Festschrift for Prof Hubscher:

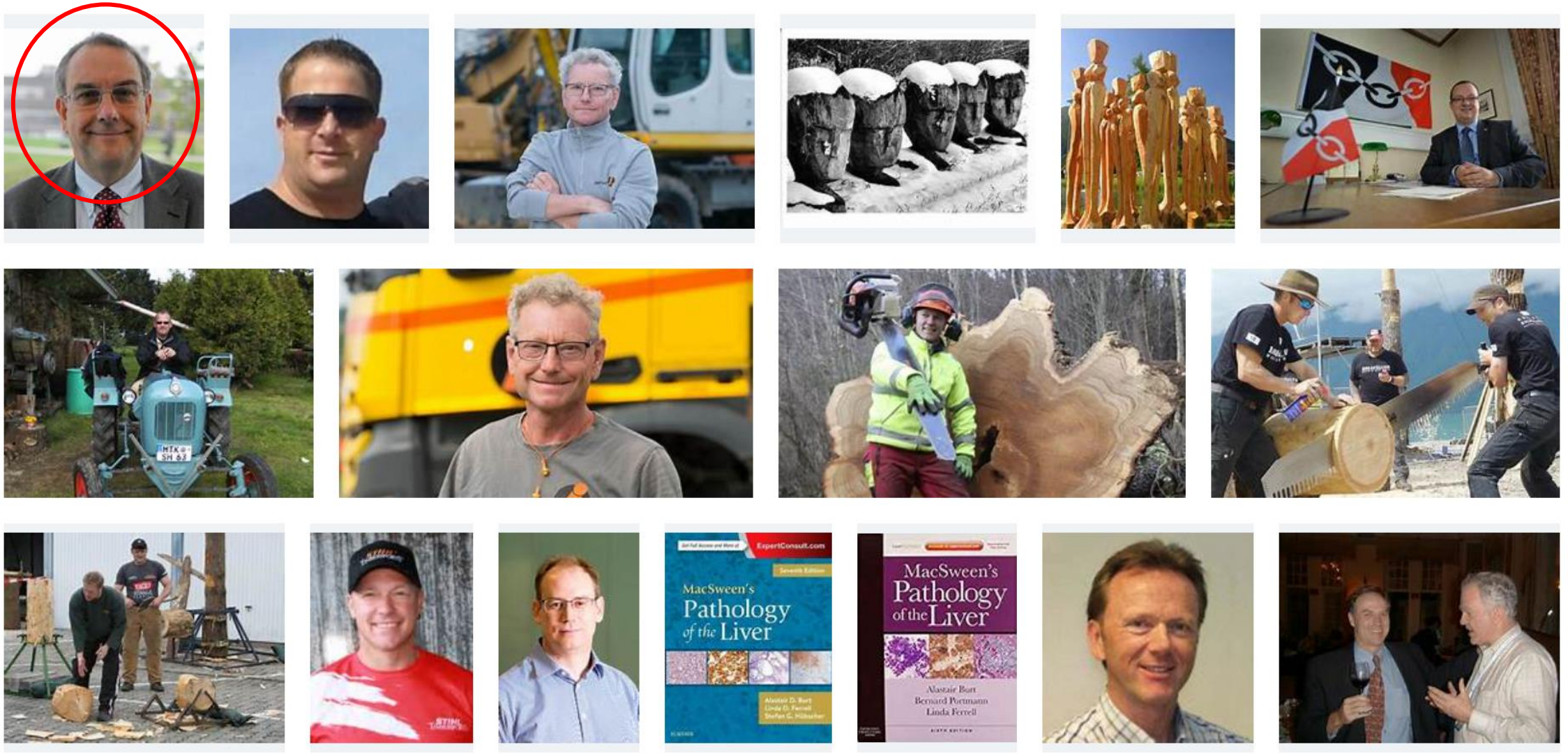
Quality Assurance in Liver Pathology

Judy Wyatt

May 2022

Stefan Hubscher - images

Recognised a familiar set of features – in appropriate context – reach a diagnosis



Born in 1956……

Biopsy findings in cases of rejection of liver allograft.

Hubscher SG, Clements D, Elias E, McMaster P.

J Clin Pathol. 1985 Dec;38(12):1366-73. doi: 10.1136/jcp.38.12.1366.

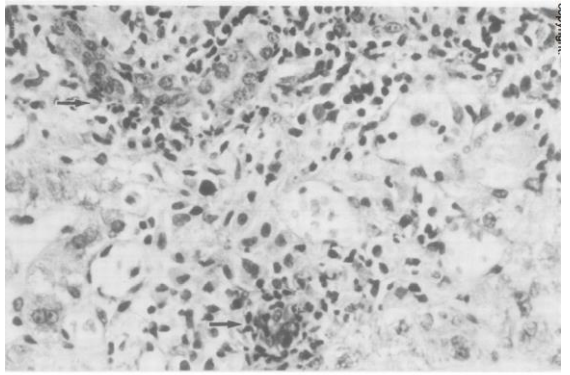


Fig. 1 *Biopsy specimen 15 days after transplantation (case 2) showing acute rejection. Mixed portal inflammatory infiltrate includes polymorphonuclear leucocytes surrounding and infiltrating epithelium of small bile ducts (arrowed). (Haematoxylin and eosin.) × 450.*

Liver membrane antibodies in alcoholic liver disease: 1. prevalence and immunoglobulin class.

Burt AD, Anthony RS, Hislop WS, Bouchier IA, MacSween RN.

Gut. 1982 Mar;23(3):221-5. doi: 10.1136/gut.23.3.221.

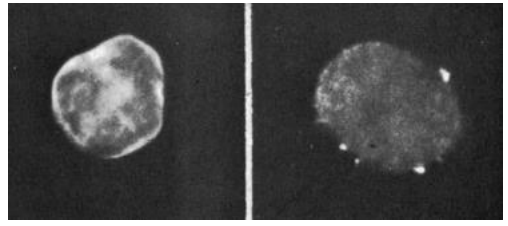


Fig. 1 *(a) Positive immunofluorescence staining of rabbit hepatocyte by a serum containing liver membrane antibody lass; note the continuous staining pattern ntensity at the periphery of the cell.*

IgA deposition in alcoholic liver disease.

Goldin RD, Cattle S, Boylston AW.

J Clin Pathol. 1986 Nov;39(11):1181-5. doi: 10.1136/jcp.39.11.1181.

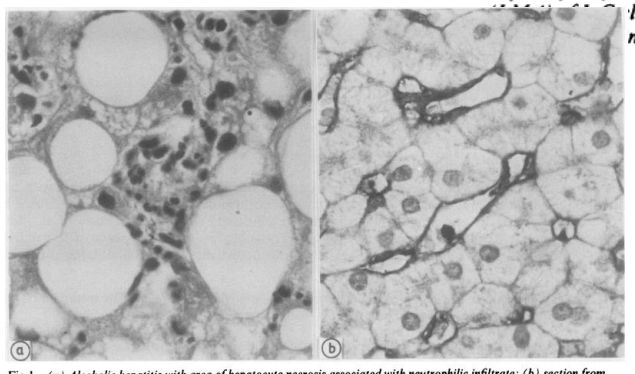
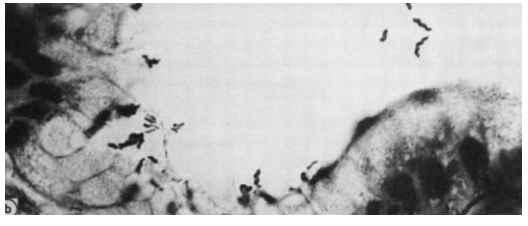


Fig 1 *(a) Alcoholic hepatitis with area of hepatocyte necrosis associated with neutrophilic infiltrate; (b) section from same biopsy specimen stained by indirect immunoperoxidase technique for IgA, illustrating continuous, sinusoidal pattern of immunoglobulin deposition.*

Local immune response to gastric Campylobacter in non-ulcer dyspepsia.

Wyatt JI, Rathbone BJ, Heatley RV.

J Clin Pathol. 1986 Aug;39(8):863-70. doi: 10.1136/jcp.39.8.863.



Quality assurance
– proactive, a system of measures to identify where there is potential error
and do something to avoid it

(v. quality control – reactive, identify errors/flaws
and make a change to prevent recurrence)

1. History of external quality assessment in cellular pathology

2. The UK Liver Histopathology EQA scheme

3. What else makes a difference?

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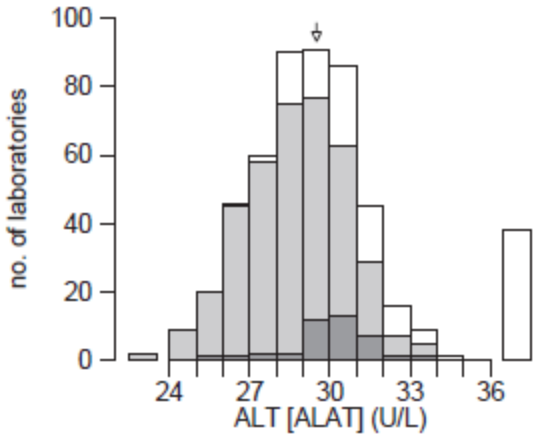
EQA schemes in laboratory medicine

Technical EQA – by laboratory:

Specimen : 993A	n	Mean	SD	CV(%)
Dry slide	33	43	3	7.8
OCD (J&J) slides [1JJ]	33	43	3	7.8
TRIS buffer with PLP [IFCC]	87	31	2	6.4
Abbott [10AB]	26	31	2	6.0
Roche Cobas/Modular [10BO]	51	31	2	5.1
Siemens (Dade Behring) [10BE]	6	39	3	6.6
TRIS buffer without PLP	390	29	2	6.7
Abbott [4AB]	110	29	2	5.9
Beckman (Olympus) [4OL]	57	31	1	4.3
Beckman [4BK]	20	30	1	4.5
Randox [4RX]	4	28		
Roche Cobas/Modular [4BO]	156	28	2	5.8
Siemens (Bayer) [4TE]	40	31	1	4.1



Your result	30
Target value (TRIS buffer without PLP)	29
Standard Uncertainty	0
Your specimen:	
%bias	+2.7
Accuracy Index	41
Method Principle mean [GLTM]	29
Method mean [MLTM]	31

Interpretive EQA:

Apply principles of technical EQA to histopathology – following diagnostic scandals in 1980's

Slide clubs – pathologists get together to discuss interesting cases – self help to avoid professional isolation

EQA schemes – rank performance, identify individual areas of weakness, and overall poor performers

Participation in EQA scheme - part of laboratory accreditation and appraisal

‘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 CS, a consultant pathologist for ‘unacceptably high level of misdiagnoses in the Bone Tumour Centre at Birmingham’s Royal Orthopaedic Hospital’

‘Dr S, 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 S’.

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……

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

A report not a result

- 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 concern

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, evidence based guidelines
- Know your limits – subspecialisation
  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

What do doctors do? – make decisions in the face of uncertainty
– use individual professional judgement.

Enable members to compare their diagnostic opinions with other pathologists reporting similar specimens.

Avoid professional isolation. Find your areas of weakness.

Individual based Interpretive EQA Schemes:

- Started with structure of technical EQA schemes
  - slide circulation, collect responses, compare diagnoses

Quorate meeting of members, discuss results, agree on how cases should be scored

Rank results, feedback on performance

Highly educational

- Lowest 2.5% = substandard performer.
- All anonymously by participant number, confidentiality assured.
- If substandard performer in 2 of 3 consecutive circulations – first action point
  alerted and monitored
- If substandard performer in 2 of the next 3 circulations – second action point
  – inform College, Responsible Officer

The Royal College of Pathologists
Pathology: the science behind the cure

EQA SCHEME	Website / email contact	annual report 2020-reviewed April 20
Breast Pathology	contact@nccbp.com	Y
Dermatopathology	www.nsdeqa.co.uk	Y
East Midlands General Histopathology EQA	EQAAdmin@uhl-tr.nhs.uk	Y
Gastrointestinal Pathology	leedsth-tr.gi.eqa@nhs.net	Y
Bowel cancer screening programme (BCSP)	phe.bowel-eqa@nhs.net	Y
Head & Neck & Oral	www.bsomp.org.uk; www.histopathologyeqa.org headandneckeqa@gmail.com	Y
National Cervical Cytology	http://csp-geqa.phe.nhs.uk/ phe.csp-geqa-admin@nhs.net	Y
National Gynaecological EQA	EQAAdmin@uhl-tr.nhs.uk	Y
National Liver Pathology	http://www.virtualpathology.leeds.ac.uk/eqa/specialist/liver/liver_circulations.php kara.o'connell@nhs.net	Y
National Musculoskeletal	portia.baines1@nhs.net	Y
National Renal	please contact RCPath for address	Y
Neuropathology	antonia.torgersen@nhslothian.scot.nhs.uk	Y
Non-Gynaecological Cytology	www.ukneqascpt.org.uk cpt@ukneqas.org.uk	Y
North West Region Histopathology EQA Scheme	http://www.virtualpathology.leeds.ac.uk/eqa/general/northwest joanne.leggott@srft.nhs.uk	Y
North West Thames General Histopathology EQA	penelopeanne.thorpe@nhs.net	Y
NorthEast England Regional Surgical Histopatholo	Susan.Teggert@nhs.net	Y
Ophthalmic	http://eyepath.org.uk/resources/ louise.simeon@ggc.scot.nhs.uk	Y
Paediatric Histopathology	sophiestenton@nhs.net	Y
Scotland & N Ireland Histopathology EQA	http://www.pathologyscotland.org/quality/ linda.mcdonald3@nhs.scot	Y
South East England	https://www.mtw.nhs.uk/gps/pathology/se-england-general-histopathology-eqa-scheme/	Y
Thames Valley General Histopathology EQA	caroline.graham7@nhs.net or angus.molyneux@mkuh.nhs.uk	Y
Uropathology EQA scheme	http://www.histopathologyeqa.org/ eqaadmin@uhl-tr.nhs.uk	Y
Wessex & SW England	via RCPath	Y
Yorkshire General Histopathology EQA	Yorks.eqa@nhs.net	Y

Specialist histopathology interpretive EQA Scheme
General histopathology interpretive EQA scheme
Cytopathology interpretive EQA Scheme

NATIONAL QUALITY ASSURANCE ADVISORY PANEL IN CELLULAR PATHOLOGY

External quality assurance (EQA) is a key technique through which pathologists and the laboratories they work in can assure themselves, their users, the wider health service and the public that their work is of an appropriate quality and is in line with their peers nationally. This is achieved through comparison of their report for each test case with those of other individuals or laboratories.

