Liver histopathology EQA Scheme

Circulation LN Spring 2016

Circulation LN – Spring 2016

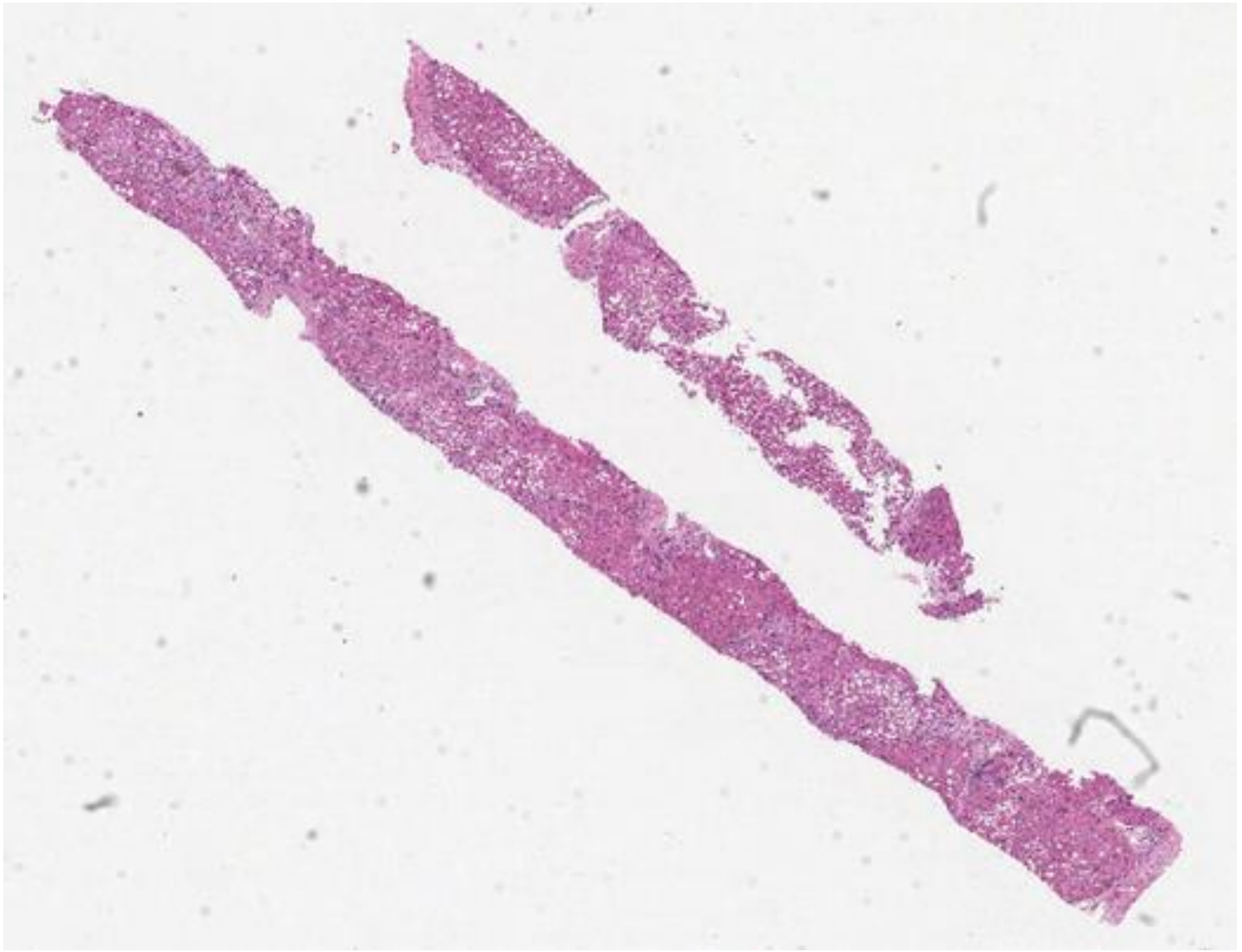
- Responses received from 86 participants.
- 80% consensus – at least 69 agree
- Responses collated and suggested scores sent to EQA members
  - half marks blue, no marks red, how to score? Green

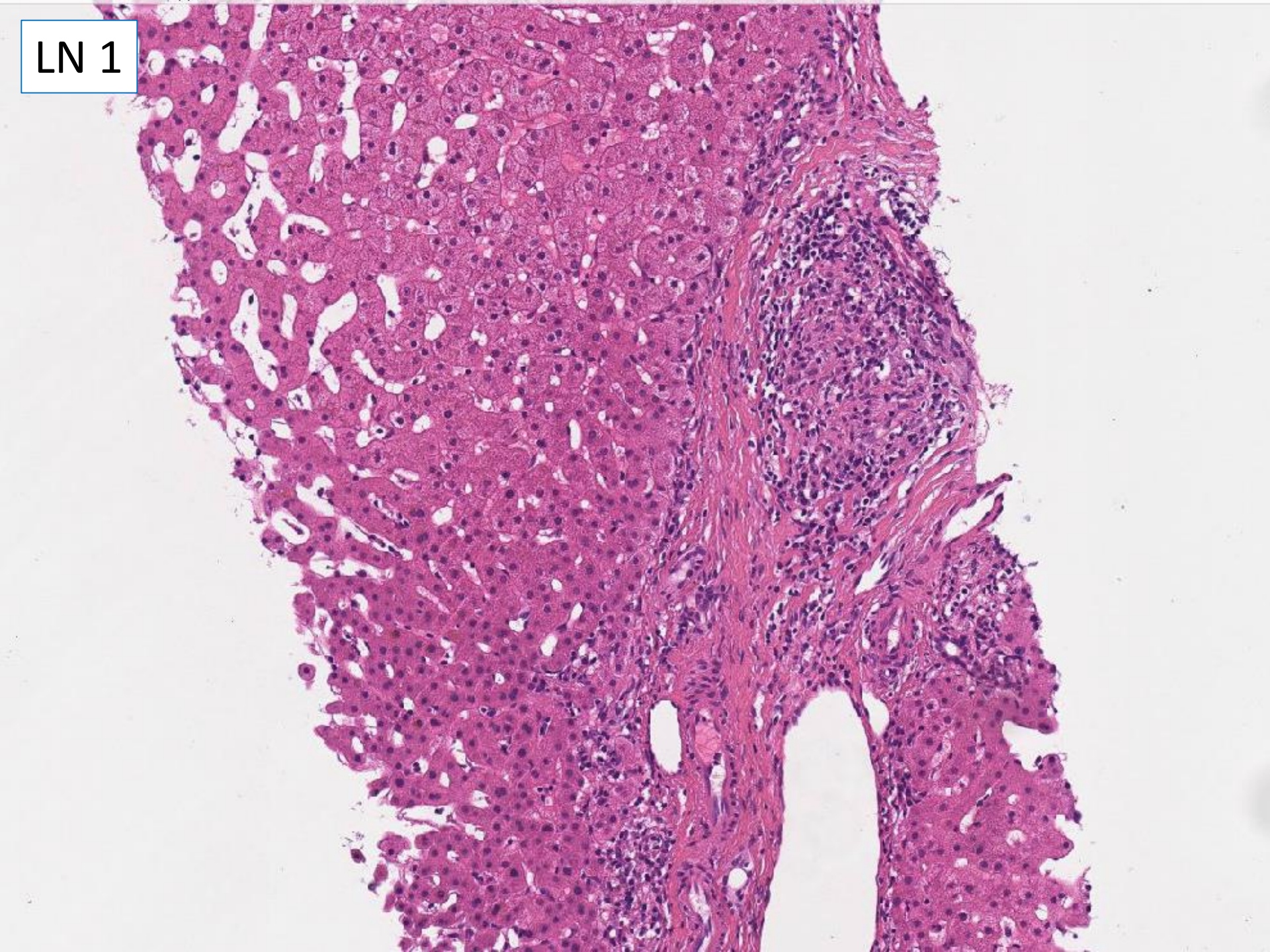
- Comments on suggested scoring received from 11
- Shown as Half marks in slides below

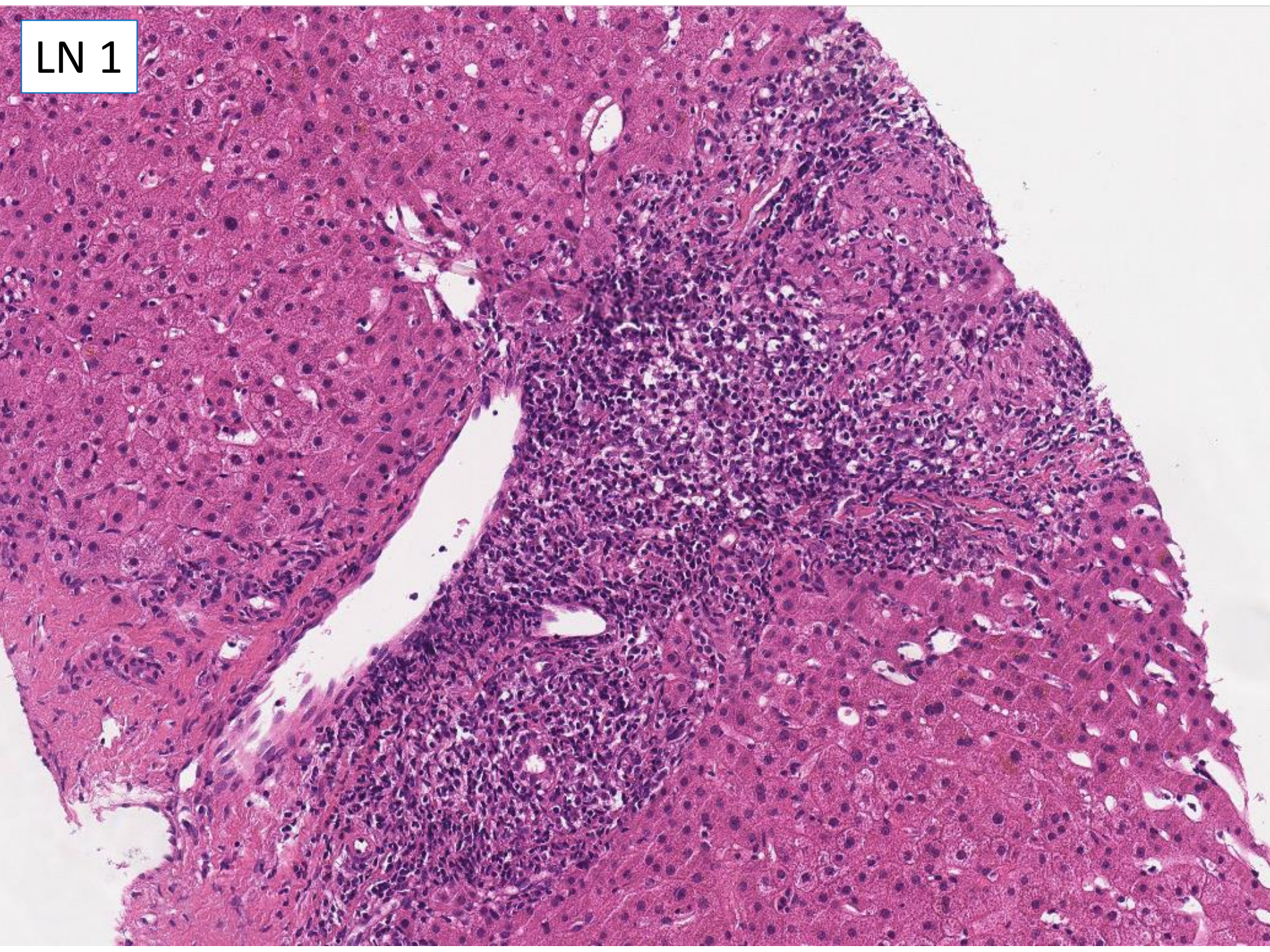
- For MCQ version – responses from 23.
- Survey monkey charts added to presentation yesterday

LN1 Female 45 years

Alk p'ase elevated 3 x normal; ALT mildly elevated; positive AMA; IgM elevated. IgG normal. Connective tissue stains (slides not provided) - no fibrosis.




LN 1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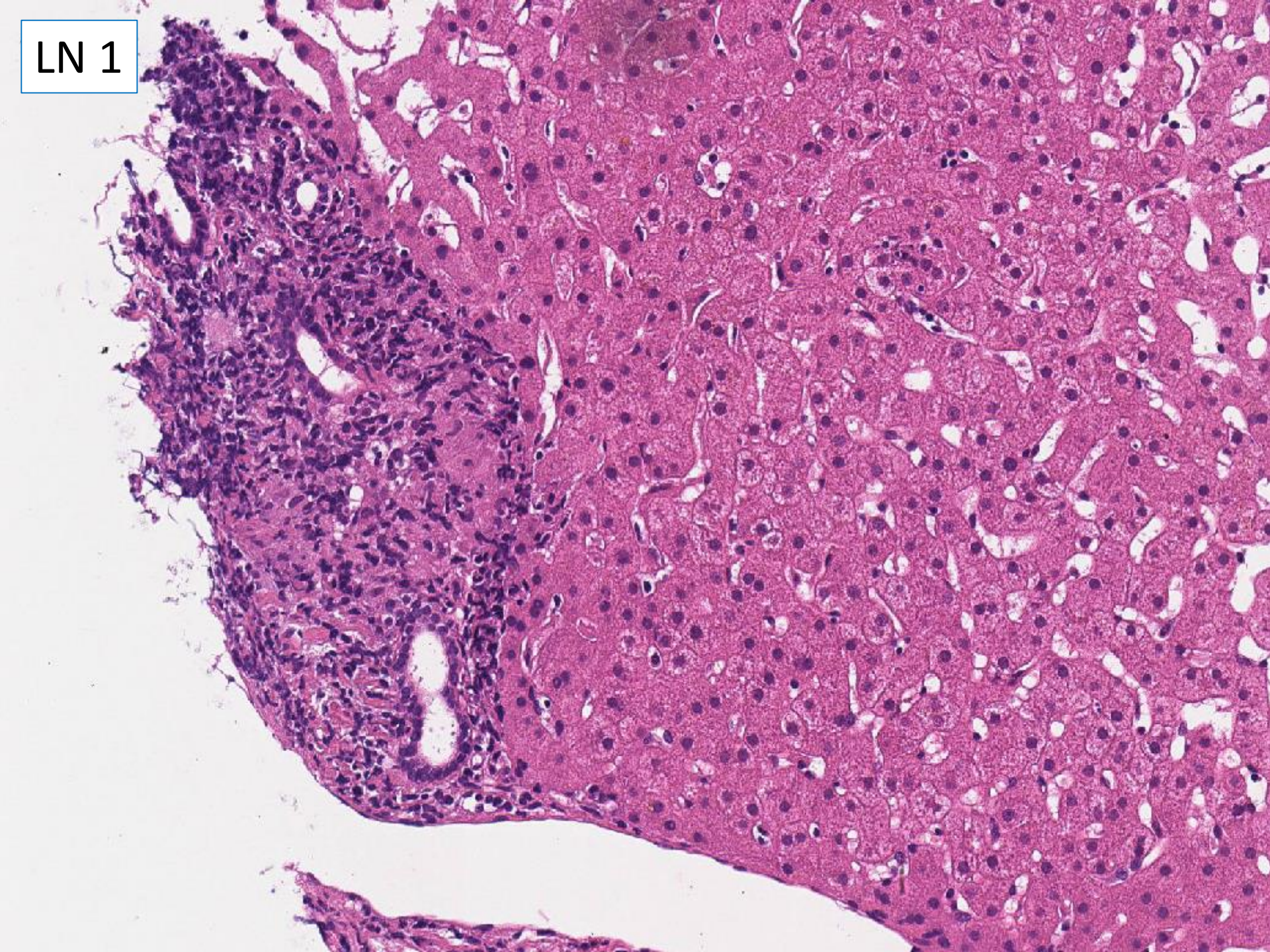
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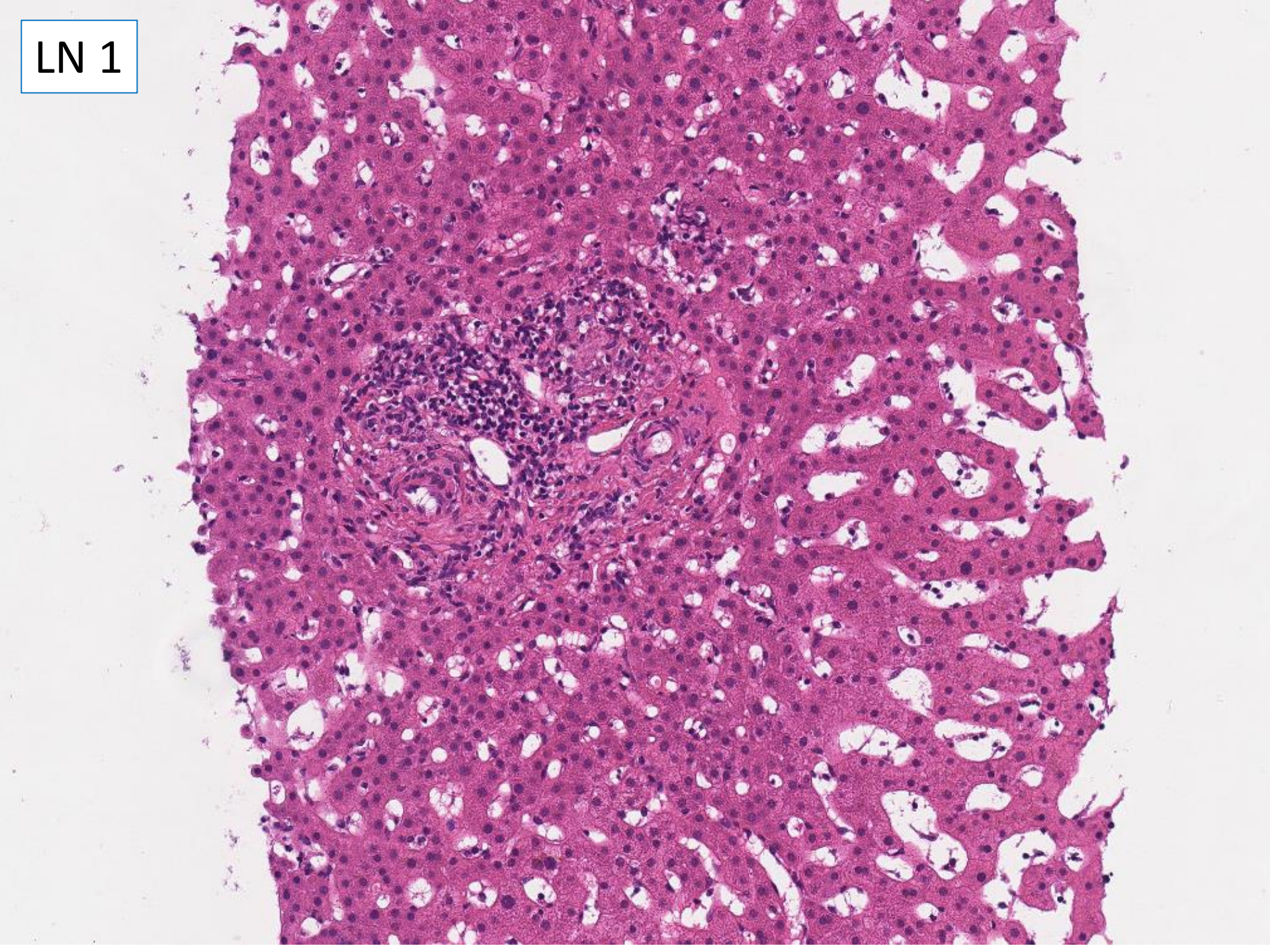
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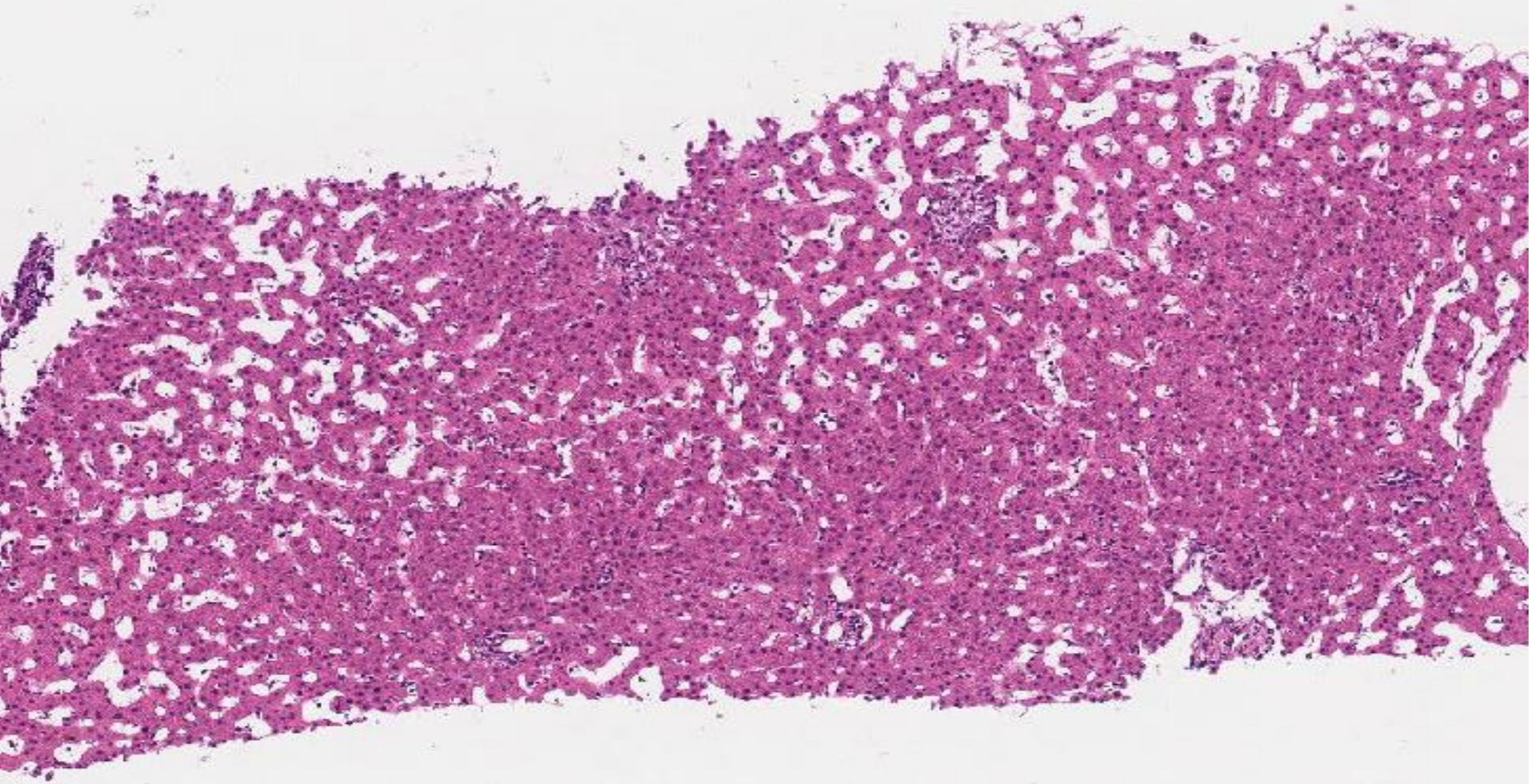


LN1 Female 45 years

Alk p'ase elevated 3 x normal; ALT mildly elevated;
positive AMA; IgM elevated. IgG normal.

Consistent with PBC	81
Consistent with primary sclerosing cholangitis	2
Suggestive of PBC, needs orcein or CK7	2
'Chronic active interface hepatitis c/w PBC'	1
COMMENTS: what name is used? Most just PBC:	
Primary biliary cholangitis	*12*
Both primary biliary cirrhosis and cholangitis	*7*
Some comment on stage – using 4 different staging systems or none	*18*
Comment on sinusoidal dilatation ? venous outflow problem 13	

Suggested scoring: for full marks, diagnosis of PBC.

No marks for PSC (is this confusion from new terminology of 'primary biliary cholangitis?).
Half marks for ambiguous terminology 'chronic active interface hepatitis c/w PBC'

LN1: Final scoring after meeting discussion:
For full marks, need a diagnosis of primary biliary cholangitis/cirrhosis/PBC.
Half marks for main diagnosis of primary sclerosing cholangitis, both responses indicated PBC was meant, but PSC was written in error.

LN1 Female 45 years

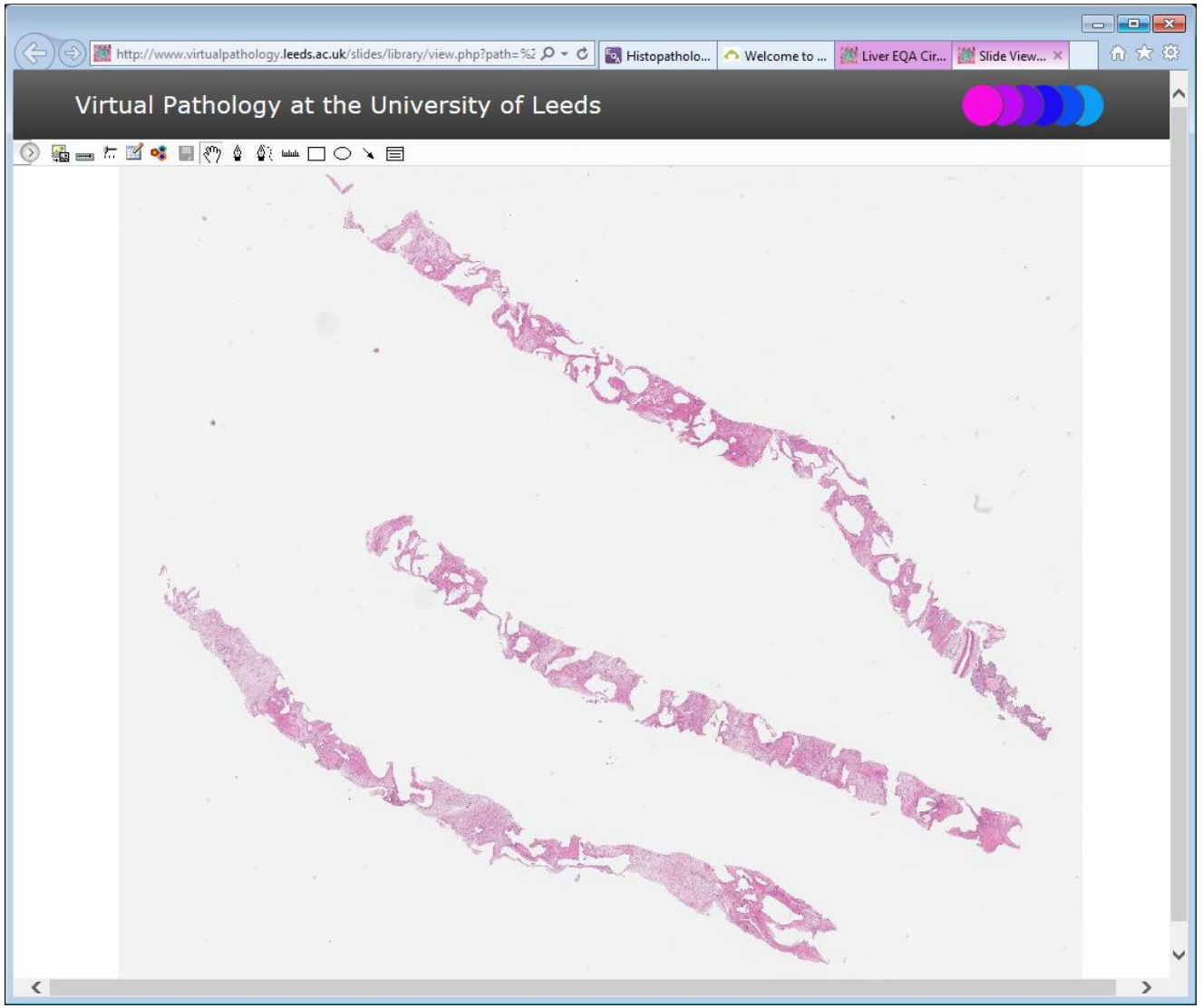
Alk p'ase elevated 3 x normal; ALT mildly elevated;
positive AMA; IgM elevated. IgG normal.

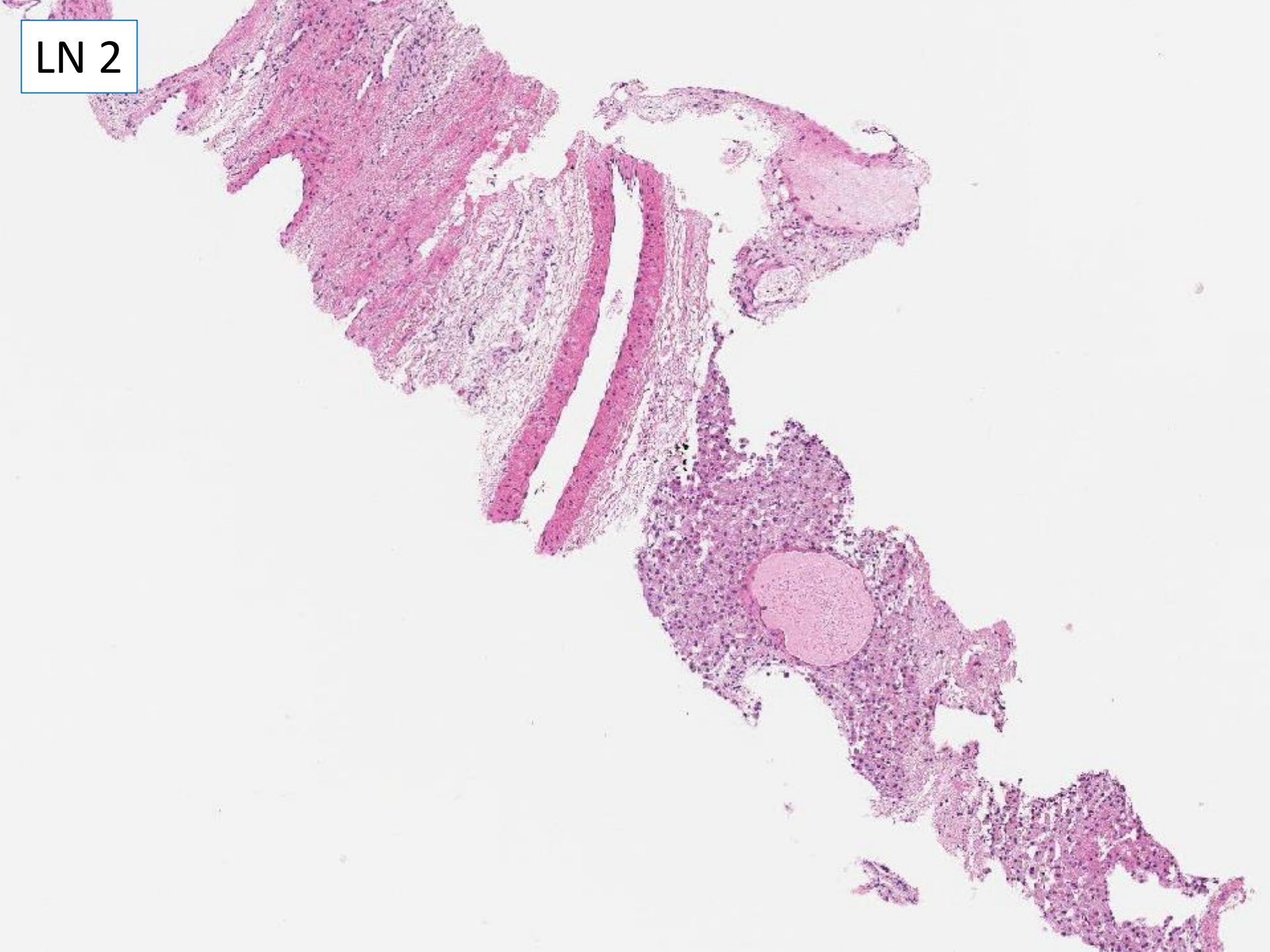
Discussion: comments on sinusoidal dilatation – not uncommonly seen in PBC, and is attributed to bystander narrowing of branches of the portal vein due to adjacent bile duct inflammation. The resulting nodular regenerative hyperplasia may result in portal hypertension before there is late stage fibrosis.

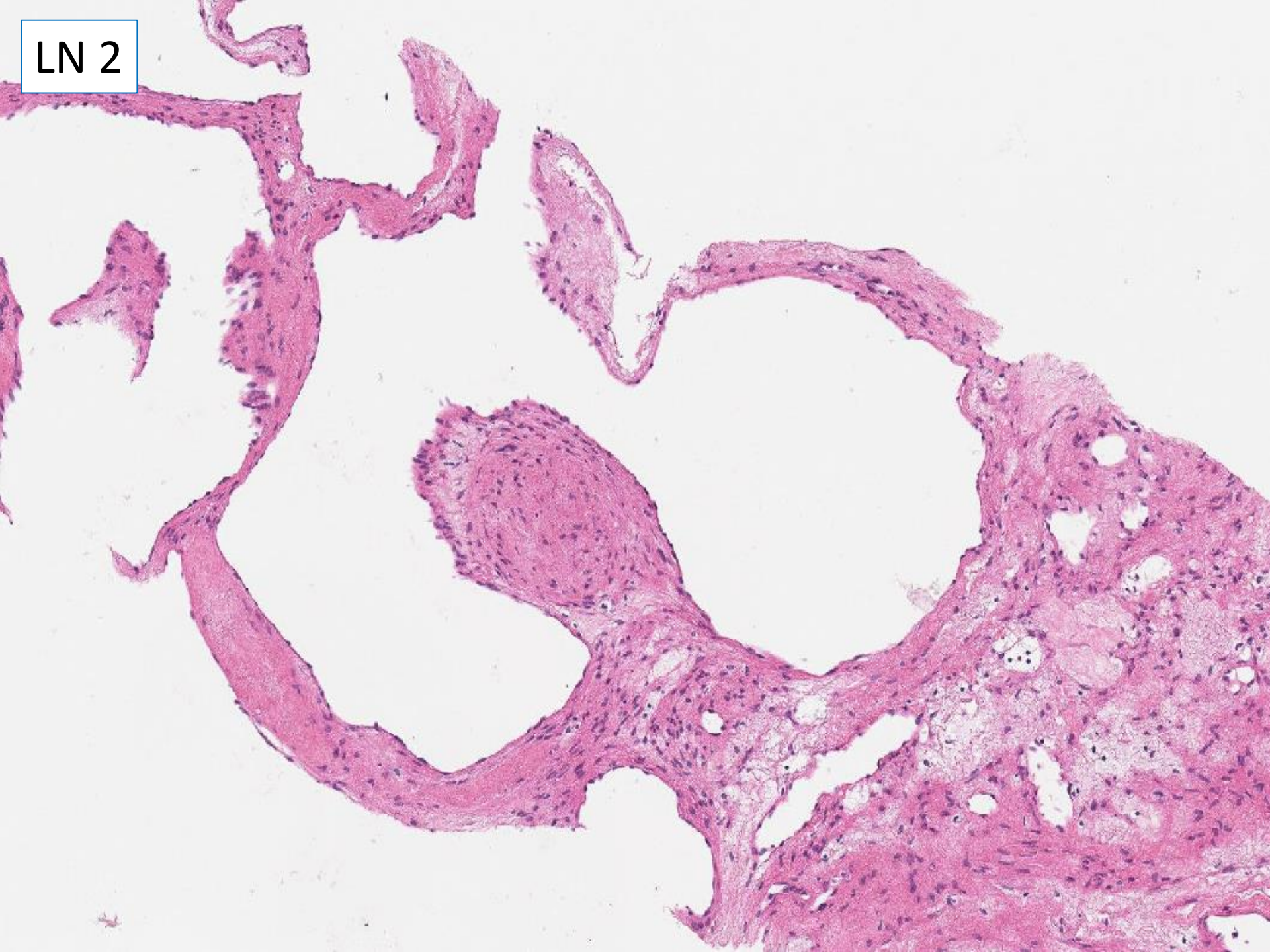
K7 immunohistochemistry can be useful in chronic biliary disease, to demonstrate ductopenia and ductular reaction. It can also show intermediate hepatobiliary cells – positivity in periportal cells which are morphologically hepatocytes is acquired in biliary disease. K7 positive hepatocytes that are not periportal are a non-specific feature of chronic liver disease.

LN2 Male 35 years

Large mass on liver ultrasound. Clinically ?? HCC fibrolamellar, patient clinically well.




LN 2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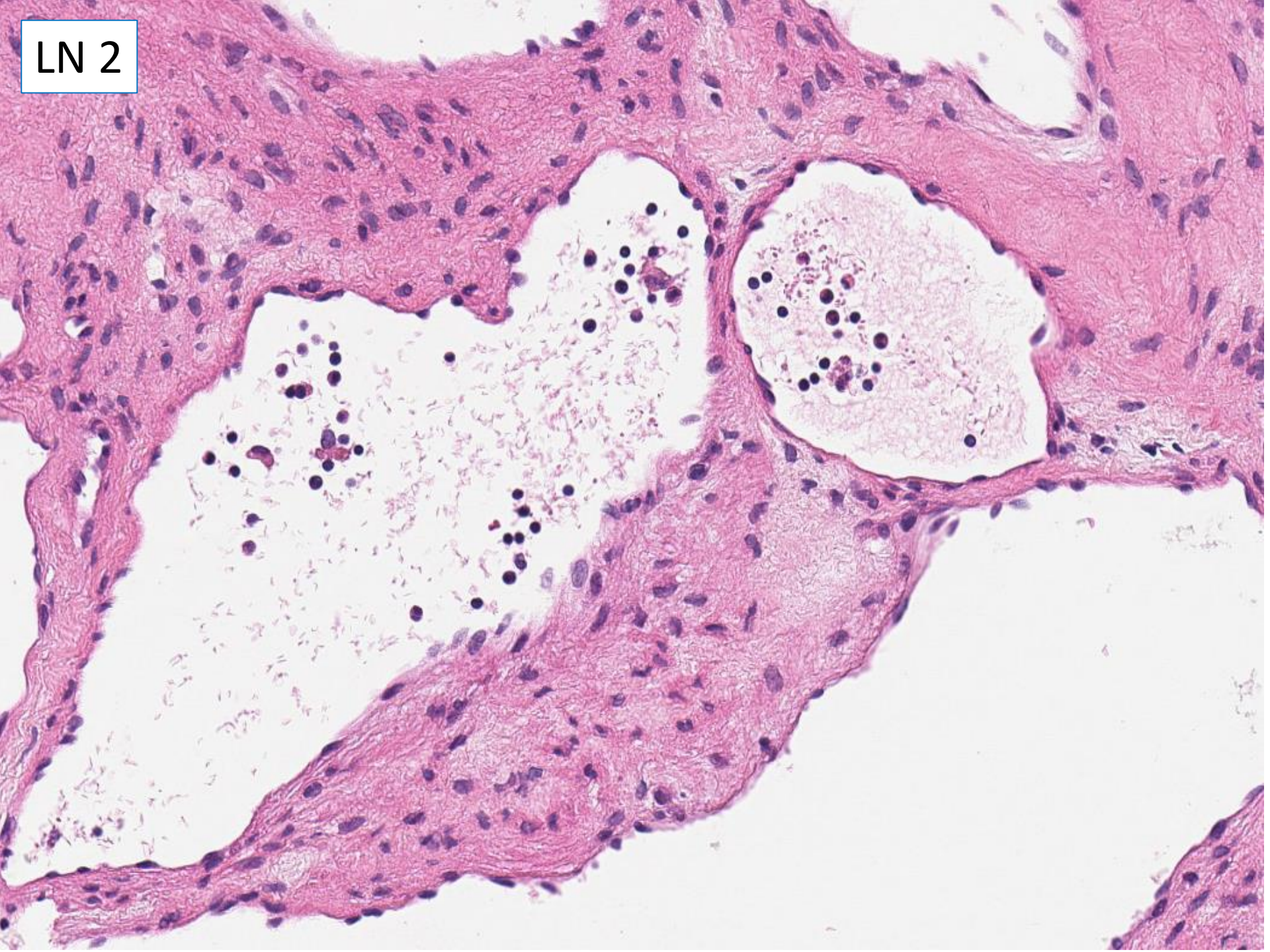

LN 2

LN2 Male 35 years

Large mass on liver ultrasound. Clinically ?? HCC fibrolamellar, patient clinically well.

