

Liver histopathology EQA Scheme

Circulation LU

Autumn 2019

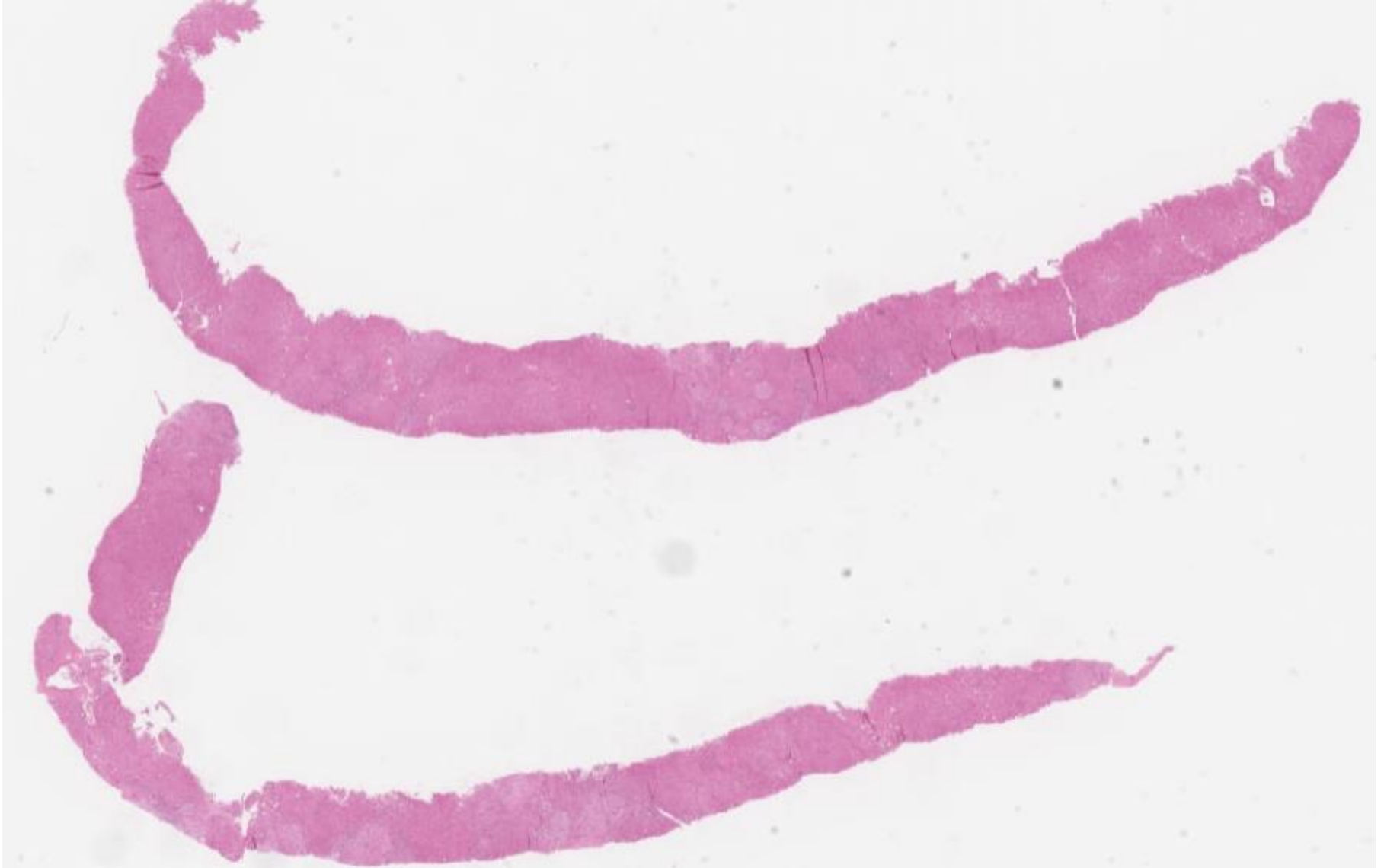
Liver EQA circulation LU Summer 2019

- Collation of 83 responses received and suggestions for scoring from the sub-committee – number required to reach >80% consensus diagnosis is 67.
- Red bars - responses proposed to lose marks
- Green bars - responses uncertain about marking - to ask the audience at the meeting on 7th.

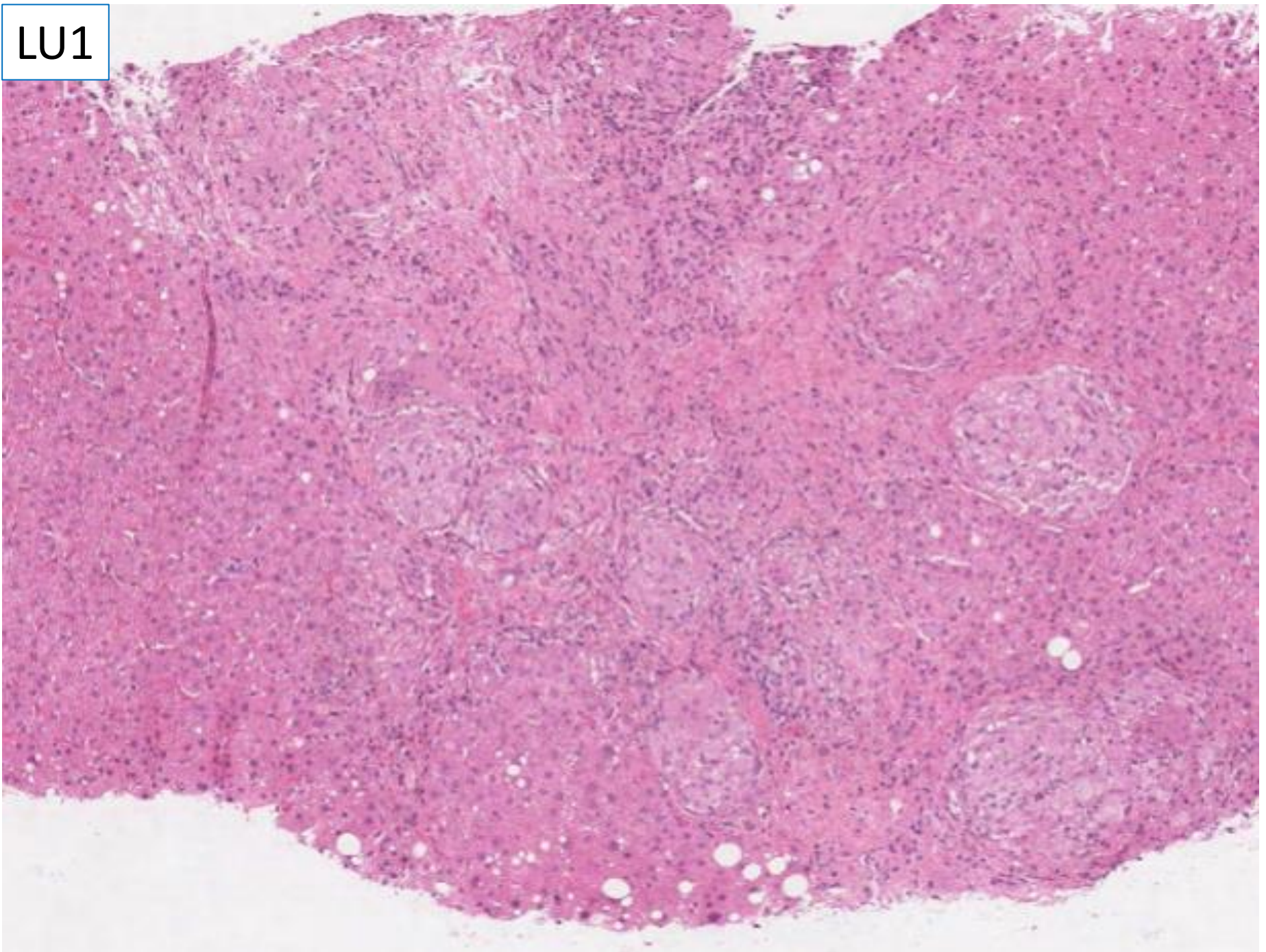
Case LU 1 45M

Deranged LFTs, increased serum ACE level.

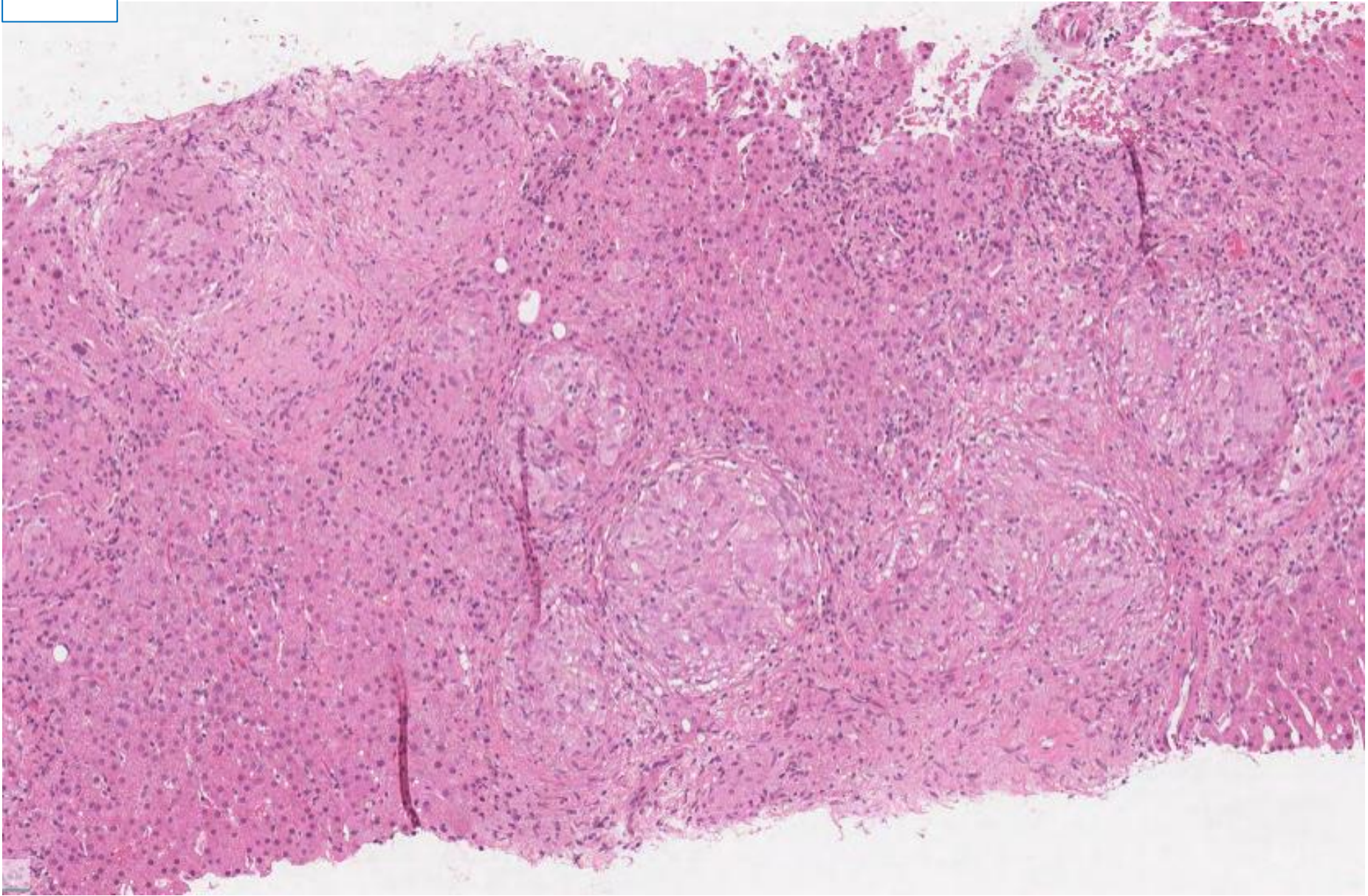
Additional stains: none



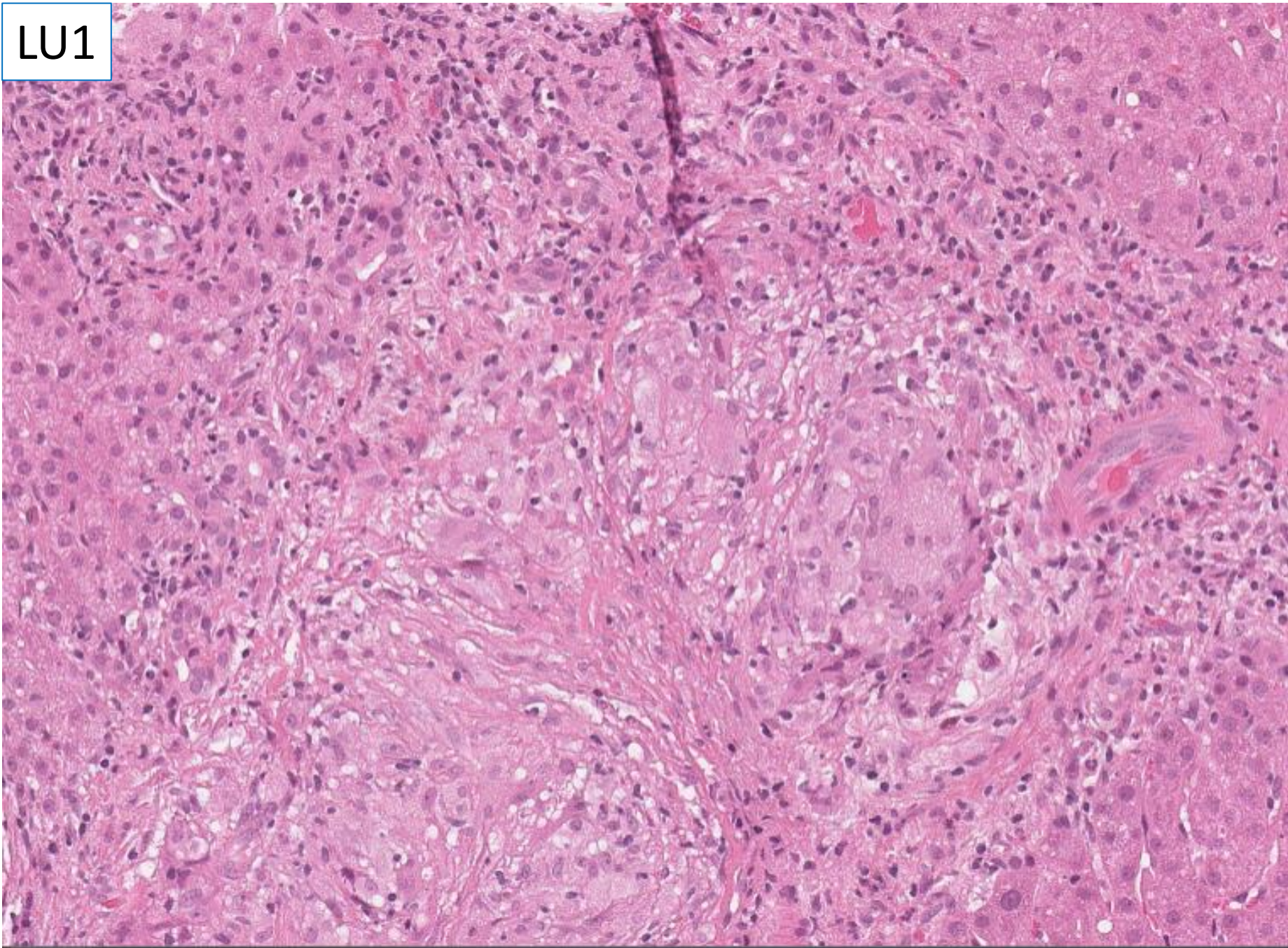
LU1



LU1



LU1



Case LU 1 45M

Deranged LFTs, increased serum ACE level.

Additional stains: none

LU1	
A	Granulomas most likely TB
B	Granulomas most likely sarcoid
C	Biliary disease
D	Primary biliary cholangitis
E	Granulomatous hepatitis, ? Drug induced liver injury

Case LU 1 45M

Deranged LFTs, increased serum ACE level.

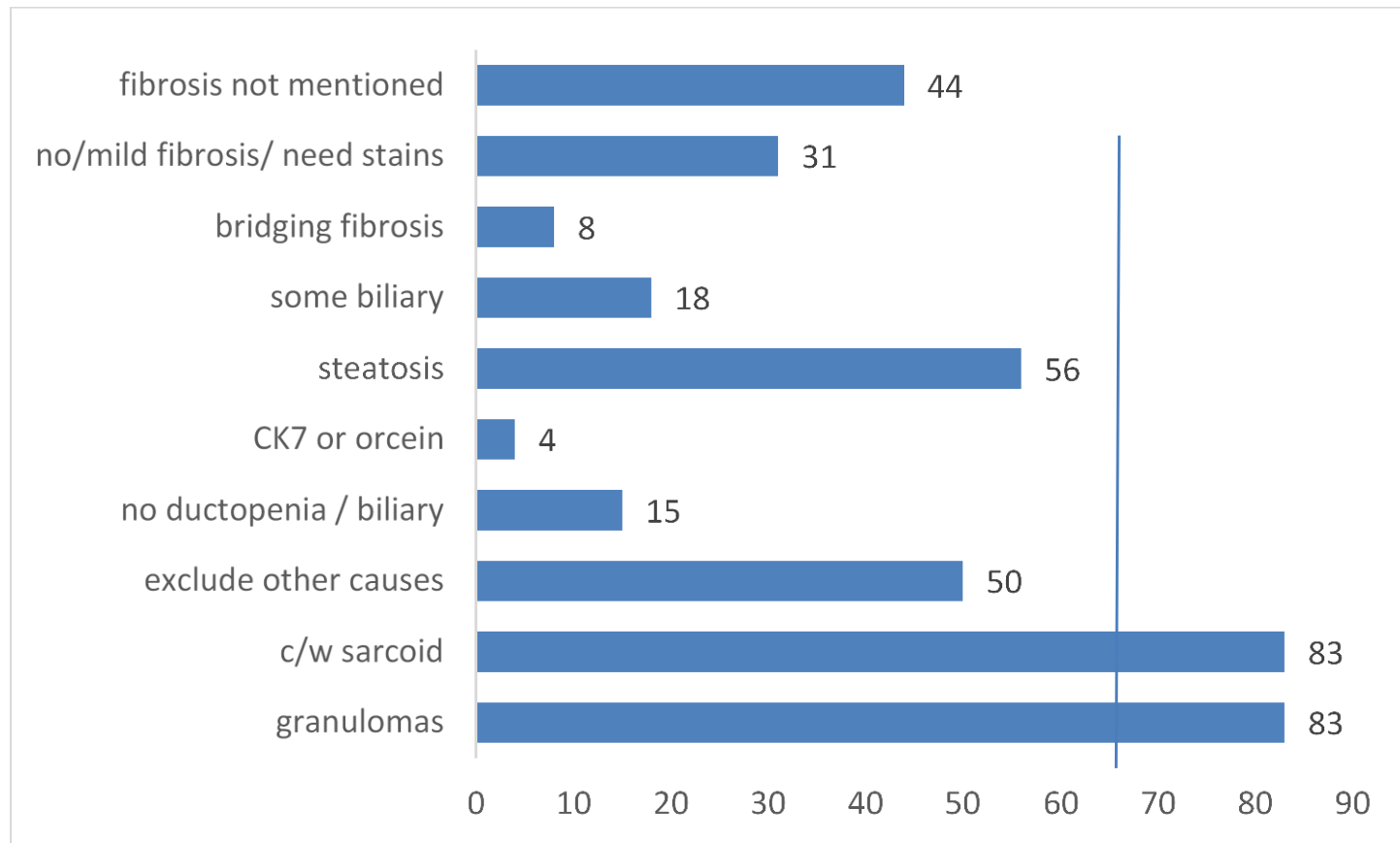
Additional stains: none

LU1	
A	Granulomas most likely TB
B	Granulomas most likely sarcoid
C	Biliary disease
D	Primary biliary cholangitis
E	Granulomatous hepatitis, ? Drug induced liver injury

Case LU 1 45M

Deranged LFTs, increased serum ACE level.

Additional stains: none



Consensus complete responses would include granulomatous inflammation with sarcoid as the only or most likely or first mentioned diagnosis.

Case LU 1 45M

Deranged LFTs, increased serum ACE level.

Additional stains: none

Suggested scoring: for 10 points include granulomatous inflammation with sarcoid as the only or most likely diagnosis. All score 10 points.

Observations/potential learning points,

Since sarcoidosis in the liver may result in chronic cholestatic disease, some comment about the presence/absence of biliary features would be appropriate.

- but insufficient for scoring: 33 mention bile ducts or ductular reaction, of which 18 consider there are biliary features/ductular reaction/ductopenia.

4 mention CK7 and/or orcein.

50 include request for other stains or need to consider alternative causes for granulomas

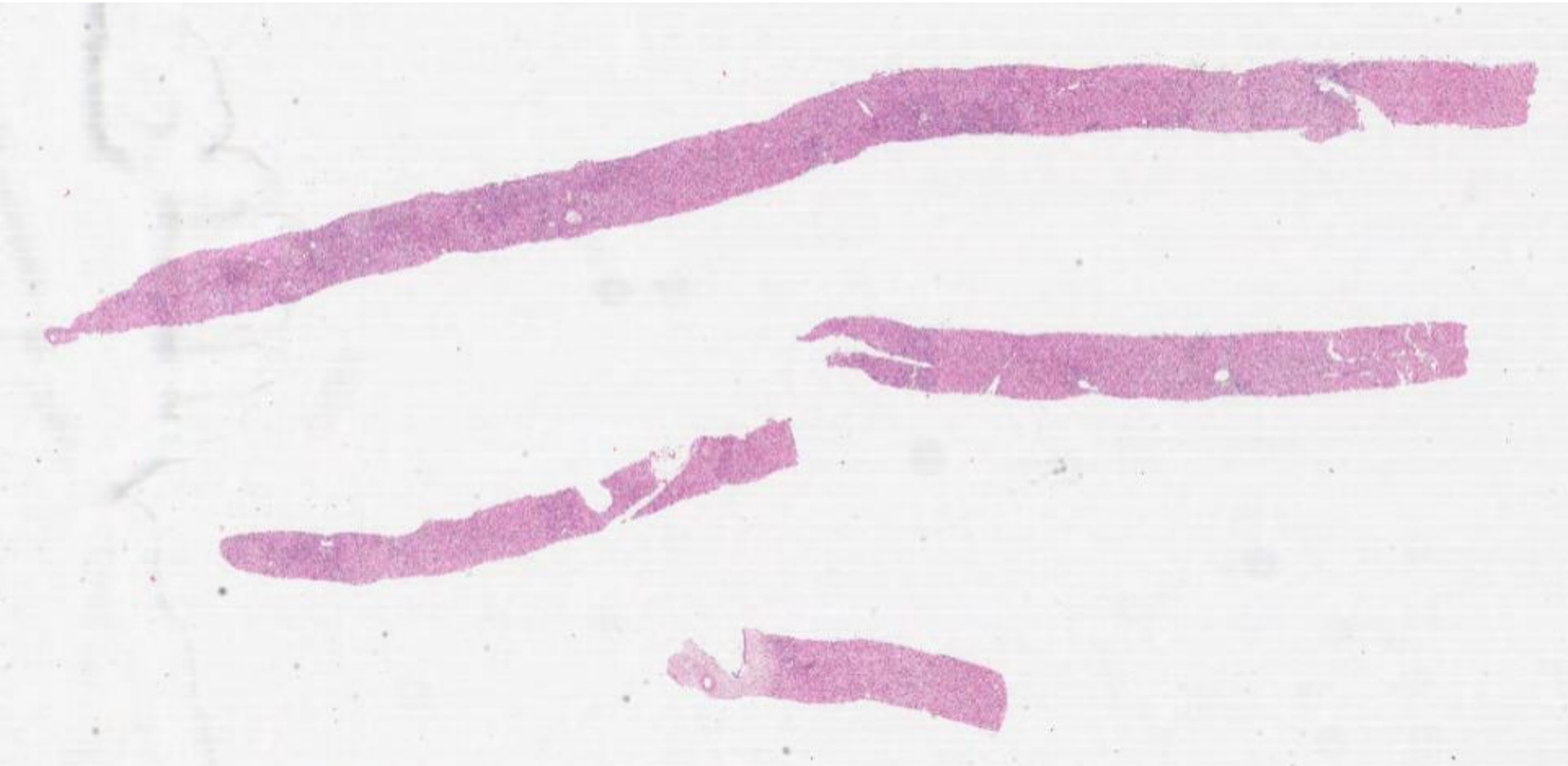
39 include some comment about fibrosis stage, of which 8 consider there is bridging; 31 either mild fibrosis or need special stains for fibrosis.

Case LU2 66F

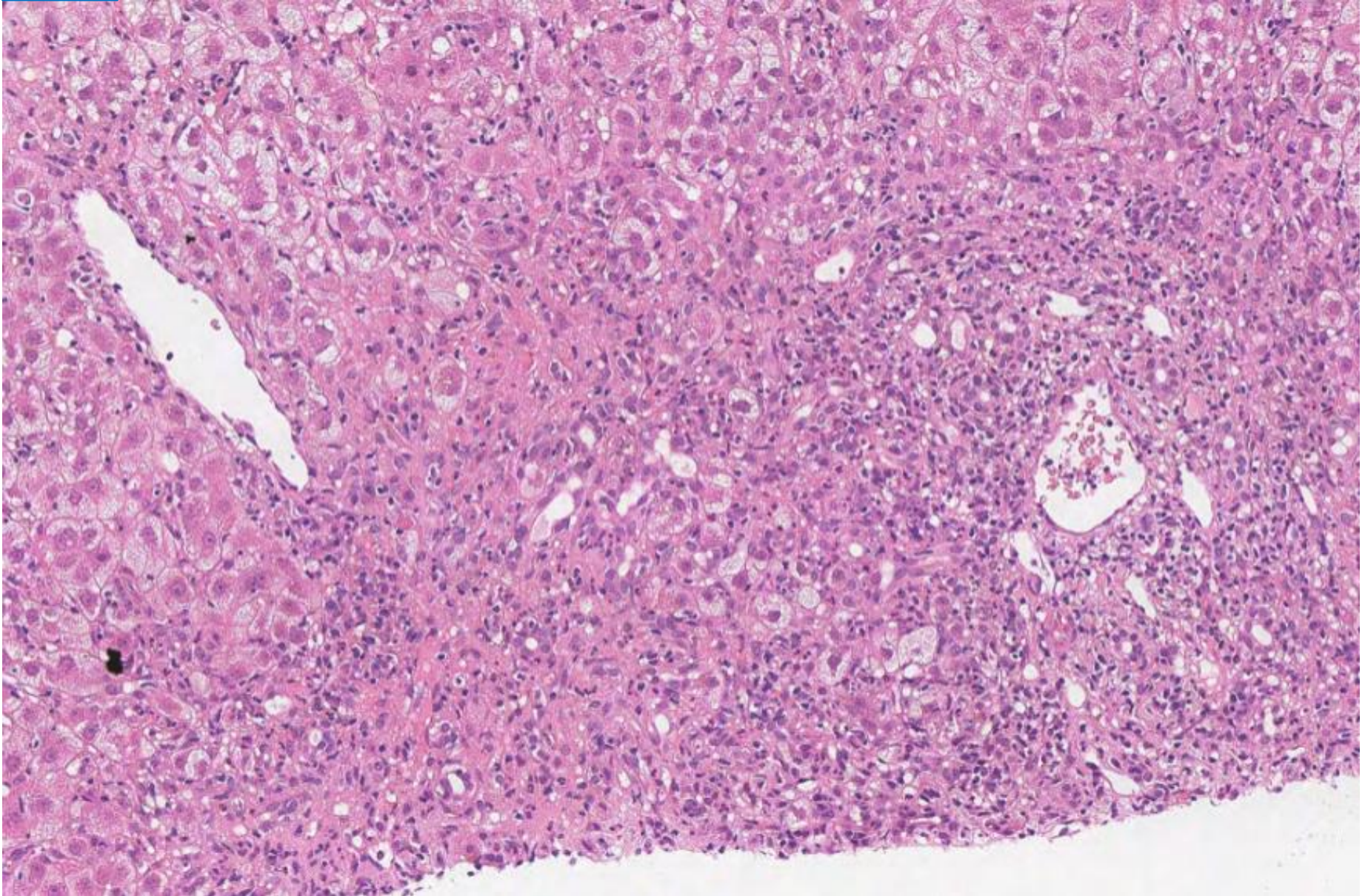
Patient presented with jaundice and diagnosed with AIH. Trial of steroids - no improvement. This is a follow up biopsy.

Specimen: Transjugular liver biopsy. 4 cores, 19,8,7 and 3mm.

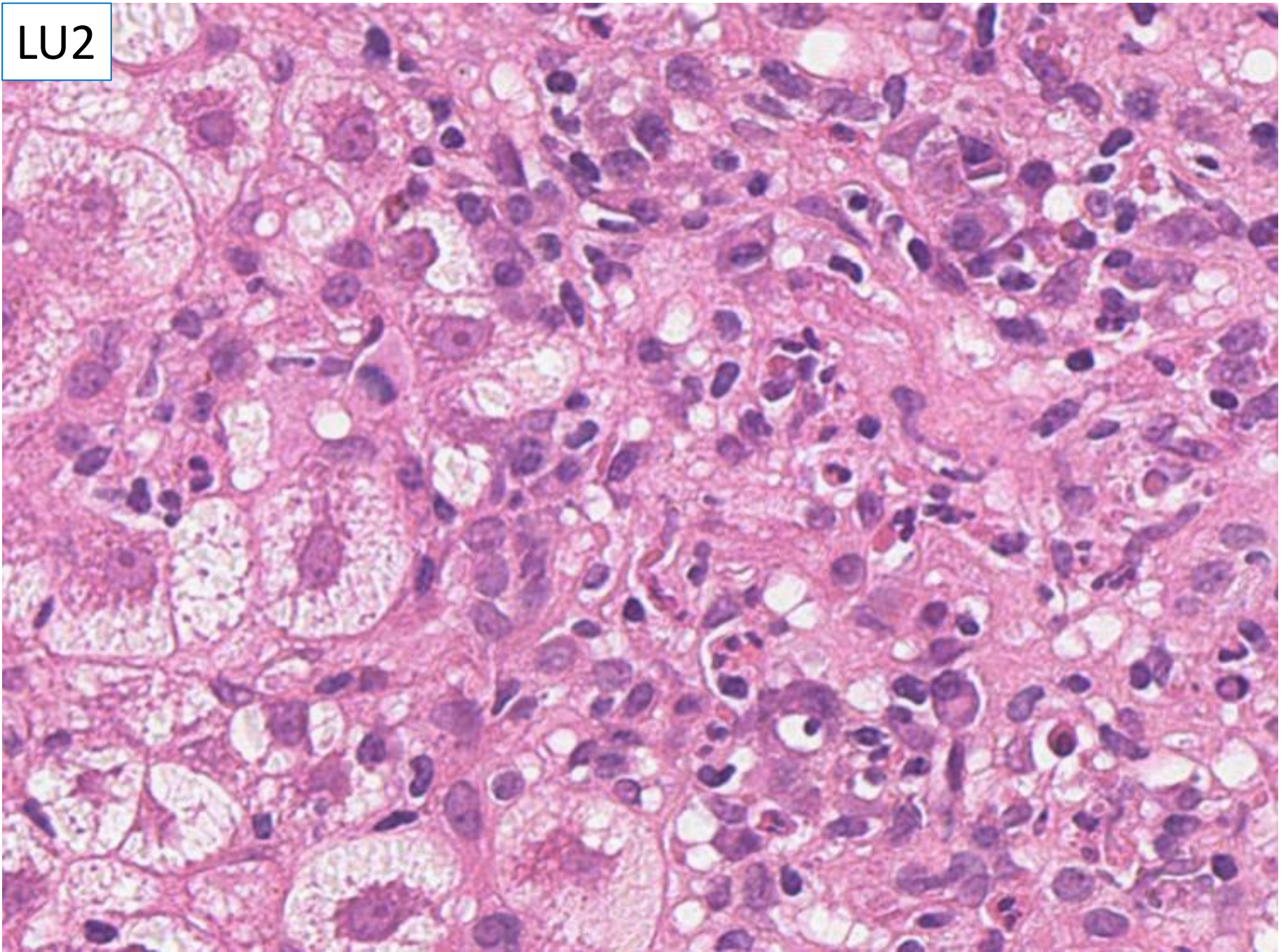
Additional stains: EPSR.



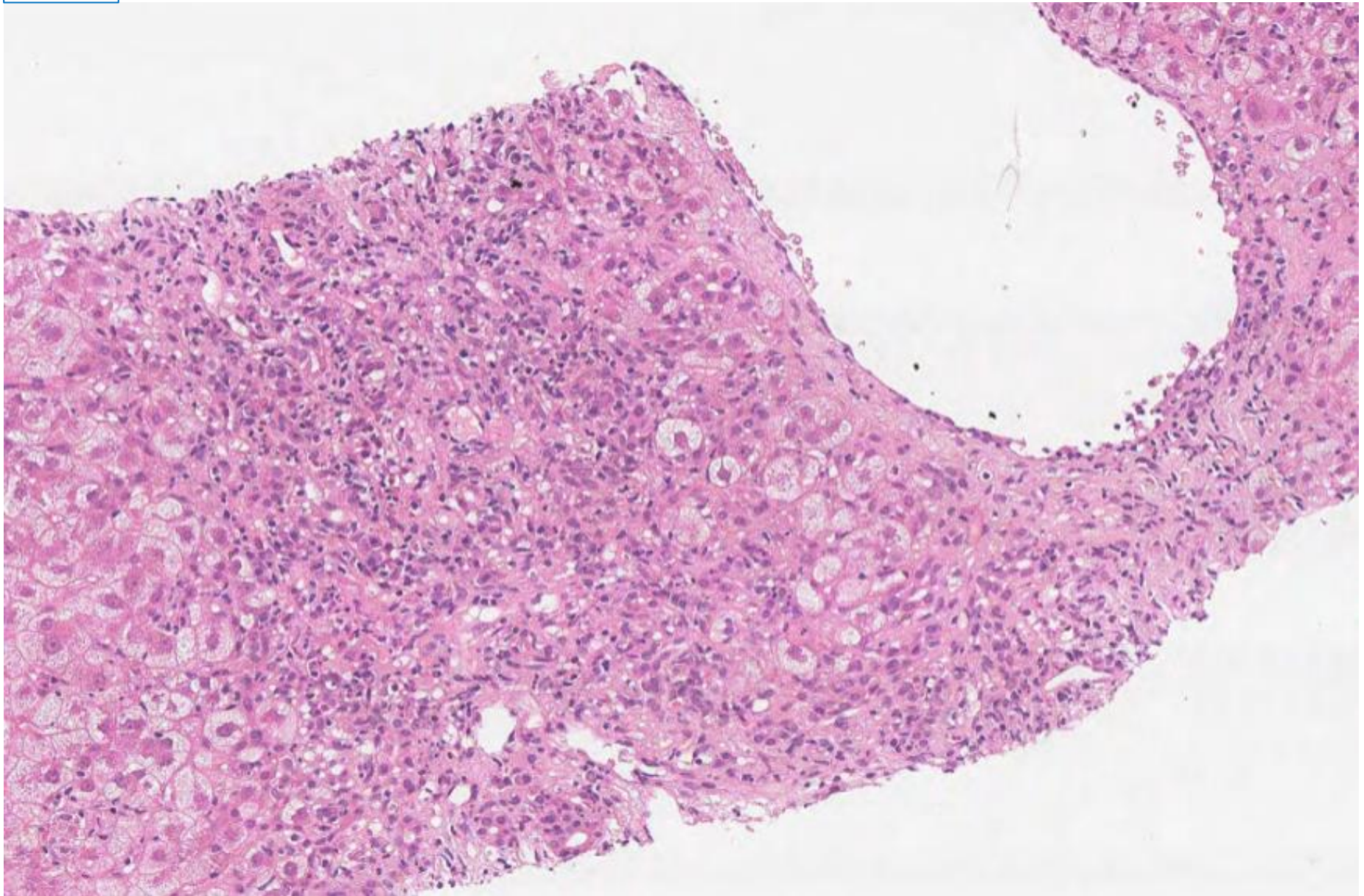
LU2



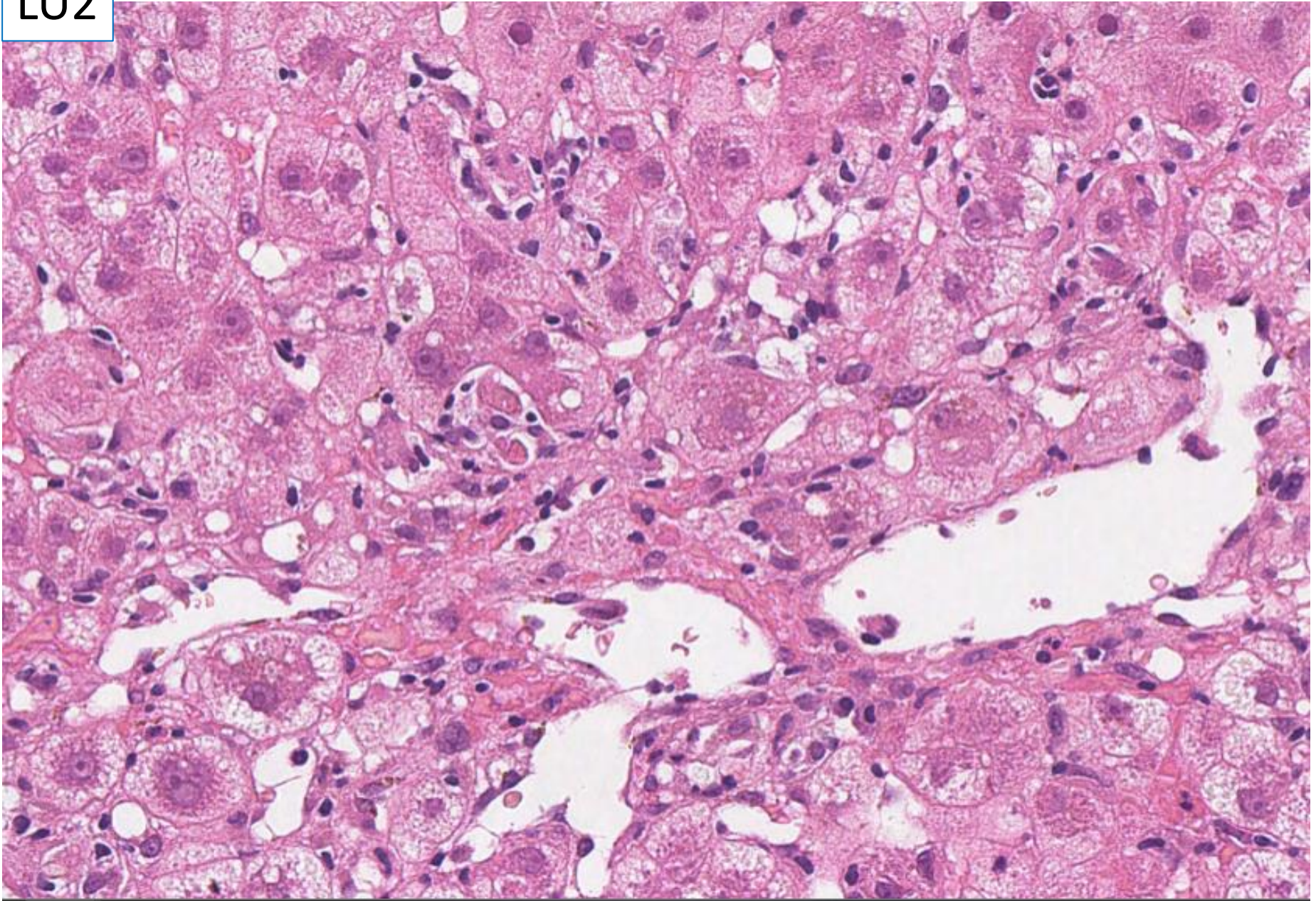
LU2



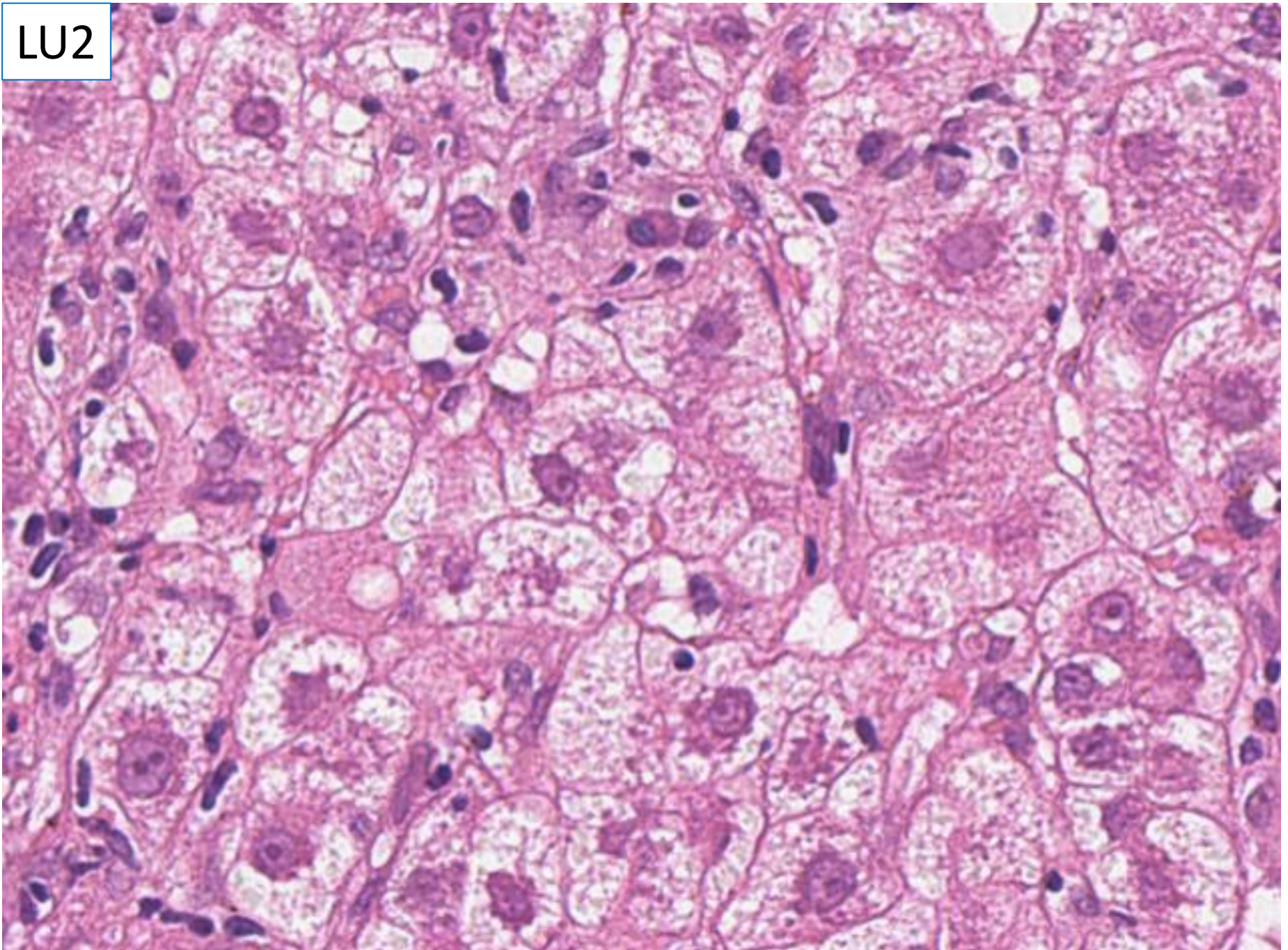
LU2



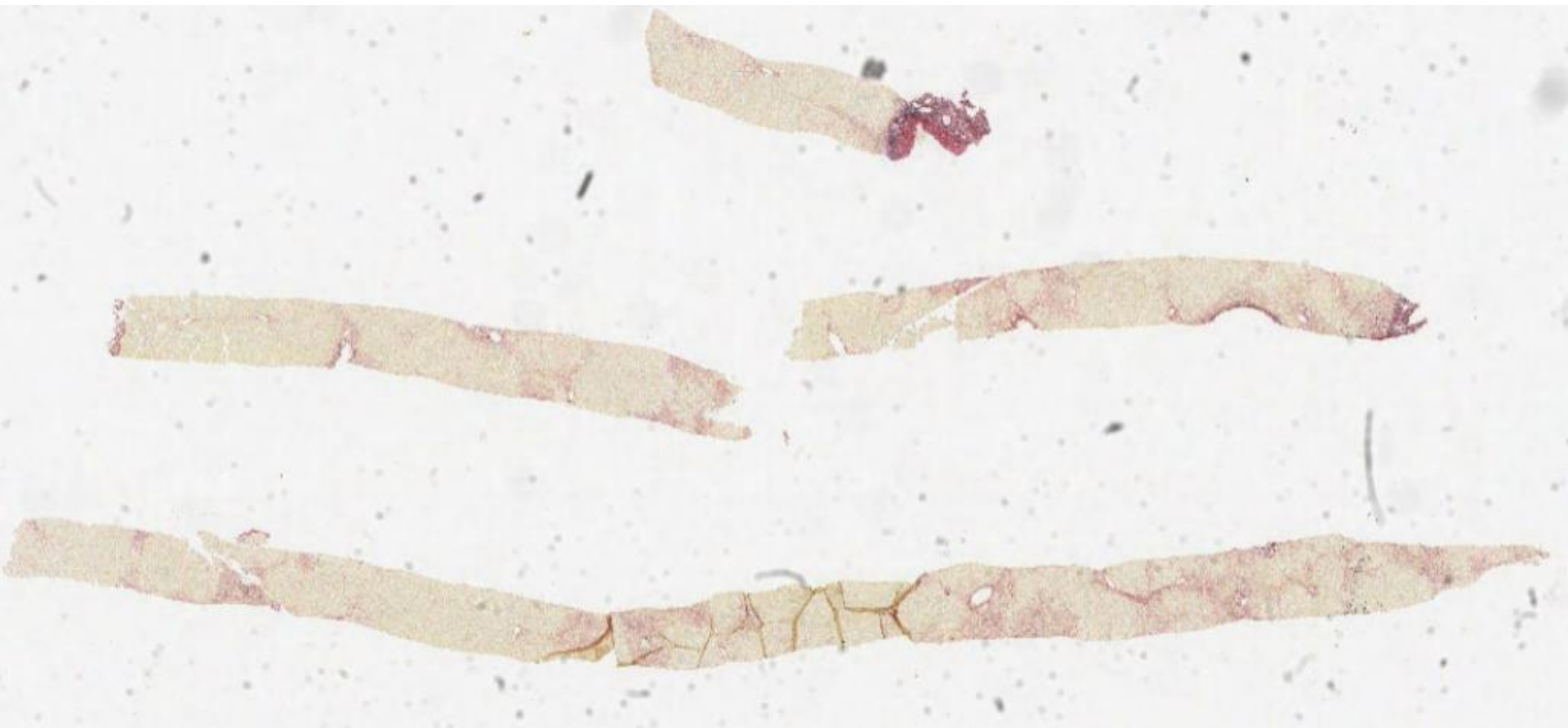
LU2



LU2

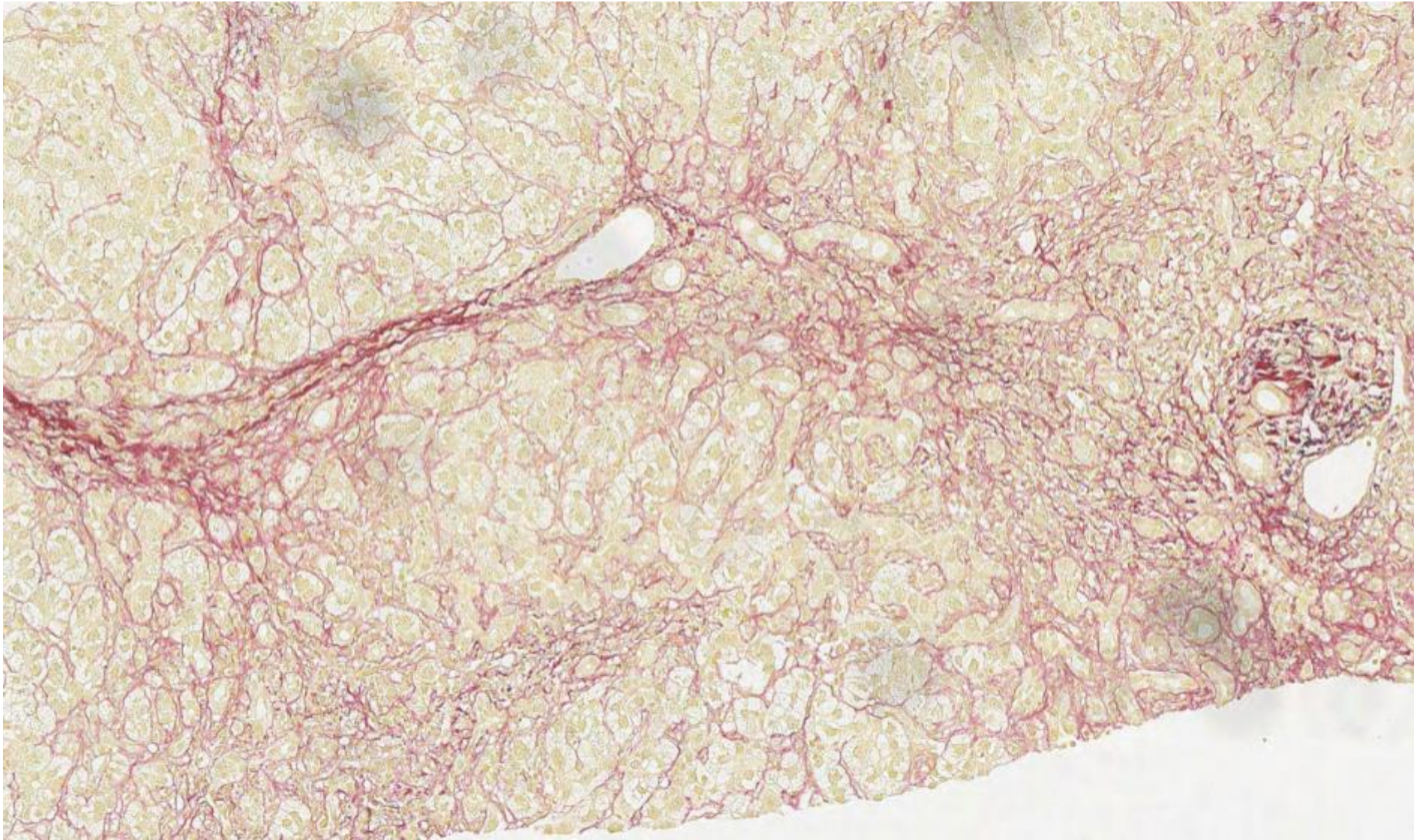


LU2



Elastic picro sirius red

LU2



Elastic picro sirius red

Case LU2 66F

Patient presented with jaundice and diagnosed with AIH. Trial of steroids - no improvement. This is a follow up biopsy.

Additional stains: EPSR.

LU2	
A	Hepatitis chronic with fibrosis ikw AIH
B	Hepatitis acute with necrosis ikw AIH
C	Hepatitis most likely drug
D	Hepatitis most likely viral e.g. Hep E
E	Wilson disease

Case LU2 66F

Patient presented with jaundice and diagnosed with AIH. Trial of steroids - no improvement. This is a follow up biopsy.

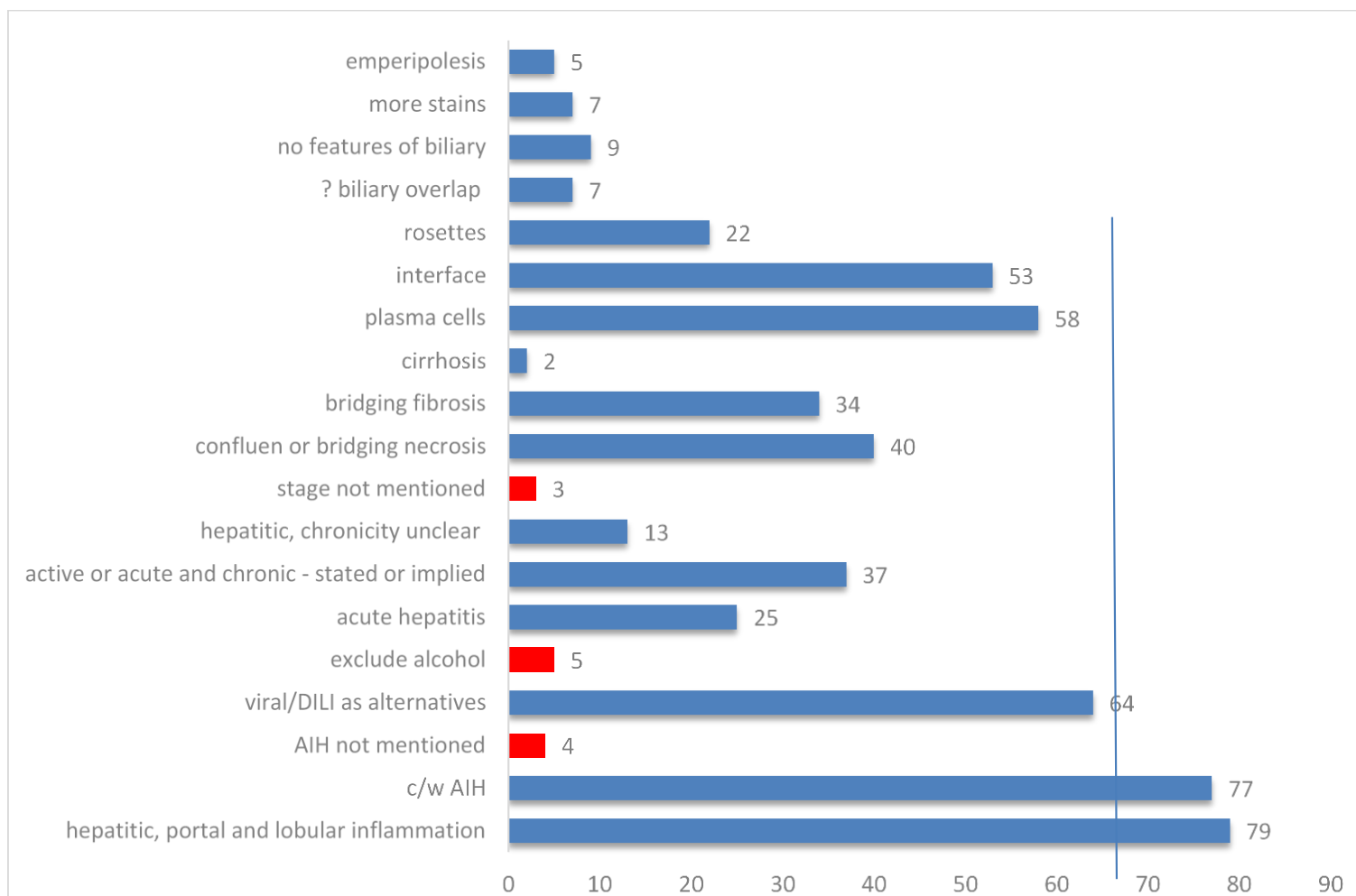
Additional stains: EPSR.

