

Liver National EQA Scheme

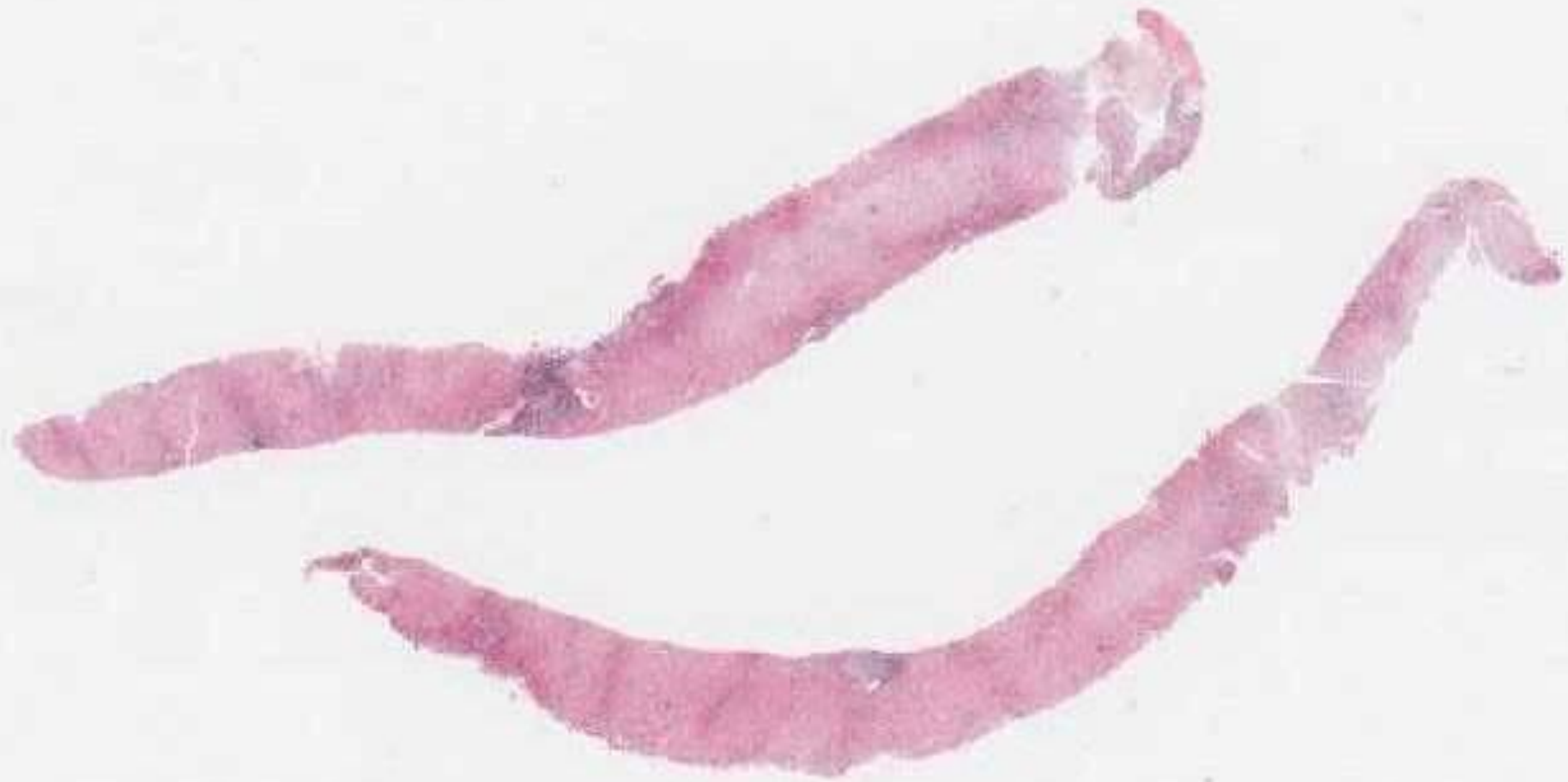
Circulation R
Newcastle July 2005

F53

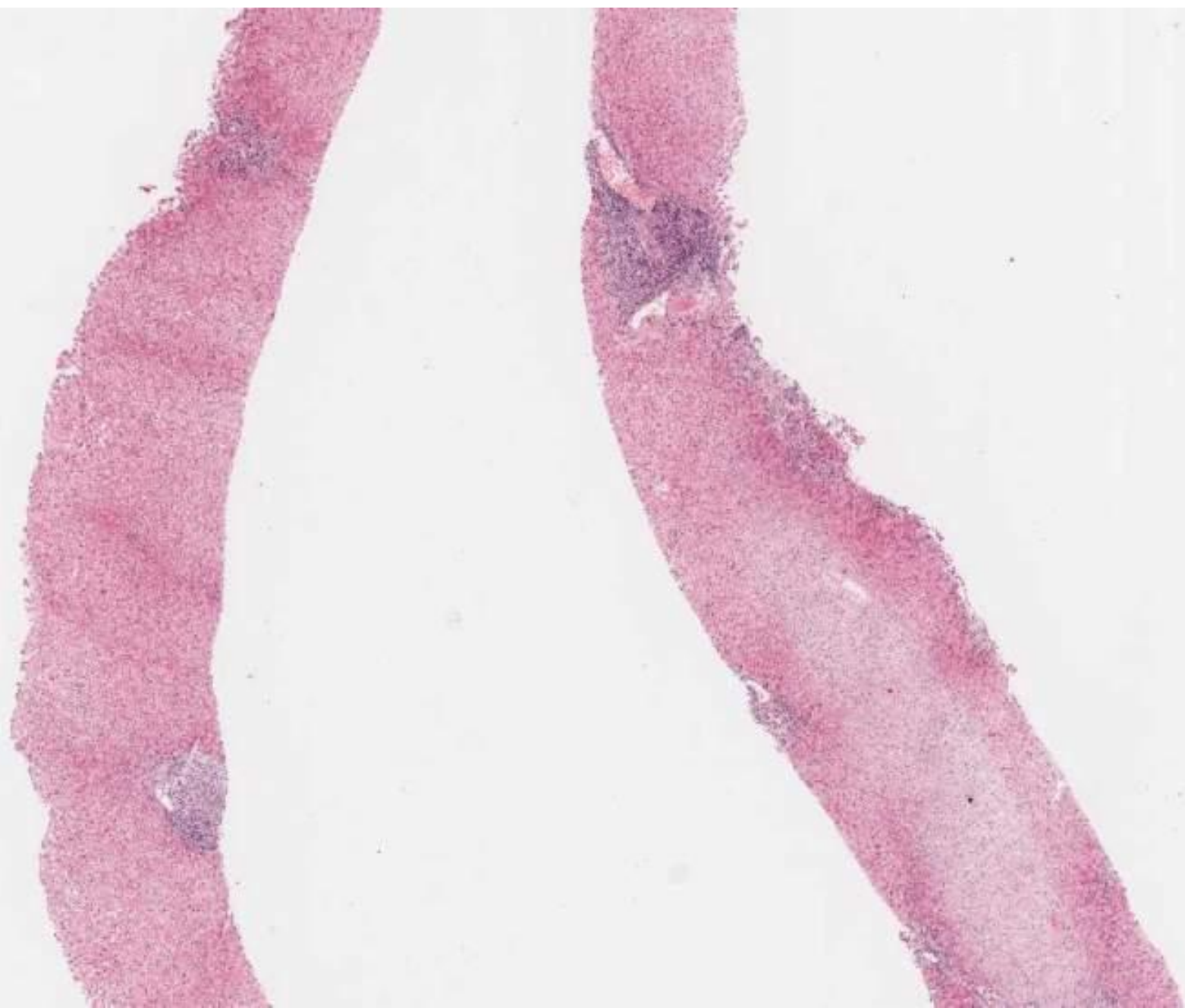
Abnormal liver function tests.

Positive anti-mitochondrial antibodies

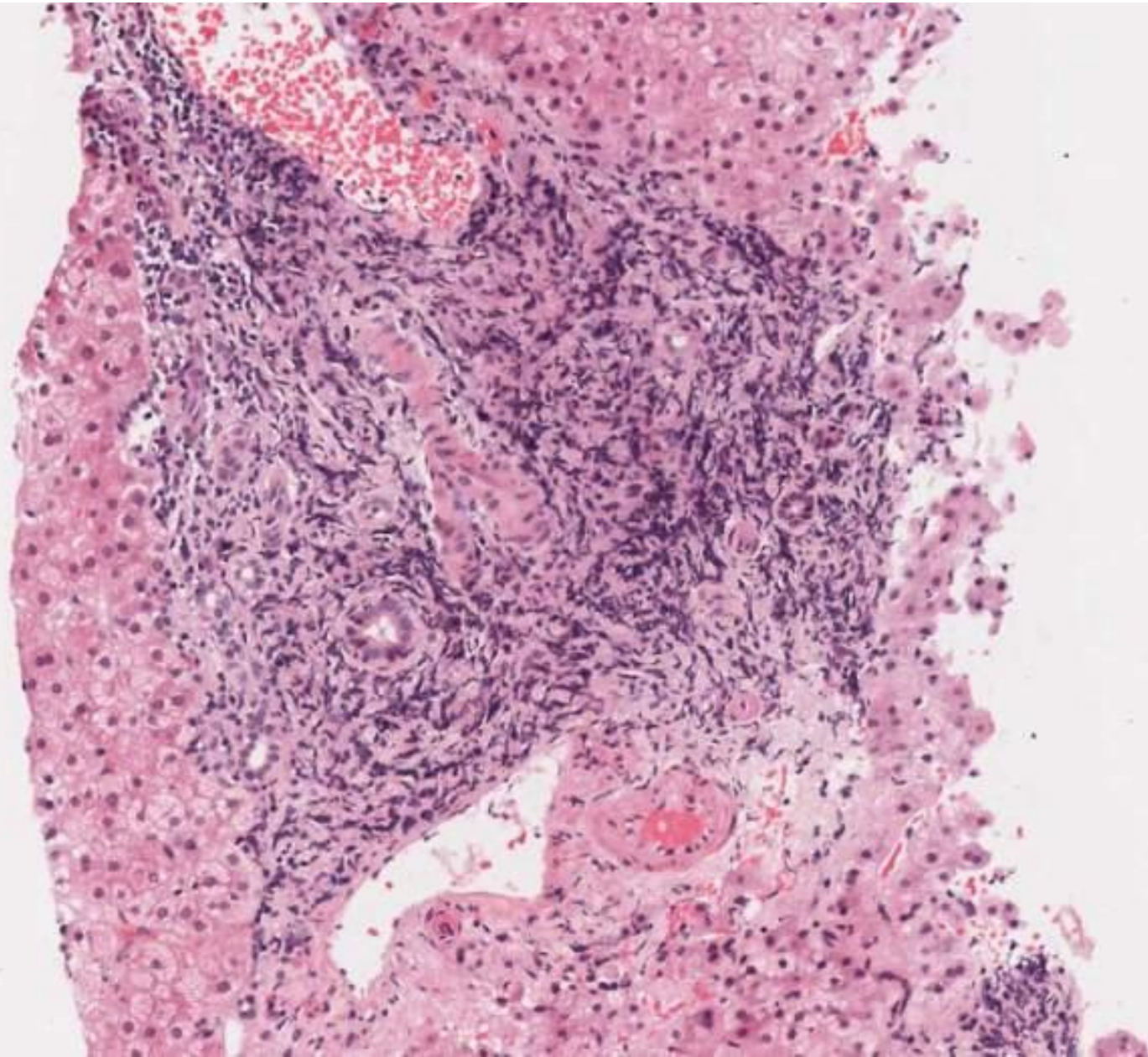
Case 218



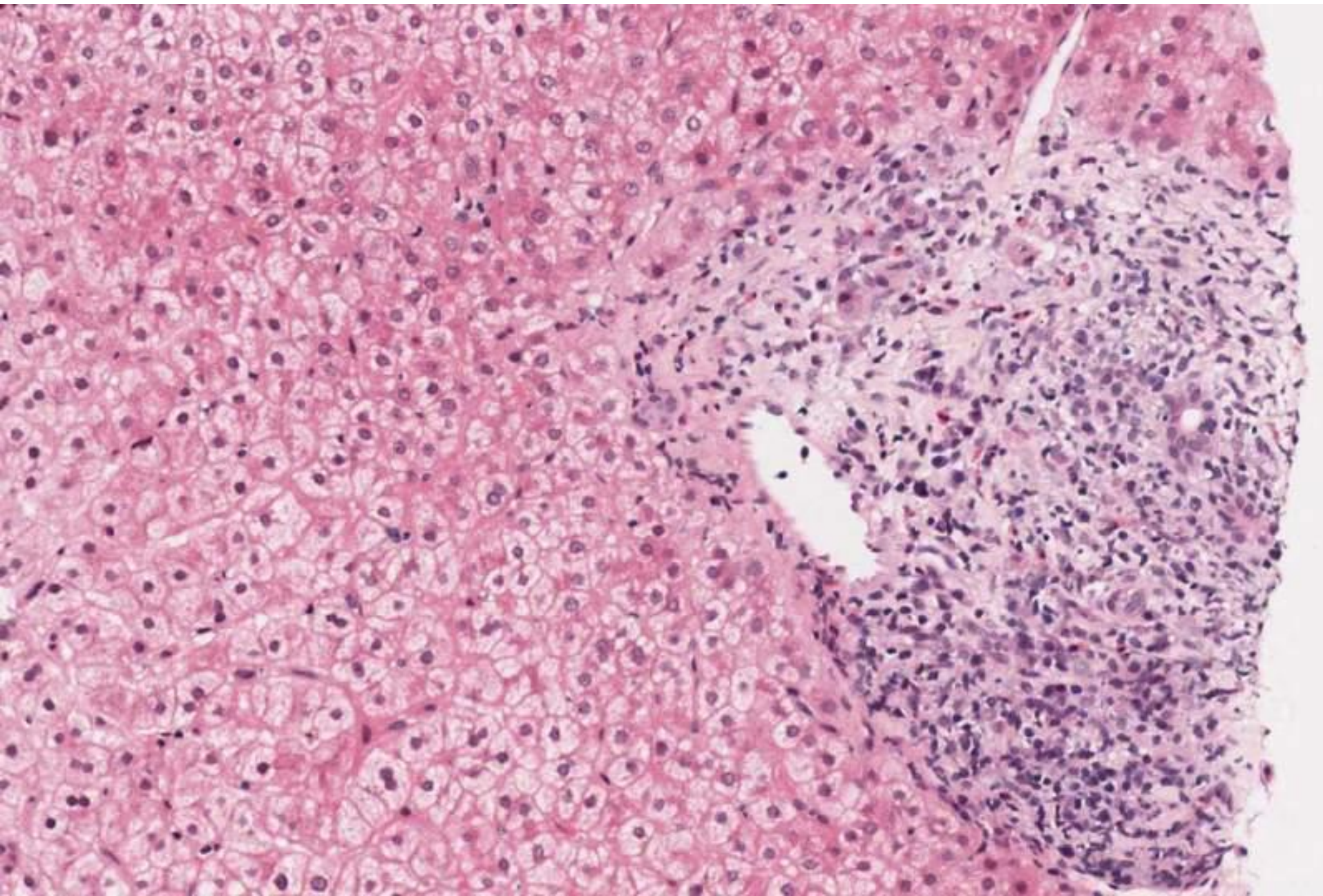
Case 218



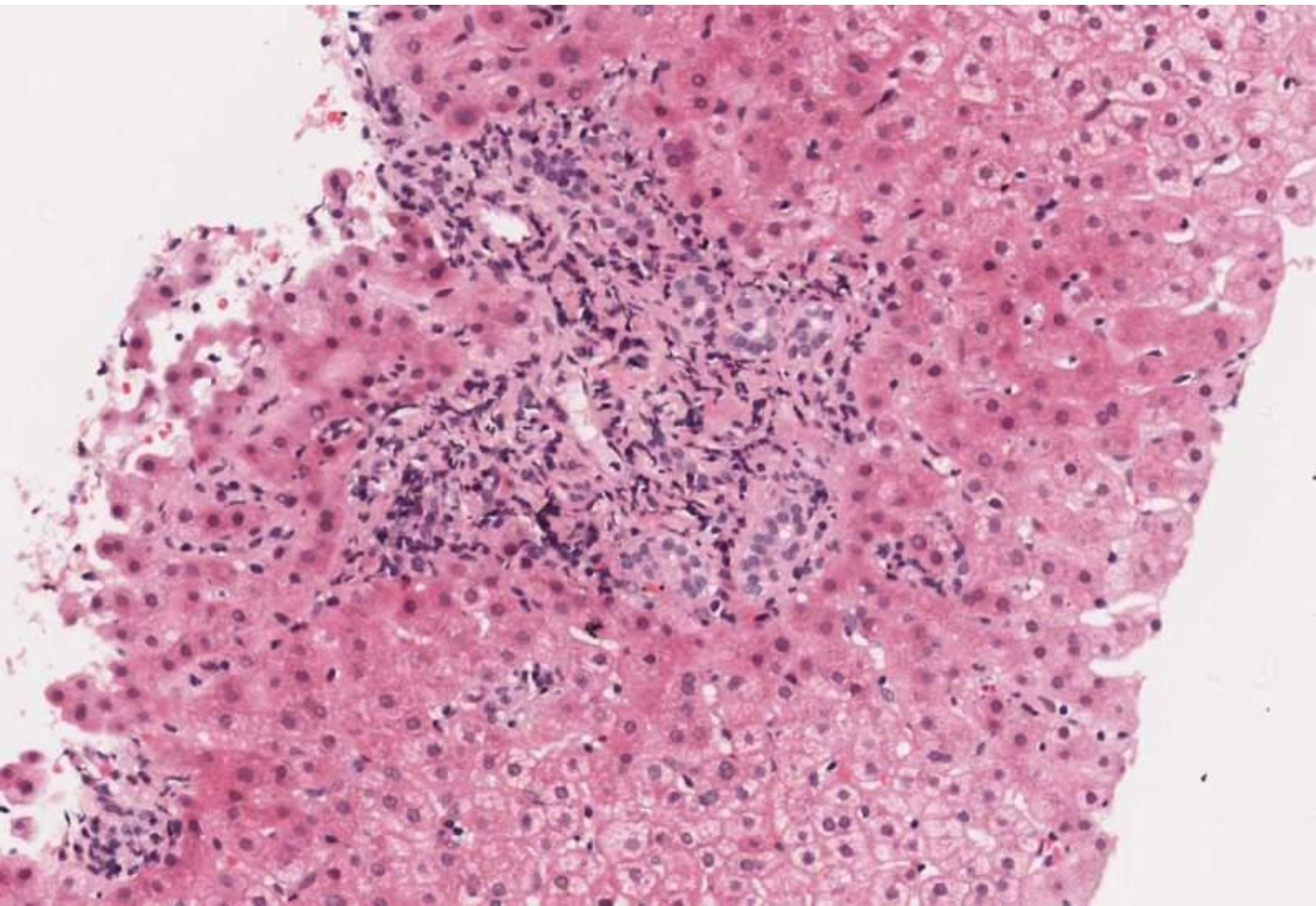
Case 218



Case 218



Case 218



Case 218

Results:

24 PBC

30 consistent with PBC

1 PBC ? overlap syndrome

1 chronic hepatitis, favours PBC

1 chronic hepatitis, not typical of PBC

Comments;

17 needs orcein

4 comment on ? overlap

2 comment on eosinophils, ? Drugs

Case 218

Discussion

All diagnoses accepted.

Follow up (Dr Shousha) – PBC; (granulomas on original sections)

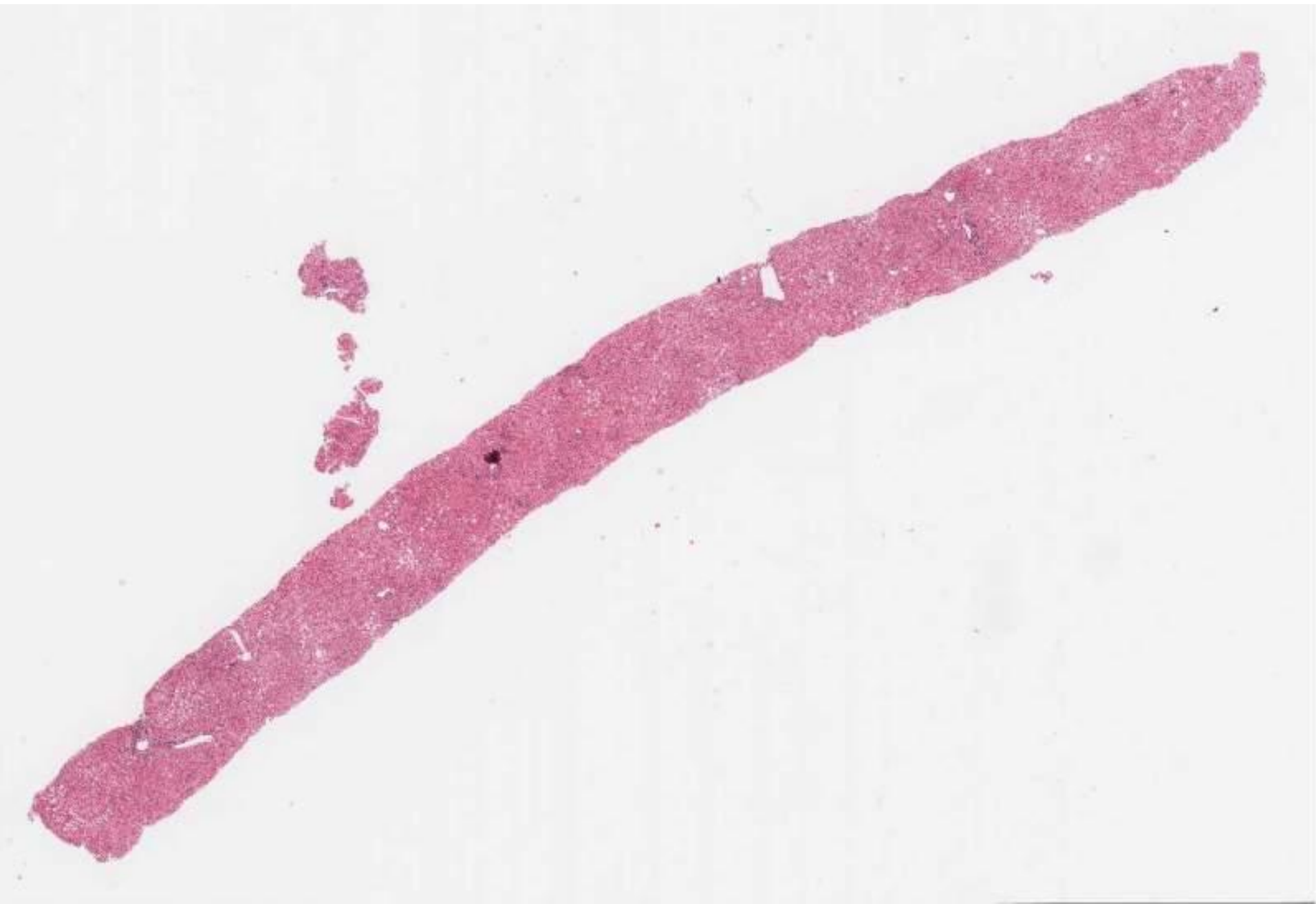
Comments: The combination of mitochondrial antibodies with histology consistent with chronic biliary disease establishes the diagnosis of primary biliary cirrhosis. In a patient with mitochondrial antibodies and cholestatic liver function tests, a biopsy is not necessarily performed, as it does not contribute to clinical management.

