Histopathology Interpretive EQA Schemes in the UK

and

UK National Liver Histopathology EQA Scheme

Judy Wyatt

December 2014

Outline:

- History of interpretive EQA schemes in the UK
  - Why did they start?
  - Governance
  - How do they work
  - Being a scheme organiser
- UK National Liver Histopathology EQA Scheme
  - How it's run
  - Examples of cases
- Future of interpretive EQA schemes

What makes a good doctor? – experience, aptitude, motivation

We want the satisfaction of being able to do a worthwhile job as well as we can.

Think of a time in the last few weeks when you made a 'good' diagnosis ……..

- What was it?
- Did you tell anyone else about it?
- Reflect on it – what does it tell you about yourself?

Think of a time when you made an error or had a ‘near miss’

- What was it?
- Did you tell anyone else about it?
- Reflect on it – what does it tell you about yourself?

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‘Bone Cancer staff escape action over mistakes’

The Independent Sept 13 1995

An independent inquiry report into the errors which occurred between 1985 and 1993 blames Dr Carol Starkie, a consultant pathologist for ‘unacceptably high level of misdiagoses in the Bone Tumour Centre at Birmingham’s Royal Orthopaedic Hospital’

‘Dr Starkie, 57, suffered from MS for many years, took early retirement shortly after the first misdiagnosis of a malignant tumour in a young boy came to light in May 1993’.

Misdiagnosis and mistreatment of 79 patients.

Recognised in 1989

1990 - surgeons gathered cases where problems had occurred

Hospital managers ignored concerns raised informally by surgeons

Report in 1995; no disciplinary action against any doctor or manager – ‘most have moved on’

2-year inquiry chaired by Dr Archie Malcolm

'A damning indictment of the mismanagement and poor communication which compounded the errors made by Dr Starkie'.

The hospital 'deeply regretted the distress caused to patients and relatives in the last 8 years'.

'We have learned from the past and have changed our management and clinical practices accordingly'

We believe this ensures the diagnosis we now employ represent the very best practice available today'

Not just the Birmingham Royal Orthopaedic Hospital……

Contributory factors

Contributary factors

'Some of the errors that were made would be unacceptable for a non-expert pathologist' *to err is human*

- Difficult area in histopathology, but with final gold standard (in patients with missed malignancy)
- Isolated pathologist, different hospital, few opportunities to discuss cases with clinicians
- Renowned specialist, confident, tends to be believed
- Opinion mis-interpreted as certainty = communication
- Cases from other hospitals for opinion
  - dispersed, no follow up information, don't know you're wrong
- Chronic illness
- Lots of people were concerned, but no-one was responsible for passing on concerns

How to stop this happening again?

- Don't be isolated – no single handed pathologists,
  if in doubt, discuss with a colleague
- Don't be misunderstood - ensure good communication
  multidisciplinary review – MDTMs
- Don't get out of date - CPD
  guidelines
- Know your limits - compare yourself with your peers
  Participation in EQA schemes
- Don't be an ostrich - Culture of individual responsibility
  to voice concerns over competence of colleagues

Having professional responsibility
– used to be assumed, now regulated

Clinical governance is

"a framework through which NHS organisations are accountable for continuously improving the quality of their services and safeguarding high standards of care by creating an environment in which excellence in clinical care will flourish"

Source: A First Class Service – Quality in the new NHS, Department of Health, 1998

Does Clinical Governance Work? Yes!.....

So long as it.....

- **a. becomes an active pursuit driven by, and focused on, quality**
- **b. recognises the complex nature of health care systems**
- **c. is not strangled by paper and procedure**

EQA schemes in laboratory medicine

- UK NEQAS (UK National External Quality Assessment Service)
  - 'helping to ensure clinical laboratory test results are accurate, reliable, and comparable wherever they are produced' = independent organisation for EQA schemes
- National Quality Assurance Advisory Panels (NQAAP) = professional groups which have executive responsibility for maintaining satisfactory standards of analytical and interpretative work in laboratories in the UK
- Chair person of NQAAP reports to the Joint Working Group on Quality Assurance (JWG)

NEQAS schemes have existed for a long time in other pathology disciplines, why not in histopathology? – *interpretive result*

- Numerical result, define poor performance as
  outside normal distribution of results (e.g. biochemistry)
- Categorical result, with specific right answer,
  no overlapping answers (e.g. microbiology)
- Subjective assessment of histology slides
  QA in histopathology and immunocytochemistry

Meanwhile, histopathologists had slide clubs

How can you apply EQA to an interpretative response?

Difficulties

- Which cases? - represent routine workload

  Generalist, specialist

- Who's right? – 80% consensus for correct diagnosis, otherwise excluded

- Marking responses – either tick box

  (e.g. breast, bowel cancer screening),

  or an interpretative element by organiser – assign text diagnoses into mutually exclusive diagnostic categories

- Persistent poor performers –

  lowest 2.5% - ? chance, or a poor pathologist

  'dangerous diagnosis' – not retained in most schemes

- Governance – can you trust the organiser with your career?

List of schemes (from RCPath)

Specialist schemes

- Lung
- Neuropathology
- Gynaecological
- Cervical cytology
- Musculoskeletal
- Breast
- Renal
- Urological
- Prostatic biopsies
- Paediatric
- Gastrointestinal
- Bowel cancer screening
- Renal transplant
- Dermatopathology
- Ophthalmic
- Head & Neck & oral
- liver

General EQA schemes

- Northern
- West midlands
- Yorkshire
- Wessex and southwest
- East anglia
- Wales
- North west non-gynae
- Thames valley
- South thames (east)
- North west
- Scotland & Ireland
- East midlands
- South Thames (west)
- North thames (west)

RCPath and interpretive EQA schemes

NQAAP (National Quality Assurance Advisory Panel)

responsible for overseeing and advising schemes, and managing poor performance.

Annual report to JWG (Joint Working Group on Quality Assurance)

Scheme organisers

provide a structured annual report

SOPs, quality management, +/- accreditation

meet once a year at RCPath

History of liver EQA scheme

- 1994 Started, 23 members,
  - Prof Burt Newcastle
  - Furness system for assessing performance – adapted to liver
- 1999 SOPs written and approval by steering committee of RCPath and NQAAP
- 2002 CPA accreditation – re-assessed every 2 years
- 2004 organiser changed to JIW, 56 members
  - New deputy = Prof Hubscher, secretary = Anne Lee
- 2006 75 members, RCpath + virtualpathology websites
- 2006-7 CPA approval due, not pursued.

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- 2006 75 members, RCpath + virtualpathology websites
- 2006-7 CPA approval due, not pursued.
- 2014 100 members. Annual meeting.
- On line submission of responses.
- 'Masterclass' presentation on problematic cases

Membership of Liver EQA Scheme

Any pathologist (UK and Ireland) with an interest in liver pathology.

- Pathologists working in hepatology centres including the 7 transplant centres
- In other departments reporting liver biopsies, there should be a recognised ‘lead’ pathologist for liver, identified in their job plan, who is a liver EQA participant.

(RCPath Tissue Pathways for Medical Liver Biopsies, May 2014)

Why be a member? Carrot and stick

You have to

Demonstrate
professional competence

CPA , appraisal, revalidation

You want to

See and discuss interesting cases,
Know you're doing OK compared with peers
Keep up to date
Challenge of test and results

Same reasons as slide clubs

Why organise it?

Someone has to
(get paid)

You want to

See and discuss interesting case,
Know you're doing OK compared with peers
Keep up to date

Enjoy teaching – make the most of experience
Provide useful CPD material
Worthwhile – contributes to good standards

What happens in a circulation?

Members register, are given a secret participants' number, and are put in 'cells'

- 17 cells of 5-6,
- Pay annual fee

2 circulations per year, 12 slides

- Members choose and submit cases – 20 copies H&E,
- +/- special stains – scanned, on website
- original clinical information
- Organiser selects cases - $\leq$ 4 resection, all 20 H&E similar
- Secretary prepares response sheets and boxes of slides and timetable for circulation
- Organiser arranges open meeting to discuss results

- Sends slides to first pathologist in cell

Contd.

- Pathologists view slides, send on to next in cell, and send responses via ‘surveymonkey’
- Slides also available for view on virtualpathology website
- After completion date, secretary downloads spreadsheet of responses, checks and deletes names *Responses are anonymous and only the secretary has the identifier key.*
- Organiser puts into diagnostic categories, and prepares presentation with clinical information, representative photos, and list of responses for open meeting.

At the meeting:

- Quorate number of participants discuss responses, and agree what is accepted as correct for scoring.
  - Some cases may not be suitable for scoring.
- Also discuss operation of the scheme and any changes to SOPs
  - Continually evolving.

After the meeting

- Organiser checks responses against agreed marking scheme, and sends scores to secretary.
- Lowest 2.5% scores (= lowest 2 if 80 participants)
  - Were they also in bottom 2.5% in either of 2 previous circulations?
    = persistant poor performer.
- Secretary sends individual scores to participants, with CPD certificate.
- Required for appraisal, revalidation and laboratory accreditation.
- Organiser completes powerpoint presentation and puts it on the internet.

IMPORTANT
The only person with access to participant number / name is the secretary

Each pathologist

- CPD points and certificate,
  necessary for annual CPD return, appraisal and revalidation
- Department – for CPA accreditation consultants must participate in an EQA scheme appropriate to their practice.

- Results are confidential to participants.
- Scheme organiser can confirm membership but not results

- From 2015 – appraisal will include a discussion of EQA results and propose a way to improve any problem areas in the Personal Development Plan

How is it funded?

Members' subscriptions

- Pay for cost of materials and postage
- Pay costs of open meetings
- Pay for secretarial support
- +/- Pay for organiser's time
- Pay for NQAAP and CPA annual fees
- Pay for quality management
  - if CPA accredited

Concern that future requirements for accreditation may make interpretive schemes un-affordable

EQA schemes as tool to improve quality

- Breast EQA scheme – essential as part of national screening programme
- Improves consistency of diagnosis, not expertise of experts
- Tick box format, so categorical results which can be analysed

Does performance in scheme improve?

Impact of a national external quality assessment scheme for breast pathology in the UK

Ellis IO, Coleman D, Wells C et al. JCP 2006;59;138-145

Individual diagnostic categories: 4 possible results

- Naturally has a high level of agreement
  (insitu and invasive carcinoma and uncomplicated benign lesion)

- Lesser agreement which can be improved by better definition of criteria
  (grading of invasive cancer)

- Lesser agreement which can be improved only by changing the system of classification
  (DCIS 6 growth patterns, 3 grades, 2 grades)

- Lesser agreement which cannot be improved
  (atypical hyperplasia, vascular invasion)

The NHS breast screening programme EQA: experience in recent years relating to issues involved in individual performance appraisal

- New members show improvement over first 3 circulations
- Members who leave tend to have lower scores
- Score is not dependent on number of cases seen in routine practice (50 -1000) or for pathologists in breast screening centres v. others
- Little difference between organising committee and members.

Parham DM et al; J Clin Pathol 2006;59;130-137

Report on circulation J b13a of a national bowel cancer screening programme EQA scheme

Version 1 of this document

1. Analysis of cases

12 SHA lead pathologists took part

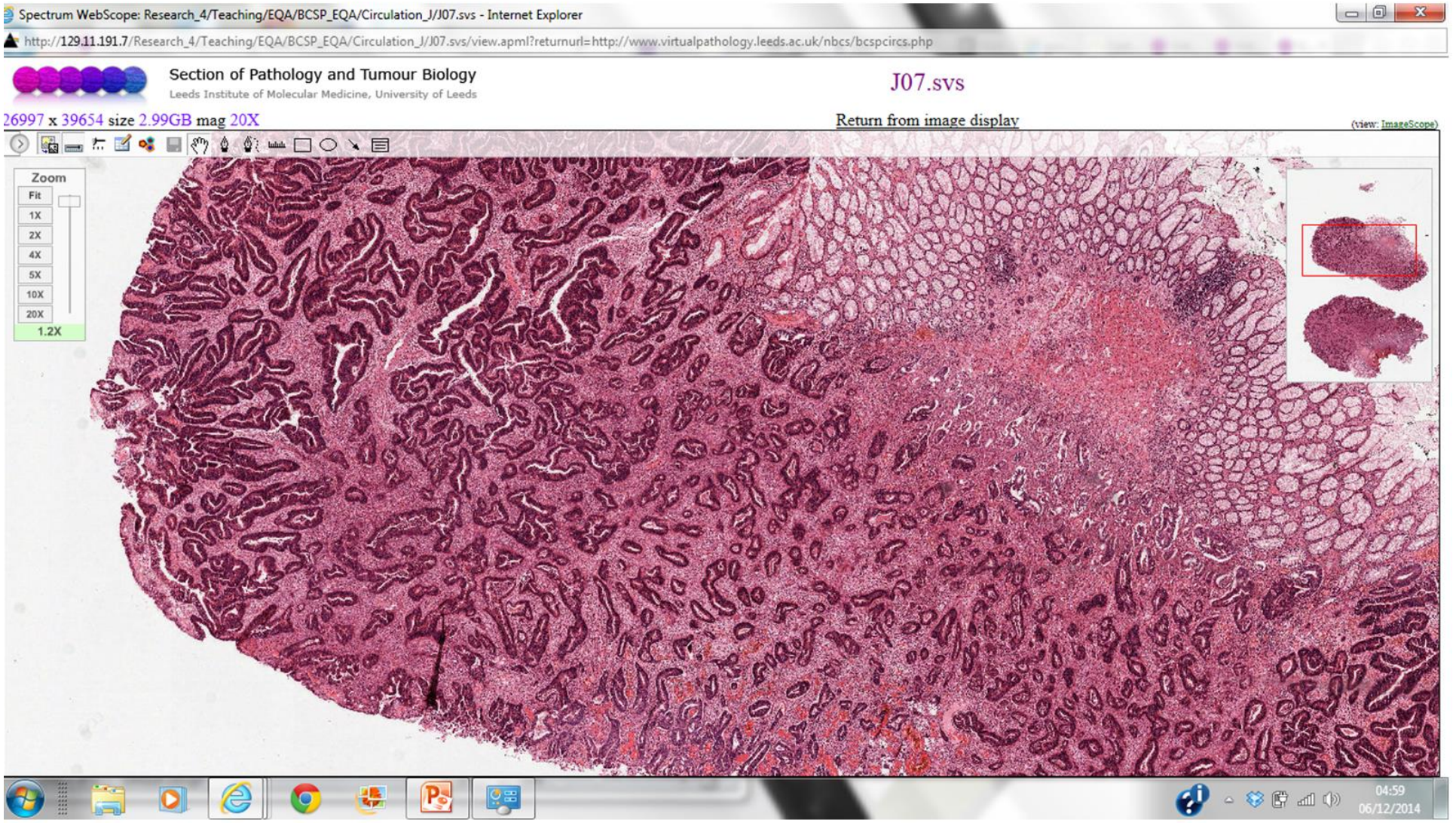
430 BCSP pathologists submitted responses that were scored

A cutoff of 80% agreement was required to make consensus - effectively 10 SHA leads had to agree on the diagnosis for a case to be used in scoring.

