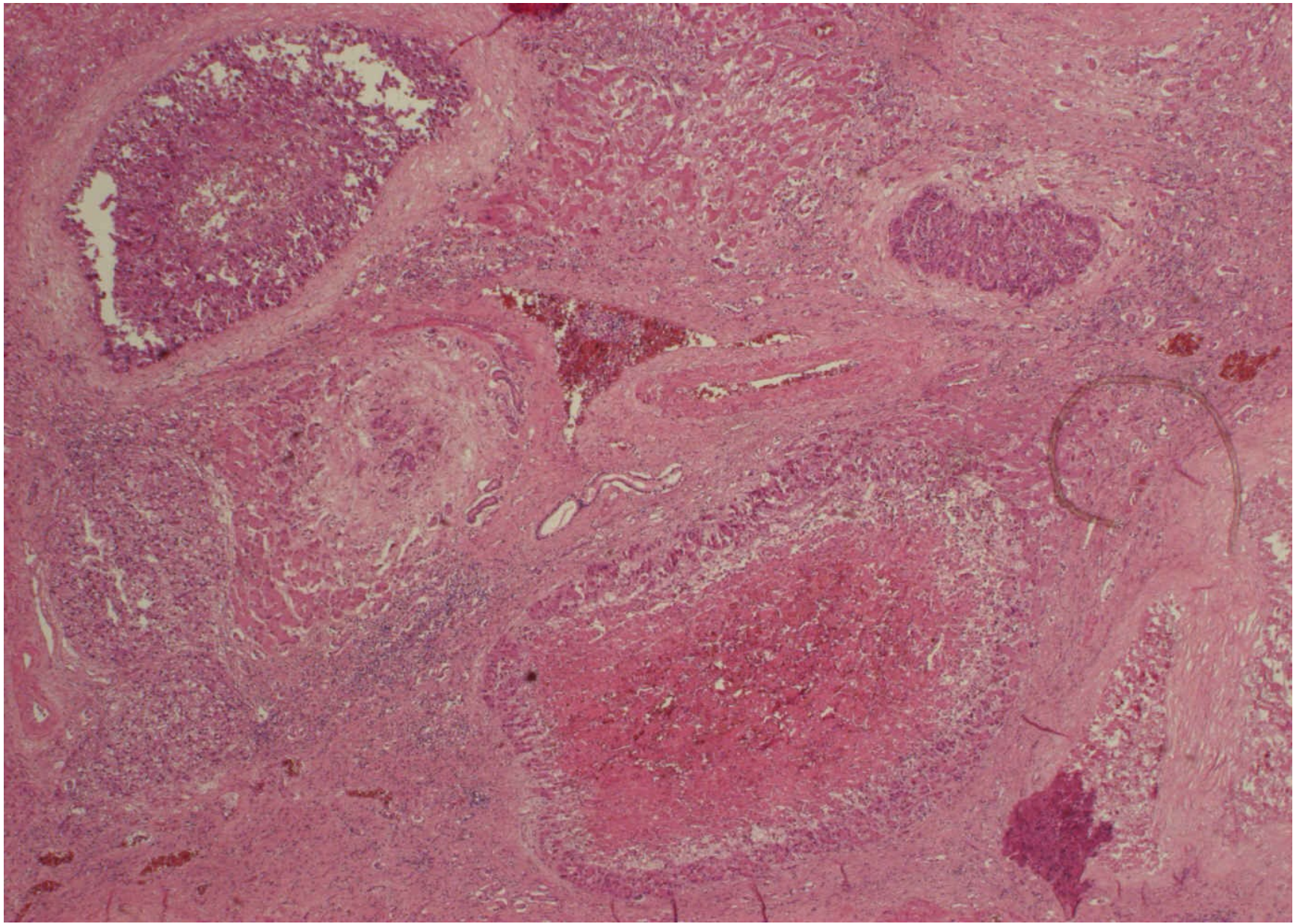
hemochromatosis– staining by Perls – detail