Individual based interpretive EQA Schemes in Cellular Pathology - Schemes which have submitted annual reports to the Royal College of Pathologists for 2020:

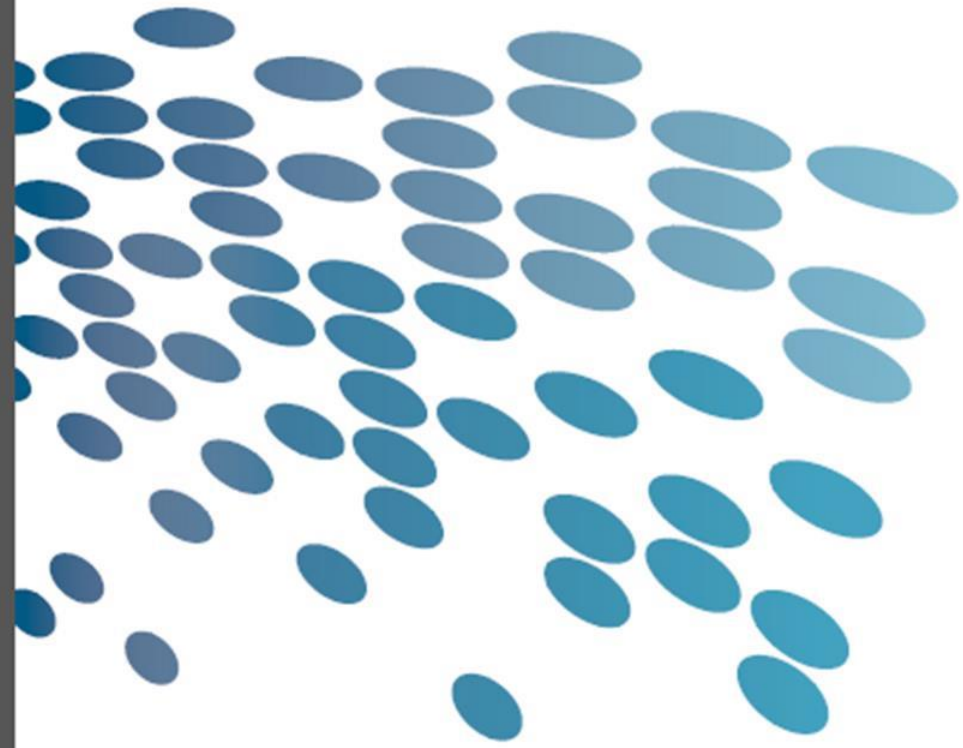
Cellular Pathology - report for National Quality Assurance in Pathology Committee,
Report for activity in 2020.

	general schemes	specialist schemes	total all schemes
schemes submitting report	9	13	22
total circulations in 2020	11	18	29
total members	1001	3099	4103
members per scheme	32-170	29-690	29-690
median members per scheme	103	188	186
circulations in 2020	11	18	29
participants	1307	3797	5104
% participation	89%	89%	89%
cases circulated	141	205	346
% used for scoring	95%	91%	93%
low score in a circulation	40	53	93
first action point	2	9	11
second action point	0	0	0

29/44 (66%) of expected,
assuming 2 circulations per scheme per year

61,248 cases with a response submitted

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.

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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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- 2014 100 members. Annual meeting.
- On line submission of responses.
- 'Masterclass' presentation on problematic cases

Governance of EQA schemes – who makes sure they're run properly?

2015 – changed to EQAlite – software that supports EQA schemes

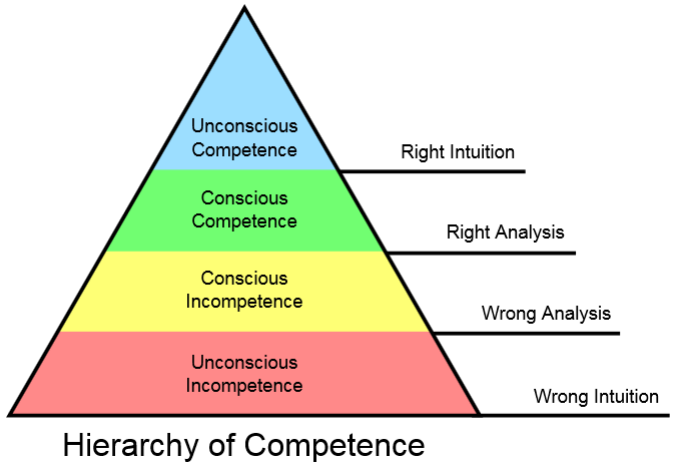
2017 – 109 members.
New deputy (Rachel Brown);
quality subcommittee does collation of responses

2020 – changed to drop down menus,
Less subjective, streamline collation of responses

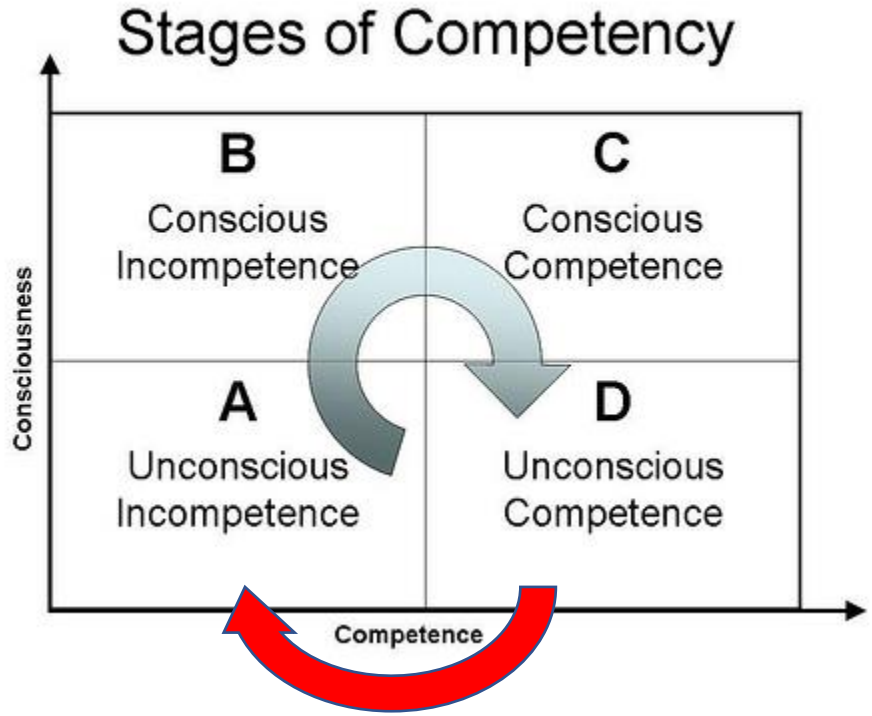
2021 – 120 members – Zoom meetings

Benefits of Interpretive EQA Schemes

- Compare your competence with peers in a safe environment, identify areas of weakness,

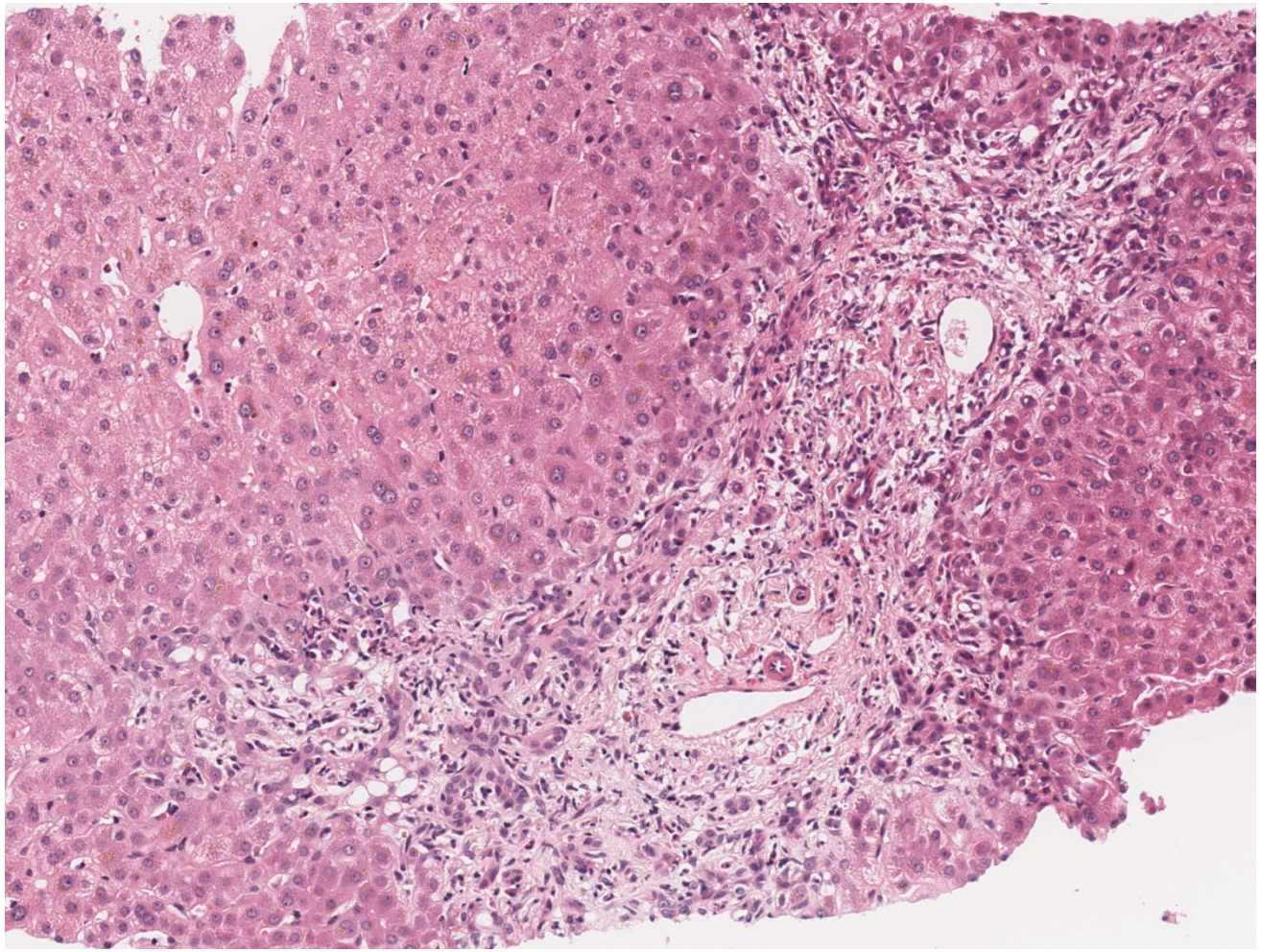


Hierarchy of Competence



Circulation N 2003: case 177 Female 50 years

Cholestatic jaundice on atorvastatin . Also nephrotic



44 participants – 10 points each

Cholestasis associated with drug	128
Hepatitis – drug related	33
Biliary obstruction	116
Portal reaction c/w drug but exclude duct obstruction	40
Chronic obstructive biliary disease	20
Biliary disease, drug or obstruction	45
? Chronic hepatitis + drug induced cholestasis	10
Cirrhosis ? Cause	5
Sepsis	12
Ductopenia and cholangiolitis ? PBC	10
Ductopenia and cholangiolitis ? Drugs/PBC	10
Cholangiolitis and paucity of ducts ? Drugs	10
Difficult – no answer	10

Case 177 comments:

Several: Biliary features

?does atorvastatin cause LBDO like changes

several: ?ductopaenia

Few – advise imaging, looks like ?PSC/PBC

Follow up: *Dr Ansell:* – *no duct obstruction found but still had high alkaline phosphatase 9 months later. Nephrotic syndrome reduced to minimal change nephropathy. Investigation of biliary tree shows no obstruction*

- Accepted diagnoses are any that mention need for investigation of biliary tree; attributing these features to the drug without raising the possibility of duct obstruction or some form of (non-drug related) chronic biliary disease is not an accepted diagnosis.

Case 177 Discussion

- ?role of statin in cholestatic liver disease.
- The statins recognised to cause hepatitis, although low risk, not associated with cholestatic injury. This biopsy shows features suggestive of biliary disease; clinicians know the patient is on statin.
- Response to this biopsy should be to recommend investigation to exclude large duct obstruction or chronic biliary disease.