Example:

Case J07

	other	low grade dysplasia	high grade dysplasia	malignant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Leeds Institute of Molecular Medicine, University of Leeds
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06/12/2014

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1. Analysis of cases

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Example:

Case J07

	other	low grade dysplasia	high grade dysplasia	malignant
SHA leads	0	0	0	12
participants	0	2	10	418

SHA leads consensus diagnosis: **malignant**

Overall for this circulation:

maximum individual score 20
actual scores compiled - numbers of participants and SHA leads scoring:

	20	19	18	17	16	15	14
SHA leads	9	2	0	0	1	0	0
participants	267	111	35	13	4	0	0

2.5% of 442 participants is 11, so the 5 participants who scored 16 are identified as poor performers in this round

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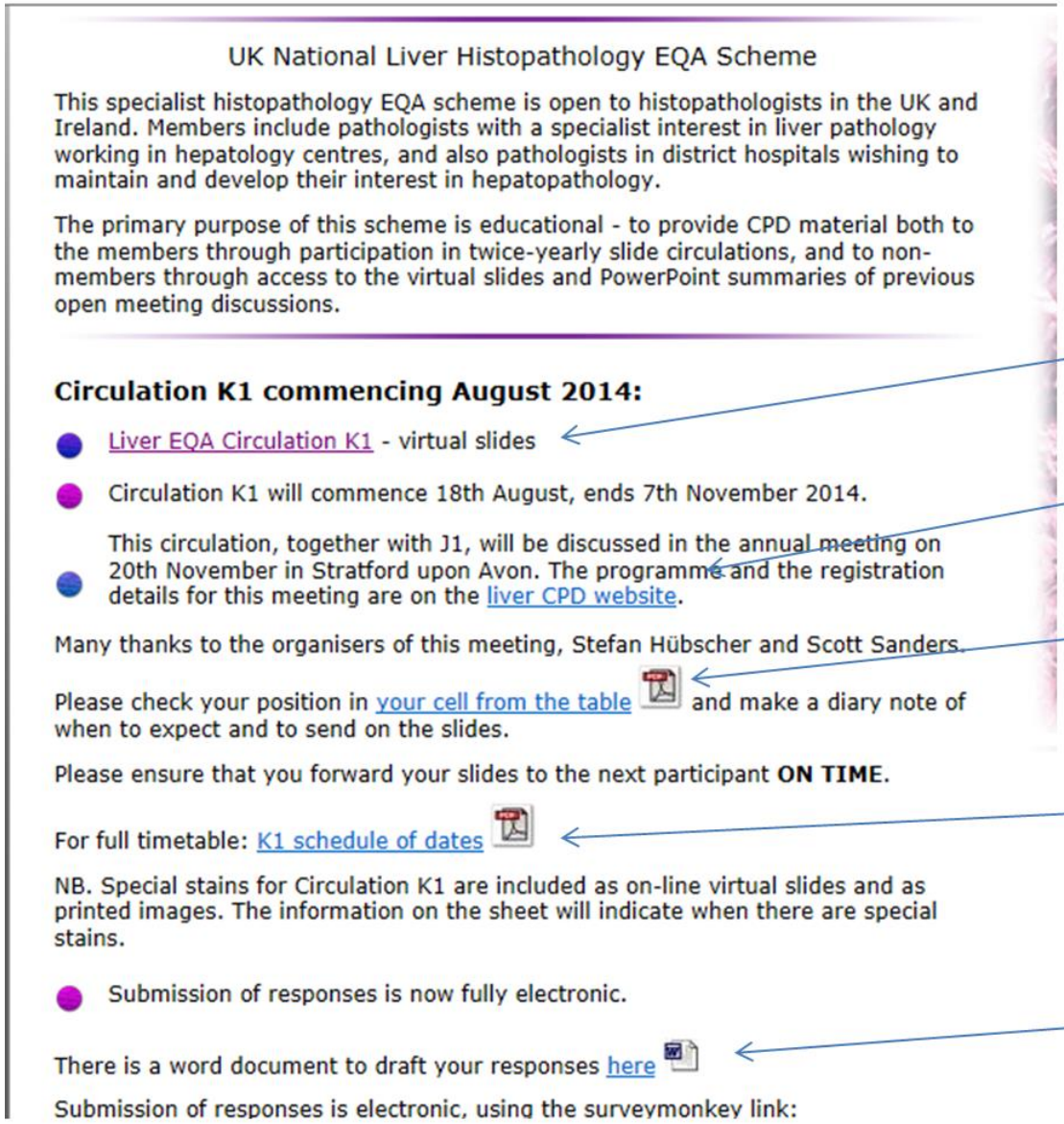
  - Examples of cases

- Future of interpretive EQA schemes

Using the website to run the EQA scheme



UK National Liver Histopathology EQA Scheme

This specialist histopathology EQA scheme is open to histopathologists in the UK and Ireland. Members include pathologists with a specialist interest in liver pathology working in hepatology centres, and also pathologists in district hospitals wishing to maintain and develop their interest in hepatopathology.

The primary purpose of this scheme is educational - to provide CPD material both to the members through participation in twice-yearly slide circulations, and to non-members through access to the virtual slides and PowerPoint summaries of previous open meeting discussions.

Circulation K1 commencing August 2014:

- Liver EQA Circulation K1 - virtual slides
- Circulation K1 will commence 18th August, ends 7th November 2014.
- This circulation, together with J1, will be discussed in the annual meeting on 20th November in Stratford upon Avon. The programme and the registration details for this meeting are on the liver CPD website.

Many thanks to the organisers of this meeting, Stefan Hübscher and Scott Sanders.

Please check your position in your cell from the table and make a diary note of when to expect and to send on the slides.

Please ensure that you forward your slides to the next participant **ON TIME**.

For full timetable: K1 schedule of dates

NB. Special stains for Circulation K1 are included as on-line virtual slides and as printed images. The information on the sheet will indicate when there are special stains.

- Submission of responses is now fully electronic.

There is a word document to draft your responses here

Submission of responses is electronic, using the surveymonkey link:

1. Virtual slides
2. Details of meeting
3. Participants in ‘cells’
4 Date to receive and send on box of slides
5. Link to surveymonkey to collect responses

1. Virtual slides

Case number 447

Male 53 years

Acute jaundice. Deranged LFT's ? Autoimmune hepatitis. 3 liver cores largest 21mm.

H&E

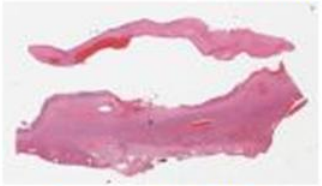
Open with WebScope
Open with ImageScope

Case number 448

Female 44 years

Hepatic cyst and gallbladder; obstructive jaundice. Collapsed cystic structure 8.3 x 7.8 x 4.7cm and weighing 93g. Dark brown smooth surface with focal roughened, haemorrhagic and pale areas.

H&E



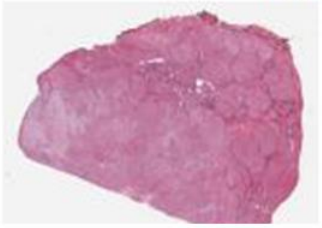
Open with WebScope
Open with ImageScope

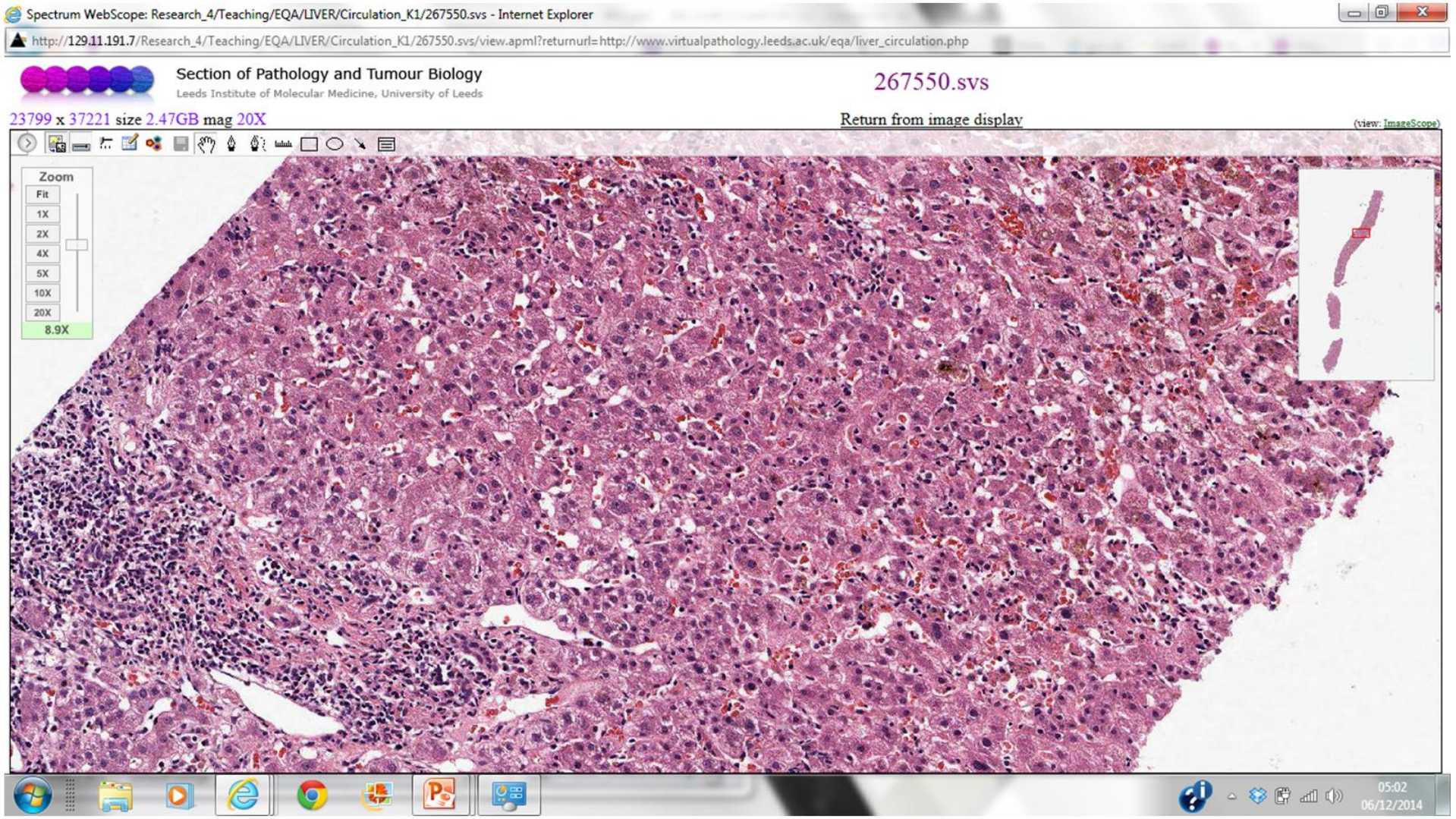
Case number 449

Male 73 years

Cirrhotic liver ? Hepatoma segment 8. segment 8 lesion. Liver 15.5g 4.2x4x3cm slicing reveals a cream tumour measuring 2.6 x 2.7 x 2.5cm. The background liver appears cirrhotic.

H&E

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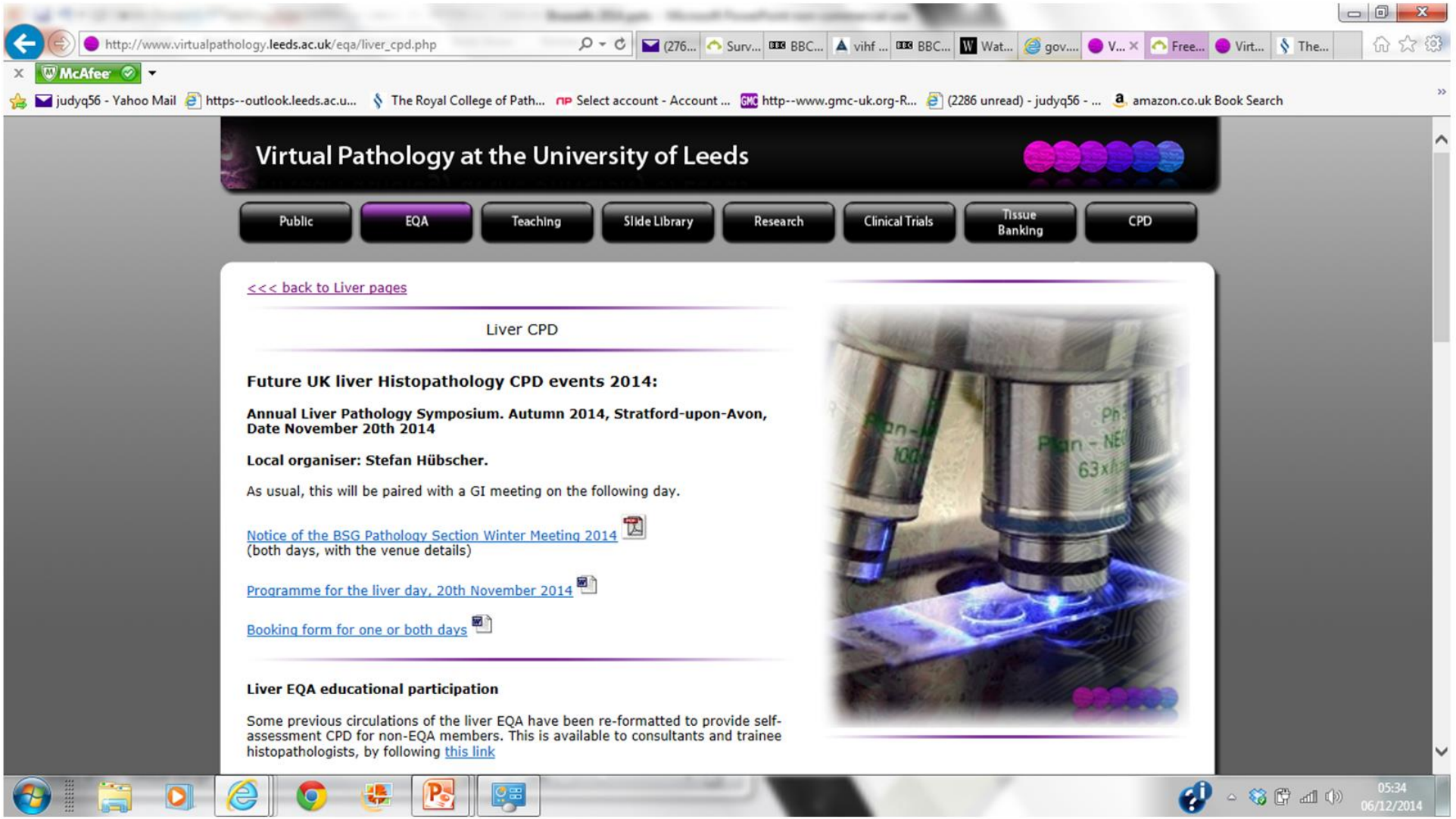
2. Details of meeting