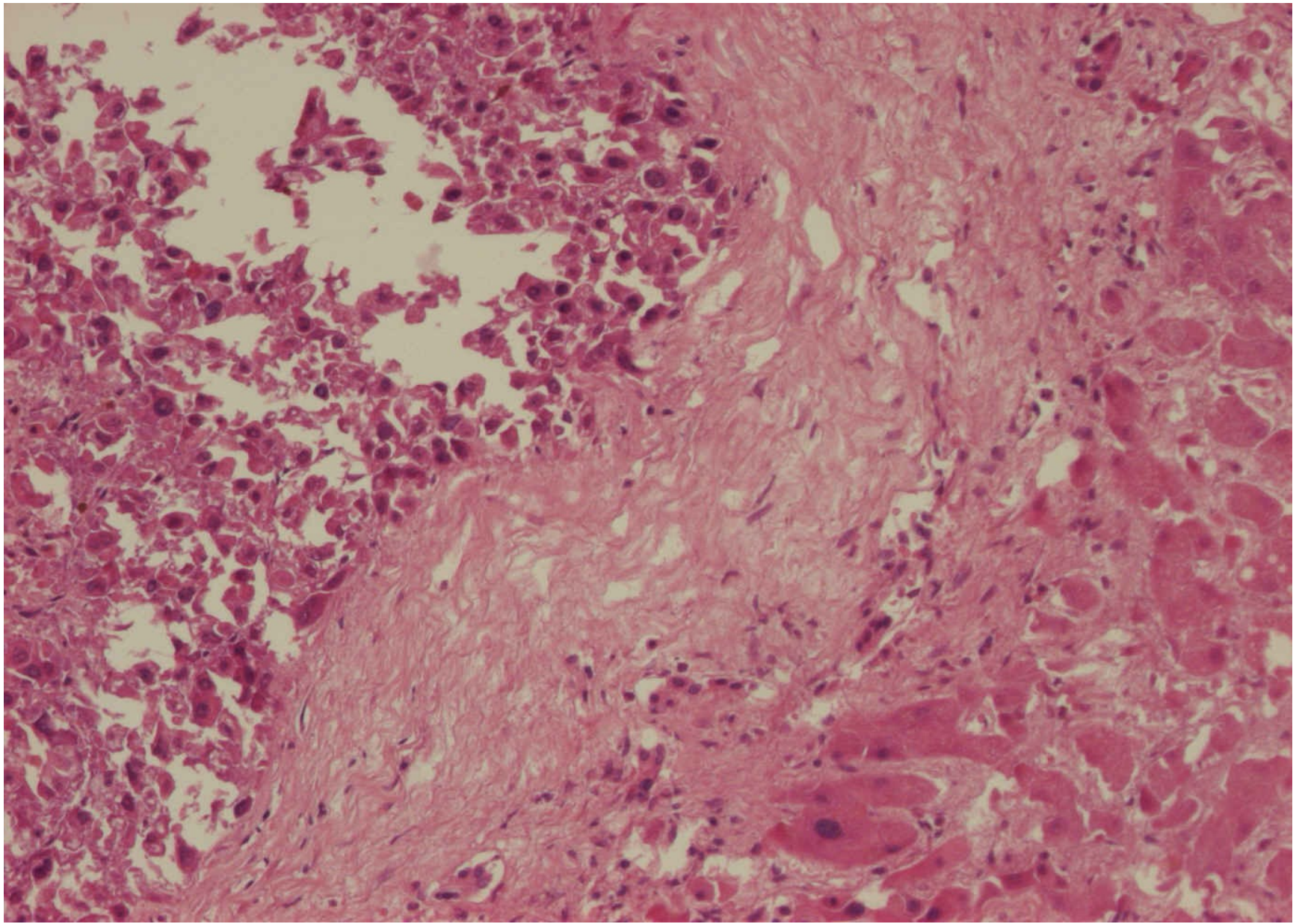
deficiency of alfa-1-AT – PAS reaction

Tumours of the Liver

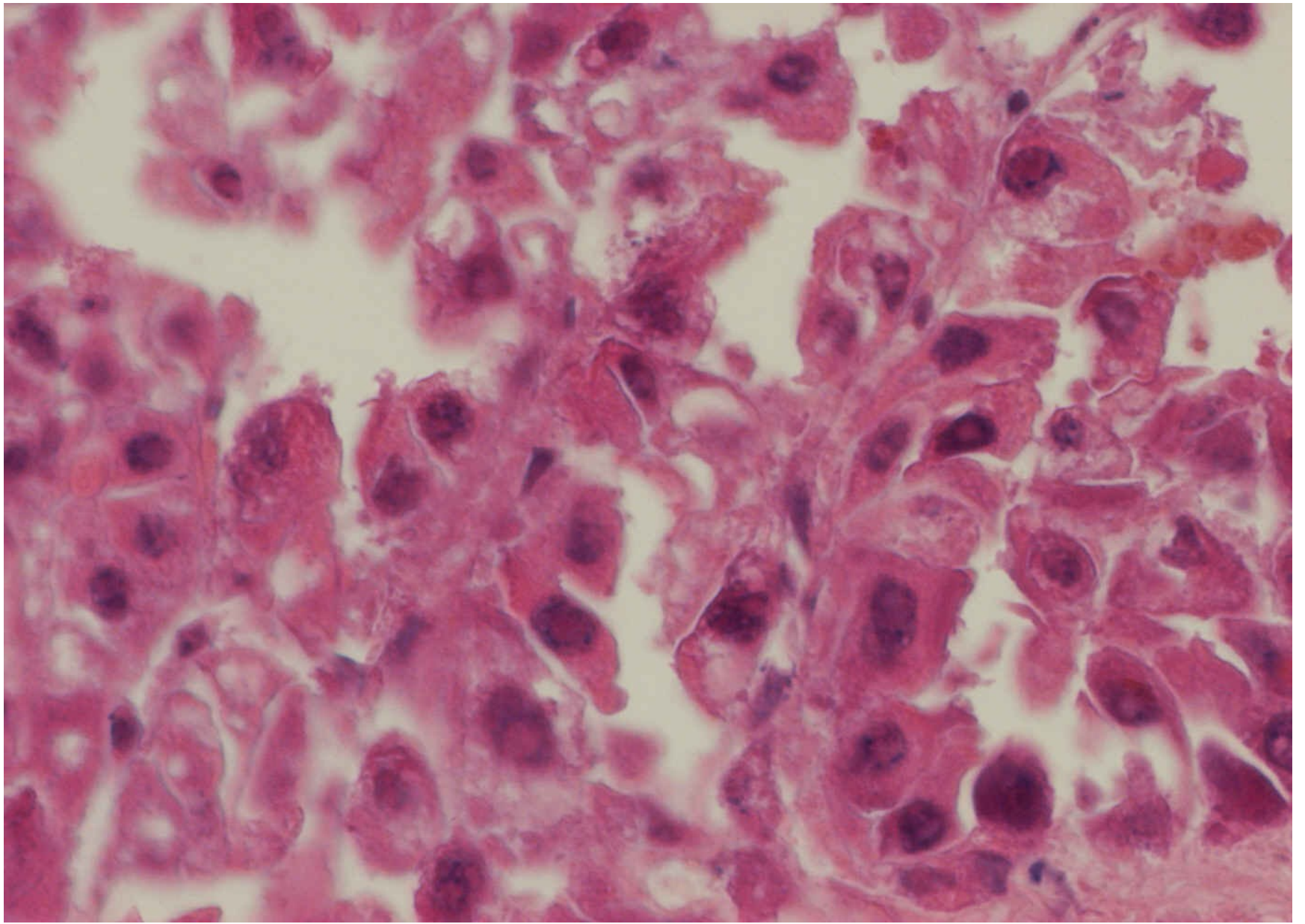
- benign
 - cavernous hemangioma
 - hepatocellular (liver cell) adenoma, cholangiocellular adenoma
 - malignant
 - hemangiosarcoma
 - hepatocellular carcinoma, cholangiocellular carcinoma
-