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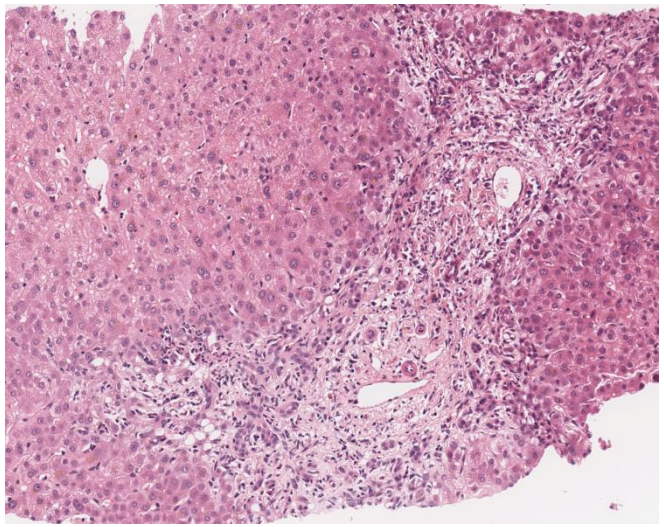
168/440 cholestasis

221/440 obstruction

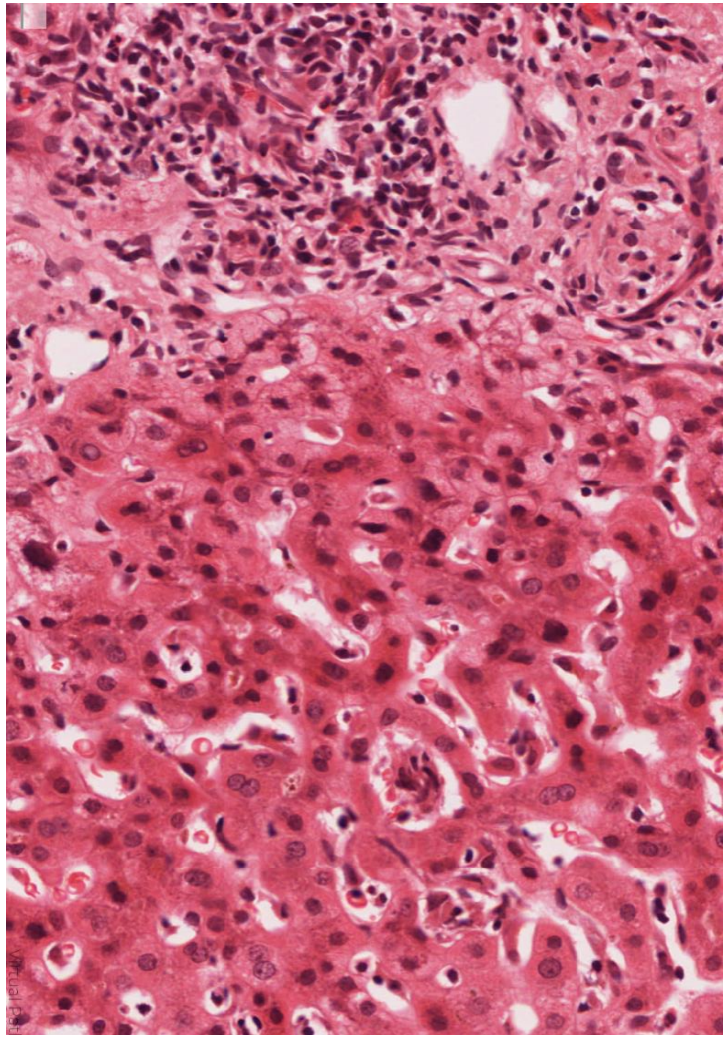
As far as possible, the conclusion of the histology report should be the same whoever has reported it – and be clinically helpful in arriving at a diagnosis.

- What we see



- How we integrate it into a disease pattern
- Many recognised a biliary pathology
- Could atorvastatin cause this??

- How we integrate that into a clinically useful report
- Investigate the biliary tree!



2004: 47 participants – how to classify responses?

Case 185: Unwell approximately 4 weeks following return from Corfu. Took herbal remedies. Also has been on Minocyclin, Bilirubin 436, ALP 259, AST 338, GGT 19, INR 0.89, immunoglobulins normal, autoantibodies – ANA 1: 60, ASM 1: 160, ?autoimmune, ?secondary to Minocyclin

Summary of Responses:

Morphological diagnosis only

acute hepatitis or cholestatic hepatitis – 40

Morphology +aetiology

possible, probable, or unqualified drug induced hepatitis – 226

hepatitis – drug or autoimmune – 70

chronic hepatitis probably drug – 10

Aetiology only

autoimmune hepatitis – 5

PBC – 6

Comments on unusual mononuclear infiltrate:

?HBV haematological disorder, lymphoproliferative, Kupffer cell hyperplasia – 5

Other c

chronic rea

florid re

no diag inv

Absence of consensus therefore case excluded from scoring

Follow up information from Bernard Portmann:

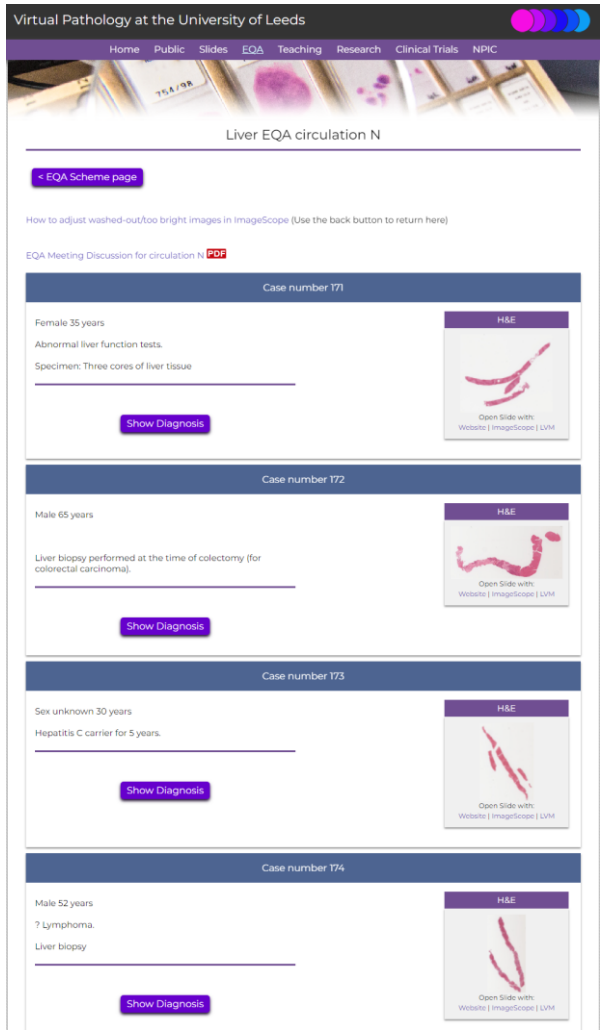
Clinicopathological diagnosis: Hepatitis with autoimmune features associated with minocyclin. There was no known haematological condition. There was no known biliary disease.

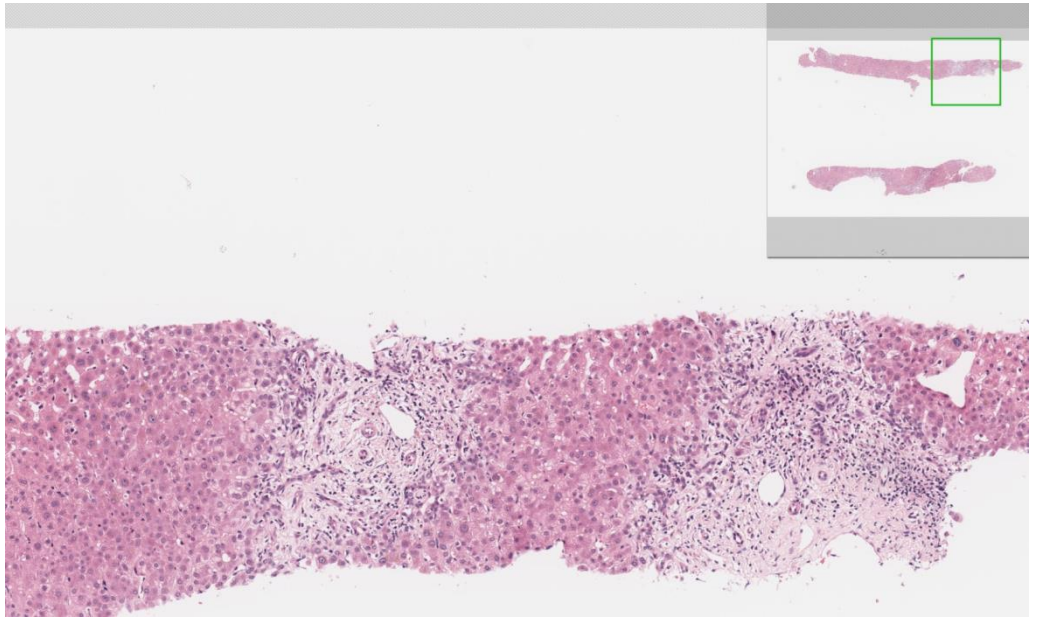
She made a slow but complete recovery over 2 months, no steroids were given.

There is a recognised association between minocycline and autoimmune hepatitis – with female preponderance, hypergammaglobulinaemia and antinuclear and smooth muscle antibodies

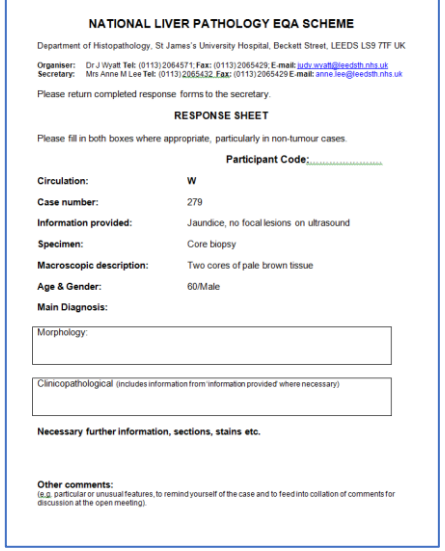
How to classify and present responses? Need morphological and clinicopathological assessments

IT to the rescue – having a website





EQAlite from 2015



NATIONAL LIVER PATHOLOGY EQA SCHEME

Department of Histopathology, St James's University Hospital, Beckett Street, LEEDS LS9 7TF UK

Organiser: Dr J Wyatt **Tel:** (0113)2065471; **Fax:** (0113)2065429; **E-mail:** judy.wyatt@leedsth.nhs.uk
Secretary: Mrs Anne M Lee **Tel:** (0113)2065432 **Fax:** (0113)2065429 **E-mail:** anne.lee@leedsth.nhs.uk

Please return completed response forms to the secretary.

RESPONSE SHEET

Please fill in both boxes where appropriate, particularly in non-tumour cases.

Participant Code:.....

Circulation:	**W**
Case number:	279
Information provided:	Jaundice, no focal lesions on ultrasound
Specimen:	Core biopsy
Macroscopic description:	Two cores of pale brown tissue
Age & Gender:	60/Male
Main Diagnosis:	

Morphology:

Clinicopathological (includes information from 'information provided' where necessary)

Necessary further information, sections, stains etc.

Other comments:
(e.g. particular or unusual features, to remind yourself of the case and to feed into collation of comments for discussion at the open meeting).

Surveymonkey from 2011

Please enter a response in each box, i.e. an morphological and clinicopathological assessment of each case.

Please be concise, write the equivalent of a 'bottom line' diagnosis rather than a detailed descriptive report.

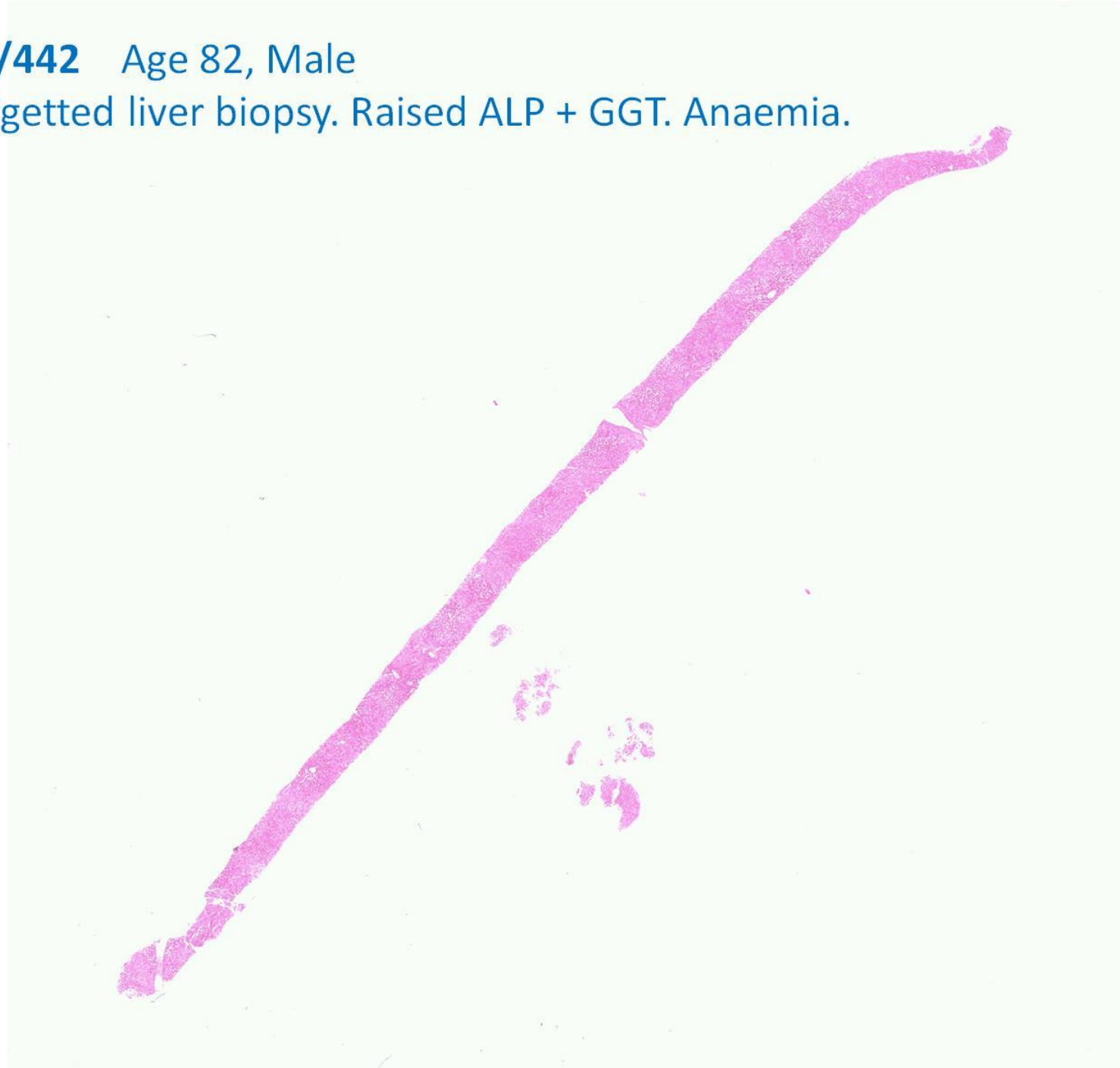
Case number D1/362 Female,48
clinical information:Gallstones,cholecystitis,lesion gall bladder bed. MRI benign.
nature of specimen:Liver resection,lobulated liver nodule 3.5x2.5x2.5cm