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C	Hepatitis most likely drug
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E	Wilson disease

Case LU2 66F

Patient presented with jaundice and diagnosed with AIH. Trial of steroids - no improvement. This is a follow up biopsy.

Additional stains: EPSR.



Consensus complete responses would include the only items achieving >80% consensus would be a hepatitic pattern, consistent with AIH.

Differential diagnosis including DILI and/or viral hepatitis (E) does not quite reach consensus.

Case LU2 66F

Patient presented with jaundice and diagnosed with AIH. Trial of steroids - no improvement. This is a follow up biopsy.

Additional stains: EPSR.

Suggested scoring: for 10 points include a hepatitic pattern consistent with AIH.

? **Lose 5 marks** if definite diagnosis of cirrhosis (2) [at meeting 16.10.19 agreed not to downscore this – only 1 connective tissue stain](#)

Lose 5 marks if no mention at all of disease stage (collagen stain included). (3)

Lose 5 marks if hepatitic pattern described, but AIH not specifically mentioned – differentials of e.g. DILI, viral, obstructive biliary disease, Wilson's (4)

Lose 5 marks if hepatitic, AIH but also addition of a definite other disease e.g. A1ATD, exclude alcohol, (5)

Lose 10 marks (score 0) if AIH not mentioned (4), a definite diagnosis of alcoholic hepatitis

Case LU2 66F

Patient presented with jaundice and diagnosed with AIH. Trial of steroids - no improvement. This is a follow up biopsy.

Additional stains: EPSR.

Observations/potential learning points,

Good agreement that this was severe hepatitis – but whether bridging necrosis or fibrosis was unclear, as was presence or not of established chronic hepatitis.

Many considered whether there was a reason for clinical unresponsiveness – only 16 commented on evidence of biliary overlap, without agreement over whether there was any.

DILI and viral hepatitis (including hepatitis E) mentioned by many – not quite consensus.

Of the histological features supporting AIH – a majority include a specific mention of plasma cells and interface hepatitis; a minority mention rosettes or emperipolesis.

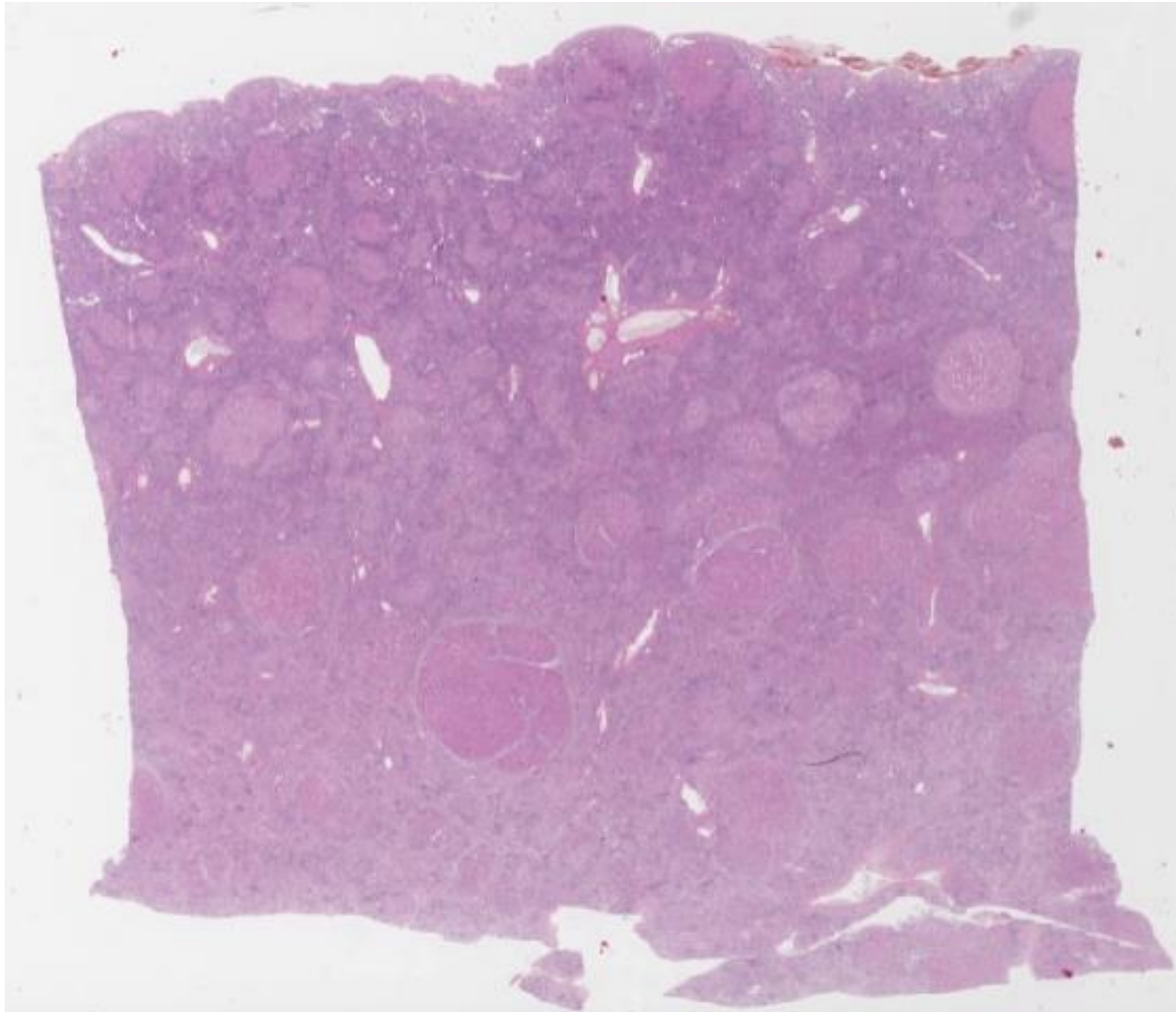
There was discussion about Sirius red v van Gieson for assessing the maturity of collagen – EPSR in this case (elastin Picro Sirius Red) – shows both elastic and collagen. Mature collagen stains darker red with both PSR and VG – but overall the PSR is darker – therefore pathologists unfamiliar with PSR may tend to overestimate the degree and maturity of collagen.

Case LU3 59M

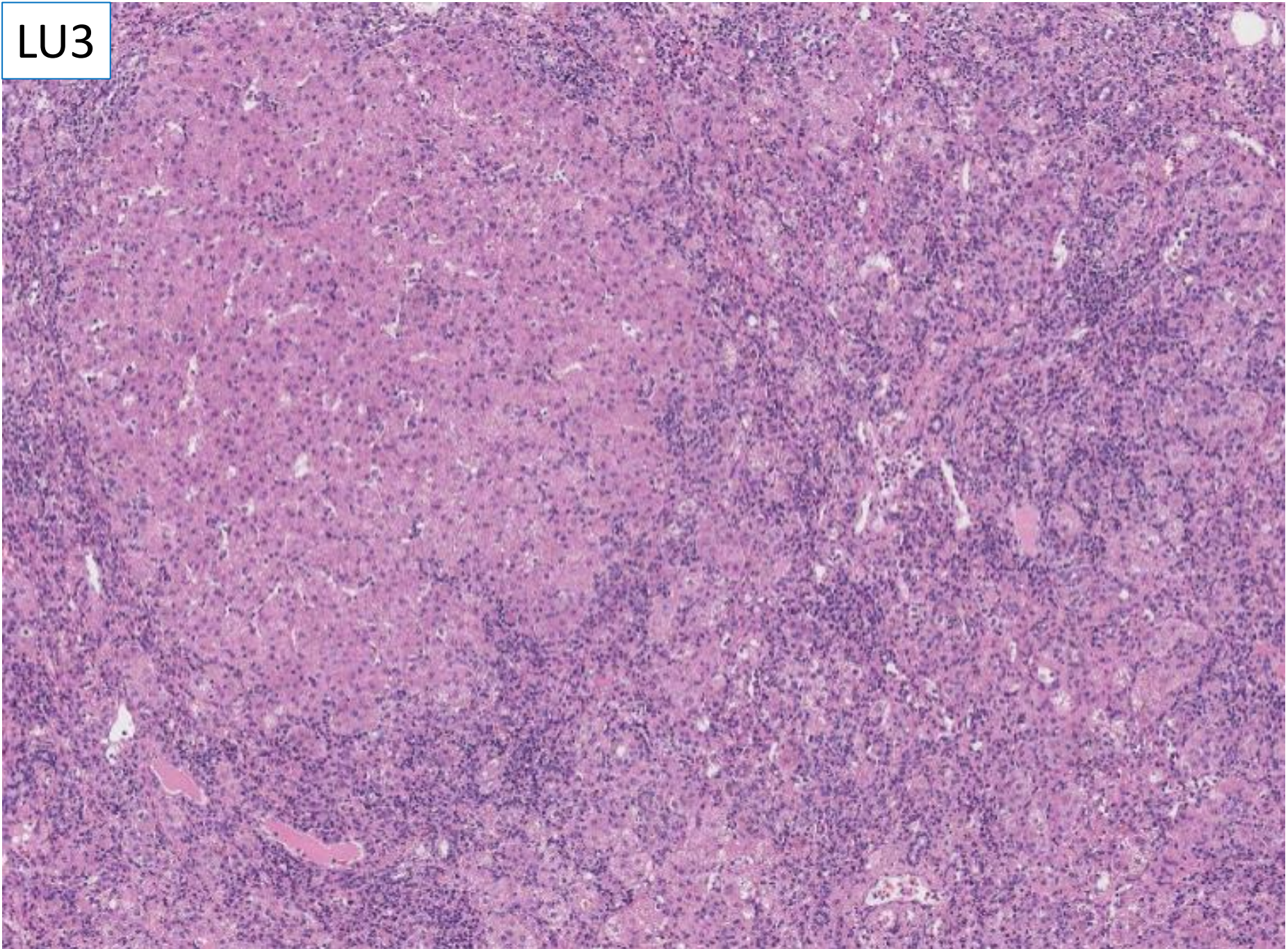
Liver transplant for autoimmune hepatitis.

Explant liver 1316g, cirrhotic appearance, no focal lesions seen.

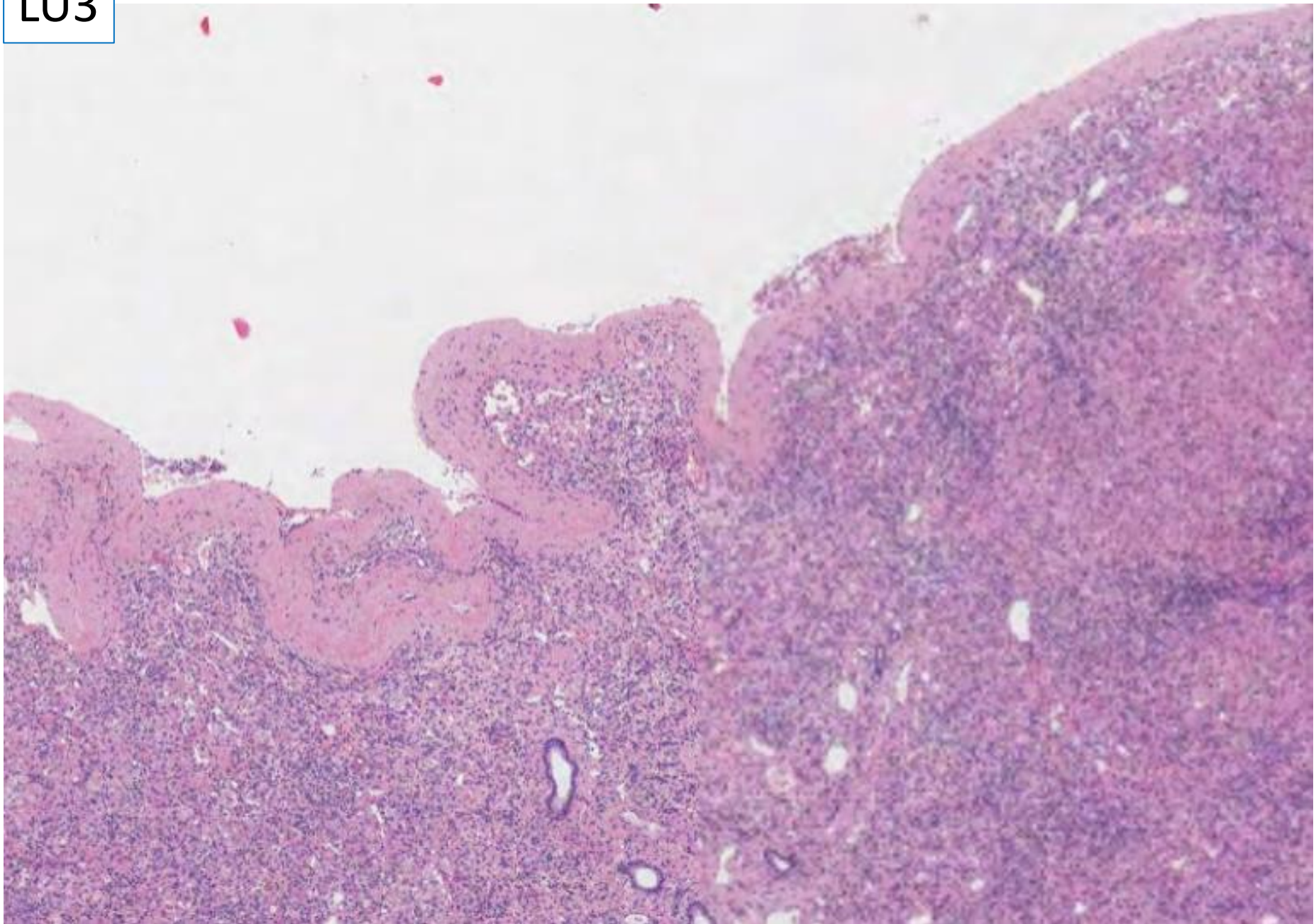
Additional stains: van Gieson, Shikata



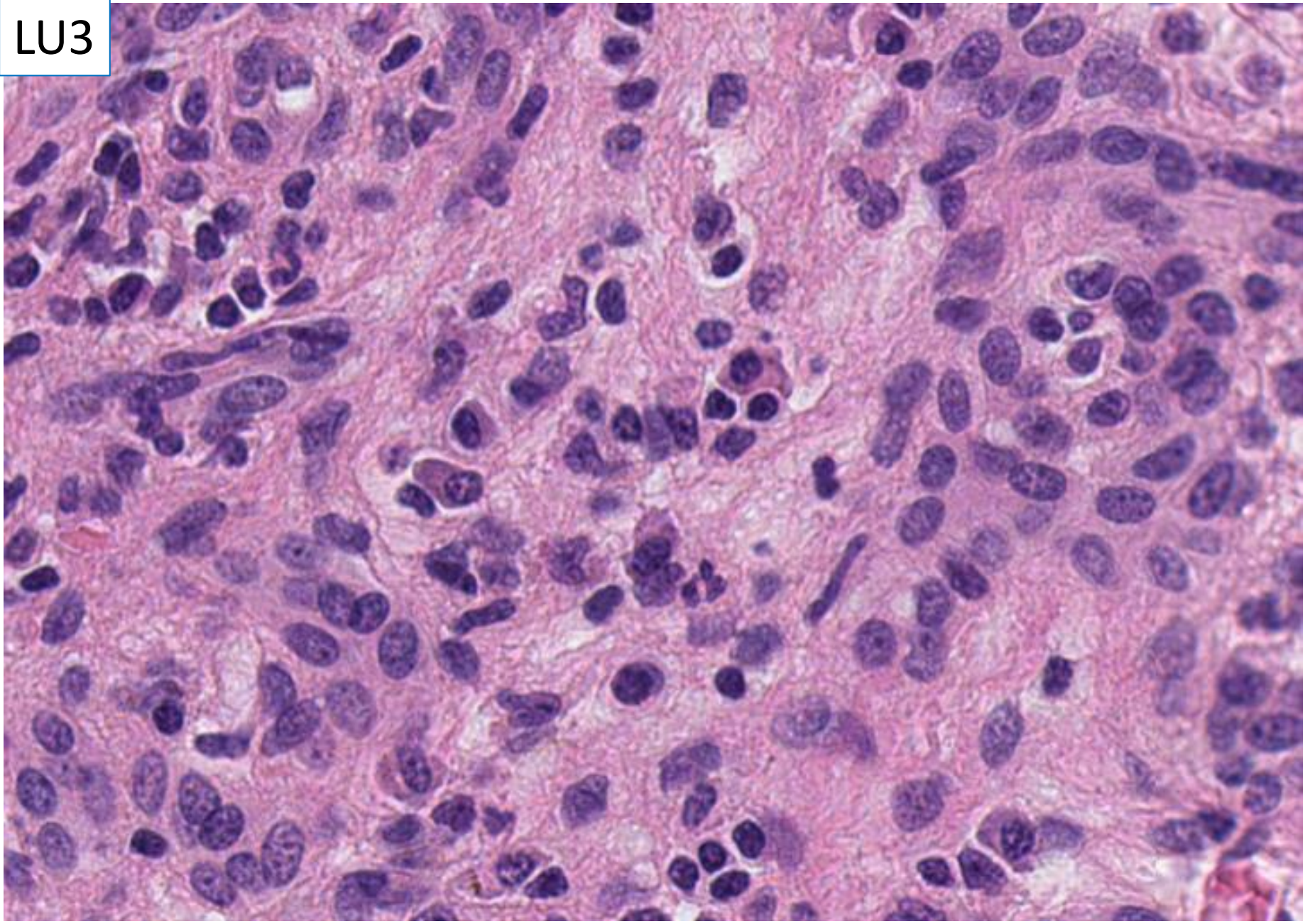
LU3



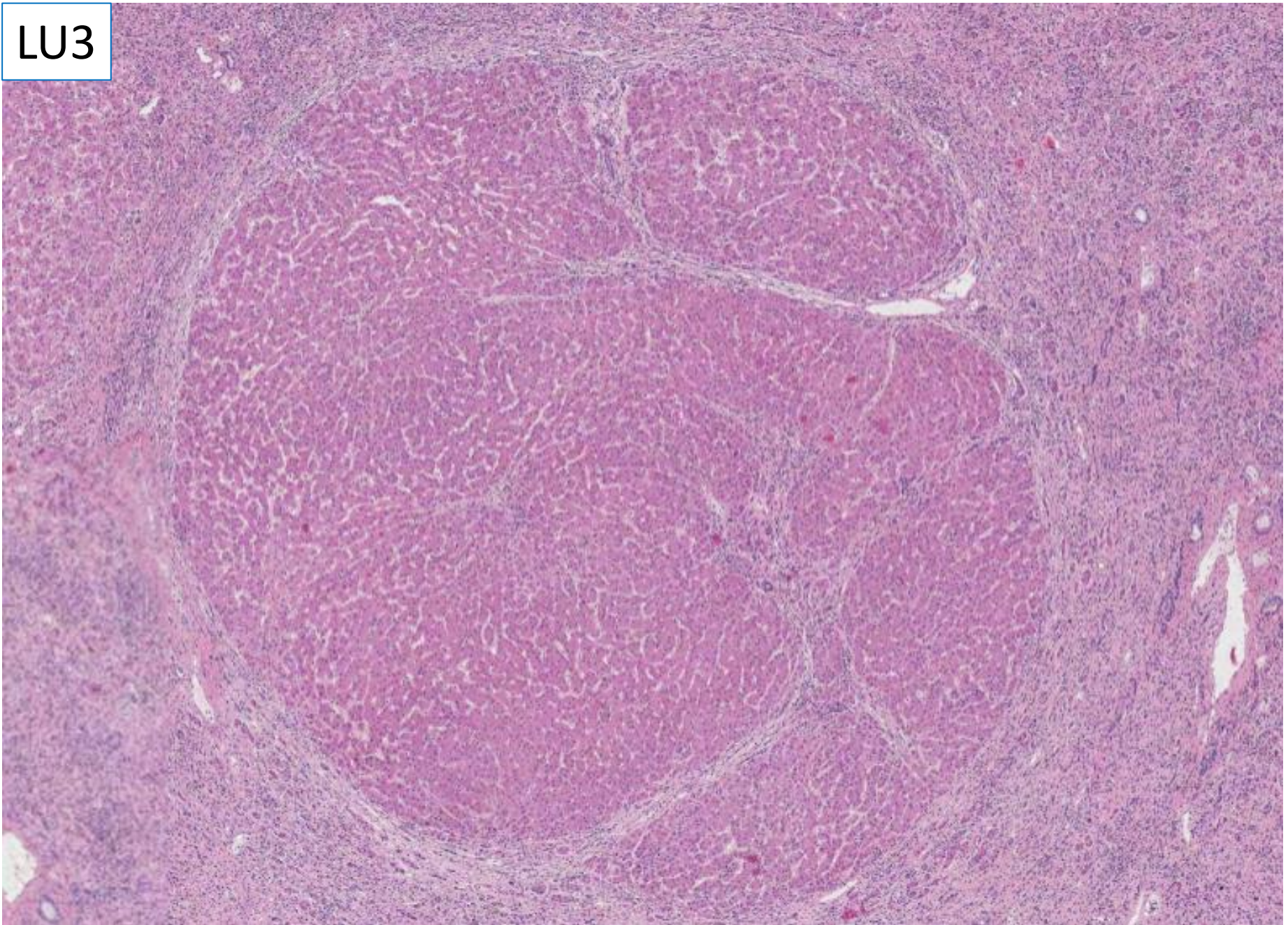
LU3



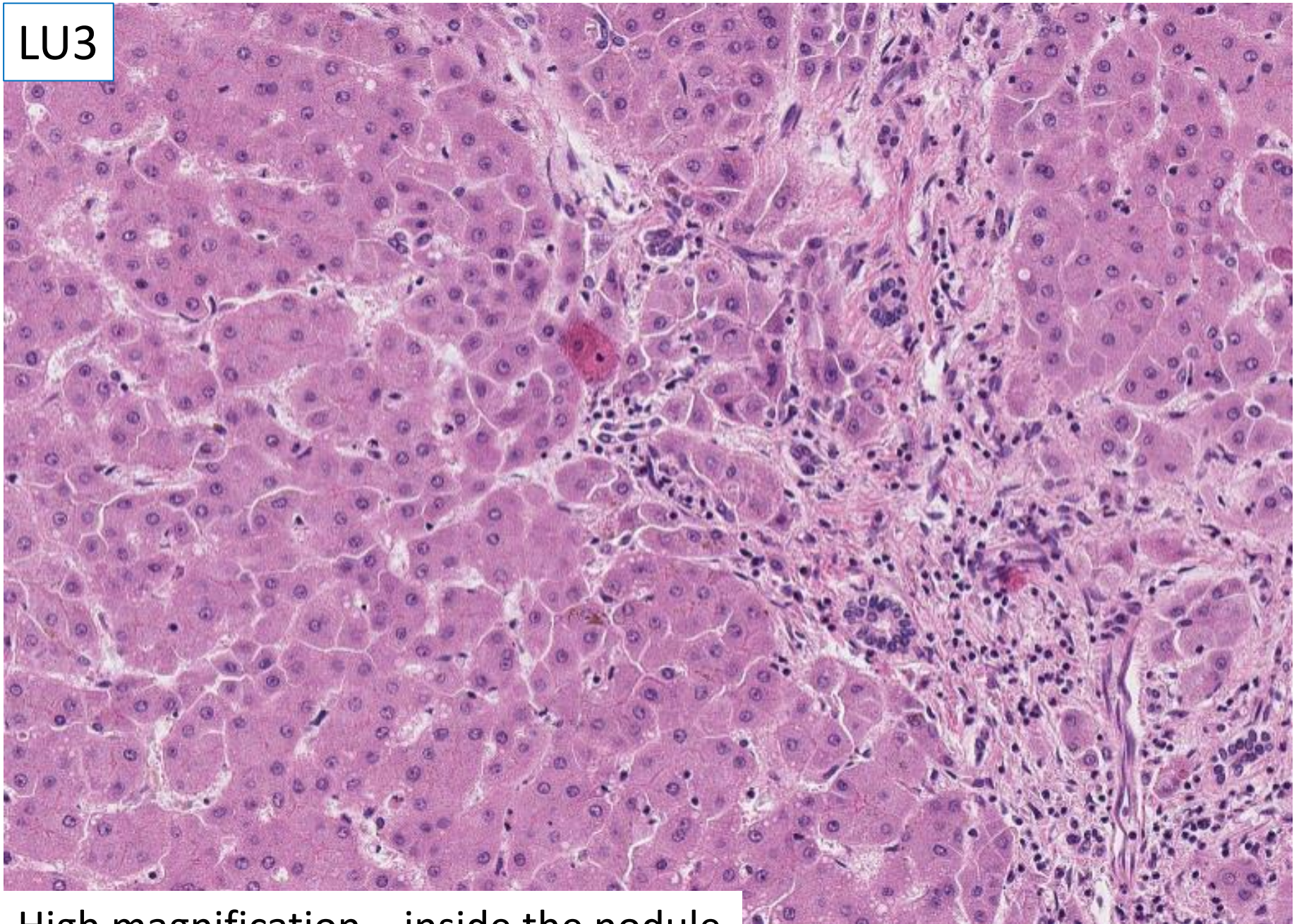
LU3



LU3

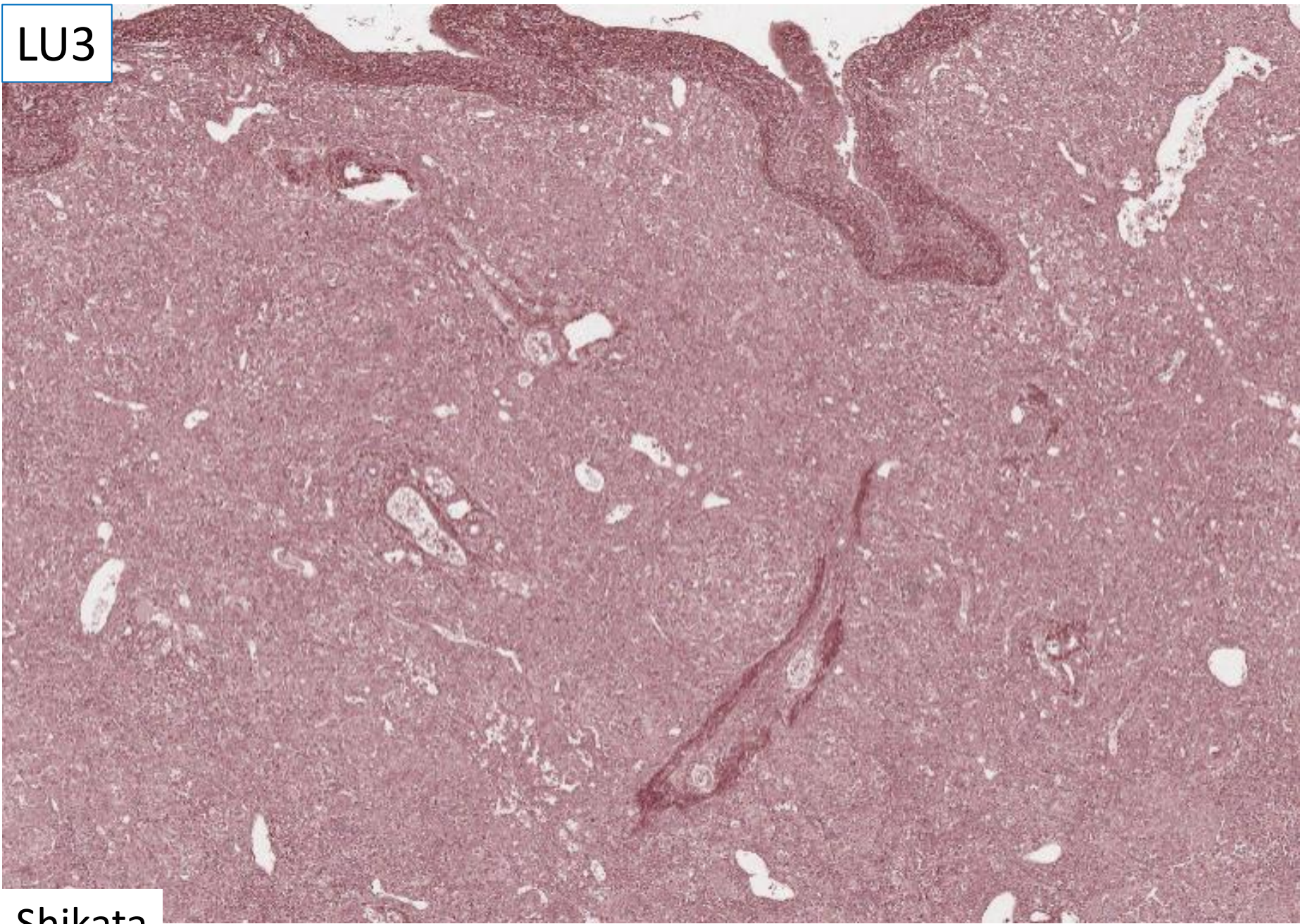


LU3



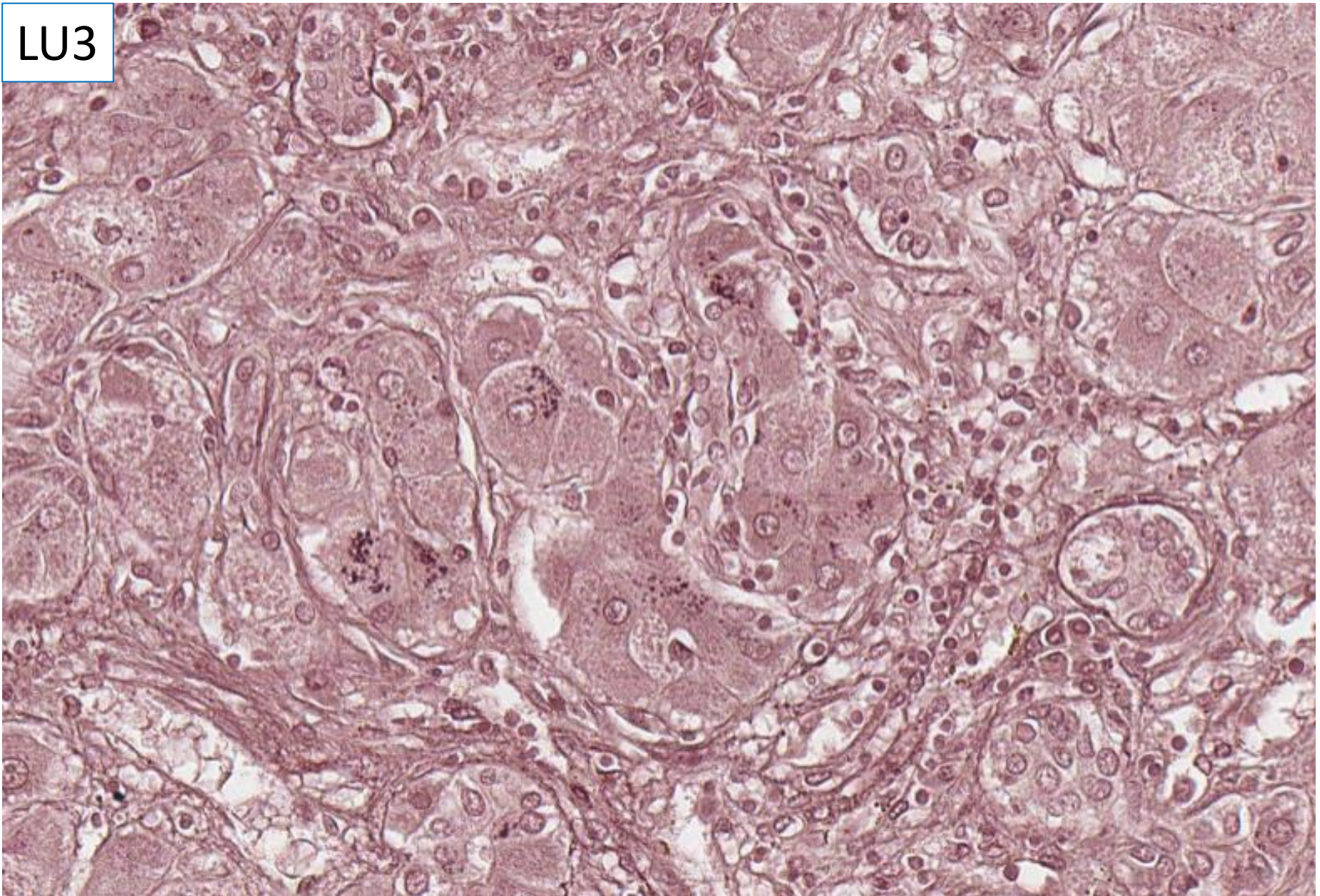
High magnification – inside the nodule

LU3



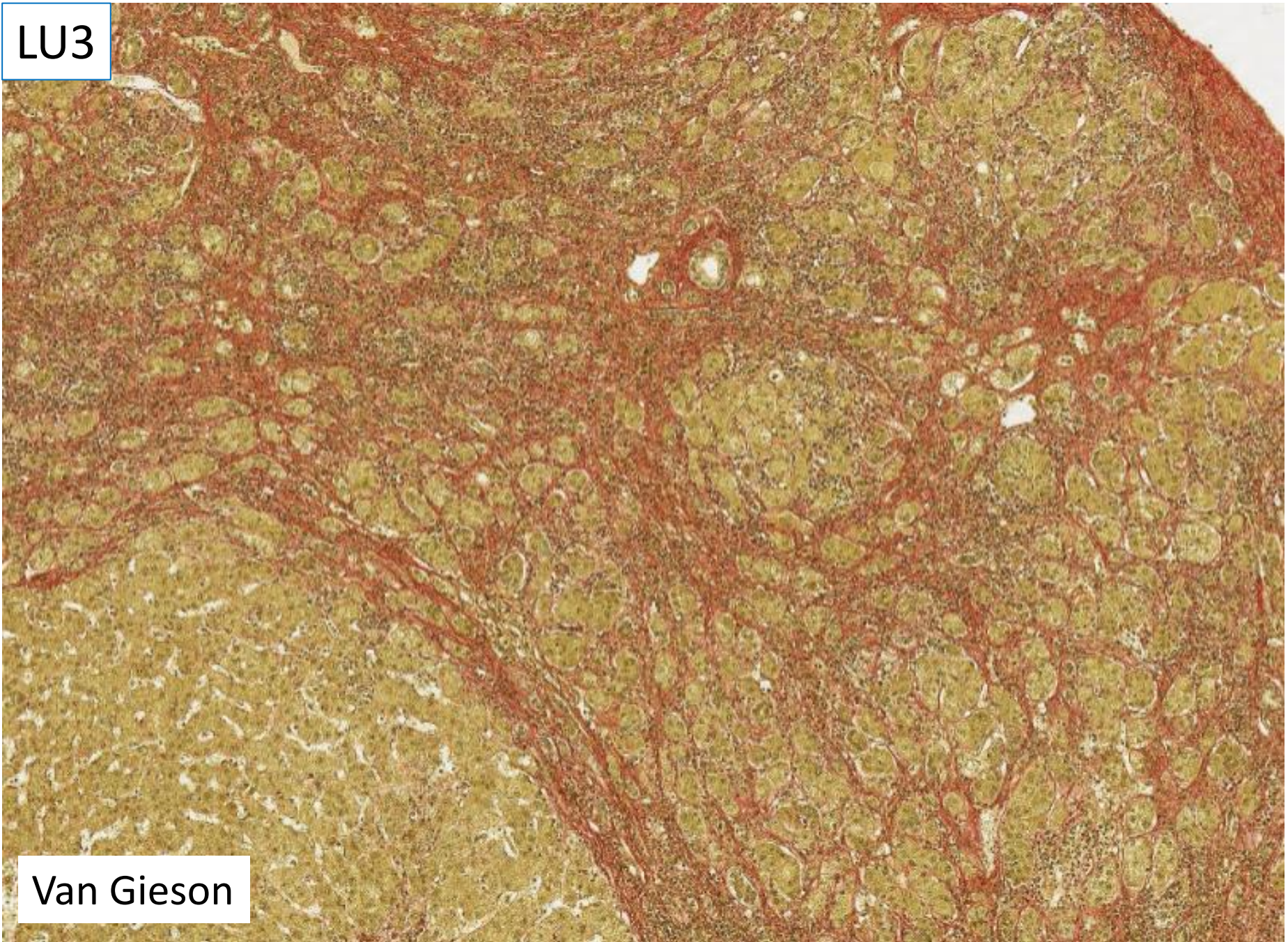
Shikata

LU3



Shikata

LU3



Van Gieson

Case LU3 59M

Liver transplant for autoimmune hepatitis.

Explant cirrhotic appearance, no focal lesions seen.

Additional stains: van Gieson, Shikata

LU3	
A	Wilson disease
B	Dysplastic nodules
C	Hepatocellular carcinoma
D	Congenital hepatic fibrosis
E	Post necrotic collapse/fibrosis ikw AIH

Case LU3 59M

Liver transplant for autoimmune hepatitis.

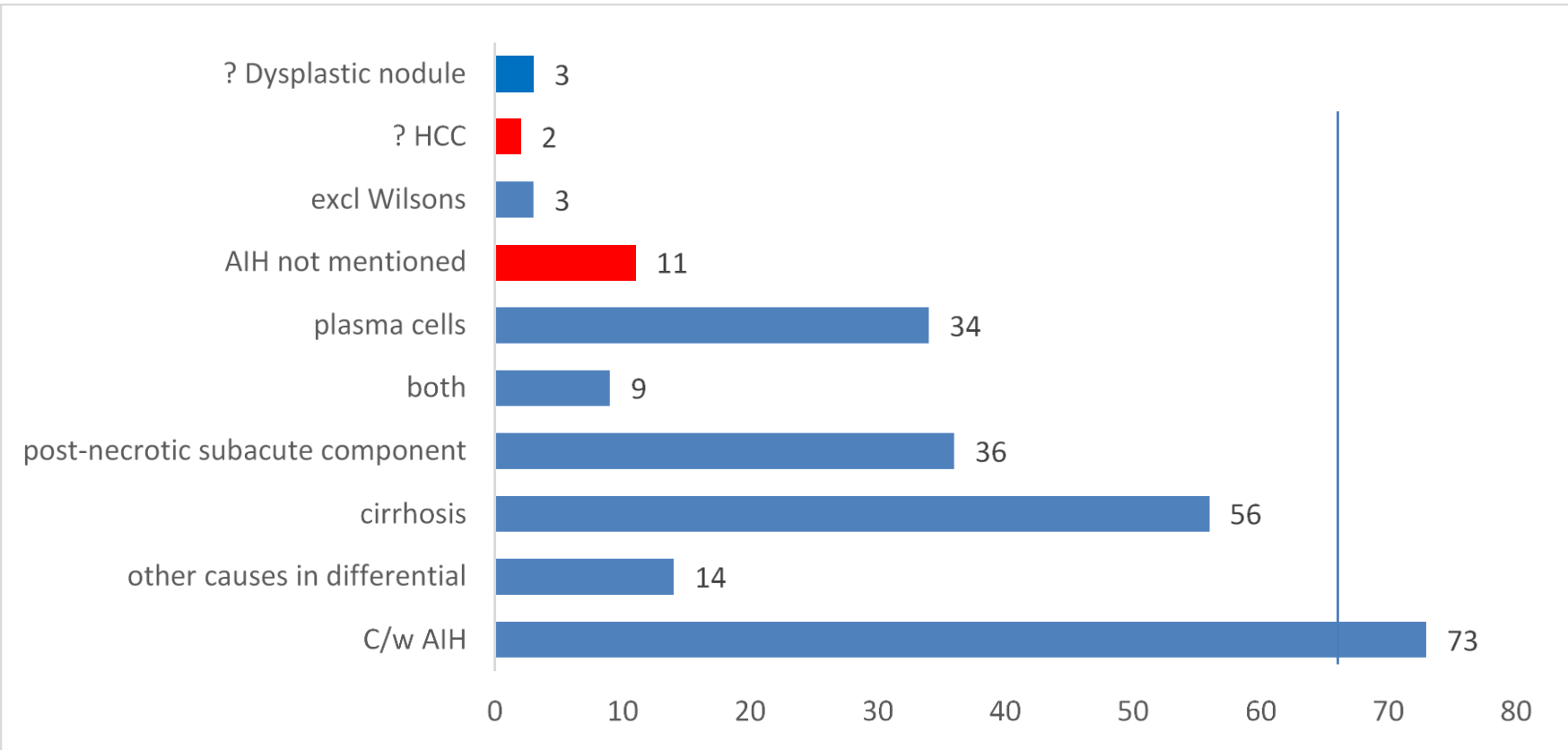
Explant cirrhotic appearance, no focal lesions seen.

Additional stains: van Gieson, Shikata

LU3	
A	Wilson disease
B	Dysplastic nodules
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D	Congenital hepatic fibrosis
E	Post necrotic collapse/fibrosis ikw AIH

Case LU3 59M

Liver transplant for autoimmune hepatitis.
Explant cirrhotic appearance, no focal lesions seen.
Additional stains: van Gieson, Shikata



Consensus complete responses would include a description of the architecture – as either cirrhosis or post necrotic subacute component, or both.
Duration of disease is unclear. Consistent with AIH.

Case LU3 59M

Liver transplant for autoimmune hepatitis.

Explant cirrhotic appearance, no focal lesions seen.

Additional stains: van Gieson, Shikata

Suggested scoring: for 10 points include description of architecture and consistent with AIH.

Lose 5 marks if no mention that this is consistent with AIH? (11) [Agreed 16.10.19](#)

Lose 5 marks if probable or definite dysplastic nodule (2)
(not if possible DN, consult with colleague).

[At meeting 16.10.19 agreed not to downscore this, difficult definitions of dysplastic nodules.](#)

Lose 10 marks (score 0) if cirrhosis with probable infiltrating HCC (2)
– IHC glypican and hepar 1.

Case LU3 59M

Liver transplant for autoimmune hepatitis.

Explant cirrhotic appearance, no focal lesions seen.

Additional stains: van Gieson, Shikata

Observations/potential learning points,

Problem with special stains – these were on the website, but the link from EQAlite didn't work, so not everyone saw them.

Post-confluent multiacinar necrosis with dominant regenerative nodules; this is a pattern consistent with autoimmune hepatitis, although non-specific and would need to know the clinical context to make the diagnosis.

Several commented on the presence of copper binding protein (13) – some considering Wilson's disease.

Five considered neoplastic nodules, of which 1 was probable infiltrating HCC with vascular invasion.

Further clinical information – there was a clinical history of at least 9 months of hepatitis prior to the transplant, and there was no clinical suggestion of Wilson's disease. The copper associated protein in this case is attributable to the chronic disease (in the absence of any other features of biliary disease). Collagen is mature on the van Gieson stain although not positive for elastic on Shikata – some indication of likely duration, not cirrhotic for years.

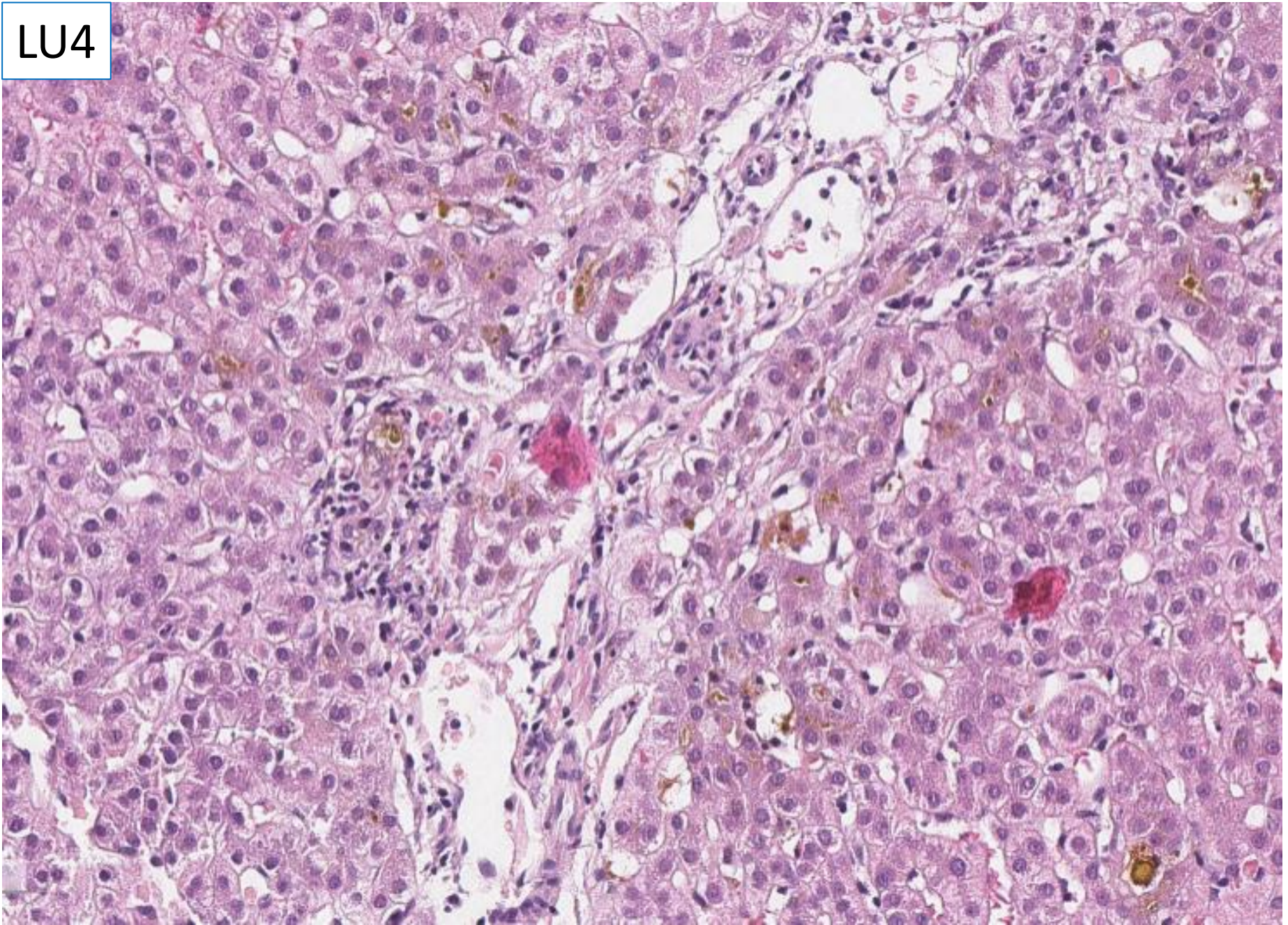
Case LU4 22M

New acute hepatitis ? cause. Persistently elevated ALT 330 and bilirubin 150 with pruritis/jaundice/headache for several weeks. ? viral ? autoimmune. USS no duct dilatation.

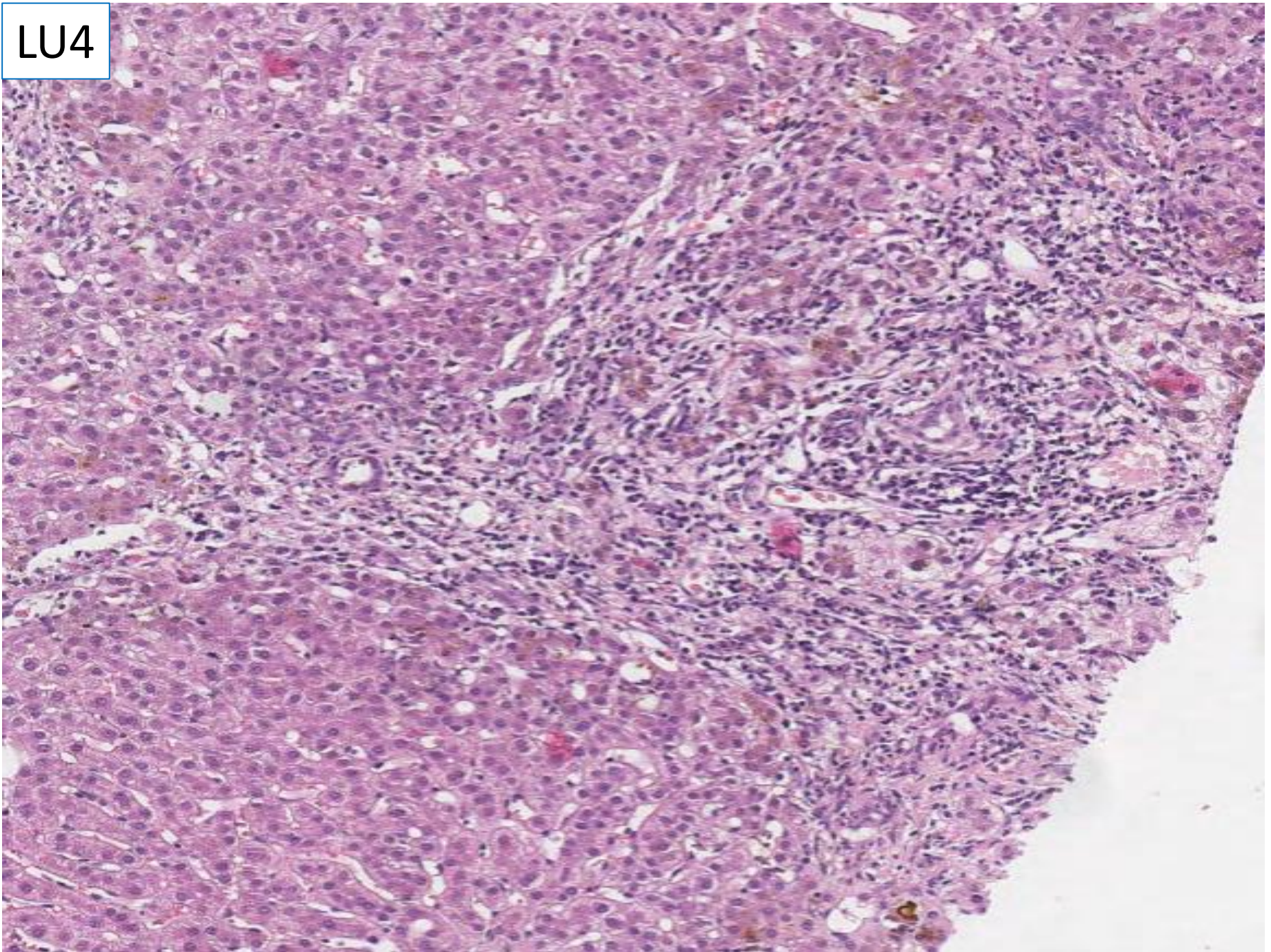
Additional stains: Shikata, CK7, Retic, EVG.



