United Kingdom Liver Pathology Group

Liver histopathology EQA Scheme

Circulation LQ Autumn 2017

www.virtualpathology.leeds.ac.uk/eqa/specialist/liver/index.php

Virtual Pathology at the University of Leeds

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UK Liver Pathology Group

The UK Liver Pathology Group (UKLPG) was formed in 2016 with the purpose:

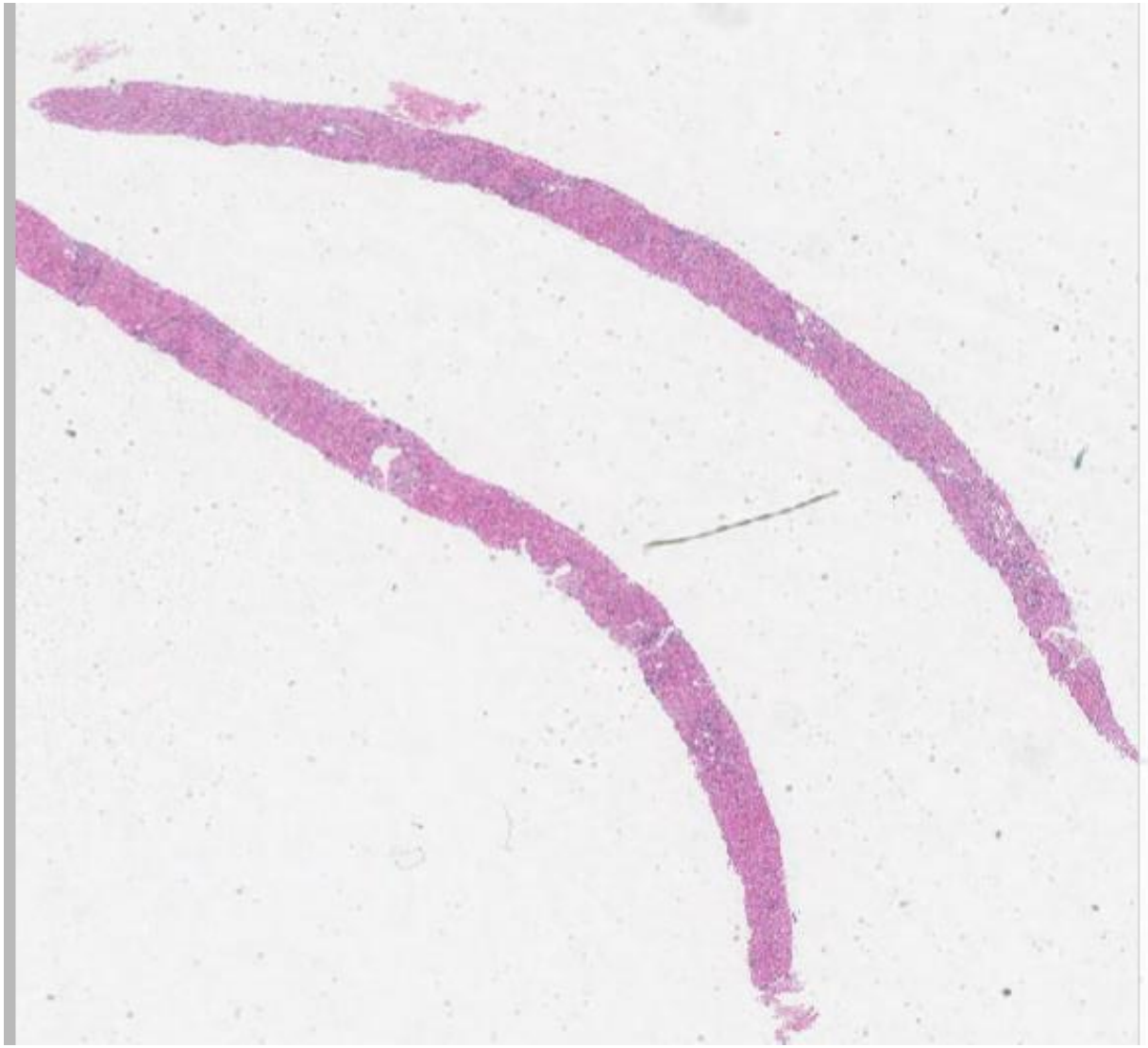
To promote excellence in liver histopathology services in the UK and Ireland, across all levels of specialisation, through professional collaboration in education, quality assurance and research.

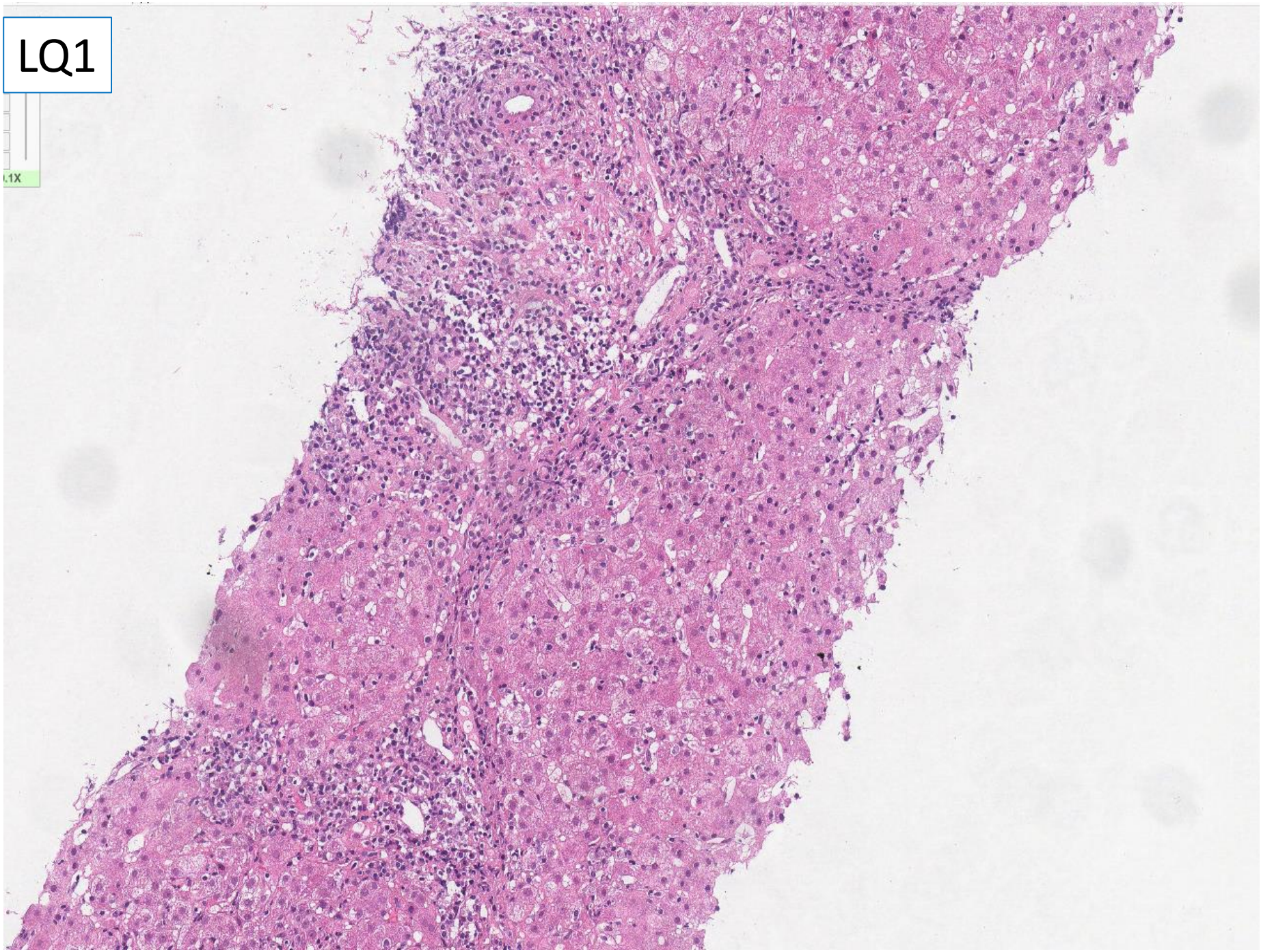
United Kingdom Liver Pathology Group

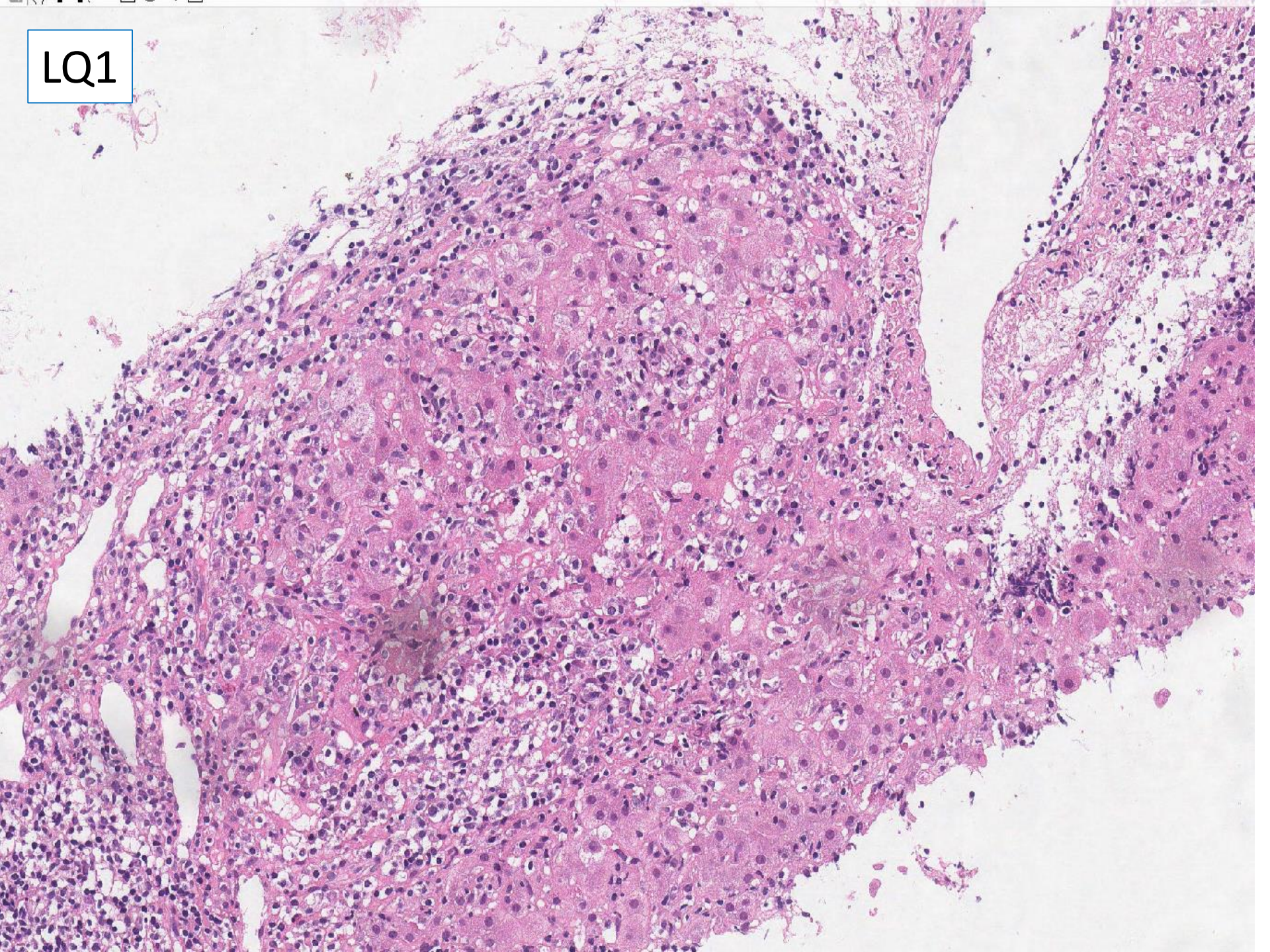
- Future CPD activities - including links for registration
- Previous CPD archive (activities from 2006 with CPD resources available)
  - Includes an original series of seminars presented by the late Professor Peter Scheuer
  - Lectures for trainee pathologists and students
  - Liver lectures from other UK CPD meetings
- UK Liver Pathology EQA Scheme
- UKLPG - background and UKLPG committee meetings
- UKLPG - membership + how to join UKLPG
- Subcommittees
- Reference images for liver biopsy reporting
- Liver transplant pathology
- Paediatric liver pathology section
- Other professional documents
- Links to other sites

Case LQ1 60F

ALT 1102; Gamma GT 84; Raised IgG. Alk phosphatase normal.
ANA positive 1:80; Anti Smooth muscle AB positive. AMA negative. IgM normal.
History breast carcinoma




LQ1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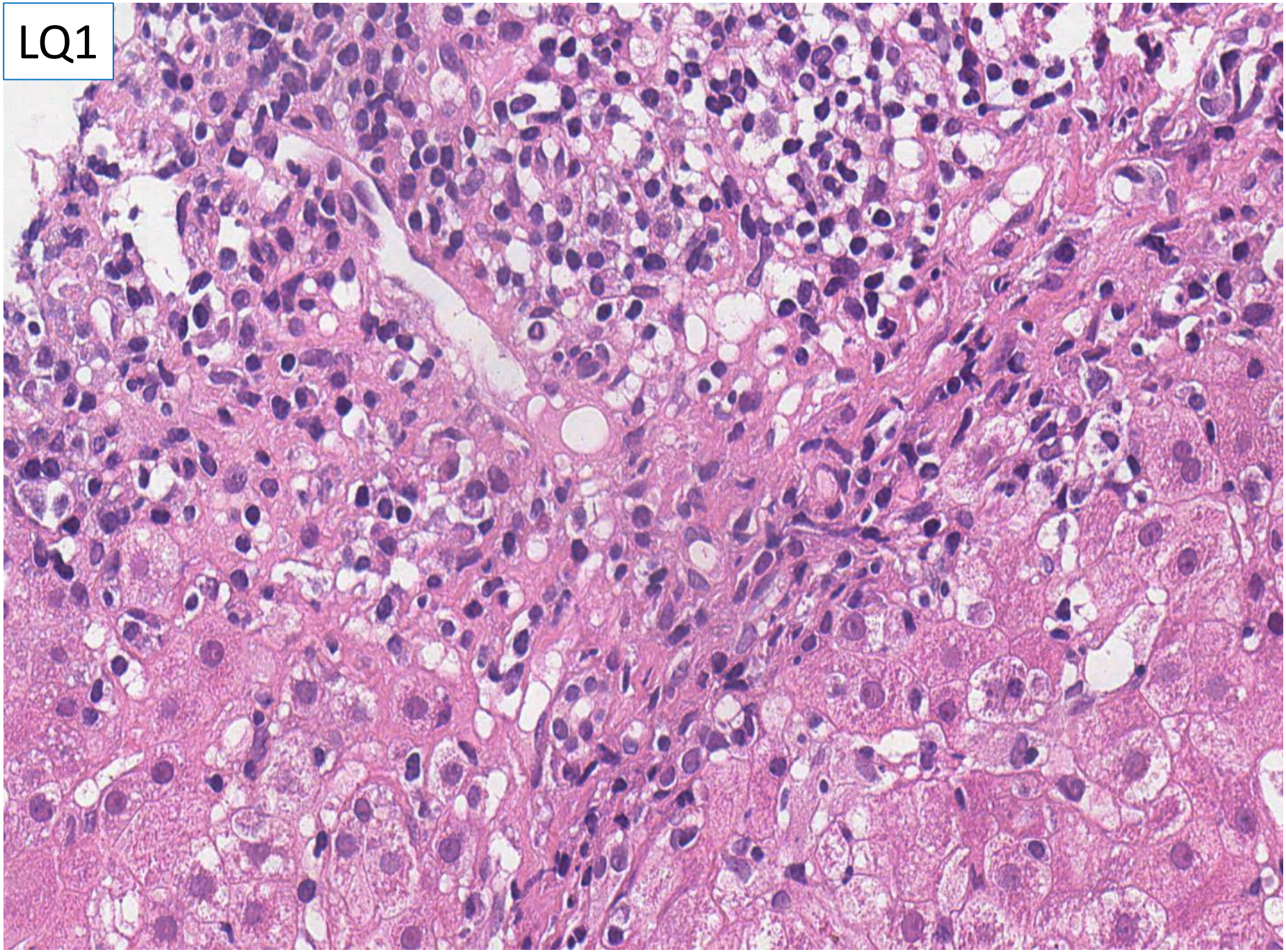
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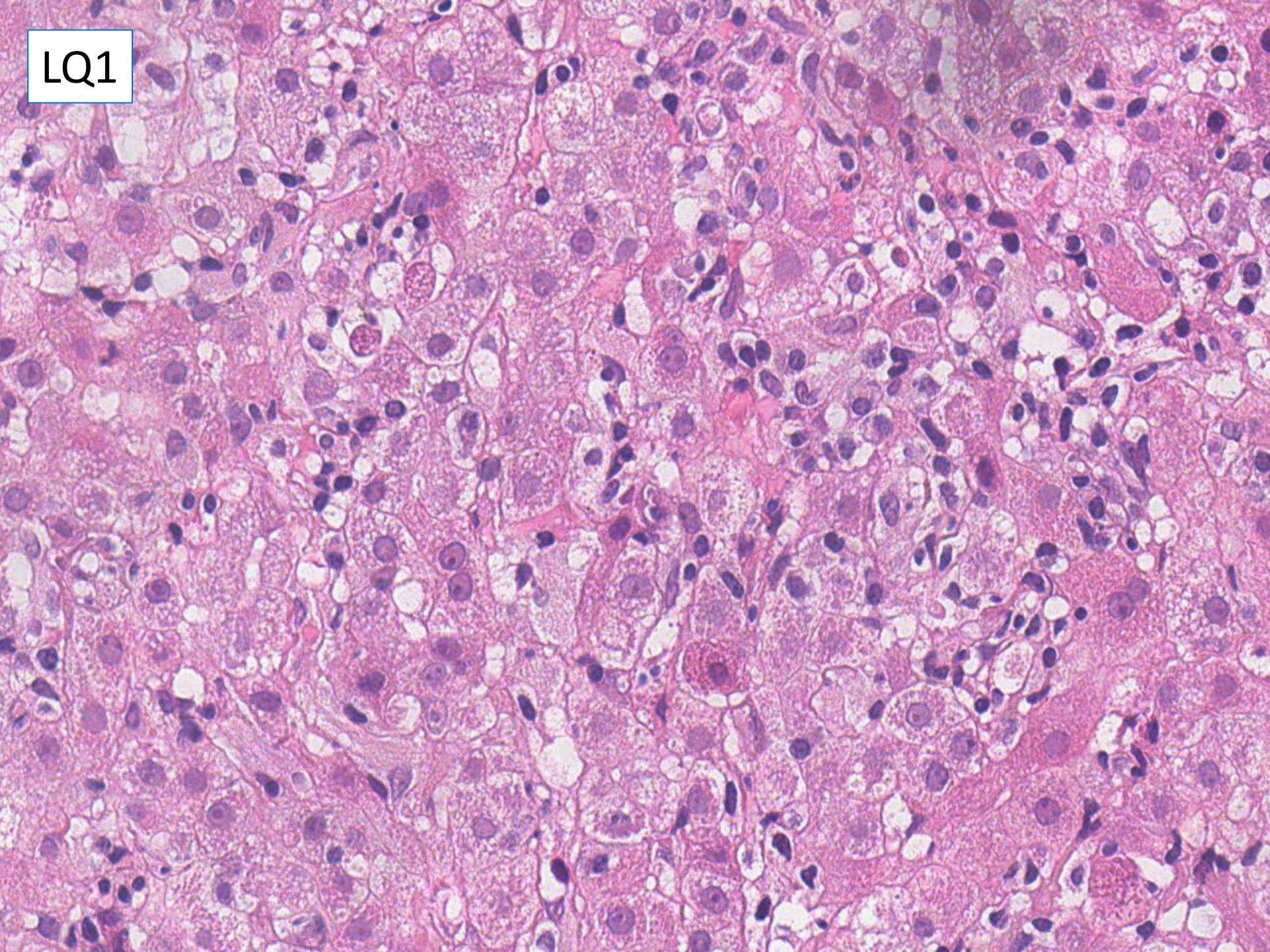
LQ1

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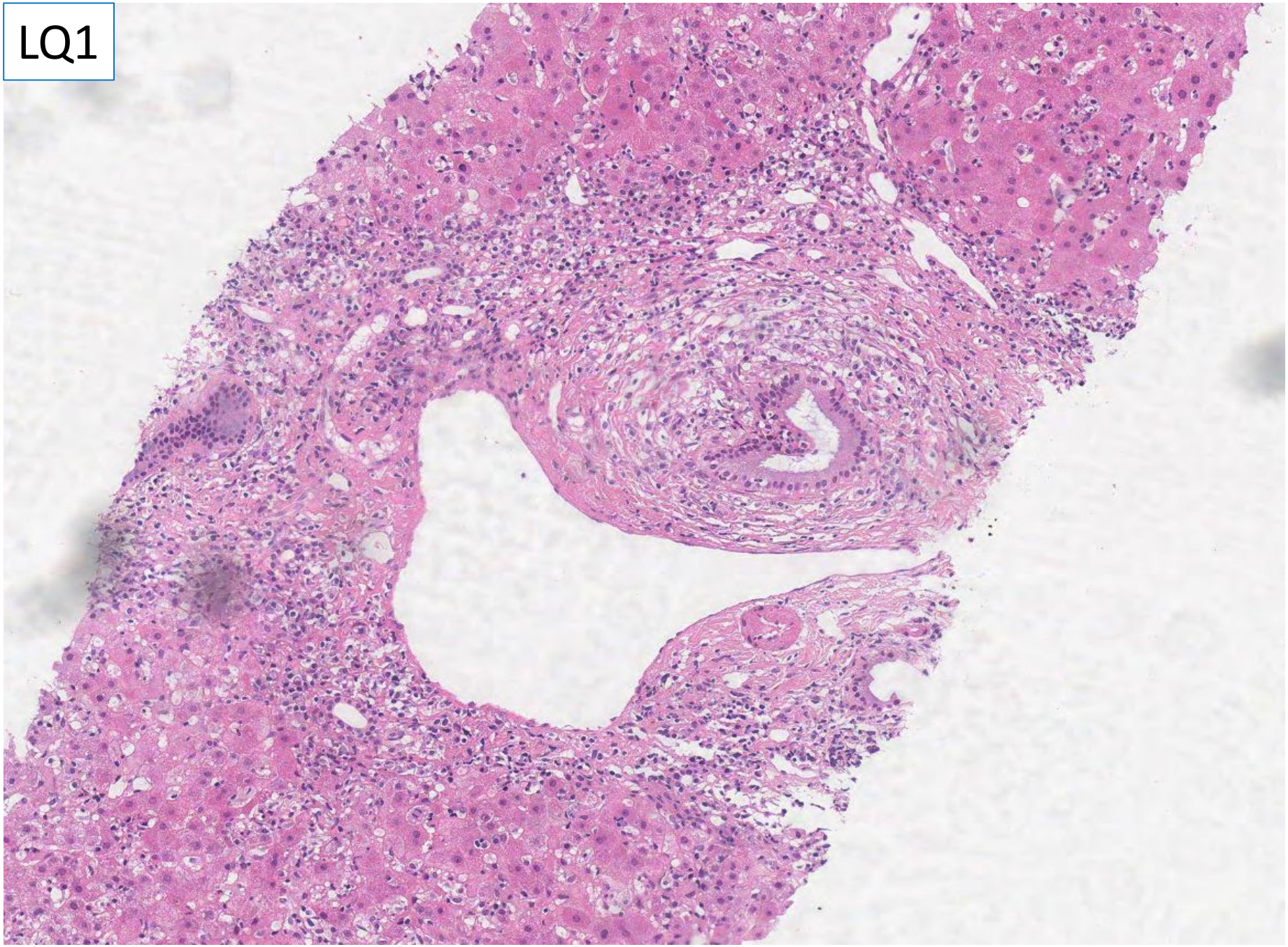
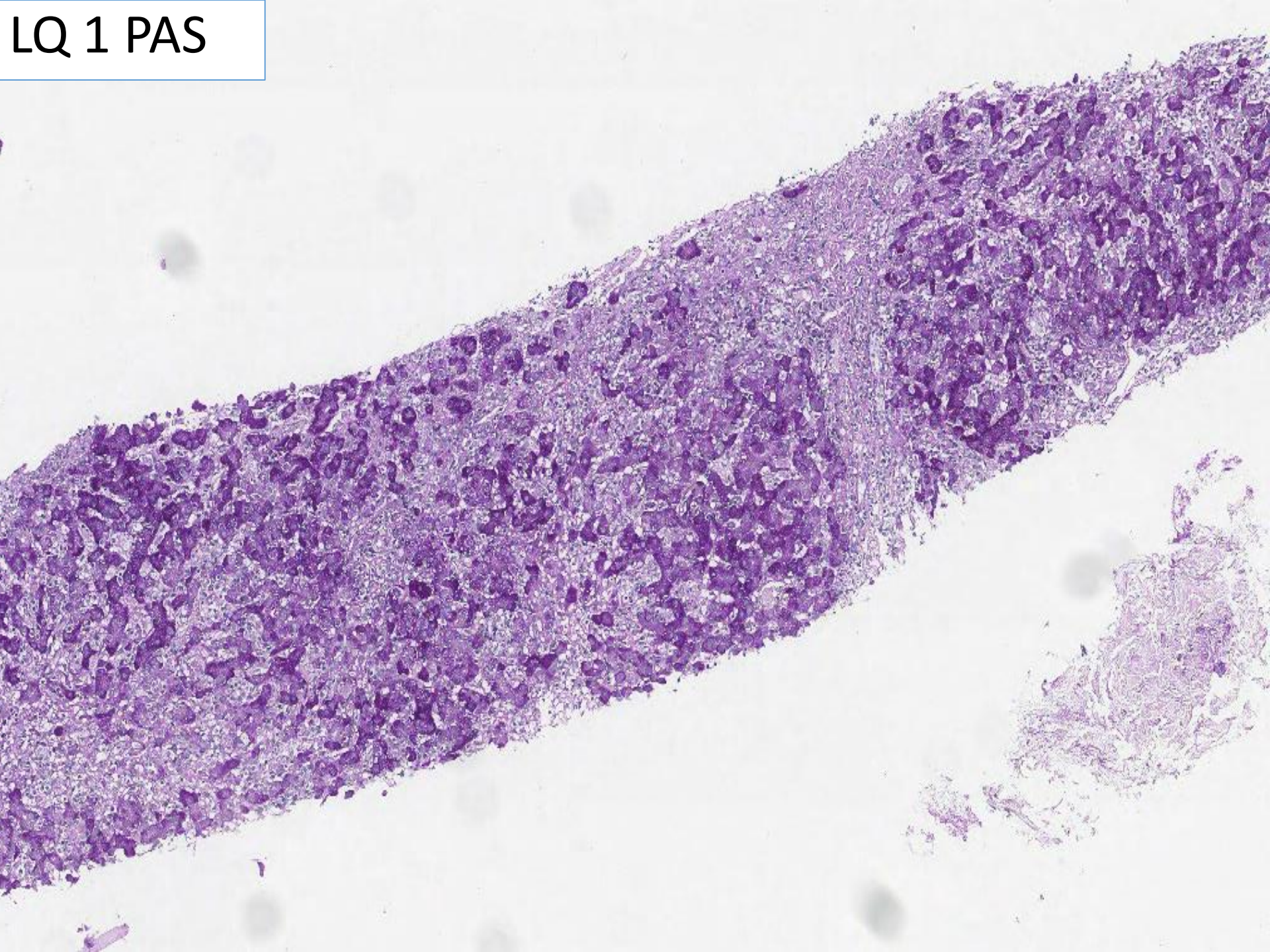
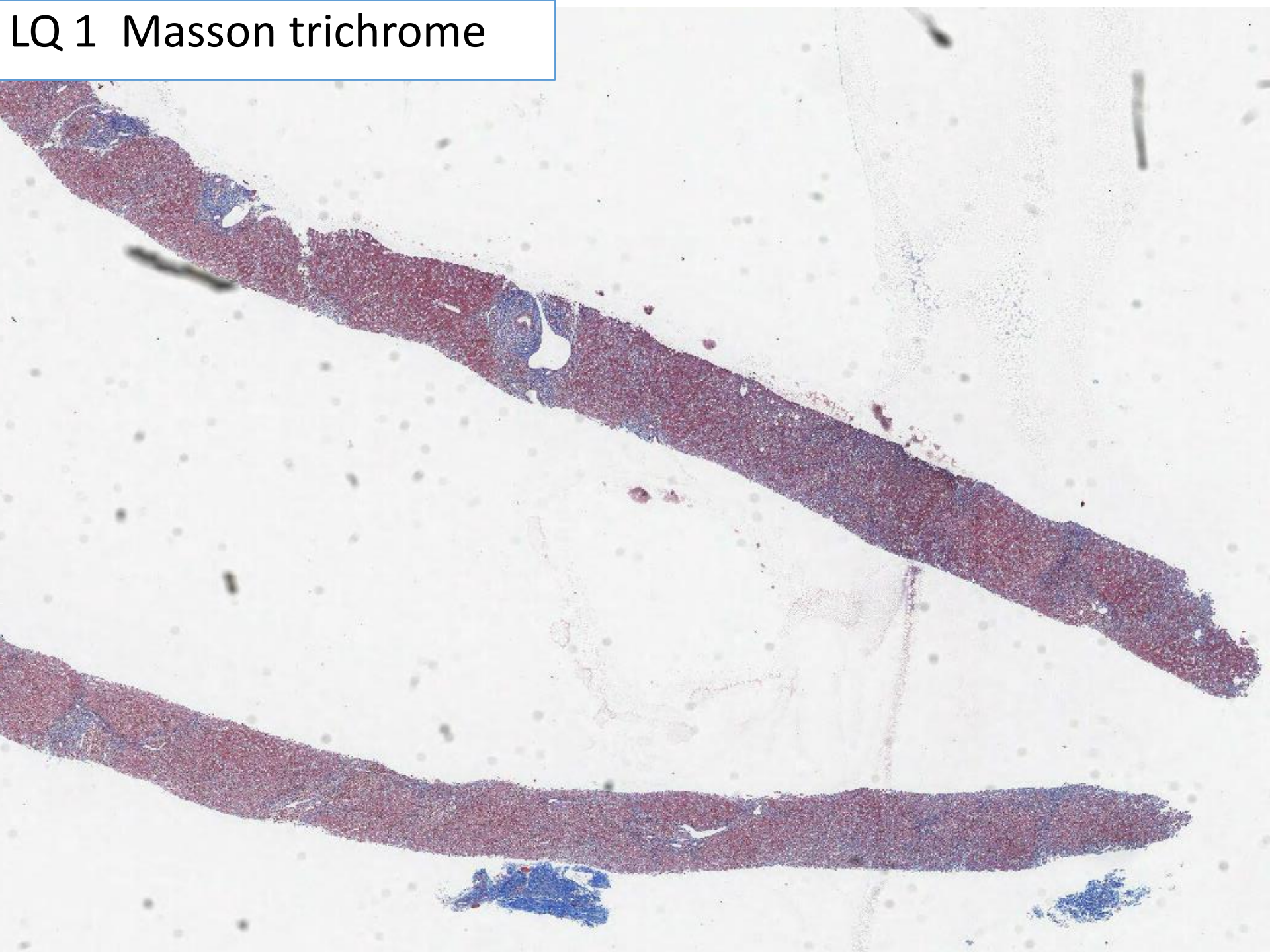


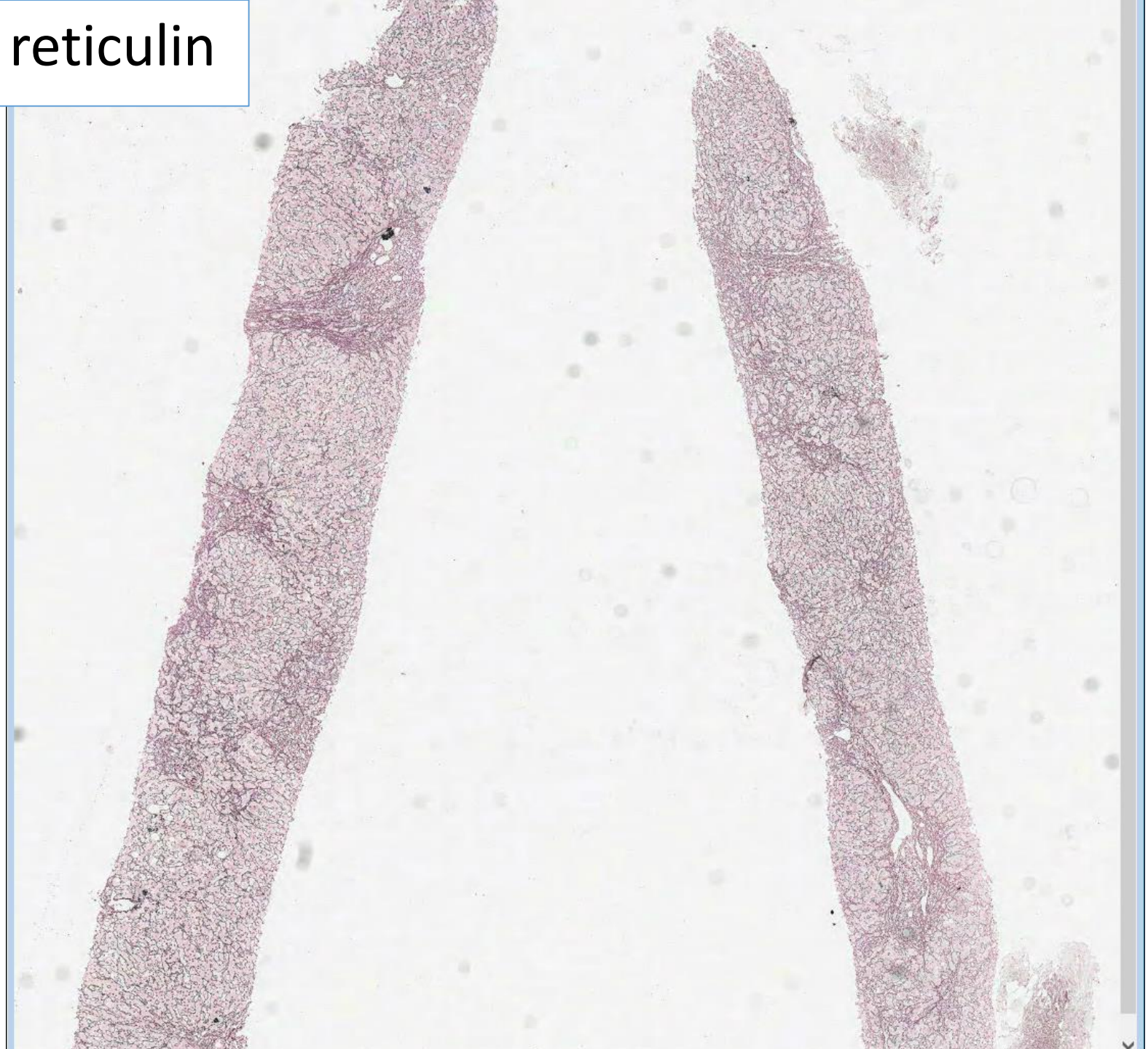
LQ 1 PAS



LQ 1 Masson trichrome



LQ 1 reticulin



Case LQ1 60F

ALT 1102; Gamma GT 84; Raised IgG. Alk phosphatase normal.
ANA positive 1:80; Anti Smooth muscle AB positive. AMA negative. IgM normal.
History breast carcinoma

Acute hepatitis c/w autoimmune hepatitis – *with any comment indicating necrosis/fibrosis/cirrhosis*	74
Of which: Confluent necrosis with bridging necrosis/collapse	32
No fibrosis, but bridging necrosis/collapse	9
States there is no fibrosis, and doesn't mention confluent/ bridging necrosis	2
Fibrosis, NOS	18
Bridging fibrosis	31
Probable/definite cirrhosis	4
Of the above: Both acute necrosis/collapse and fibrosis	16
Acute hepatitis c/w autoimmune hepatitis – *with no comment on fibrosis/bridging necrosis*	9
Several commented on periductal fibrosis, ? PSC etc – not separately counted.	
Acute hepatitis with possible malignant infiltration	4
Primary biliary cholangitis, cirrhosis	2
Hepatitis viral infection/chronic active hepatitis, evolving cirrhosis (autoimmune not mentioned)	1

Consensus diagnosis:
acute hepatitis consistent with autoimmune hepatitis.

Recognition that there is bridging necrosis and/or fibrosis. 74 responses included an opinion about fibrosis or acute hepatitis with bridging necrosis, with a majority view favouring at least some component of fibrosis (53/83).

Several commented in some way on periductal fibrosis ? PSC, need for additional stains, but we have not separately recorded those.

Case LQ1 60F

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Case LQ1 60F

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ANA positive 1:80; Anti Smooth muscle AB positive. AMA negative. IgM normal.
History breast carcinoma

Agreed scoring:

For full marks – acute hepatitis, consistent with autoimmune hepatitis and with some comment about architectural disturbance with a bridging process. Most considered there to be some fibrosis, but insufficient consensus on bridging necrosis v. fibrosis.

Score 5 marks if states no fibrosis, but gives no indication that there is bridging necrosis, or makes no comment about fibrosis/necrosis.

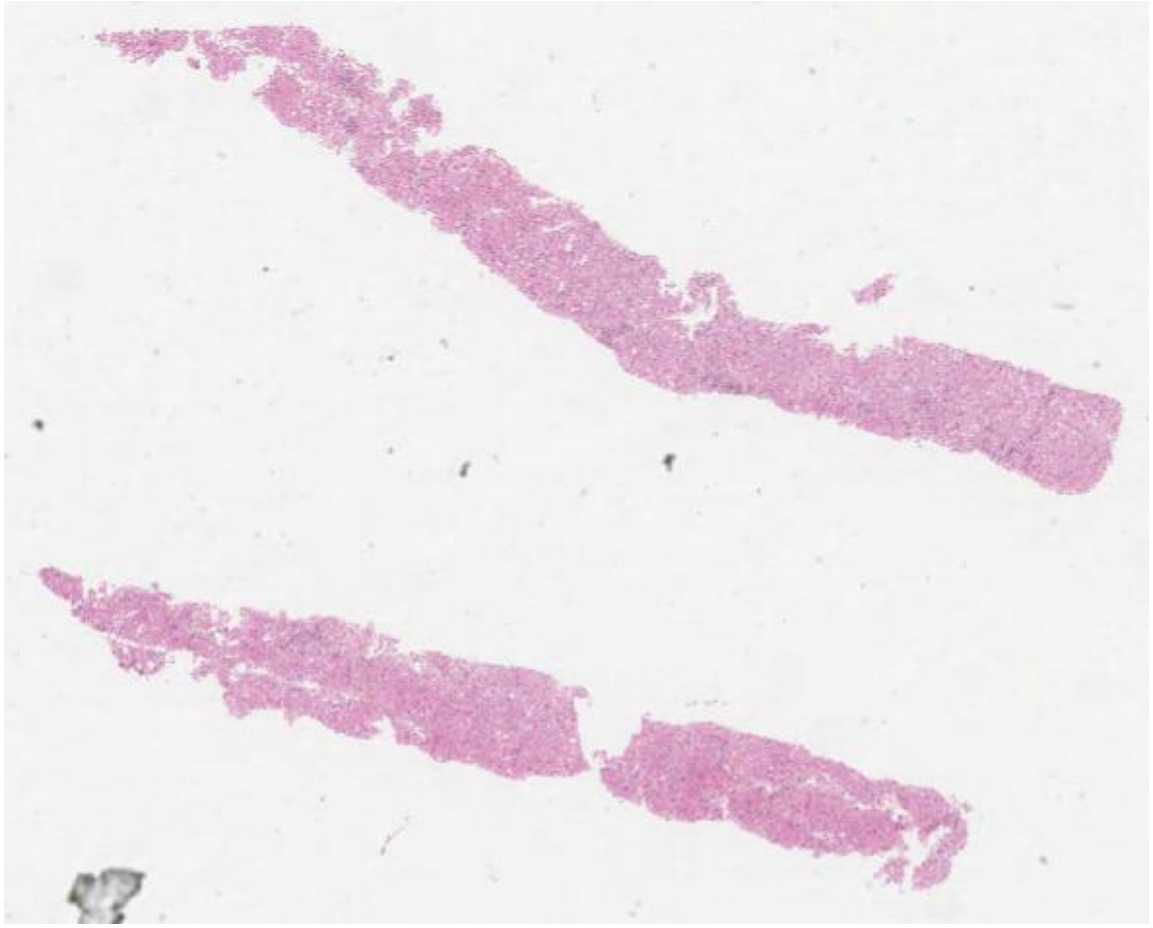
On a show of hands - to score no points for a diagnosis of probable or definite cirrhosis, or for acute hepatitis with possible malignant infiltration.

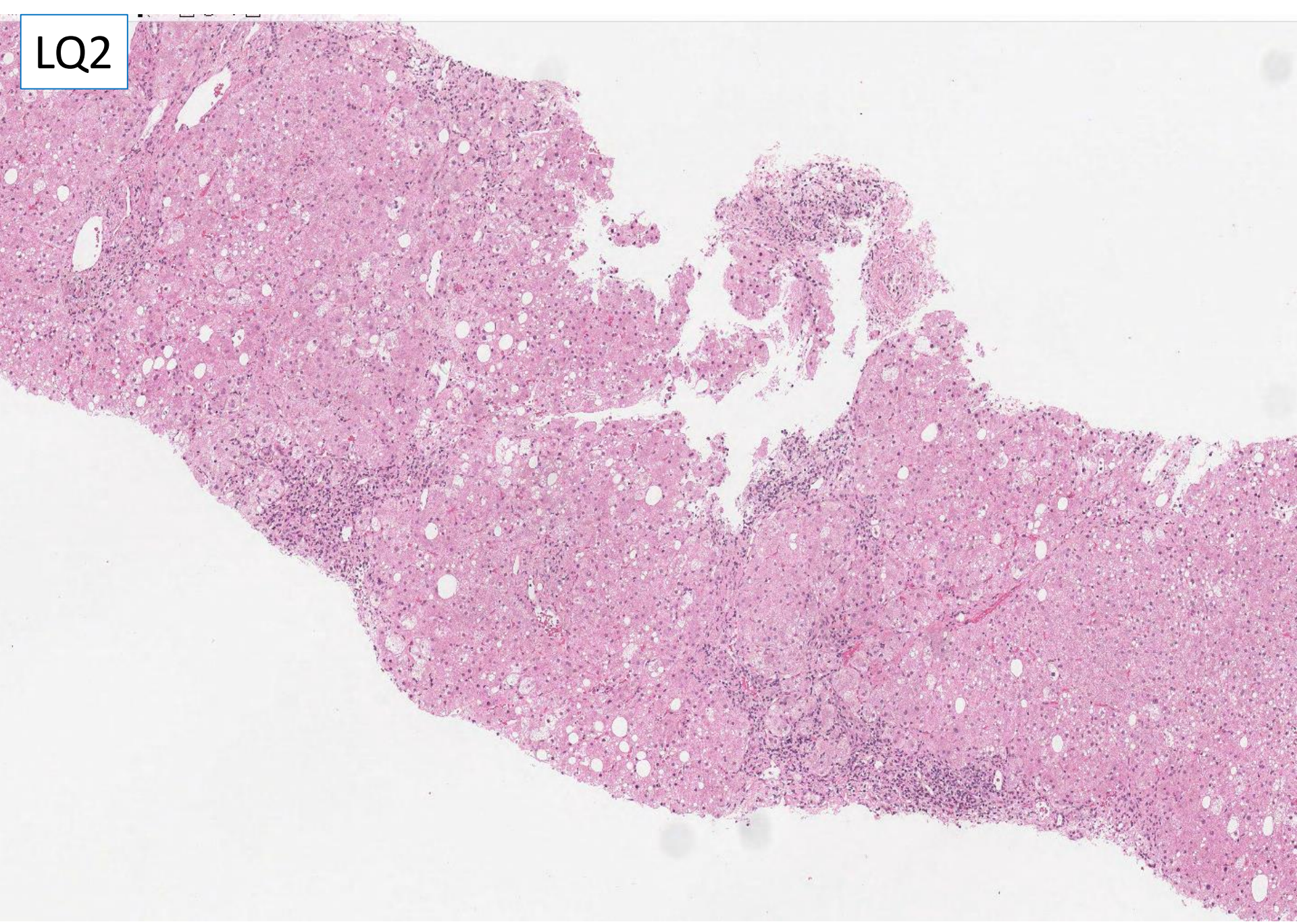
Also – no points for unequivocal PBC, cirrhosis, or viral infection/chronic active hepatitis with no mention of autoimmune hepatitis.

Discussion focussed on the terminology of bridging necrosis – which is regarded as always involving central veins, whereas bridging fibrosis may be purely portal-portal.

