## **BASL** abstracts

Hepascore were best at detecting advanced fibrosis, but other panels performed adequately with similar AUCs.

**Conclusion** In this cohort of previous non-responders to HCV treatment, the ability to discriminate advanced fibrosis appears to be similar amongst most of the markers tested. In contrast, of the eight markers tested in this study, the ELF panel appears to have the most consistent diagnostic performance across all stages of fibrosis and performs well in the detection of minimal and mild fibrosis. ELF testing would permit stratification of previous non-responders for further anti-HCV and anti-fibrotic therapy and for screening for complications of cirrhosis.

P16

NON-INVASIVE DETECTION OF OESOPHAGEAL VARICES: COMPARISON OF NON-INVASIVE ASSESSMENT OF SYSTEMIC HAEMODYNAMICS WITH LABORATORY PARAMETERS AND PREDICTIVE SCORES

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**Introduction** Endoscopic screening for varices (OV) is advised in cirrhosis, repeated every 1–3 years, with primary prophylaxis given to large OV. This is costly to endoscopy units, unpleasant for patients and multiple procedures may affect compliance. Cirrhosis is characterised by a hyperdynamic circulation; novel tools make noninvasive assessment possible.

**Aim** Toassess accuracy of non-invasive tests of systemic haemodynamics (Cardiac output and index (CO, CI), systemic vascular resistance (SVR), baroreceptor sensitivity (BRS) to detect OV and compare with other non-invasive methods (Child–Pugh score (CPS), albumin, platelet count, prothrombin time (PT), AST/ALT ratio, platelet count/spleen diameter ratio).

**Method** Prospective study of 29 cirrhotic patients. Systemic haemodynamics were assessed non-invasively with the Finometer®, hepatic venous pressure gradient (HVPG) assessed portal pressure and gastroscopy for variceal size (none/small (absent), medium/large (large)).

**Results** 69% male, median age 47 (42–55) years, CPS 6 (Class A 18, B 10, C 1) and MELD 10 (8–13). Prevalence OV 79%, large 38%. Significant differences in haemodynamics were seen between patients classified as absent or large OV (CO 5.6 vs 8.0lpm, CI 3.0 vs 4.5 l/min/m2, SVR 1.17 vs 0.77MU, HVPG 14 vs 19 mm Hg, BRS 5.8 vs 3.2 ms/mm Hg, CPS 5 vs 7, respectively). Comparisons summarised in the Abstract P16 Table 1. At a cutoff of 7.15lpm, CO predicted large OV with 73% sensitivity, 78% specificity and correctly classified 76% of patients. At a cutoff of 3.66 l//min/m2, CI predicted large OV with 82% sensitivity, 83% specificity and correctly classified 79% of patients. This compares to 78% correctly classified using HVPG, 76% CPS and 59% PT.

Abstract P16 Table 1 Results

Non-invasive test	Presence OV AUROC (95% CI)	Statistical significance	Large OV AUROC (95% CI)	Statistical significance
CO	0.71 (0.49 to 0.92)	p=0.029	0.84 (0.69 to 0.99)	p<0.001
CI	0.76 (0.51 to 0.95)	p=0.004	0.86 (0.71 to 1.0)	p<0.001
SVR	0.63 (0.40 to 0.86)	NS	0.77 (0.59 to 0.94)	p=0.002
HVPG	0.91 (0.74 to 1.0)	p<0.001	0.81 (0.64 to 0.98)	p<0.001
BRS	0.81 (0.53 to 0.96)	p=0.014	0.81 (0.64 to 0.98)	NS
Platelet count	0.75 (0.53 to 0.96)	p=0.011	0.59 (0.36 to 0.82)	NS
Albumin	0.32 (0.02 to 0.61)	NS	0.63 (0.42 to 0.84)	NS
PT	0.89 (0.79 to 0.99)	p=0.015	0.70 (0.52 to 0.88)	p=0.015
CPS	0.85 (0.75 to 0.95)	p<0.001	0.74 (0.54 to 0.94)	p=0.009
AST/ALT ratio	0.73 (0.55 to 0.91)	p=0.006	0.68 (0.46 to 0.91)	NS
Platelet/spleen	0.78 (0.59 to 0.97)	p=0.002	0.69 (0.48 to 0.90)	p = 0.040

**Conclusion** Non-invasive assessment of systemic haemodynamics appears a promising technique to identify cirrhotic patients at risk of having large oesophageal varices. Larger prospective validation studies need to be performed. Standard laboratory tests and predictive scores (except Child—Pugh score) are not reliable to accurately predict large oesophageal varices.

P17

## ANTICOAGULATION FOR LIVER FIBROSIS: A PILOT STUDY IN HEPATITIS C INFECTED PATIENTS

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Introduction Therapy directed towards the underlying cause of liver disease is not always successful, and anti-fibrotics are urgently required to slow disease progression in these patients. Recent evidence demonstrates a role for the coagulation cascade in promoting liver fibrosis. It is accepted that thrombin can activate hepatic stellate cells via PAR1 cleavage and we have previously shown that the prothrombotic Factor V Leiden mutation is associated with accelerated fibrosis in chronic HCV infection. Further, in animal studies, we have shown that warfarin anticoagulation ameliorates hepatic fibrosis. There is now an urgent need to evaluate the effect of anticoagulation on hepatic fibrosis in human studies.

**Aim** To evaluate the safety and impact of warfarin anticoagulation on the progression of liver fibrosis using non-invasive tests in HCV patients: a pilot study.

**Method** HCV patients (n=10, mean age 49.5 years, range 42–62 years, 6=M, 4=F) with moderate fibrosis (Ishak stage 3–4), who had previously failed anti-viral therapy were enrolled. Routine blood tests, liver stiffness measurements and serum markers of fibrosis (ELF testing), were performed at 0, 8 and 16 weeks. A subset of patients (n=5) had hepatic transit times performed at each time point. Patients were given no anticoagulation between 0 and 8 weeks (observation period) and anticoagulated with warfarin to maintain an INR of 2–3 between 8 and 16 weeks (treatment period). Wilcoxon signed ranks test used to compare paired data.

Results Warfarin anticoagulation significantly reduced median liver stiffness values (8 vs 16 weeks: 9.60 vs 6.90 kPa, p=0.012; 0 vs 16 weeks 7.70 vs 6.90 kPa, p=0.043). There was no significant difference in liver stiffness values between the start and end of the observation period. A non significant trend towards prolongation was seen in mean hepatic transit times following anticoagulation (11.0 vs 12.1 s; p=0.23). There were no significant differences between ELF test scores, serum ALT values and APRI scores following anticoagulation. No serious adverse events were reported during the anticoagulation period. One patient had a minor adverse event, requiring temporary cessation of warfarin treatment, and was excluded from the analysis. **Conclusion** A short period of warfarin anticoagulation demonstrated a significant improvement in liver stiffness measurements in HCV patients with pre-existing moderate fibrosis, with no major adverse events. These results suggest that warfarin anticoagulation may have a beneficial effect on liver fibrosis in HCV patients. Larger human studies are required to further evaluate its anti-fibrotic potential.

P18

## THE IMPACT OF HEALTH PROTECTION AGENCY GUIDELINES ON THE MANAGEMENT OF HEPATITIS B IN WEST KENT

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**Introduction** In 2006, the Health Protection Agency (HPA) issued standards regarding the follow-up of Hepatitis B infection. West