Case 219

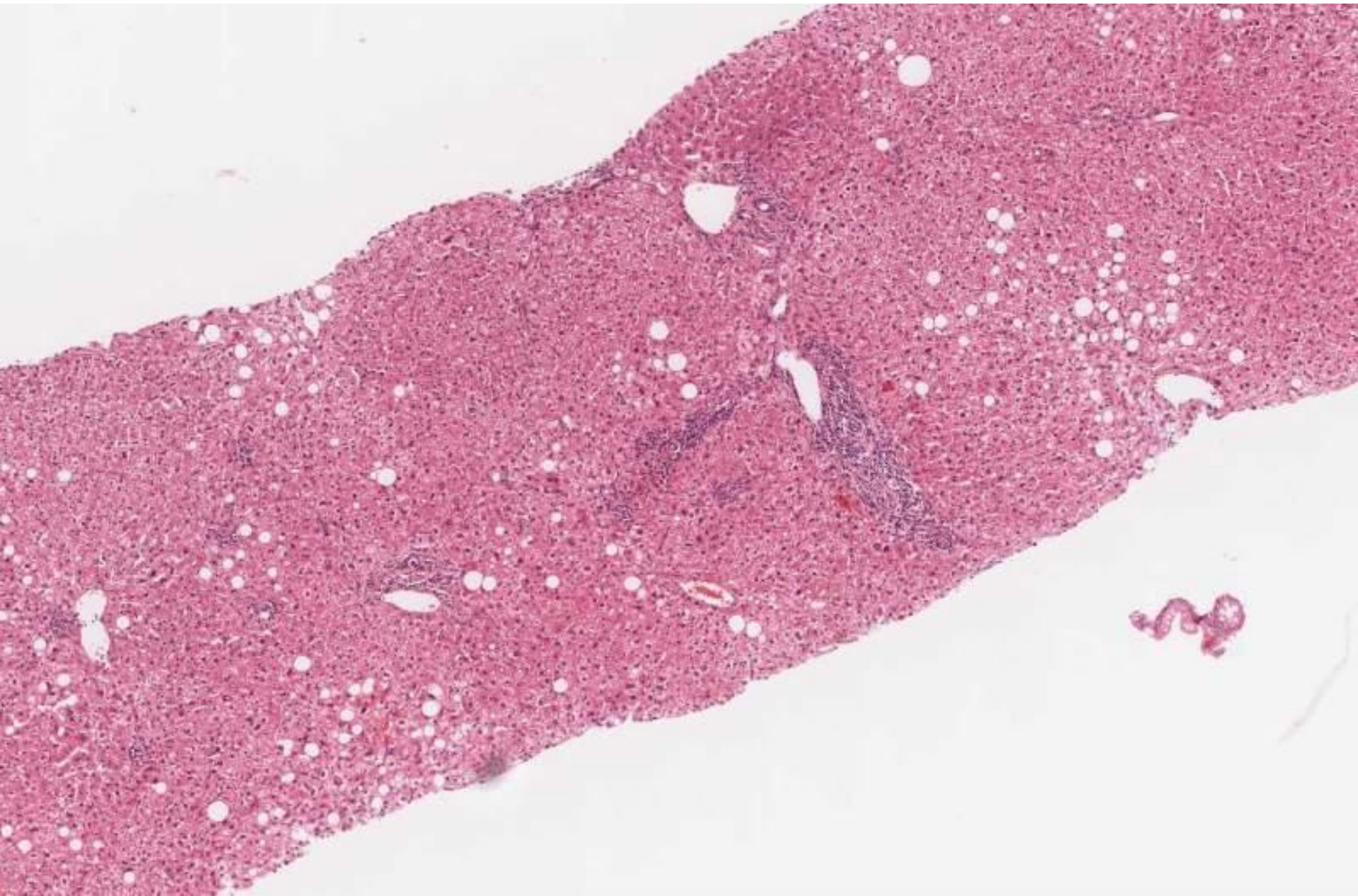
43M

ALT 1.5x normal, BMI 27

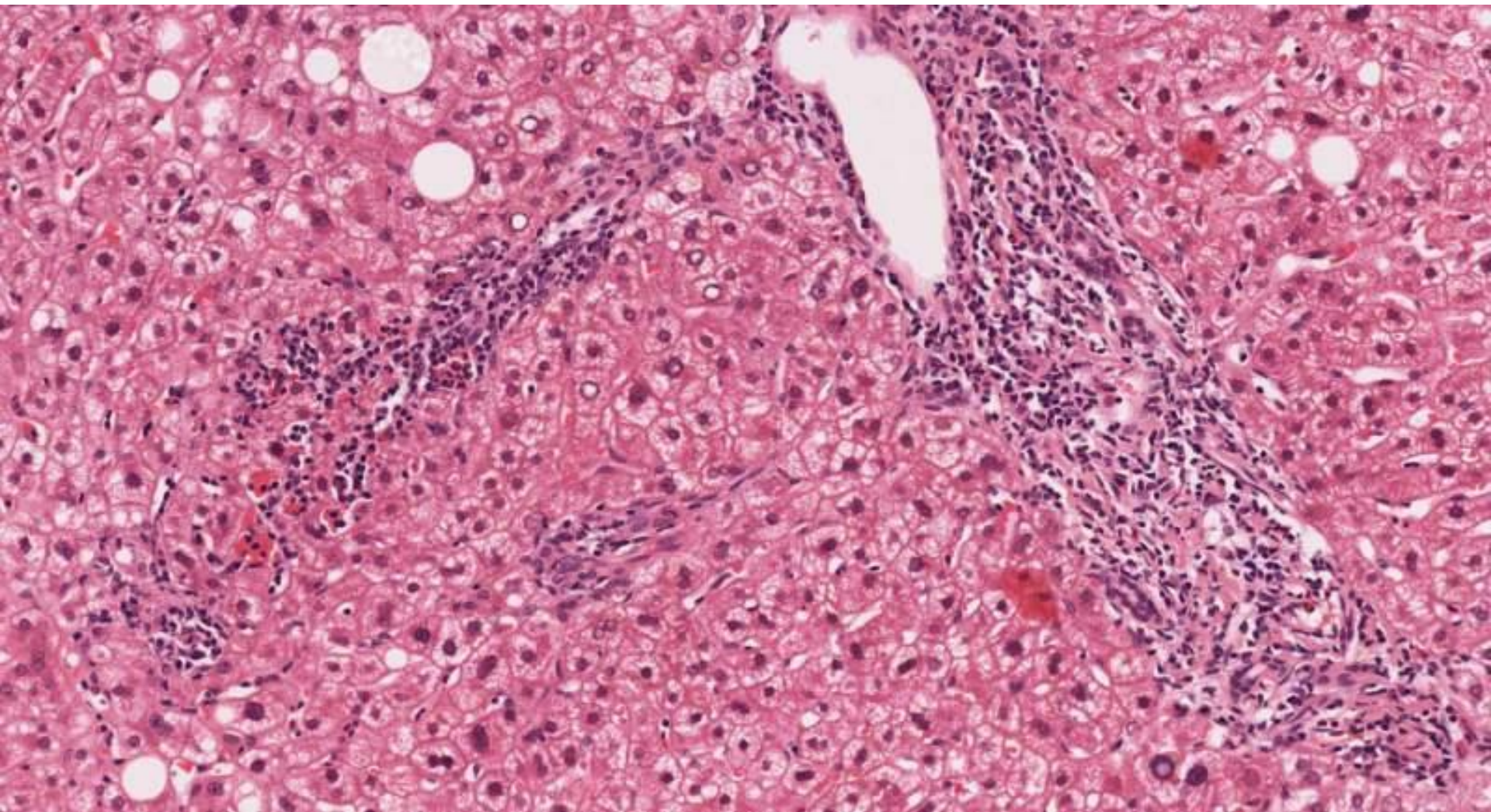
Case 219



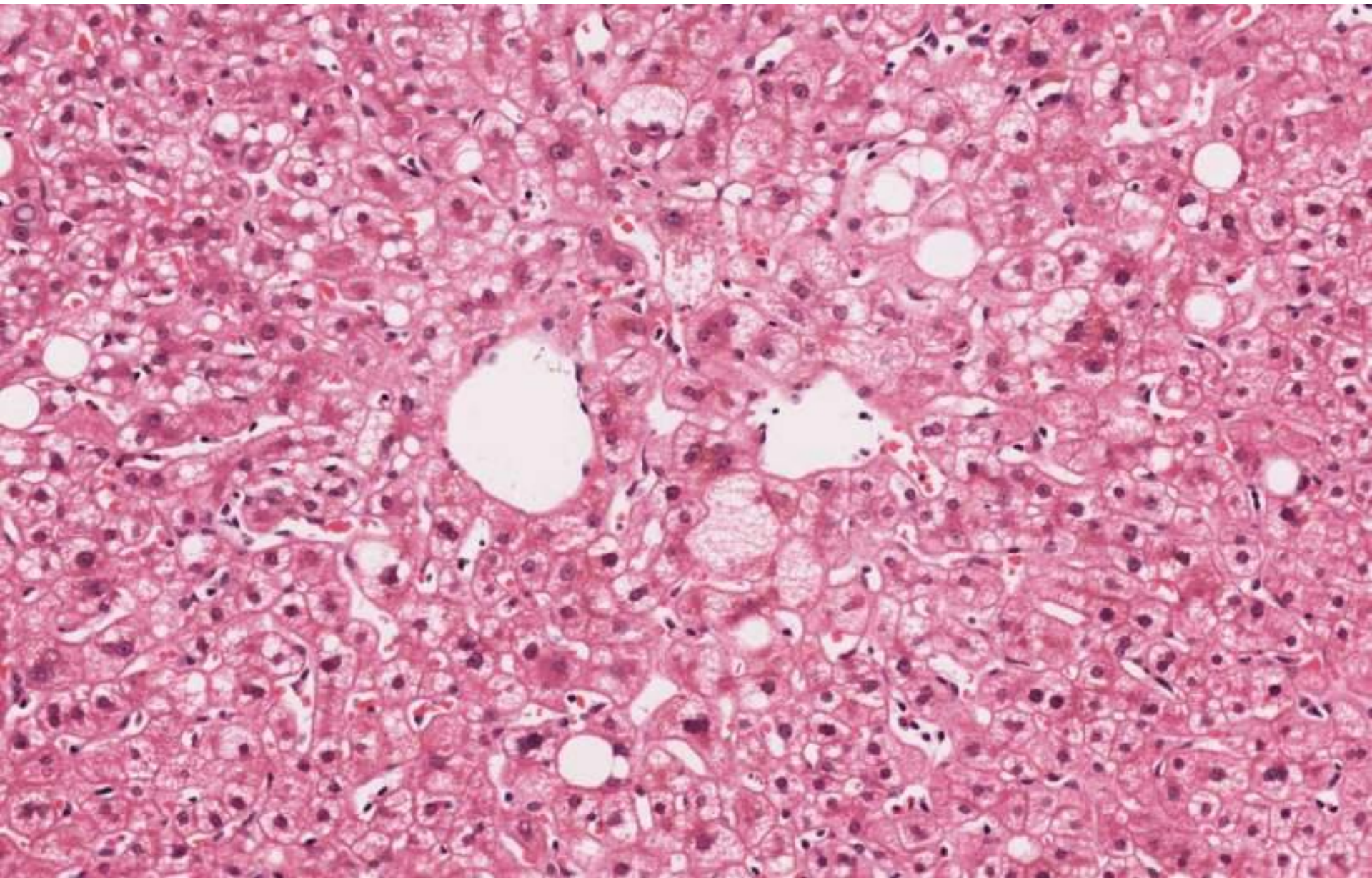
Case 219



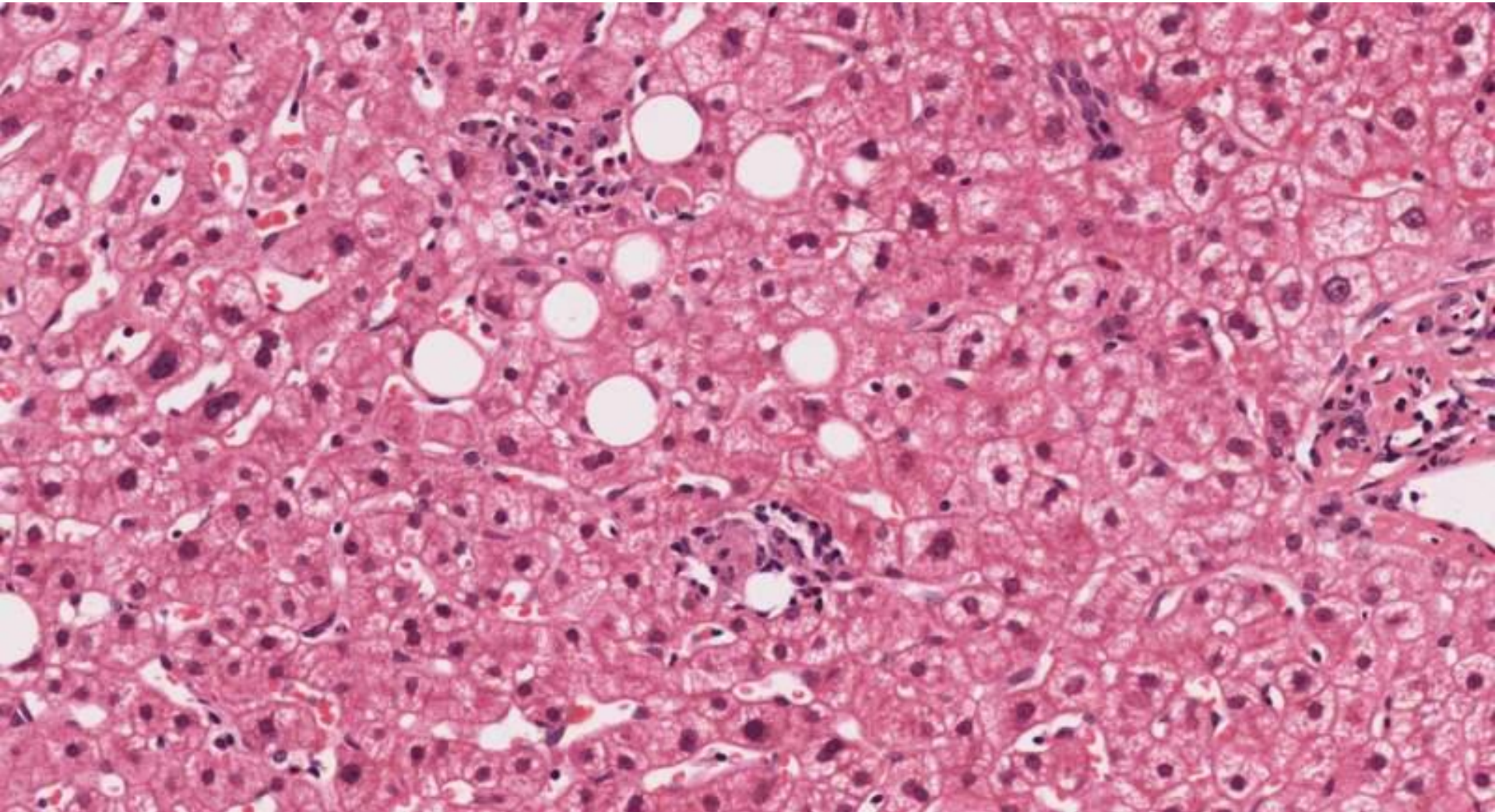
Case 219



Case 219



Case 219



Case 219

Results:

36 steatohepatitis

7 steatosis

3 minimal steatohepatitis

4 NAFLD

1 steatosis/steatohepatitis

1 ring granulomas, ? Q fever etc.

1 mild fatty change and lobular inflammation ? drug reaction

1 steatohepatitis with eosinophils ? Drugs

1 steatohepatitis (Ishak 1) nil else

Case 219

Comments:

23 needs alcohol history

7 VG for fibrosis

9 ? drugs in view of eos (5) or inflammation (4)

Follow up (Dr Kaye) – no significant alcoholic history and no drugs; treated as NAFLD

Case 219

Discussion:

Two diagnoses rejected – fibrin ring granulomas, ?Q fever etc and mild fatty change with lobular inflammation, ?drugs.

Follow up (Dr Kaye) – no significant alcoholic history and no drugs; treated as NAFLD

Comments:

Steatohepatitis – it was not possible to determine aetiology because of insufficient clinical information. The optimum answer would be ‘steatohepatitis, requires alcohol history’. The diagnosis NALFD may be inappropriate since without that history this case cannot be classified as non-alcoholic; however, glycogenated nuclei were present, and these show a much stronger association with non-alcoholic steatohepatitis than alcoholic liver disease.

/contd.

Case 219

Comments (contd)

Illustrating photomicrographs showed features of steatohepatitis, but these may not have been present on all of these slides circulated. (It was suggested that those claiming the diagnostic features were not on their slides could have that particular slide reviewed but this was felt to be impractical). Features of steatohepatitis were present on the illustrated section.

The distinction between steatosis and steatohepatitis was felt to be clinically important as follow up and future management may be different. However, both disease patterns may be seen within the same biopsy, and perhaps in this case in different levels of the same biopsy.

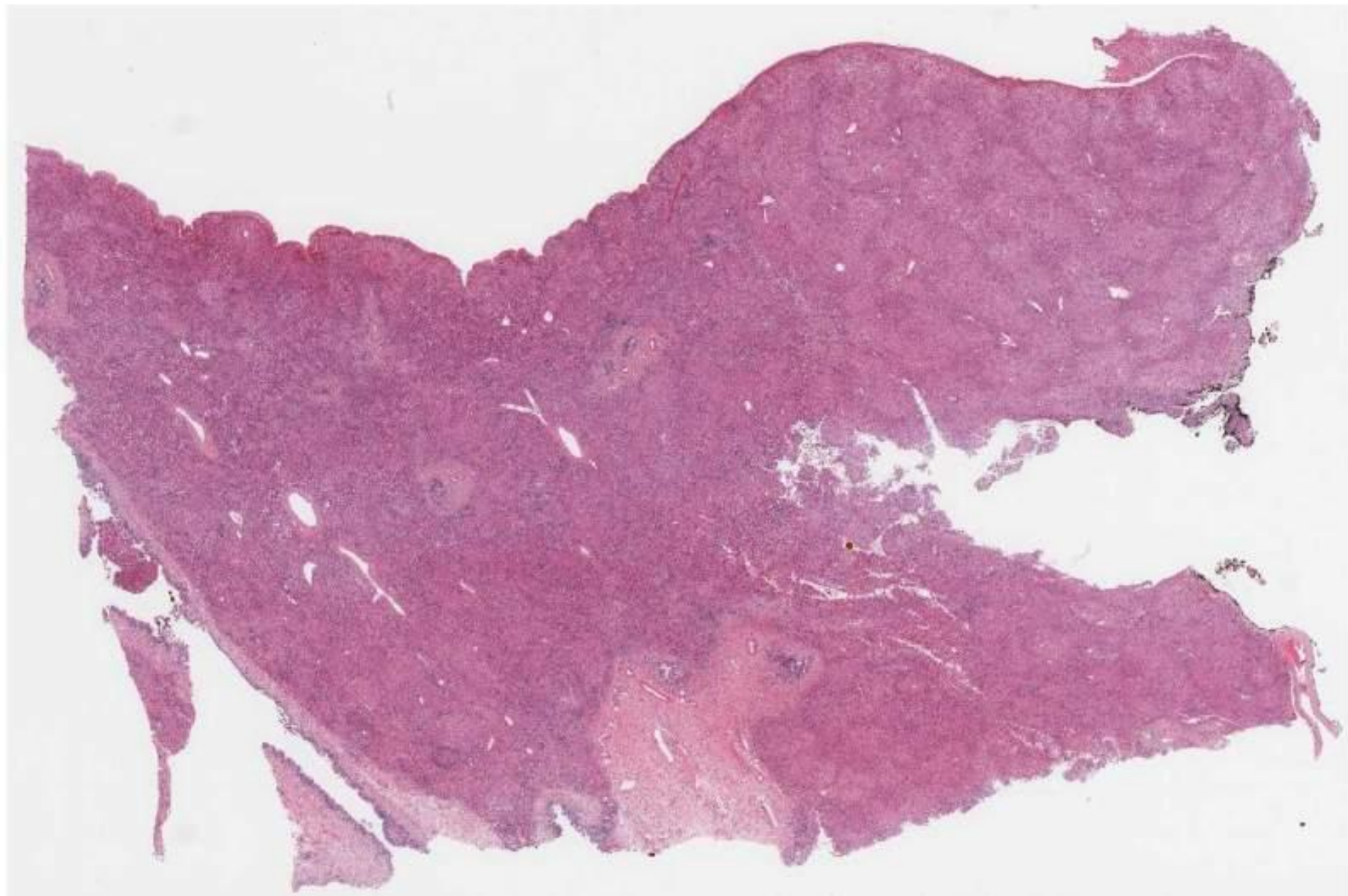
Case 220

60M

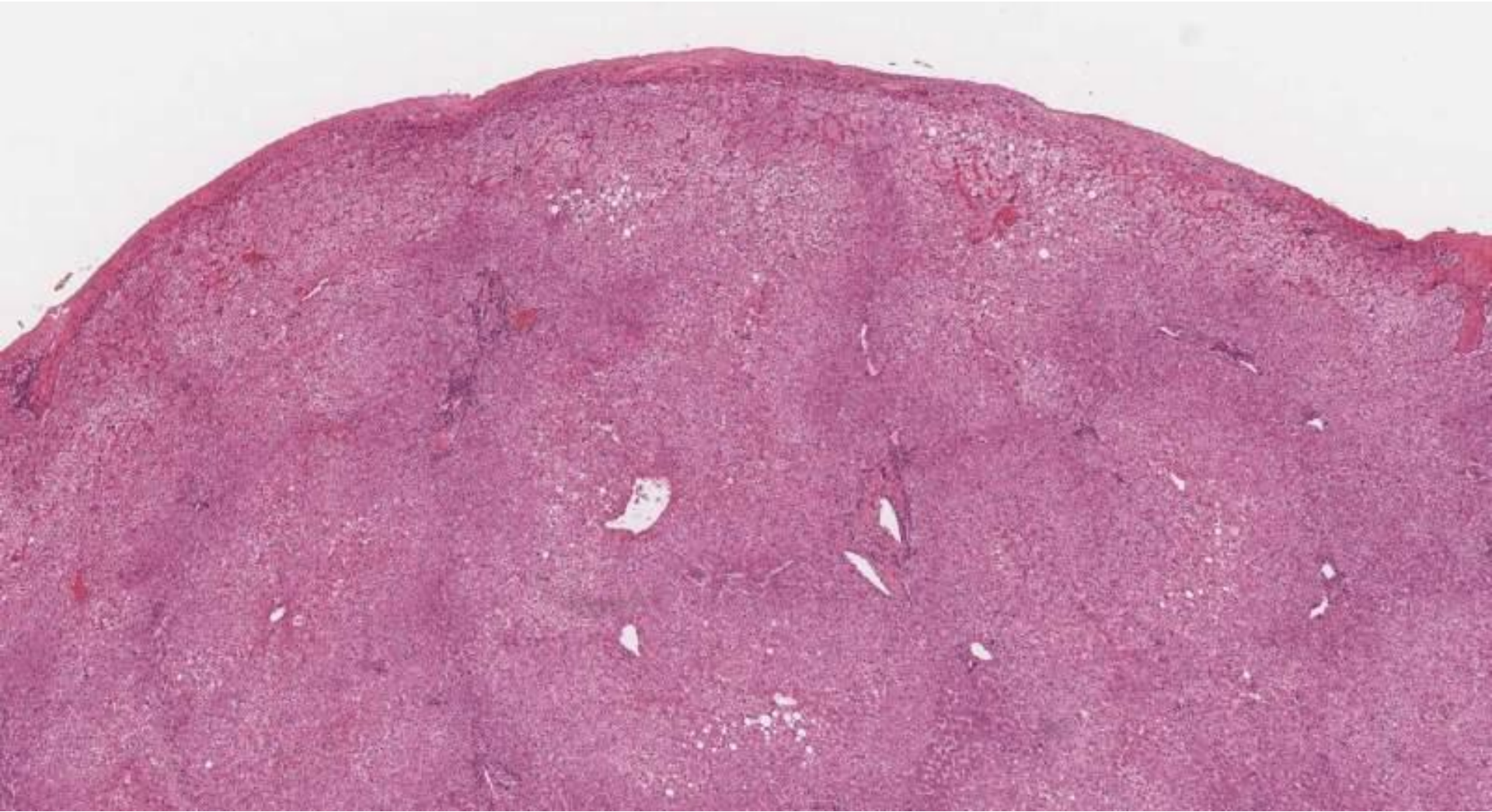
Laparoscopic cholecystectomy for gall stones 2 years ago
Recurrent episodes of septicaemia/cholangitis for last 9 months.
ERCP showed a stricture in the left main hepatic duct and a cyst nearby (2cm diameter). At surgery the cyst was found to contain small gall stones. Special stains not contributory.

Specimen: liver 13x12x4cm, containing a 2cm cyst that is full of bile debris and small stones. Section of liver adjacent to cyst.