Haemangioma	61
Lymphangioma	4
Favours haemangioma > lymphangioma	3
Favours lymphangioma >haemangioma	4
Either haemangioma or lymphangioma	11
Haemangioma v angiomyolipoma	1
Angiomyolipoma	1
Venous malformation	1
COMMENT: *Would do IHC (any vascular, D2 40)*	*13*

Half marks

<u>LN2 Final scoring after meeting discussion:</u>

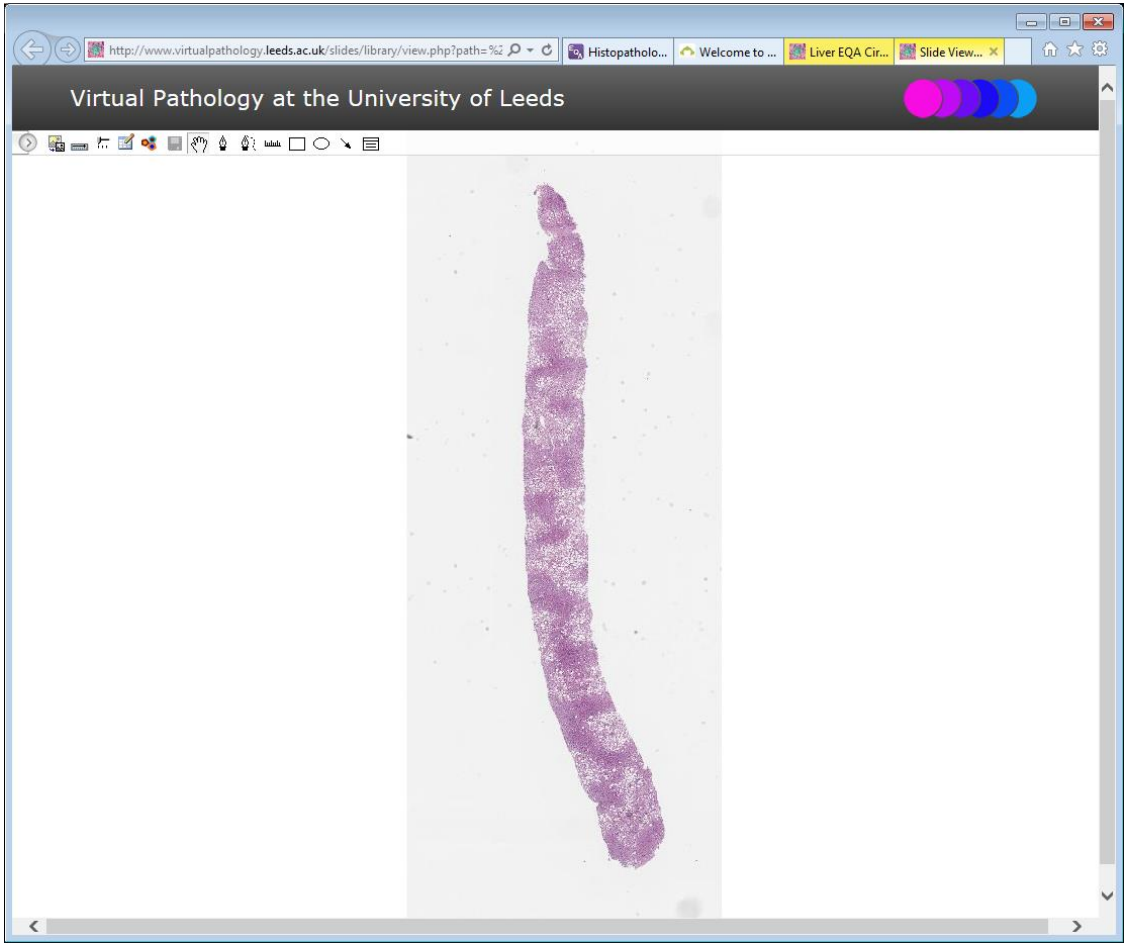
For full marks – either haemangioma or lymphangioma

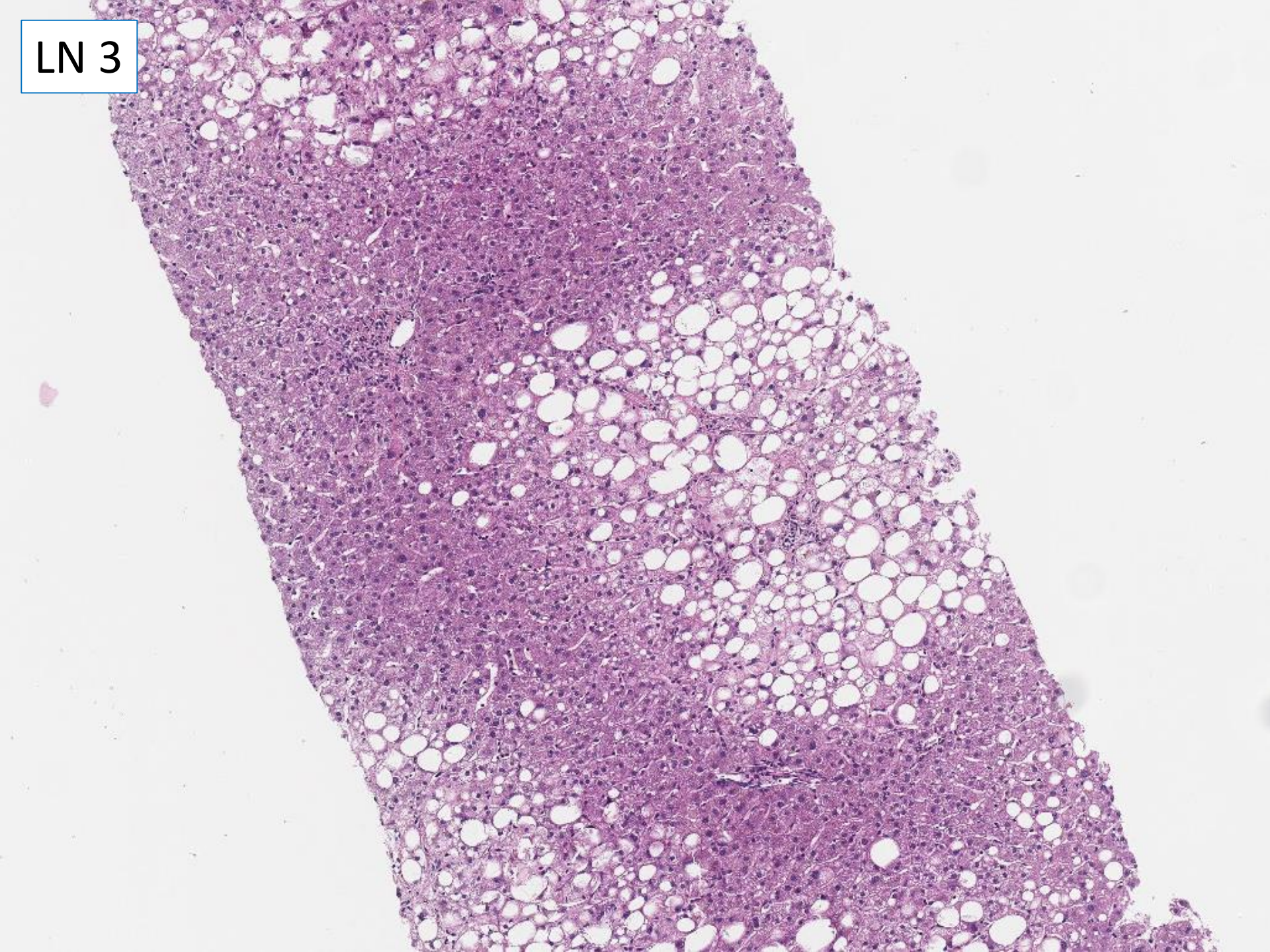
No marks for angiomyolipoma.

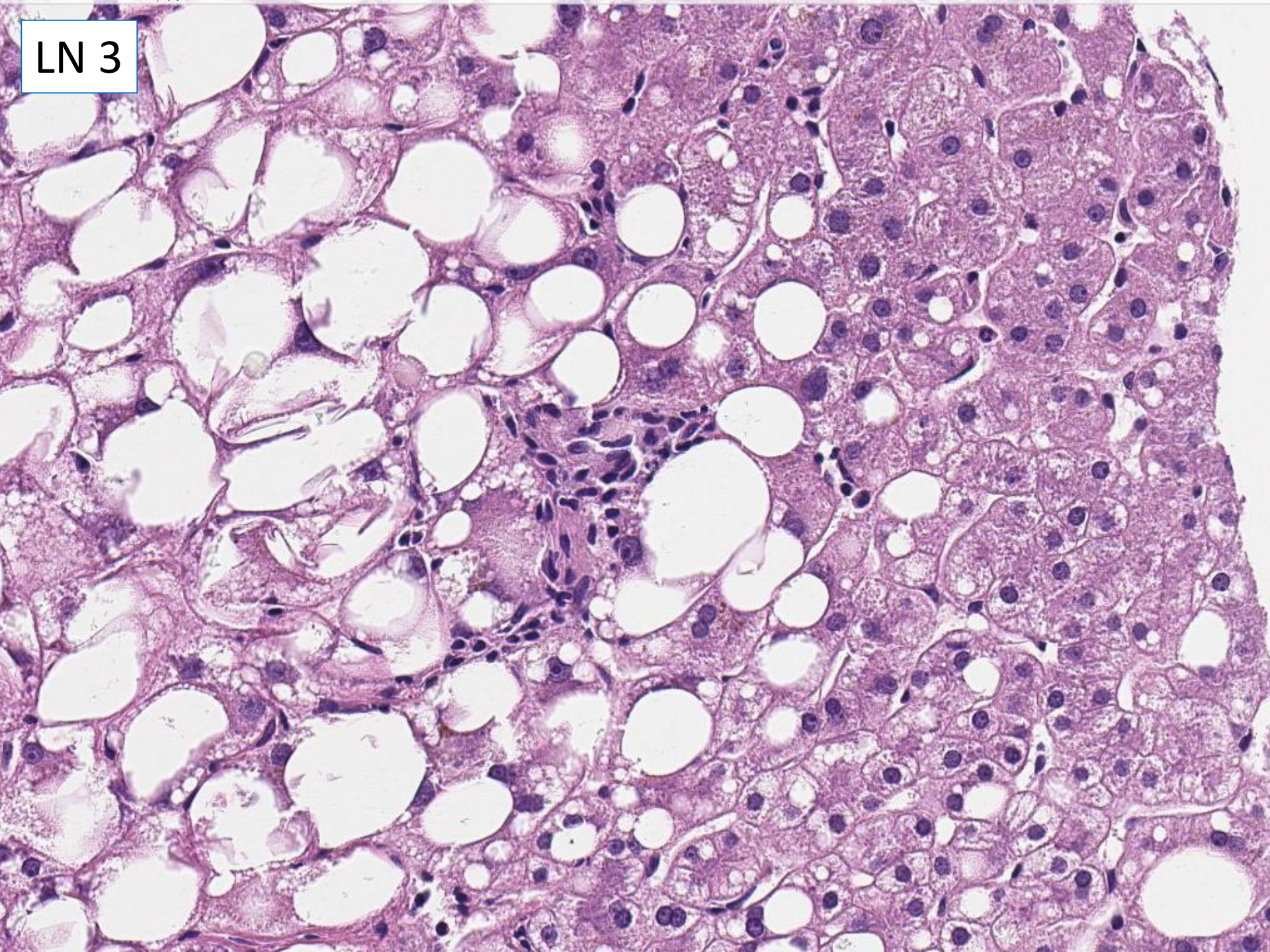
Half marks for venous malformation.

LN3 Male 60 years

Fatty liver on ultrasound scan. Raised ALT, raised BMI (32), Type 2 diabetes. NAFLD, fibrosis score 1.81. Fibroscan 35.3kPa.




LN 3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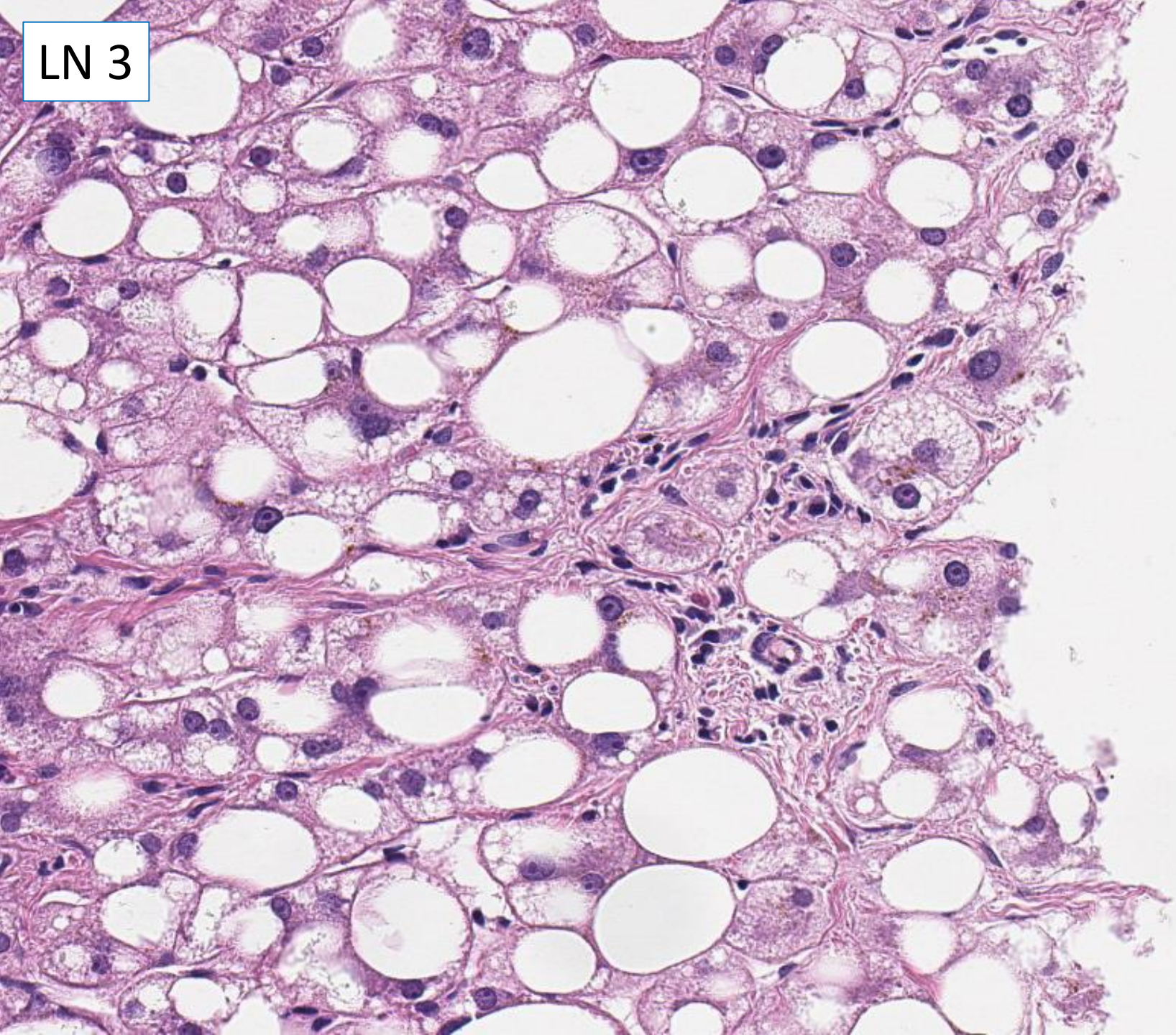
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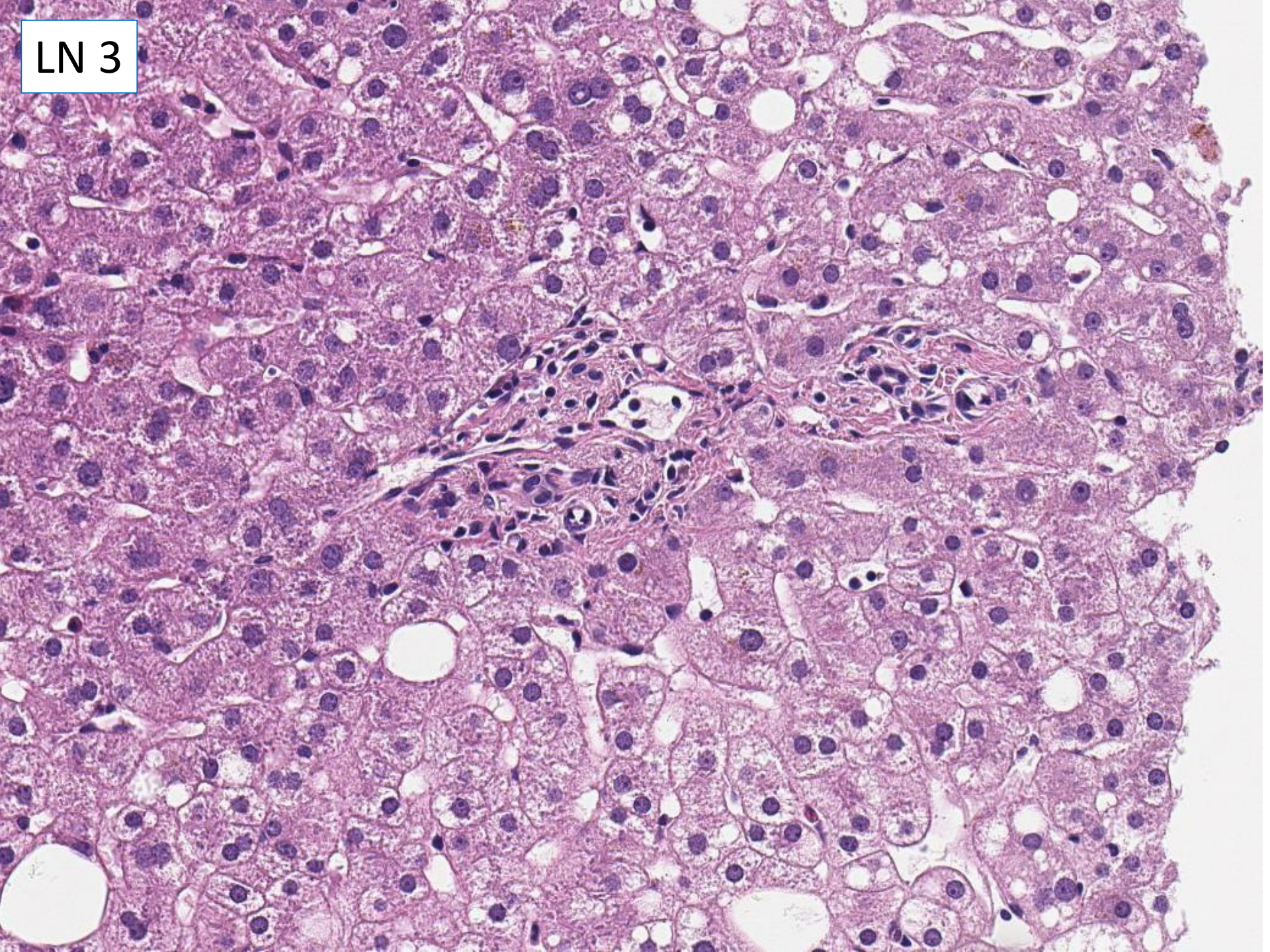
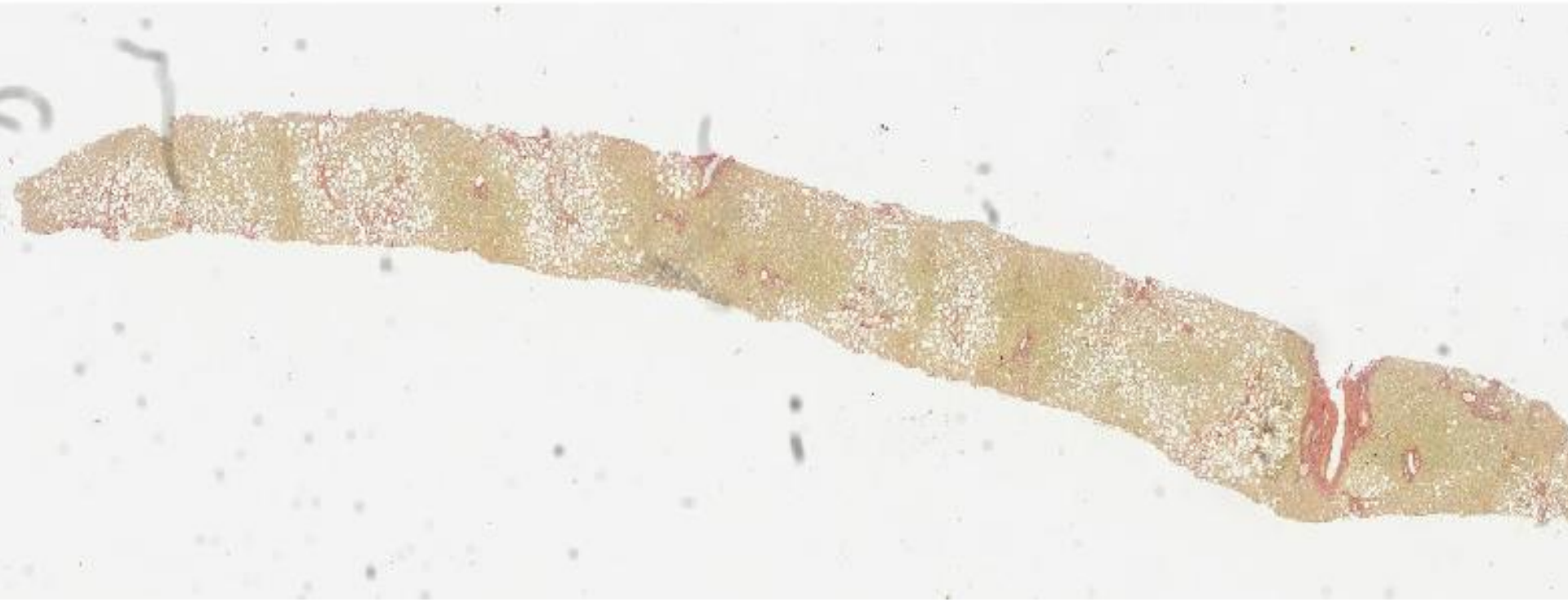
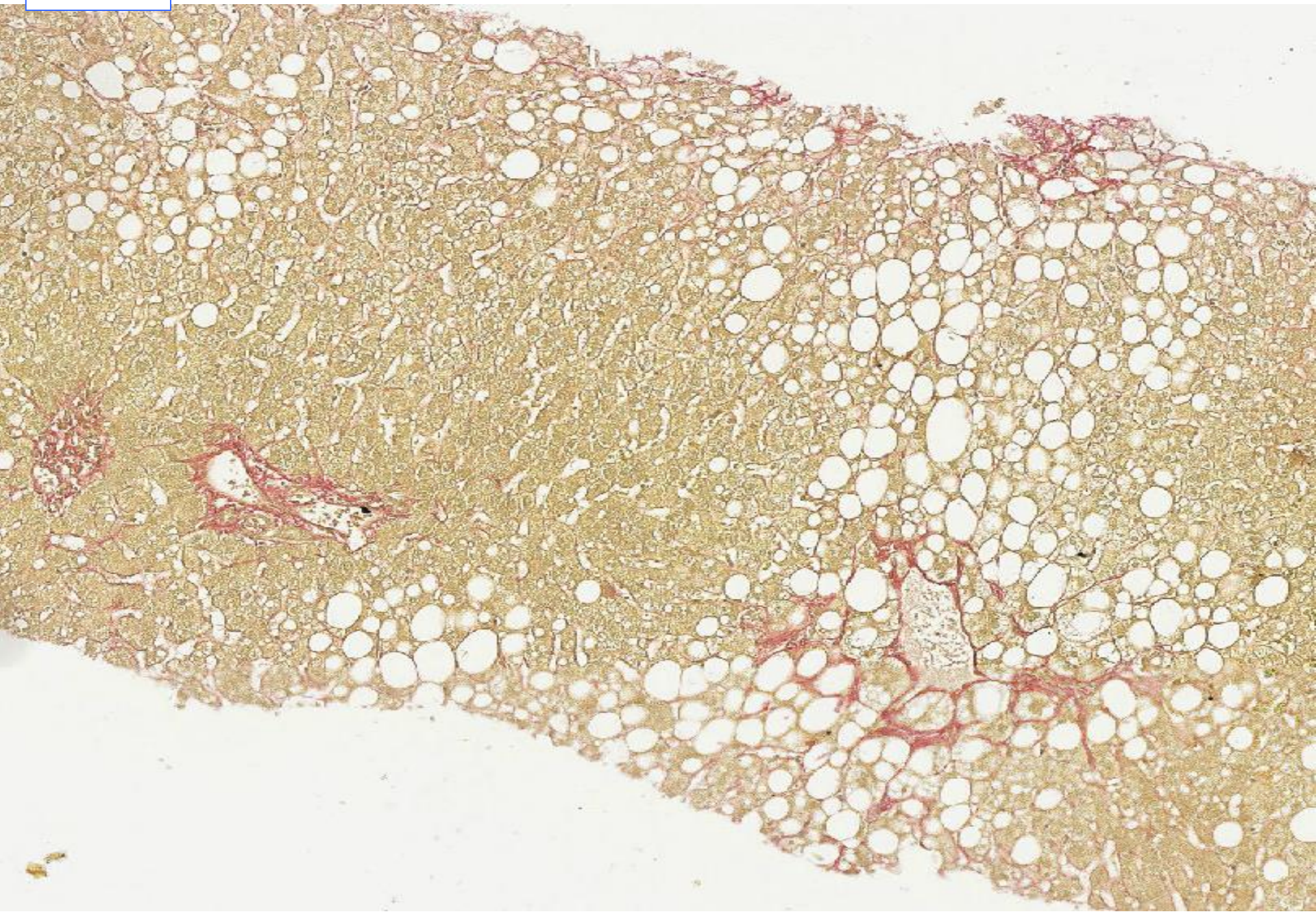

LN 3

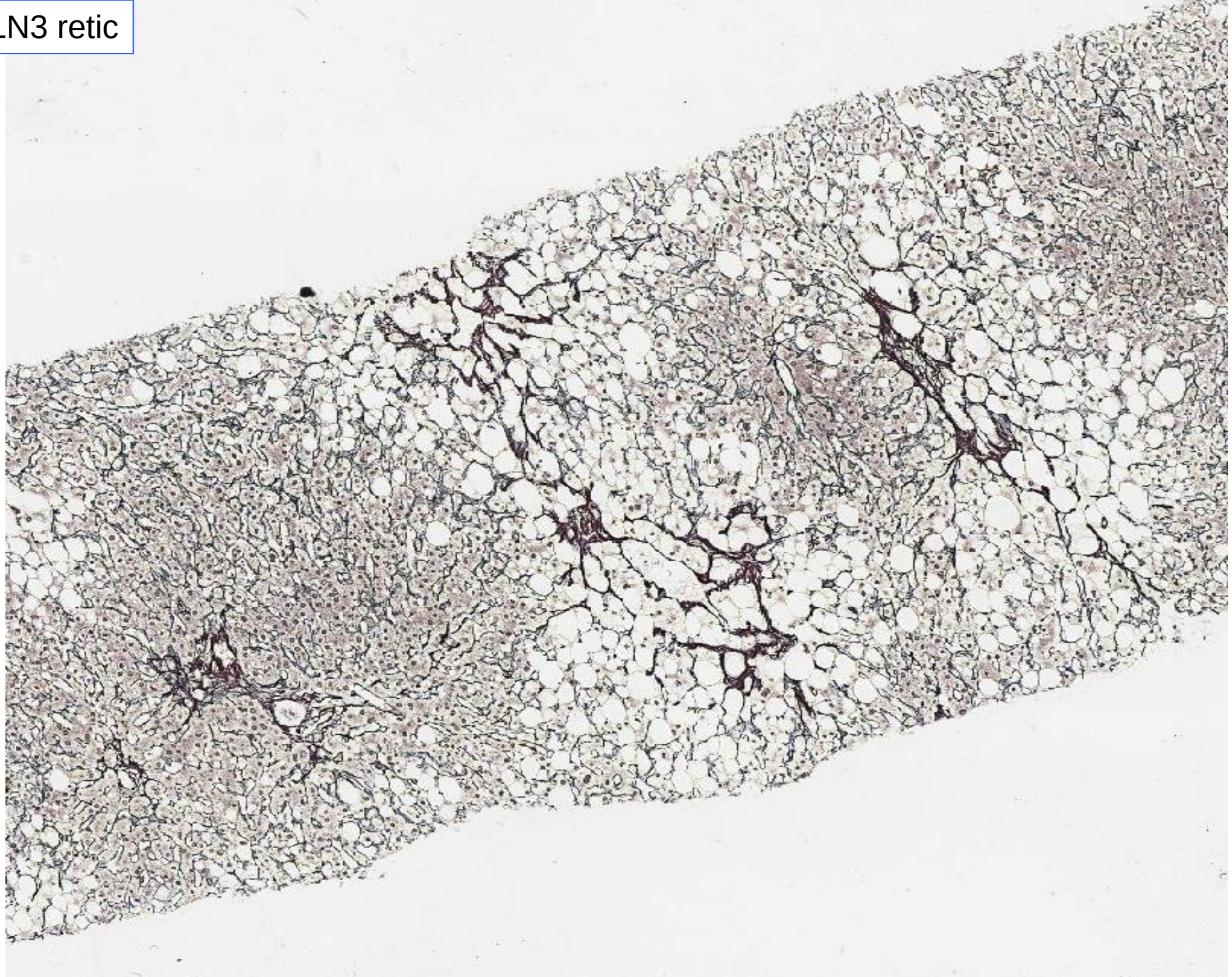
LN3 VG



LN3 VG



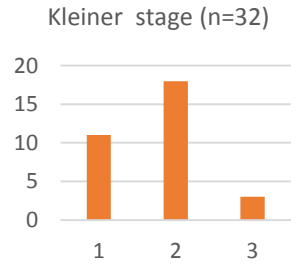
LN3 retic



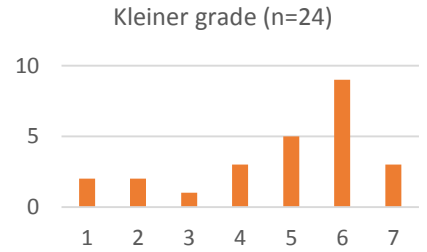
LN3 Male 60 years

Fatty liver on ultrasound scan. Raised ALT, raised BMI (32), Type 2 diabetes. NAFLD, fibrosis score 1.81. Fibroscan 35.3kPa.

Steataohepatitis	81
Steatofibrosis	3
Steatosis	1
Fatty liver disease	1
AETIOLOGY:	
c/w non-alcoholic fatty liver disease	76
Of which need to exclude alcohol mentioned	6
No cause mentioned	10
STAGE: all made some comment on fibrosis stage – descriptive (mild) or Kleiner (32) or Ishak (2)	86
commented that the fibrosis stage was less than predicted by fibroscan	*6*



24 included a Grade (Kleiner) –



LN3 Male 60 years

Fatty liver on ultrasound scan. Raised ALT, raised BMI (32), Type 2 diabetes. NAFLD, fibrosis score 1.81. Fibroscan 35.3kPa.

scoring: Clear consensus for steatohepatitis.

1. How should other fatty liver disease (steatofibrosis, steatosis, fatty liver disease) be scored?

Half marks

2. Should marks be deducted if there is no comment on link to metabolic/non-alcoholic fatty liver disease?

Half marks

3. Stage - all responses included a comment on fibrosis – Kleiner stage/grade included for information, not scored.

LN3 Male 60 years

Fatty liver on ultrasound scan. Raised ALT, raised BMI (32), Type 2 diabetes. NAFLD, fibrosis score 1.81. Fibroscan 35.3kPa.

Steataohepatitis	81
Steatofibrosis	3
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Fatty liver disease	1
AETIOLOGY:	
c/w non-alcoholic fatty liver disease	76
Of which need to exclude alcohol mentioned	6
No cause mentioned	10
STAGE: all made some comment on fibrosis stage – descriptive (mild) or Kleiner (32) or Ishak (2)	86
commented that the fibrosis stage was less than predicted by fibroscan	*6*

LN3: final scoring after meeting discussion: for full marks, a diagnosis of steatohepatitis and comment on fibrosis. Half marks for steatofibrosis, steatosis, fatty liver disease. Half marks if no mention of the aetiology consistent with NAFLD. No marks if fatty liver disease with neither steatohepatitis nor aetiology.

LN3 Male 60 years

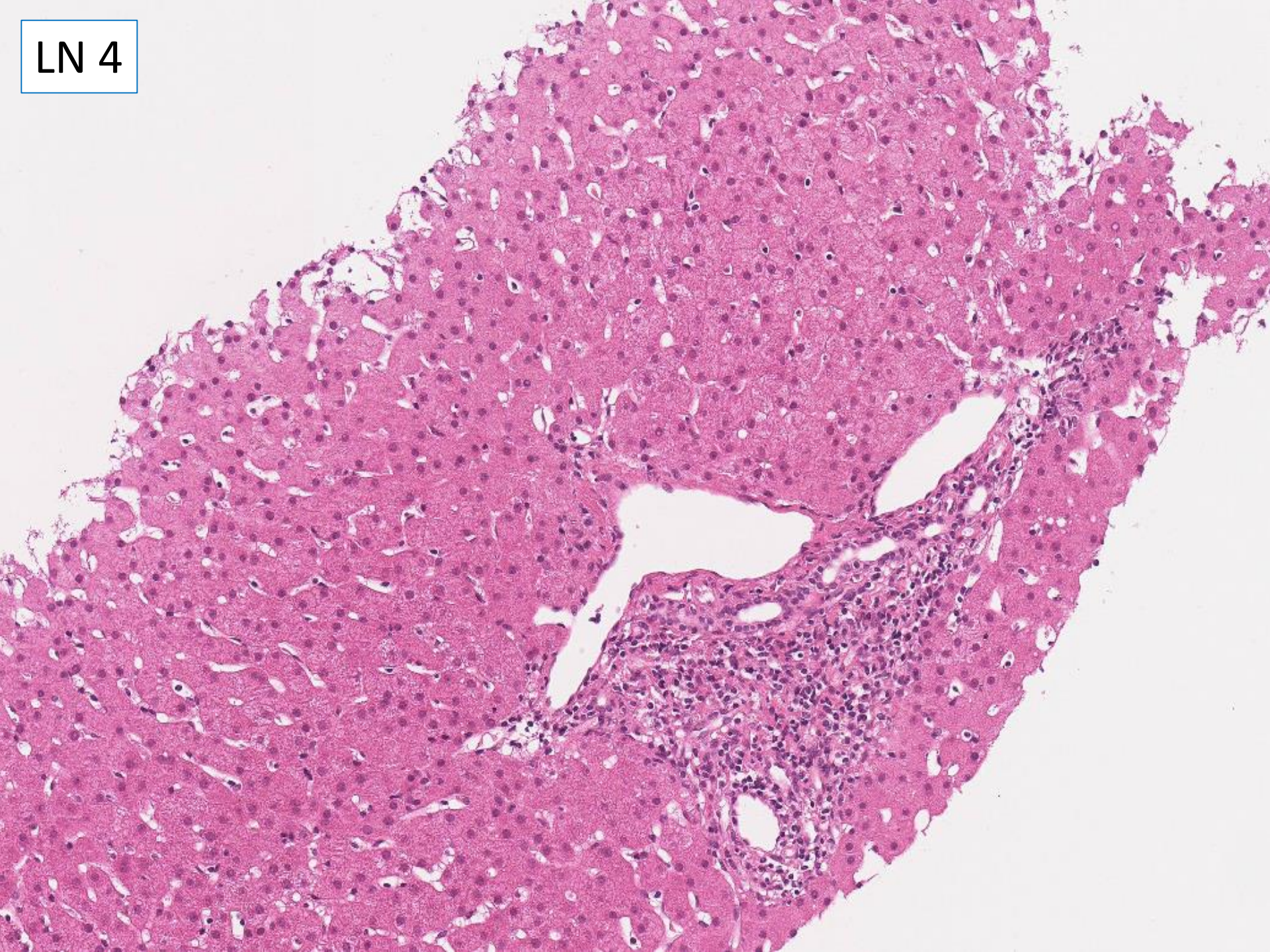
Fatty liver on ultrasound scan. Raised ALT, raised BMI (32), Type 2 diabetes. NAFLD, fibrosis score 1.81. Fibroscan 35.3kPa.

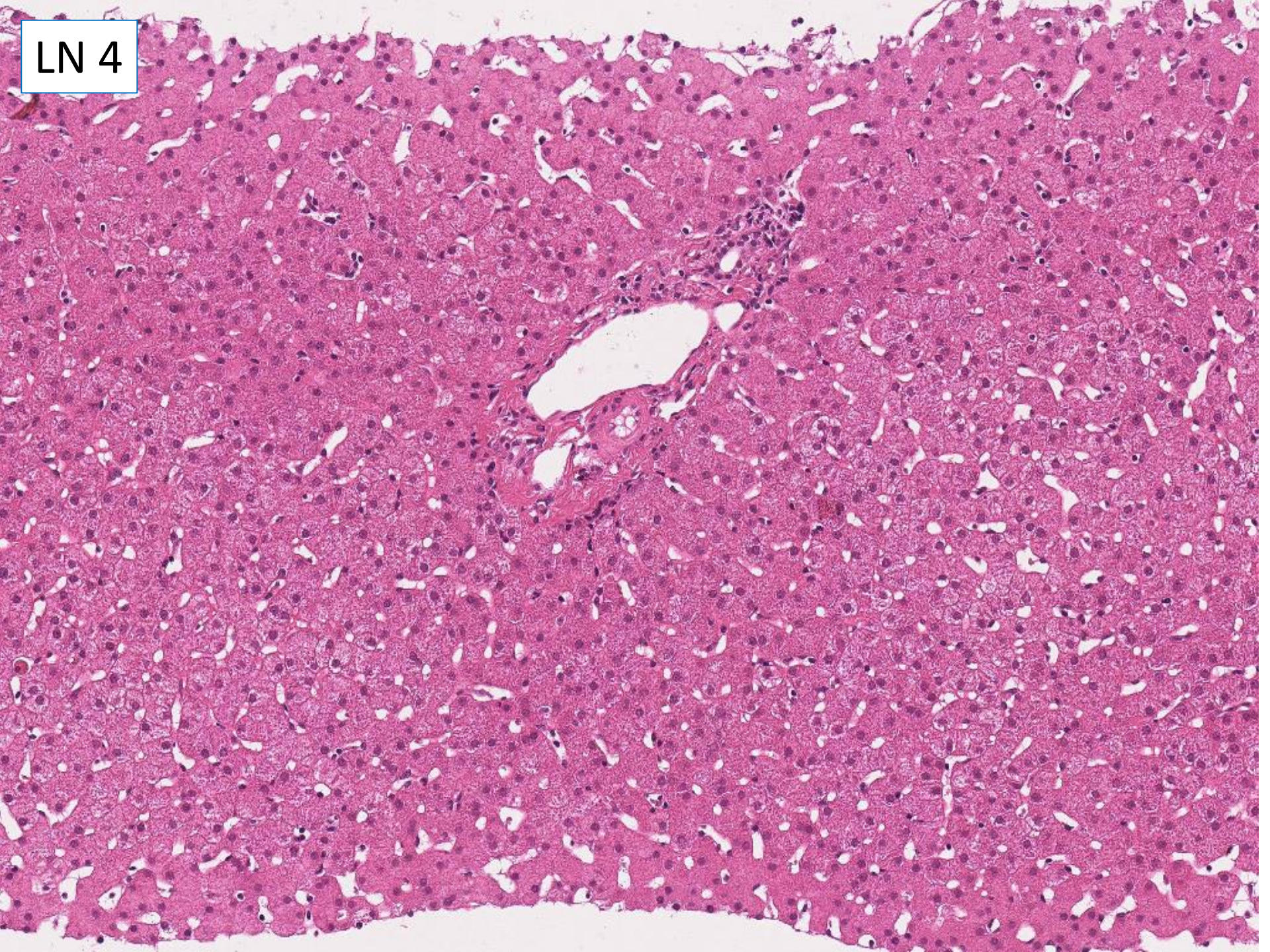
Comments: which stage to use? Kleiner for NAFLD, or text comment. Kleiner stage 2 is fibrosis affecting portal tracts and perivenular zone 3, but no bridging fibrosis. Ishak, designed for chronic hepatitis and not including perivenular fibrosis, doesn't work well for fatty liver disease. Staging systems are designed for research, morphological description is sufficient for diagnostic practice (whether or not fibrosis, bridging, late stage with nodules).

Discrepancy between fibroscan and biopsy stage – we have found that low fibroscan is good for excluding significant fibrosis, but high level can have other reasons.

LN4 Female 31 years

Hepatitis C Virus.