3. Participants in 'cells'

Cell Distribution List K1

Date slides to be received	Participant	Cell Group	Cell Position	Address
18/08/2014	Dr J I Wyatt	1	1	St James's University Hospital, Department of Histopathology, Level 5 Bexley Wing, Beckett Street, Leeds, LS9 7TF
01/09/2014	Dr O Rotimi	1	2	St James's University Hospital, Department of Histopathology, Level 5 Bexley Wing, Beckett Street, Leeds, LS9 7TF
15/09/2014	Dr D Treanor	1	3	St James's University Hospital, Department of Histopathology, Level 5 Bexley Wing, Beckett Street, Leeds, LS9 7TF
29/09/2014	Dr S Mane	1	4	Barnsley Hospital, Department of Histopathology, Gawber Road, Barnsley S75 2EP
13/10/2014	Dr T H W Barker	1	5	Norfolk & Norwich University Hospital , Department of Histopathology, Colney Lane, Norwich, NR4 7UY
27/10/2014	Dr L Igali	1	6	Norfolk & Norwich University Hospital , Department of Histopathology, Colney Lane, Norwich, NR4 7UB
18/08/2014	Dr K Kalyanasundaram	2	1	University Hospital of North Staffordshire, Cellular Pathology , Floor 2, Main Building, Newcastle Road, Stoke-on-Trent, ST4 6QG
01/09/2014	Dr C Howitt	2	2	University Hospital of North Staffordshire, Cellular Pathology , Floor 2, Main Building, Newcastle Road, Stoke-on-Trent, ST4 6QG
15/09/2014	Dr A Darne	2	3	Royal Victoria Infirmary, Department of Cellular Pathology, Queen Victoria Road , Newcastle Upon Tyne , NE1 4LP
29/09/2014	Dr B Haugk	2	4	Royal Victoria Infirmary, Department of Cellular Pathology, Queen Victoria Road , Newcastle Upon Tyne , NE1 4LP
13/10/2014	Dr Y Bury	2	5	Royal Victoria Infirmary, Department of Pathology, Queen Victoria Road , Newcastle Upon Tyne , NE1 4LP
27/10/2014	Dr D Tiniakos	2	6	Royal Victoria Infirmary, Department of Cellular Pathology, Queen Victoria Road , Newcastle Upon Tyne , NE1 4LP
18/08/2014	Dr P Wright	3	1	Manchester Royal Infirmary, Department of Histopathology , Oxford Road, Manchester , M13 9WL
01/09/2014	Dr S M McGrath	3	2	Manchester Royal Infirmary, Department of Histopathology, First Floor CSB1, Oxford Road, Manchester , M13 9WL
15/09/2014	Dr G Howarth	3	3	Manchester Royal Infirmary, Department of Pathology, First Floor Clinical Sciences Building, Oxford Road , Manchester M13 9WL
29/09/2014	Prof R McMahon	3	4	Manchester Royal Infirmary, Department of Histopathology, Oxford Road , Manchester M13 9WL
13/10/2014	Dr E W Benbow	3	5	Manchester Royal Infirmary, Department of Pathology, Oxford Road, Manchester , M13 9WL
18/08/2014	Dr R J Prescott	4	1	Royal Blackburn Hospital, Department of Histopathology, Haslingden Road, Blackburn, BB1 3HH
01/09/2014	Dr G Langman	4	2	Birmingham Heartlands Hospital, Department of Cellular Pathology, Bordesley Green East , Birmingham, B9 5SS
15/09/2014	Dr D Neil	4	3	Queen Elizabeth Hospital , Department of Cellular Pathology, Mindelsohn Way, Edgbaston, Birmingham B15 2WB
29/09/2014	Dr R Brown	4	4	Queen Elizabeth Hospital , Department of Cellular Pathology, Mindelsohn Way, Edgbaston, Birmingham B15 2WB
13/10/2014	Prof S G Hubscher	4	5	Queen Elizabeth Hospital , Department of Cellular Pathology, Mindelsohn Way, Edgbaston, Birmingham B15 2WB
18/08/2014	Prof R D Goldin	5	1	St Mary's Hospital, Department of Cellular Pathology, 4th Floor, Clarence Wing, Praed Street, London W2 1NY
01/09/2014	Dr J Lloyd	5	2	St Mary's Hospital, Department of Cellular Pathology, 4th Floor, Clarence Wing, Praed Street, London W2 1NY
15/09/2014	Dr C P Johnson	5	3	Royal Liverpool University Hospital , Department of Pathology, 5th Floor, Duncan Building, Prescott Street, Liverpool L69 3GA
29/09/2014	Prof F Campbell	5	4	Royal Liverpool University Hospital , Department of Pathology, 5th Floor Duncan Building, Daulby Street , Liverpool L69 3GA
13/10/2014	Dr T Andrews	5	5	Royal Liverpool University Hospital , Department of Pathology, 5th Floor, Duncan Building, Prescott Street, Liverpool L7 8XP
27/10/2014	Dr Z Abdul-Rahman	5	6	Royal Liverpool University Hospital , Department of Pathology, 5th Floor Duncan Building, Daulby Street , Liverpool L69 3GA
01/09/2014	Dr T Walker	6	2	St Richards Hospital , Department of Histopathology, Spitalfield Lane, Chichester, West Sussex PO19 6SE
15/09/2014	Dr D Cowlishaw	6	3	St Richards Hospital , Department of Histopathology, Spitalfield Lane, Chichester, West Sussex PO19 6SE
29/09/2014	Dr T Umar	6	4	St. Richard's Hospital, Department of Pathology, Spitalfield Lane, Chichester, West Sussex PO19 5EB
13/10/2014	Dr N Cope	6	5	Royal Devon and Exeter Hospital, Department of Pathology, Church Lane , Exeter, EX2 5AD
27/10/2014	Dr T Clarke	6	6	Royal Devon and Exeter Hospital, Department of Pathology, Church Lane , Exeter, EX2 5AD

4 Date to receive and send on box of slides

National Liver EQA Scheme

CIRCULATION K1

Autumn 2014

Schedule for slide circulation **K1**

Louise to send out the slides on week commencing 11th August 2014 to Cell Position 1 in each Cell Group

	Date Slides to be received	Slides to be sent to next participant on
Cell Position 1	18th August 2014	29th August 2014
Cell Position 2	1st September 2014	12th September 2014
Cell Position 3	15th September 2014	26th September 2014
Cell Position 4	29th September 2014	10th October 2014
Cell Position 5	13th October 2014	24th October 2014
Cell Position 6	27th October 2014	Slides kept at final location

Responses for Circulation K1 to be electronically submitted by 7th November 2014

The meeting to discuss cases J1 and K1 will be held on 20th November 2014.

5. Link to surveymonkey to collect responses

Case K1/446 **Age 50, Male**

Haemochromatosis, raised ferritin. Assess fibrosis ? Cirrhosis
(please also see 3 special stains on website – Perls, Sirius Red, Retic)

1 core 17mm long

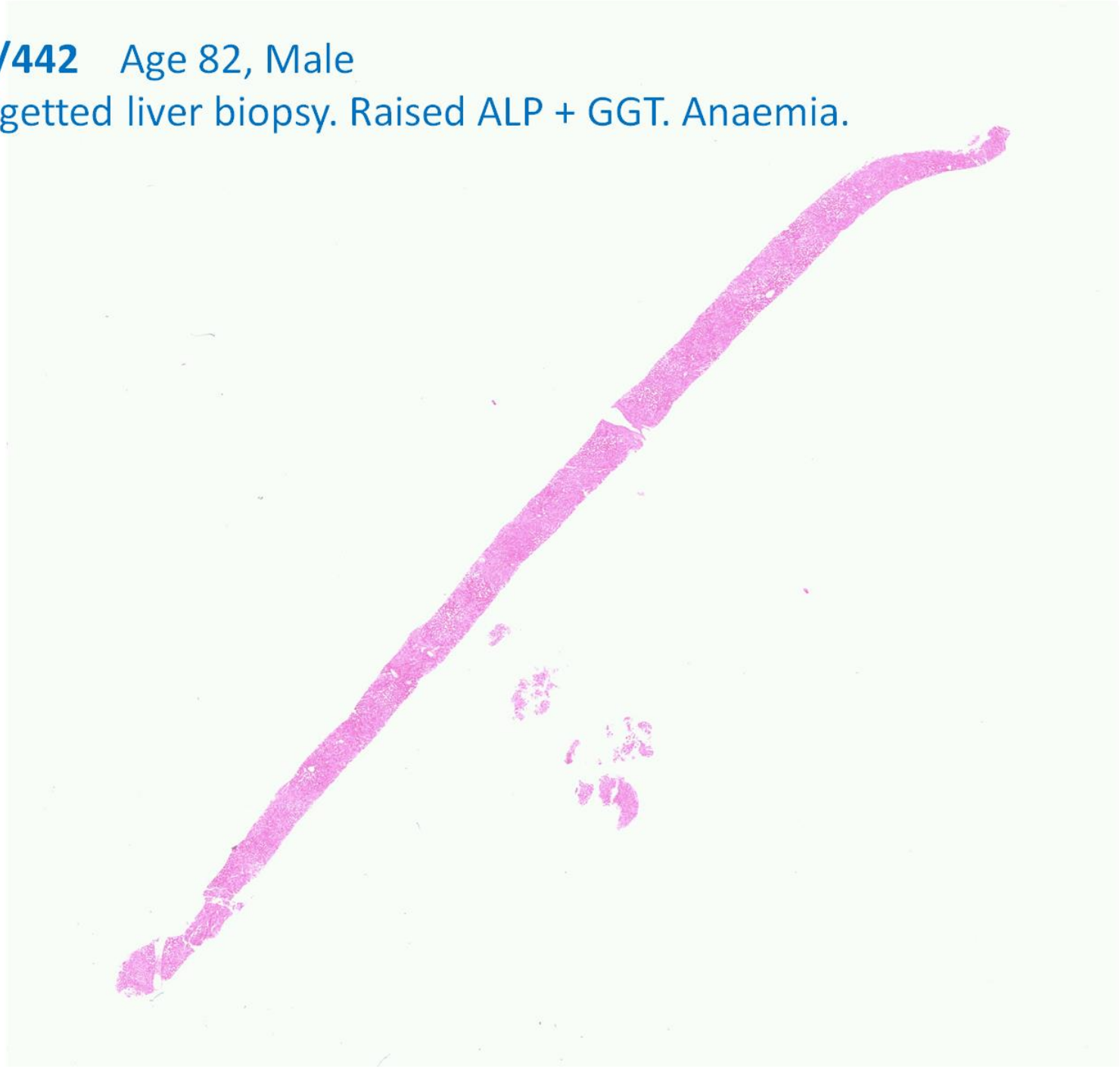
Morphological assessment:
Normal architecture, no inflammation. Grade 4 iron, no fibrosis/very mild on Sirius red

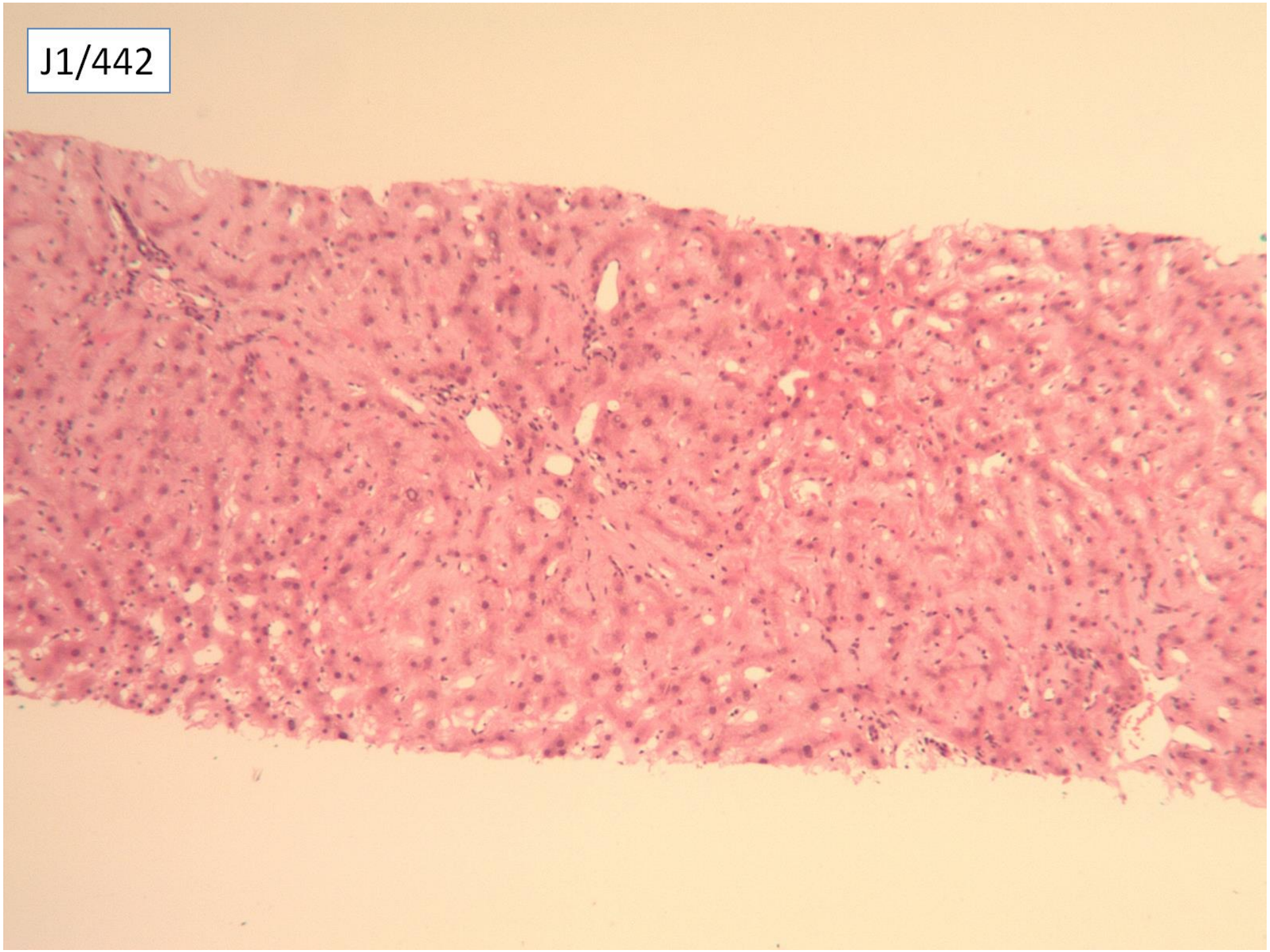
Clinicopathological diagnosis:
Consistent with genetic haemochromatosis. Minimal fibrosis HFE gene studies.