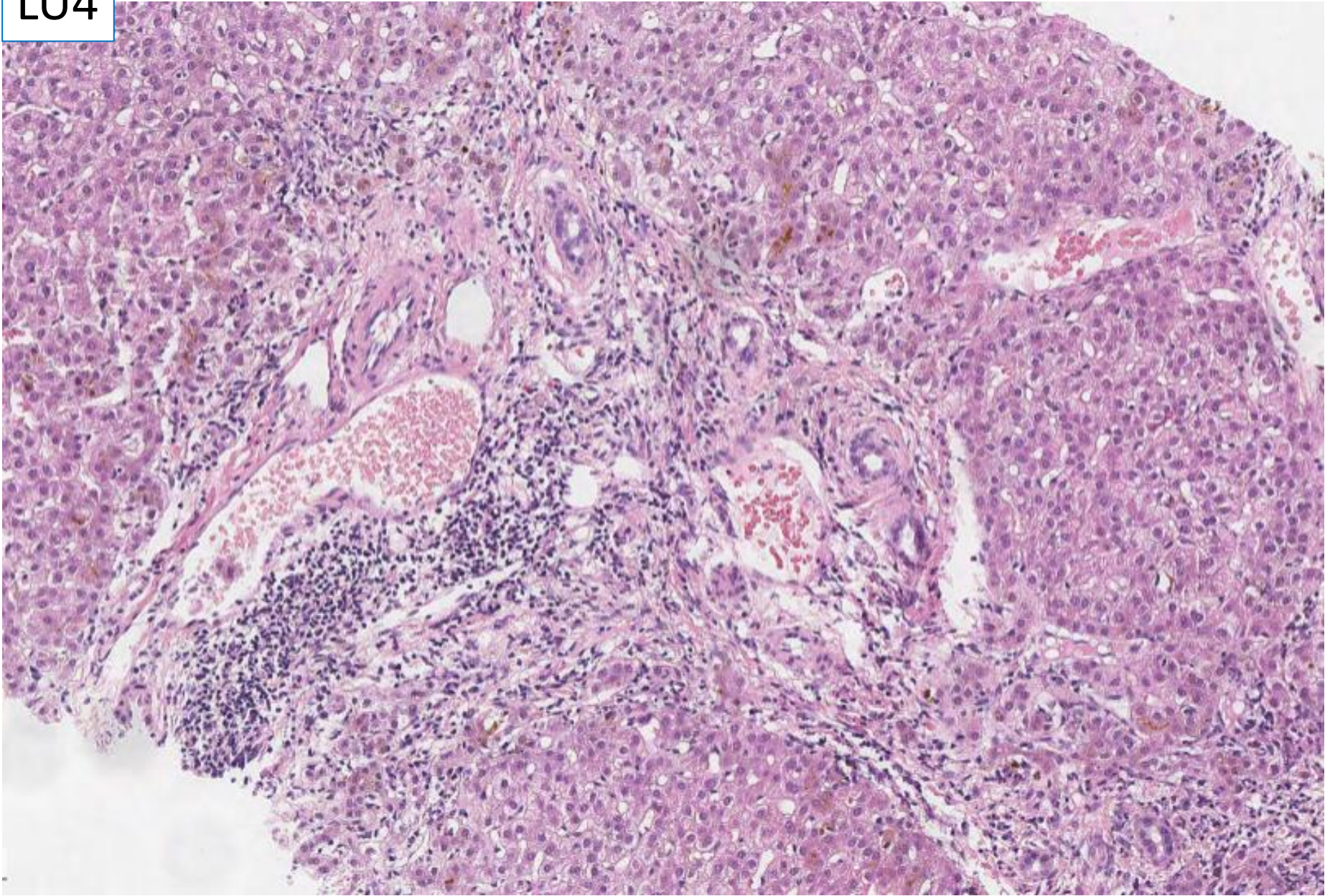
LU4



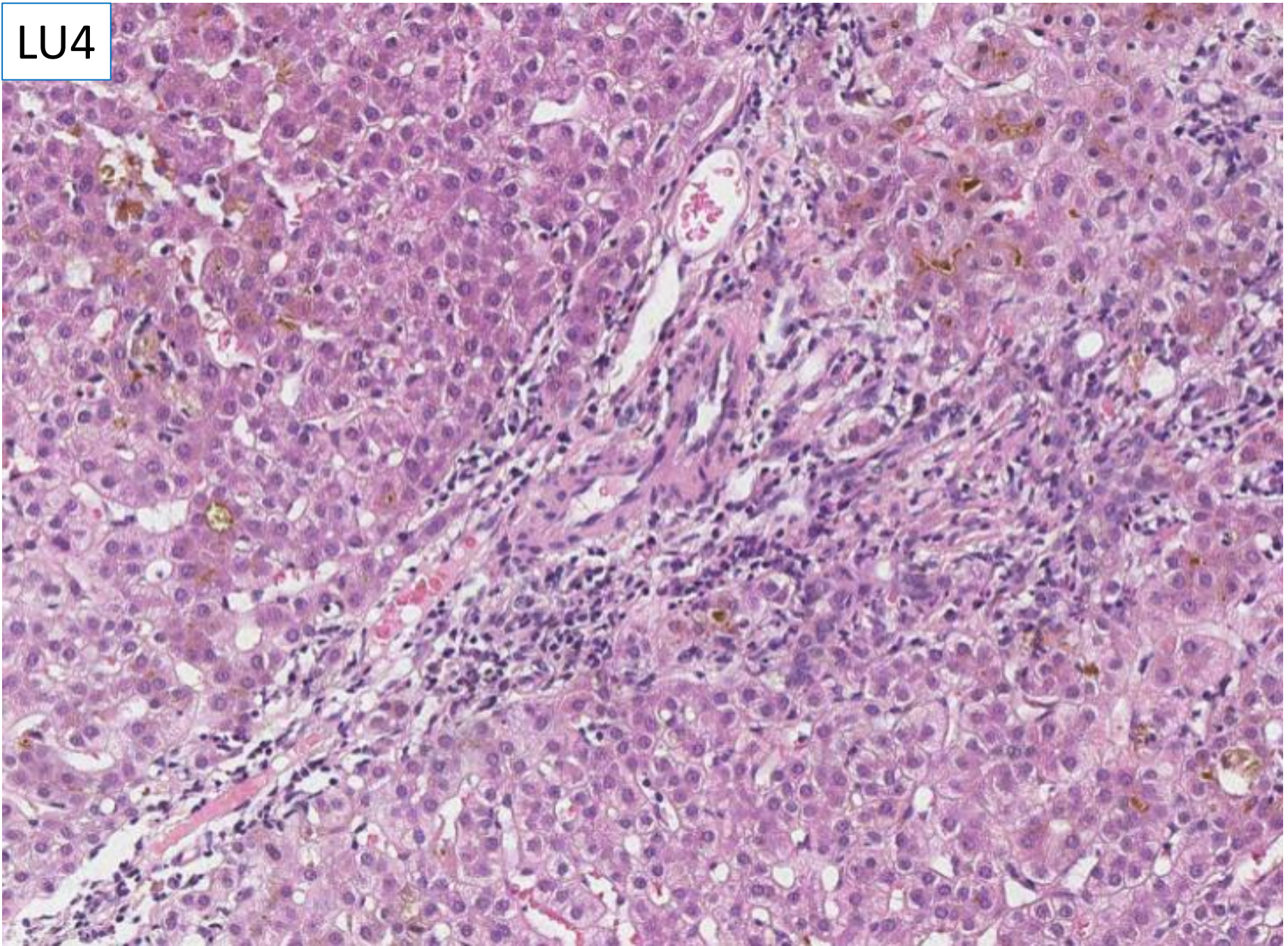
LU4



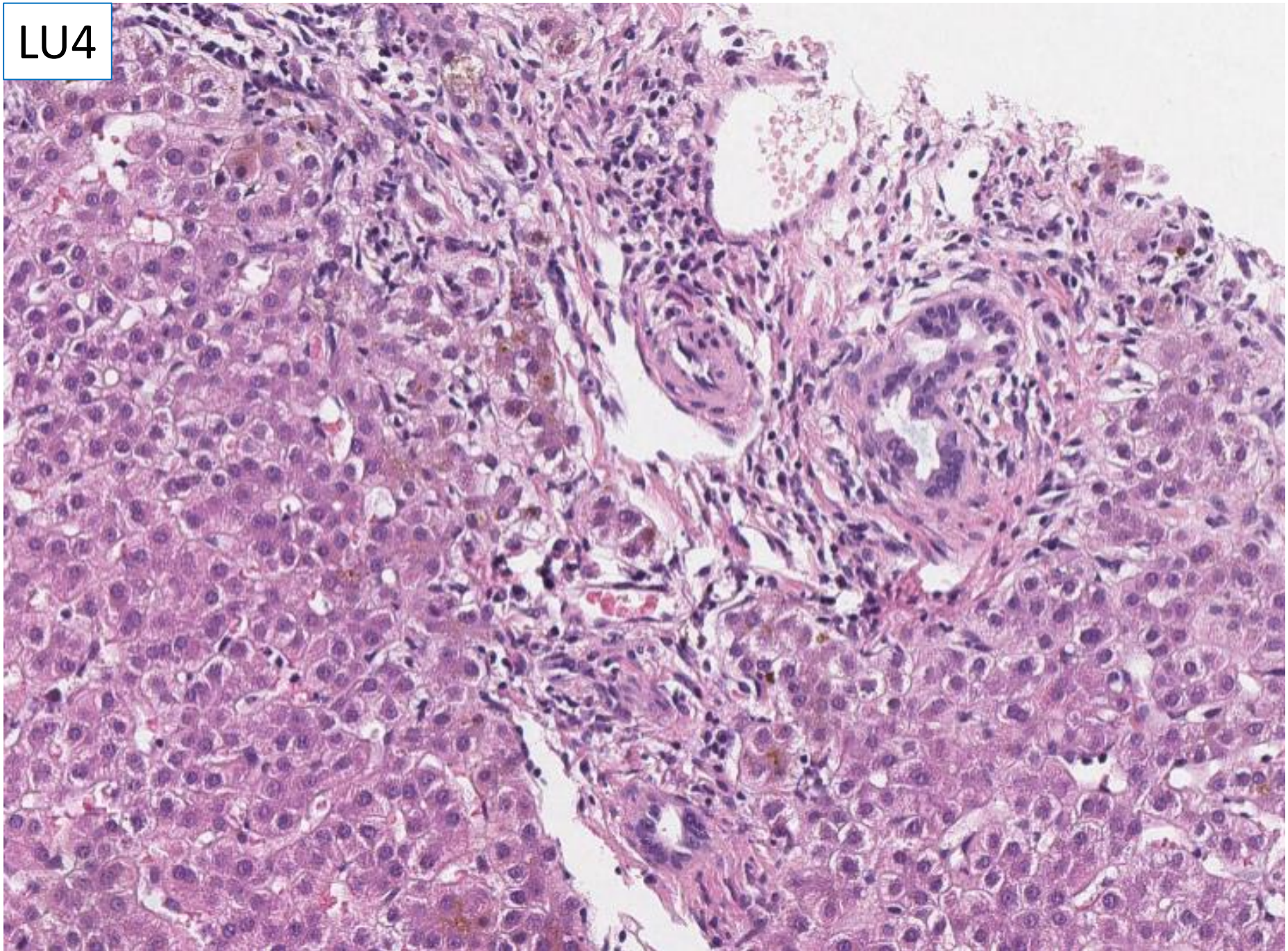
LU4



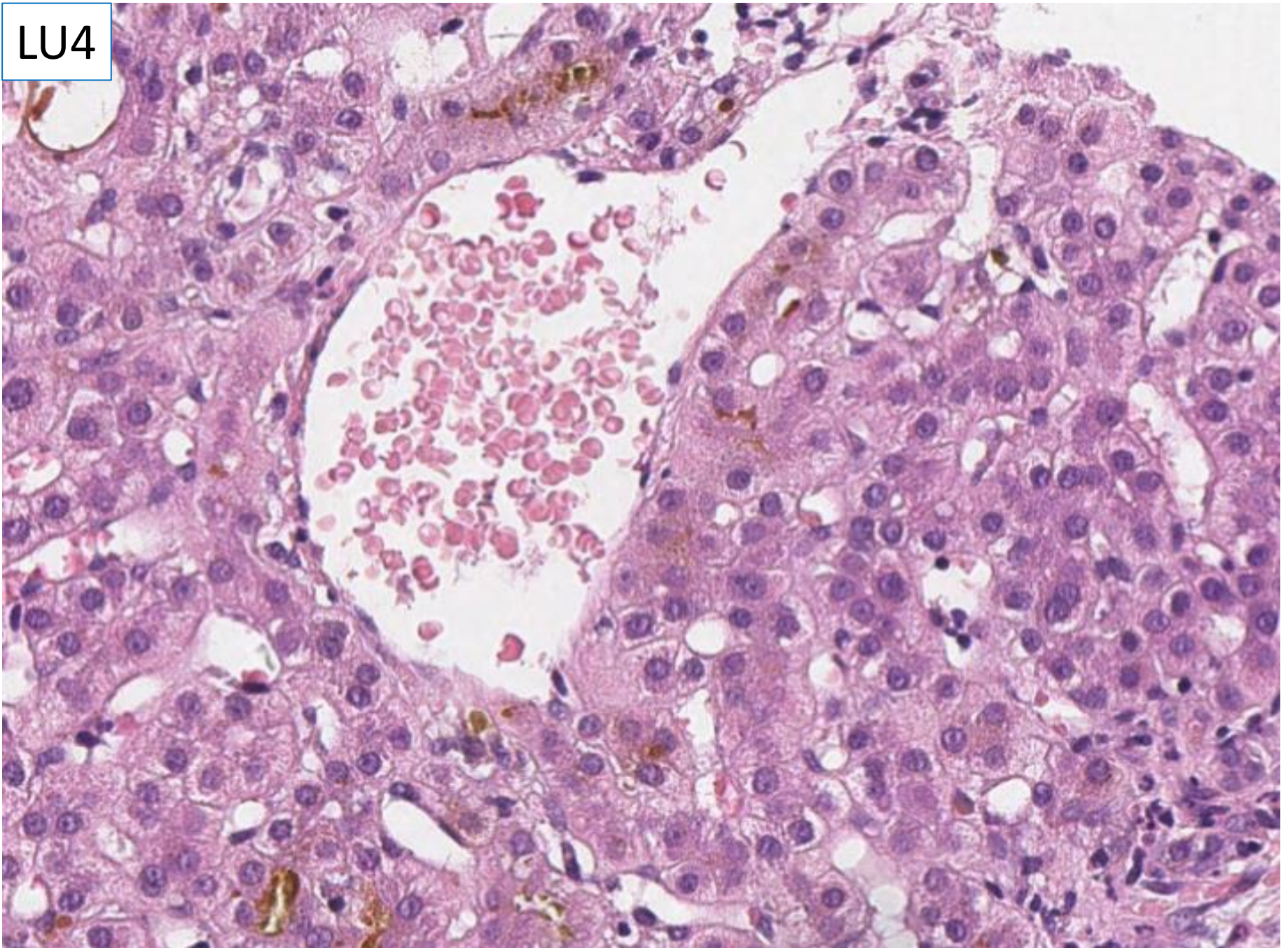
LU4



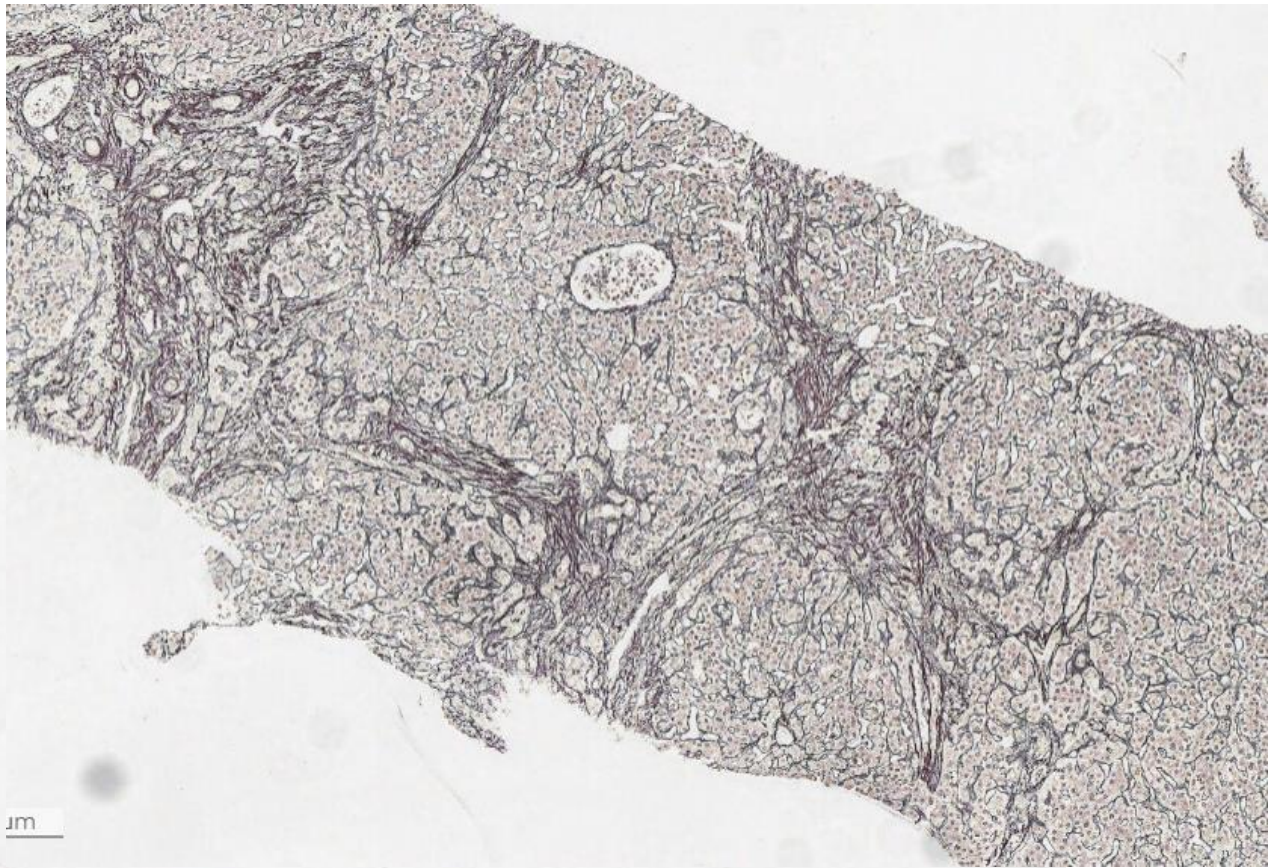
LU4



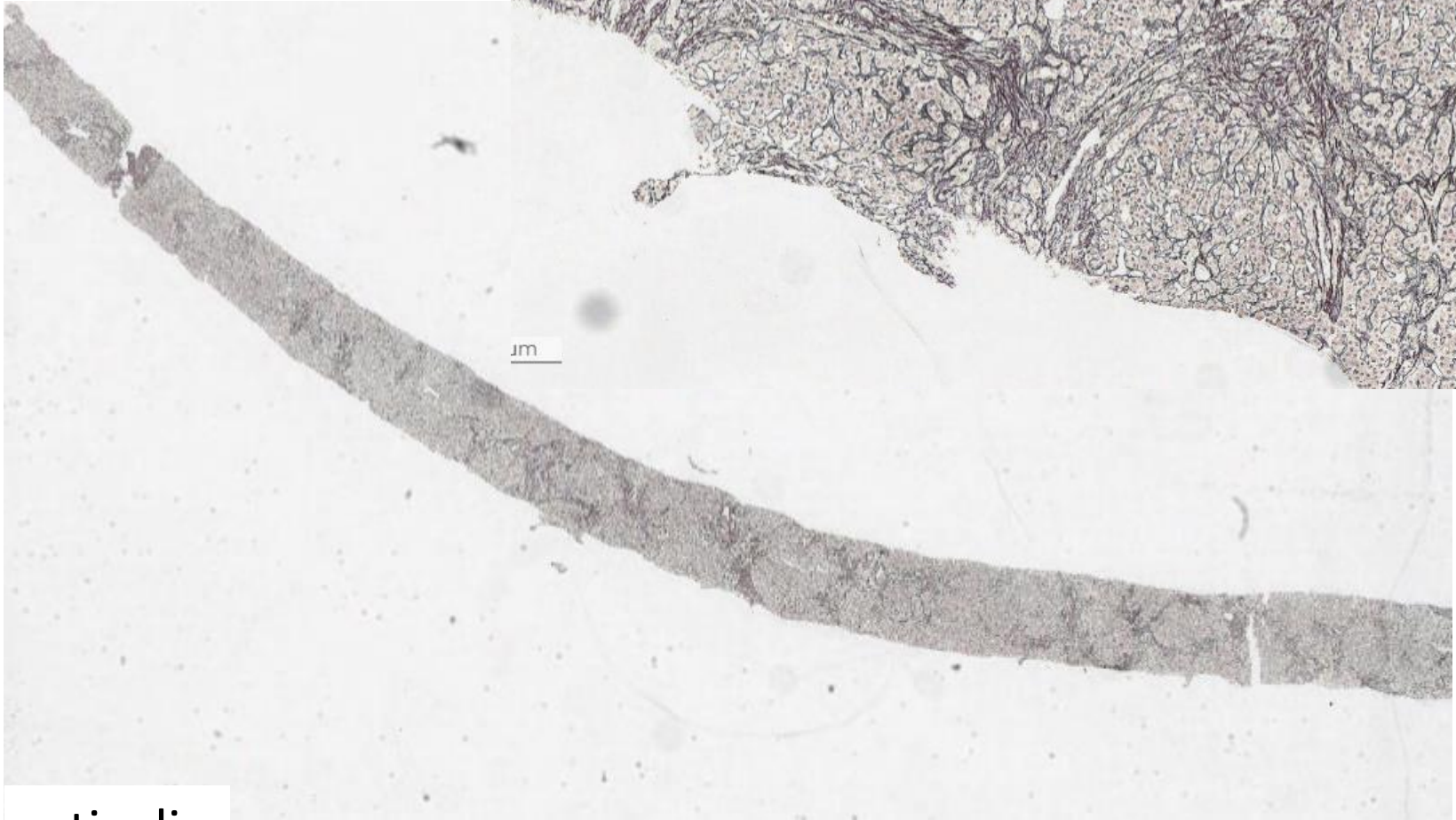
LU4



LU4

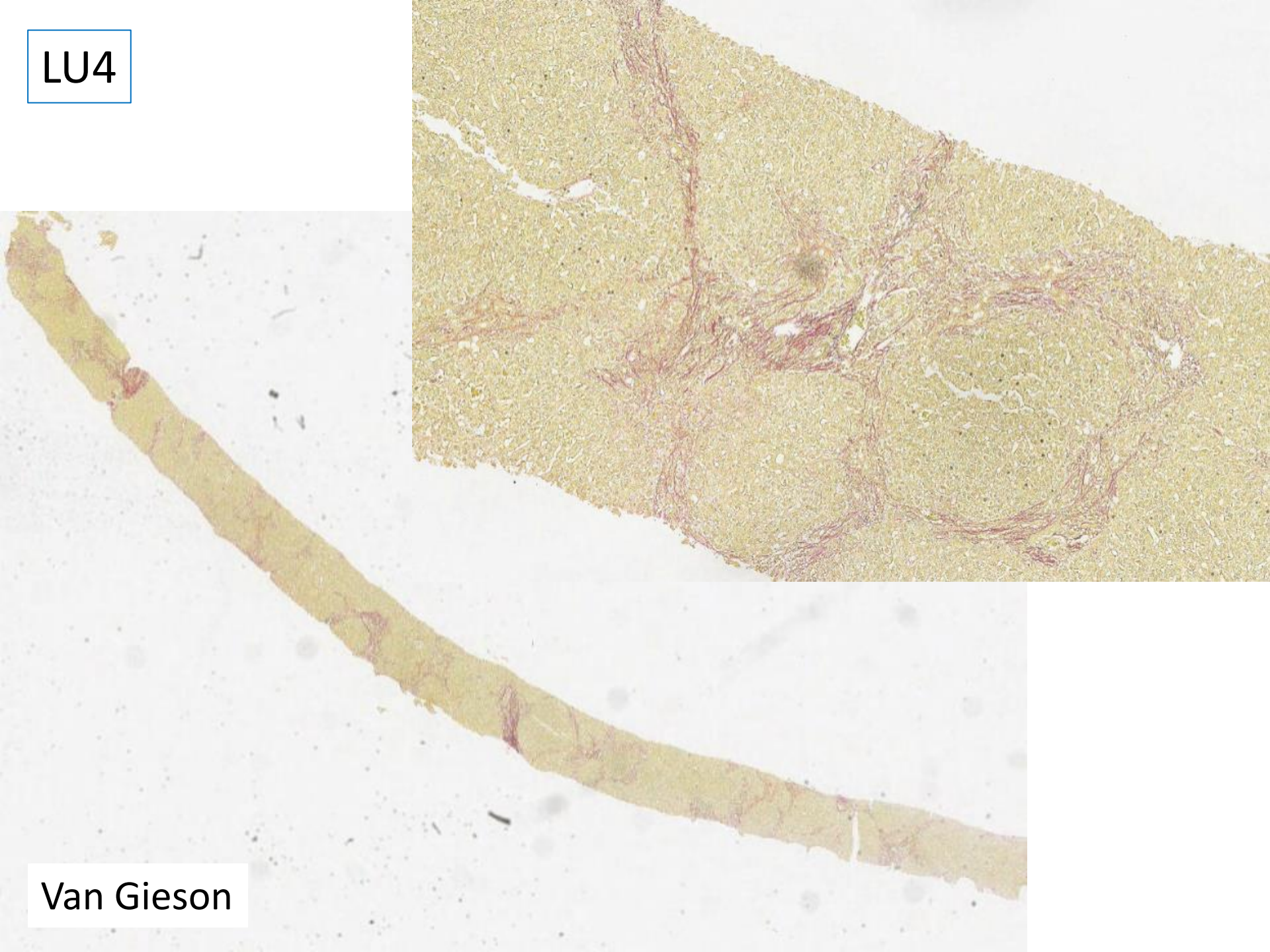


1µm



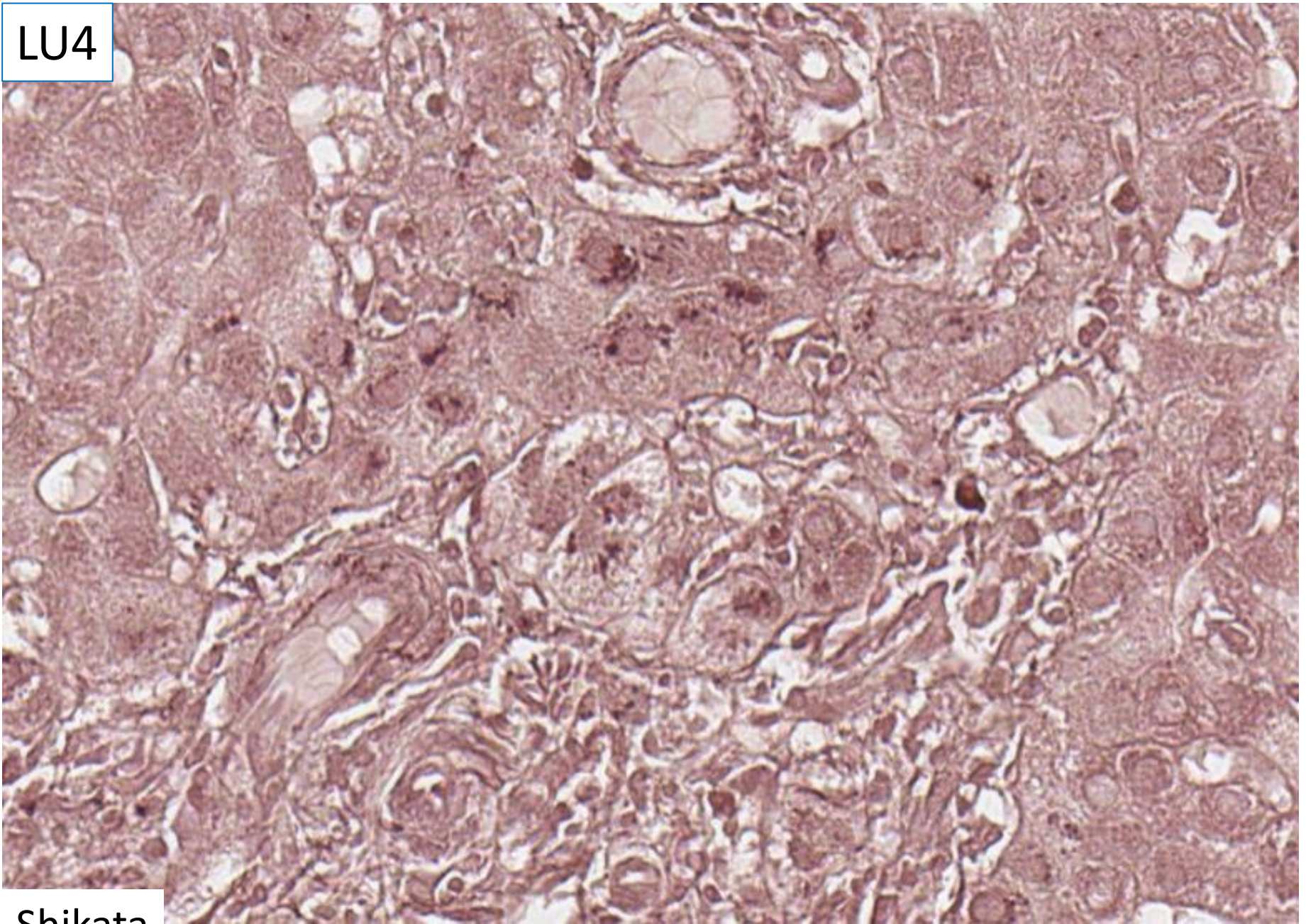
reticulin

LU4



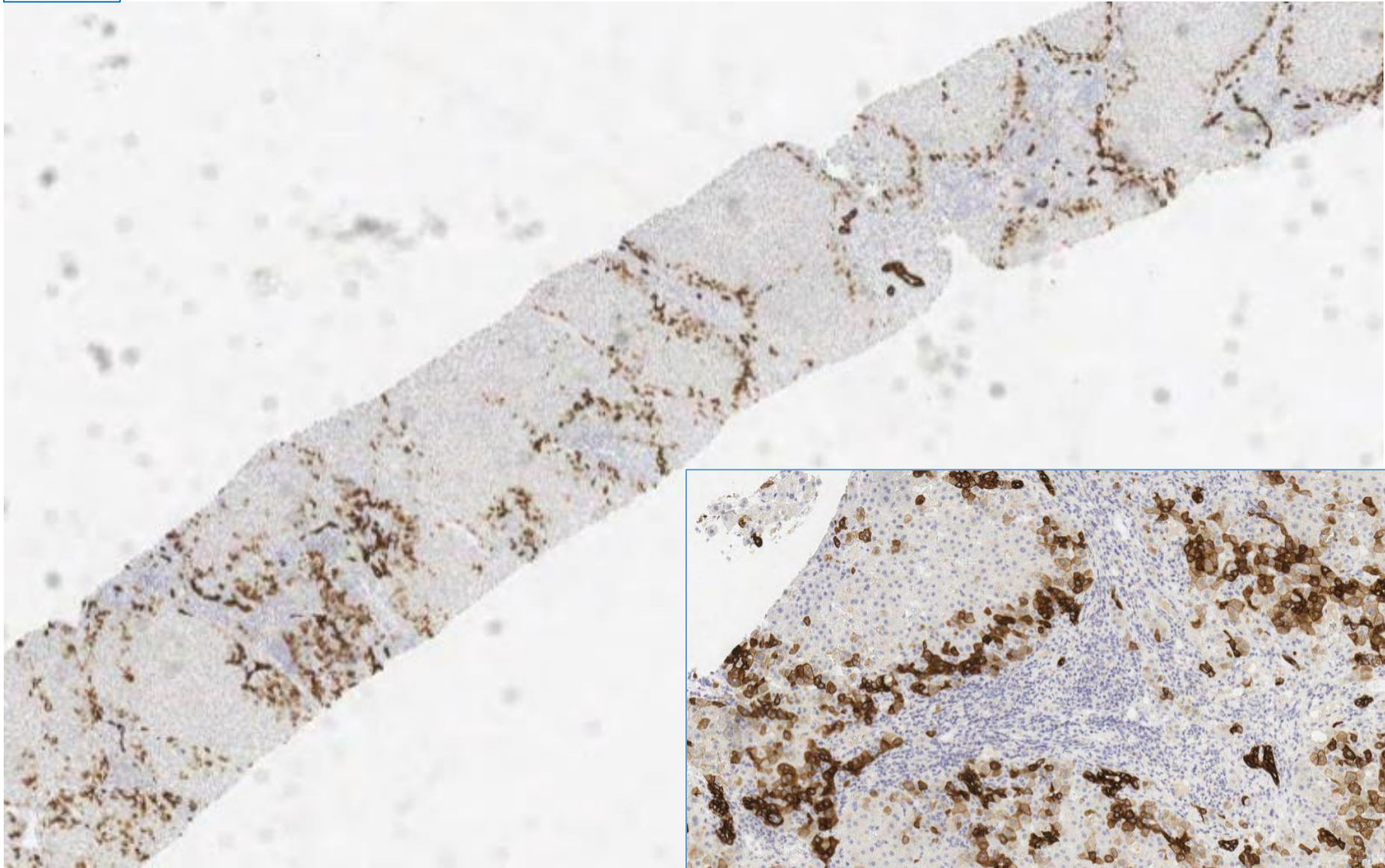
Van Gieson

LU4

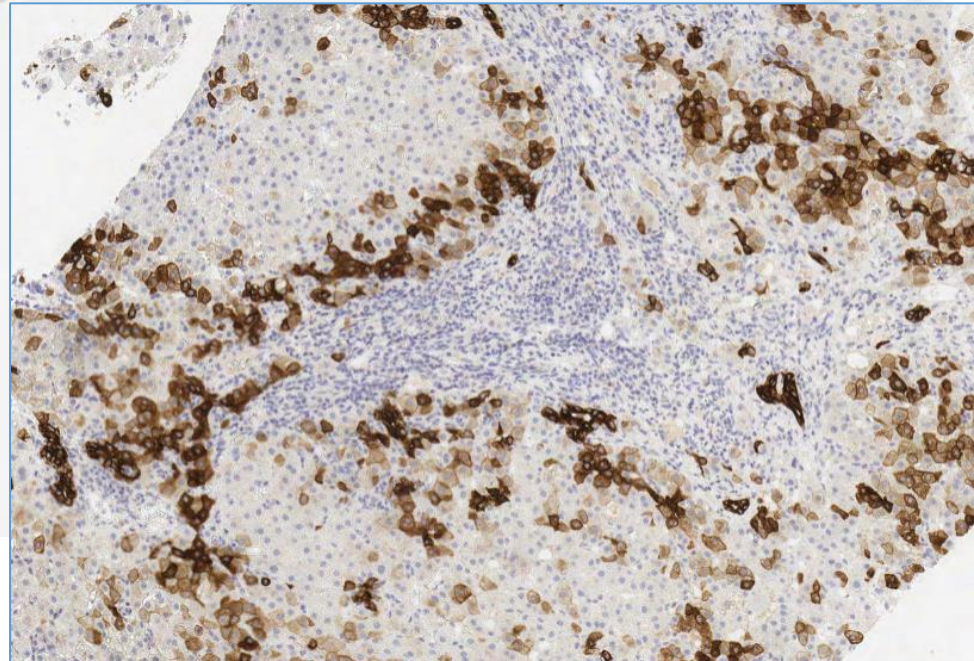


Shikata

LU4



Keratin 7



Case LU4 22M

New acute hepatitis ? cause. Persistently elevated ALT 330 and bilirubin 150 with pruritis/jaundice/headache for several weeks. ? viral ? autoimmune. USS no duct dilatation.

Additional stains: Shikata, CK7, Retic, EVG.

LU4	
A	Chronic biliary features, ? large bile duct obstruction, PSC
B	Acute hepatitis ikw drug
C	Acute hepatitis ikw AIH
D	Chronic hepatitis
E	Wilson disease

Case LU4 22M

New acute hepatitis ? cause. Persistently elevated ALT 330 and bilirubin 150 with pruritis/jaundice/headache for several weeks. ? viral ? autoimmune. USS no duct dilatation.

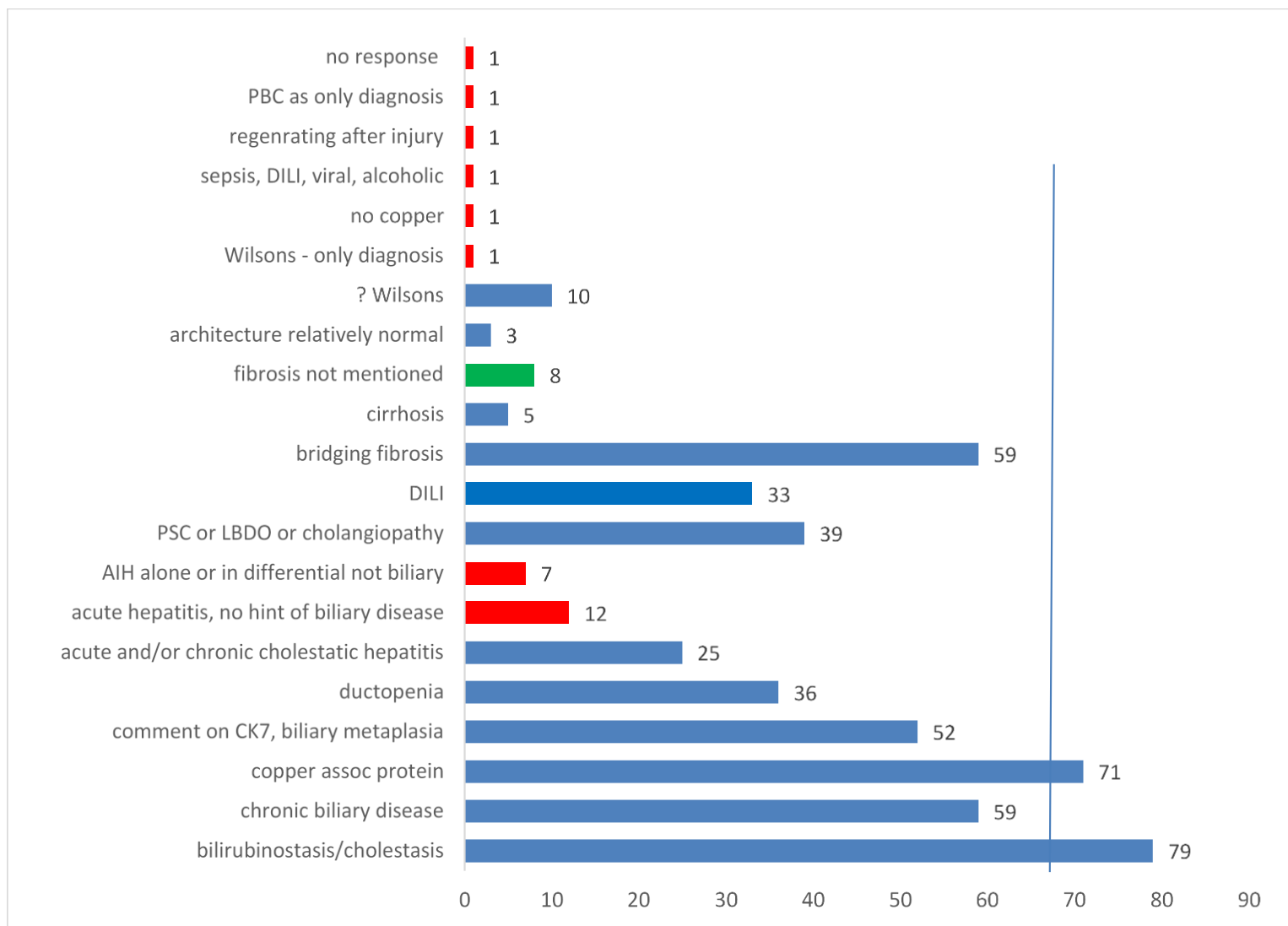
Additional stains: Shikata, CK7, Retic, EVG.

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New acute hepatitis ? cause. Persistently elevated ALT 330 and bilirubin 150 with pruritis/jaundice/headache for several weeks. ? viral ? autoimmune. USS no duct dilatation.

Additional stains: Shikata, CK7, Retic, EVG.



Case LU4 22M

New acute hepatitis ? cause. Persistently elevated ALT 330 and bilirubin 150 with pruritis/jaundice/headache for several weeks. ? viral ? autoimmune. USS no duct dilatation.

Additional stains: Shikata, CK7, Retic, EVG.

Suggested scoring: for 10 points include any response in which a chronic cholestatic/biliary disease is included in the differential diagnosis.

However, responses which didn't clearly indicate this did not reach the 80% cut off – so I think better not to score.

- show of hands at meeting: majority view that this is suitable for scoring

Many commented that this was difficult and required CPC discussion.

Lose 5 marks if – mention cholestasis but don't mention possible chronic biliary disease / cholangiopathy of any sort (12)

? Lose 5 marks if – don't mention architecture (8)

Lose 10 marks (score 0) if – make an entirely different diagnosis e.g. unqualified diagnosis of PBC, Wilson's or acute hepatitis (drugs, viral AIH) or no response. (5)

Case LU4 22M

New acute hepatitis ? cause. Persistently elevated ALT 330 and bilirubin 150 with pruritis/jaundice/headache for several weeks. ? viral ? autoimmune. USS no duct dilatation.

Additional stains: Shikata, CK7, Retic, EVG.

Observations/potential learning points, suitable for masterclass?

– the assessment of a biopsy that shows likely features of chronic biliary tract disease.

Additional comments from Stefan Hubscher – difficult case:

1. Because of history – despite acute hepatitis presentation, the striking thing is the lack of inflammation
2. The disconnect between portal areas and lobules – chronic cholestatic disease with copper associated protein but also severe bilirubinostasis.

Most people with chronic cholestatic disease don't develop jaundice until disease is very advanced – if severe bilirubinostasis either severe fibrosis or advanced vanishing bile ducts – neither in this case – so consider either a dominant stricture (not here) or other cause like DILI.

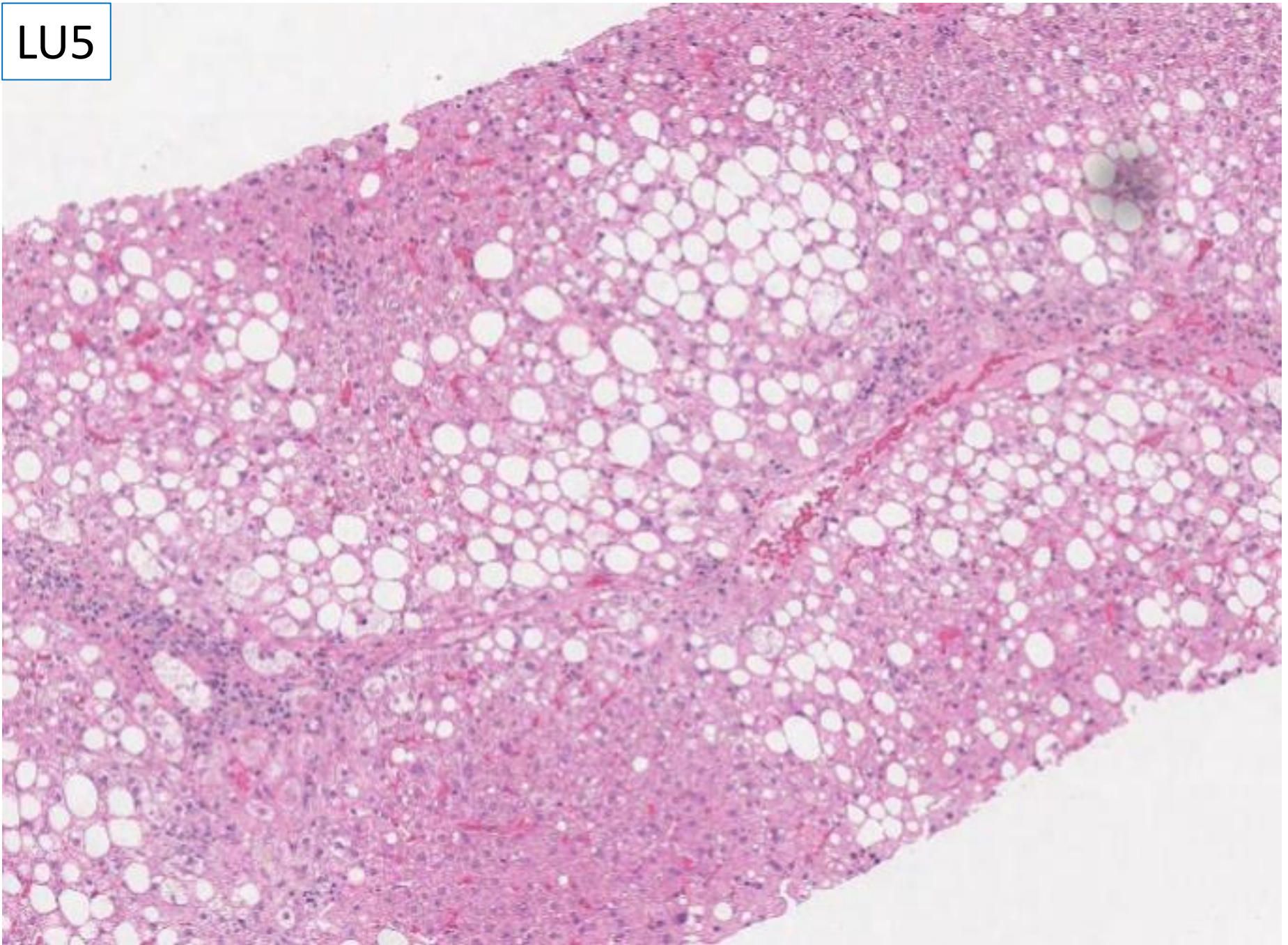
Follow up information for this case: biliary imaging showed irregular stricturing of left and right bile ducts – diagnosis of PSC. No dominant stricture, and appearances unchanged 6 months later. The alk phos was between 500 and 600 at the time of the biopsy. Subsequently pANCA +ve, slightly raised IgG (18), no other autoantibodies. Has not yet had colonoscopy.

Case LU5 55M

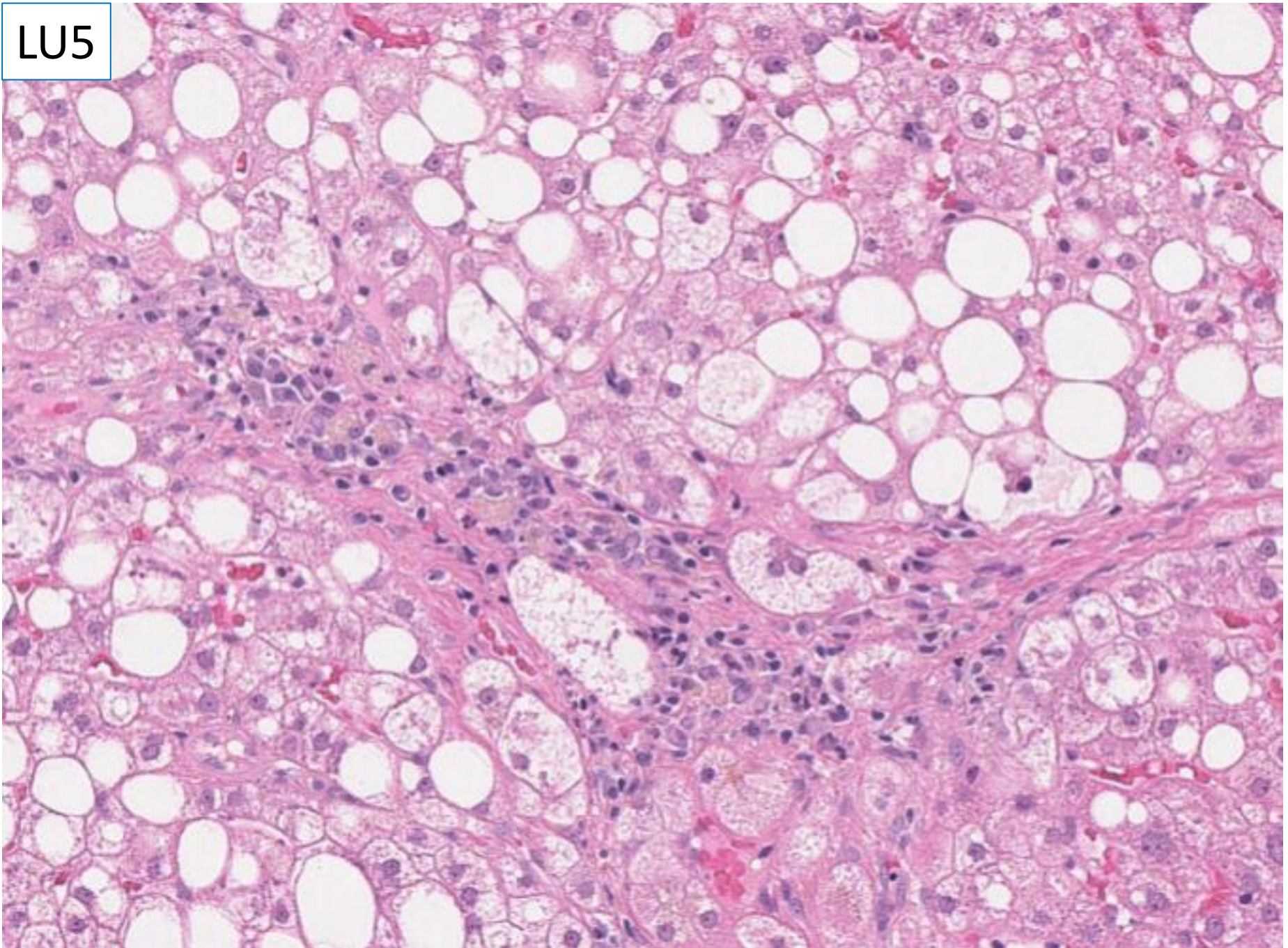
NAFLD. Fibroscan 12. Moderate fibrosis? on Humira for rheumatoid arthritis.
Additional stains: HVG.