LN 4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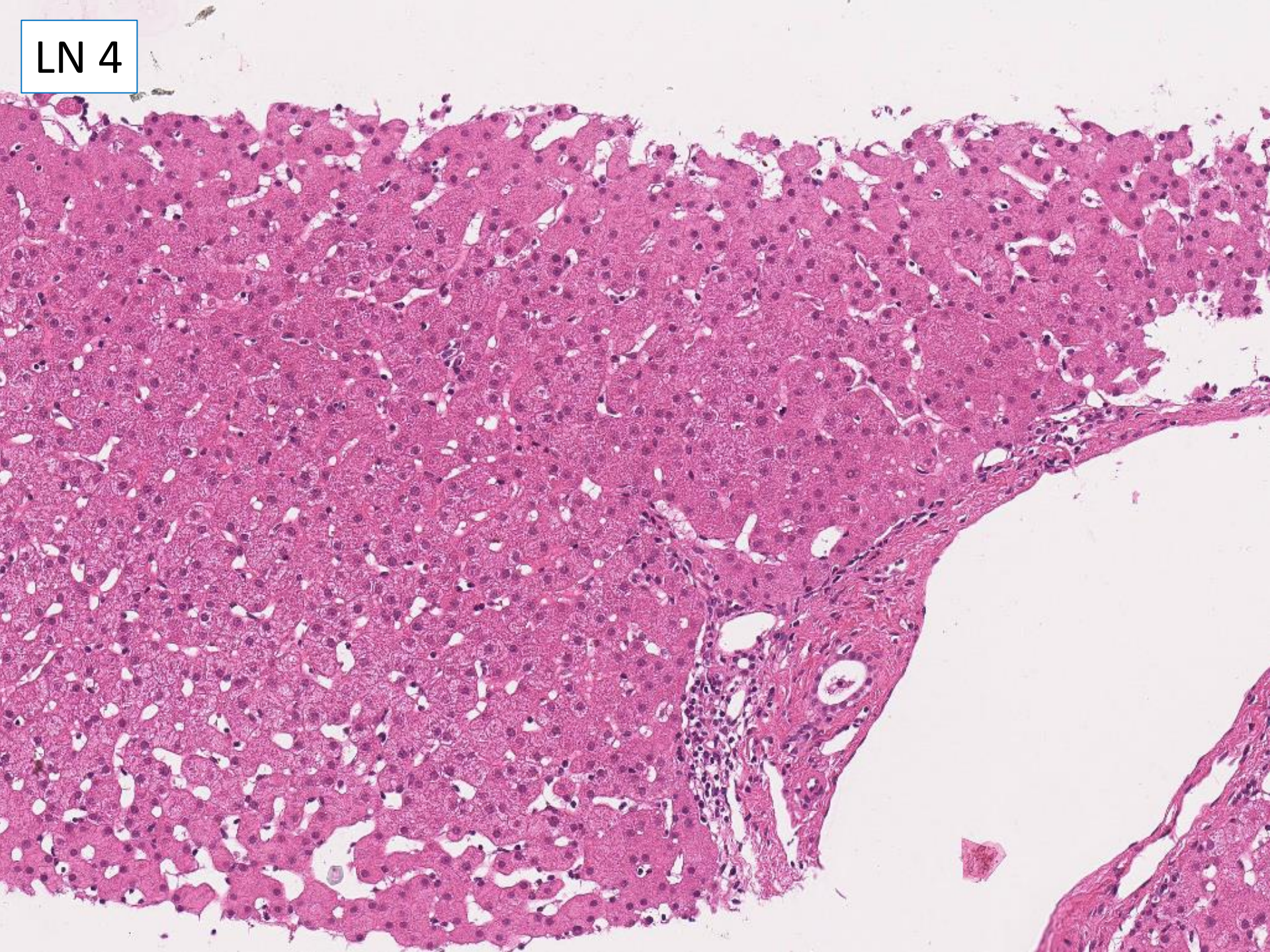
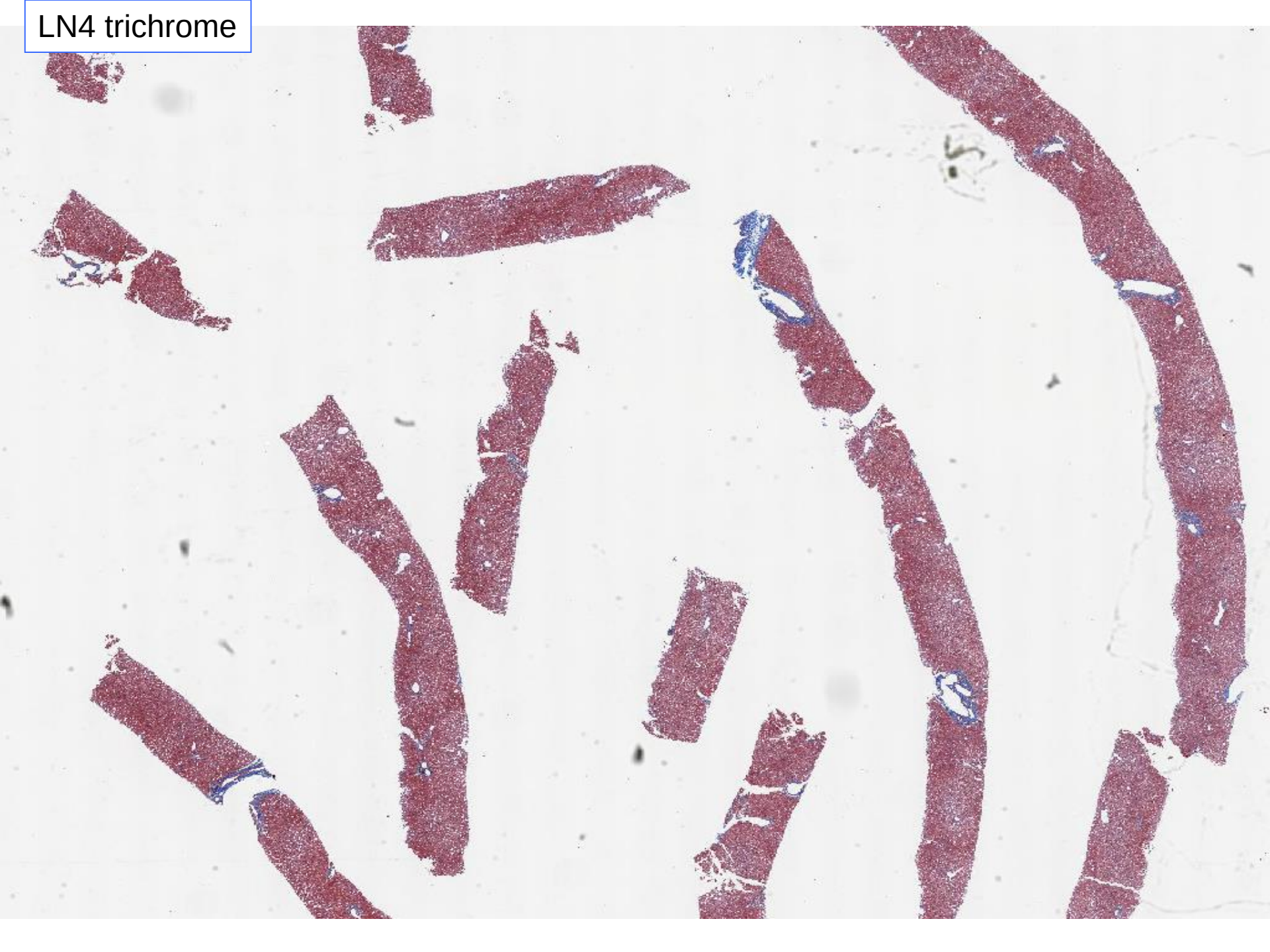
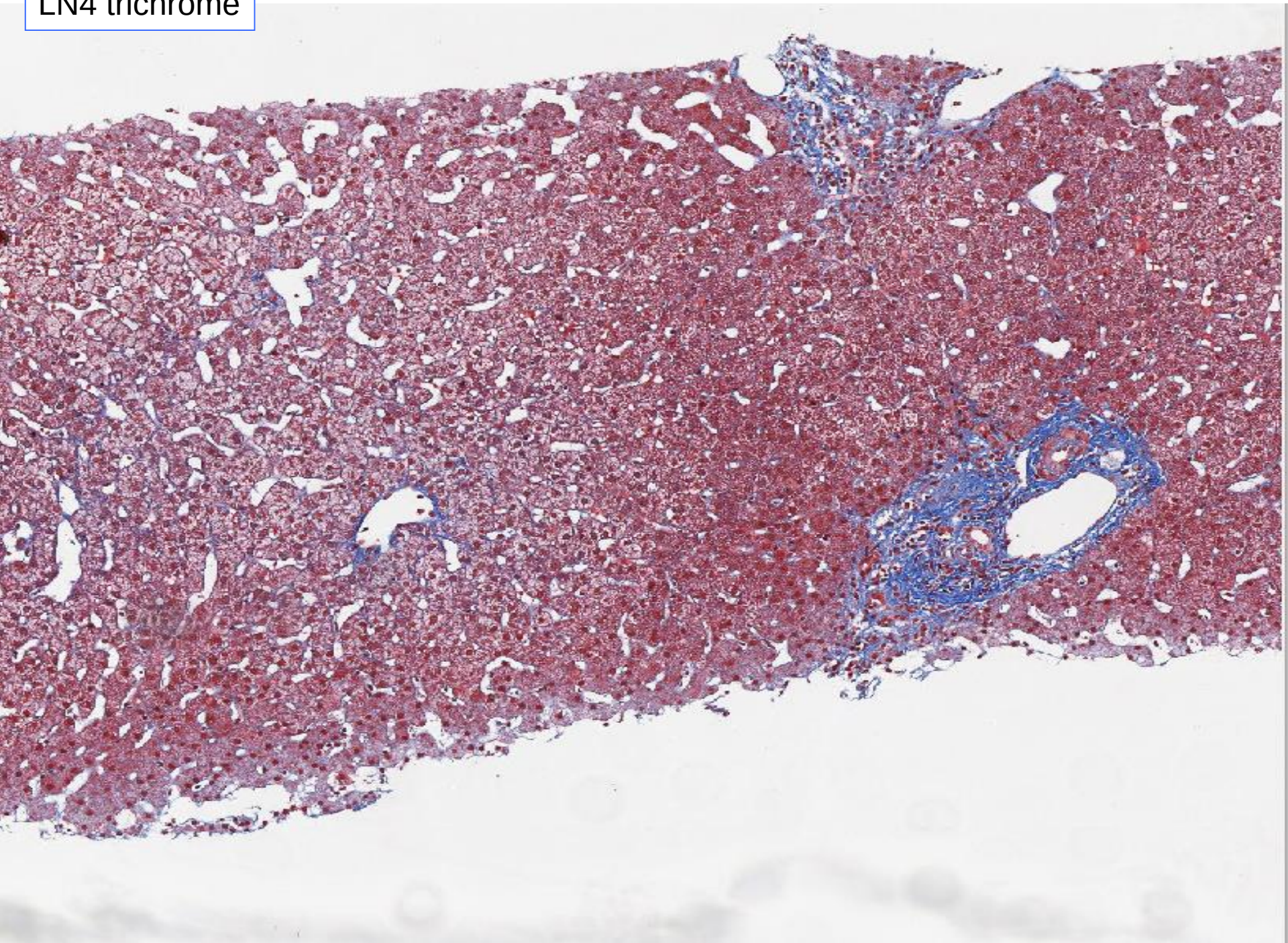

LN 4


LN4 trichrome

LN4 trichrome



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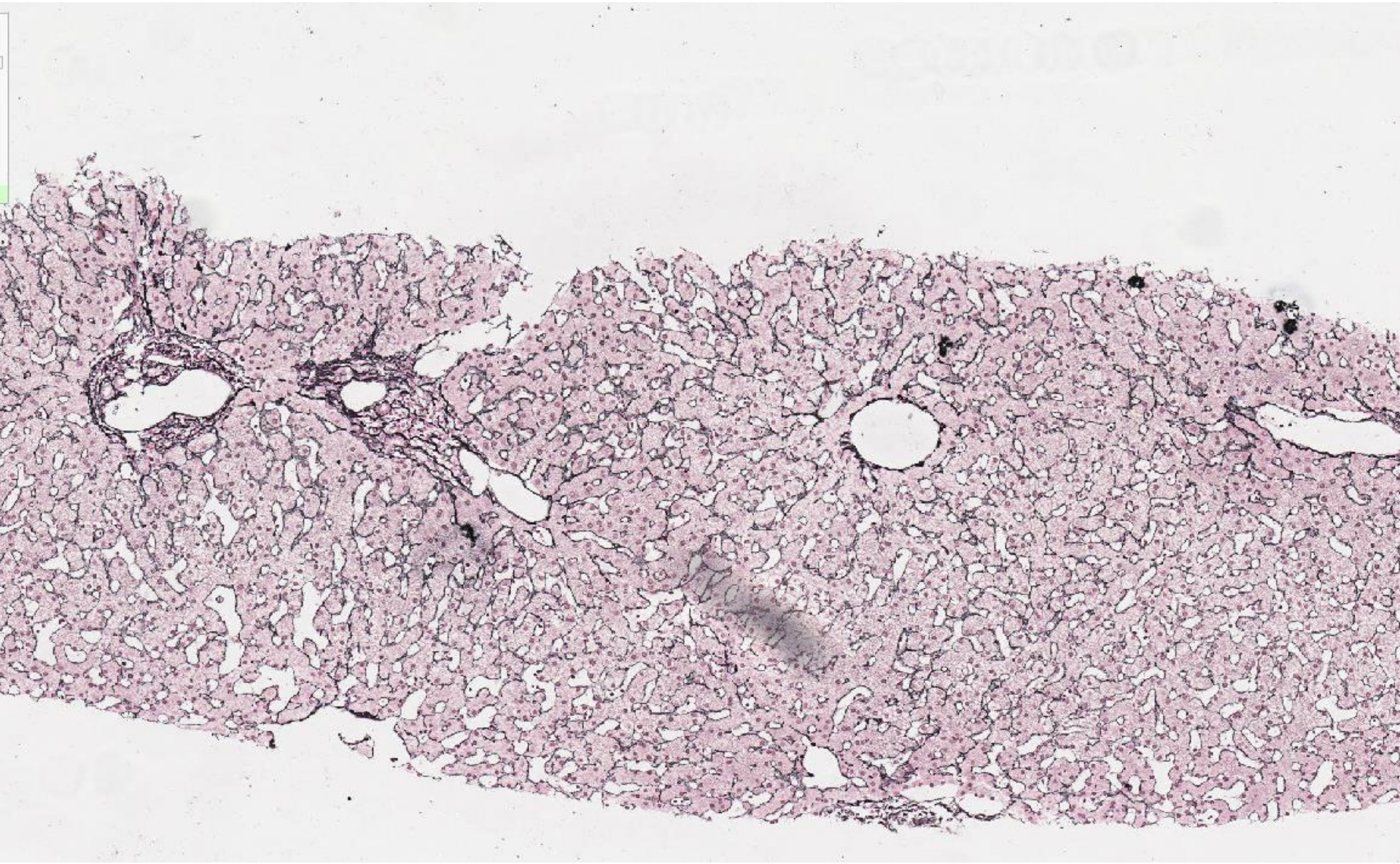


LN4 Female 31 years

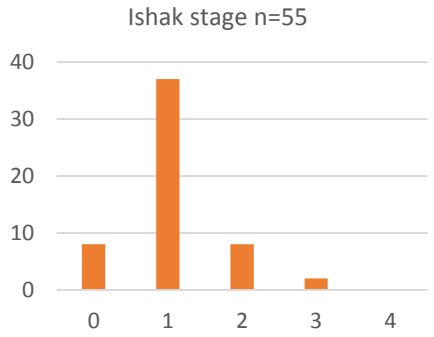
Hepatitis C Virus.

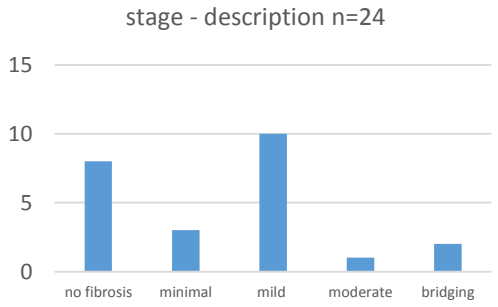
Description of chronic hepatitis, + state consistent with Hepatitis C	84
Hepatitis C not mentioned	2
STAGE/GRADE: no fibrosis stage or grade mentioned	2
Any comment on fibrosis stage	84

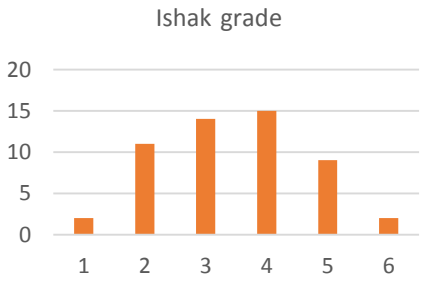
Suggested scoring:

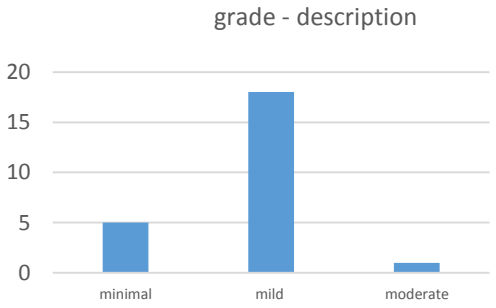
For full marks need to include hepatitis C, and some comment on stage. (based on similar cases in the past).

Stage/grade included for information. Only 3 used Metavir (A1/F1, A0/F1, A1/F1).









LN4 Female 31 years

Hepatitis C Virus.

Description of chronic hepatitis, + state consistent with Hepatitis C	84
Hepatitis C not mentioned	2
STAGE/GRADE: no fibrosis stage or grade mentioned	2
Any comment on fibrosis stage	84

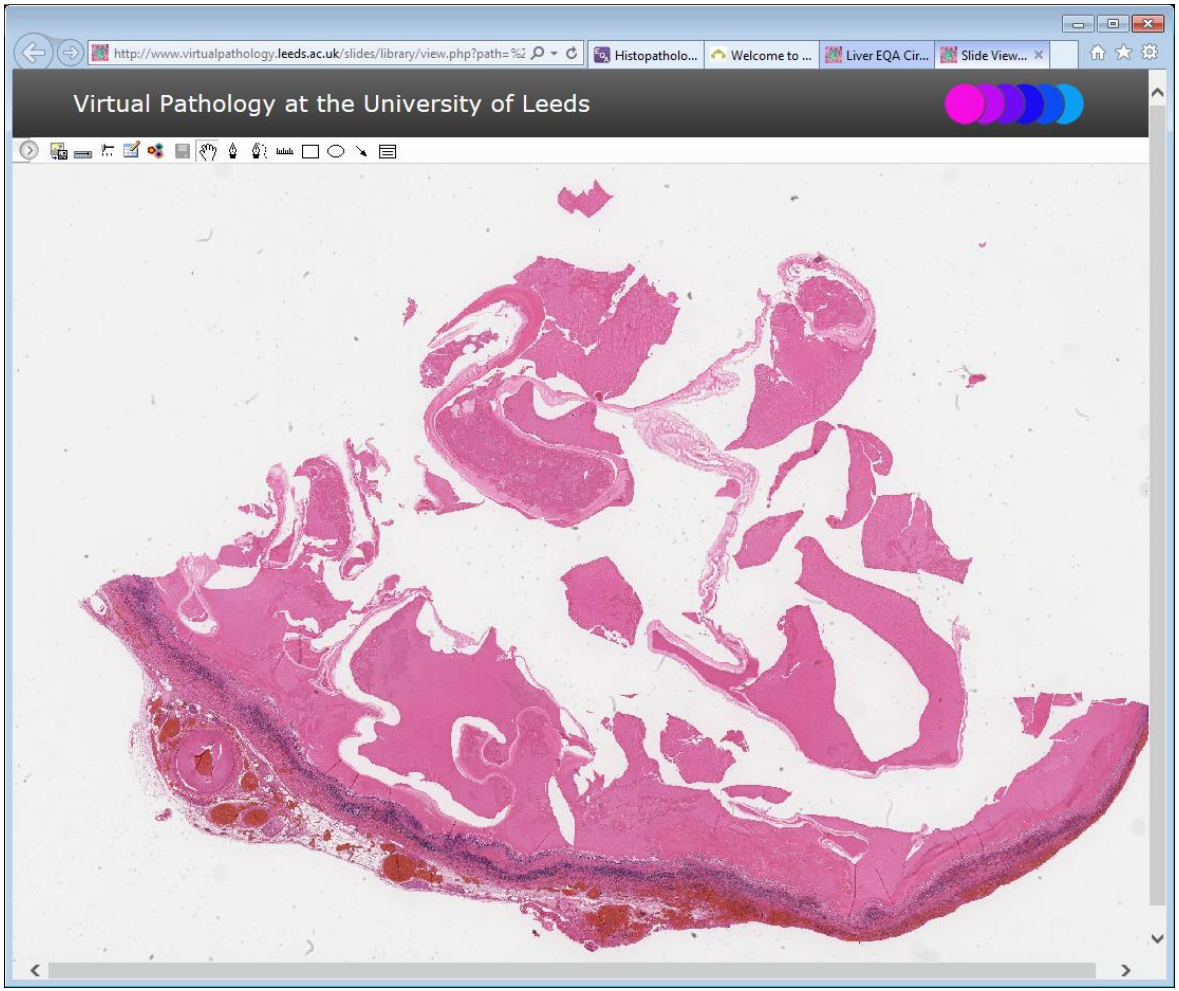
LN4 agreed scoring after meeting dicussion:

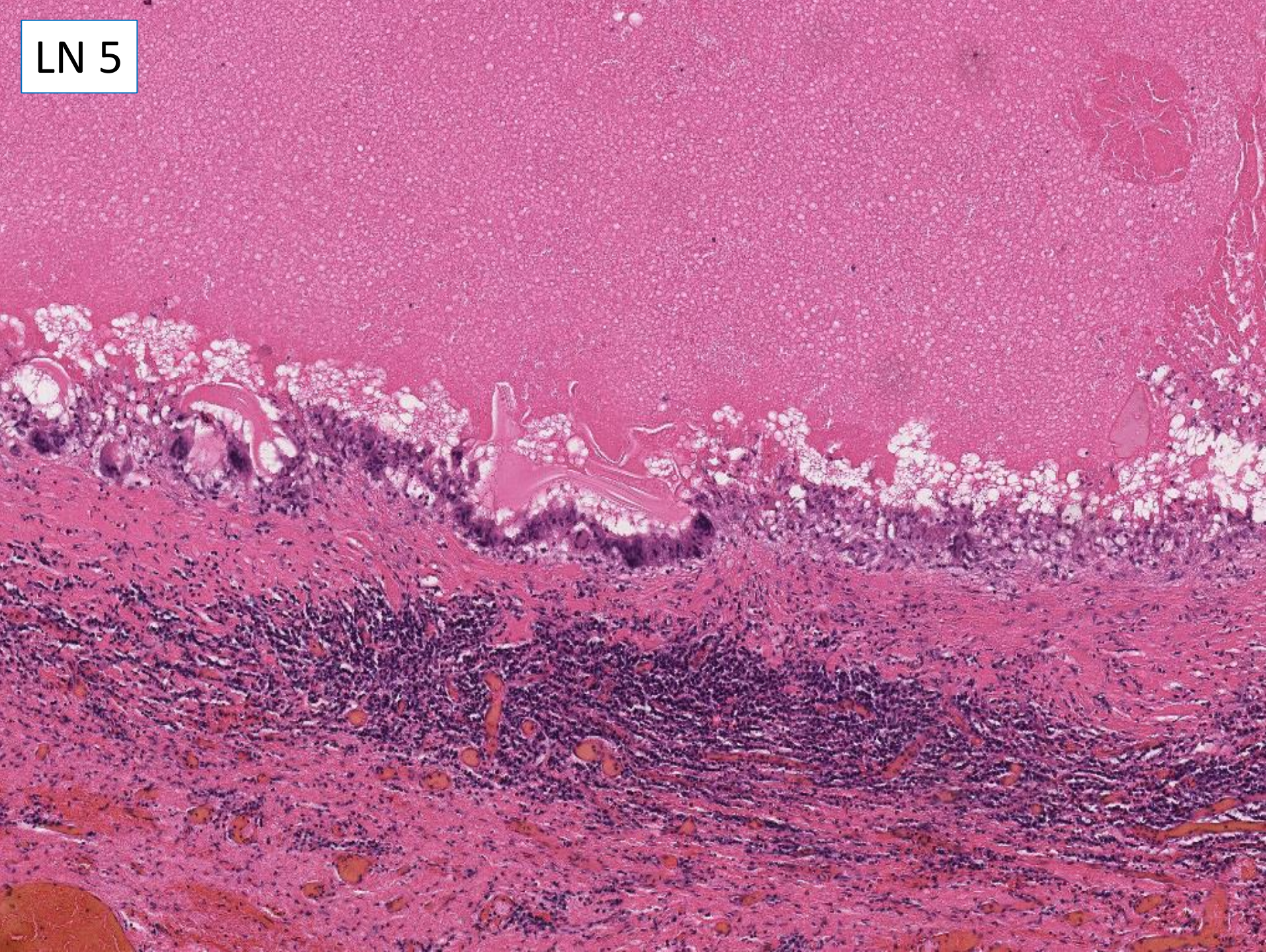
For full marks, include chronic hepatitis, consistent with hepatitis C, and a comment on fibrosis stage, either Ishak or description. Half marks if either is missing.

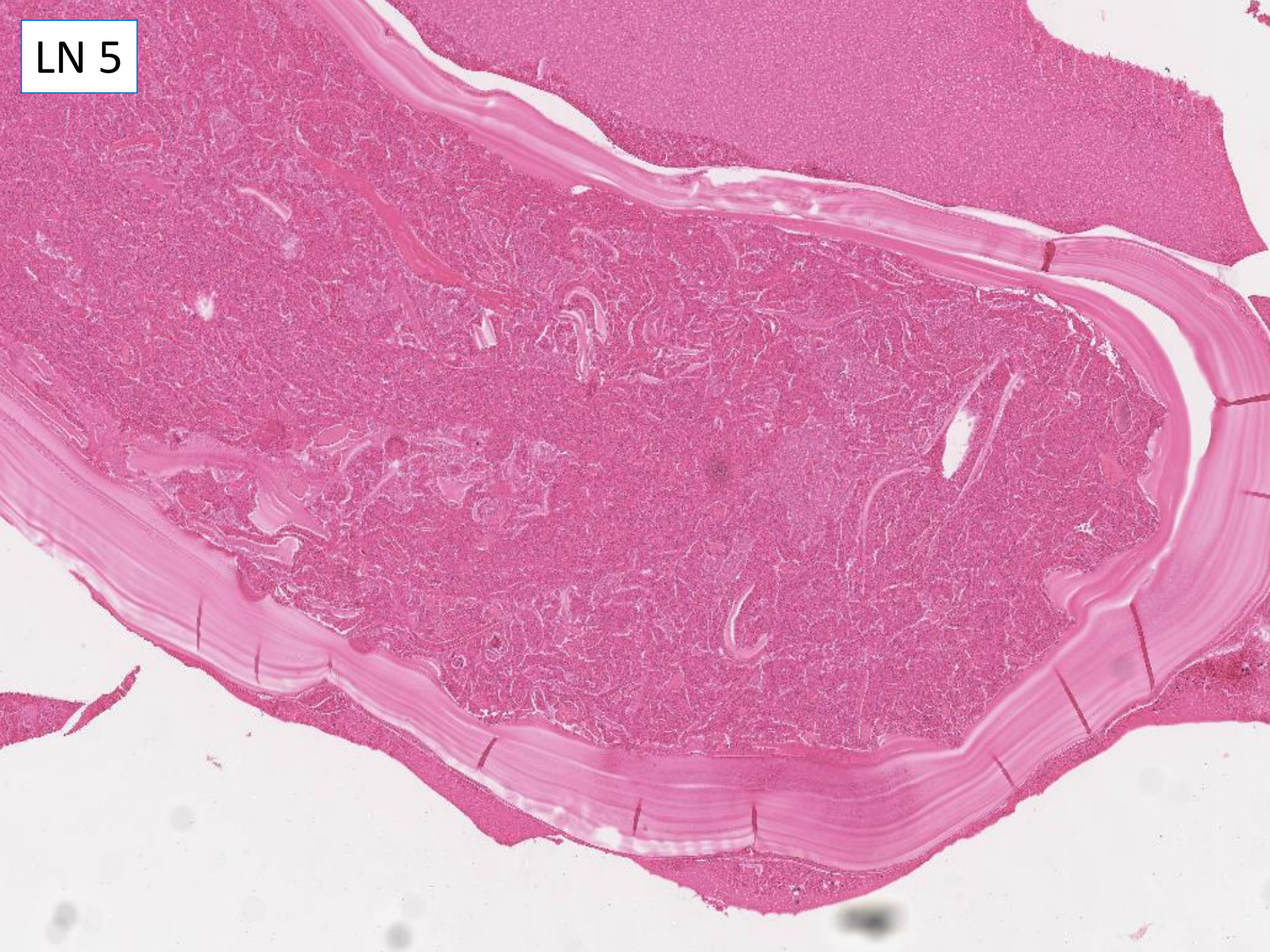
Comment – avoid “chronic active hepatitis” – not current terminology, was part of previous morphological classification of chronic persistent/chronic active or aggressive/ chronic lobular hepatitis, which has been replaced by aetiology and stage/grade terminology.

LN5 Male 34 years

Hydatid cyst 4B. Specimen: Hydatid Cyst from liver.




LN 5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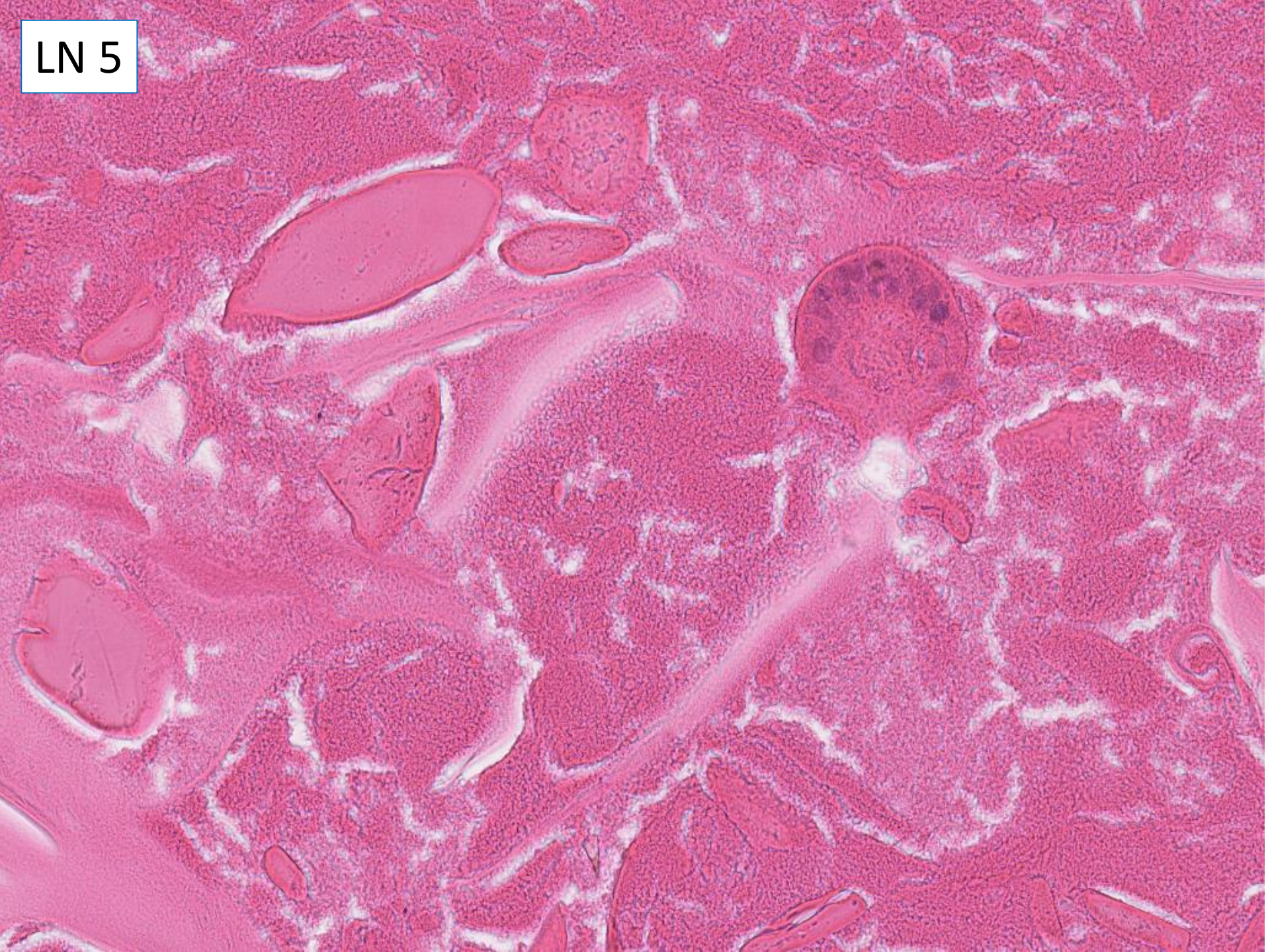

LN 5

LN5 Male 34 years

Hydatid cyst 4B. Specimen: Hydatid Cyst from liver.

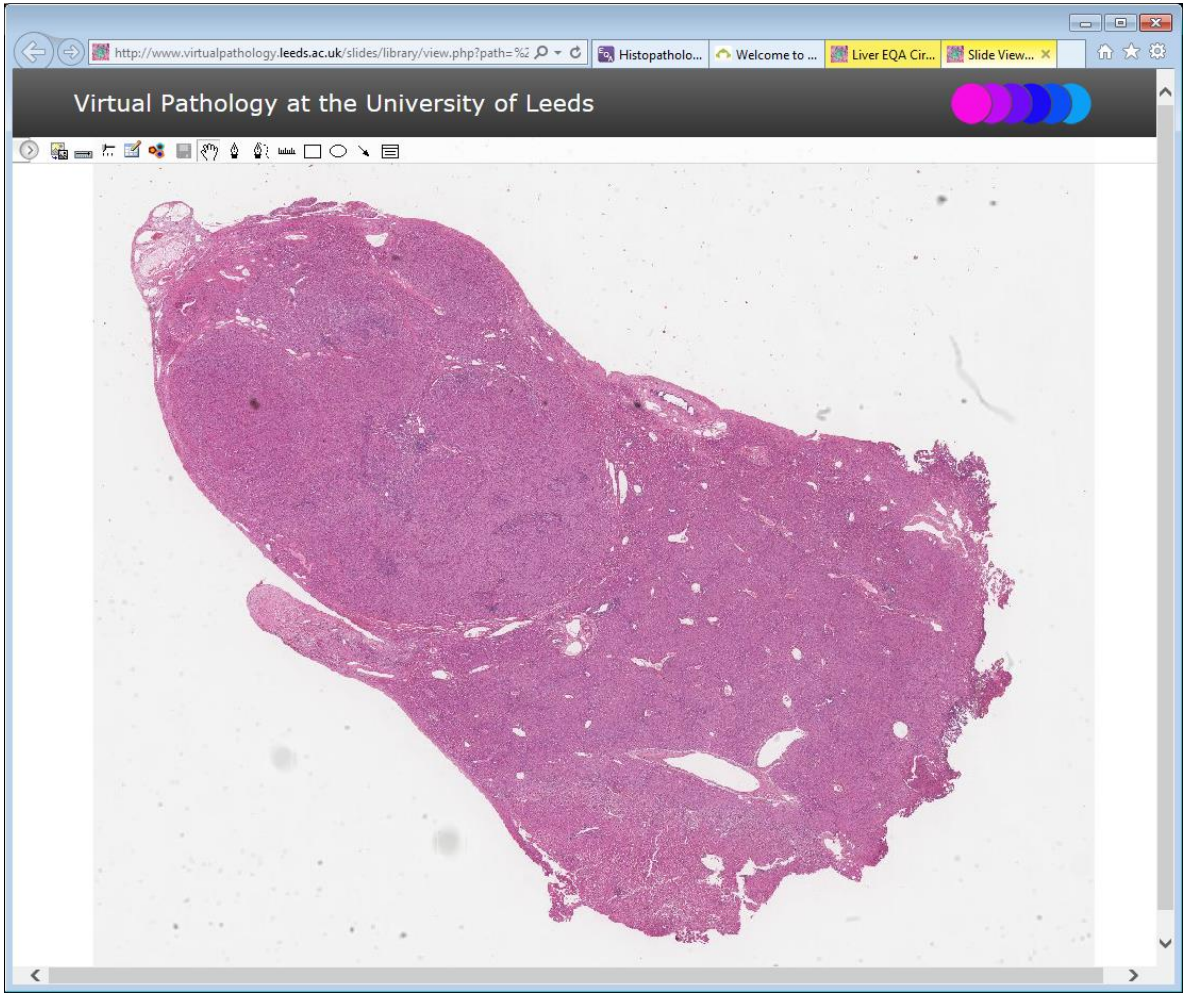
Hydatid cyst	86

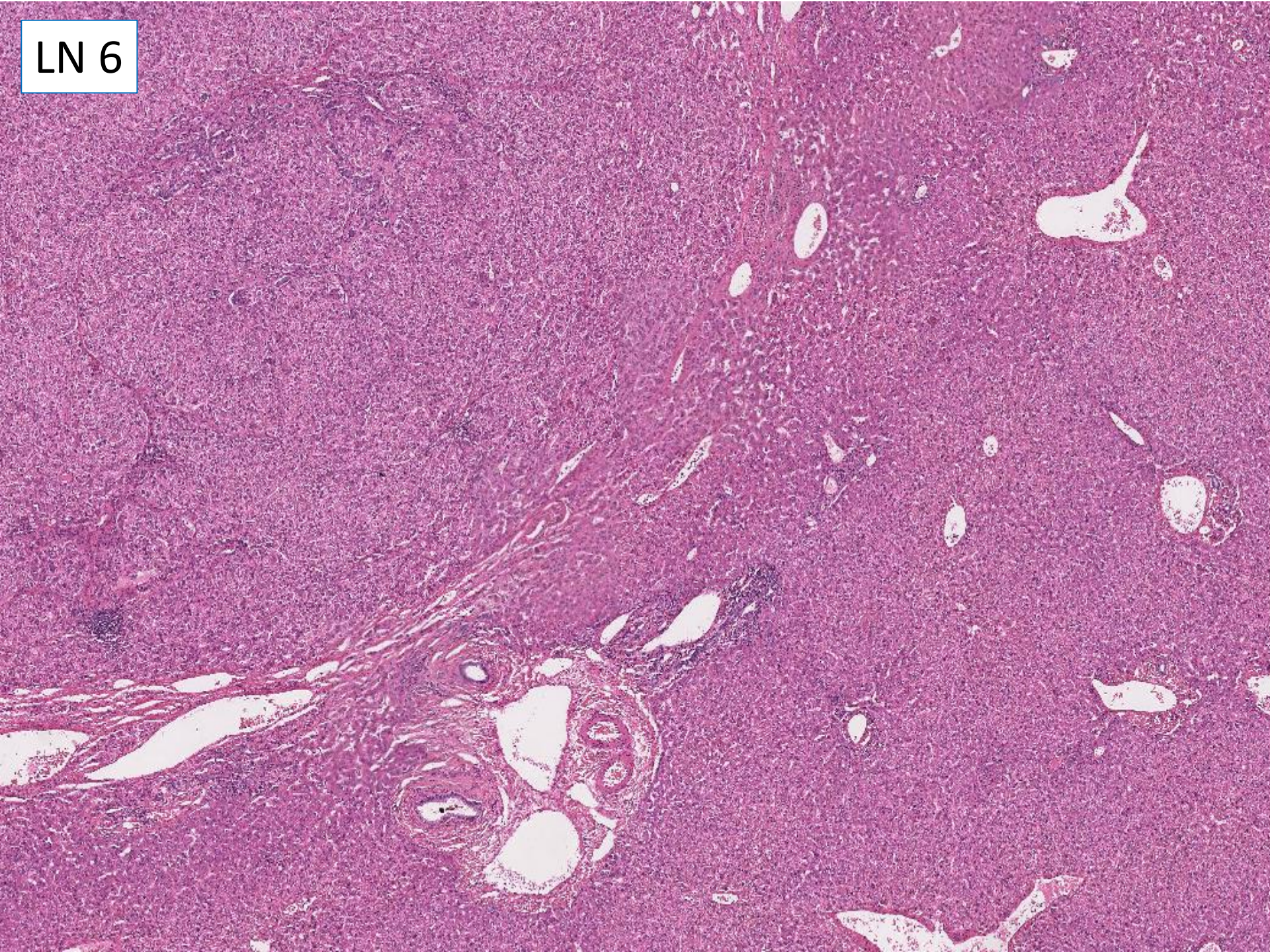
LN5 final scoring after meeting discussion:
for full marks, diagnosis of hydatid cyst.
All scored full marks

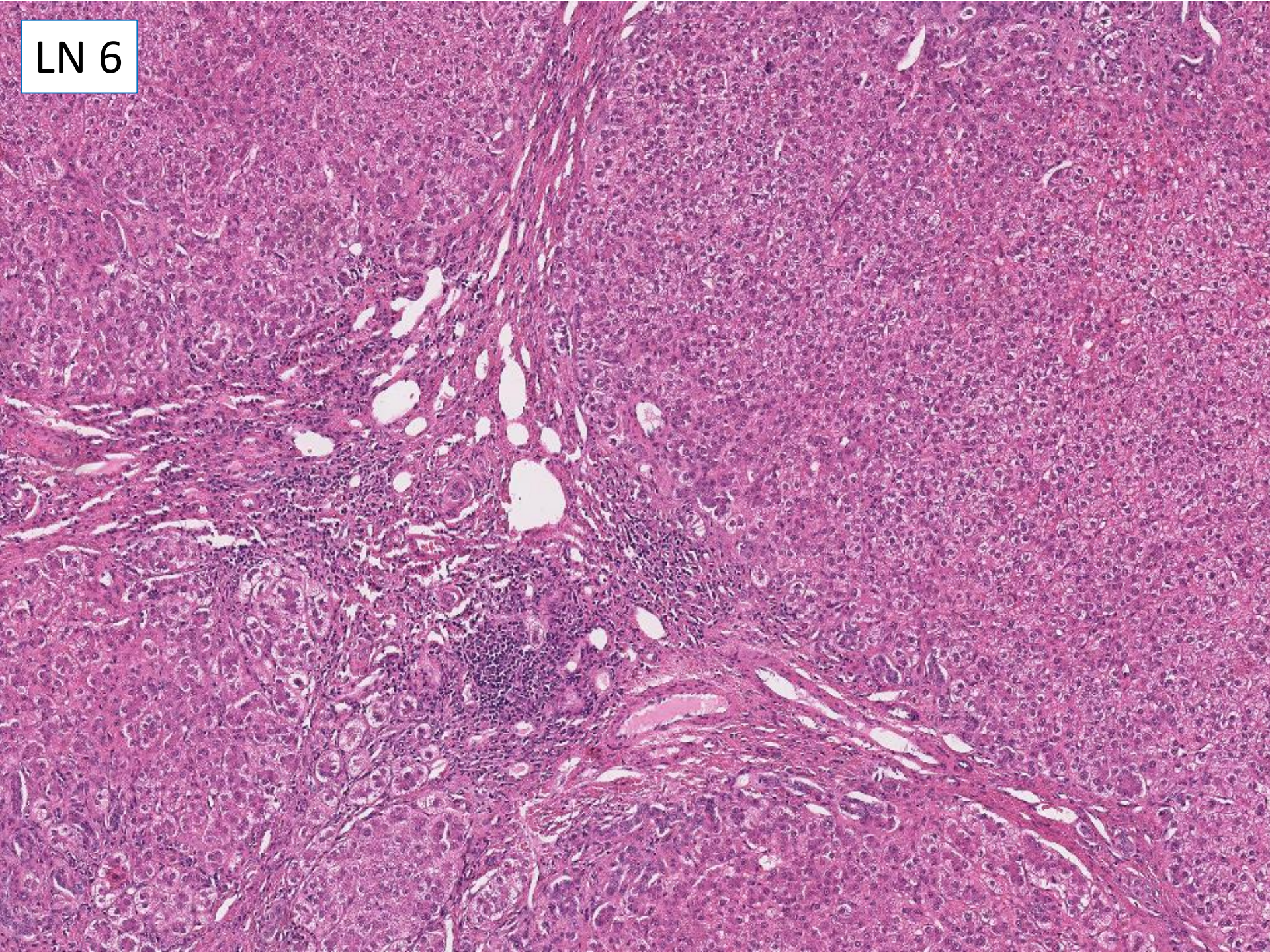
LN6 Male 34 years

Hydatid cyst 4B (NB this is an incidental lesion discovered during resection of the cyst).