Morphological assessment: 0

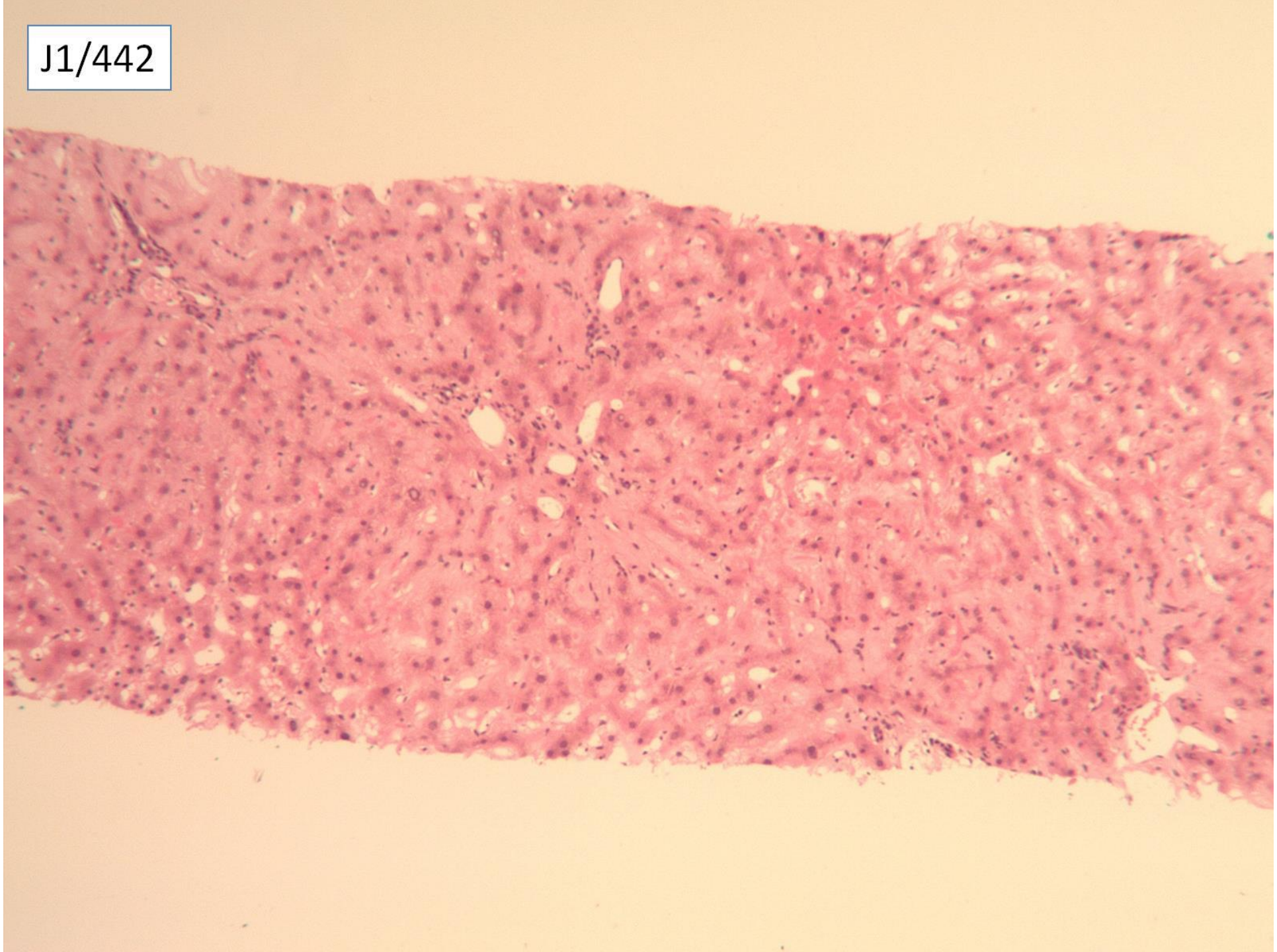
clinicopathological diagnosis: 0

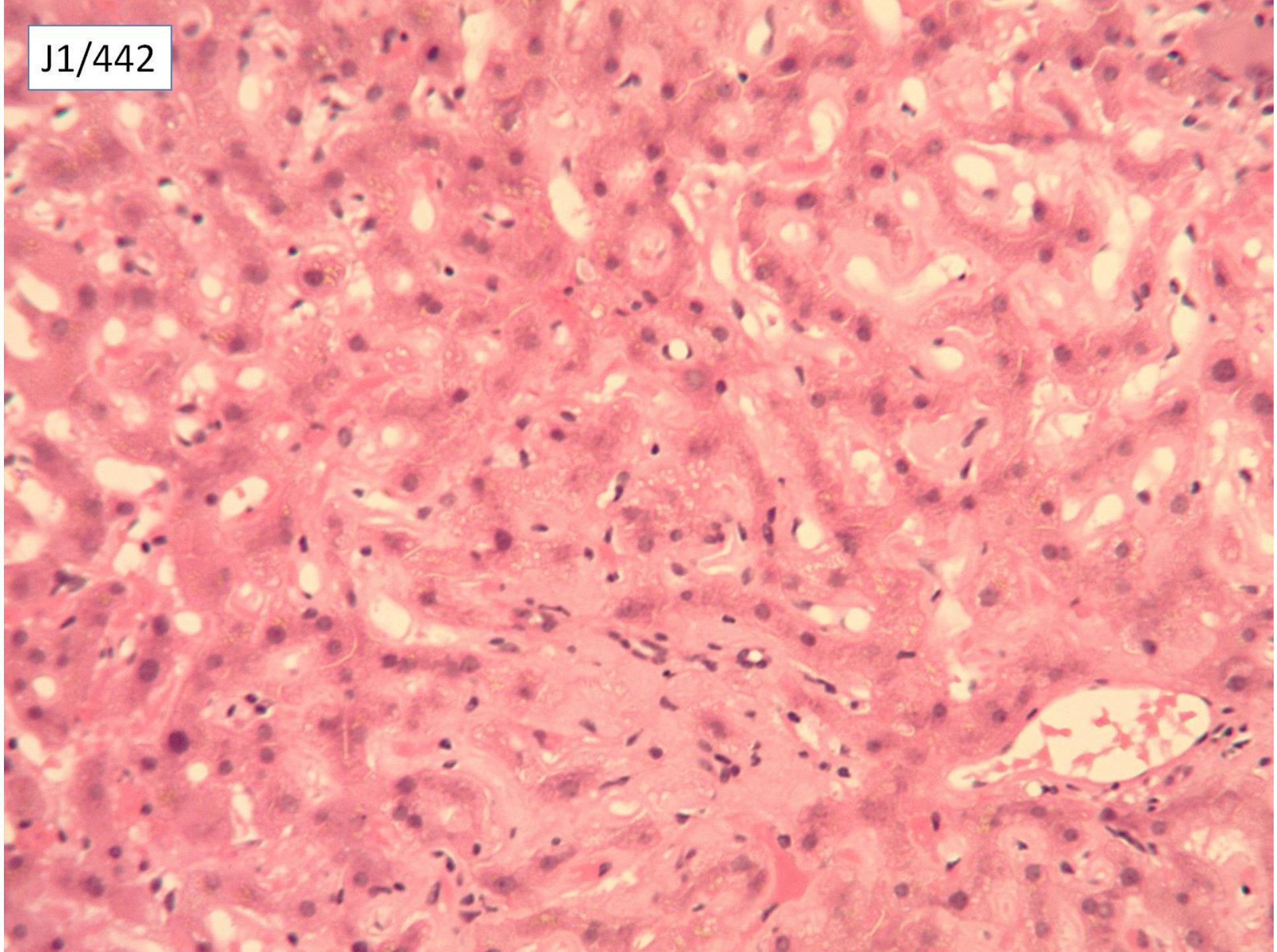
Case J1/442 Age 82, Male

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



Spring 2014:
82 participants




J1/442

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

Spring 2014: 82 participants

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

For 10 points: amyloid as only or main diagnosis

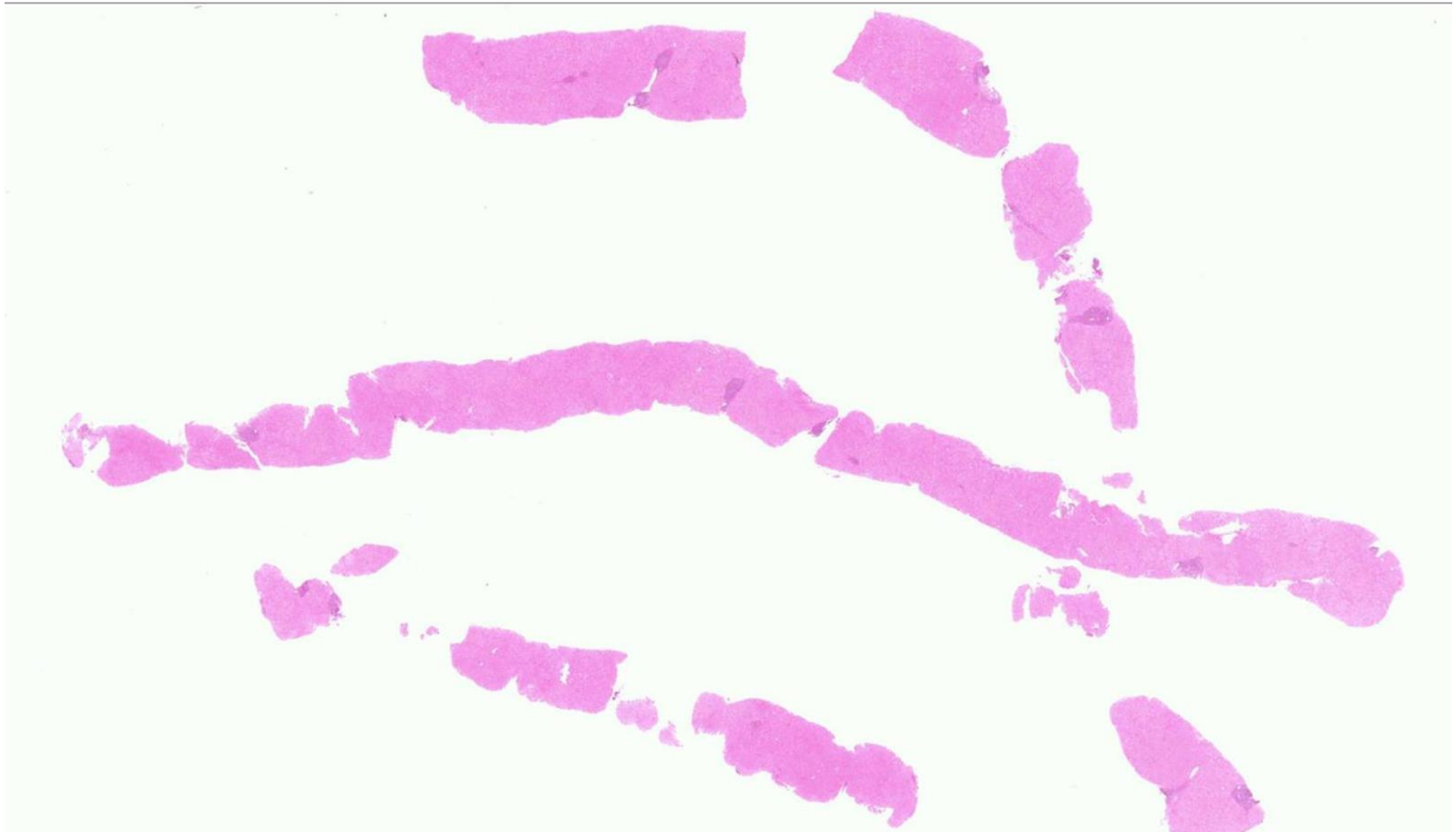
Amyloid in differential but not first – score 5 points