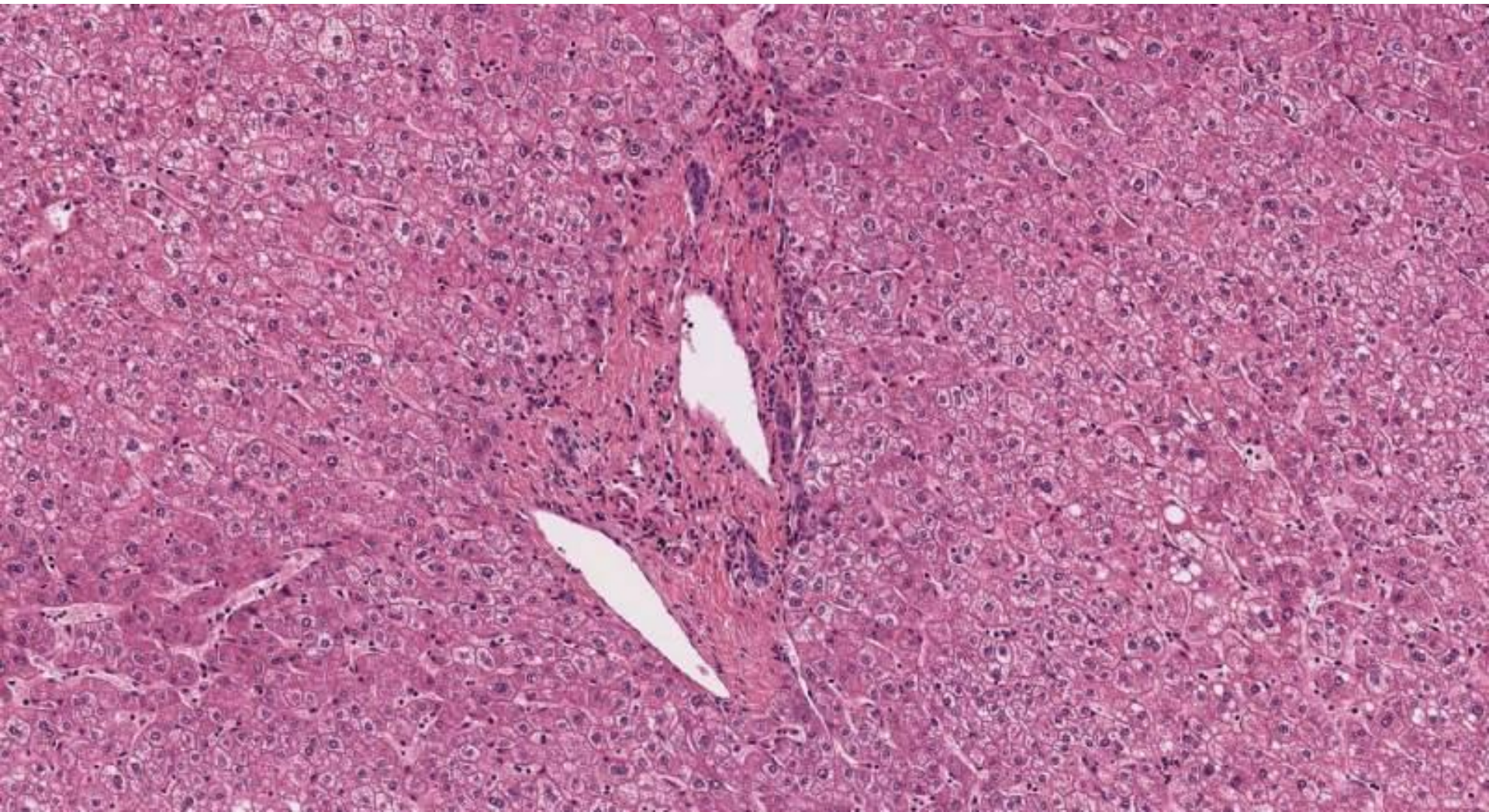
Case 220



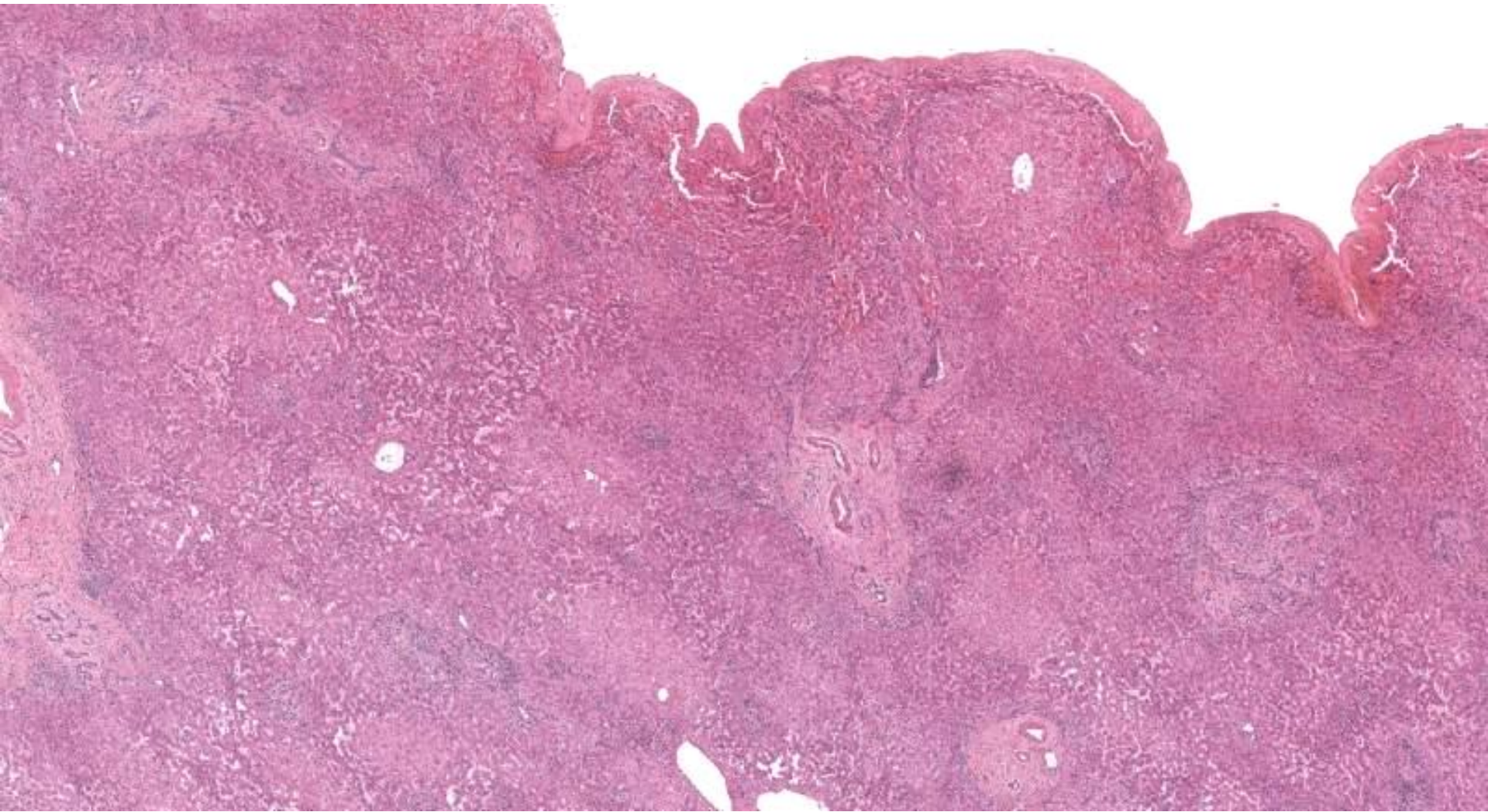
Case 220



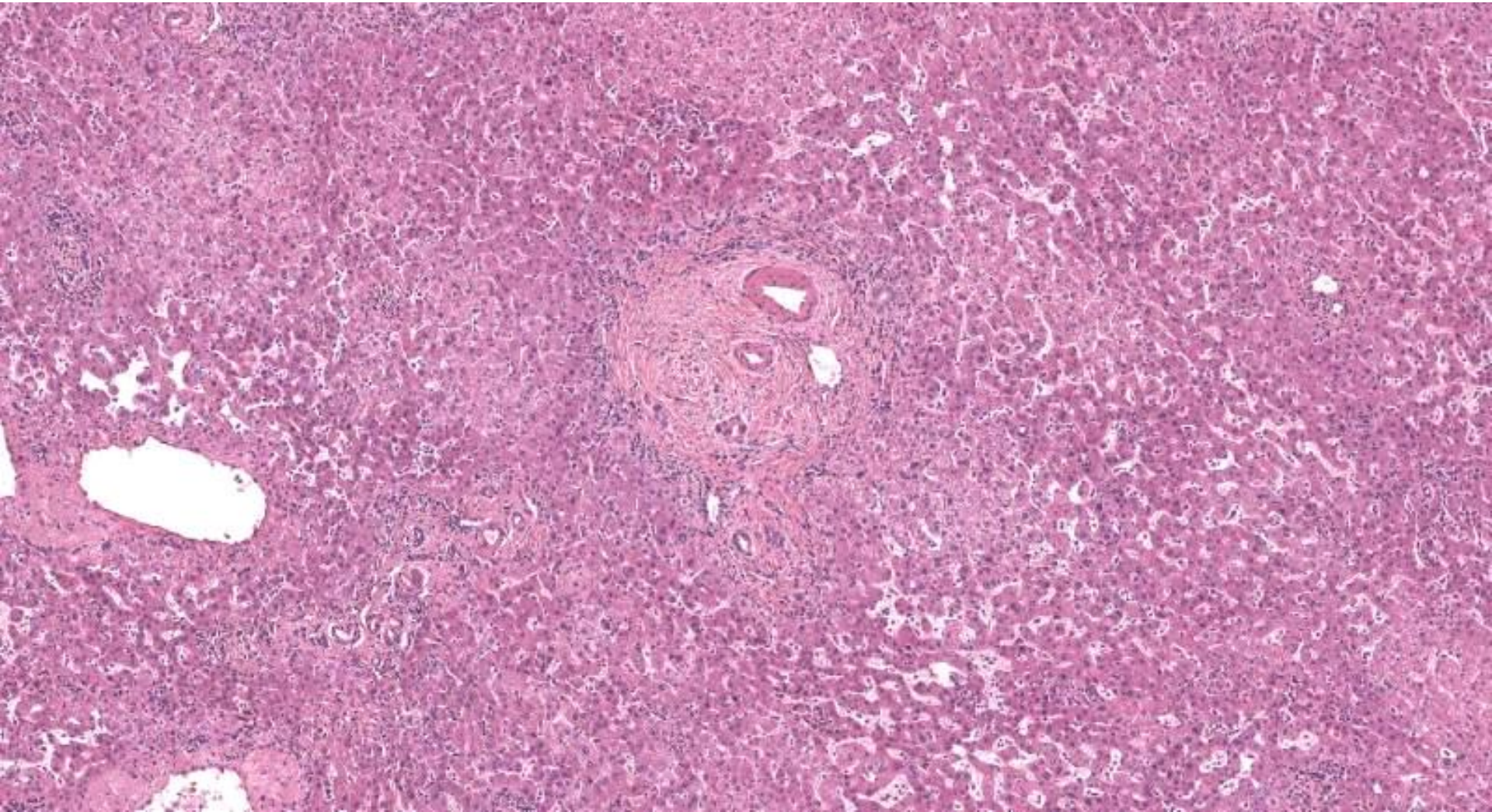
Case 220



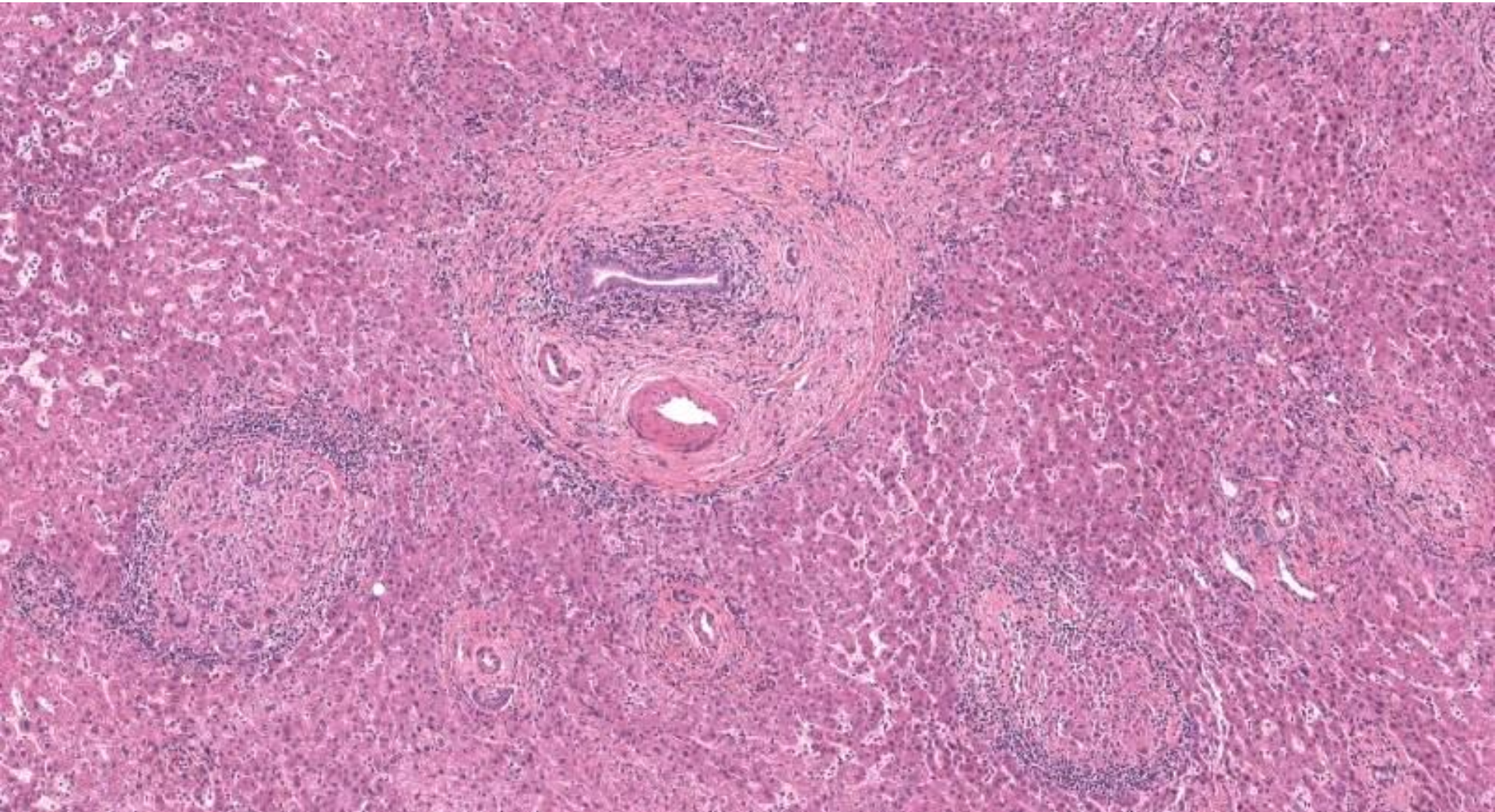
Case 220



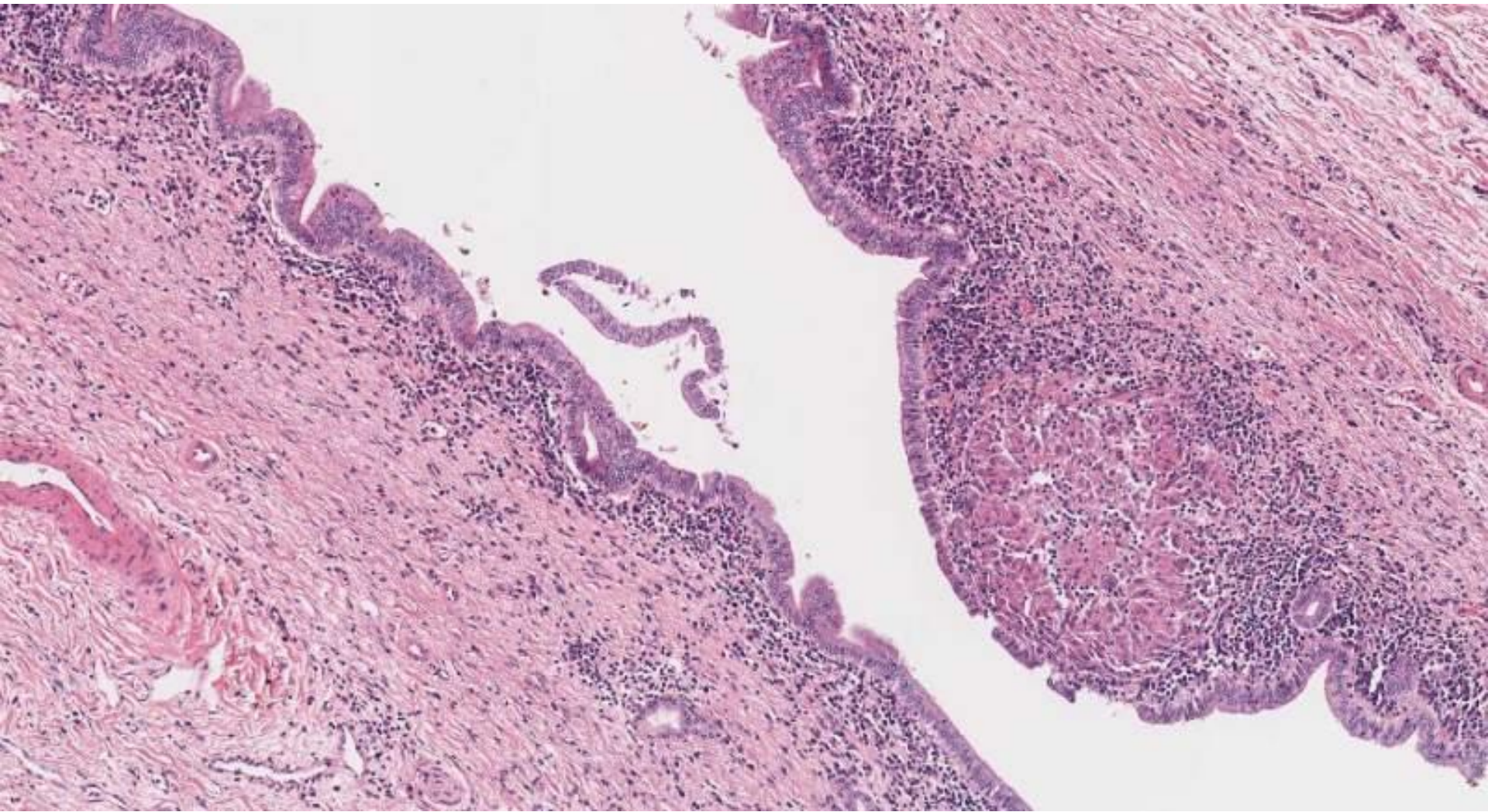
Case 220



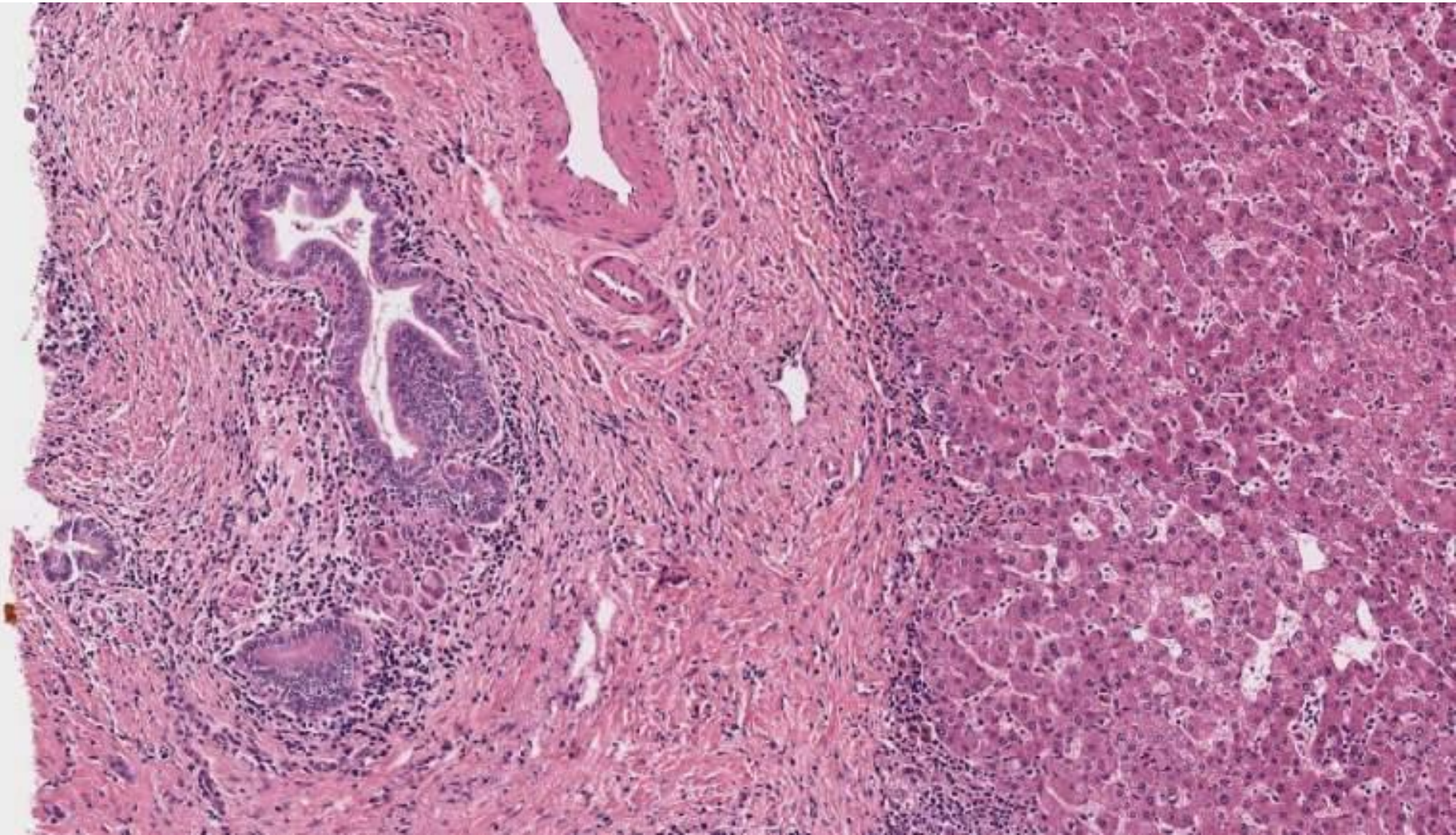
Case 220



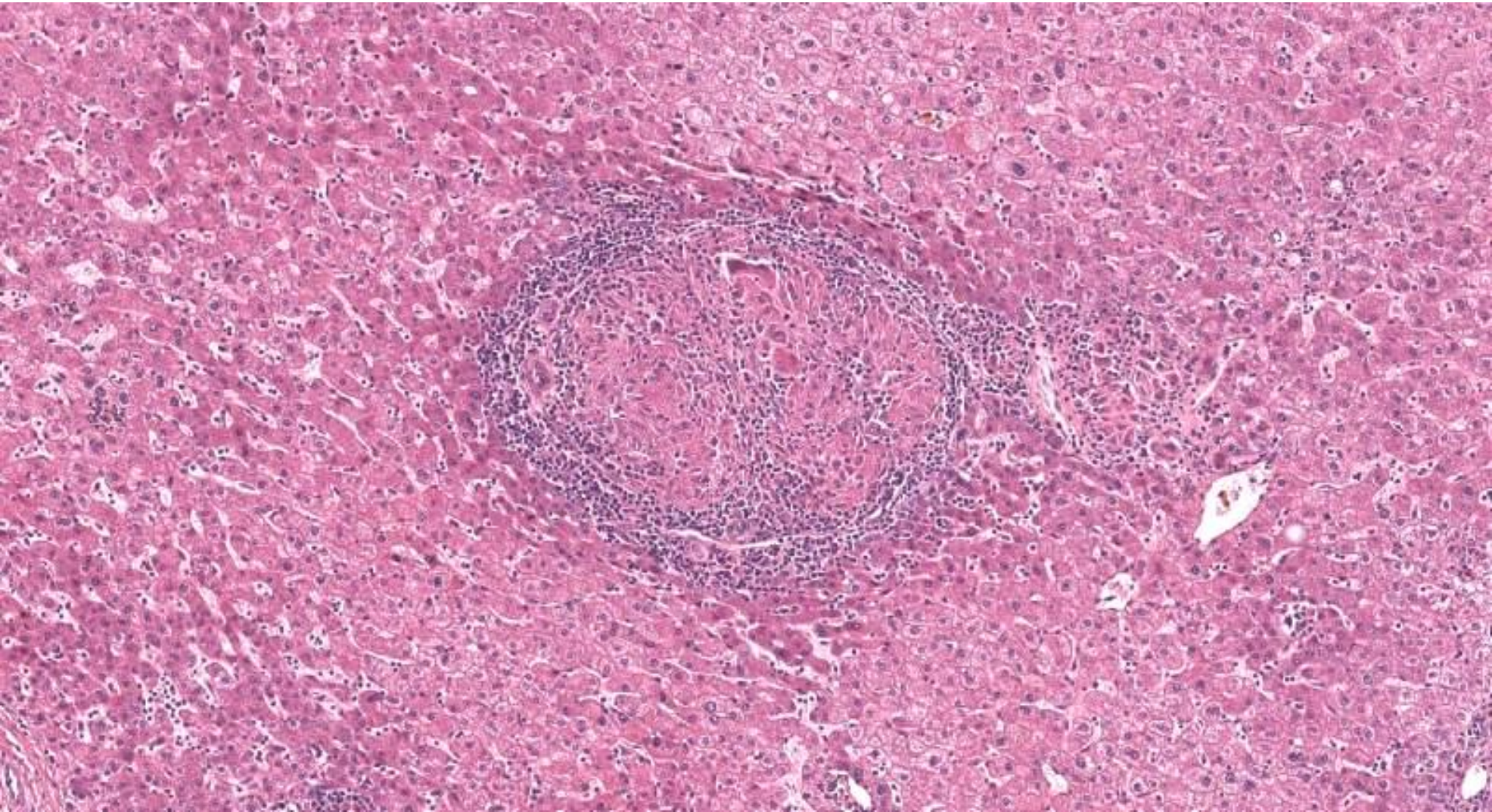
Case 220



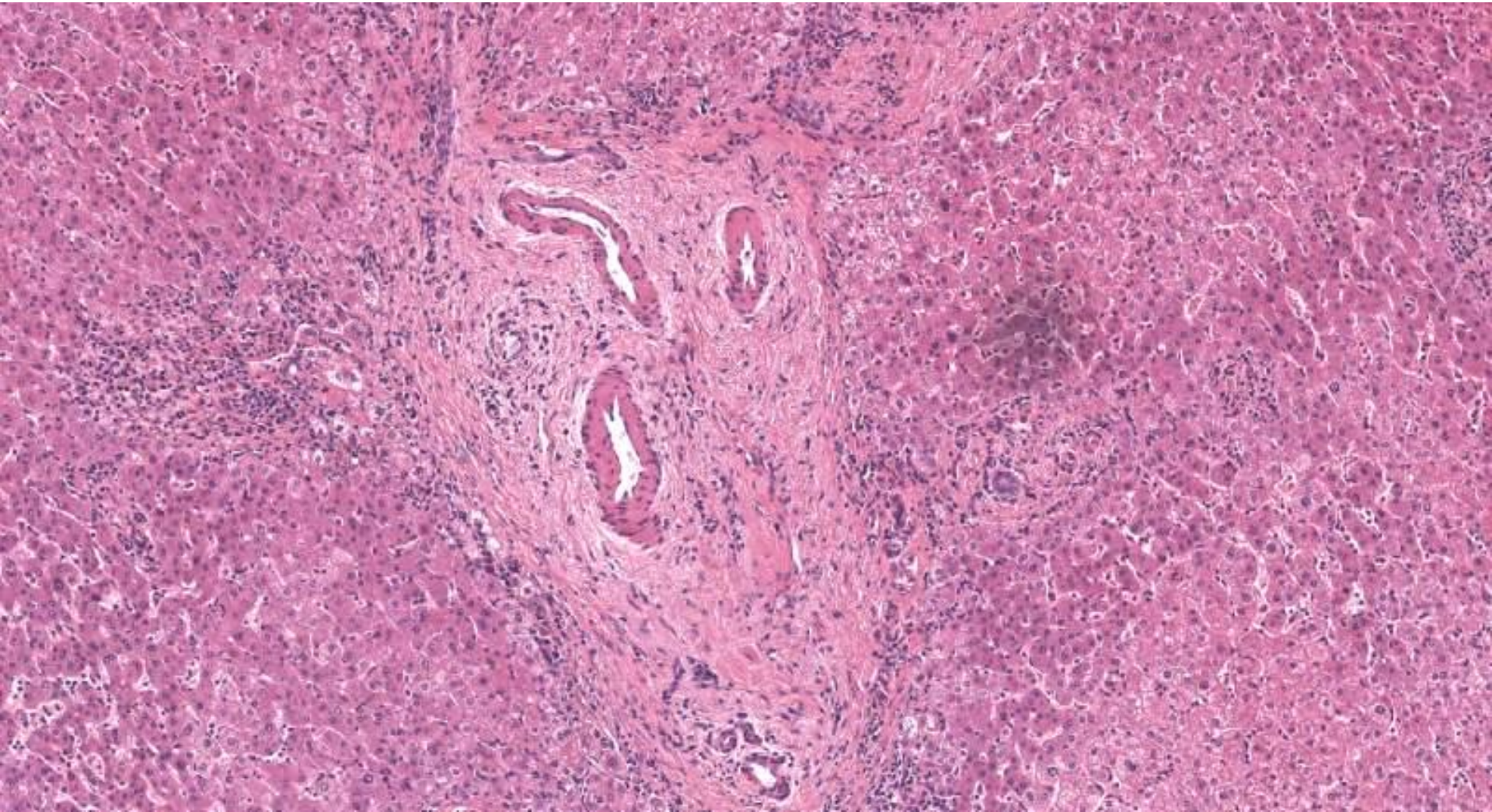
Case 220



Case 220



Case 220



Case 220

Results:

- 34 cholangitis with granulomas, due to stricture/stones/surgery
- 1 cholangitis due to gallstones (granulomas not mentioned)
- 2 granulomatous hepatitis

- 1 chronic biliary disease with granulomas
- 3 sclerosing cholangitis with granulomas
- 5 ?PSC
- 4 ?PBC or sarcoid

- 2 choledochal cyst with granulomas
- 2 biliary cystadenoma with granulomas
- 2 ? localised Caroli's
- 1 didn't see slide

Case 220

comments:

exclude sarcoid 5

needs AMA 5

Case 220

Discussion

Accept all except granulomatous hepatitis.

Comments:

Accepted results should indicate that this is a granulomatous chronic biliary disease. Further clinical information from Professor Williams – details of original gall bladder surgery not available; it is not recorded whether or not the left duct was damaged during this procedure.

It would be uncommon for cystic dilatation of the duct to occur just as a result of surgical stricture, and there may have been a pre-existing cyst in the left lobe that has become inflamed consequent on surgical stricturing.

A granulomatous response to bile escaping through the damaged basement membrane occurs in PSC, and in other causes of bile duct injury, and is presumably the explanation in this case. Detailed scrutiny of the notes shows no clinical evidence of primary biliary cirrhosis or sarcoid. The original diagnosis is of granulomatous cholangitis following surgical stricture.

Case 221

M70

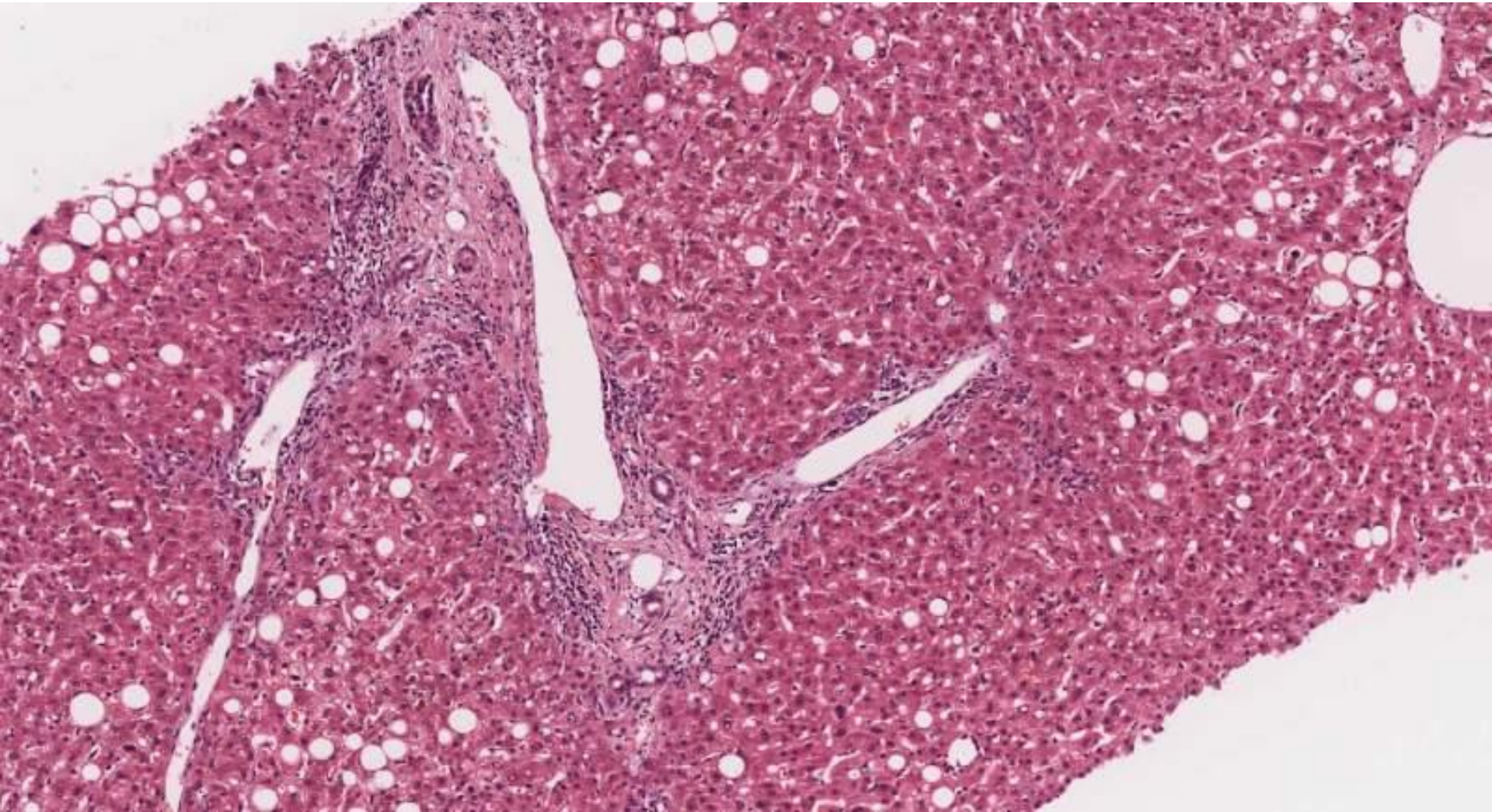
Cholestatic jaundice (bil 221) Normal ERCP. Negative liver screen.

On Atorvastatin, ? drug reaction, special stains grade 2 iron.