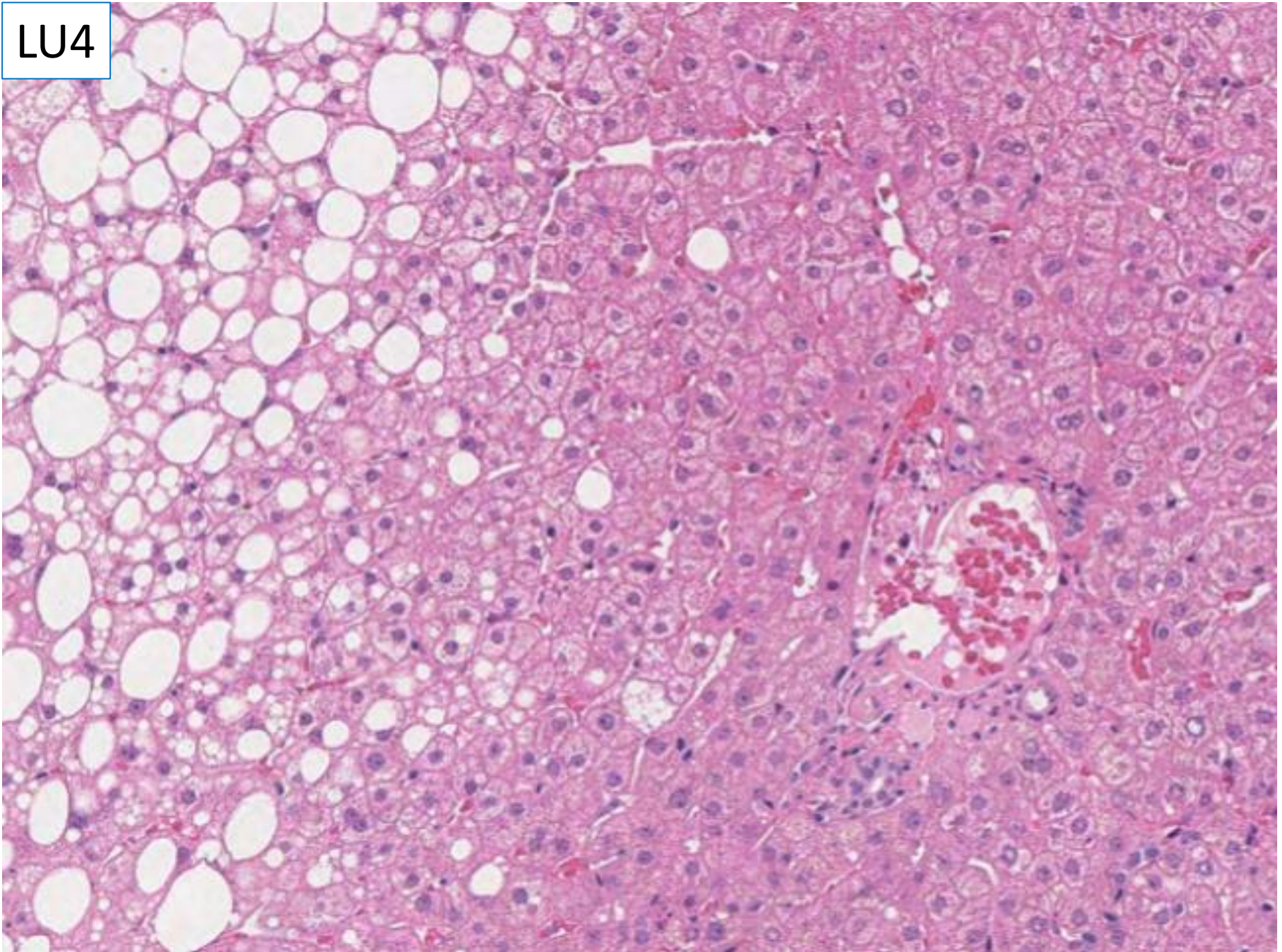
LU5



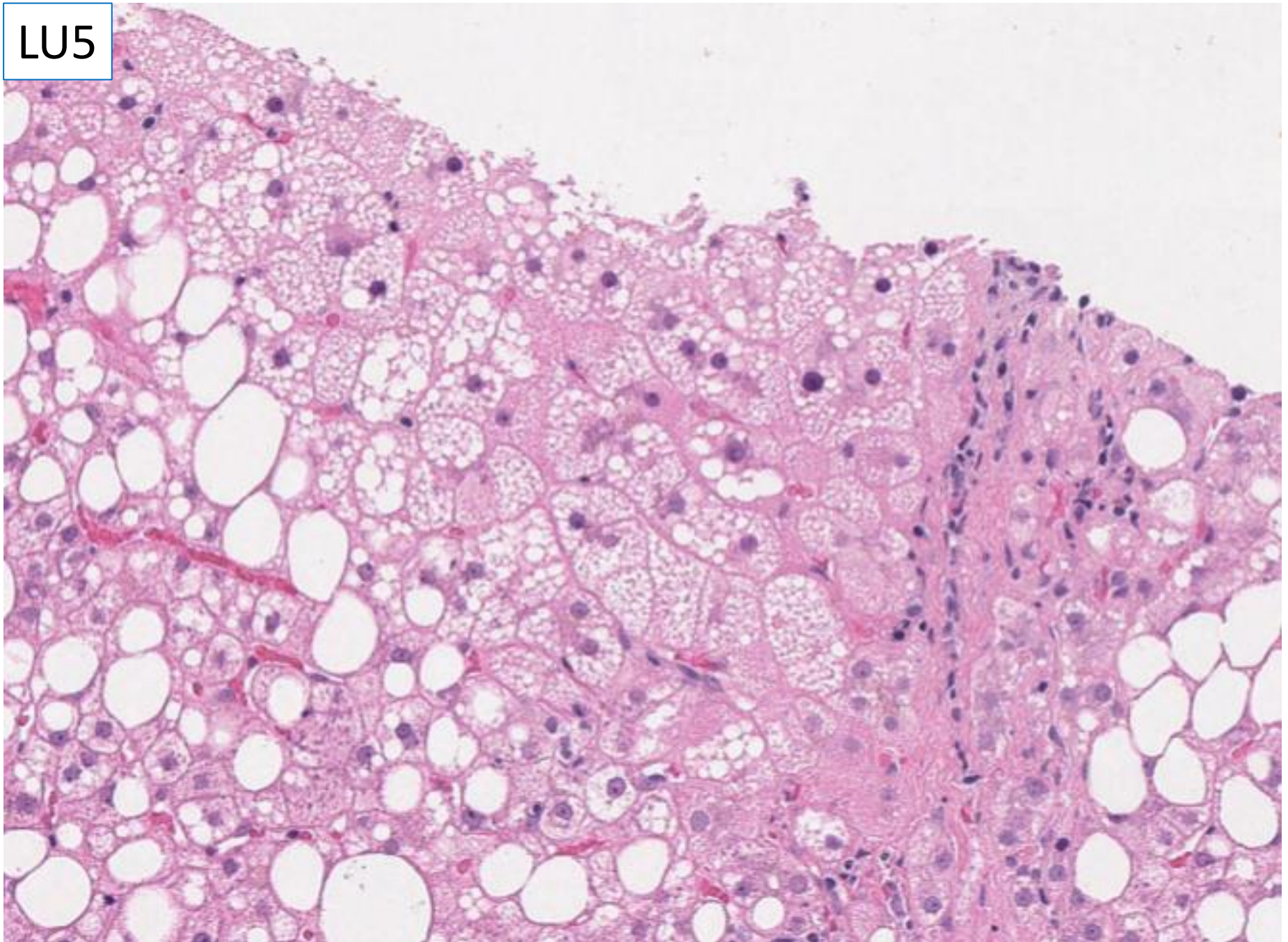
LU5



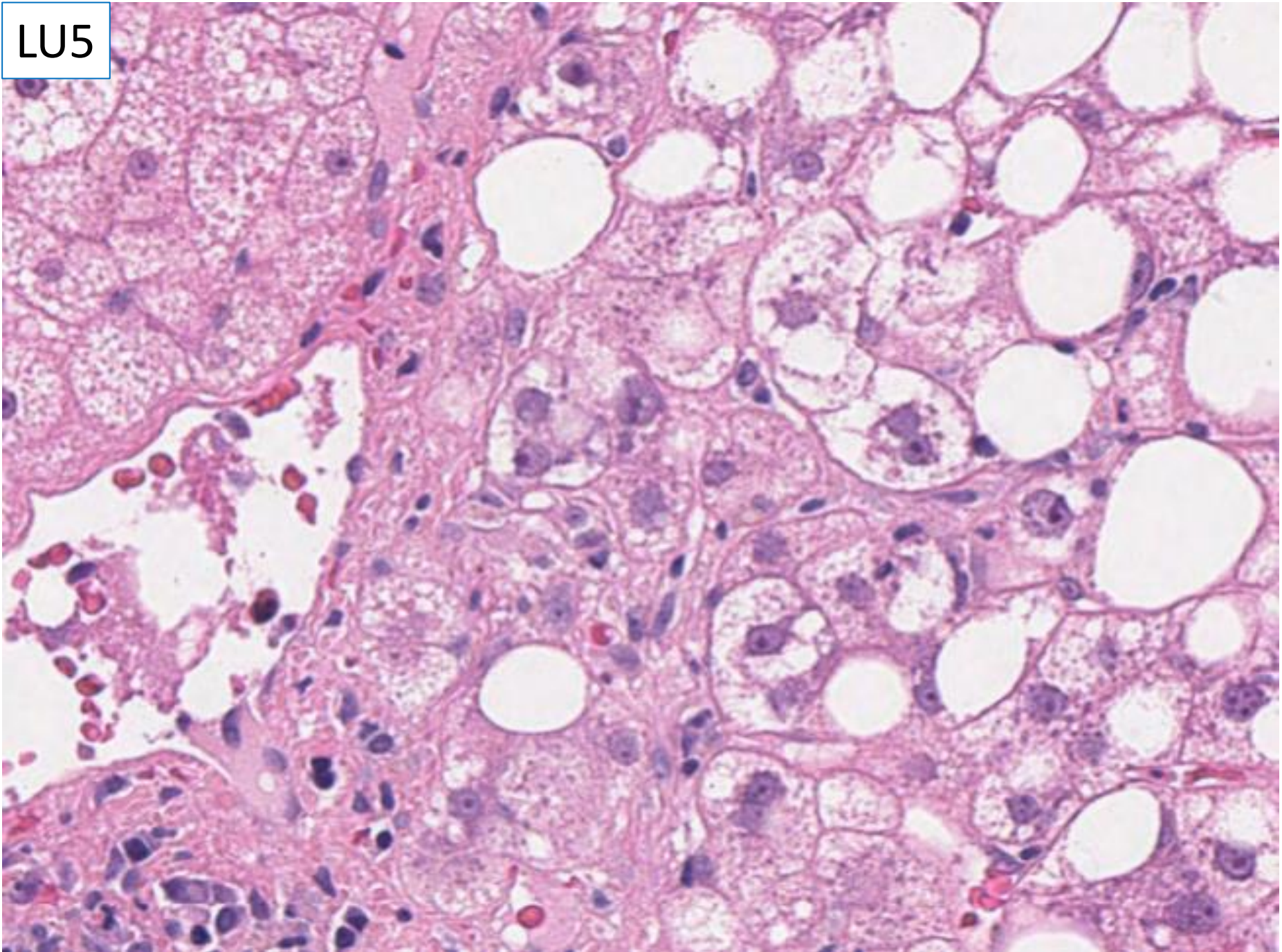
LU4



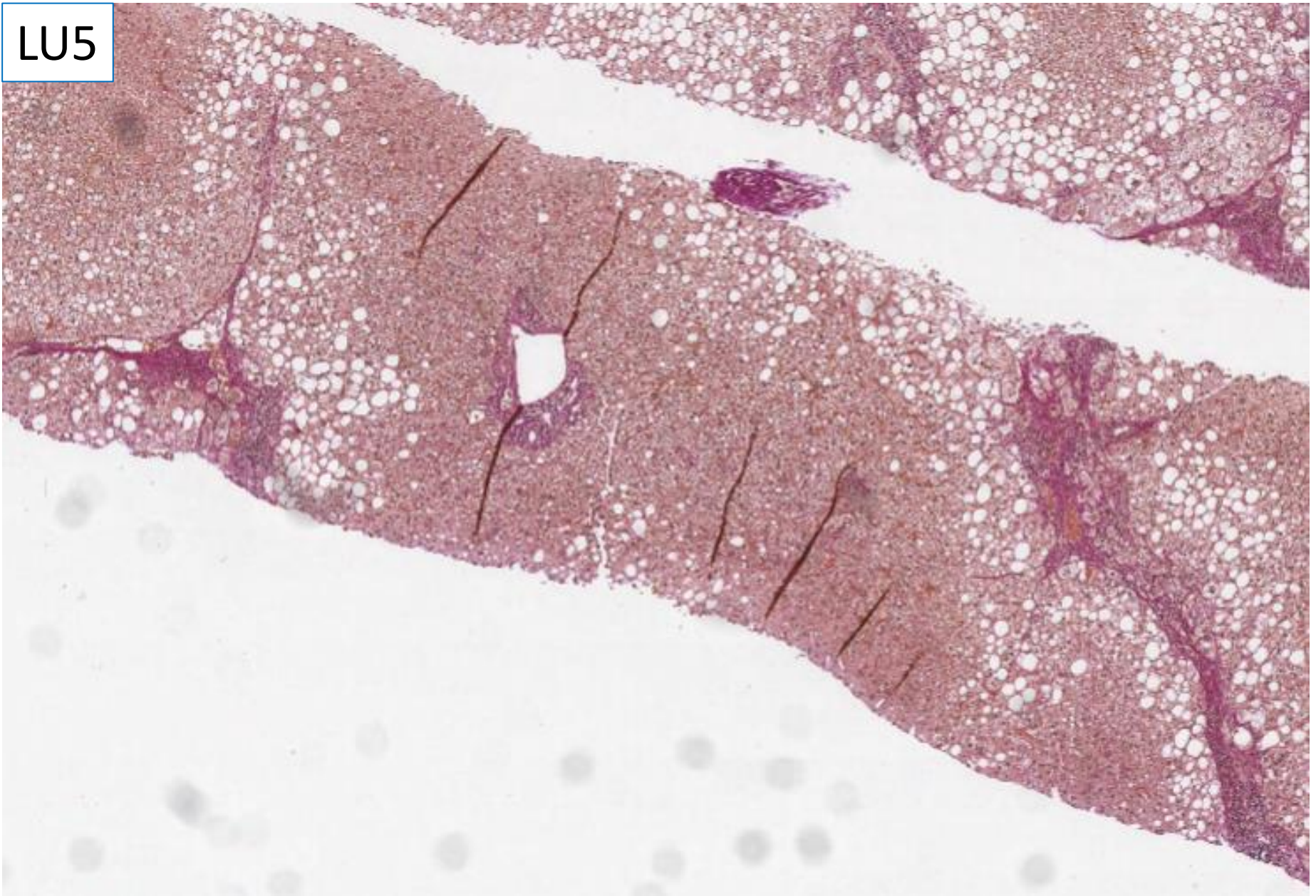
LU5



LU5

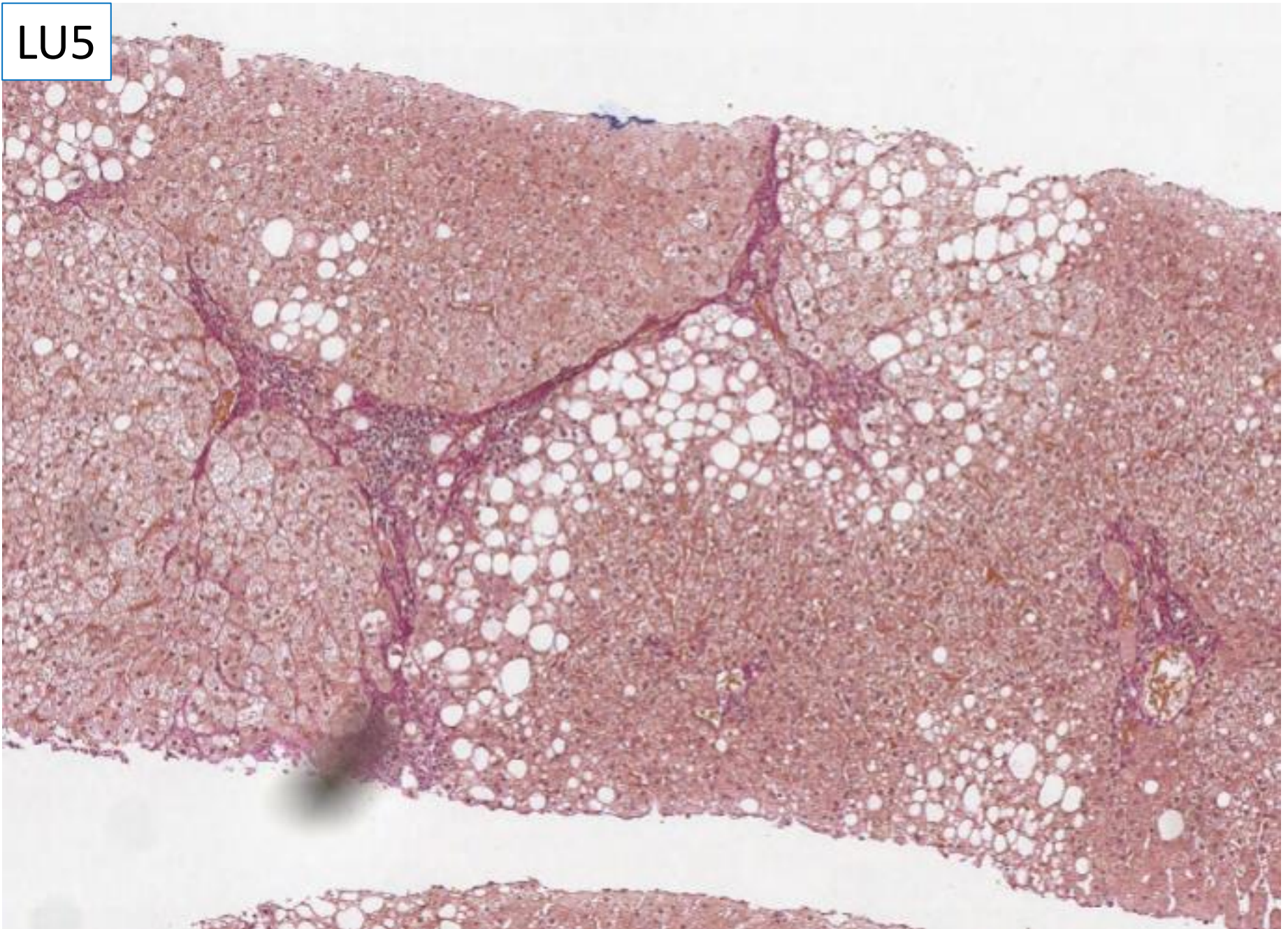


LU5



HVG

LU5



Case LU5 55M

NAFLD. Fibroscan 12. Moderate fibrosis? on Humira for rheumatoid arthritis.
Additional stains: HVG.

LU5	
A	DILI with background steatosis
B	steatohepatitis
C	steatosis
D	Rheumatoid related liver disease
E	Biliary disease with background steatosis

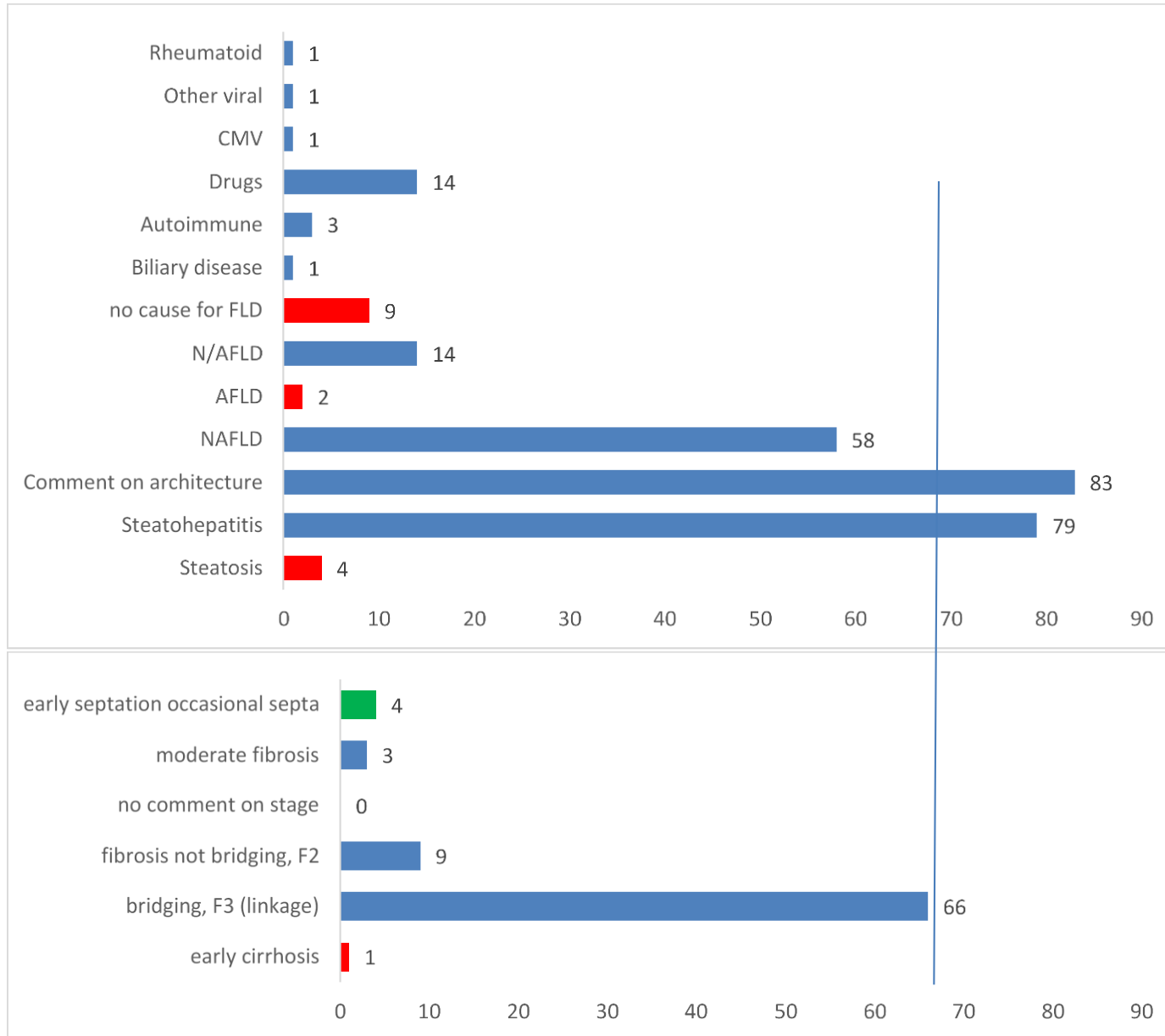
Case LU5 55M

NAFLD. Fibroscan 12. Moderate fibrosis? on Humira for rheumatoid arthritis.
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E	Biliary disease with background steatosis

Case LU5 55M

NAFLD. Fibrosan 12. Moderate fibrosis? on Humira for rheumatoid arthritis.
Additional stains: HVG.



Case LU5 55M

NAFLD. Fibroscan 12. Moderate fibrosis? on Humira for rheumatoid arthritis.
Additional stains: HVG.

Consensus complete responses would include – Steatohepatitis with comment on architecture and comment on NAFLD, AFLD or both as likely aetiology.

After recording actual fibrosis stage – 69 indicated recorded either bridging fibrosis or ‘moderate fibrosis’ – only 1 indicated early cirrhosis. Also 4 use terminology ‘early septation; or ‘occasional septa’ – do we use septa to imply bridging fibrosis or not?

Suggested scoring: for 10 points include – good description of steatohepatitis and consistent with the clinical context of NAFLD

Lose 5 marks if – describe steatosis but not steatohepatitis (4)

Lose 5 marks if – alcohol or drugs as the only cause without including NAFLD (2)

Lose 5 marks if – no mention of NAFLD as an aetiology (9)

Lose 5 marks if – no comment on architecture – but all included a comment.

Lose 5 marks for cirrhosis (1).

? lose 5 marks for early septation or occasional septa (4) – *not marked down but discussion on terminology – see below*

Lose 10 marks (score 0) if – make an inappropriate diagnosis as favoured option e.g. viral hepatitis, autoimmune hepatitis – but all had included these as additional diagnoses to fatty liver disease.

Case LU5 55M

NAFLD. Fibroscan 12. Moderate fibrosis? on Humira for rheumatoid arthritis.
Additional stains: HVG.

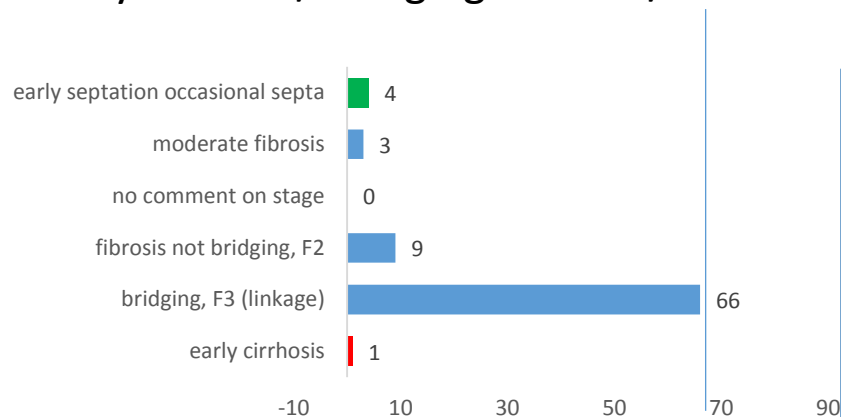
Observations/potential learning points,

– assessing steatohepatitis activity

Additional collation for disease stage – all responses included some comment on stage.

If add 'moderate fibrosis' to bridging, F3 fibrosis, then there is a consensus for this result

What do responses 'early septation' or 'occasional septa' intend to convey to clinicians – can we converge on a staging description that is early fibrosis / bridging fibrosis / cirrhosis or advanced fibrosis?



Discussion: Some use 'septa' for portal-portal linkage, but 'bridging' for portal-central bridges. Terminology may be drifting with time.

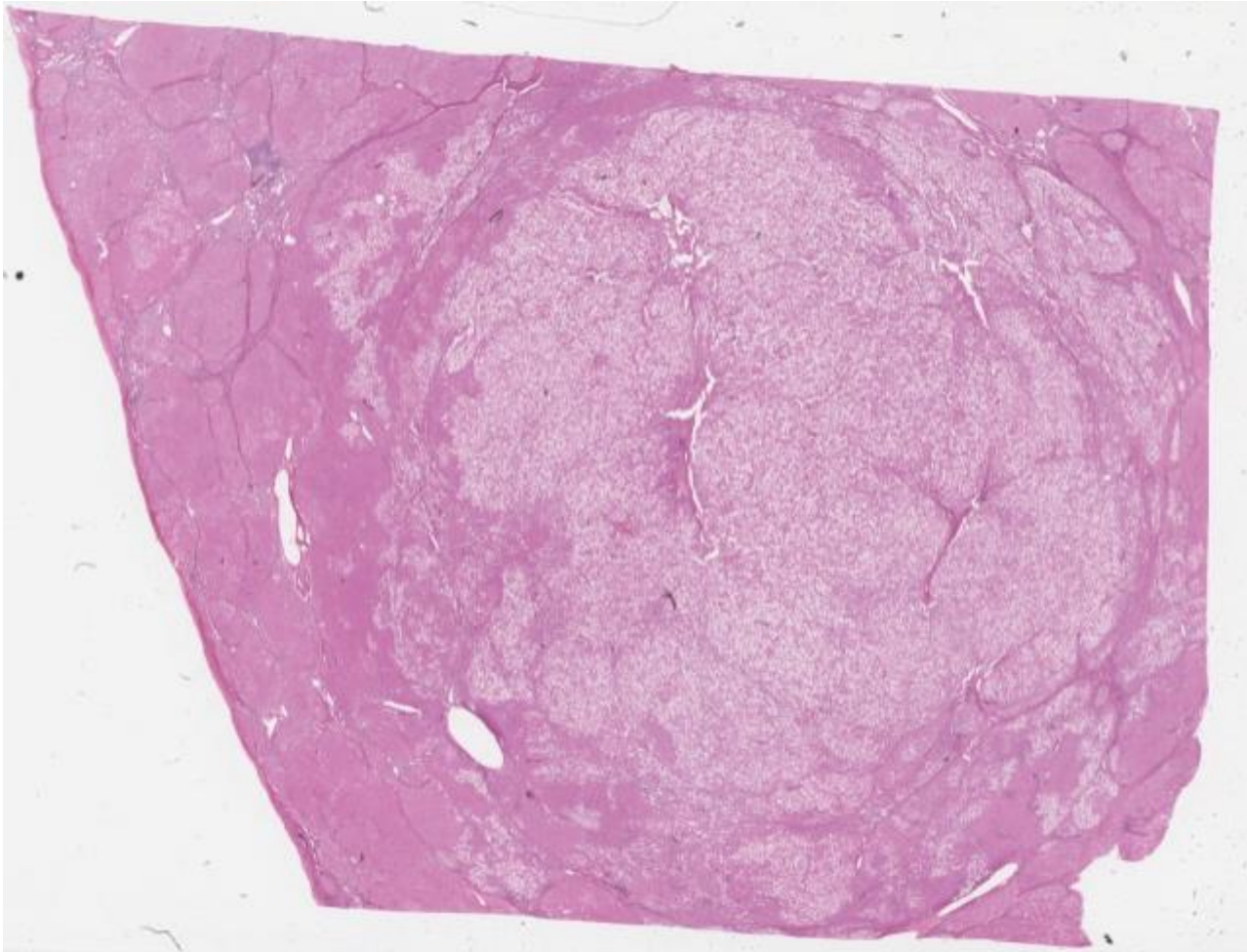
The agreement on stage seems to have improved over time compared with previous EQA circulations. We should have a session on staging at next year's meeting.

Case LU6 80F

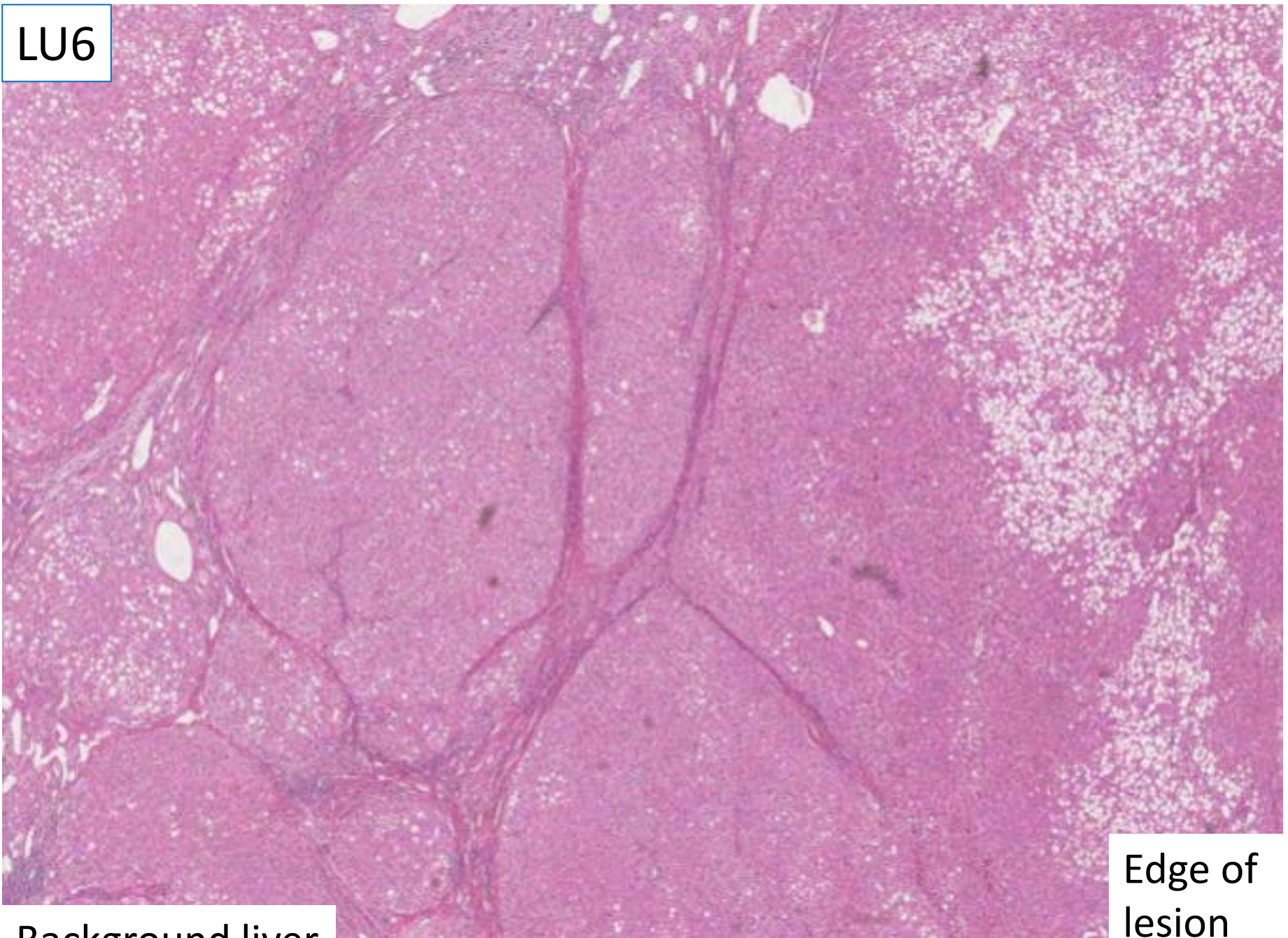
Liver cirrhosis, mass lesion in segment VIII/IVa, ?HCC.

Specimen: Segment VIII/IVa segmentectomy, 70 x 68 x 35 mm containing a mass lesion 22 x 22 x 18mm.

No additional stains.



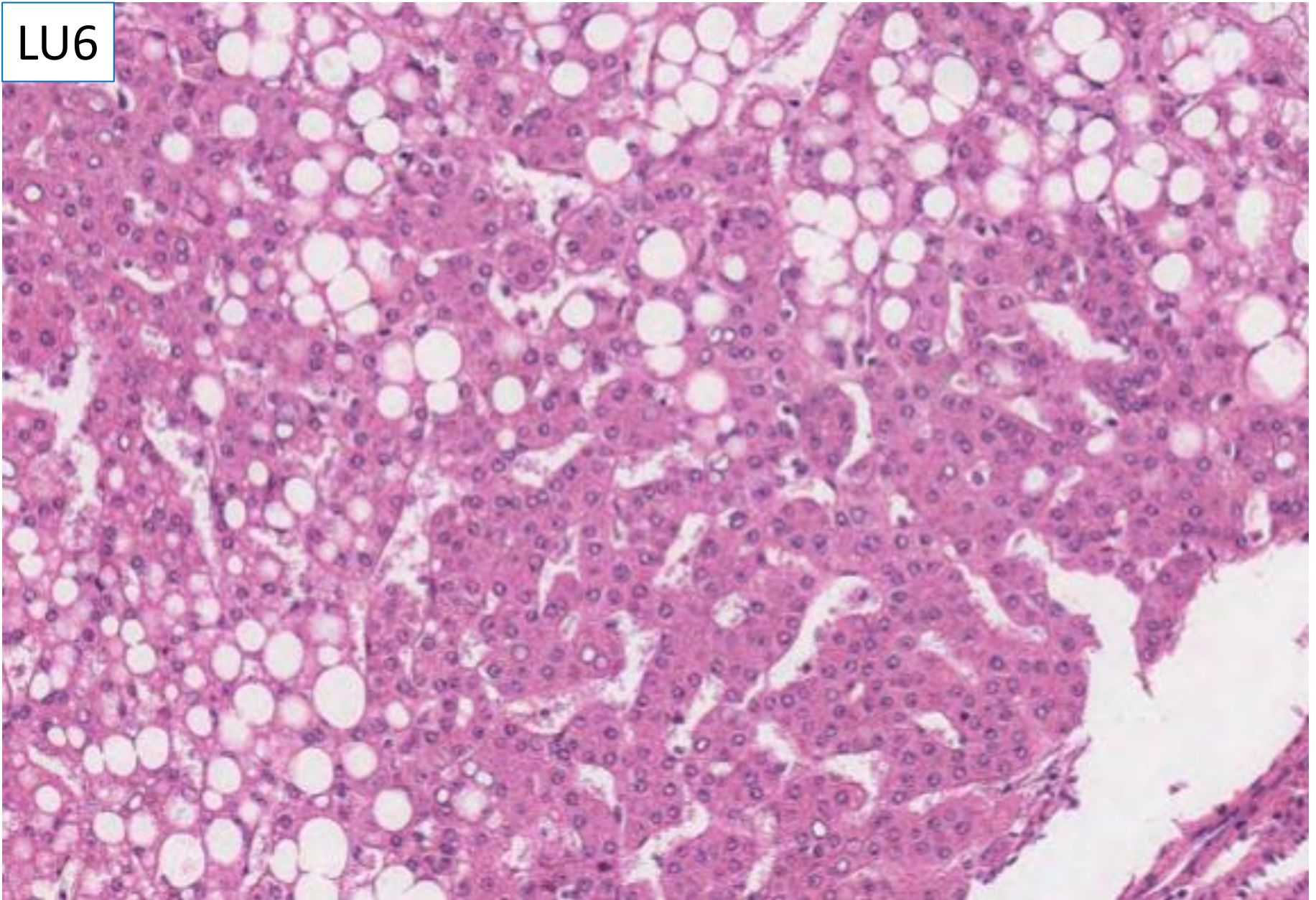
LU6



Background liver

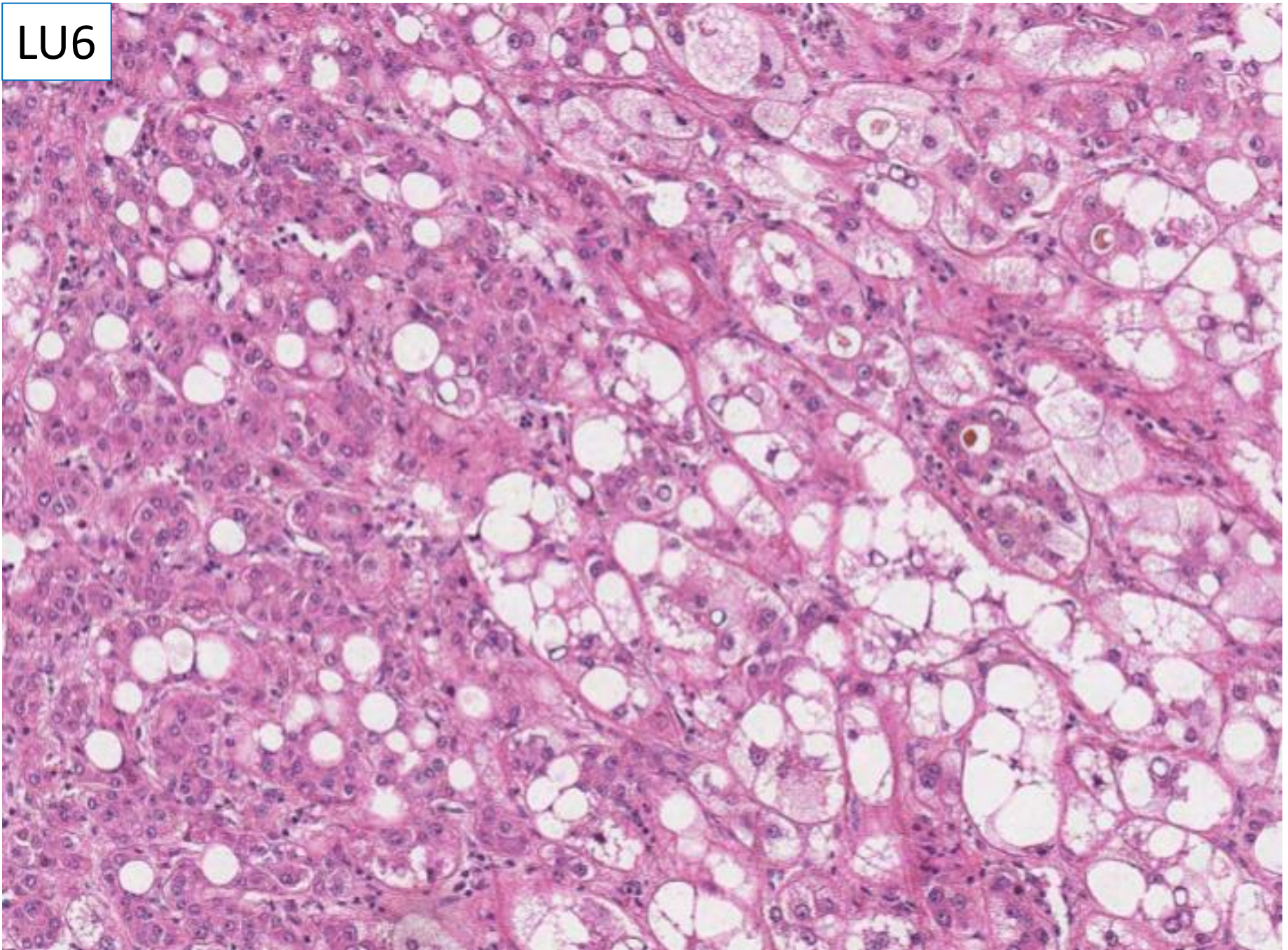
Edge of
lesion

LU6

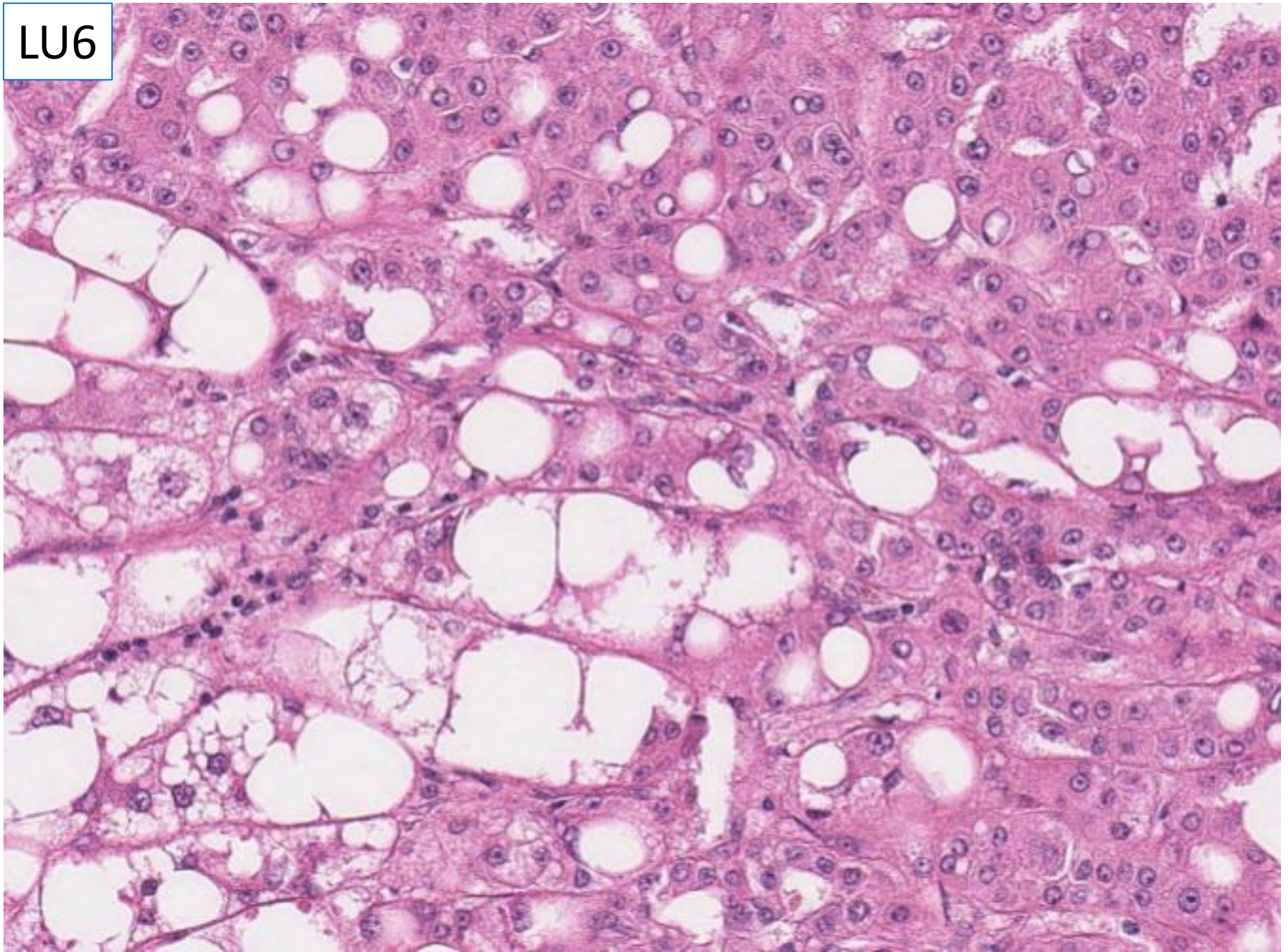


Inside lesion

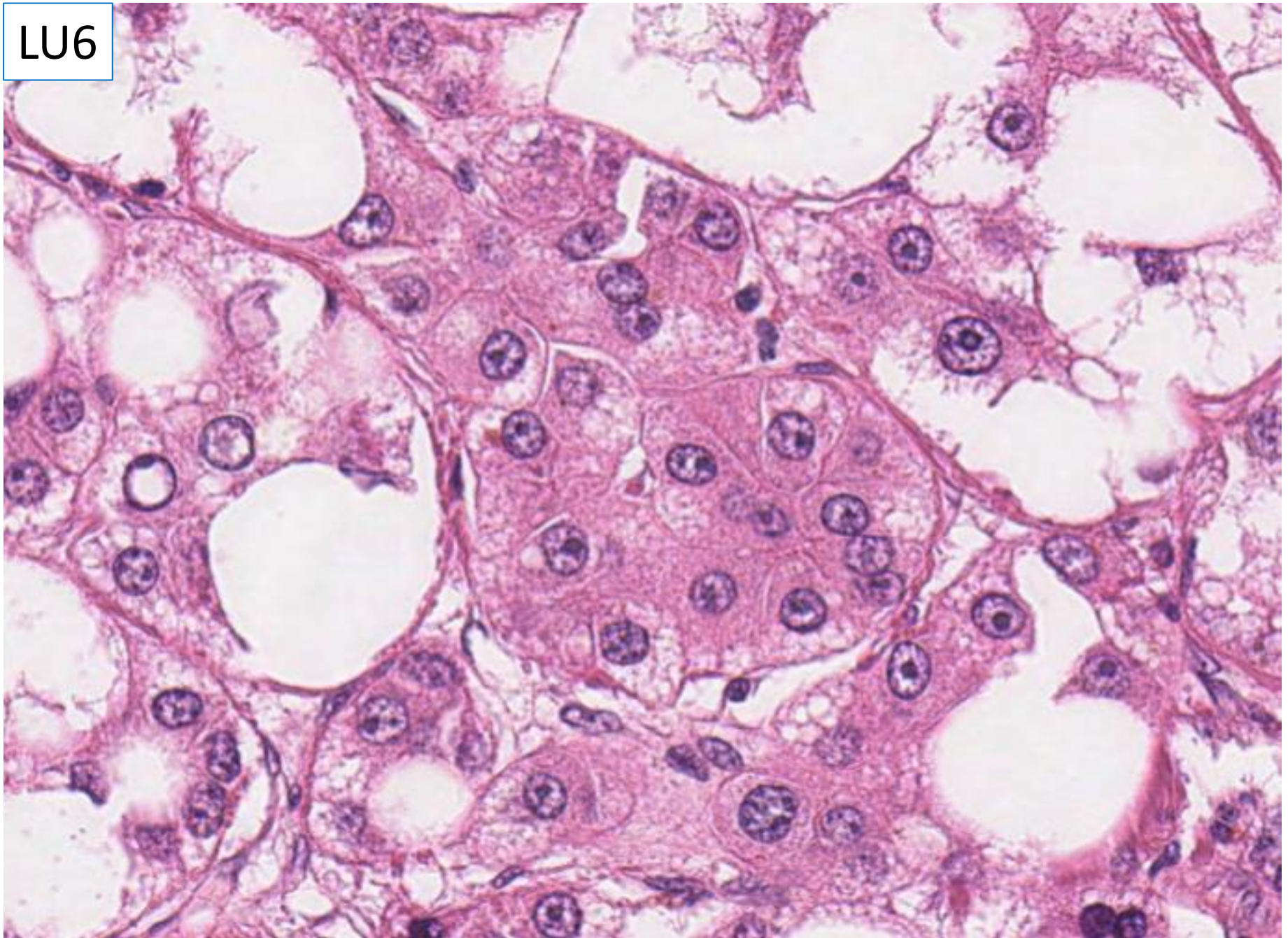
LU6



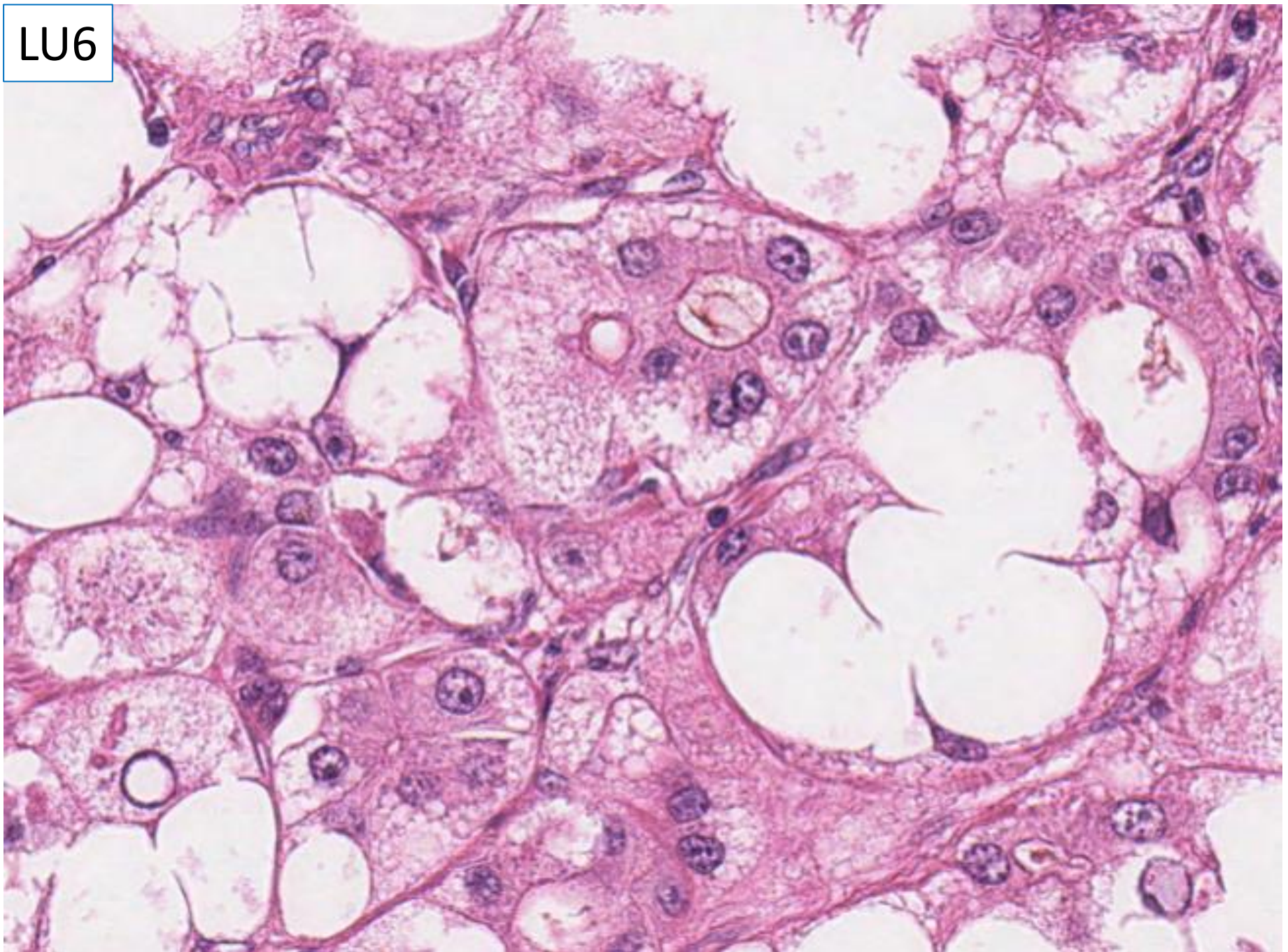
LU6



LU6



LU6



Case LU6 80F

Liver cirrhosis, mass lesion in segment VIII/IVa, ?HCC.

Specimen: Segment VIII/IVa segmentectomy; mass lesion 22 x 22 x 18mm.

No additional stains.

LU6	
A	Regenerative nodule background cirrhosis
B	HCC background cirrhosis
C	FNH background cirrhosis
D	Adenoma background cirrhosis
E	Focal Steatotic nodule

Case LU6 80F

Liver cirrhosis, mass lesion in segment VIII/IVa, ?HCC.

Specimen: Segment VIII/IVa segmentectomy; mass lesion 22 x 22 x 18mm.

No additional stains.

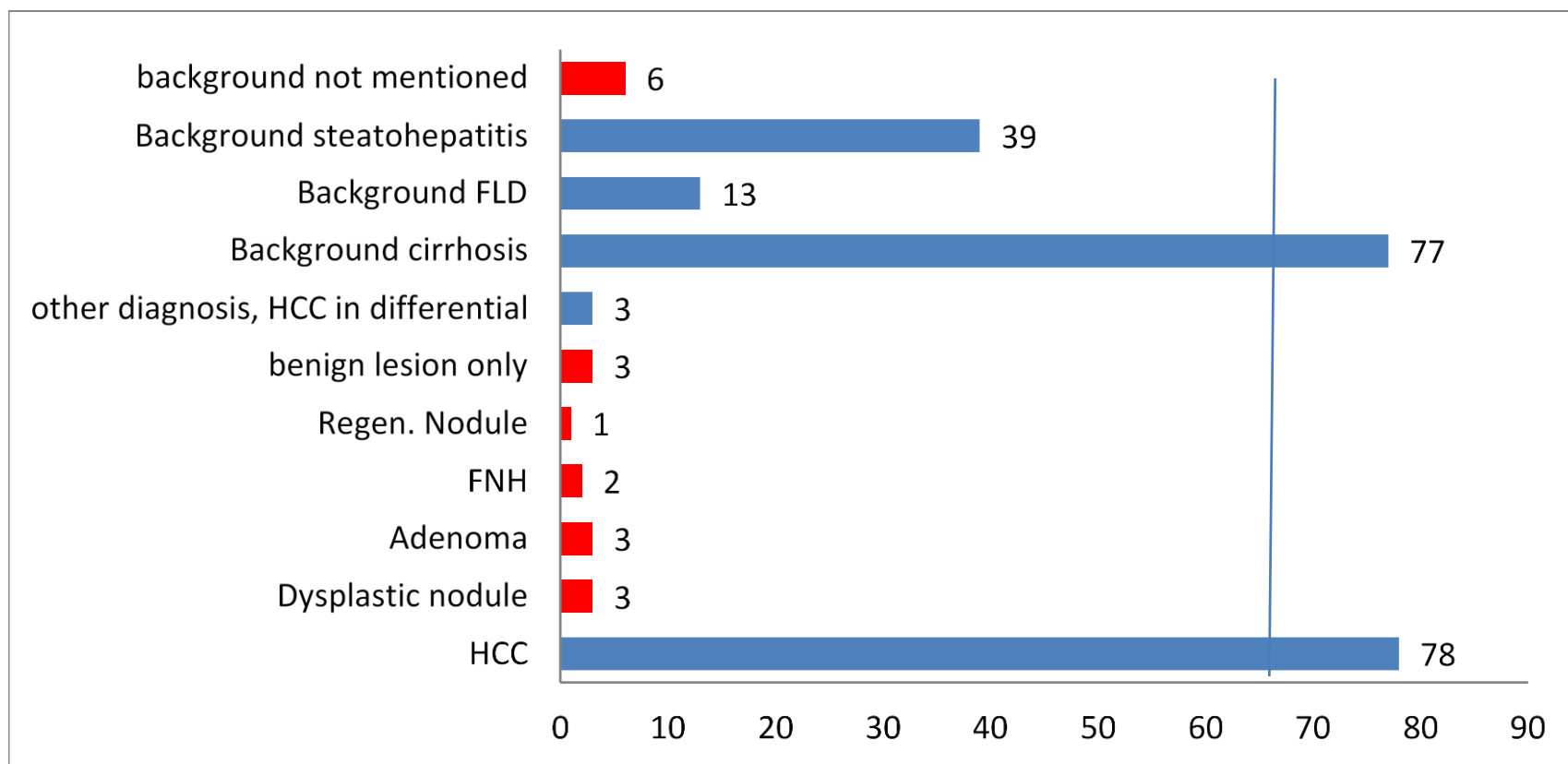
LU6	
A	Regenerative nodule background cirrhosis
B	HCC background cirrhosis
C	FNH background cirrhosis
D	Adenoma background cirrhosis
E	Focal Steatotic nodule

Case LU6 80F

Liver cirrhosis, mass lesion in segment VIII/IVa, ?HCC.

Specimen: Segment VIII/IVa segmentectomy; mass lesion 22 x 22 x 18mm.

No additional stains.



Consensus complete responses would include – hepatocellular carcinoma and mention of cirrhosis in non-lesional liver

Case LU6 80F

Liver cirrhosis, mass lesion in segment VIII/IVa, ?HCC.

Specimen: Segment VIII/IVa segmentectomy; mass lesion 22 x 22 x 18mm.

No additional stains.