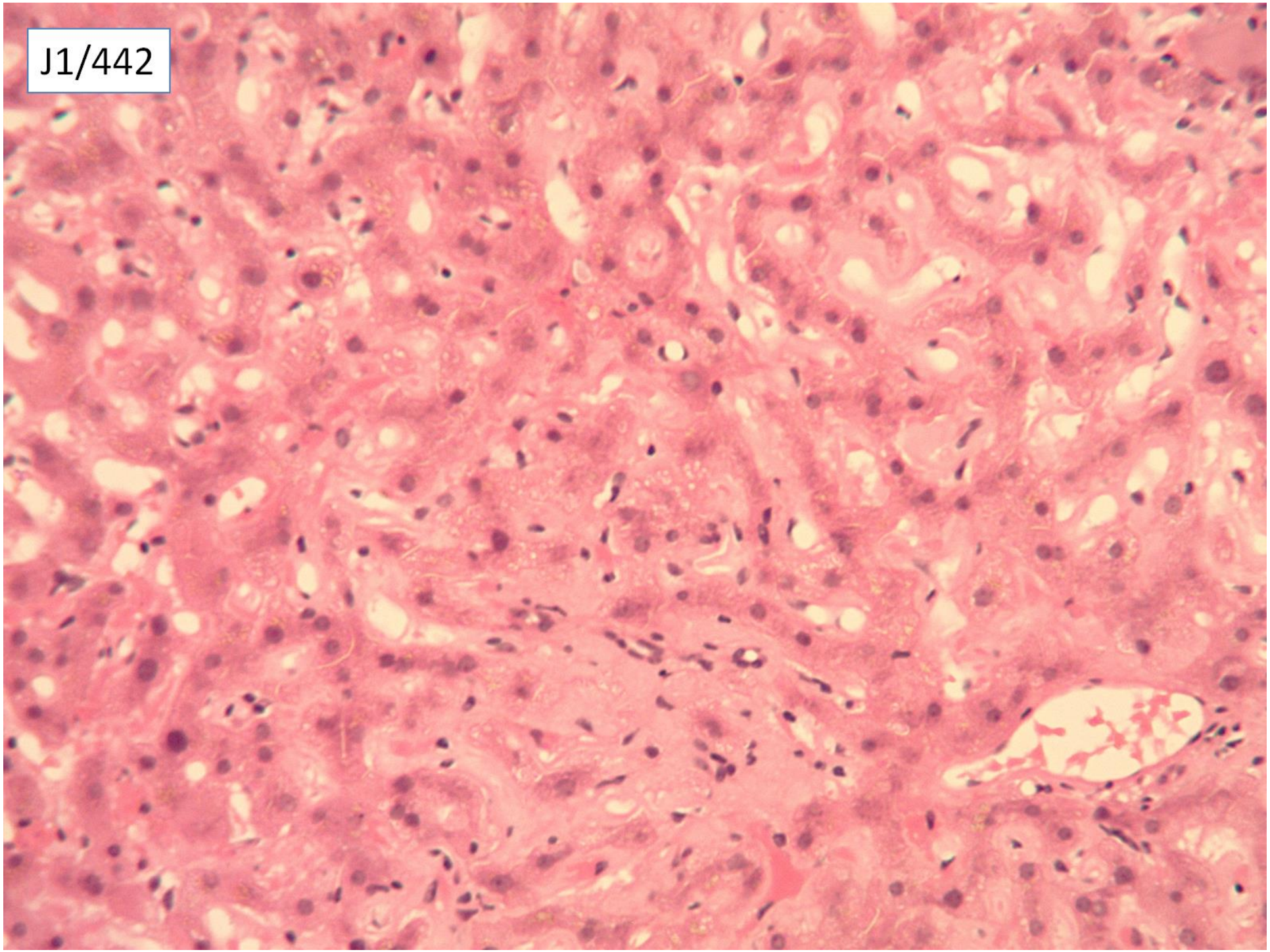
en-End	Case K1/4	clinicopath	Case K1/4	Clinicopath	Case K1/4	Clinicopat	Case K1/4	Clinicopath	Case K1/4	Clinicopath	Case K1/4	Clinicopath	C
2	Core of live	Severe sic	Cores of li	Severe, m	Cyst lining	Mucinous	Tumour co	Moderatel	Cores of li	Moderatel	Core of liv	Mild chron	C
6	The histol	This is in k	This biops	There is n	Cyst, with c	This lesio	Histology	The aetiol	This liver c	Features a	This liver b	The degre	T
8	Normal an	Consistent	Portal and	Consistent	Mucinous	Mucinous	HCC, mod	HCC, cirrh	Normal ar	Cholestas	Chronic he	Chronic he	S
10	Architectu	chronic he	Some Por	Severe he	Simple cys	Biliary cys	Hepatocyt	Moderatel	Architectu	The appea	There is m	This is a c	A
11	Severe sic	Severe sic	Portal and	In keeping	Mucinous	Mucinous	1. Backgro	1.Cirrhosis	1.Mild port	Further clir	1.Moderat	1.Some fe	S
15	Archiectur	Severe sic	Hepatits w	Acute hep	Cyst lined	Mucinous	Well differ	HCC arisi	Normal ar	Acute chol	Architectur	Midlly acti	N
16	Liver show	Grade 3/4	Acute lobu	Acute lobu	Liver with c	Mucinous	Lesion is a	Mod diff H	Liver with r	Acute chol	Liver show	Chronic HE	L
21	Fibrosis Is	Consistent	Focal brid	? Auto-imr	Lined my s	Biliary mu	Establishe	Liver cell c	Portal trac	Probably c	Portal trac	Moderate	N
23	Haemosic	In keeping	Acute port	Acute hep	Cyst lined	In keeping	Hepatoce	In keeping	Bilirubinos	Histologic	1.Chronic H	1.Hepatitis	N
24	Grade 4 (c	In keeping	Florid acu	Would be	Mucinous	Benign mu	Well differ	Pedophilia	Predomina	Suggestiv	Chronic he	Features c	S
26	Marked ha	Genetic ha	Portal and	In keeping	Cyst lined	Cystadenc	Fatty lesio	Hepatoce	Portal and	Cholestati	Chronic pc	Chronic HE	N
28	Grade 3 (c	Features c	Severe lol	Severe he	Mucinous	Mucinous	Cirrhosis v	Hepatoce	Perivenula	Cholestati	Mild chron	Features c	A
30	Florid grad	Haemochr	Lobular ar	Acute hep	Mucinous	Mucinous	Moderatel	Hepatoce	Acute can	Acute chol	Chronic he	Chronic he	S
31	There is n	The appea	This liver s	The overa	This show	Given the	This biops	The appea	The liver a	The appea	The liver a	The Orcei	T
33	Grade 4 d	Siderosis	Marked ex	Chronic he	Cyst lined	Mucinous	Nodular liv	Hepatoce	Some por	Some feat	Portal trac	Chronic he	S
34	16 portal tr	The featur	Liver biop	This is an	Cyst lined	This a ber	Cirrhotic liv	Hepatoce	18 portal tr	Features n	Approxima	Chronic ac	A
37	Normal an	Severe sic	Acute hep	Acute hep	Biliary epit	Biliary cys	Grade2/3 I	Hepatoce	Normal ar	Acute chol	mild chron	mild chron	c
38	Liver with	Features c	Liver with r	Acute hep	Hepatic cy	Mucinous	Liver cirrh	Moderatel	Liver with r	Features n	Liver with r	Chronic he	L
39	Architectu	Haemochr	Architectu	Would be	Cyst lined	Hepato-bi	Lesion cor	Hepatoce	Architectu	Cholestas	Architectur	Hep B, mc	A
40	Normal an	Haemochr	Acute hep	Acute hep	Cyst wall li	Biliary cys	In the back	Hepatoce	Portal trac	Acute hep	Brisk pred	Hepatitis B	N
41	Mild portal	Mild portal	Moderate	Moderate	Apparent u	Mucinous	Backgrour	Cirrhosis w	Bland hep	Bland cho	Mild portal	Mild steato	N
42	Grade 4 ha	hereditary	Acute hep	consistent	cyst lined I	Biliary cys	cirrhotic liv	moderatel	Normal ar	Acute chol	Portal fibro	Chronic he	N
43	Abundant	Haemachr	Acute hep	Acute hep	Benign co	Cystadenc	Mixed trab	HCC - see	Mild active	Mild active	Moderate	Mild active	N
47	GRADE 4	HAEMOCH	ACUTE H	?HEPATIT	MUCIN SE	BILIARY C	CIRRHOT	HEPATOC	HEPATOC	CHOLEST	MODERAT	ALCOHOL	N
51	extensive	Mild chron	mixed por	in keeping	cyst lined I	hepatobili	establishe	well differe	cellular ba	acute hepa	Parencym	Chronic he	C
57	Marked sig	Grade 4 si	Acute pan	No seroloc	Cyst wall- I	Hepato-bi	Part of noc	Well differ	Cholestati	No seroloc	Mild steato	Consistent	N
59	Abundant	Consistent	Acute hep	Would be	Mucinous	See abov	Hepatoce	As above	Portal exp	I'd want a f	Steatohep	c/w hepati	N
60	Severe pa	c/w geneti	Severely a	Severely a	Low grade	Low grade	Moderatel	Hepatoce	Bile duct a	Acute chol	Portal hep	1. Chronic	A
61	Core of live	Iron overlo	Moderate	Moderate-	Fibrous- w	Hepatobil	Multinodul	Hepatoce	Mild polyn	Cholestati	Mild-mod	Mild chron	N

Case J1/442 Age 82, Male

Non-targetted liver biopsy. Raised ALP + GGT. Anaemia.




J1/442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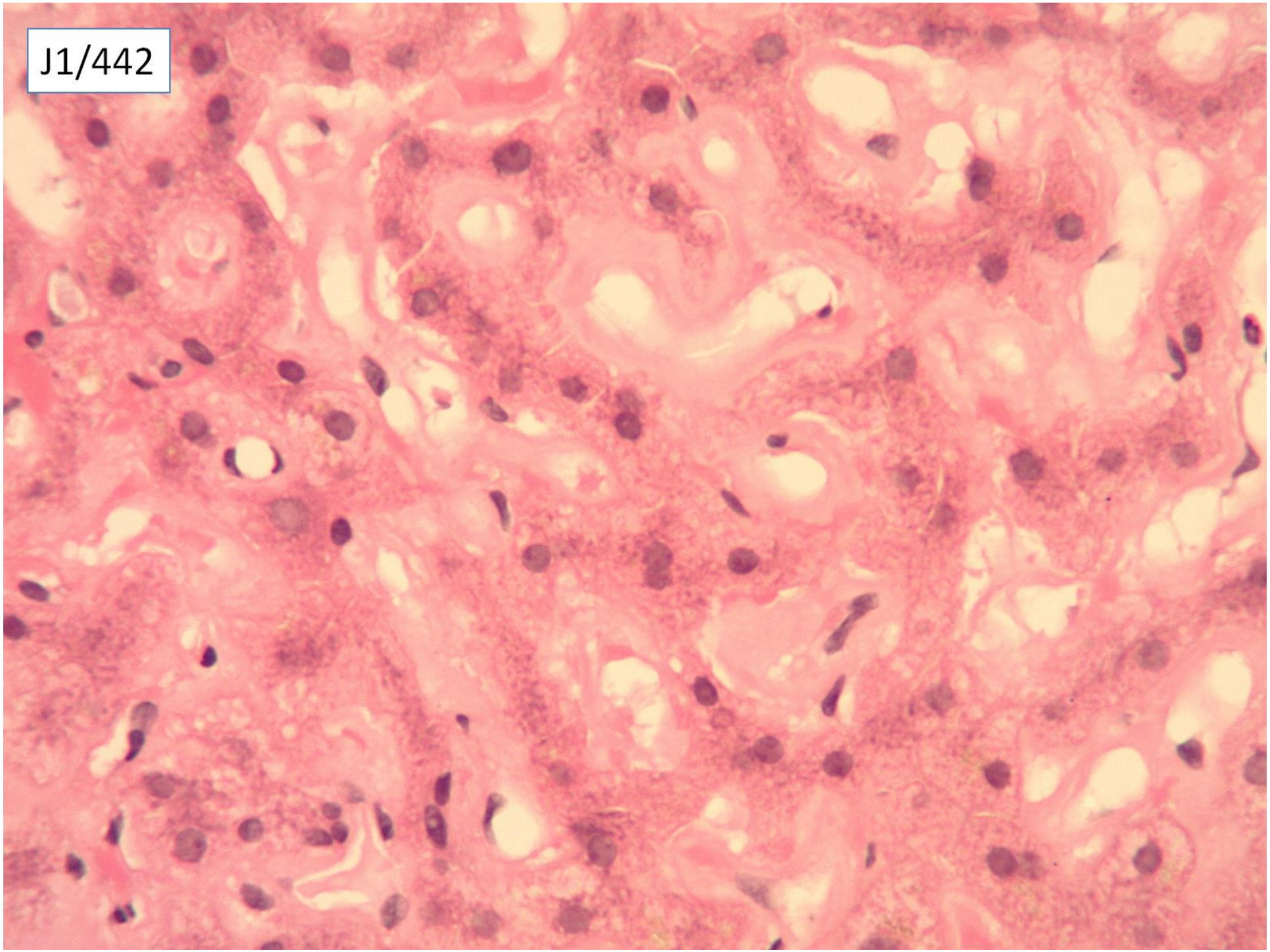
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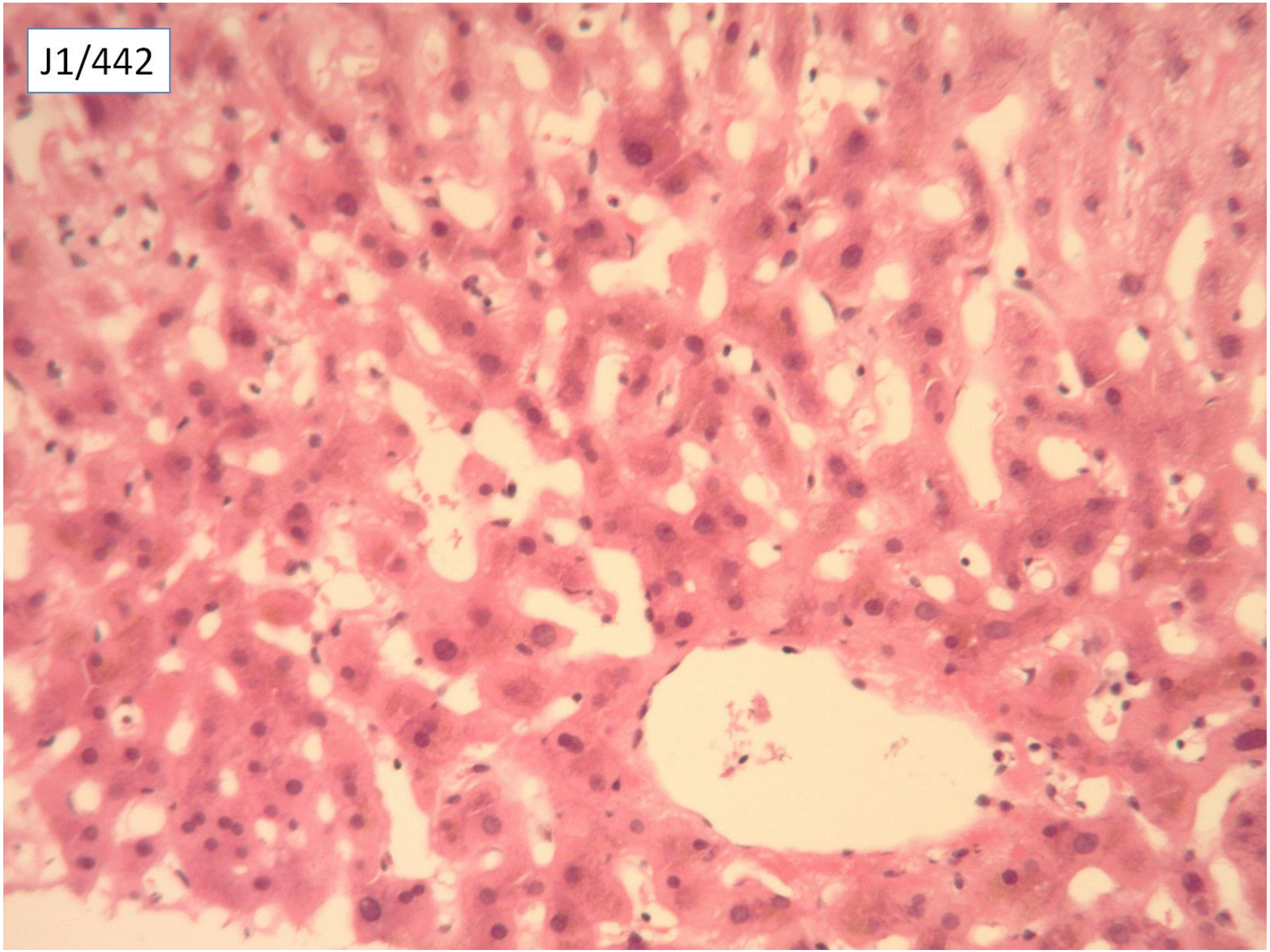
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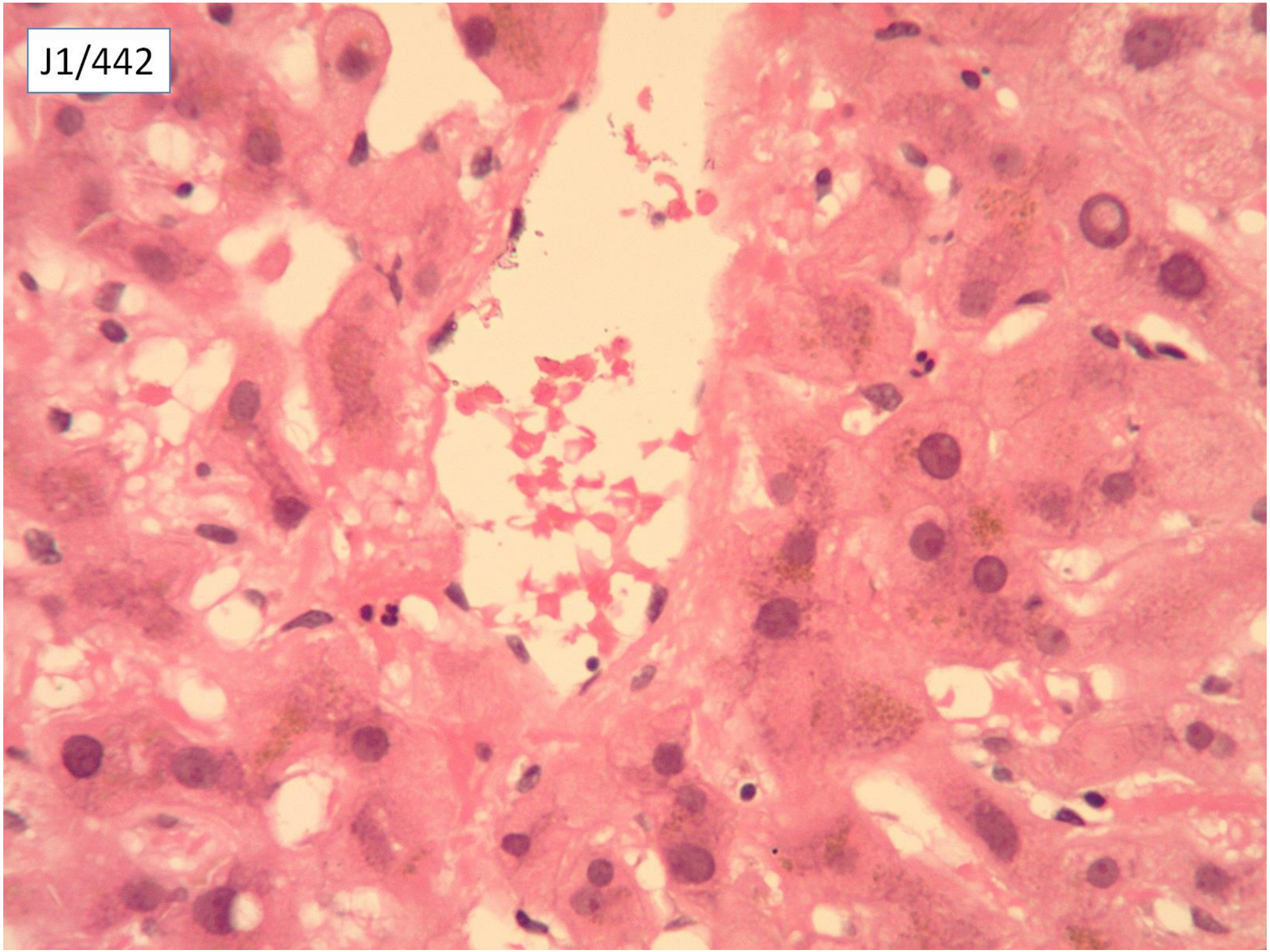