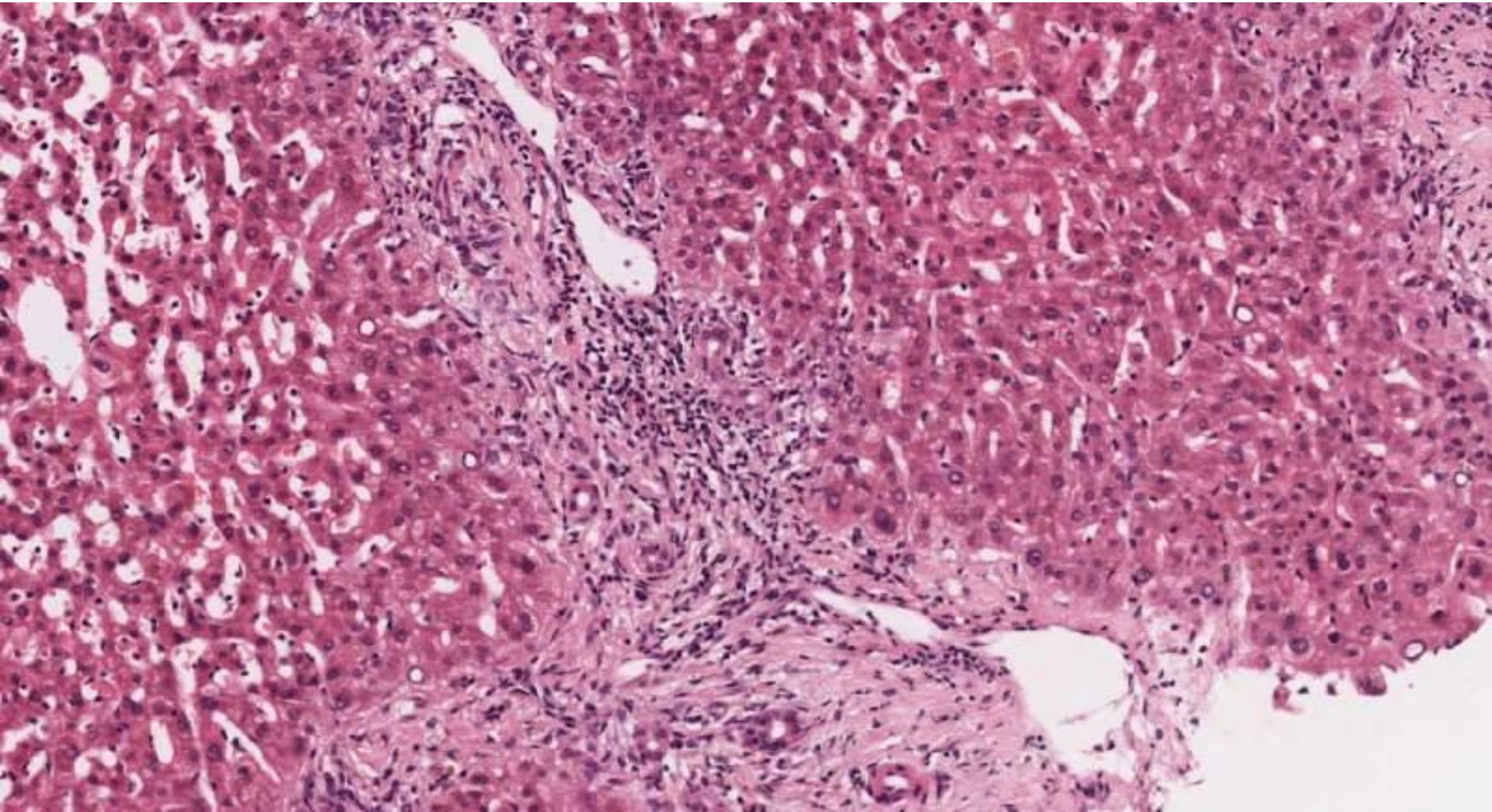
Case 221



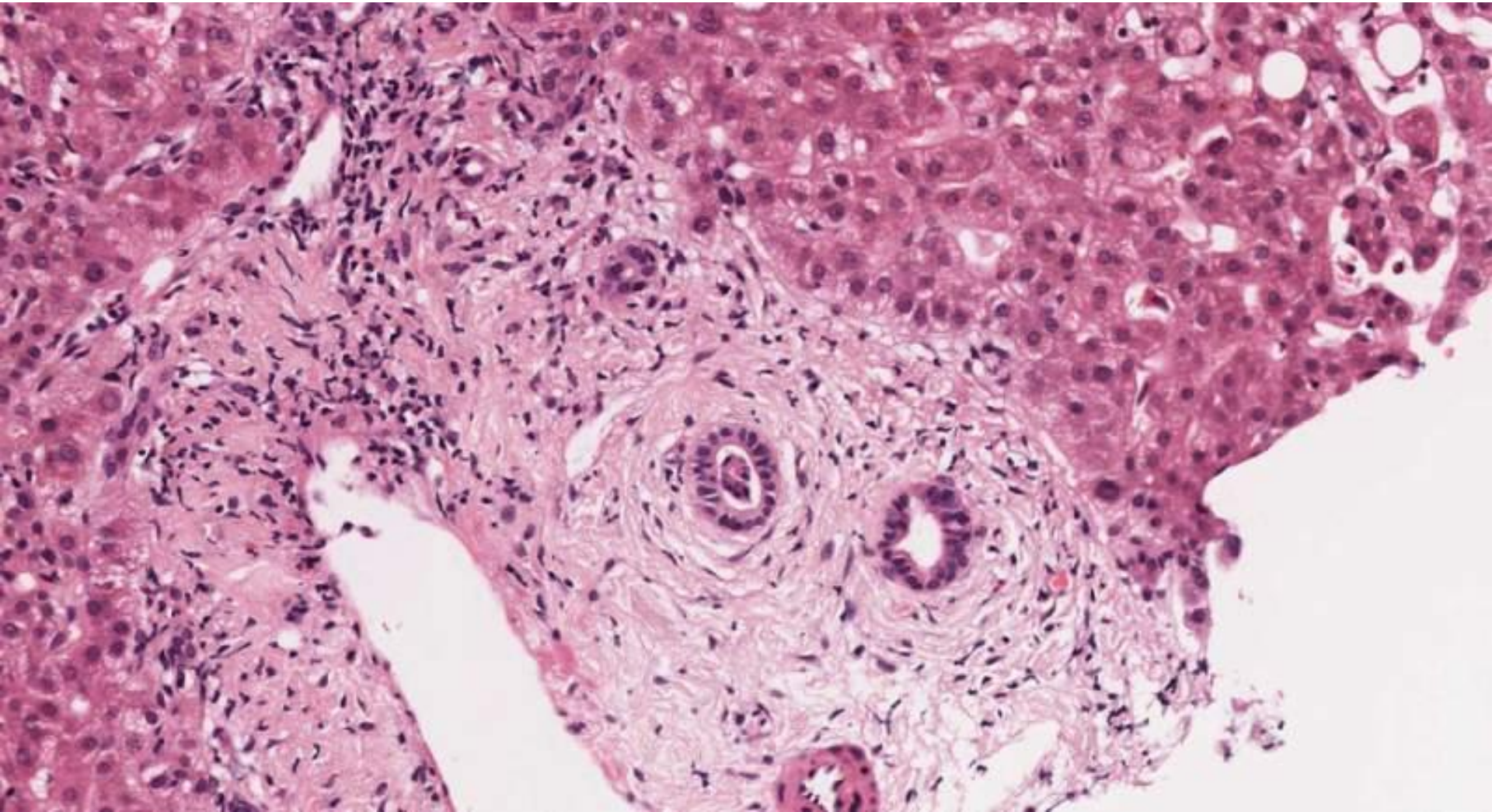
Case 221



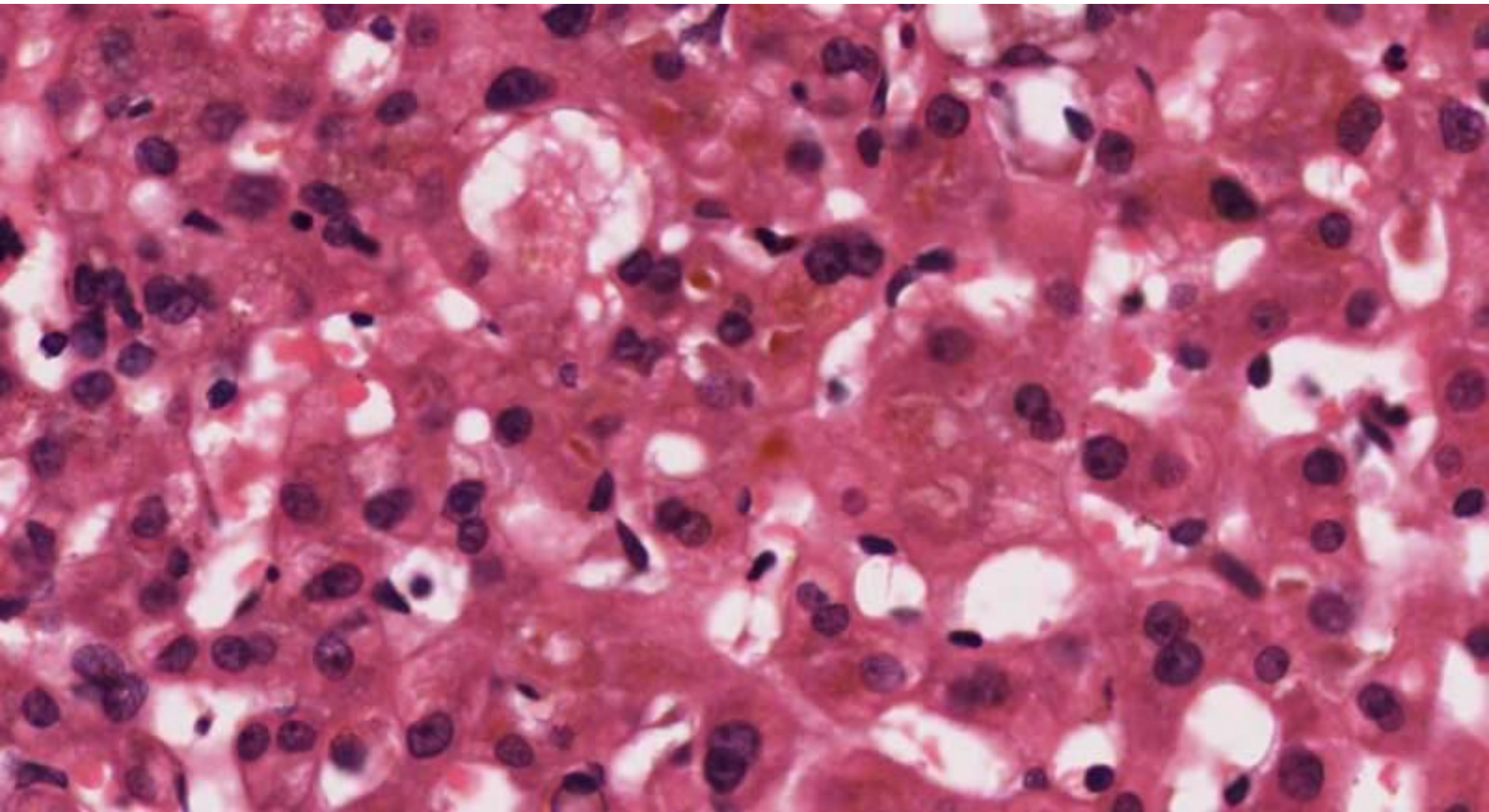
Case 221



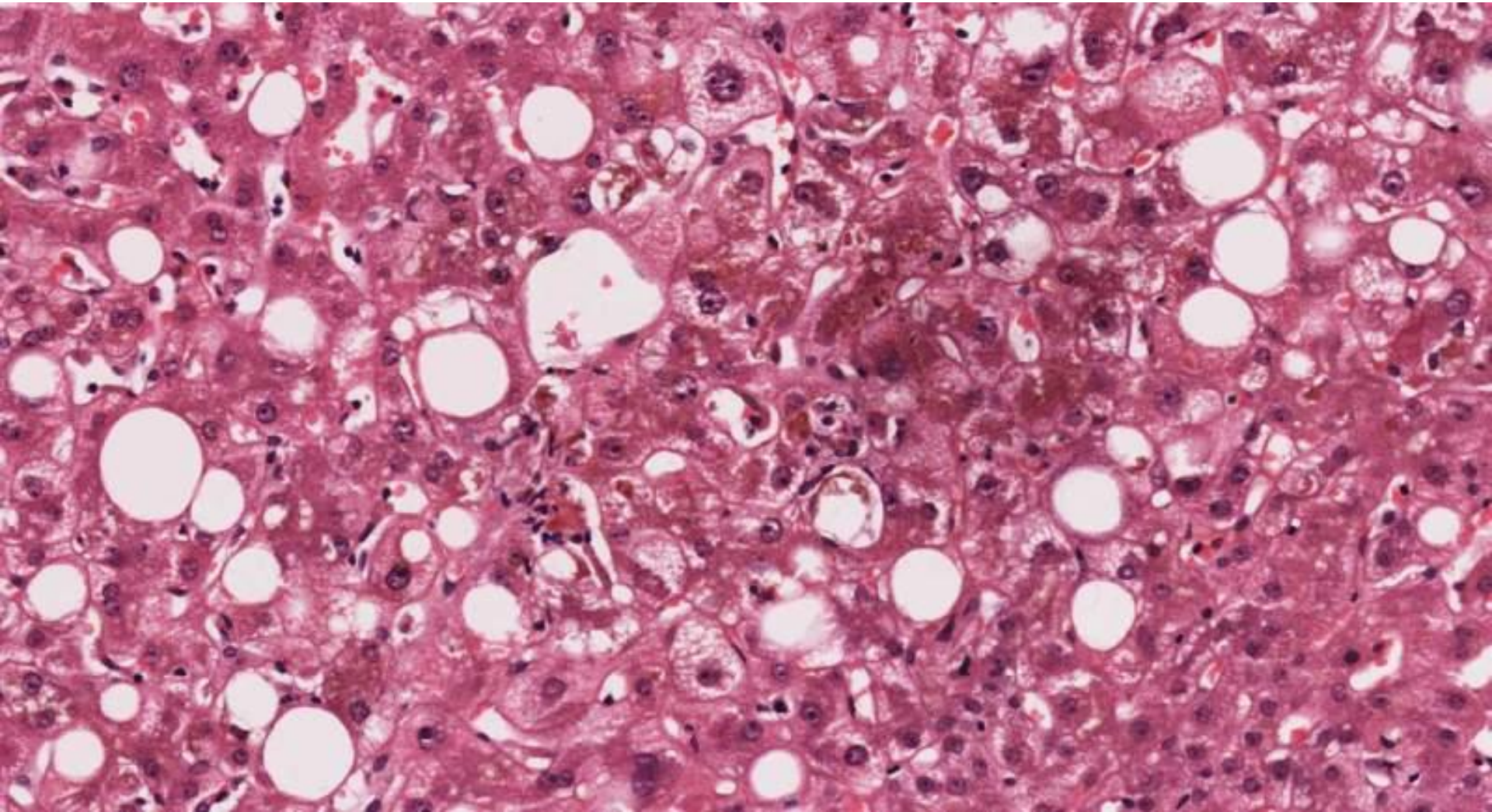
Case 221



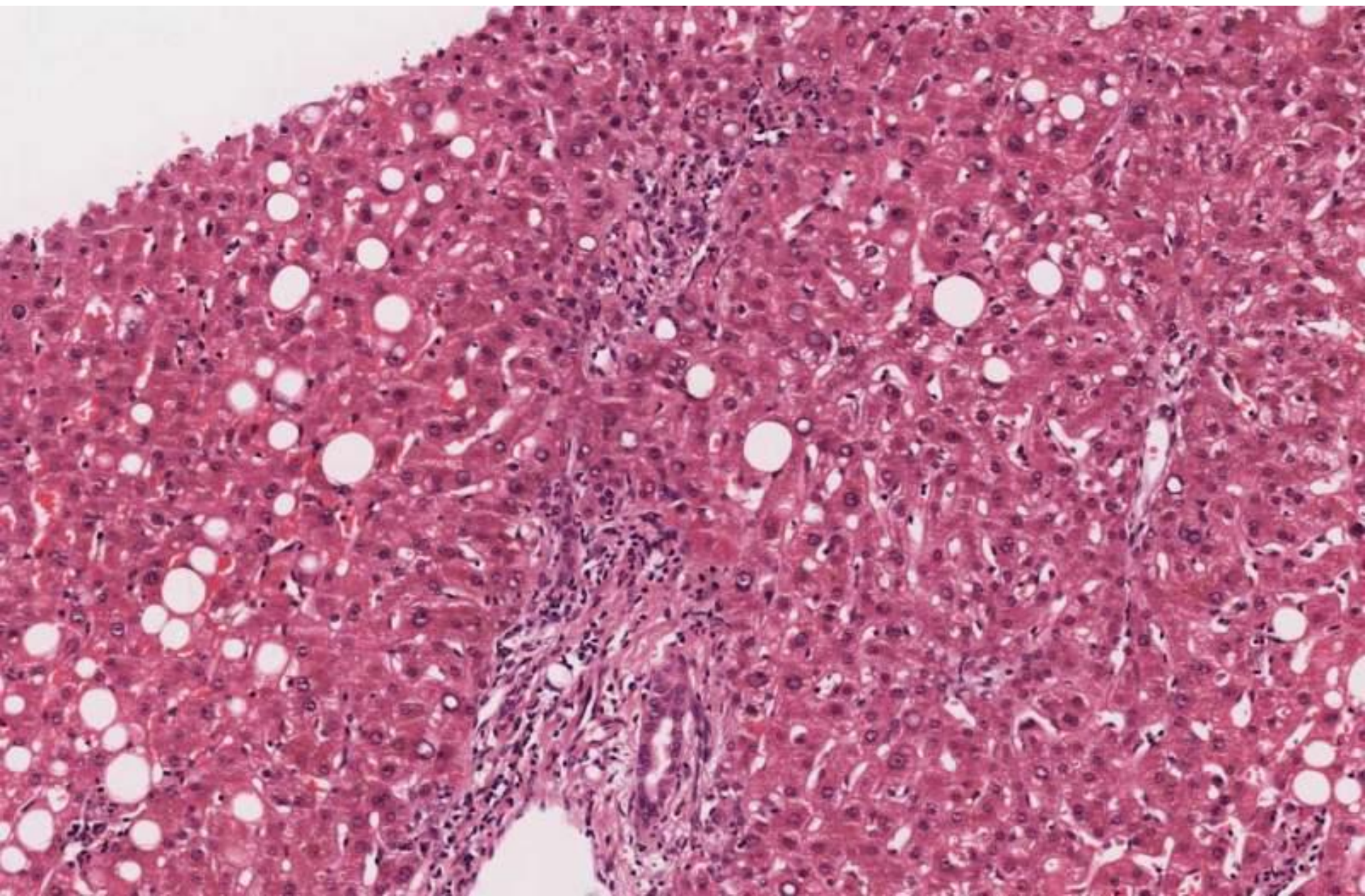
Case 221



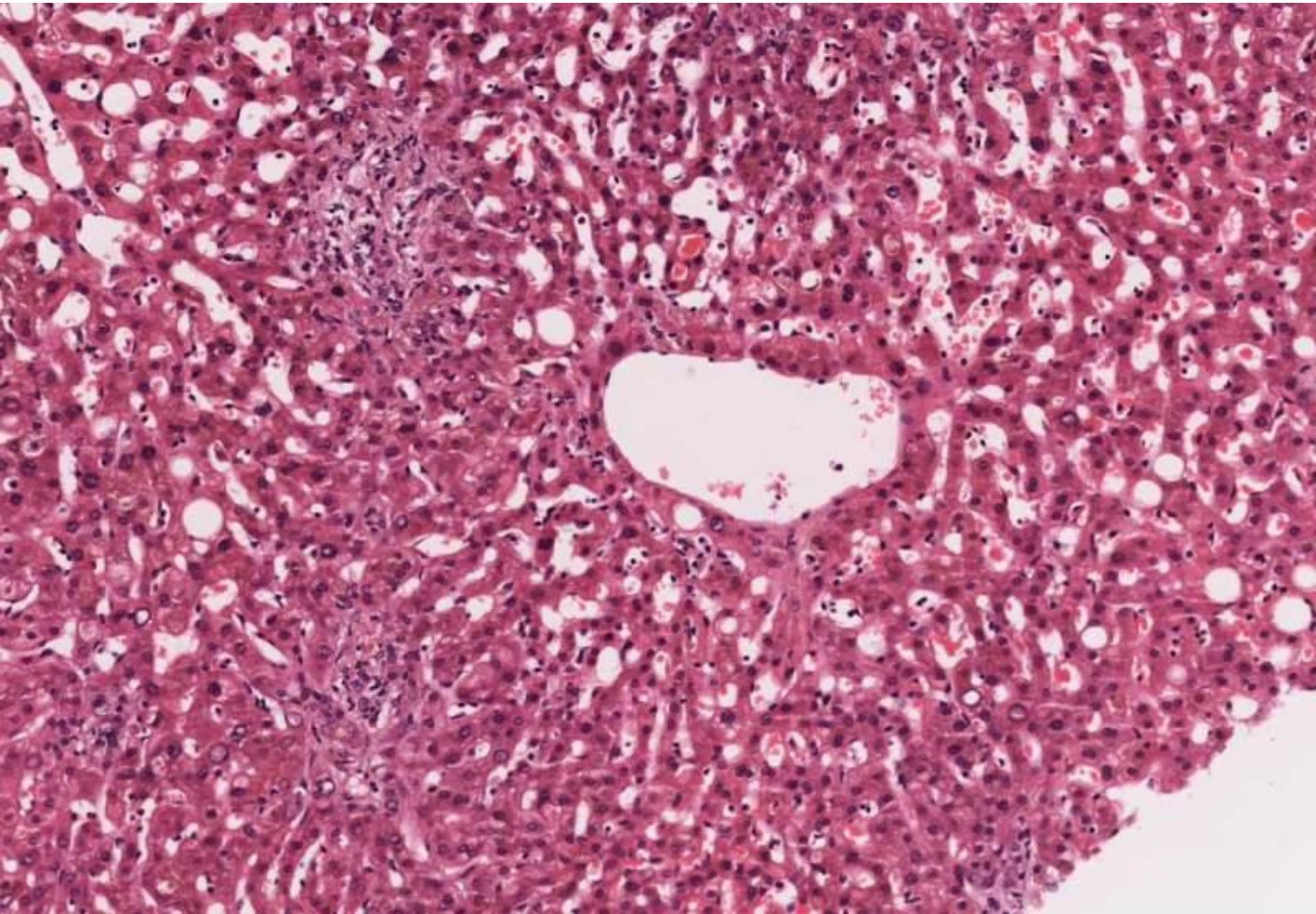
Case 221



Case 221



Case 221



Case 221

Results:

17 Cholestasis

14 cholestatic hepatitis

11 cholestasis with cholangiolitis /bile duct injury

12 cholestasis and steatohepatitis

5 fatty liver disease, cholestasis not mentioned

2 Budd Chiari /CCF as main diagnosis

29 due to drugs (statin)

20 possibly due to drugs

3 unlikely to be due to drugs

2 no mention of drugs

Case 221

Comments:

Do ERCP/? Early PSC 3

Also steatosis 17

Sinusoidal dilatation 2

Check ? genetic haemochromatosis 6

Follow up (Dr Kaye) – Initially diagnosed as cholestasis probably drug induced

Second CT showed stricture and bile duct adenocarcinoma confirmed on brushings. On review, features consistent with large duct obstruction.

The patient died rapidly after diagnosis therefore haemochromatosis not checked. No venograms

Case 221

Discussion

Accept all except fatty liver disease cholestasis not mentioned and Budd Chiari/CCF as main diagnosis.

Comments:

For EQA scoring, recognition that the main disease process was cholestatic is the minimum accepted.

This case shows portal changes of cholangiolitis and some oedema – features that would not be characteristic of statin related injury and suggests the need for further exploration past the biliary tree.

The “take home” message of this case is that liver function tests, requested because of the prescribing of statins, may be abnormal because of other previously unsuspected liver disease. Lobular hepatitis due to statins was found to be very rare during surveillance studies, and biopsy changes should be attributed to the statin with caution; they should be a low threshold for further investigation in this situation. The three participants who commented ‘unlikely to be due to drugs’ were correct.

Case 222

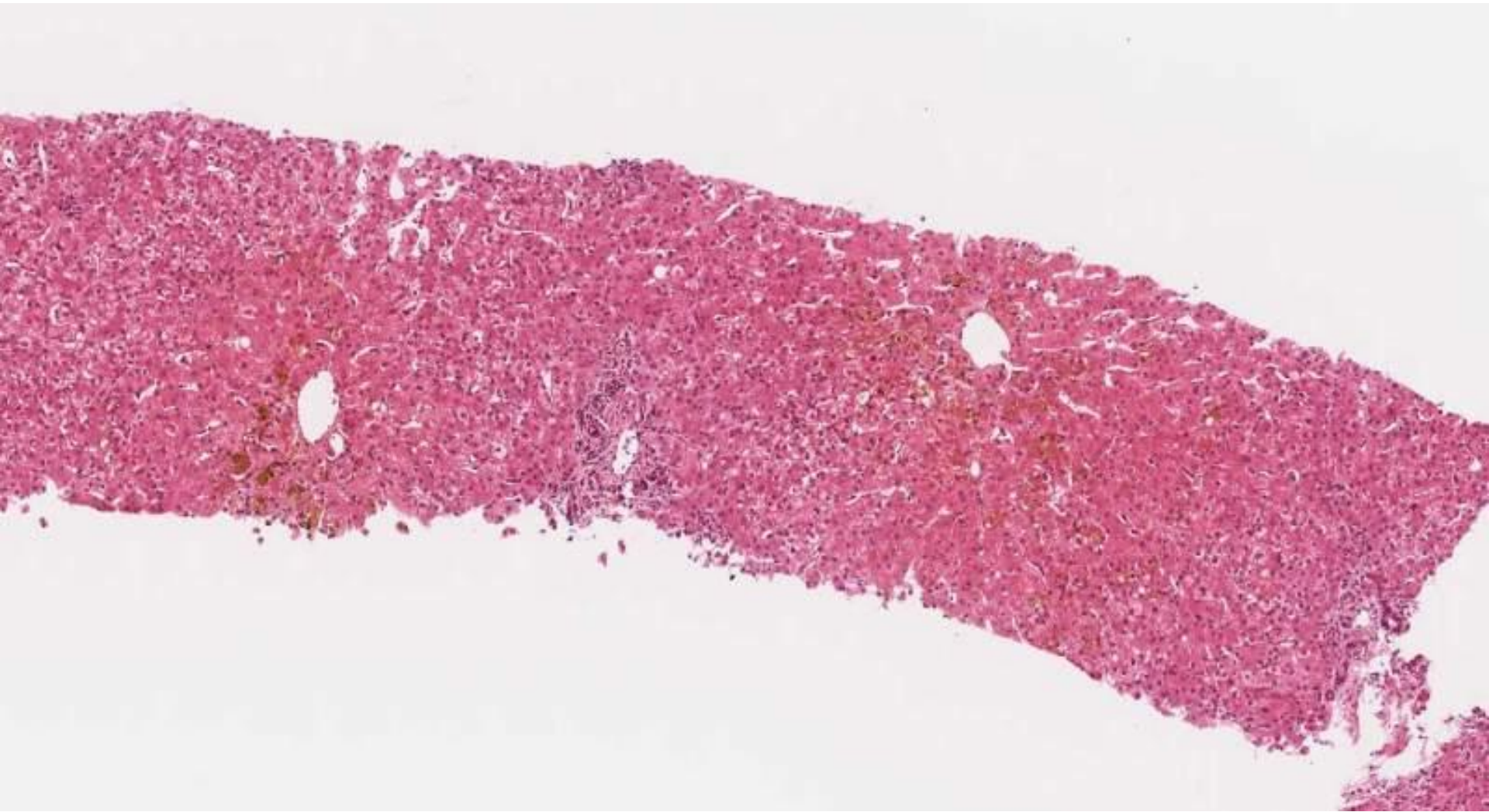
F37

Crohn's disease. TPN for 4 months. Jejun-ileal resection with only 40cm small bowel remaining. ?short bowel – related steatohepatitis. ? TPN related