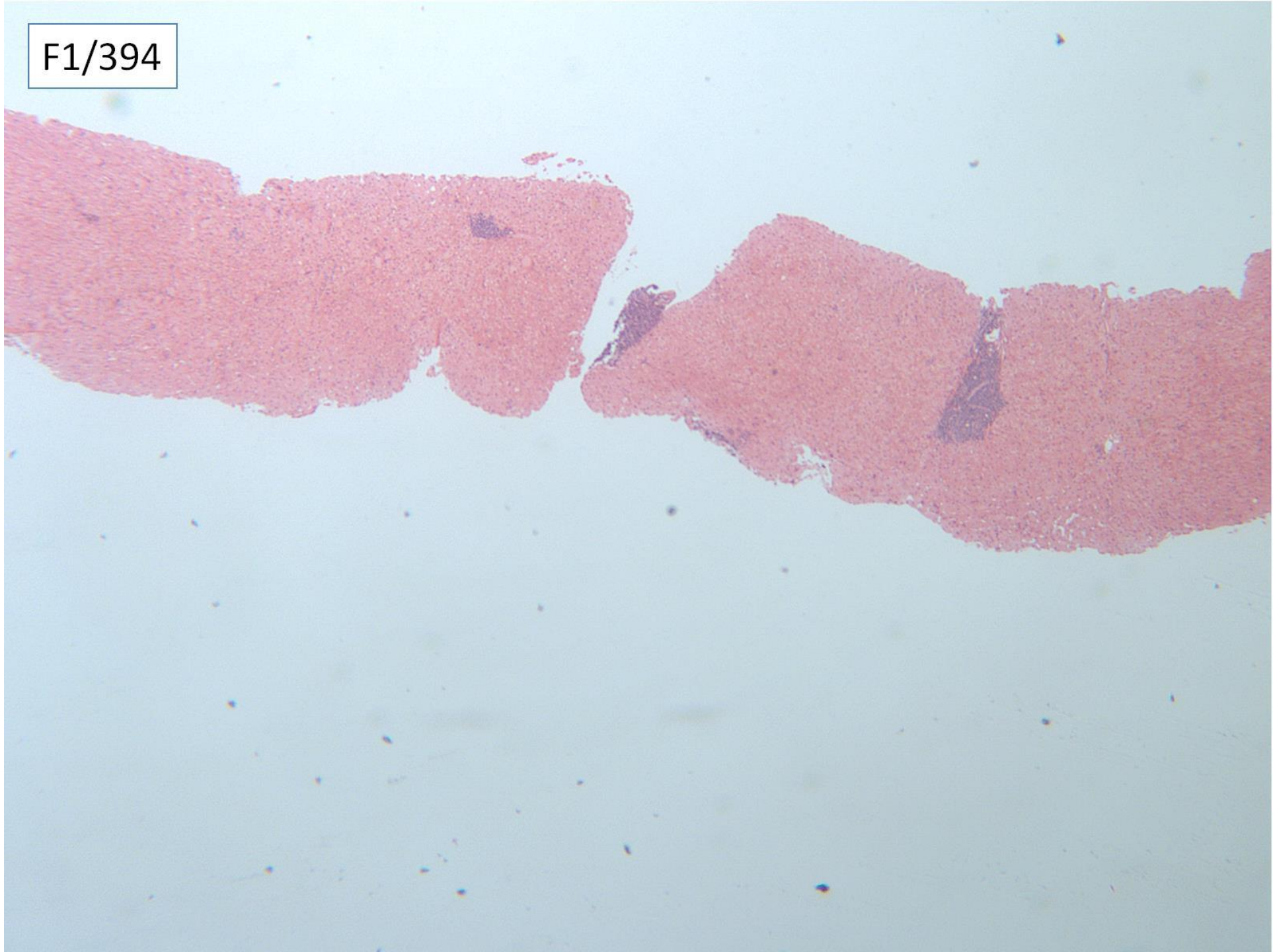
No mention of amyloid – score 0 points

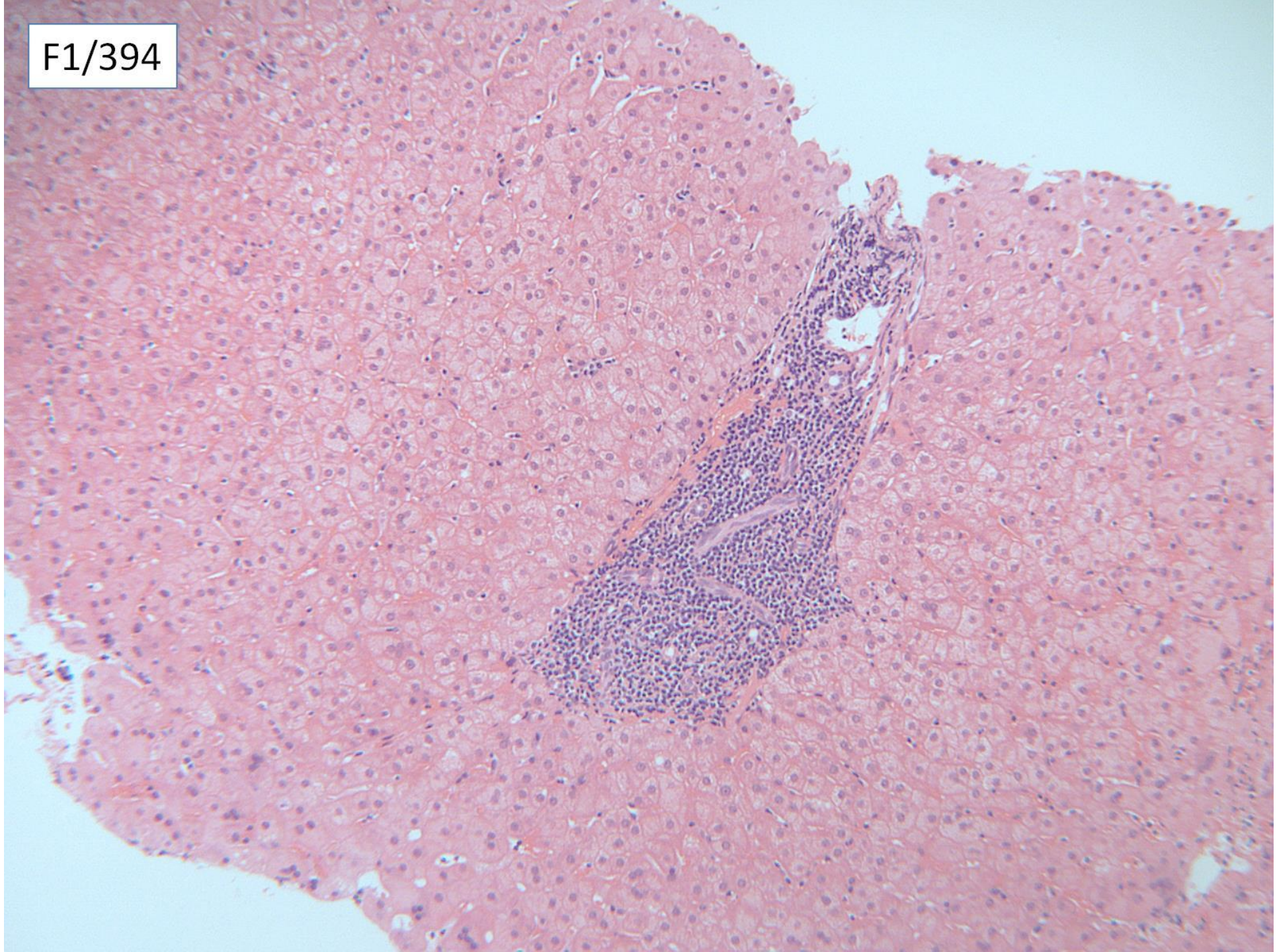
Spring 2012: 81 participants

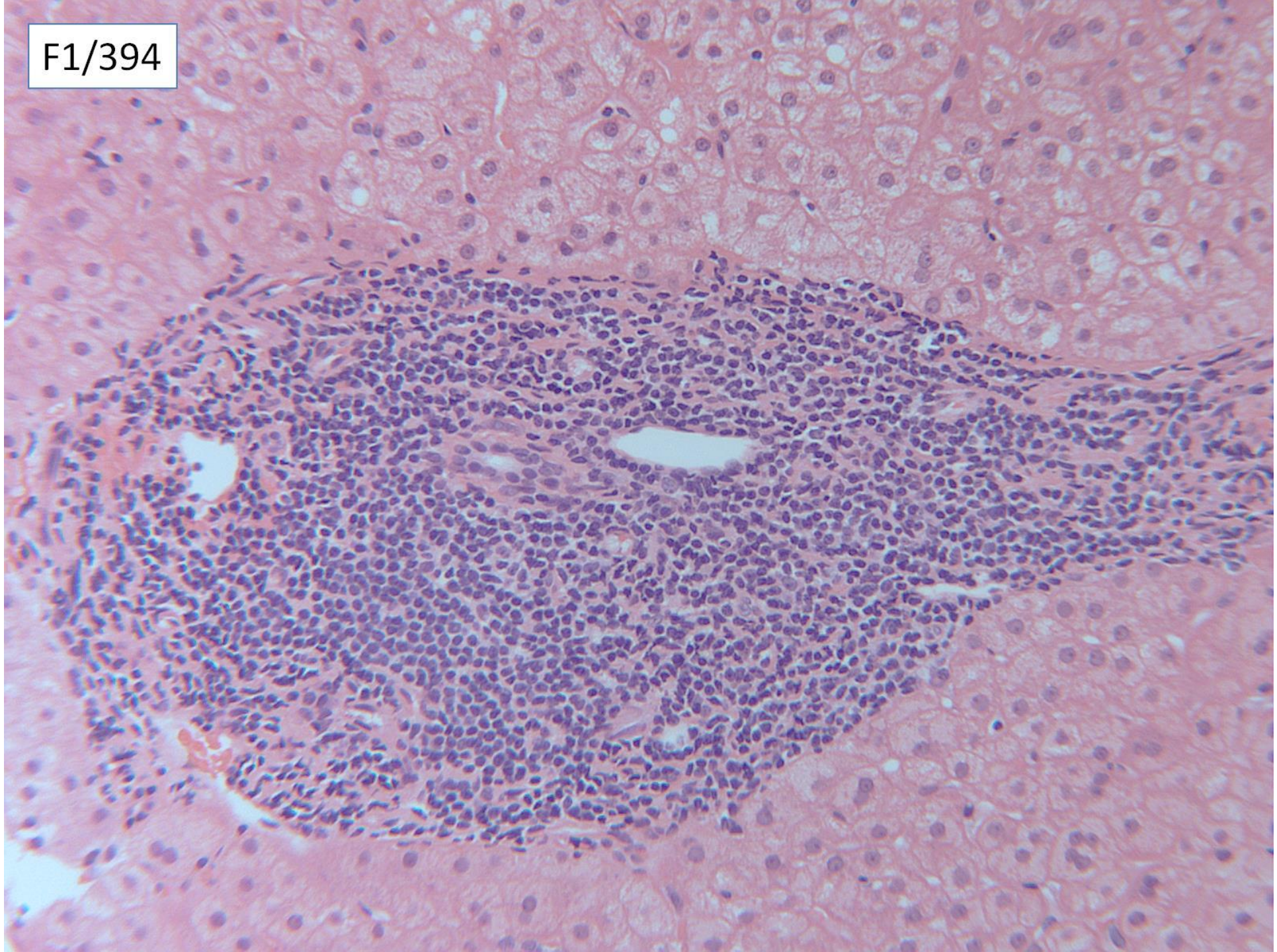
EQA Case F1/ 394 58 M

HCV genotype, RNA –ve after treatment




F1/394


F1/394


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.

Spring 2012: 81 participants

Case 15: EQA Case 394: 58M,

HCV genotype, RNA –ve after treatment

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

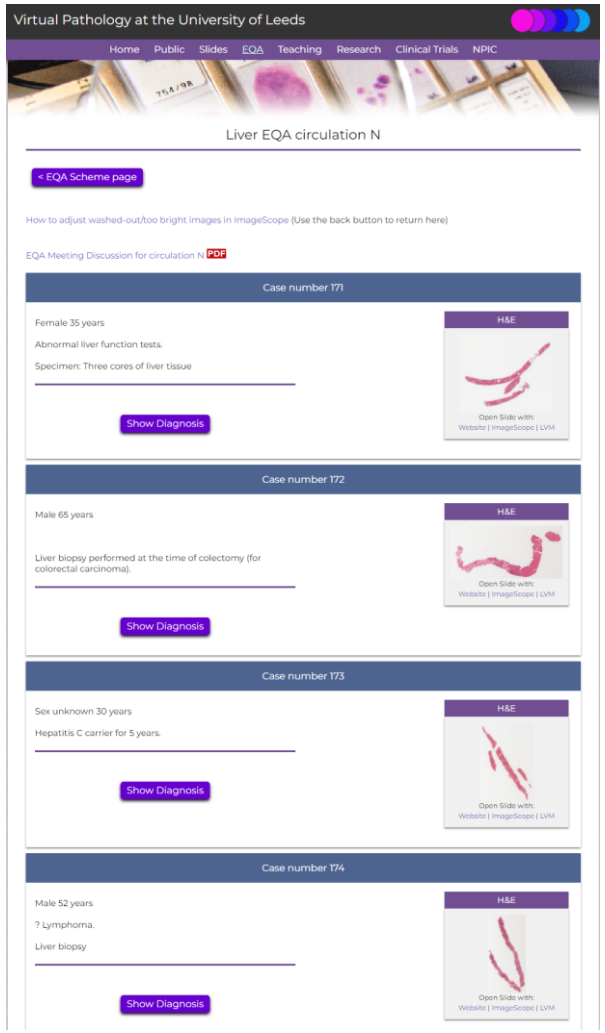
Suggested scoring
No consensus,
not suitable for scoring.

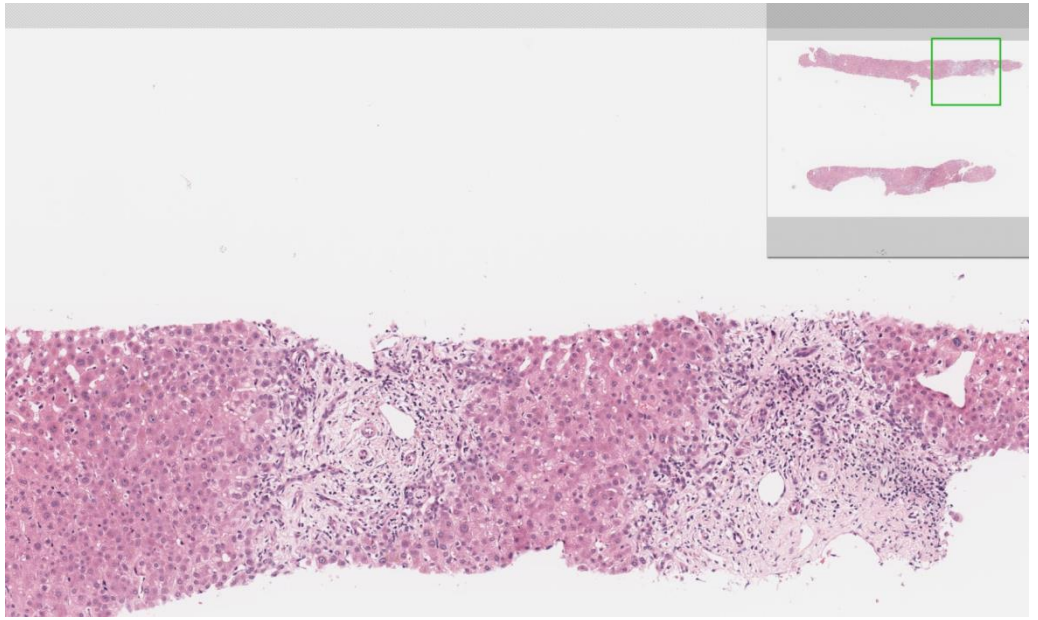
Very interesting result – original diagnosis was lymphoma, but clinical details frame the problem as a case of hepatitis C.