Specimen: Segment III Lesion.




LN 6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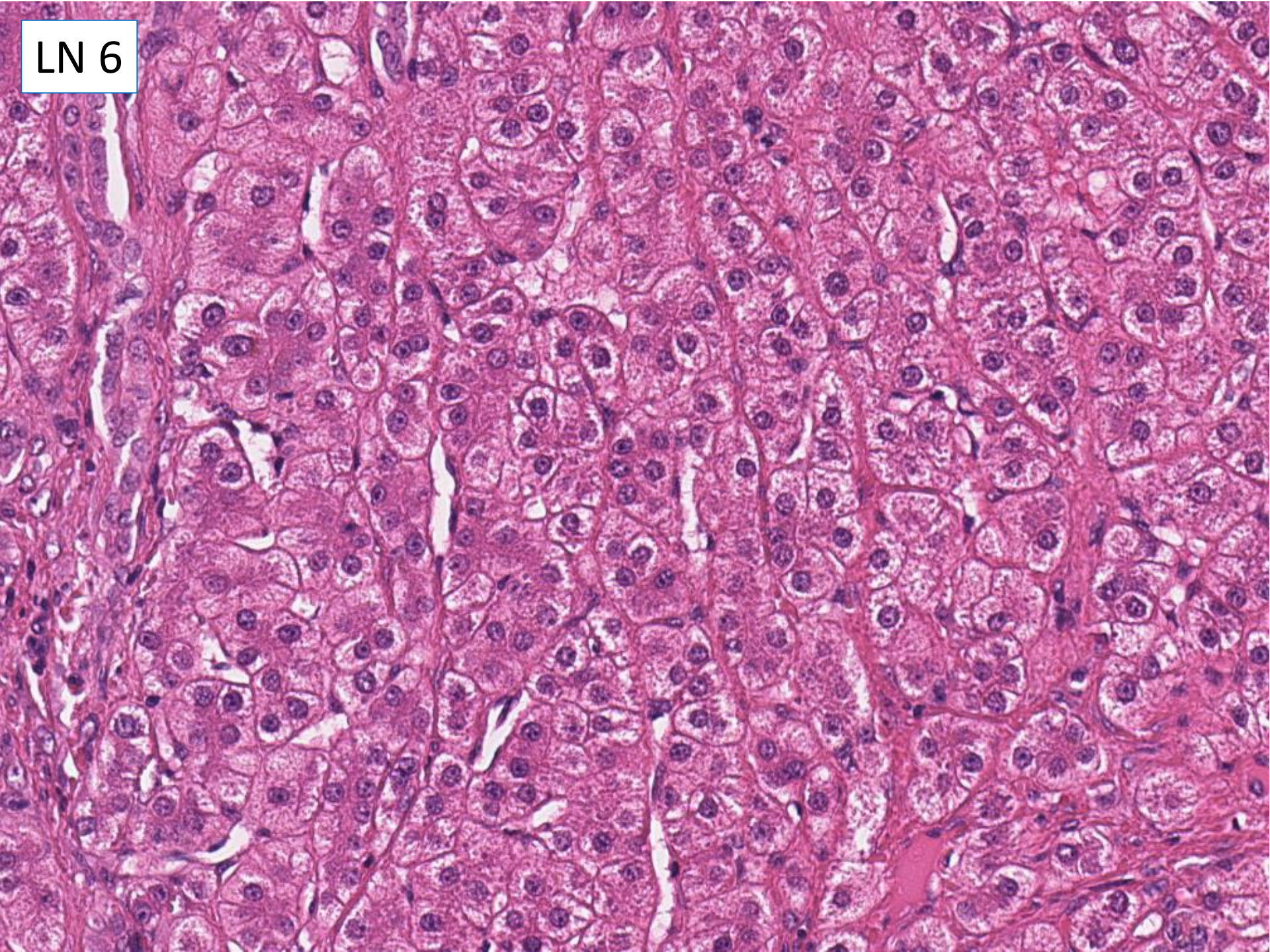

LN 6

LN6 Male 34 years

Hydatid cyst 4B (NB this is an incidental lesion discovered during resection of the cyst). Specimen: Segment III Lesion.

Focal nodular hyperplasia	73
Focal nodular hyperplasia – glutamine synthetase to confirm	11
Consistent with focal nodular hyperplasia – correlate with imaging	Full marks
FNH differential diagnosis well differentiated HCC	1
Of interest – *'focal nodular hyperplasia is described in close proximity to hydatid cyst'*	

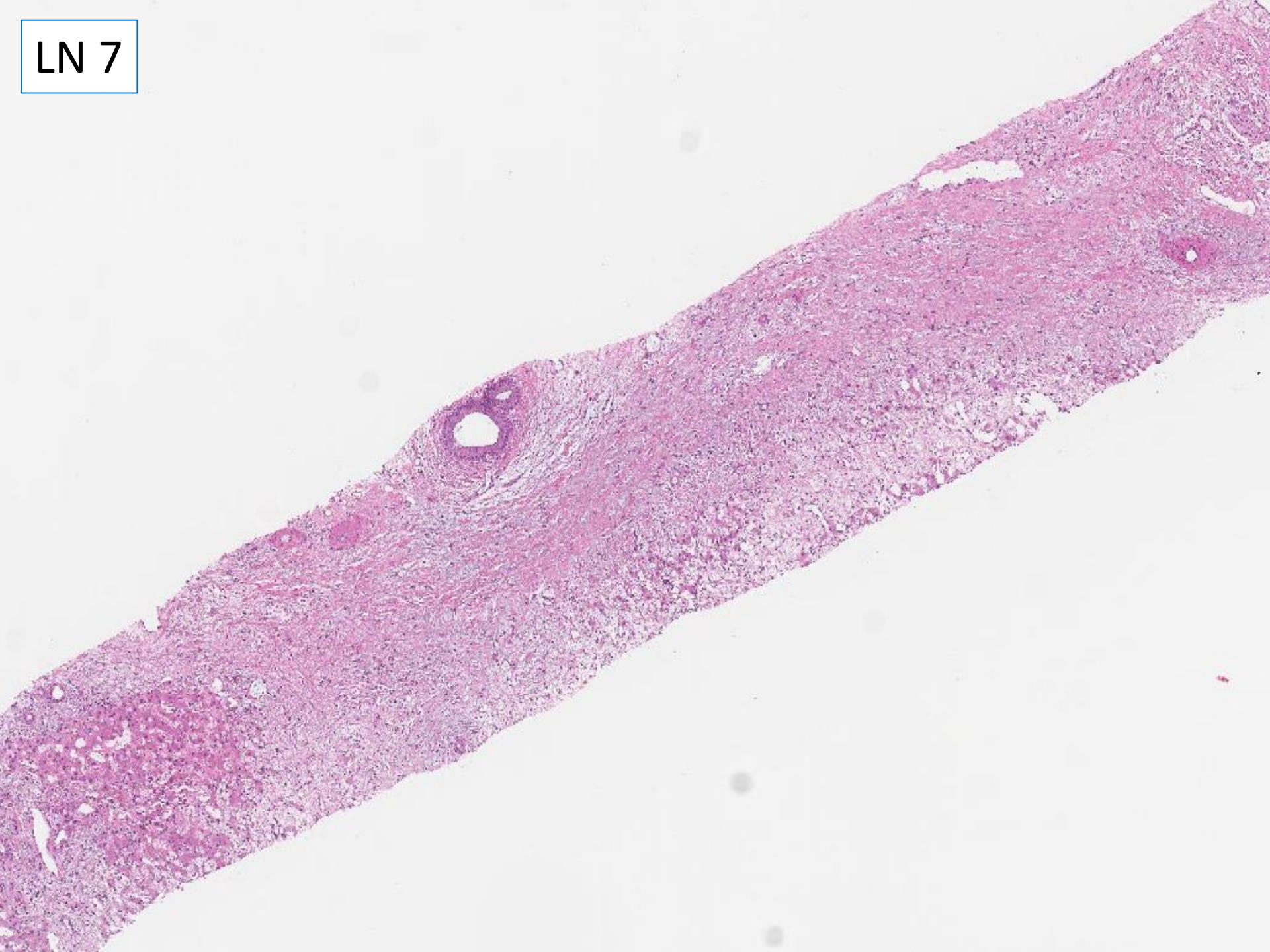
Suggested scoring: for full marks, clear statement that this is FNH.

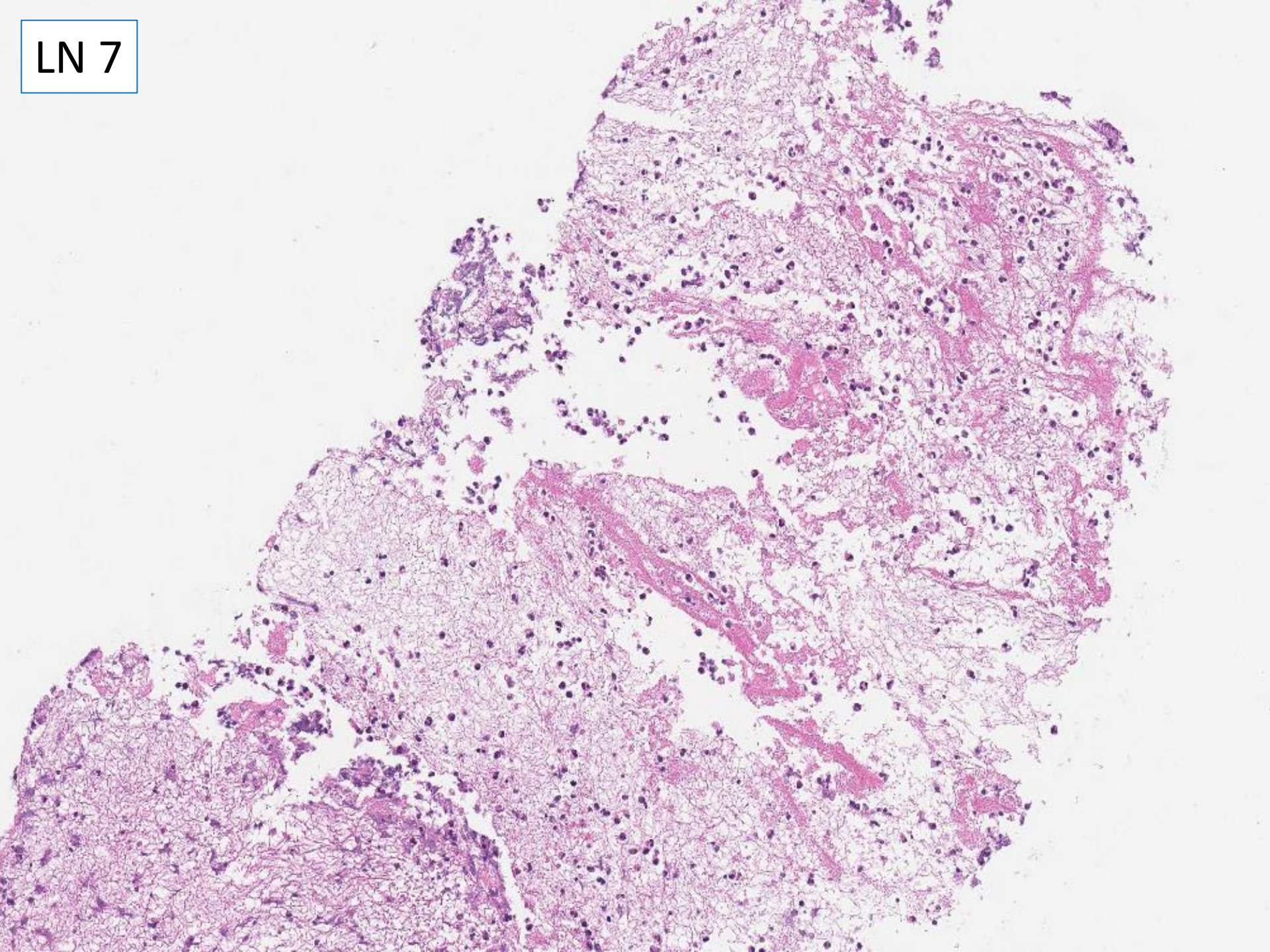
LN6 Final scoring after meeting discussion: for full marks, clear diagnosis on FNH.
On review of responses, this was clearly the intended diagnosis of responses where a differential diagnosis was raised, so all score full marks.

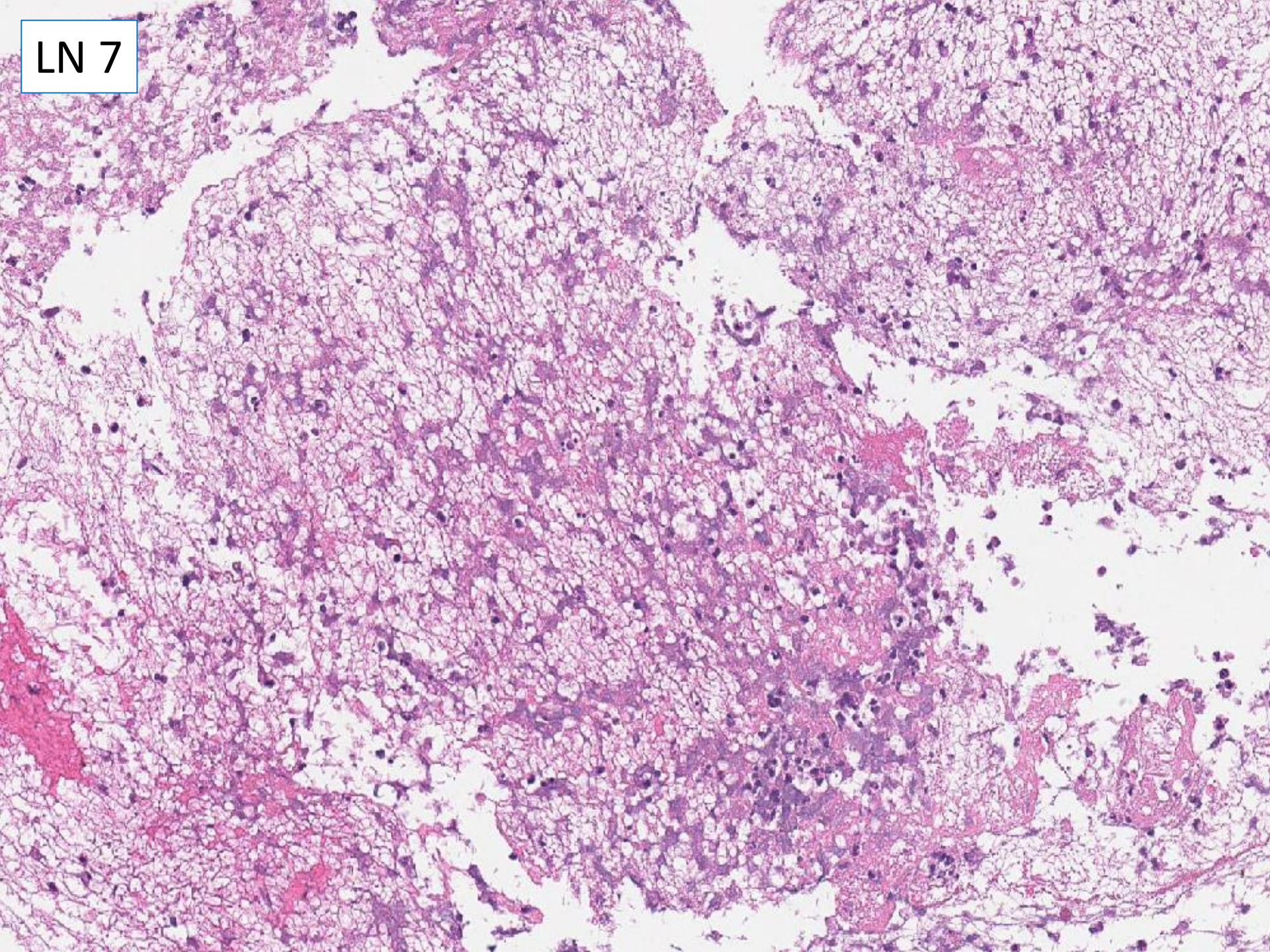
Focal nodular hyperplasia has been described in the vicinity of various other focal lesions in the liver, and the vicinity of hydatid cyst (this case) would be an example of that.

LN7 Male 63 years

Right liver abscess drained. Residual solid tissue in wall ?? abscess wall. Rule out tumour. No previous history of malignancy.


LN 7


LN 7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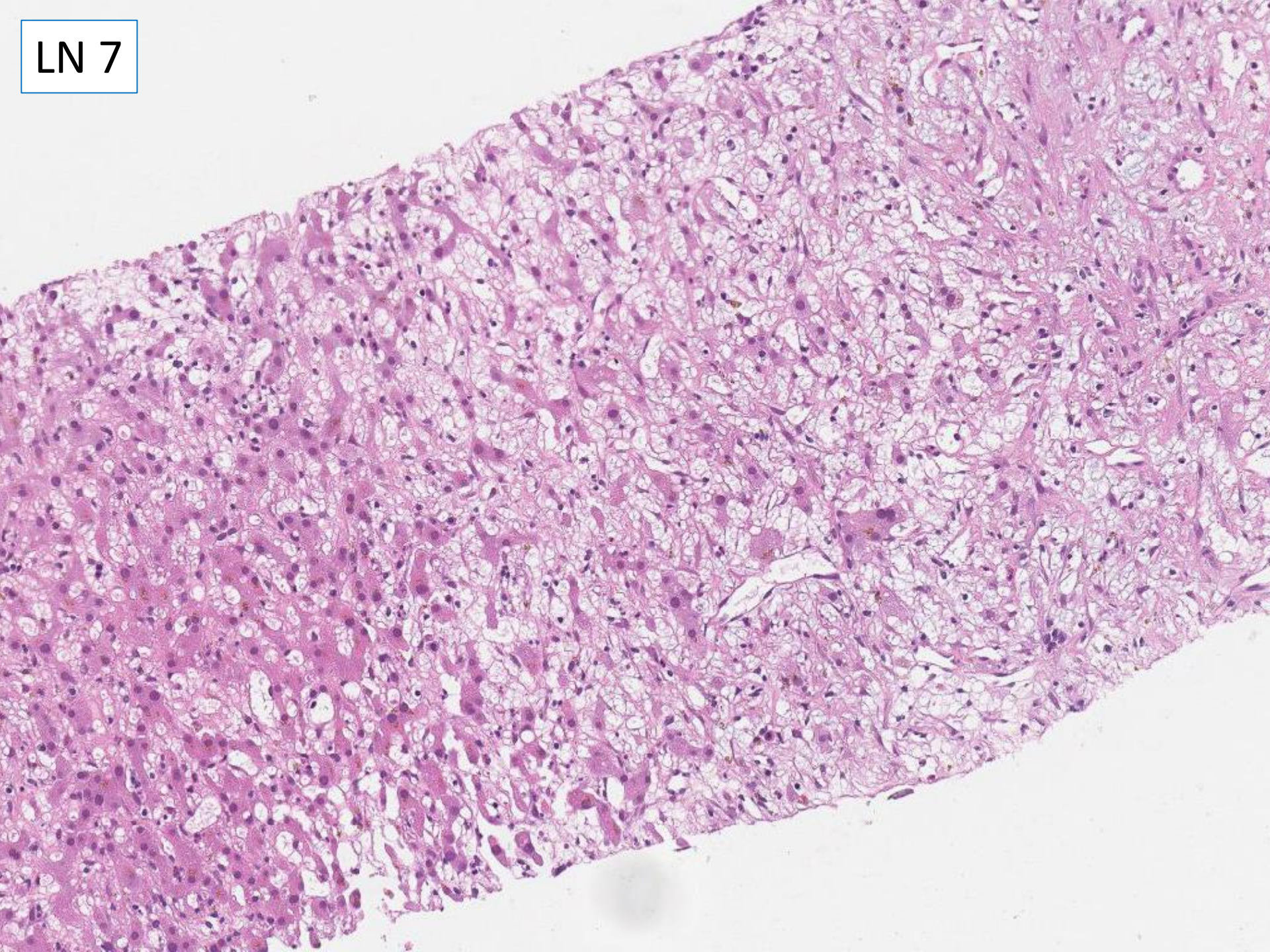
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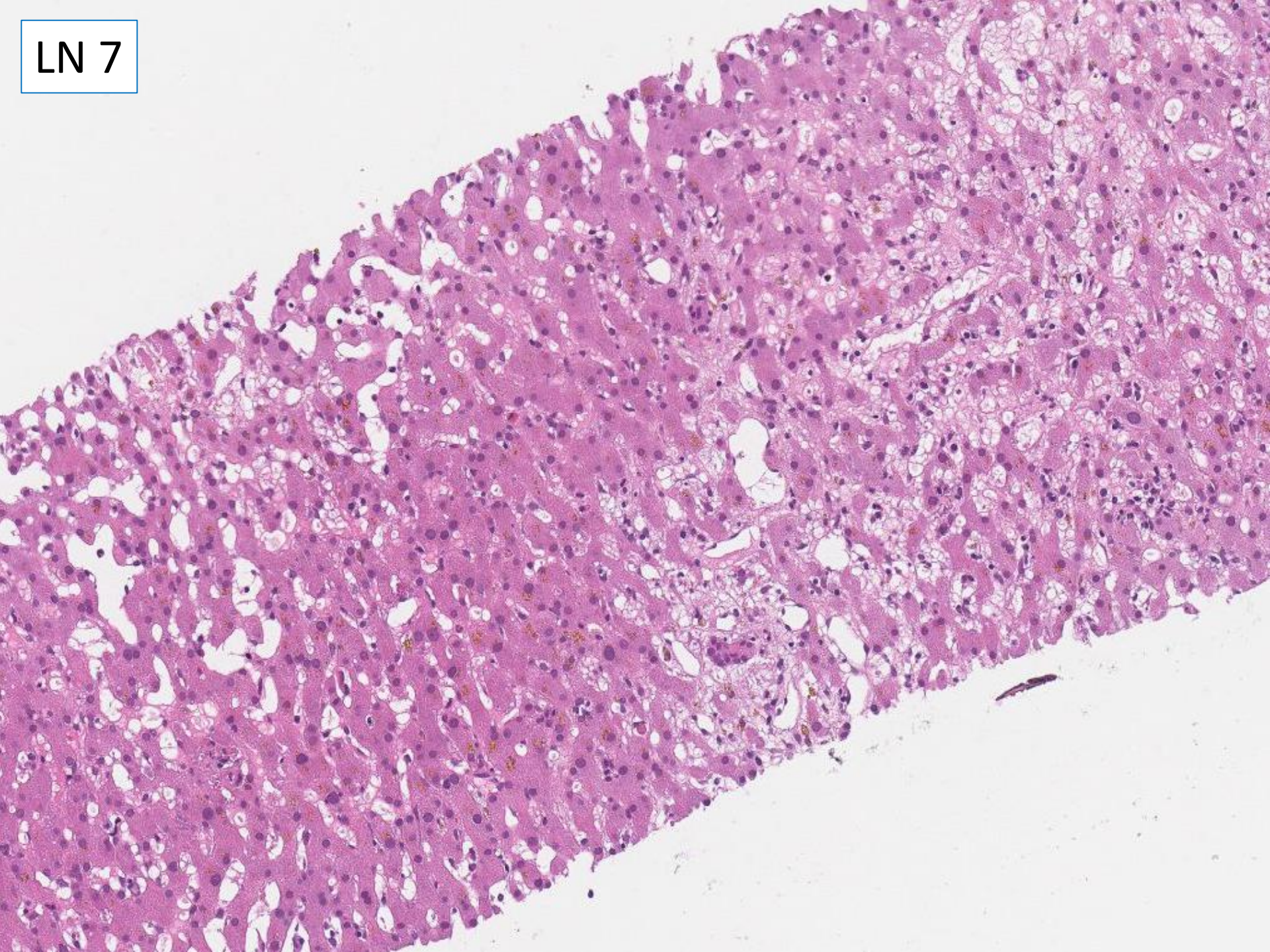
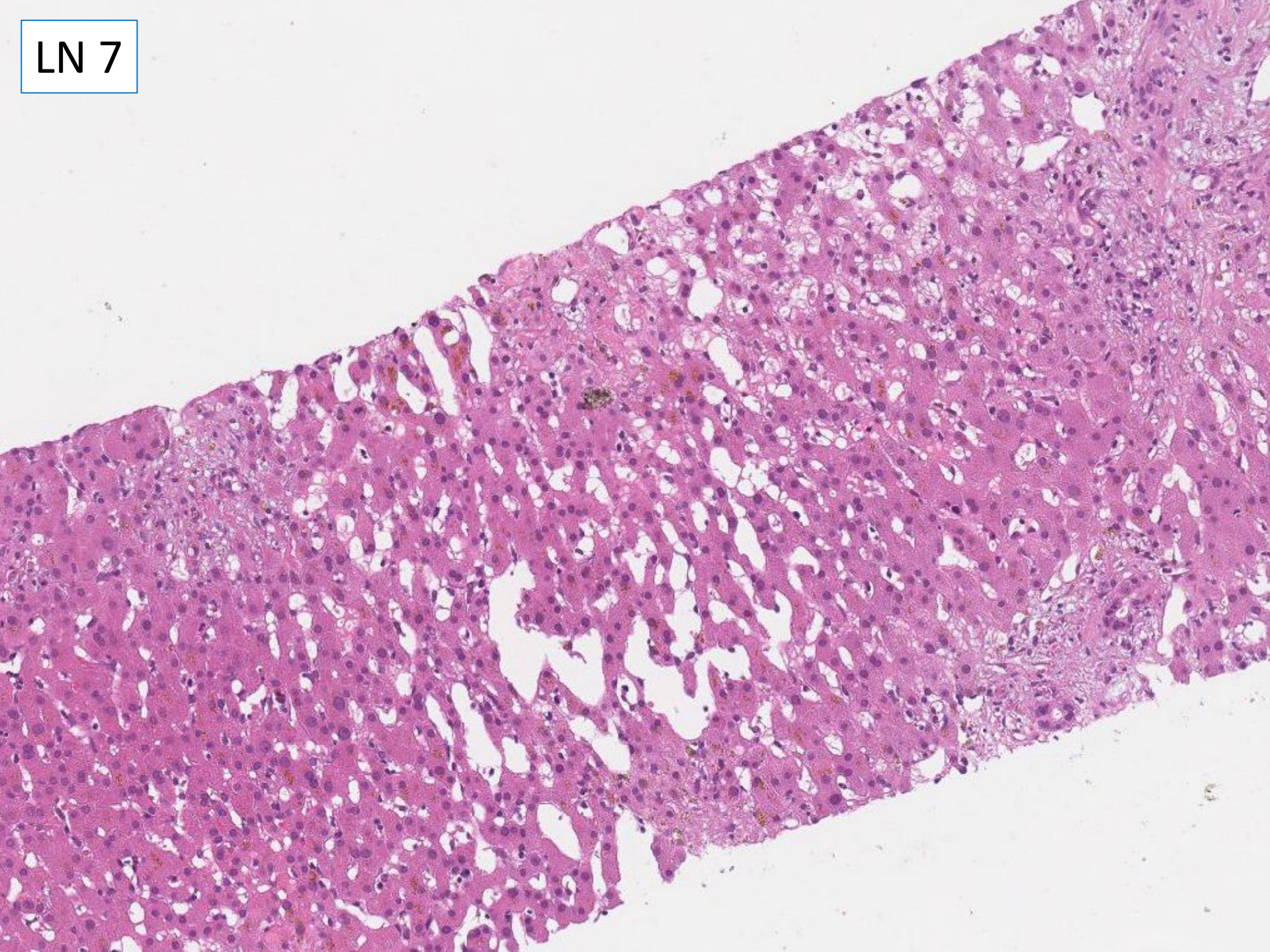

LN 7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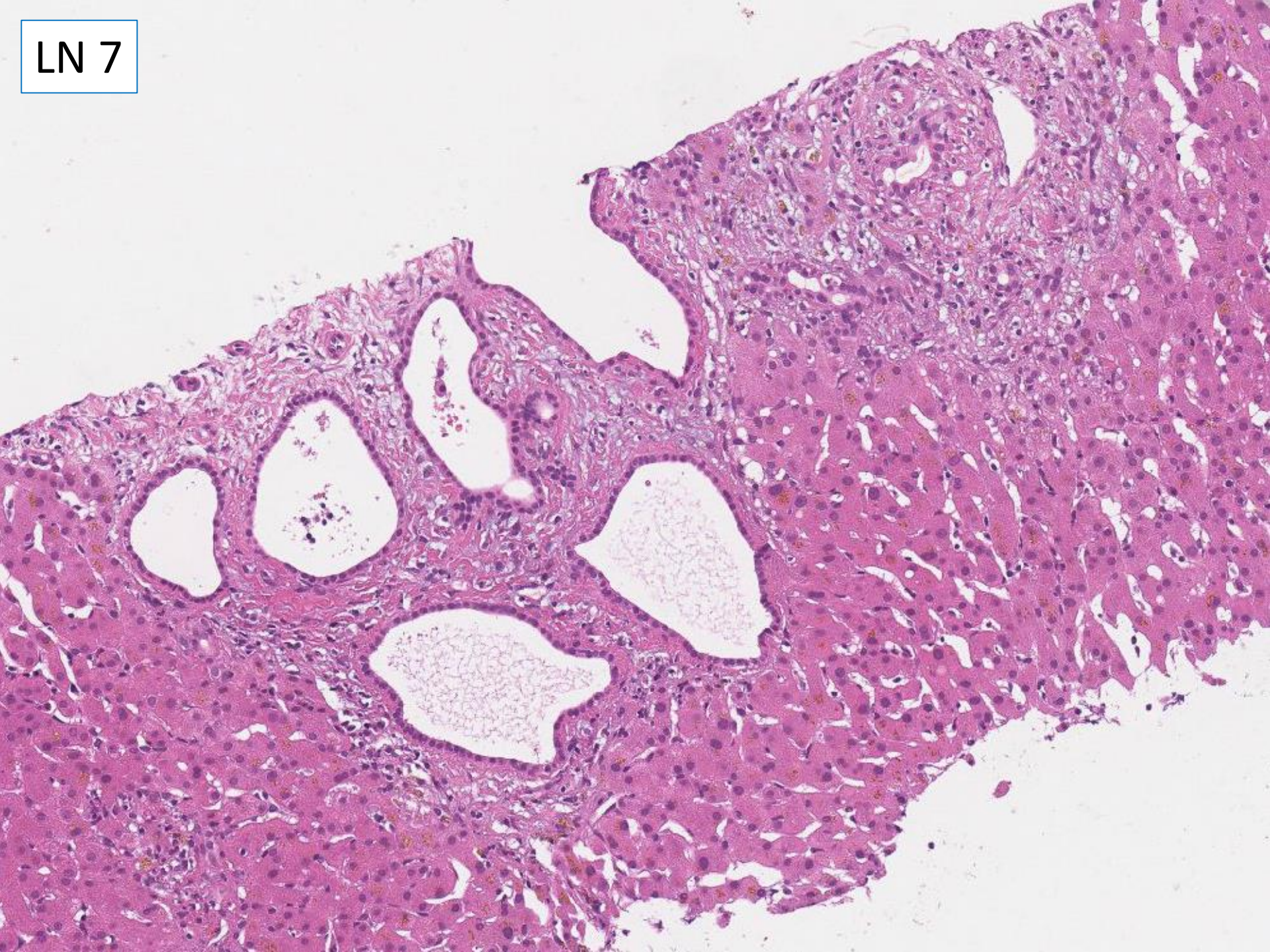
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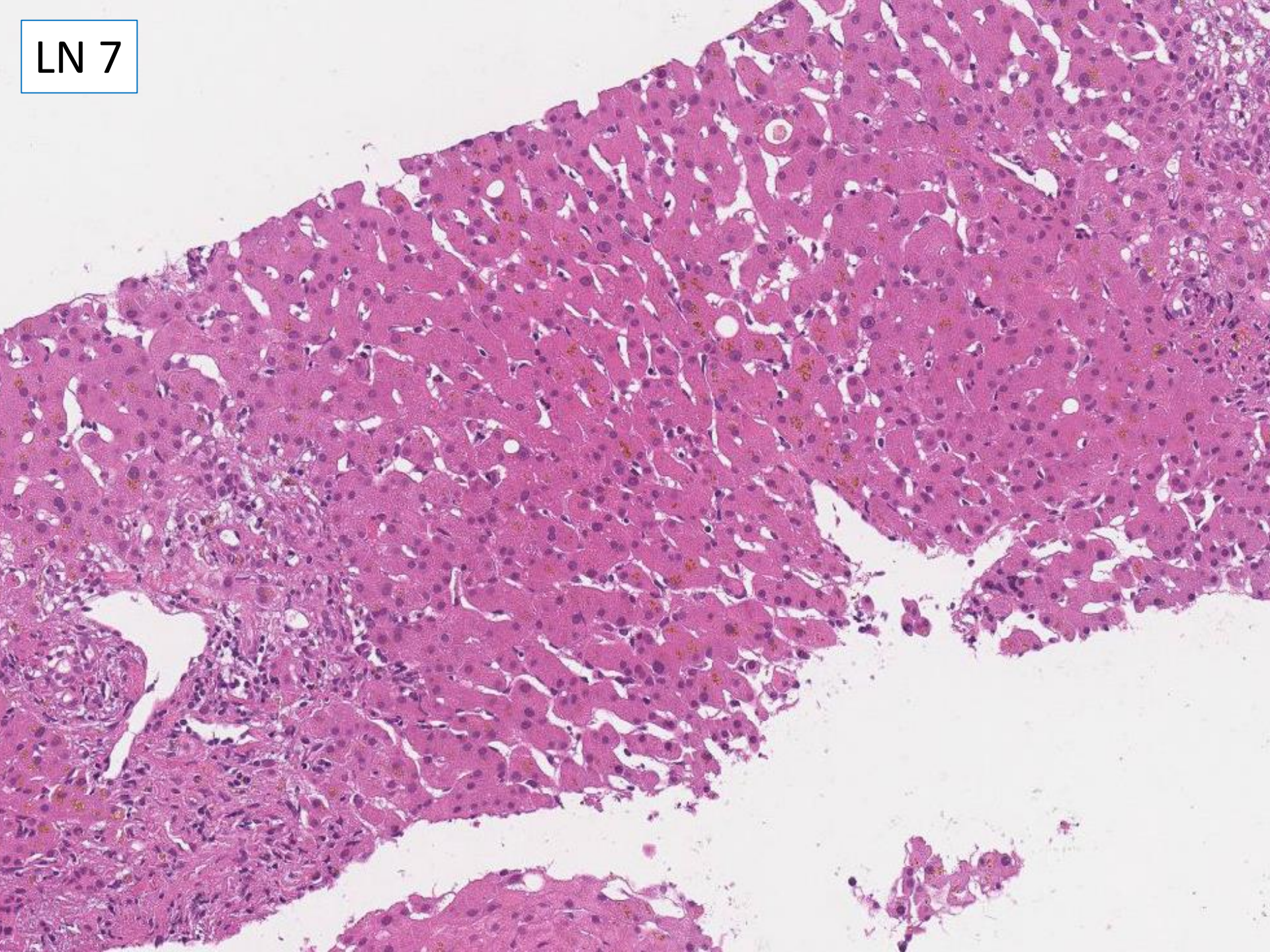
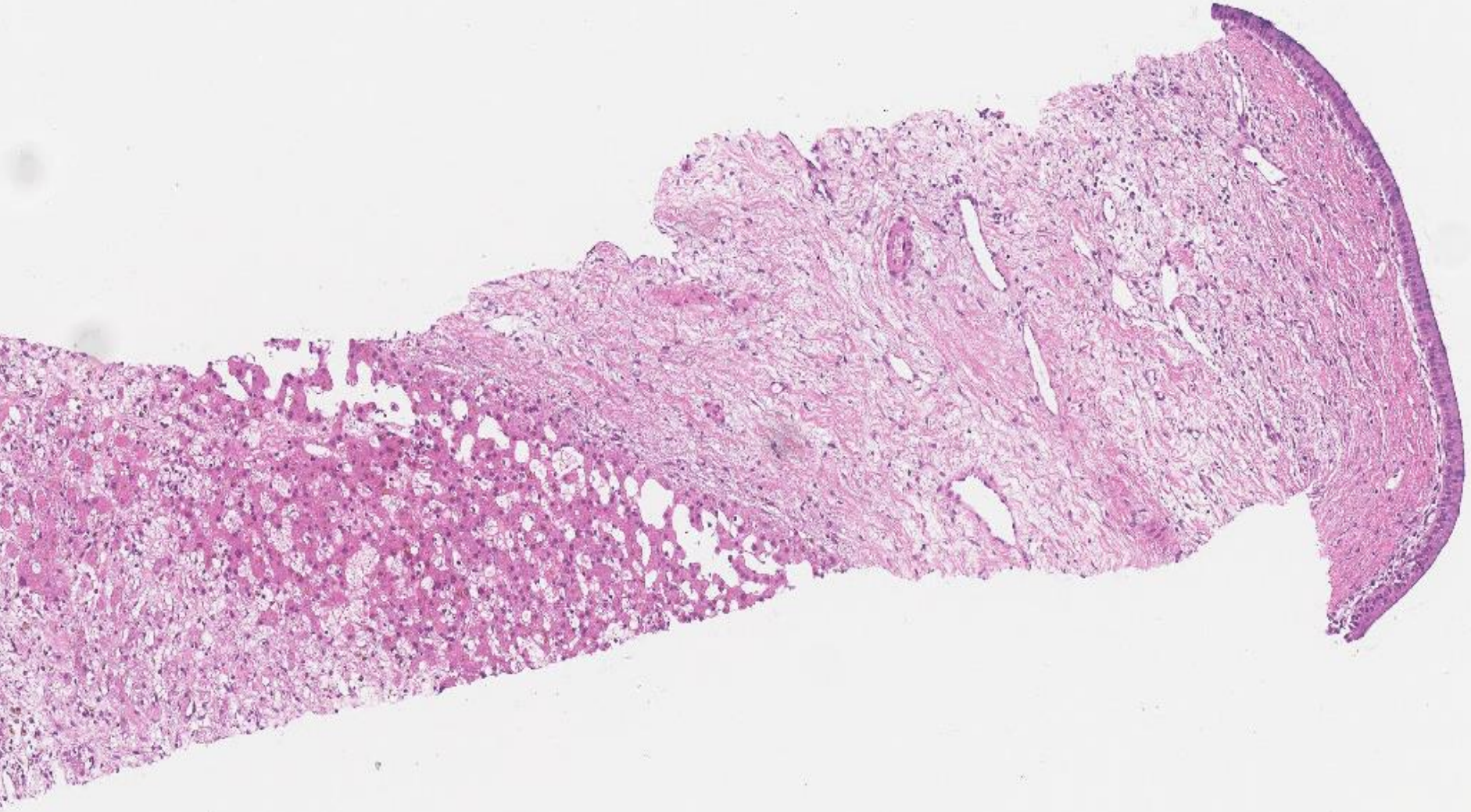

LN 7

LN 7



LN7 Male 63 years

Right liver abscess drained. Residual solid tissue in wall ?? abscess wall.

Rule out tumour. No previous history of malignancy.

Abscess wall	56
Favour abscess but exclude neoplasia (EHE, HCC, angiosarc, RCC)	7
Also mentioned von Meyenberg complex	30
Underlying cause: biliary cyst (epithelial lining seen)	11
?parasitic	3
Others – not neoplastic: n=11	11
Granulation tissue, edge of ulcer	1
Granulation tissue, no neoplasia	1
? Infective, no malignancy	1
Description only – needs more stains	1
Reactive 'inflammatory pseudotumour like'	1
Solitary bile duct cyst (no mention of inflammation)	2
Infarct, no mention of inflammation	1
Pressure effect of tumour, with SOS and fibrosis	1
Organising blood clot, no significant inflammation	1
Non-cirrhotic necrosis, exclude PVT/BCS	1
Neoplastic n=11:	
Favour neoplastic – vascular/EHE/PEComa do IHC	3
Hepatobiliary mucinous cystadenoma/MCN	2
Angiomyolipoma	3
Likely angiosarcoma	1
Adenocarcinoma, favour intrahepatic cholangiocarcinoma	1
HCC (only text)	1

<u>Suggested scoring</u>:

63 mention the abscess in some way – insufficient consensus.

? suitable for scoring.

6 yes, 5 no

If accept any responses that are not diagnosing/ favouring underlying neoplasia, then sufficient consensus to score.

<u>If so:</u>

suggest half marks for suggested low grade neoplasia and

no marks for likely angiosarcoma/ adenoocarcinoma/ HCC.

LN7 Male 63 years

Right liver abscess drained. Residual solid tissue in wall ?? abscess wall.

Rule out tumour. No previous history of malignancy.

Abscess wall	56
Favour abscess but exclude neoplasia (EHE, HCC, angiosarc, RCC)	7
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Underlying cause: biliary cyst (epithelial lining seen)	11
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Others – not neoplastic: n=11	
Granulation tissue, edge of ulcer	
Granulation tissue, no neoplasia	
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Reactive 'inflammatory pseudotumour like'	
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Pressure effect of tumour, with SOS and fibro	
Organising blood clot, no significant inflamm	
Non-cirrhotic necrosis, exclude PVT/BCS	
Neoplastic n=11:	
Favour neoplastic – vascular/EHE/PEComa do	
Hepatobiliary mucinous cystadenoma/MCN	
Angiomyolipoma	
Likely angiosarcoma	
Adenocarcinoma, favour intrahepatic cholang	
HCC (only text)	

LN7 after discussion at meeting : not suitable for scoring.

Discussion – insufficient consensus on abscess.

In addition to neoplastic diagnosis, others e.g. edge of ulcer, 'inflammatory pseudotumour like', edge of tumour etc. are also incorrect or misleading.