Case LQ2 63F

Chronic pancreatic insufficiency. Type 2 diabetic on insulin, Deranged LFTs, fatty liver on ultrasound, elevated fibroscan

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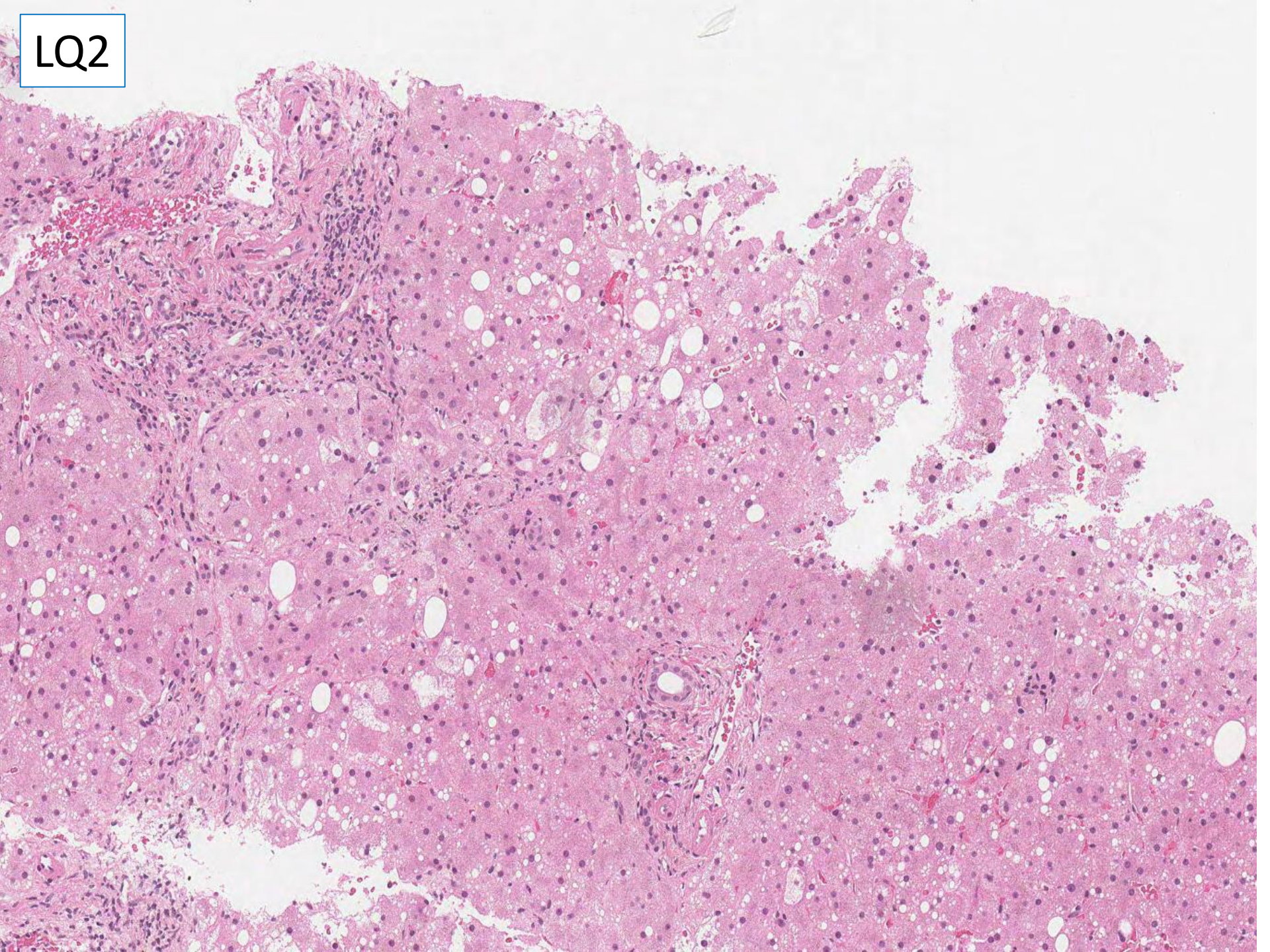
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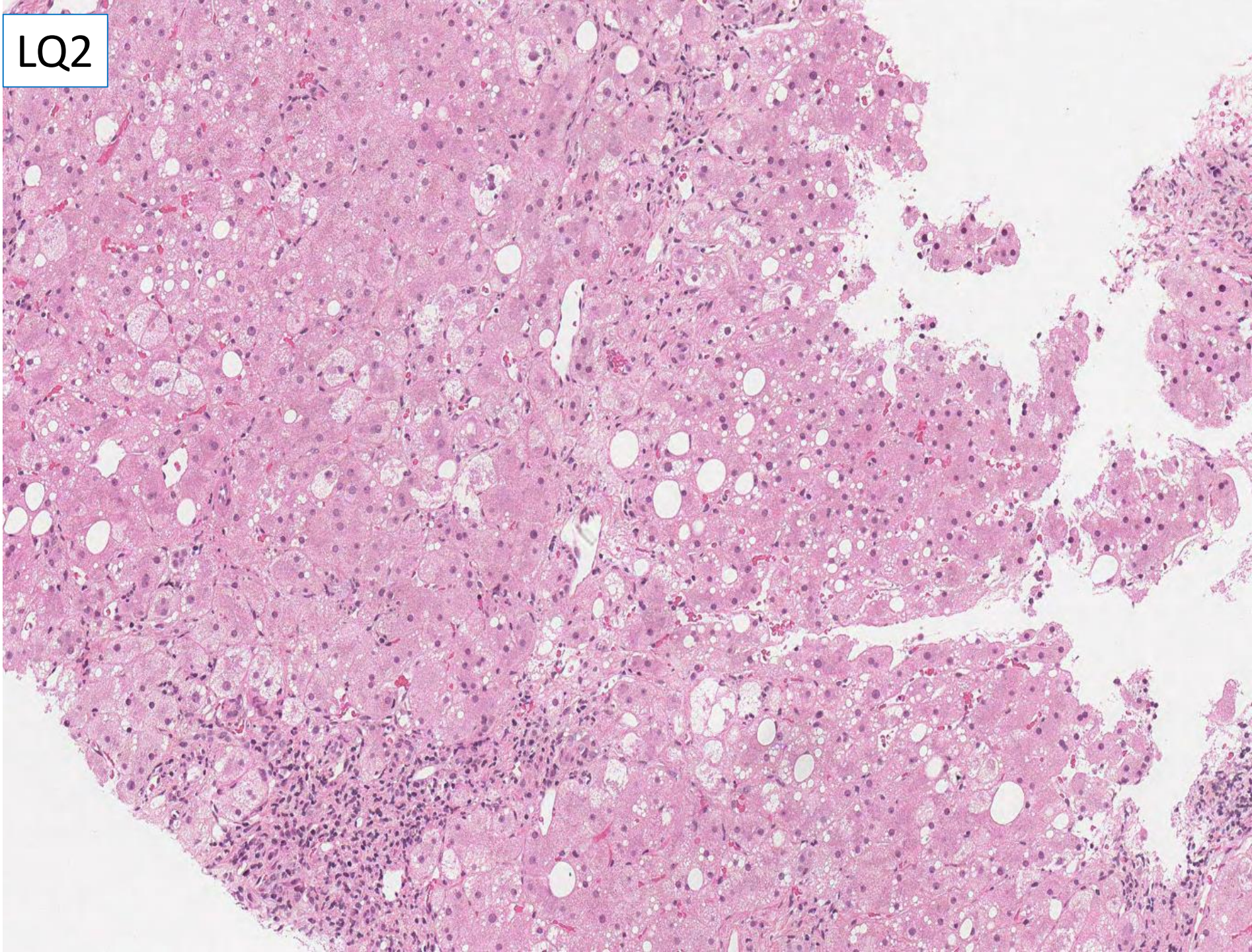
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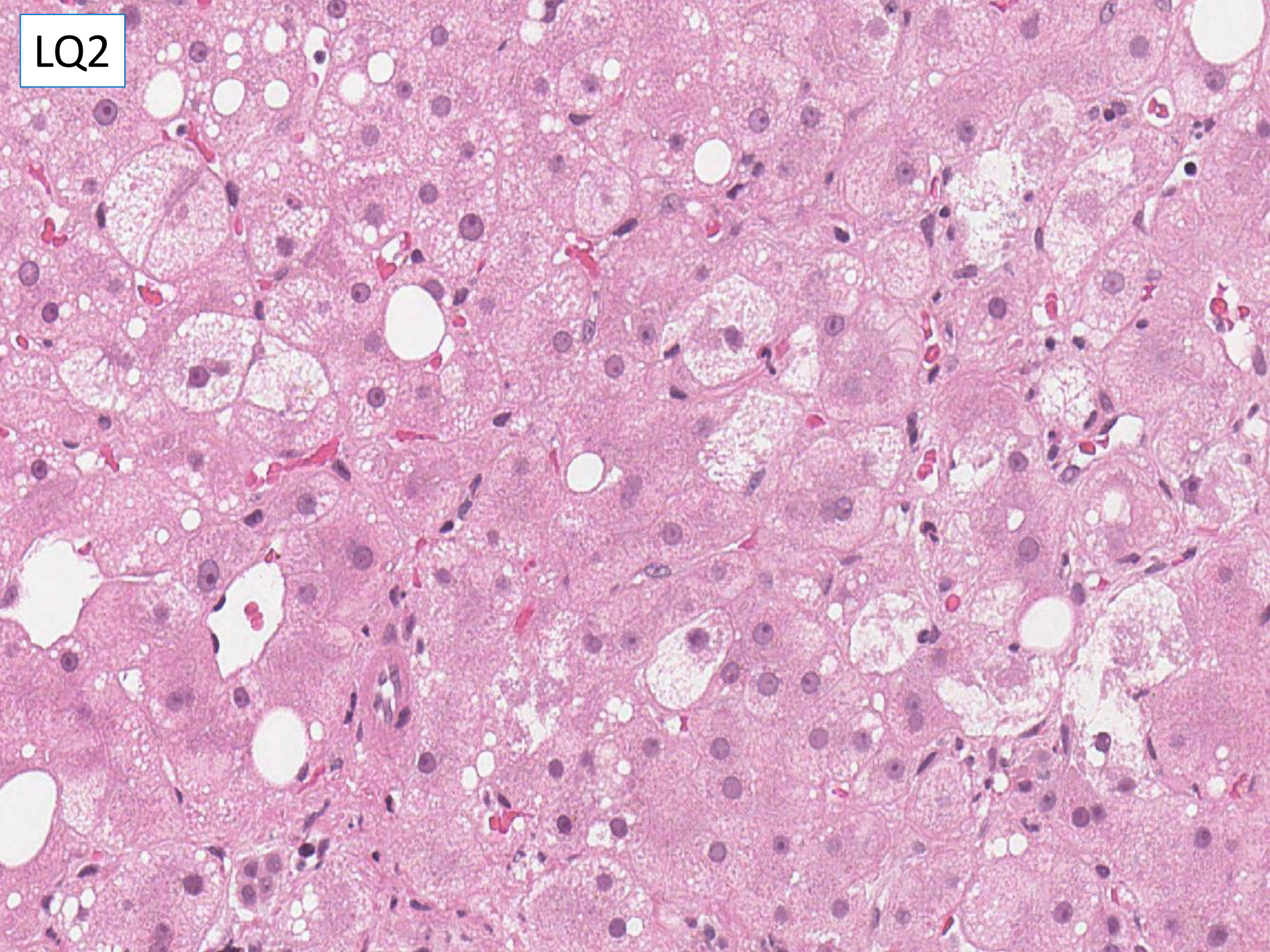
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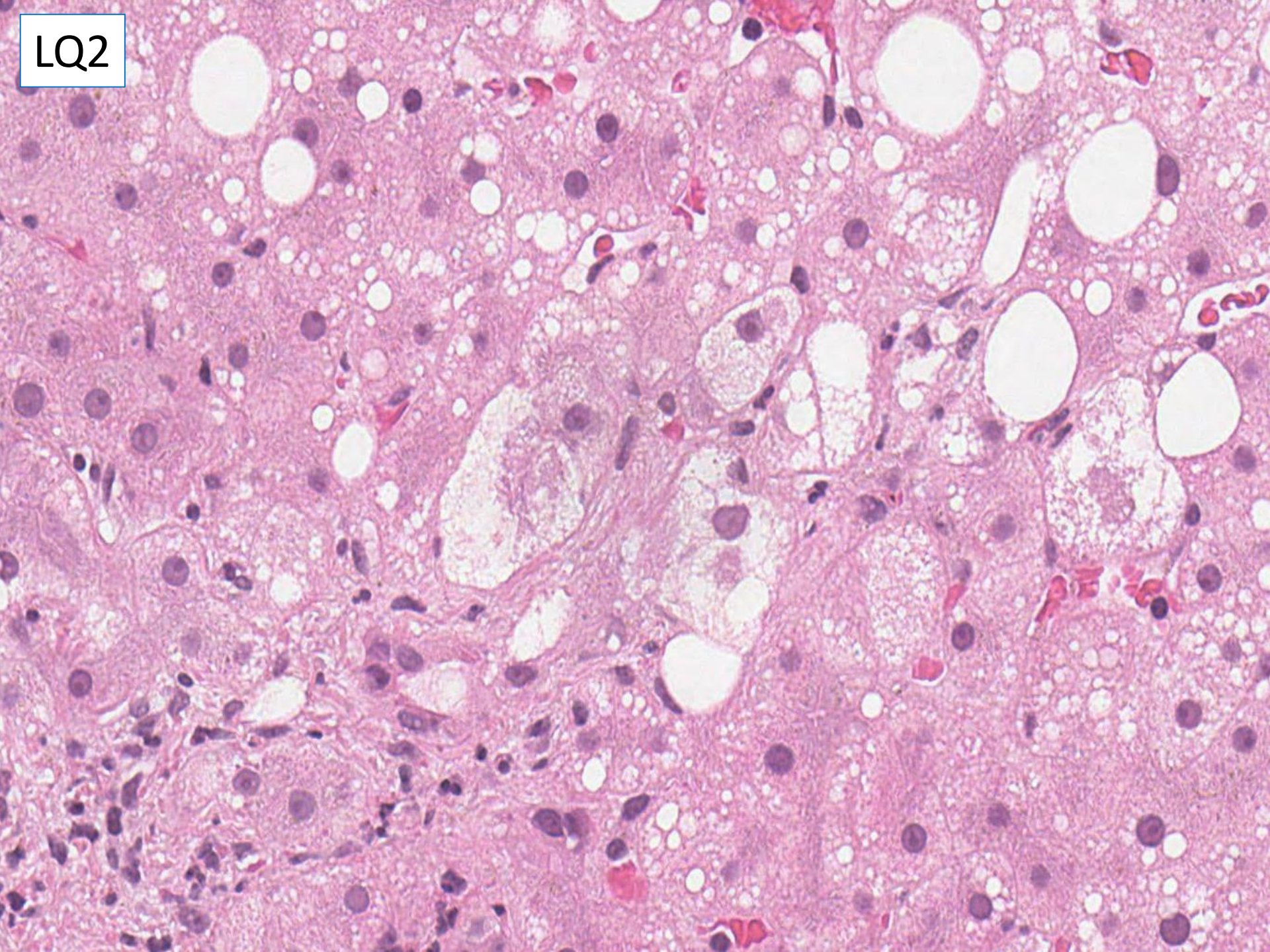
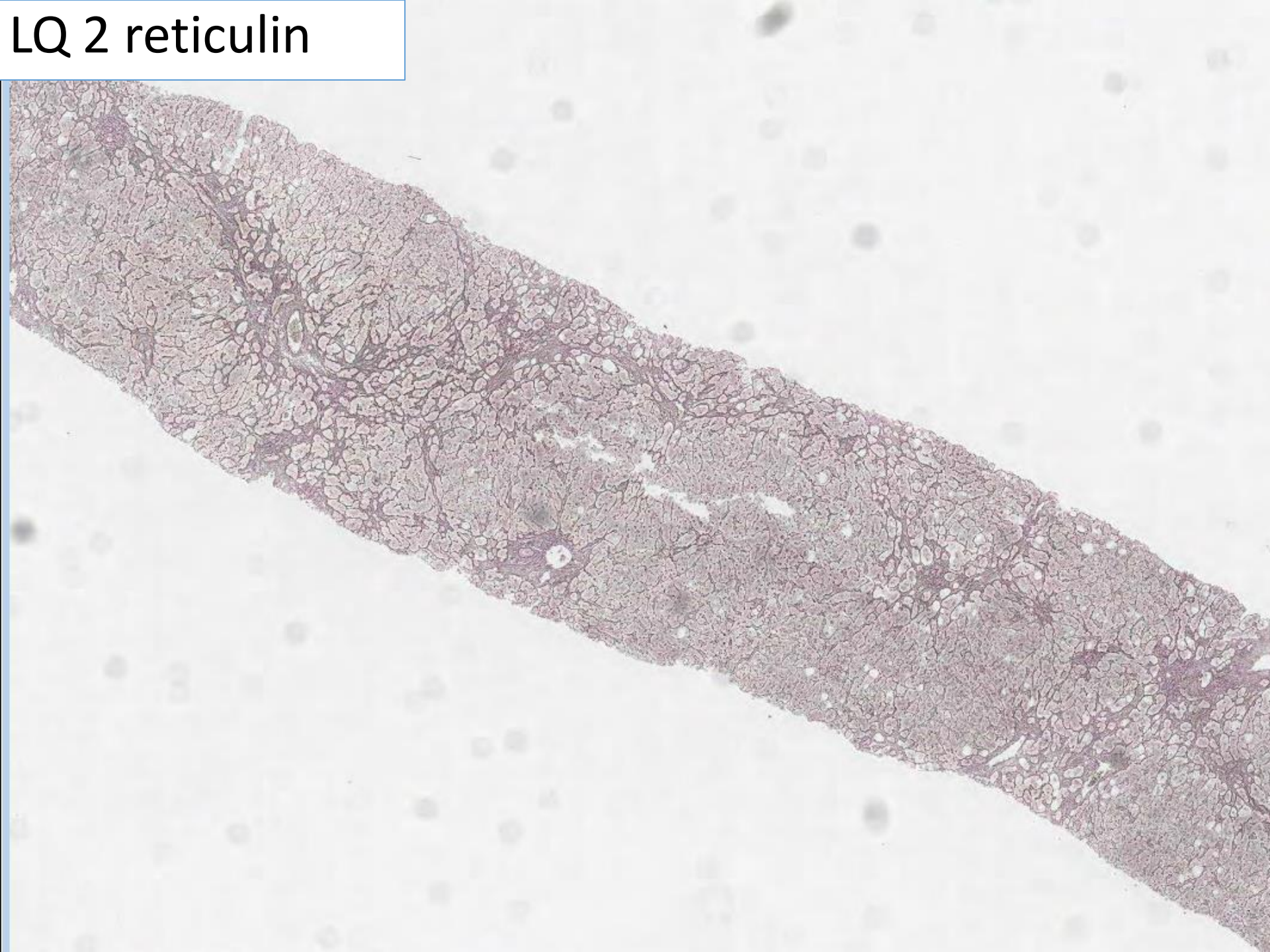
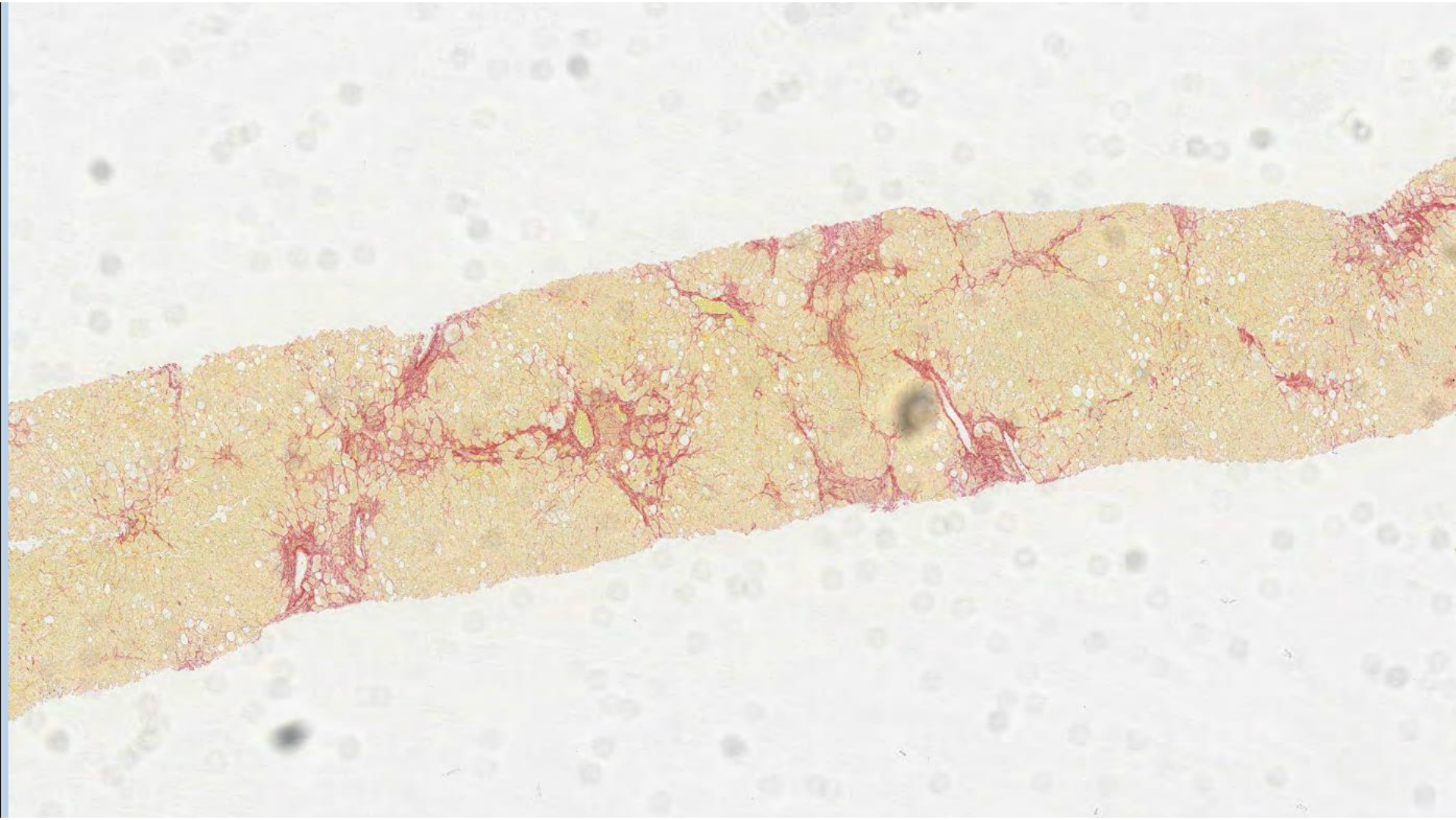

LQ2

LQ 2 reticulin



LQ 2 Picro sirius red



Case LQ2 63F

Chronic pancreatic insufficiency. Type 2 diabetic on insulin, Deranged LFTs, fatty liver on ultrasound, elevated fibroscan

Steatohepatitis (or fatty liver hepatitis) or NASH abbreviation and implied steatohepatitis	75
Steatohepatitis features described but word not used	2
steatosis	3
Fibrosis – bridging or earlier stage	69
- Advanced/possible/developing cirrhosis	9
- Definite cirrhosis	1
Fibrosis stage not mentioned	1
Aetiology – NAFLD/NASH +/- mention of alcohol	69
Favours alcohol over non-alcoholic	5
No mention of aetiological factor	10

Consensus diagnosis: for full marks, a clear diagnosis of steatohepatitis, bridging fibrosis, and NAFLD the likely aetiology +/- consider alcohol

- For steatohepatitis described but word not used – score 10/5/0 = 1/5/0

Case LQ2 63F

Chronic pancreatic insufficiency. Type 2 diabetic on insulin, Deranged LFTs, fatty liver on ultrasound, elevated fibroscan

Agreed scoring: for full marks, a clear diagnosis of steatohepatitis, bridging fibrosis, and NAFLD the likely aetiology +/- consider alcohol.

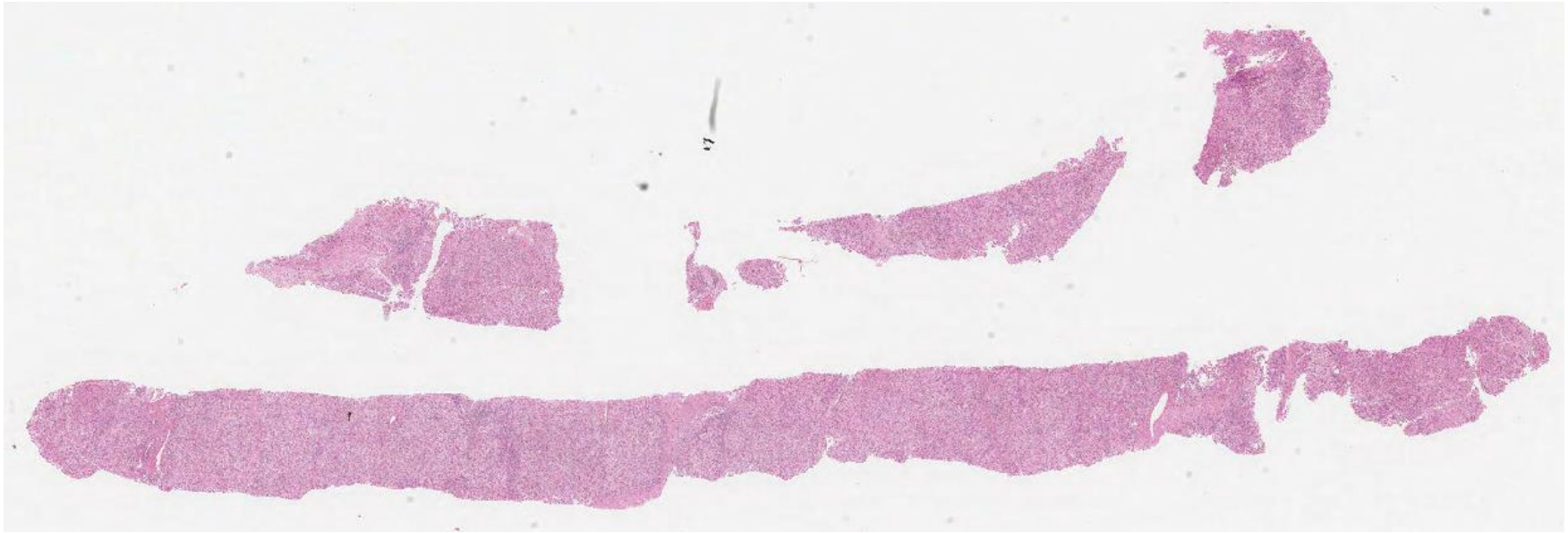
Score 5 marks for steatohepatitis described but neither word nor 'NASH' used. Score 5 marks if no fibrosis stage mentioned, or for definite cirrhosis.

Score 5 marks if there is no mention of any aetiological factor – 'NASH' and 'ASH' are taken to indicate the aetiology as well as the presence of steatohepatitis so don't lose marks if include these.

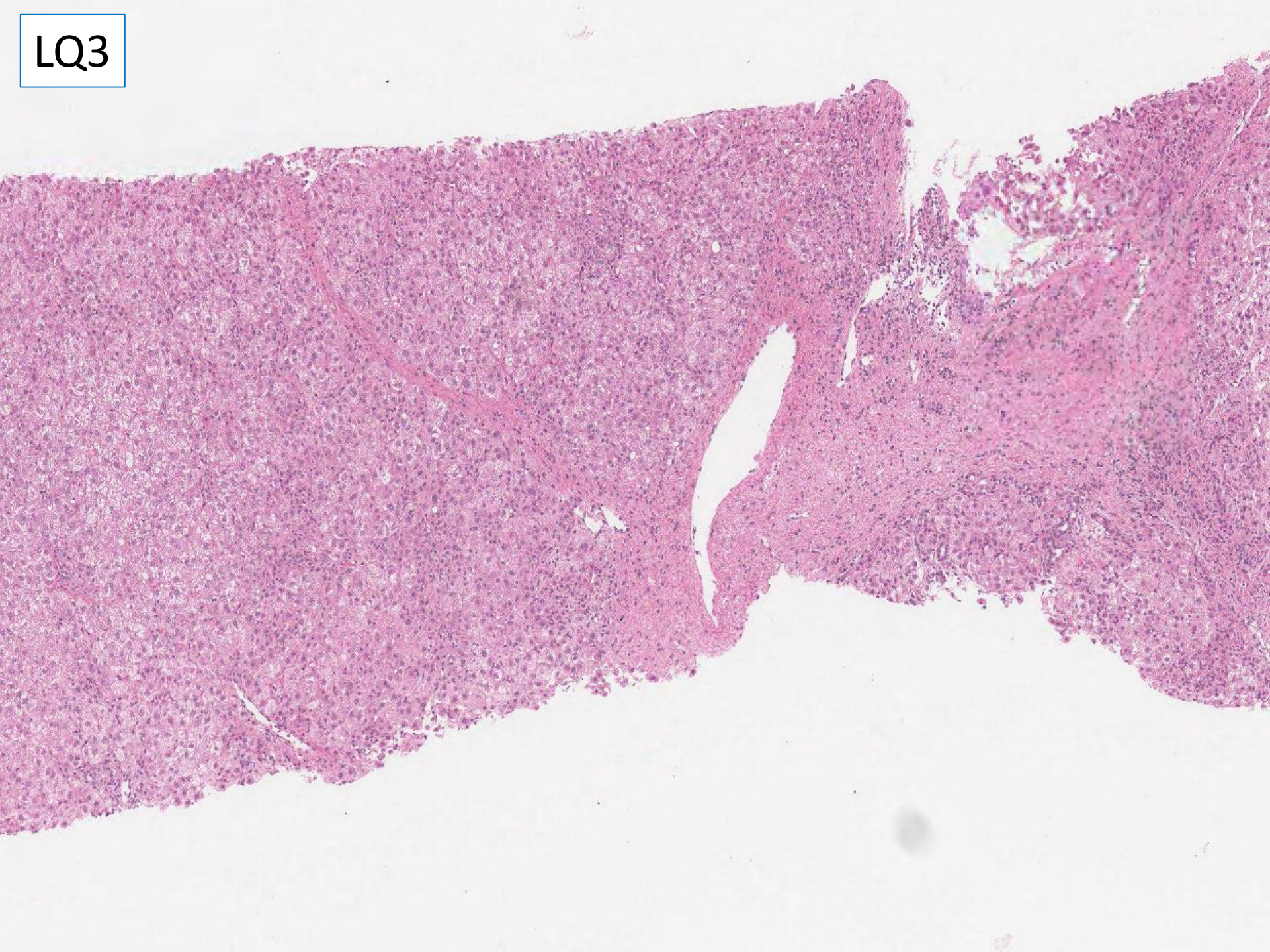
Of those who mentioned the aetiology – 69 included that this is consistent with non-alcoholic fatty liver disease – in keeping with the given history. Fifty mentioned alcohol as a potential co-factor, and 5 favoured alcoholic as the most likely aetiology.

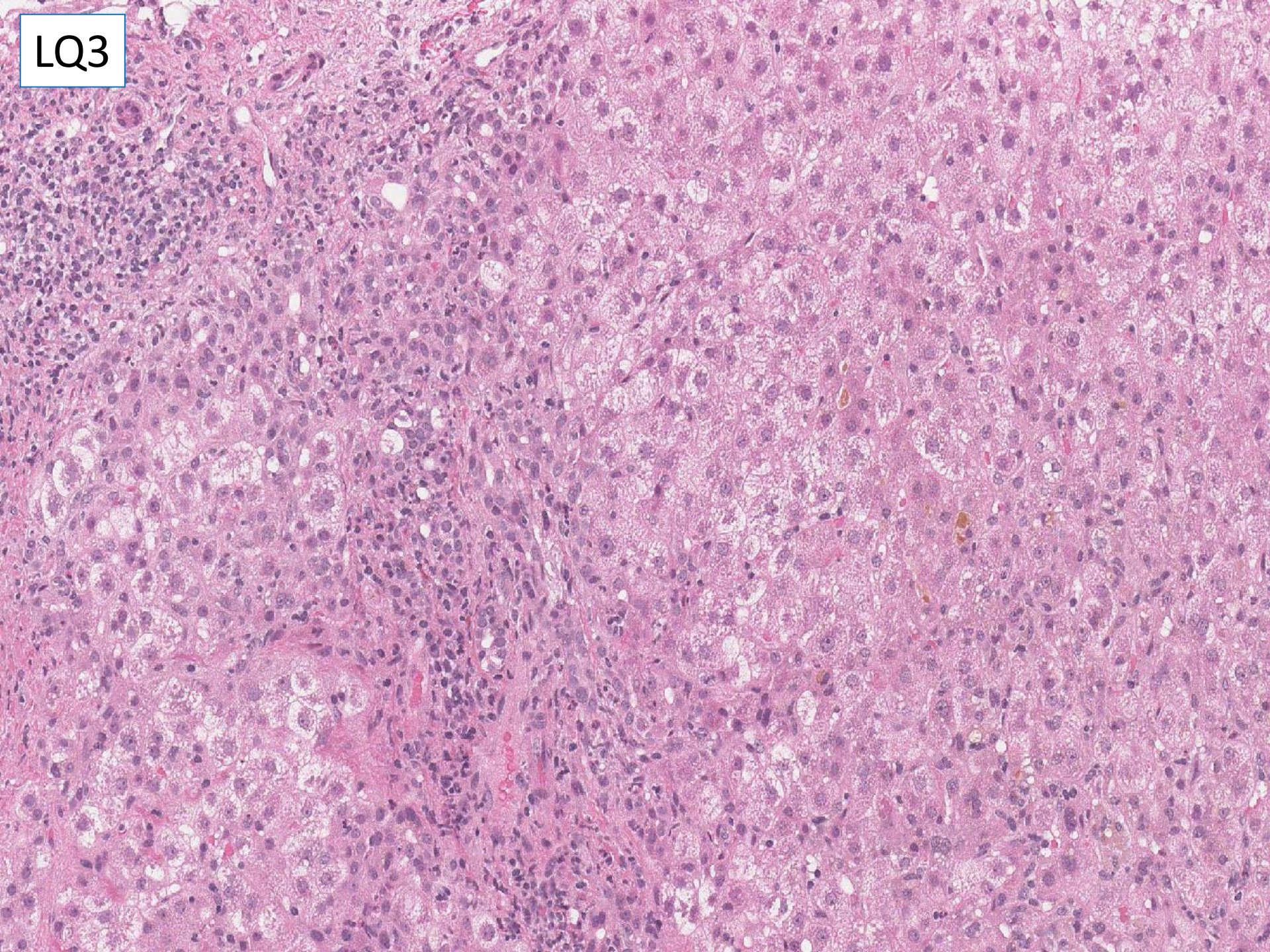
Case LQ3 57M

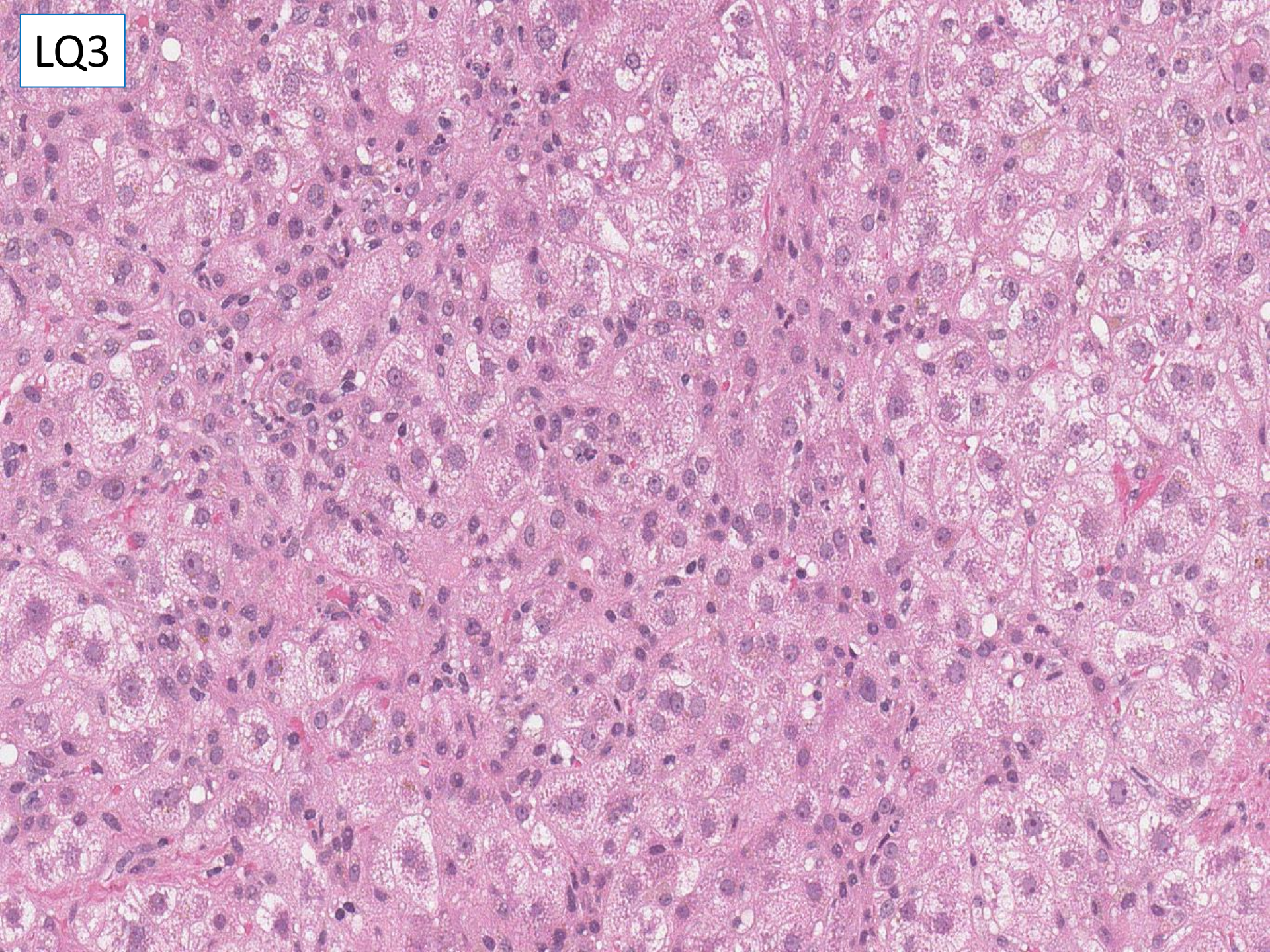
History of alcohol excess, currently abdominal pain, jaundice.
Liver screen normal, ?fatty liver on CT, oesophageal varices

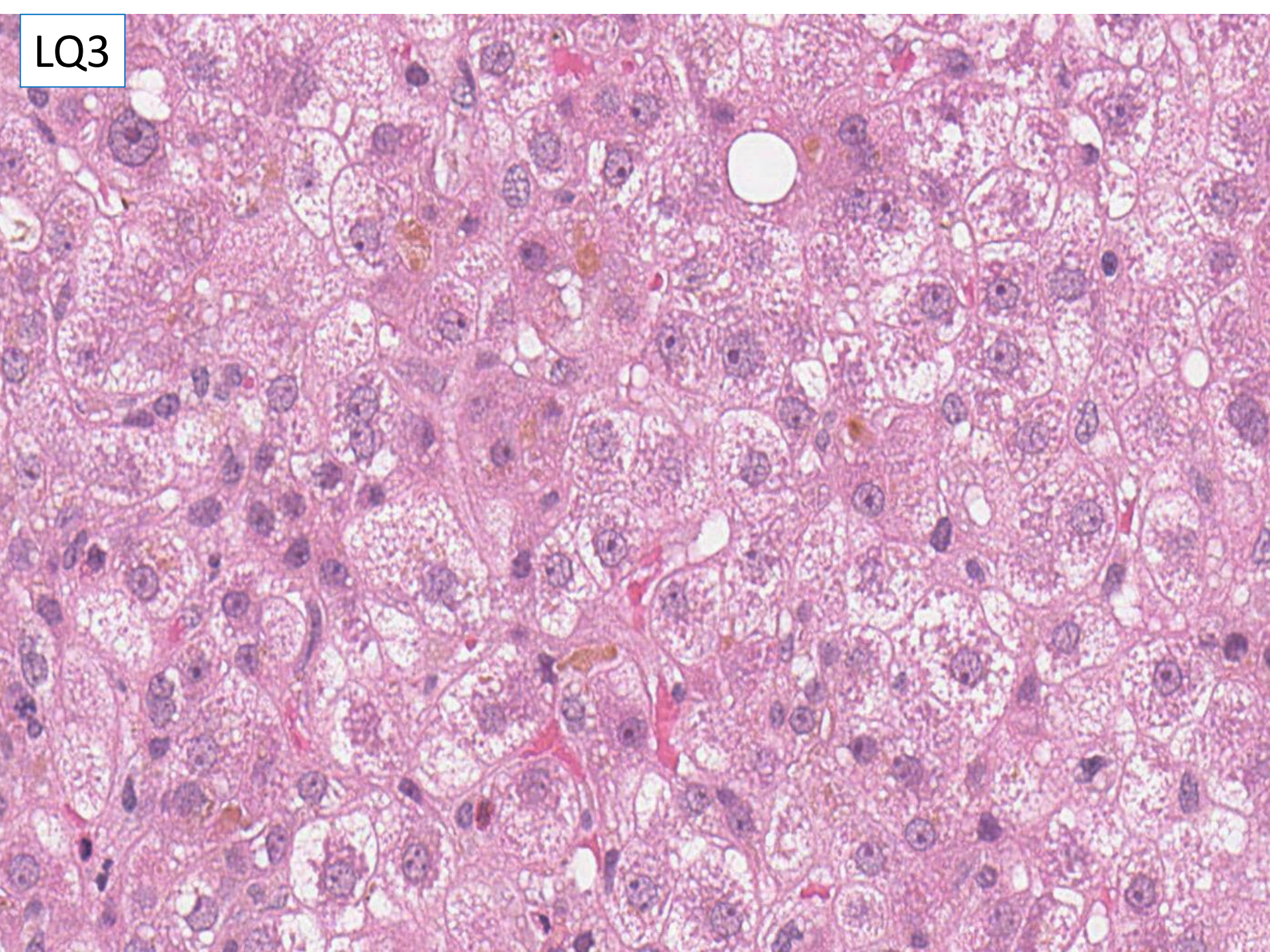


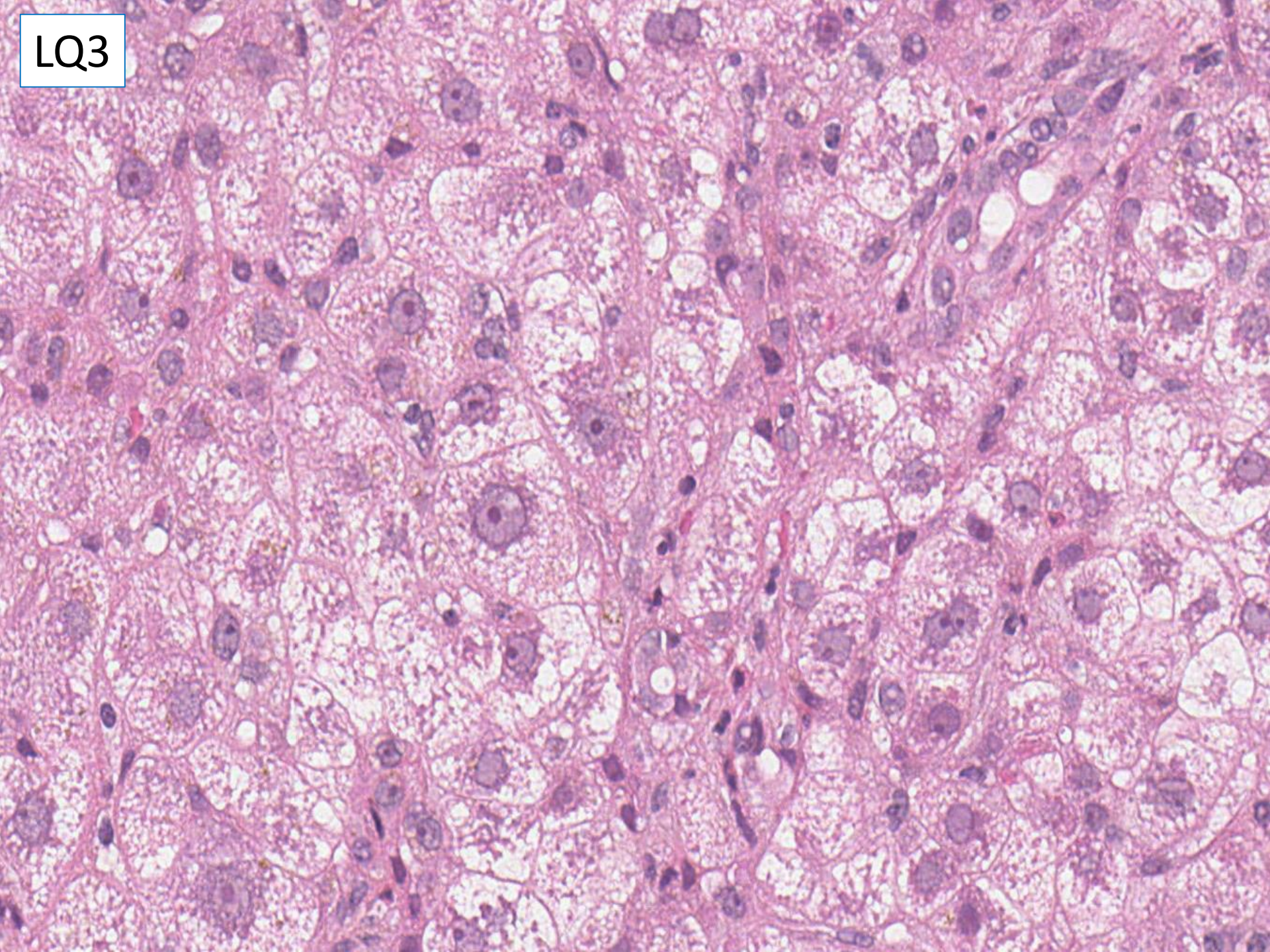
LQ3



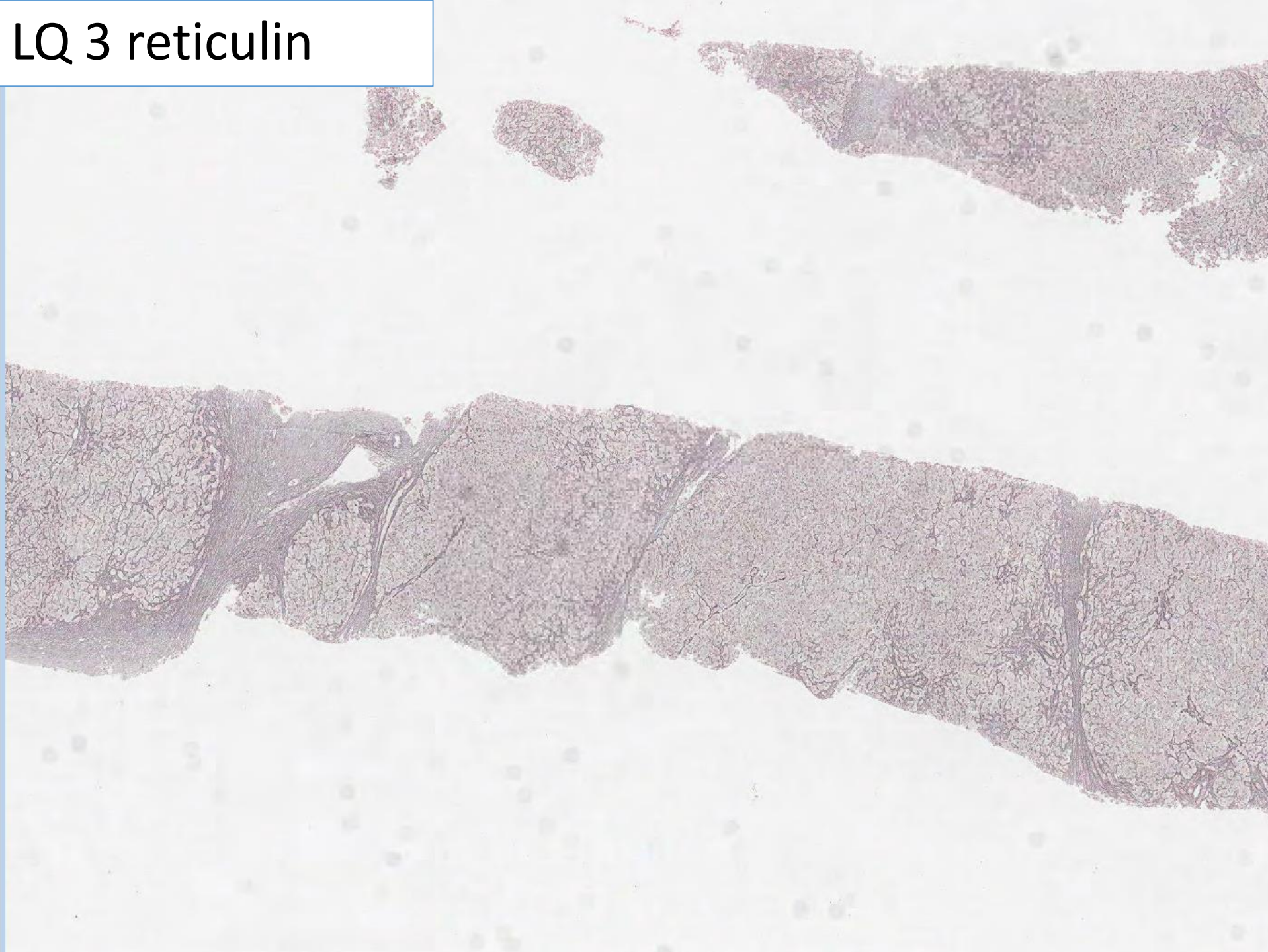

LQ3


LQ3


LQ3


LQ3

LQ 3 reticulin



Case LQ3 57M

History of alcohol excess, currently abdominal pain, jaundice.
Liver screen normal, ?fatty liver on CT, oesophageal varices

Cirrhosis or advanced fibrosis	82
No significant fibrosis	1
Consistent with ethanol or comment on alcohol	71
No mention of alcohol, abstinence, or reference to clinical history	9
Includes - Associated with fatty liver disease (alcohol not mentioned)	4
Comment on not typical for alcohol or uncertain cause	7
Co-diagnosis with	
acute lobular hepatitis/cholestatic hepatitis	25
Alcoholic hepatitis	25
Steatohepatitis NOS	7
Differential diagnosis / rule out:	
Biliary obstruction/ pancreatitis	13
DILI	20
Hepatitis virus	14
Infection/ sepsis	10
Acute hepatitis with cholestasis, background cirrhosis. ? drug or viral – does not look like alcohol	4
Cholestasis	55
No mention of cholestasis	28
Biliary disease as main diagnosis – PSC or PSC/PBC, no mention of alcohol	2

<u>Consensus</u> diagnosis:

cirrhosis with consideration of alcohol as the cause/contributing factor.