hepatocellular carcinoma

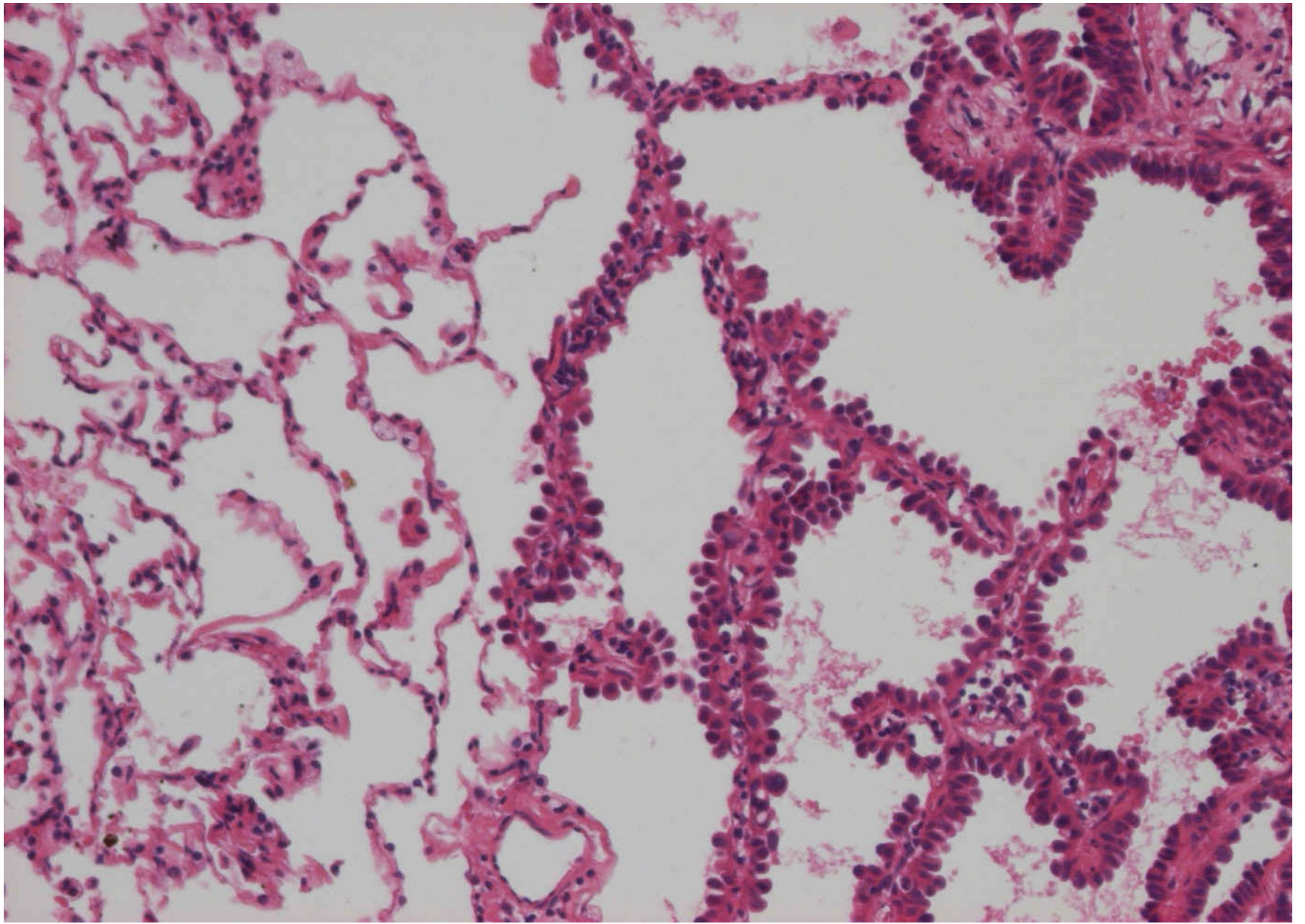


hepatocellular carcinoma – detail

