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	2
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	

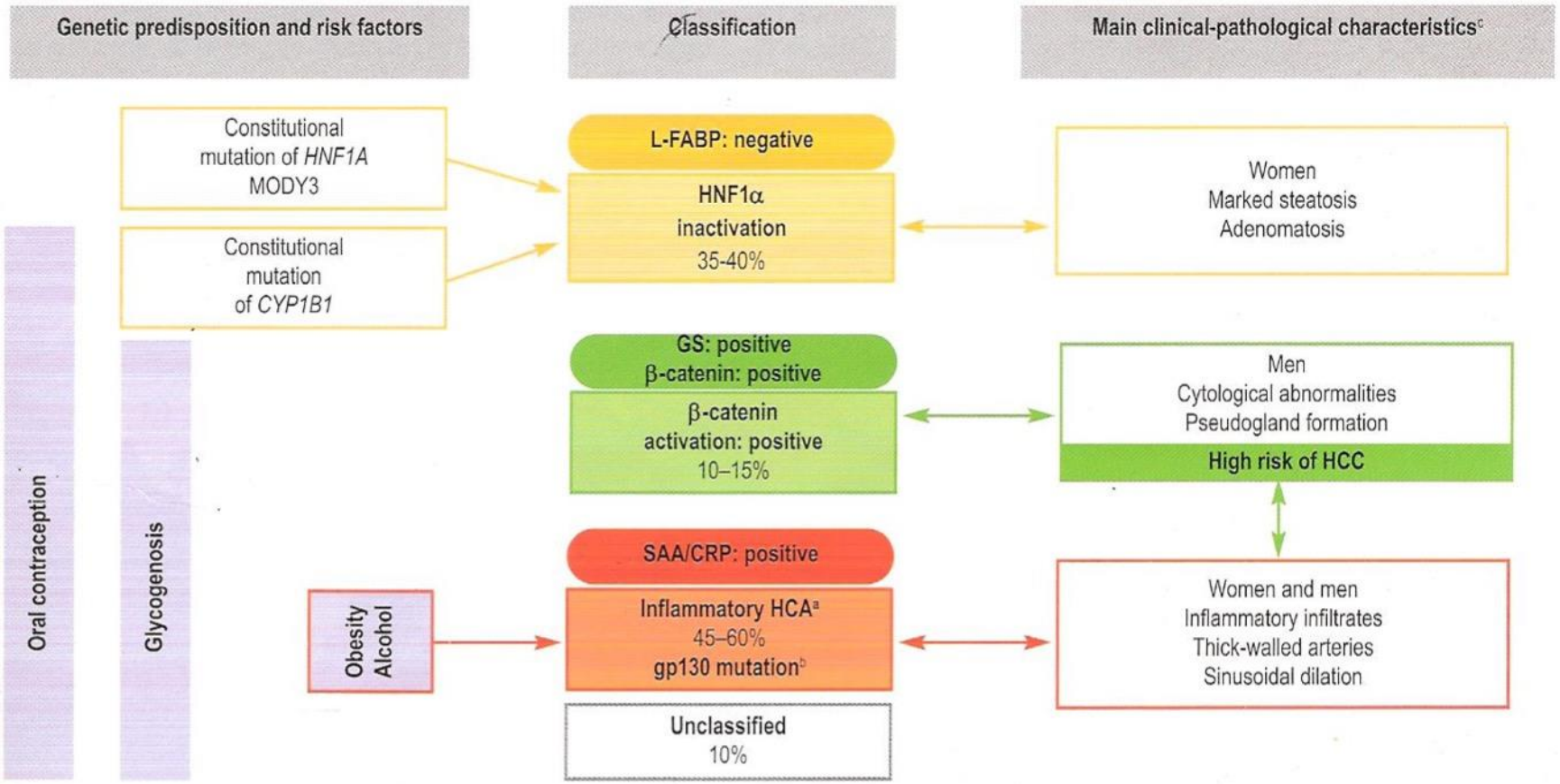


Fig. 10.14 Classification of hepatocellular adenoma (HCA) by genotype and phenotype.
GS, glutamine synthetase; HCC, hepatocellular carcinoma; SAA/CRP, serum amyloid A/C-reactive protein.
[a] 10% of HCAs also have mutant β-catenin; [b] Mutations in the gene encoding gp130 are found in 60% of inflammatory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

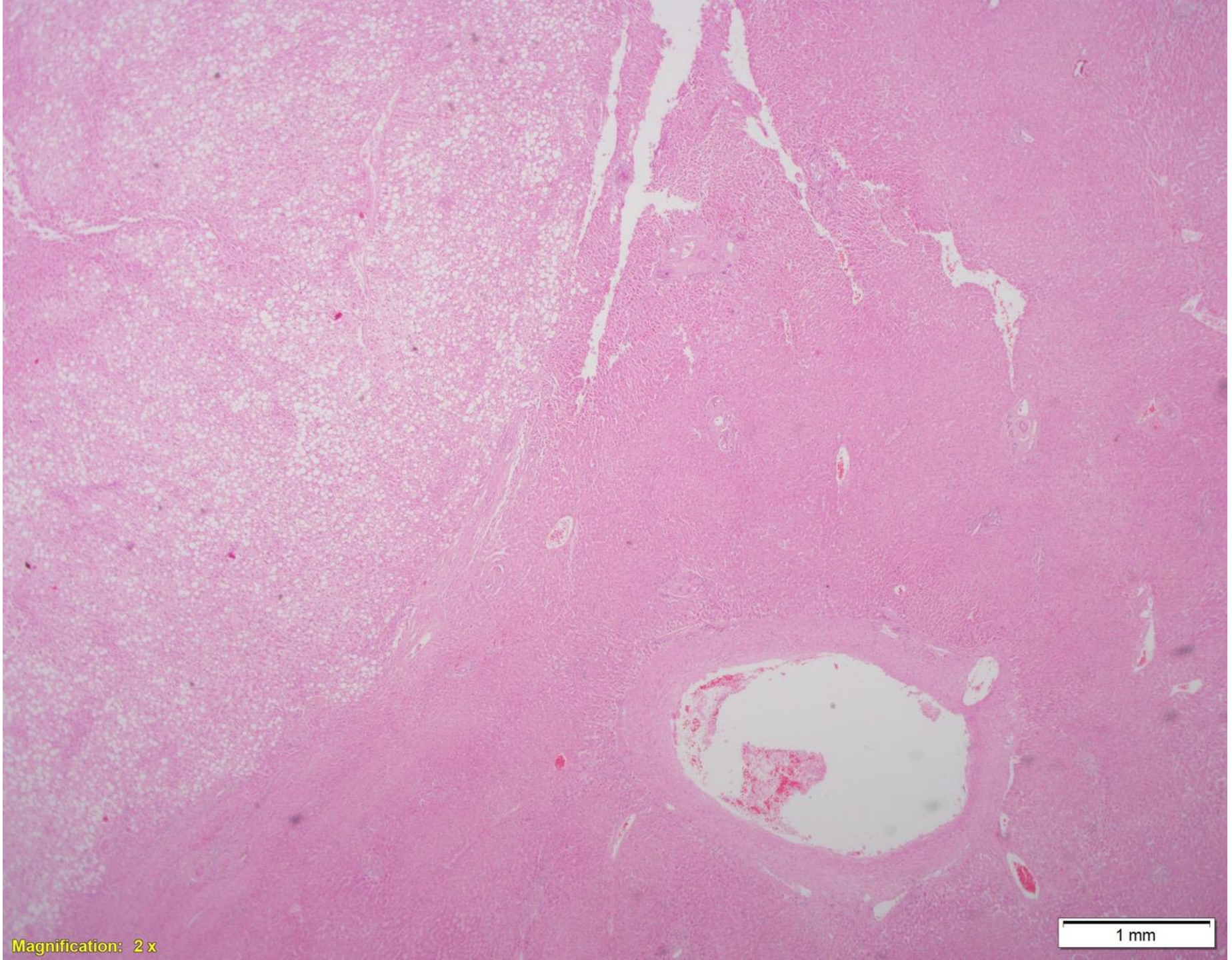
Table 10.03 Histological features of subtypes of hepatocellular adenoma (HCA).

Histological feature	HNF1α– inactivated HCA	β-Catenin-activated HCA	Inflammatory HCA[a]	Unclassified HCA
Steatosis	Usually +++ Rarely + Extremely rarely –	–	– to ++ (heterogeneous) rarely +++	+/–
Sinusoidal dilatation and peliosis	– to +	– to +/-	– to +++	– to ++
Ductular reaction	–	–	+/– to +++	–
Abnormal thick arteries	–	–	+ to ++	– to +/–
Inflammatory reaction	– to +/–	–	+ to +++	–/+
Cytological abnormalities	–	+	–[b]	–
Remodelling (necrosis, haemorrhage, fibrotic bands)	– to +/–	– to +/–	– to ++	– to +/–
Immunohistochemistry				
L-FABP[c]	–	+	+	+
Glutamine synthetase[d]	–	+	–[b]	–
β-Catenin (aberrant nuclear expression)	–	+	–[b]	–
SAA/CRP[e]	–	–	+ to +++	–

L-FABP, liver fatty acid-binding protein; HNF1α, hepatocyte nuclear factor 1 α; SAA/CRP, Serum amyloid A/C reactive protein. Grading: +, mild; ++, moderate; +++, severe; –, absent.

[a] Can also have mutant β-catenin; [b] Except if β-catenin is mutated; [c] Normally expressed in nontumoral liver; [d] Glutamine synthetase is occasionally expressed at the periphery and around veins of any HCA without β-catenin mutation;