Suggested scoring: for 10 points include – confident diagnosis of hepatocellular carcinoma and mention of cirrhosis in non-lesional liver

Lose 5 marks if – favour the lesion representing a dysplastic nodule, HCC in differential with additional work. (3) – *at meeting – will not mark these down.*

Lose 5 marks if – don't mention cirrhosis in background liver. (6)

Lose 10 marks (score 0) if – favour the lesion representing a benign process e.g. regenerative nodule, FNH or adenoma without mention of HCC in differential or any further work. (5)

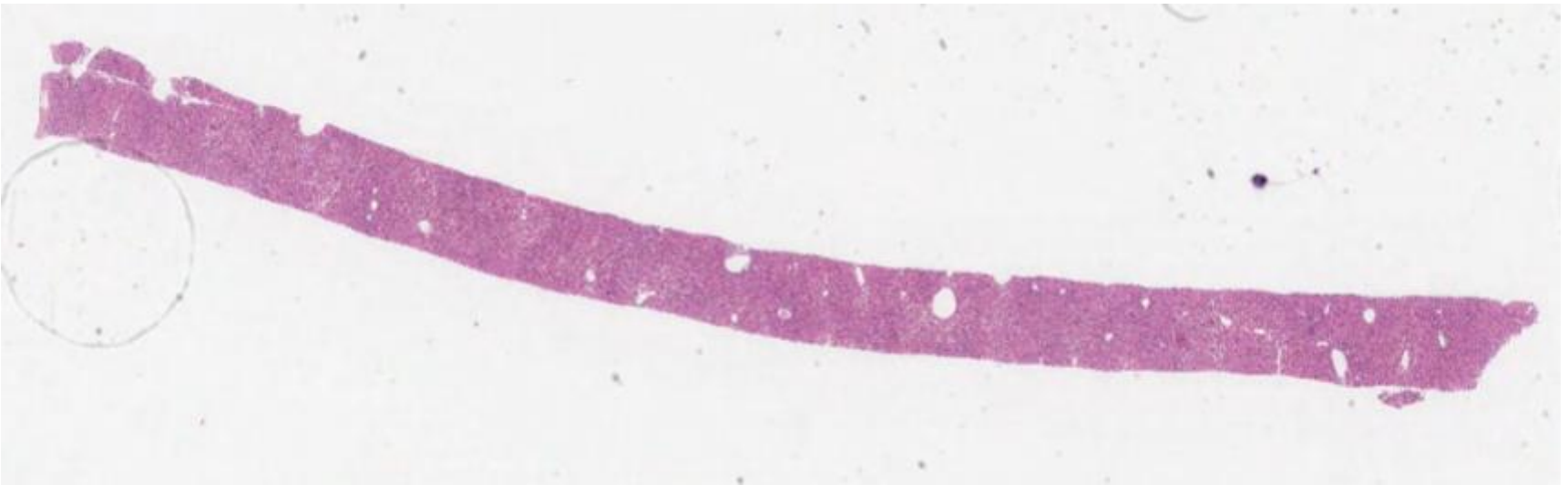
Meeting 16.10.19 unequivocal benign diagnosis score 0 if HCC in DD but not favoured score 5

Observations/potential learning points,

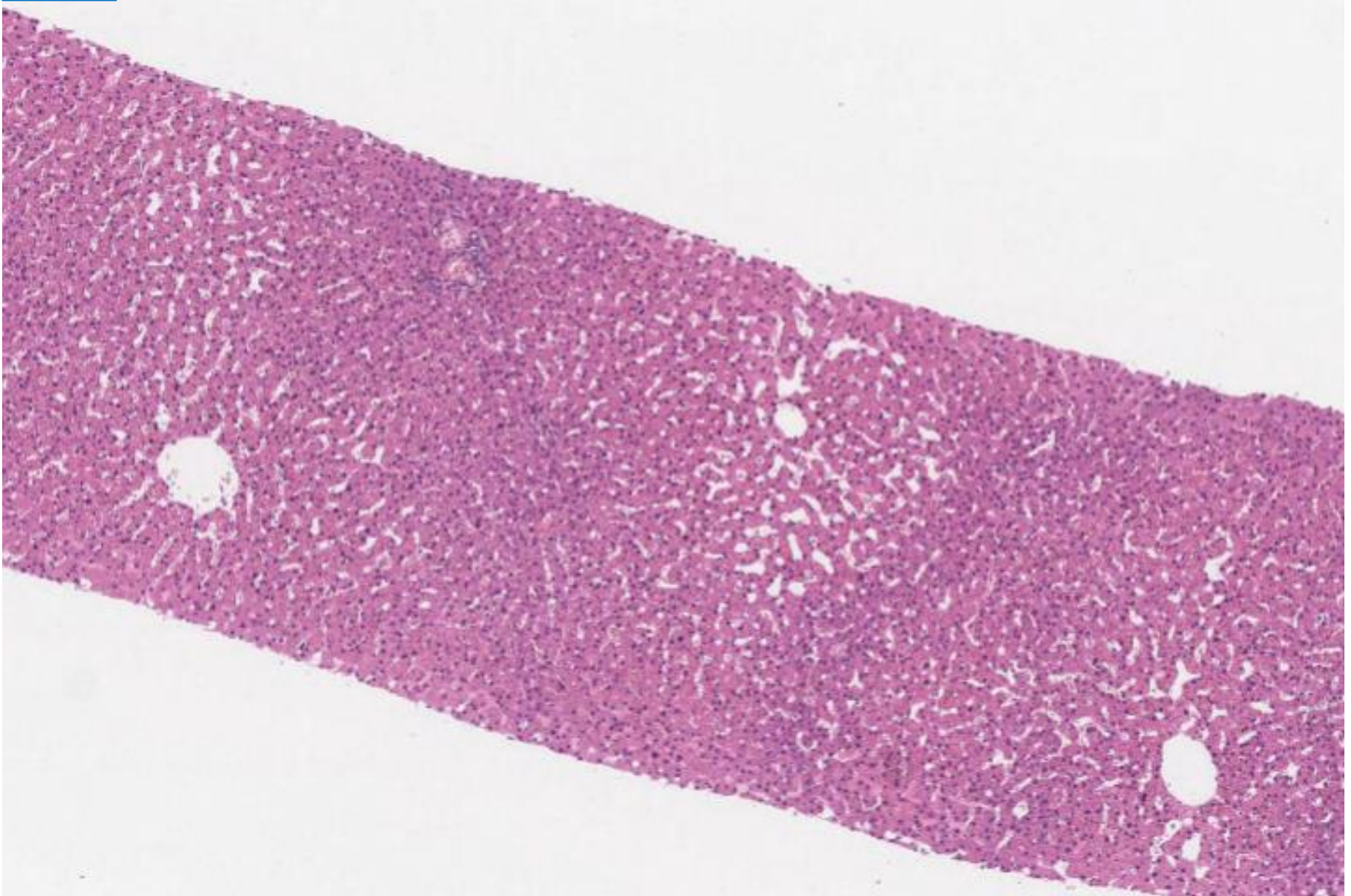
– assessment of well differentiated hepatocellular lesions

Case LU7 24M

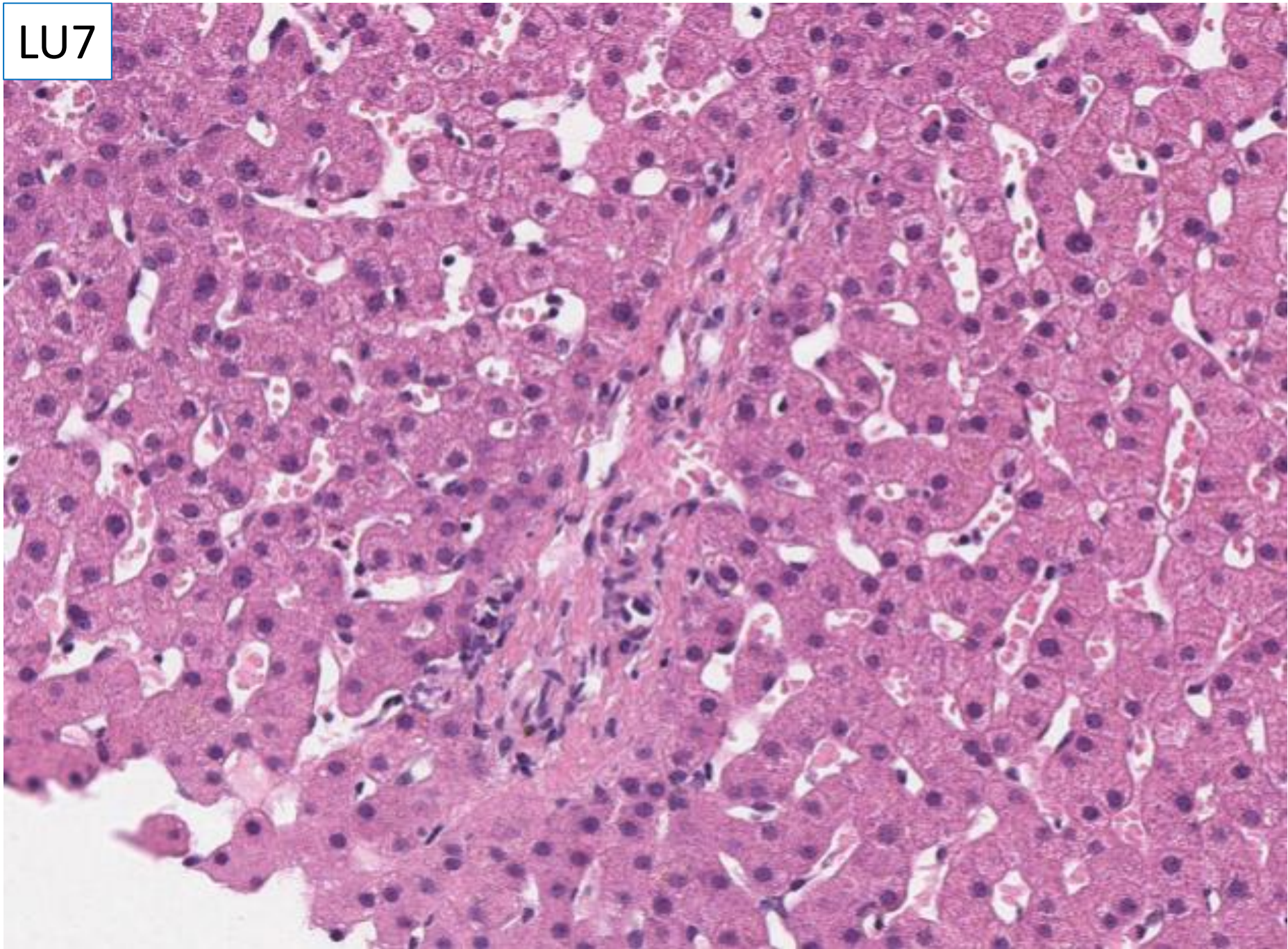
Portal hypertension. being assessed for TIPPS; ? cirrhosis.
Also PSR, Victoria Blue.



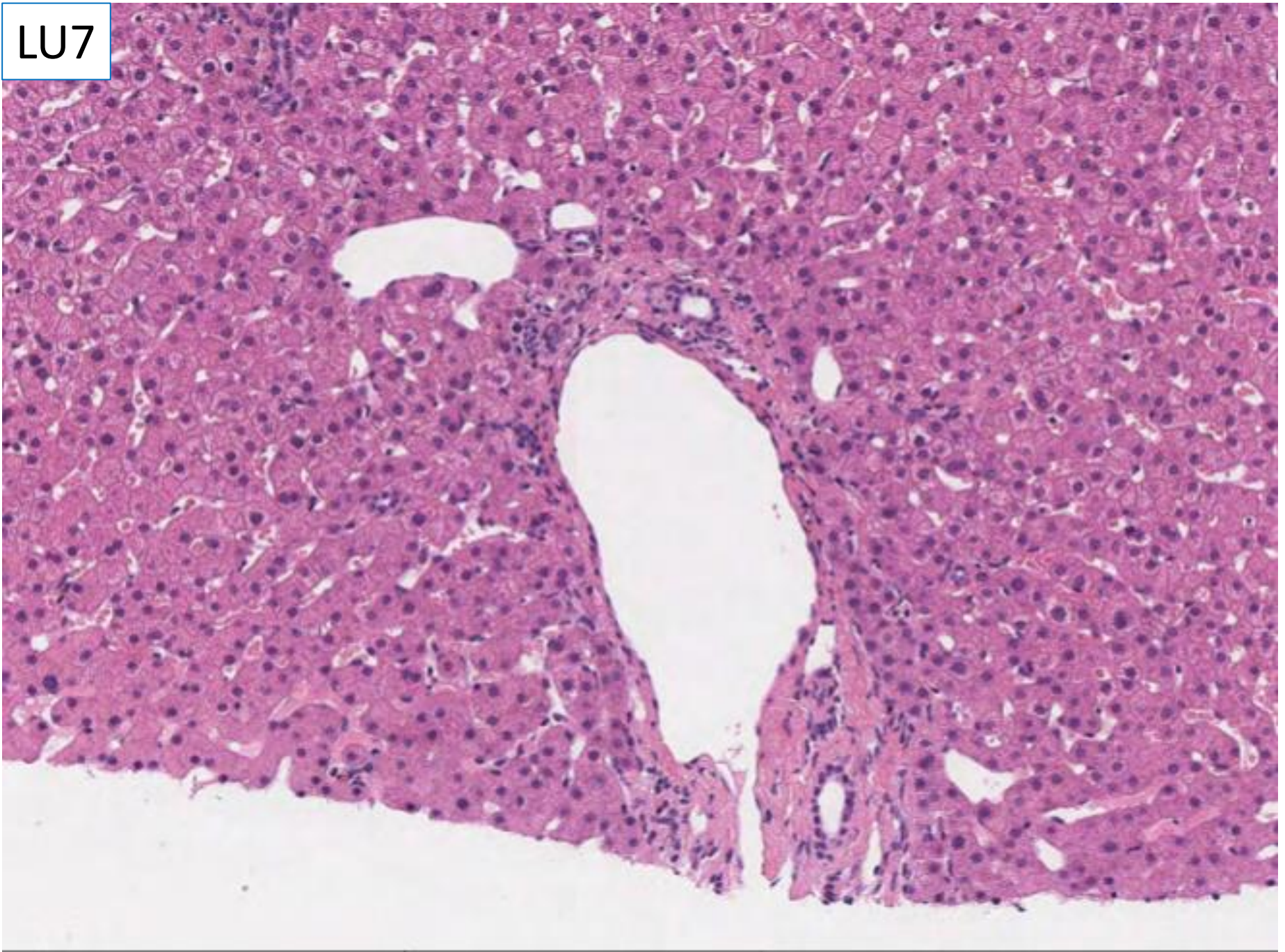
LU7



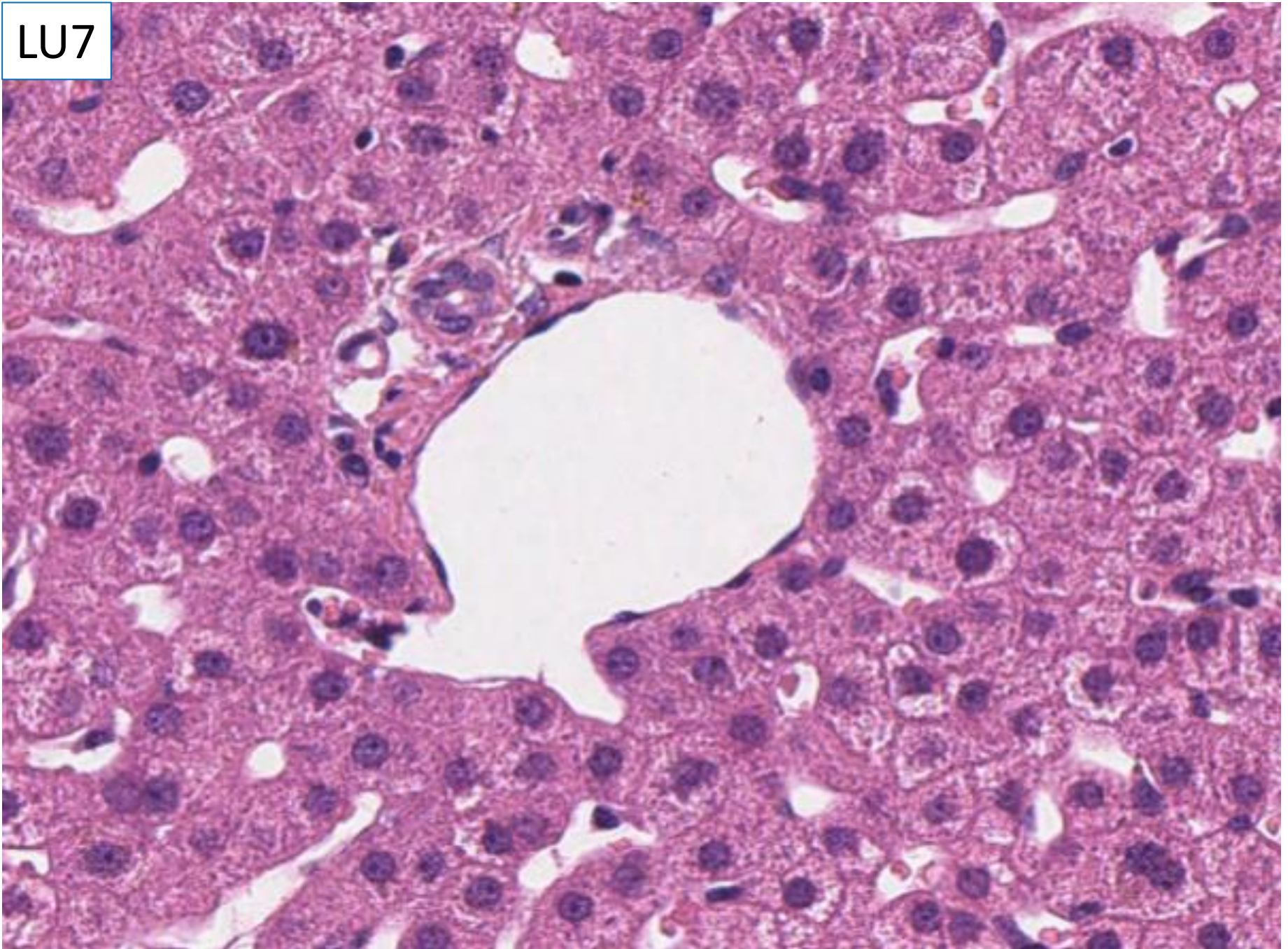
LU7



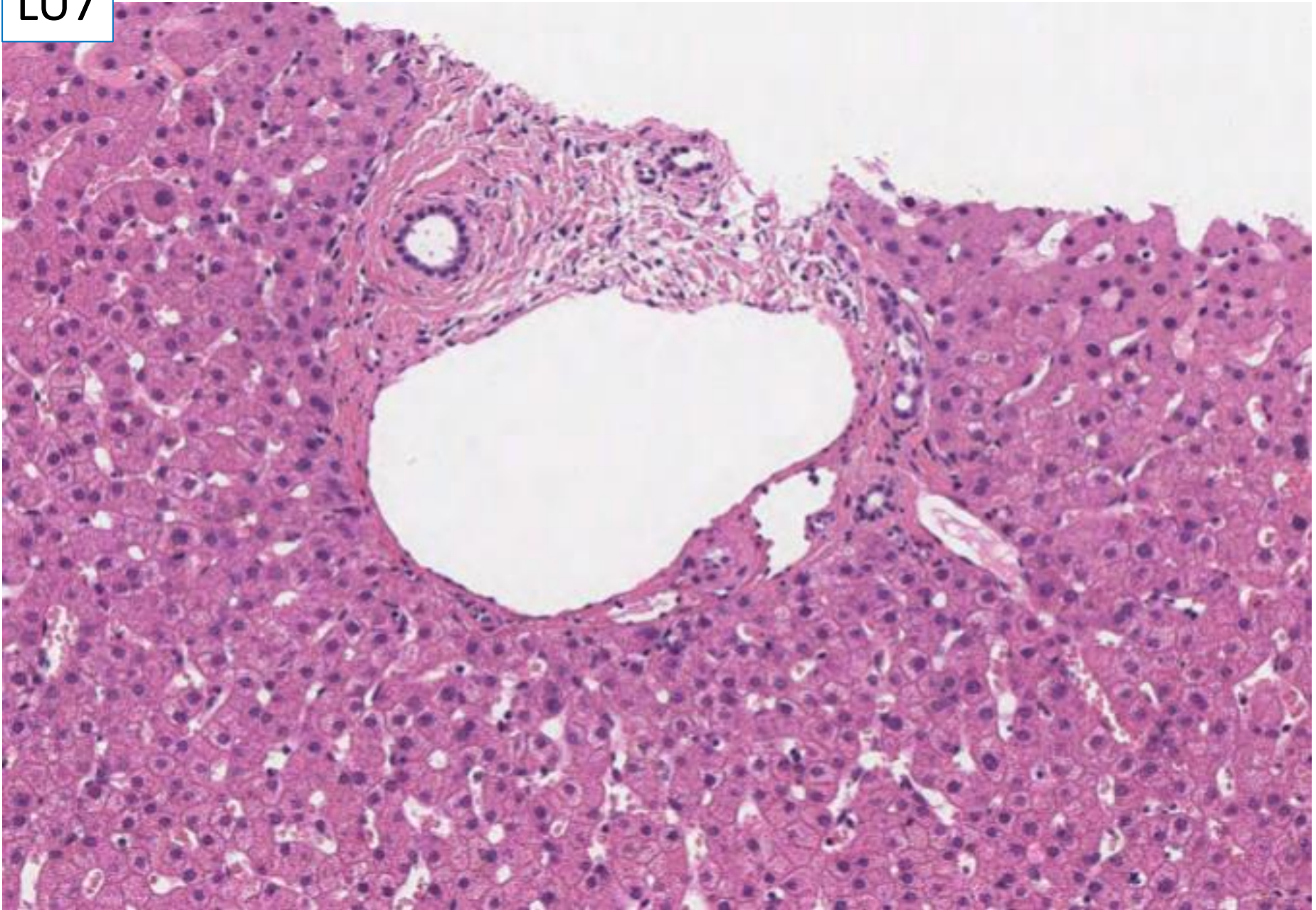
LU7



LU7



LU7

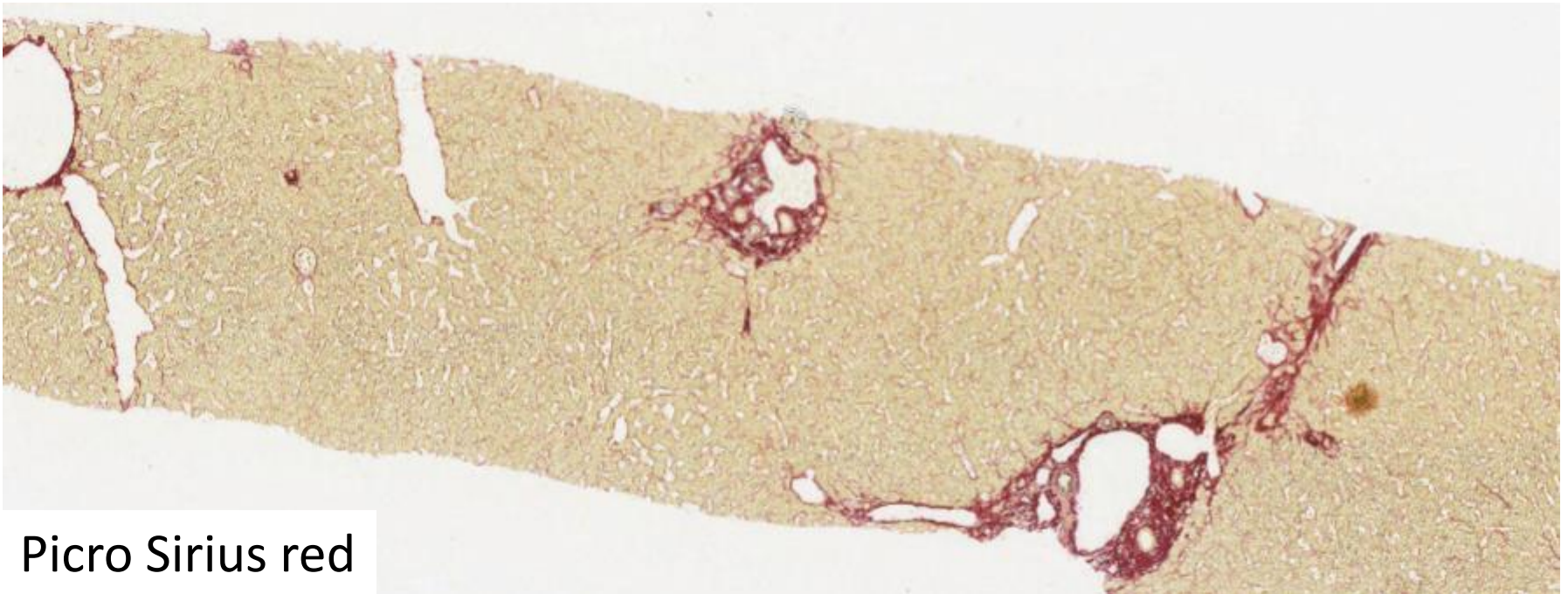
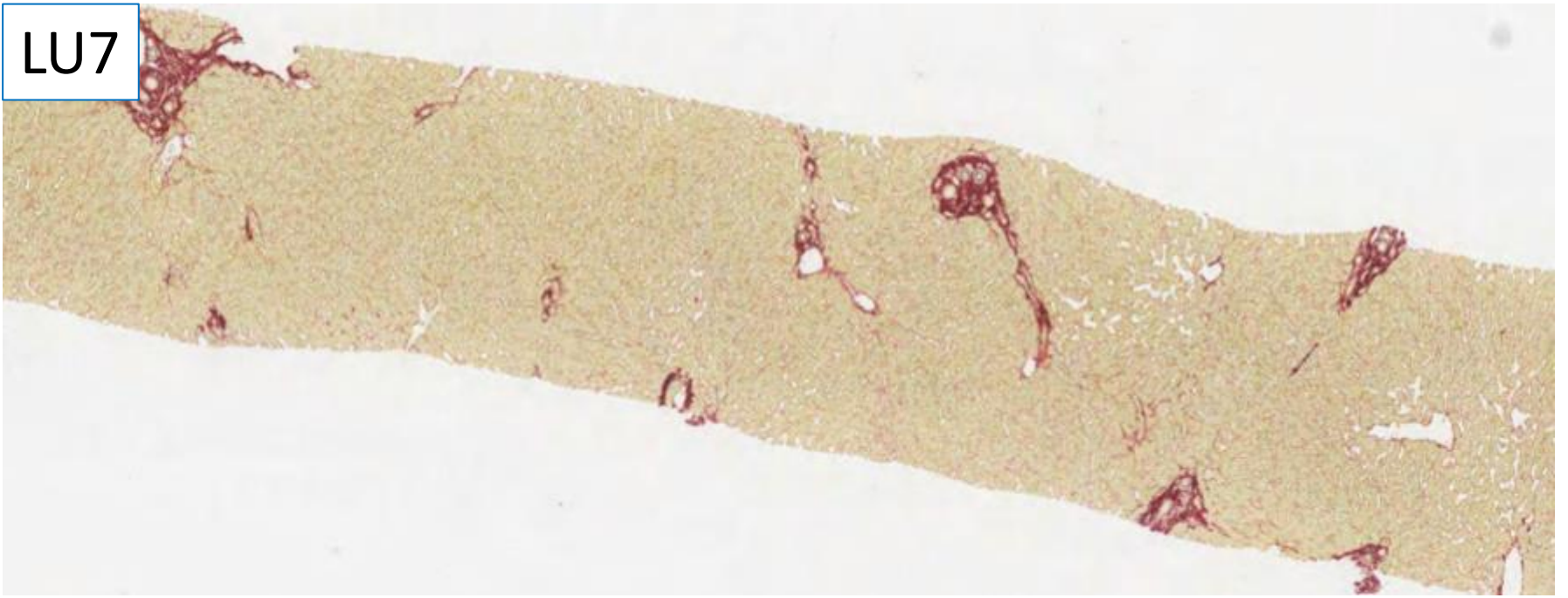


LU7



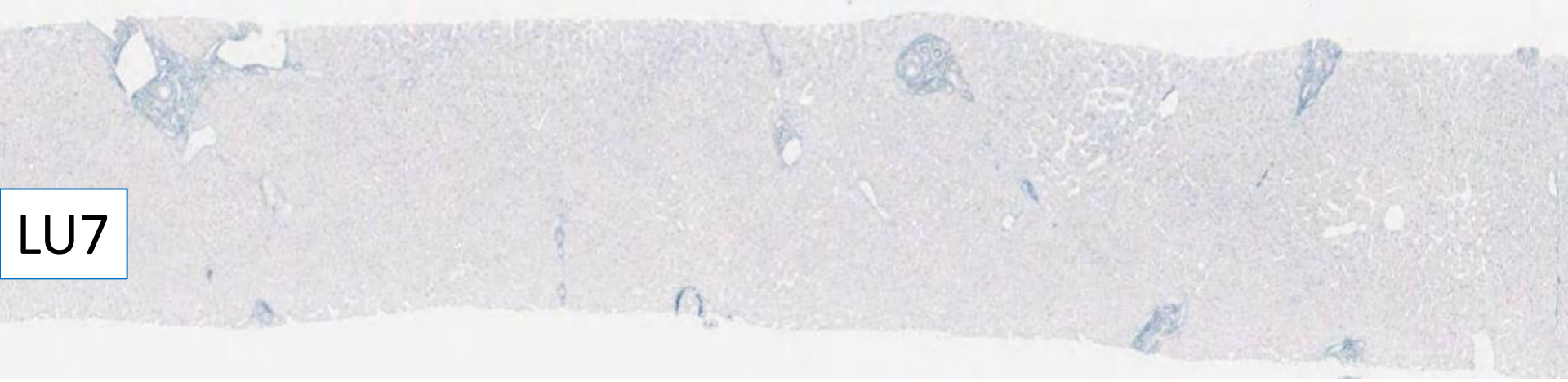
Picro Sirius red

LU7

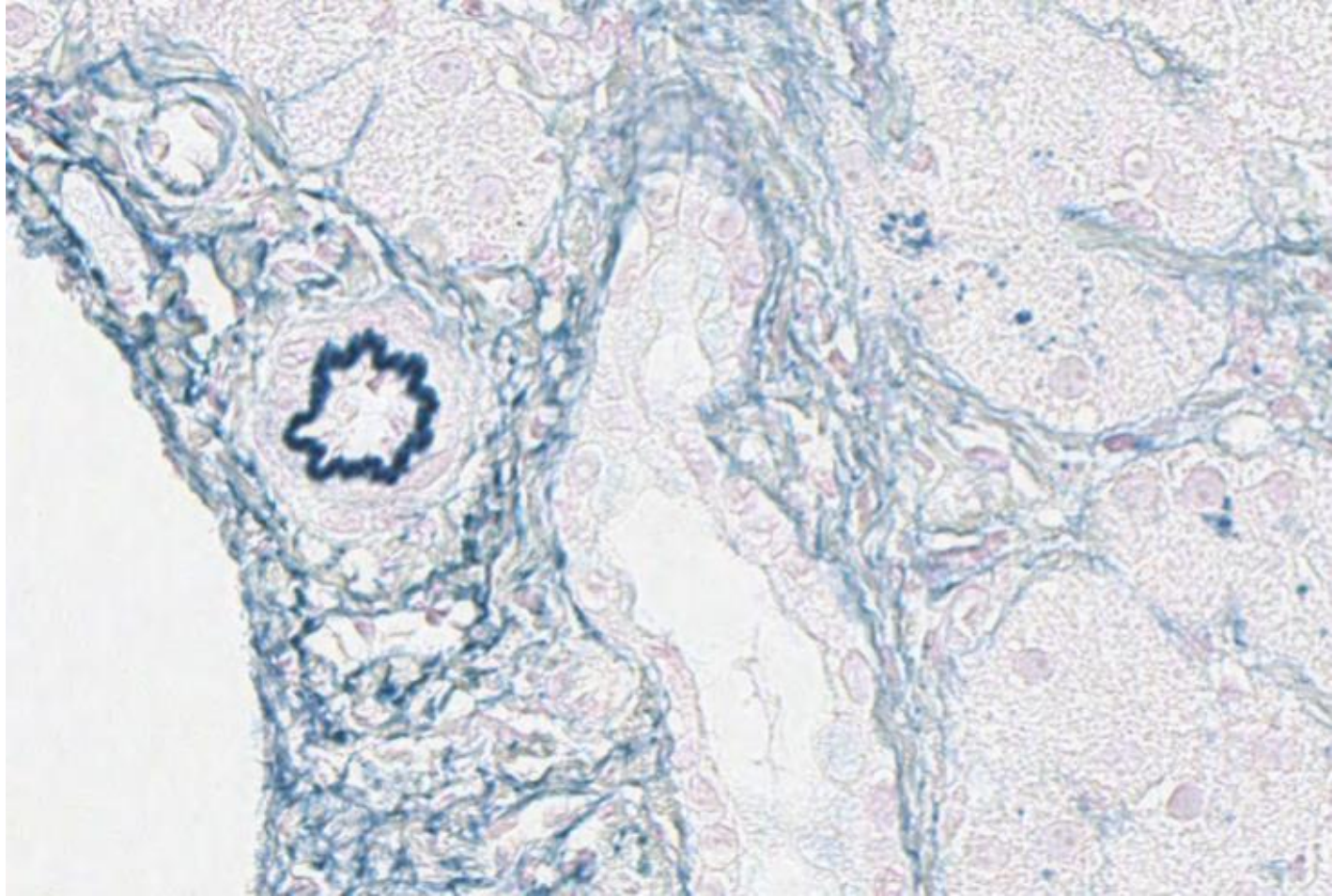


Picro Sirius red

LU7



Victoria
blue



Case LU7 24M

Portal hypertension. being assessed for TIPPS; ? cirrhosis.

Also PSR, Victoria Blue.

LU7	
A	Venous outflow obstruction
B	Biliary disease +/- possible sclerosing cholangitis
C	Normal liver
D	Vascular disease 'Non cirrhotic portal hypertension'
E	Wilson disease

Case LU7 24M

Portal hypertension. being assessed for TIPPS; ? cirrhosis.

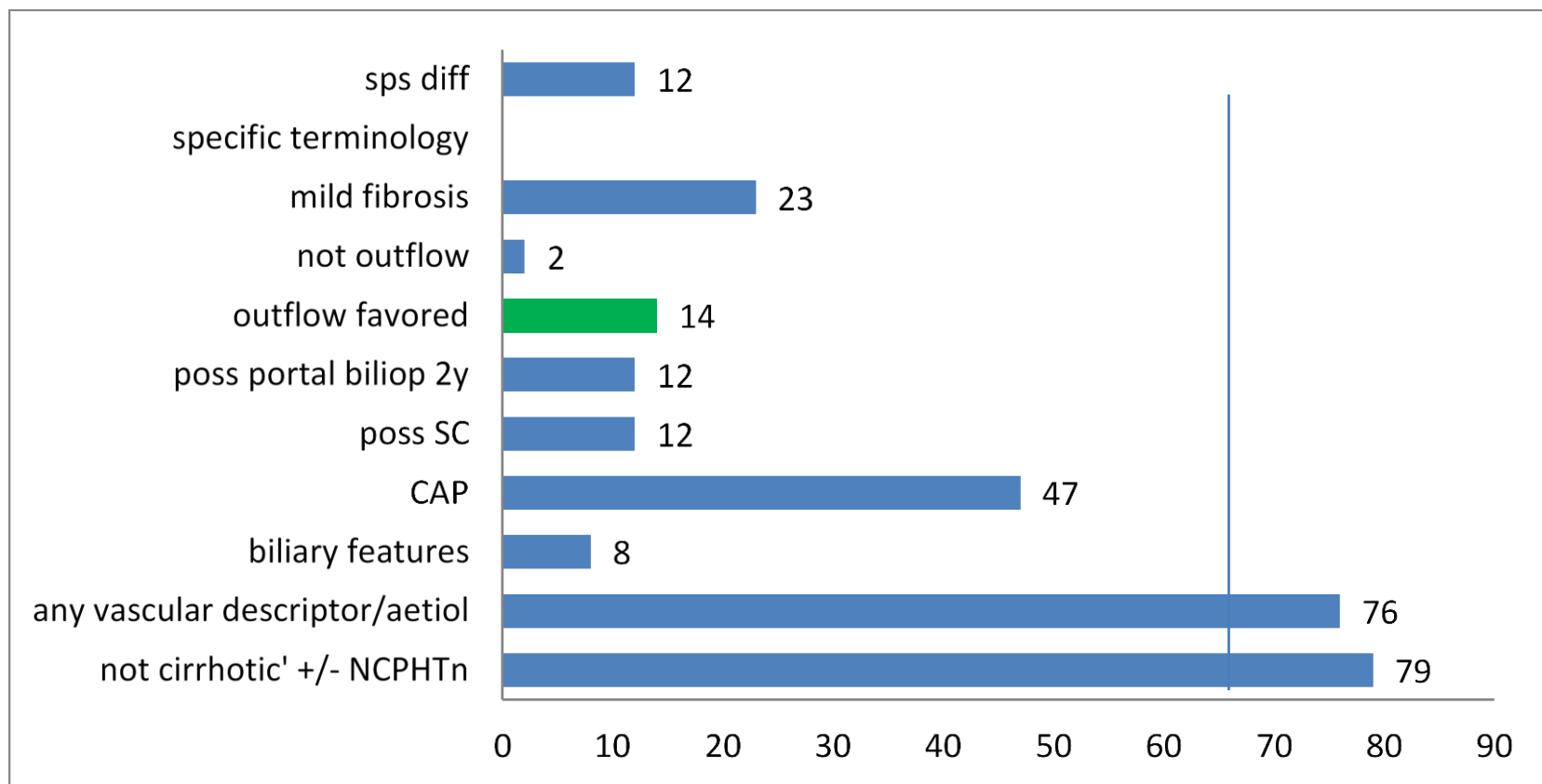
Also PSR, Victoria Blue.

LU7	
A	Venous outflow obstruction
B	Biliary disease +/- possible sclerosing cholangitis
C	Normal liver
D	Vascular disease 'Non cirrhotic portal hypertension'
E	Wilson disease

Case LU7 24M

Portal hypertension. being assessed for TIPPS; ? cirrhosis.

Also PSR, Victoria Blue.



Consensus complete responses would include; 'not cirrhosis' and/or 'non cirrhotic portal hypertension'

AND some indication of a vascular cause or description of vascular lesions.

Case LU7 24M

Portal hypertension. being assessed for TIPPS; ? cirrhosis.

Also PSR, Victoria Blue.

Suggested scoring: for 10 points include 'not cirrhosis' and some intimation that might be a vascular cause

Lose 5 marks if say not cirrhotic but favour another disease e.g primary biliary disease (PSC where specified) =6, Wilsons =1, 'background congenital hepatic fibrosis' =1

Lose 5 marks if 'essentially normal' (1) [meeting 16.10.19 this to score 0 – at meeting, not worse than favouring other disease, so score 5 as well.](#)

Lose 5 marks if 'not cirrhotic' not stated and no comment fibrosis (where anything less than fibrosis described assumed 'not cirrhotic') (3)

? Lose 5 marks if strongly favour outflow obstruction

- *show of hands agreed to score 5*

Case LU7 24M

Portal hypertension. being assessed for TIPPS; ? cirrhosis.

Also PSR, Victoria Blue.

Observations/potential learning points, suitable for masterclass? yes

Sometimes 'no cirrhosis' implied as above. 'non-cirrhotic portal hypertension' taken to imply vascular cause

11 observed specials seemed to be from a different case!

Meeting 16.10.19 they are not, submitting lab separated cores into different cassettes to ensure sufficient tissue left for multiple EQA sections.

Terminology – mostly generic vascular descriptions, 14 suggested outflow obstruction including one 'Budd Chiari?', 2 'not outflow'.

Hepatoportal sclerosis 2

Incomplete septal fibrosis 1

vascular postsinusoidal disease 1

Obliterative portal venopathy 4

NRH 3

Ischaemic liver disease 1

8 biliary features including 12 possible sclerosing duct lesion

12 'portal biliopathy' or suggestion biliary changes secondary to vascular

LU7

Masterclass – Sue Davies

Also LT7 and LT11

Vascular disease in liver biopsies

Case LU8 73M

Cirrhosis. Left lateral lesion, ?HCC. Ferritin 1708. AFP 5, CEA 2, CA19-9 42.

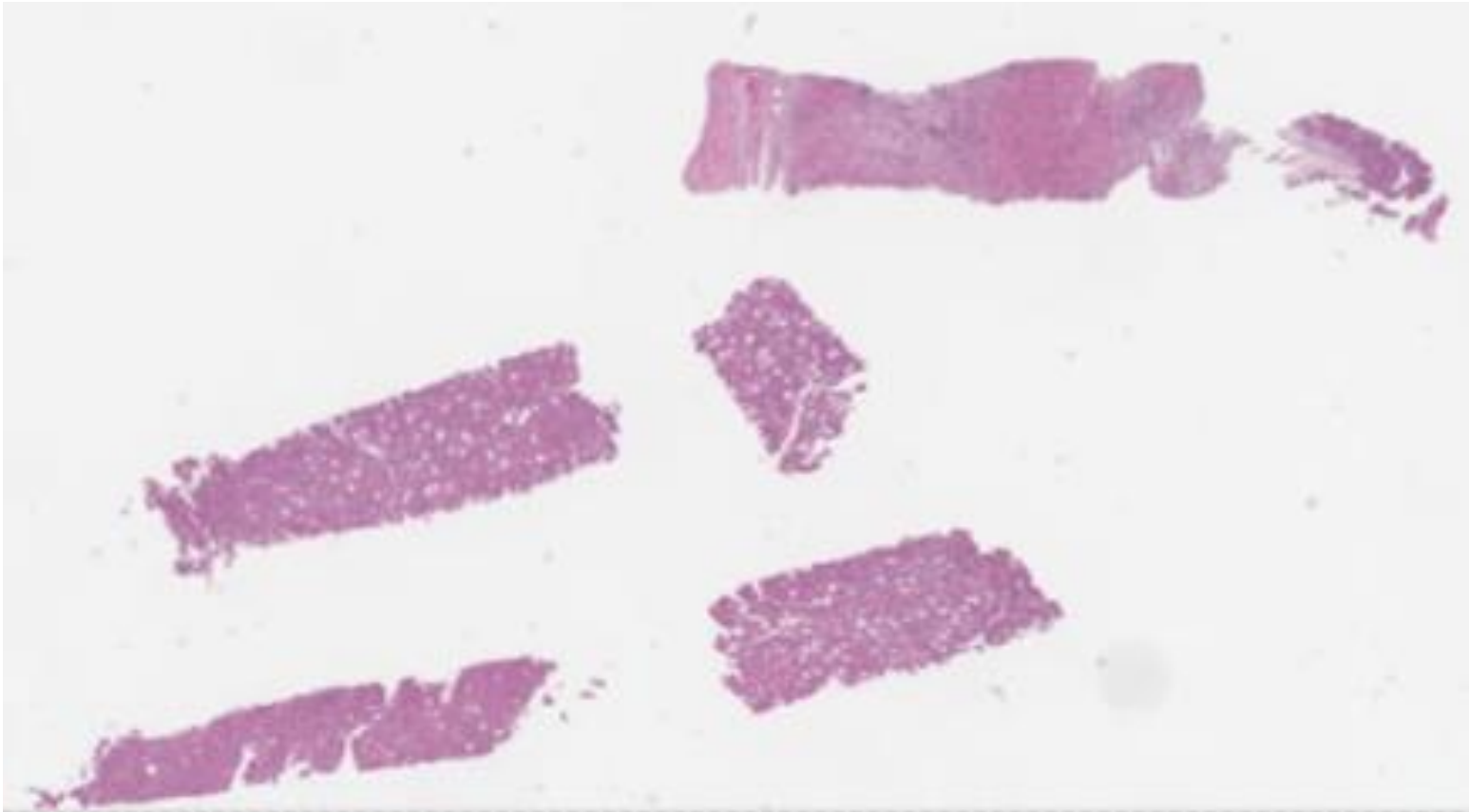
MRI liver: Established cirrhosis with associated portal hypertension.

Segment II lesion demonstrates radiological features suspicious for HCC.

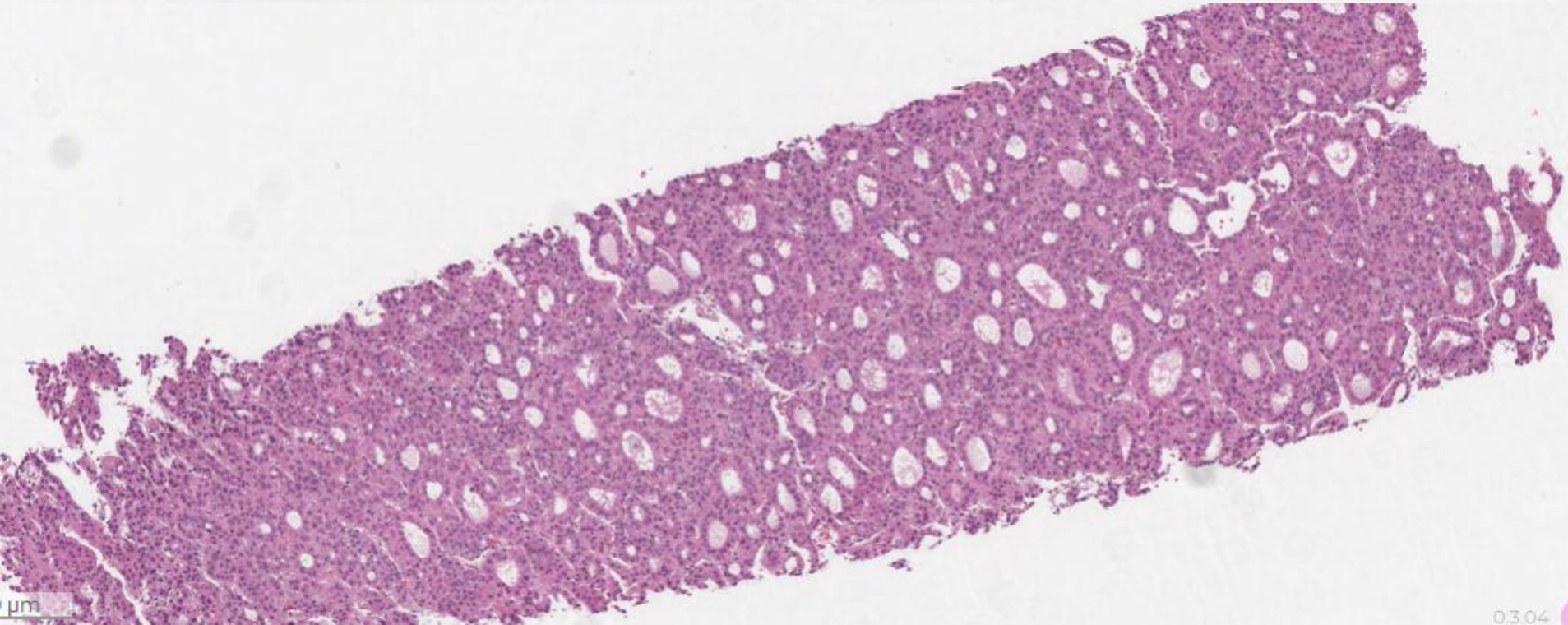
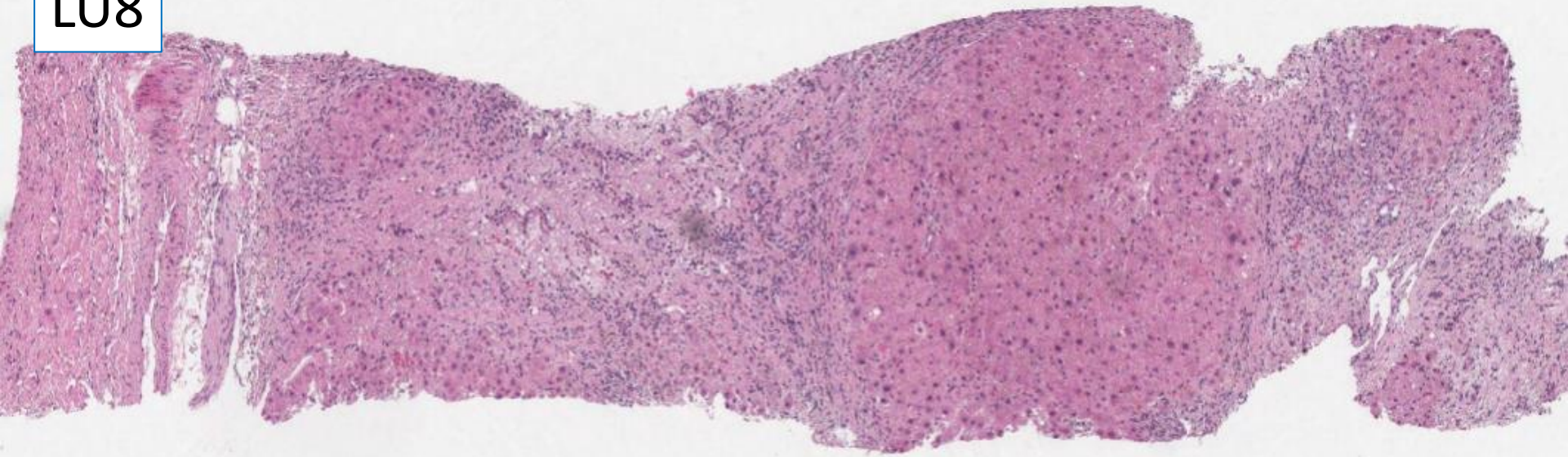
Specimen: Lesional liver biopsy, left lobe,

Two cores 10mm and 11mm in length.

Also: Reticulin, Perls, van Gieson, Hepato, Arginase, Glutamine Synthetase.

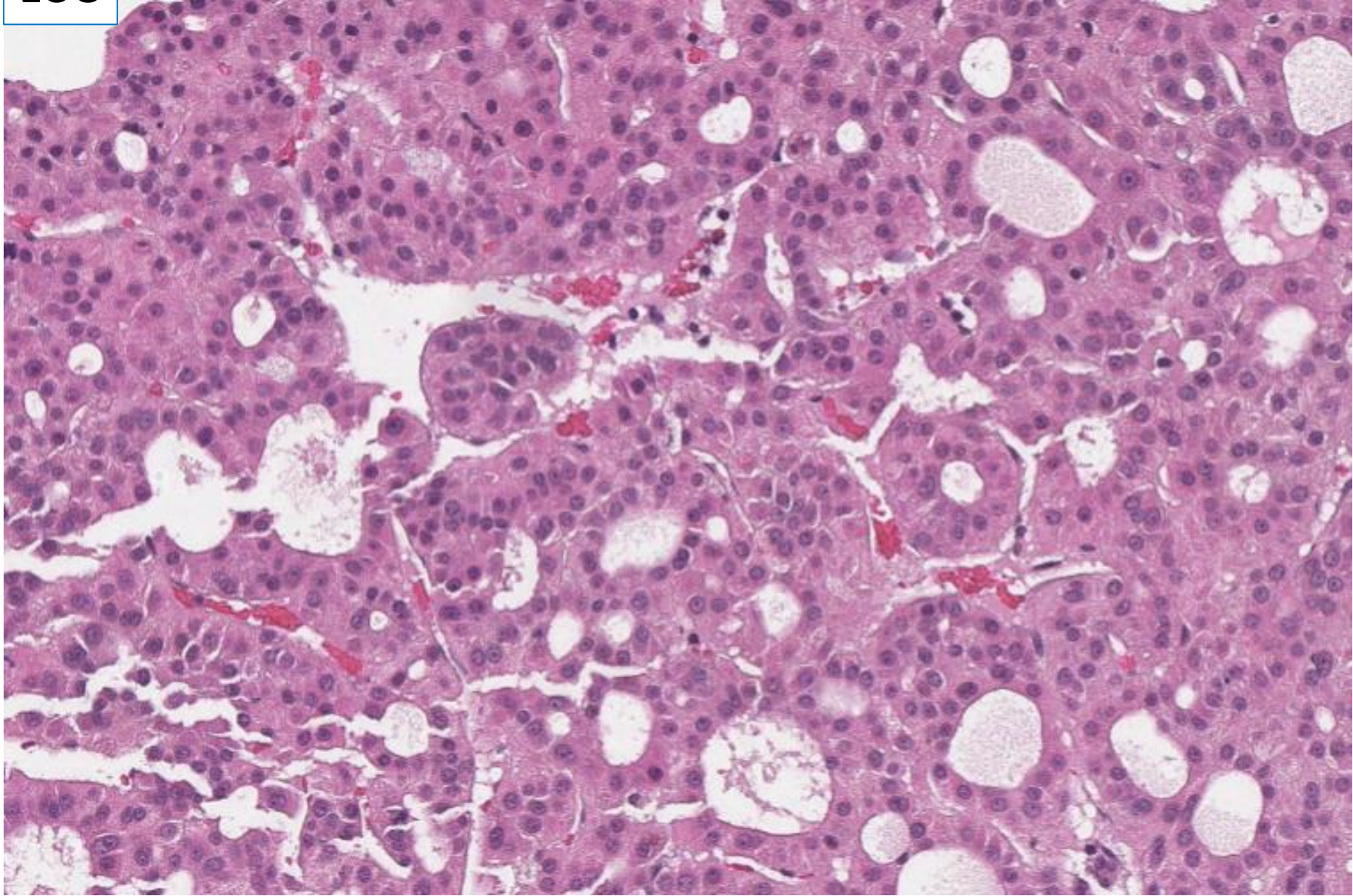


LU8



µm

LU8

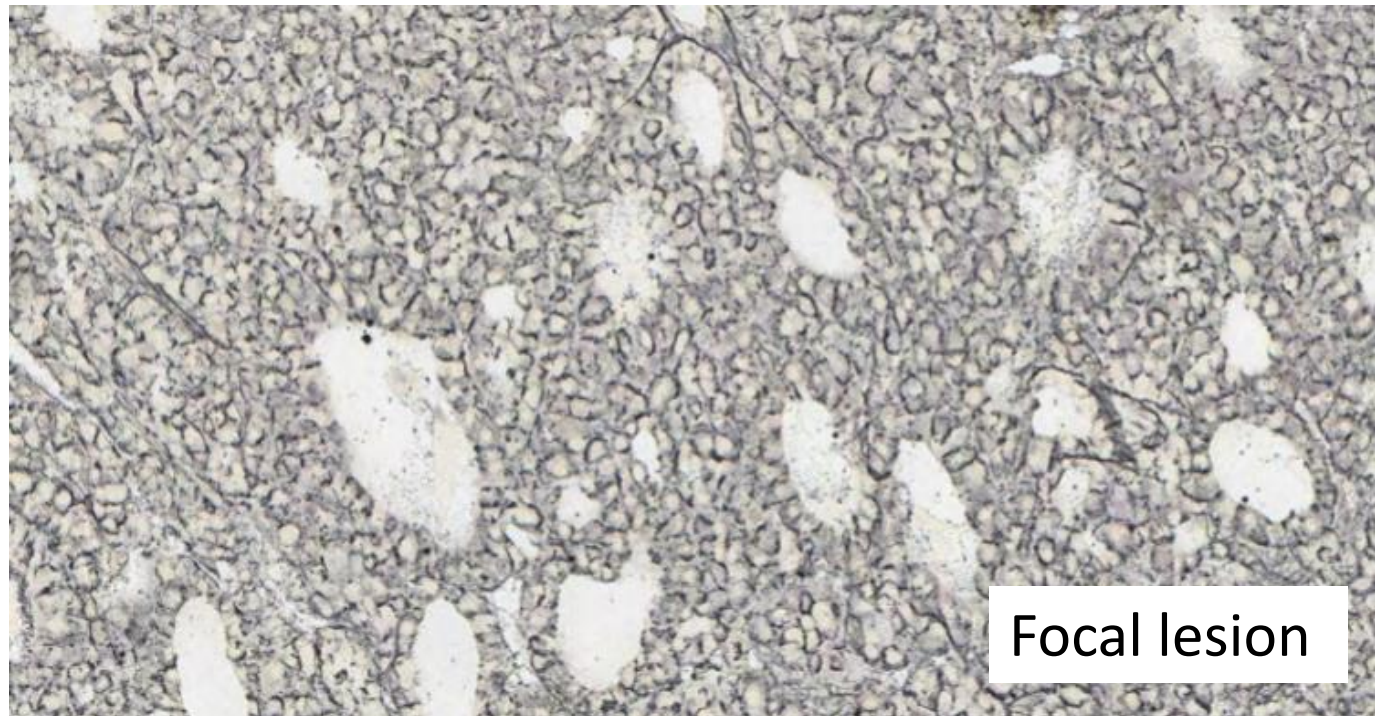
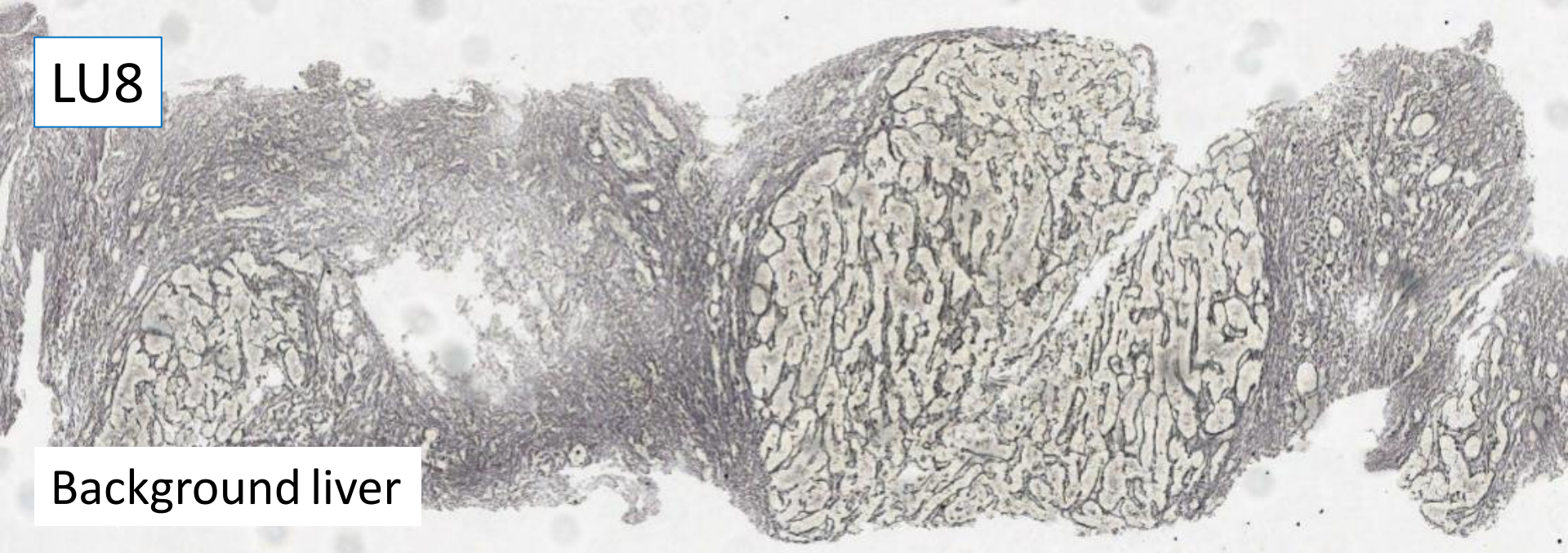