No consensus on what else may be going on here.

Case LQ3 57M

History of alcohol excess, currently abdominal pain, jaundice.
Liver screen normal, ?fatty liver on CT, oesophageal varices

Agreed scoring:

For full marks – A diagnosis of cirrhosis, with some comment on the role played by alcohol.

Score 5 marks if there is no reference to alcohol, abstinence, or the clinical history of risk factor.

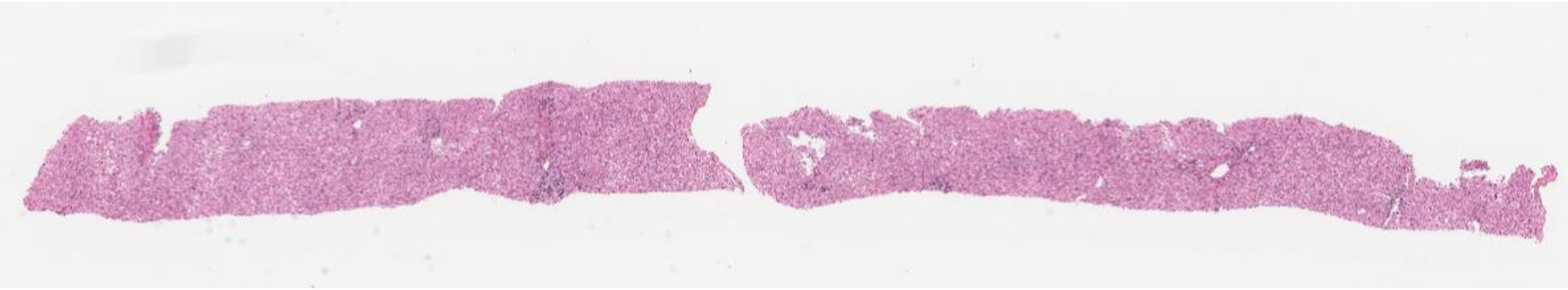
Score 0 marks for biliary disease as the main diagnosis with no mention of alcohol, or for report indicating no significant fibrosis.

There was much discussion about the likely cause of cholestasis and lobular hepatitis, which was considered not to show features of steatohepatitis, but to favour an alternative cause of disease activity against a background of cirrhosis without specific features of alcoholic liver disease – although that may have been the original cause. Therefore would be consistent with abstinence – there is no steatosis or steatohepatitis in the biopsy. The pathologist should flag up to the clinician that they should look for another cause – e.g. hepatitis E, drugs, to account for cholestasis.

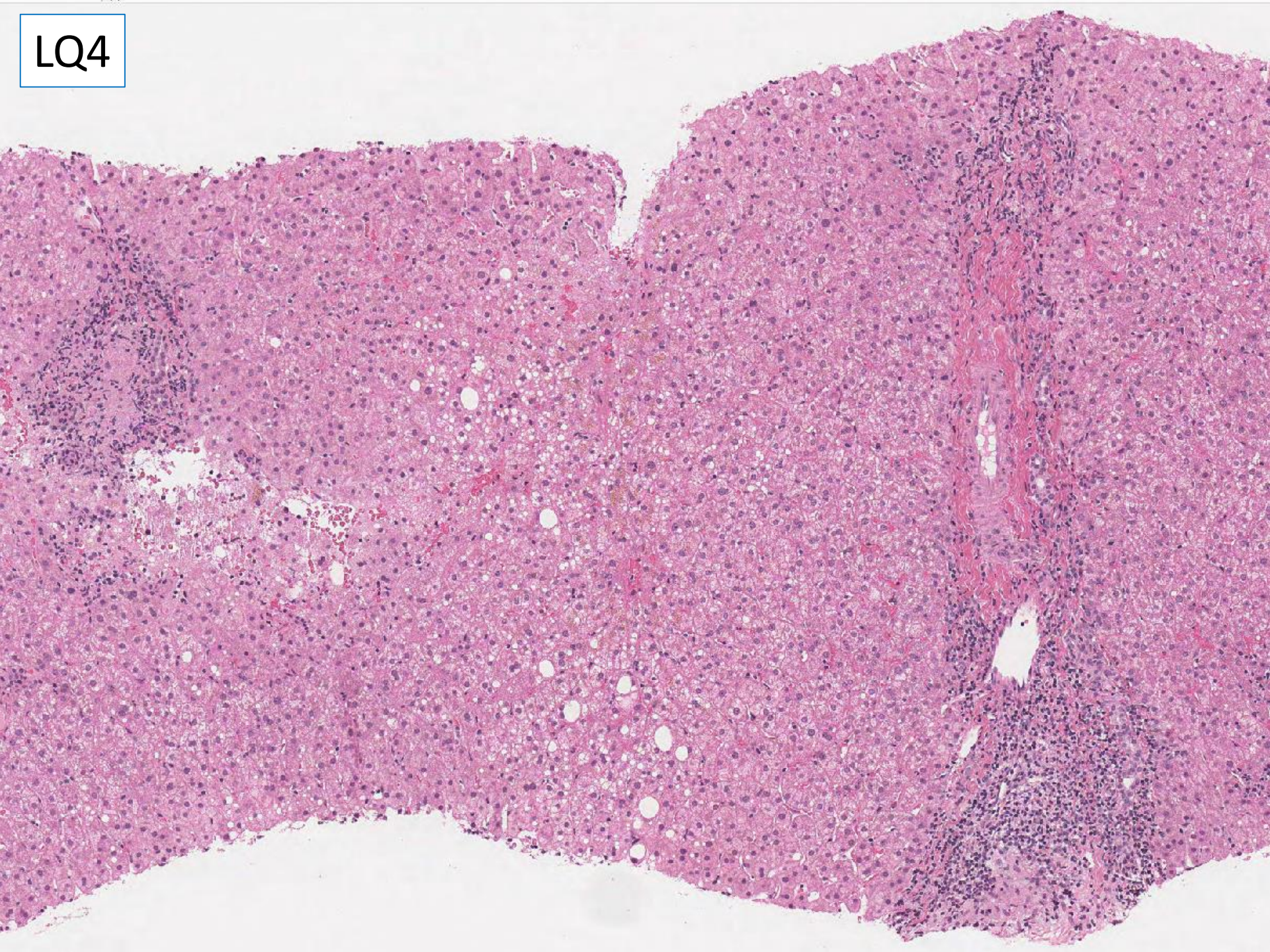
Follow up information: I can confirm that alternative causes were sought and not found autoantibodies-ve, viral screen-ve, no apparent drug history and patient probably not abstinent.

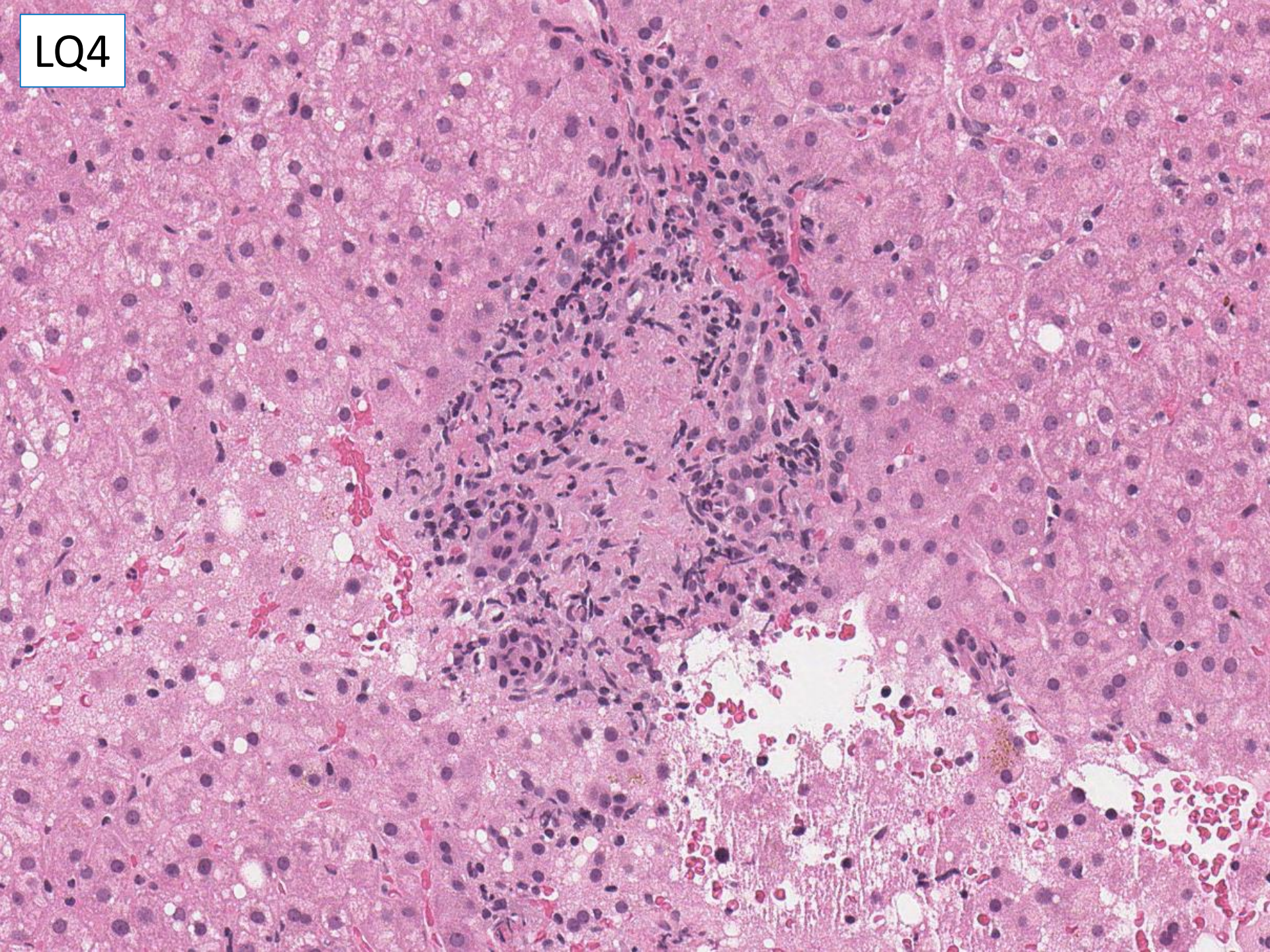
Case LQ4 44M

Abnormal LFTs, AMA strong +ve, fatty liver on ultrasound, ?PBC



LQ4

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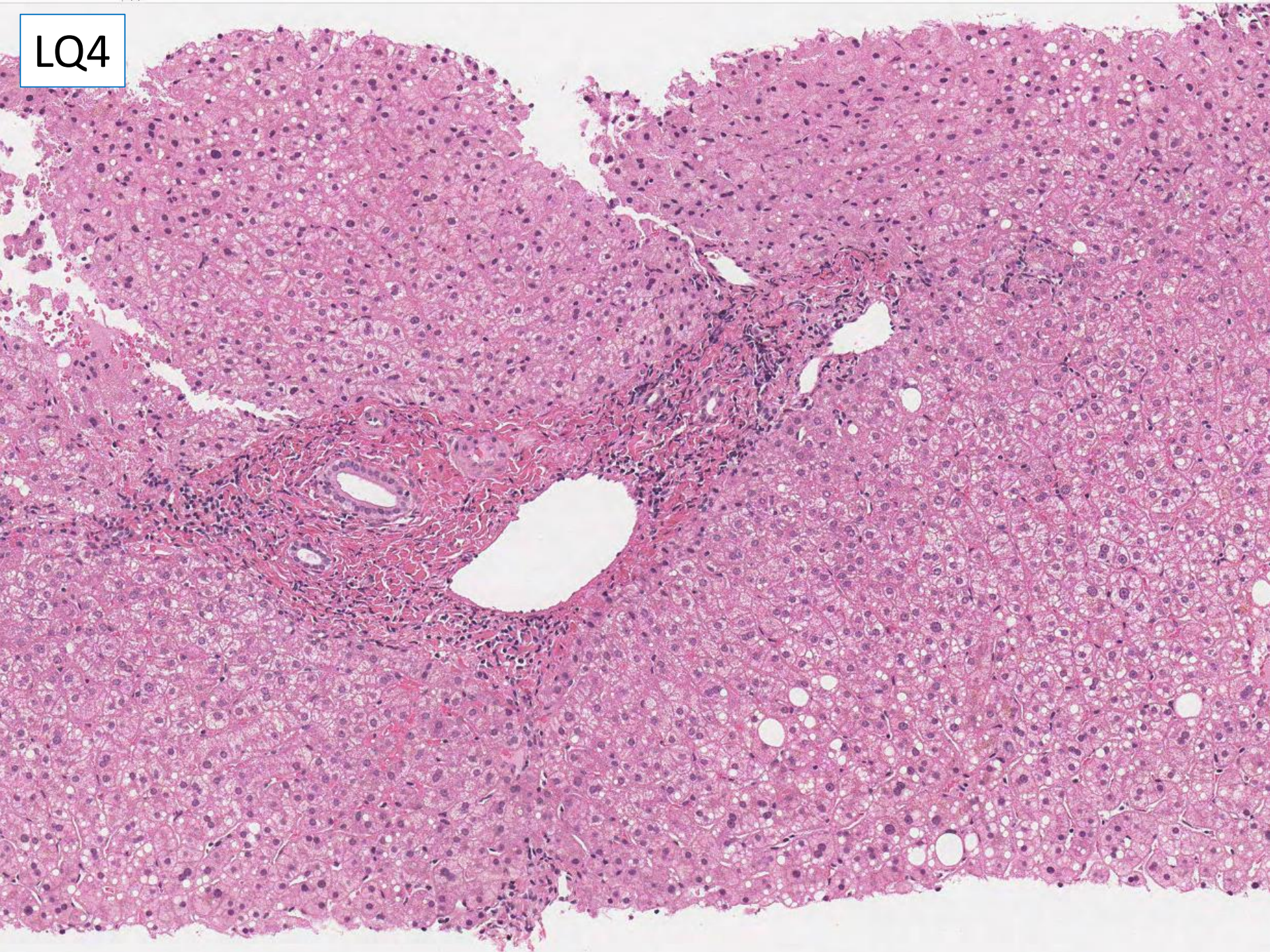
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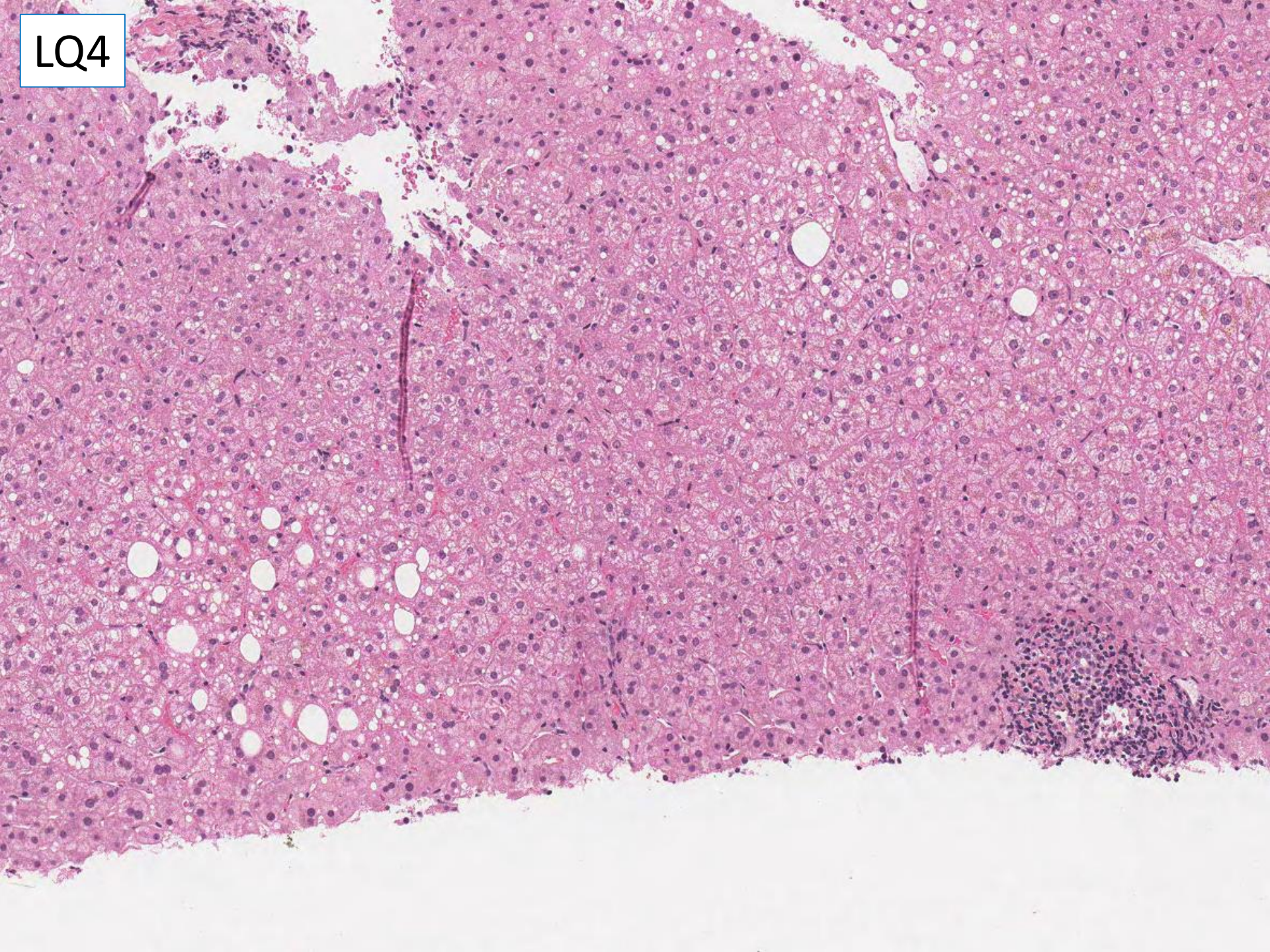
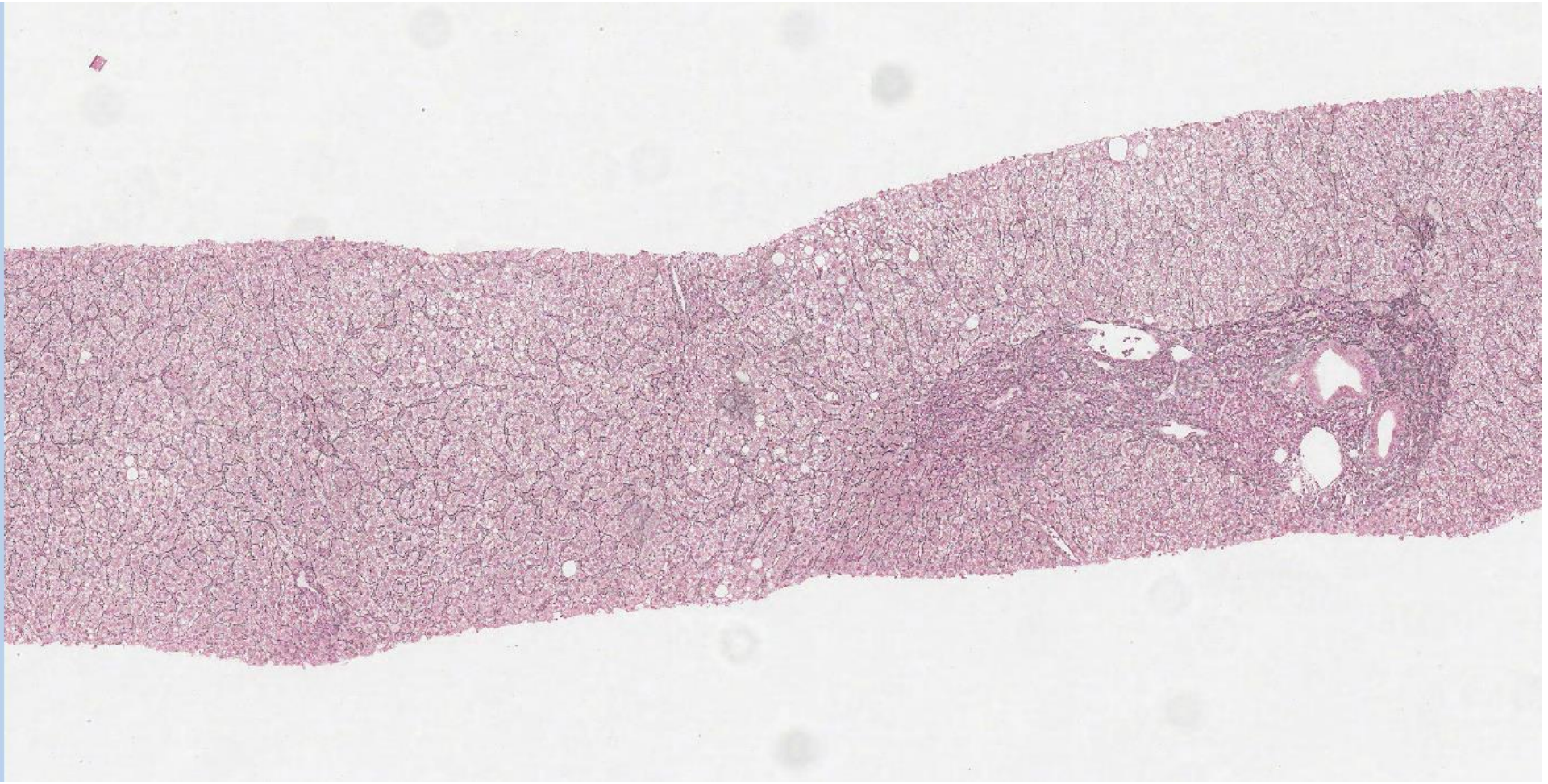
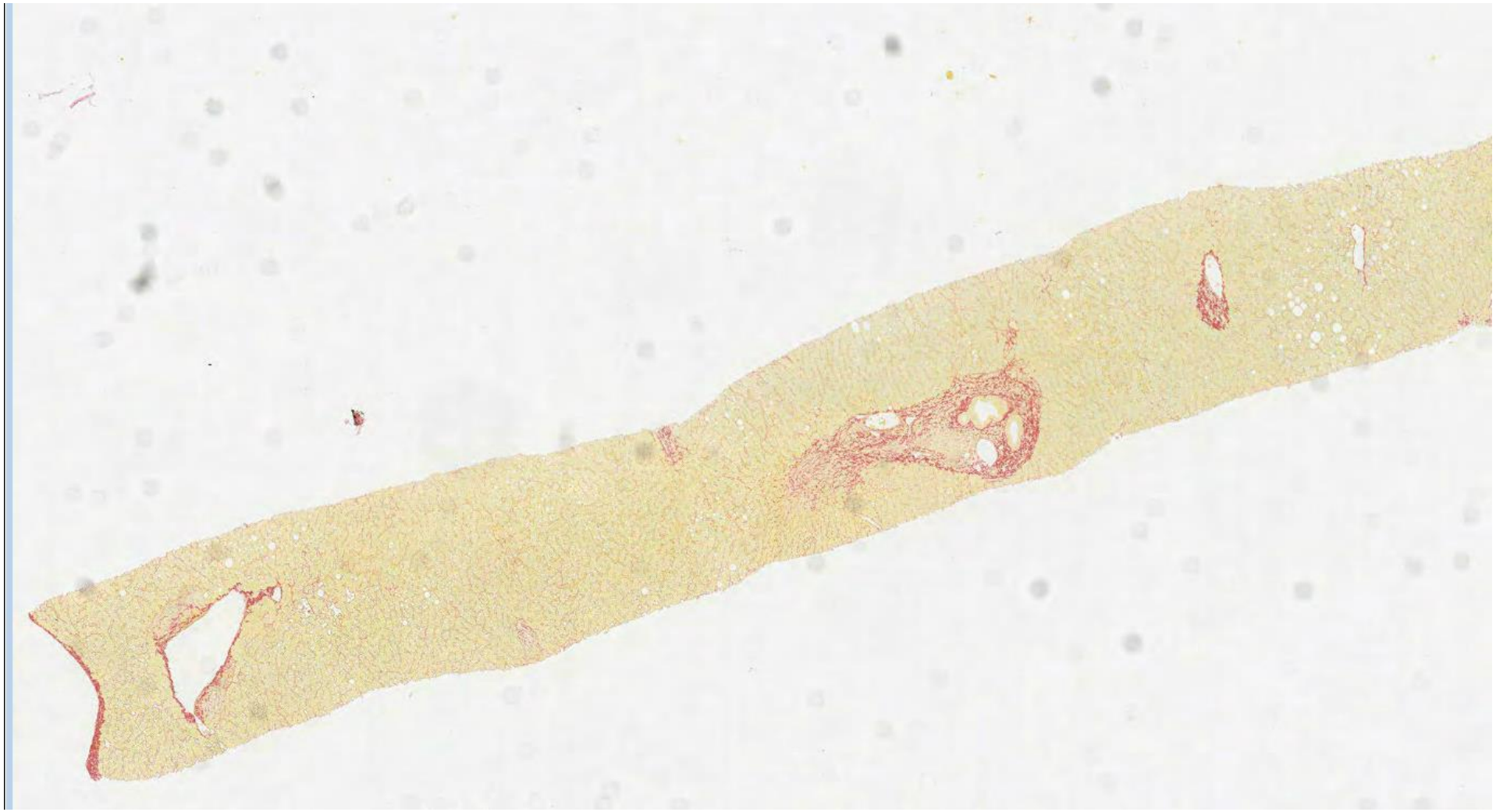

LQ4

LQ4 reticulin



LQ 4 Picro Sirius red



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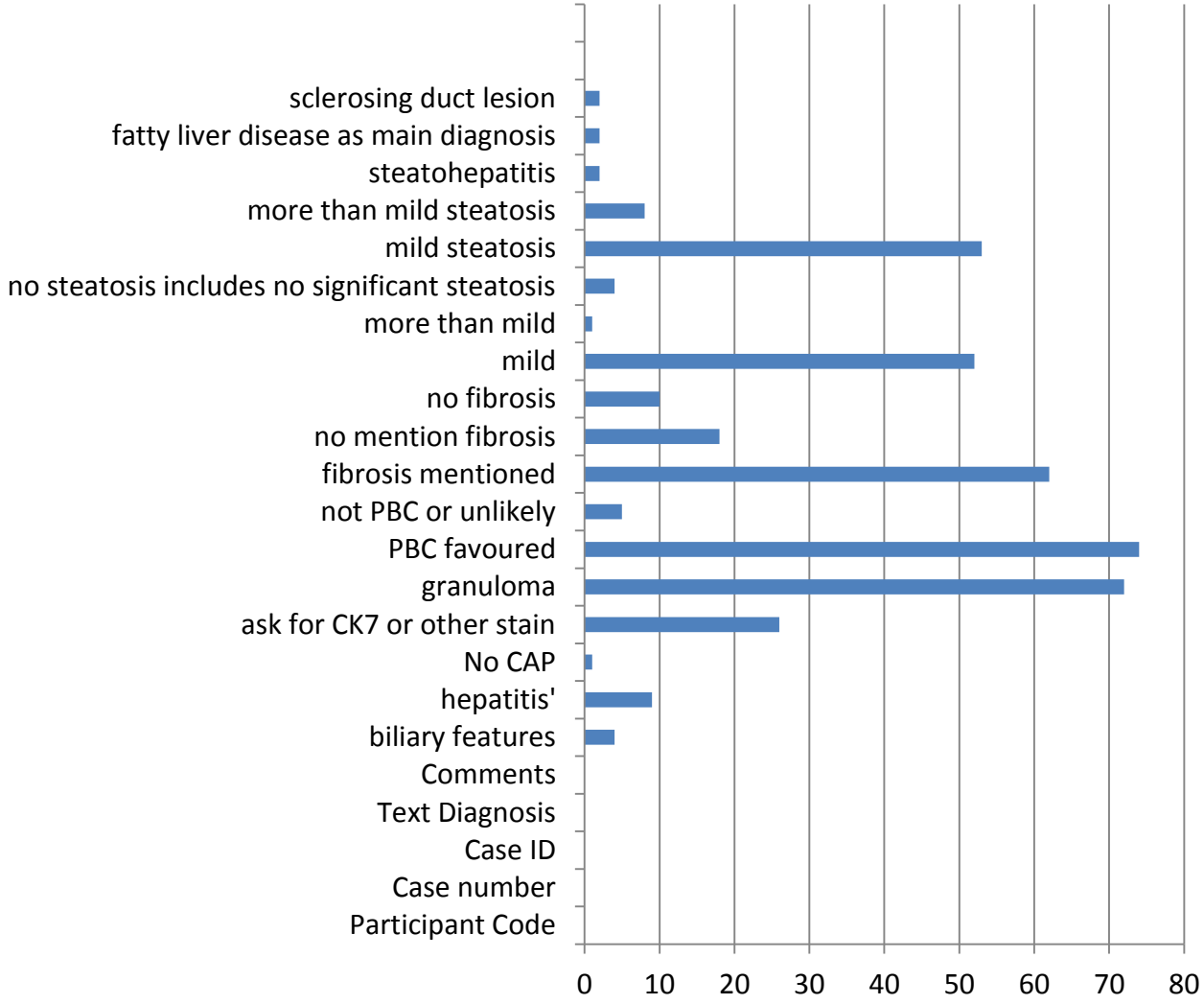


Consensus diagnosis

PBC (74, many said ‘early’ or ‘because of AMA’) as favoured diagnosis
and presence of granulomas.

Majority mentioned fibrosis (62).

26 asked for extra stains (CK7)

One given benefit of doubt? Survey results: all vote for full marks

Case LQ4 44M

Abnormal LFTs, AMA strong +ve, fatty liver on ultrasound, ?PBC

Agreed scoring:

For full marks – a favoured diagnosis of primary biliary cholangitis (PBC) including a reference to the stage of disease.

Score 5 marks if no mention of fibrosis or any reference to the disease stage.

Score no marks for responses giving an alternative diagnosis to PBC – fatty liver disease, autoimmune hepatitis, granulomatous hepatitis – or that this is not PBC or PBC unlikely.

Additional stains were suggested by 22 – including keratin 7 (16), cytokeratin (3), Orcein/Shikata (12), rhodamine (3). See masterclass presentation by Alberto Quaglia – these are not equivalent to each other, and a combination can be useful in investigating for chronic liver disease. During the last year many have experienced problems with orcein – this appears to be due to a bad batch of synthetic orcein, which has been rectified, and should now improve.

Masterclass: Alberto Quaglia

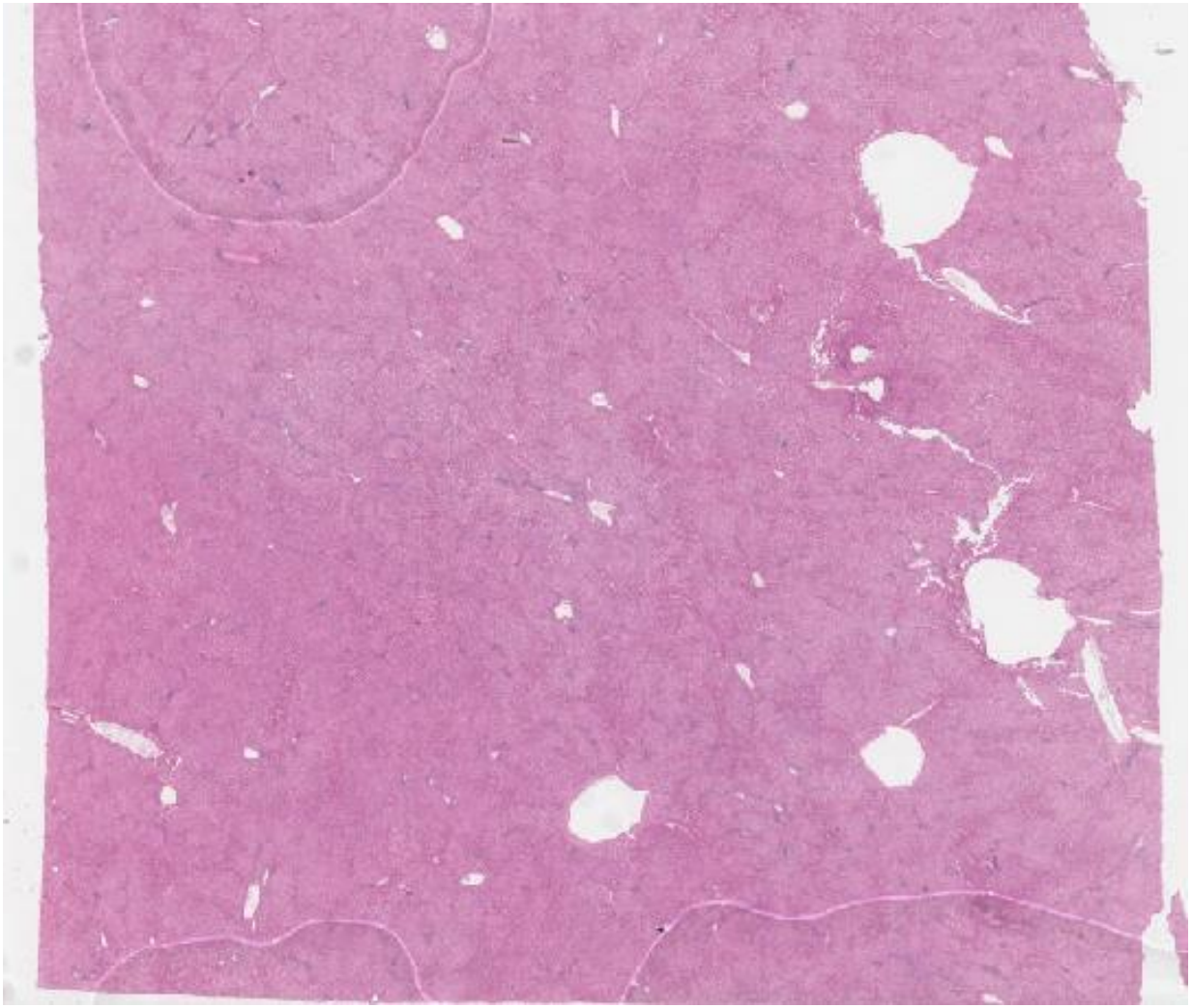
- Shikata, rhodamine and keratin 7 in biliary disease

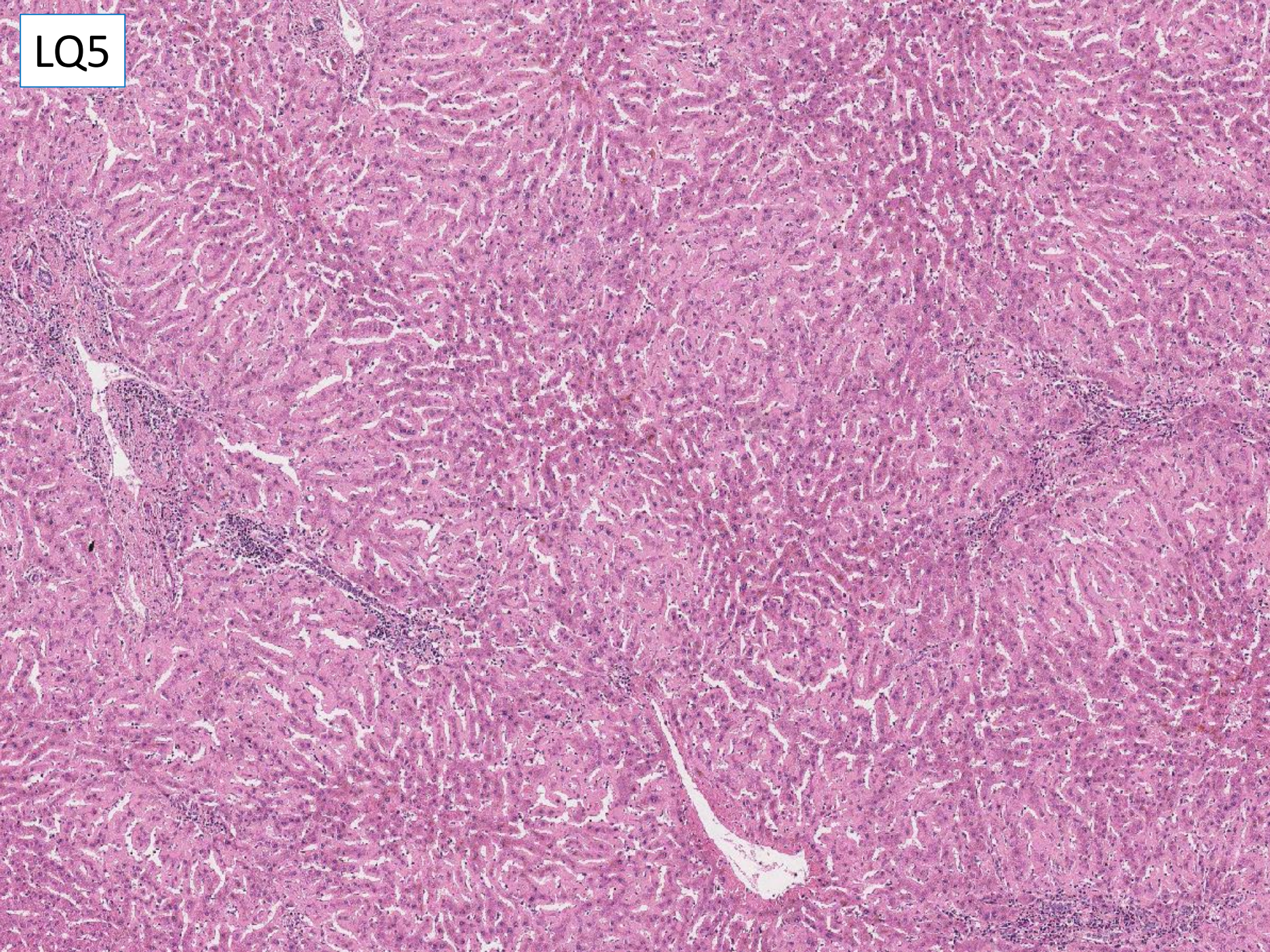
Case LQ5 65F

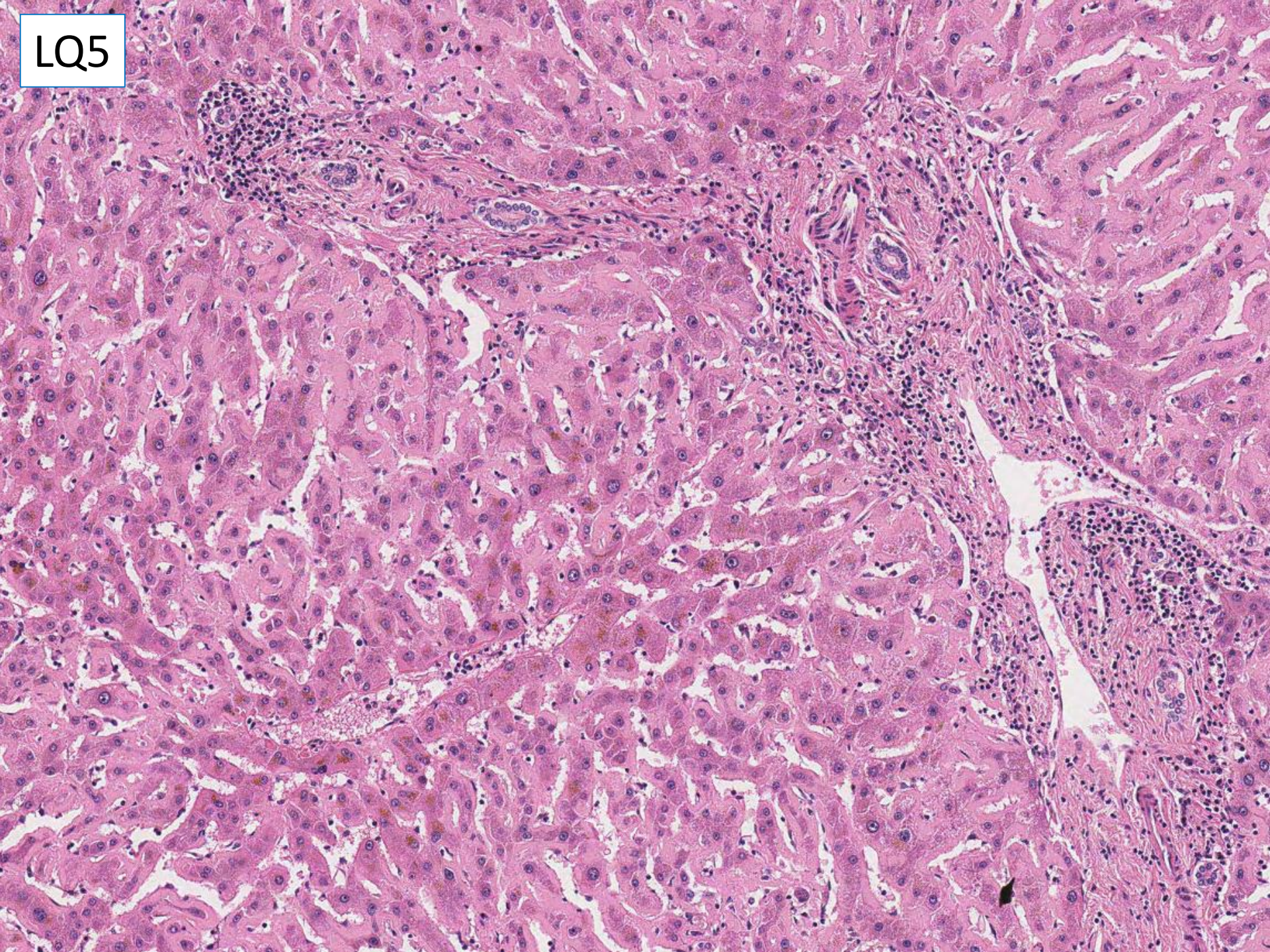
'Liver lesion segment 6/7'.

From electronic patient record IgG kappa paraproteinaemia.