[e] Rarely, only one is expressed; occasionally, both can be overexpressed in nontumoral liver as a general inflammatory reaction (i.e. in response to bleeding).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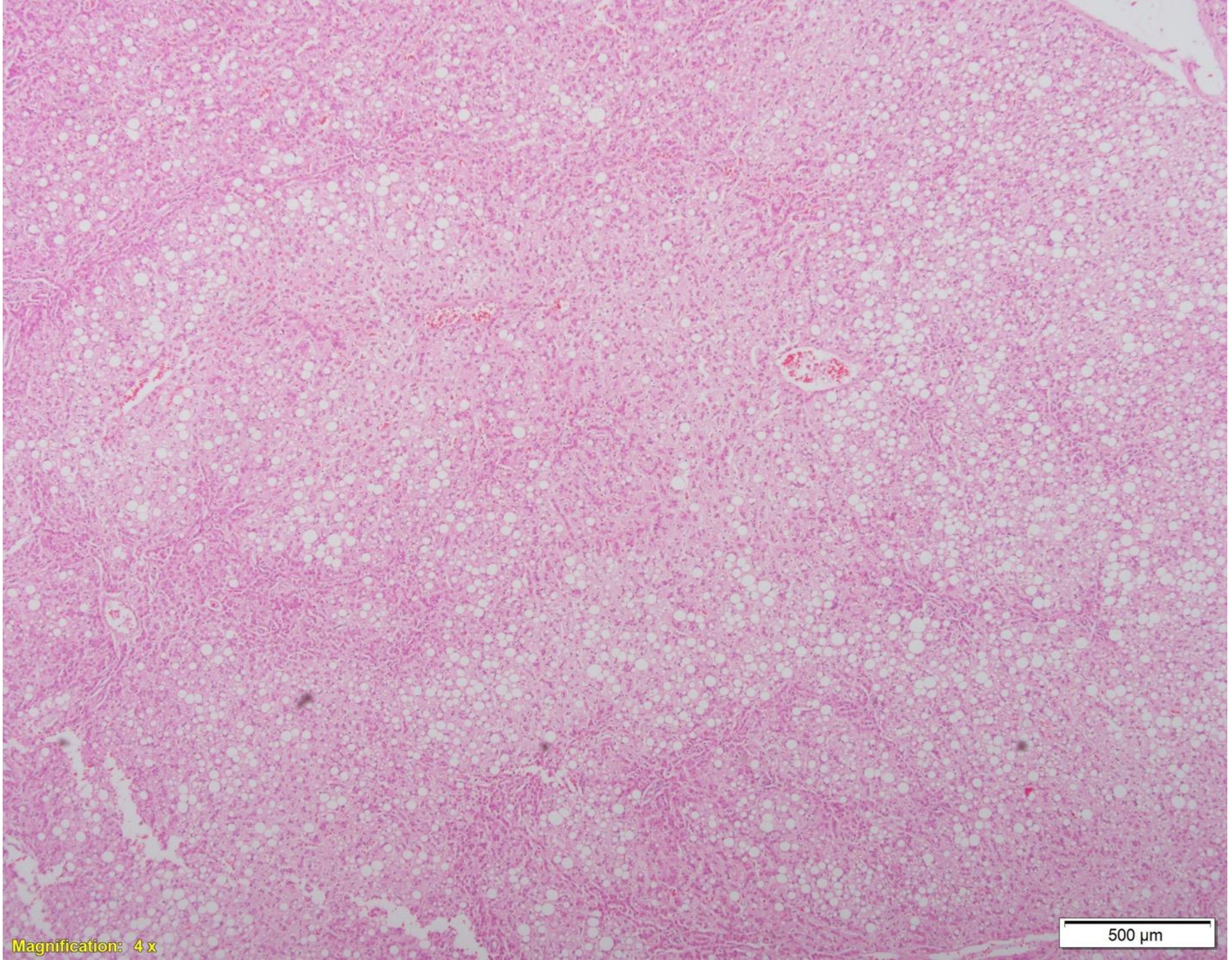
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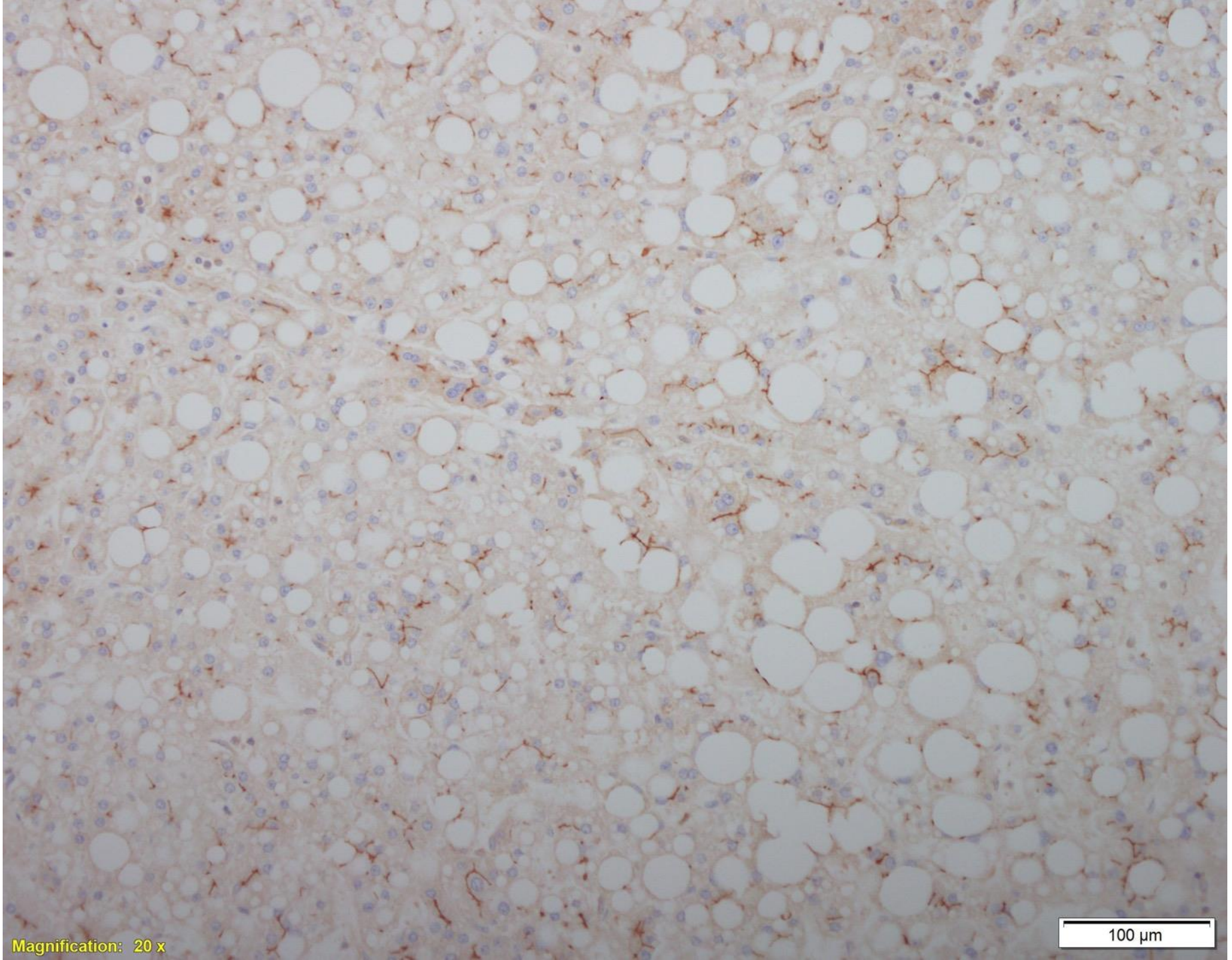
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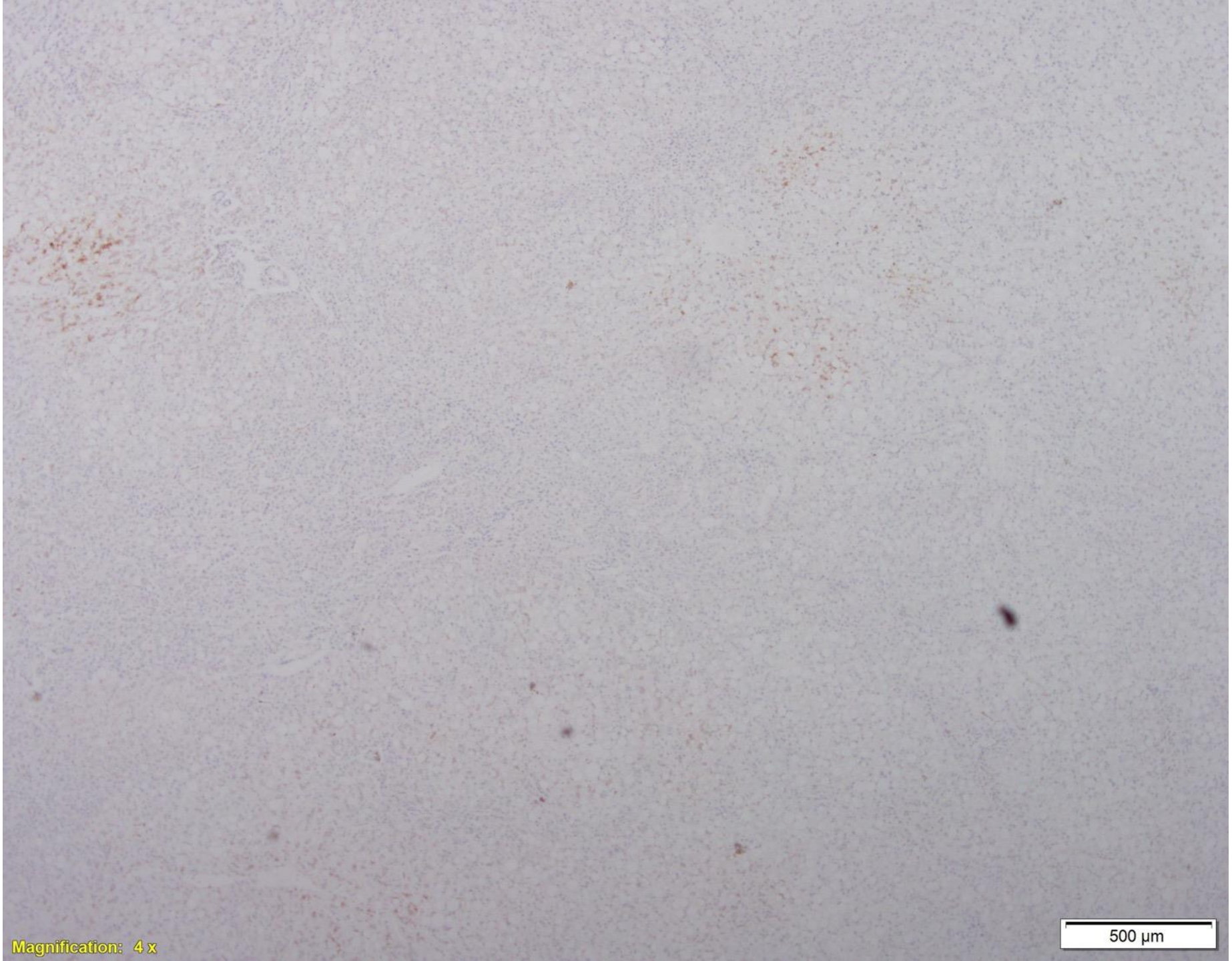