LU8

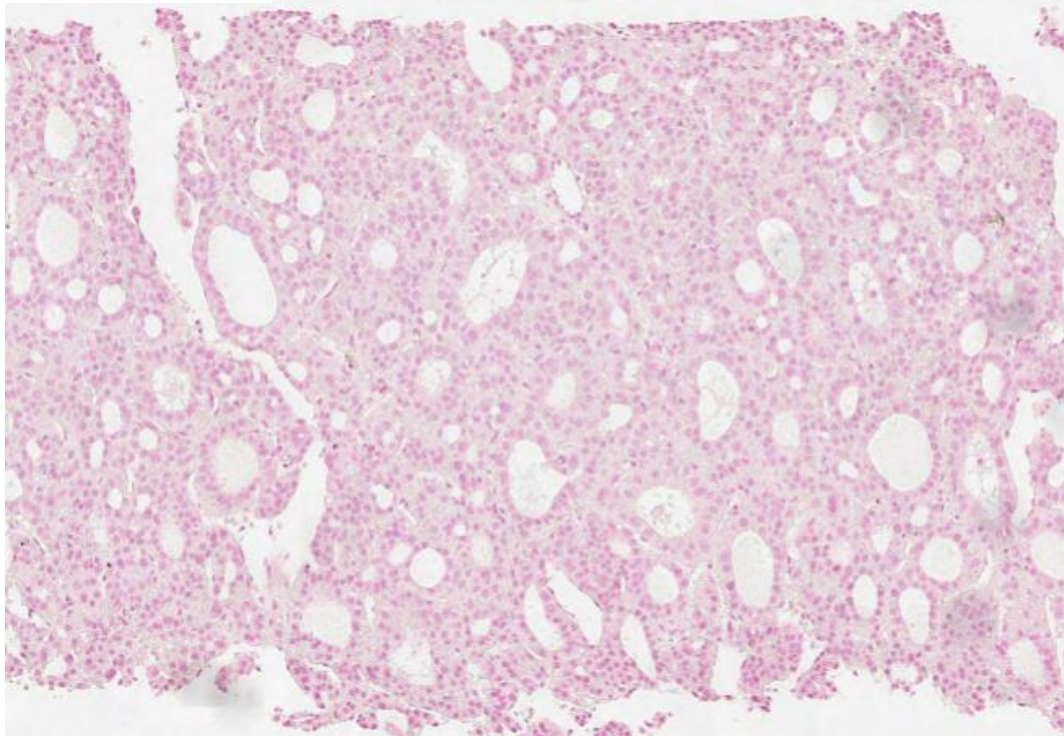
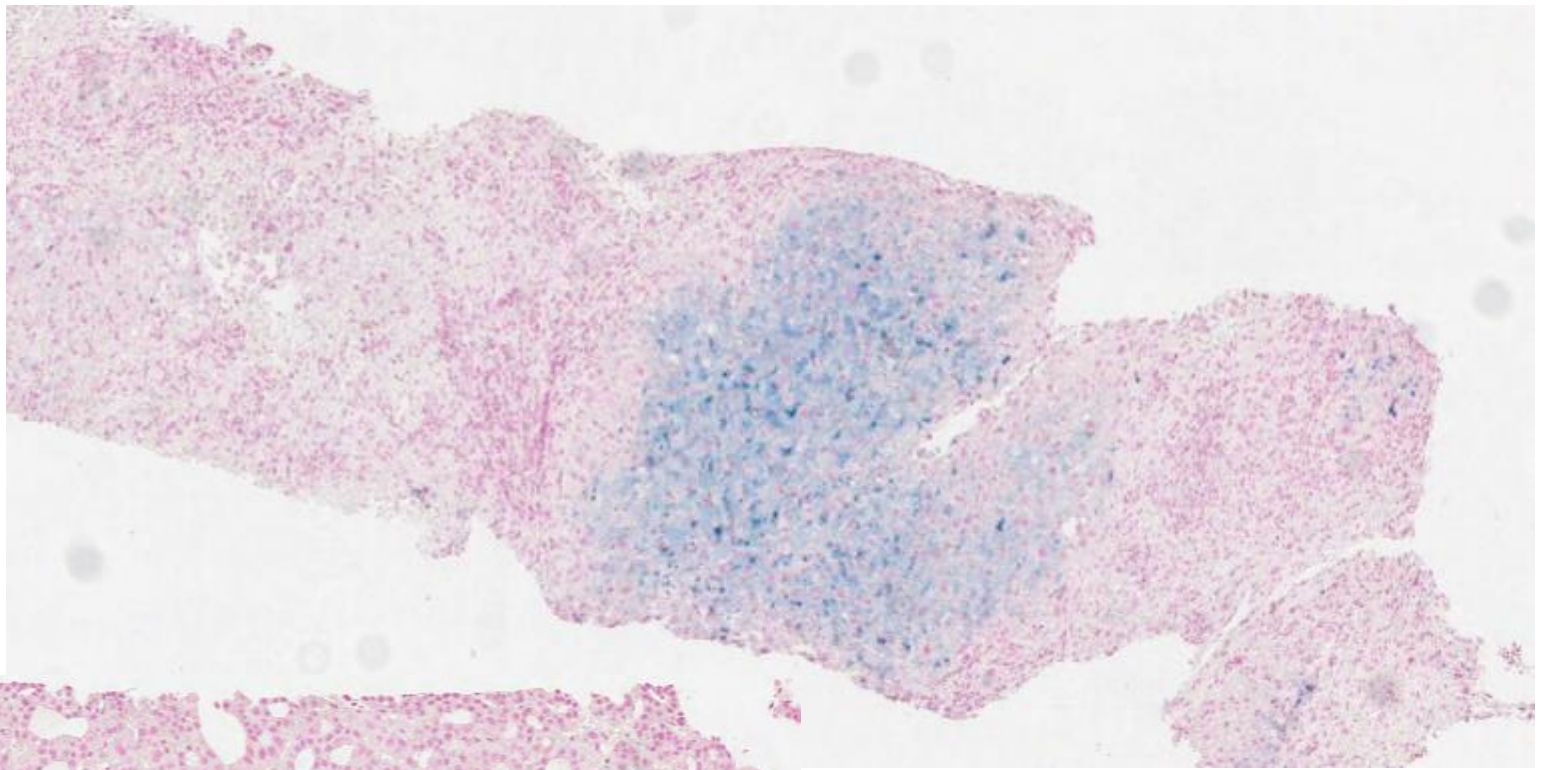
Background liver

reticulin

Focal lesion

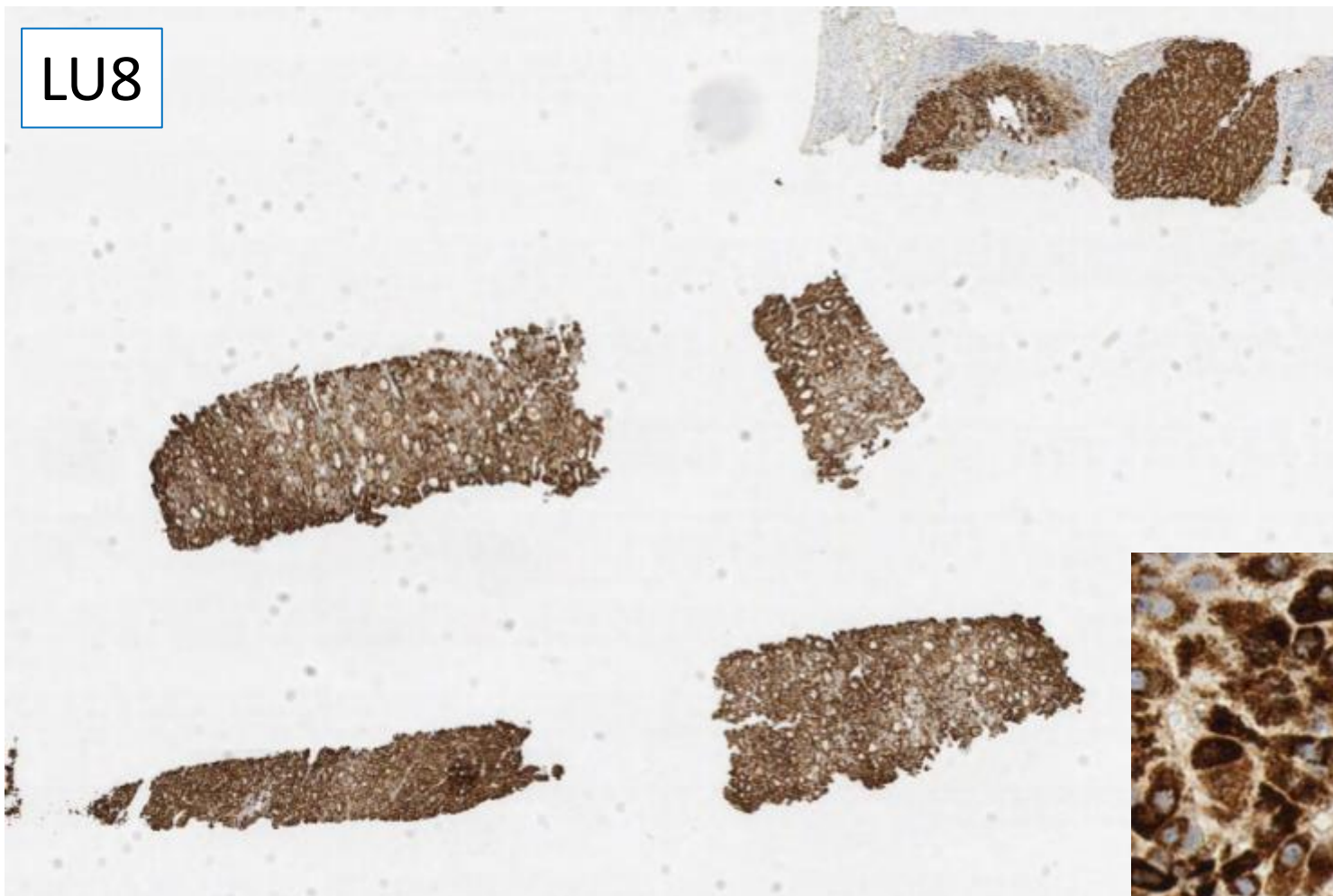


LU8



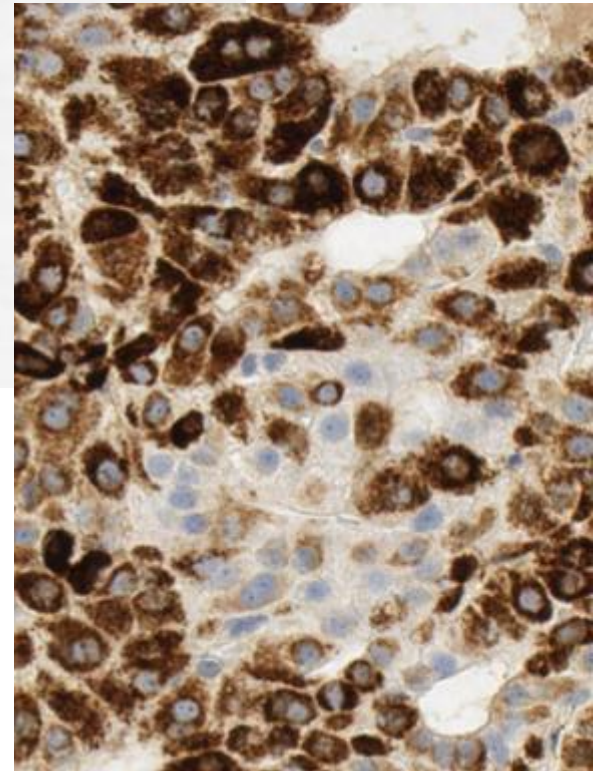
Perls

LU8



Hepato

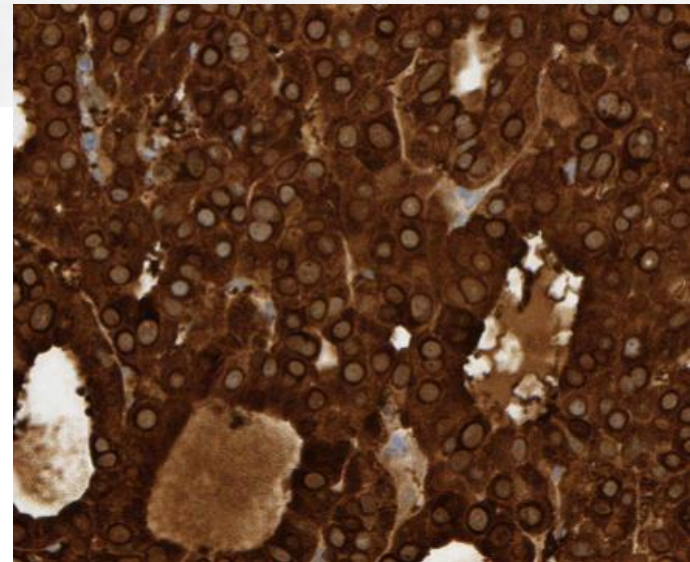
Lesion



LU8

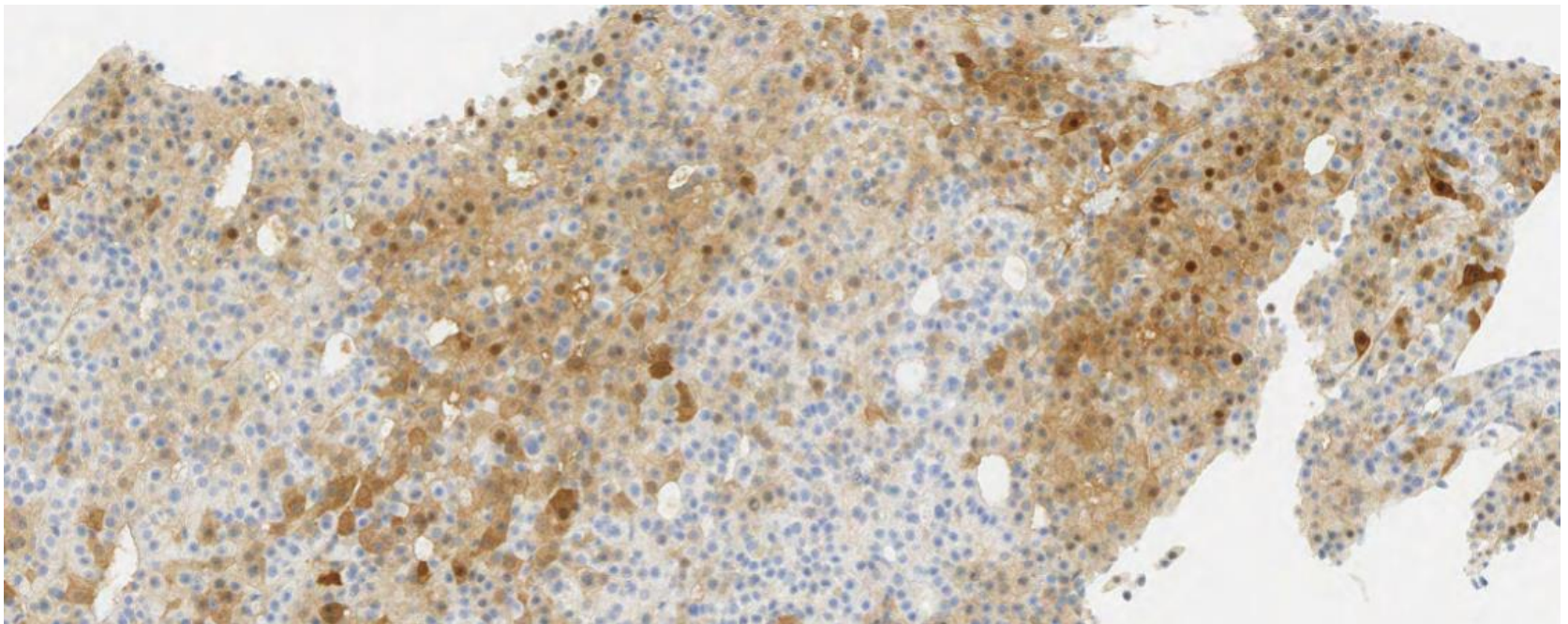
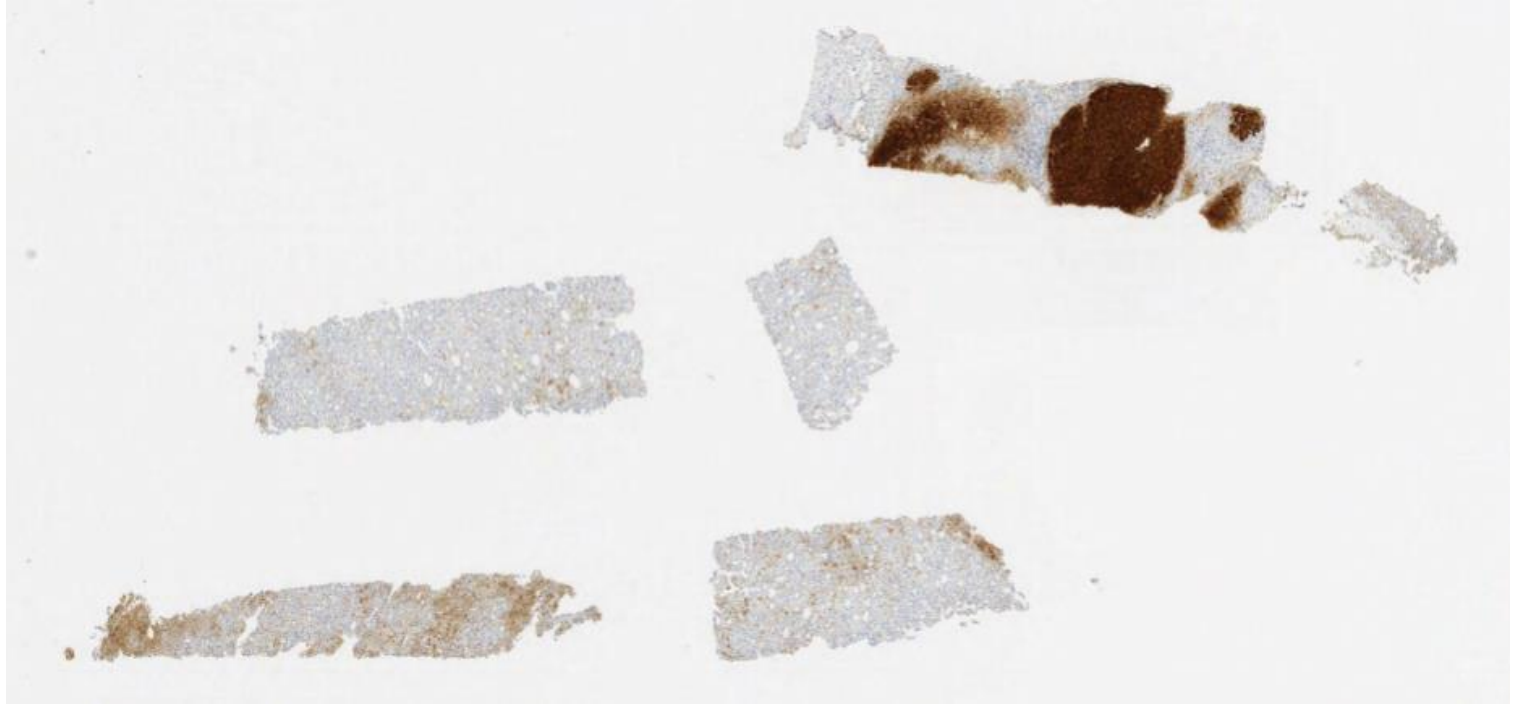


Glutamine synthetase



LU8

Arginase



Case LU8 73M

Cirrhosis. Left lateral lesion, ?HCC. Ferritin 1708. AFP 5, CEA 2, CA19-9 42.

Also: Reticulin, Perls, van Gieson, Hepato, Arginase, Glutamine Synthetase.

LU8	
A	HCC and iron
B	Dysplastic nodule, cirrhosis and iron deposition
C	HCC, cirrhosis and iron deposition
D	HCC and cirrhosis
E	Hepatocellular adenoma and iron deposition

Case LU8 73M

Cirrhosis. Left lateral lesion, ?HCC. Ferritin 1708. AFP 5, CEA 2, CA19-9 42.

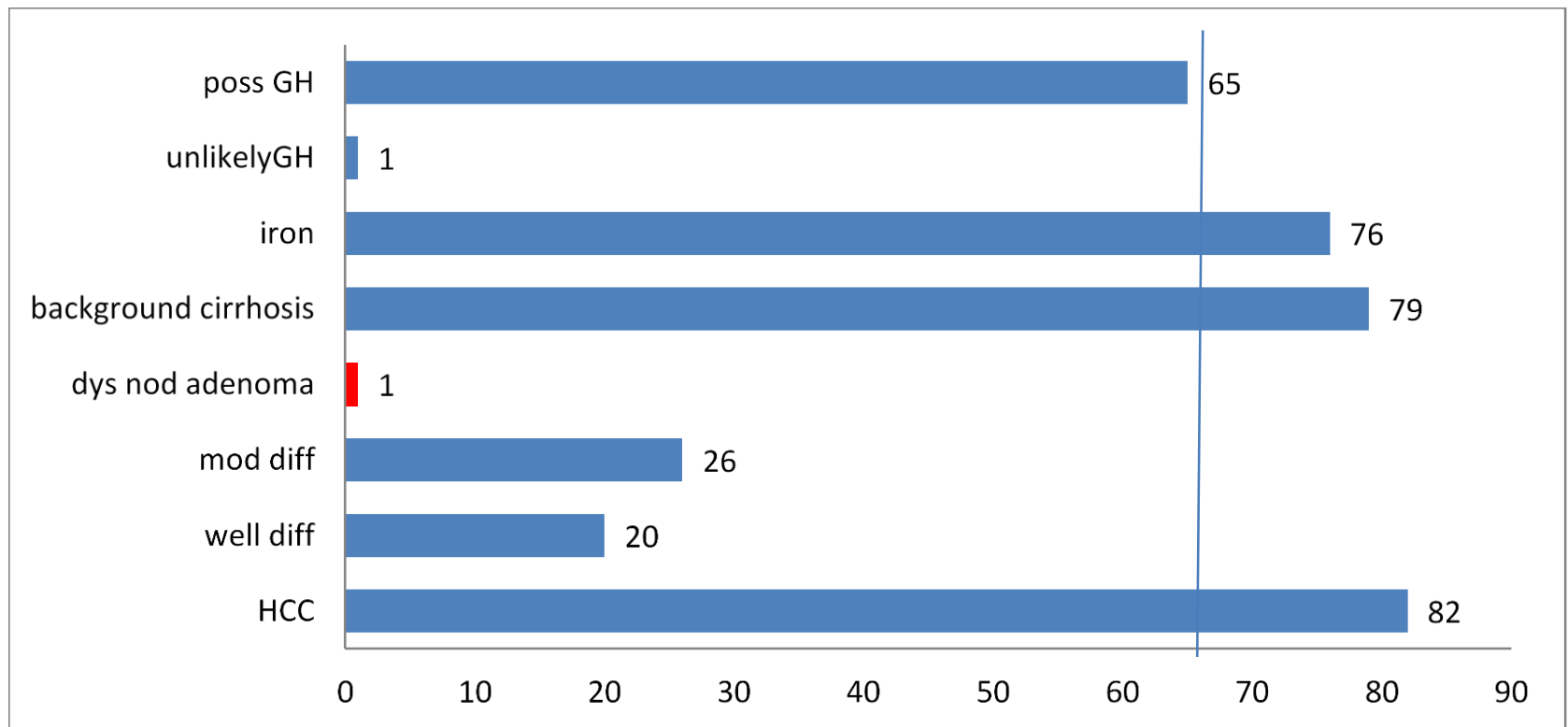
Also: Reticulin, Perls, van Gieson, Hepato, Arginase, Glutamine Synthetase.

LU8	
A	HCC and iron
B	Dysplastic nodule, cirrhosis and iron deposition
C	HCC, cirrhosis and iron deposition
D	HCC and cirrhosis
E	Hepatocellular adenoma and iron deposition

Case LU8 73M

Cirrhosis. Left lateral lesion, ?HCC. Ferritin 1708. AFP 5, CEA 2, CA19-9 42.

Also: Reticulin, Perls, van Gieson, Hepato, Arginase, Glutamine Synthetase.



Consensus complete responses would include, HCC, background cirrhosis and background siderosis.

Possibility of genetic haemochromatosis suggested by 65, just under consensus.

Case LU8 73M

Cirrhosis. Left lateral lesion, ?HCC. Ferritin 1708. AFP 5, CEA 2, CA19-9 42.

Also: Reticulin, Perls, van Gieson, Hepato, Arginase, Glutamine Synthetase.

Suggested scoring: for 10 points include, HCC cirrhosis and iron.

Lose 5 marks if no mention of one of these. No cirrhosis (1),

Lose 5 marks No iron (4)

Lose 5 marks neither cirrhosis or iron (1)

Lose 10 marks (score 0) if not giving HCC diagnosis (1)

Observations/potential learning points,

46 offered differentiation comment 20 well 26 moderate

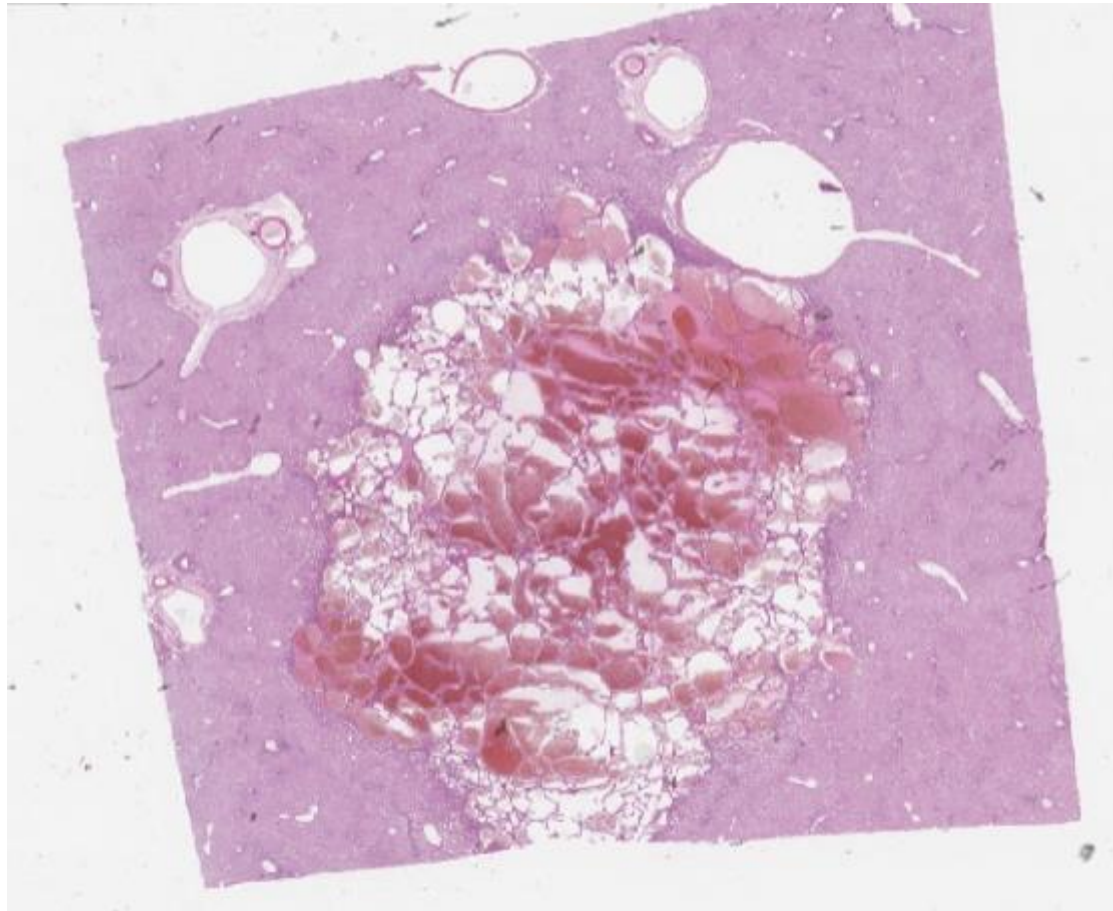
Case LU9 30F

History of COCP.

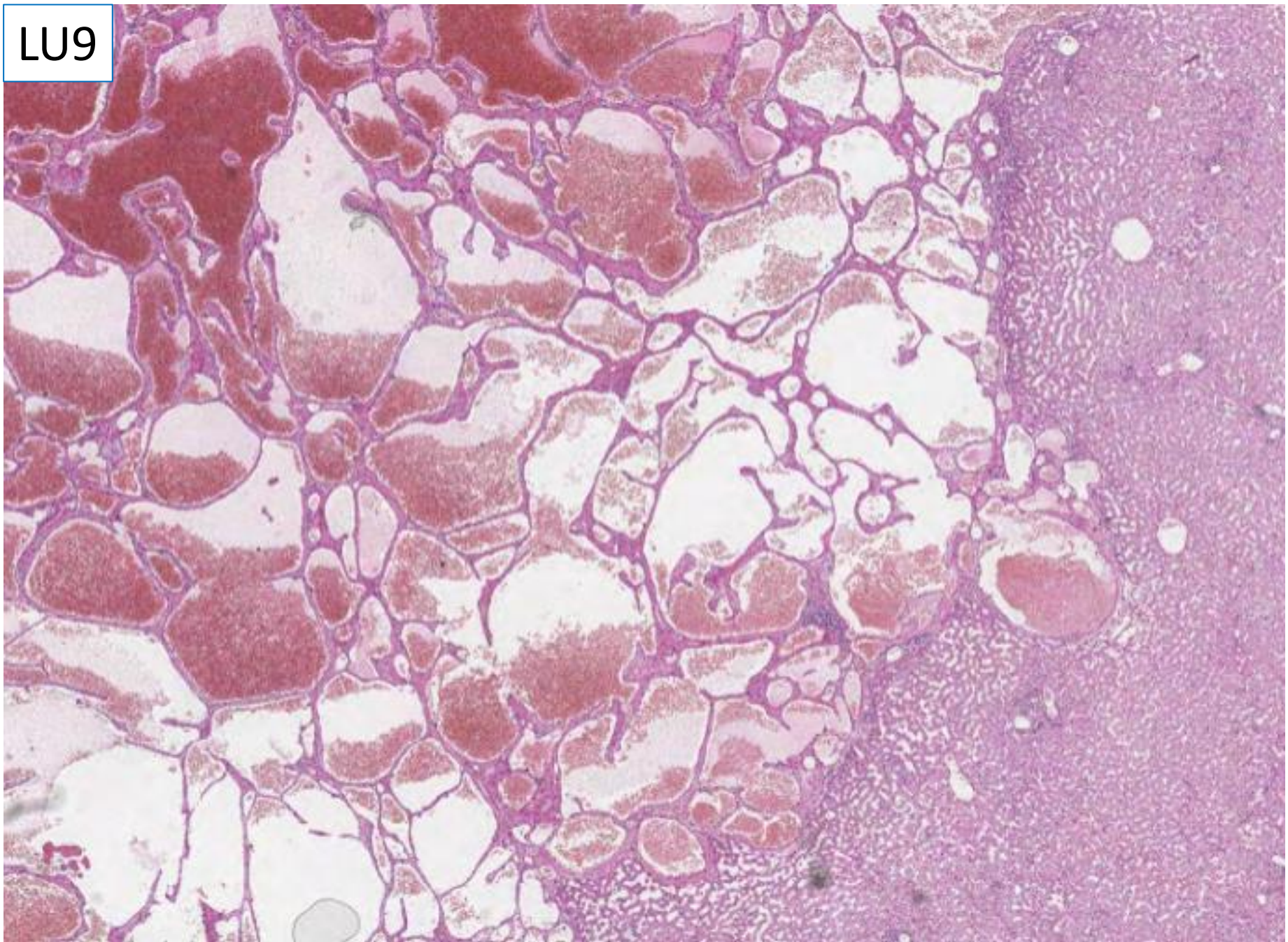
Left lateral liver resection.

Incidental 13 mm well-circumscribed haemorrhagic lesion in resection for larger lesion.

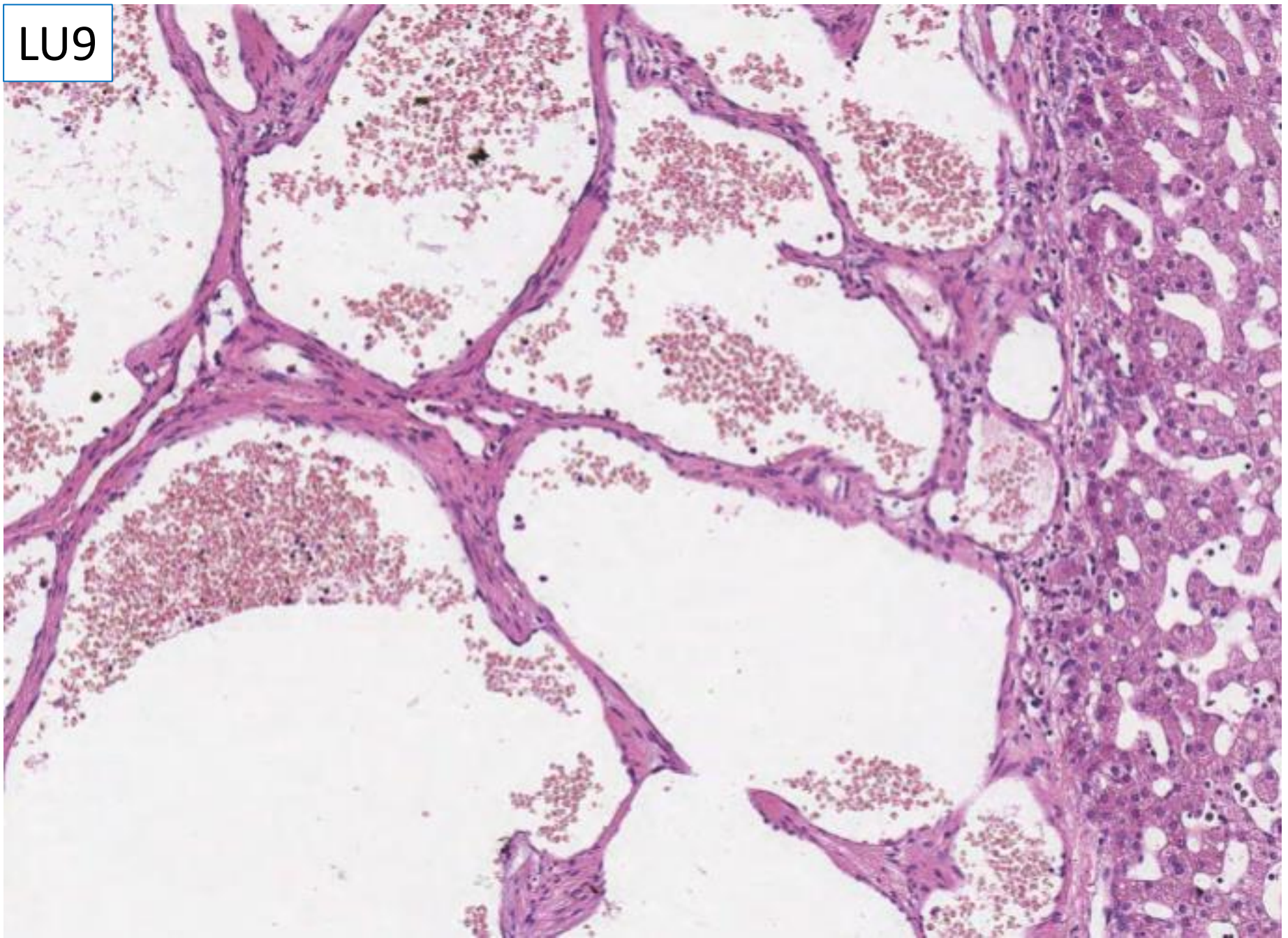
No additional stains



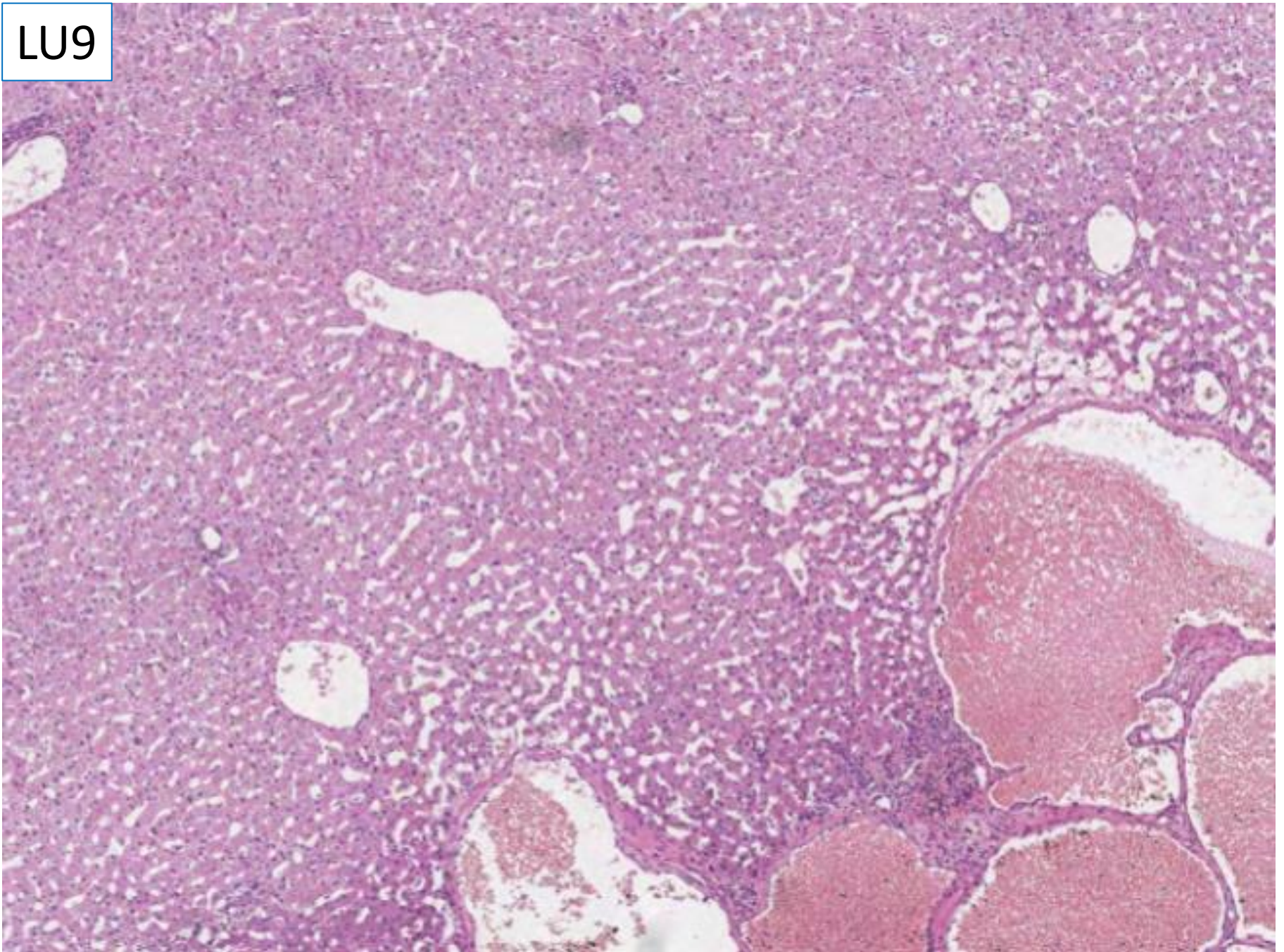
LU9



LU9



LU9



Case LU9 30F

History of COCP. Left lateral liver resection.

Incidental 13 mm well-circumscribed haemorrhagic lesion in resection for larger lesion.

No additional stains

LU9	
A	Epithelioid haemangioendothelioma
B	Haemangioma/cavernous haemangioma
C	Peliosis hepatis
D	Haemangiomatosis
E	Angiosarcoma

Case LU9 30F

History of COCP. Left lateral liver resection.

Incidental 13 mm well-circumscribed haemorrhagic lesion in resection for larger lesion.

No additional stains

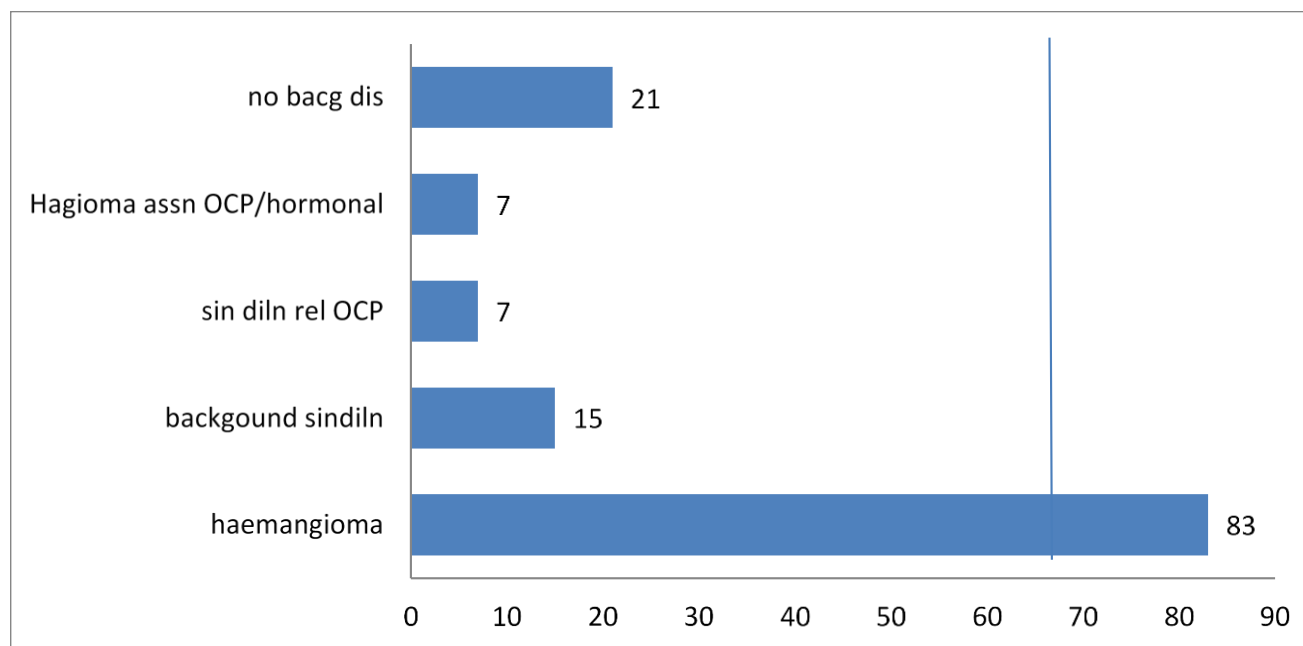
LU9	
A	Epithelioid haemangioendothelioma
B	Haemangioma/cavernous haemangioma
C	Peliosis hepatis
D	Haemangiomatosis
E	Angiosarcoma

Case LU9 30F

History of COCP. Left lateral liver resection.

Incidental 13 mm well-circumscribed haemorrhagic lesion in resection for larger lesion.

No additional stains



Consensus complete responses would include haemangioma or cavernous haemangioma, all participants

All score 10

Case LU9 30F

History of COCP. Left lateral liver resection.

Incidental 13 mm well-circumscribed haemorrhagic lesion in resection for larger lesion.

No additional stains

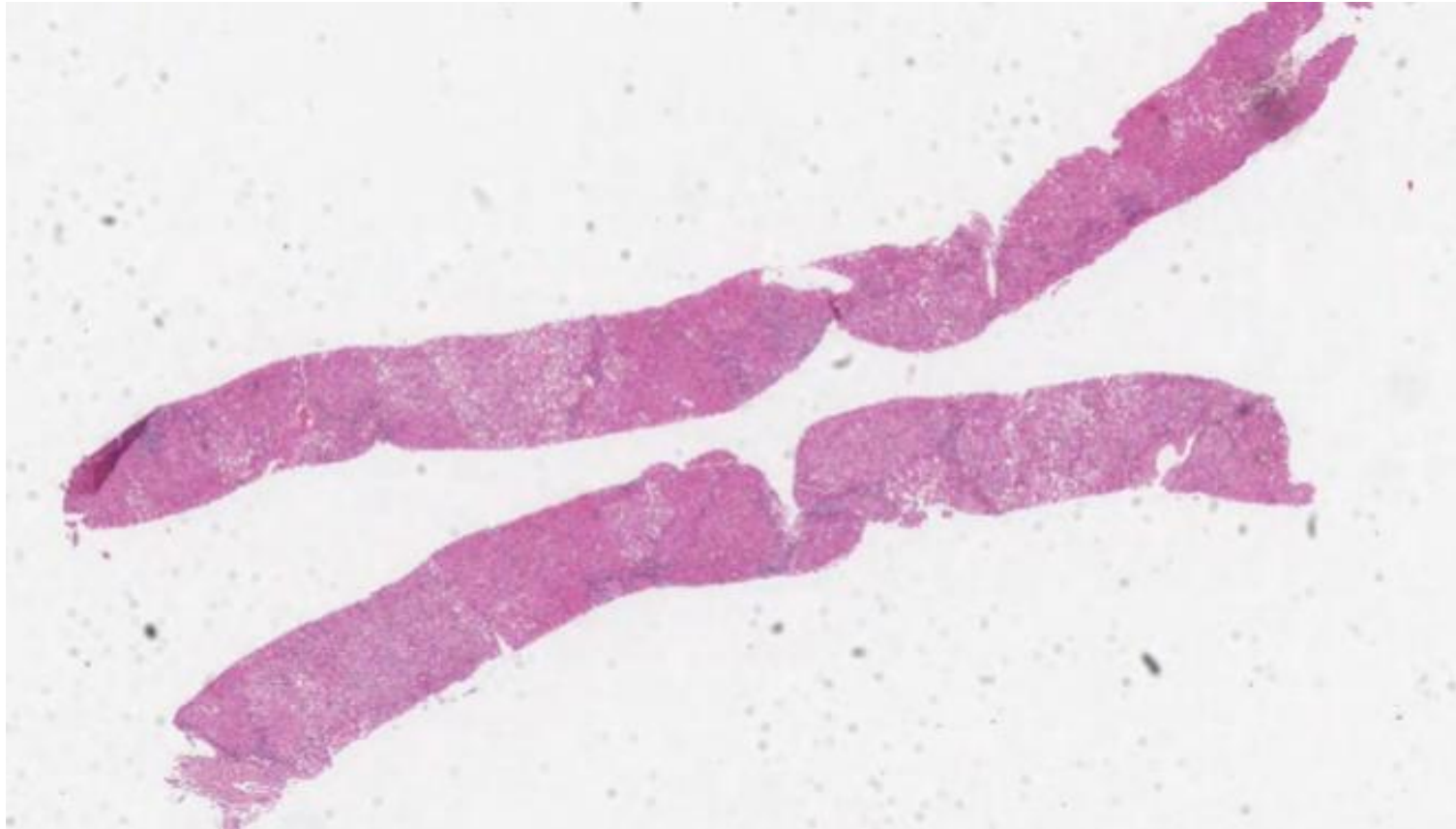
Observations/potential learning points,

Few comments regarding background liver – sinusoidal dilatation possibly OCP related or proximity to space occupying lesion, couple commented haemangioma – hormones may encourage growth but not incidence.

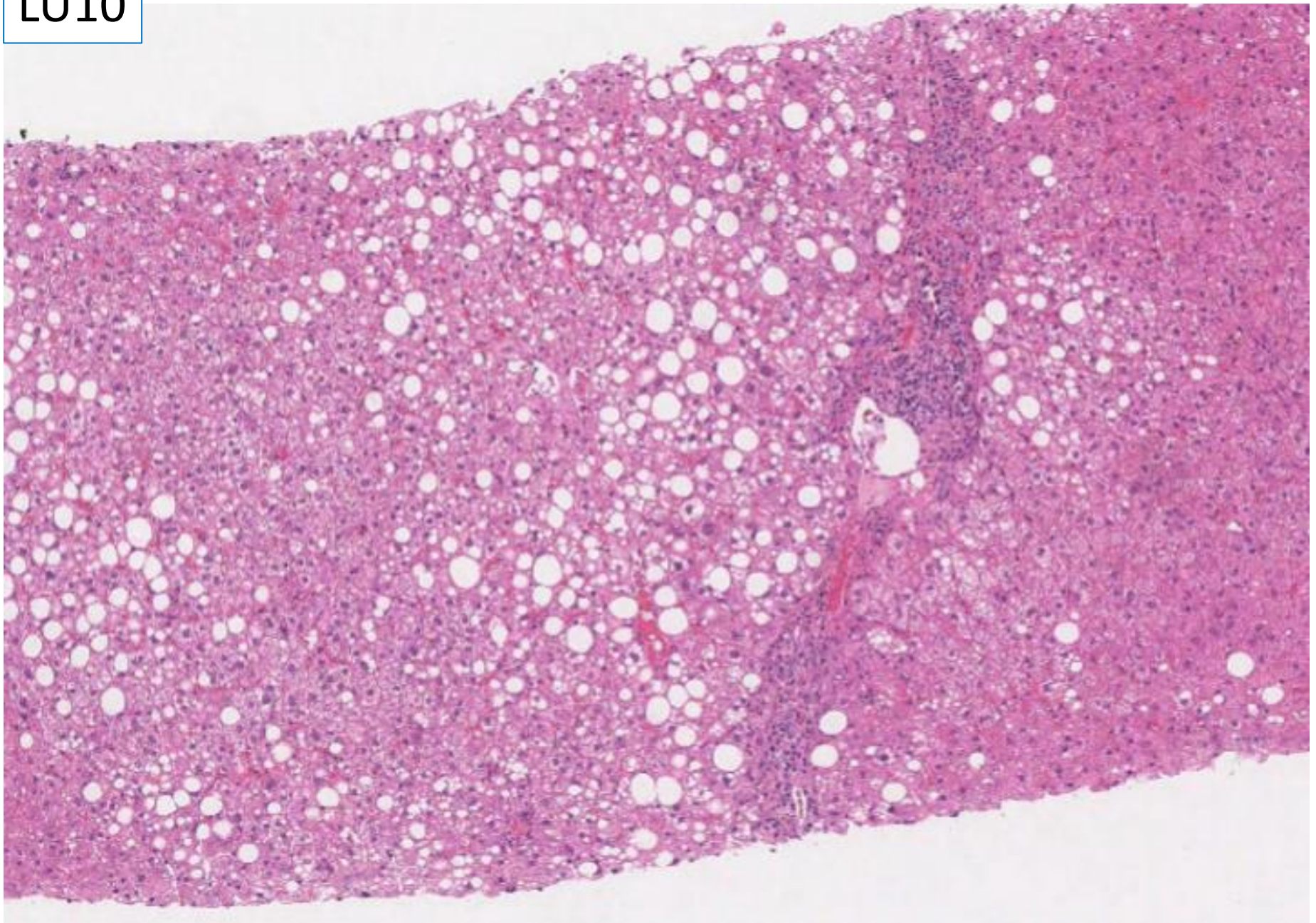
Case LU10 74M

A1AT ?extent of fibrosis.

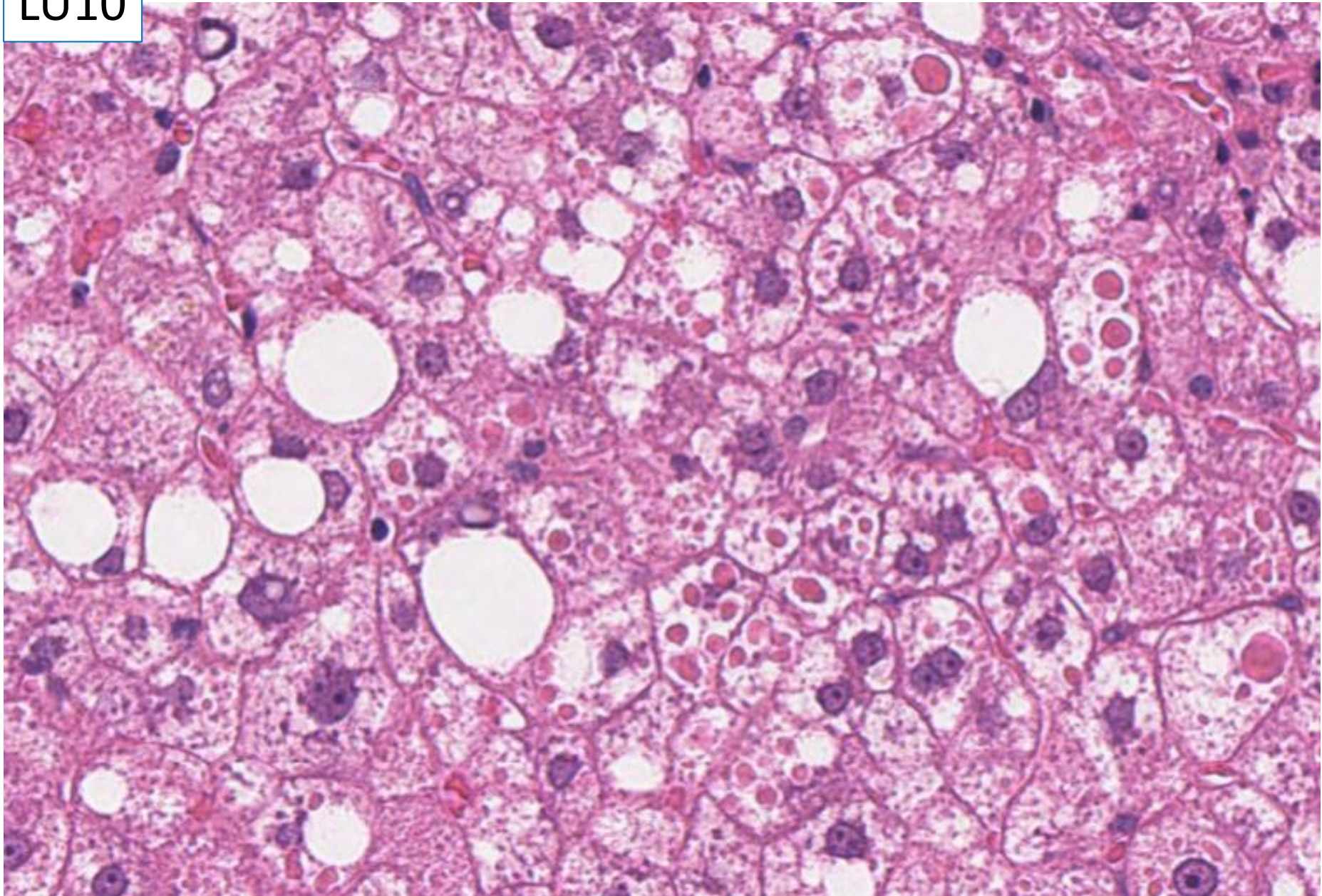
Additional stains: PASD, HVG.