Section is from background liver




LQ5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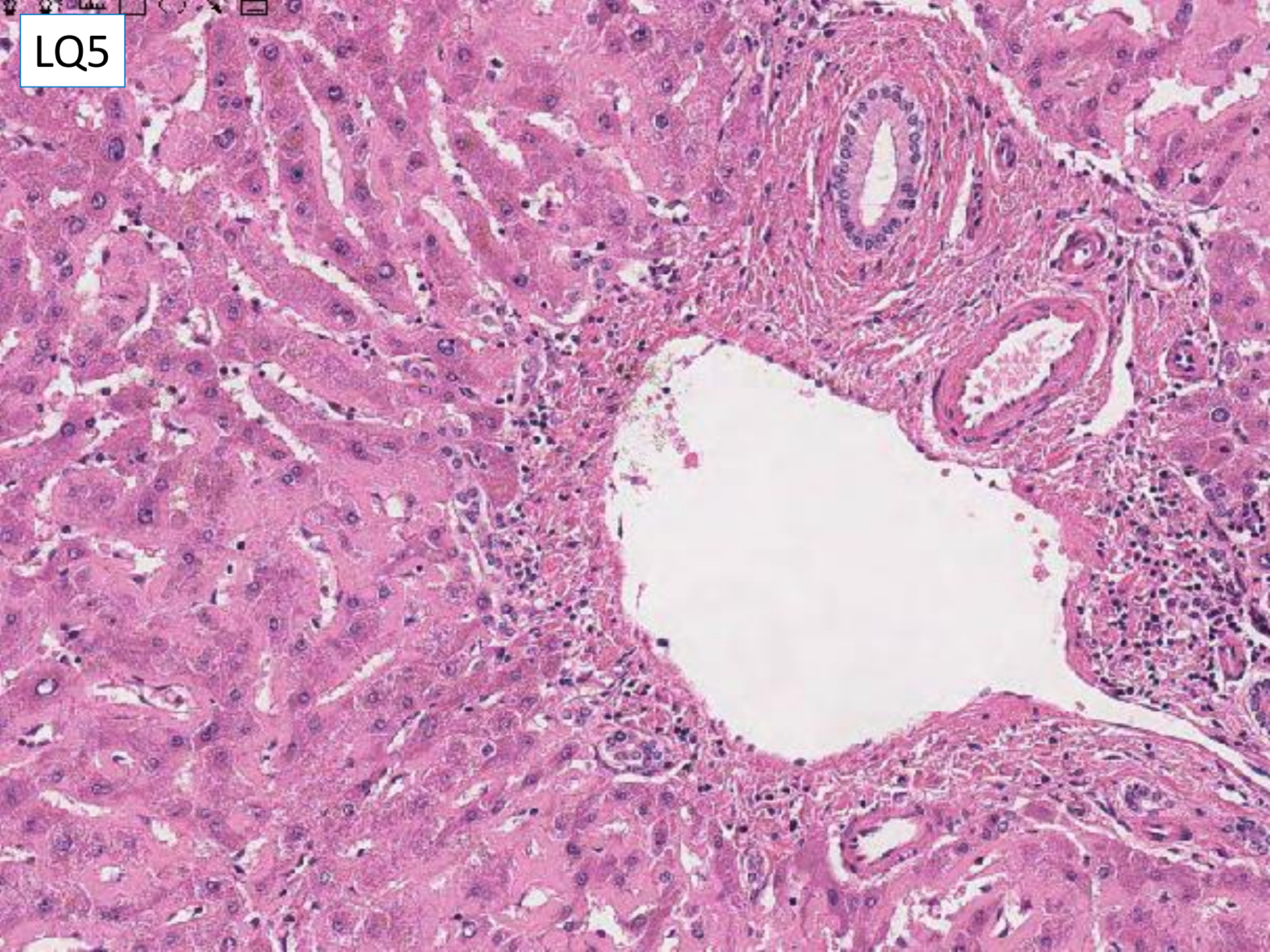
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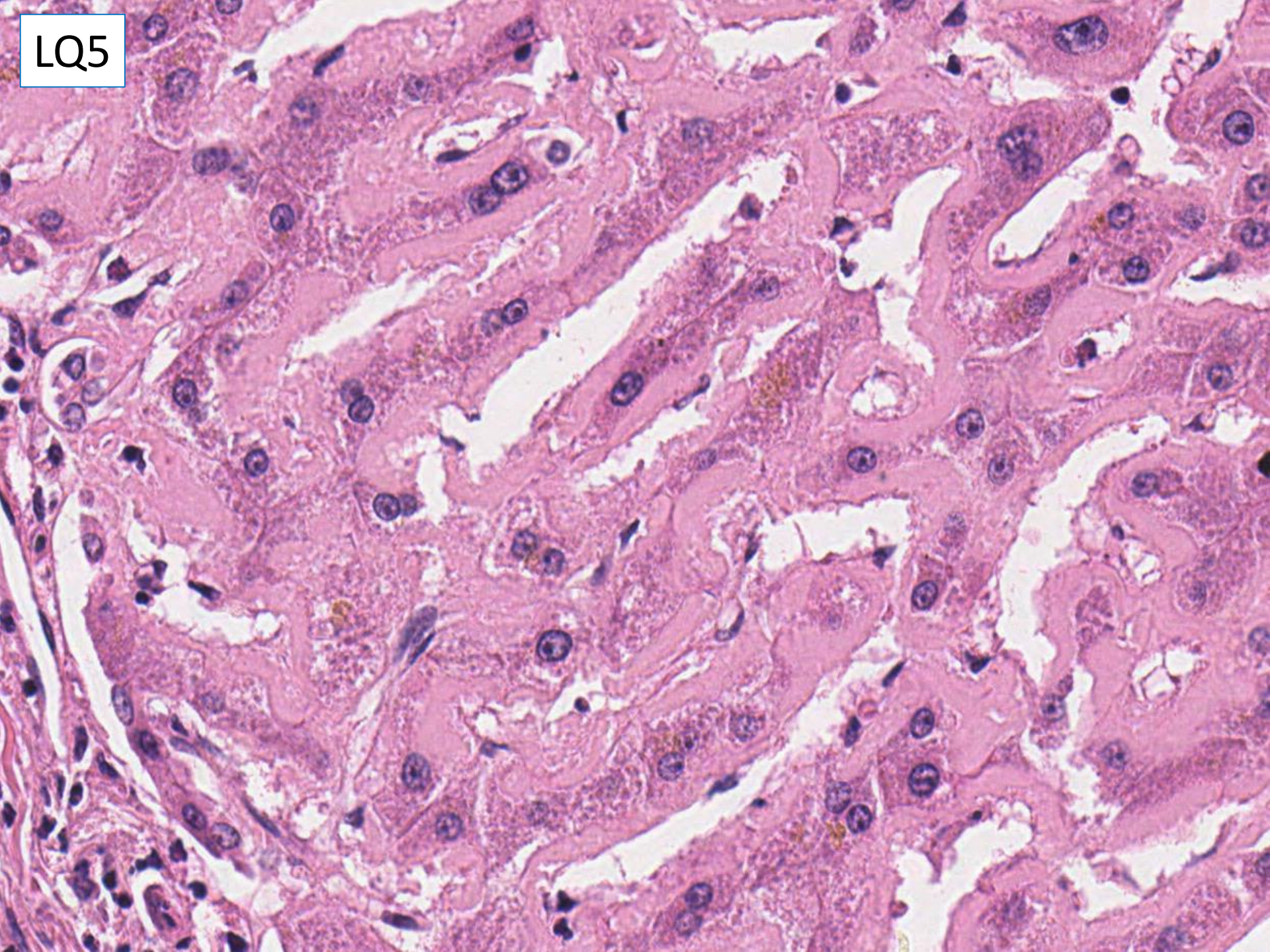
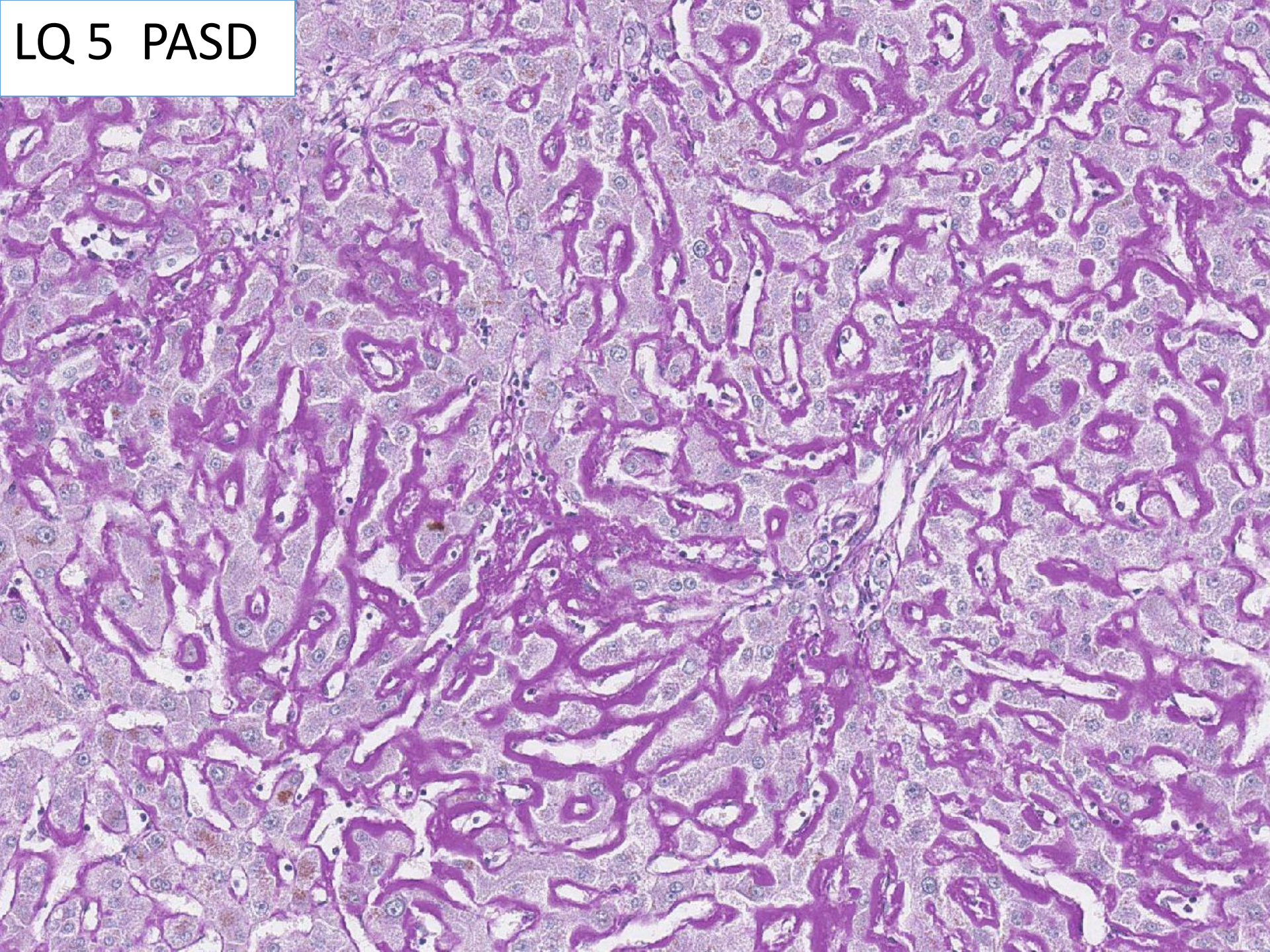
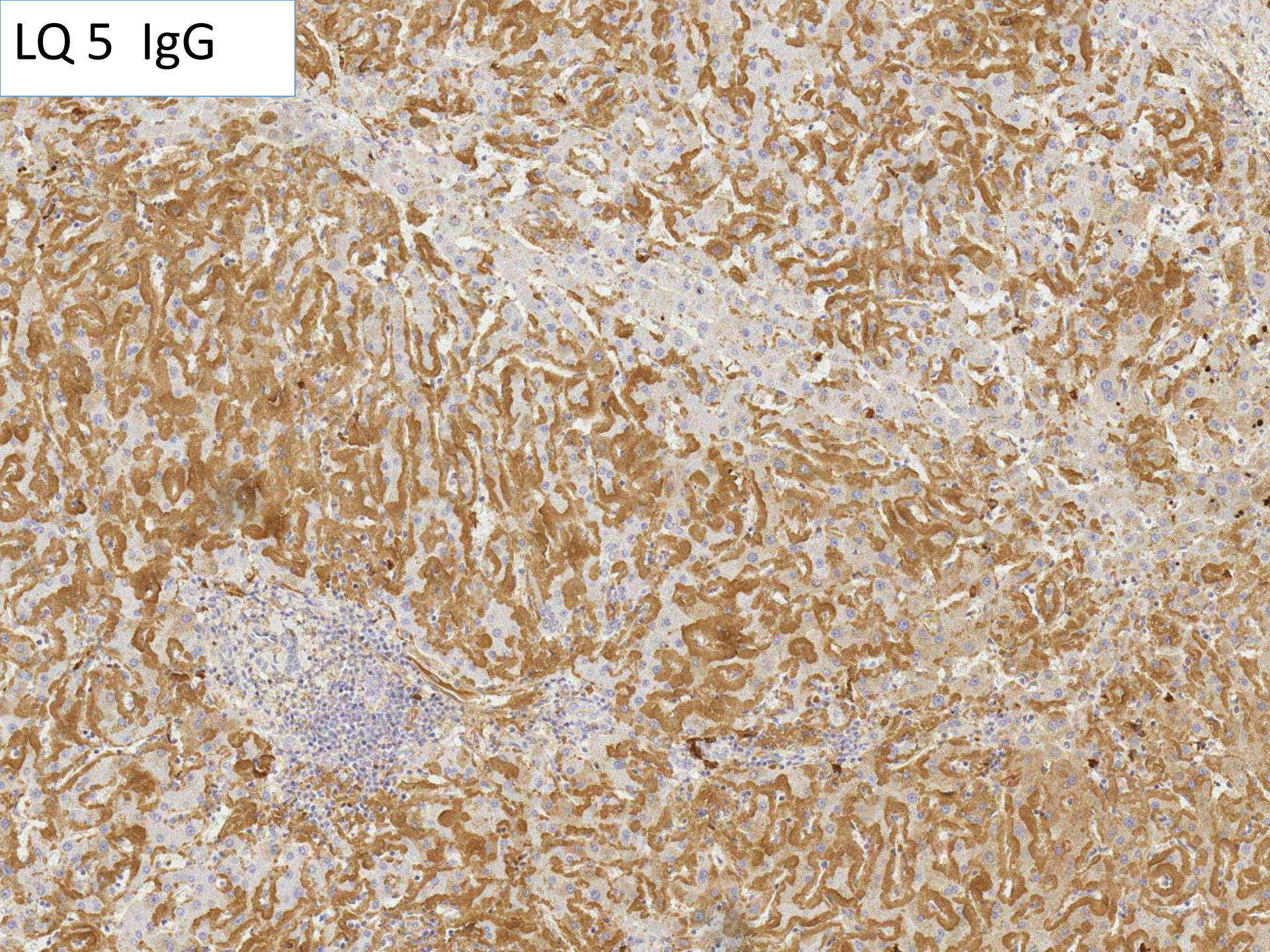
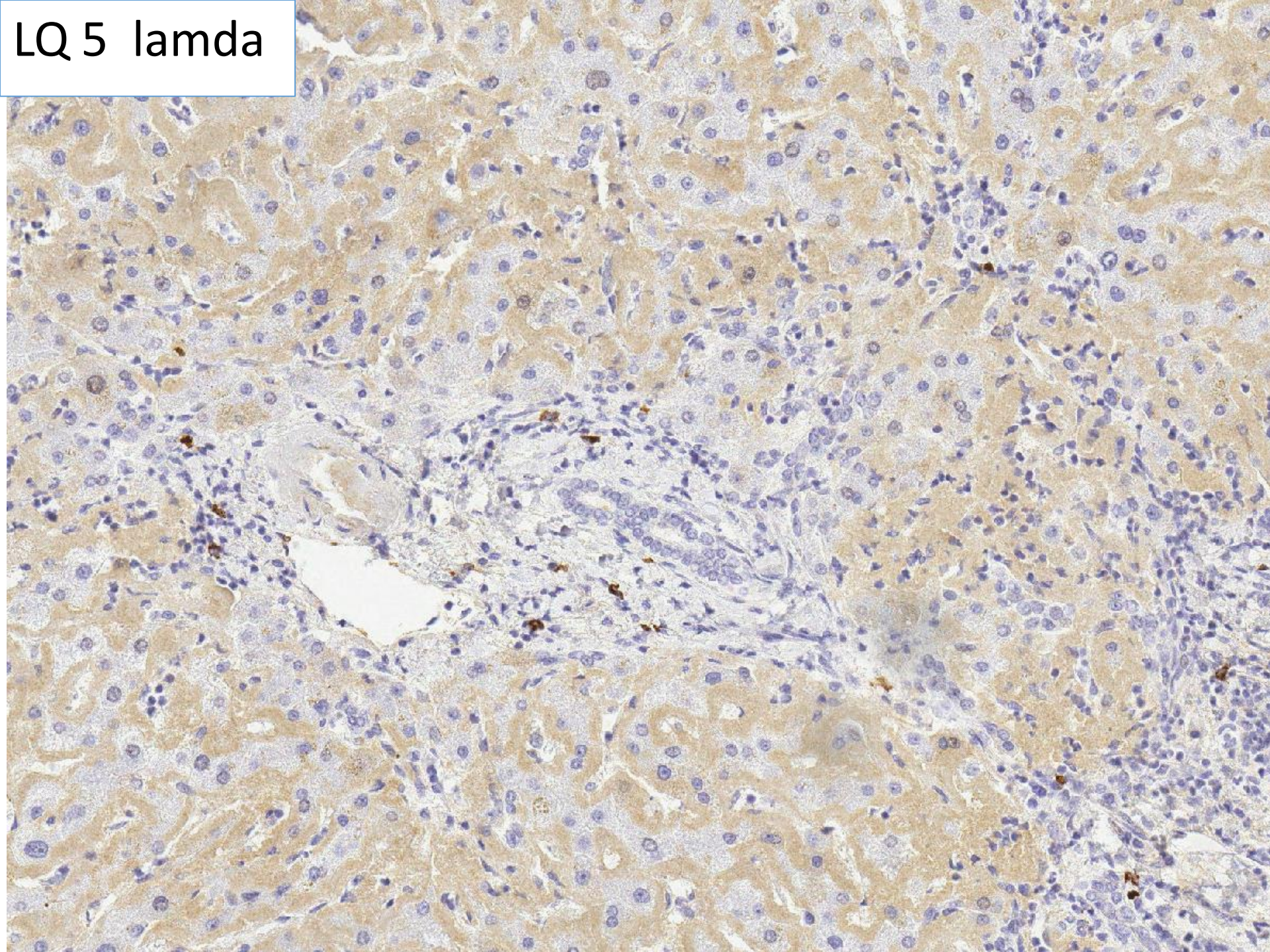

LQ5

LQ 5 PASD




LQ 5 IgG

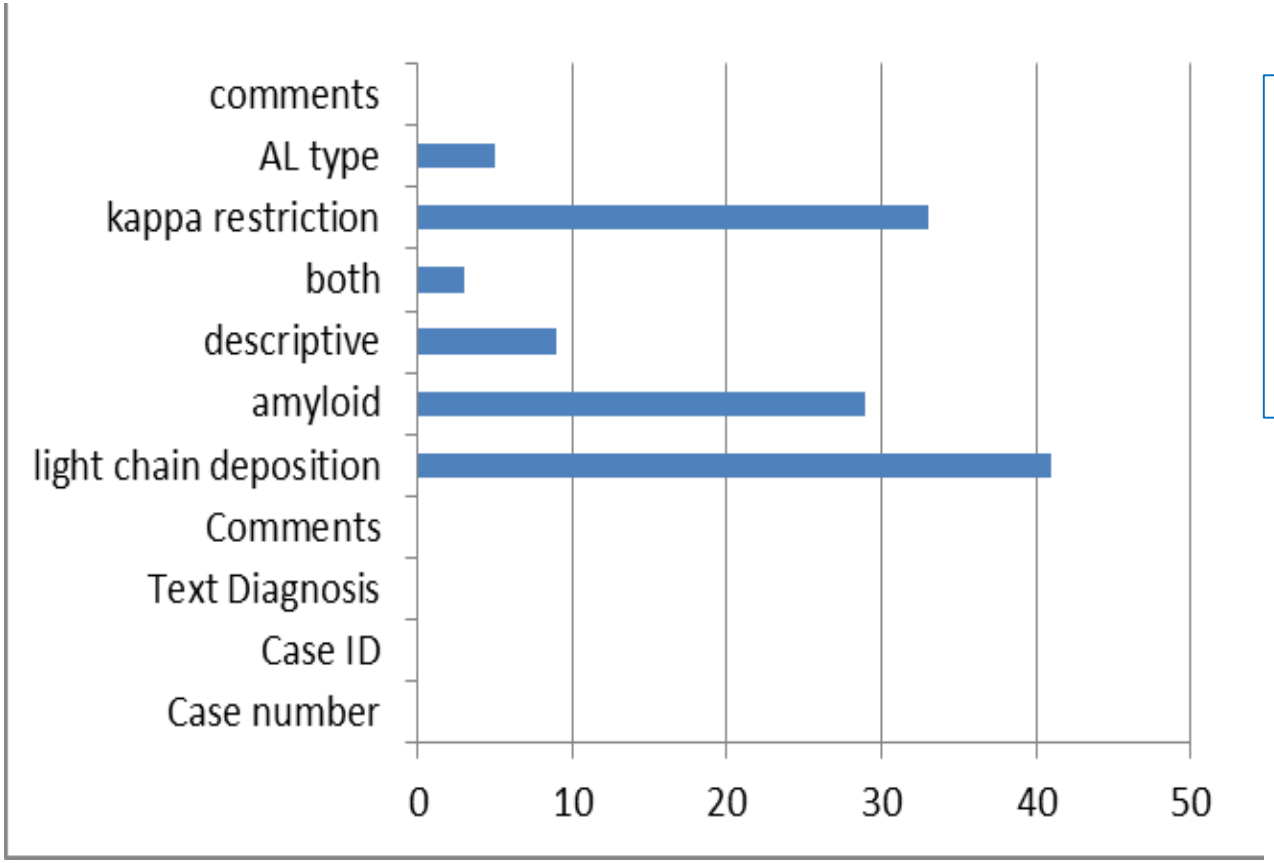

LQ 5 lamda

Case LQ5 65F

'Liver lesion segment 6/7'.

From electronic patient record IgG kappa paraproteinaemia.

Section is from background liver



Consensus diagnosis:

Light chain deposition disease
and/or amyloid

Just a description = 9

Or exclude case?

Case LQ5 65F

'Liver lesion segment 6/7'.

From electronic patient record IgG kappa paraproteinaemia.

Section is from background liver

Agreed scoring:

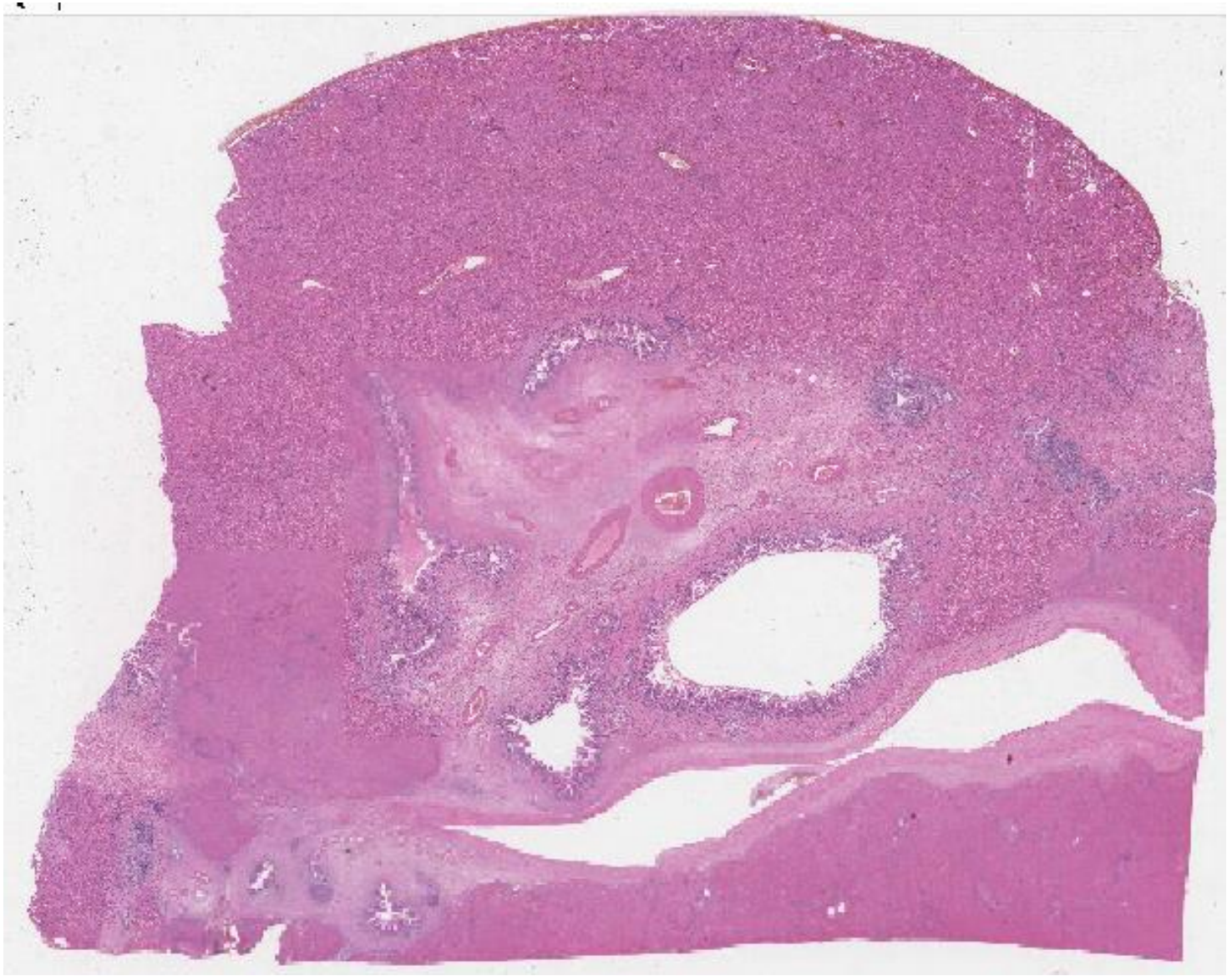
For full marks – need to include either light chain deposition disease, or amyloid.

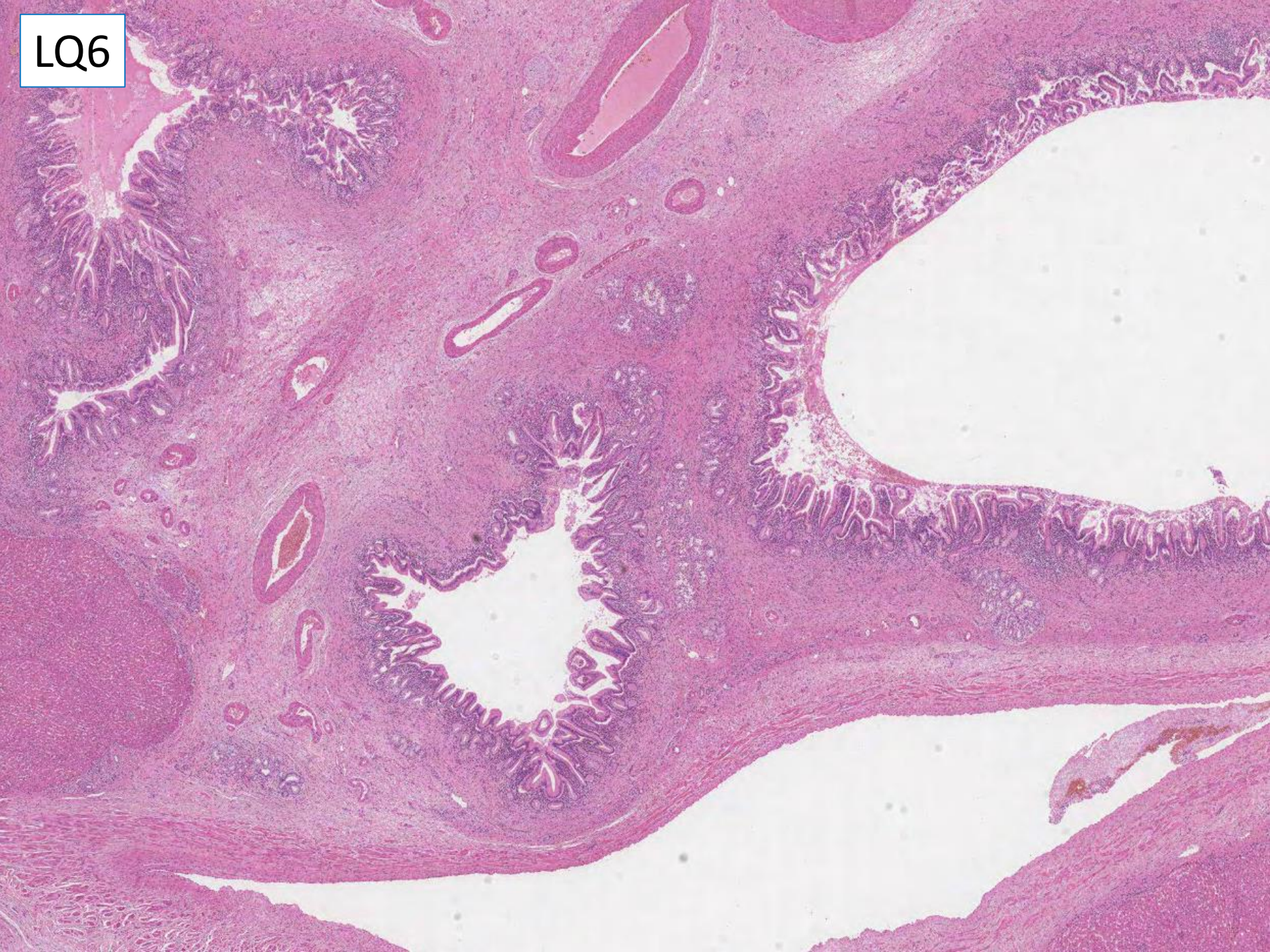
Score 5 points for a description of sinusoidal deposition of material without either of these diagnoses. Score 0 points for a diagnosis of abnormal plasmacytic cells in sinusoids, consistent with myeloma (deposition of material not mentioned).

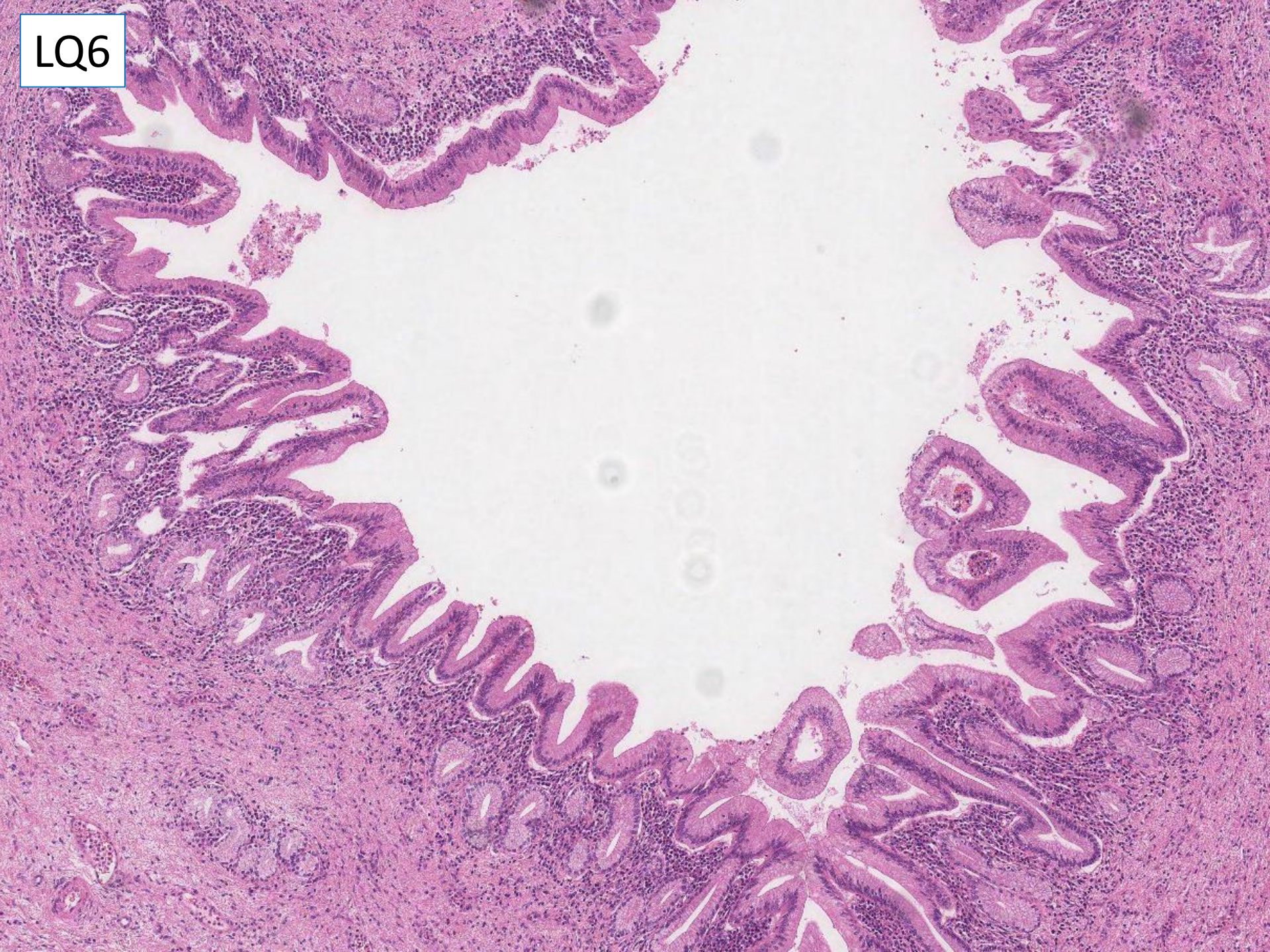
The PASD positive, Congo red material is characteristic of kappa light chain deposition disease, in keeping with the clnical history of IgG kappa paraproteinaemia.

Case LQ6. 85M

Recurrent abscess in left lobe of liver.




LQ6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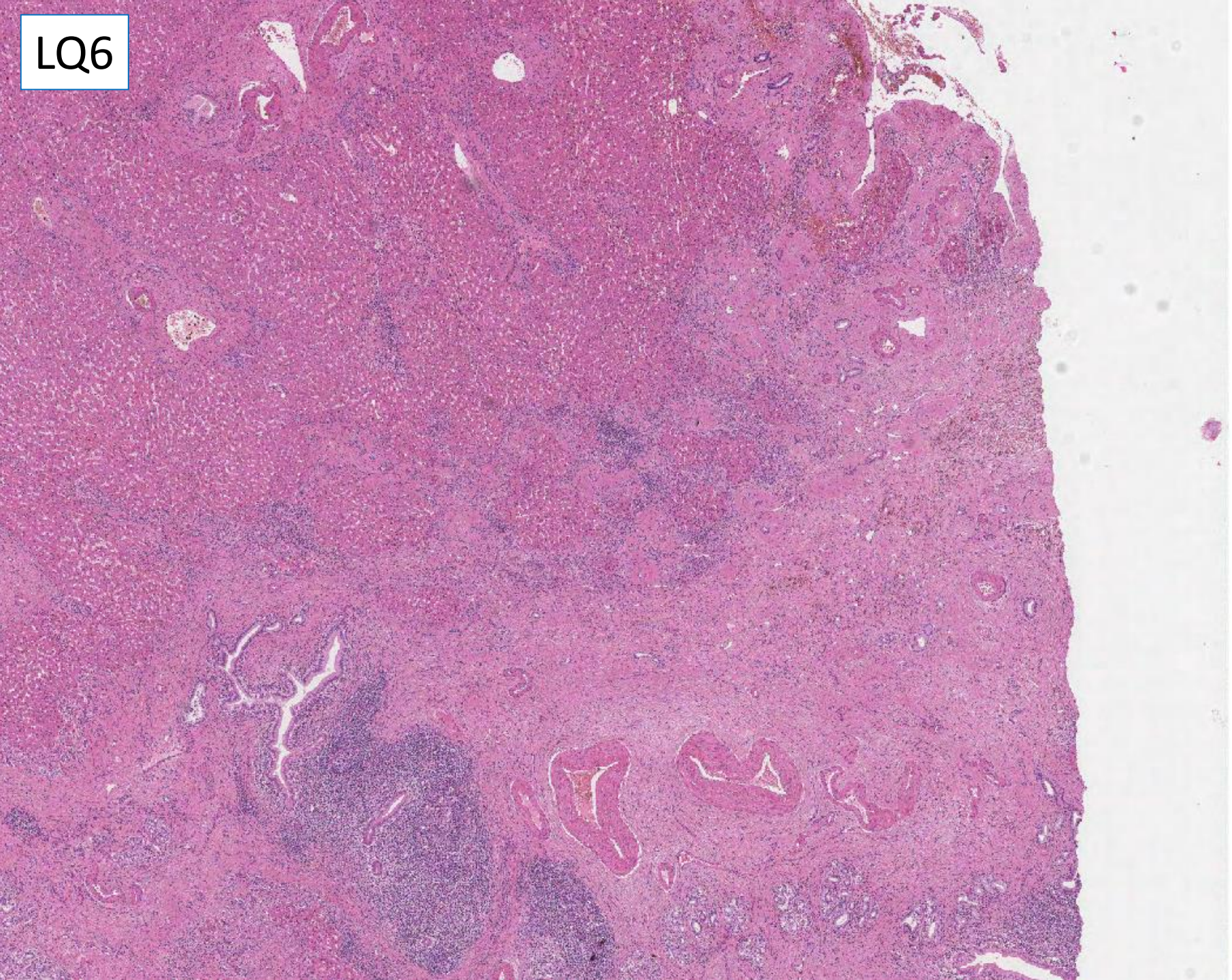
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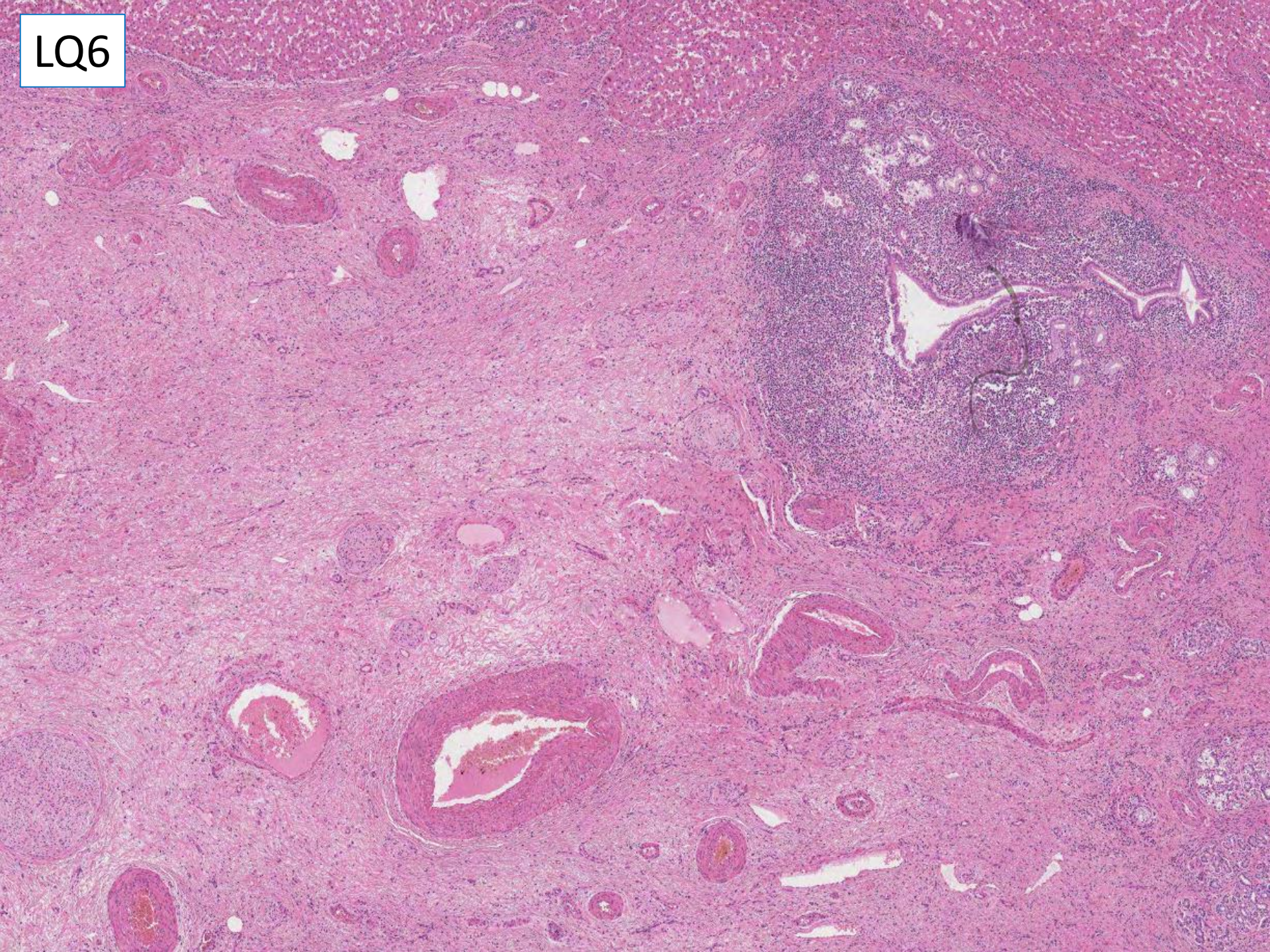
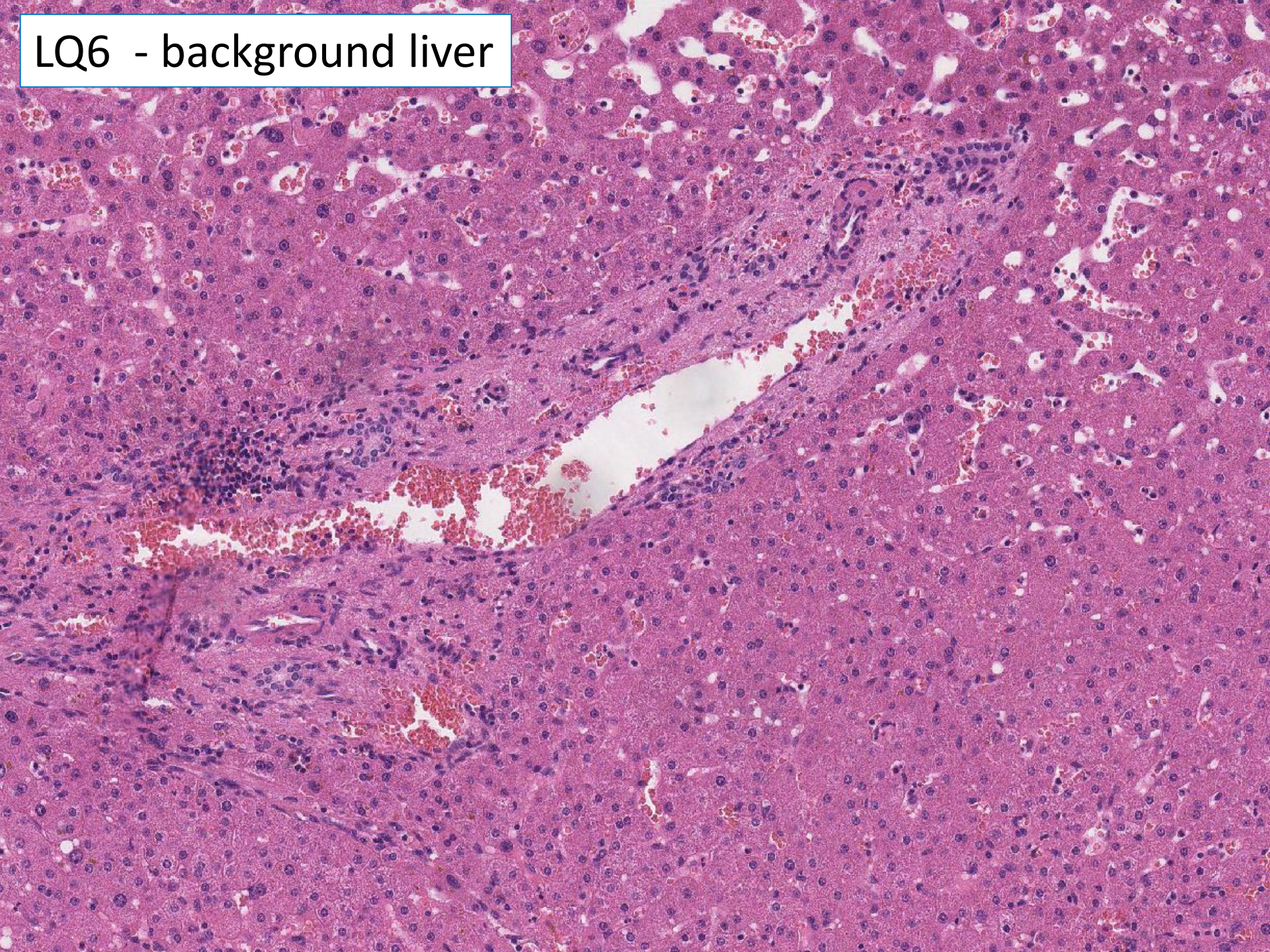

LQ6


LQ6 - background liver

Case LQ6. 85M

Recurrent abscess in left lobe of liver.

Chronic cholangitis with differential diagnoses – primary sclerosing cholangitis, IgG4-related disease, gallstones, malformation/scarring	35
Intraduct papillary neoplasm of bile ducts (IPNB)/intraduct papillary mucinous neoplasm (IPMN)/biliary intra-epithelial neoplasia (BilIN)	20
Caroli's disease	9
Choledochal cyst/Simple cyst	6
Biliary cystadenoma/mucinous cystadenoma	3
Hamartoma	3
Don't know/no clear diagnosis/biliary lesion	3
IgG4-related disease (as only diagnosis)	1
Fungal abscess	1
Autoimmune hepatitis/hepatitis C overlap	1
Own case	1

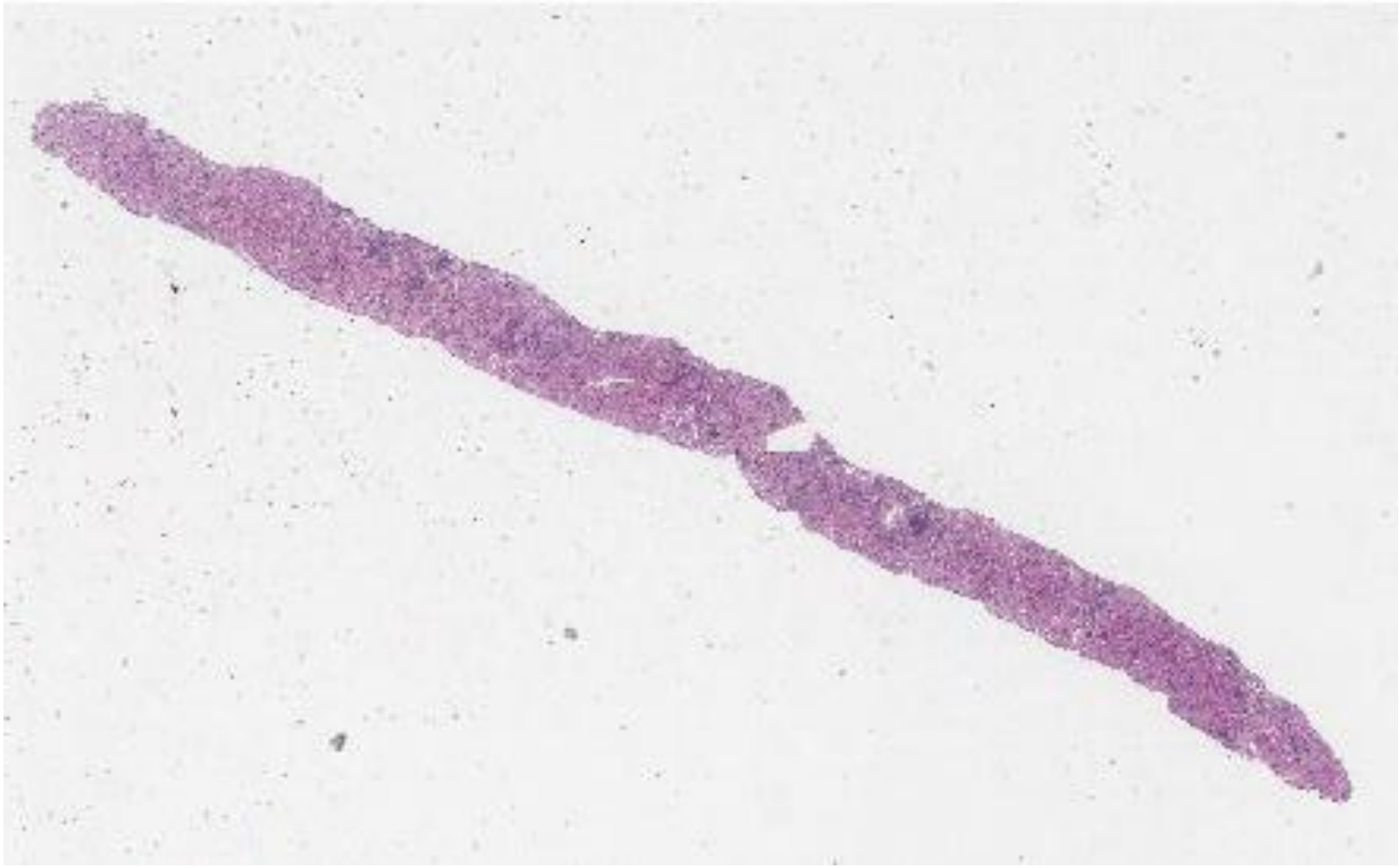
Consensus diagnosis:

no consensus for this case.