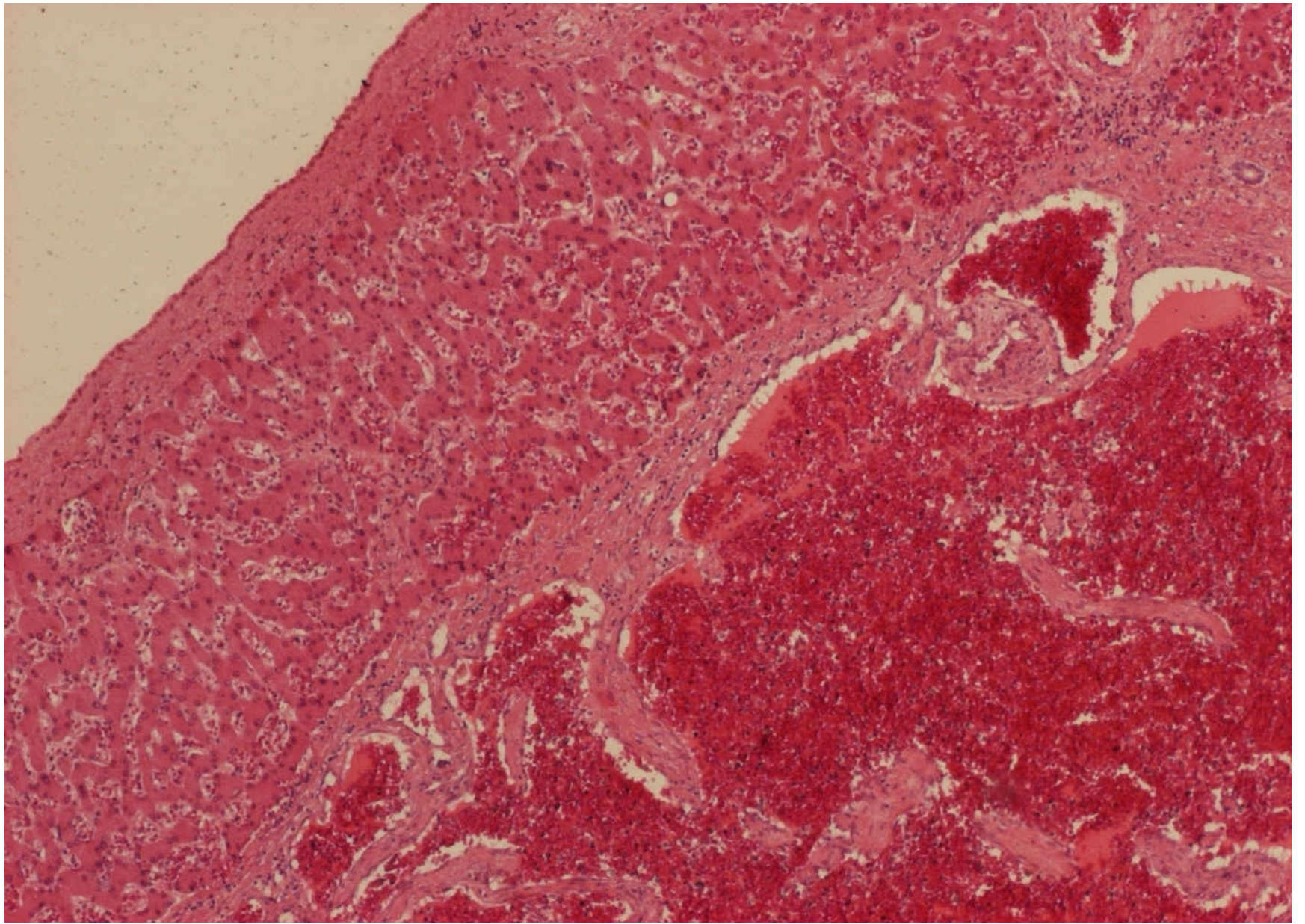


hepatocellular carcinoma – detail

-
- Repetition is the mother of learning!