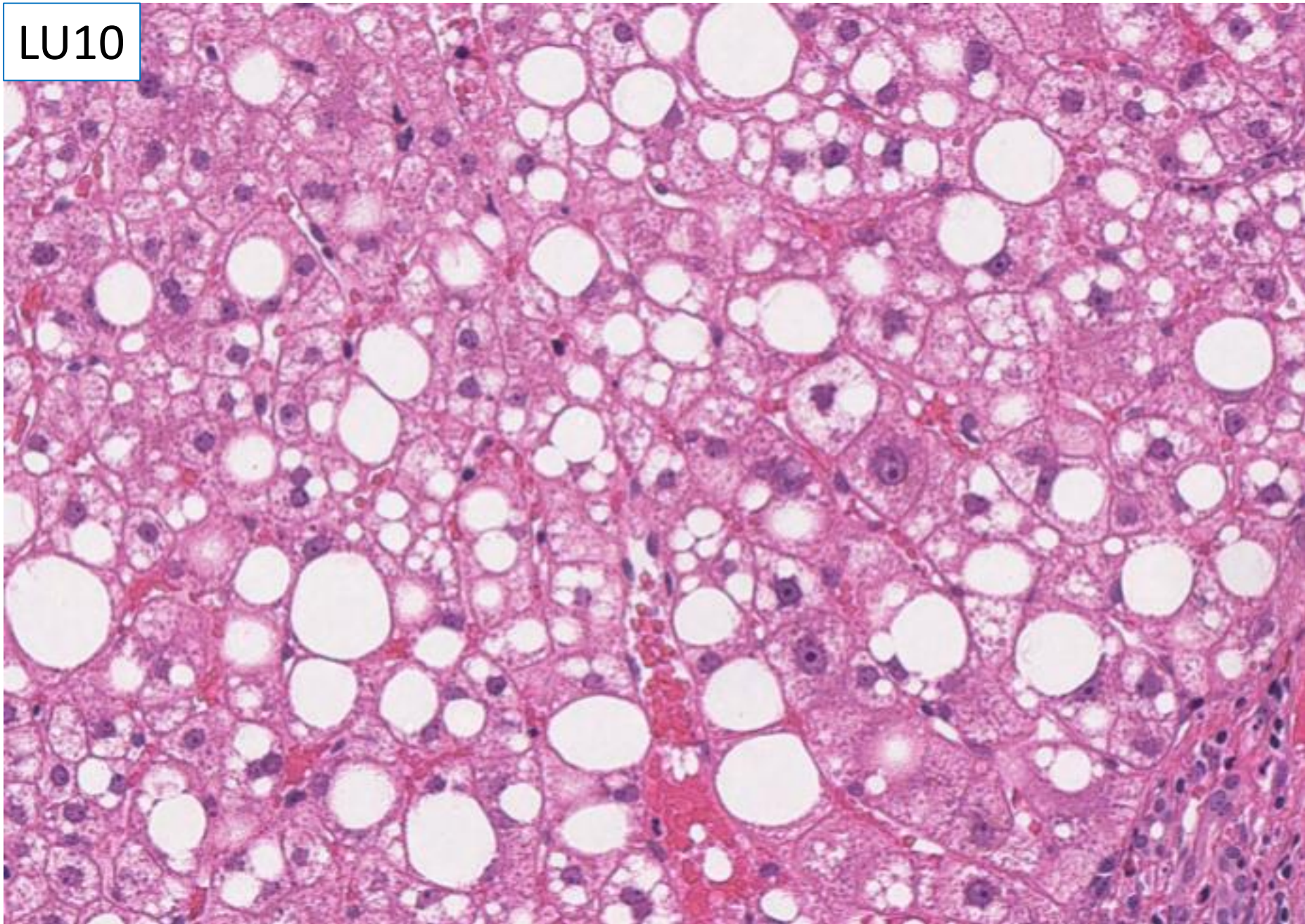
LU10



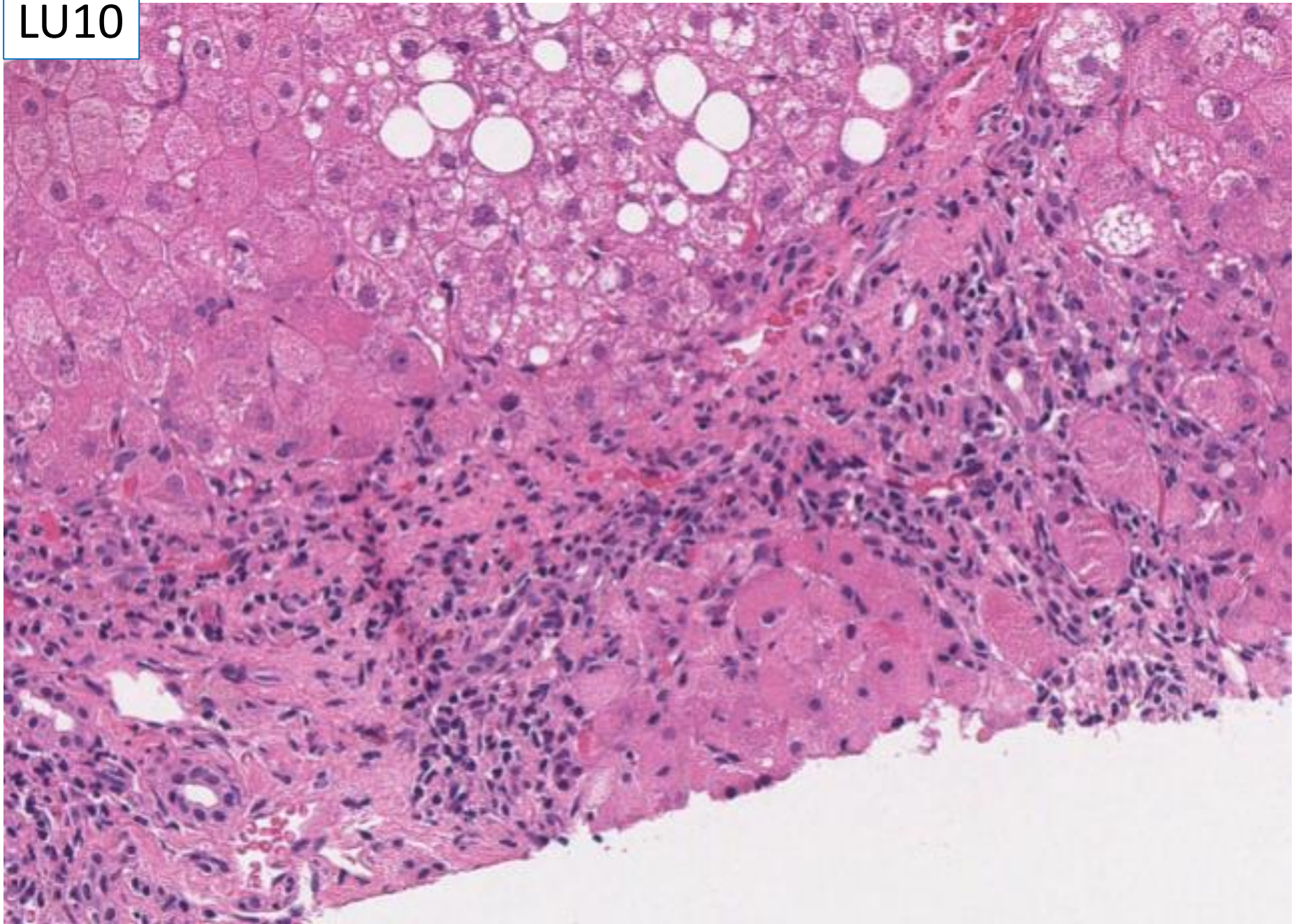
LU10



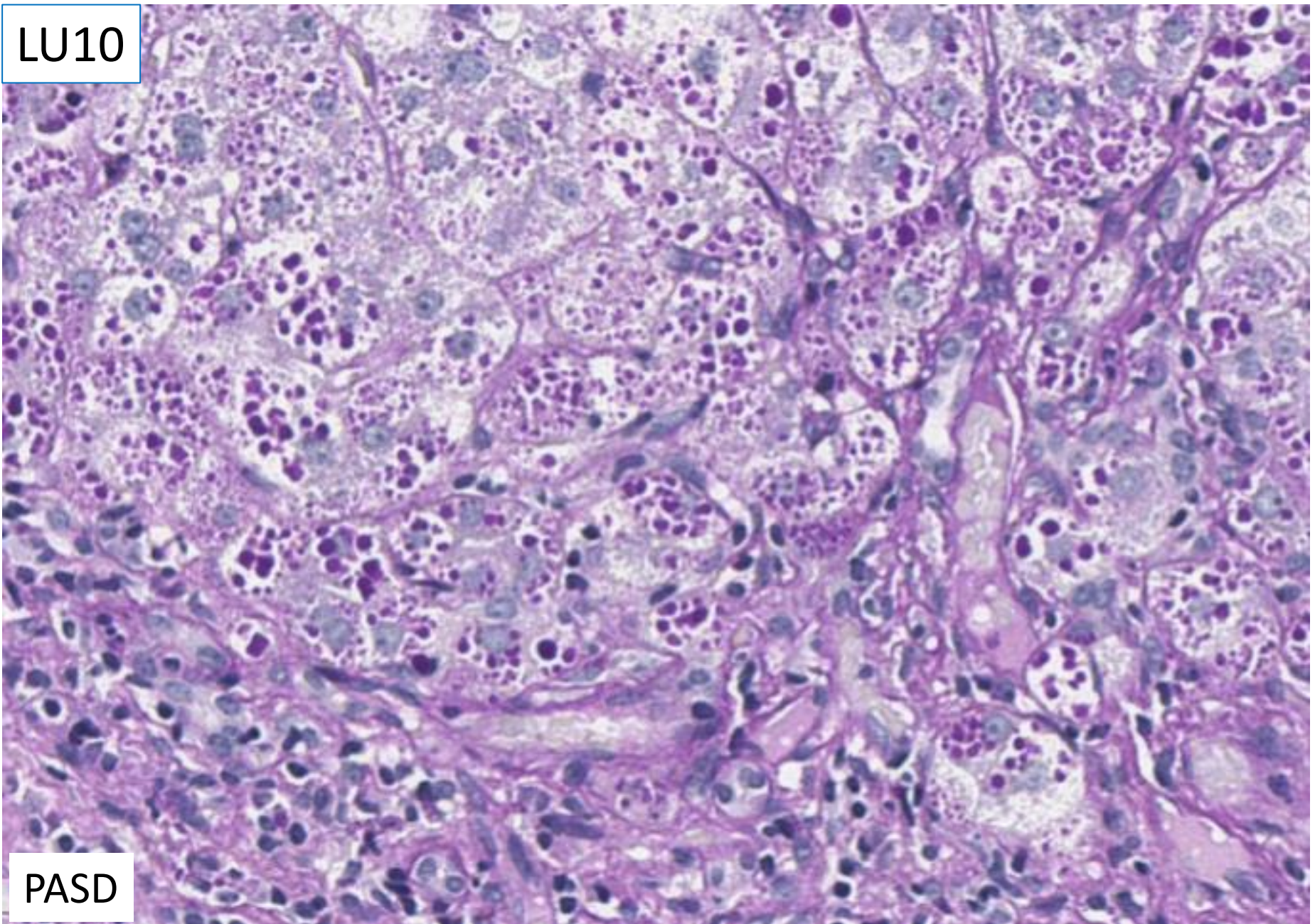
LU10



LU10

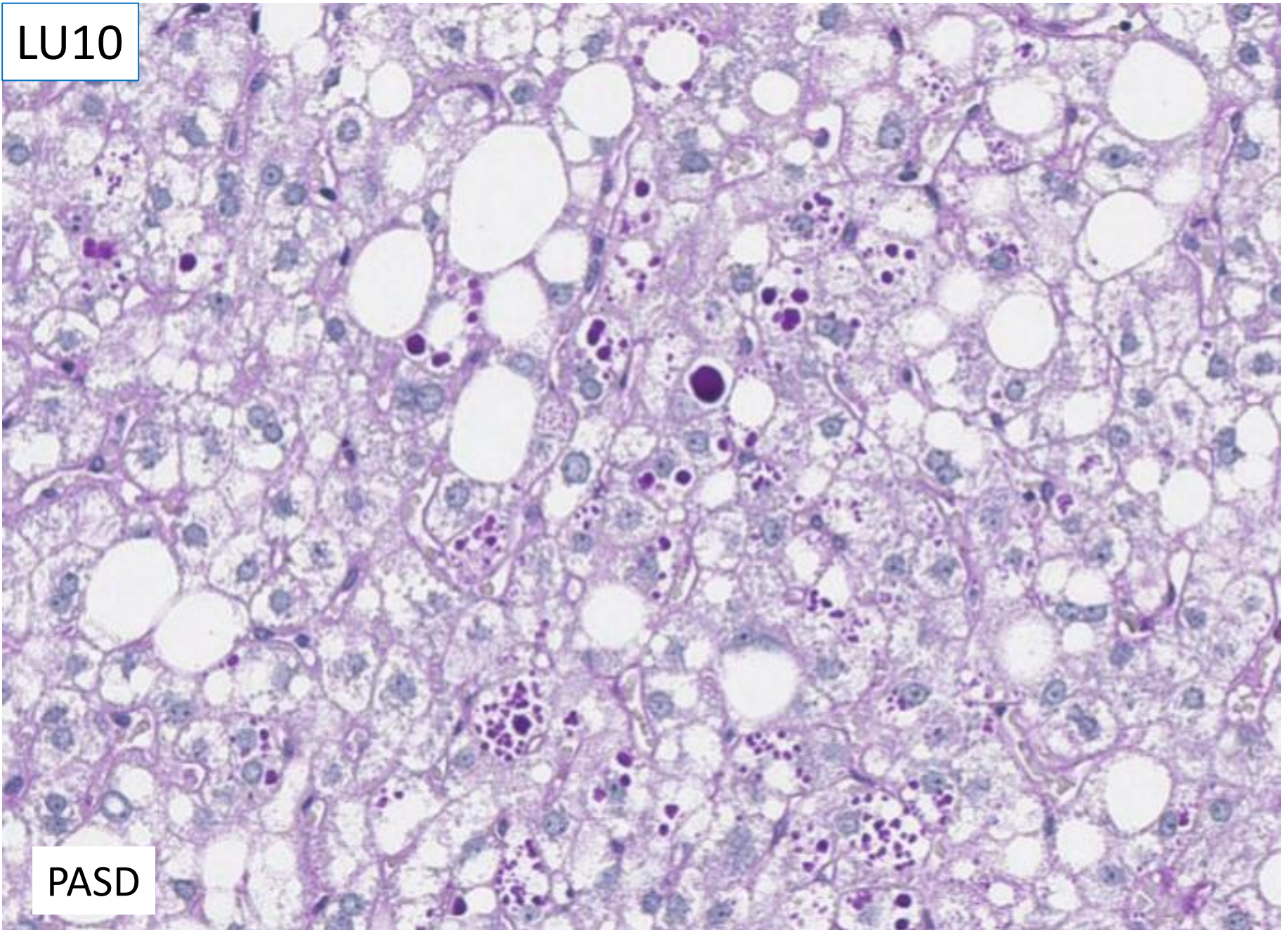


LU10



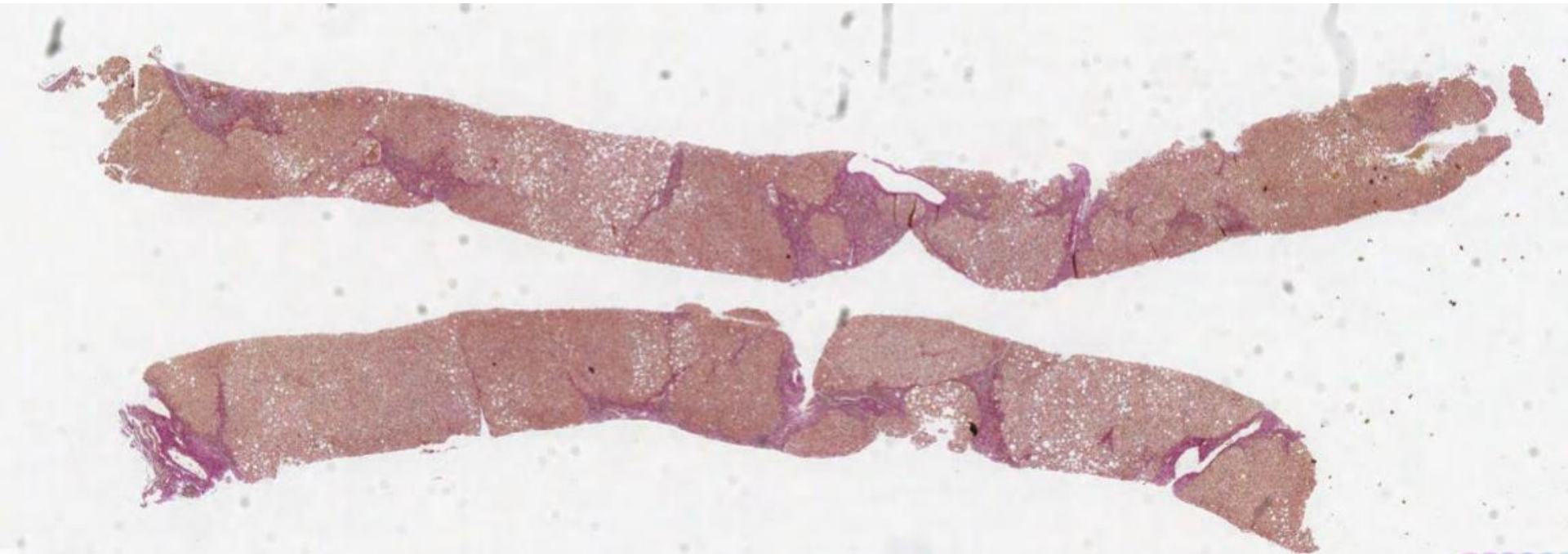
PASD

LU10



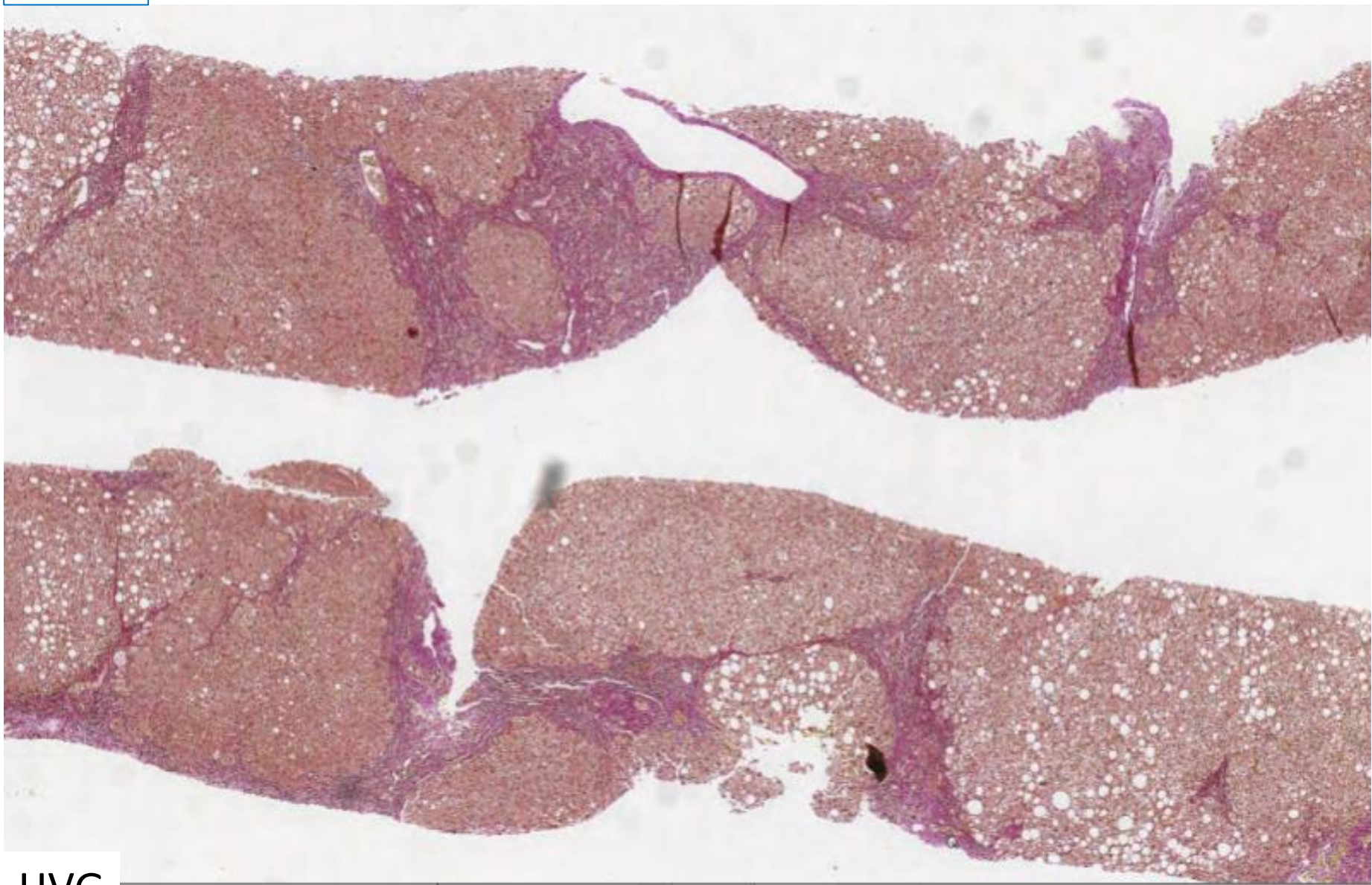
PASD

LU10



HVG

LU10



HVG

Case LU10 74M

A1AT ?extent of fibrosis.

Additional stains: PASD, HVG.

LU10	
A	A1AT accumulation
B	A1AT accumulation, steatosis and severe fibrosis/cirrhosis
C	A1AT accumulation, steatosis and mild fibrosis
D	A1AT-like globules due to alcoholic cirrhosis
E	Chronic hepatitis with severe fibrosis/cirrhosis

Case LU10 74M

A1AT ?extent of fibrosis.

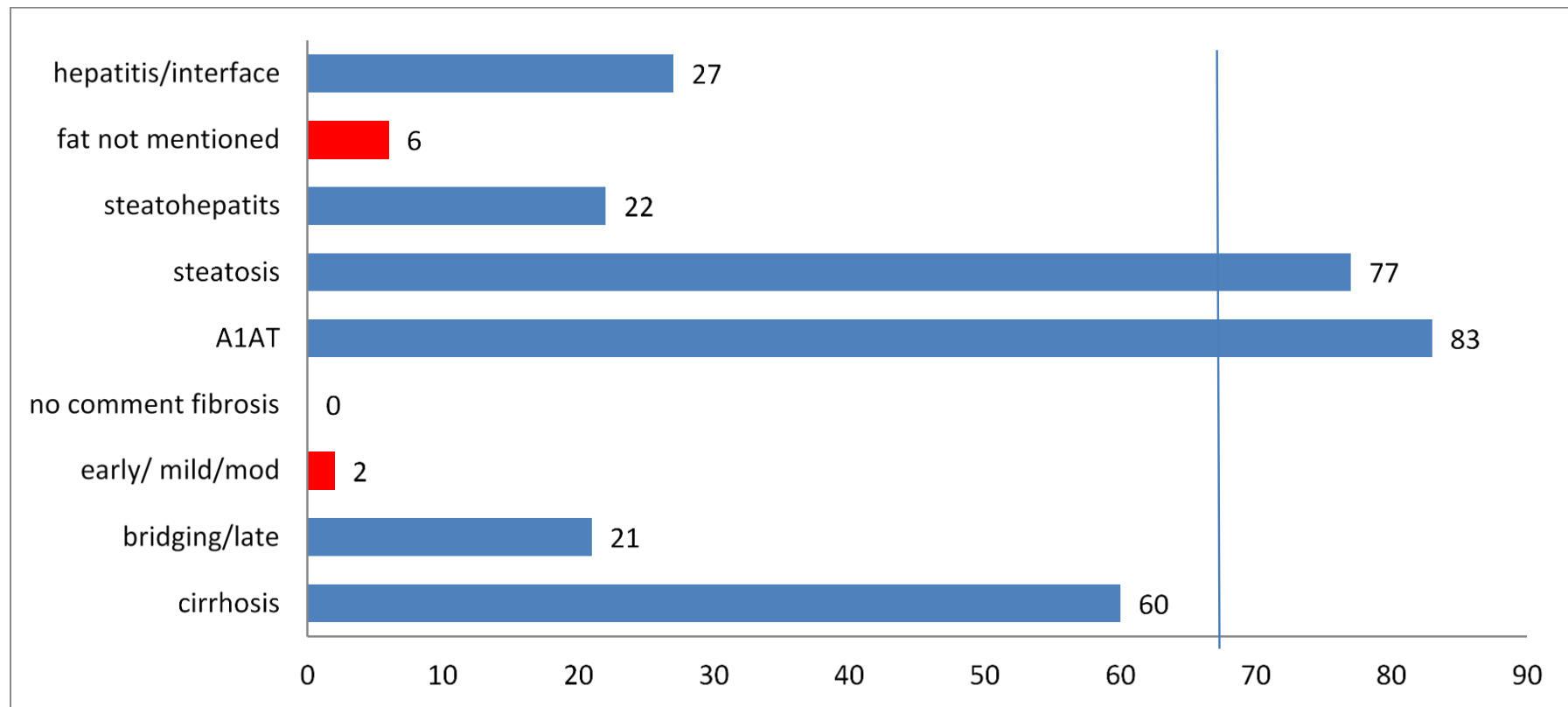
Additional stains: PASD, HVG.

LU10	
A	A1AT accumulation
B	A1AT accumulation, steatosis and severe fibrosis/cirrhosis
C	A1AT accumulation, steatosis and mild fibrosis
D	A1AT-like globules due to alcoholic cirrhosis
E	Chronic hepatitis with severe fibrosis/cirrhosis

Case LU10 74M

A1AT ?extent of fibrosis.

Additional stains: PASD, HVG.



Consensus complete responses would include ; A1AT disease (83), comment on fibrosis (83, 60 cirrhosis 23 less than but often 'severe' only 2 saying 'early fibrosis') and mention of steatosis (77)

Case LU10 74M

A1AT ?extent of fibrosis.

Additional stains: PASD, HVG.

Suggested scoring: for 10 points include A1ATD, fibrosis cirrhosis or bridging, and steatosis

Lose 5 marks if no mention of steatosis? (6)

meeting 16.10.19 agreed also lose 5 for early fibrosis as there is consensus for at least moderate/bridging (2)

Observations/potential learning points,

‘cirrhosis’ includes ‘early’ ‘developing’ ‘incomplete’ ‘evolving’ sometimes the word ‘cirrhosis’ not used – numbers or ‘nodules’ used – have to make assumptions.

Recorded as ‘hepatitis’ if used as word or any mention interface or moderate inflammation

27 mentioning hepatitis either ‘chronic hepatitis’ as the major pattern of damage (5, one possible AI) or moderate portal infiltrate +/- interface.

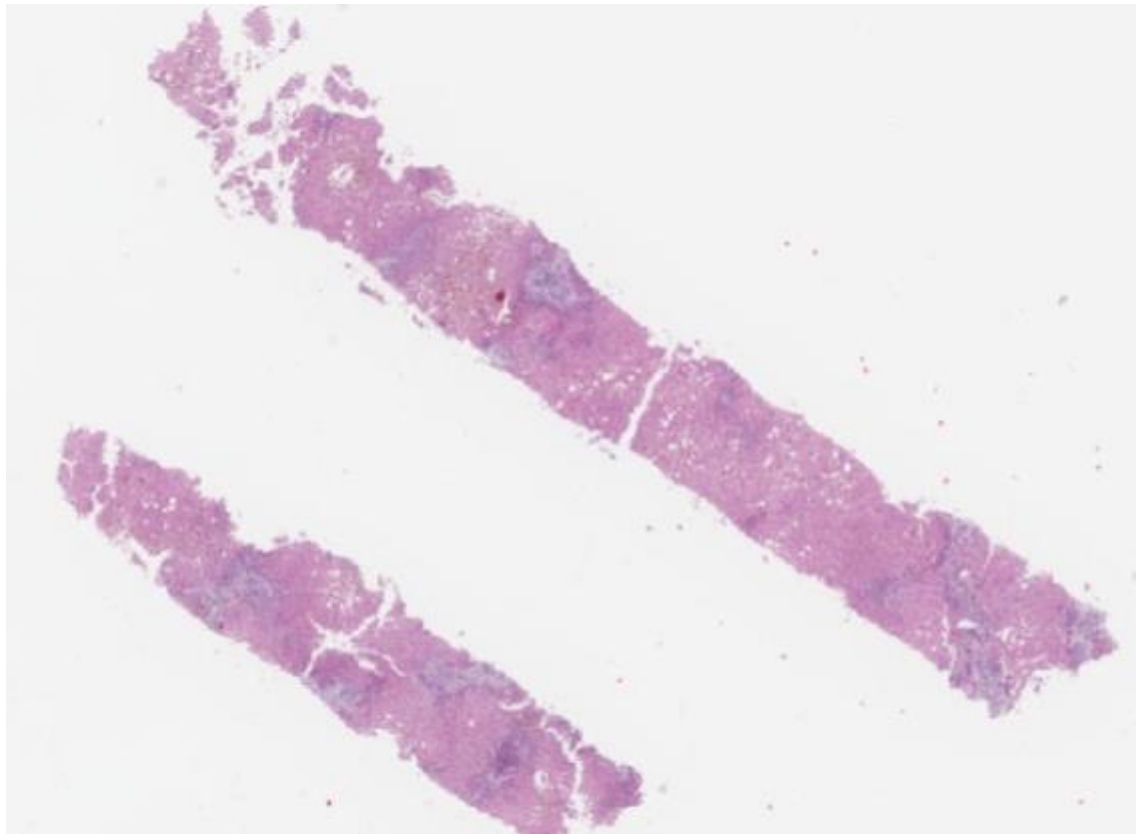
22 thought amounted to steatohepatitis

Case LU11 66M

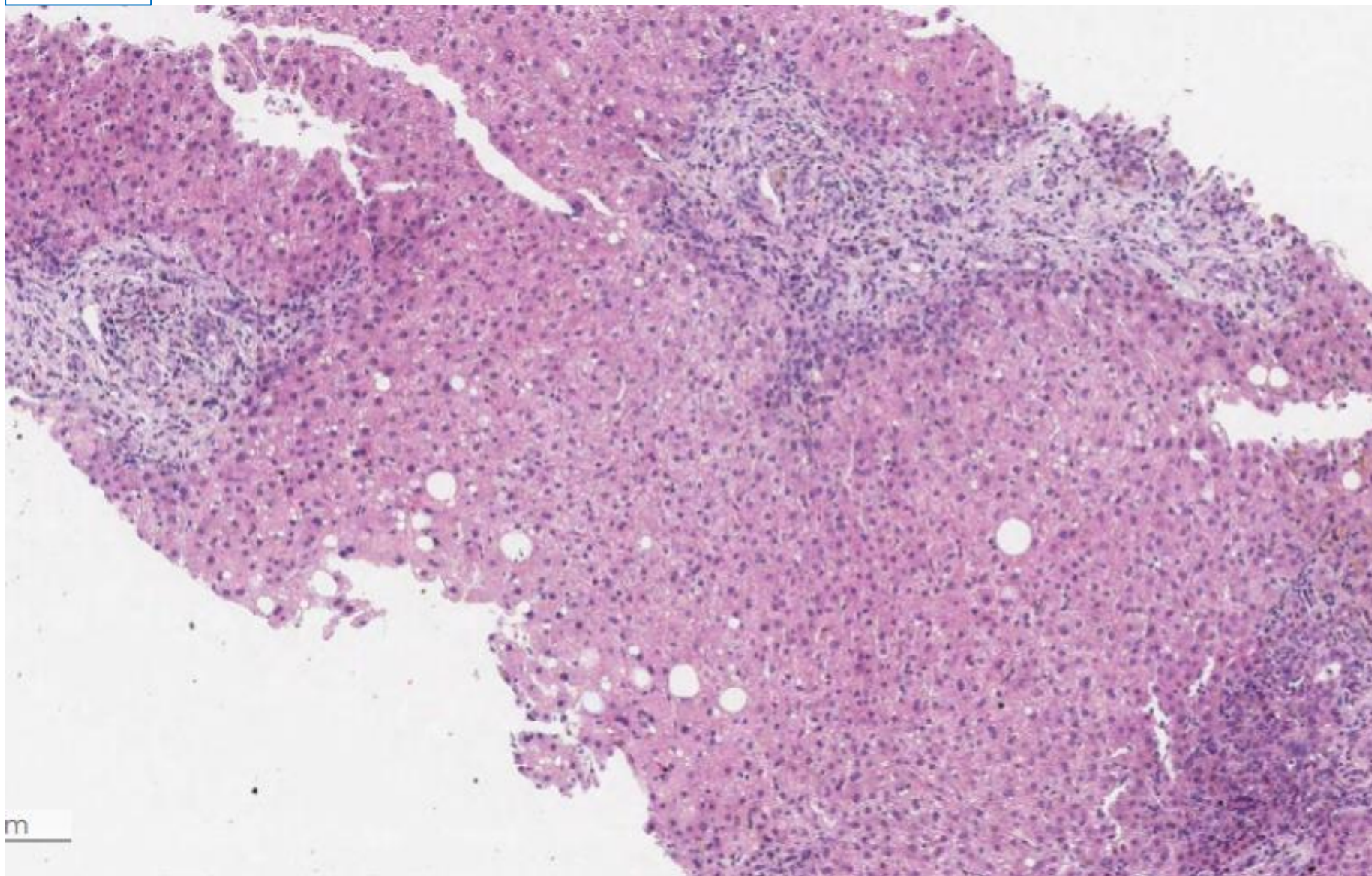
Abnormal LFTs. ANCA positive. Imaging normal. ? inflammation ? cirrhosis.

Additional information - presented 3 weeks ago with 2 day history of nausea and vomiting; found to have deranged liver function tests. Had taken 2 courses of flucloxacillin from GP. Usual medications: levothyroxine, simvastatin, atenolol, ranitidine. Bili 100, ALT 878, Alk phos 193.

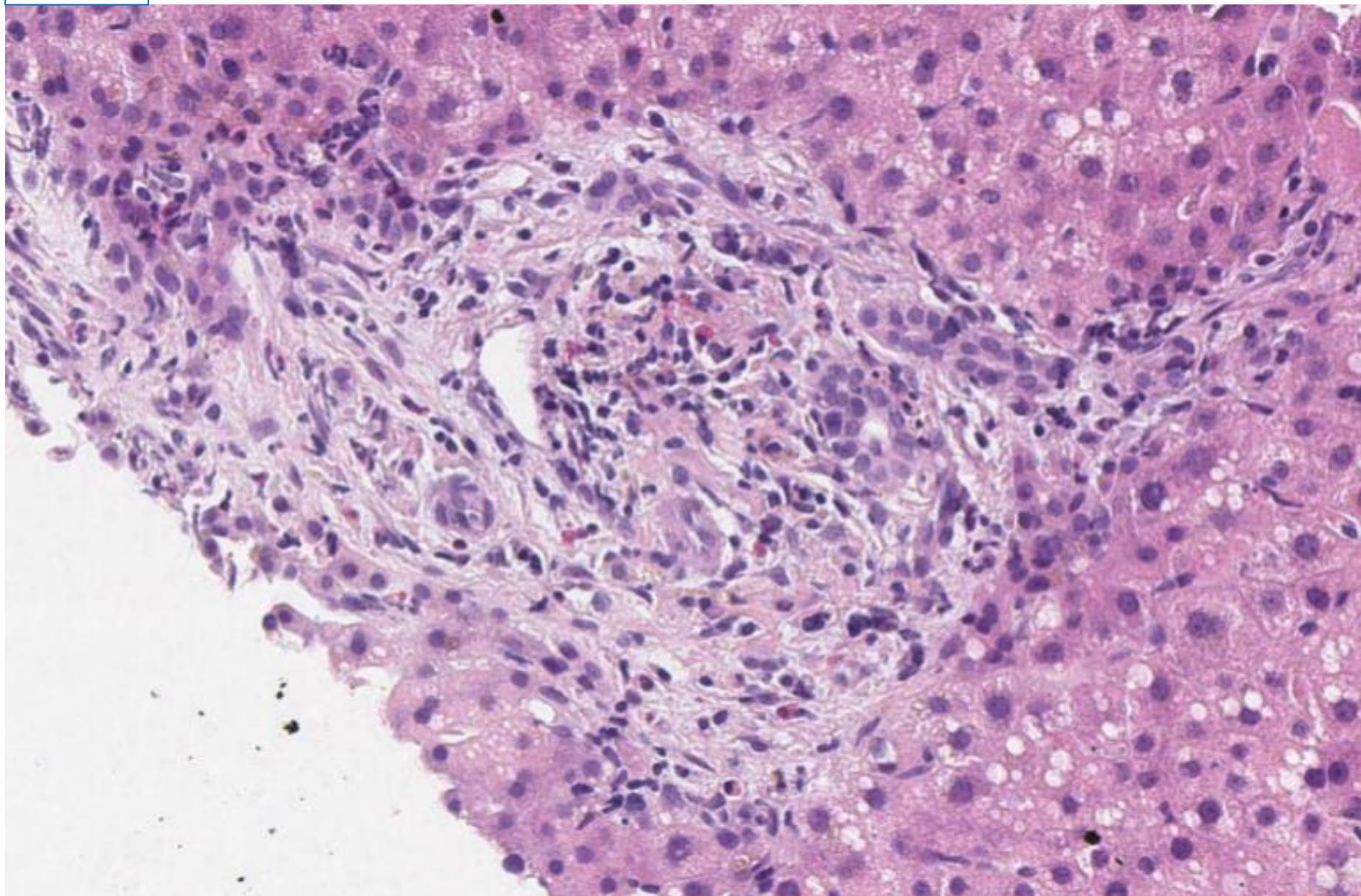
Additional stains: Retic, van Gieson, PASD.