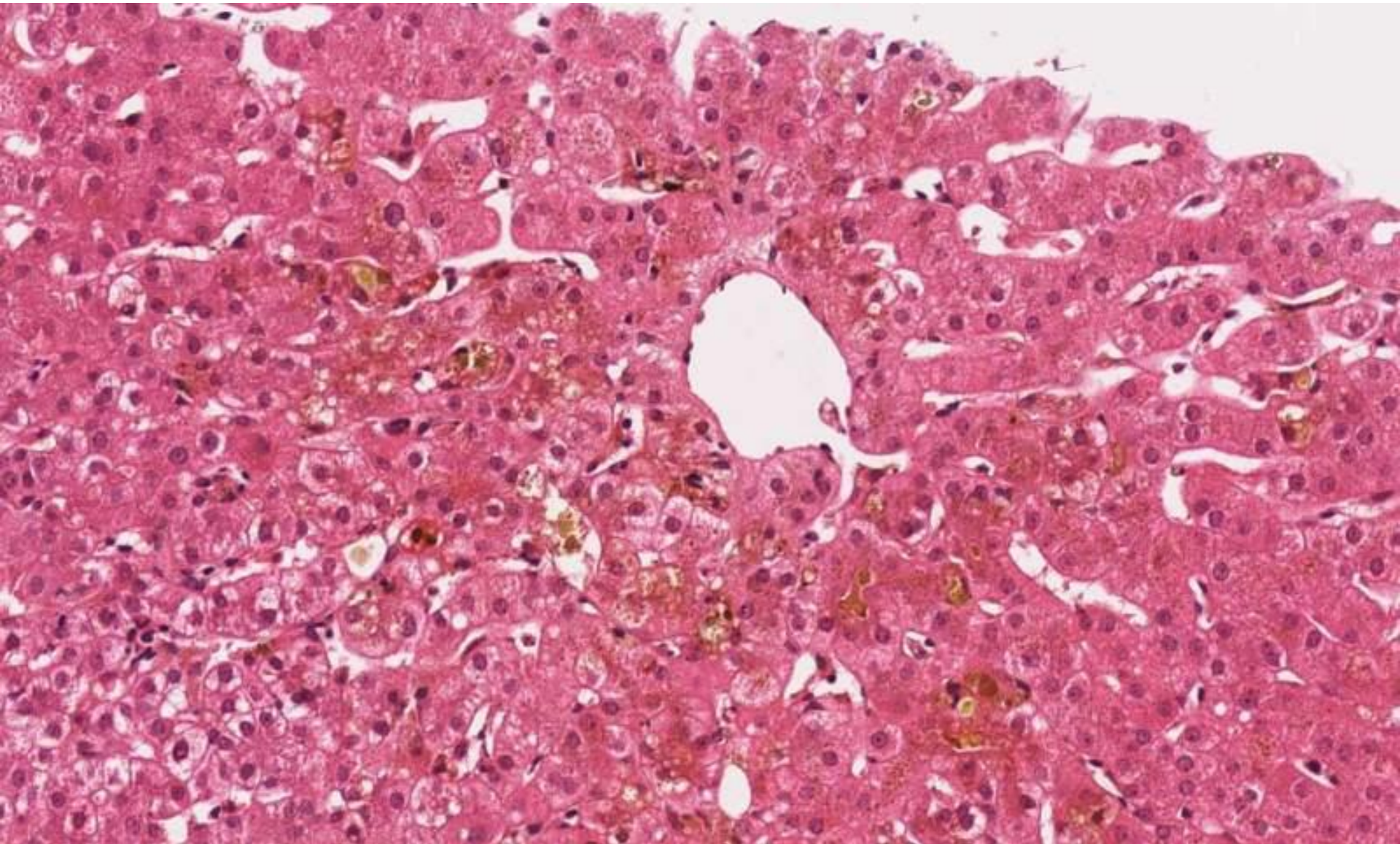
Case 222



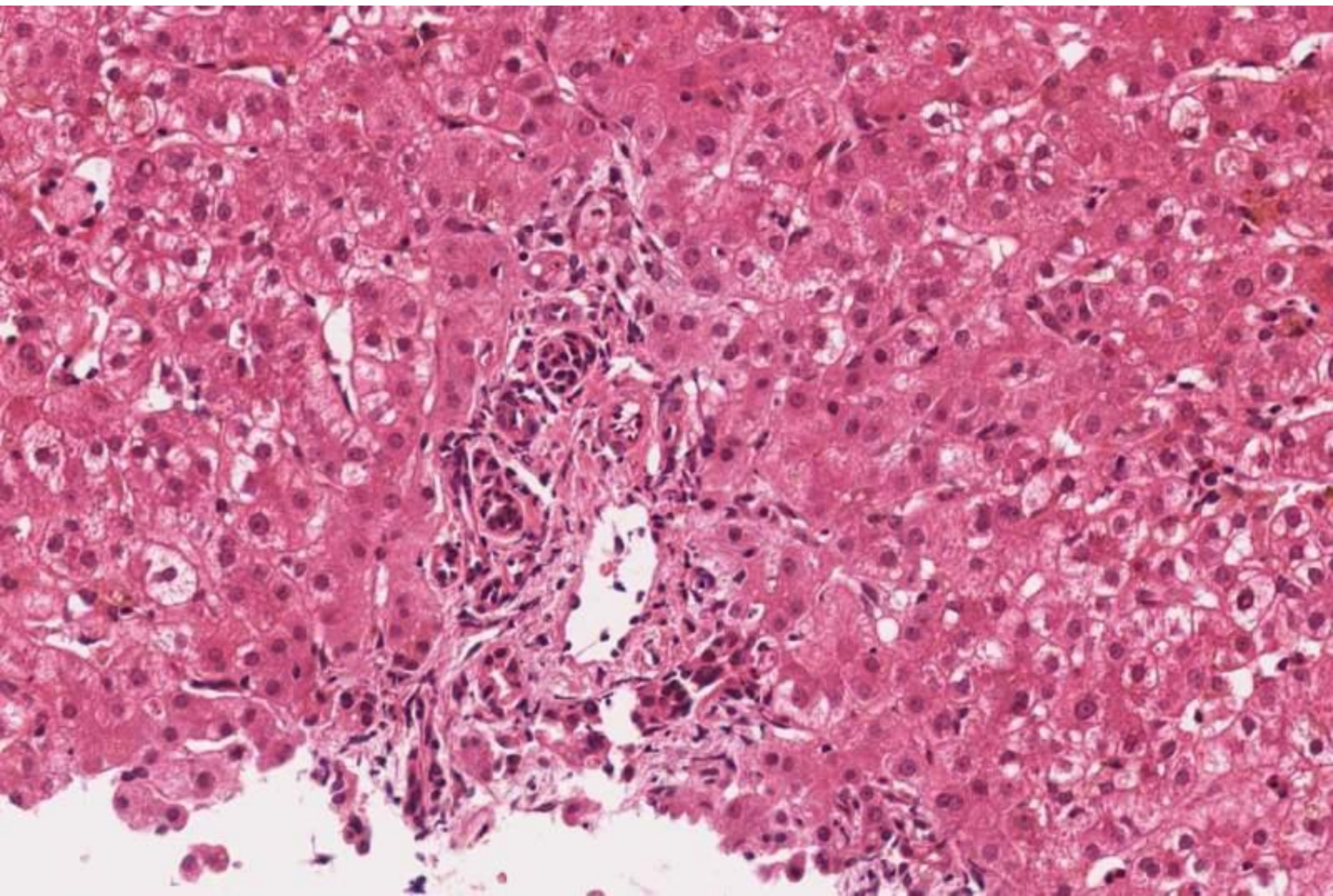
Case 222



Case 222



Case 222



Case 222

Results

55 Cholestasis etc. related to TPN

1 cholestasis, no steatosis (TPN not mentioned)

1 probable PSC, not TPN

Case 222

Comments:

Not steatohepatitis specifically stated: 6

Exclude LBDO 11

Exclude PSC, needs ERCP 2

Orcein 2

? drugs 4

Follow up (Dr Feakins): multiple fistulas and sepsis, died.

*Not investigated for LBDO, no clinical features to suggest obstruction;
cholestasis attributed to TPN*

Case 222

Discussion

Accept all except 'cholestasis, no steatosis (TPN was not mentioned)',
and 'probable PSC not TPN'.

Comments:

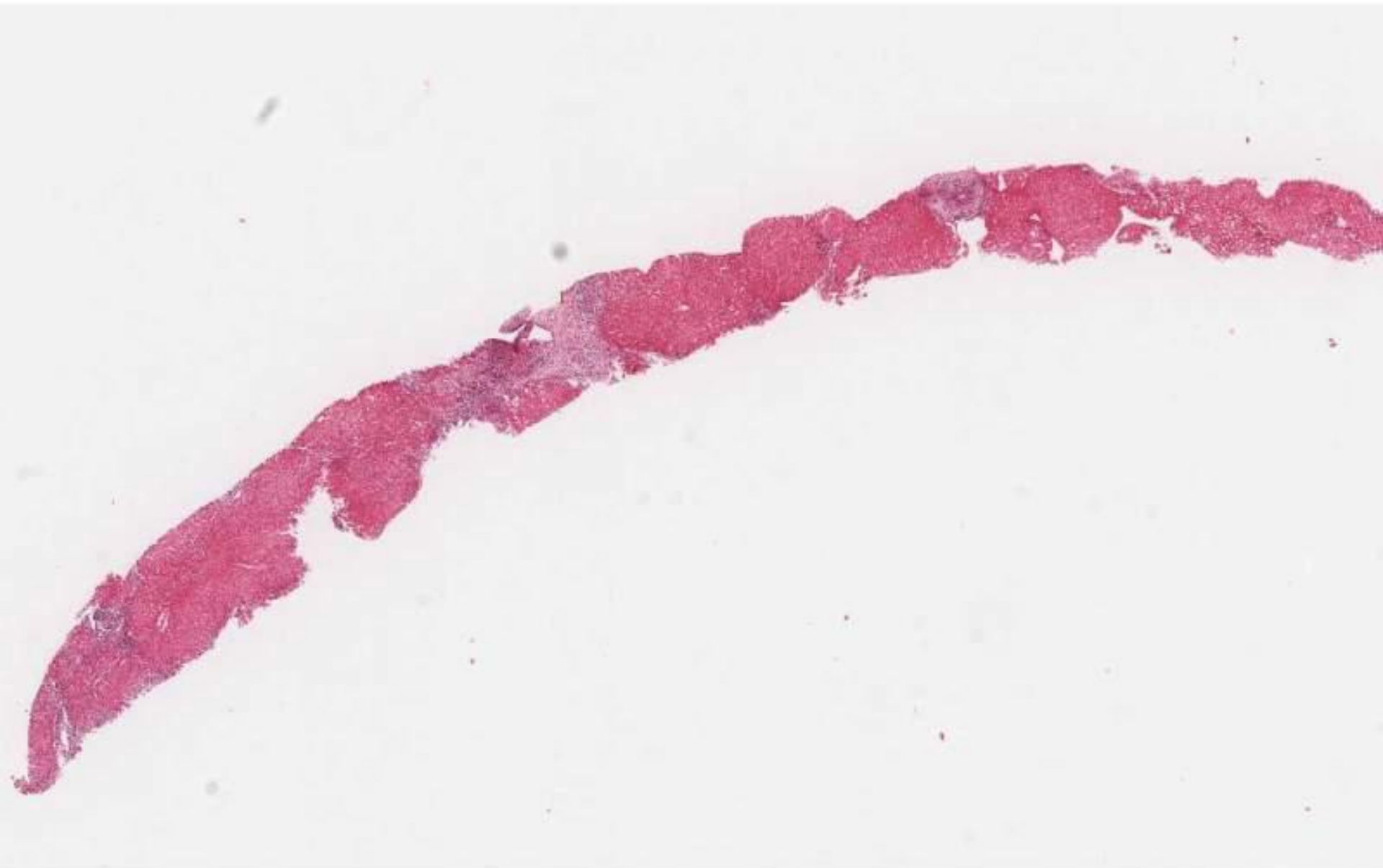
Clinically attributed to TPN – biliary imaging not appropriate.

Case 223

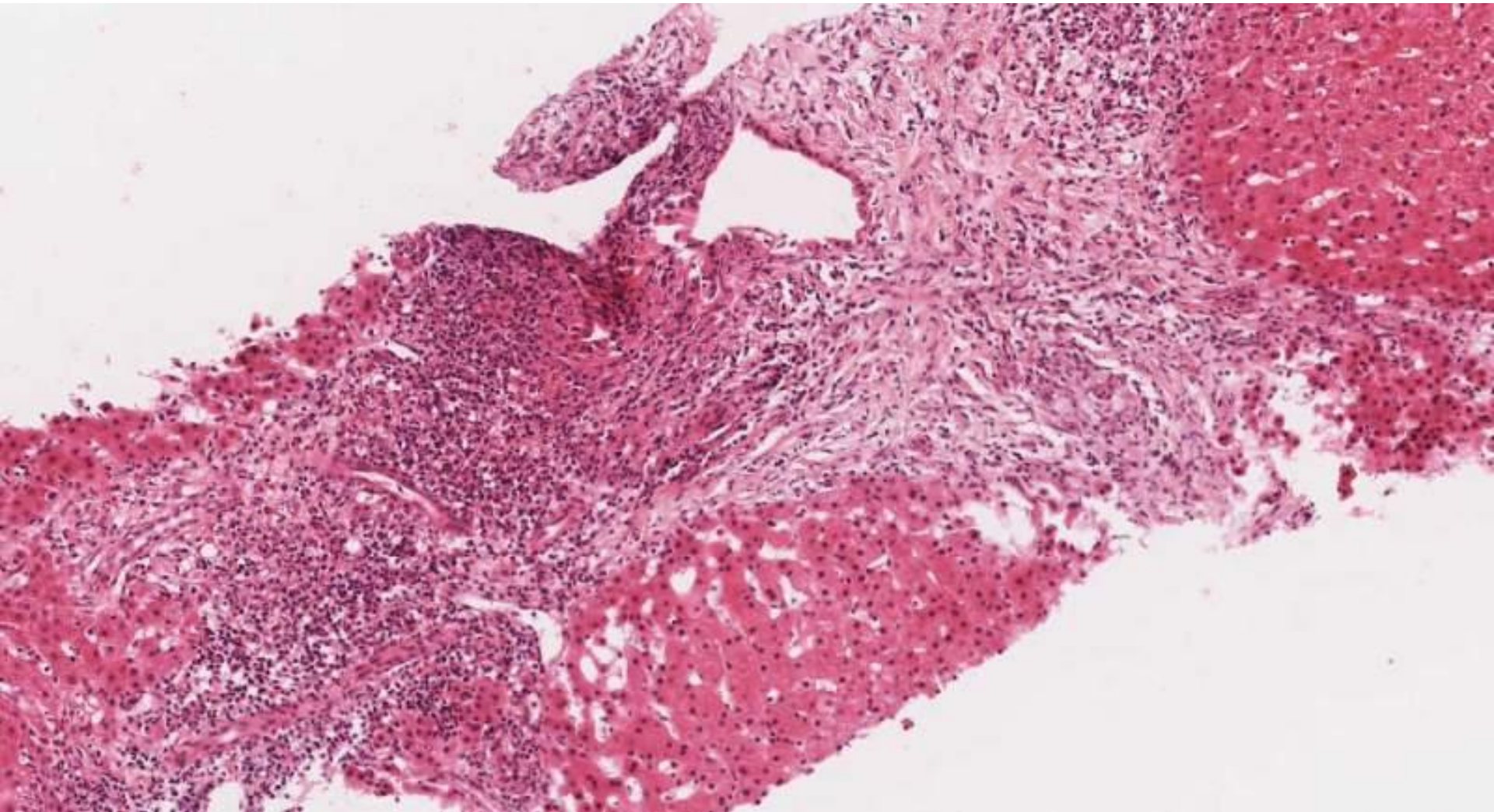
F49

Abnormal LFTs

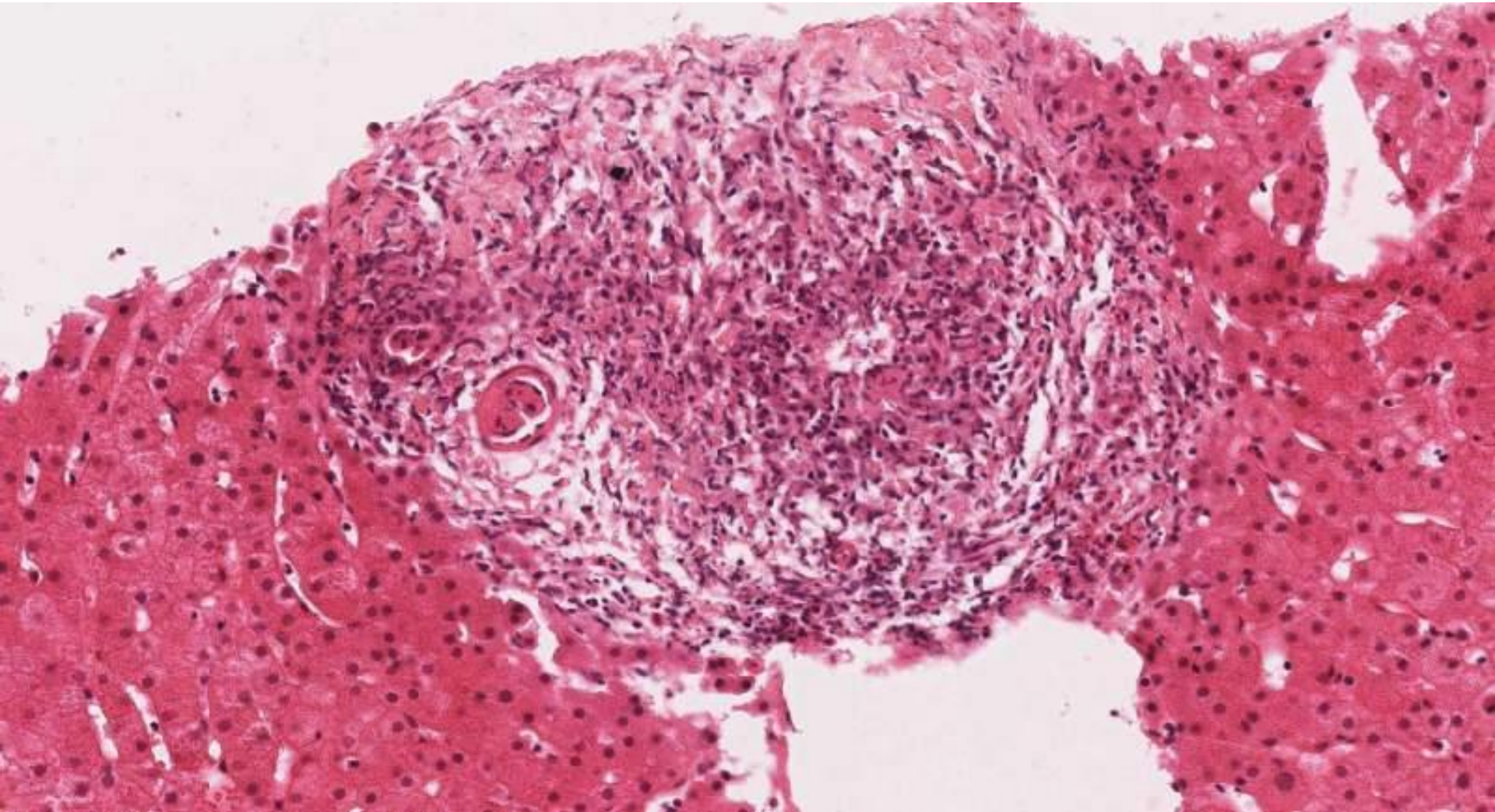
Case 223



Case 223



Case 223



Case 223

Results:

54 PBC with description and discussion

2 PBC with no other comments

1 granulomatous hepatitis, Differential diagnosis PBC, drug-induced hepatitis, sarcoid, obstruction

1 exclude drug reaction, lymphoma, other tumour ???PBC

Comments:

Needs AMA/serology/autoantibodies - 50

Orcein - 10

Imaging - 1

Comments on possibility of overlap syndrome, drugs – several

Case 223

Discussion

Accept all except 'granulomatous hepatitis with differential and exclude drug reaction lymphoma etc'.

Comments:

Discussion related to necessity of mitochondrial antibodies information for diagnosing PBC. This case was felt to show florid granulomatous duct lesions which were so characteristic of primary biliary cirrhosis, as to make any other diagnosis extremely unlikely. The only other possible differential would be sarcoidosis. There are patients with PBC who do not have mitochondrial antibodies. Many of these would have antinuclear antibodies, previously designated 'autoimmune cholangitis', but the current term for this is 'mitochondrial antibody negative PBC'. Clinically they behave the same as patients with PBC.

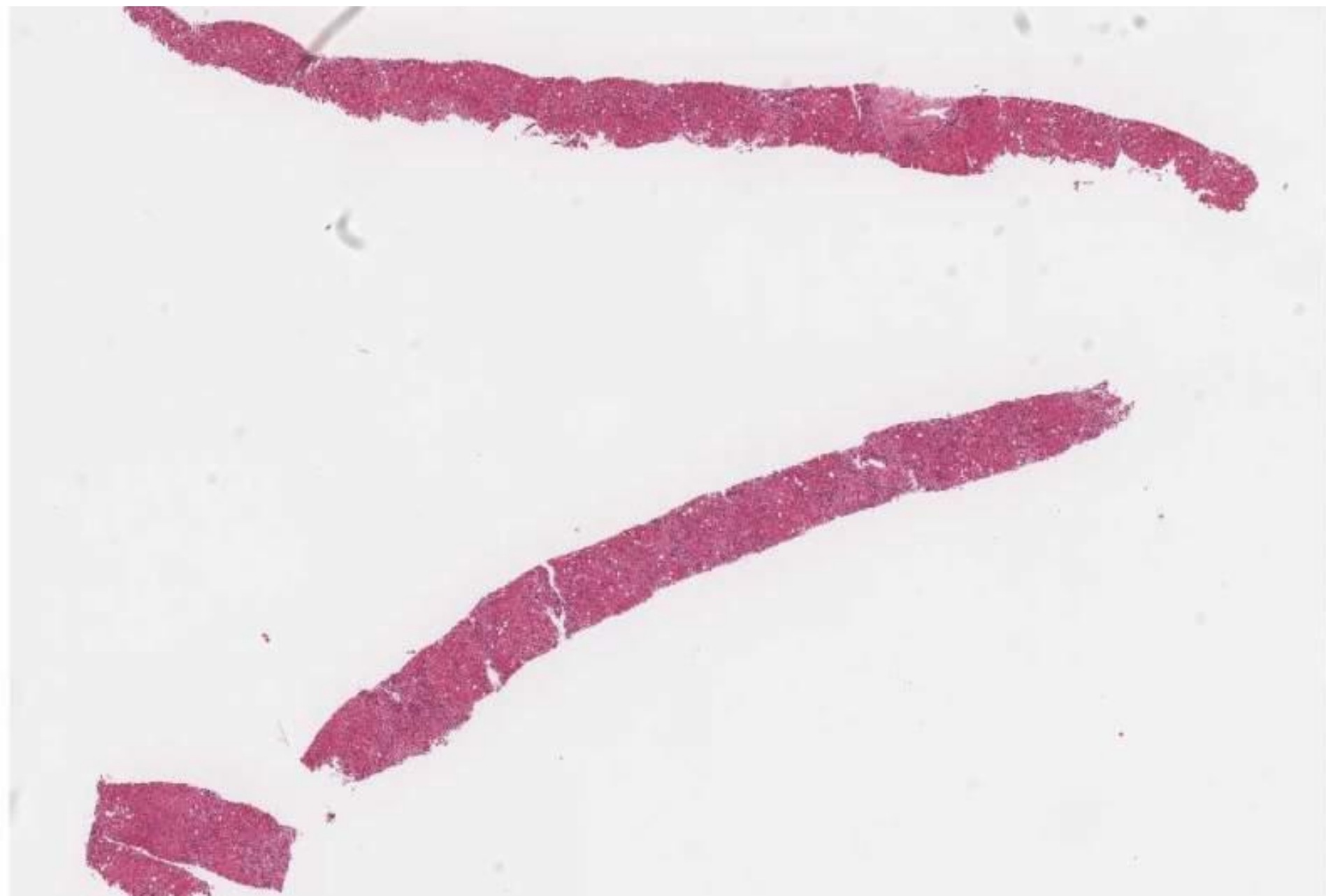
The question whether PBC can therefore be a purely morphological diagnosis – in practice this does not arise – the additional clinical information would be available. Also discussion about biopsy not being required for management in patient with appropriate LFTs and serology, although they are often still obtained in practice.

'Overlap syndrome' with autoimmune hepatitis is diagnosed when there are clinical (LFTs, autoantibodies) *and* histological features of both diseases – can be suggested by prominent interface hepatitis with plasma cells in a PBC biopsy, but not thought to be sufficient in this case.

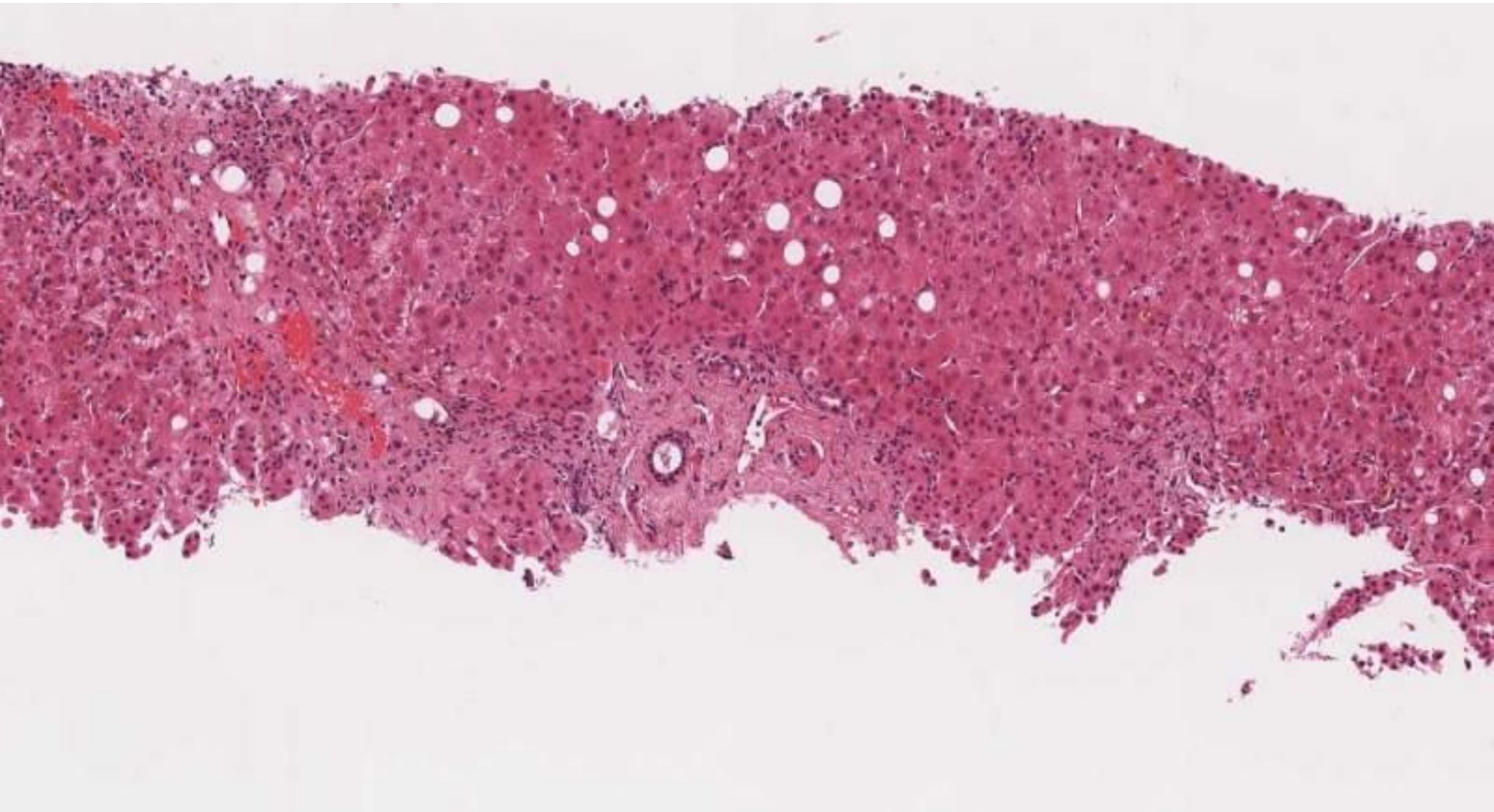
M58

Alcoholic liver disease – DPAS – prominent activated Kupffer cells. Orcein – no CAP detected. EVG – pericellular fibrosis,
Bilirubin 135, ALP 183, ALT 51