-



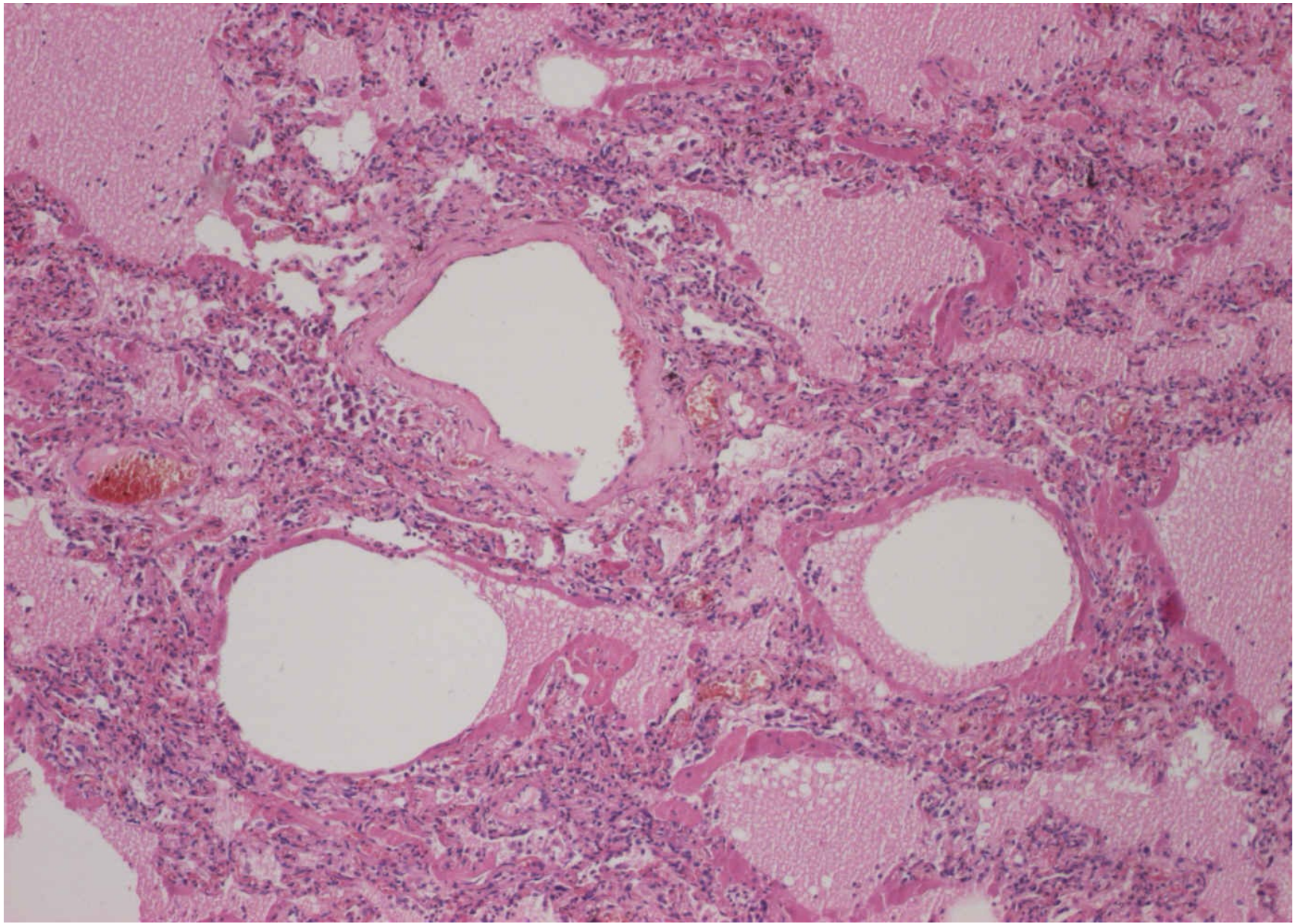
lungs – your diagnosis?



liver – your diagnosis?