J1/442

A	B	C	D
articipant	Have yo	Case J1/442 Age 82, Male Non-targetted liver biopsy. Raised ALP + GGT. Anaemia. Needle biopsy core 25x1x1mm Morphological assessment:	Clinicopathological diagnosis:
2	Yes - I've	Eosinophilic perisinusoidal material. Congo red and immunostaining (AA & light chains) required.	Amyloidosis (probably). Consider causes underlying AA and AL amyloidosis.
3	Yes - I've	11 non-inflamed portal tracts, overall vascular relationships preserved, prominent perisinusoidal deposition of pale pink material with significant hepatocyte atrophy and sinusoidal dilatation, also eosinophilic material in portal tracts.	Morphologically highly suggestive of amyloid, confirm by congo red, sirius red stains and refer to national amyloidosis centre, in differential diagnosis – light chain deposition, correlate with haematology and cause for anaemia
6	Yes - I've	This liver biopsy shows pale amorphous, eosinophilic material filling the canalicular eye and causing atrophy of hepatocytes. The appearances are in keeping with amyloid deposition which should be confirmed by staining with Congo red which is positive with apple green birefringence.	This should be correlated with the patient's clinical history for evidence of chronic inflammatory disease and the presence of free light chains in the serum. Pre-treatment of Congo red with potassium permanganate can distinguish primary and secondary amyloidosis.
8	Yes - I've	amyloid, or light chain deposition disease. Needs PASD and congo red.	check paraprotein, other associations of amyloid. consider clinical referral to national amyloidosis centre
10	Yes - I've	Overall architecture is preserved. The portal tracts are normal. The lobule shows marked sinusoidal dilatation and congestion with some atrophy of hepatocytes and eosinophilic material lining the sinusoids.	The appearances strongly suggest amyloid. I would do congo red and immunos for SAA/P, light chains.
11	Yes - I've	Eosinophilic material in sinusoids, highly suggestive of amyloid. Need Congo Red staining for confirmation.	In keeping with hepatic amyloidosis.
12		Sinusoidal infiltrate of hyaline material ? amyloid ? light chain disease. Portal tracts normal ?? arteriolar	Suggestive of amyloidosis but would not report without special AND clinical correlation

Case J1/442 Age 82, Male

Non-targetted liver biopsy. Raised ALP + GGT. Anaemia.

77 Amyloid

1 heart failure ? BCS ?? amyloid - needs stains

2 sinusoidal dilatation, congestion, no mention of amyloid

1 cholestatic histology and biochemistry, Large bile duct obstruction

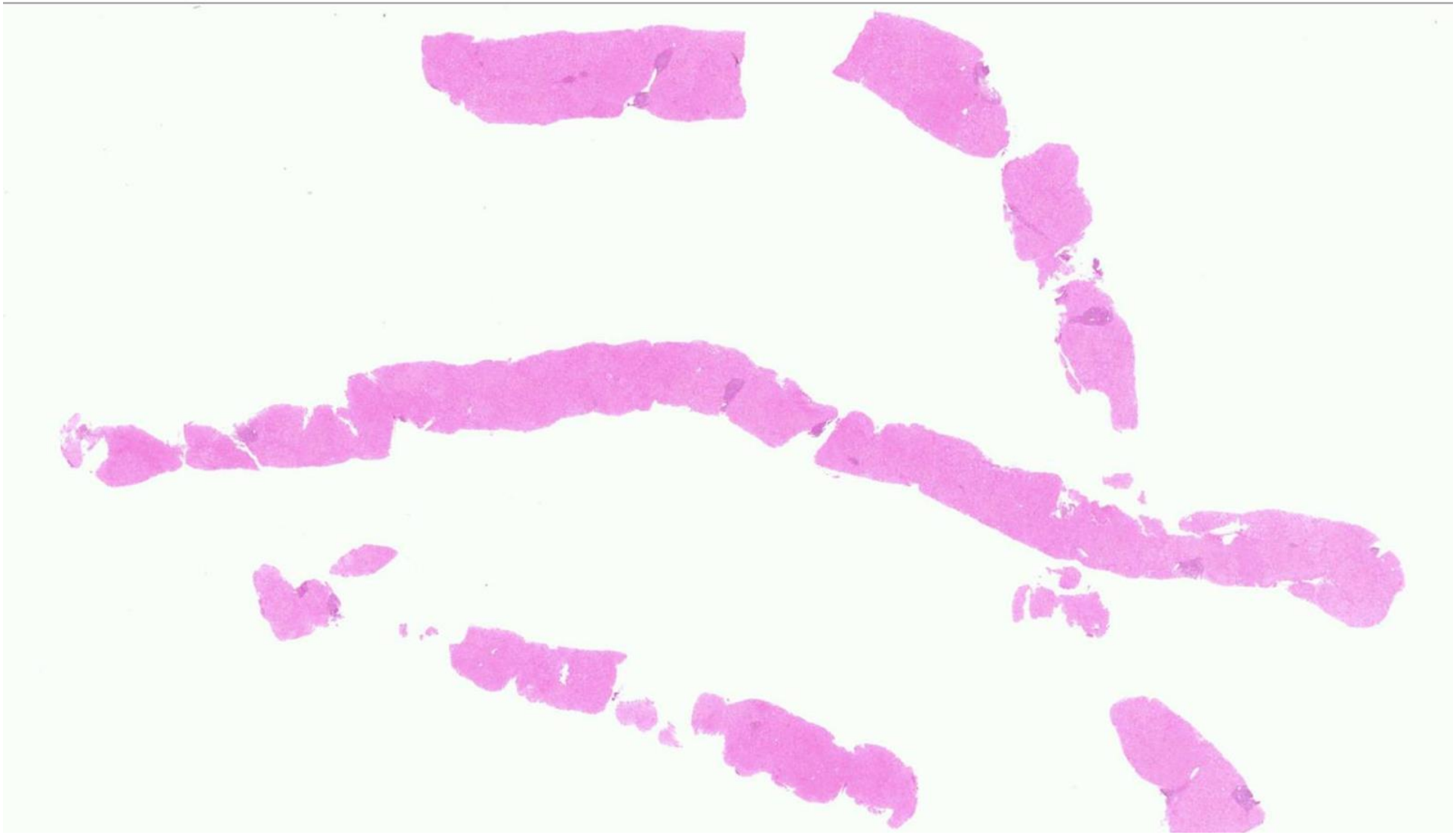
1 cholestasis and bile duct damage ? PSC

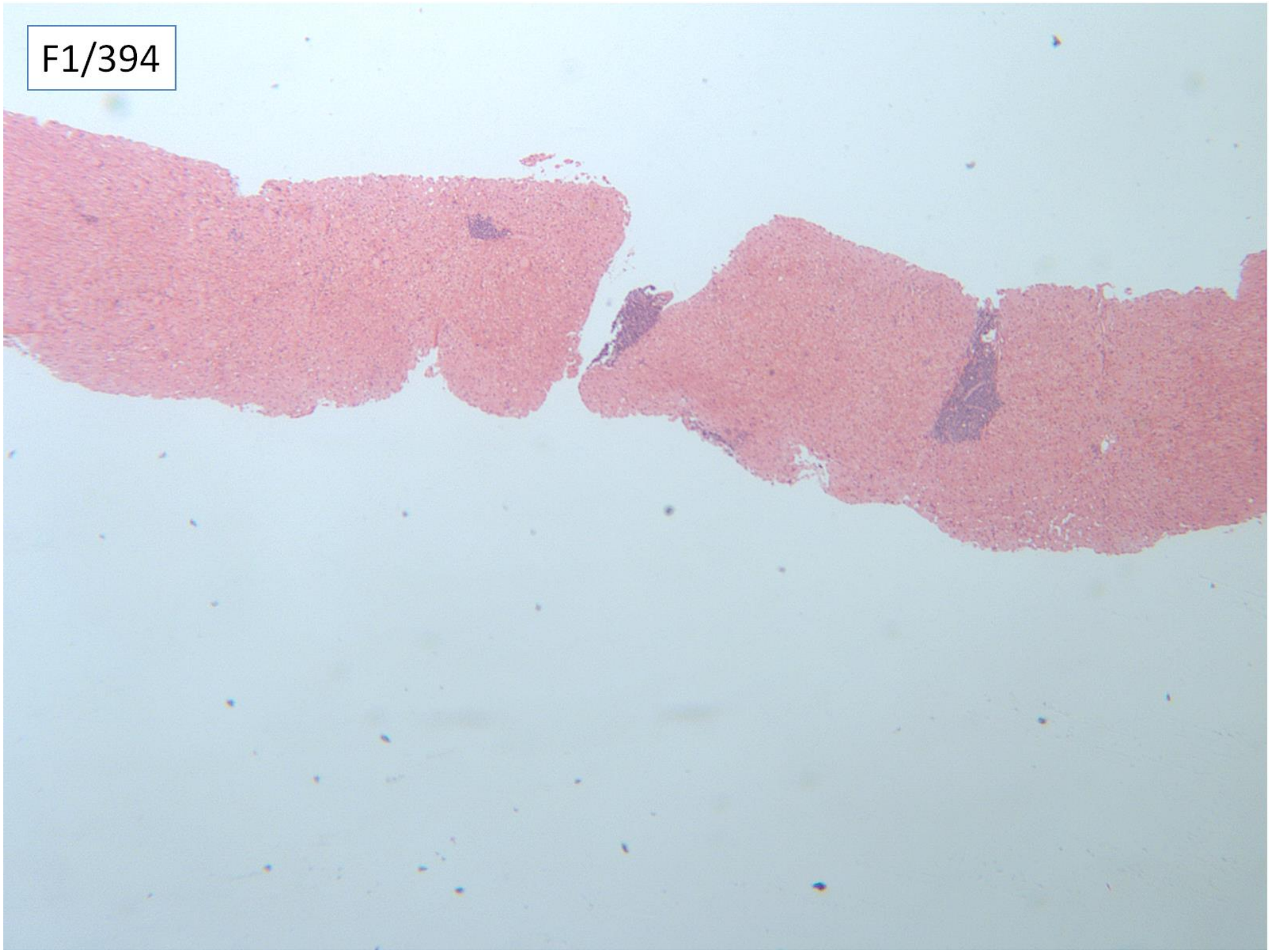
Suggested scoring: at last an easy one to score.
For 10 points - amyloid as only or main diagnosis
Amyloid in differential but not first score 5 points.
No mention of amyloid, score 0 points.