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

IT to the rescue – having a website





EQAlite from 2015

NATIONAL LIVER PATHOLOGY EQA SCHEME

Department of Histopathology, St James's University Hospital, Beckett Street, LEEDS LS9 7TF UK

Organiser: Dr J Wyatt **Tel:** (0113)2064571; **Fax:** (0113)2065429; **E-mail:** judy.wyatt@leedsth.nhs.uk
Secretary: Mrs Anne M Lee **Tel:** (0113)2065432 **Fax:** (0113)2065429 **E-mail:** anne.lee@leedsth.nhs.uk

Please return completed response forms to the secretary.

RESPONSE SHEET

Please fill in both boxes where appropriate, particularly in non-tumour cases.

Participant Code:.....

Circulation:	**W**
Case number:	279
Information provided:	Jaundice, no focal lesions on ultrasound
Specimen:	Core biopsy
Macroscopic description:	Two cores of pale brown tissue
Age & Gender:	60/Male

Main Diagnosis:

Morphology:

Clinicopathological (includes information from 'information provided' where necessary)

Necessary further information, sections, stains etc.

Other comments:
(e.g. particular or unusual features, to remind yourself of the case and to feed into collation of comments for discussion at the open meeting).

Surveymonkey from 2011

Please enter a response in each box, i.e. an morphological and clinicopathological assessment of eac

Please be concise, write the equivalent of a 'bottom line' diagnosis rather than a detailed descriptive re

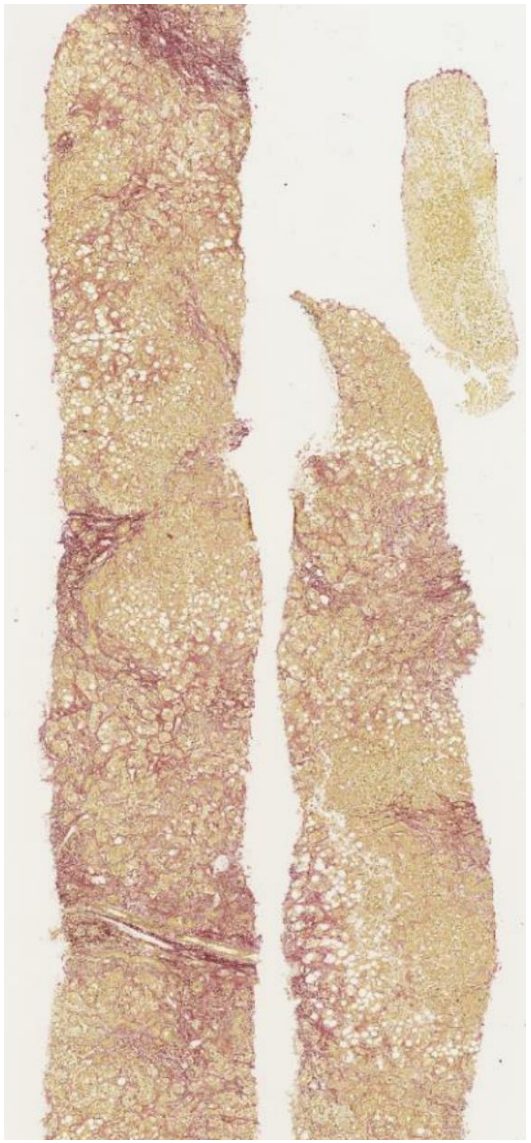
Case number D1/362 Female,48
clinical information:Gallstones,cholecystitis,lesion gall bladder bed. MRI benign.
nature of specimen:Liver resection,lobulated liver nodule 3.5x2.5x2.5cm

Morphological assessment: 0

clinicopathological diagnosis: 0

Drop-down menus from 2020

Circulation LW – pilot drop down menus
2021 – 95/116 participated



Case number 10

Male 38 years

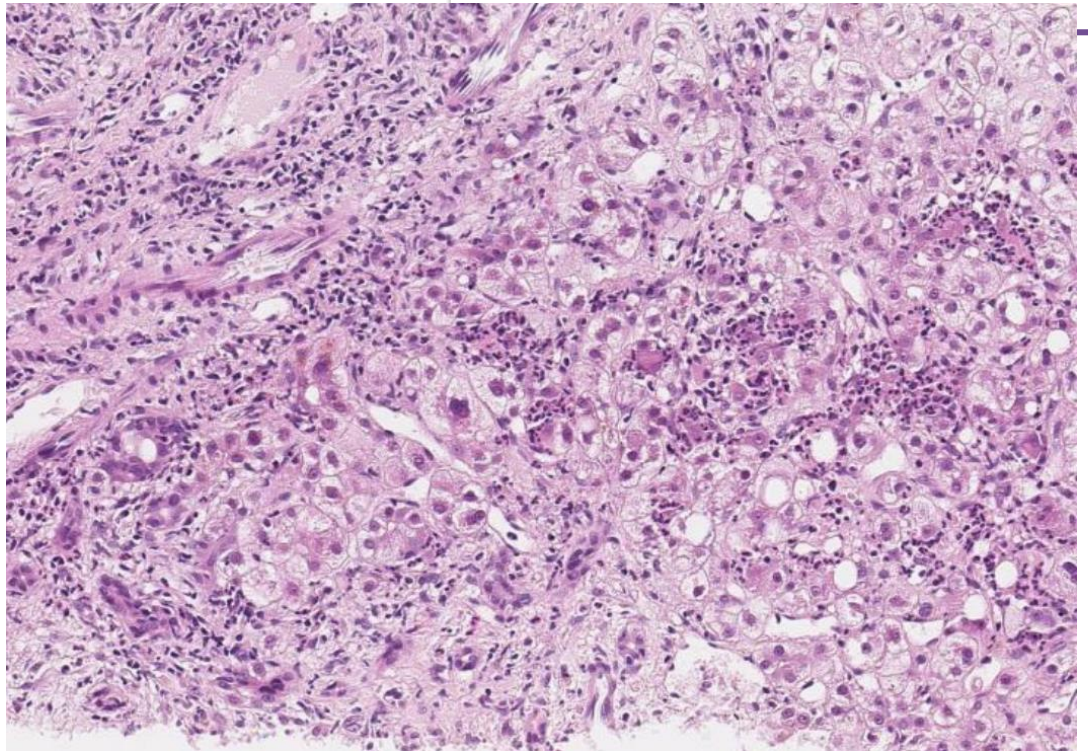
Alcoholic hepatitis on a background of probable cirrhosis. We are considering starting steroids.

Specimen:

Liver biopsy.

Macroscopic description:
2 cores of tissue, 1.5cm + frags.

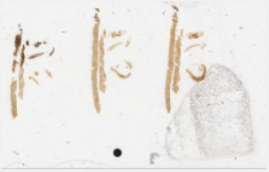
Immunohistochemistry:
EPSR, retic, PASD, Perls, Orcein.



H&E
477606

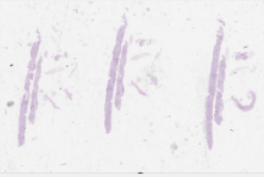
Open Slide with:
Website | ImageScope

Reticulin
477610



Open Slide with:
Website | ImageScope

PAS diastase (PASD)
477611



Open Slide with:
Website | ImageScope

Orcein
477712

Open Slide with:
Website | ImageScope

Clinical: Male 38. Alcoholic hepatitis on a background of probable cirrhosis.

Tumour:	Popularity:
- No tumour/lesion present	97.9%
Other (please specify in Comments)	2.1%

Pattern:	Popularity:
steatohepatitis	92.6%
cholestasis, bilirubinostasis	52.6%
lobular hepatitis	6.3%
steatosis	3.2%
Other (please specify in Comments)	2.1%
not applicable	1.1%

Stages:	Popularity:
advanced fibrosis with bridging and nodularity/cirrhosis	47.4%
fibrosis with bridging between vascular structures	34.7%
hepatocyte loss or bridging - favour collapse not fibrosis	7.4%
Other (please specify in Comments)	7.4%
mild/early fibrosis without bridging	3.2%

Diagnostic categories:	Popularity:
fatty liver disease - alcohol related liver disease	88.4%
Other (please enter alternative diagnosis in comments box)	6.3%
drug induced liver injury (please specify in comments box)	2.1%
fatty liver disease - either alcohol or non-alcohol	2.1%
acute / subacute hepatitis - autoimmune / drug / viral	2.1%
iron overload - acquired, secondary	1.1%
ascending cholangitis	1.1%
fatty liver disease - non-alcohol related fatty liver disease	1.1%

Diagnosis Combination:	Count:
fatty liver disease - alcohol related liver disease	76
[No selections made]	4
fatty liver disease - alcohol related liver disease, Other (please enter alternative diagnosis in comments box)	4
fatty liver disease - either alcohol or non-alcohol	2
Other (please enter alternative diagnosis in comments box)	2
acute / subacute hepatitis - autoimmune / drug / viral	1
acute / subacute hepatitis - autoimmune / drug / viral, fatty liver disease - alcohol related liver disease	1
ascending cholangitis, fatty liver disease - alcohol related liver disease	1
drug induced liver injury (please specify in comments box)	1
drug induced liver injury (please specify in comments box), fatty liver disease - alcohol related liver disease	1
fatty liver disease - alcohol related liver disease, iron overload - acquired, secondary	1
fatty liver disease - non-alcohol related fatty liver disease	1

for 10 points - steatohepatitis, alcohol related and more than mild fibrosis.

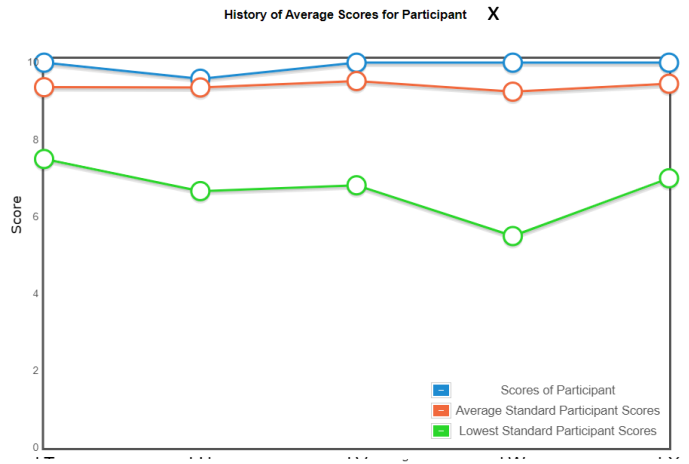
Lose 5 marks for mild/early fibrosis, or bridging/collapse

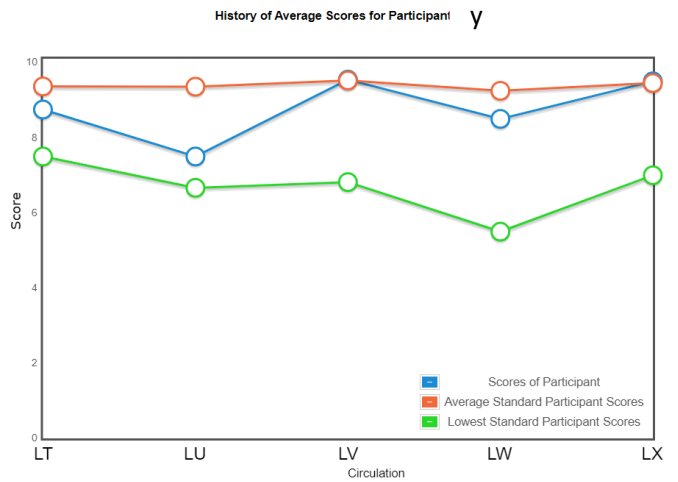
Lose 5 marks for steatohepatitis with no mention of alcohol,

Lose 10 marks (score 0) for lobular hepatitis, cholestasis, DILI, alcohol not mentioned = neither alcohol nor steatohepatitis mentioned.

Benefits of Interpretive EQA Schemes

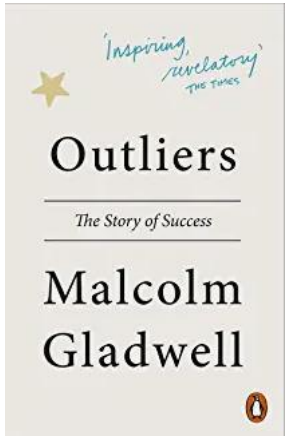
- Compare your competence with peers in a safe environment, identify areas of weakness,
- Avoid professional isolation
- Harmonise diagnostic criteria and terminology
- Awareness of new developments/diagnostic entities





Challenge: to retain the primary value of case discussion as a learning tool,
identifying what pathologists find difficult,
and how approach the problem.

Cases without consensus are learning opportunities – ‘masterclass’ presentations from someone with knowledge and experience



10,000 hours experience with feedback

How do we get better at what we do?