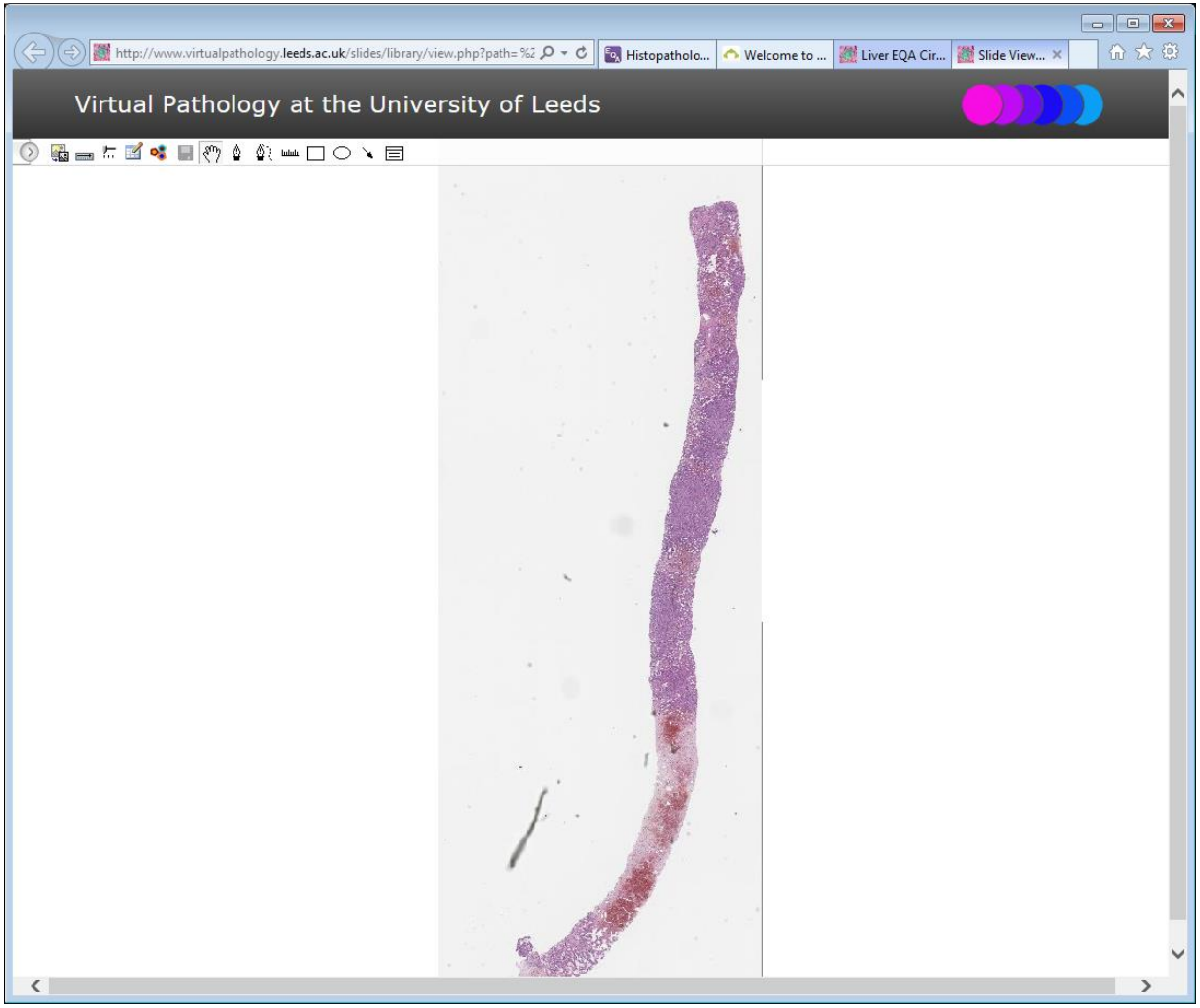
Dangerous to try to contrive consensus when there isn't a clear consensus, and in real life for a case like this a malignant diagnosis wouldn't be firmly made on the basis of this slide in isolation.

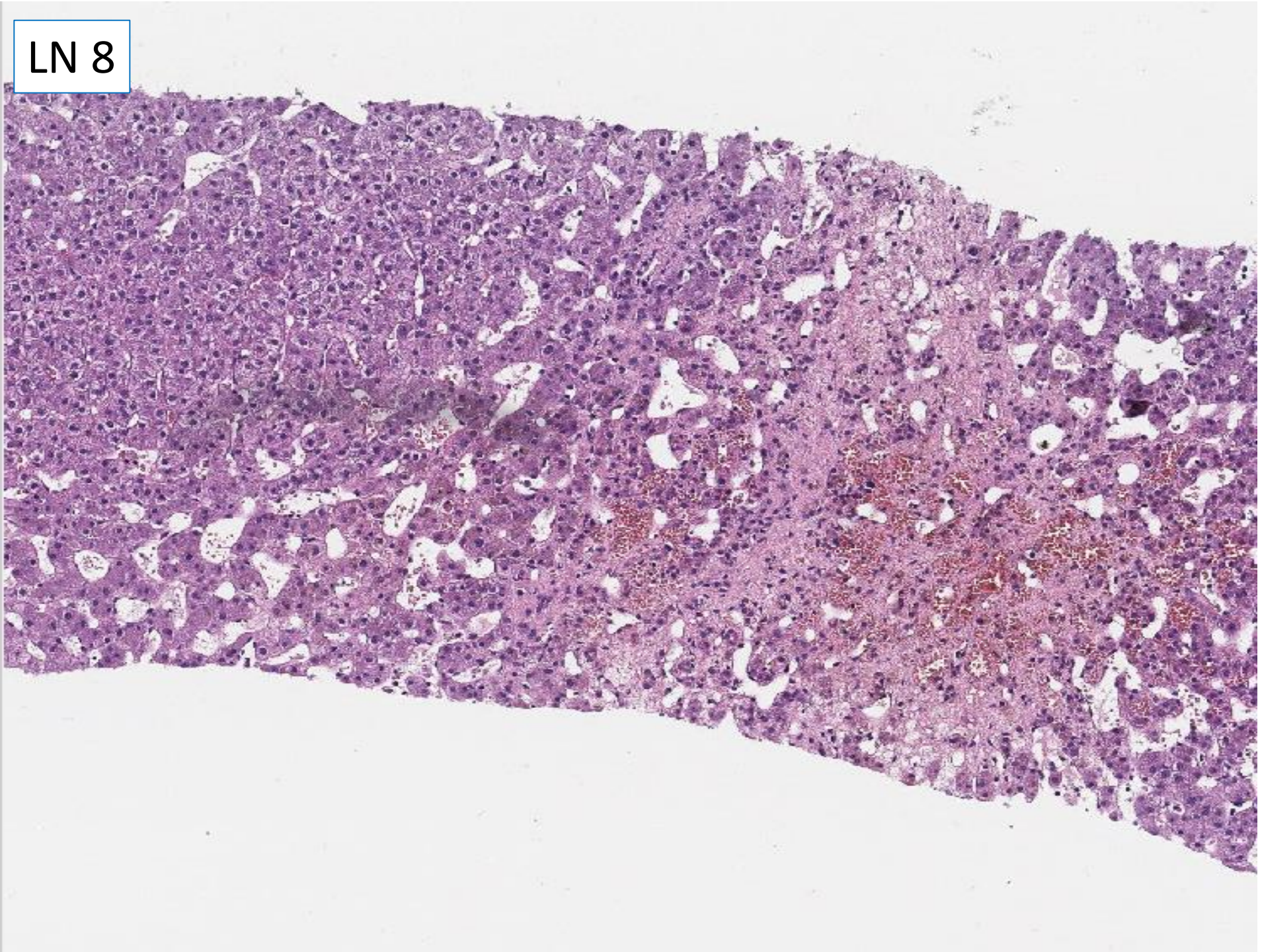
This is interesting and educational but not suitable for EQA scoring.

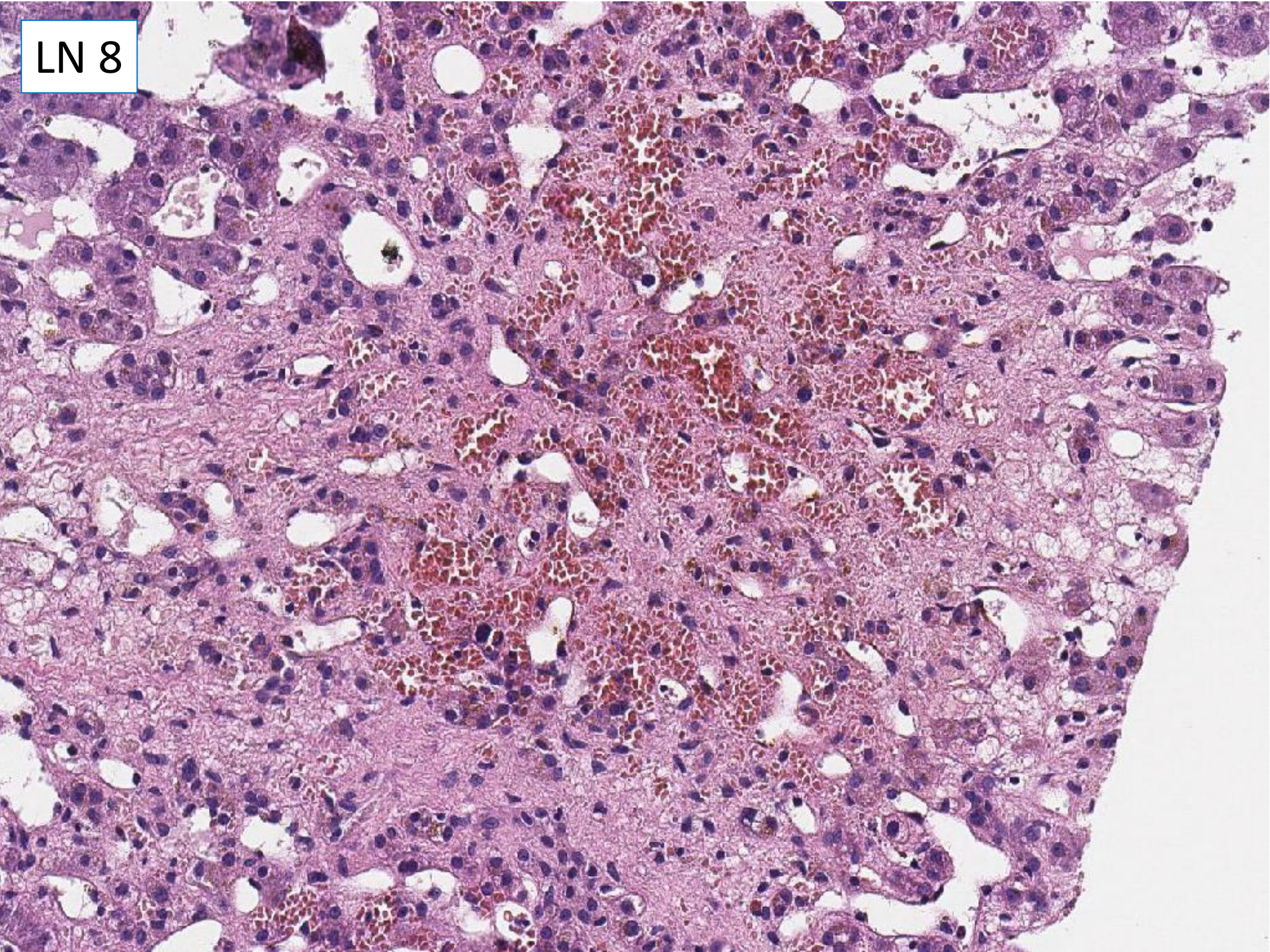
Follow up on this case – after 18 months no evidence of further problem.

LN8 Female 31 years

Background childhood AML. Initially presented under cardiology with heart failure and ascites. Found to have multiple liver masses. Biosy to confirm diagnosis.




LN 8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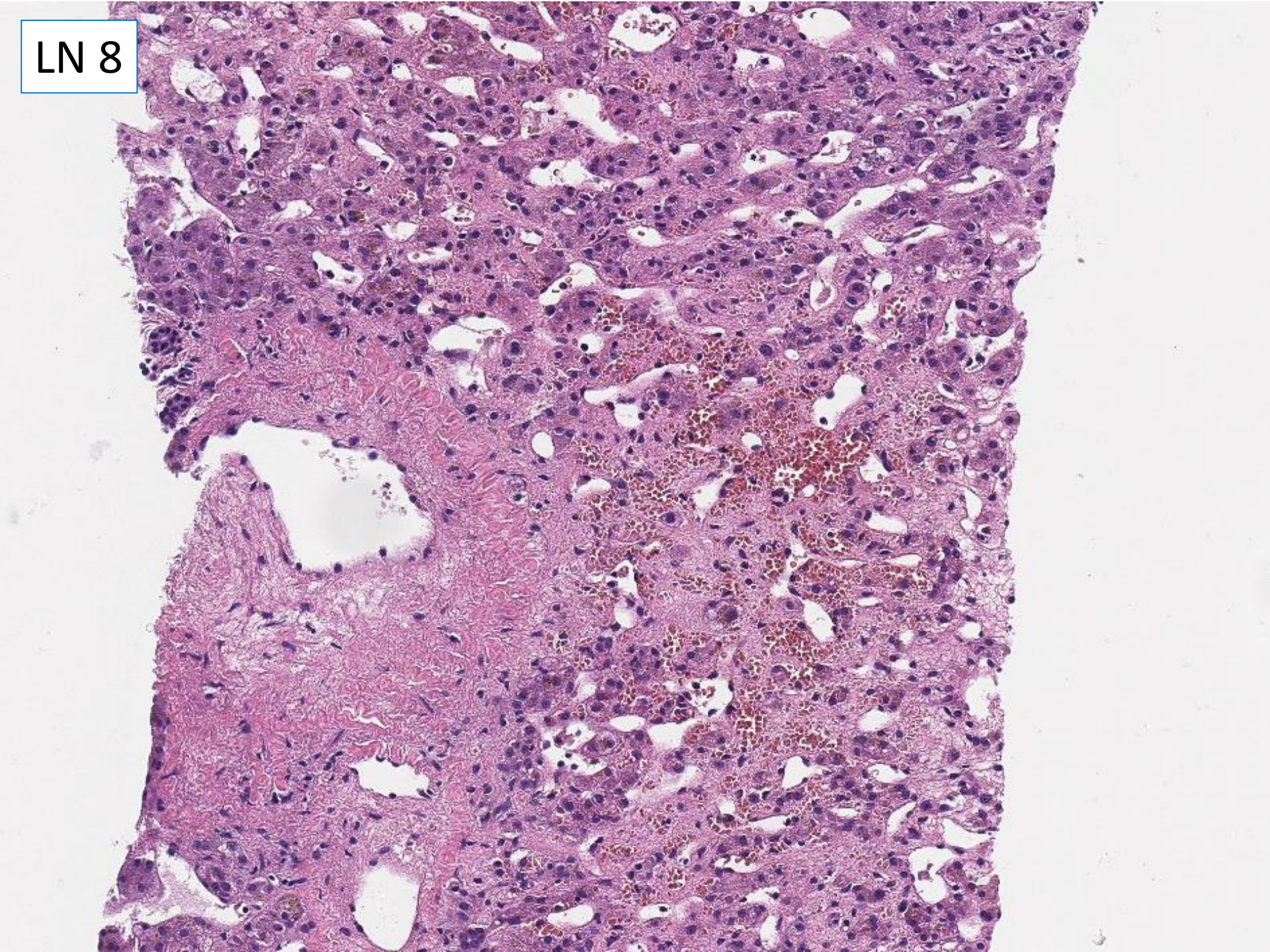
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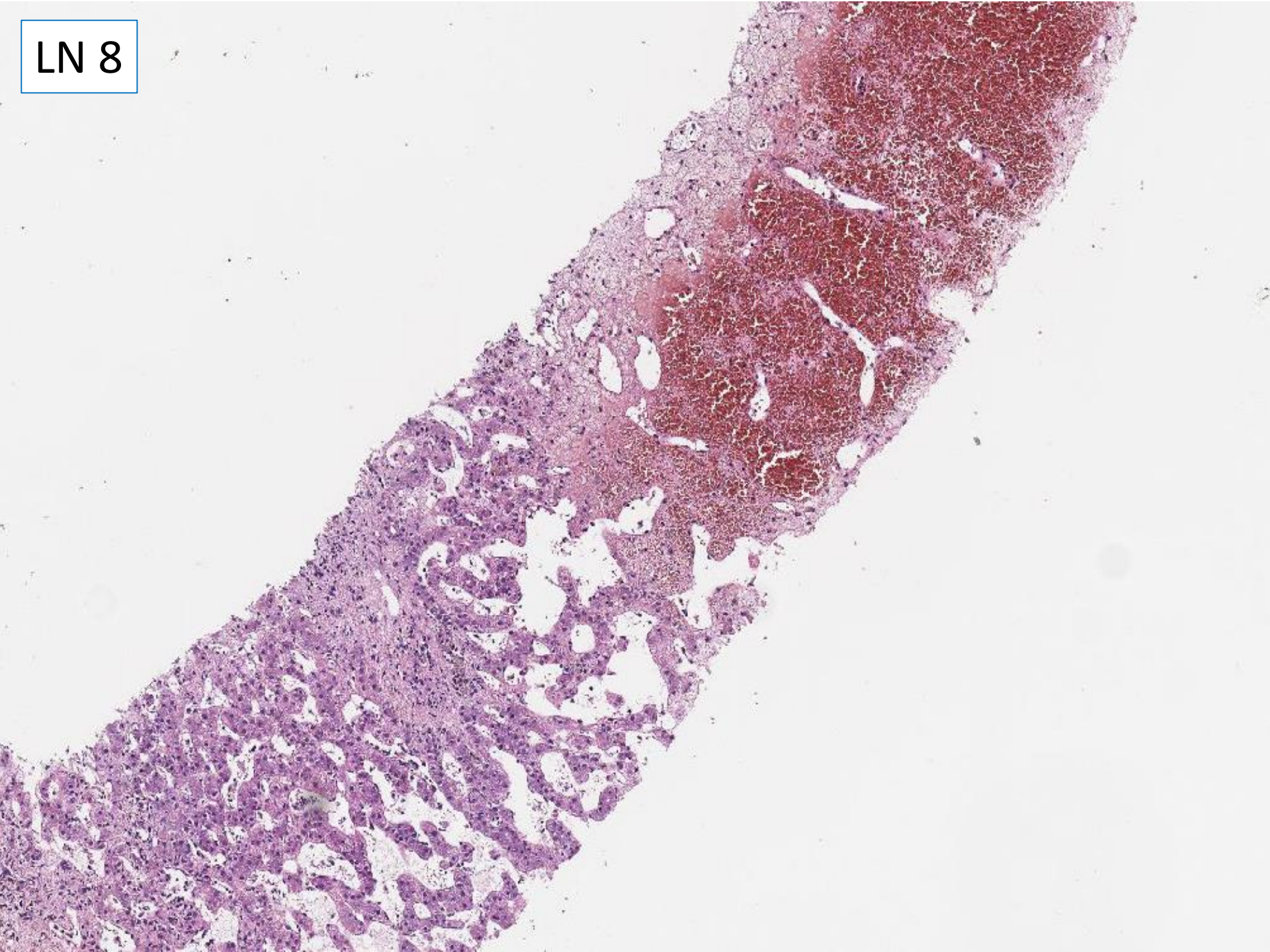
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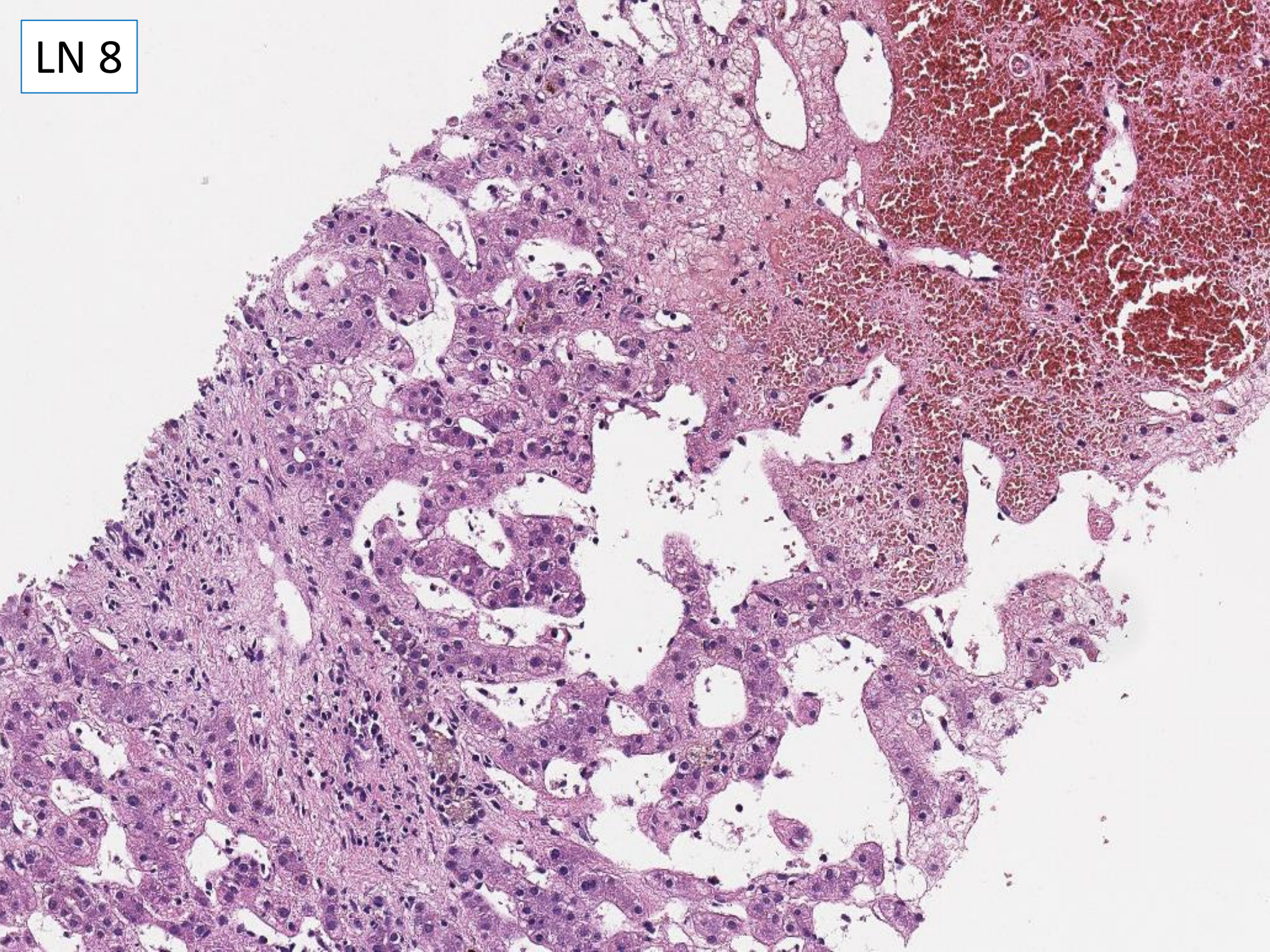
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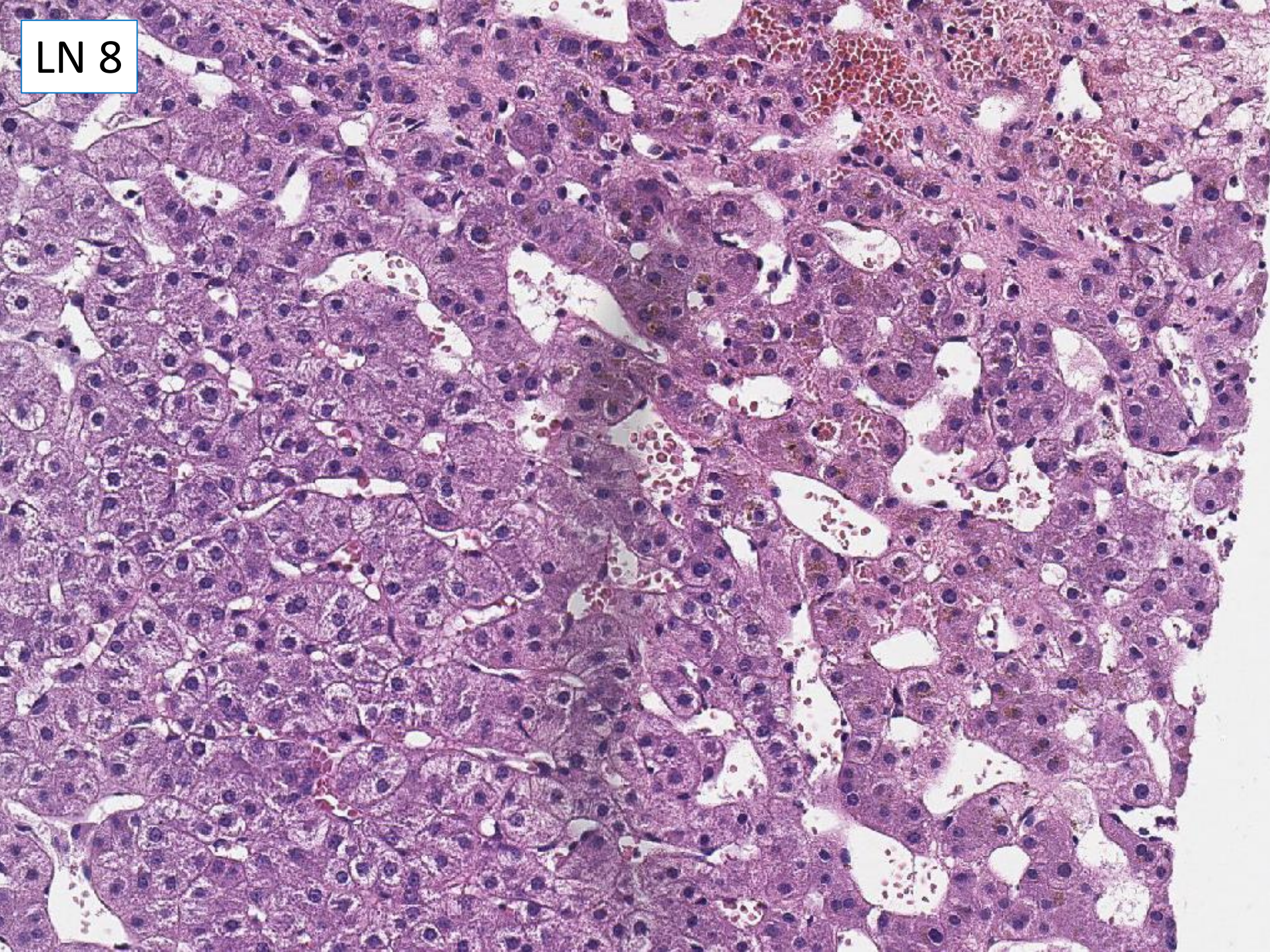
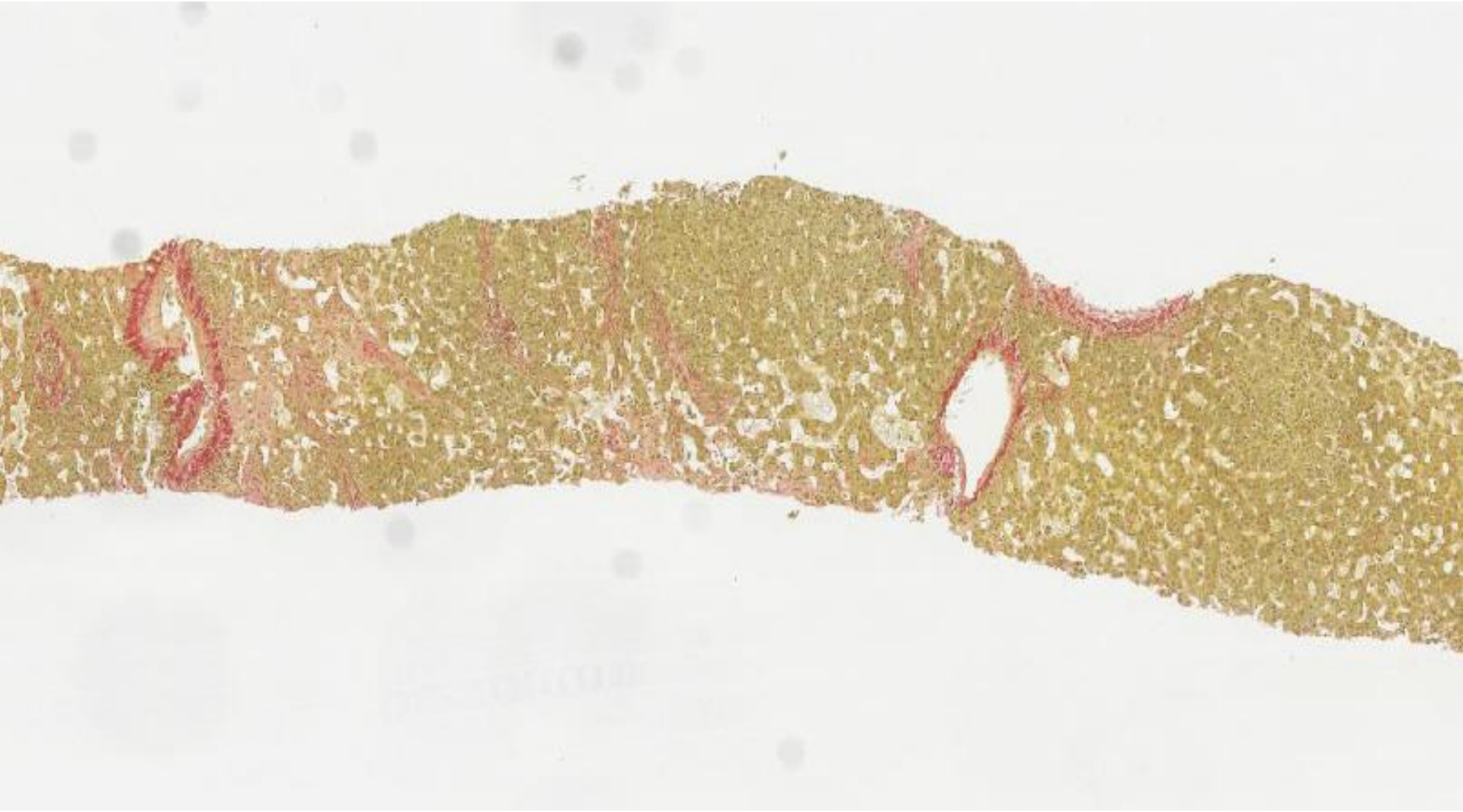
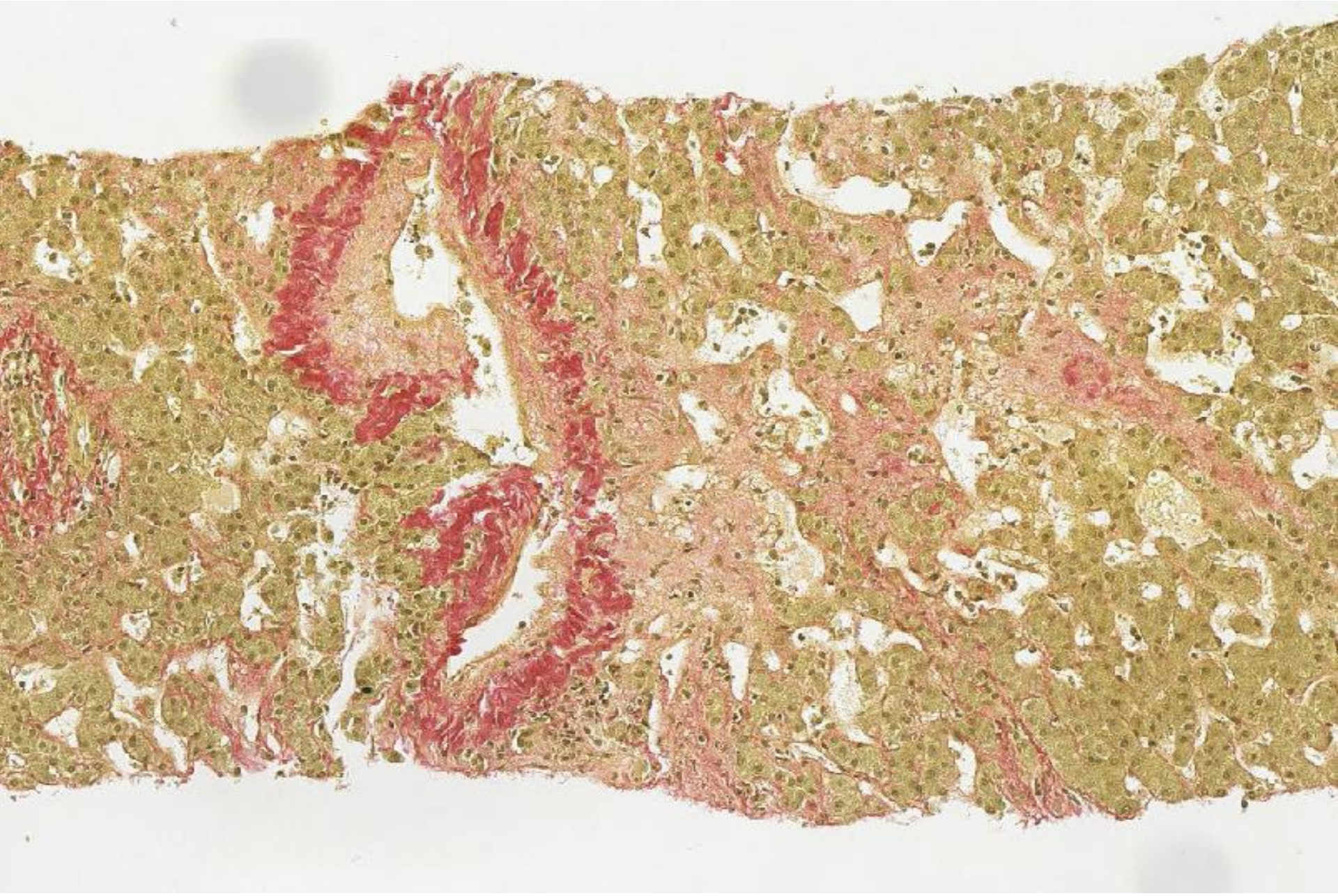

LN 8

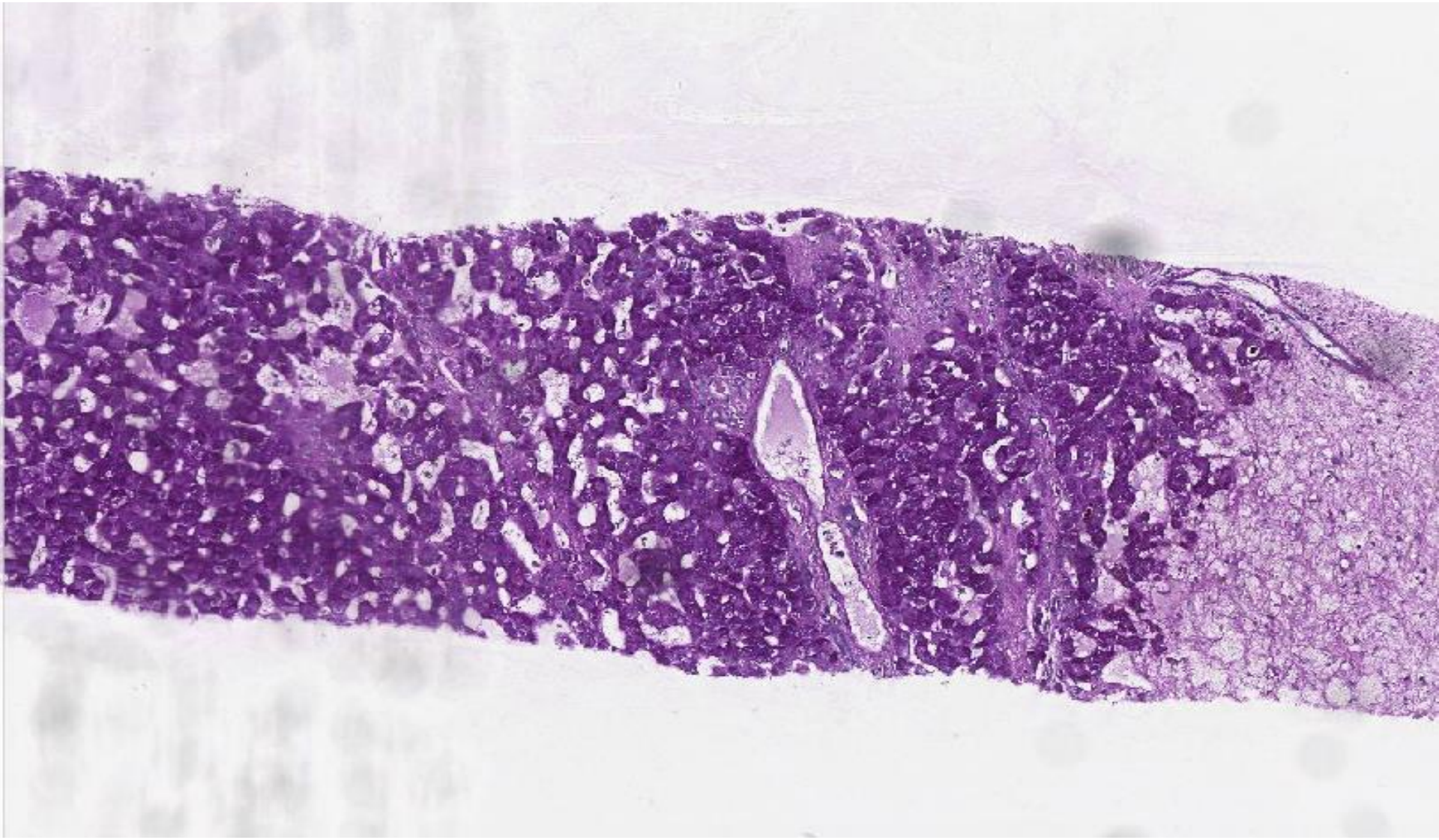
LN8 VG



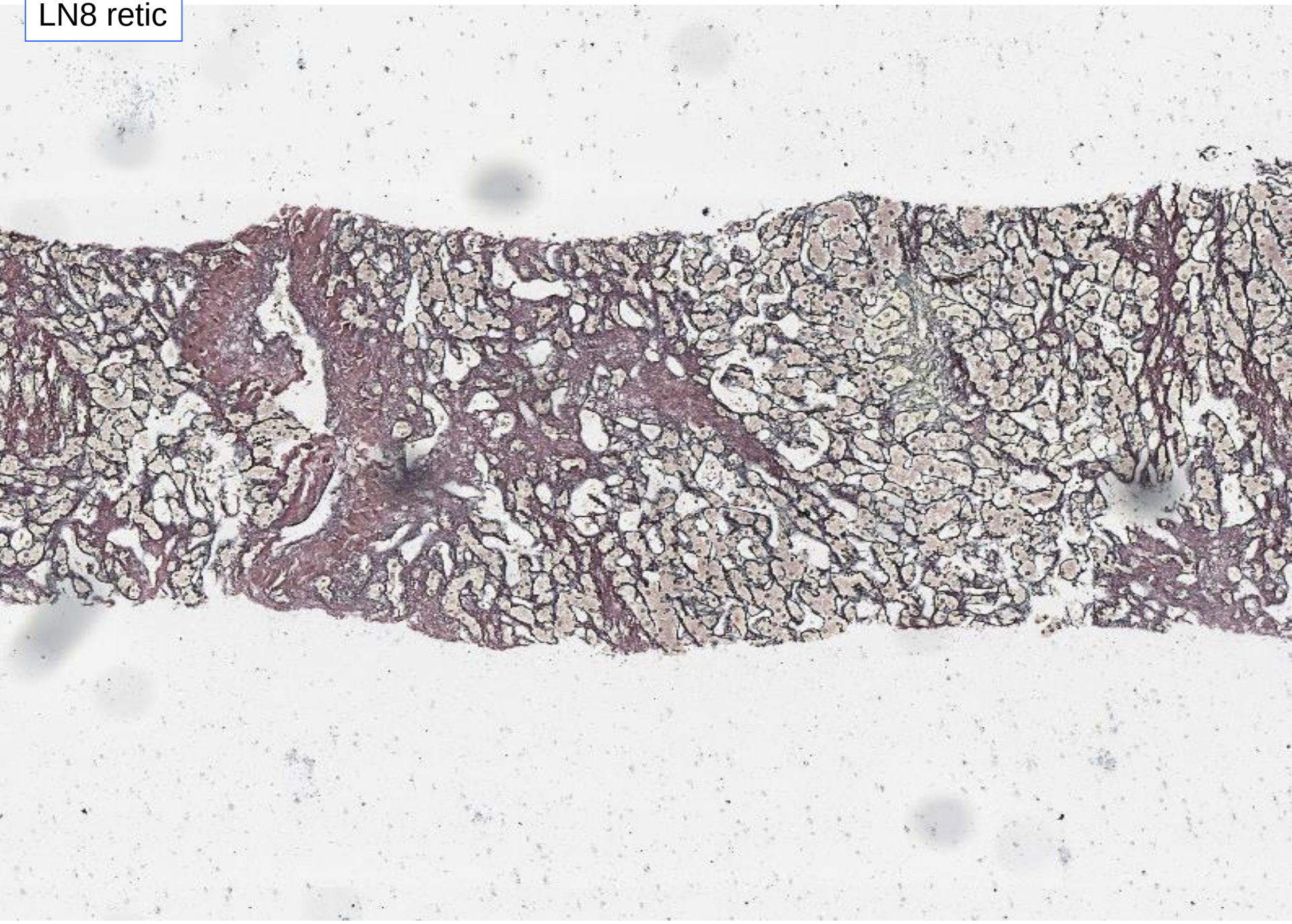
LN8 VG



LN8 PAS



LN8 retic



LN8 Female 31 years

Background childhood AML. Initially presented under cardiology with heart failure and ascites. Found to have multiple liver masses. Biopsy to confirm diagnosis.