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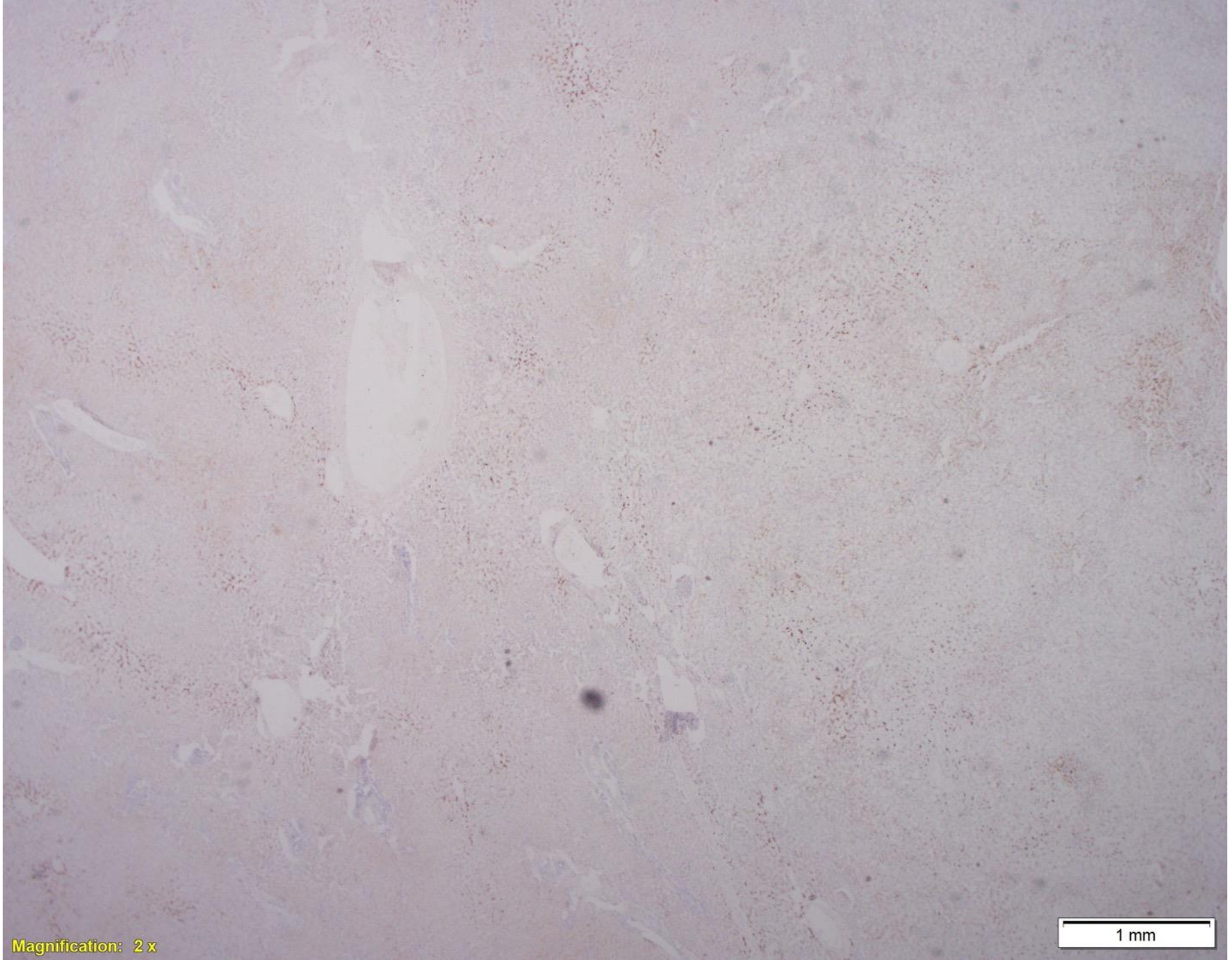
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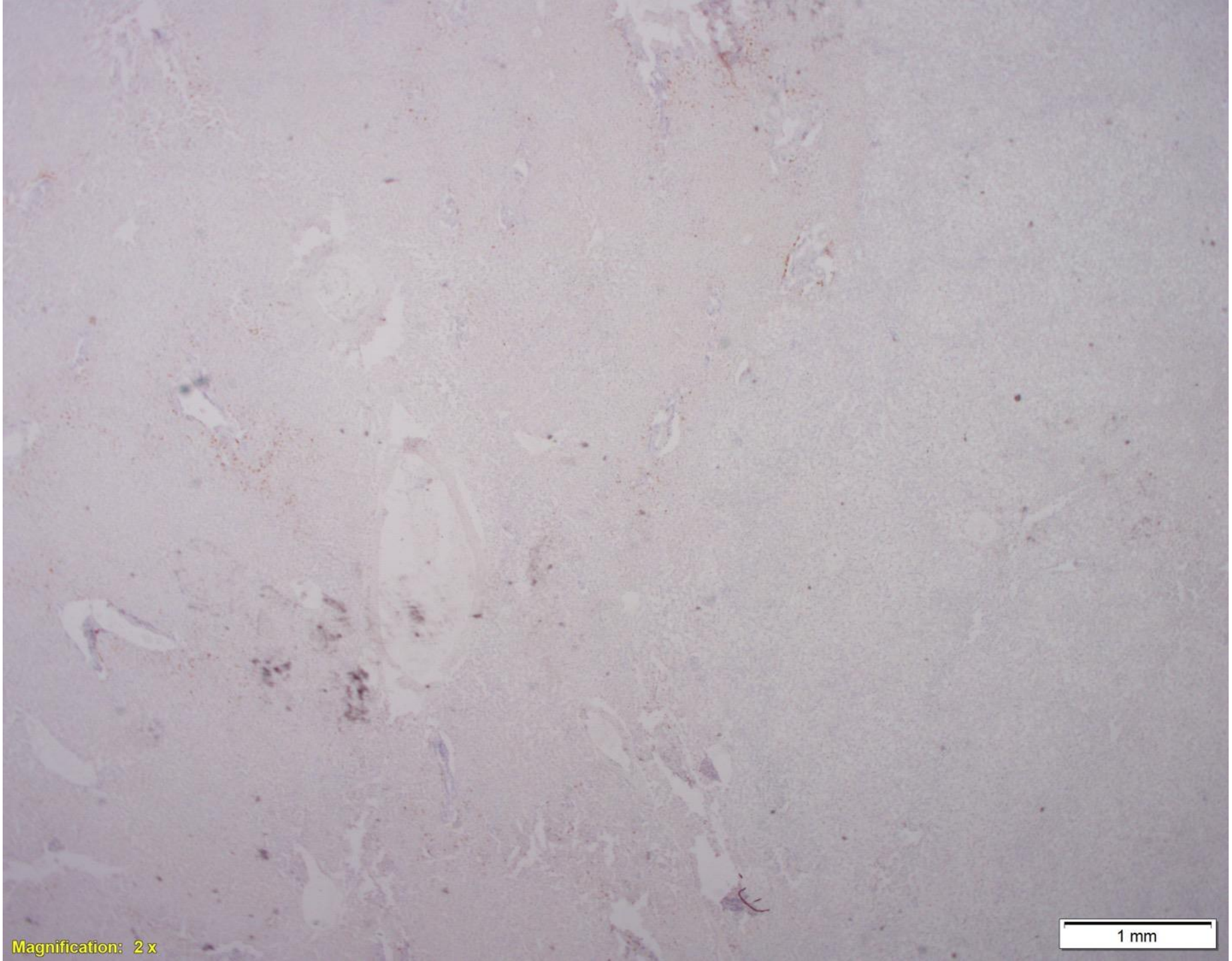
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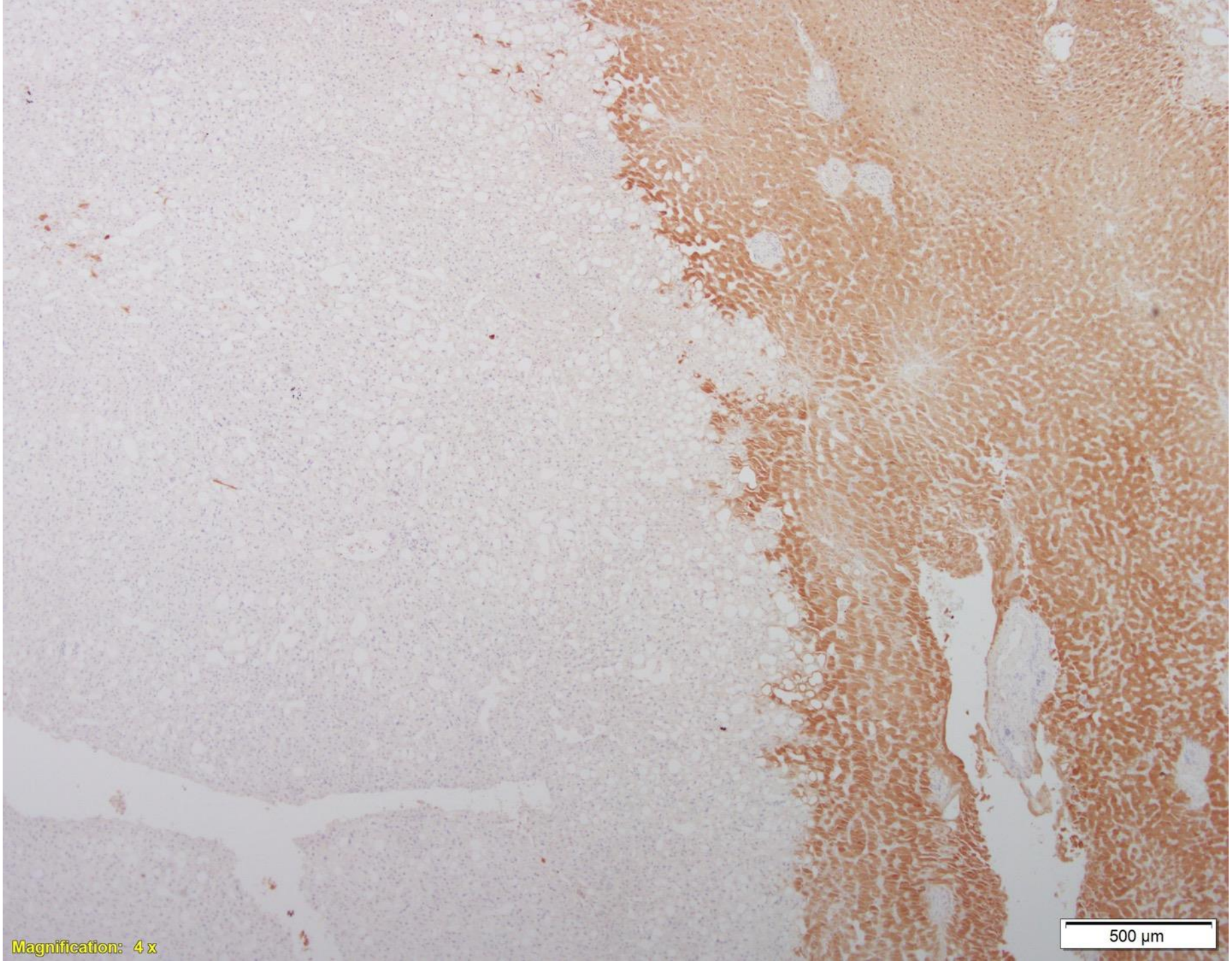
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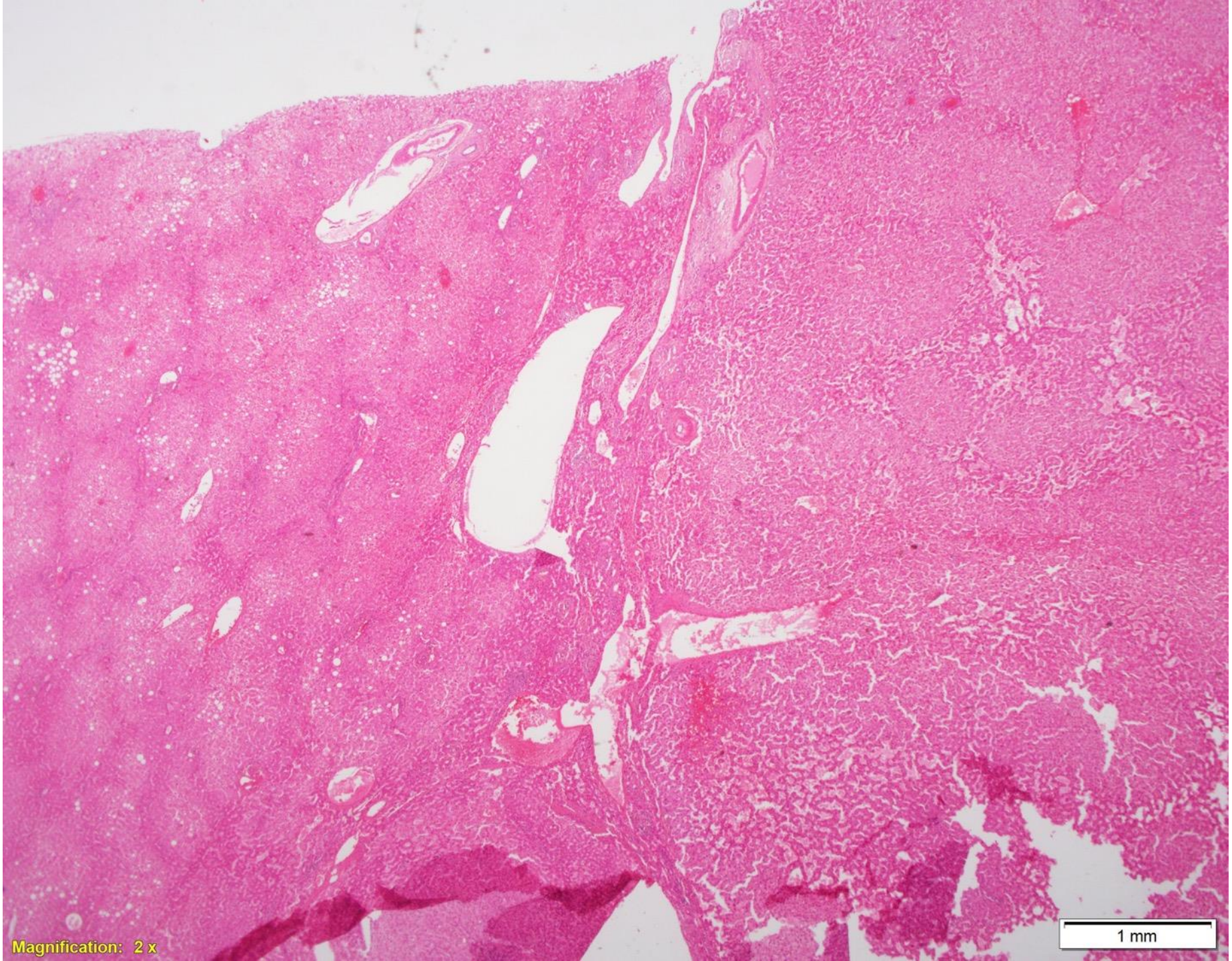