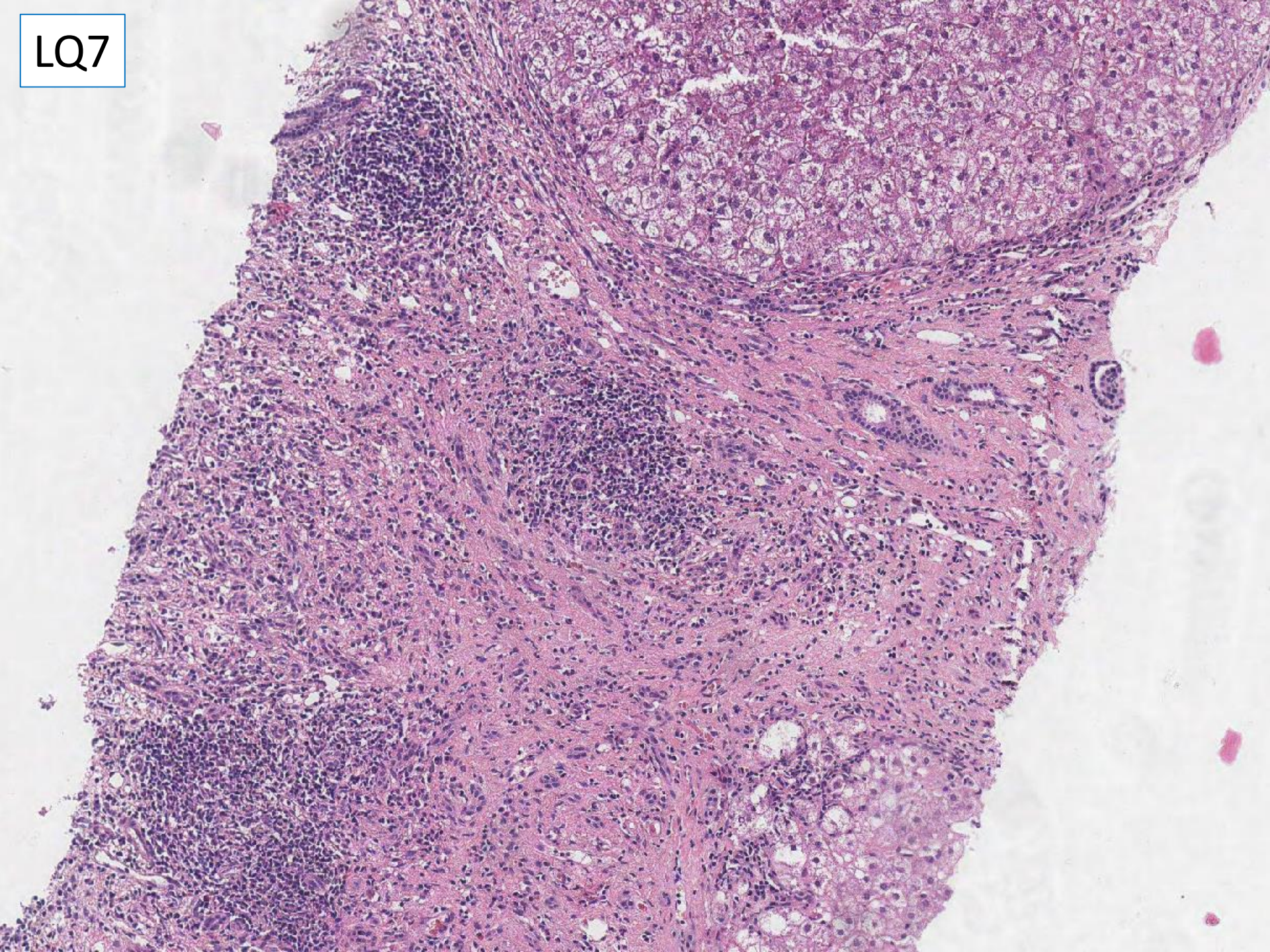
Not suitable for scoring

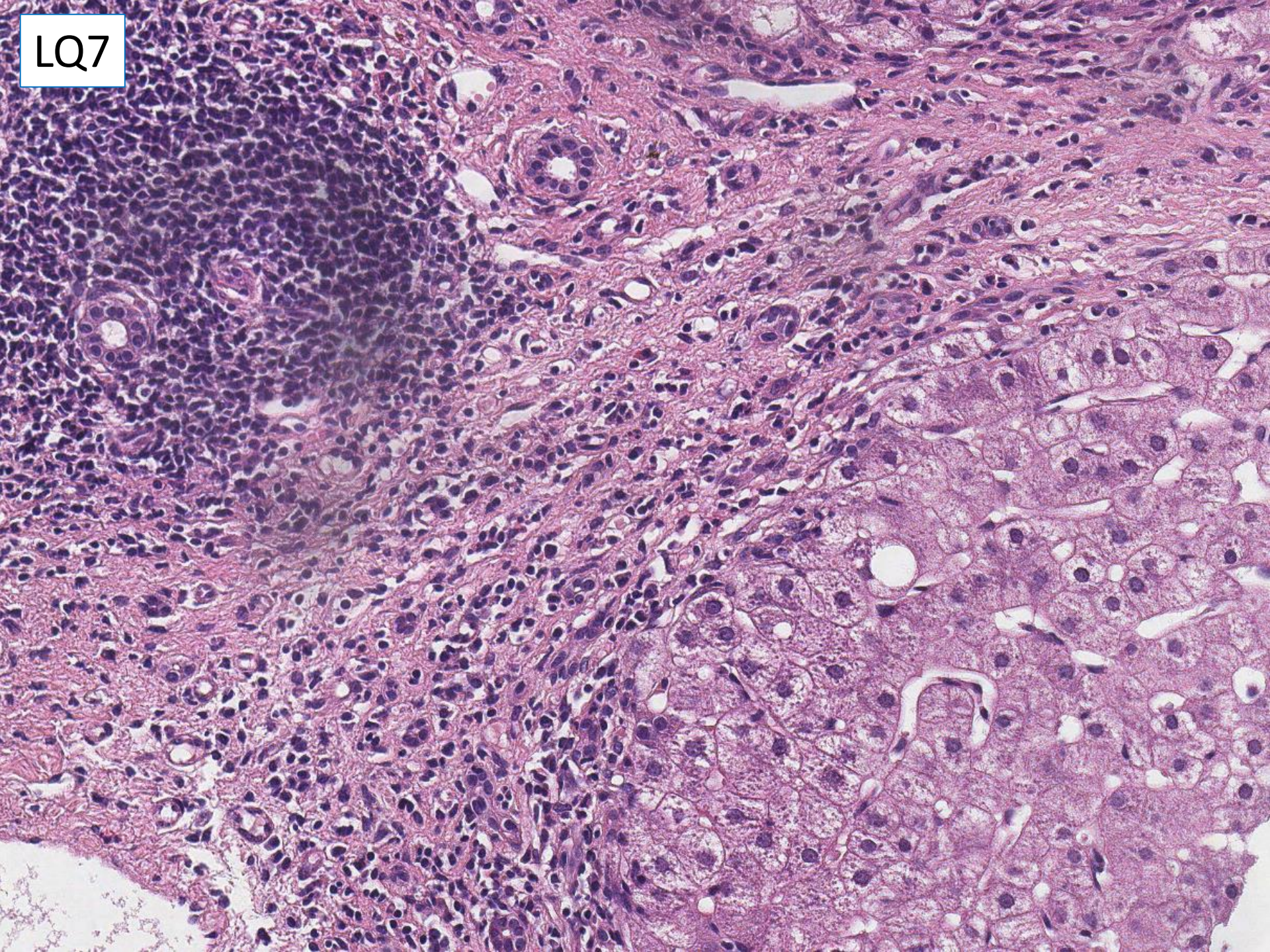
Case LQ7 60F

Chronic hepatitis C. Positive mitochondrial antibodies and raised IgG. Type II diabetes and obesity. Liver biopsy to rule out autoimmune hepatitis. (also VG, CK8/18)



LQ7

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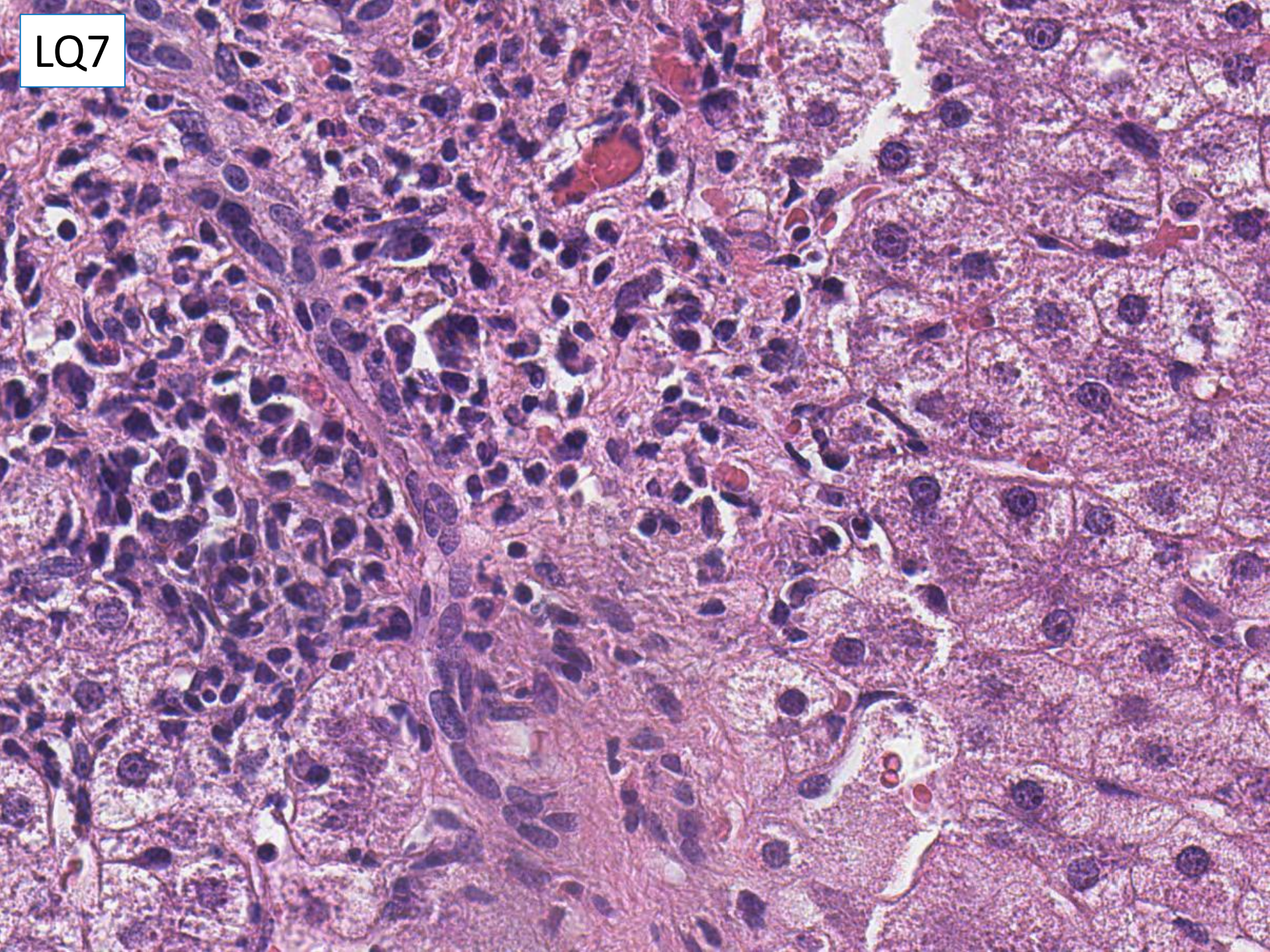
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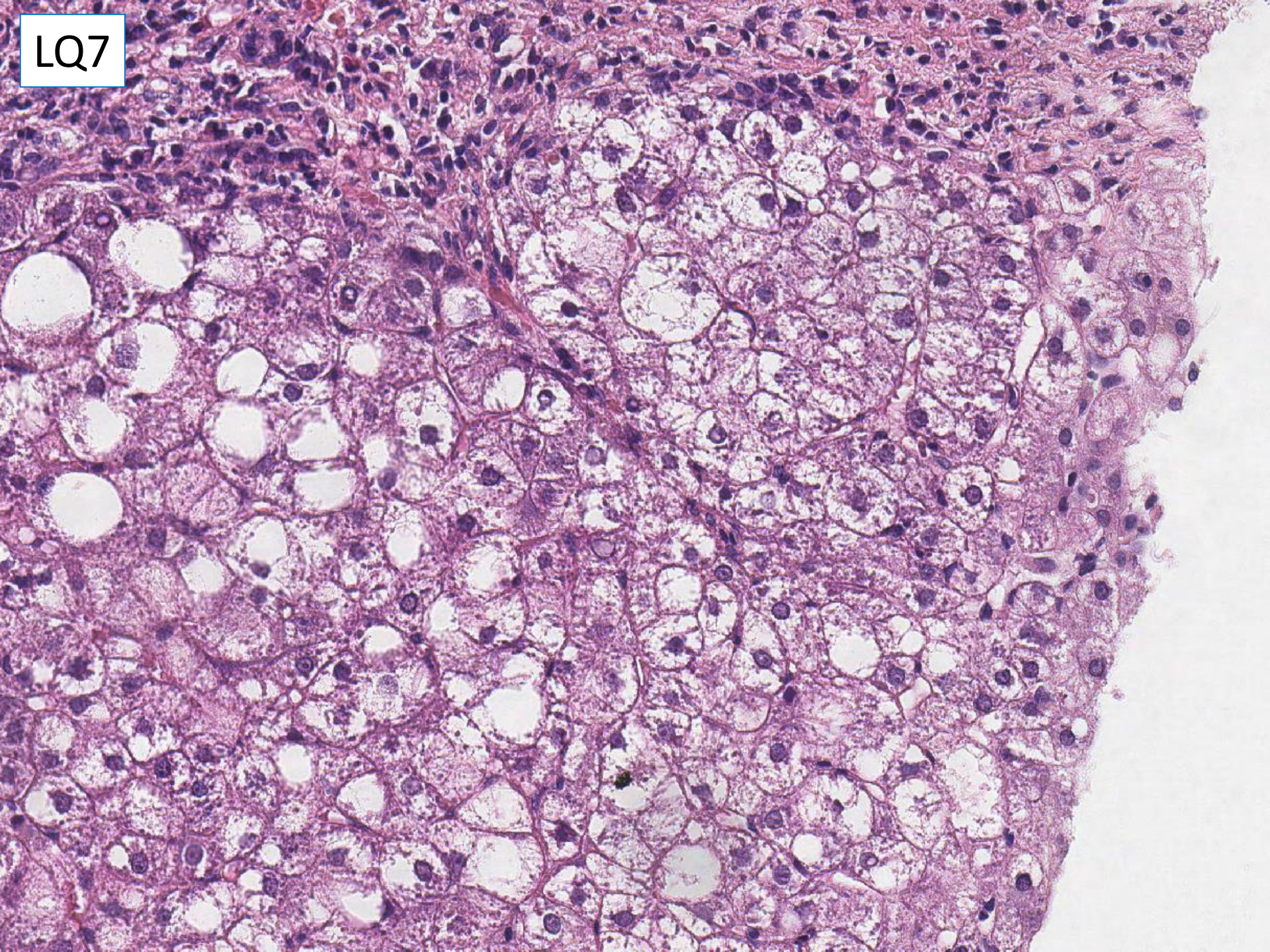
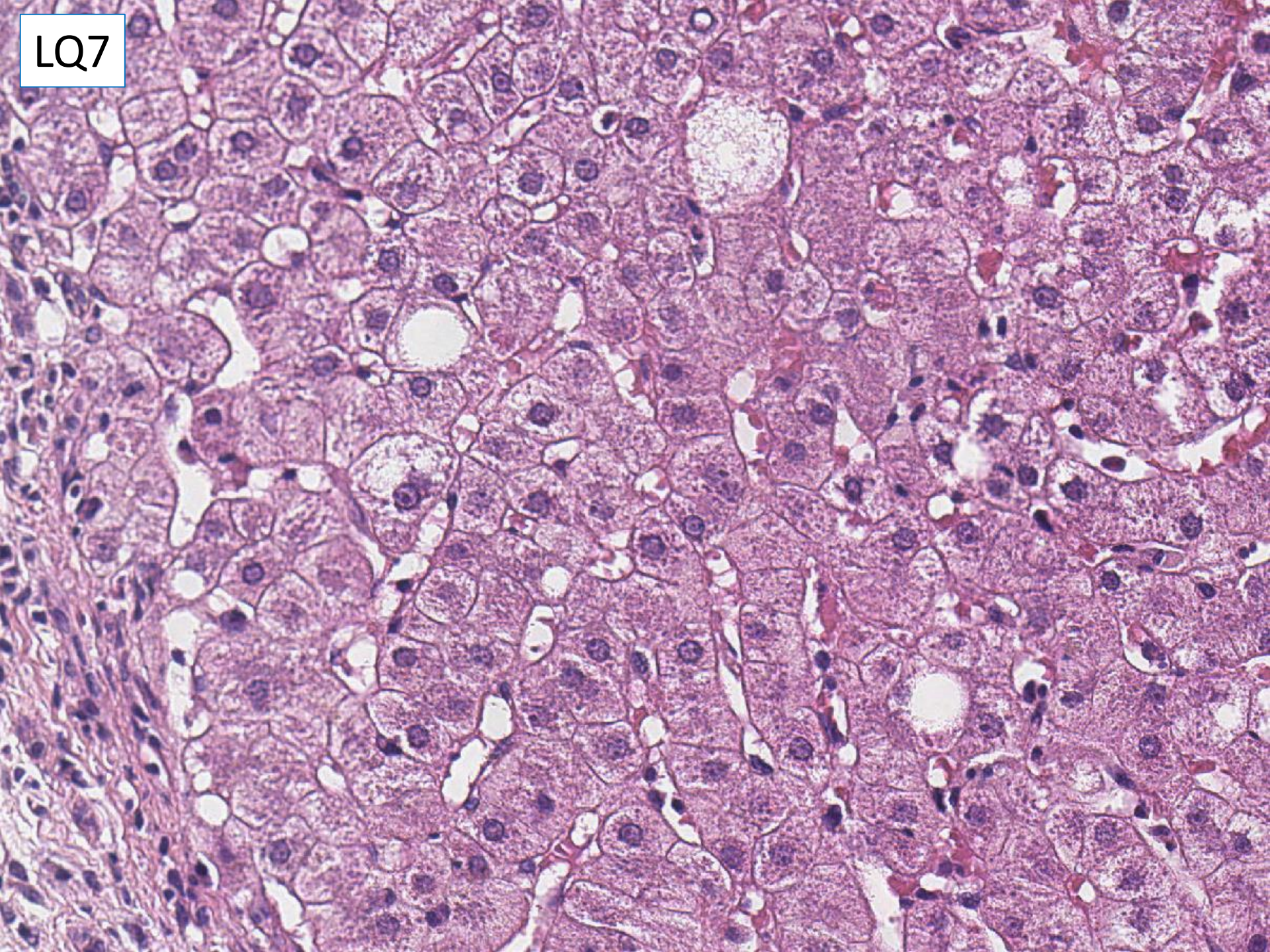
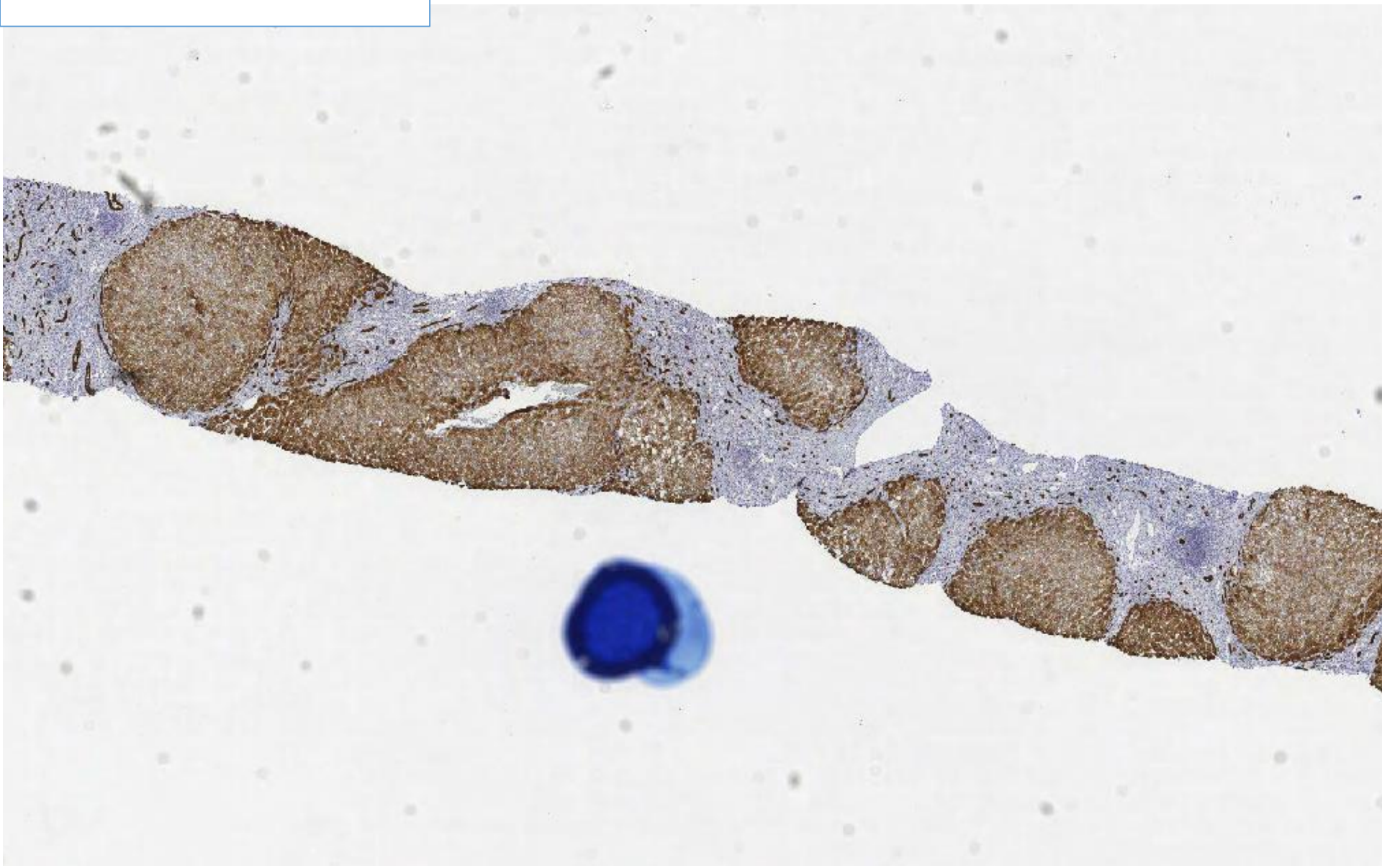
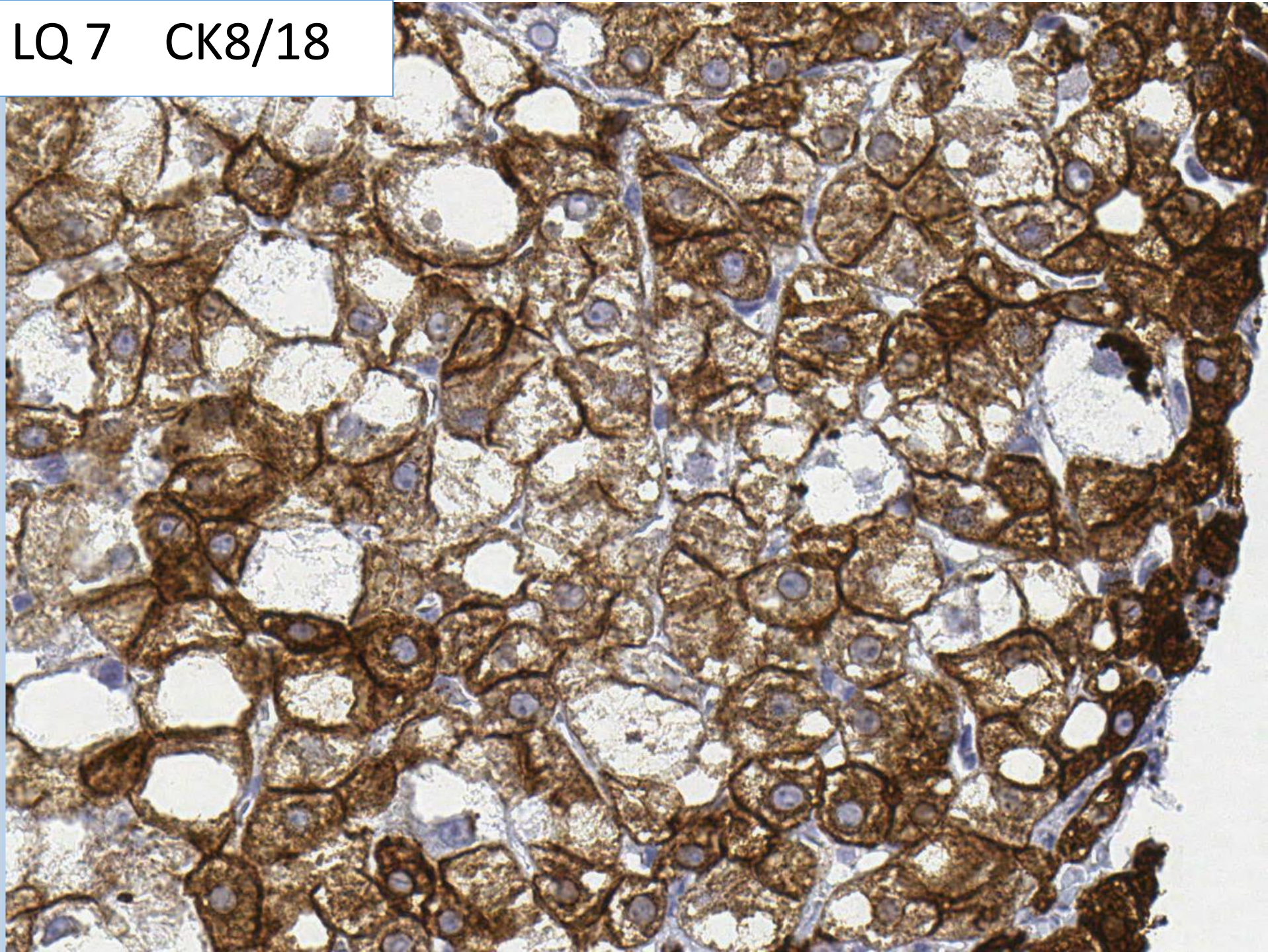

LQ7


LQ7

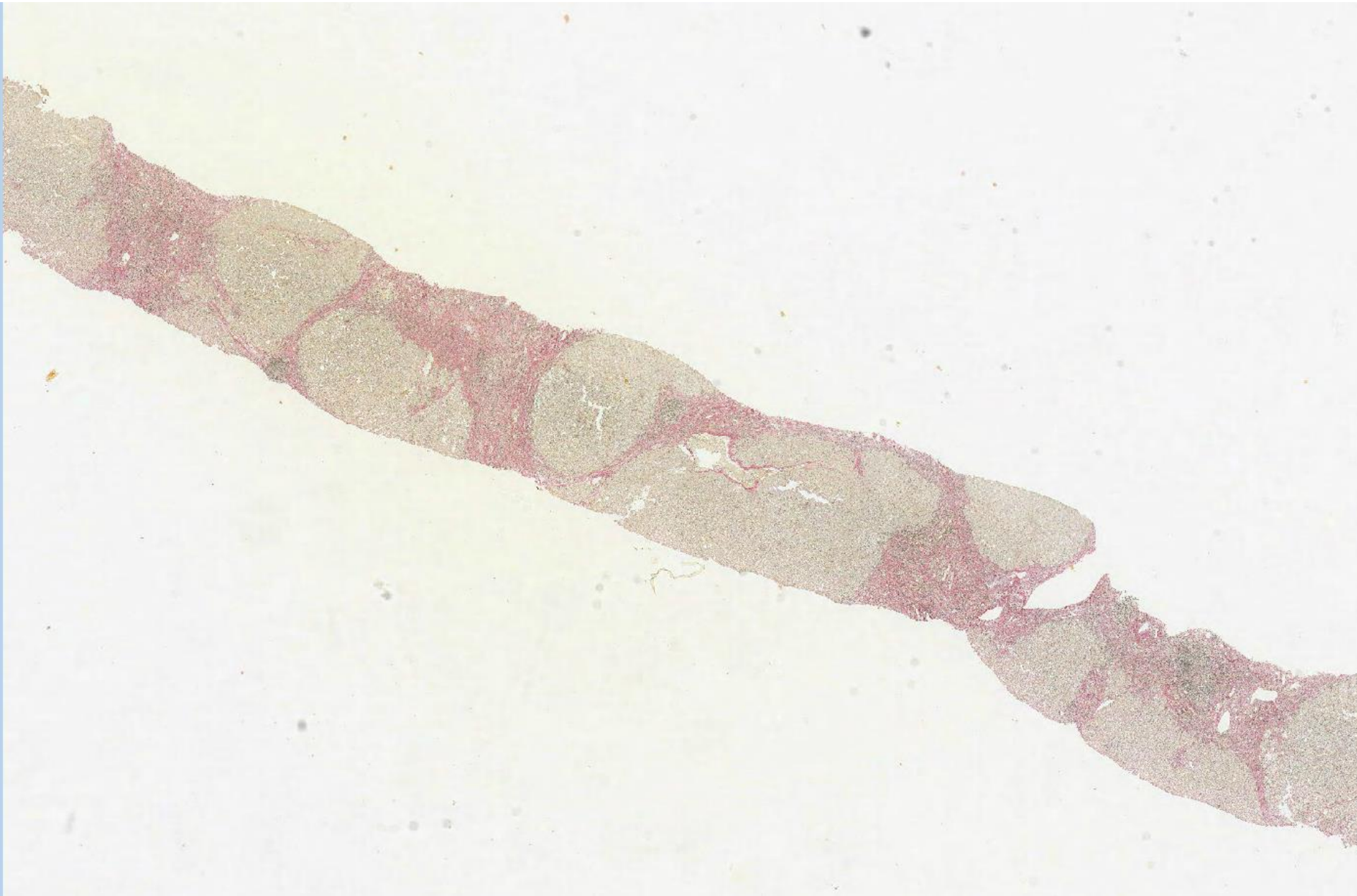
LQ 7 CK8/18



LQ 7 CK8/18



LQ 7 van Gieson



Case LQ7 60F

Chronic hepatitis C. Positive mitochondrial antibodies and raised IgG. Type II diabetes and obesity. Liver biopsy to rule out autoimmune hepatitis. (also VG, CK8/18)

Cirrhosis	83
Hepatitis C	76
Hepatitis C not mentioned	4
Unlikely/not in keeping with hepatitis C	2
Hepatitiis B	1
Non-alcoholic steatohepatitis	41
Describe features of steatohepatitis, but don't use that word	9
Steatosis	15
No steatohepatitis / no steatosis	6
Fatty liver disease not mentioned	11
Autoimmune hepatitis likely or possible	48
Not features of autoimmune hepatitis	21
Autoimmune hepatitis not mentioned	12
Primary biliary cholangitis/PBC likely or possible	17
No features of biliary disease	25
Biliary disease not mentioned	30
Needs Orcein/CK7 to look for biliary features	4
Overall:	
HCV only	10
HCV and steatohepatitis	17
HCV and autoimmune hepatitis, not PBC	14
HCV and PBC, not AIH	3
HCV and PBC and AIH - not steatohepatitis	6
Full house: HCV, steatohepatitis and autoimmune disease	26

Consensus response:

cirrhosis, mixed aetiology - not just hepatitis C.

But there is no consensus about the relative contributions of hepatitis C, steatohepatitis, and autoimmune disease either AIH or PBC.

In practice this biopsy would need to be discussed at a CPC meeting.

Hep C only – half marks

Case LQ7 60F

Chronic hepatitis C. Positive mitochondrial antibodies and raised IgG. Type II diabetes and obesity. Liver biopsy to rule out autoimmune hepatitis. (also VG, CK8/18)

Agreed scoring:

For full marks – Cirrhosis (all responses included cirrhosis), hepatitis C, and consideration of other potential aetiological factors.

Score 5 points for responses where hepatitis C was not mentioned, hepatitis B instead – or where features were considered unlikely/not in keeping with hepatitis C.
On review – there were two cases with a definite diagnosis of PBC/AIH with no mention of hepatitis C – scored 0 points.

Score 5 points for responses where only hepatitis C was mentioned, without any consideration of potential role of other aetiological factors – although there was no consensus on steatohepatitis/autoimmune hepatitis / primary biliary cholangitis. On review – all these responses included some discussion of potential role of other aetiologies, but concluded hepatitis C alone was sufficient to account for the histology.

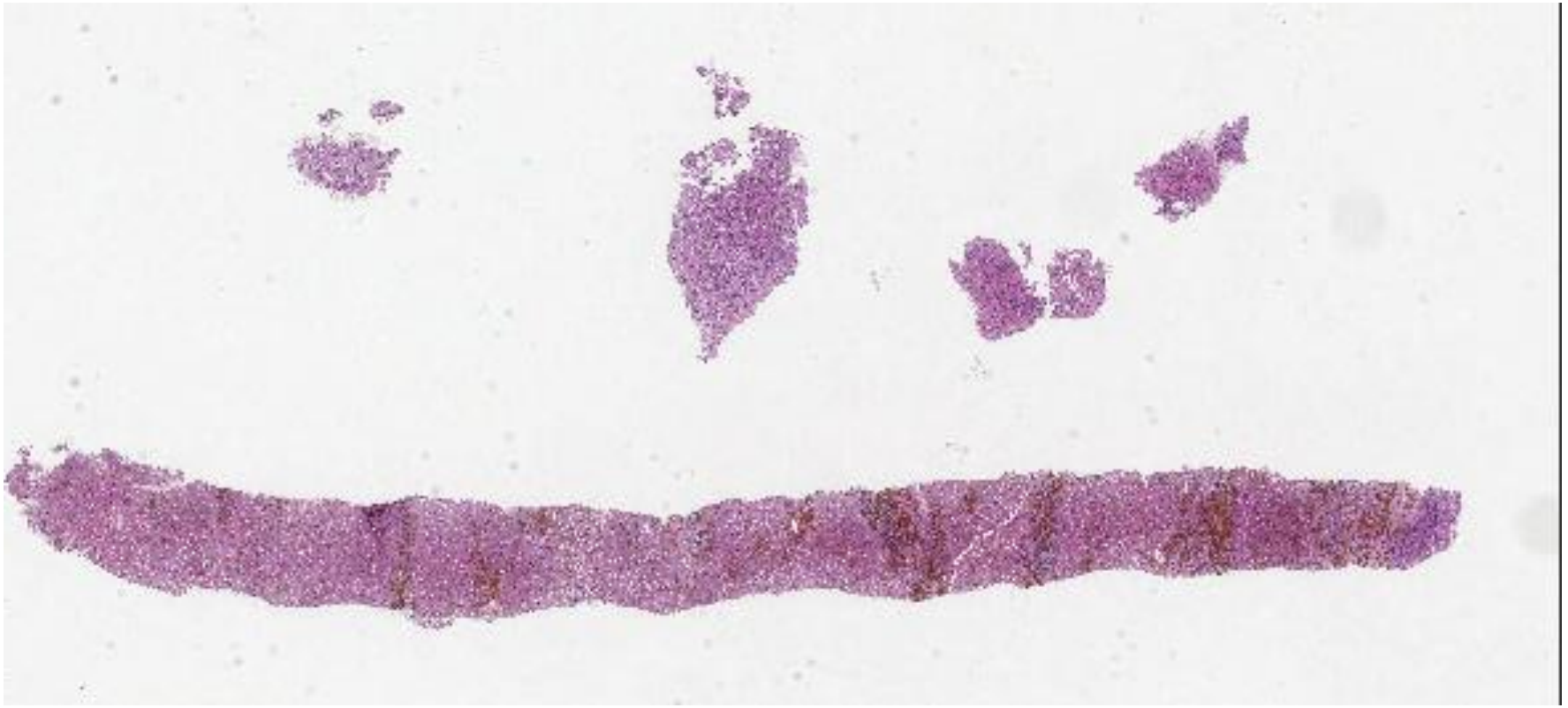
Follow up: the patient was treated with antivirals 9 months after this biopsy, ALT fell to normal within 4 weeks (having been previously raised for years) and she achieved a sustained viral response.

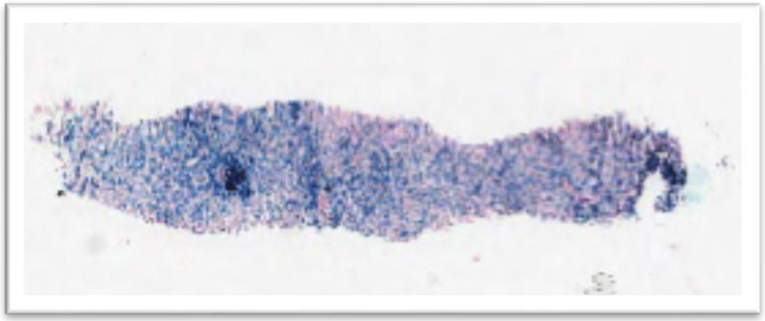
Discussion: role of hepatitis C interacting with NAFLD – the role is synergistic, genotype 3 can cause steatosis alone, other genotypes potentiate steatosis in patients with other risk factors. So improvement in ALT doesn't mean that there wasn't also a component of metabolic steatohepatitis as well as chronic hepatitis from hepatitis C.

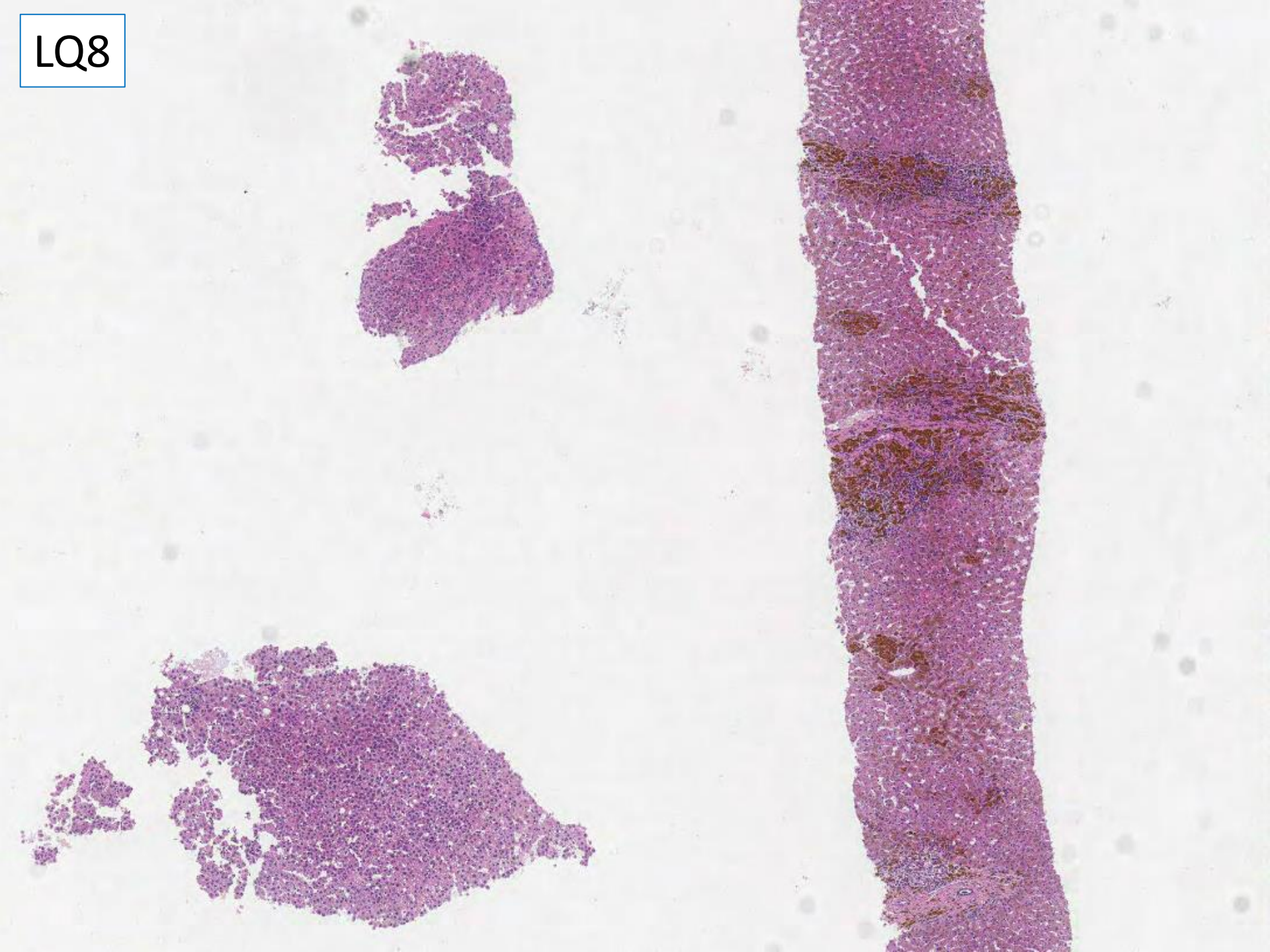
Autoantibodies are common at low titre in hepatitis C – quoted 20% SMA, 5% AMA.