14/14 agree, 0 unsuitable

EQA Case F1/ 394 58 M

HCV genotype, RNA –ve after treatment

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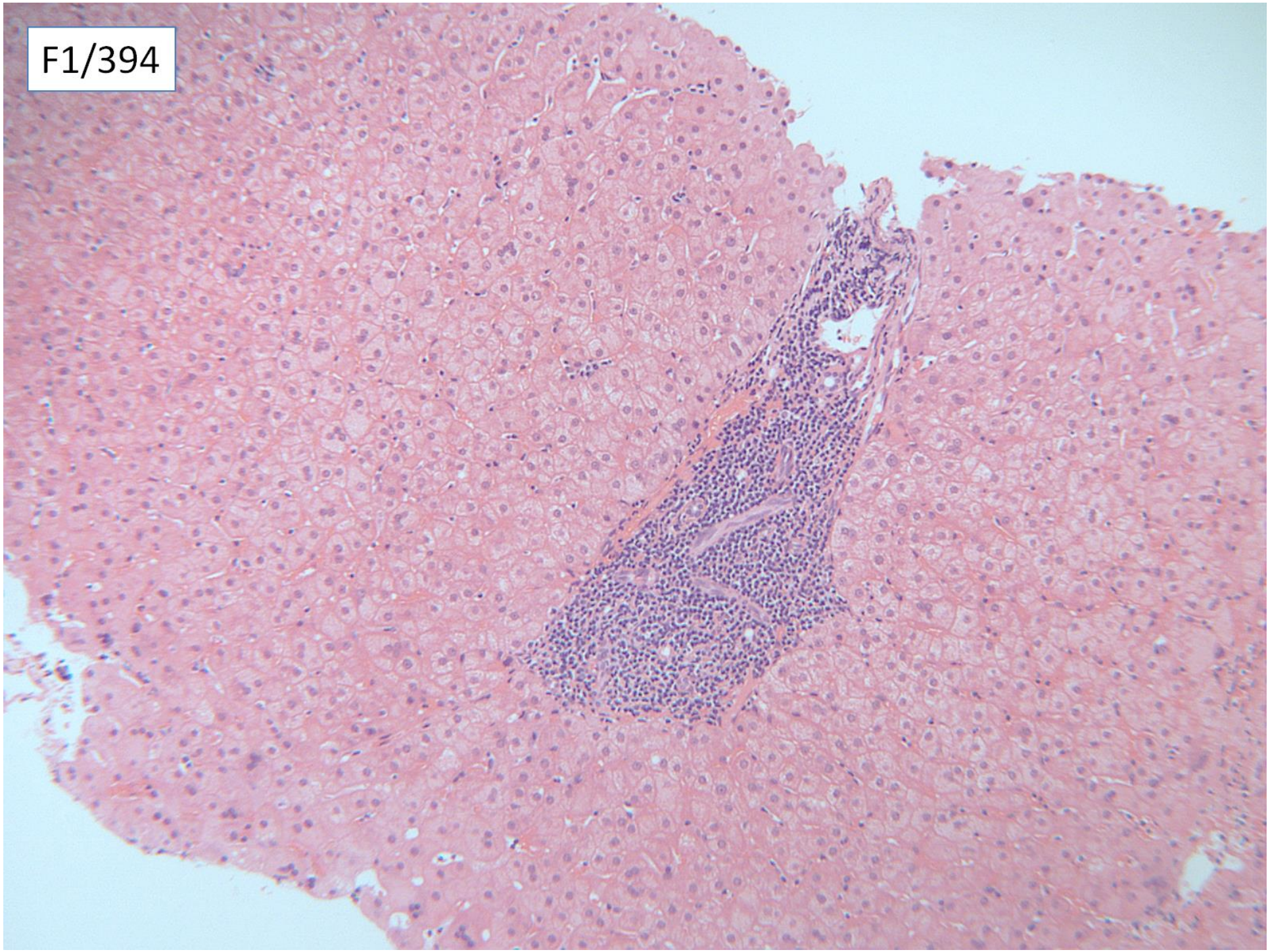
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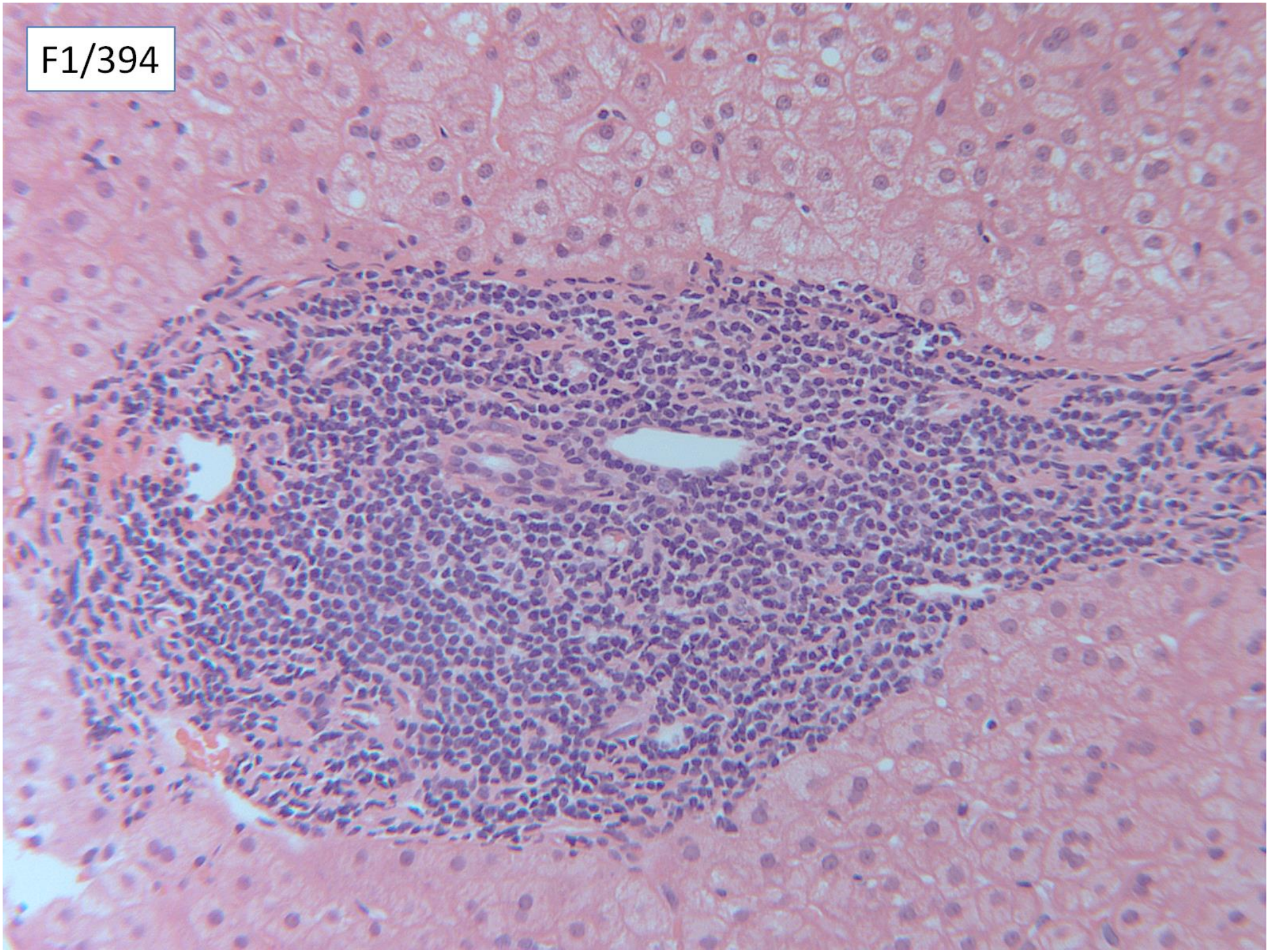
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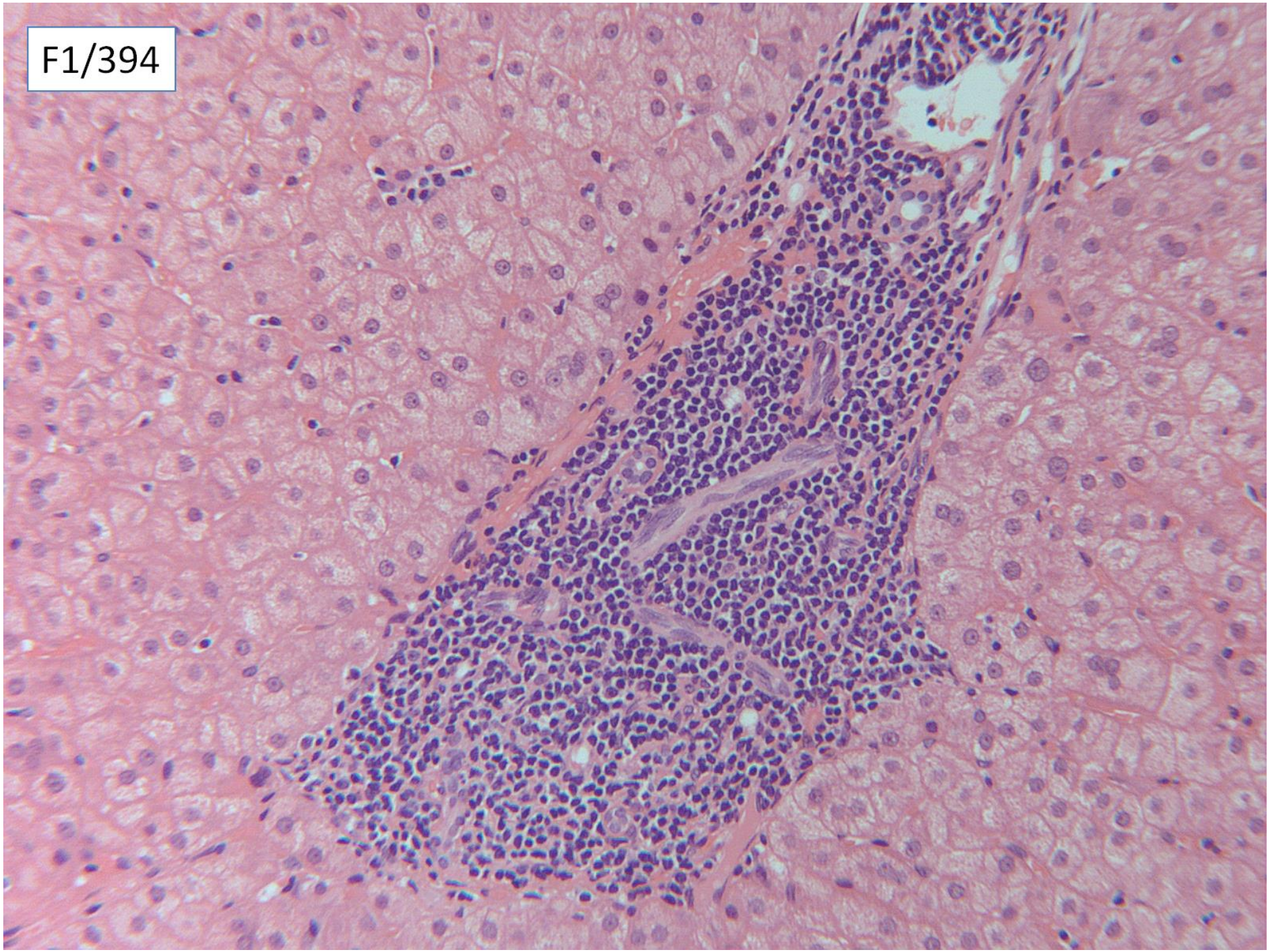

F1/394

A	B	C
2	Monotonous, dense portal lymphocytic infiltrate suggestive of B-cell lymphoma. "Indian file" sinusoidal permeation by lymphocytes not evident. Some multinucleate hepatocytes.	Further clinical and laboratory investigations (including immunohistochemistry) for consideration of lymphoma are required.
3	21 portal tracts, mild focal fibrosis, monotonous lymphoid infiltrate in portal areas, focal interface hepatitis but not prominent, multinucleation of hepatocytes with ground glass type cytoplasmic appearances, lobular infiltrate with occasional foci of spotty necrosis but mainly sinusoidal	Dense portal infiltrate and giant cell change in parenchyma DD: unusual appearance of chronic HCV infection post treatment with giant cell change, stage 2/6, grade 6-7/18, but because of monotonous infiltrate exclude low grade B cell lymphoma by ICC with characterisation of lymphoid cells as B and T cells (chronic hepatitis in DD) check with ICC, correlate with history, LFTs etc
6	Mild to moderate portal inflammation in all portal tracts. No confluent necrosis. Mild focal interface hepatitis round most portal tracts occasional foci of lobular inflammation.No fibrosis (stage 0). Necroinflammatory score 2,0,1,2. Consistent with ild to moderately active hepatitis C infection. Multinucleate hepatocytes may be related to treatment witn interferon.	Hepatitis C infection. 2,0,1,2. No fibrosis.
7	Portal chronic inflammation, minimal necroinflammation, multinucleation of hepatocytes. Needs collagen stain to assess (likely minimal) fibrosis	Chronic hepatitis C virus infecton with minimal activity. Pending collagen stain.
8	Early stage, portal inflammation with monotonous lymphocytes - looks like CLL. Not more than stagei 1 fibrosis. Parenchyma - some increase in lymphocytes but not necroinflammatory activity. Frequent multinucleate hepatocytes ? why - ? drugs for CLL	Probably CLL, would account for most/all portal inflammation.

Case 15: EQA Case 394: 58M,

HCV genotype, RNA –ve after treatment

Suggested scoring
No consensus,
not suitable for scoring.

Responses - 81

41 Hepatitis C

13 Hepatitis C but lymphocytes monomorphic, possible/want to exclude lymphoma

26 Lymphoma as most likely diagnosis

1 Hepatitis C and autoimmune hepatitis

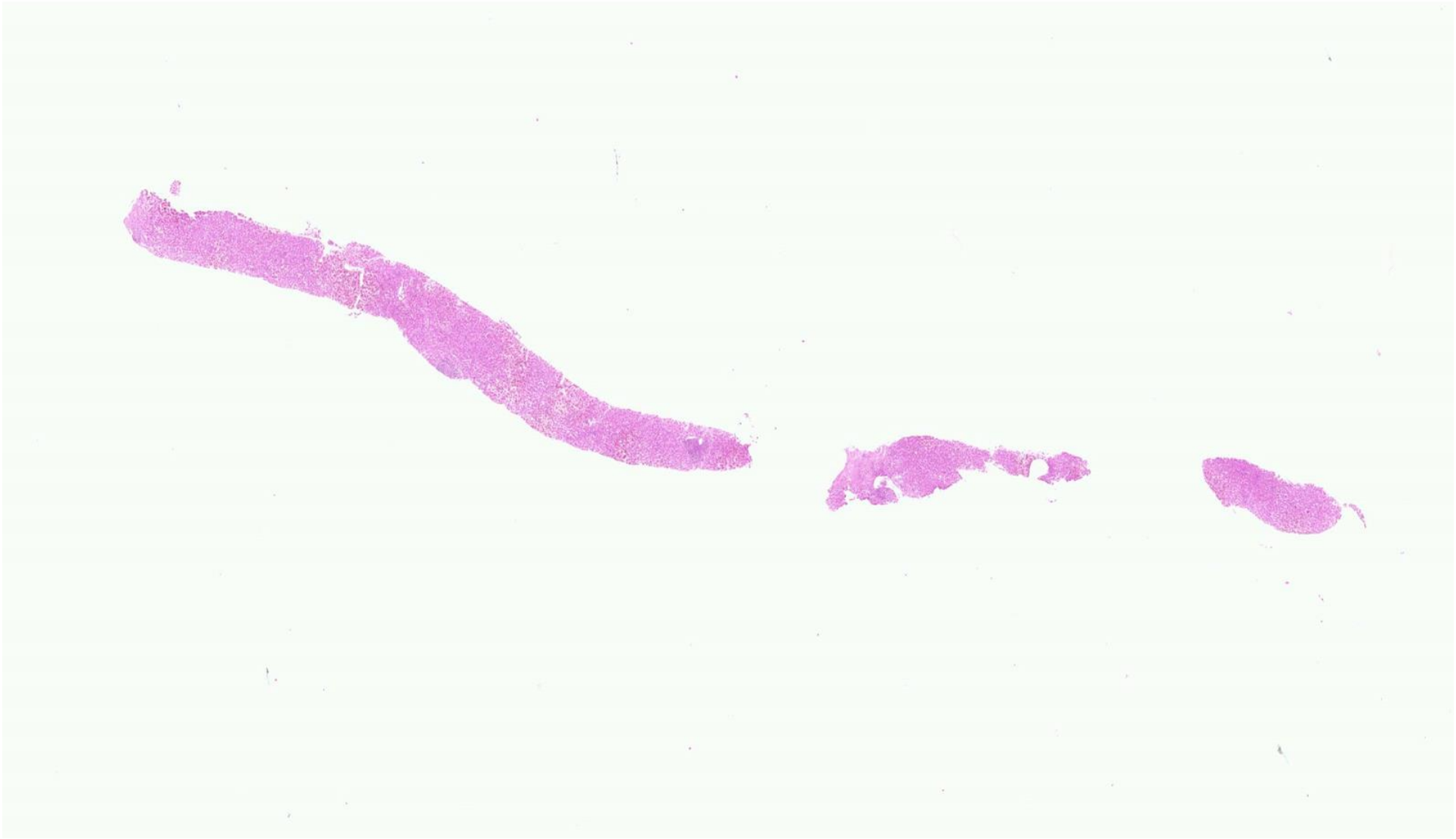
Very interesting result – original diagnosis was lymphoma, but clinical details frame the problem as a case of hepatitis C.