Aptitude x Experience x motivation

Quality assurance
– proactive, a system of measures to identify where there is potential error
and do something to avoid it

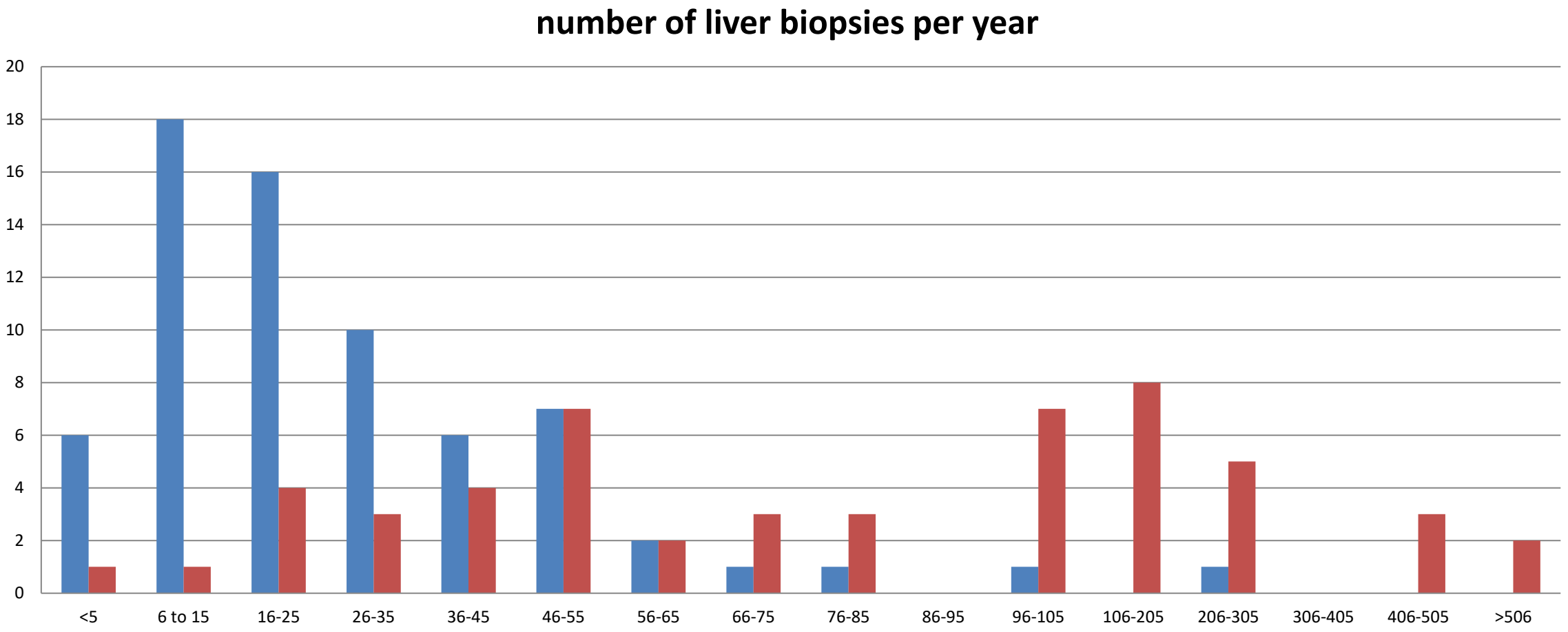
(v. quality control – reactive, identify errors/flaws
and make a change to prevent recurrence)

1. History of external quality assessment in cellular pathology

2. The UK Liver Histopathology EQA scheme

3. What else makes a difference?

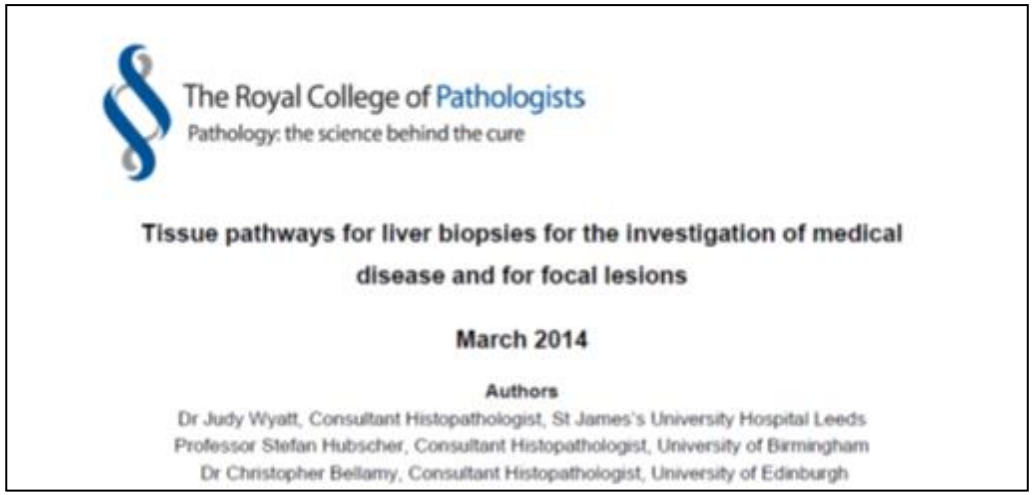
- Good specimen
- Pre and post test – good communication
- Standard practice guidance



Replies from 69 UK consultantsfrom Gloucester GI course in 2011 (in blue), (13 liver EQA members excluded)

and from 53 liver EQA members, 2013 (in red).

Reduce variation within the UK


The Royal College of Pathologists
Pathology: the science behind the cure

Tissue pathways for liver biopsies for the investigation of medical disease and for focal lesions

March 2014

Authors

Dr Judy Wyatt, Consultant Histopathologist, St James's University Hospital Leeds
Professor Stefan Hubscher, Consultant Histopathologist, University of Birmingham
Dr Christopher Bellamy, Consultant Histopathologist, University of Edinburgh

Tissue Pathways, RCPath

Guidelines on – staffing and workload

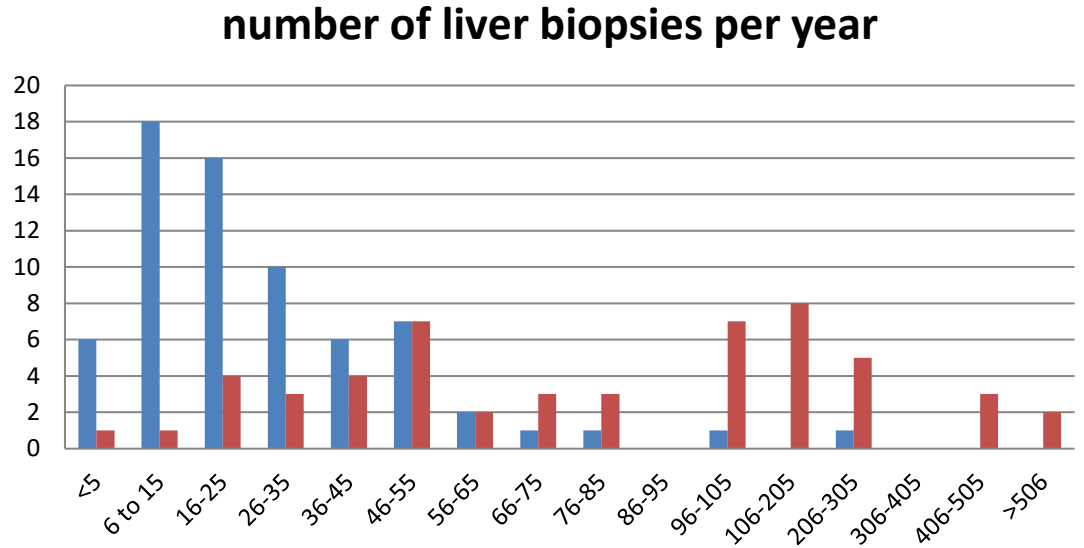
biopsy size, embedding, sectioning, staining

report content

referral, second opinion

Training, continuing professional development

- Lead pathologist for liver, in job plan
- Participates in Liver EQA Scheme



Reporting liver biopsies: Guidelines in 2020

Tissue Pathways, RCPath

Guidelines on – staffing and workload

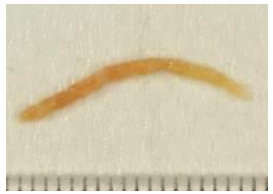
biopsy size, embedding, sectioning, staining

report content

referral, second opinion

Biopsy – a verb if you're a physician

- a noun if you're a pathologist



Essential to have good communication
– what do you want to find out from this biopsy?

The Royal College of Pathologists
Pathology: the science behind the cure

Tissue pathways for liver biopsies for the investigation of medical disease and focal lesions

October 2020

Authors: Dr Judy Wyatt, St James's University Hospital Leeds
Professor Stefan Hubscher, University of Birmingham
Dr Christopher Bellamy, University of Edinburgh
Dr Susan Davies, Addenbrookes Hospital, Cambridge

Guidelines

OPEN ACCESS

Guidelines on the use of liver biopsy in clinical practice from the British Society of Gastroenterology, the Royal College of Radiologists and the Royal College of Pathology

James Neuberger ,[1] Jai Patel,[2] Helen Caldwell,[3] Susan Davies,[4] Vanessa Hebditch,[5] Coral Hollywood,[6] Stefan Hubscher,[7] Salil Karkhanis,[8] Will Lester,[9] Nicholas Roslund,[10] Rebecca West,[5] Judith I Wyatt,[11] Mathis Heydtmann[12]

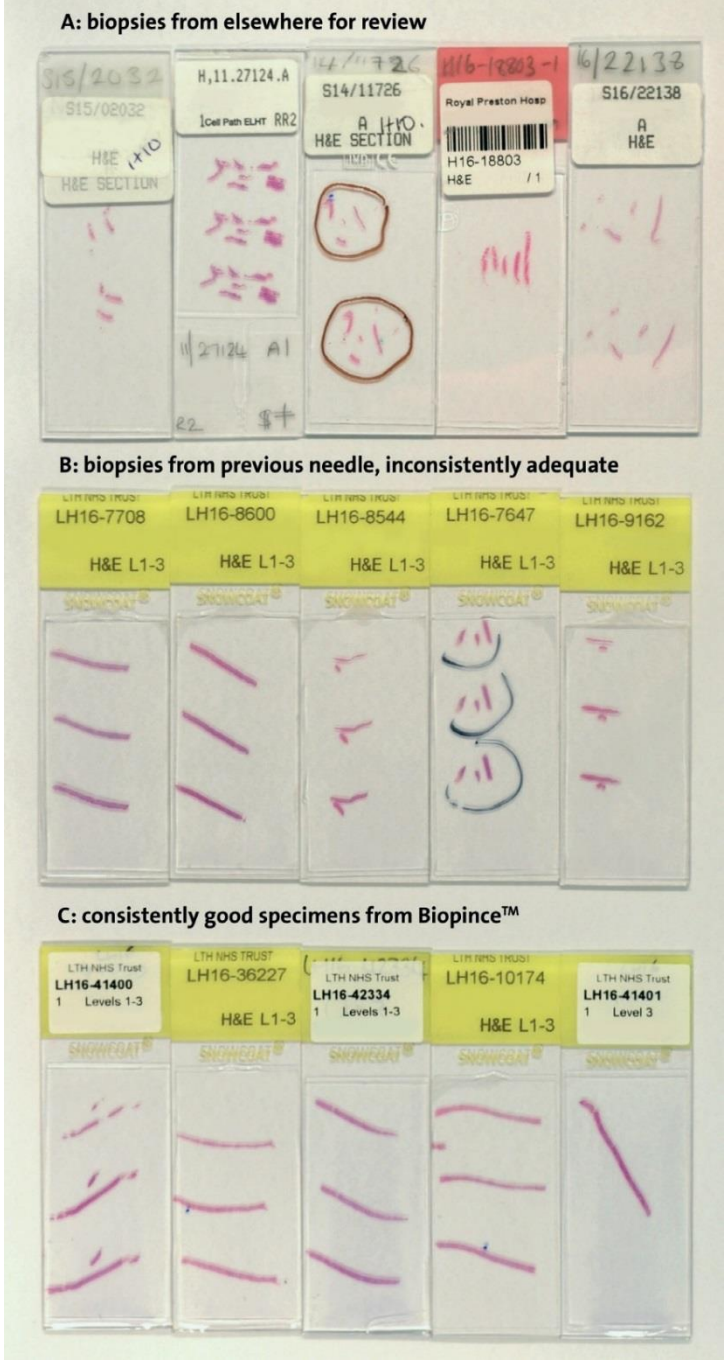
Neuberger J, et al. Gut 2020;69:1382–1403.

Liver biopsy adequacy and biopsy needle type

Various biopsies sent for second opinion

Biopsies using previous 16G side cut needle

Biopsies using current 16G full core needle



A good sample is an essential starting point for a good biopsy report.