Case LQ8 69M

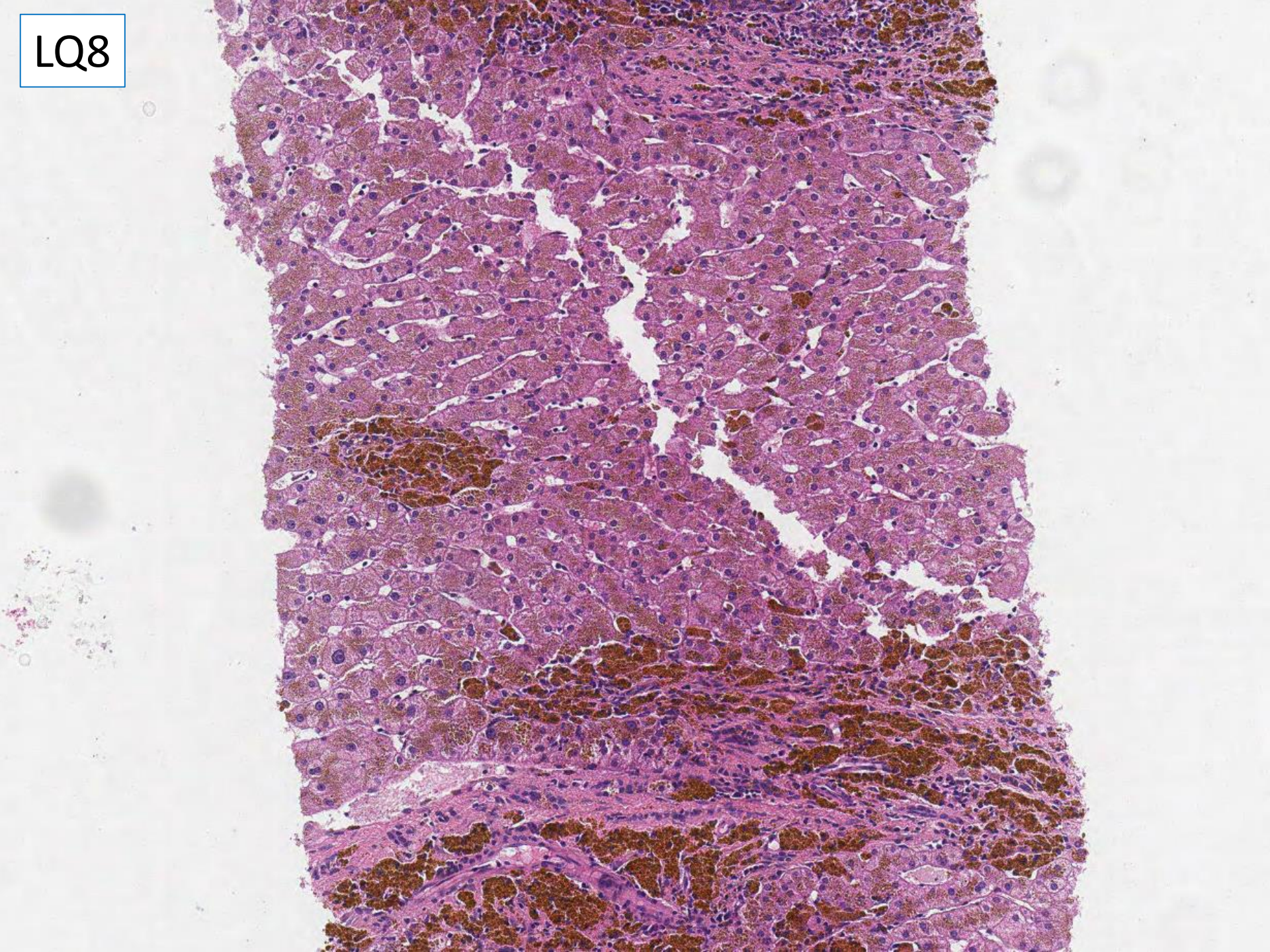
? HCC pre-ablation. 18G biopsy. Also Perls stain (little tissue left in block)



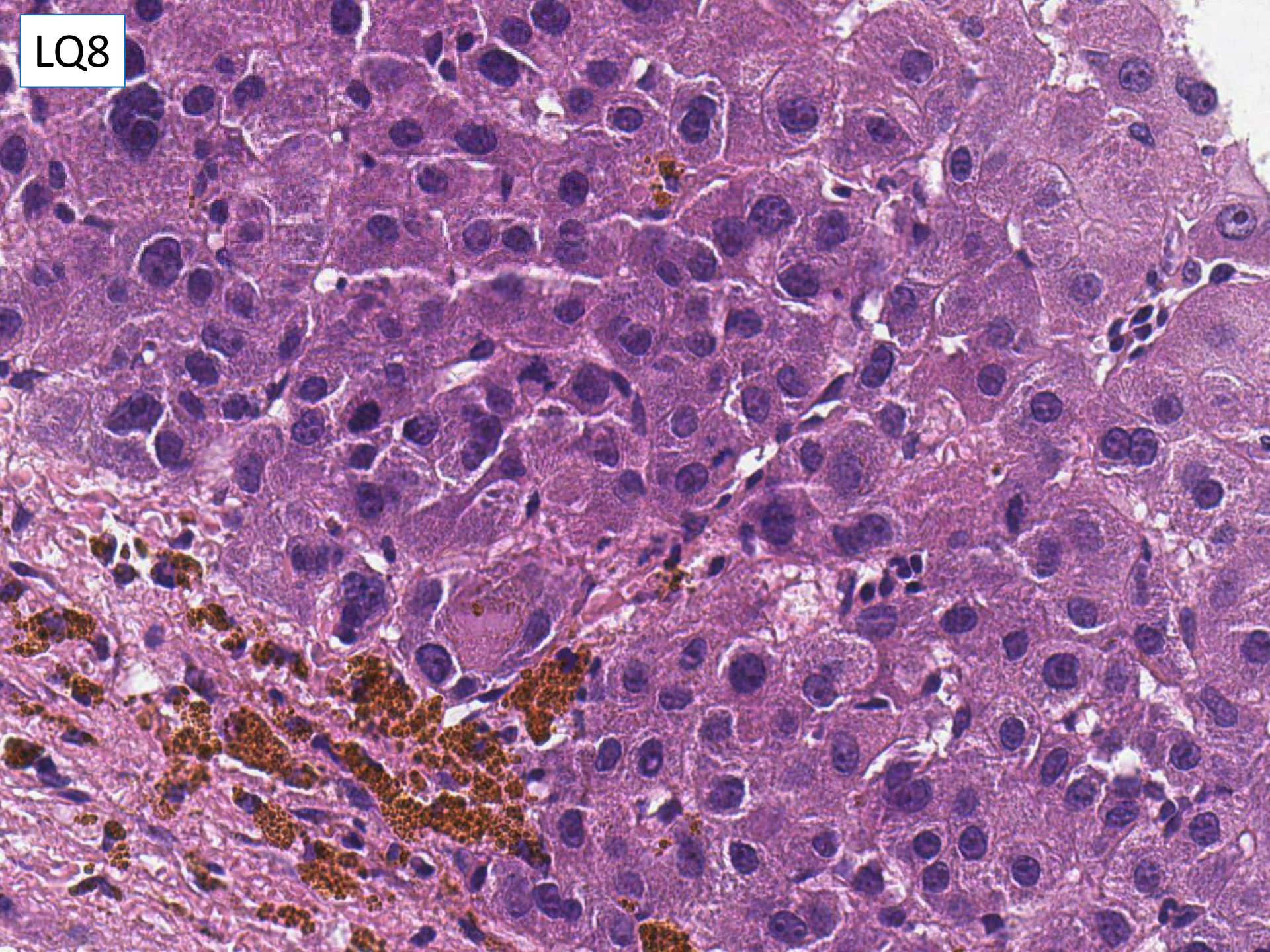



LQ8

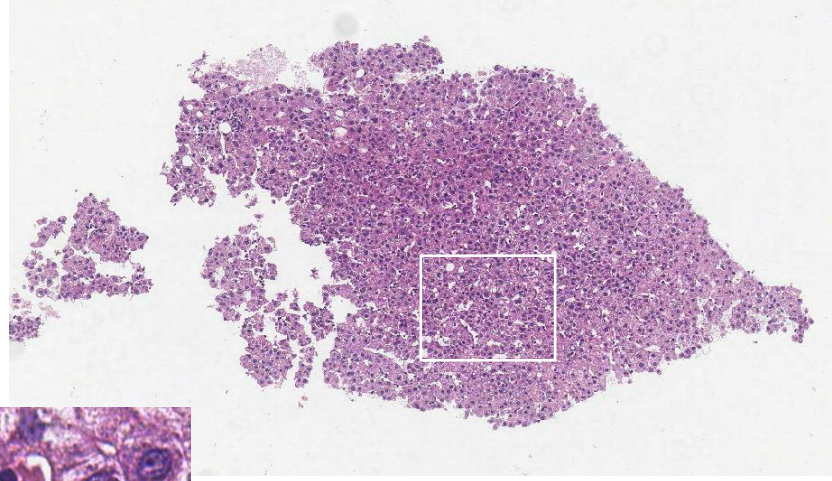
LQ8

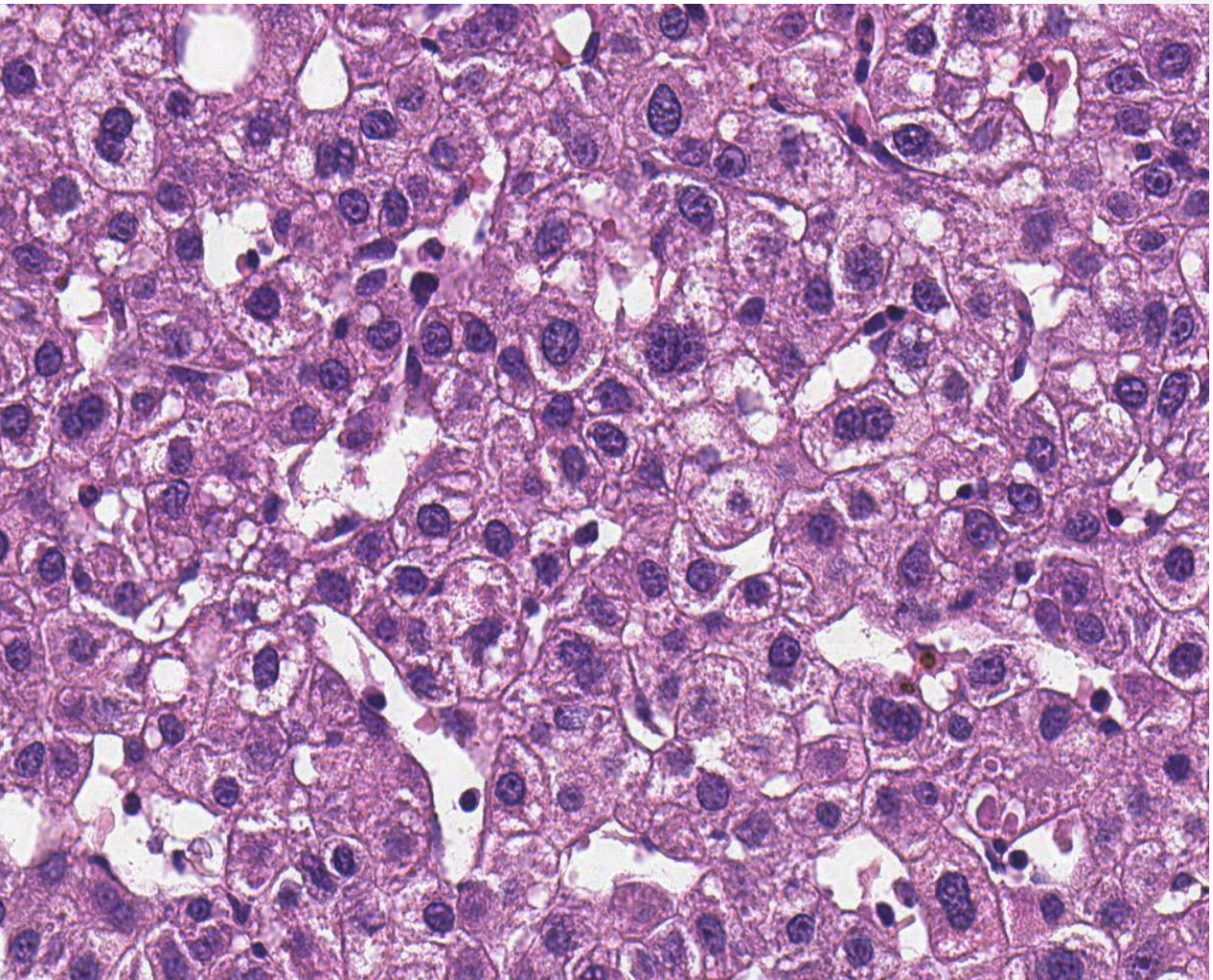


LQ8

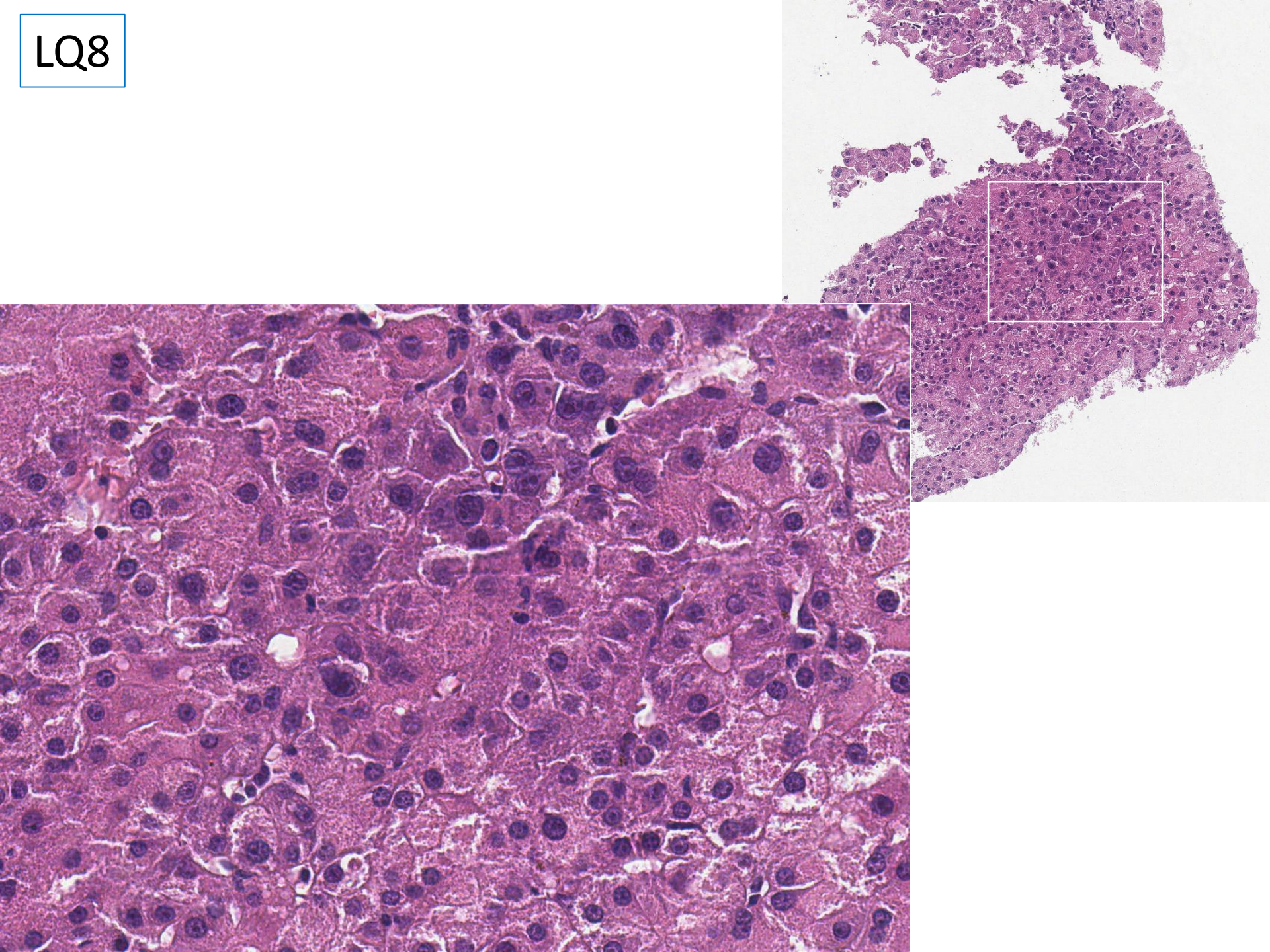

LQ8

LQ8

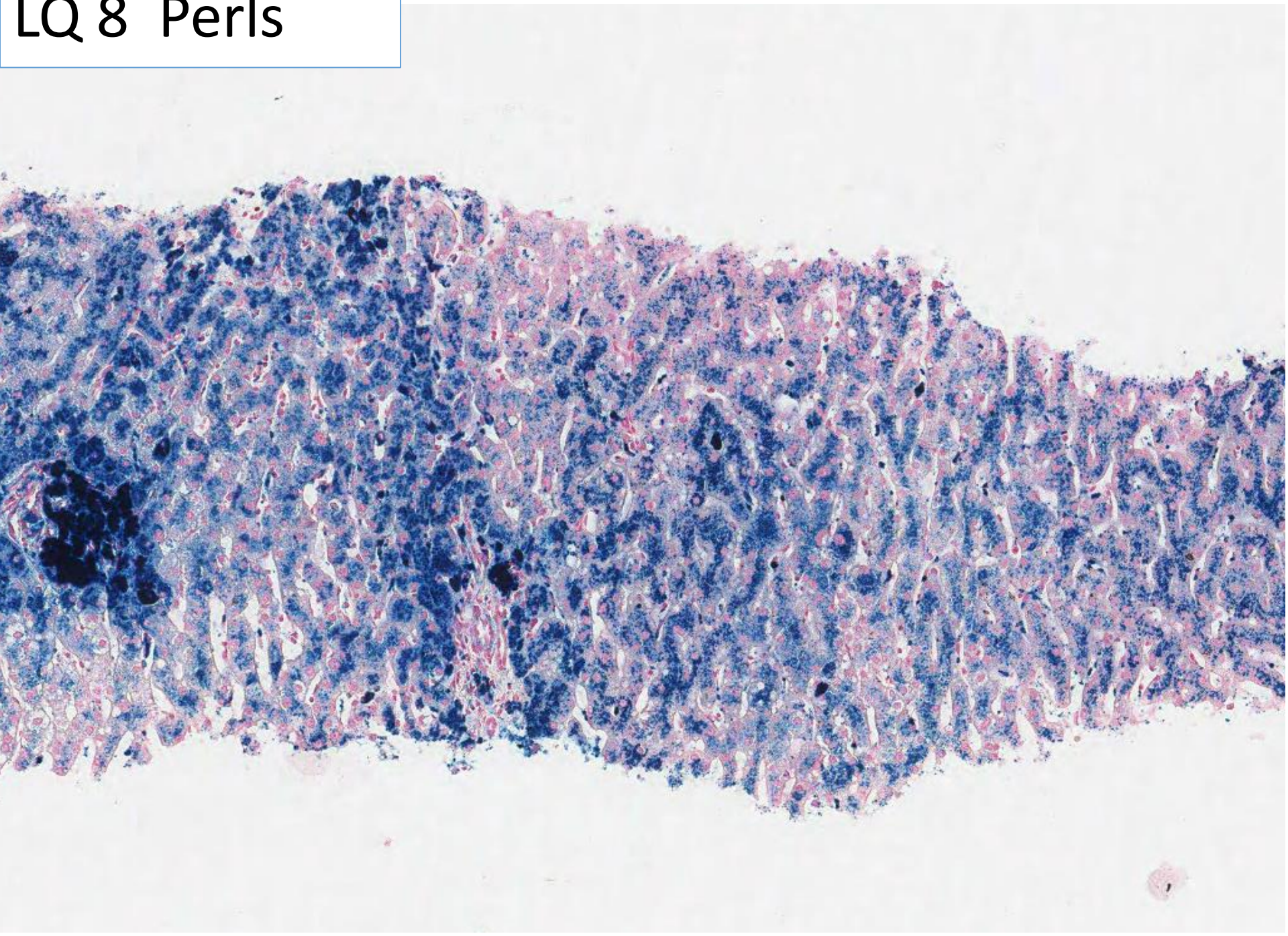




LQ8



LQ 8 Perls



Case LQ8 69M

? HCC pre-ablation. 18G biopsy. Also Perls stain (little tissue left in block)

Hepatocellular carcinoma	50
Suspect HCC, do additional stains	17
HGDN or HCC	1
Small cell dysplasia	2
No HCC	3
No mention of a focal lesion	11
Severe iron overload and either haemochromatosis or discussion of possible cause	83
Of which Haemochromatosis/genetic cause/transfusion related?	78
No cause of siderosis	5
Cirrhosis probable or definite	9
Bridging fibrosis, portal fibrosis	14
'not cirrhotic'	10
Insufficient for stage/need special stains	16
No mention of stage	31

Consensus diagnosis:

hepatocellular carcinoma, or suspect HCC and do additional stains;

background iron overload, investigate for cause,

probably not cirrhotic but insufficient in the biopsy to assess the stage.

Survey results: Small cell dysplasia 0/2/3
No cause for siderosis 0/5/1

Case LQ8 69M

? HCC pre-ablation. 18G biopsy. Also Perls stain (little tissue left in block)

Agreed scoring:

For full marks: a diagnosis of hepatocellular carcinoma, or suspect HCC and do additional stains. Also include siderosis with a consideration of the cause, haemochromatosis/genetic cause/transfusion related.

Lose 5 points for small cell dysplasia.
Score 0 points if no mention of a focal lesion, or specifically stated that HCC was not present.

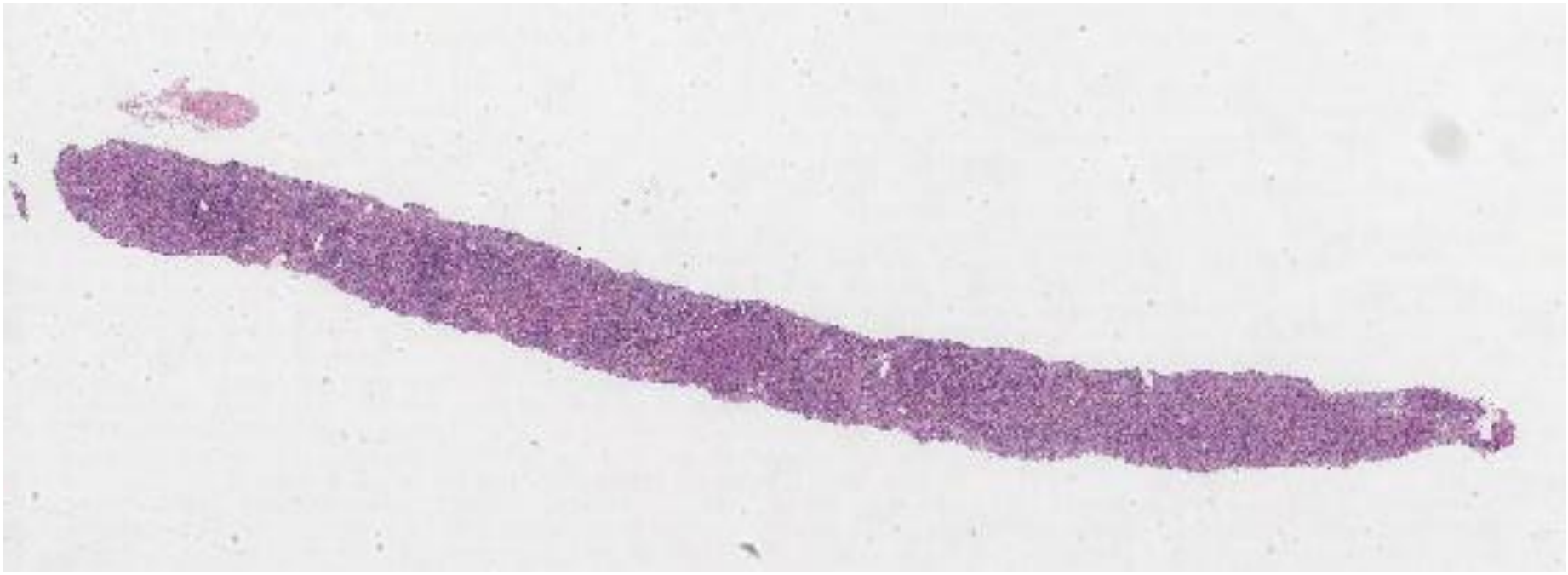
Lose 5 points if there is no mention of a clinical cause for siderosis.

Assessment of fibrosis was not scored – this was perilesional tissue, and without connective tissue stains provided.

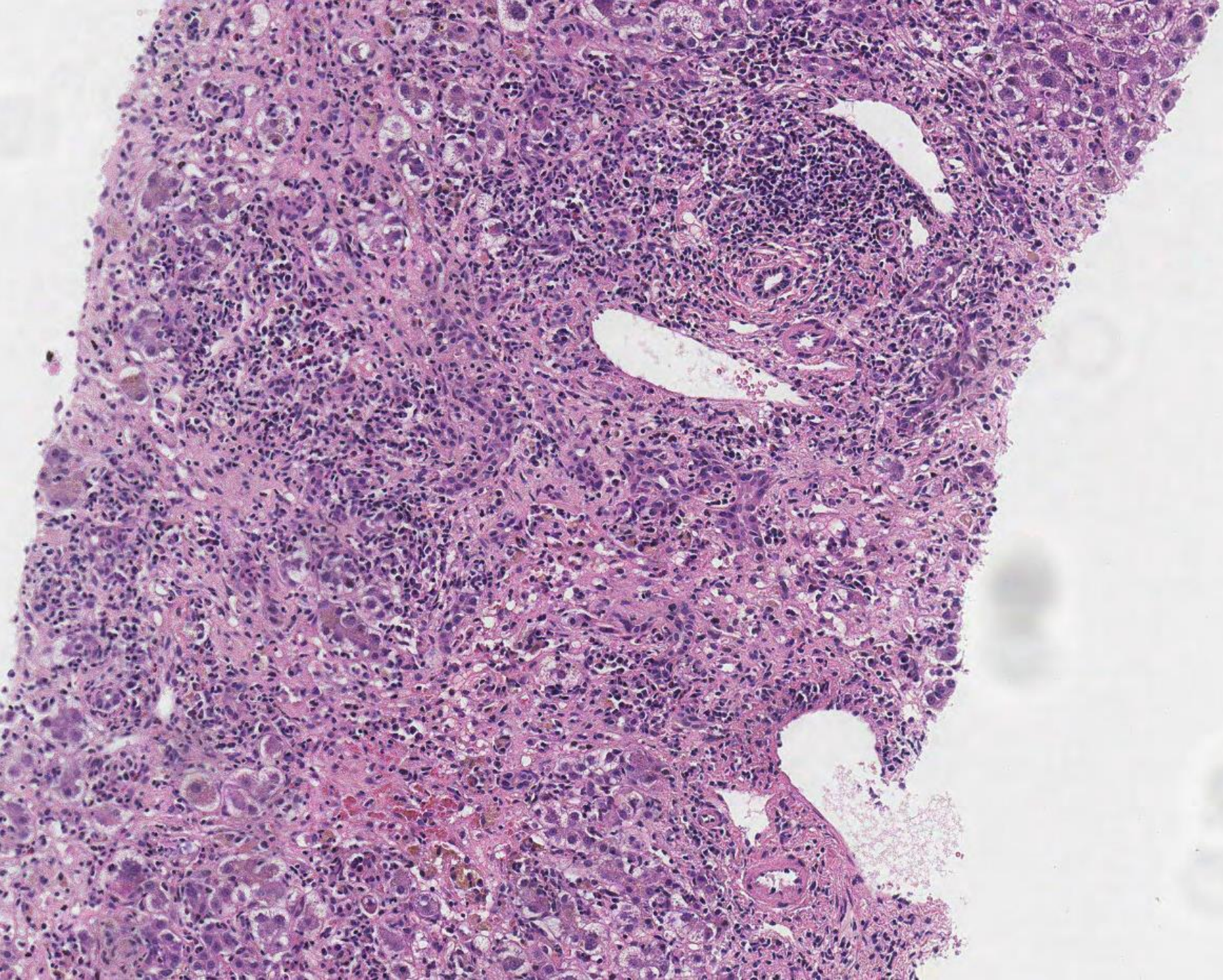
Follow up information: found to by C282y homozygous haemochromatosis. So far no recurrence of HCC following RFA.

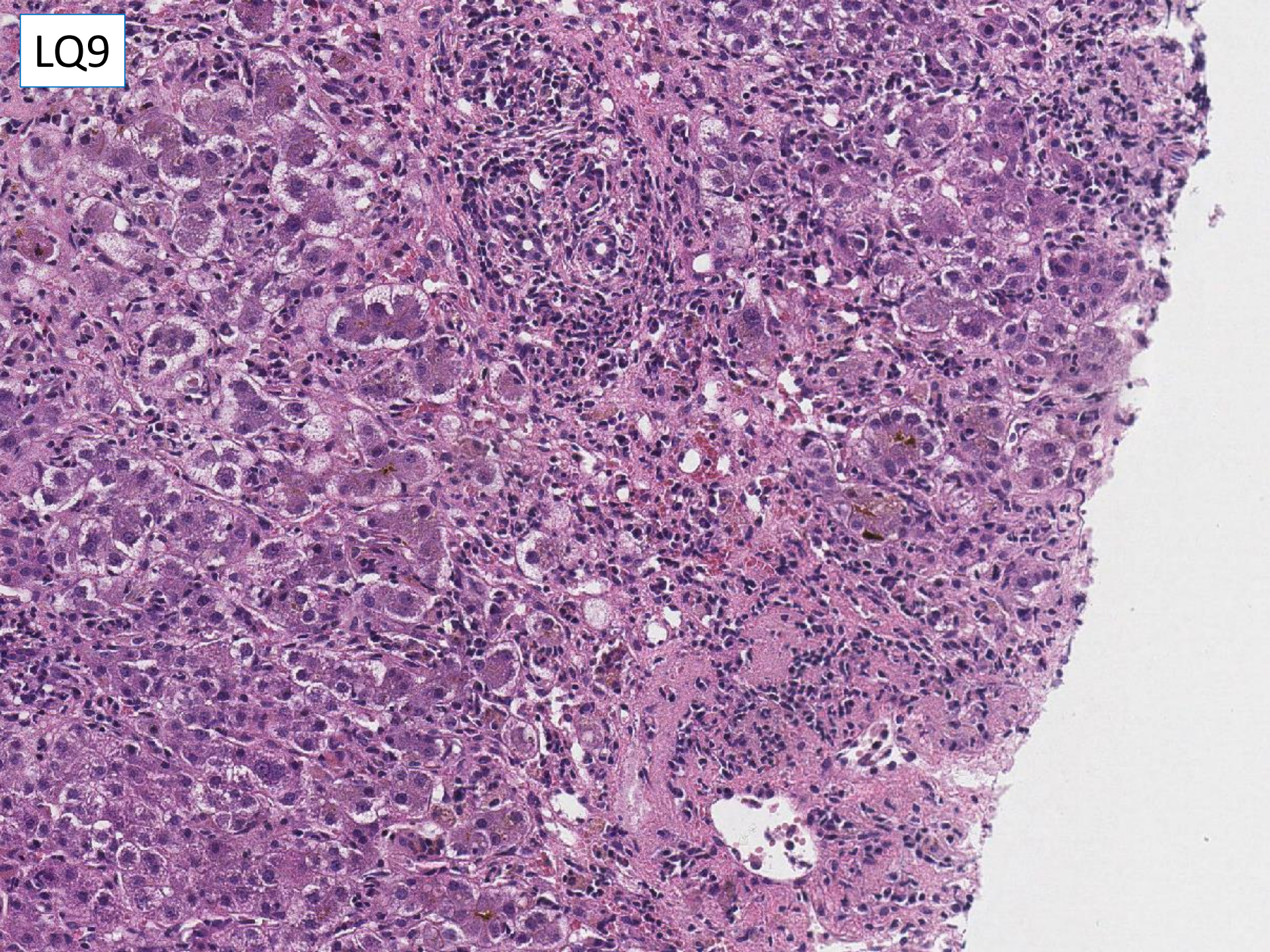
Case LQ9 49F

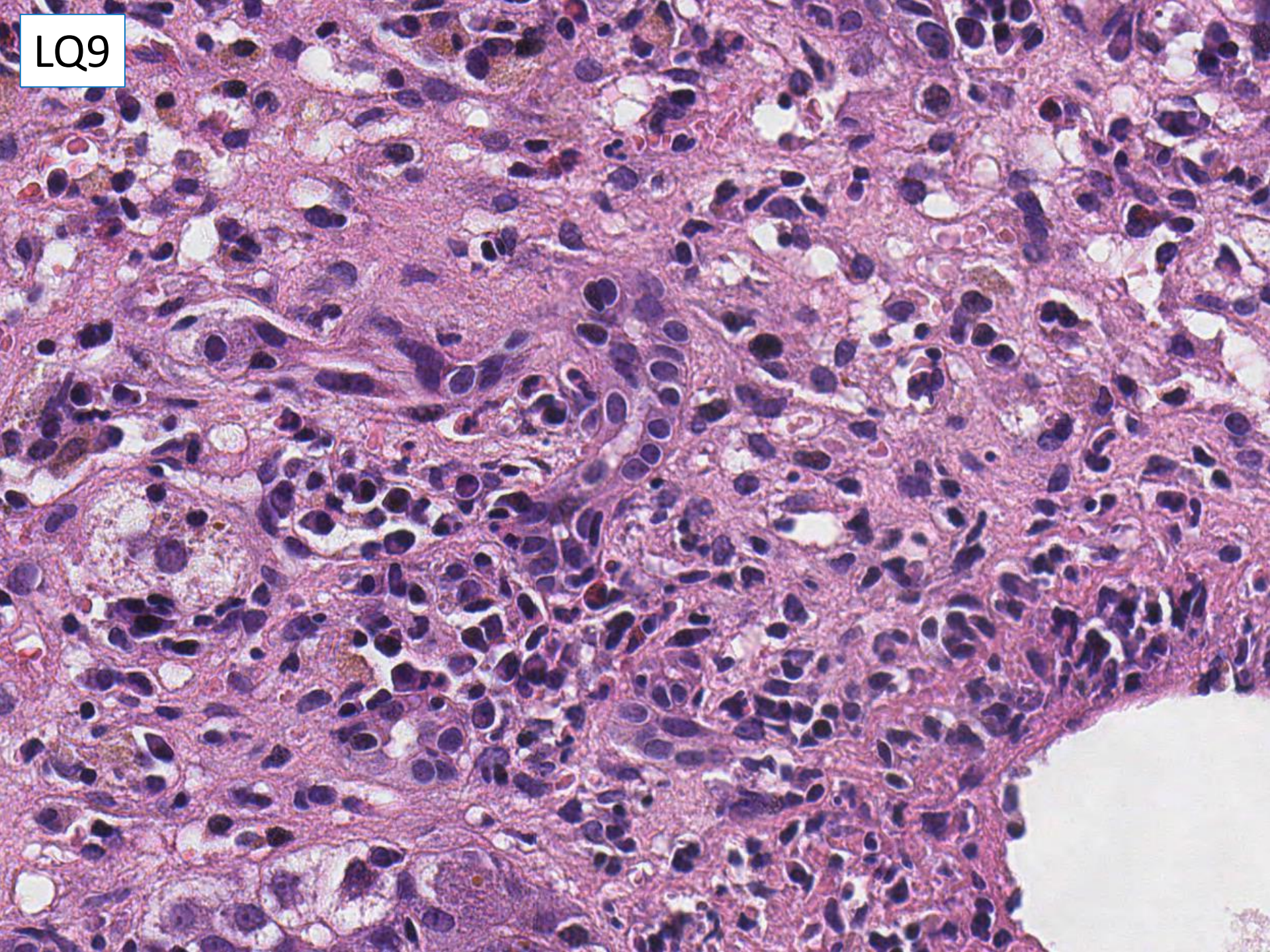
Acute Hepatitis. Liver biopsy. Additional information from urgent Liver biopsy booking from 'BG hypothyroidism. A/W Deranged LFT's. Acute hepatitis, Coagulopathy, high IgG hepatitis negative, ? Autoimmune hepatitis'.

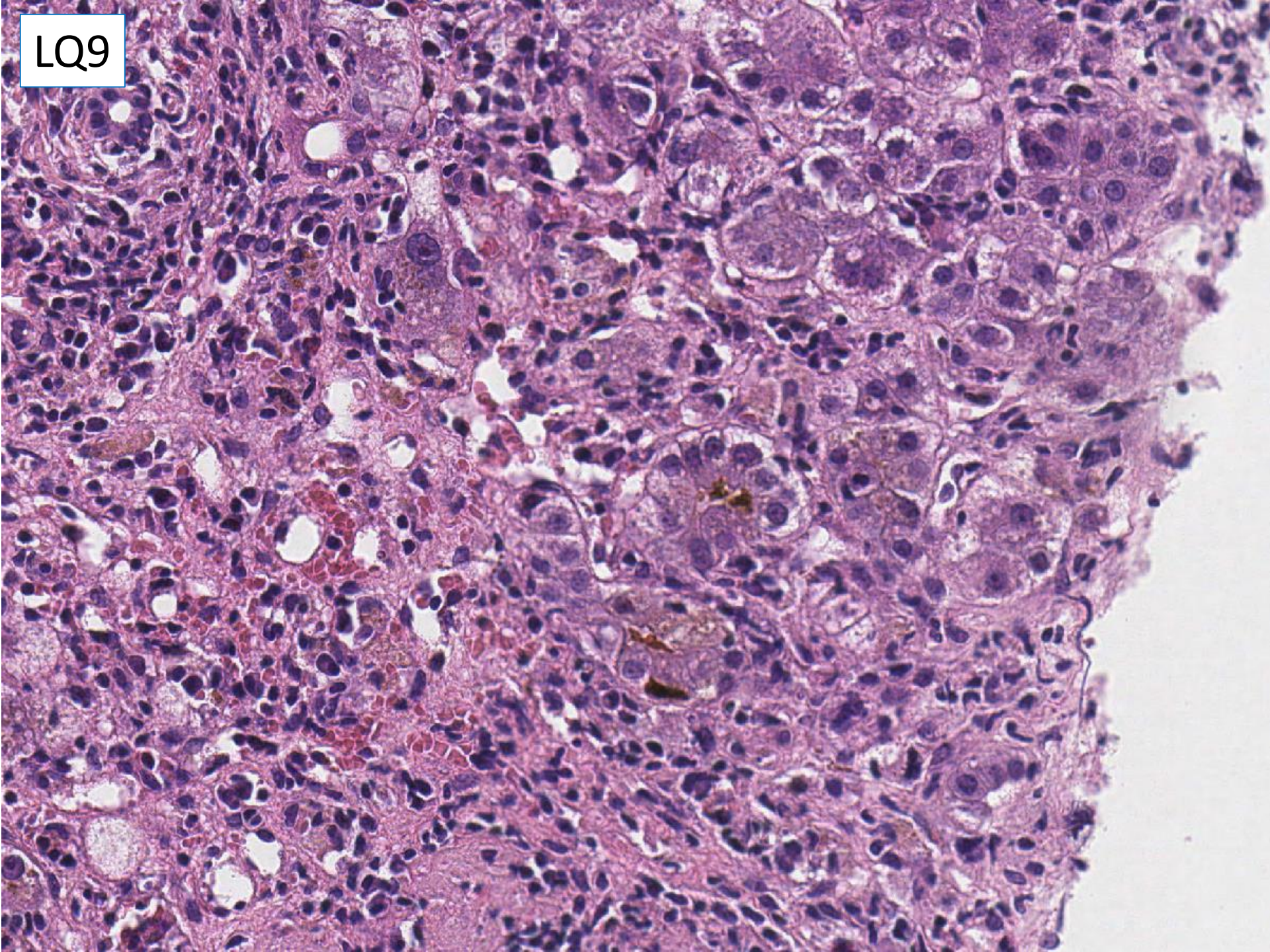


Also Reticulin, Shikata, PASD, van Gieson




LQ9


LQ9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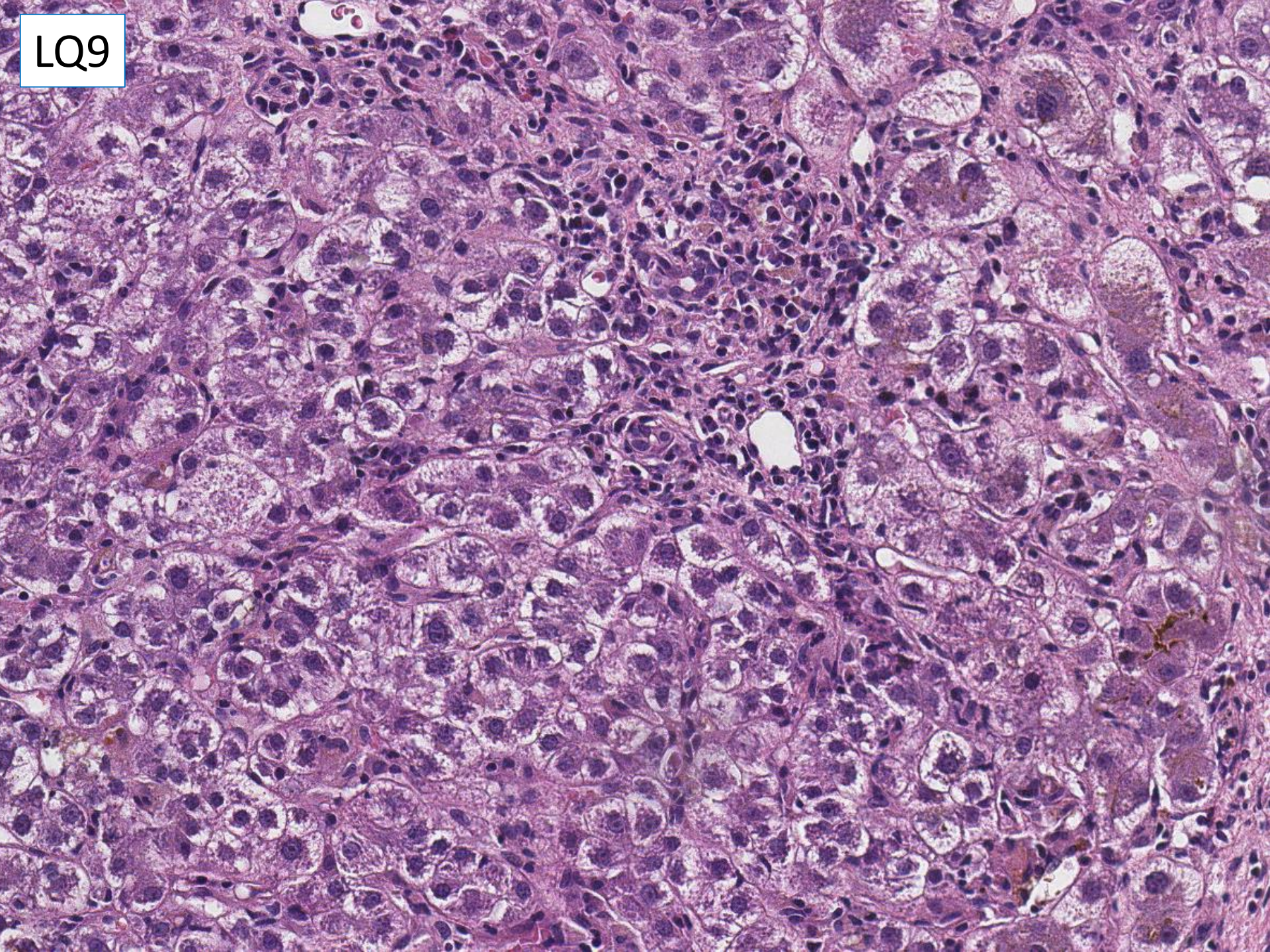
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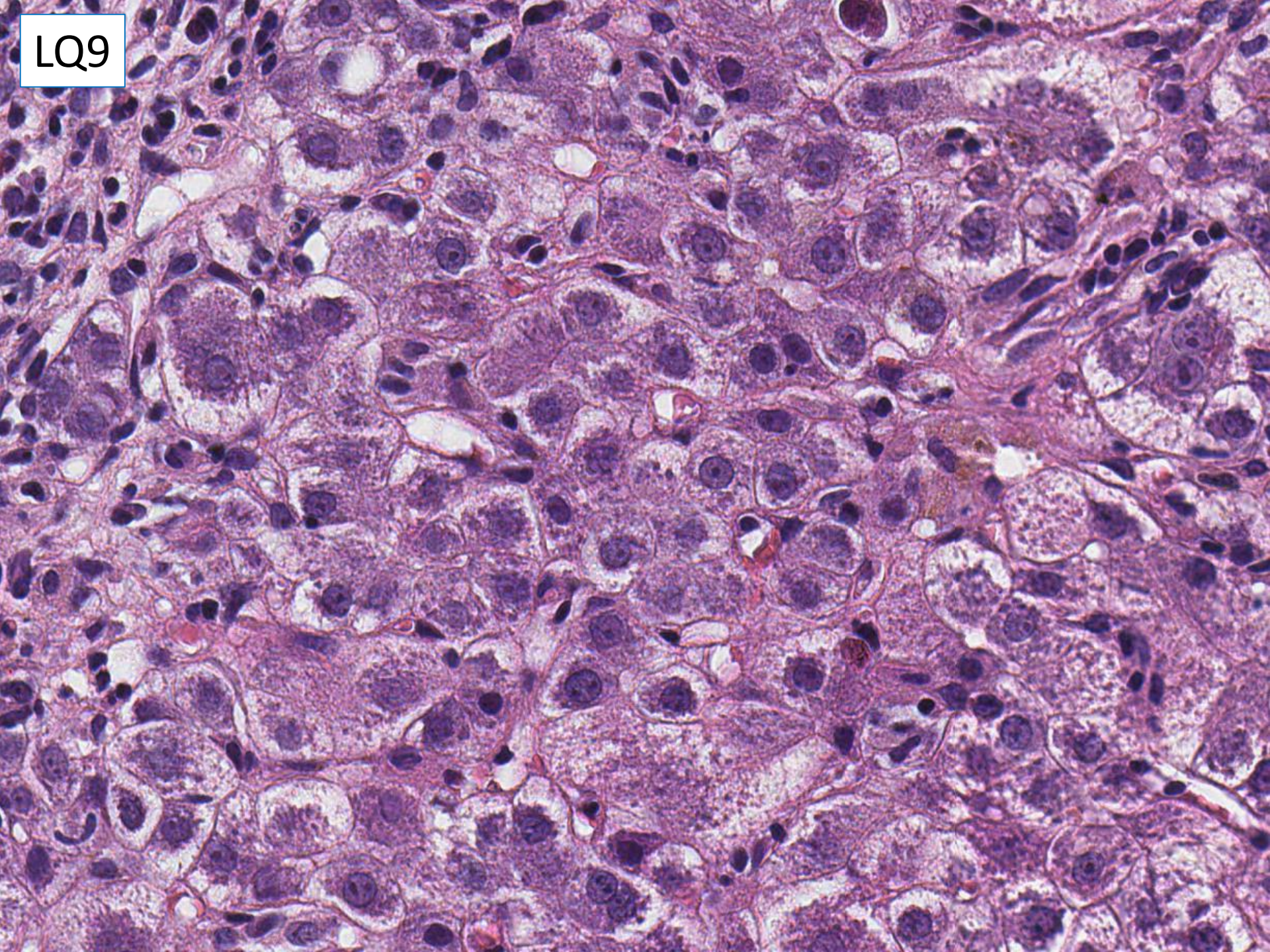
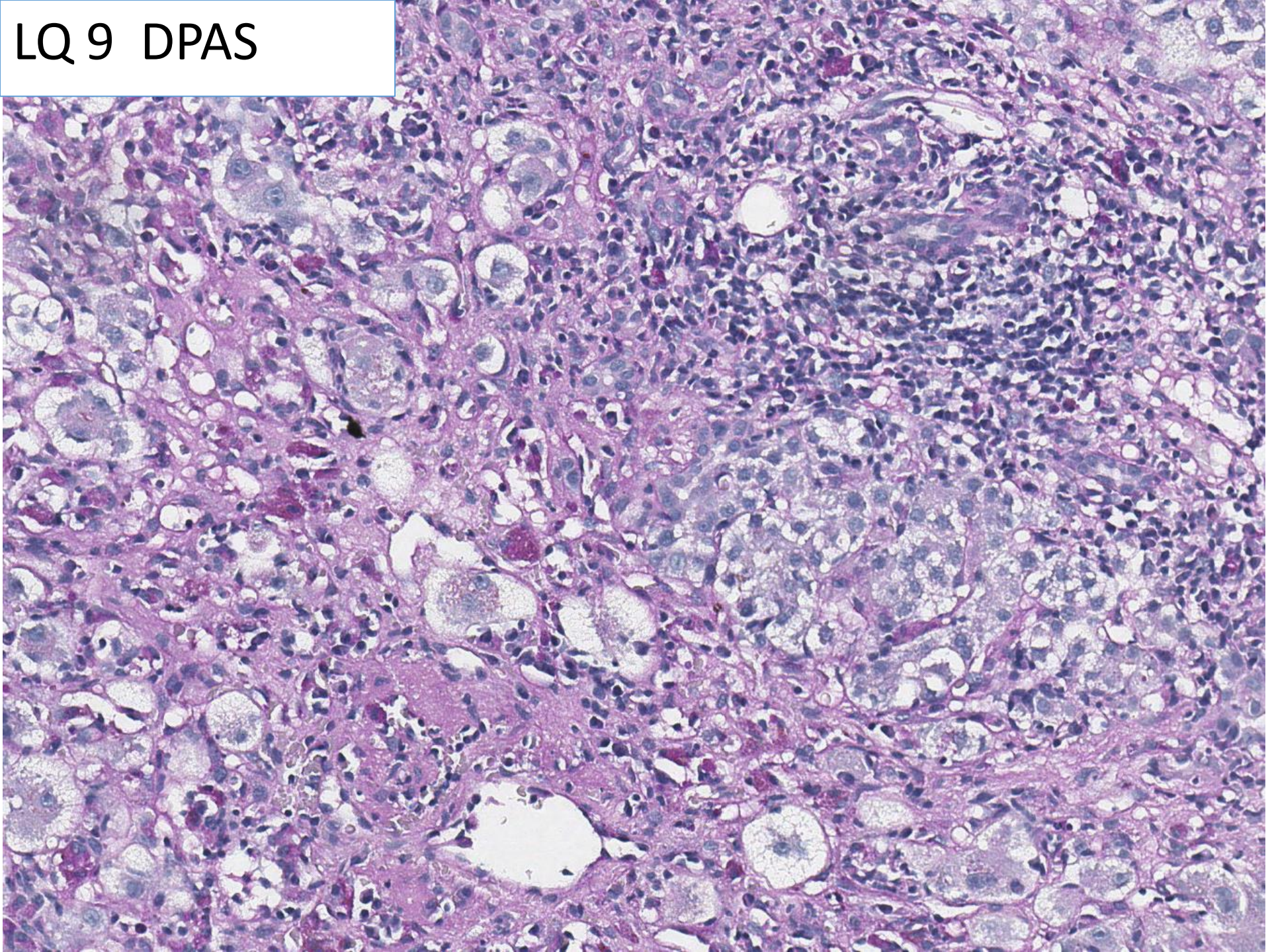
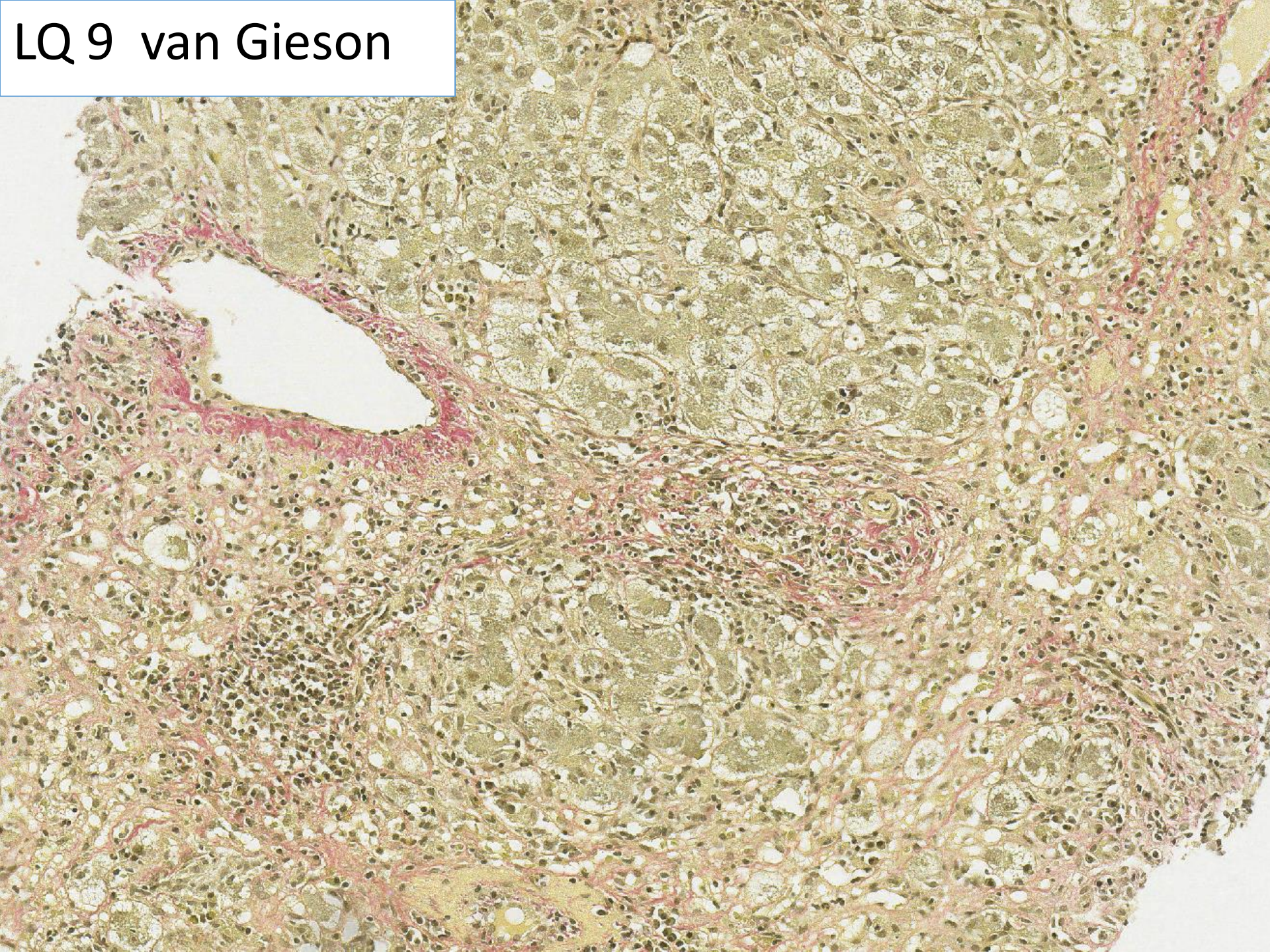