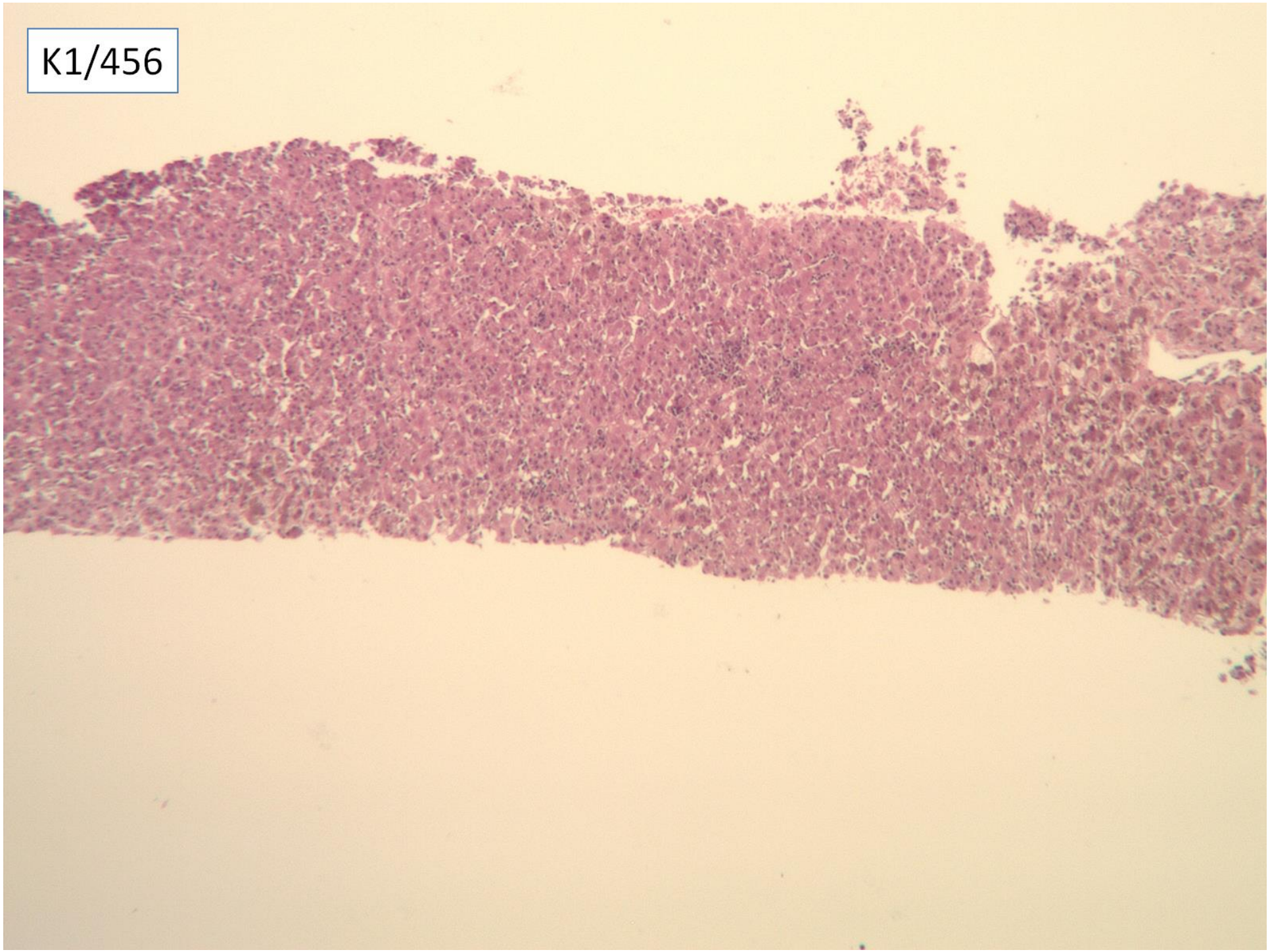
Are you more likely to recognise the lymphoma if you don't read the clinical information before looking at the slide?

Following the meeting: responses reviewed: 41 had some comment raising the possibility of lymphoproliferative process/lymphoma.
40 responses – hepatitis C only

Case K1/456 **Age 47, Female**

Unwell jaundice 1 week. High LFT's ?acute hepatic failure.

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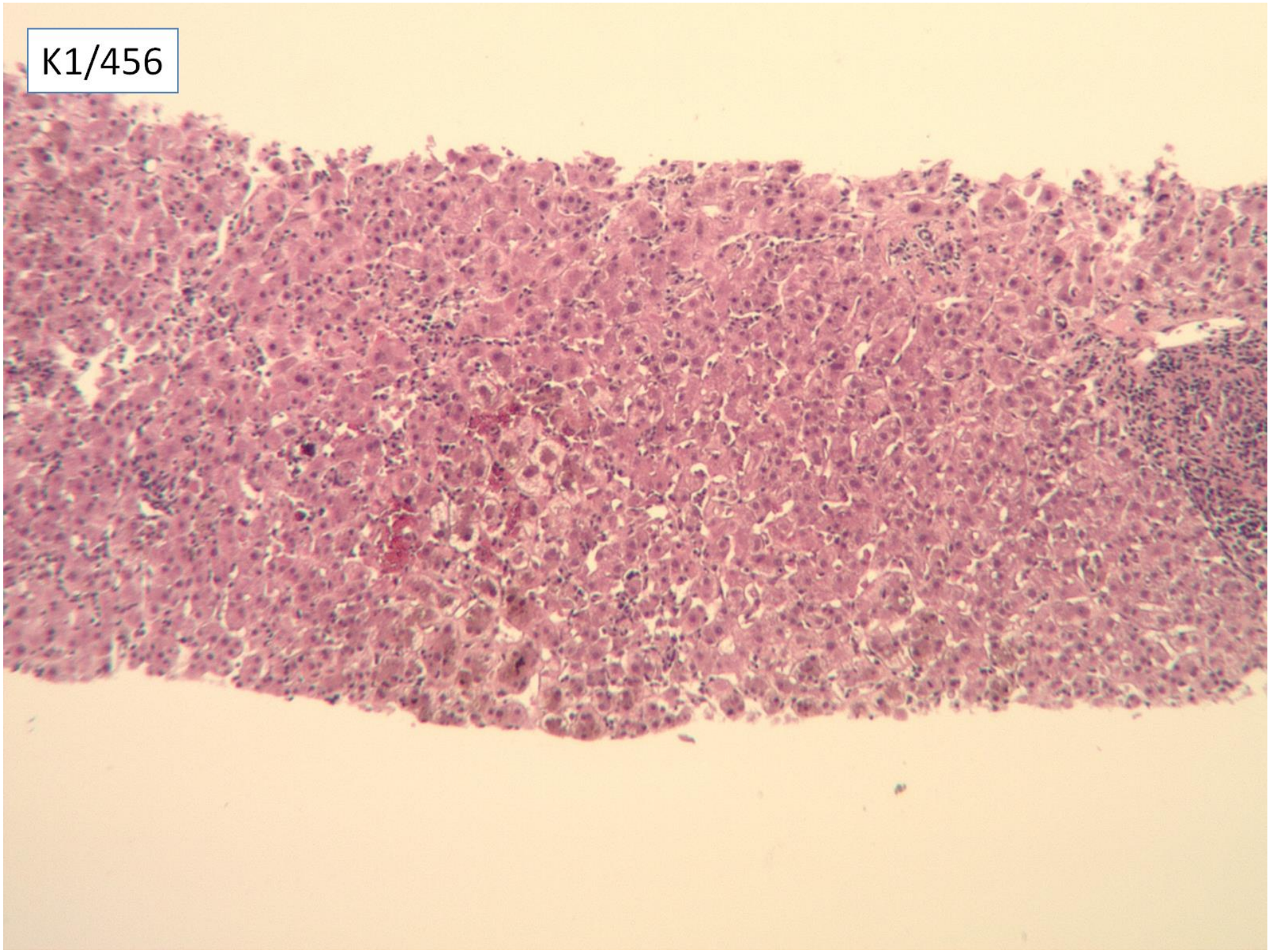
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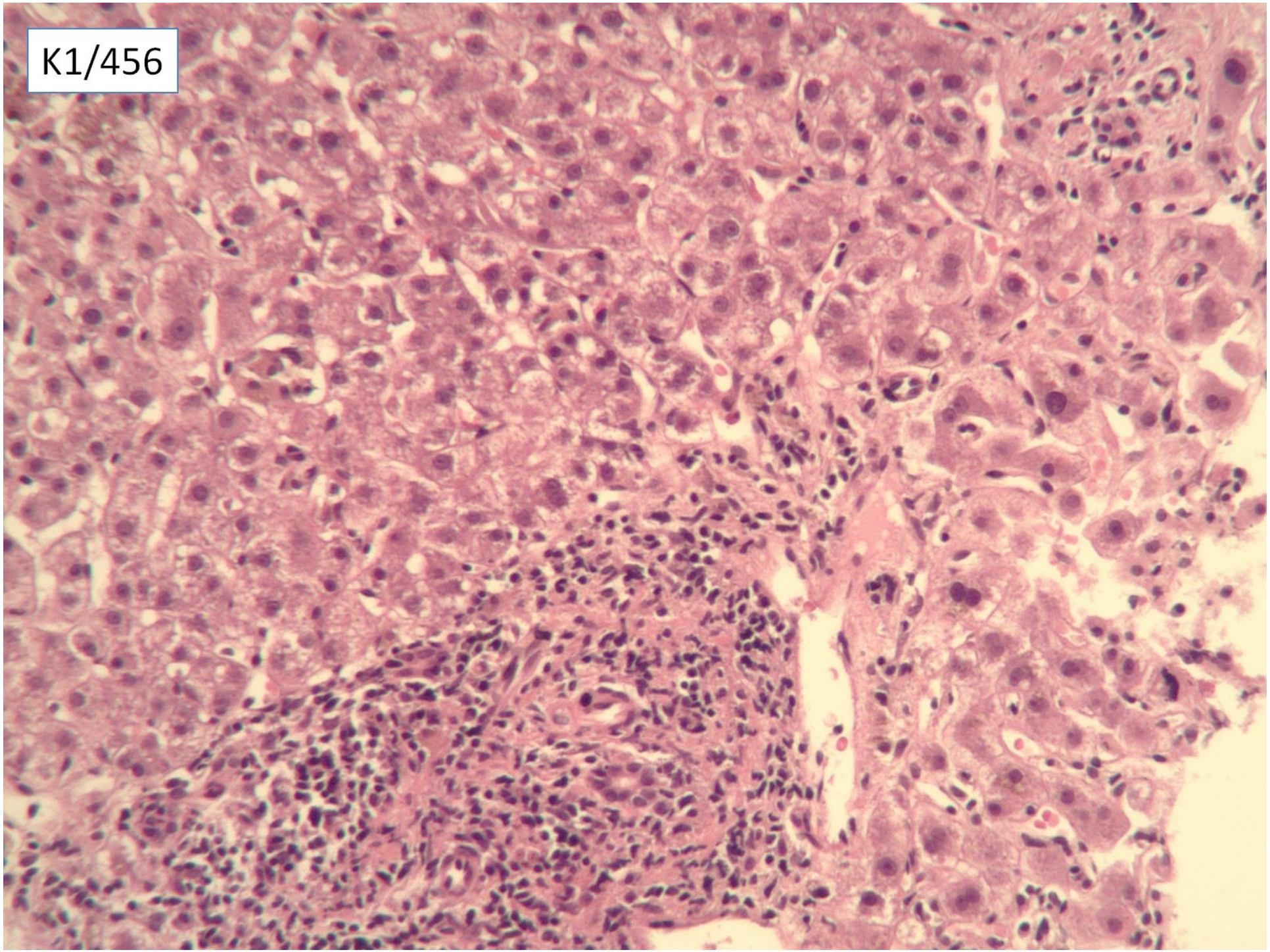
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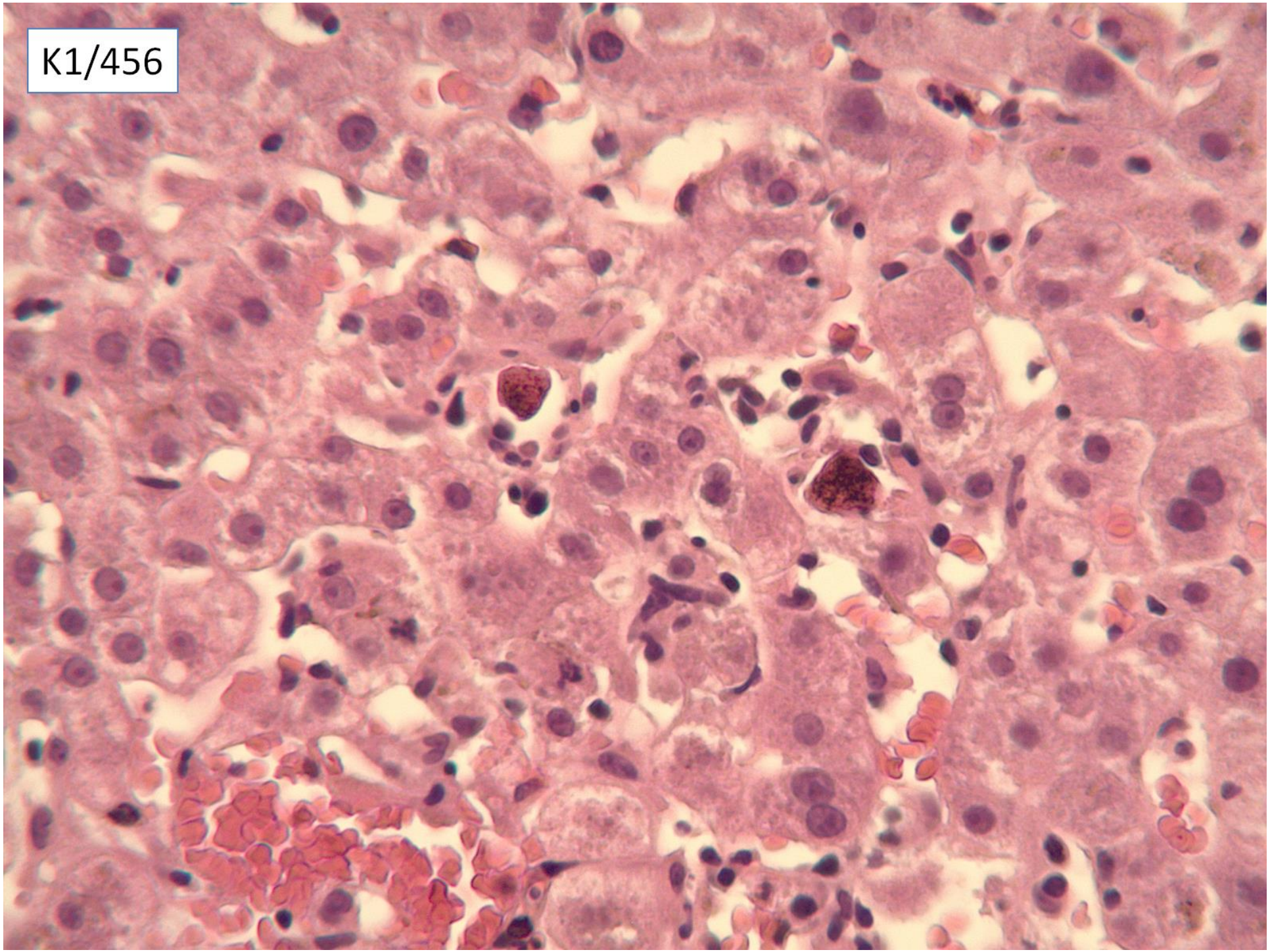
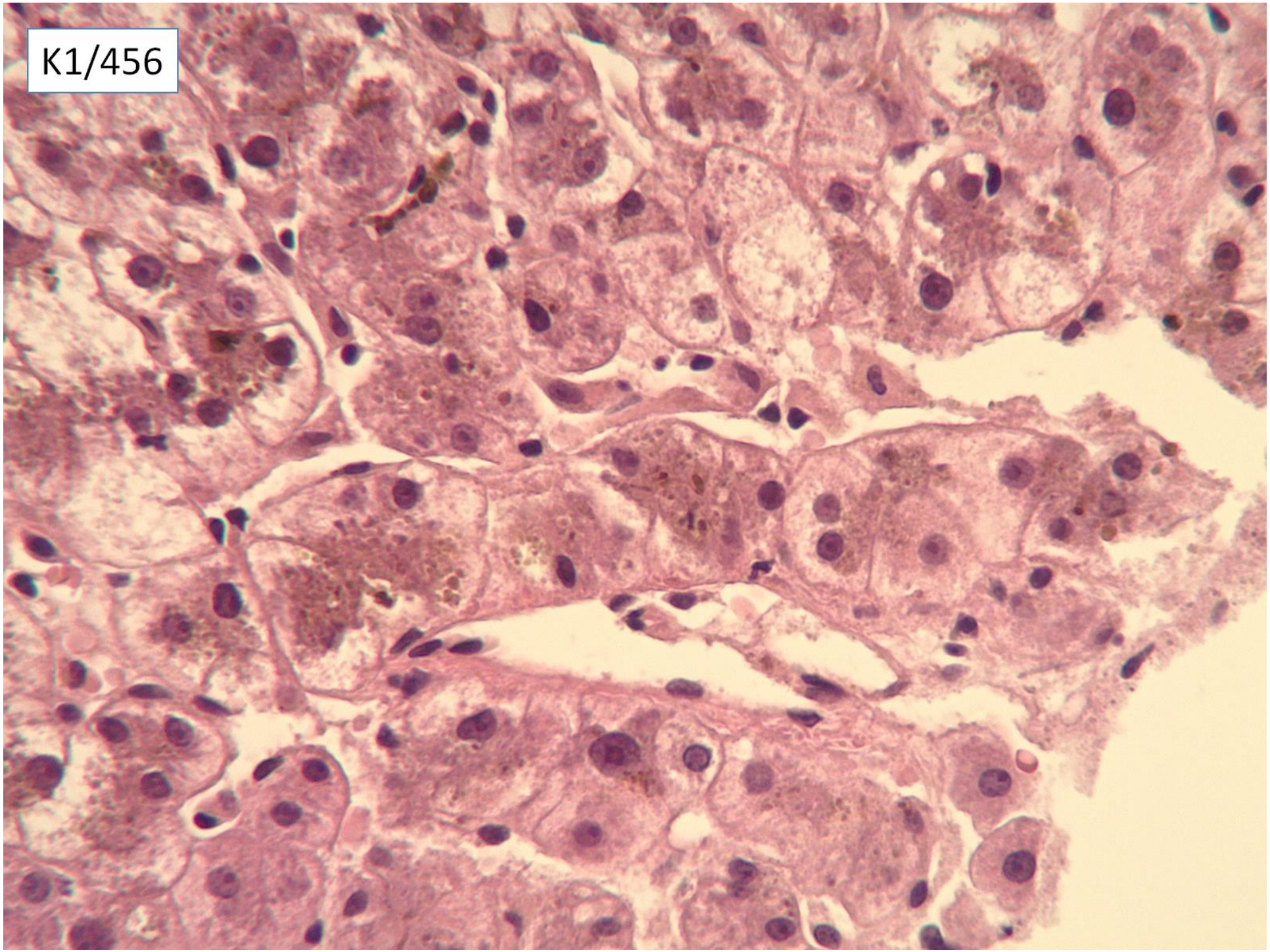