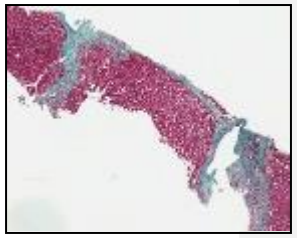
Recommended 2cm core from at least a 16-G needle

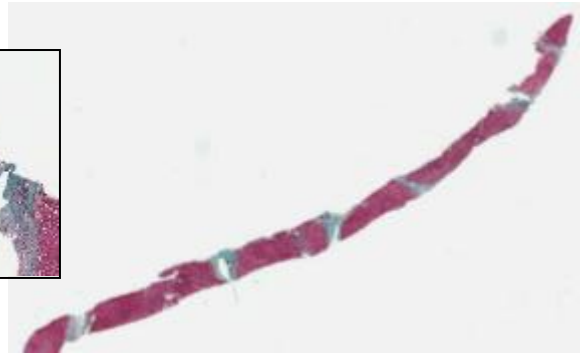
Consistently obtain >10portal tracts per section

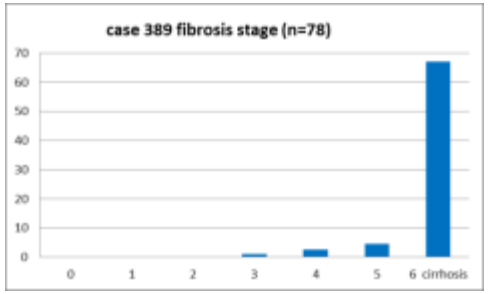
Colloredo G et al *J Hepatol* 2003:38:1448-57
Kleiner DE and Bedossa P *Gastroenterology* 2015;149;1305-1308

Example from EQA Scheme

389 - cirrhosis

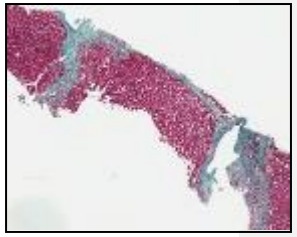


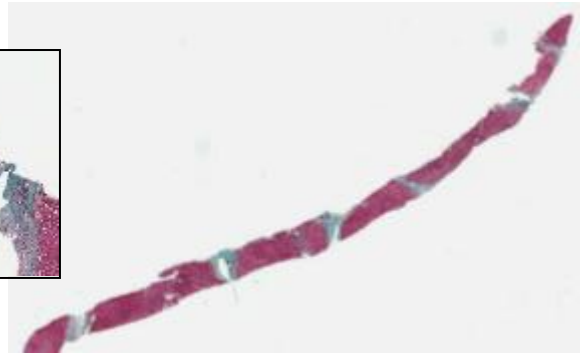


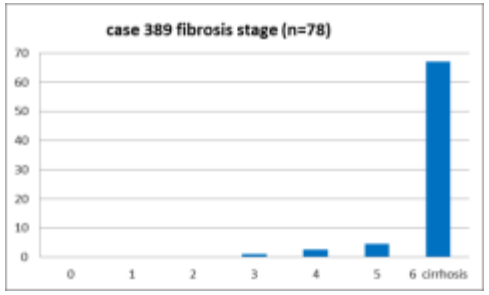


Example from EQA Scheme

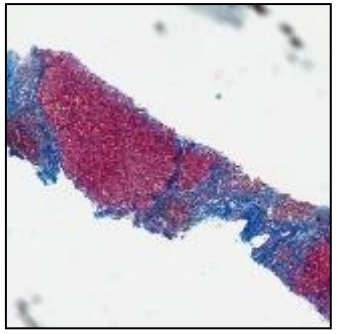
389 - cirrhosis

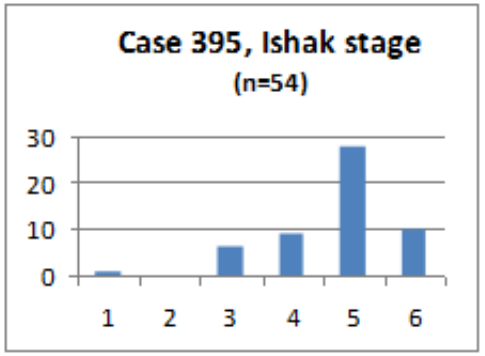






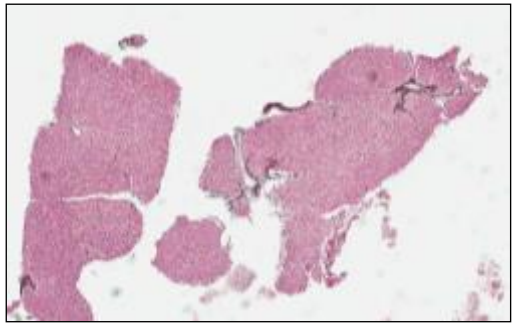
395 hep C – multiple cores, total 40mm

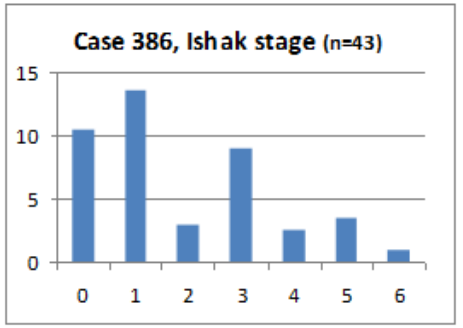




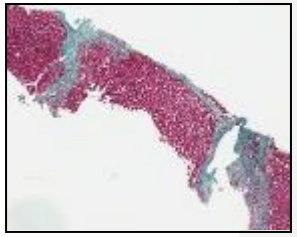
Example from EQA Scheme

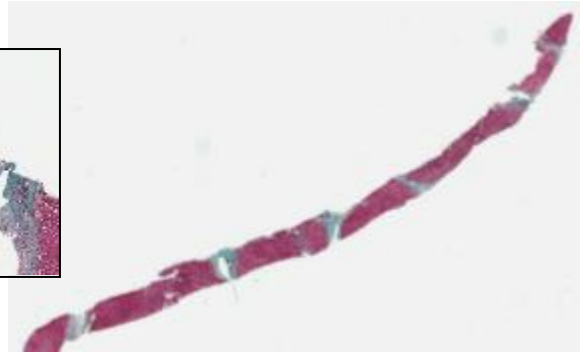
386 Hepatitis B – to assess inflammation and fibrosis
4 cores up to 7mm long

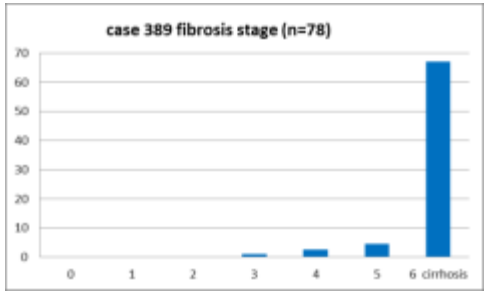




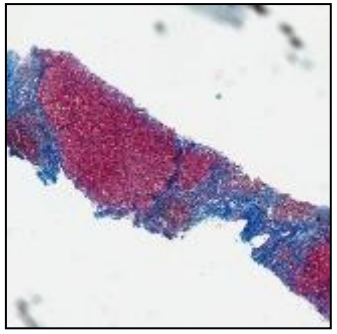
389 - cirrhosis

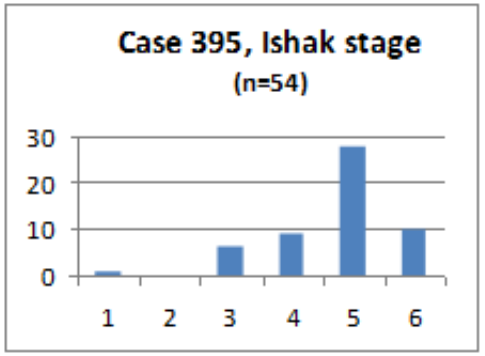






395 hep C – multiple cores, total 40mm





Q1 What type of dog is this?

Q2 How spotty is it?

Reference images

What can we do to improve agreement in pathologists' staging of fibrosis?
(Liver EQA meeting, Leeds, December 2012)

- Circulate slides with collagen stain

- Nottingham – 100 digital slides of various stages, good size biopsies.
- Create two sets matched for stage. Invite participants from EQA scheme

- Any liver disease – clinically important stage, not a named stage that depends on diagnosis
  - No fibrosis,
  - Early fibrosis
  - Bridging fibrosis
  - Late stage (cirrhosis or suspect/developing cirrhosis)

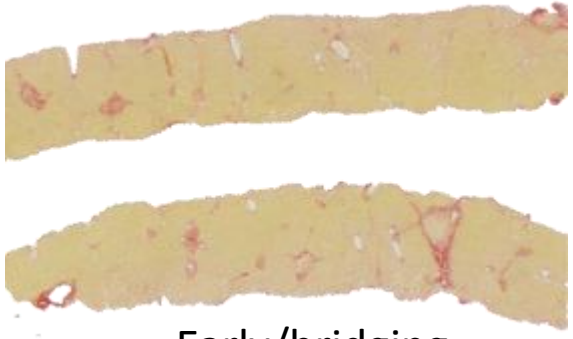
- Illustrate consensus view of biopsy stage from first set

Sounds easy …….

Examples: Reference images, based on complete agreement or 4:4 split of 8 liver pathologists

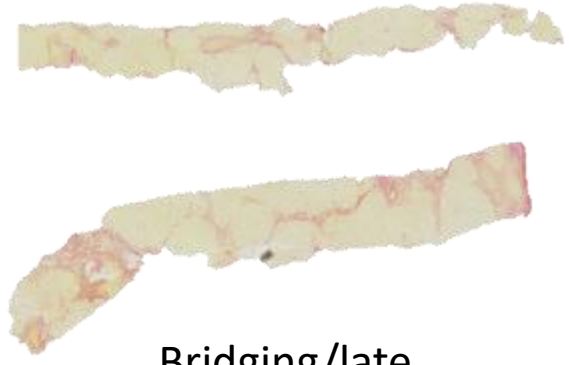
None/early

Early

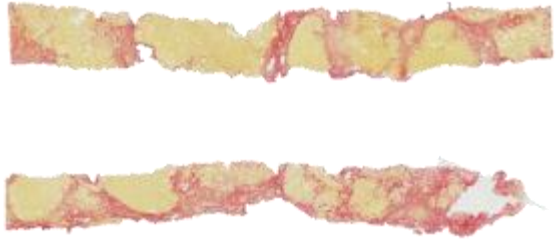


Early/bridging

Bridging



Bridging/late



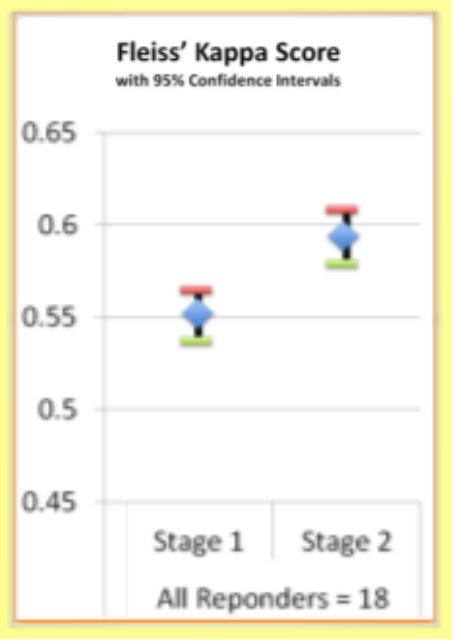
Late

Eight UK pathologists staged 47 Sirius red stained liver biopsies

Cases with full agreement or 4:4 split used to illustrate stage and threshold between stages

The 8 liver pathologists repeated the exercise on 47 other biopsies using reference images as a guide

Ten ‘generalist’ histopathologists looked at set 1 without reference images, and set 2 with reference images



UK Liver Pathology Group

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

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

United Kingdom Liver Pathology Group

- Future CPD activities - including links for registration
- UK Liver Pathology EQA Scheme

CPD resources:

- Our group's activities from 2006 which have CPD resources available
- An original series of seminars presented by the late Professor Peter Scheuer
- Liver lectures from other UK CPD meetings

Quick guide to our website PDF

Index of topics in annual update meetings PDF

From 2007 – liver subcommittee of the pathology section of the BSG

In 2016 – formed the UK Liver Pathology Group

Chair:
Stefan Hubscher

Succeeded by
Rachel Brown

Virtual Pathology at the University of Leeds

Home Public Slides EQA Teaching Research Clinical Trials NPIC

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United Kingdom Liver Pathology Group

Reference images for liver biopsy reporting

< UKLPG pages

- Reference images for staging fibrosis PDF
- Reference images of steatosis PDF

Bridging/late

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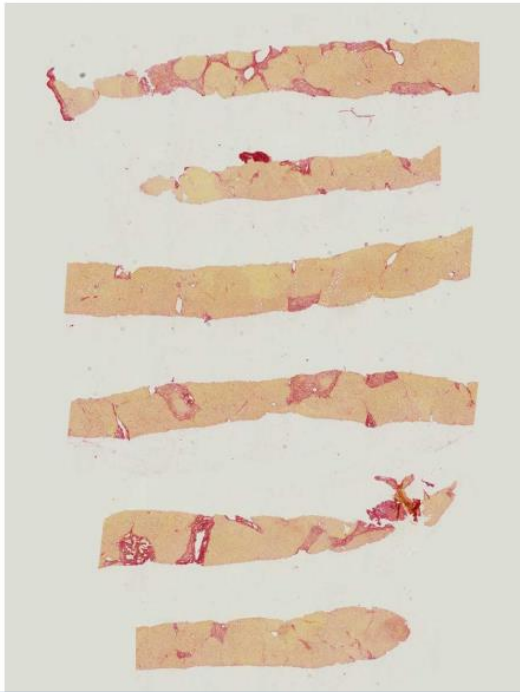


Reduce variation
– use the same terminology and criteria

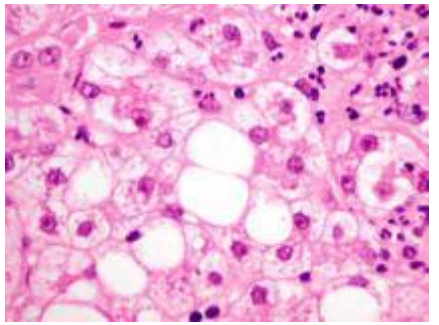
- Text books – training with Peter Scheuer

- Liver Biopsy Interpretation 1st edition 1968
  - Now in 9th edition, author Jay Lefkowitch

- For my generation – Prof Scheuer, tapes and slides
- Still available on virtualpathology

Liver Pathology Seminars

A series of Seminars by **Peter J Scheuer**, Professor Emeritus,
Royal Free and University College Medical School
of University College London 2001



- Biopsy interpretation – an opinion, not a fact

Quality assurance
– proactive, a system of measures to identify where there is potential error
and do something to avoid it

1. History of external quality assessment in cellular pathology

2. The UK Liver Histopathology EQA scheme

3. What else makes a difference? – system of measures
- Good specimen
- Pre and post test – good communication
- Standard practice guidance
- Easy to access

Thanks to Stefan for leadership, perspective, friendship and support
- avoiding the oozlum bird……..

The **oozlum bird** is a legendary creature found in Australian and British folk tales. The bird has a habit of taking off and flying around in ever-decreasing circles until it manages to fly up its own infundibulum,