LQ9

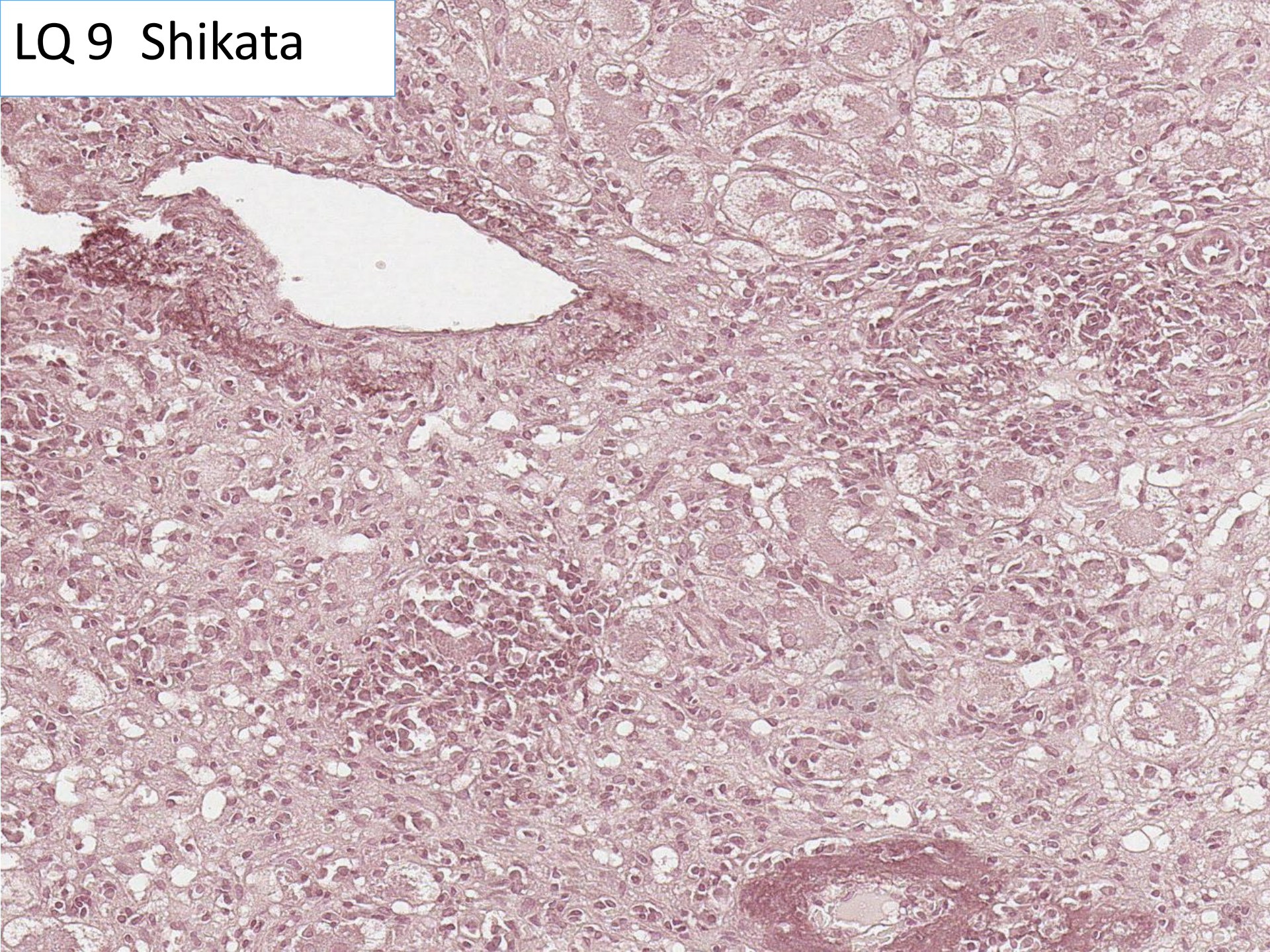
LQ 9 DPAS



LQ 9 van Gieson



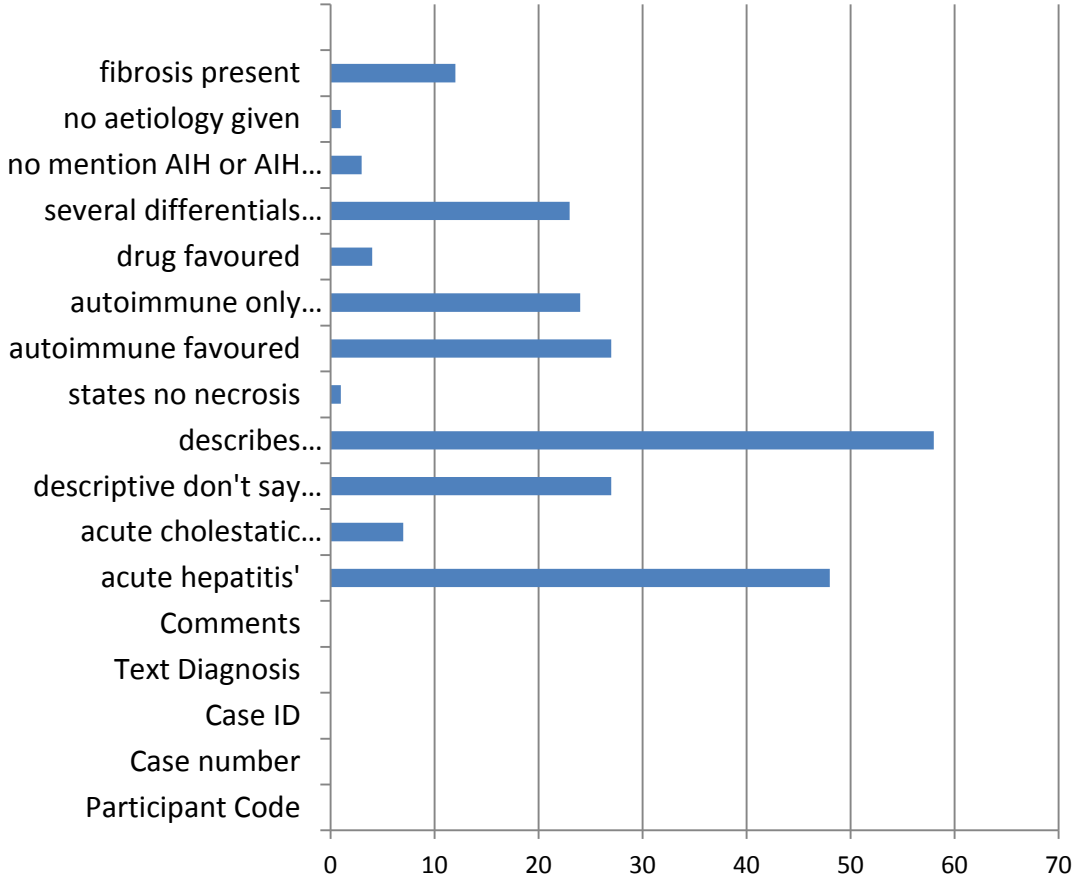
LQ 9 Shikata



Case LQ9 49F

Acute Hepatitis. Liver biopsy.

Additional information from urgent Liver biopsy booking form: hypothyroidism. deranged LFT's. Acute hepatitis, Coagulopathy, high IgG, hepatitis negative, ? Autoimmune hepatitis'.



Concensus diagnosis:

If acute hepatitis combined with ‘acute cholestatic hepatitis’ and descriptions which might have included ‘hepatitis’ NOS or ‘active’ = 82/83.
Autoimmune aetiology mentioned by 76/83

12 included fibrosis, of which
1 cirrhosis
5 bridging fibrosis
6 mild/early

Aetiology – response includes autoimmune: 76. no aetiology mentioned 1: drugs +/- viral: 4

Case LQ9 49F

Acute Hepatitis. Liver biopsy.

Additional information from urgent Liver biopsy booking form: hypothyroidism. deranged LFT's. Acute hepatitis, Coagulopathy, high IgG, hepatitis negative, ? Autoimmune hepatitis'.

Agreed scoring:

For full marks – a diagnosis of acute hepatitis, ‘acute cholestatic hepatitis’ or descriptions including ‘hepatitis’ NOS or ‘active’ and autoimmune hepatitis included in the aetiology.

Lose 5 points for bridging fibrosis and no points for cirrhosis. The connective tissue stains illustrated absence of mature collagen in the areas of collapse, indicating this was confluent necrosis and not established fibrosis.

Lose 5 points if no mention of autoimmune hepatitis.

Case LQ10 53F

Increased fibroscan (11) ?NAFLD ?degree of fibrosis.

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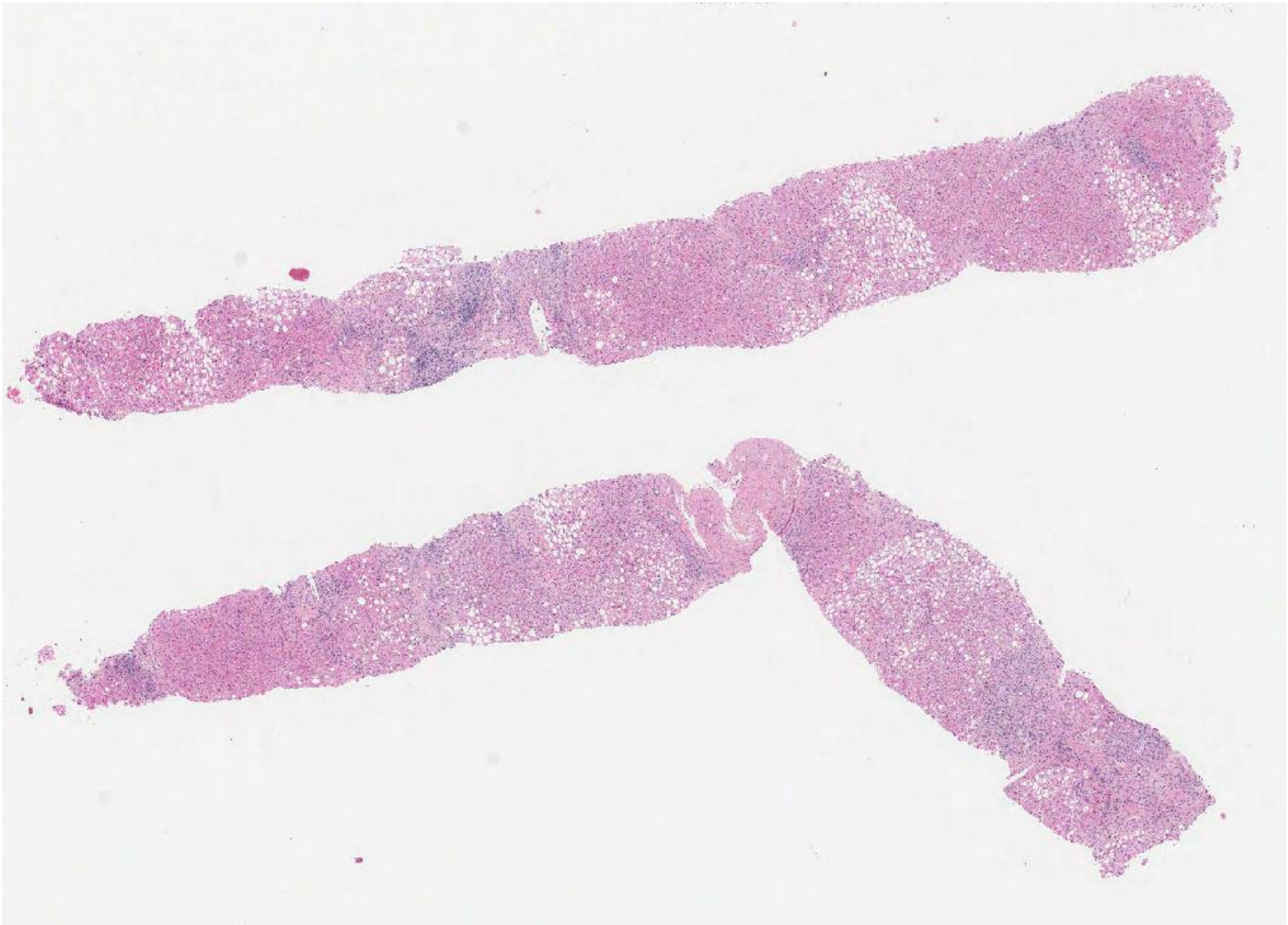
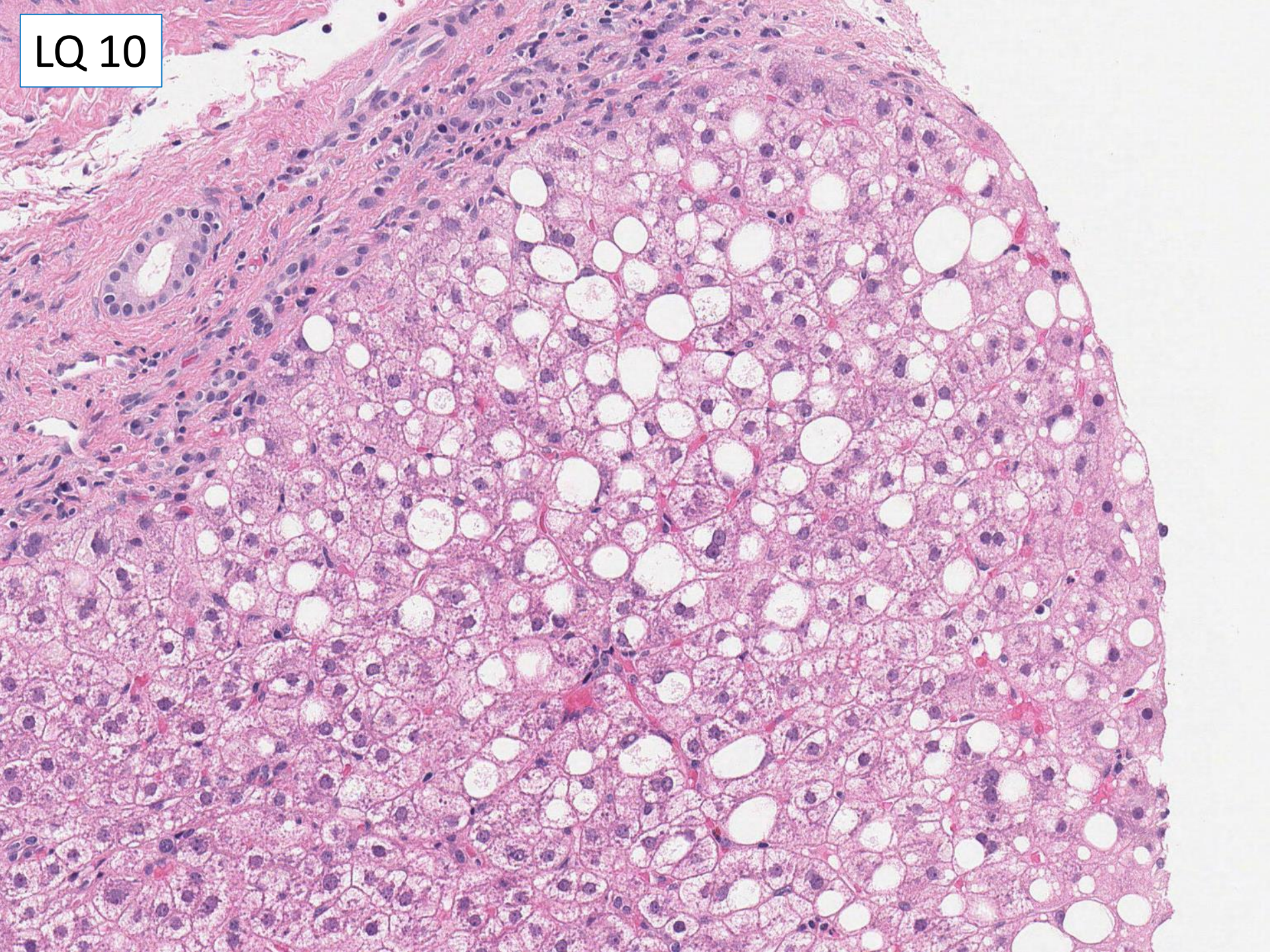
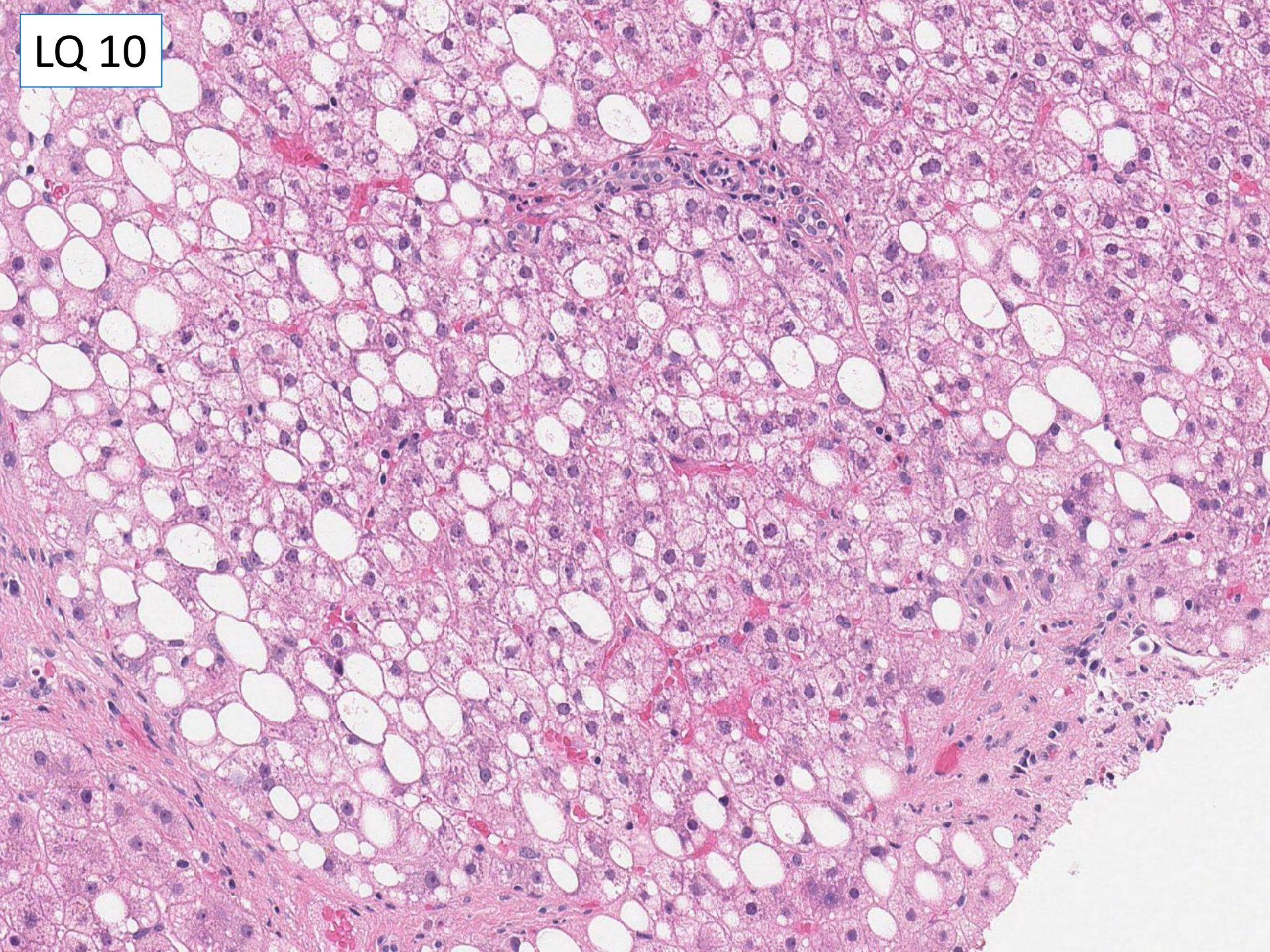
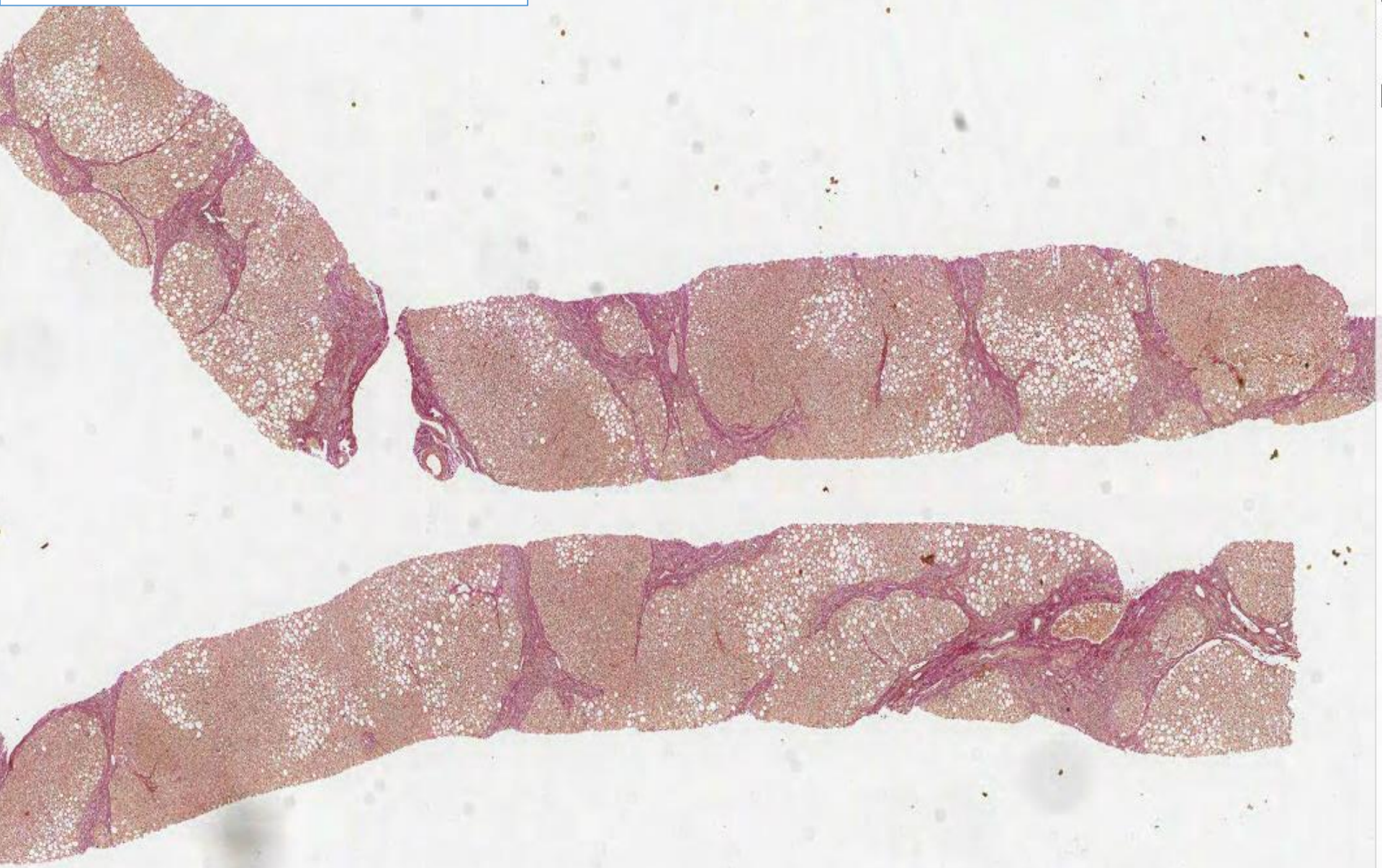


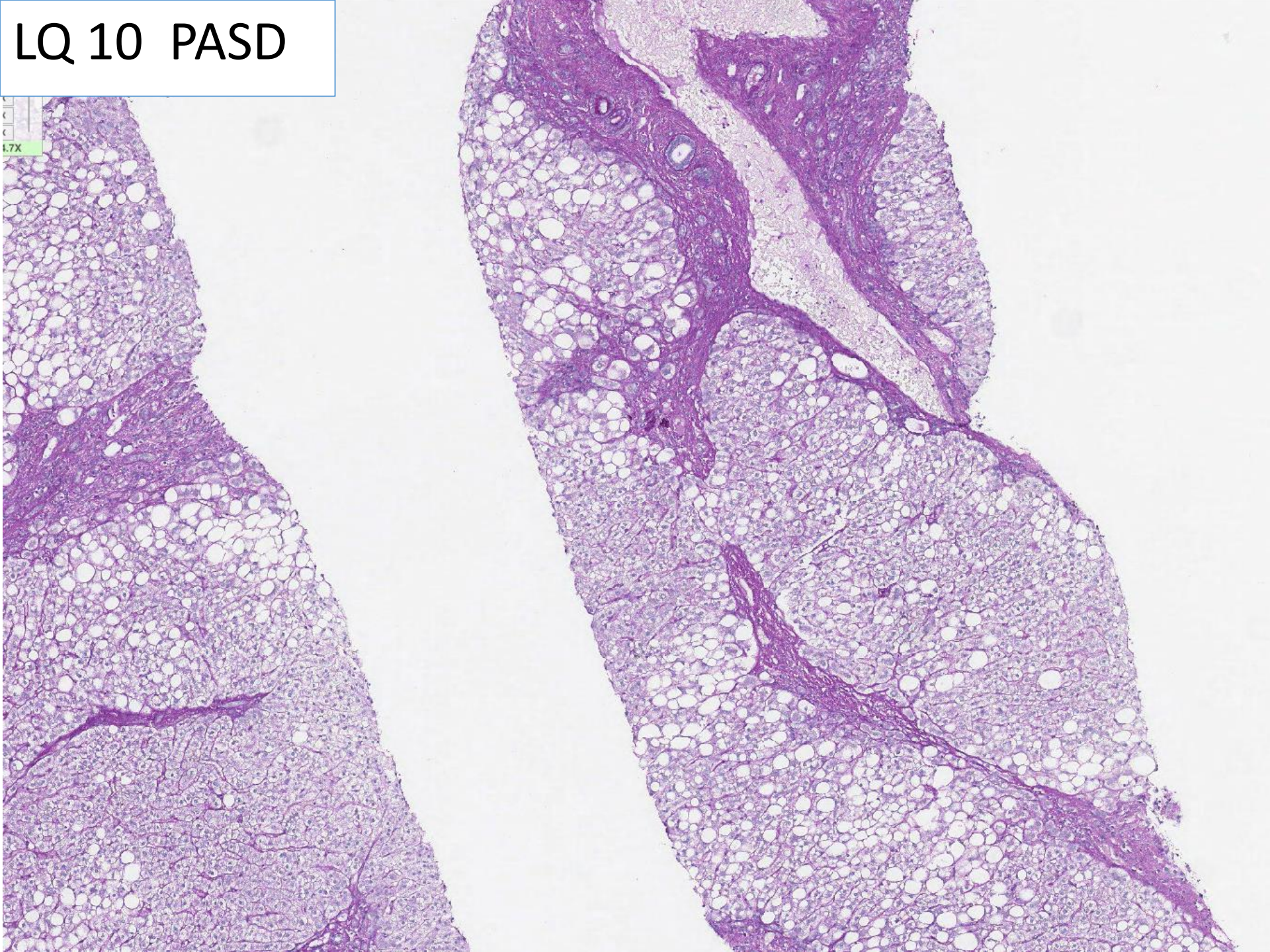

LQ 10


LQ 10

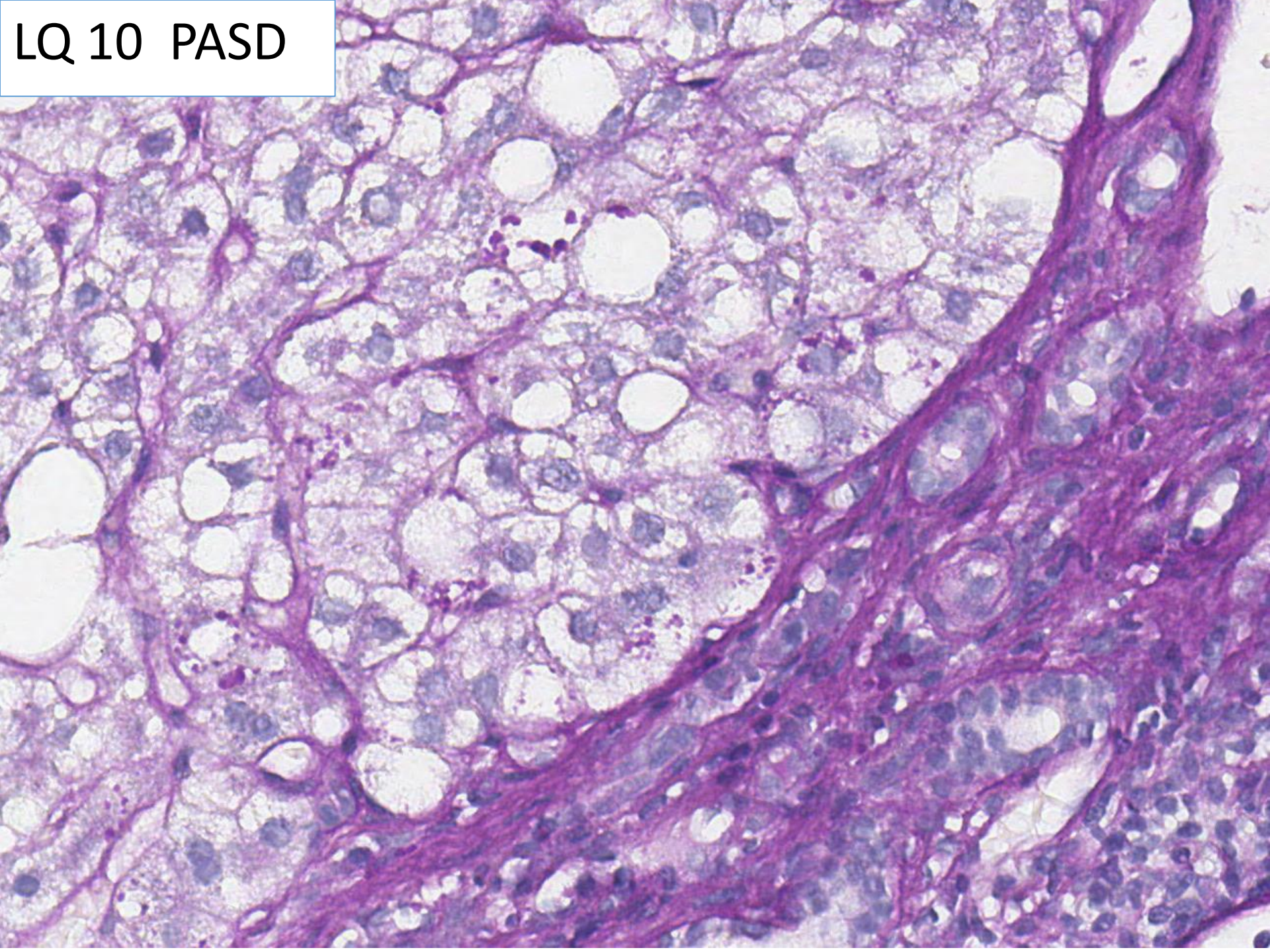
LQ 10 van Gieson



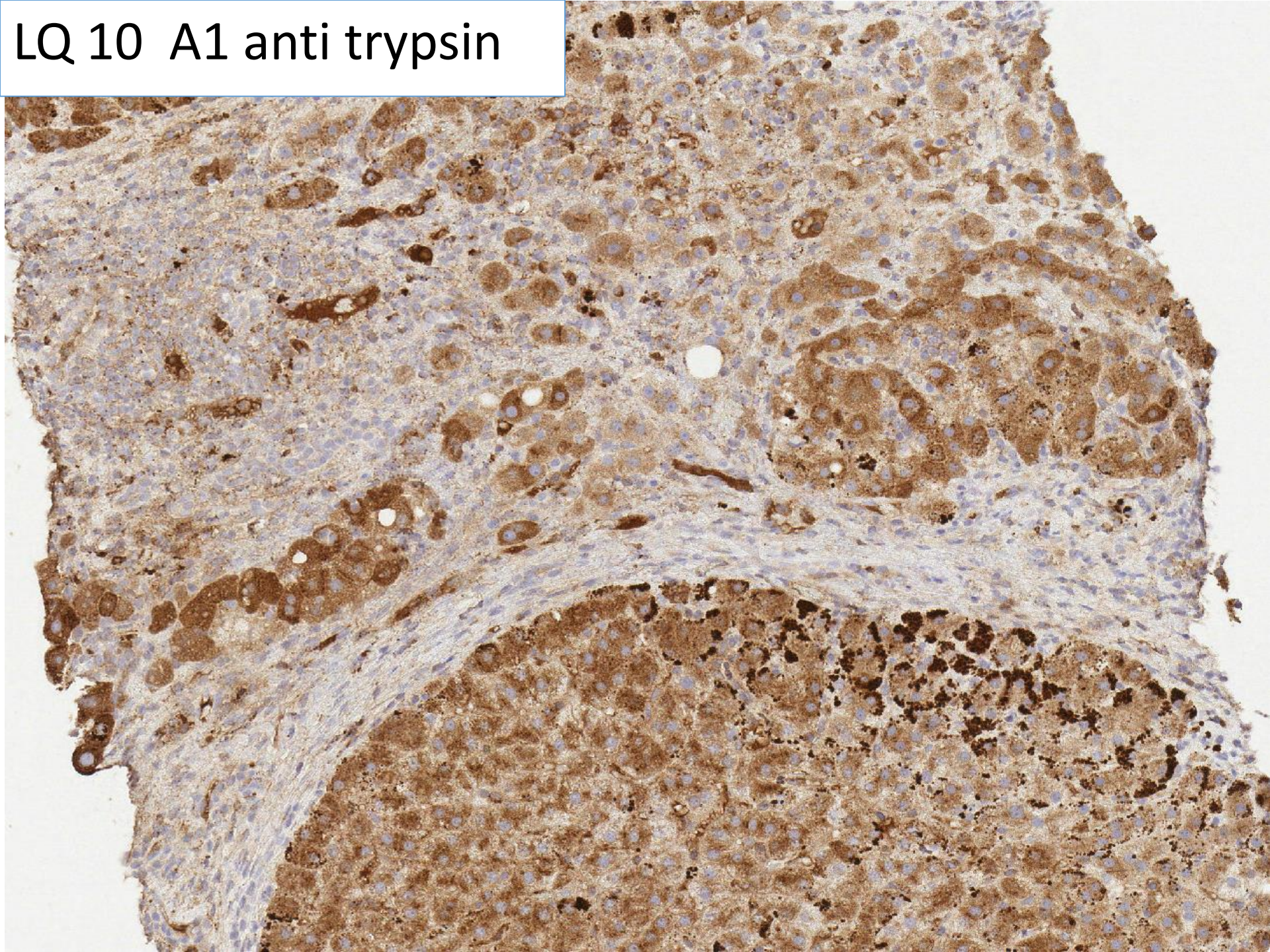
LQ 10 PASD



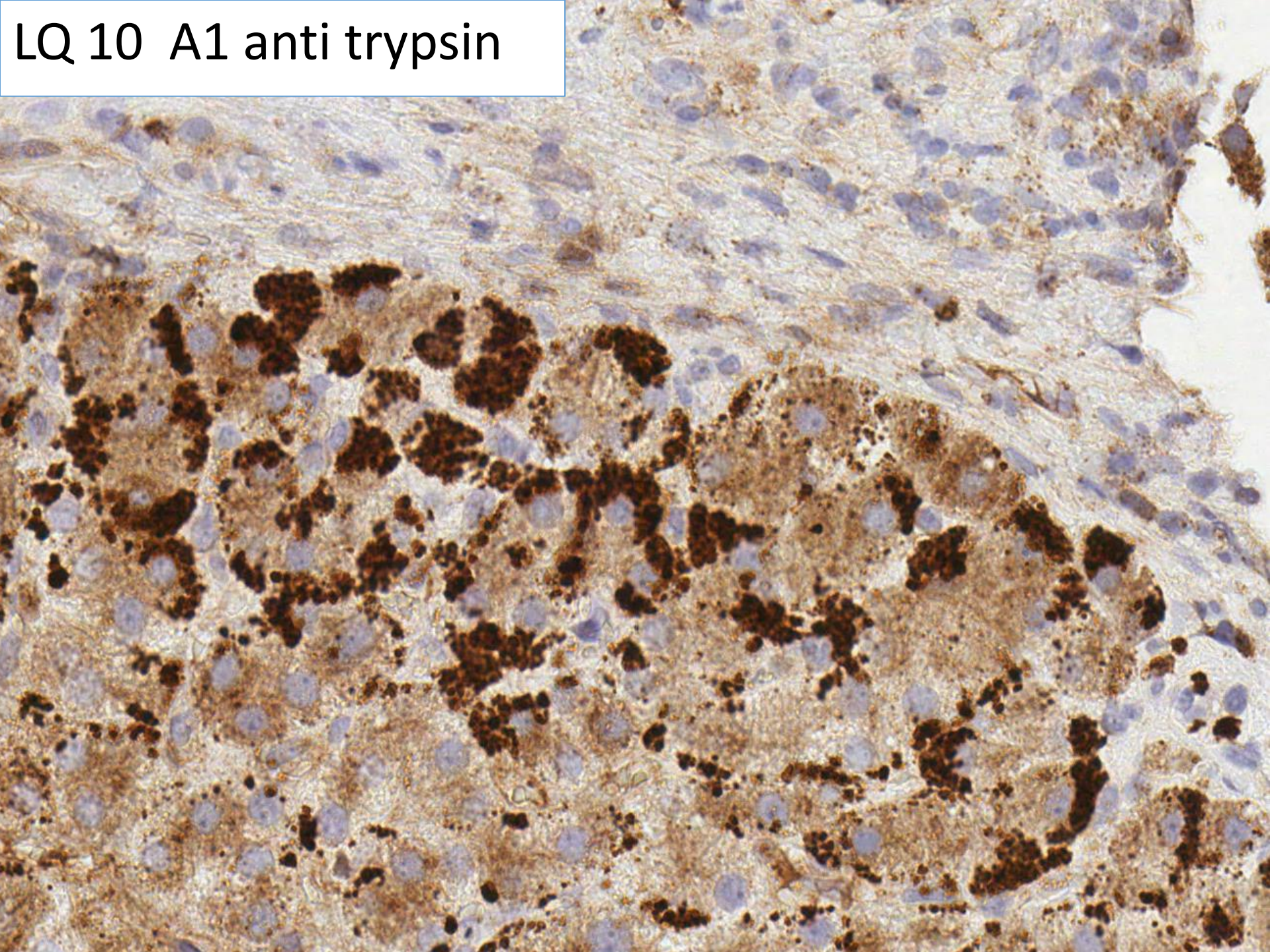
LQ 10 PASD



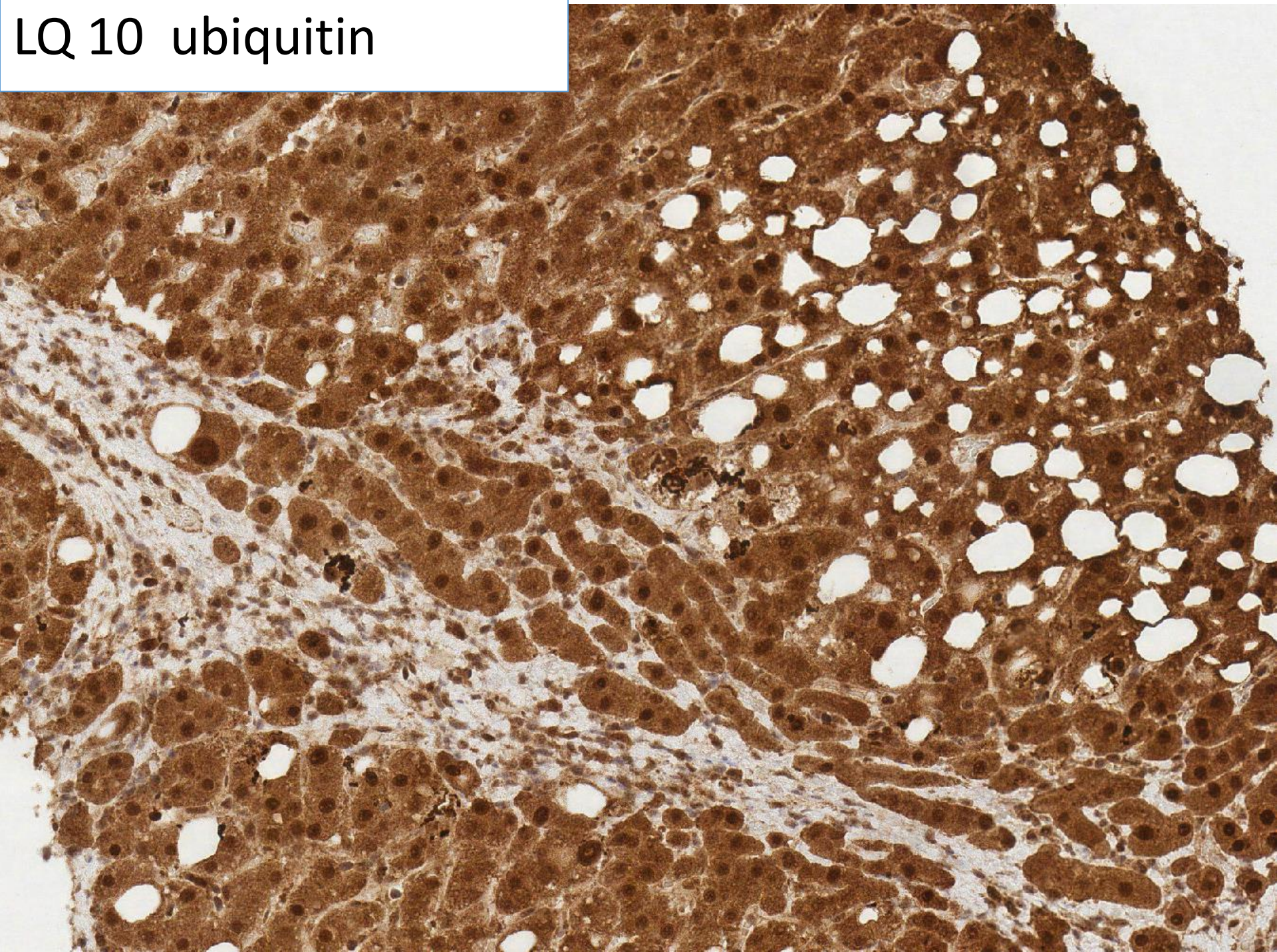
LQ 10 A1 anti trypsin



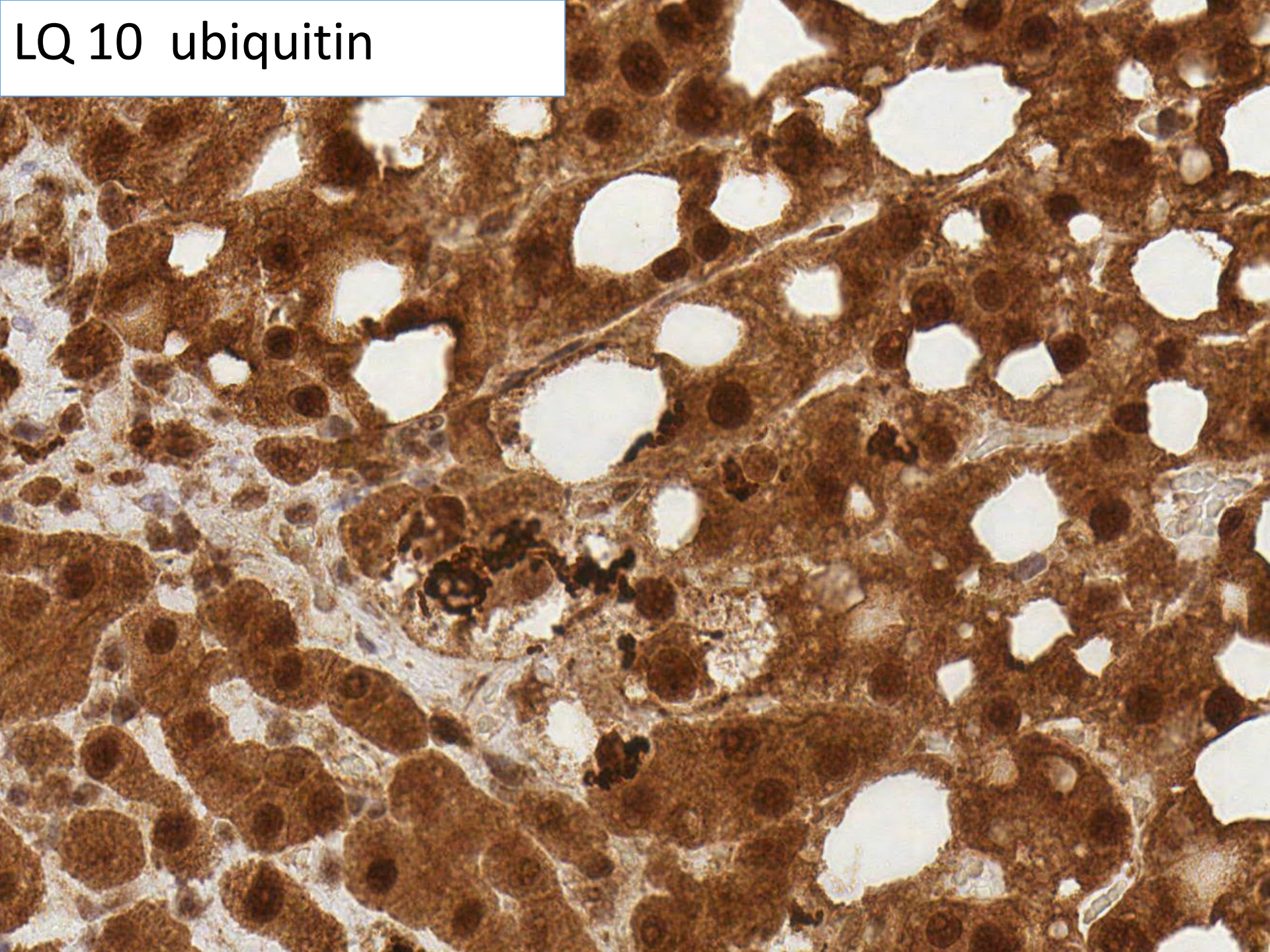
LQ 10 A1 anti trypsin



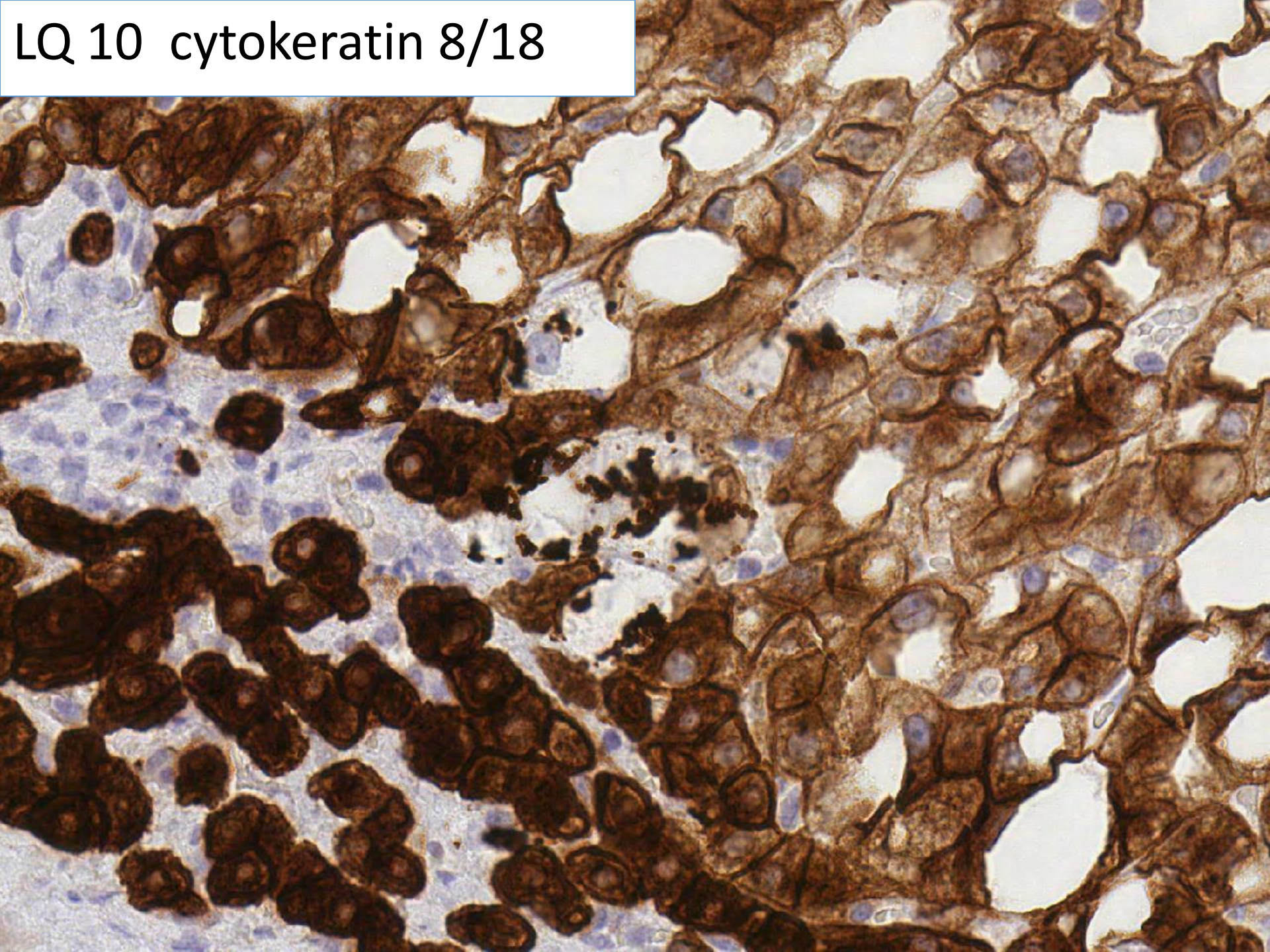
LQ 10 ubiquitin



LQ 10 ubiquitin



LQ 10 cytokeratin 8/18



Case LQ10 53F

Increased fibroscan (11) ?NAFLD ?degree of fibrosis.