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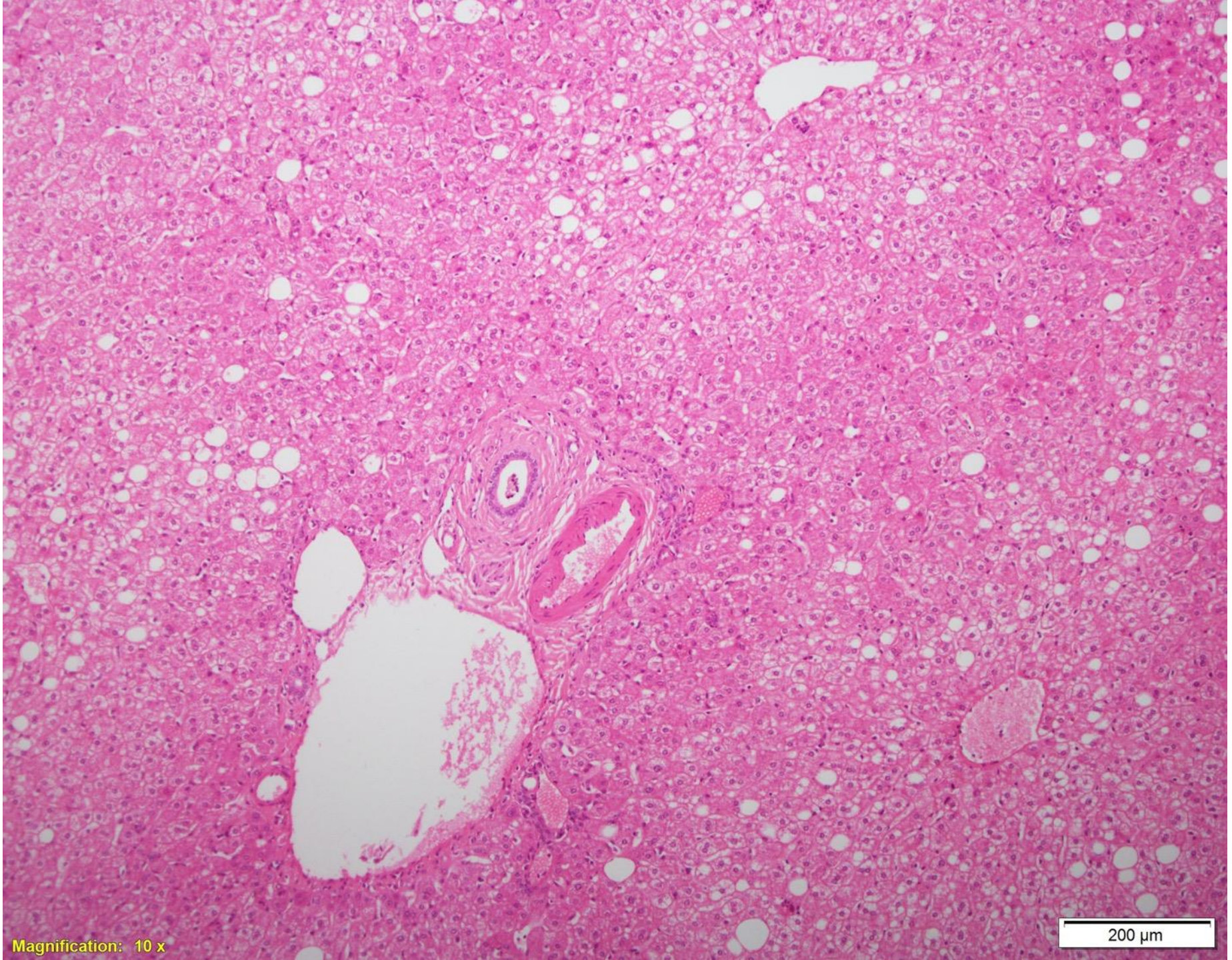
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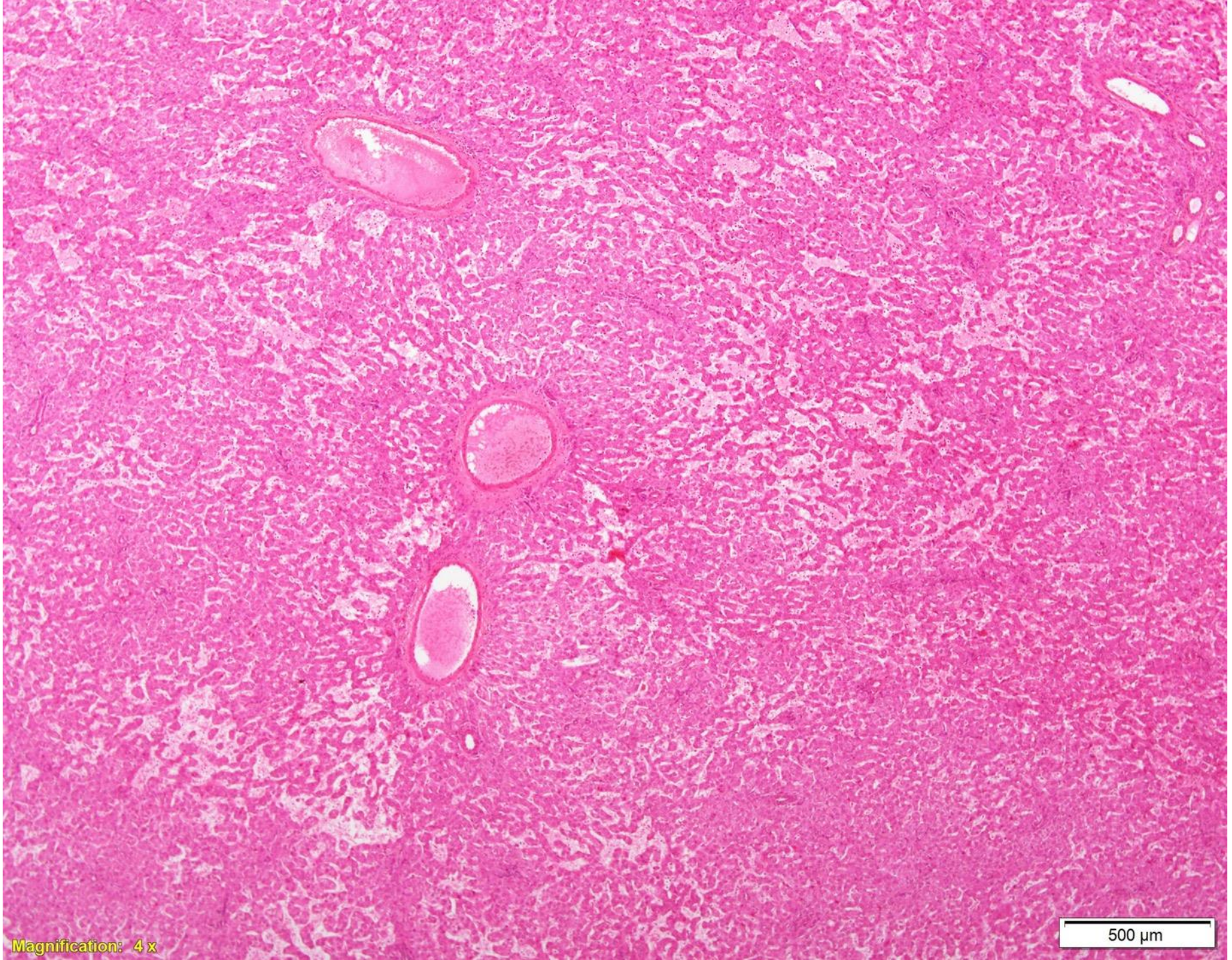
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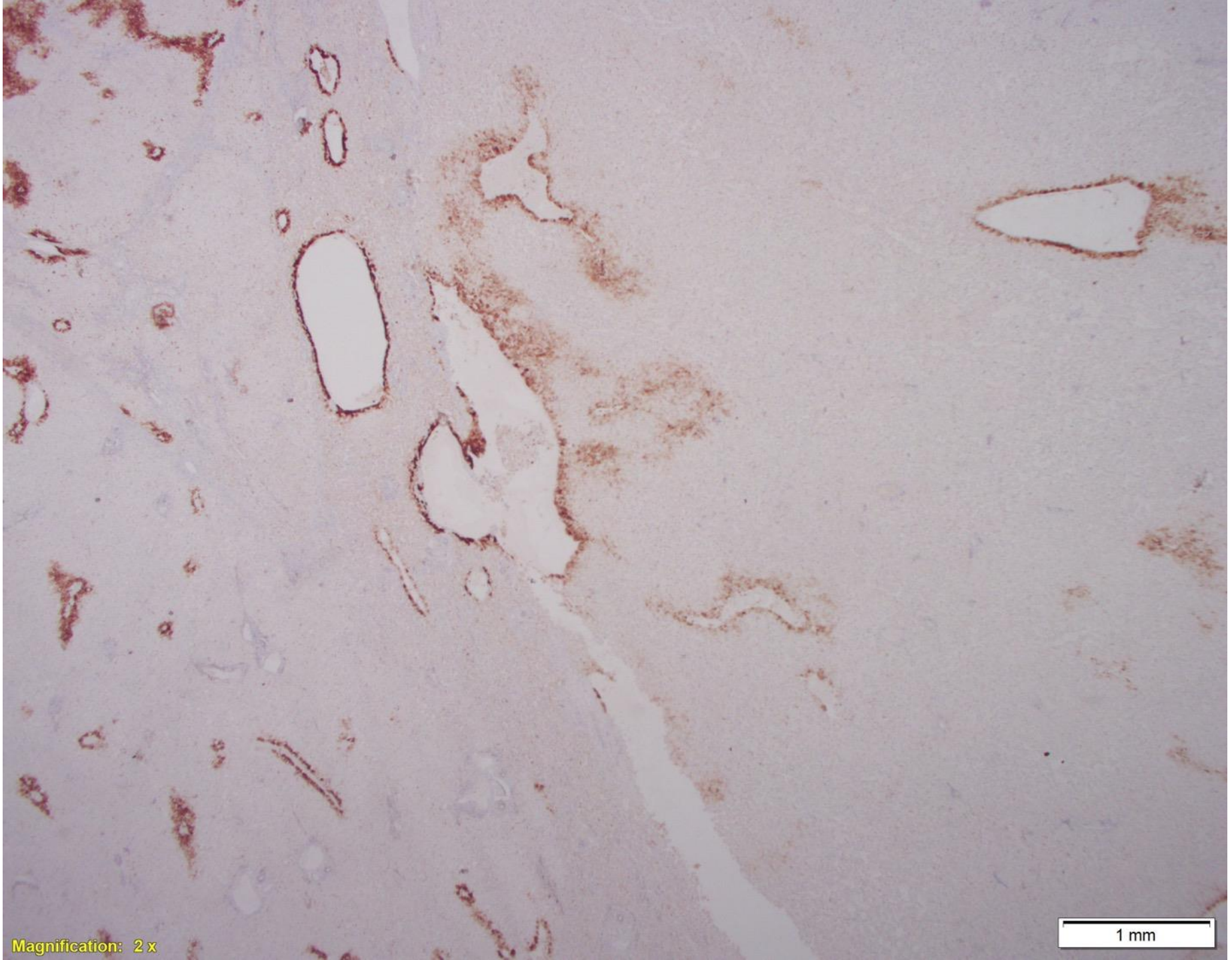
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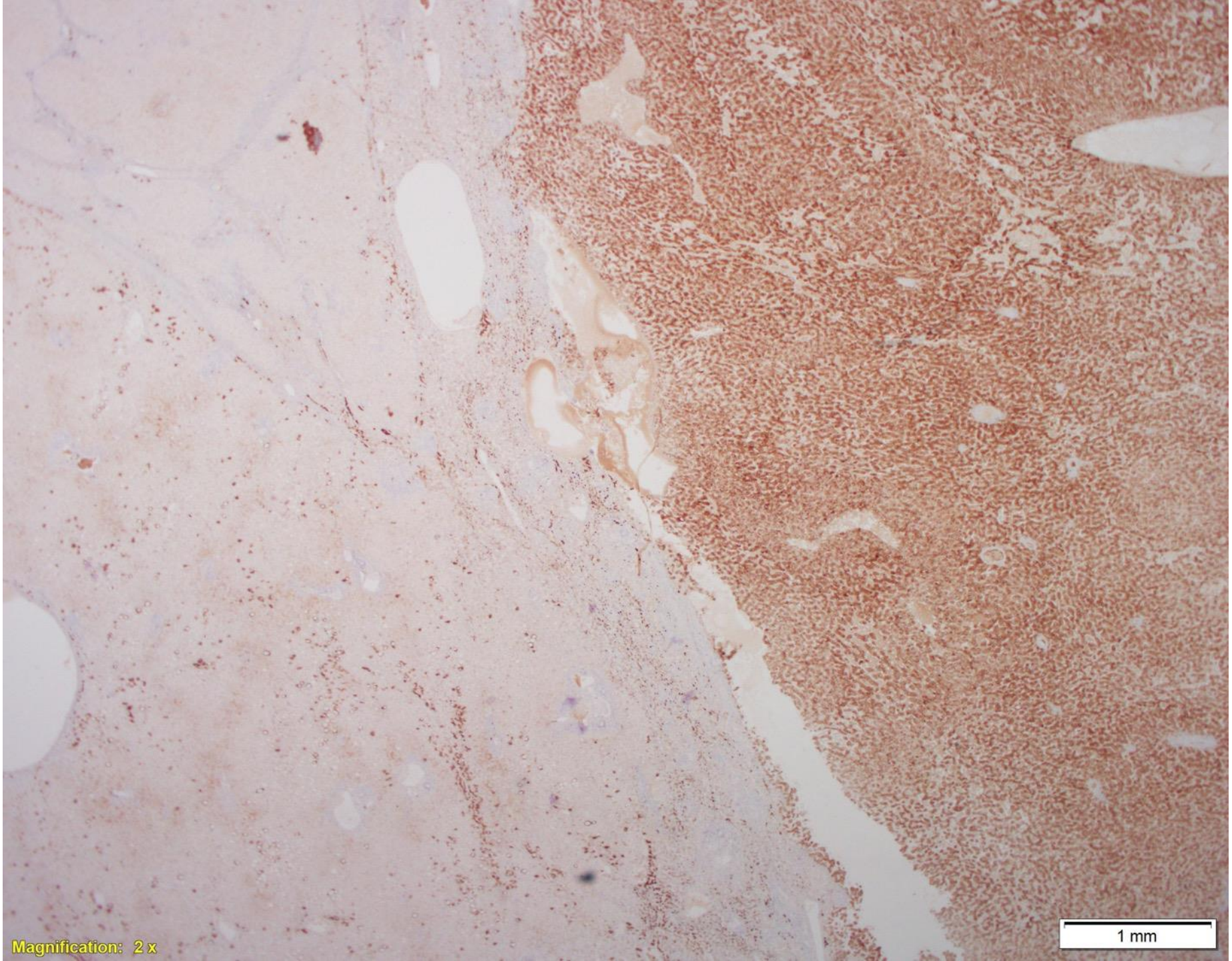
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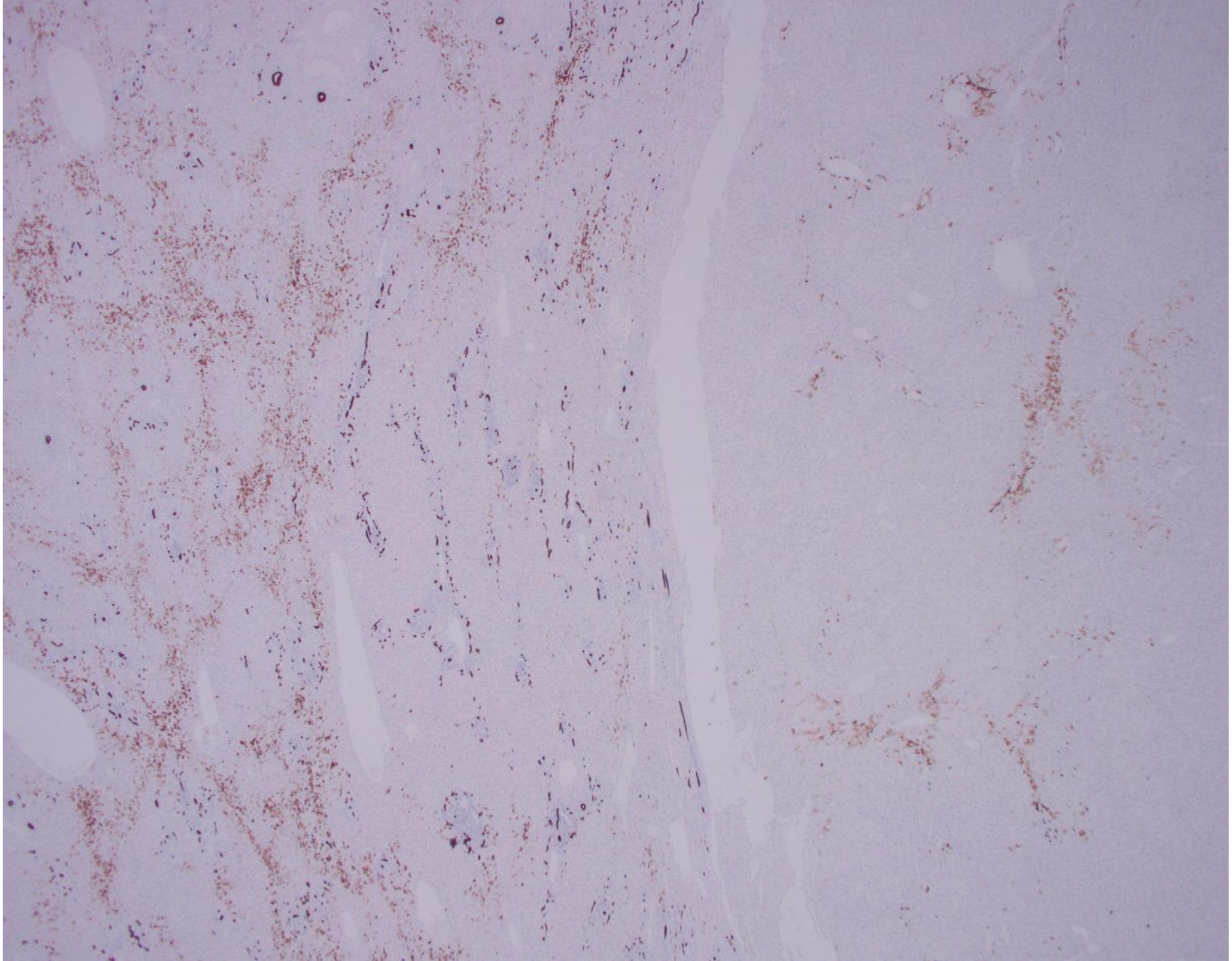