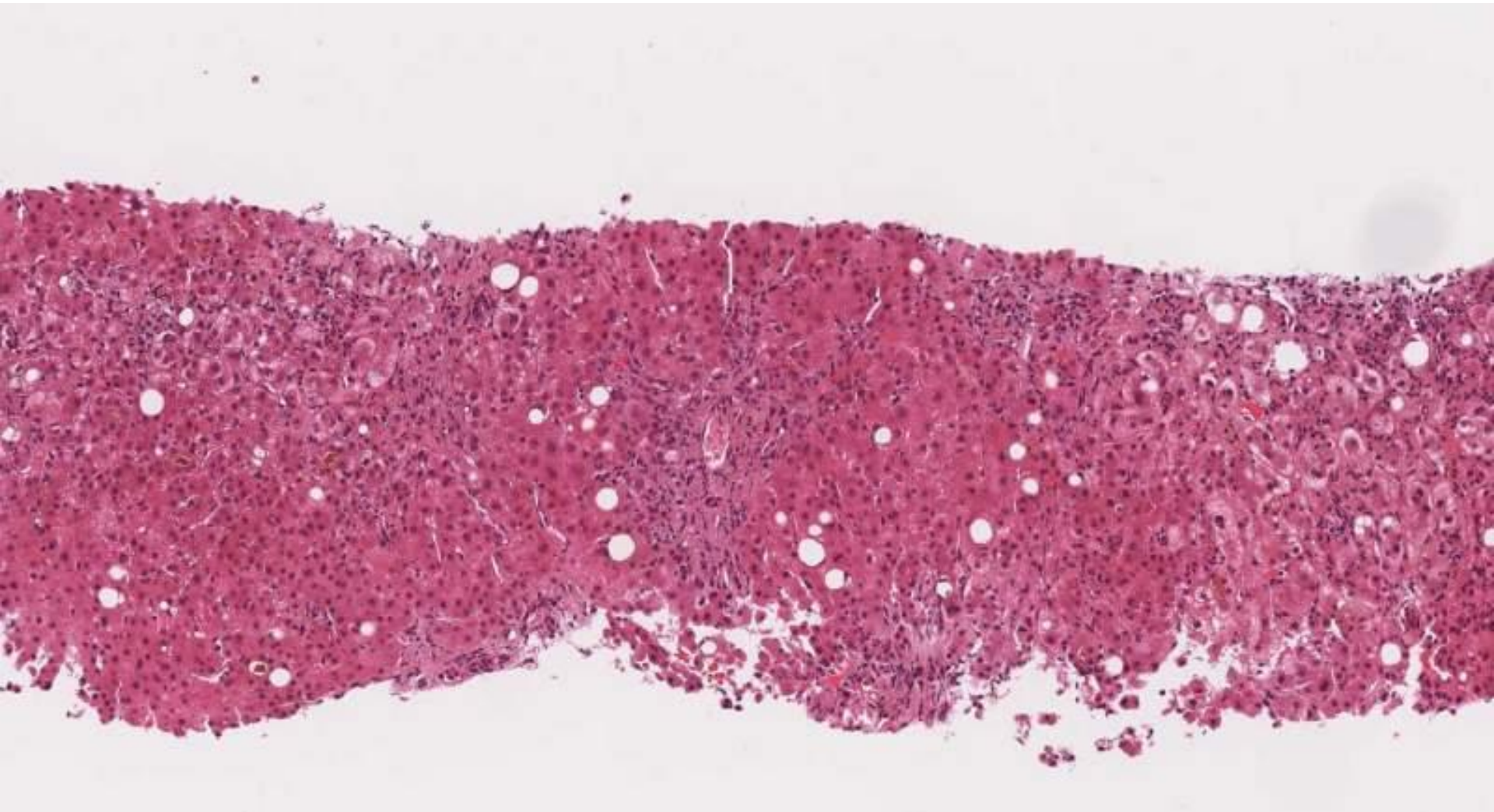
Case 224



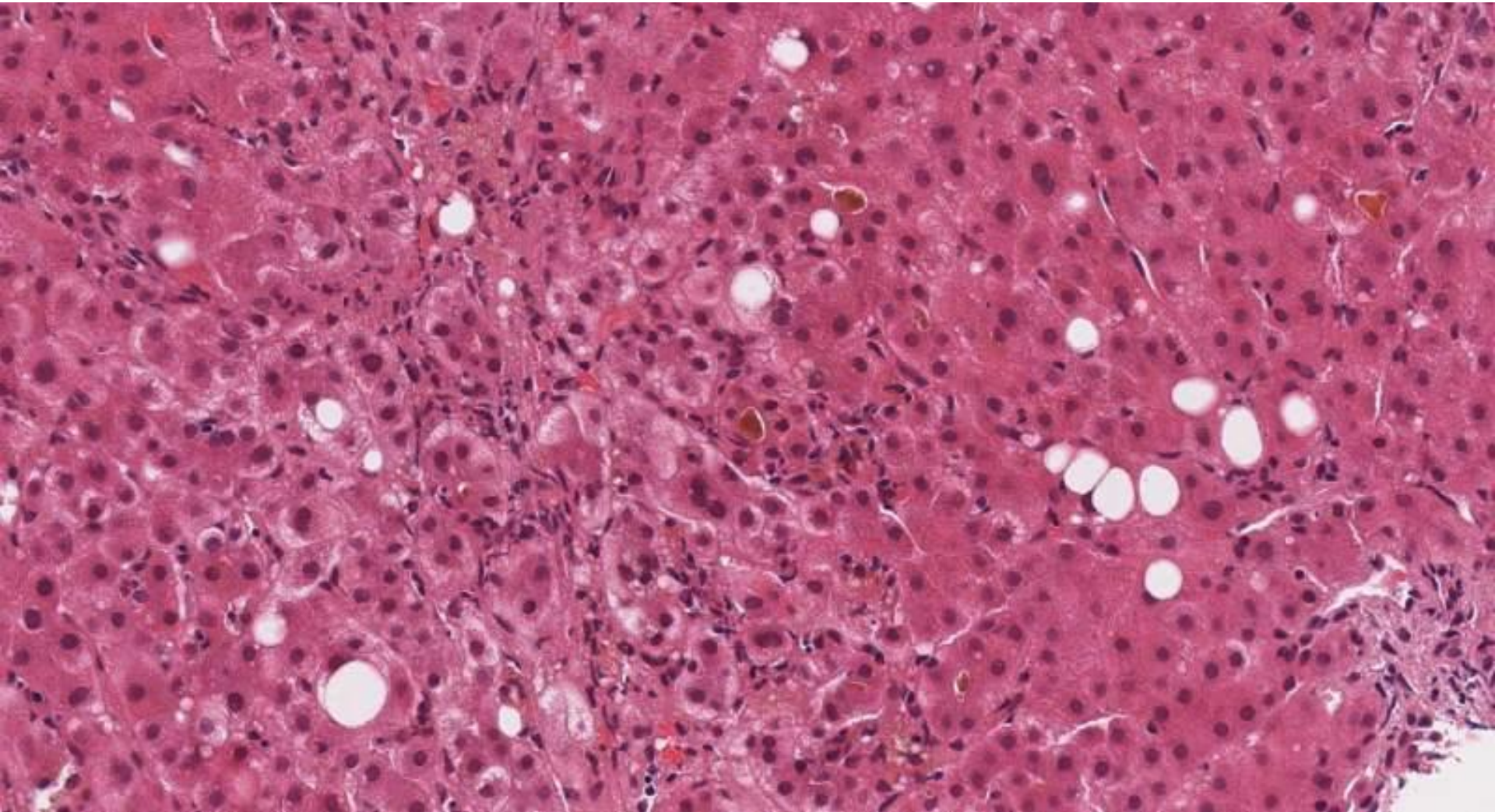
Case 224



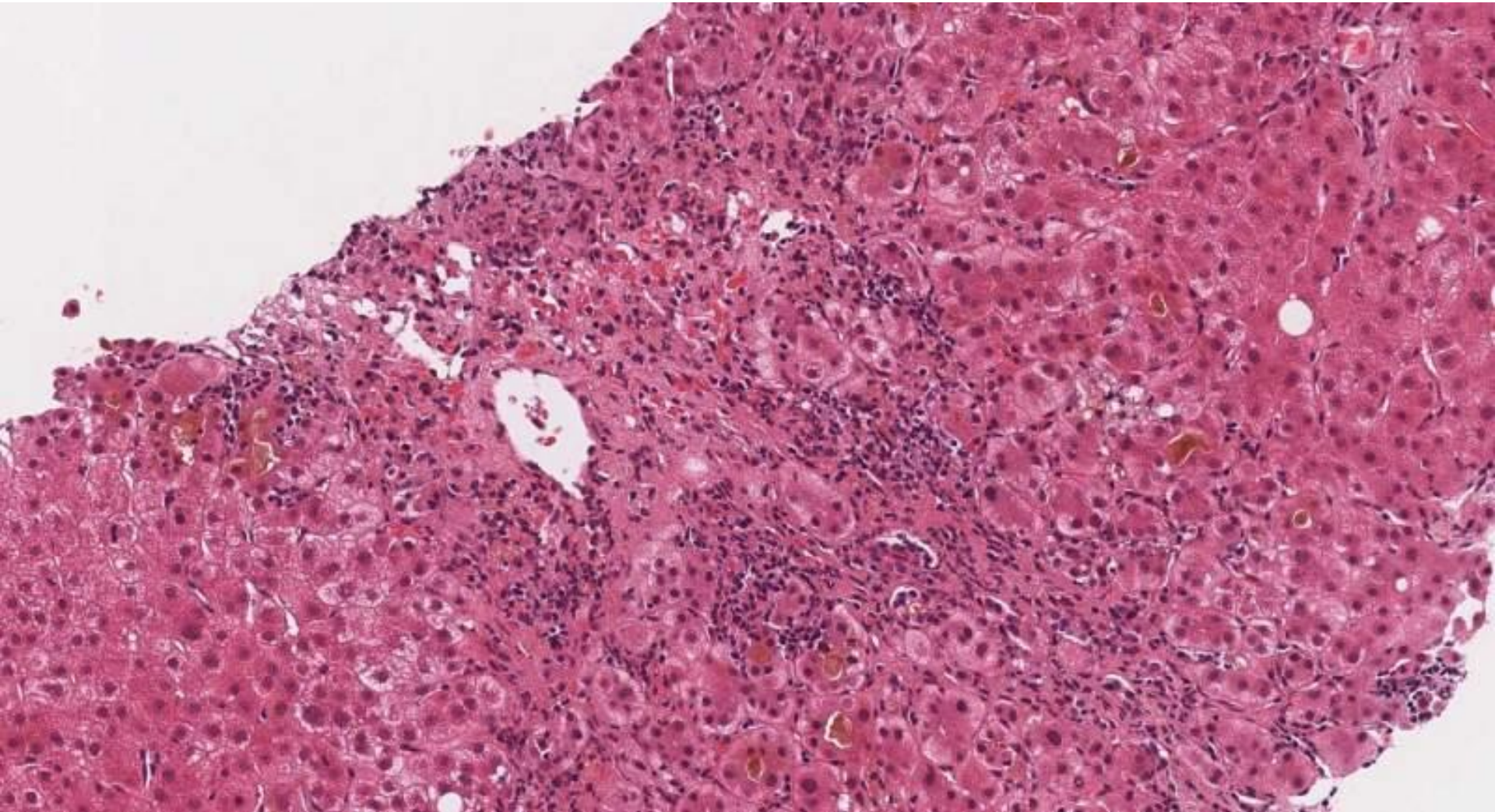
Case 224



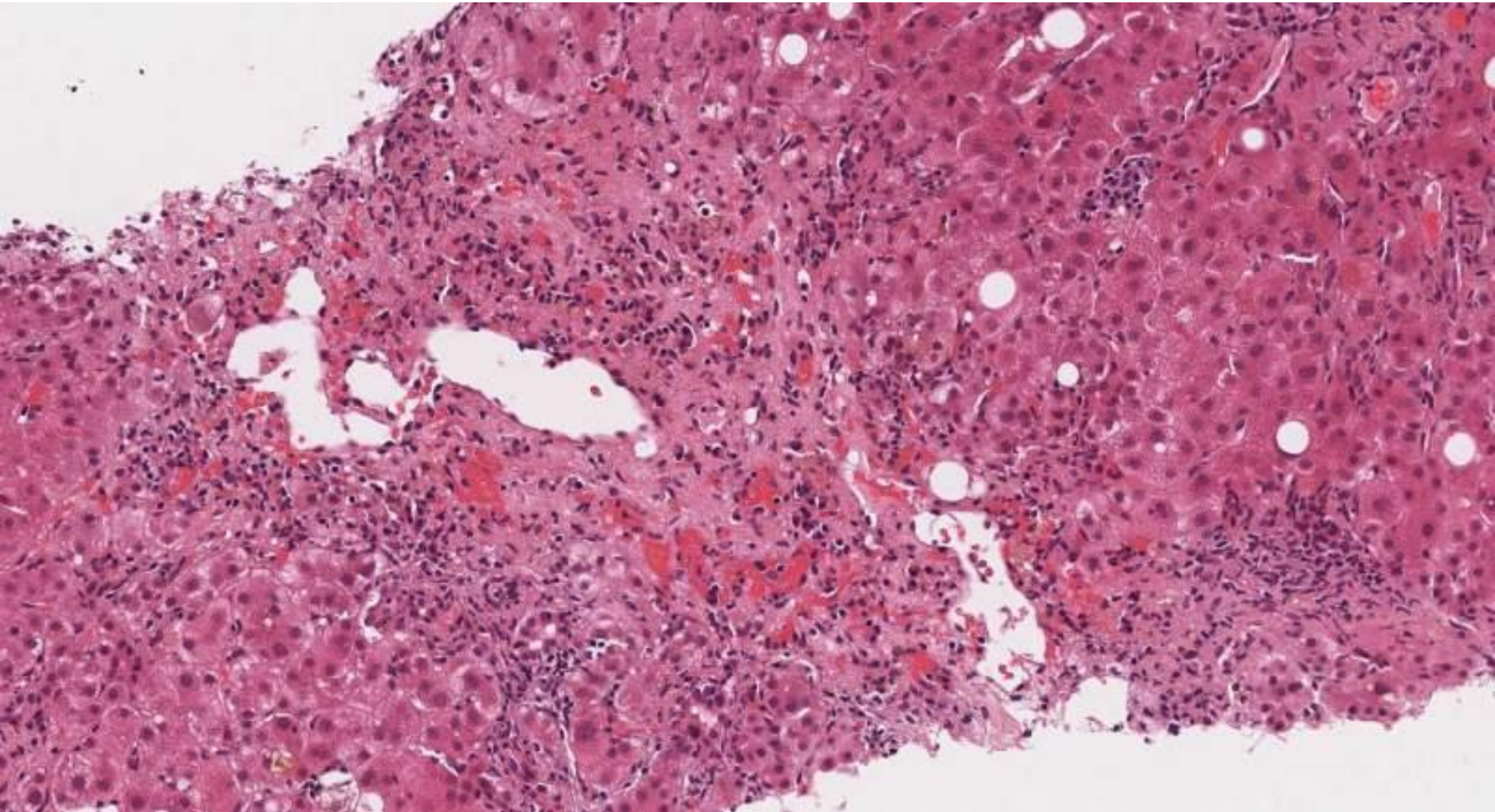
Case 224



Case 224



Case 224



Case 224

Results:

25 different groups of diagnoses:

24 alcohol alone – of which

7 steatohepatitis

8 steatohepatitis + cholestasis

7 cirrhosis

2 sclerosing hyaline necrosis

2 cholestatic hepatitis c/w alcohol

26 alcoholic liver disease plus something else to account for necrosis/cholestasis/inflammation

16 steatohepatitis plus drugs

6 autoimmune

1 viral

3 some additional, doesn't say what

3 alcohol unlikely/not alcohol

2 cholestasis, hepatitis, collapse, ? drugs, not alcohol

1 perivenular dropout and congestion ? acute BCS

Case 224

Follow up (Dr Kitching): Diagnosis: alcoholic steatohepatitis with cholestasis of a severity suggesting a second process, ? Drug toxicity

Further information after MDT: Disabled man with a history of alcoholism, admitted with exacerbation of liver disease. No definite history but paracetamol level high on admission, probable overdose of paracetamol.

Liver tests normalised over next 8-12 weeks.

Case 224

Discussion

Not suitable for scoring.

Comments:

The pattern of zonal perivenular necrosis and inflammatory infiltrate is not part of the spectrum of alcoholic liver disease; the correct response to this case is to suspect an additional pathology super-added onto fatty change and fibrosis from alcohol.

The clinical diagnosis is probable Paracetamol toxicity in addition to alcoholic liver disease. Architectural distortion is due to perivenular zonal necrosis rather than established fibrosis/cirrhosis; the features are not characteristic of sclerosing hyaline necrosis where the fibrosis is pericellular and surrounds ballooned hepatocytes, generally with obliteration of the efferent veins.

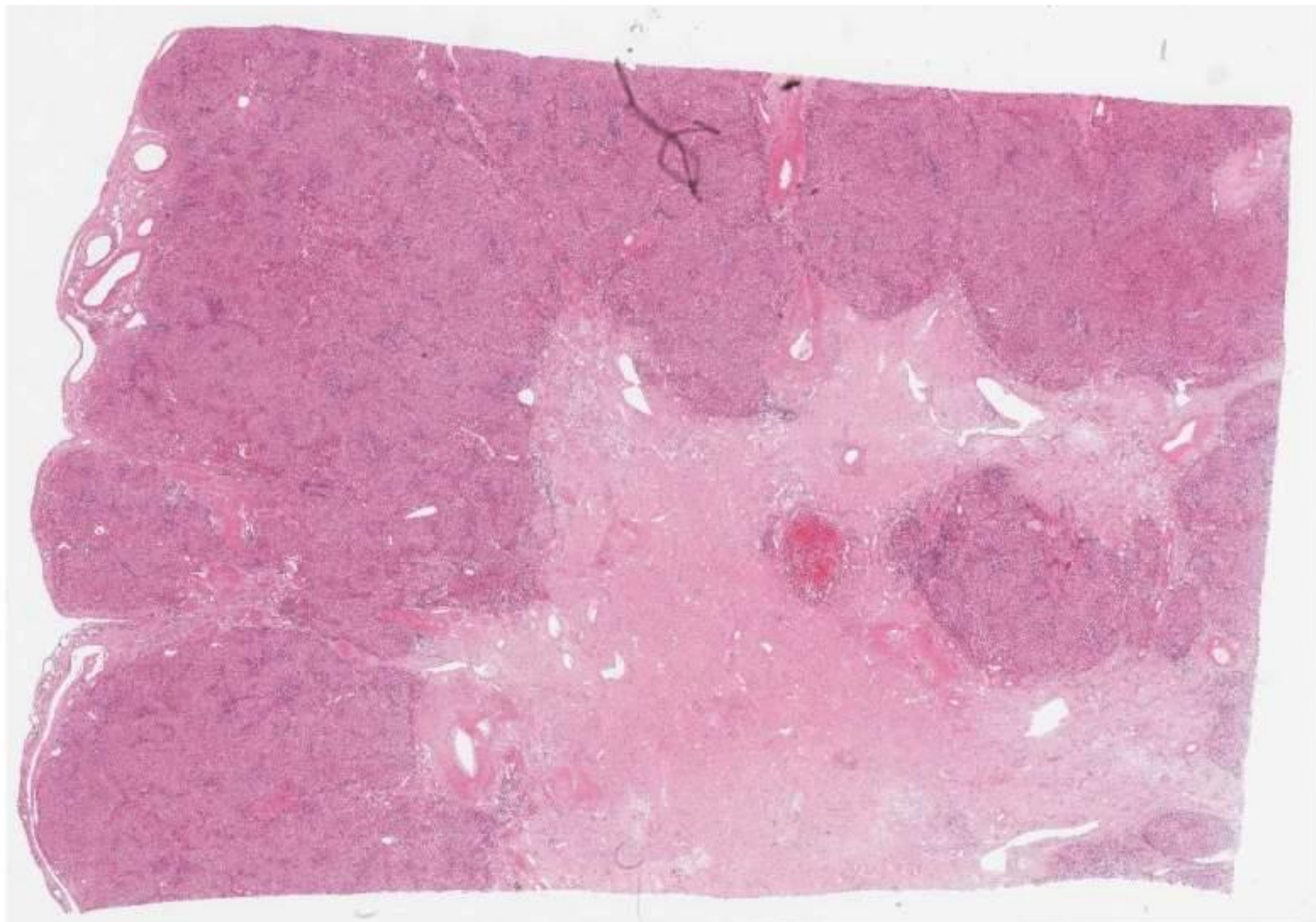
The degree of inflammatory infiltrate is more than you would expect from Paracetamol, and autoimmune hepatitis should be fully excluded. It was commented that although the early stages of Paracetamol toxicity were characterised by coagulative necrosis without an inflammatory cell component, after one to two weeks (rarely seen in practice) there is collapse and an inflammatory infiltrate becomes more prominent, so that the pattern of injury more closely resembles acute hepatitis with zonal necrosis from other causes.

F35

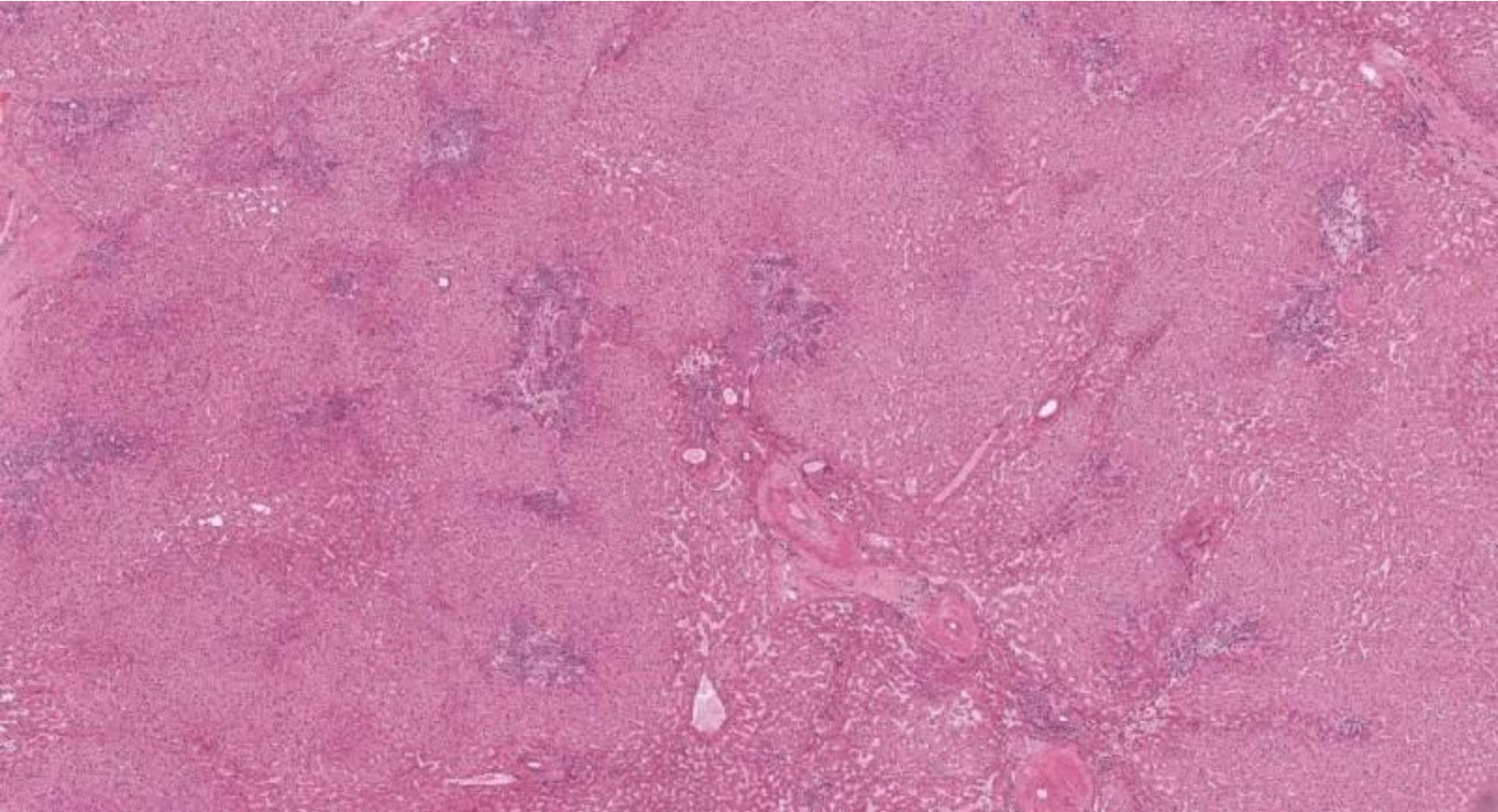
Liver tumour, segment 3

Largely circumscribed mass measuring
5.5x4.5x3.5cm with attached liver on one bare
surface 4.5x2.5cm

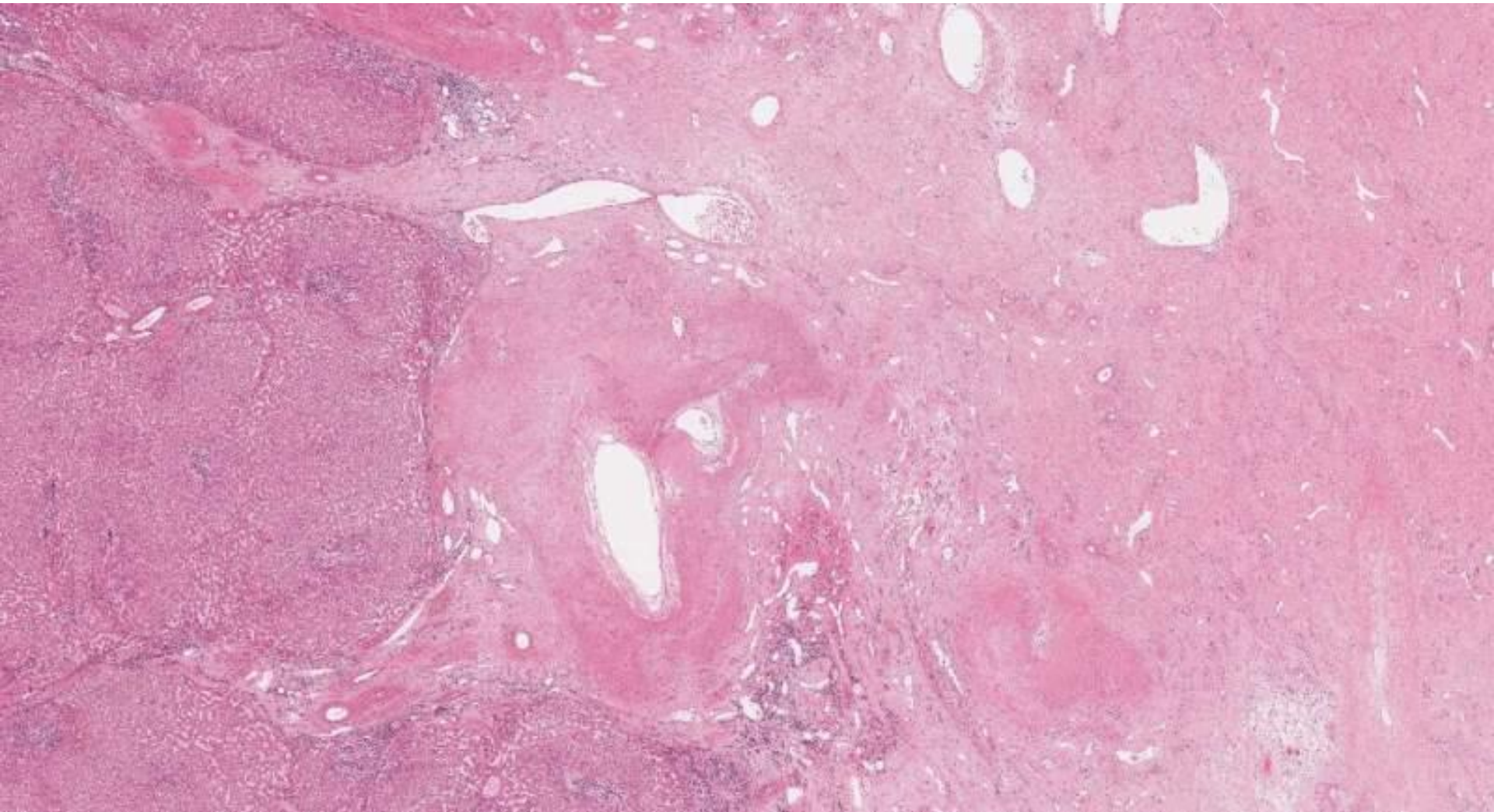
Case 225



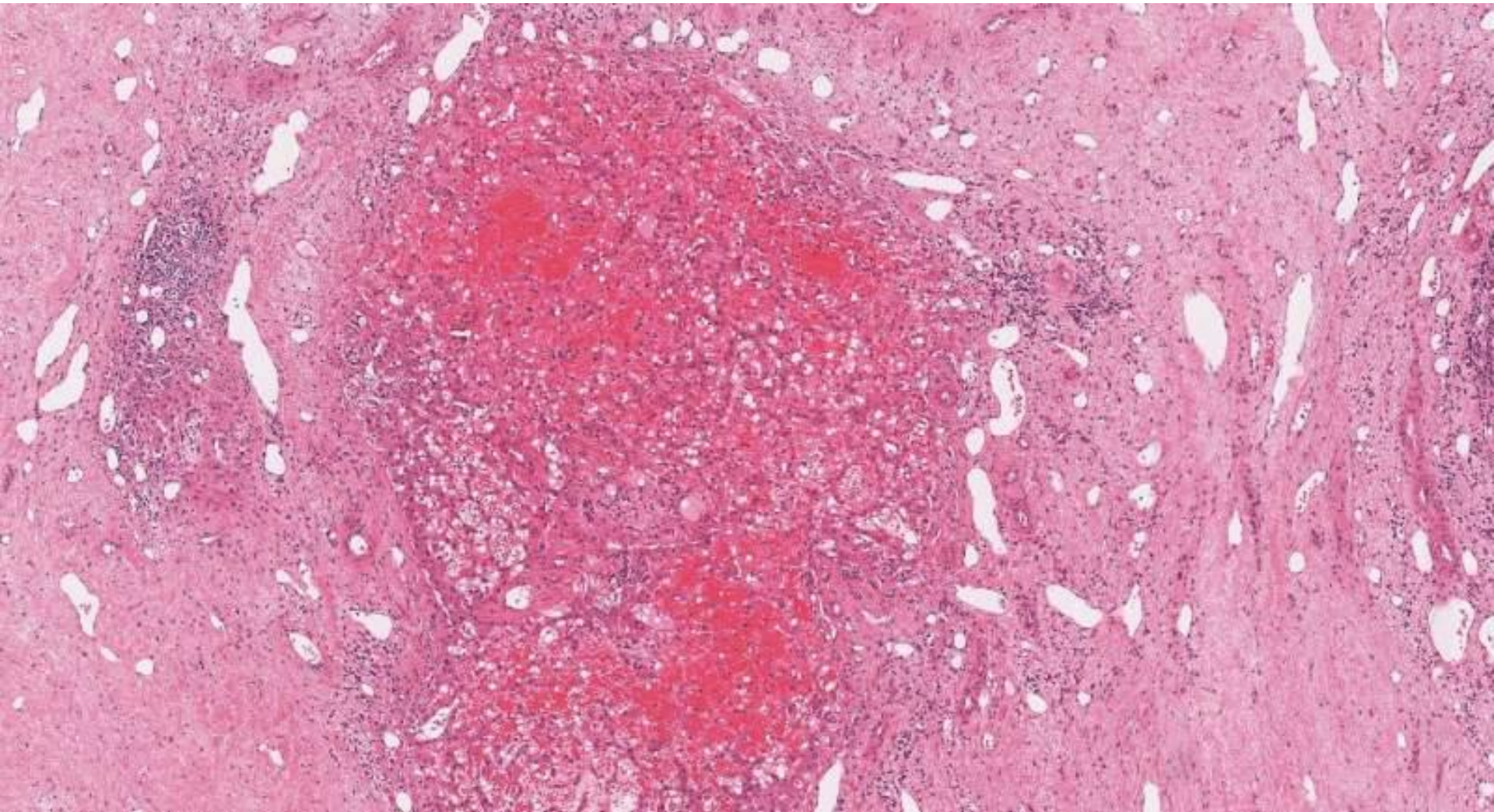
Case 225



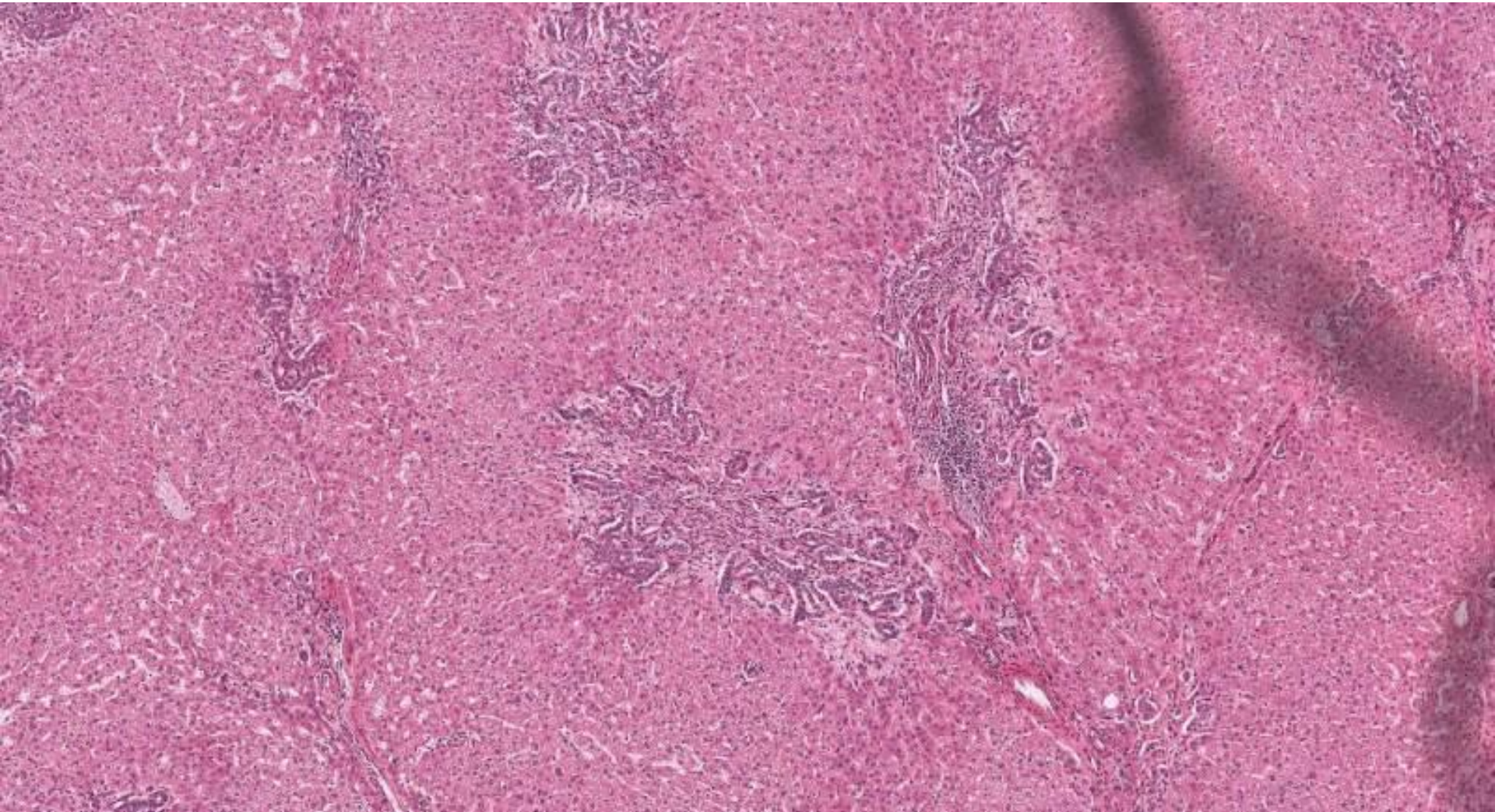
Case 225



Case 225



Case 225



Case 225

Results:

48 Focal nodular hyperplasia

2 sclerosed haemangioma

1 fibrosed haemangioma/embolised vascular malformation

1 sclerotic vascular malformation, suggestive of FNH

1 angiomyofibroma? FNA ??

1 hyalinised vascular malformation (“FNH”)

1 mesenchymal hamartoma

1 hyperplasia in association with vascular malformation,
now occluded

Case 225

Comments

Adjacent liver with focus of necrosis and granulomas 2
? previously embolised 2

*Follow up (Dr Sherwood) – Diagnosis: FNH
(no history of previous embolisation)*

Discussion

Reject 'sclerosed haemangioma', 'fibrosed haemangioma', 'angiomyofibroma', 'mesenchymal hamartoma', and 'hyperplasia associated with vascular malformation now occluded'. Accept diagnosis that include 'FNH'

Comments:

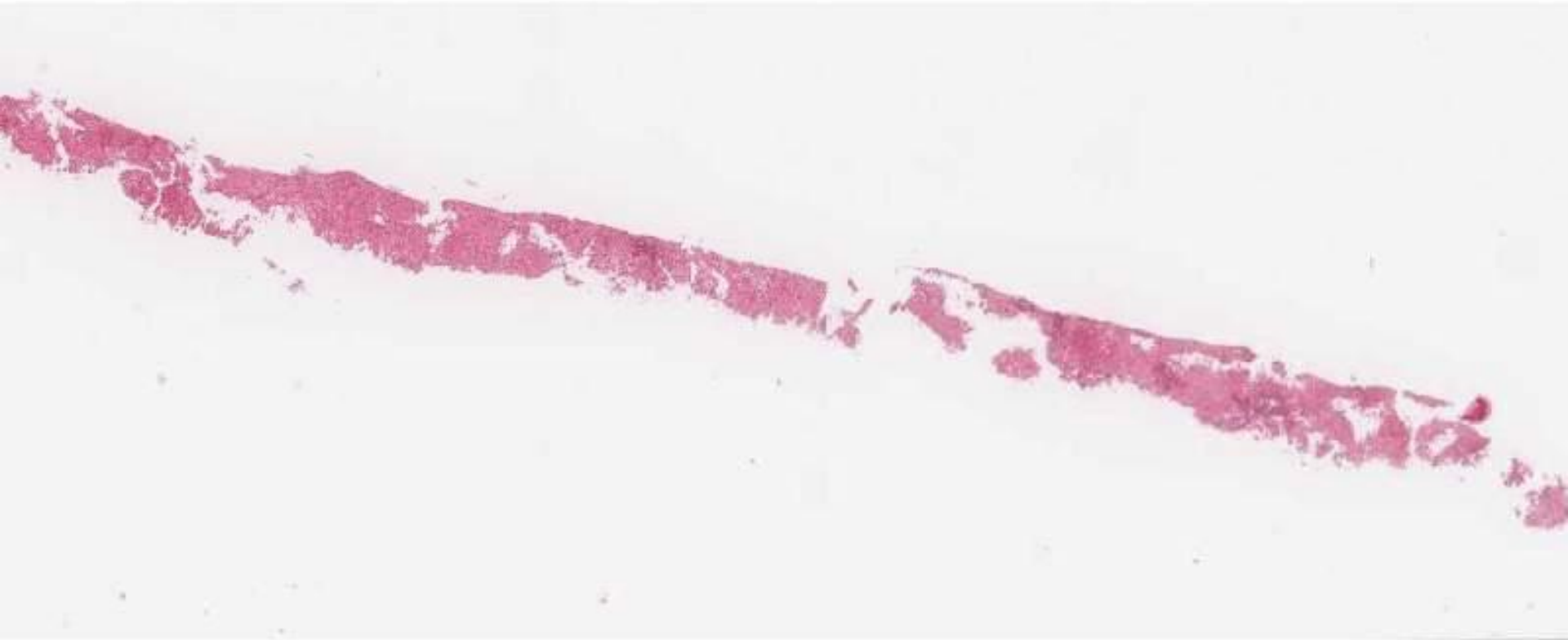
The biopsy shows characteristics of FNH, albeit with an unusually large central scar. FNH features were apparent in the adjacent parenchyma, so this was more than just a sclerosed haemangioma. There was no clinical history of prior embolisation for this lesion.

Case 226

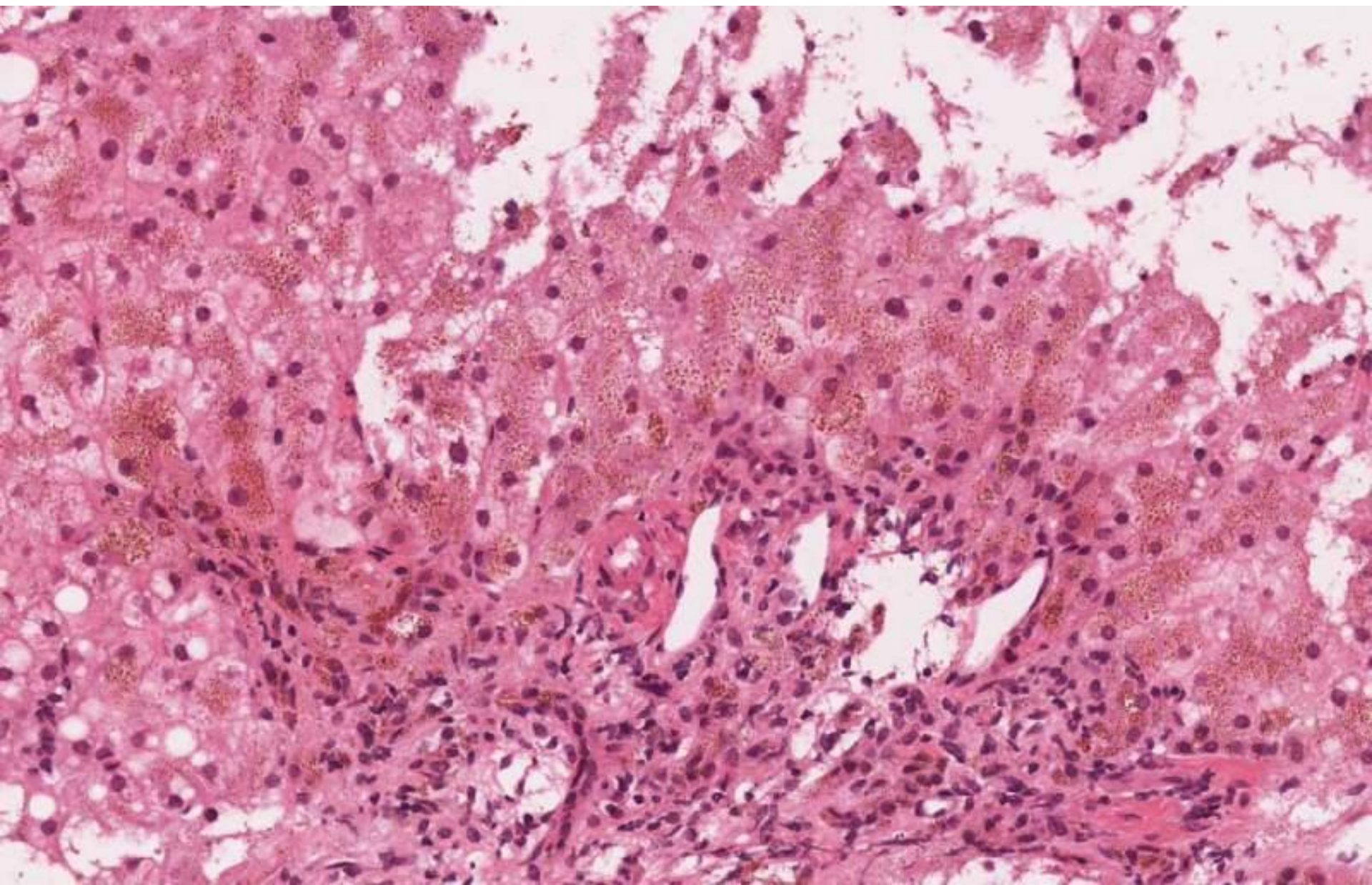
M40

Abnormal LFTs for 1 year. ALT increased. Positive anti-smooth muscle antibodies Ferritin increased. Negative viral screen. Perls stain strongly positive for Iron.

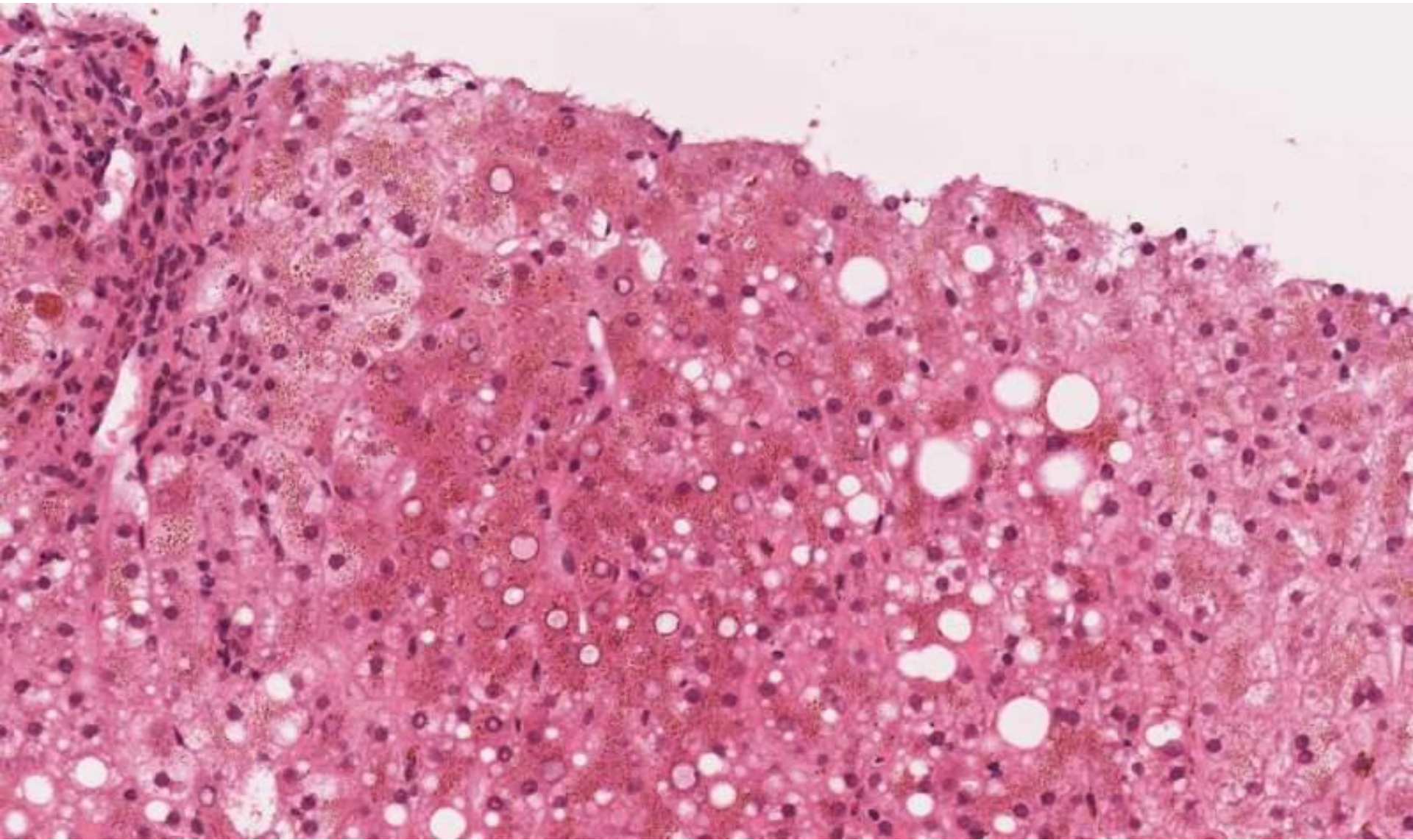
Case 226



Case 226



Case 226



Case 226

Results:

37 haemochromatosis, or consistent with haemochromatosis

6 haemochromatosis + steatosis

4 haemochromatosis + steatohepatitis

2 haemochromatosis +?AIH

4 haemosiderosis with fatty change, ? alcohol related (no mention of haemochromatosis)

1 autoimmune hepatitis,+ test for haemochromatosis

1 CAH ? history of genetic haemochromatosis

1 steatohepatitis, ?autoimmune, ?haemochromatosis

1 siderosis; steatosis/steatohepatitis, ?alcohol HFE typing

1 looks like PBC

Case 226

Comments:

Genetic test for haemochromatosis – 31

No evidence of AIH - 13

More tests for AIH, ANA titres – 4

May be consistent with AIH – 1

Exclude alcohol – 4

Exclude PCT – 1

Case 226

Discussion

Accept any with haemochromatosis or siderosis suggests HFE typing as main diagnosis.

Reject haemosiderosis with no mention of haemochromatosis or genetic typing and diagnoses where the main pathology implied was autoimmune hepatitis.

Comments:

The history of smooth muscle antibodies raise the possibility of autoimmune hepatitis but there was felt to be no histological evidence of that diagnosis on the biopsy. There is no indication therefore to treat the patient for autoimmune hepatitis.

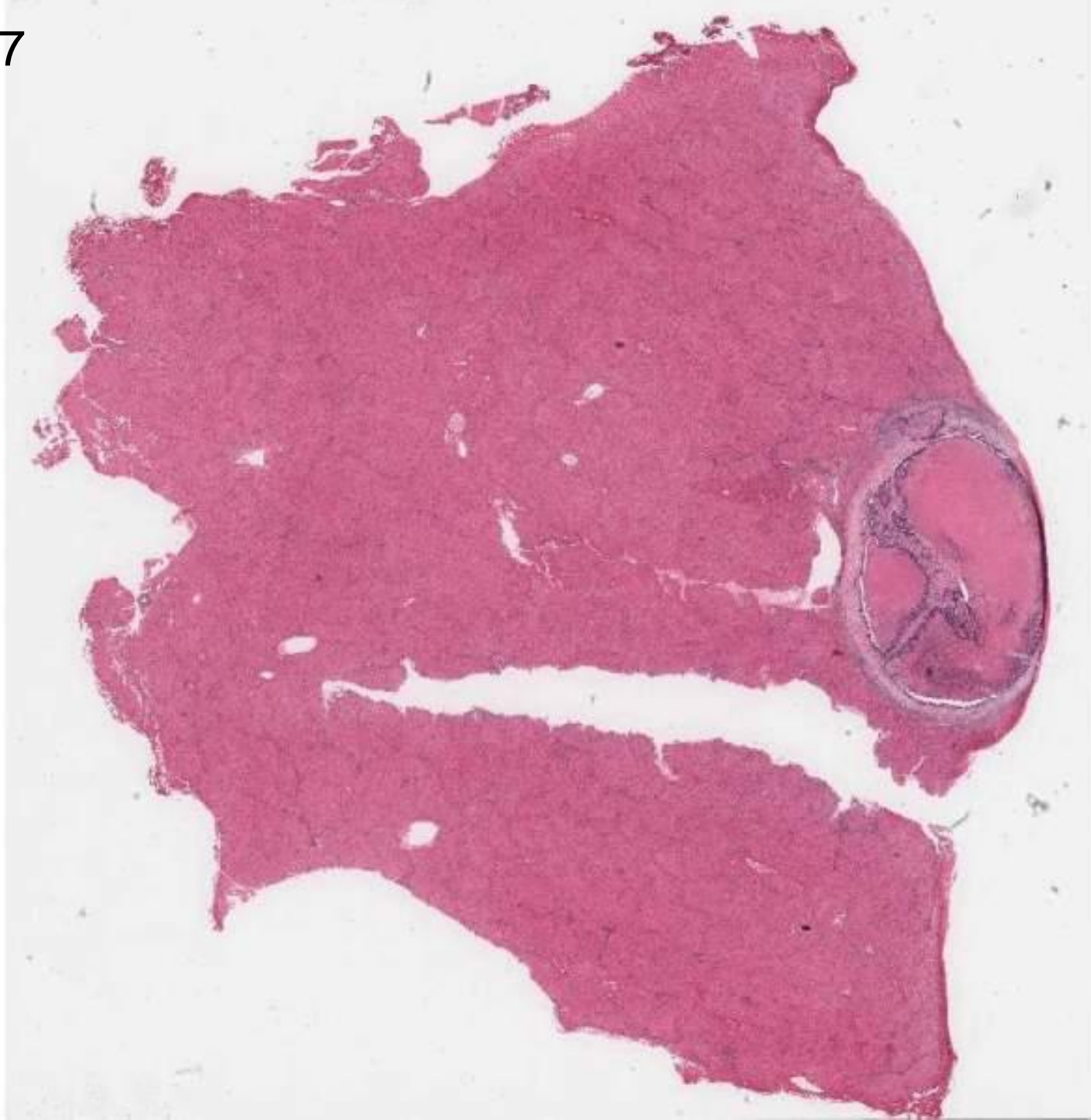
Case 227

F60

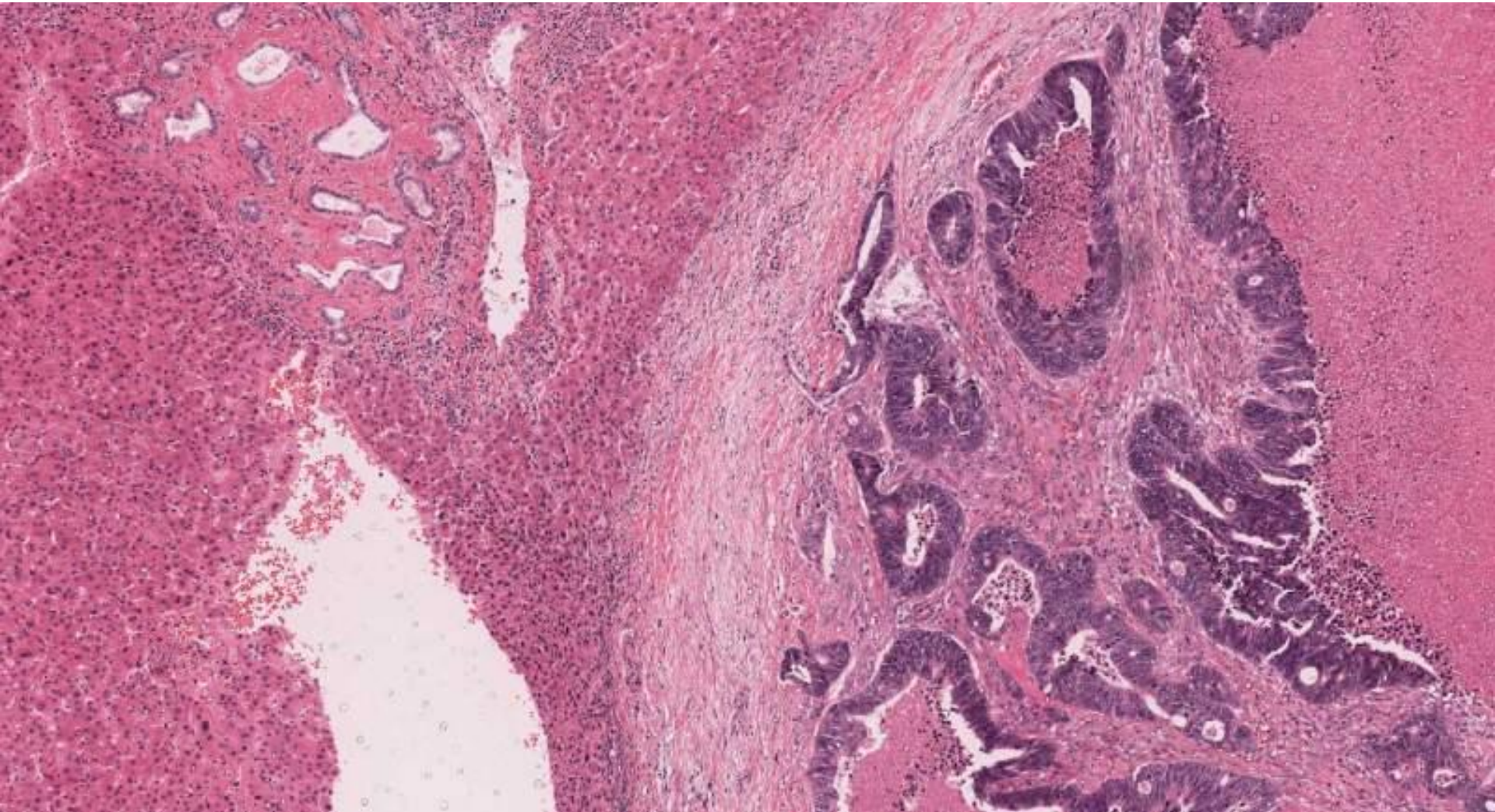
Previous Dukes' C adenocarcinoma of sigmoid colon 1991 (12 years previously). Also previous gastrointestinal stromal tumour (5cm diameter, low mitotic rate) in stomach one year previous.

Wedge resection, 3x2x2cm, central white nodule 6mm diameter

Case 227



Case 227



Case 227

Results:

51 metastatic adenocarcinoma, consistent with large bowel primary site

1 metastatic adenocarcinoma, consistent with GIT primary site

4 metastatic adenocarcinoma; no mention of primary site

2 + bile duct hamartoma

1 + acute hepatitis, ?sclerosing cholangitis, vasculitis

Case 227

Comments:

Do CK20 – 17 (1 person – *this information should have been provided*)

Possible new primary in view of the long interval to recurrence - several

*Follow up: (Dr Sheehan) – metastatic carcinoma, consistent with colonic primary;
(sufficiently characteristic that immunos were not considered necessary).*

Case 227

Discussion:

Accept all except acute hepatitis, ?sclerosing cholangitis.

Follow up: (Dr Sheehan) – metastatic carcinoma, consistent with colonic primary; (sufficiently characteristic that immunos were not considered necessary).

Comments:

CK 20 not generally thought to be necessary for diagnosis with this very characteristic histological pattern of metastatic colorectal cancer.

