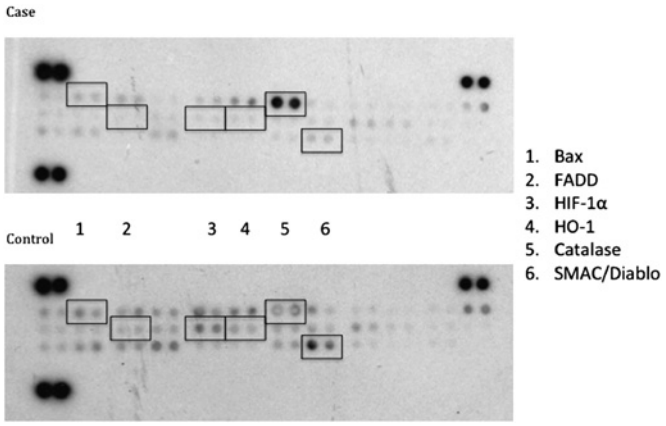


Abstract P02 Figure 3 Examples of apoptosis array for case and control.

($p<0.03$). Systemic arterial M30 levels were intermediate between the PV and HV levels (Abstract P02 figure 1). Analysis of apoptosis arrays showed significant downregulation of a number of apoptosis-associated proteins, including Bax, HIF-1, FADD and SMAC/Diablo ($p<0.04$) (Abstract P02 figure 2). Catalase was markedly elevated compared to controls in three of the four AALF patients. Histological evaluation revealed confluent hepatocellular loss, epithelial regenerative activity and an absence of apoptotic bodies.

**Conclusion** In this large cohort of PALF patients we have demonstrated the presence of elevated M30 levels and a correlation between caspase three activation and poor clinical outcome. The transhepatic M30 gradient, down regulation of apoptosis-associated proteins and histological appearances indicate that hepatocellular apoptosis might not be the major source of circulating M30. Our data also indicate that in established PALF, apoptosis in non-hepatic epithelial tissues may predominate and is likely to reflect incipient multi-organ failure, with resulting poor outcomes.

## P03 THE DIAGNOSTIC VALUE OF TRANSIENT ELASTOGRAPHY COMPARED TO CLINICAL ACUMEN, LABORATORY TESTS AND ULTRASOUND? IS THERE ADDED VALUE?

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**Introduction** The diagnostic performance of transient elastography (TE), and other non-invasive biomarkers of fibrosis, is assessed by direct comparison with liver biopsy. However clinical acumen, laboratory tests and ultrasonography are utilised for the assessment of fibrosis in clinical practice.

**Aim** The aim of this study was to assess the incremental value of elastography compared to routine diagnostic tools.

**Method** We included consecutive patients with both fibroscan and biopsy data. Patients with decompensated cirrhosis or suboptimal fibroscan readings were excluded (success rate <60% or IQR/median >0.21). Four consultant/attending hepatologists (who were blinded to TE and biopsy results) were asked to assess the severity of fibrosis on the basis of anonymised clinical data. Simple laboratory tests (eg, full blood count, liver function tests and clotting) and ultrasonography for each case were then given to the clinicians to assess the incremental increase in diagnostic performance. One independent pathologist formally assessed the degree of fibrosis on biopsy, which was the reference standard. Receiver Operating Characteristics (ROC) curves were calculated for (1) clinical acumen (2) clinical acumen + laboratory tests + ultrasonography and (3) TE, for the prediction of significant fibrosis (greater or equal to F2) and cirrhosis.

**Results** 130 patients were enrolled in the study with paired data and a biopsy deemed adequate for staging. The cohort (65% male; mean age 46 years) was of mixed aetiology (15% ALD, 48% chronic viral hepatitis, 24% NAFLD, 24% other). The average biopsy length was 23 mm with 16 portal tracts. The median TE reading was 6.3 (median IQR 0.8 and 100% success rate).

**Conclusion** There appears to be little additional benefit in AUC performance of transient elastography to diagnose cirrhosis compared to clinical acumen and routinely available tests. There is however incremental diagnostic benefit for the assessment of significant fibrosis. The baseline performance of simple diagnostic tools, which will vary depending on the stage of fibrosis, needs to be accounted for when assessing liver biomarker performance.

## P04 A DOSE EFFECT OF THE DISEASE RISK GENE HLA DR3 CONTRIBUTES TO NUMERICAL AND FUNCTIONAL IMPAIRMENT OF CD4+CD25+ REGULATORY T CELLS IN PATIENTS WITH AUTOIMMUNE HEPATITIS

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**Introduction** Autoimmune diseases are frequent among first degree relatives (FDR) of patients with autoimmune hepatitis (AIH), but concordance for AIH is rare. A numerical and functional impairment of $CD4^{pos}CD25^{pos}$ regulatory T cells (Tregs) is described in AIH patients, but no study has addressed Treg status in their FDR.

**Aim** To define whether the defect of Tregs in AIH is inherited and is associated with disease predisposing HLA genes.

Abstract P03 Table 1 AUC performance for the assessment of significant fibrosis and cirrhosis

| | Clinician | Clinical acumen AUC | Clinical acumen/lab tests/radiology AUC | TE alone AUC | p Value for AUC of TE vs clinical/lab/radiology* |
|---|---|---|---|---|---|
| Detection of significant fibrosis | 1 | 0.56 | 0.55 | 0.78 | 0.0003 |
| | 2 | 0.52 | 0.56 | 0.81 | 0.0001 |
| | 3 | 0.56 | 0.53 | 0.87 | <0.0001 |
| | 4 | 0.59 | 0.52 | 0.78 | <0.0001 |
| Detection of cirrhosis | 1 | 0.63 | 0.70 | 0.87 | 0.0268 |
| | 2 | 0.65 | 0.73 | 0.82 | 0.6693 |
| | 3 | 0.77 | 0.77 | 0.89 | 0.1367 |
| | 4 | 0.65 | 0.80 | 0.84 | 0.6705 |