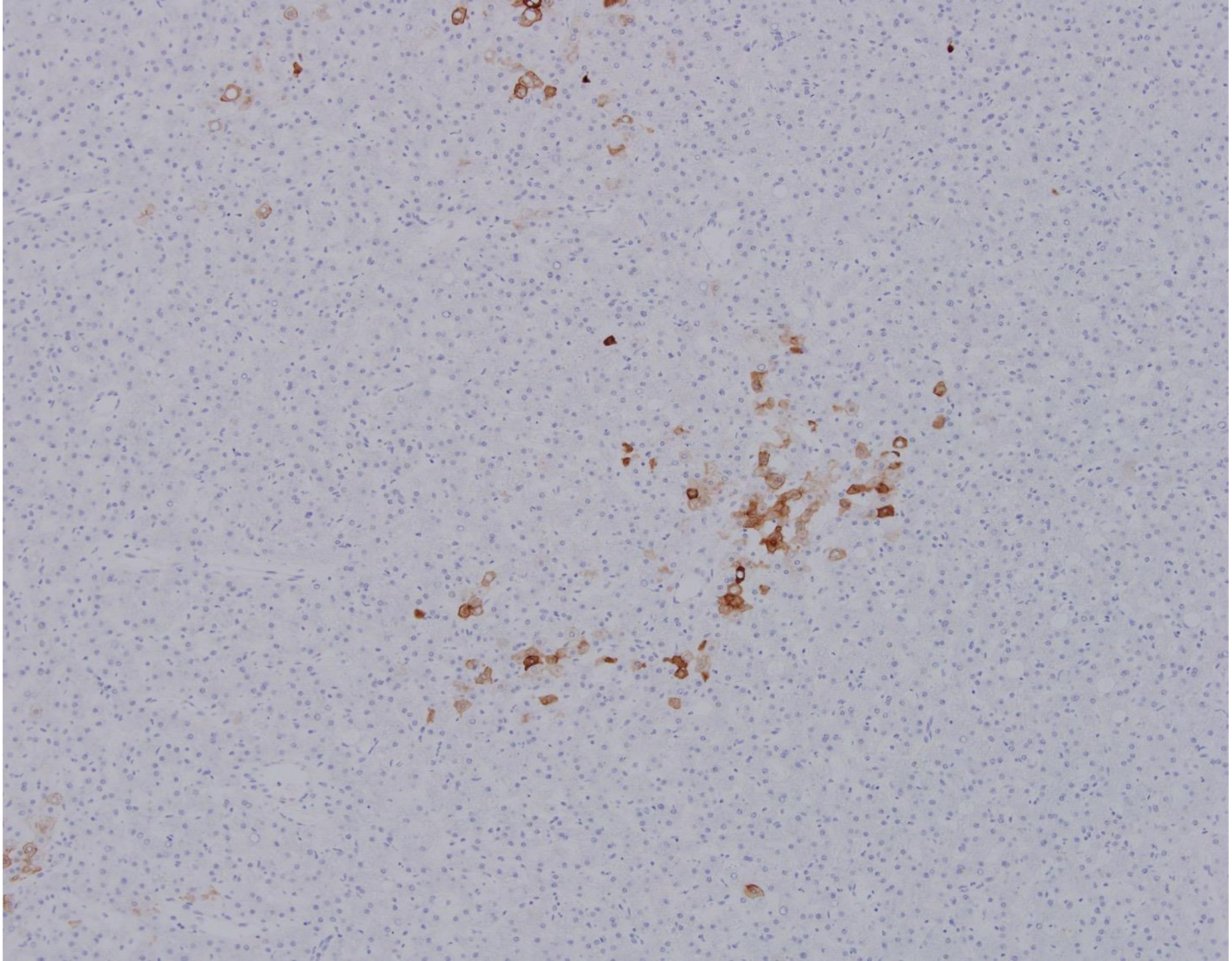

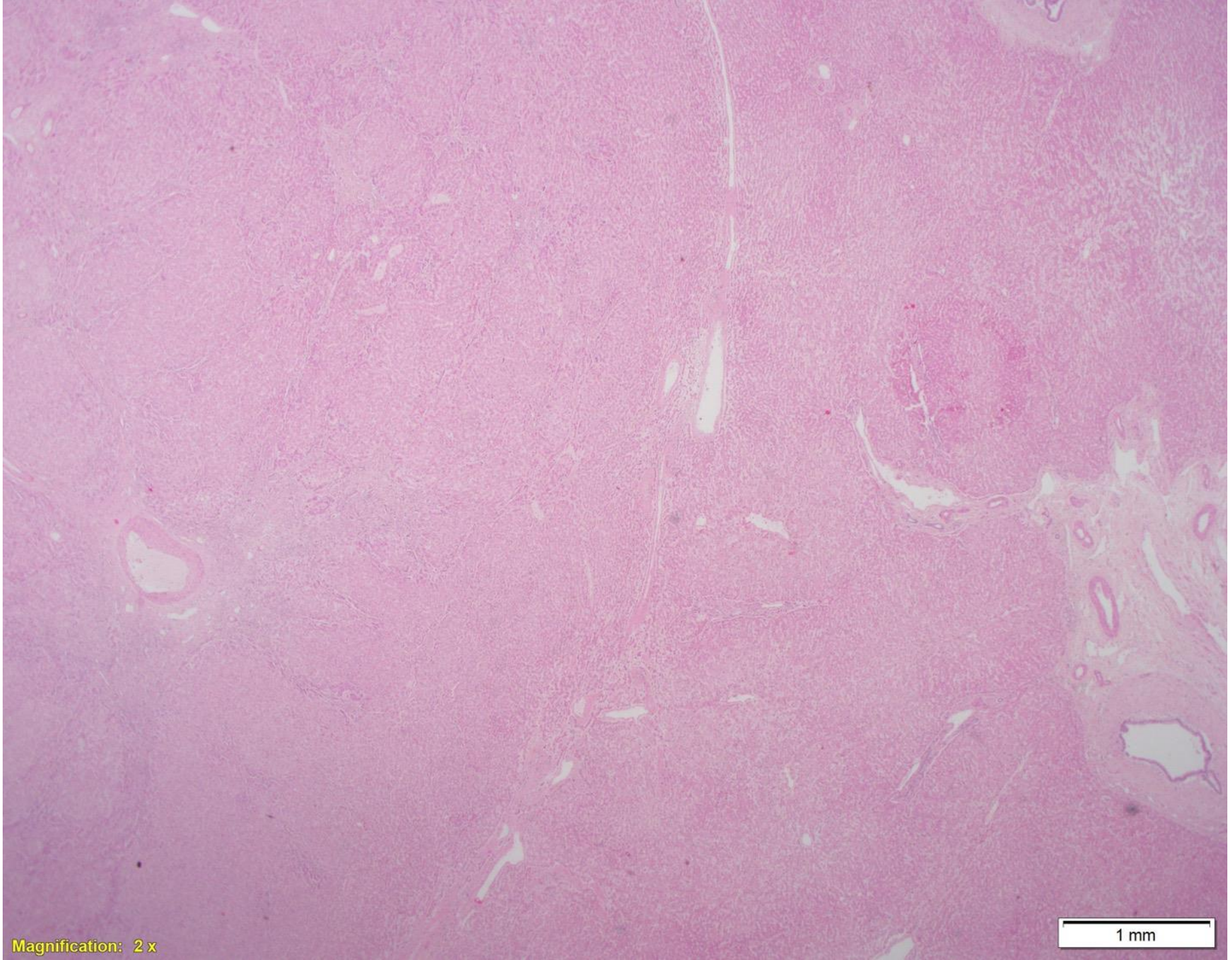
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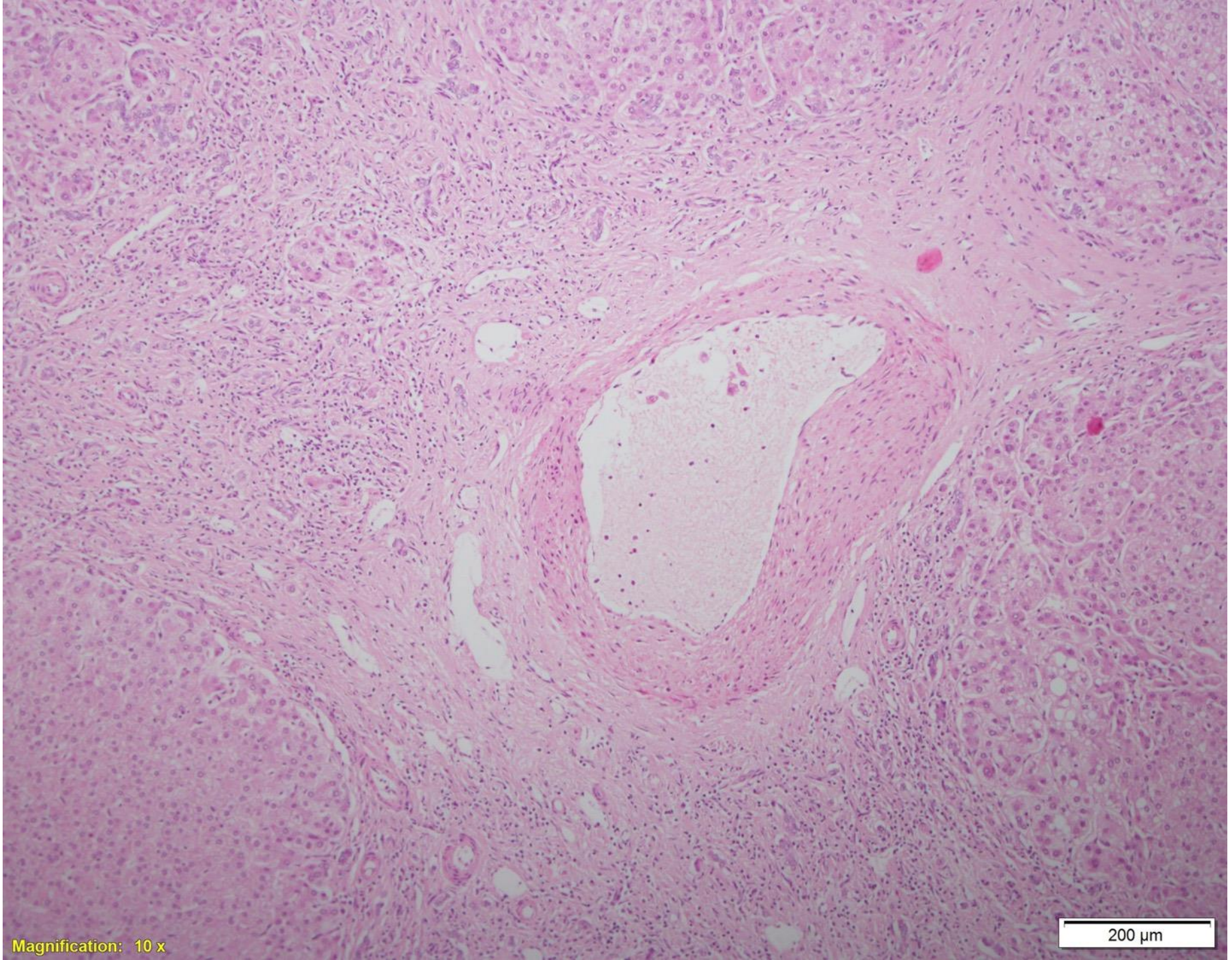
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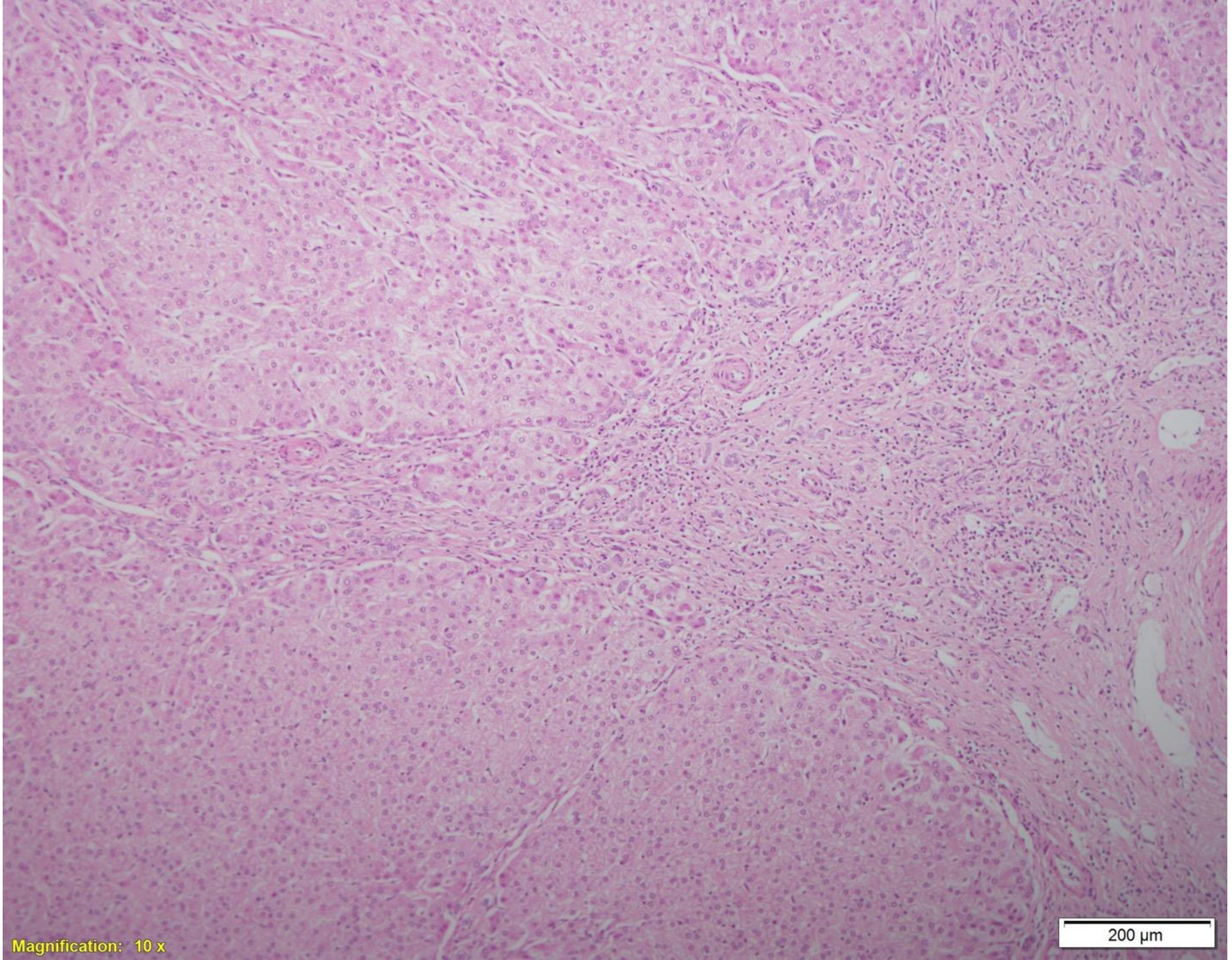
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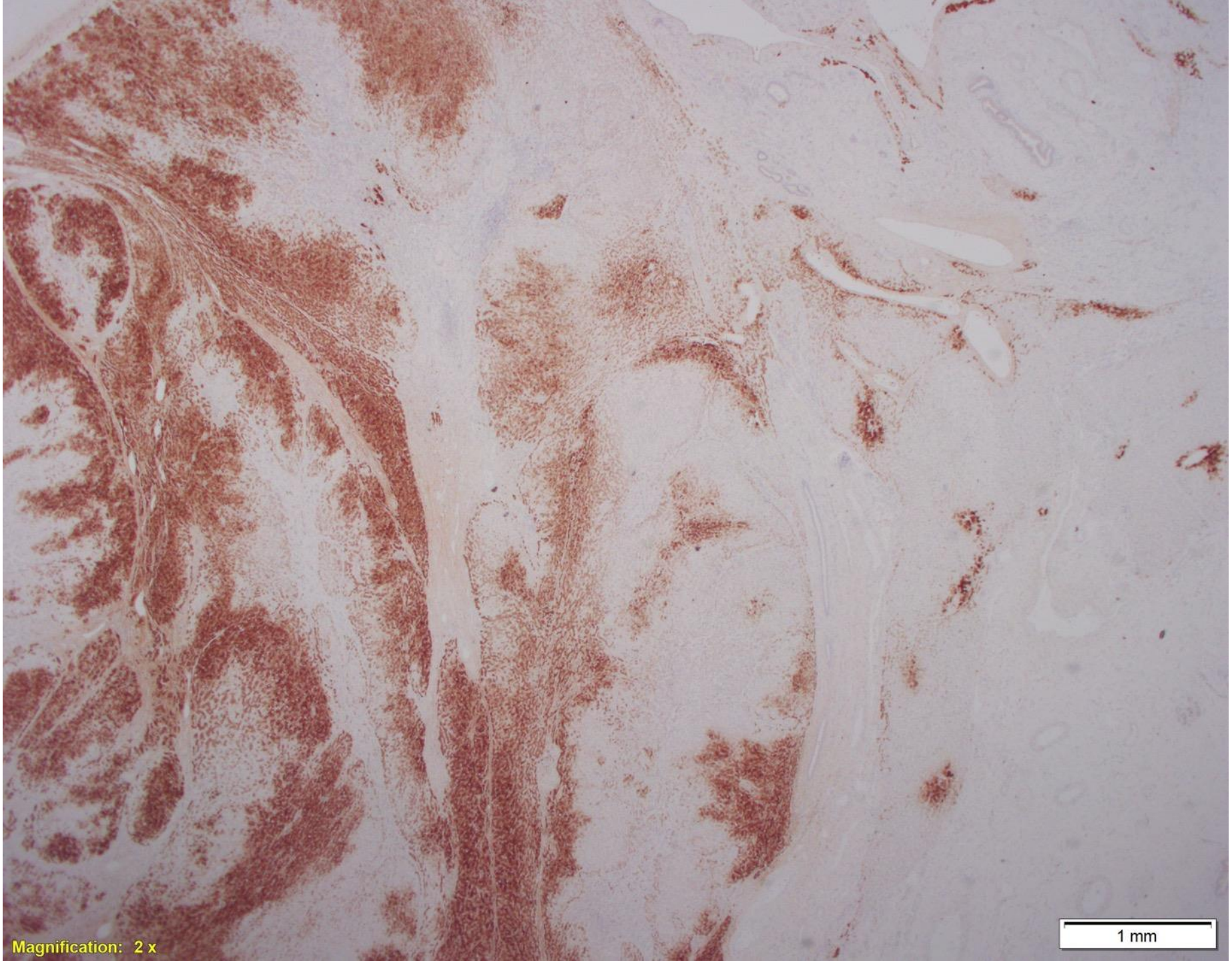