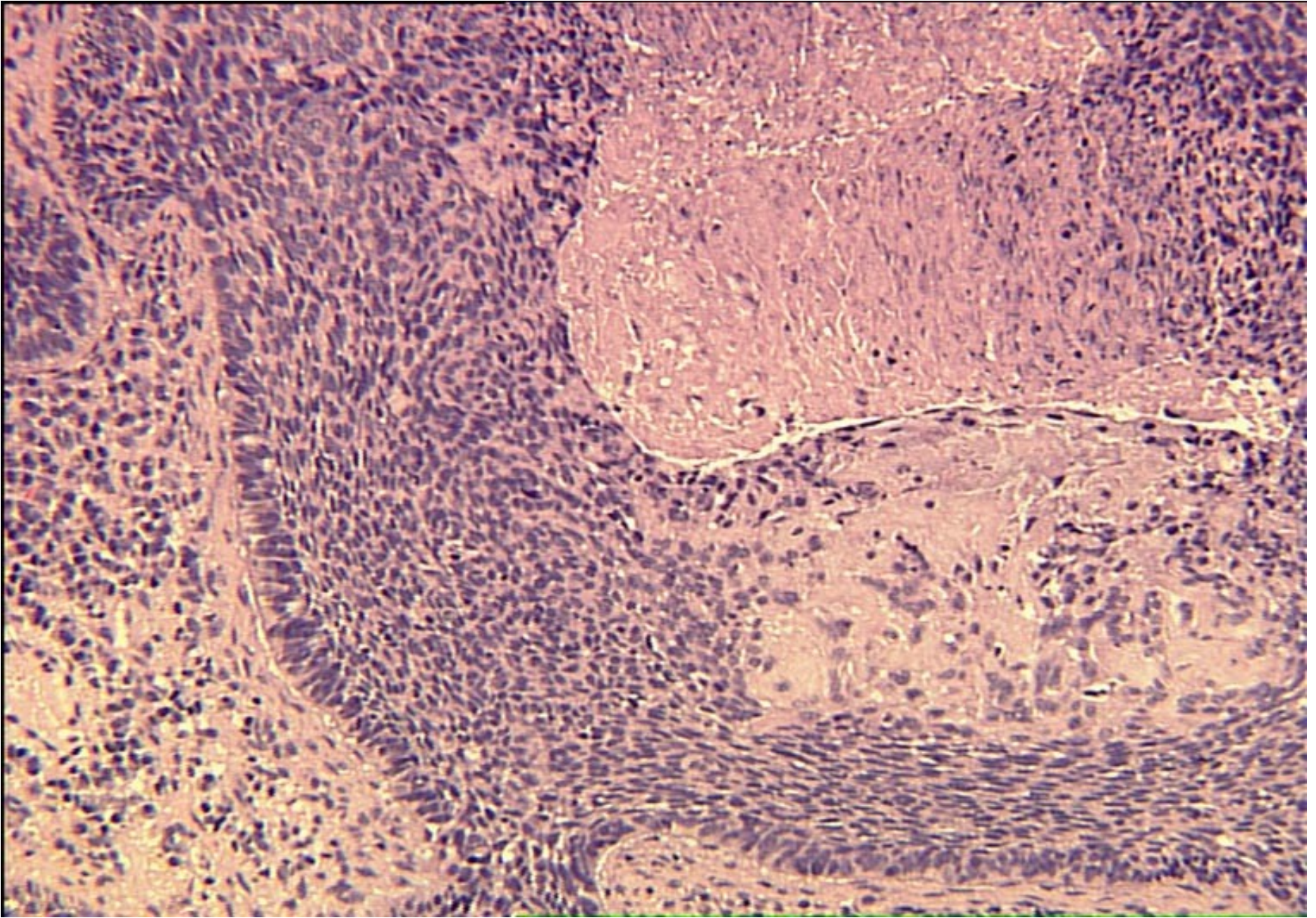
large intestine – your diagnosis?



lungs – your diagnosis?



small intestine – your diagnosis?



excision from the skin – your diagnosis?