K1/456


K1/456

Ended Res	Case K1/456 Age 47, Female Unwell jaundice 1 week. High LFT's ?acute hepatic failure. 3 cores - max 10mm Morphological assessment:	Clinicopathological diagnosis:
2	Core of liver. Marked portal and parenchymal inflammation. Few plasma cells or eosinophils. No lymphoid aggregate or granuloma. Marked parenchymal cholestasis. Liver-cell ballooning. Occasional apoptoses.	Severe, morphologically acute hepatitis with cholestasis. The aetiology is not evident morphologically and possibilities include drug reaction, viral infection, and autoimmune hepatitis. The degree of cholestasis favours drug reaction.
6	This liver biopsy shows severe cholestasis, hepatocellular and canalicular. There is portal inflammation and focal ductular reaction. Native bile ducts are present with no obvious cholangiography there are many apoptotic cells in the sinusoids. There is no definite malignant infiltrate. This is a cholestatic hepatitis	There is no evidence of fatty liver disease. This is a cholestatic hepatitis and a possible drug-related cause should be considered.
8	Acute cholestatic hepatitis. Not confluent necrosis, some portal inflammation with plasma cells - AIH possible.	Differential diagnosis includes autoimmune, drugs, viral hepatitis (includes hepatitis E)
10	About 50% of the portal tracts are normal. The rest show some fibrosis and lymphoid inflammation with mild focal interface hepatitis. No plasma cell aggregates are seen. The lobules show severe disarray with swollen hepatocytes, acidophils and widespread sinusoidal inflammation. There is bile stasis with some central drop out.	This is a moderately severe acute hepatitis. would prefer virus but want to exclude drug. There is no evidence of AiH.
11	Lobular hepatitis with spotty necrosis and severe cholestasis. No evidence of bridging necrosis or panacinar necrosis. A few atypical portal lymphoid cells ? significance.	Acute cholestatic hepatitis ? cause. Consider viral agents, drugs and AIH in differential diagnosis. Further clinical information required.
15	Preserved architecture. Moderate centrilobular cholestasis with associated ballooning. Moderate portal and parenchymal inflammation. Mild ductular proliferation. Spotty necrosis	Acute cholestatic hepatitis. Differential diagnosis of drug, viral or less likely autoimmune hepatitis.
	Acute lobular hepatitis with marked intracellular/intracanalicular cholestasis. Small foci of	Acute lobular/cholestatic hepatitis ?drug reaction ?autoimmune

Case K1/456 Age 47, Female

Unwell jaundice 1 week. High LFT's ?acute hepatic failure.

45 acute cholestatic hepatitis

16 acute hepatitis

2 acute liver injury, or description 'hepatitis' not stated

1 "acute intrahepatic cholestasis ? ductopenia, - drugs or underlying ductopenic disease"

2 descriptive, no conclusion

1 lymphoma with cholestatic liver injury

1 large bile duct obstruction – no mention of hepatitis

41 drugs/viral/autoimmune, none favoured
13 virus/drugs, AIH less likely/not AIH
8 differential, favours drug
4 drugs as only cause
4 favours AIH
2 drugs or AIH (not virus)
1 favours viral

Specific ones suggested:
4 hepatitis E, 3 hepatitis A, 2 EBV
2 ? haemochromatosis
3 ? Wilson's
1 exclude lymphoma,
1 suspect lymphoproliferative disorder or large bile duct obstruction
2 ? alcohol

6 drugs not mentioned
5 no aetiology suggested

Suggested scoring:.
for 10 points need acute hepatitis. Lose 5 points for report not mentioning hepatitis. Lose 10 points for misleading diagnosis.

12/17 agree, 3 unsuitable

Case K1/456 **Age 47, Female**

Unwell jaundice 1 week. High LFT's ?acute hepatic failure.

- Additional clinical information - initial ALT >2500, coagulopathy (peak INR 2), now normalised. Bilirubin 255. Hepatitis A/B/C –ve. Alcohol >42u/week, not dependant.

- Original diagnosis: Acute cholestatic hepatitis. Subsequently acute hepatitis E. IgM +ve, PCR $2x10^5$.

- Treated with steroids before the IgE results was known. ALT steady fall, normalised in 4 weeks. Follow up in local hospital.

- See presentation by Chris Bellamy for discussion of histology of hepatitis E.

Outline:

- History of interpretive EQA schemes in the UK
  - Why did they start?
  - Governance
  - How do they work
  - Being a scheme organiser
- UK National Liver Histopathology EQA Scheme
  - How it's run
  - Examples of cases
- Future of interpretive EQA schemes

Sherwood Forest NHS Trust could 'run out of money'

21 September 2012

Health regulator Monitor is intervening in the running of a Nottinghamshire NHS trust over concerns about its finances.

The trust's chair, Tracy Doucet, said despite financial "challenges" no concerns over patient care were raised.

In 2006, the trust embarked on a £320m PFI expansion scheme

Sherwood Forest NHS Trust chairman stands down

6 October 2012

The chairman of a Nottinghamshire NHS trust facing financial difficulties has stood down from her post.

In 2006, work began on a £320m PFI expansion at King's Mill.

Monitor wrote to Ms Doucet on 21 September saying it was "extremely concerned by the very serious issues facing the trust".

King's Mill Hospital admits breast cancer test errors

8 October 2012

Breast cancer treatment at a hospital in Nottinghamshire is to be reviewed after problems with tests on 120 women.

Seventy-nine women are being recalled with regard to the results of breast tissue biopsies between 2004 and 2010.

Hospital officials have said the fault is due to a technical issue and not medical error.

The trust apologised to those directly affected and other patients worried by the news

Monitor has already raised concerns over the financial viability of the trust because of the rising costs of a private finance initiative to rebuild King's Mill Hospital.

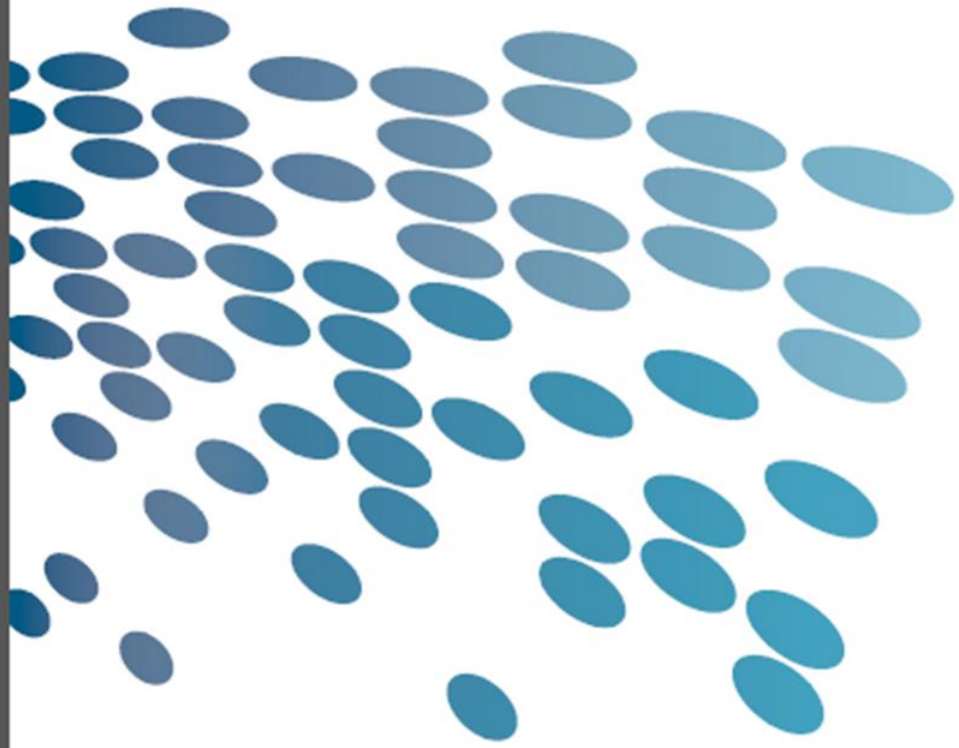
It stepped in and appointed Chris Mellor as interim chairman following the resignation of Tracey Doucét as trust chairman on Thursday.

Analysis:

Suspicions were first raised about the reliability of the tests in late 2010 when it was noticed that King's Mill had different results compared to other hospitals.

King's Mill has now stopped processing these tests and samples are sent to Nottingham's Queen's Medical Centre.

Pathology Quality Assurance Review



Chaired by Dr Ian Barnes

January 2014

Individual performance

4.32. The professional bodies, led by RCPath, should develop methodologies for assessing the performance of individuals in EQA schemes that will give a fair and accurate picture of their competence to practice.

4.33. All practicing individuals responsible for reporting pathology results and providing clinical advice should be registered with current EQA individual assessment schemes and demonstrate regular participation as defined by the JWGQA.

They should achieve appropriate levels of performance as determined by the professional bodies. Performance in individual schemes should be discussed and noted at annual appraisal.

4.34. Where opportunities or a need to improve are identified, additional remedial training should be required, or practice in the area of concern should be stopped until appropriate retraining has been undertaken and revalidation achieved. This process should be noted formally as part of governance procedures, with support from the employing organisation.

4.35. EQA schemes are designed to assess and improve individual performance and employing organisations should ensure that resources are made available to support participation and remedial action if required.

4.36. Provider organisations and professional bodies should ensure that individuals understand that EQA schemes are designed to assess and improve individual performance, and that attempts at collusion are considered matters of professional probity.

Interpretive EQA schemes in the future

- Separated from analytical EQA schemes,
- Re-named Personal Proficiency Assessment
- New regulatory framework through RCPath
- ? Requirement for accreditation of schemes through ISO 17043
- Opportunity for digital slides, web-based, more ‘professionally run’ schemes
- Strengthen their role in improving standards and consistency of reporting – as long as the regulation remains proportional.

Overview:

What makes a good doctor? – experience, aptitude, motivation

A good diagnosis. An error or near-miss

- History of interpretive EQA schemes in the UK
  - Why did they start?
  - Governance
  - How do they work
  - Being a scheme organiser

30 years of evolution

- UK National Liver Histopathology EQA Scheme
  - How it's run
  - Examples of cases

Educational
Identifies weaknesses

- Future of interpretive EQA schemes

Thank you.