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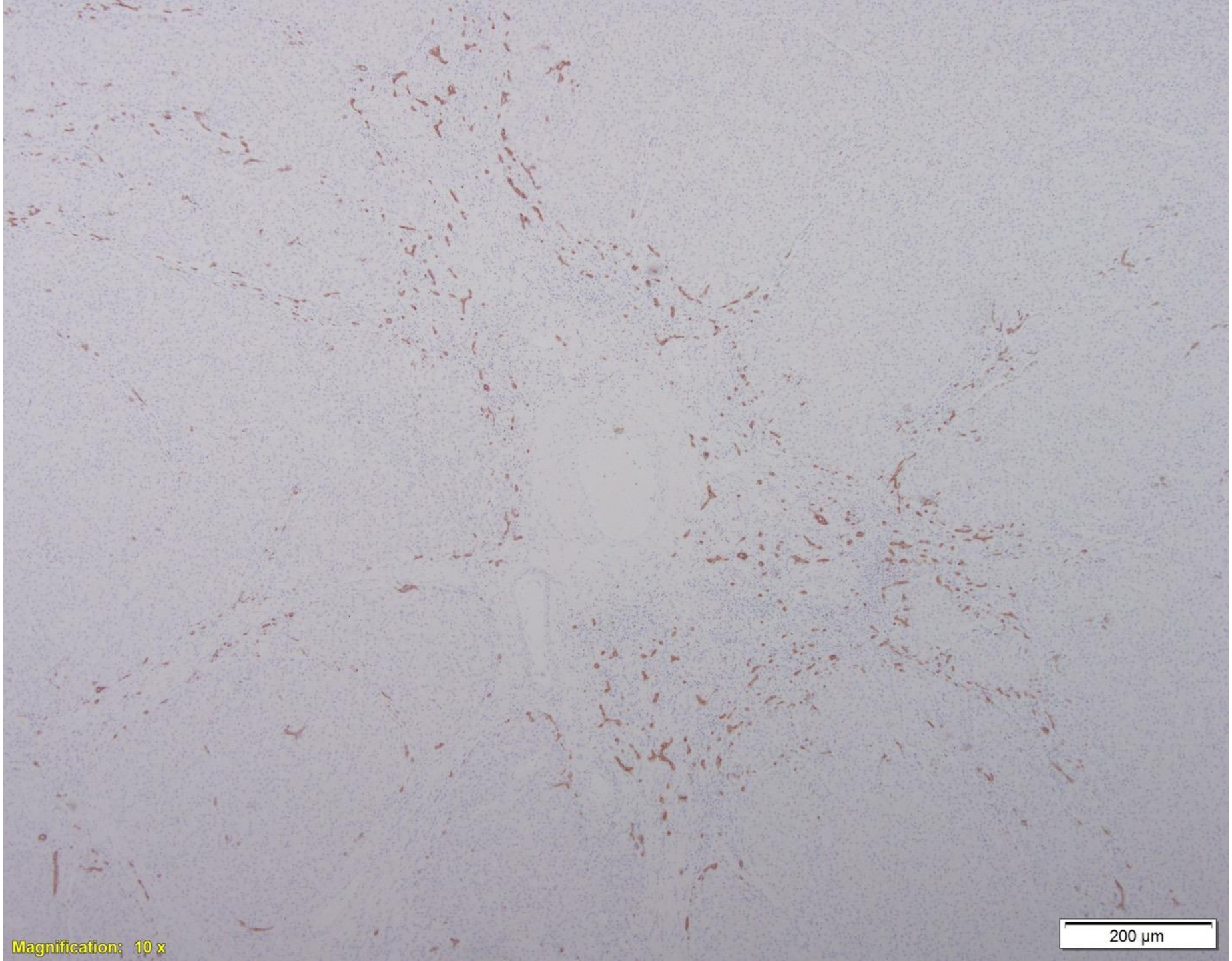
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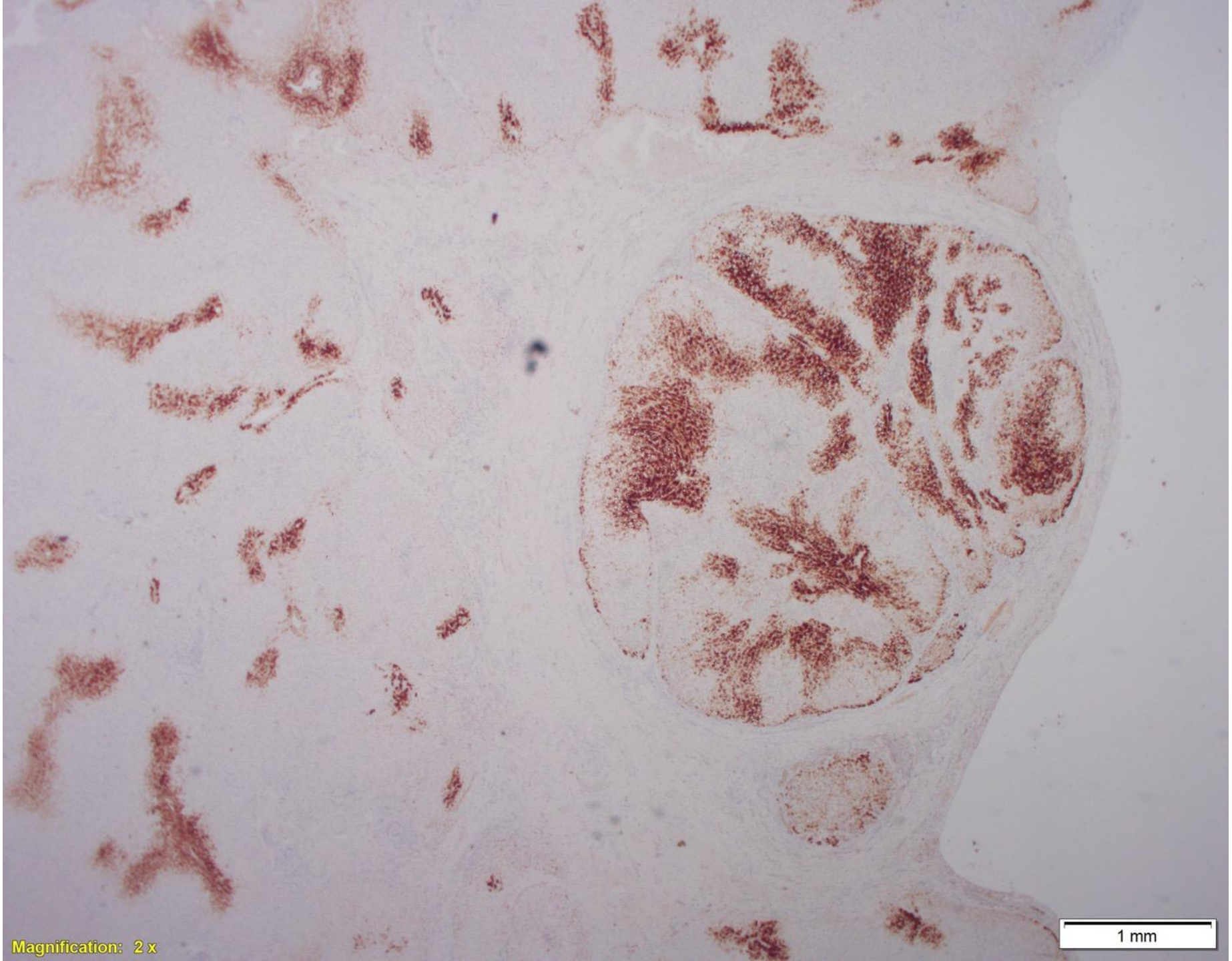
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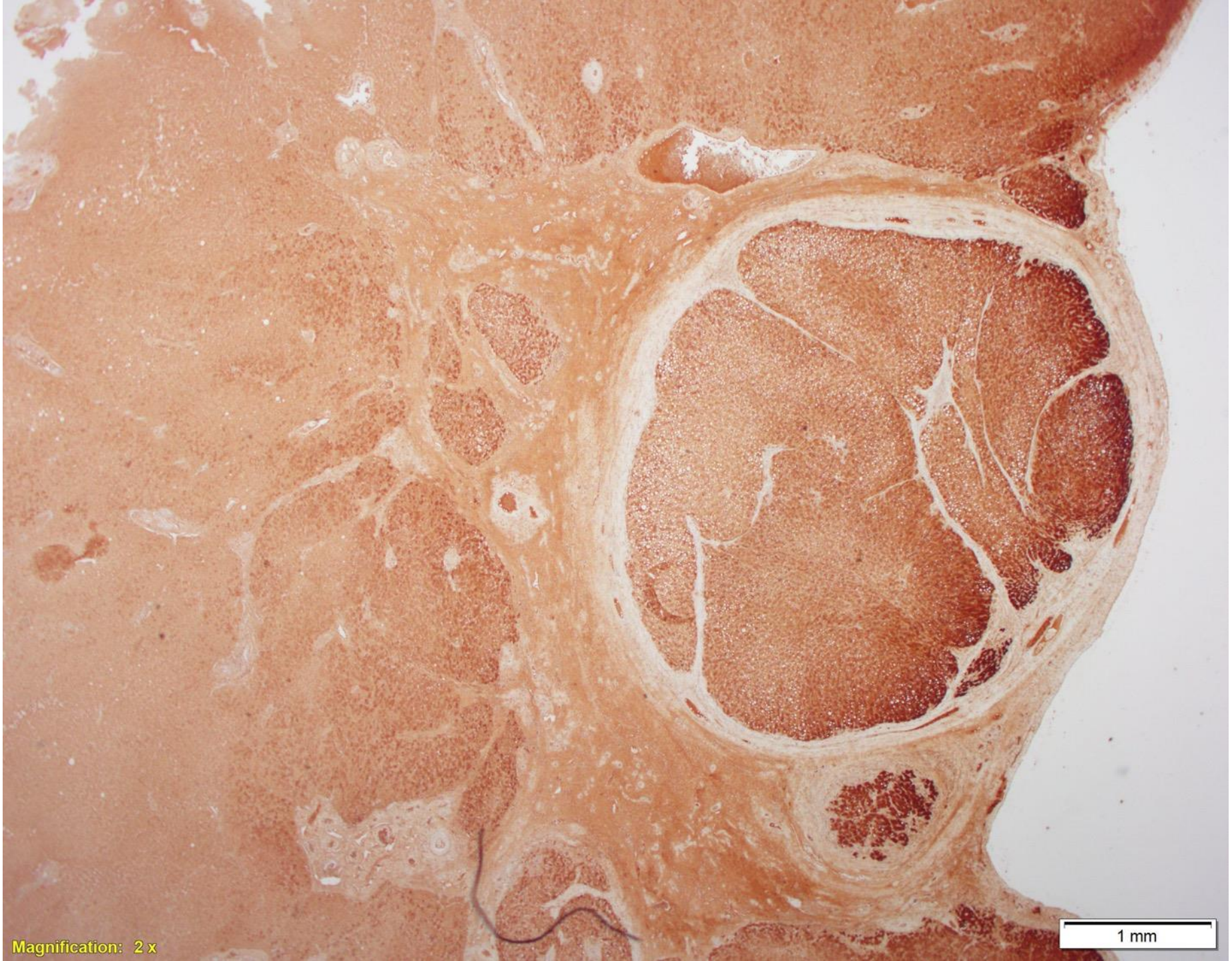