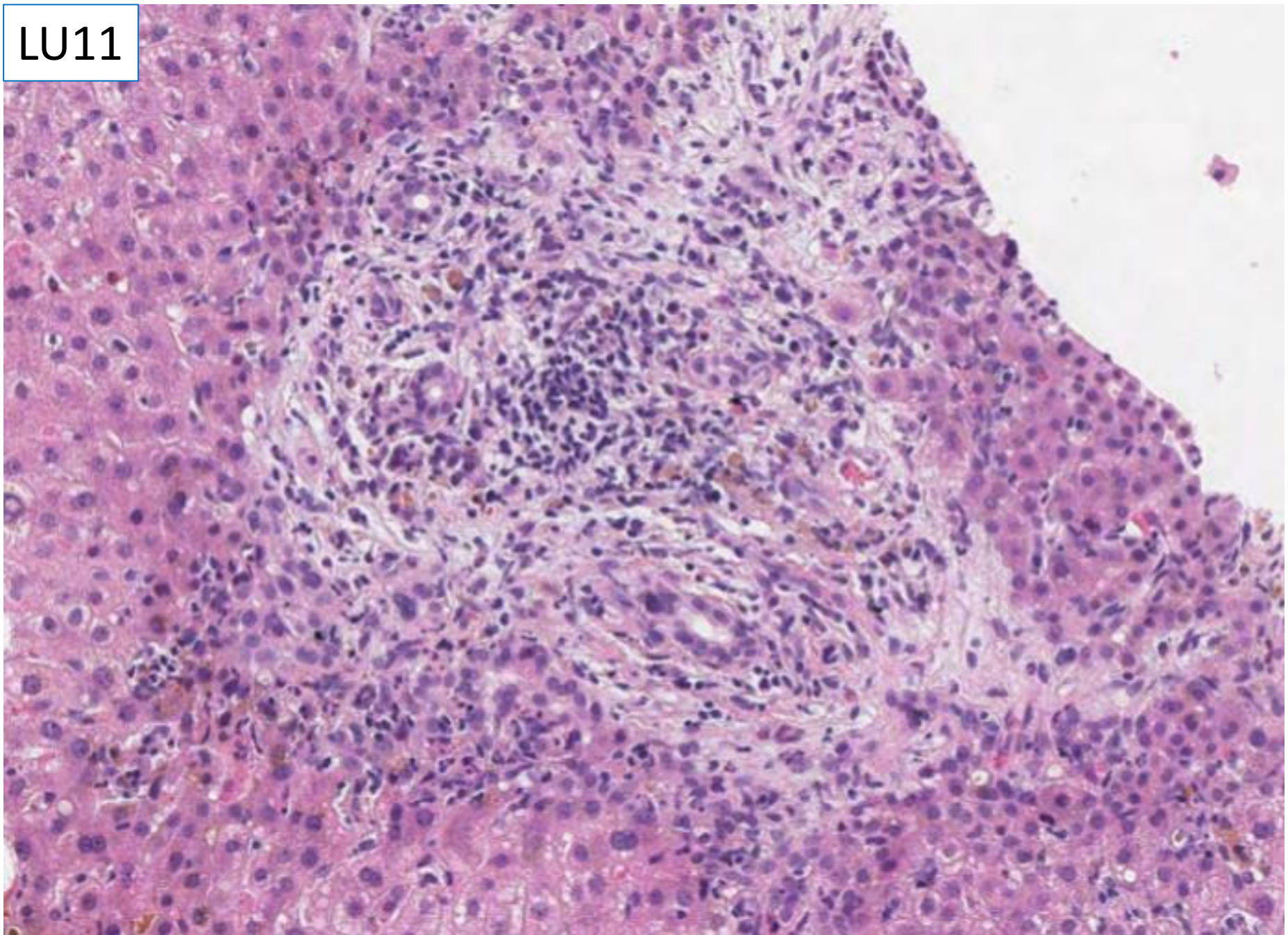
LU11



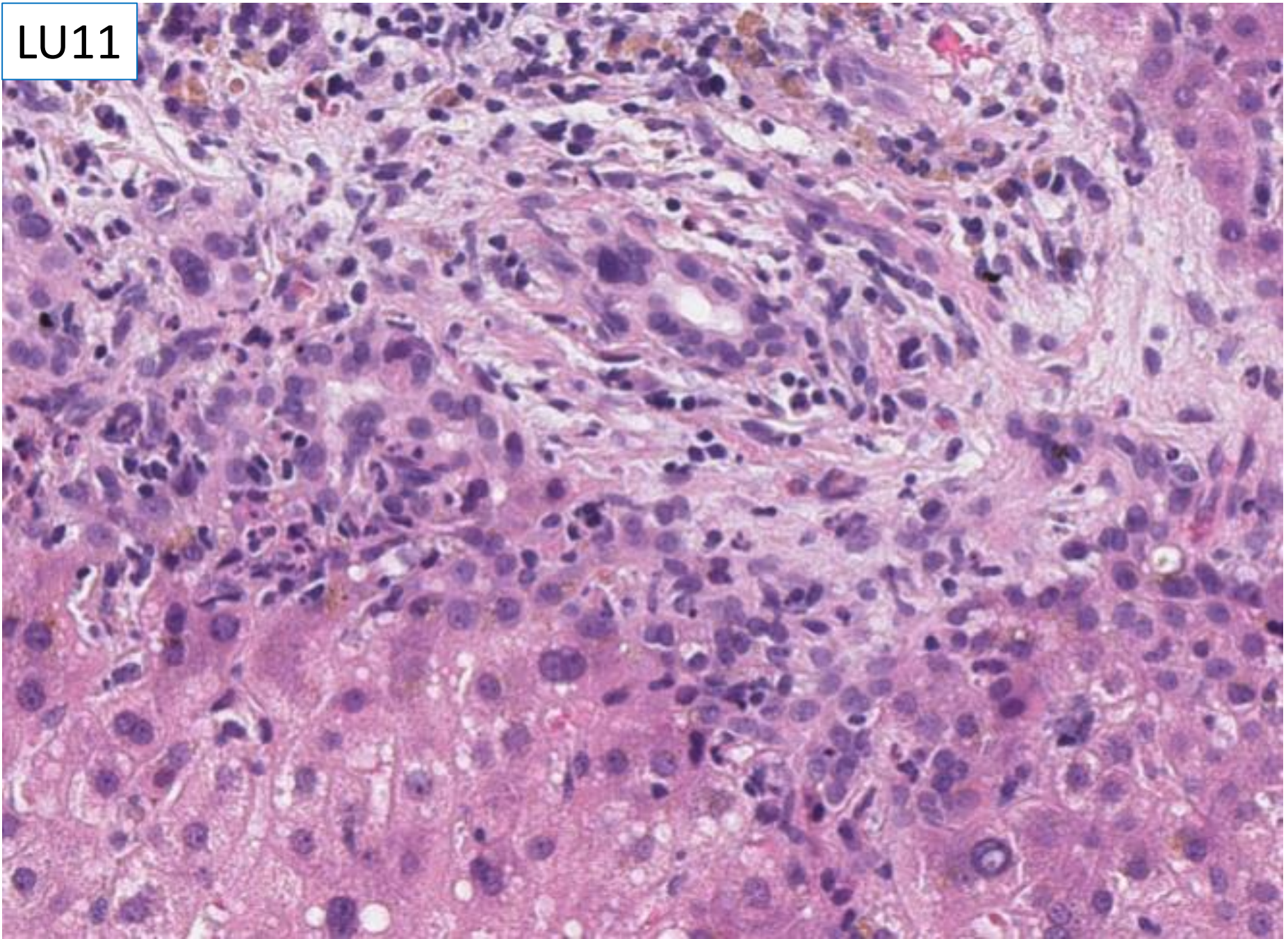
LU11



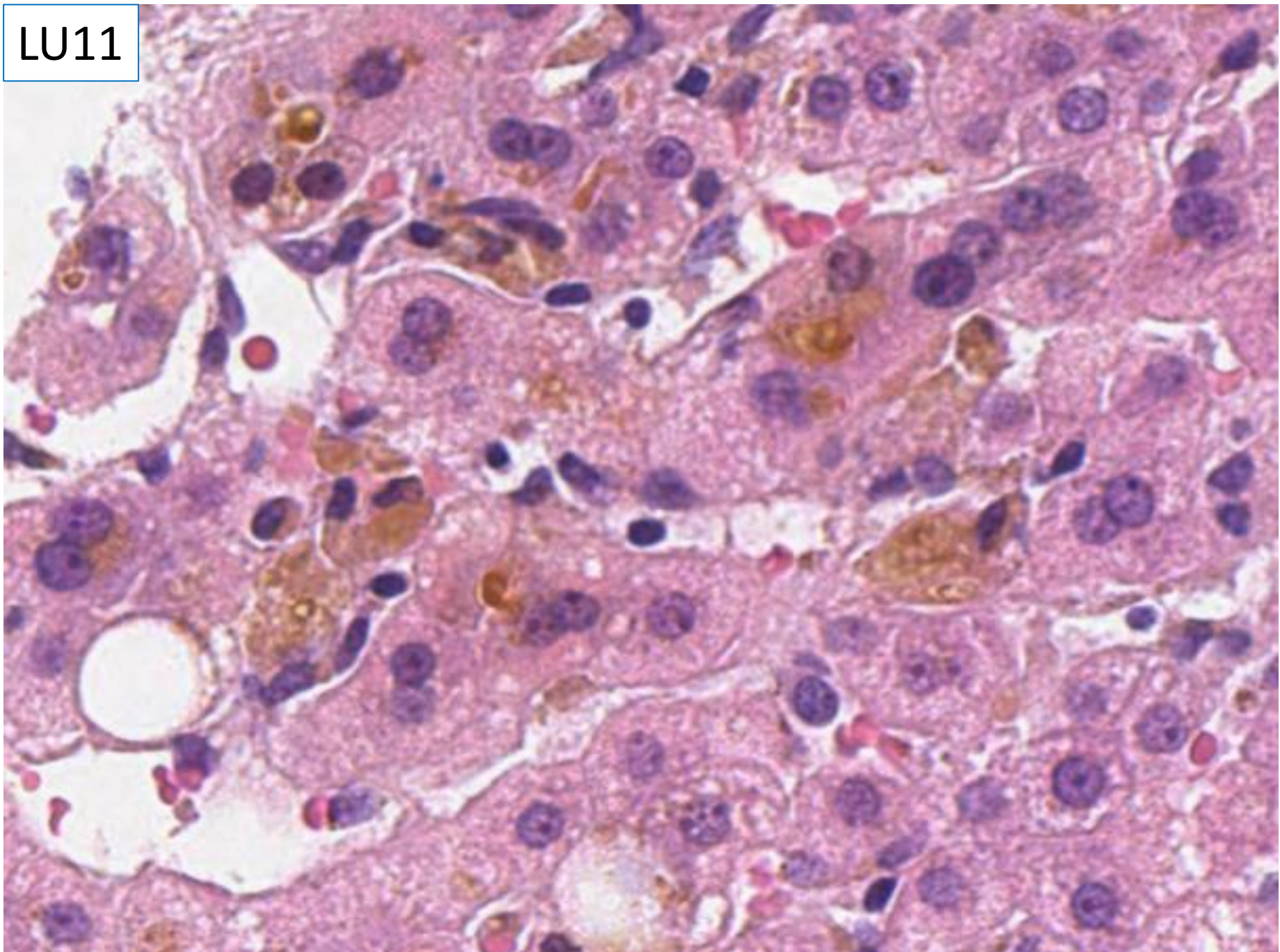
LU11



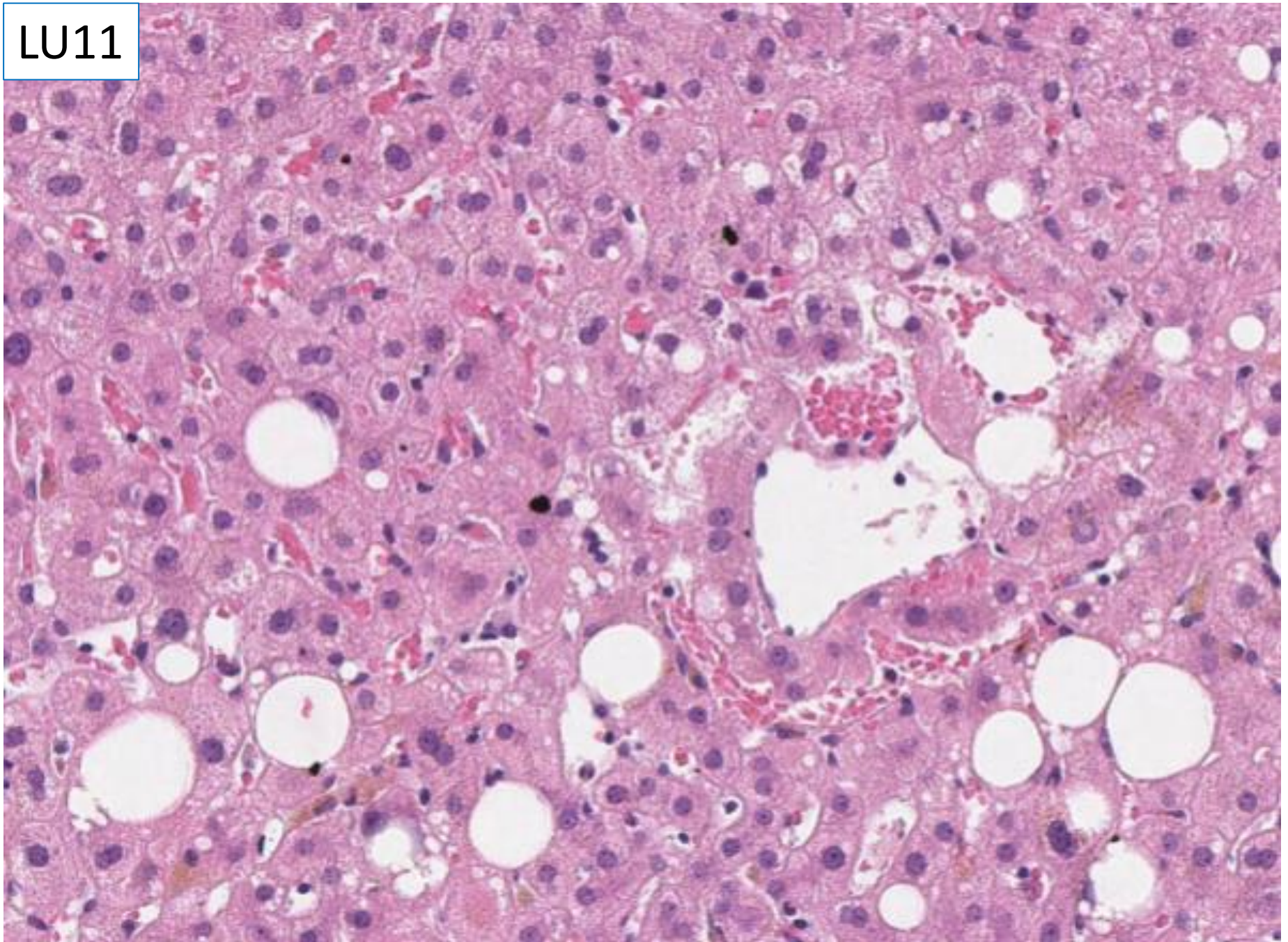
LU11



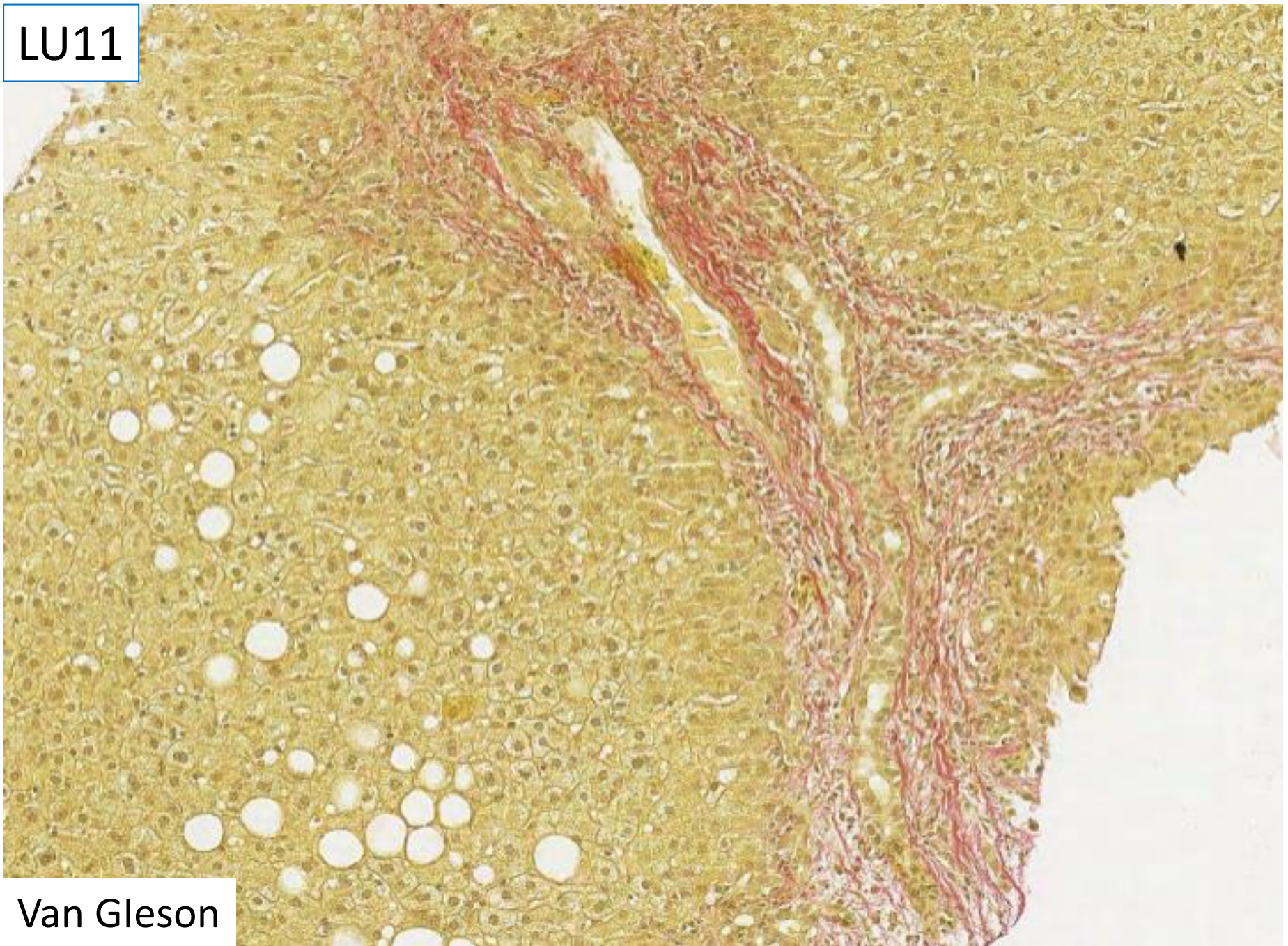
LU11



LU11

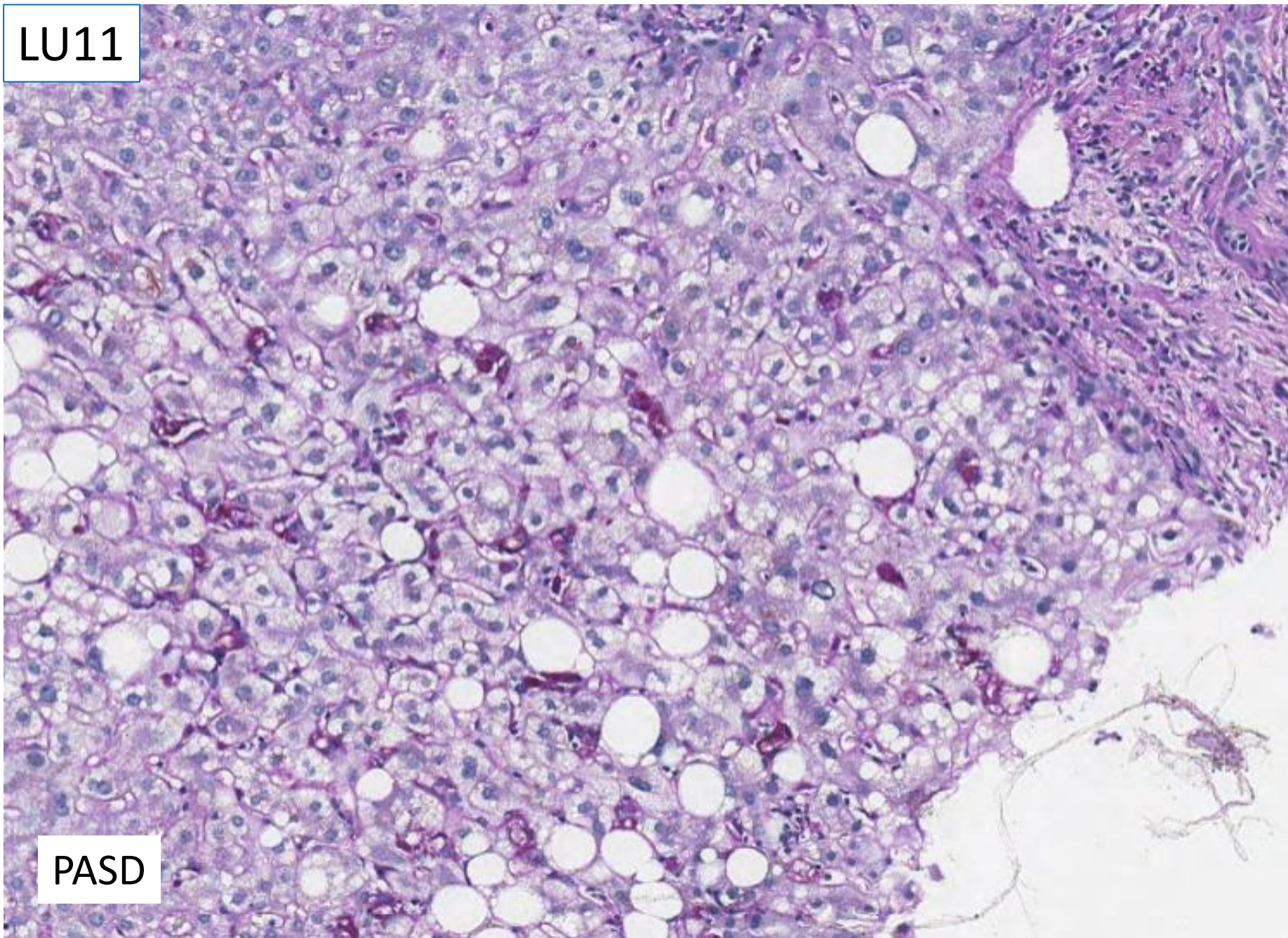


LU11



Van Gleson

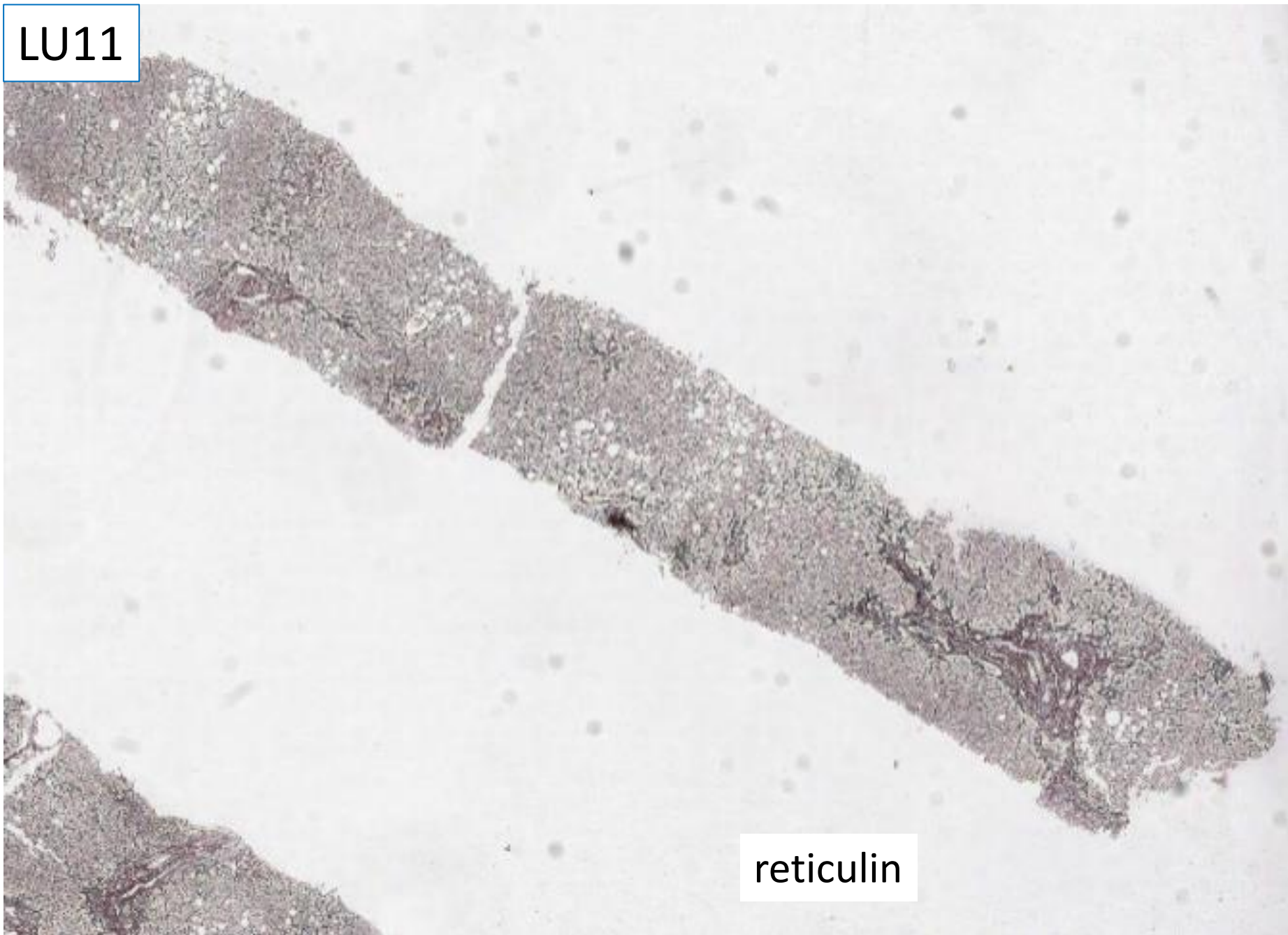
LU11



PASD

LU11

reticulin



Case LU11 66M

Abnormal LFTs. ANCA positive. Imaging normal. ? inflammation ? cirrhosis.

Additional information - presented 3 weeks ago with 2 day history of nausea and vomiting; found to have deranged liver function tests. Had taken 2 courses of flucloxacillin. . Bili 100, ALT 878, Alk phos 193.

Additional stains: Retic, van Gieson, PASD.

LU11	
A	Chronic hepatitis, possible AIH
B	Biliary features most likely drug
C	Biliary features most likely biliary tract disease e.g. PSC or large duct obstruction
D	Biliary features could be drug can't exclude biliary tract disease/obstruction
E	Cholestatic hepatitis

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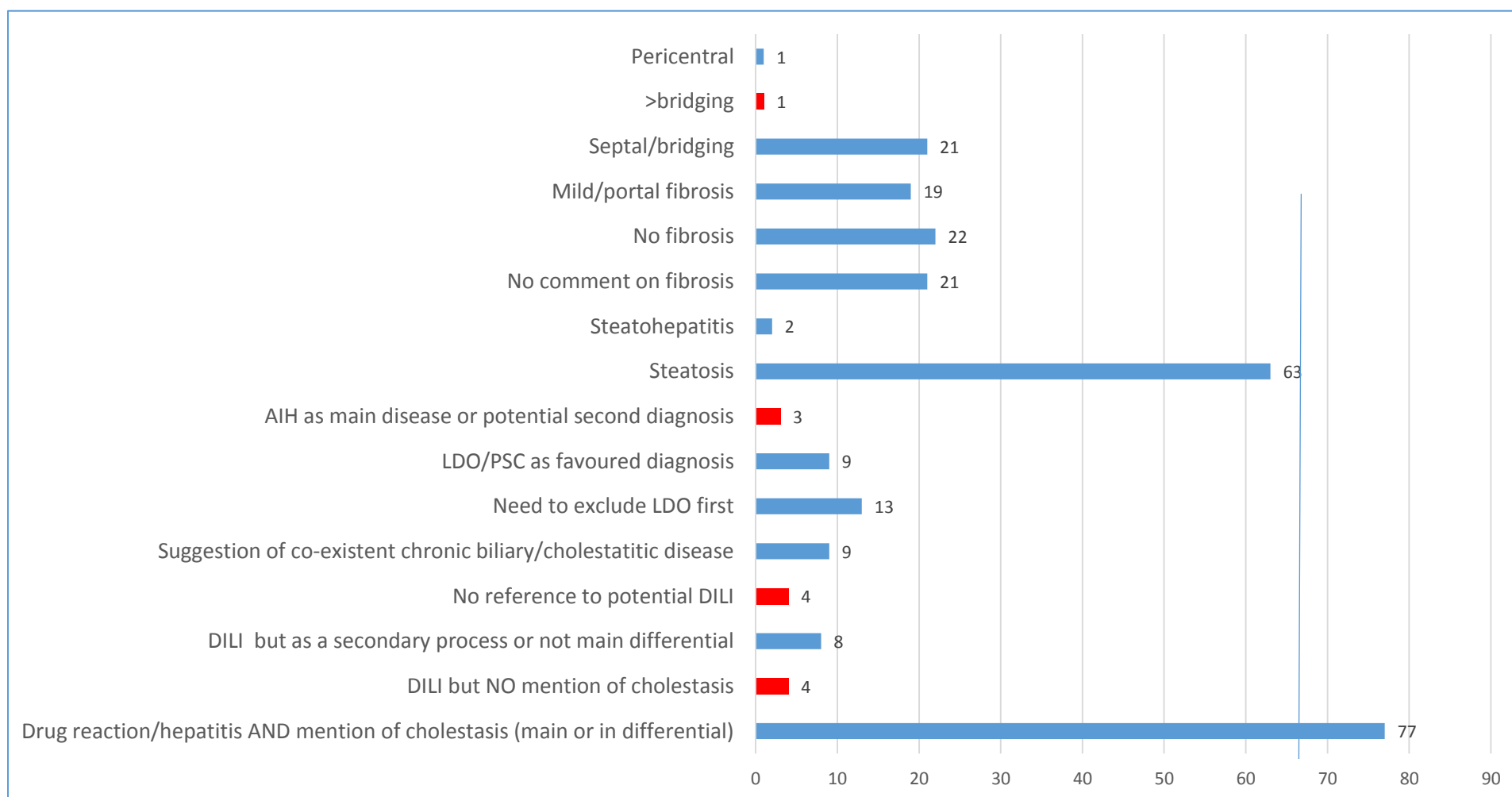
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Consensus complete responses would include DILI (including drug induced hepatitis and drug reaction) with mention of cholestasis

51 people mentioned cholestasis/cholestatic hepatitis in text/diagnostic summary

76 people mentioned cholestasis in histological description

64 people mentioned flucloxacillin or drug reaction (41 referenced flucoxacin specifically)

Meeting 16.10.19 PK to revisit. Tricky case. Felt OK to lump together cholestasis and cholestatic hepatitis or drug induced hepatitis, not unreasonable to have LBDO or biliary tract disease in DD

Case LU11 66M

scoring: for 10 points Drug induced reaction with mention of cholestasis anywhere.

Lose 5 marks DILI/flucloxacillin mentioned but no mention of cholestasis

Lose 5 marks if favour large duct obstruction (+/- related to PSC) as primary cause but possible drug reaction mentioned (concurrent, cholestasis accentuated or precipitated by drug)

Lose 5 marks if AIH as probable/possible diagnosis but mention of potential DILI/cholestasis

Lose 10 marks (score 0) if no reference DILI as likely or possible cause

Comments

- Range of fibrosis comments in this case
- Several respondents suggested chronic cholestatic disease or acute on chronic, or fluclox related cholestasis superimposed on some other liver disease. Wide spread of responses – but could be suitable for scoring based on above.

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Follow up information: clinical follow up – improved and LFTs returned to normal within a few weeks. Biliary imaging was normal.

Learning point – that antibiotic cholestatic DILI can mimic large bile duct obstruction very closely.

If it wasn't for the clinical history of flucloxacillin, the histological diagnosis would be large duct obstruction – so biliary imaging to exclude large duct obstruction should also be recommended.

Case LU12 45F

Left lateral segmentectomy laparoscopically. ?FNH ?adenoma

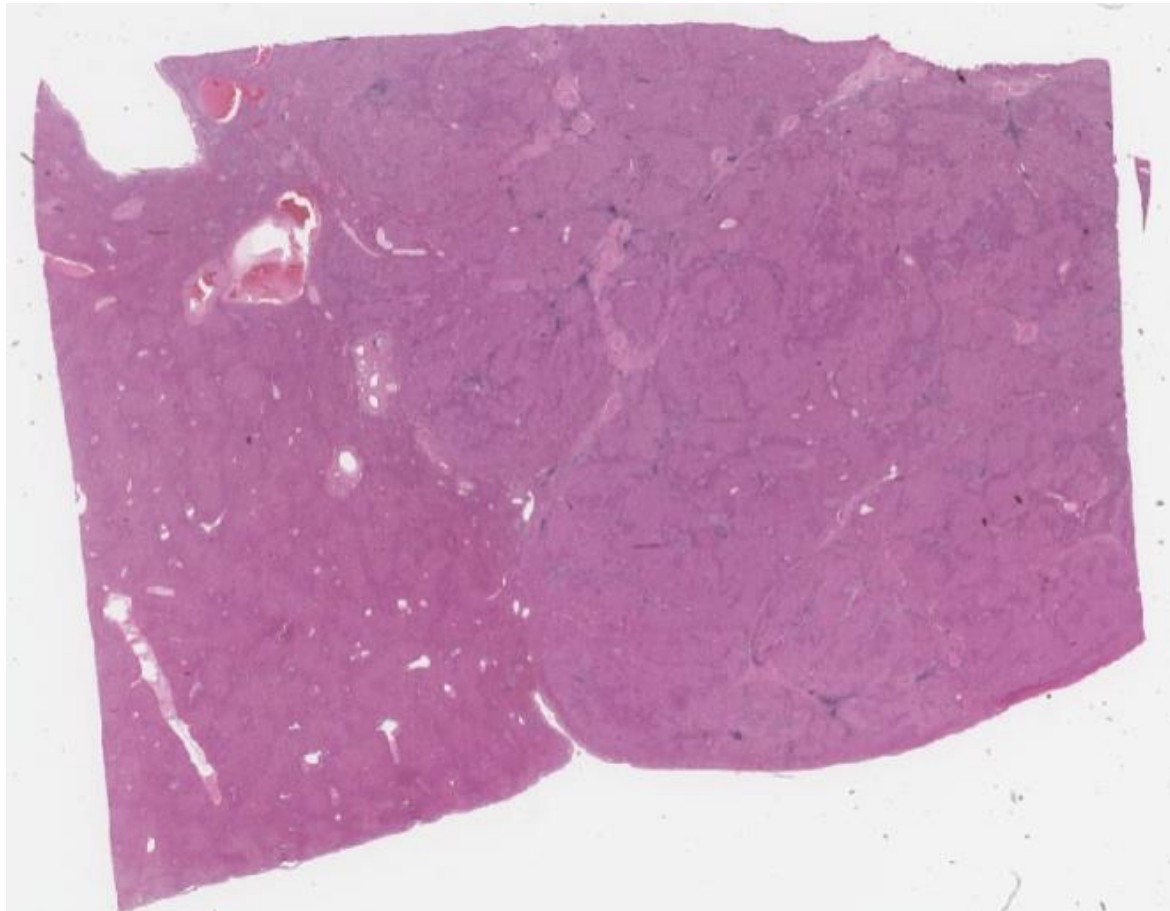
Macroscopic description:

Specimen 187g. 129x 80x 43mm Capsule intact.

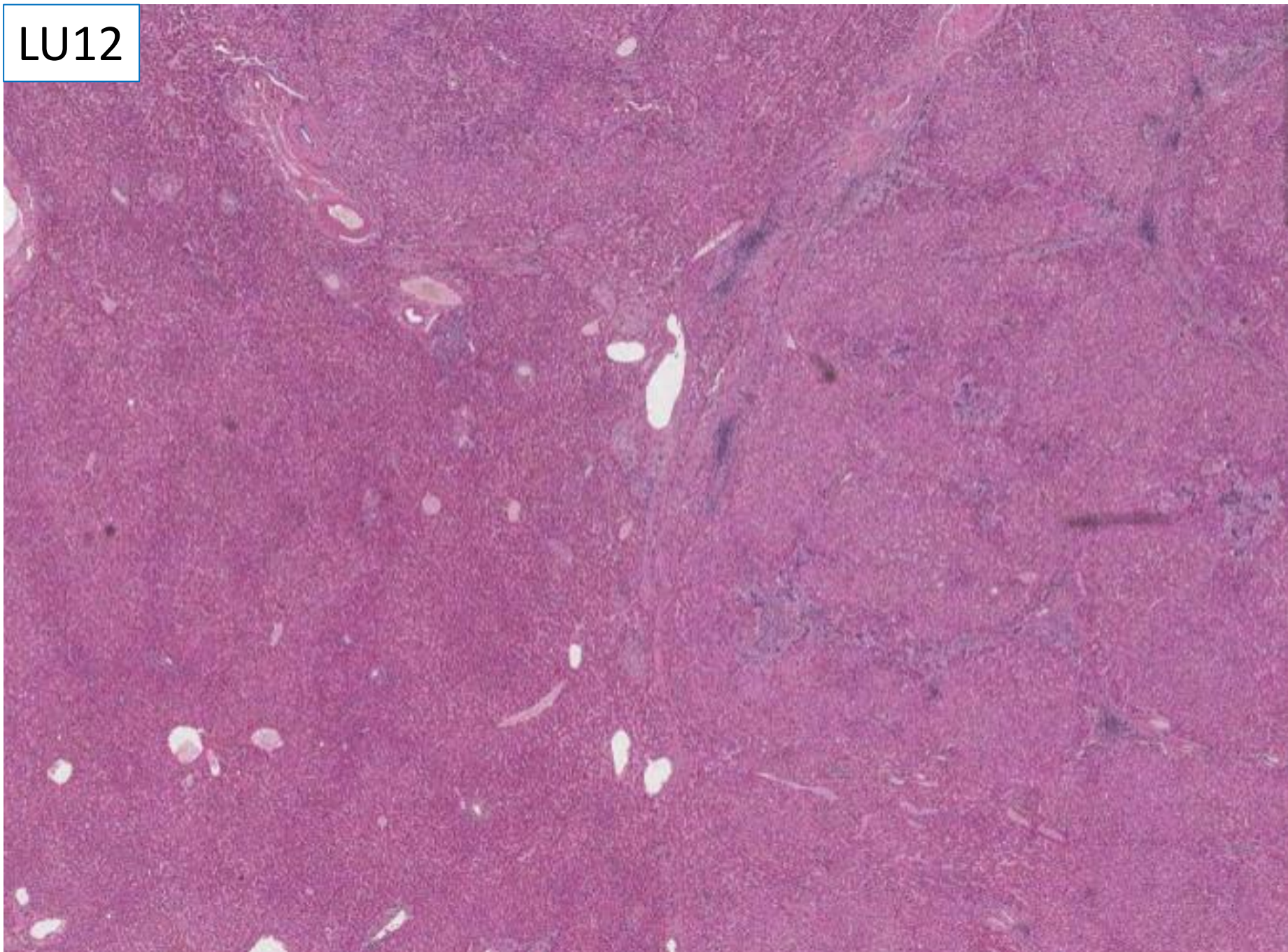
Lesion 44 x 38 x 25mm with a cream, tan, congested appearance.

Second lesion 13mm diameter, 20 mm from main lesion.

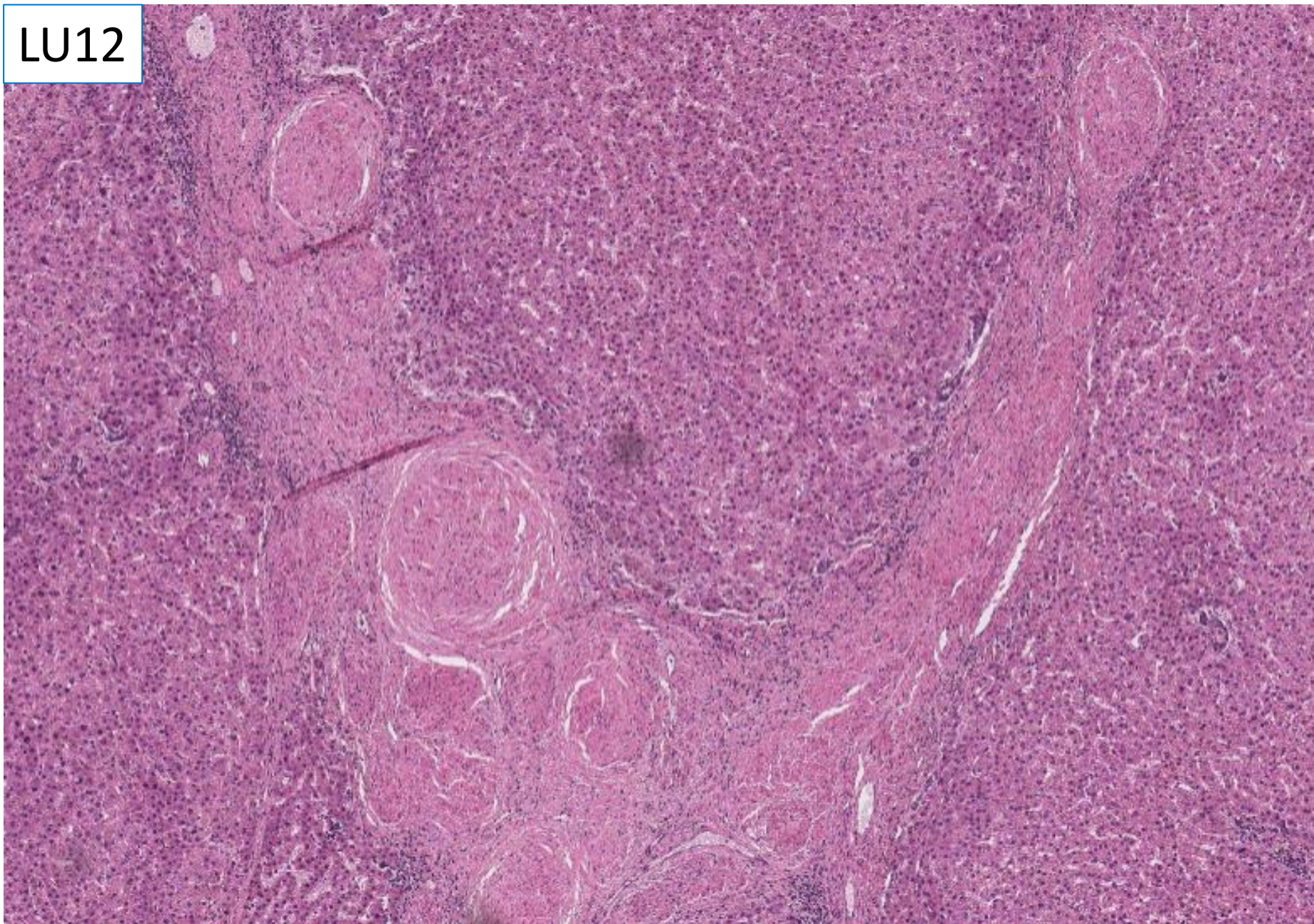
Additional stains: Glutamine synthetase, Keratin 7, van Gieson.



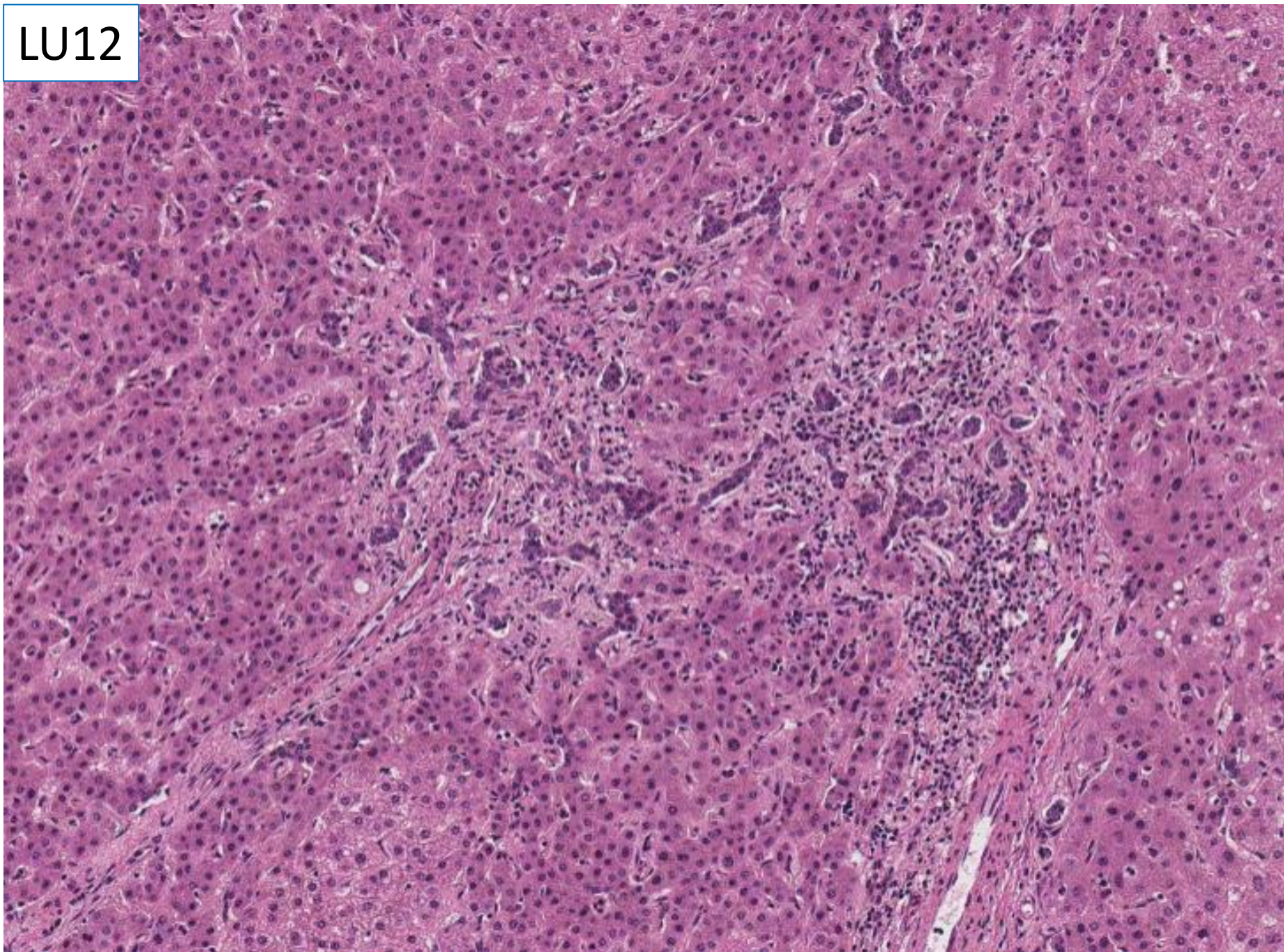
LU12



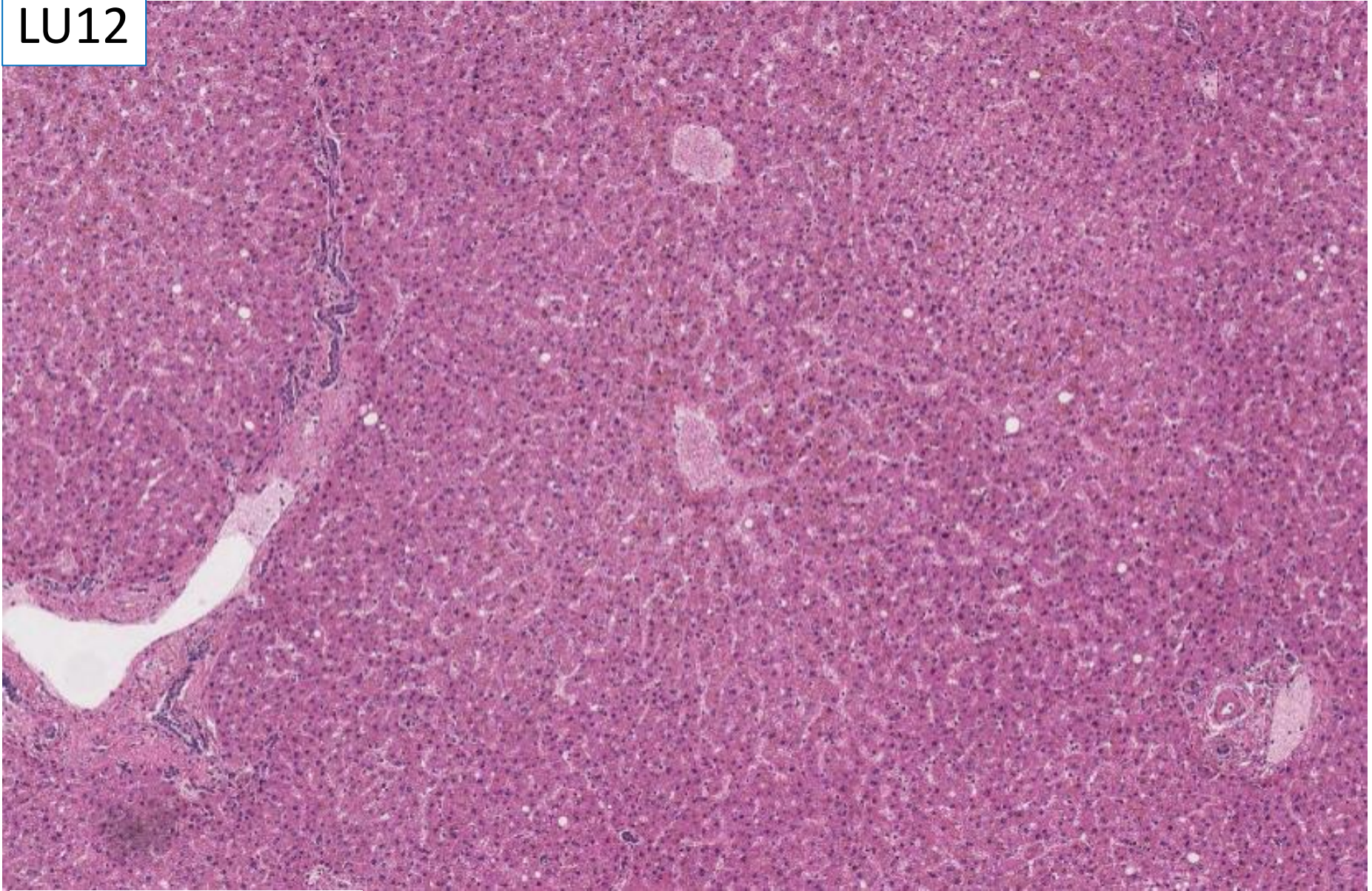
LU12



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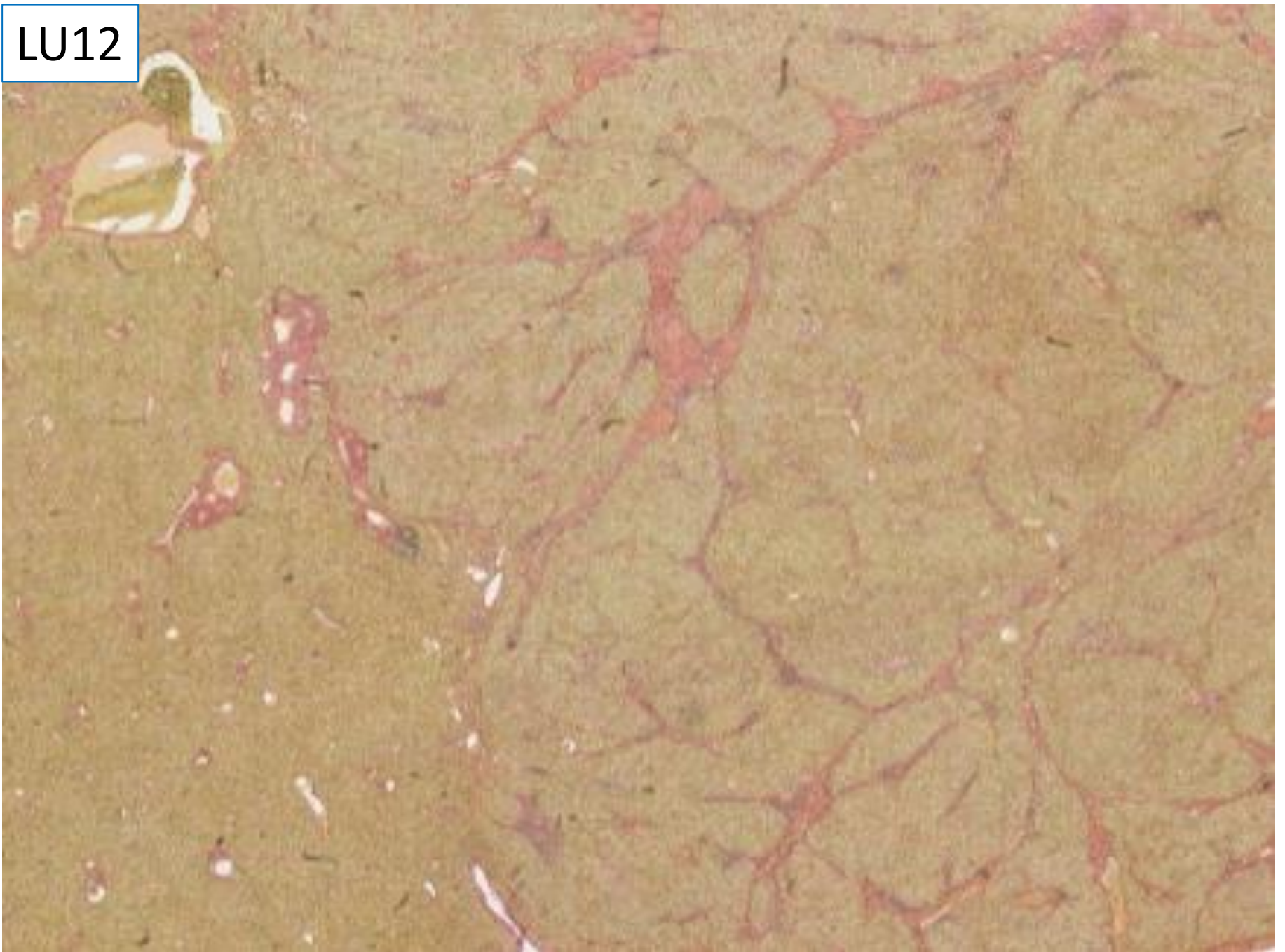


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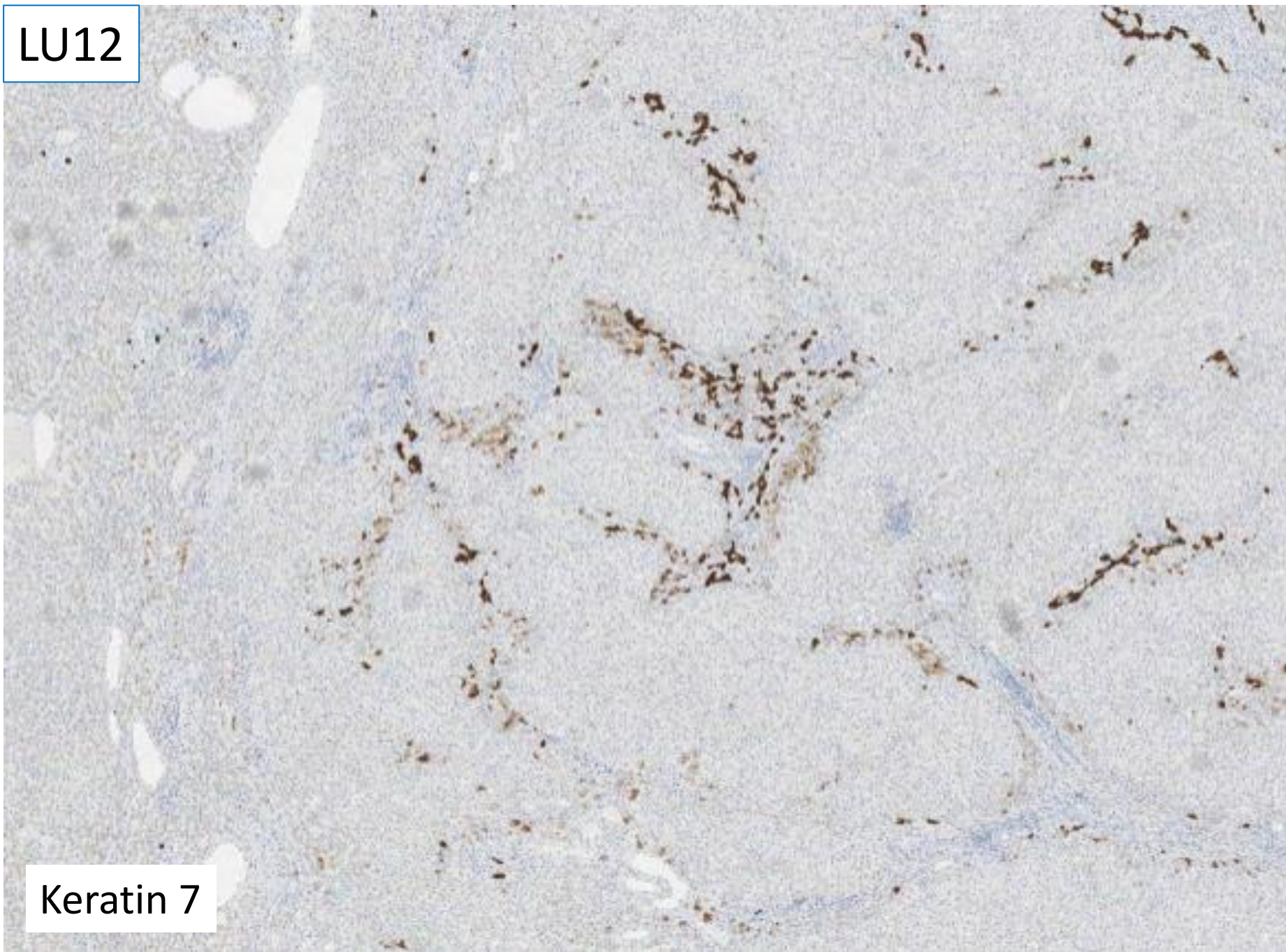
Background liver

LU12

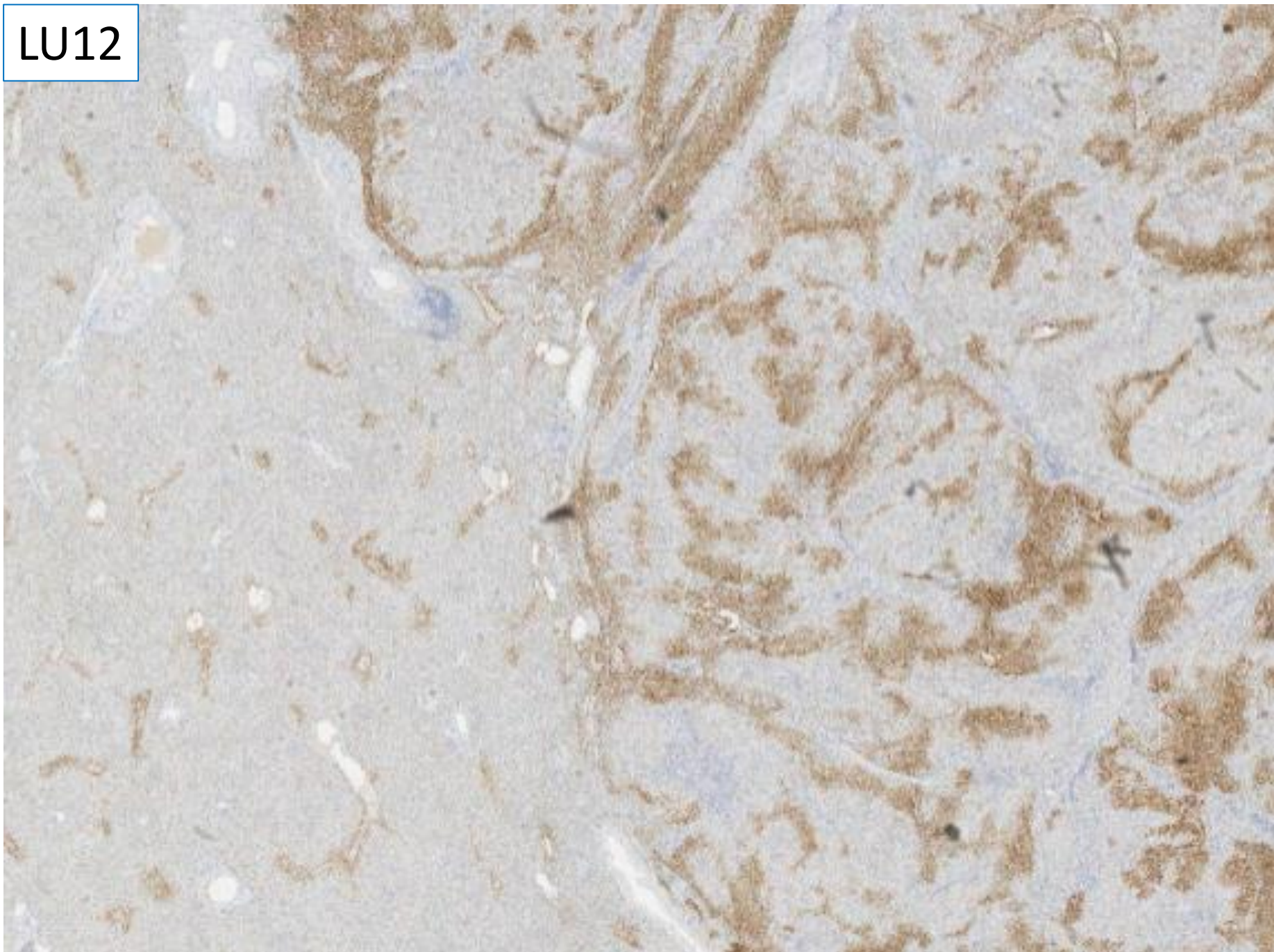


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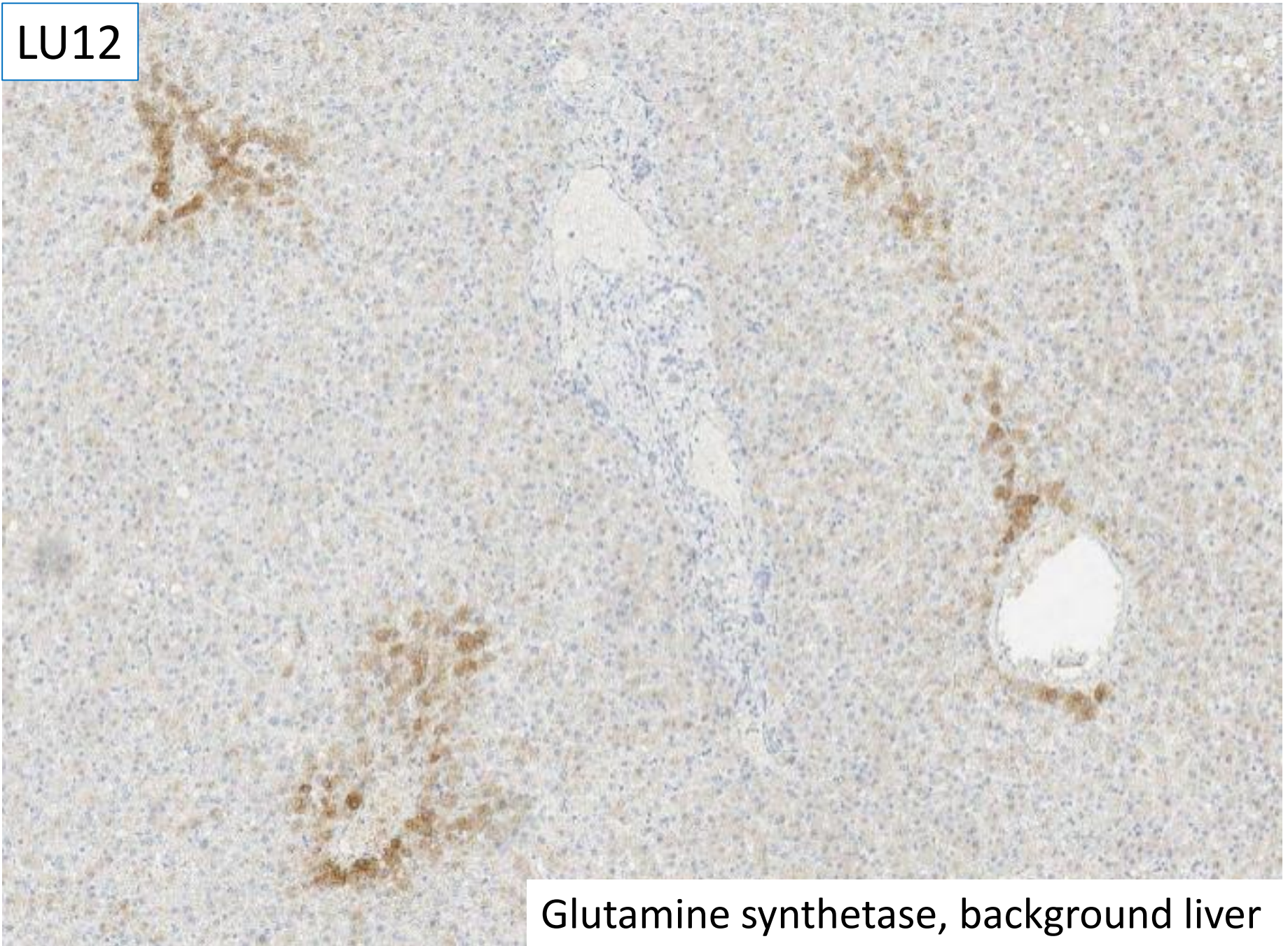
Keratin 7



LU12



LU12



Glutamine synthetase, background liver

Case LU12 45F

Left lateral segmentectomy laparoscopically. ?FNH ?adenoma

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A	Focal nodular hyperplasia
B	Hepatocellular adenoma
C	Cirrhosis
D	Nodular regenerative hyperplasia
E	Hepatocellular carcinoma

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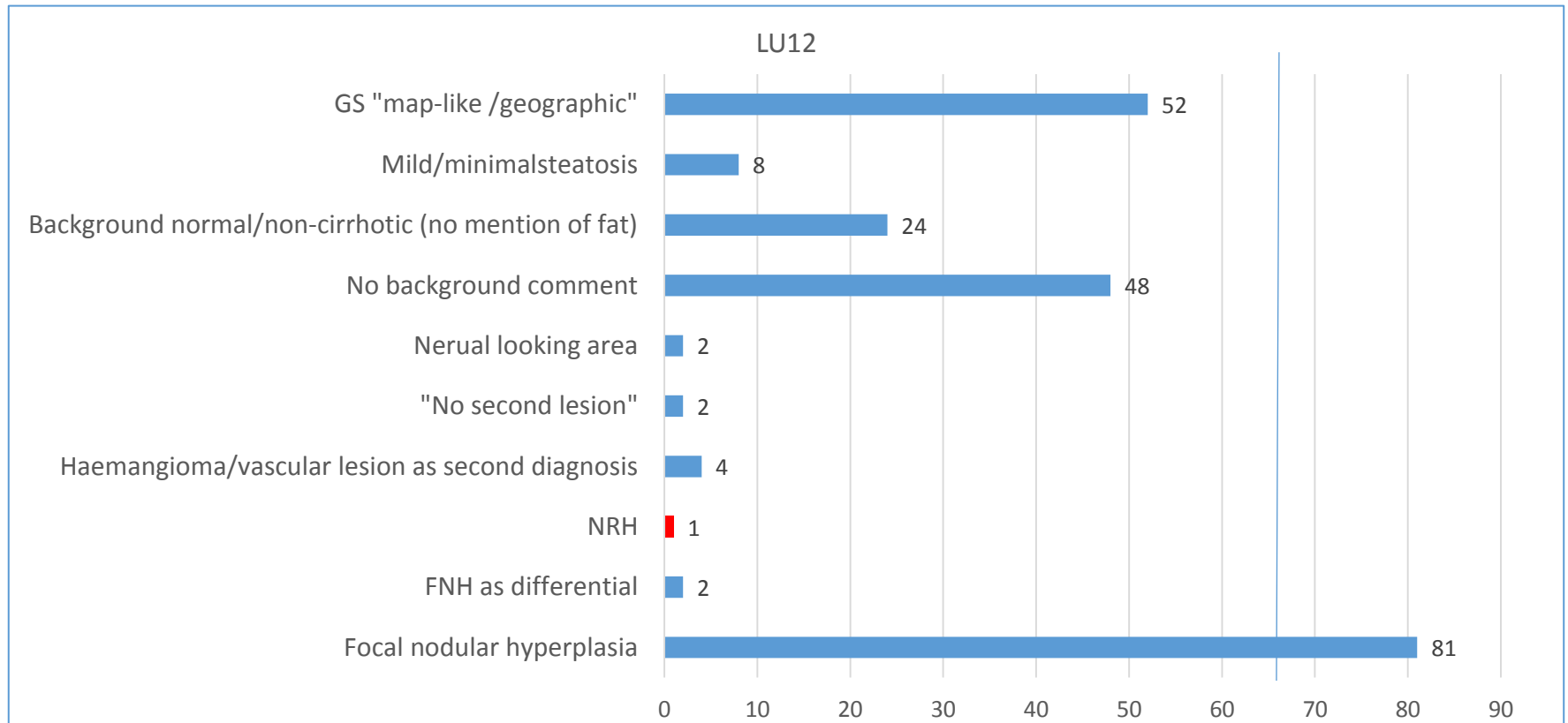
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LU12: Scoring proposal

Consensus complete responses would include Focal Nodular hyperplasia.

Second lesion is referred to in clinical information, which the submitting pathologist has copied from the request form – it is 20mm away in the macro description, and so is not present in the submitted section; 4 responses thought there may also be a haemangioma as the second lesion – these have not been marked down, since the information provided was rather misleading

Suggested scoring: for 10 points include FNH or as main diagnosis with differential including FNH

Lose 10 marks (score 0) if not FNH – nodular regenerative hyperplasia (1)

The End