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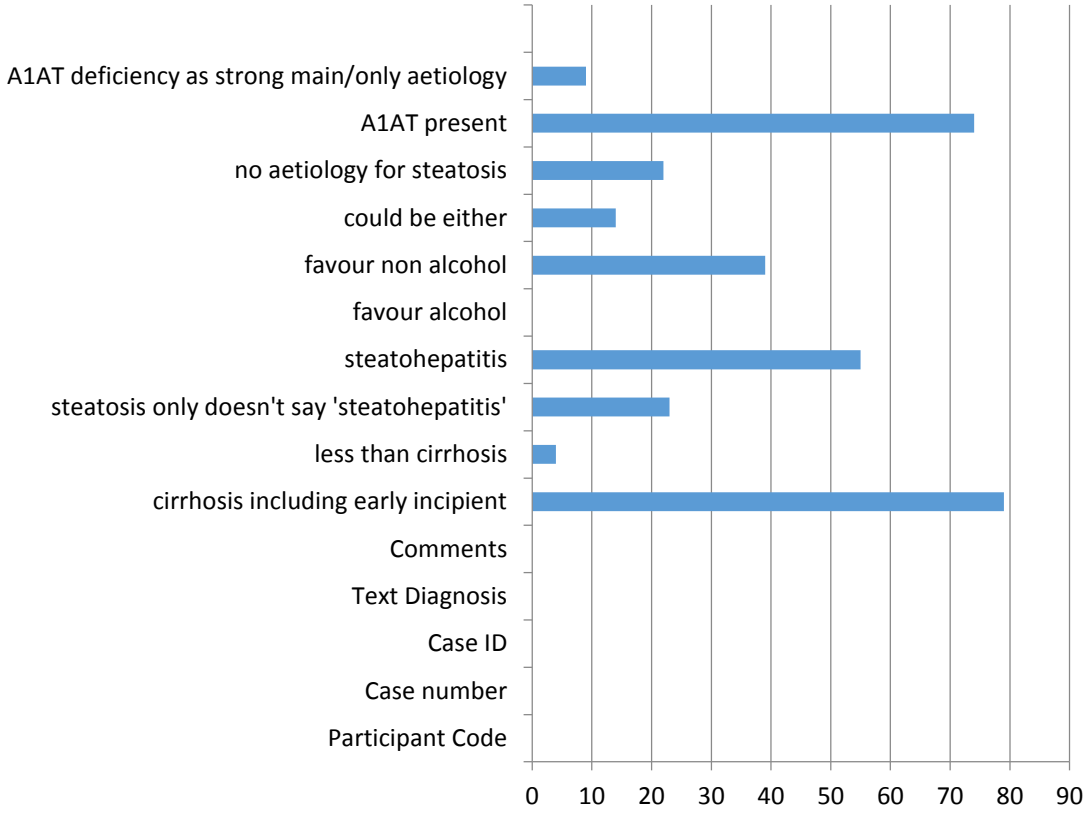

Consensus diagnosis: cirrhosis and the presence of A1AT globules

– variable interpretations offered as to relevance, most had fatty liver disease (majority steatohepatitis) as main diagnosis with possibility of additional A1AT disease and recommended further Ix.

Suggested scoring: half marks if: Less than cirrhosis = 4 of which:
Ishak 5/6, Advanced bridging fibrosis and nodularity, Bridging fibrosis x2
No mention A1AT at all = 9; A1AT deficiency given as main diagnosis = 9

Case LQ10 53F

Increased fibroscan (11) ?NAFLD ?degree of fibrosis.

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Agreed scoring:

For full marks - need all three components of cirrhosis or late stage fibrosis/probable cirrhosis, and a consideration of both steatohepatitis and alpha 1 antitrypsin deficiency as potential contributory factors in the aetiology.

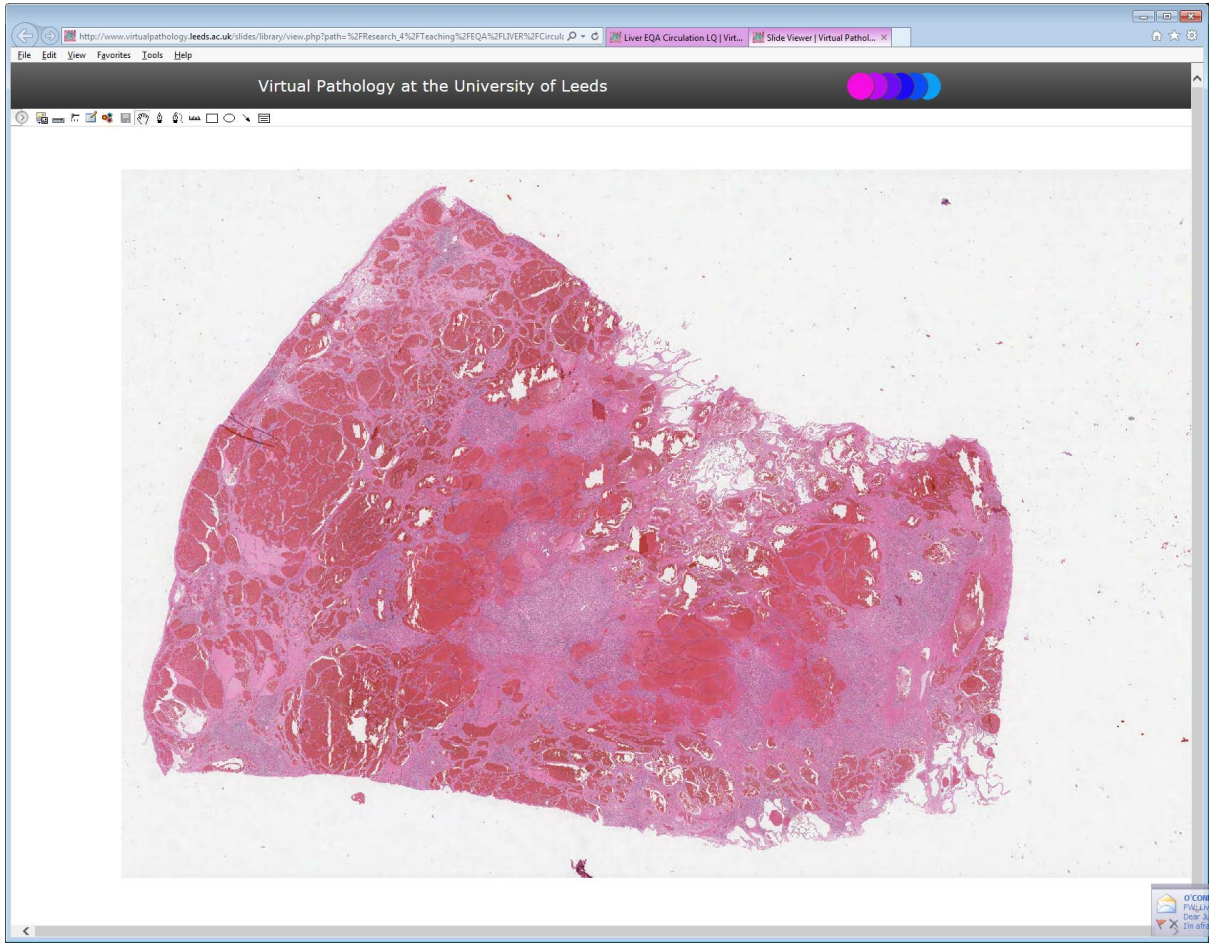
Lose 5 points if fibrosis stage of bridging, but does not suggest the presence of cirrhosis or late stage disease.

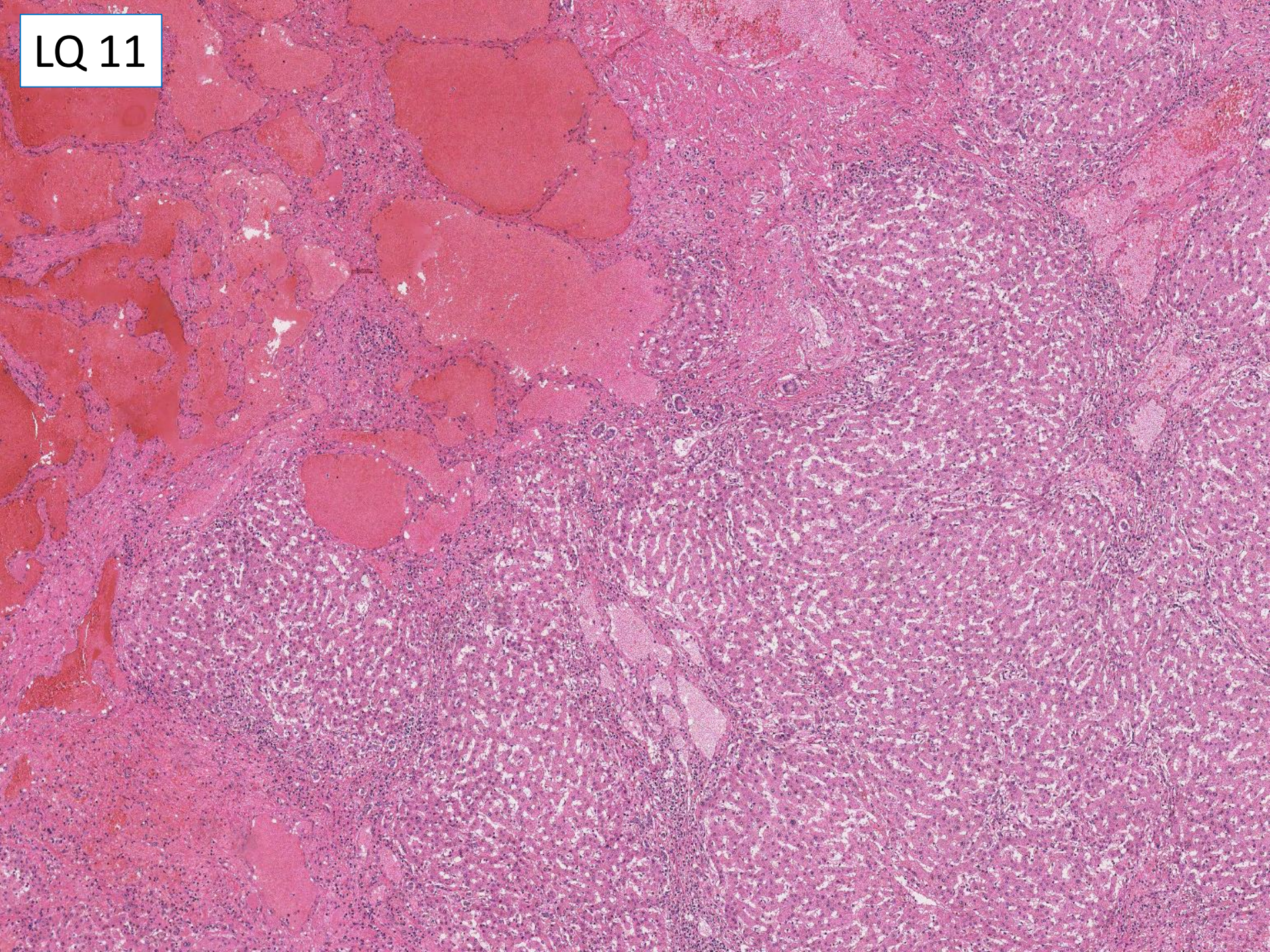
Lose 5 points if no mention of alpha 1 antitrypsin deficiency, or gives this as main diagnosis, with no mention of steatohepatitis/NASH as an aetiological factor, or description of Mallory bodies without indicating that this implies a diagnosis of steatohepatitis.

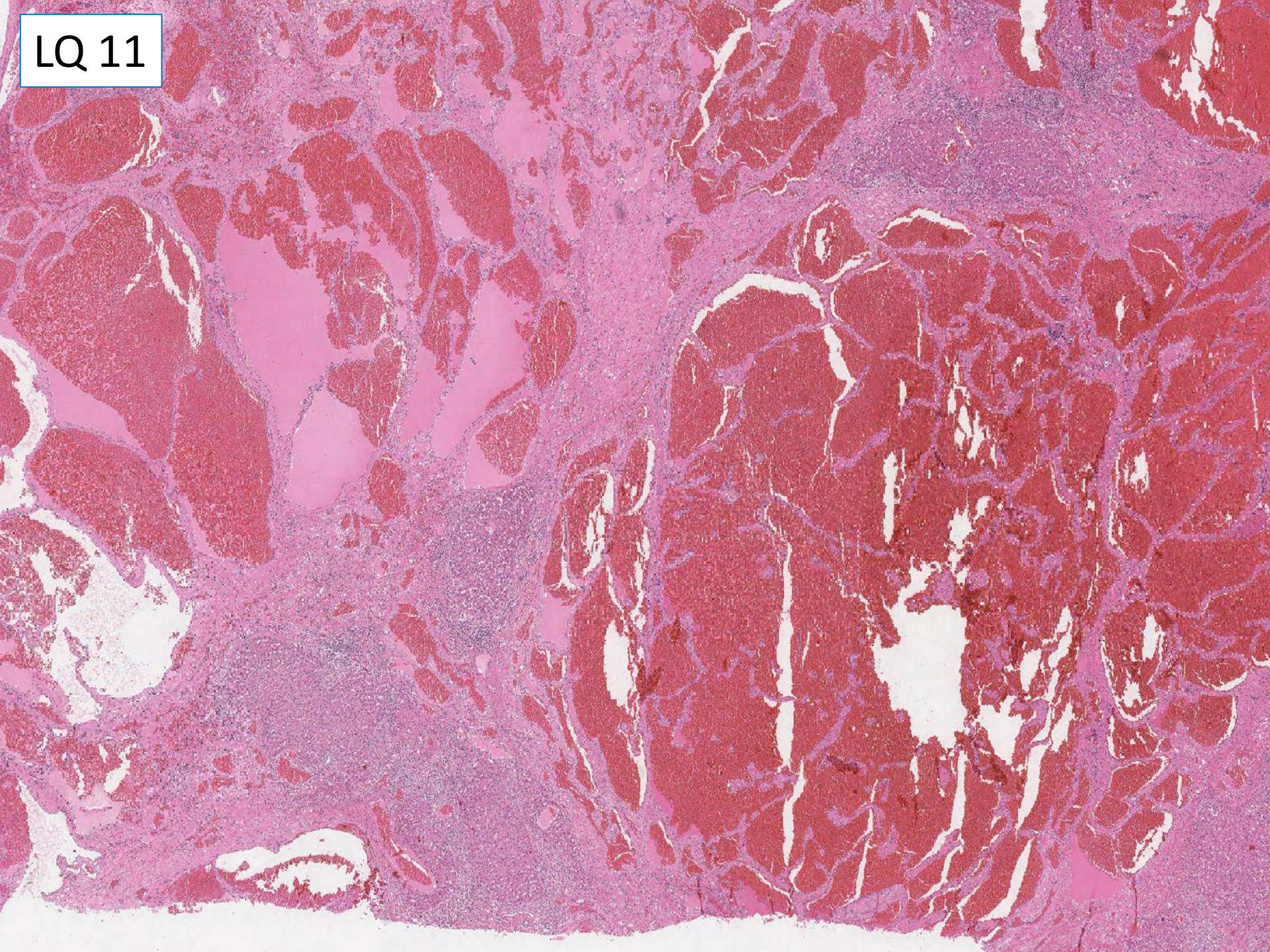
There was not a consensus on the most likely cause. Do we know A1AT phenotype?

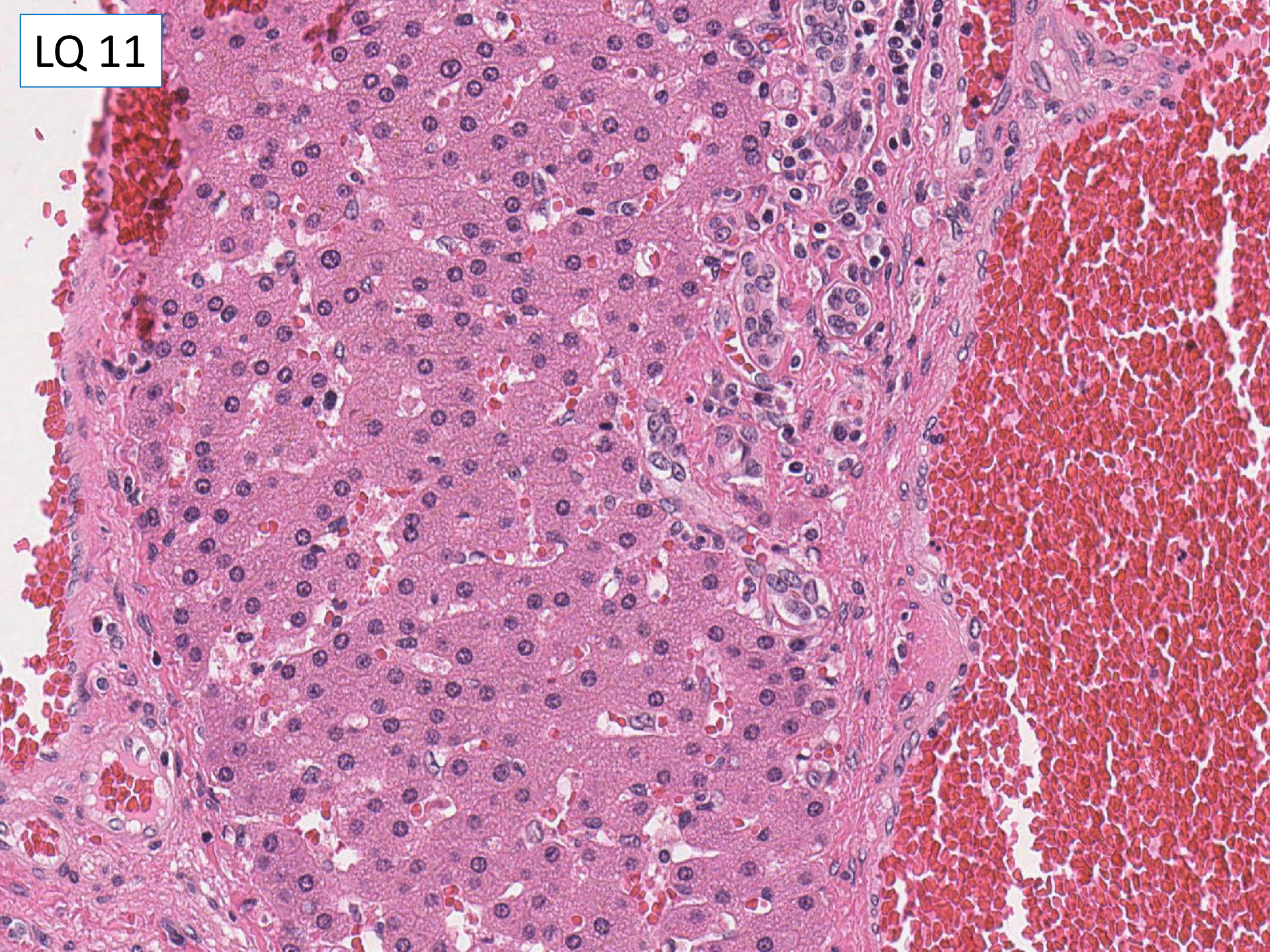
Case LQ11 59M

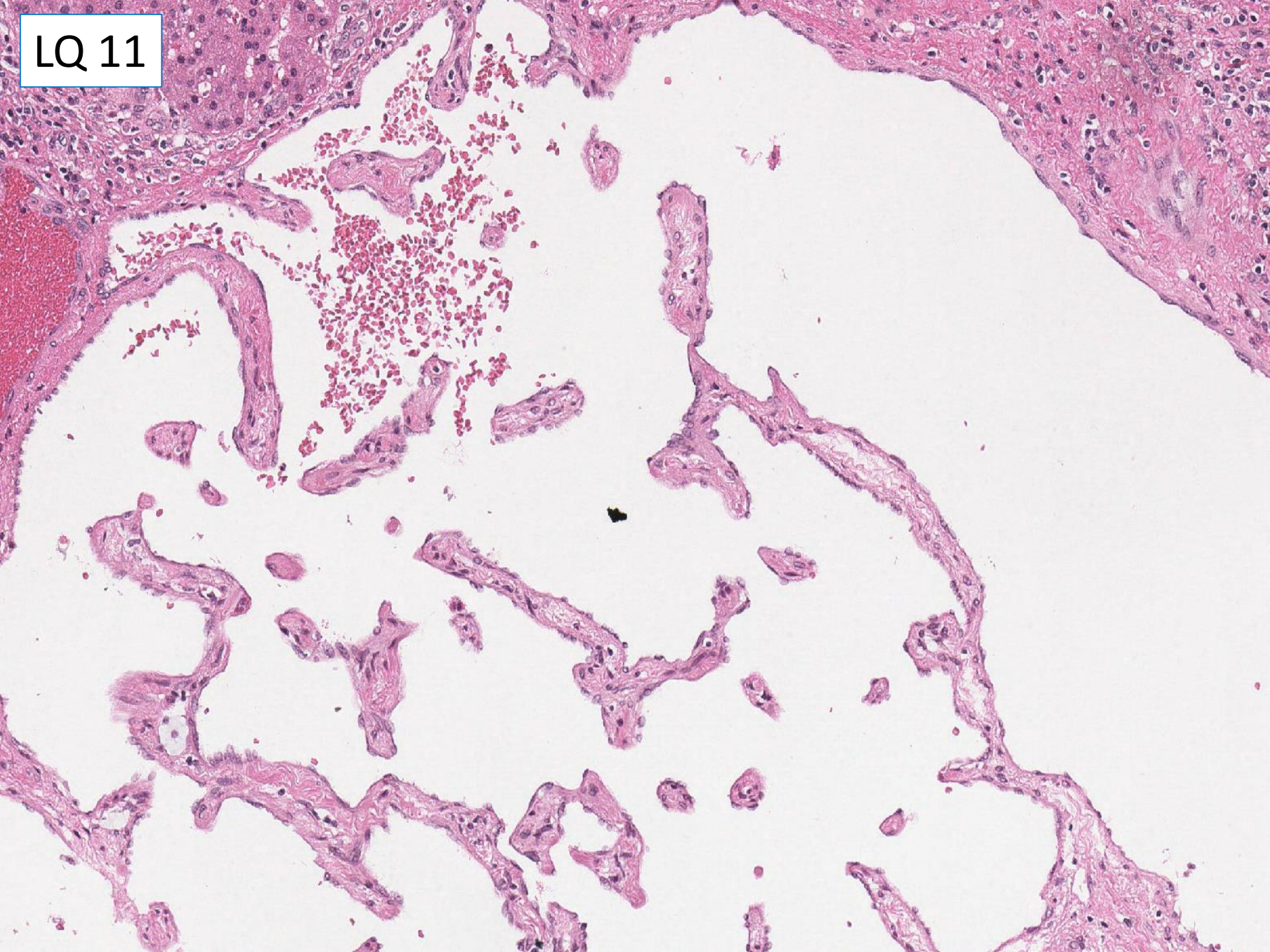
Intra-abdominal mass involving right lobe of liver




LQ 11


LQ 11


LQ 11


LQ 11

Case LQ11 59M

Intra-abdominal mass involving right lobe of liver

Cavernous haemangioma	53
Haemangioma	27
Cavernous haemangioma with atypia	1
haemangioma with atypia	1
Haemangioma, rule out malignancy: intravascular papillary hyperplasia	1

Consensus: Cavernous haemangioma or haemangioma.

Suggested scoring: For full marks, either cavernous haemangioma or haemangioma (cavernous haemangioma in WHO classification).

How to score responses with atypia or rule out malignancy? 0 / 4 / 2

Case LQ11 59M

Intra-abdominal mass involving right lobe of liver

Agreed scoring:

For full marks, either cavernous haemangioma or haemangioma (cavernous haemangioma in WHO classification).

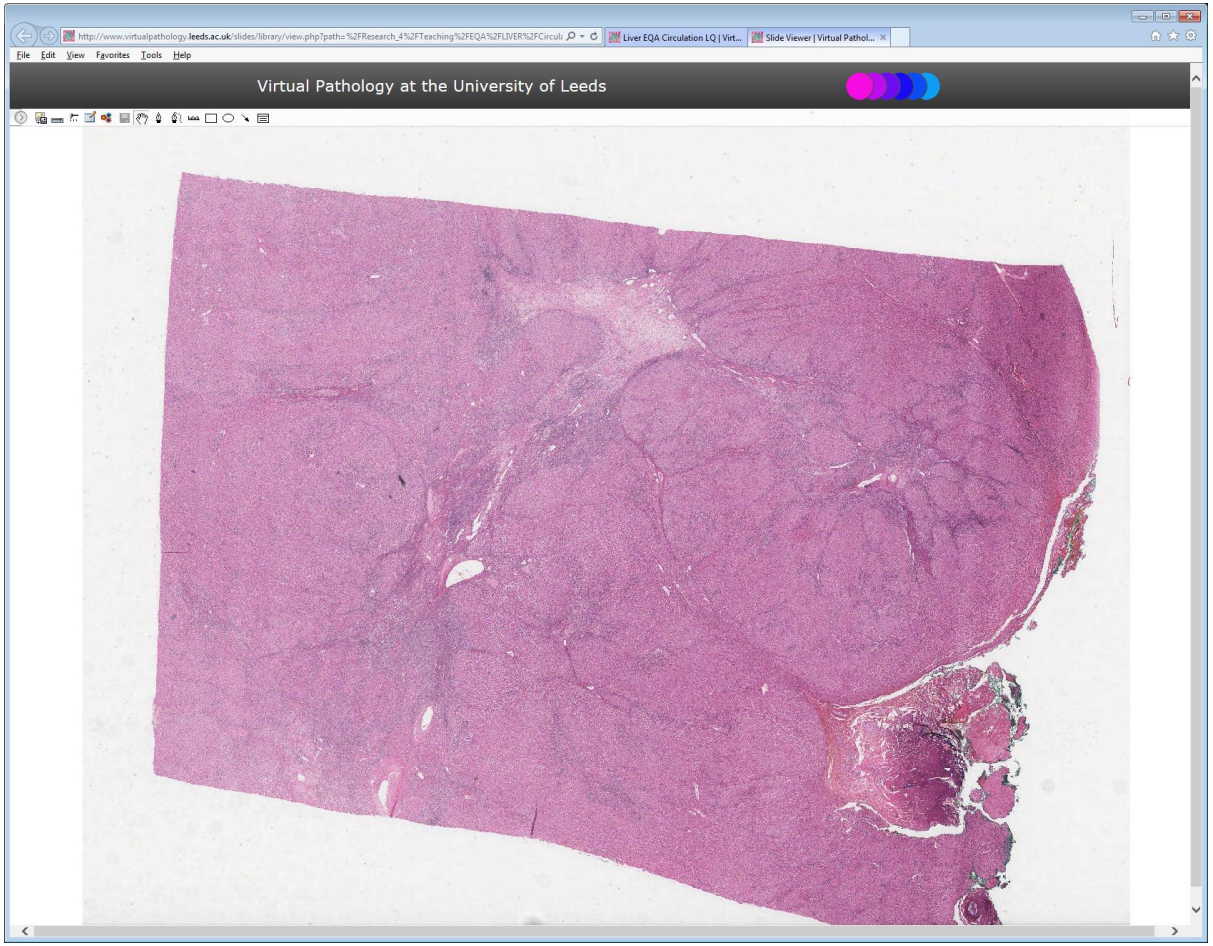
How to score responses with atypia or rule out malignancy? – on show of hands these would score half marks.

On review of responses, all had an overall diagnosis of haemangioma. None had wording which suggested that this may be part of a malignant lesion e.g. ‘haemangioma with some atypical features’ referring to the growth pattern rather than cytology. Also ‘haemangioma with areas of intravascular papillary hyperplasia’ – therefore no marks deducted, all responses scored full marks.

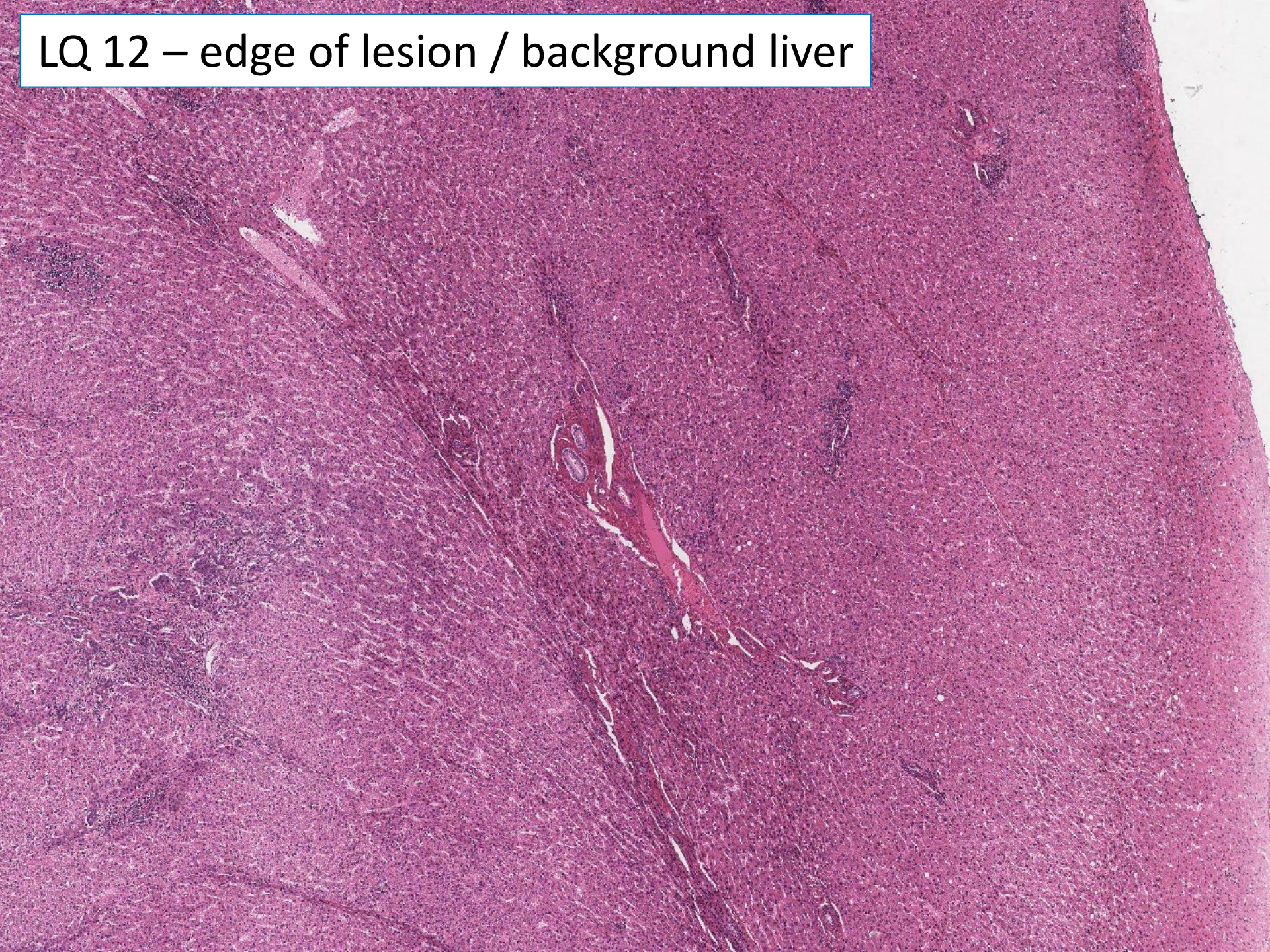
Several commented that there are features of haemangiomatosis, since the lesion spreads into the adjacent liver parenchyma.

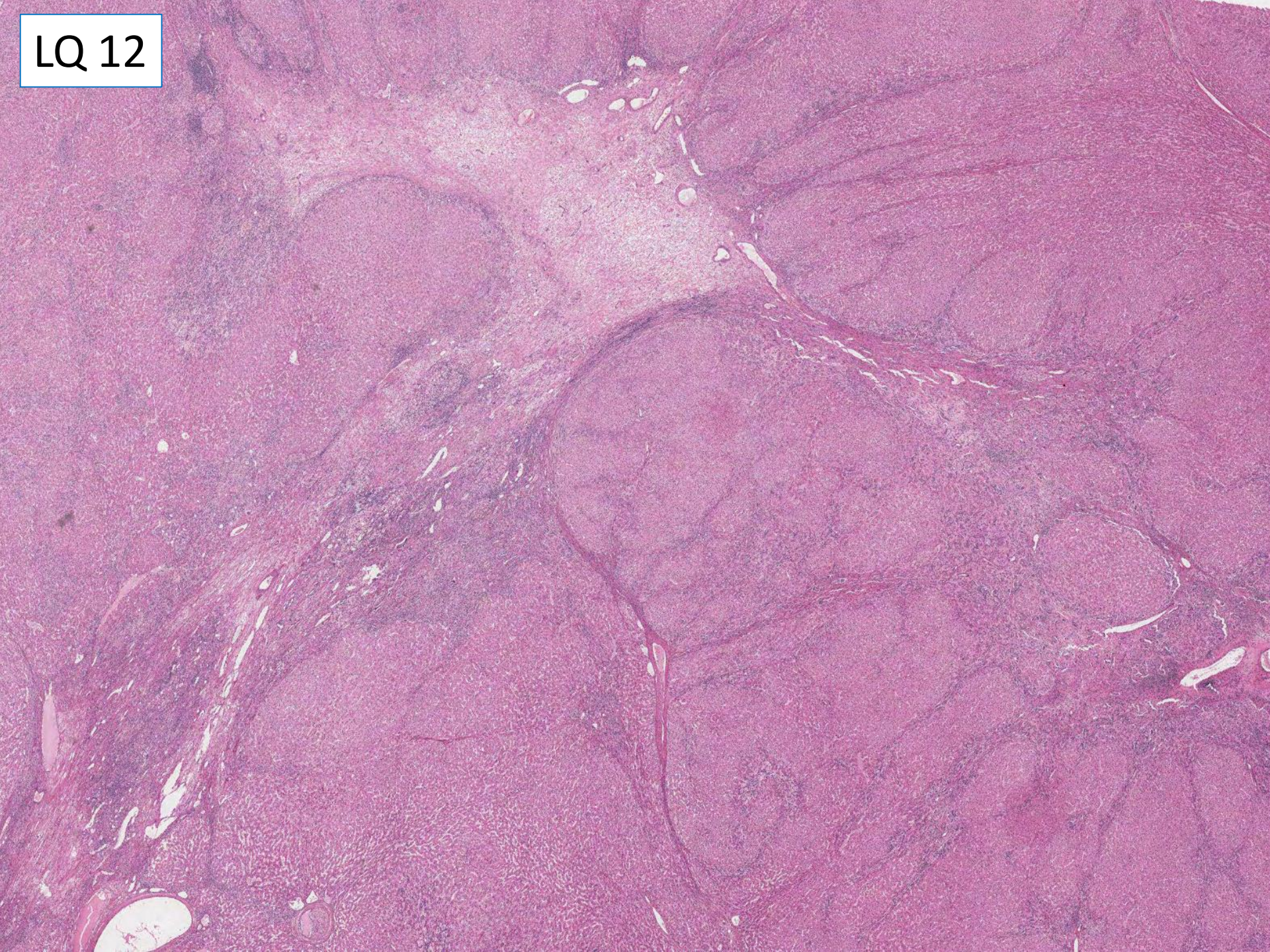
Case LQ12 28M

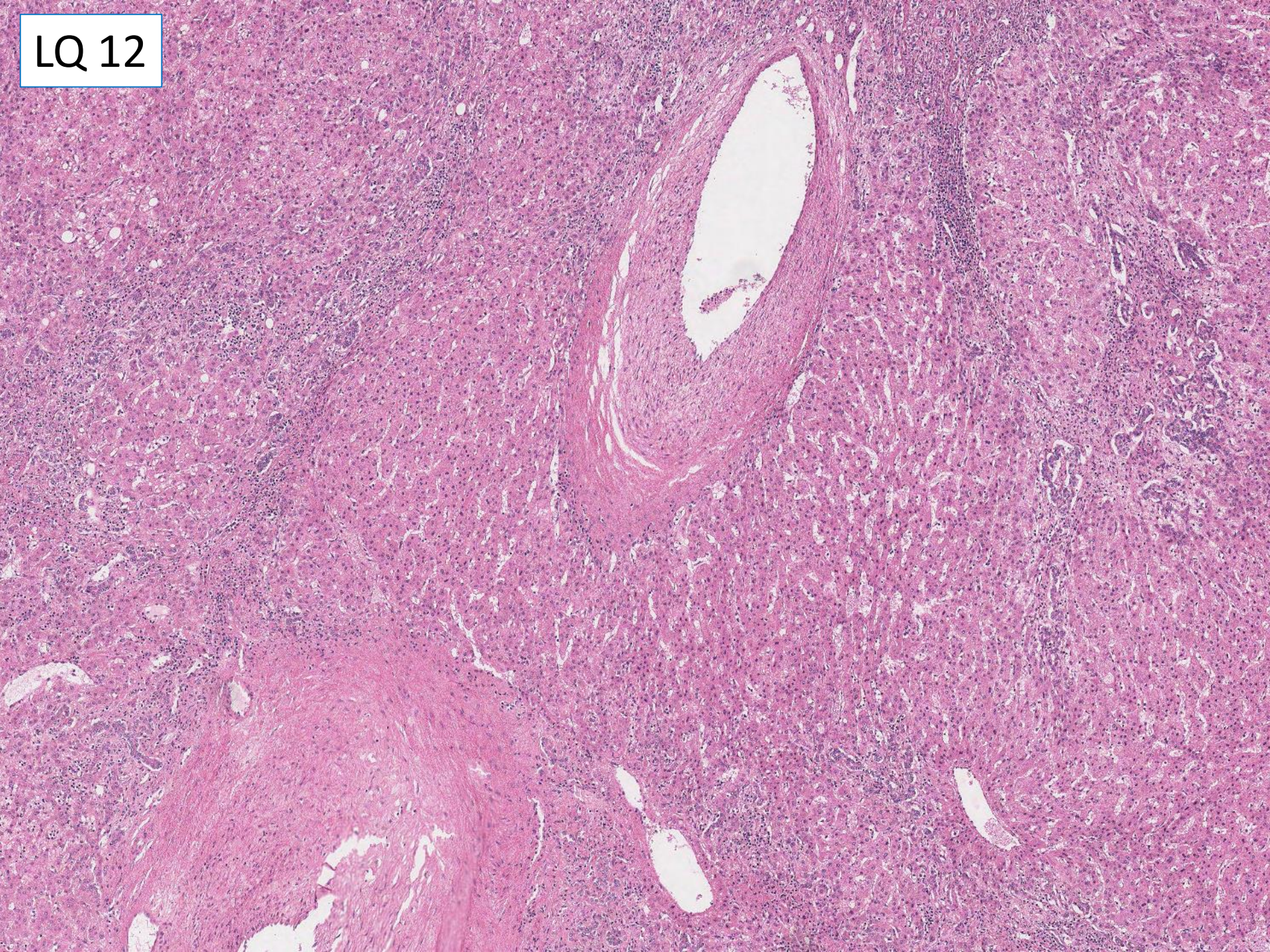
Lesion left lobe liver. Liver resection 135x95x60mm with a circumscribed lesion 35x32x24mm

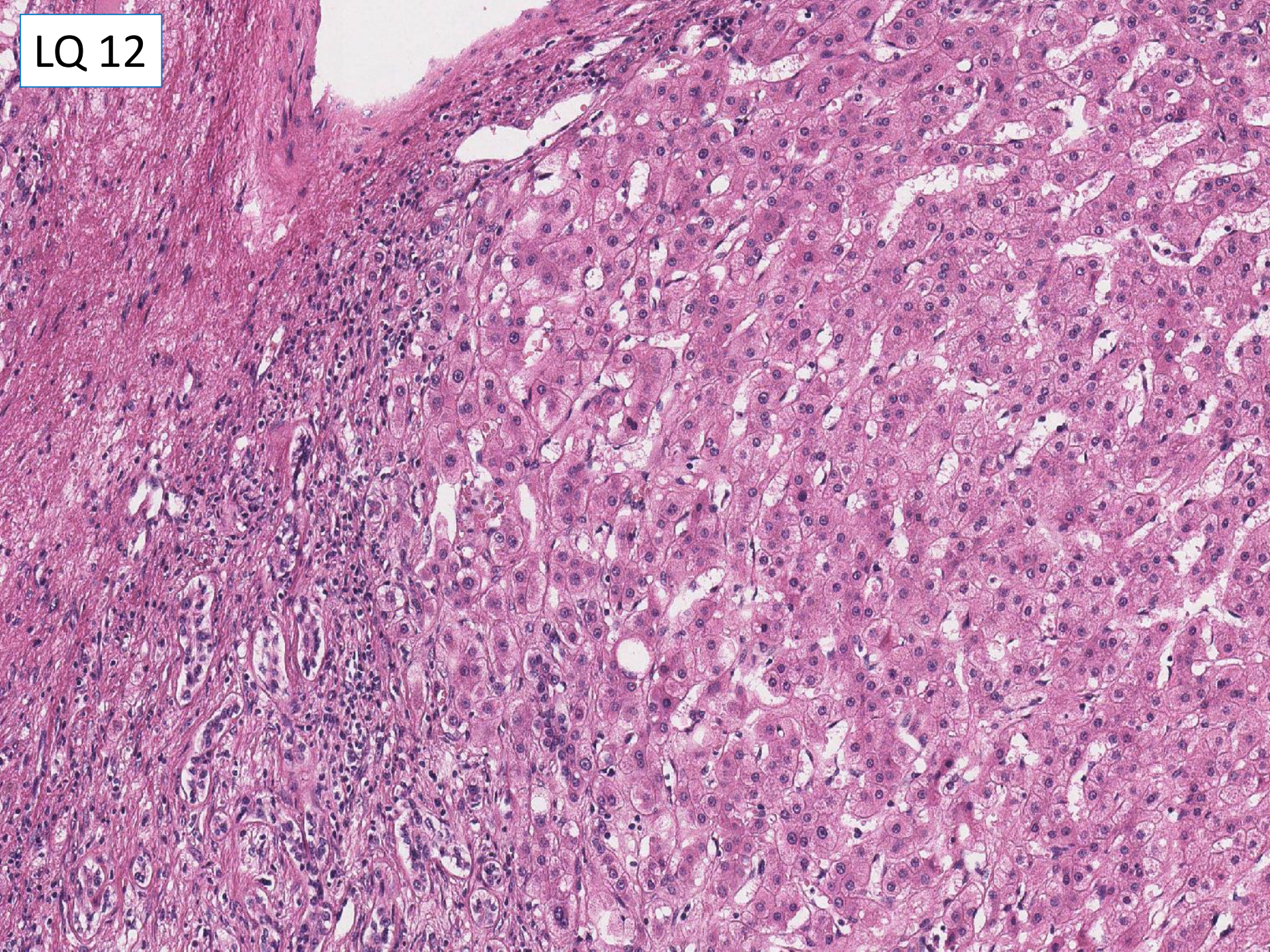


LQ 12 – edge of lesion / background liver




LQ 12


LQ 12


LQ 12

Case LQ12 28M

Lesion left lobe liver. Liver resection 135x95x60mm with a circumscribed lesion 35x32x24mm

Focal nodular hyperplasia	82
Hepatic adenoma - - do stains to confirm.	1

Agreed scoring:
For full marks, a diagnosis of focal nodular hyperplasia.

Score half marks for hepatic adenoma – do stains to confirm.

UK Liver Pathology Group

United Kingdom Liver Pathology Group

Opportunity for more discussion

The end