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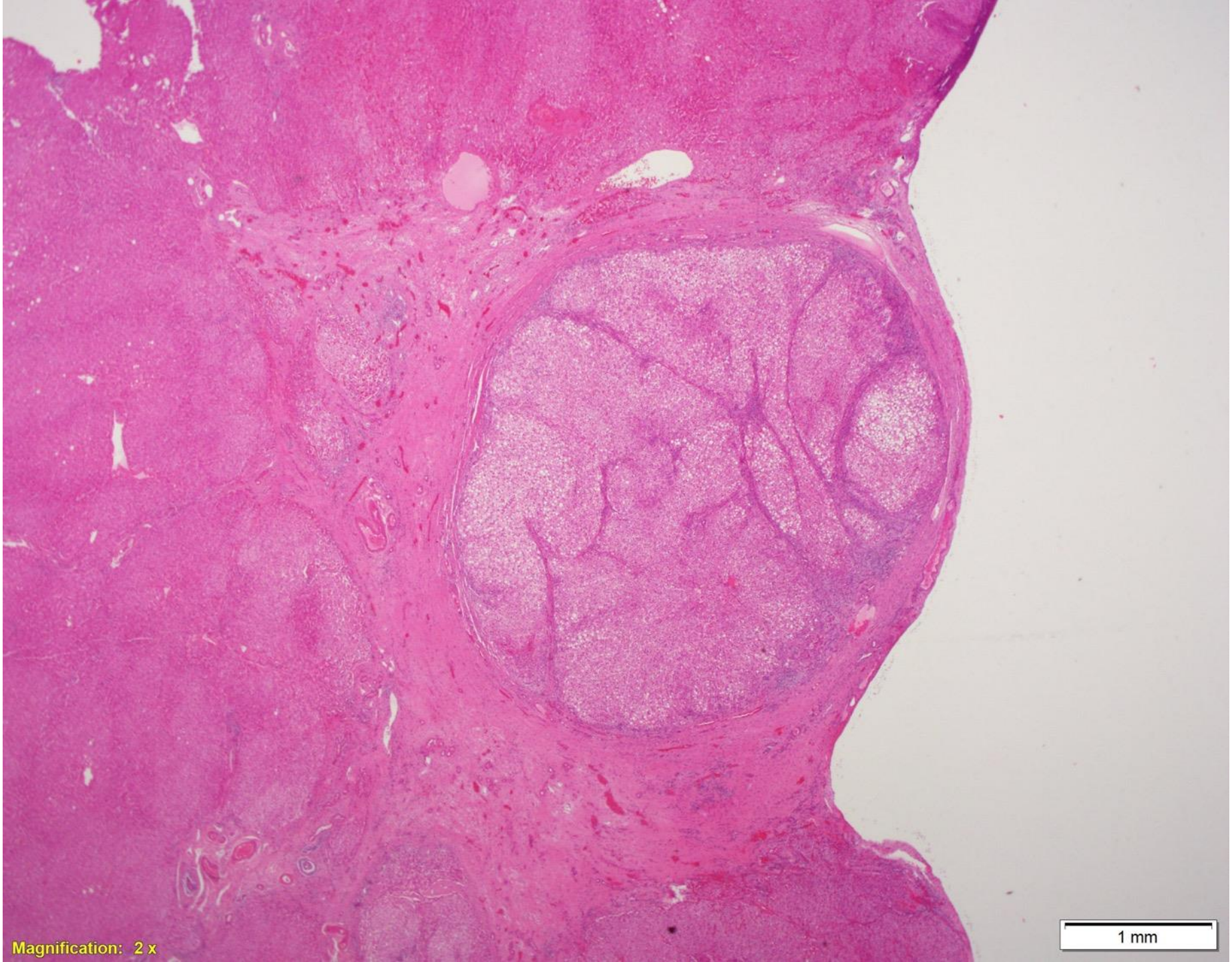
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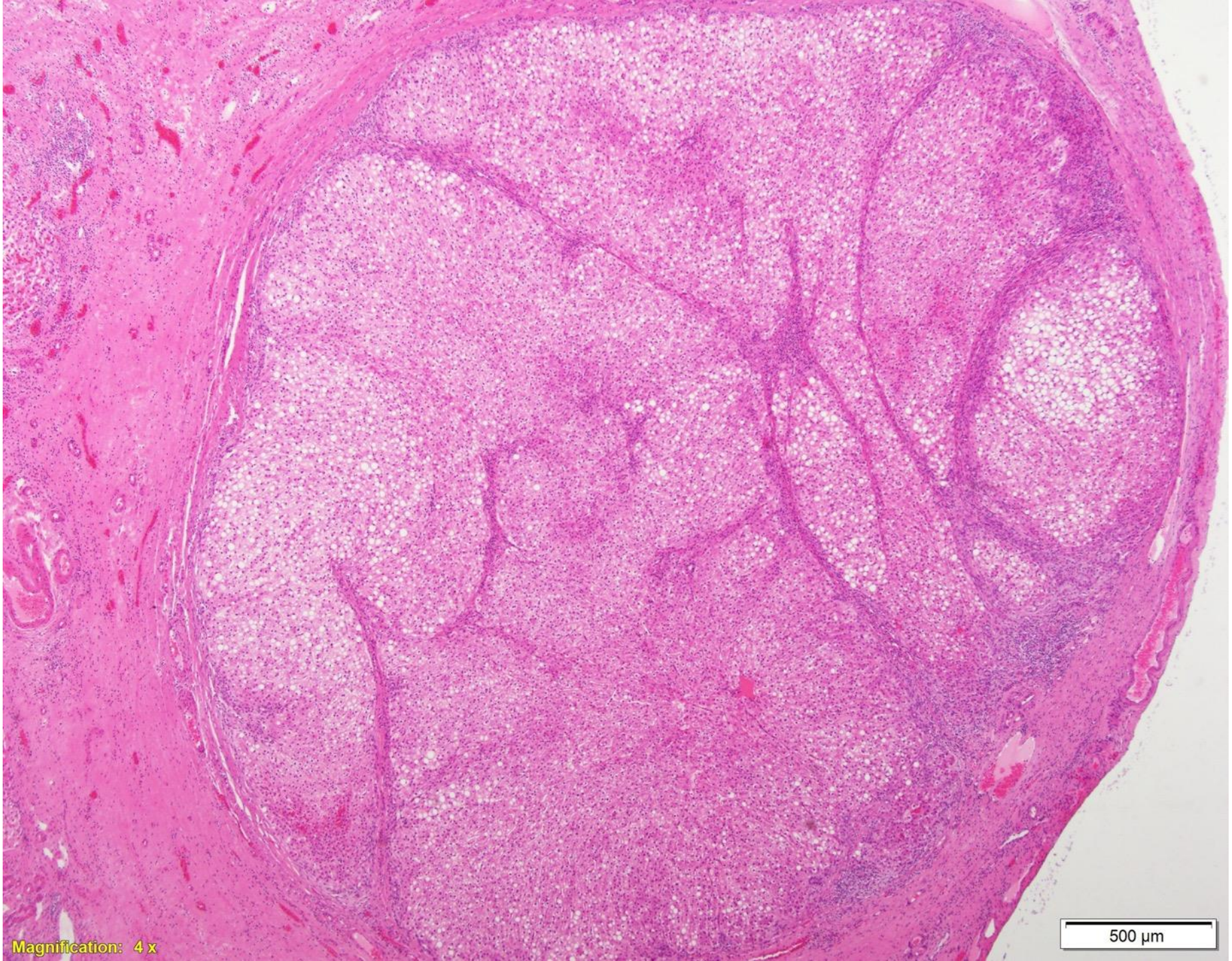
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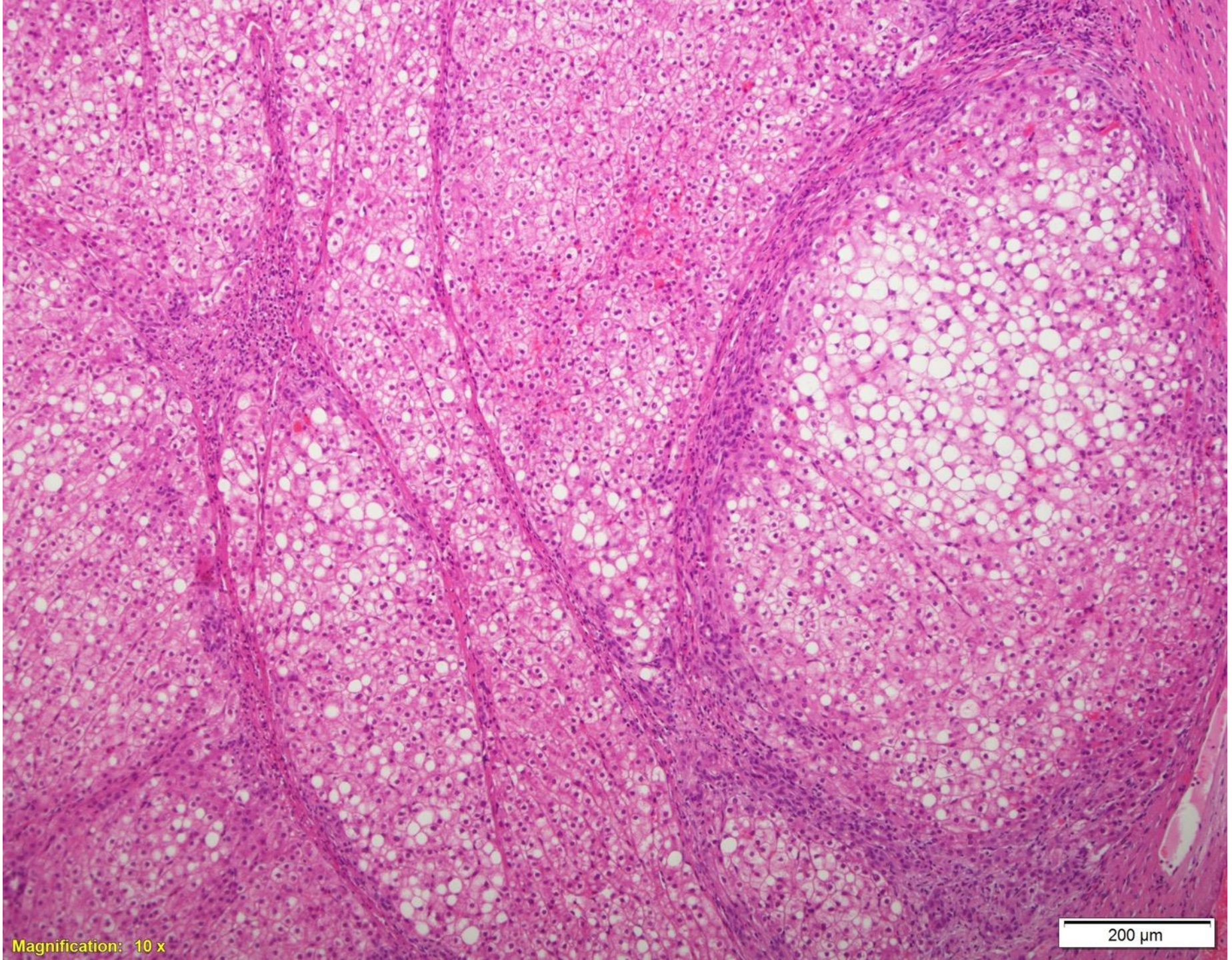
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HEPATOBILIARY-PANCREAS