Venous outflow obstruction - any	82
Peliosis hepatis as only diagnosis	2
Vascular dilatation and tumour - haemangioma	1
Hepatocellular carcinoma	1
Of venous outflow obstruction (82)	82
Cardiac failure	34
Related to drugs for AML, includes SOS/VOD	20
Cardiomyopathy related to chemotherapy	2
Budd Chiari syndrome	17
Veno-occlusive disease/VOD	11
Cardiac cirrhosis	1
Cardiac sclerosis	1

No marks

<u>LN8 final scoring after meeting discussion</u>: for full marks – need a response that includes venous outflow obstruction.

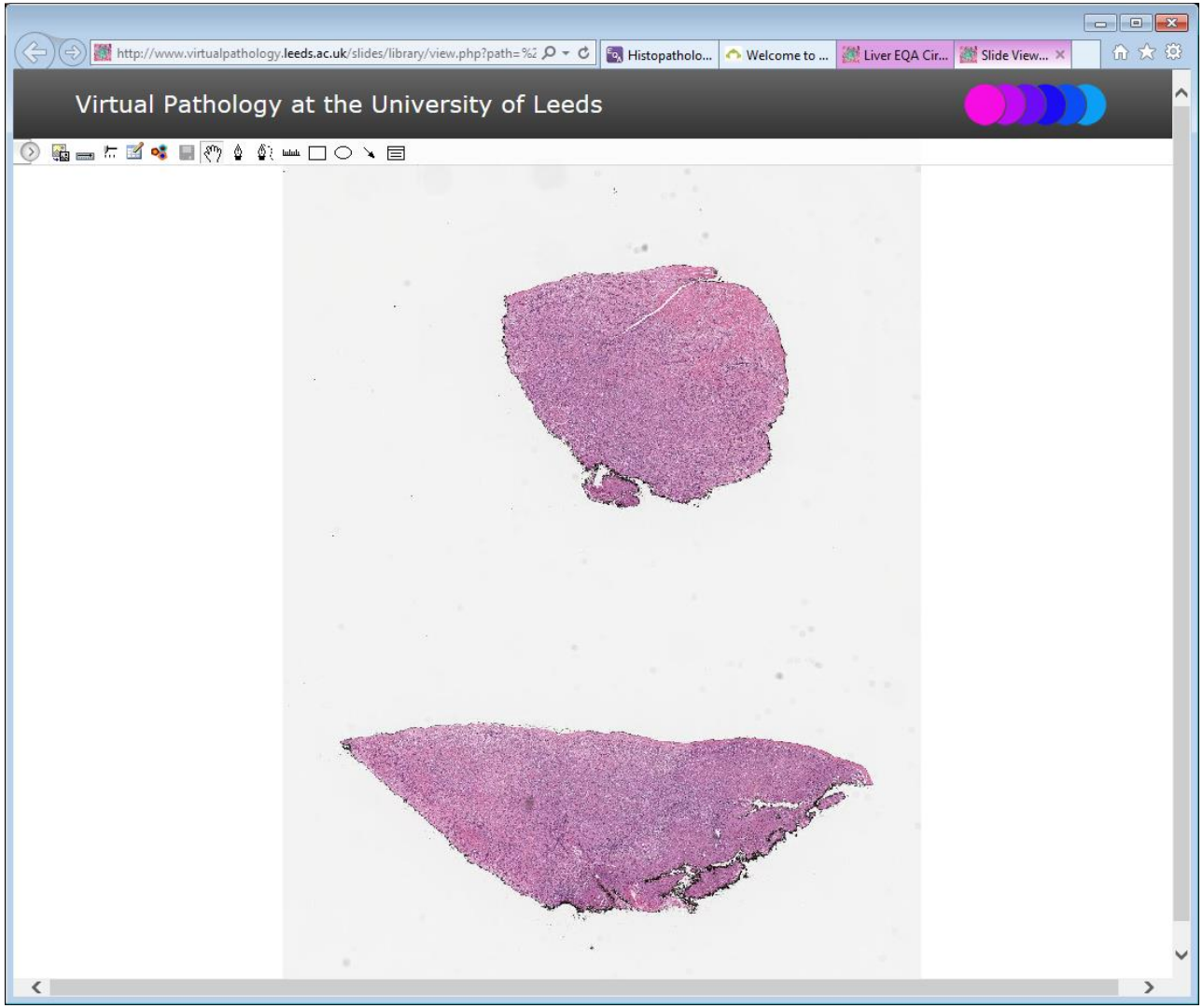
No marks for peliosis hepatis, vascular dilatation and tumour ? haemangioma, and for HCC.

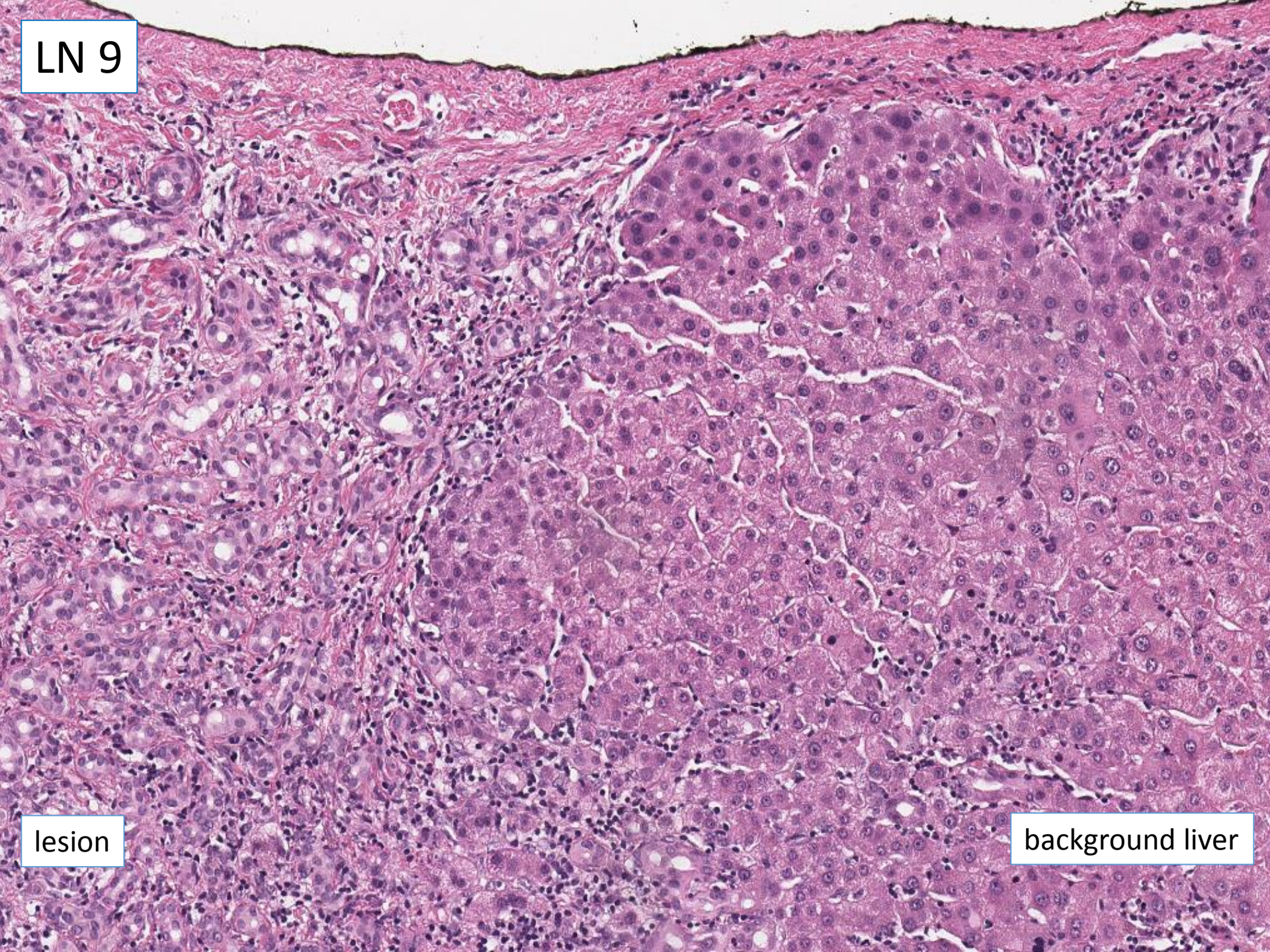
There was no consensus on the likely level of the venous obstruction, or on the likely relationship with the history of childhood AML and subsequent heart failure.

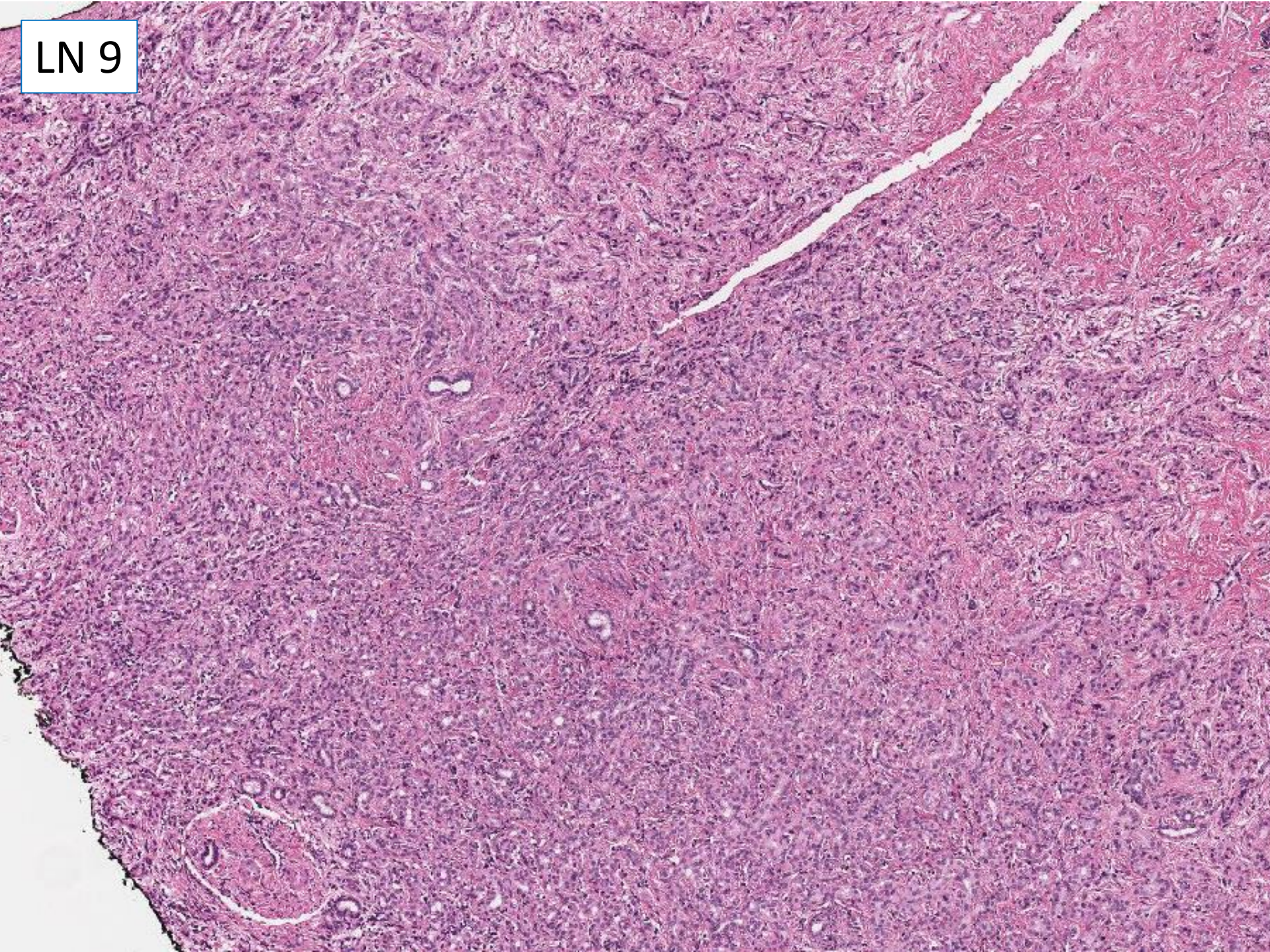
From clinical follow up, this was attributed to cardiomyopathy, a late side effect from the drugs used to treat AML at the age of 2 years.

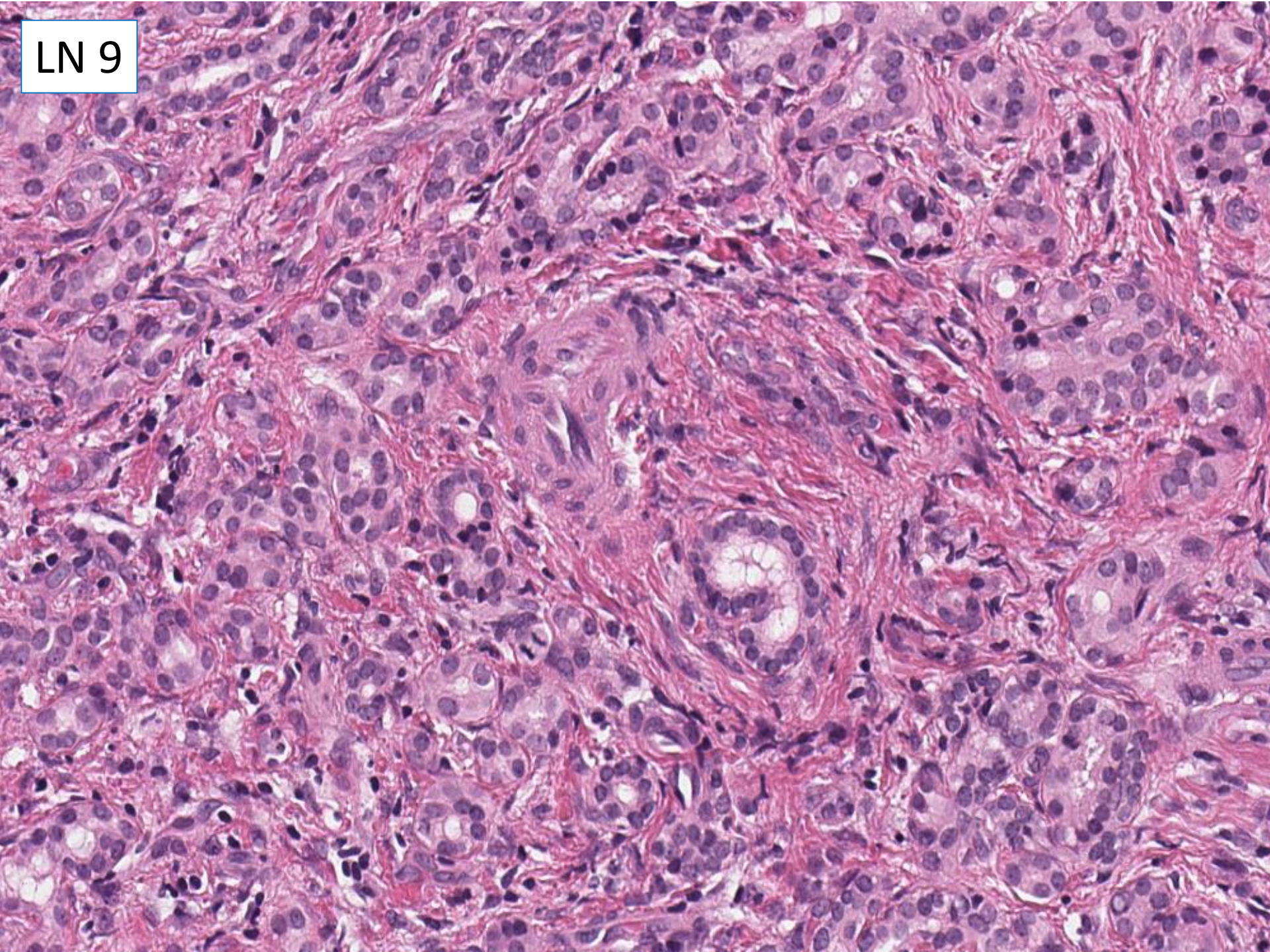
LN 9 Male 57 years

Elective laparoscopic cholecystectomy, sub-capsular liver lesion noted.




LN 9
lesion
background liver


LN 9


LN 9

LN 9 Male 57 years

Elective laparoscopic cholecystectomy, sub-capsular liver lesion noted.

Bile duct adenoma	50
Bile duct adenoma/peribiliary gland hamartoma – both names	29
Peribiliary gland hamartoma	5
Ductal plate malformation	1
Bile duct adenoma/von Meyenberg complex, biliary microhamartoma	1
Do immunohistochemistry	3
Comment: 'it's an adenoma again'	

Divided 5:6

Half marks

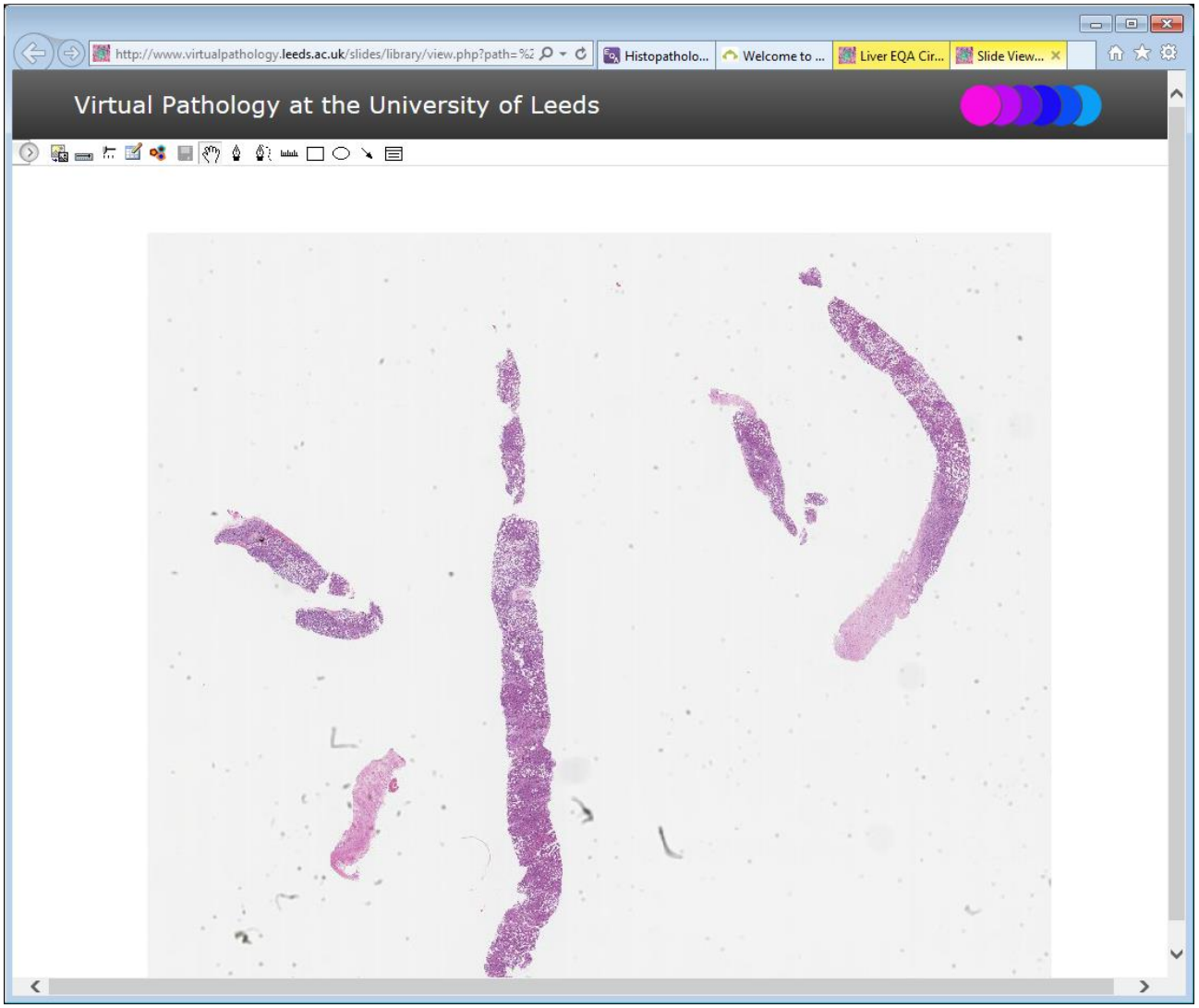
Suggested scoring: Consensus in the tumour, although not in what to call it.

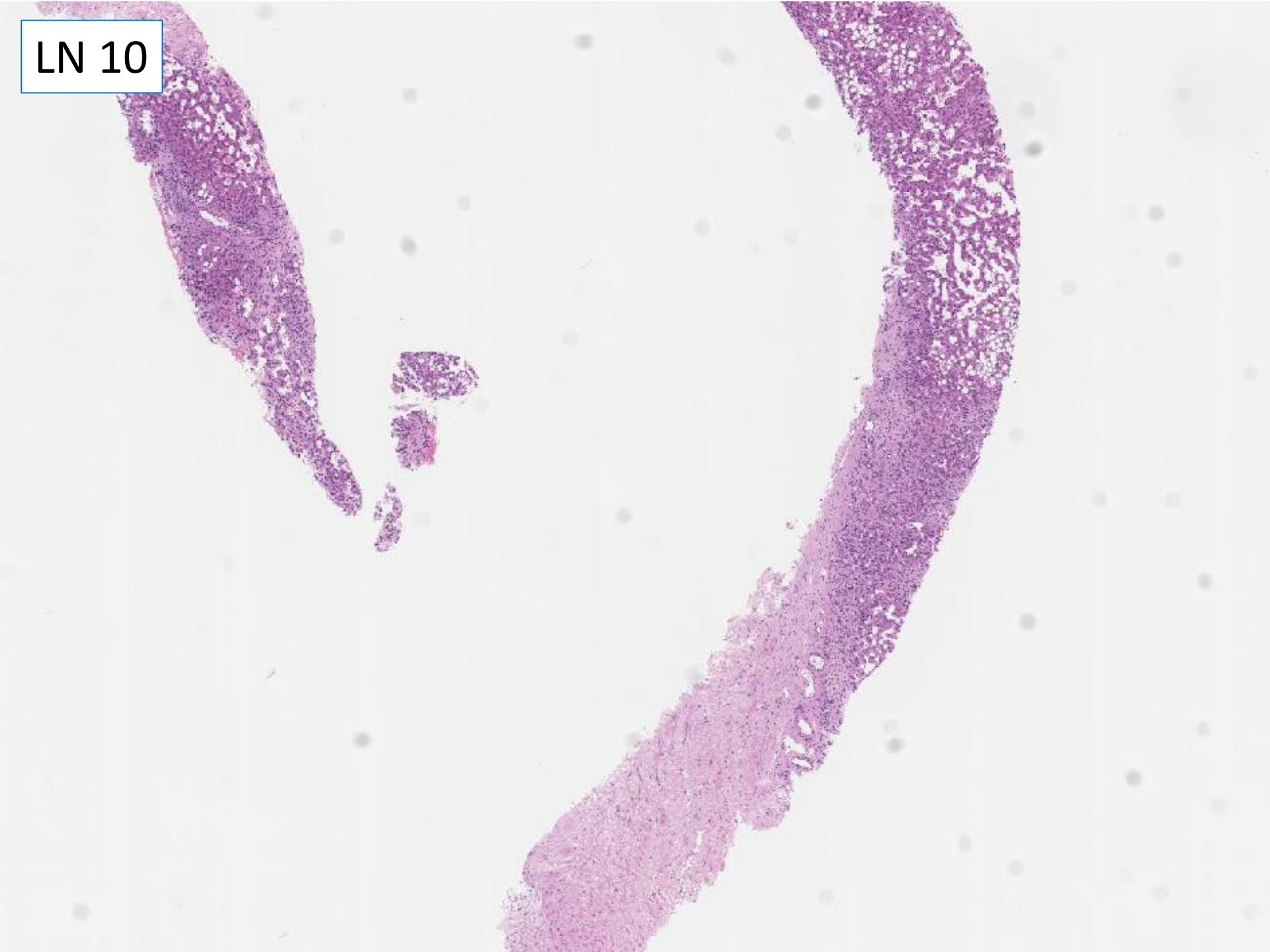
LN9 final scoring after meeting discussion: for full marks – bile duct adenoma or peribiliary gland hamartoma.
No marks for ductal plate malformation. Marks not deducted for von Meyenberg complex because the main diagnosis in this response was BDA.

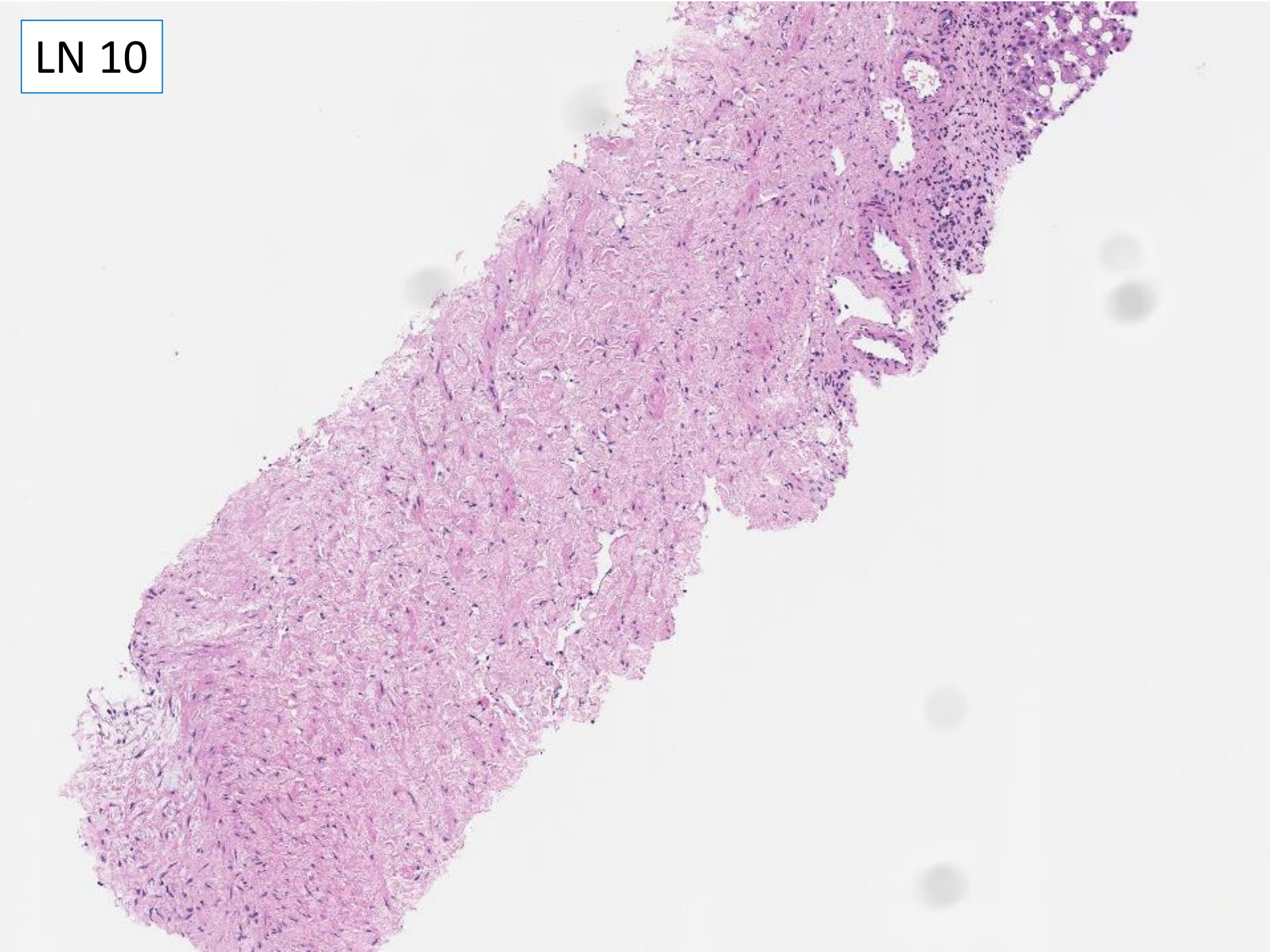
Comment: these lesions have now been shown to be clonal, with mutation, therefore genuinely neoplastic, and so bile duct adenoma is the appropriate terminology, although peribiliary gland hamartoma is in common use as a synonym.

LN10 Male 32 years

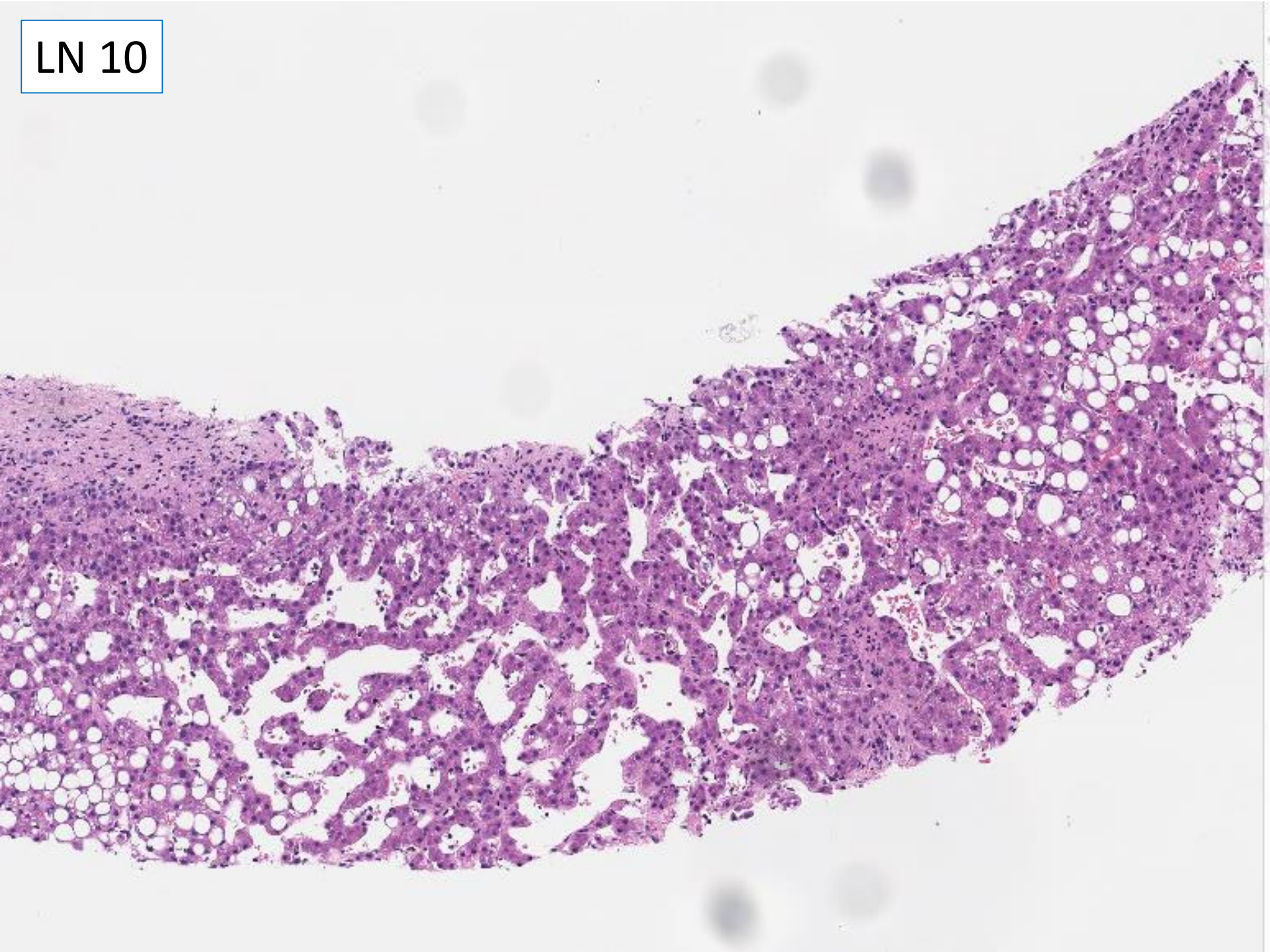
Renal transplant 6 months ago. Presented with ascites ?cirrhosis. ?Amyloid please do staining for amyloidosis.

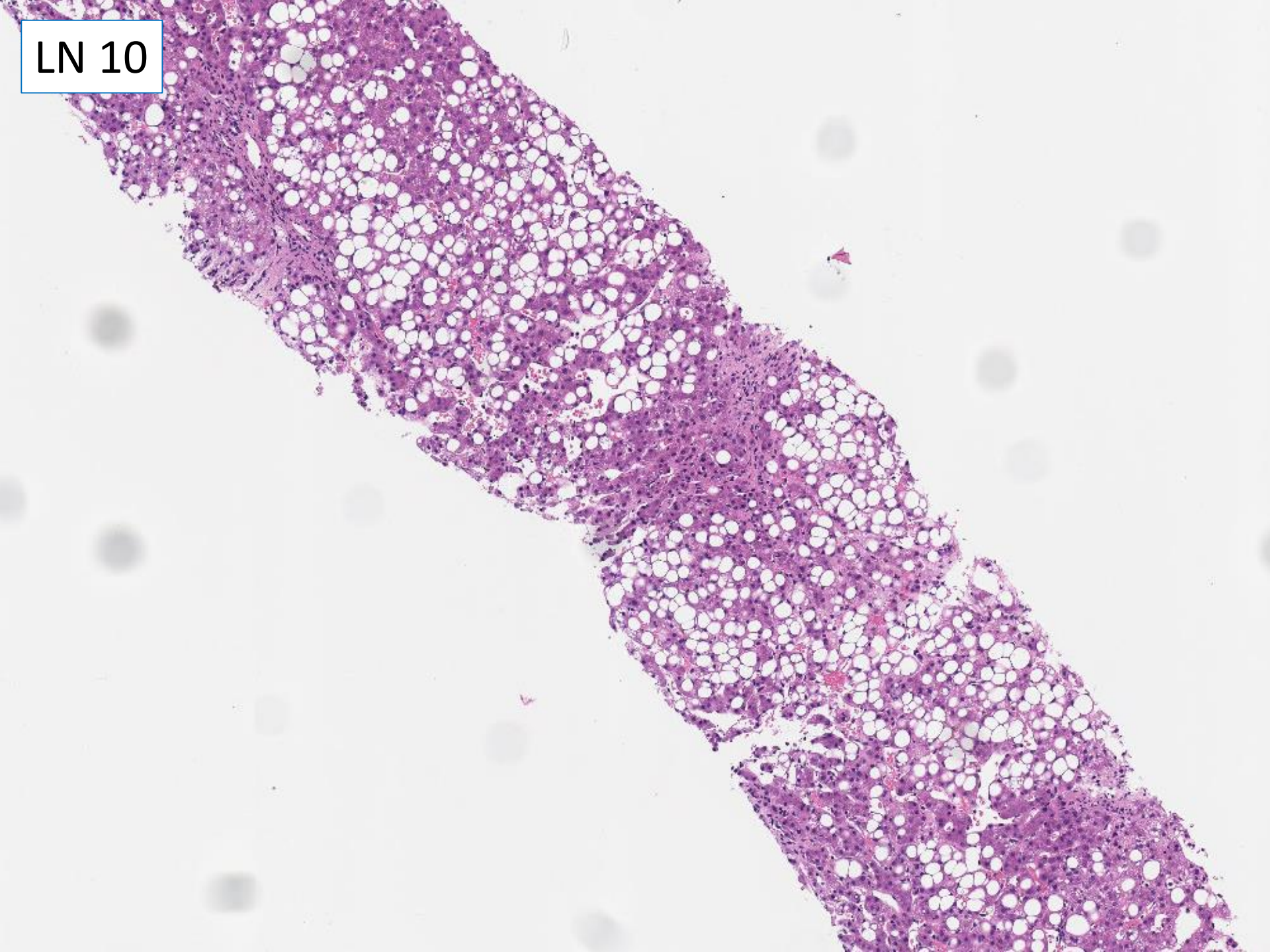



LN 10

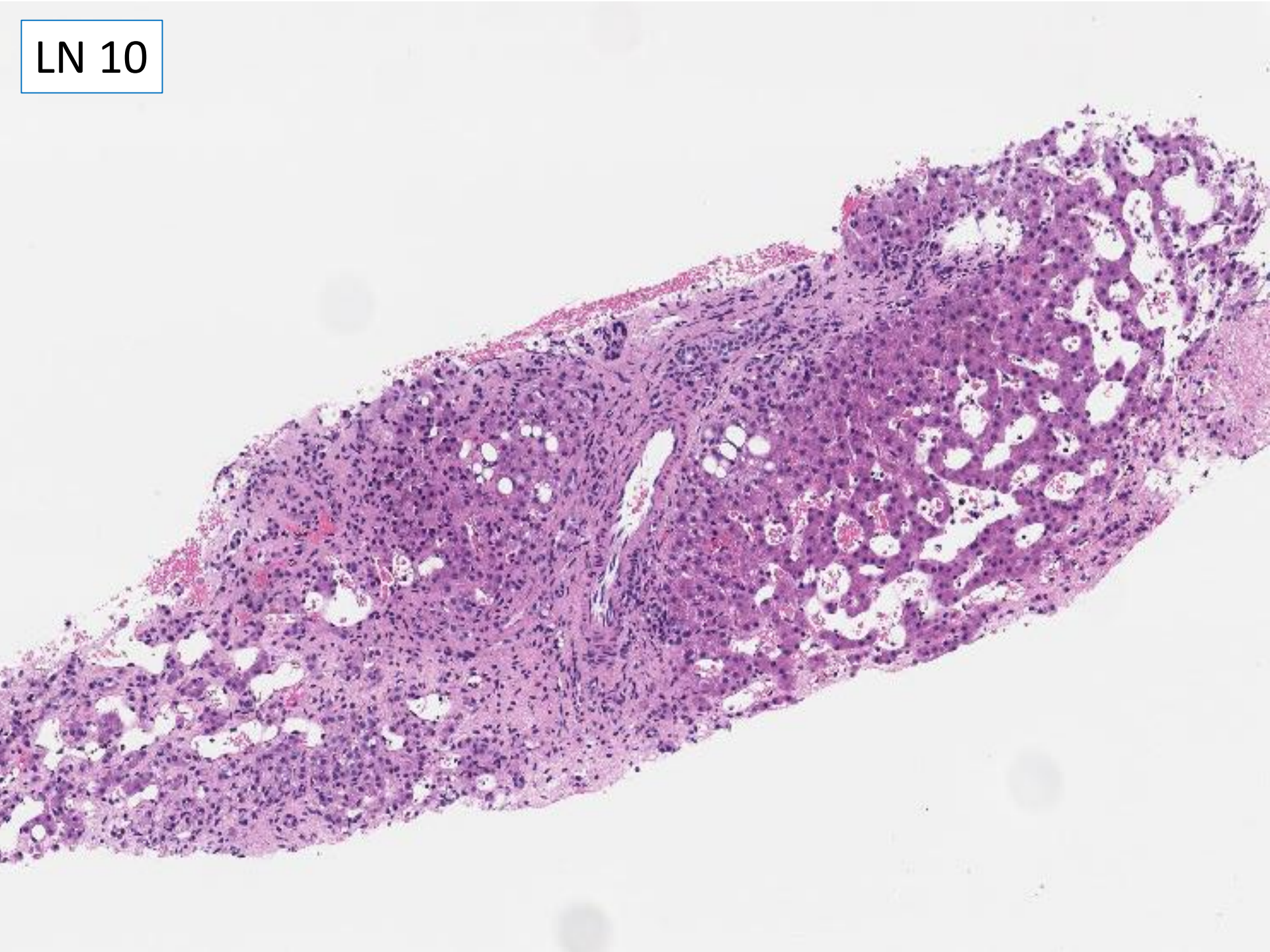

LN 10

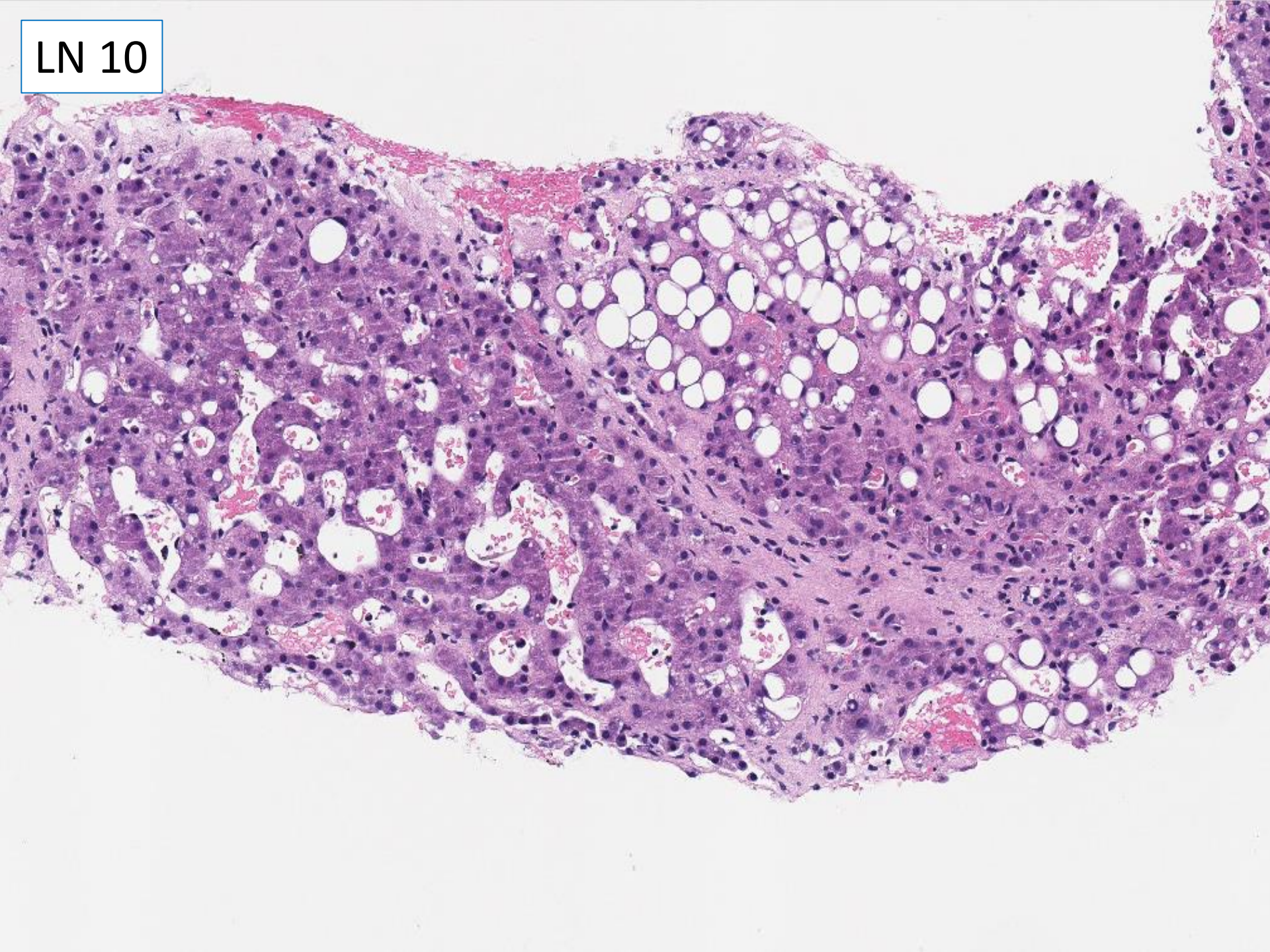
LN 10




LN 10

LN 10

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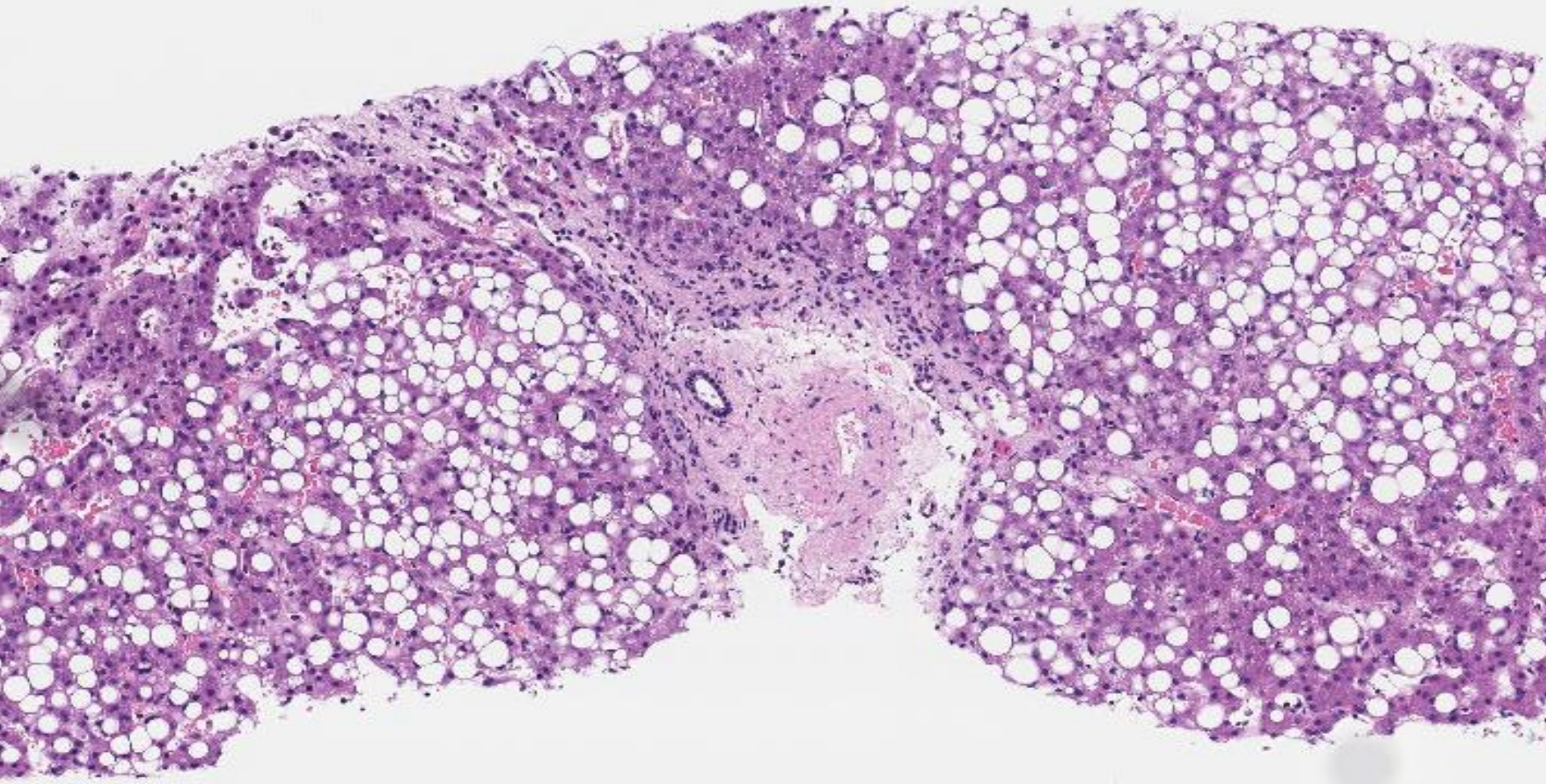