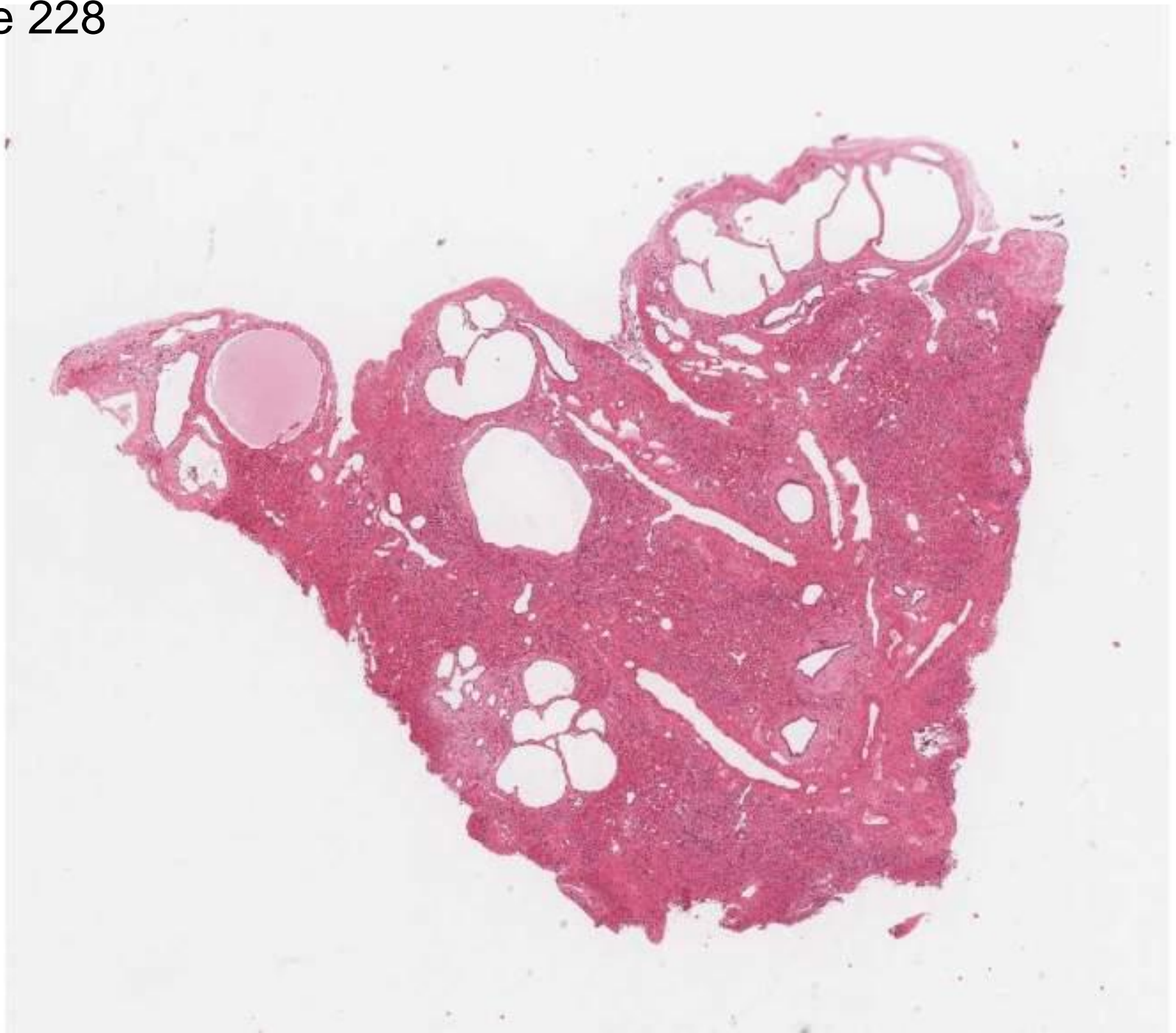
Case 228

F63

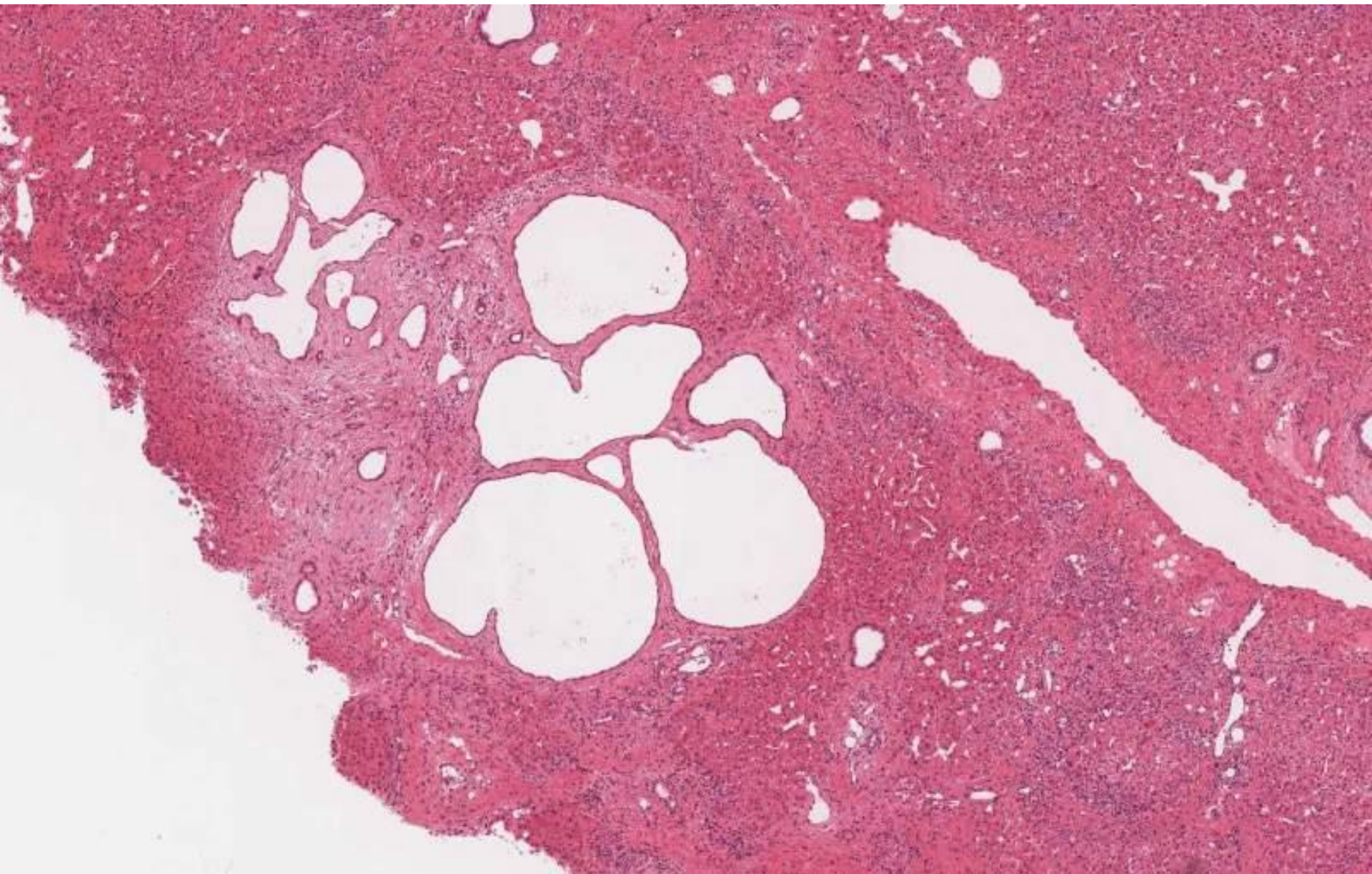
End stage renal failure.

Liver found to be thickened and cystic at incisional
hernia repair.

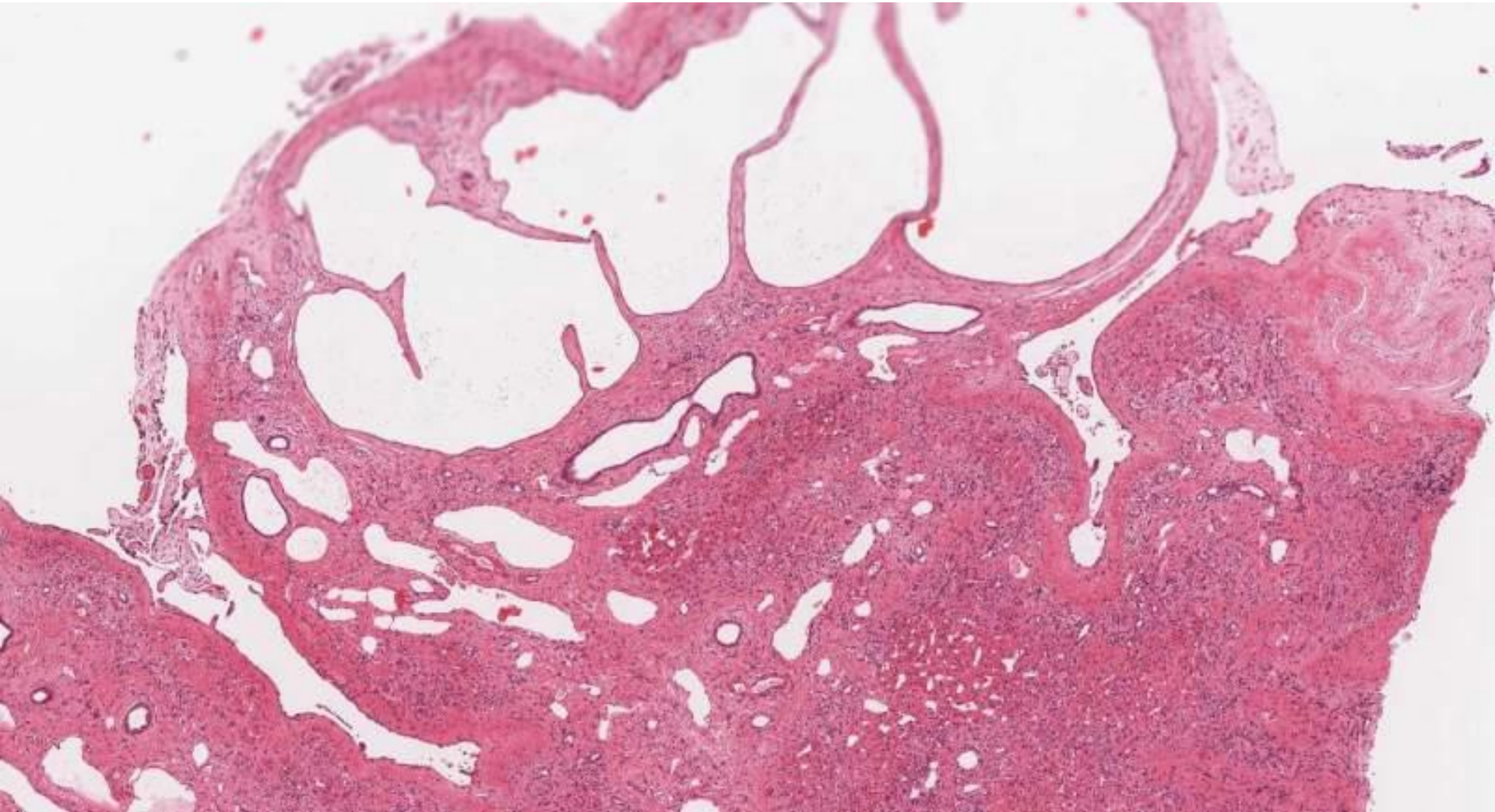
Case 228



Case 228



Case 228



Case 228

Results:

39 Cystic liver, adult polycystic renal disease

17 fibropolycystic liver disease (renal not mentioned)

1 von Meyenberg complex/hamartoma

1 cystic spaces, ?haemangioma/lymphangioma ?multicystic liver

1 didn't see slide

Discussion

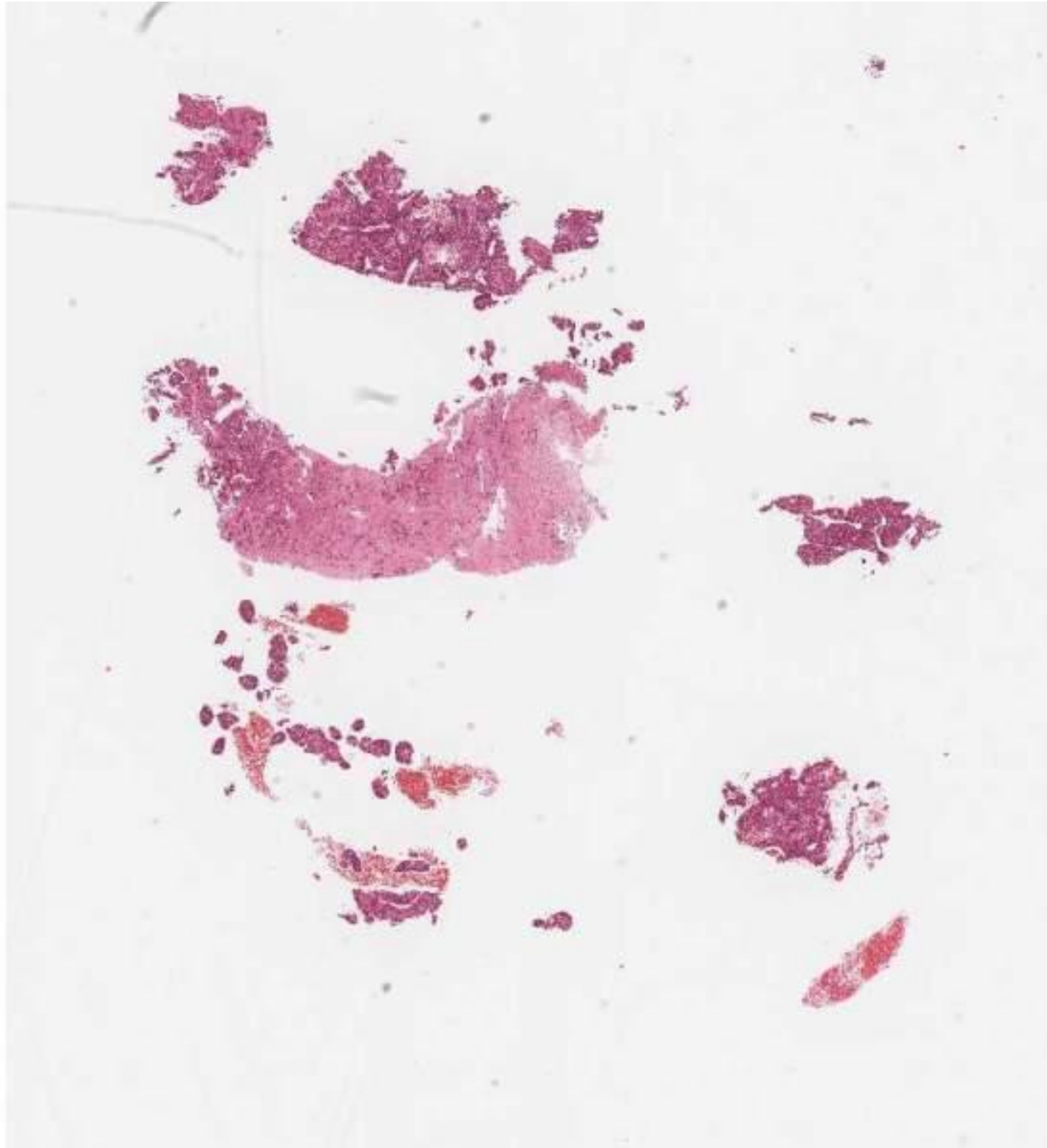
Accept all except von-Meyenberg complex and
?haemangioma/lymphangioma.

Case 229

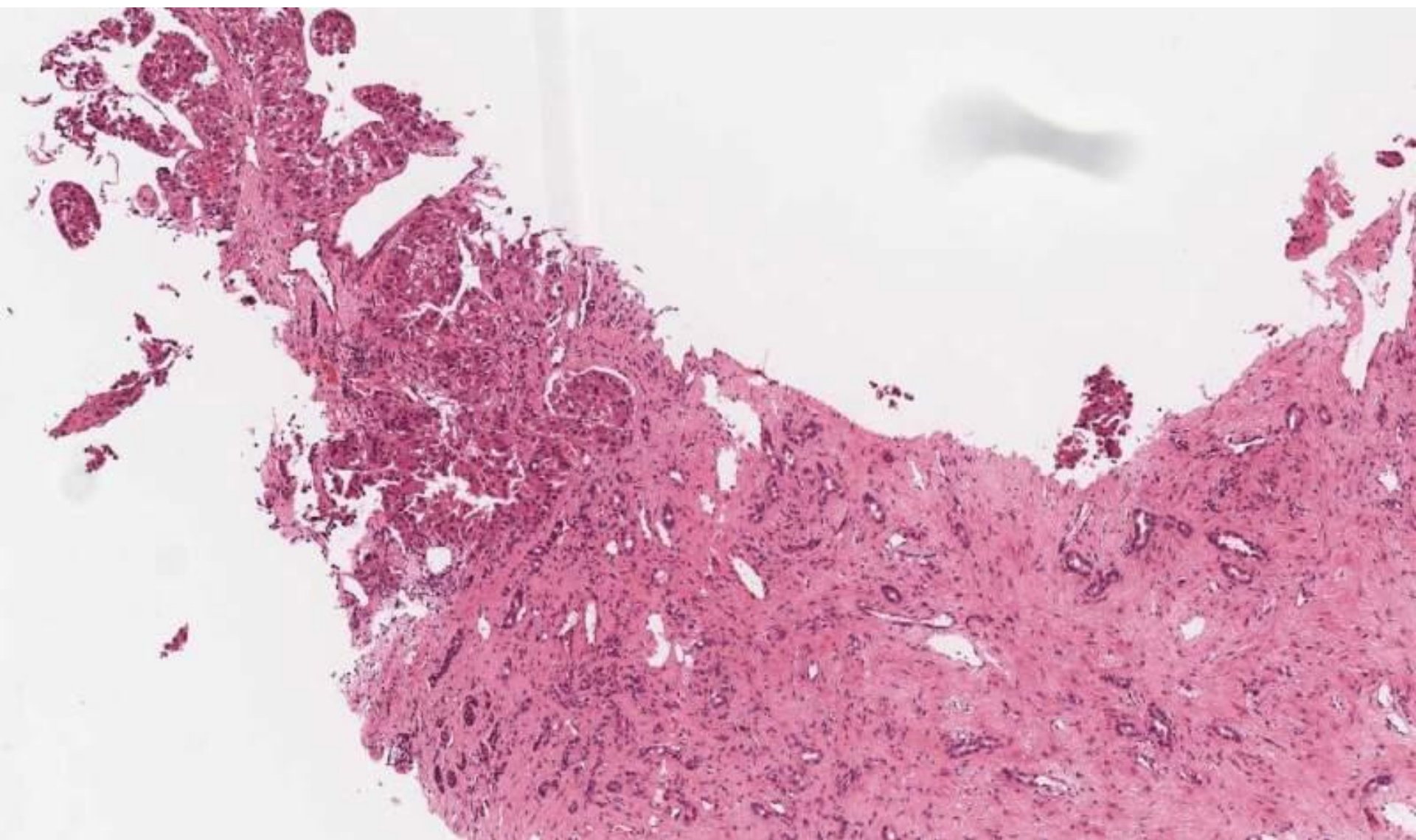
M65

? metastasis in liver

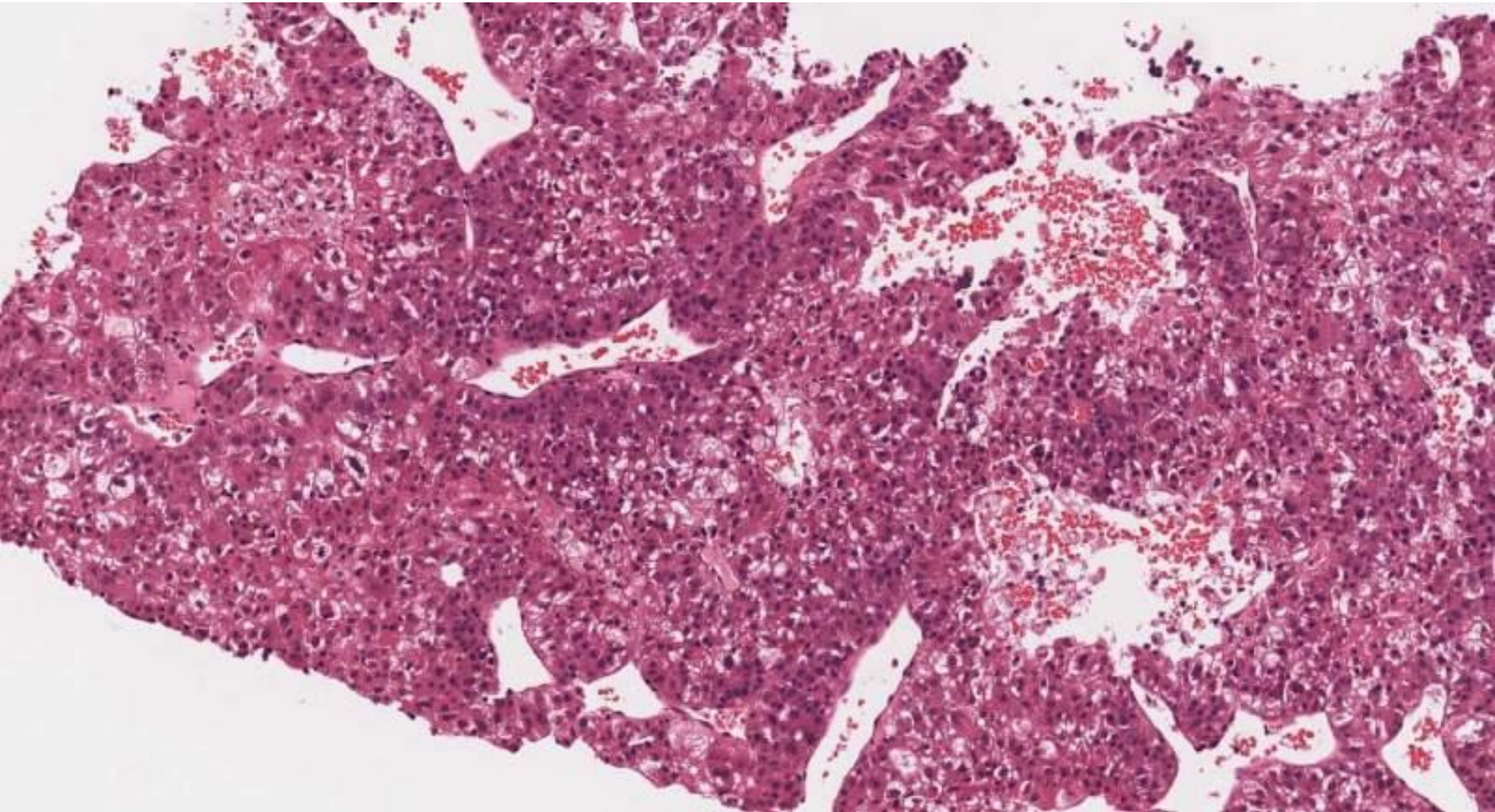
Case 229



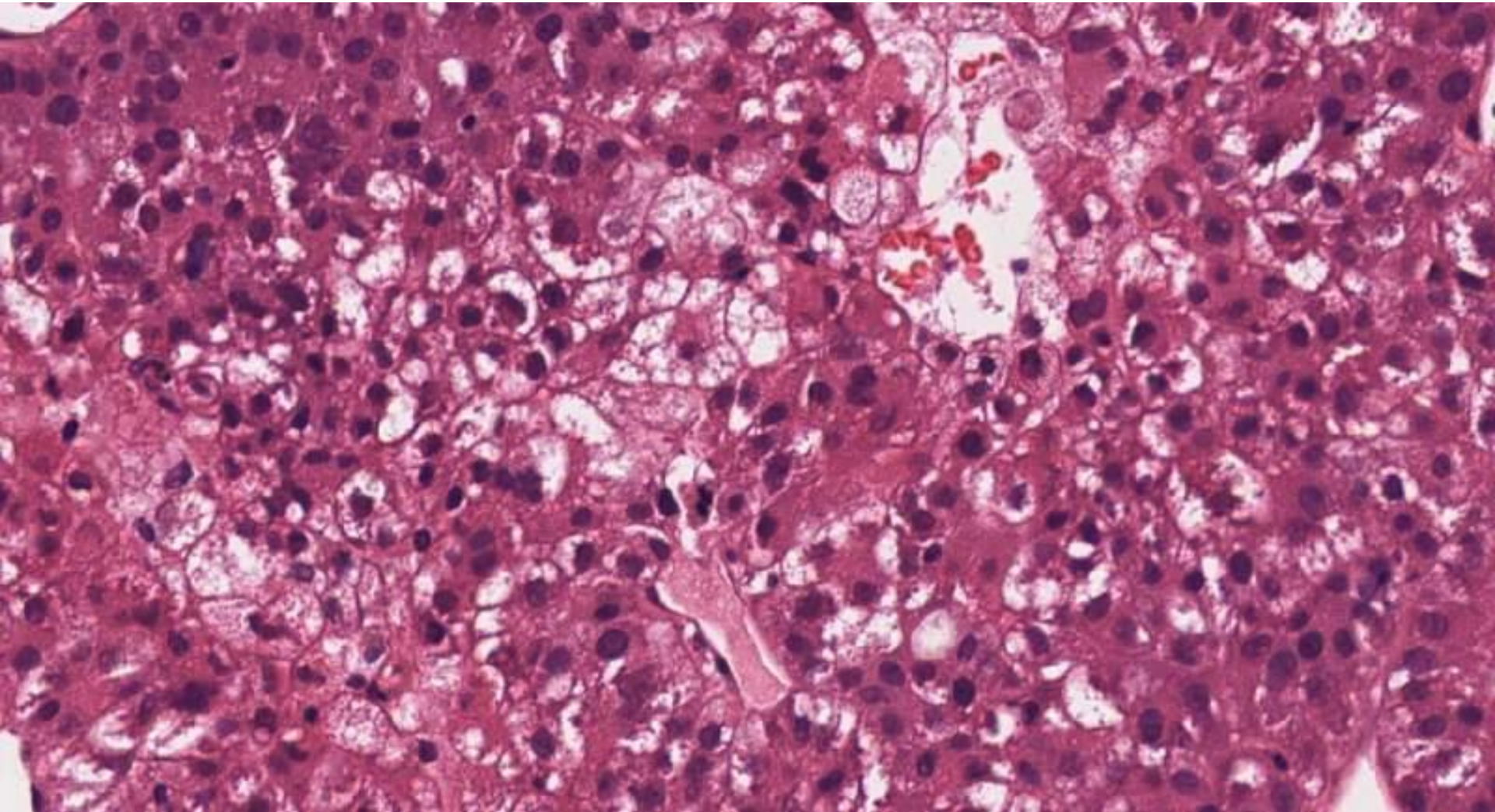
Case 229



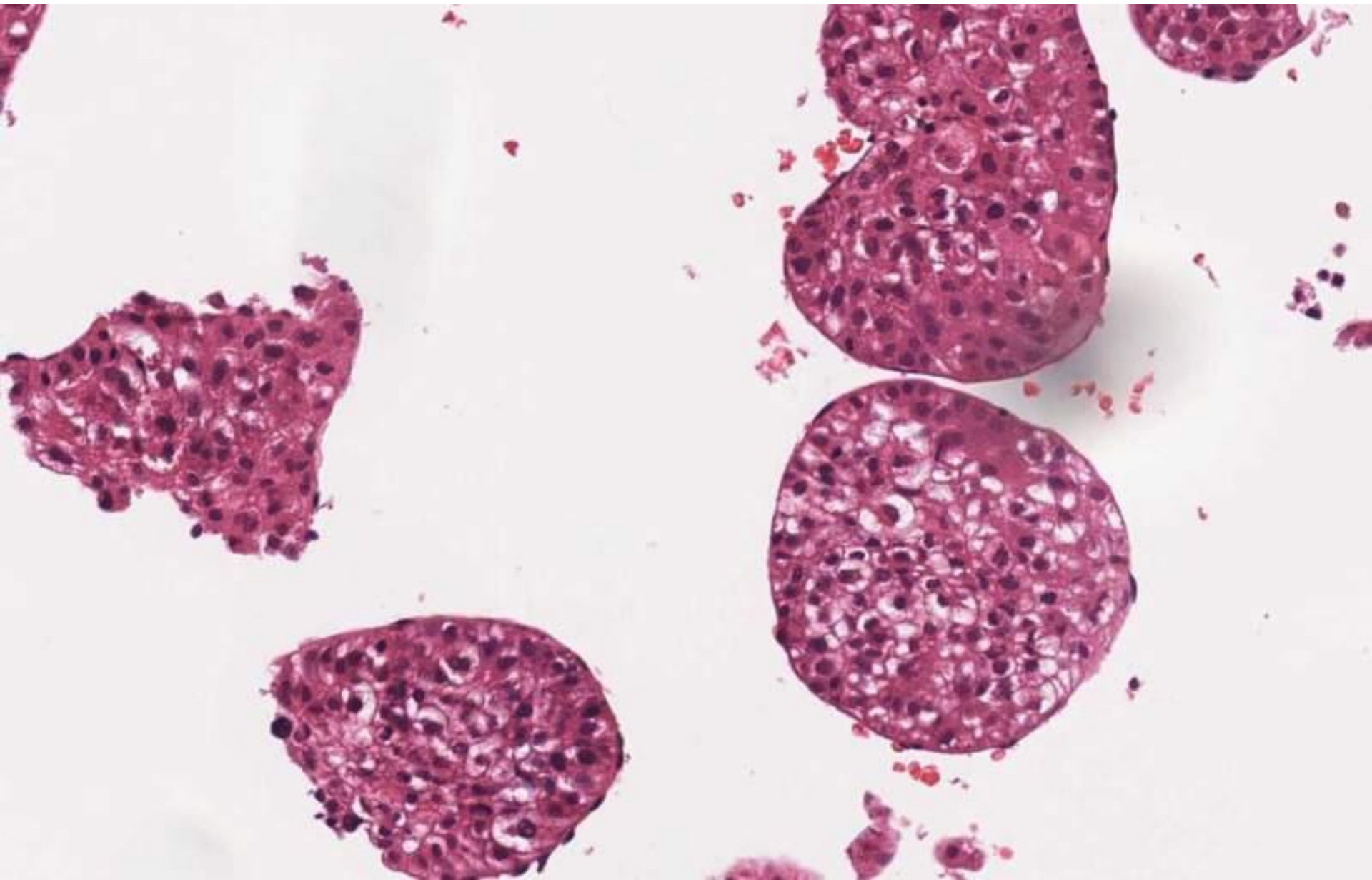
Case 229



Case 229



Case 229



Case 229

Results:

45 hepatocellular carcinoma

5 hepatocellular carcinoma (do immunos to exclude renal)

1 carcinoma, HCC favoured over metastasis

1 HCC or adenoma

1 clear cell carcinoma, favour renal over HCC

1 metastatic renal cell carcinoma

Comments:

Some sort of immunohistochemistry – 29

Immunohistochemistry not mentioned - 25

Case 229

Discussion

Reject 'HCC or adenoma', and 'clear cell carcinoma favouring renal over HCC'.

Comments:

The trabecular architecture, and particularly the isolated round groups of tumour cells surrounded by endothelium were felt to be so characteristic of hepatocellular carcinoma as to make this the overwhelmingly likely diagnosis. The architecture and cytology were not consistent with adenoma. There was a spread of views on the appropriateness of immunohistochemistry but there was not time to discuss this aspect of the case.