MR findings of steatotic focal nodular hyperplasia and comparison with other fatty tumours

Maxime Ronot · Valérie Paradis · Rafael Duran · Anne Kerbaol · Marie-Pierre Vullierme · Jacques Belghiti · Dominique-Charles Valla · Valérie Vilgrain

The mean percentage of intralesional steatosis was 26 % (5–60 %). The mean percentage of intralesional steatosis in groups 1, 2 and 3 was 12.2 % (5–30 %), 48.7 % (10–60 %) and 27.4 % (5–60 %), respectively (P=0.001, 1 vs. 2 P=0.028; 2 vs. 3 P=0.023 and 1 vs. 3 P=0.0043). When looking only at steatotic FNHs in group 3, the mean percentage of intralesional steatosis was 25.4 % (5–50 %). It was not significantly different from other fatty tumours from group 3 (25.4 % vs. 28.9 %, P=0.85). There was no correlation between the percentage of intralesional steatosis and the size of the lesions.

The architecture of the adjacent liver was normal in all cases, with no fibrosis or necroinflammation. Liver steatosis was found in 20/41 (49 %) patients with a mean of 36 % (5–80 %) when present. Liver steatosis was not significantly more frequent in patients with steatotic FNHs compared to the other fatty tumours (11/21 patients, 52 % vs. 9/20 patients, 43 %, P=0.26). On the other hand, liver steatosis was significantly more pronounced in patients with non-FNH fatty tumours (48.9 % vs. 25.5 %, P=0.0002). Six out of the 10 patients (24 %) with ≥33 % of liver steatosis belonged to the control group and had telangiectatic/inflammatory HCAs.

The presence of steatosis within FNH assessed on histology is not uncommon. In the study reported by Hussain et al., up to 50 % of FNHs contained fat, including some FNHs with very mild steatosis [27]. Similarly, Ferlicot et al. reported up to 85 % of intralesional steatosis in a series of 27 atypical FNHs [22]. In our large series of FNHs that were resected, intralesional steatosis was found in 22 % of FNHs at pathology. Indeed, this prevalence is probably overestimated as the presence of fat is associated with atypical findings on MR imaging. For instance, most of our steatotic FNHs were typical on histology but half of them were atypical on MR imaging.

In our series, semiquantitative histological analysis showed that half of our patients with steatotic FNH had some degree of liver steatosis ranging from 5 % to 60 %. This relatively high prevalence compared to the general population confirms previous reports suggesting that liver steatosis is frequent in steatotic FNH [25, 26, 30]. Nevertheless, only four patients with steatotic FNH had significant steatosis ≥30 %. The concomitant presence of fat in the liver and FNH might be related to the same metabolism in FNH and the surrounding liver. Liver steatosis was also found in 43 % of patients with other fatty tumours. This may be explained by the fact that our series includes a large number of telangiectatic HCAs. It has been previously reported that these lesions are often associated with liver steatosis [31].

A quick survey at KCH

- 7 cases steatosis fnh and background
- 8 cases no steatosis in fnh or background
- 10 cases steatosis in background liver none in fnh
- 1 cases steatosis (focal) in fnh, none in background liver