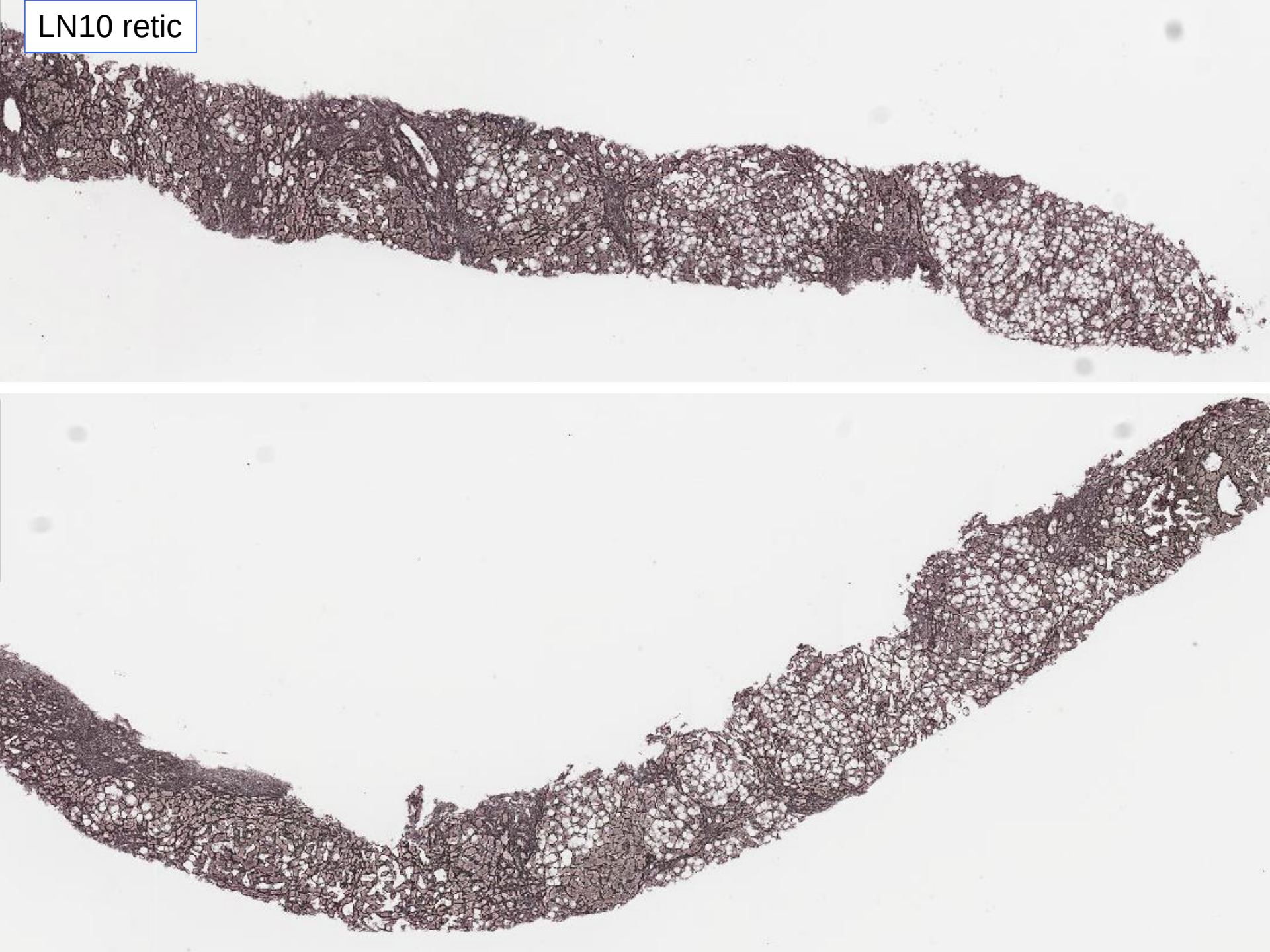
LN10 retic

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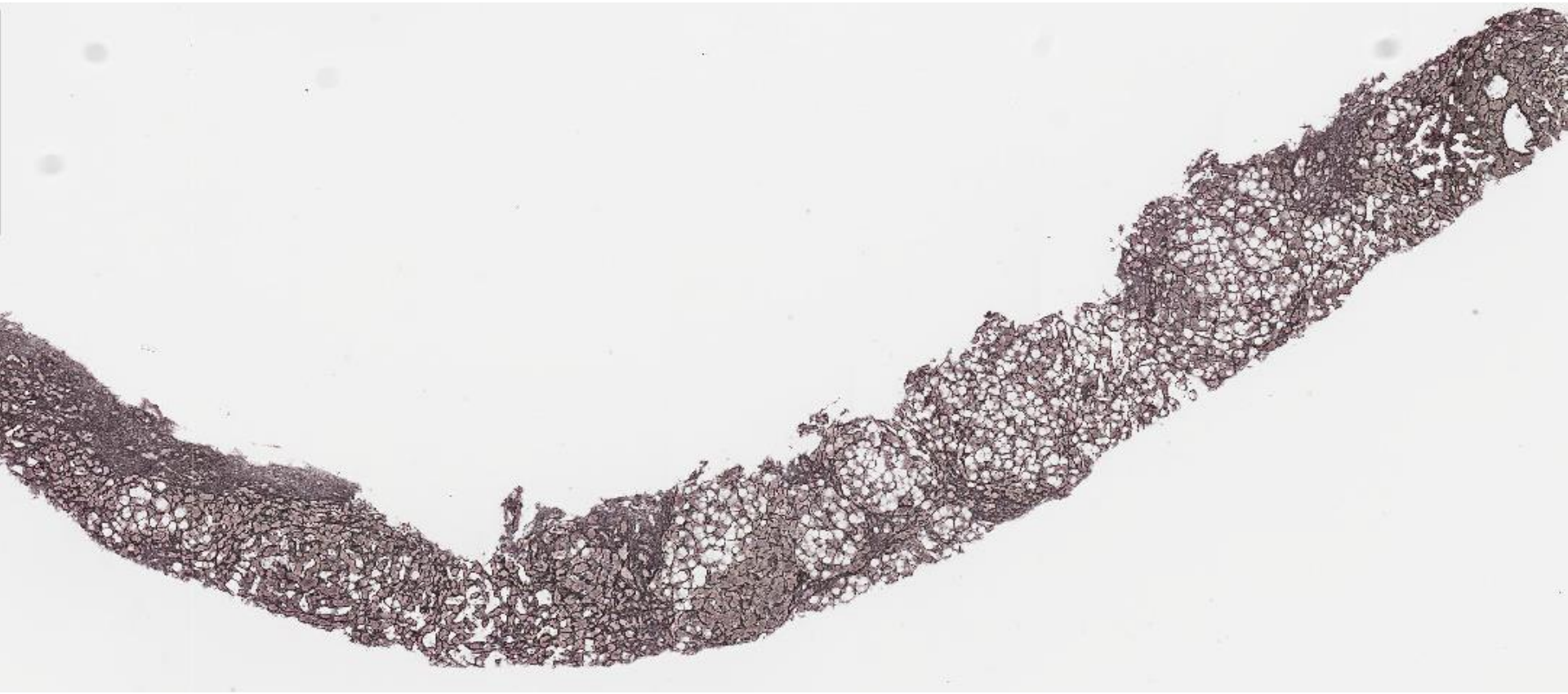
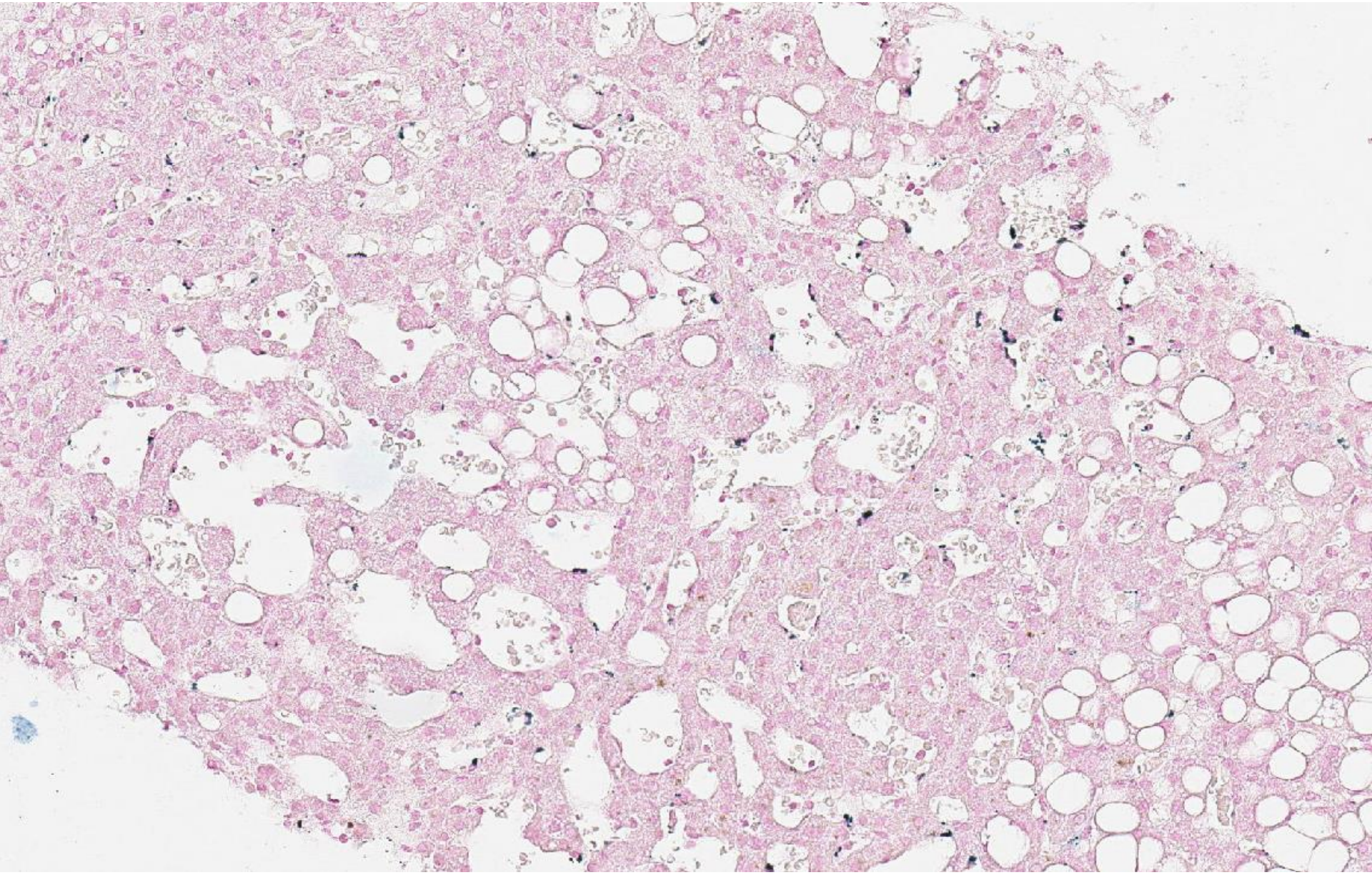
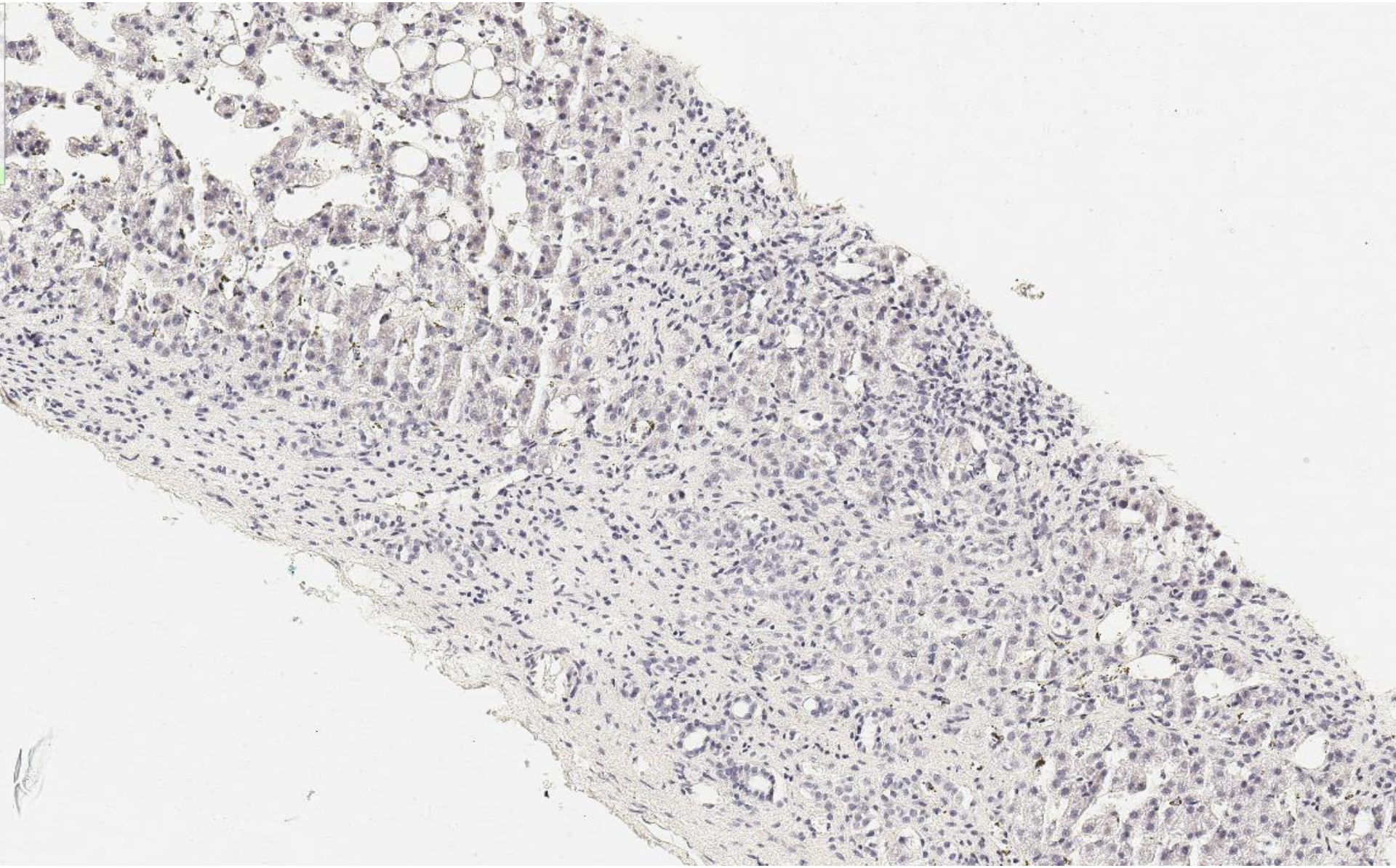
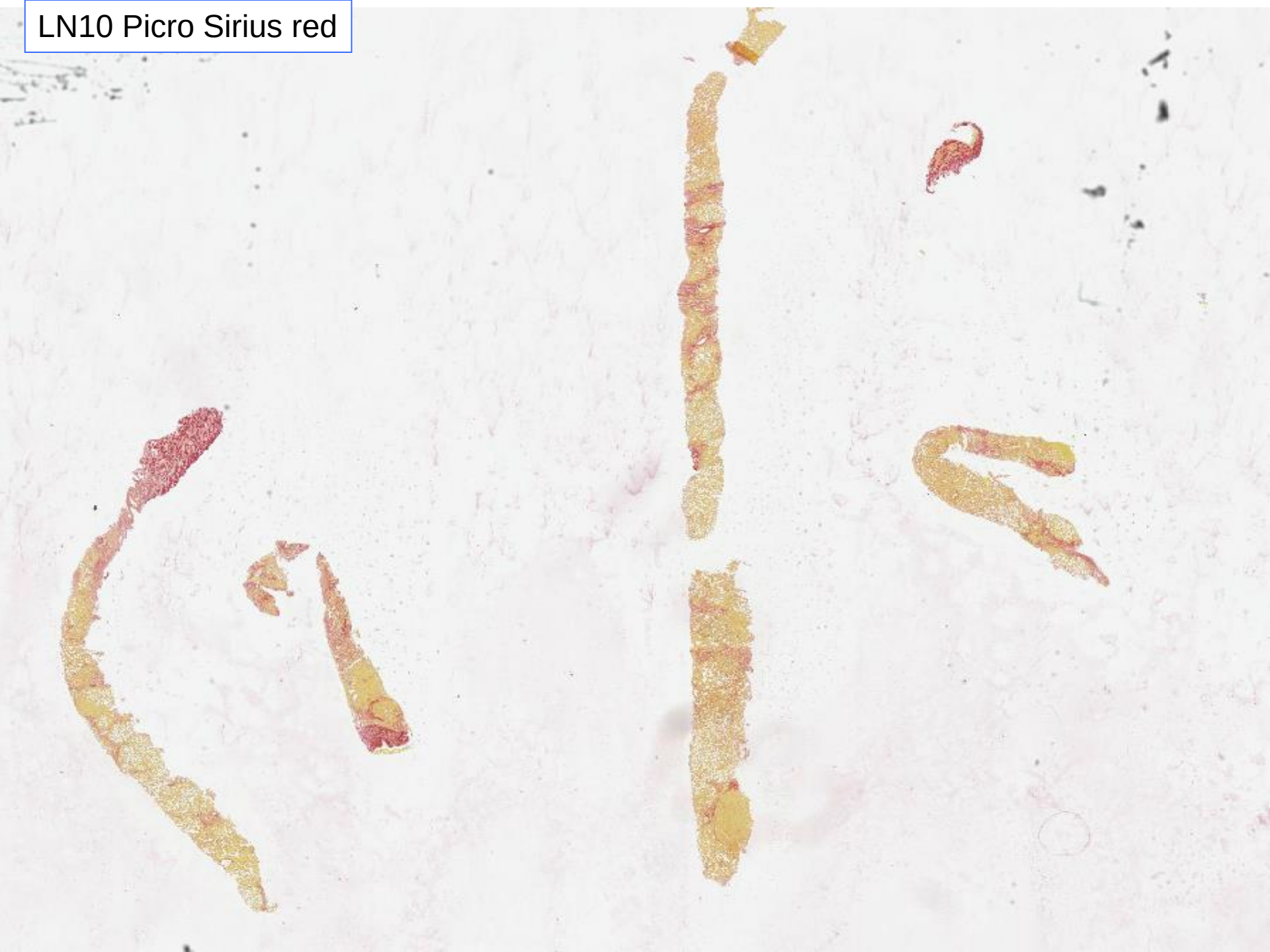


LN10 perls

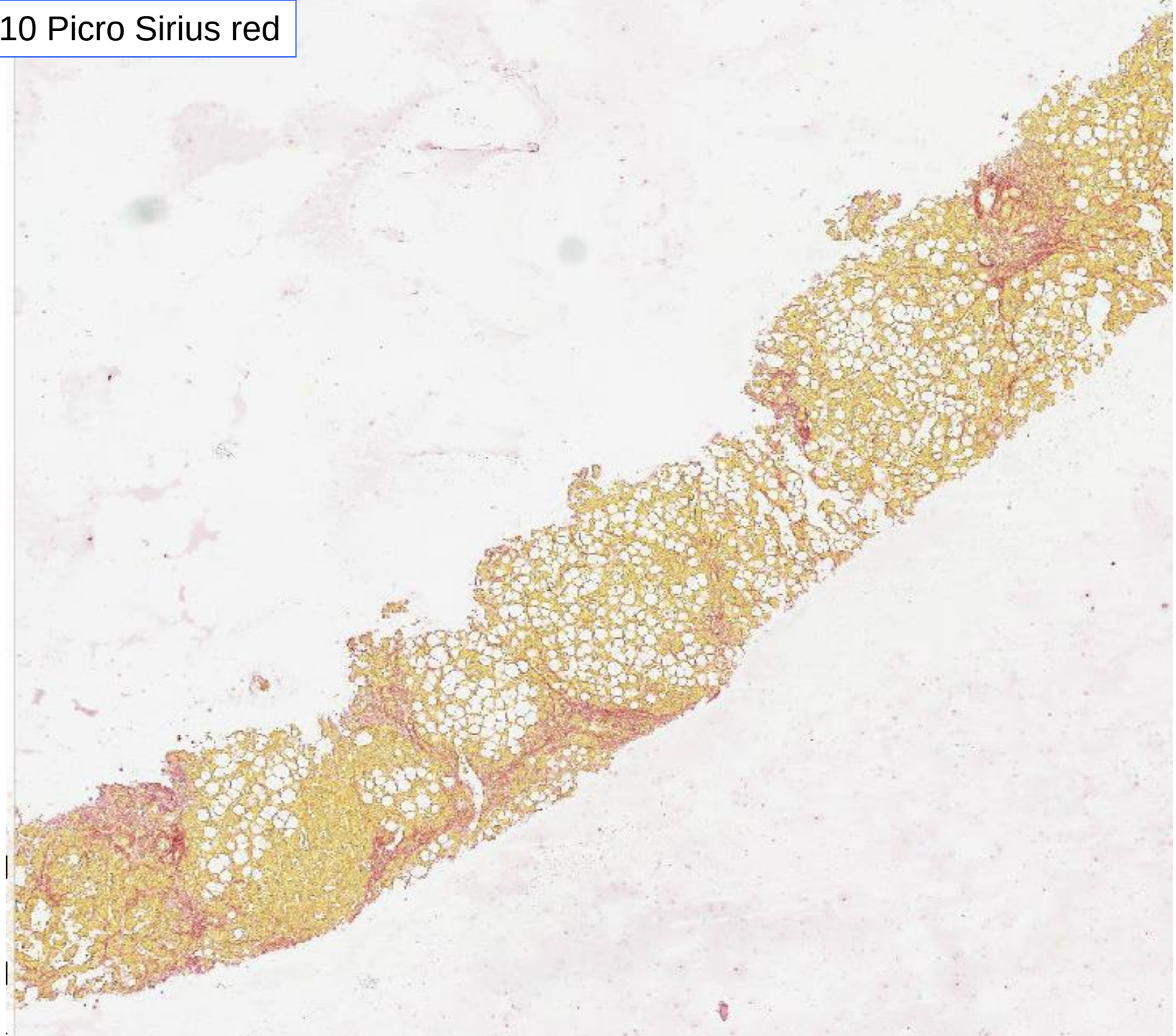


LN10 congo red




LN10 Picro Sirius red

LN10 Picro Sirius red



LN10 Male 32 years

Renal transplant 6 months ago. Presented with ascites ?cirrhosis. ?Amyloid please do staining for amyloidosis.

Vascular problem – Venous outflow obstruction or BCS	34
No mention of vascular pathology/outflow obstruction	31
Sinusoidal dilatation – outflow obstruction not mentioned	18
Peliosis hepatis	2
Fatty liver – steatosis	59
Steatohepatitis	19
Neither fatty liver nor vascular problem mentioned	6
Amyloid as main diagnosis (of which 1 – if confirmed by IHC)	3
Specifically stated not amyloid	70
No mention of amyloid	2
Light chain deposition disease – renal and liver	1
'drug induced changes' – various, as cause of fatty liver, vascular pathology, not separately counted.	
Others – single line diagnoses:	
Cirrhosis, probably alcohol	1
Micronodular cirrhosis	1
Hepatitis ? viral ? drug related	1
Viral hepatitis v. GVHD v. NHL	1
Viral hepatitis, chronic drug damage	1
Unsure, sorry	1

LN10 : this case is unsuitable for scoring!

(Only consensus agreement is that this is not amyloid!)

Submitting pathologist diagnosis:

Dual diagnosis of venous outflow obstruction and fatty liver disease.

Philip Kaye – masterclass case LN10

– discussion of approach to diagnosis, photographs already included.

Dual diagnosis of venous outflow obstruction and fatty liver disease.

Post biopsy information:

- Free hepatic vein pressures 21mm
- Wedge pressure 3
- Hepatic veins patent
- Echocardiogram – calcific pericardium with constrictive pericarditis
- Impaired systolic function and dilated IVC
- worsening ascites and oedema

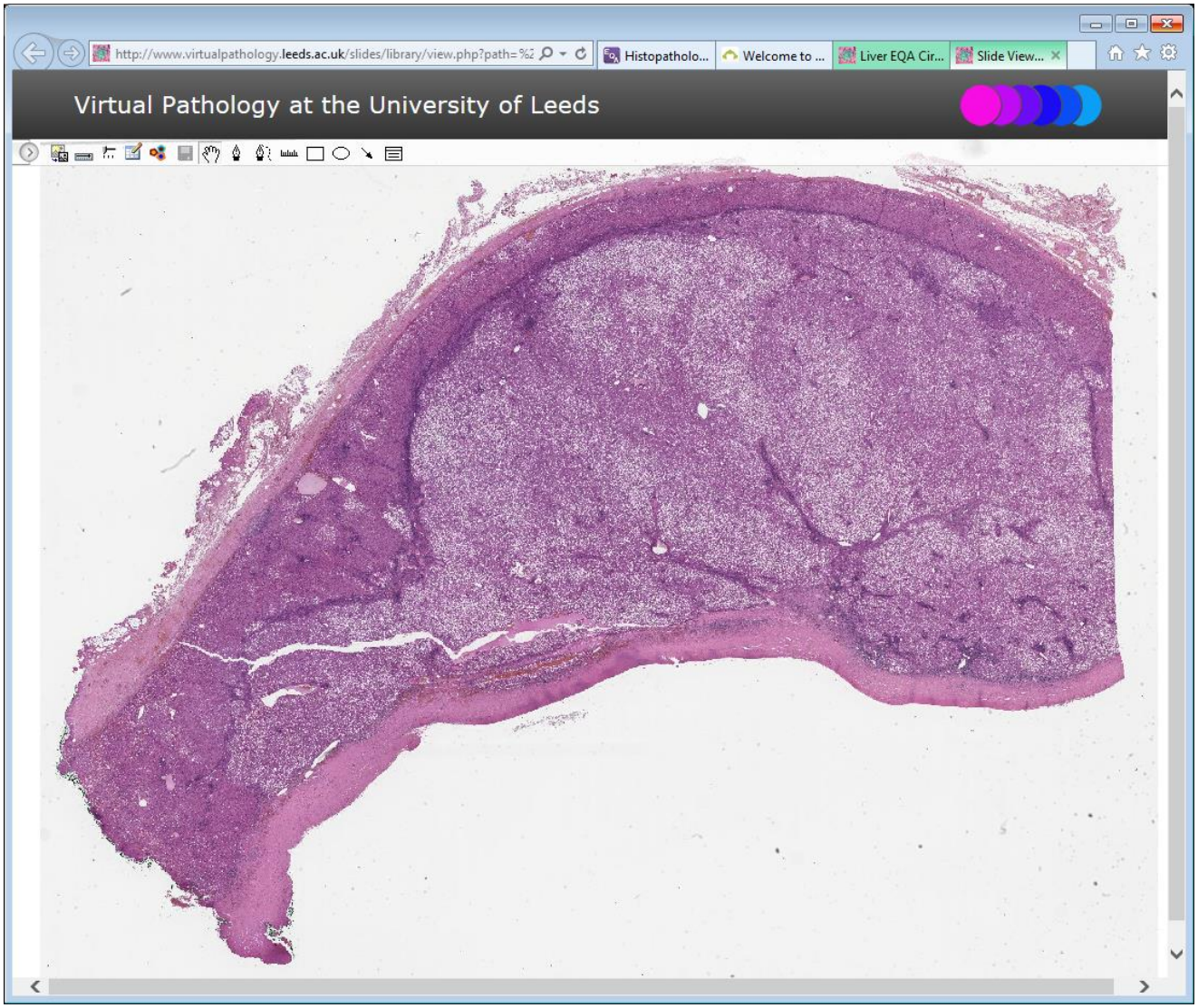
Follow up:

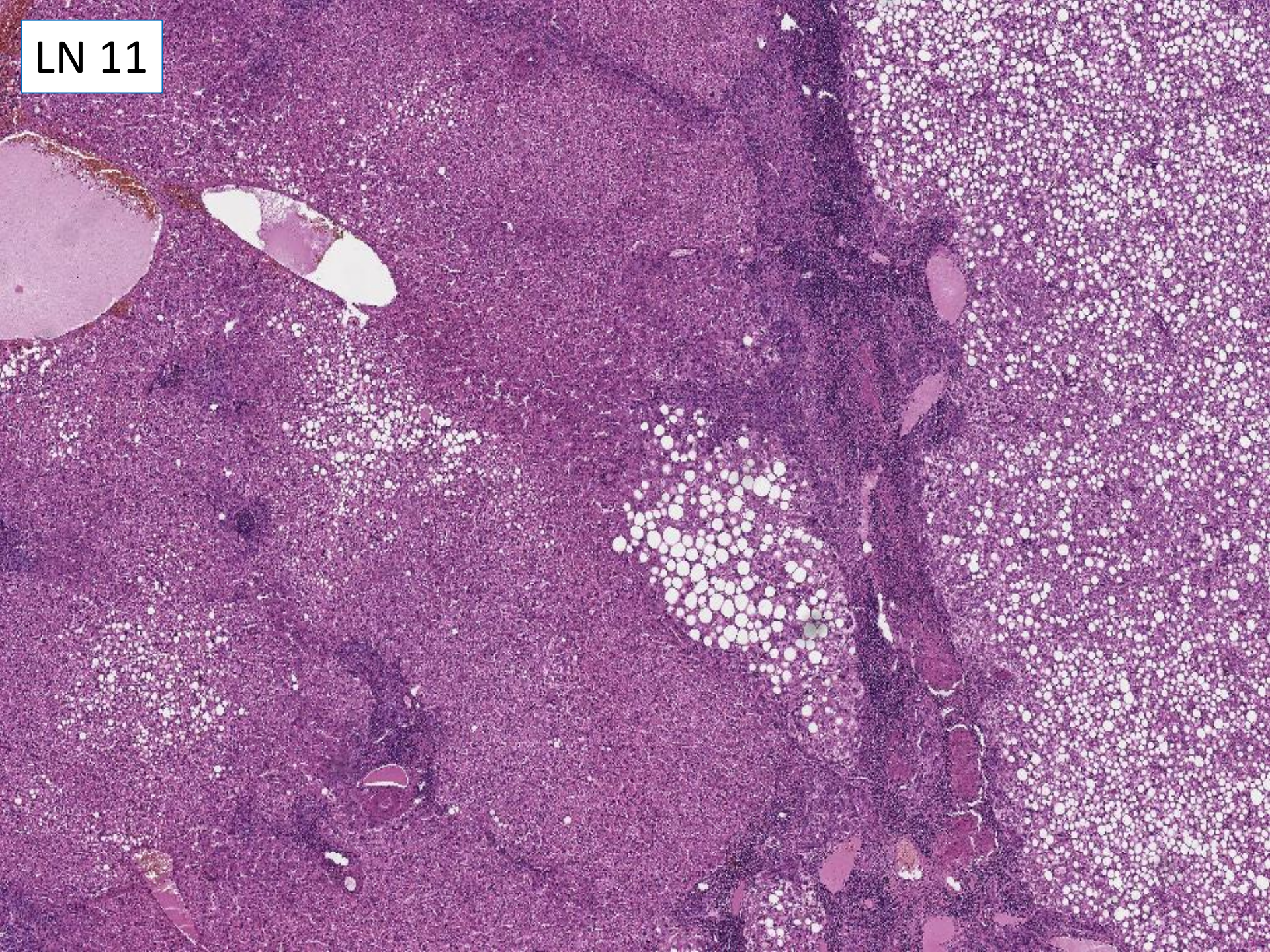
- Underwent pericardiectomy
- Bleeding complications, and died post op.

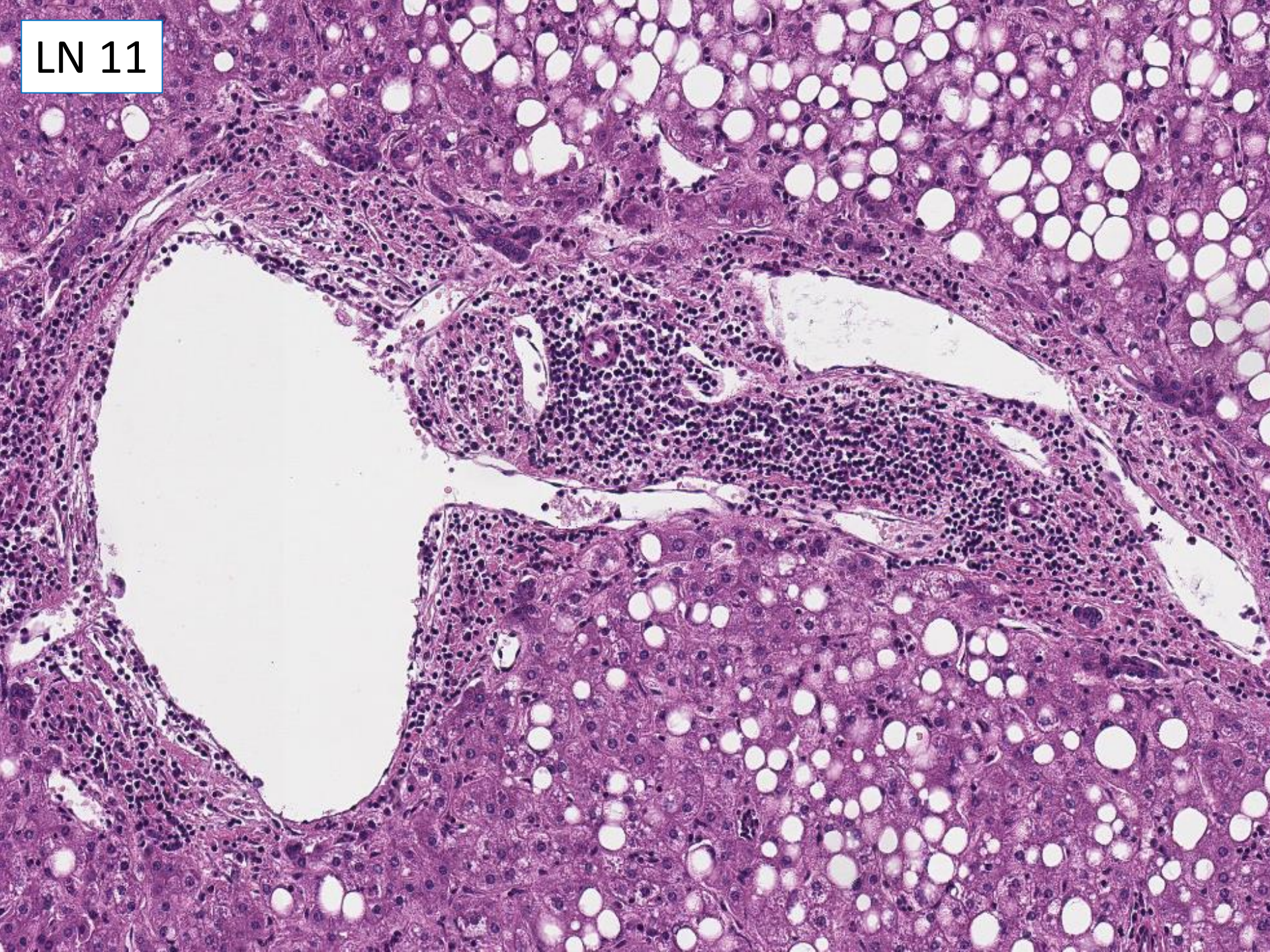
The powerpoint and video presentation are on the CPD page, 2016 update meeting, Cheltenham

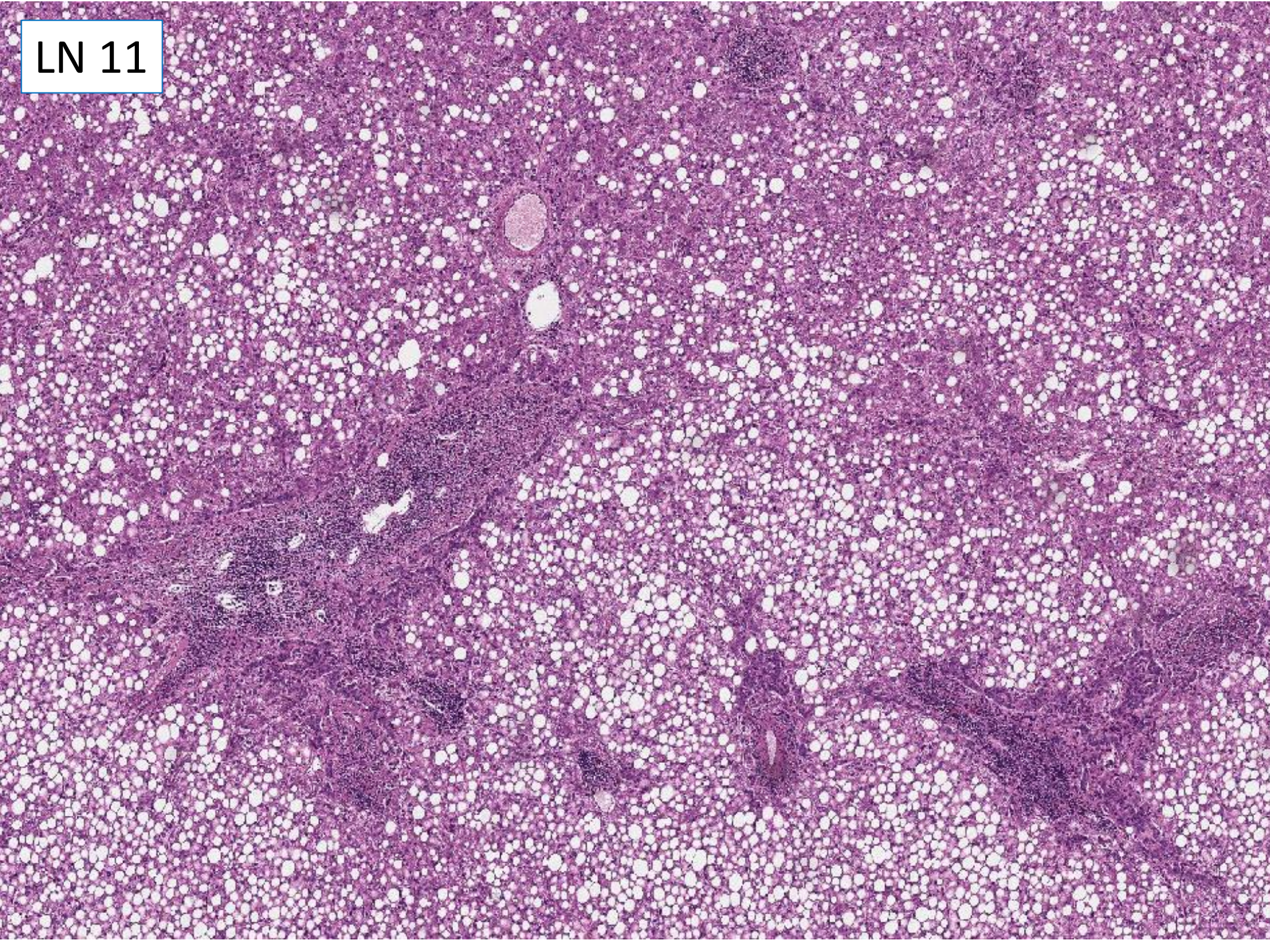
LN11 Female 27 years

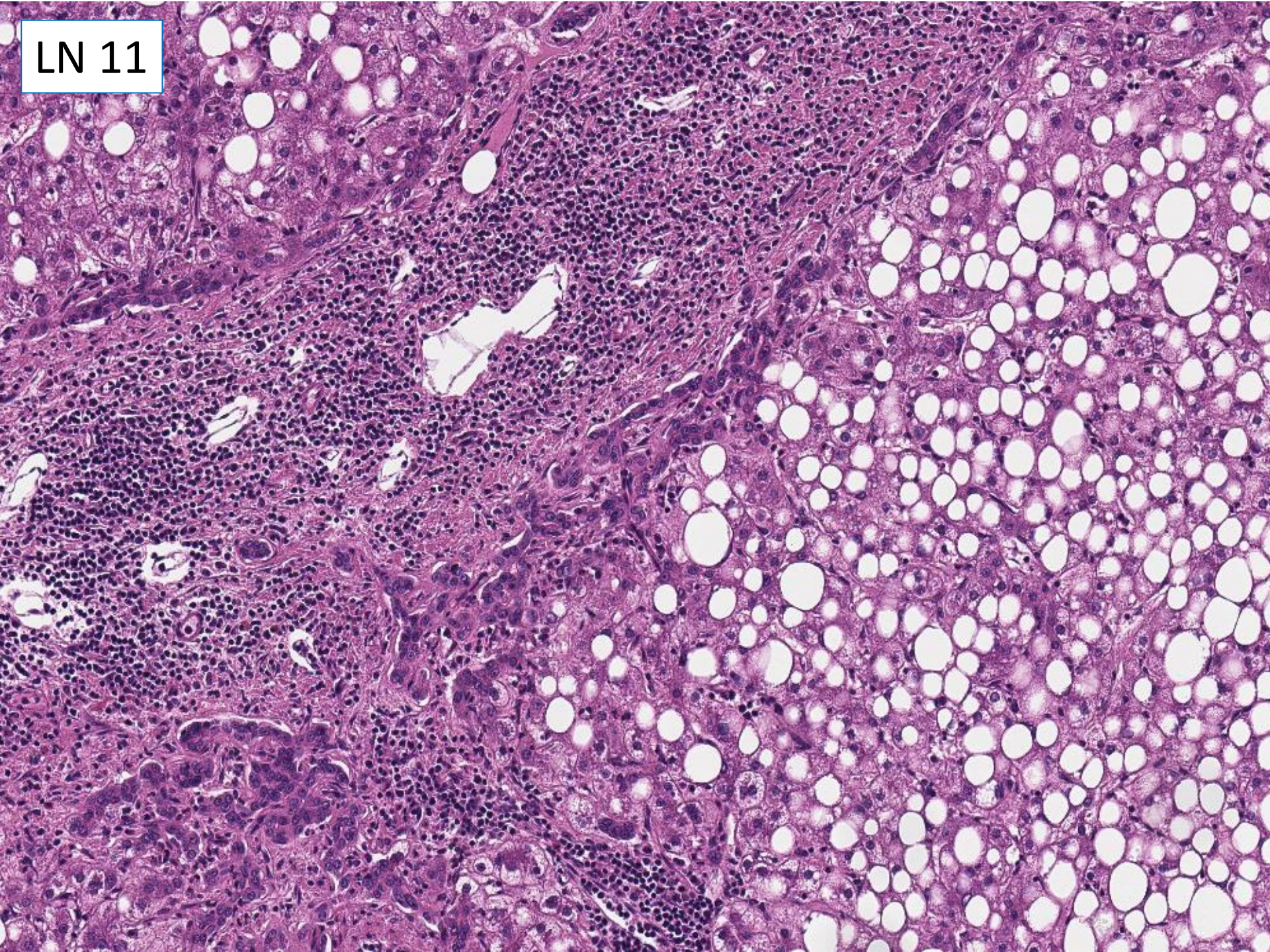
? Adenoma in segment 6.

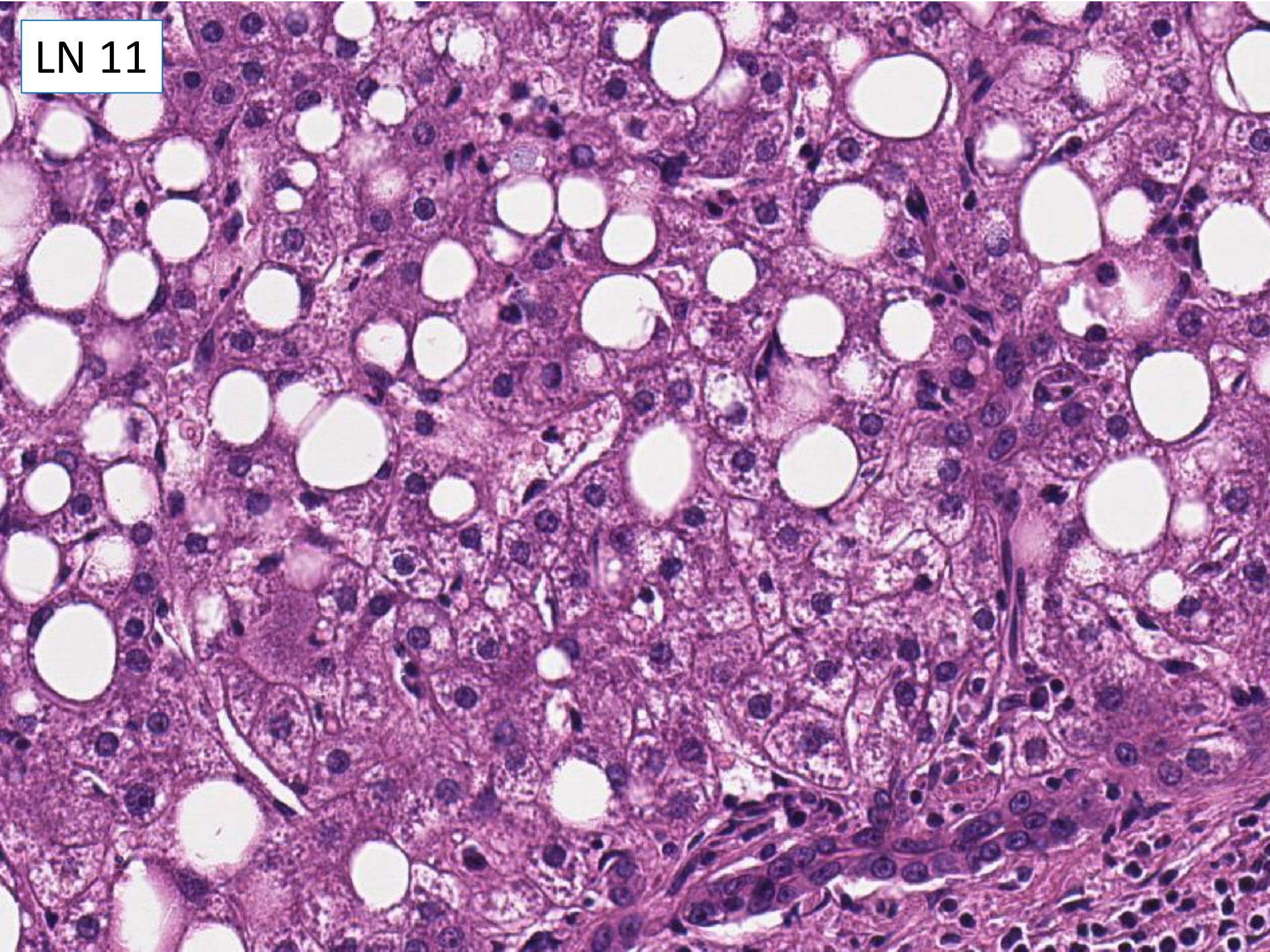



LN 11


LN 11


LN 11


LN 11


LN 11

LN11 Female 27 years ? Adenoma in segment 6.

Hepatocellular adenoma	53
Of which: Not further specified	22
Needs immunos for subtype	5
Most likely steatotic/HNF1a mutated	20
Most likely inflammatory	6
Focal nodular hyperplasia	14
Either adenoma or FNH, of which neither favoured	6
Either, favour adenoma	7
Either, favour FNH	4
Adenoma, differential diagnosis HCC, needs reticulin	Half marks
NASH, fatty change with lymphoid infiltrate, focal lesion not mentioned	1
Of all responses – 38 commented need IHC, 45 not mentioned IHC, 3 needs reticulin	

Suggested scoring: 70 responses are of adenoma alone or with differential diagnosis of FNH. Sufficient consensus for scoring.

LN11 Final scoring after meeting discussion : not scored. Final diagnosis unclear.

See masterclass separate powerpoint presentation (32 slides): diagnosis of focal hepatocellular lesions, especially steatotic.

Masterclass LN11 – Alberto Quaglia

The powerpoint and video presentation are on the CPD page, 2016 update meeting, Cheltenham

Alberto Quaglia, diagnosis of hepatocellular adenoma, Classification from WHO book.

This case LN11 doesn't fit with either steatotic adenoma or inflammatory adenoma, and may be a small FNH with steatosis – for further immunohistochemistry.

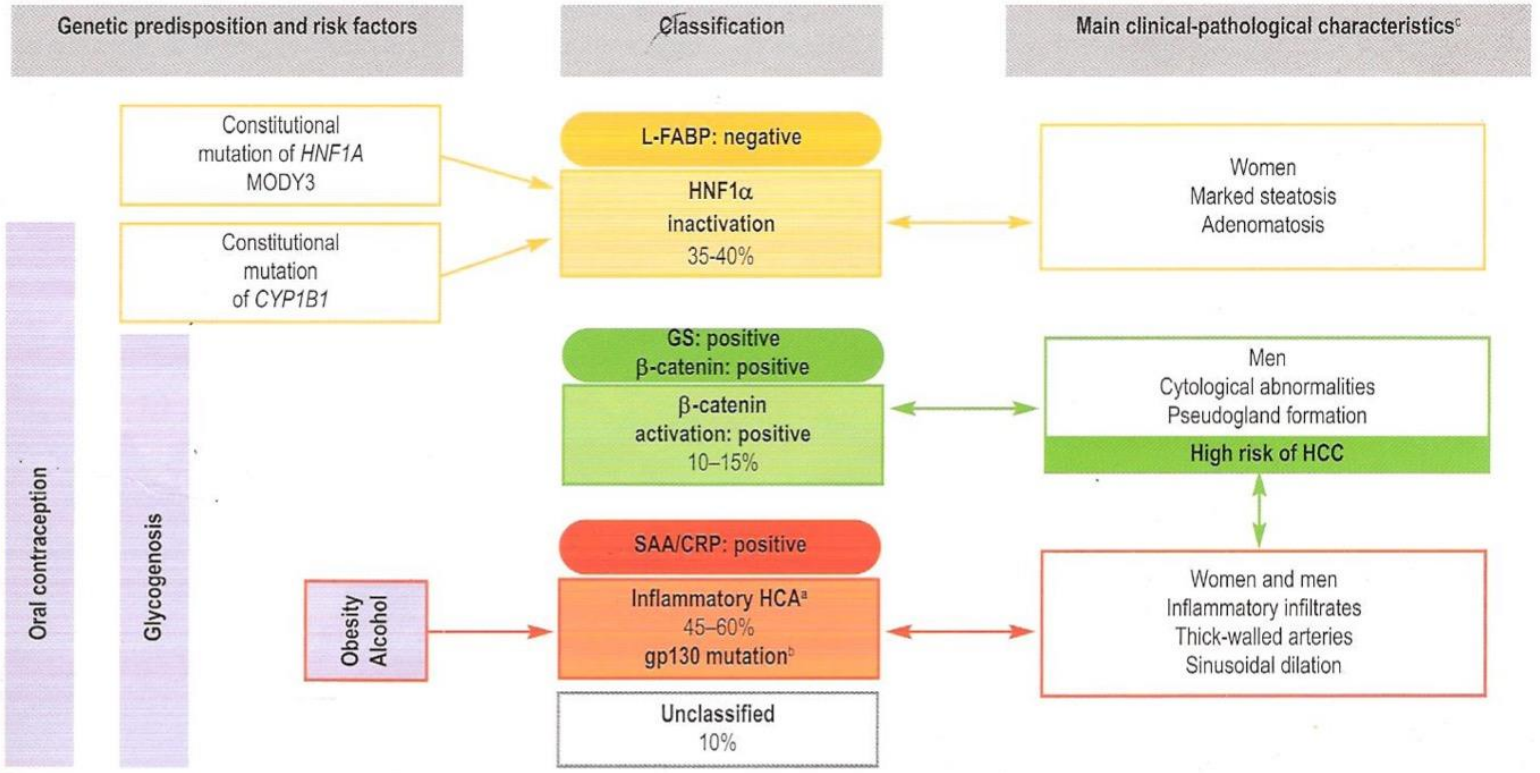
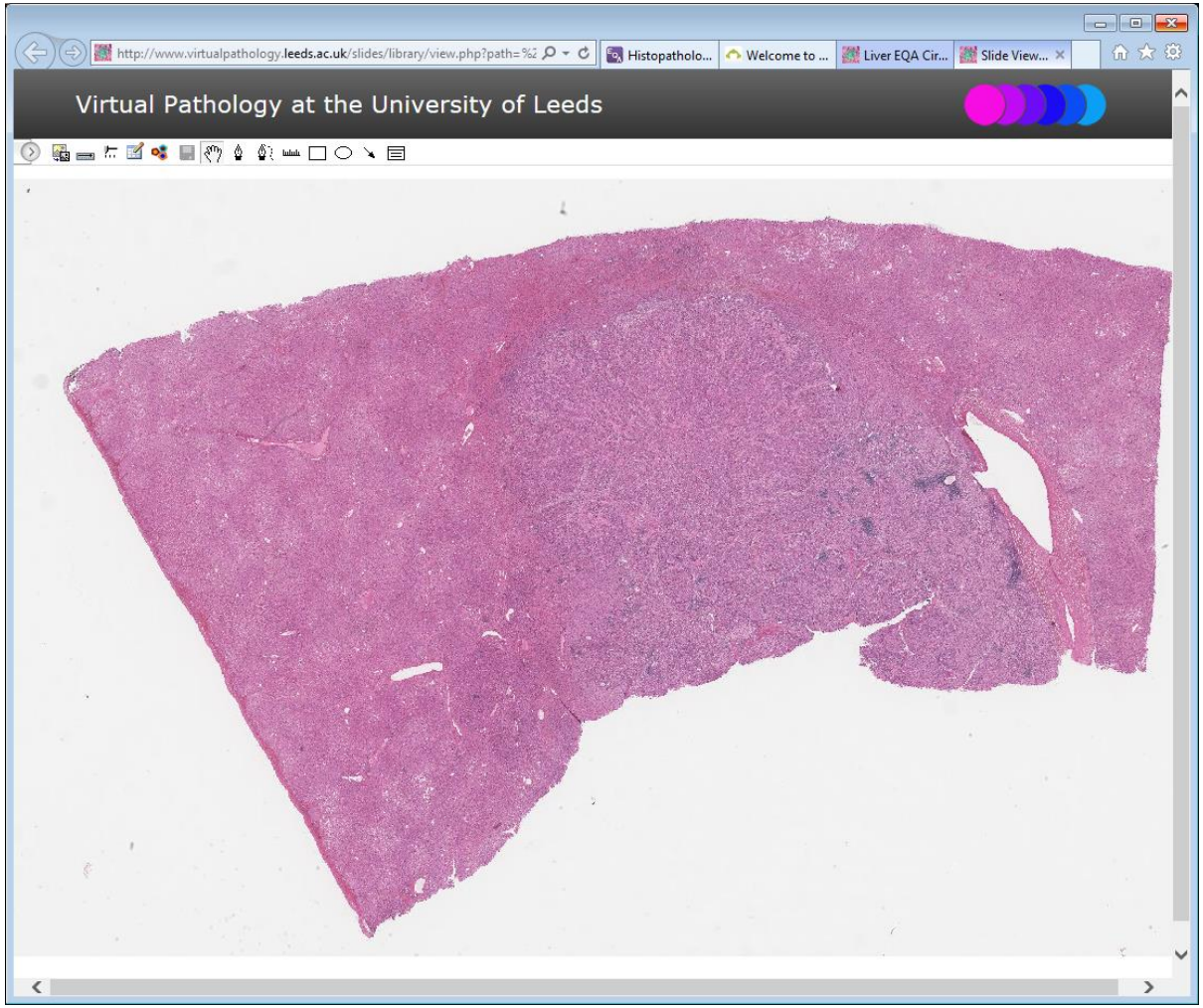
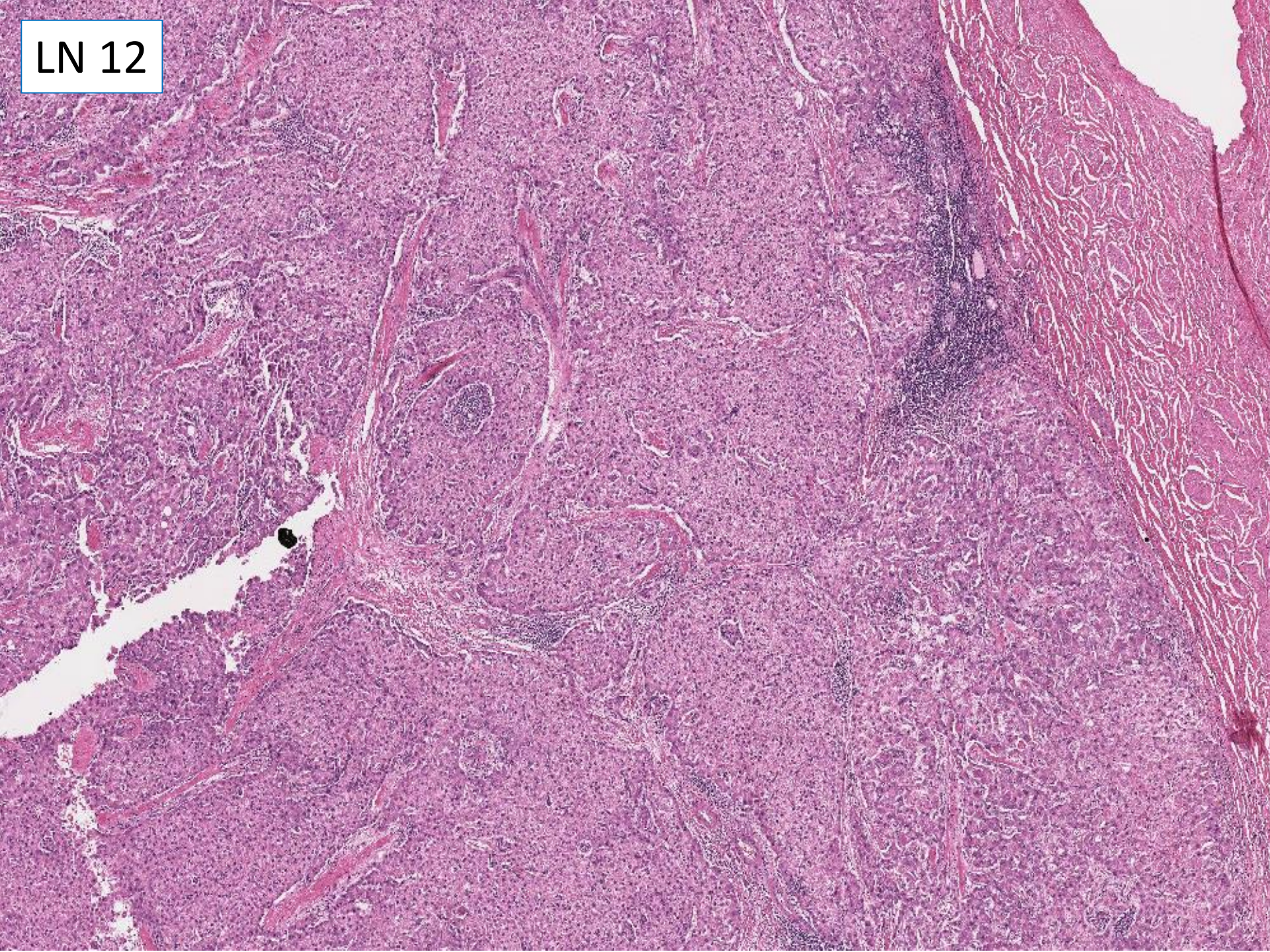


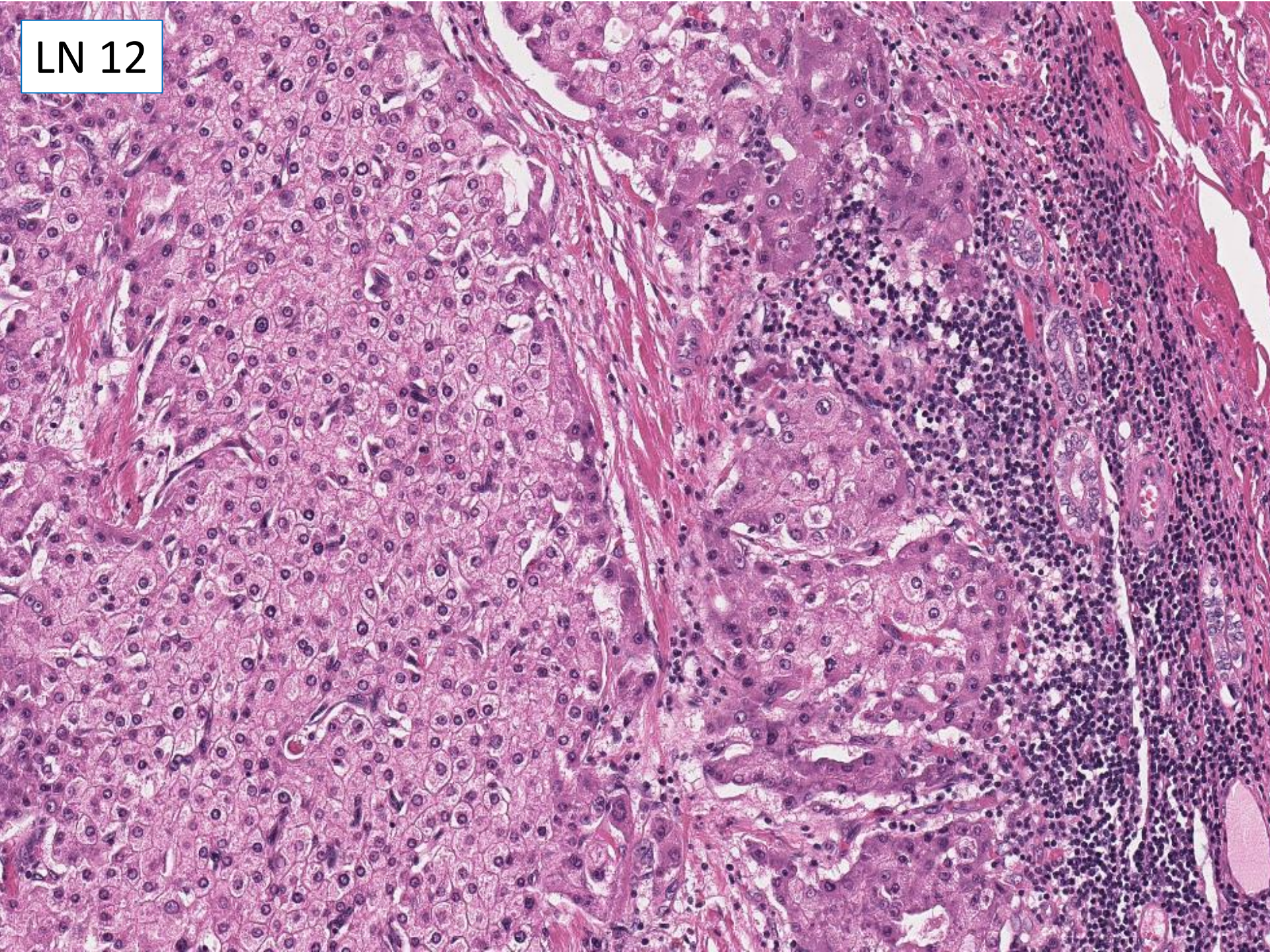
Fig. 10.14 Classification of hepatocellular adenoma (HCA) by genotype and phenotype.
GS, glutamine synthetase; HCC, hepatocellular carcinoma; SAA/CRP, serum amyloid A/C-reactive protein.
[a] 10% of HCAs also have mutant β-catenin; [b] Mutations in the gene encoding gp130 are found in 60% of inflammatory HCAs; [c] These characteristics are frequent, but not exclusive.

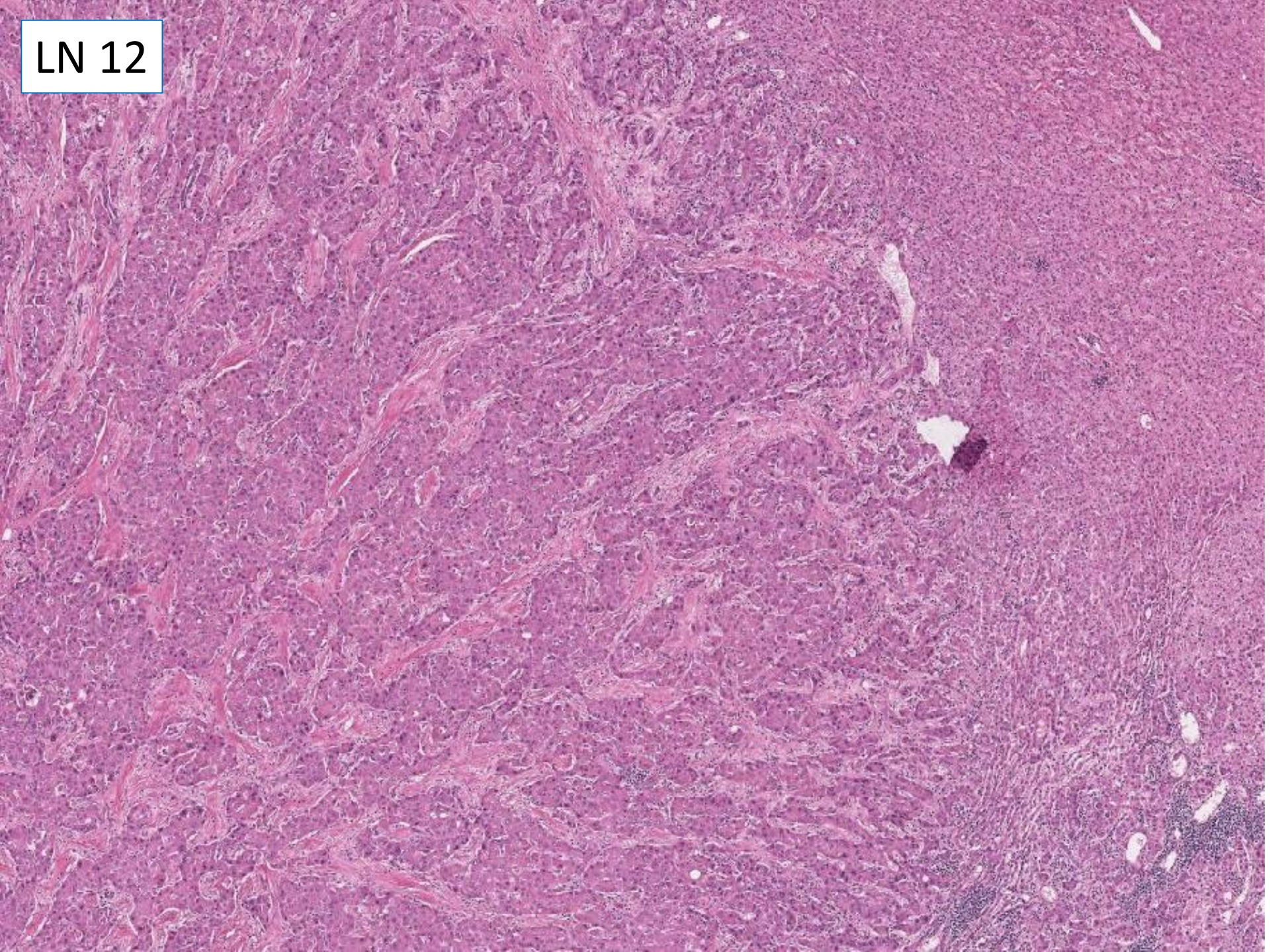
LN12 Male 73 years

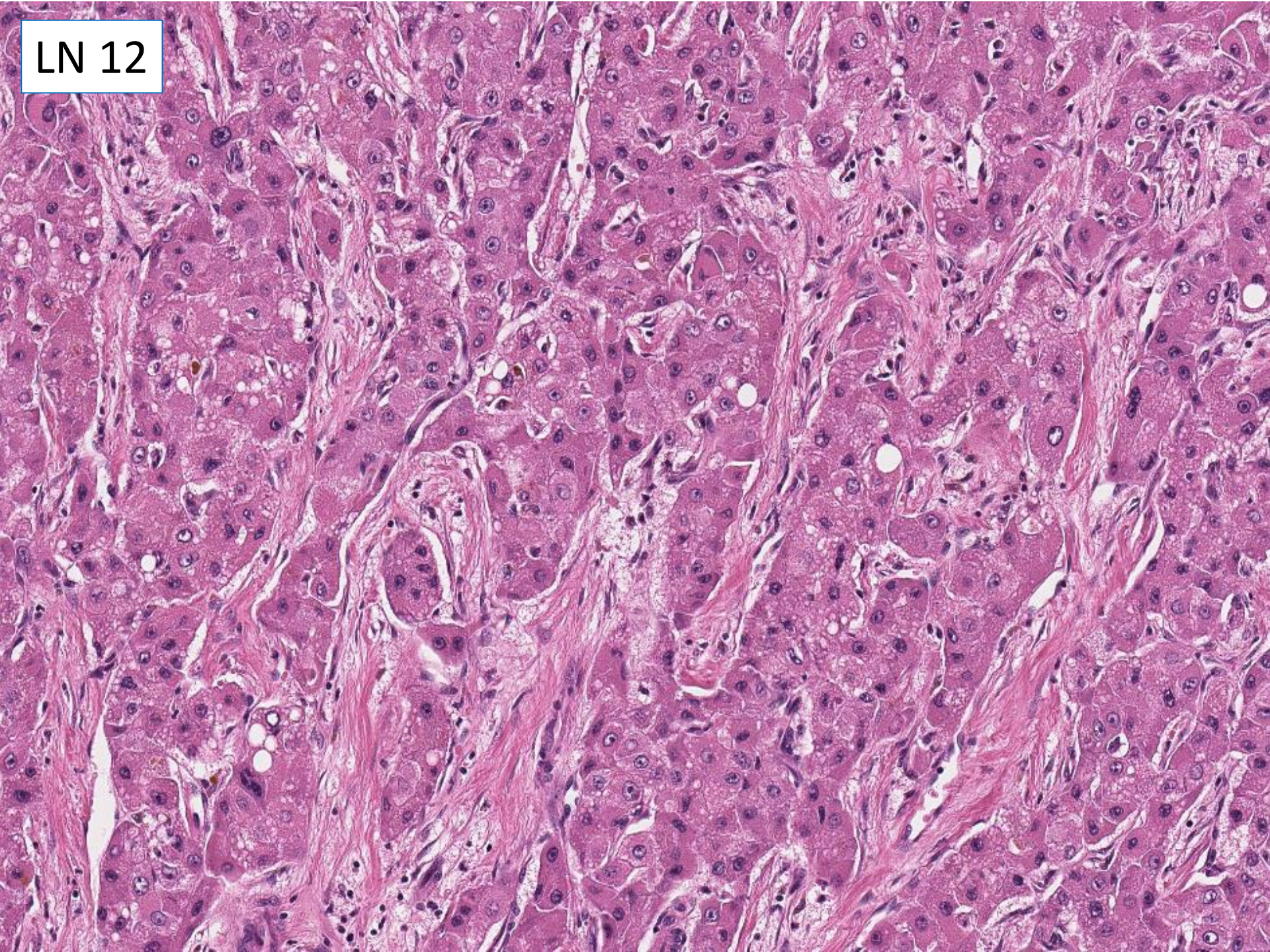
Colorectal liver mets. Laparoscopic left liver lobectomy.




LN 12


LN 12


LN 12


LN 12

LN12 Male 73 years

Colorectal liver mets. Laparoscopic left liver lobectomy.

Hepatocellular carcinoma	39
Hepatocellular carcinoma, fibrolamellar type	42
Favours HCC over metastatic carcinoma	4
'hepatoid adenocarcinoma, needs IHC'	1
Background liver – not cirrhotic	36
– needs collagen stains for stage	6
Suggestive of cirrhosis	1
Not mentioned	37

Full marks

no marks

LN12 final scoring after meeting discussion: for full marks – diagnosis of hepatocellular carcinoma, or favours HCC over metastasis.
No marks for 'hepatoid adenocarcinoma needs IHC'.
Insufficient comment on background liver to include that in the scoring.

Comment from Stefan Hubscher: "Mixed FLC" more frequently occurs in older people, have higher AFP levels and worse prognosis with more intrahepatic recurrence/metastases (compared with "pure" FLC)

Liver EQA – Circulation LN, Case 12

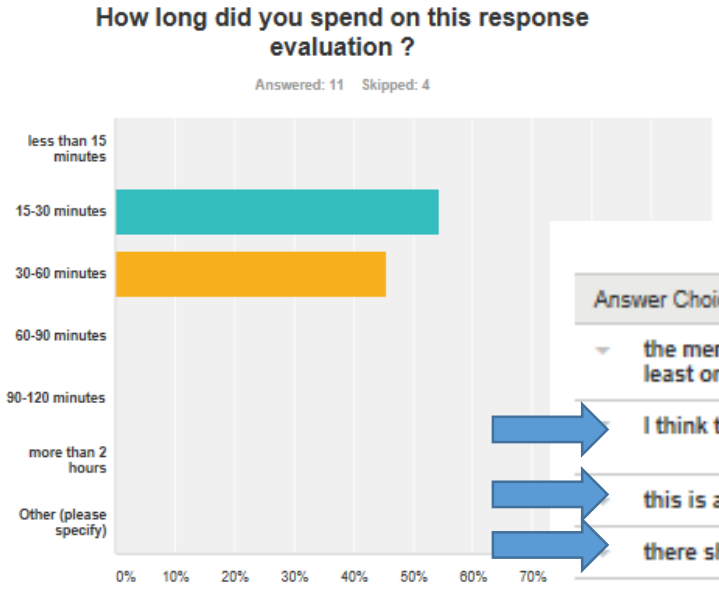
‘Masterclass’ presentation by Stefan Hubscher

The PowerPoint and video presentation are on the CPD page, 2016 update meeting, Cheltenham

Fibrolamellar carcinoma versus conventional HCC

1. Clinico-pathological features
2. Tumour biology
3. Problems with histological diagnosis

Circulation LN – pre-meeting comments on suggested scoring received from 11 members.



Answer Choices	Responses
the members should be requried to comment on the proposed scoring (e.g. for at least one circulation per year?) as part of thier membership	36.36% 4
I think that commenting on the scoring was a useful and important thing for me to do.	100.00% 11
this is a good format to enable members' comments to be made	90.91% 10
there should be another CPD point for members who subit comments on the scoring	72.73% 8
the scoring should be decided during the open meeting, no need to do this first	0.00% 0
the scores can just be assigned by the organiser and presented at the open meeting without further discussion -focus discussion on the diagnostic points of the case	9.09% 1
it is useful to also see the full text of the responses in the excel spreadsheet	36.36% 4
I didn't look at the excel spread sheet of full respones - just the collation	54.55% 6
I expect that I will do this again	100.00% 11
I started, but didn't manage to finish doing this	0.00% 0
scoring should be done in a different way, e.g. cases should be assigned to individual EQA member sto be collated, not done by the organiser	9.09% 1
any comments? Responses	36.36% 4
Total Respondents: 11	

Routine reporting liver biopsies in the UK –

Pre-course questionnaire – **Gloucester GI meeting 2012**:
What liver pathology do you do, and how do you do it?

110 responses received = half of registrants

of which 76% report liver biopsies

– 'no special interest in liver' (53)

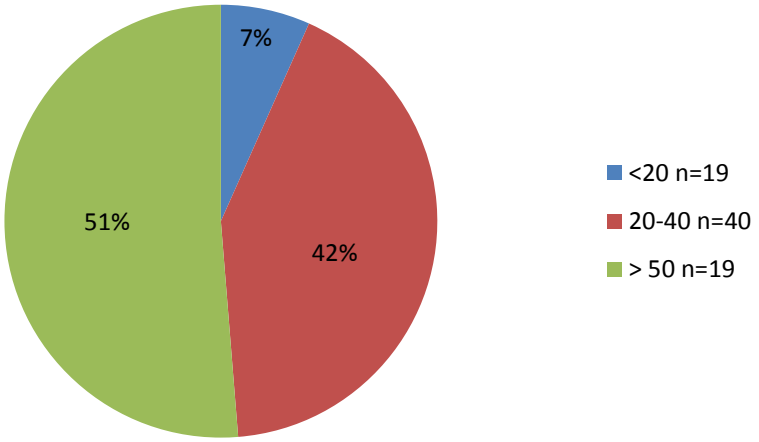
- 'others would see me as a local specialist' (25)

Roughly how many medical liver biopsies do you report each year?

between 3 and 200, 6 don't know,

medical liver biopsies per year	% of histology reports	Generalist (n=53, 68%)	'local specialist' (n=25, 32%)	total
≤20 (n=19)	0.3%	17	2	19
20-40 (n=40)	1.0%	31	9	40
≥ 50 (n=19)	2.1%	5	14	19

2696 biopsies per year reported by 78 of you



UK Liver Histopathology EQA Scheme

- Since 1994 – on line since 2003,
  - 324 cases with results and comments.
- Now 103 members – 30 here today, with 70 ‘guests’

- UK Liver Pathology Group – created this year,
- **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.**
- Full members, associate members, trainee members
- http://www.virtualpathology.leeds.ac.uk/eqa/specialist/liver/liver_group.php

The UK Liver Pathology Group

< back to Liver pages

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.

Constitution of the UKLPG DOC

The UKLPG developed from the UK Liver EQA Scheme and the liver subcommittee of the Pathology Section of the BSG, following meetings in September 2014 and May 2016.

The committee members were elected in June 2016.

There are three sub-committees responsible for education, quality assurance and research. Please see the UKLPG constitution for full details.

The committee membership in September 2016 DOC

The committee aims to meet by teleconference four times a year, and the minutes of those meetings will be on this website.

Meeting 14th September 2016 DOC

Initial meeting to finalise the constitution of the UKLPG Meeting 4th May 2016 DOC

UK Liver Pathology Group

- Full members = EQA membership
  - Pathologists in hepatology and transplant centres
  - Others with an interest in liver pathology,
  - Recommended for ‘lead for liver pathology’
    (RCPath Tissue Pathways)
- Associate members = other Consultants who report liver biopsies, who are not in the EQA scheme
- Trainee members = trainees interested in liver pathology
- Receive emails about educational events etc.

The End