PWE-283 | SURVEILLANCE FOR HEPATOCELLULAR CARCINOMA: A CLINICAL AUDIT

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Introduction It is thought that mortality due to hepatocellular carcinoma (HCC) is rising in the Western world. This has been primarily attributed to the hepatitis C viral epidemic.[1] HCC detected after the development of symptoms carries an extremely poor prognosis (0–10 per cent survival at 5 years),[2] whereas those detected at surveillance can often be cured. One study demonstrated reduced mortality of 37% in cirrhotic patients undergoing 6 monthly surveillance with ultrasound scanning (USS) and α feto-protein (AFP) levels; this method is currently recommended by the BSG.[3] However, the American Association for the study of Liver Diseases (AASLD) now advocates 6 monthly USS without AFP testing: studies showed that AFP levels increased detection rate but also increased false positive detection rates with an added cost of $2000 to $3000 per tumour found.[4] The clinical lead for hepatology in Swansea, Dr C L Ch'ng, has primarily adopted these guidelines.

Methods A list of 246 patients entered into the HCC surveillance programme between January 2006 and December 2011 was reviewed and the frequency of USS and AFP levels measured for each patient was recorded. 62 patients were excluded:

▶ 20 DNAs
▶ 14 new patients
▶ 13 had insufficient data
▶ 12 developed serious comorbidities or died before follow-up was complete
▶ 3 were inappropriate for surveillance

Results were compared with standards set by BSG and AASLD guidelines.

Results 184 patients were appropriate for surveillance

▶ 183 had at least 1 USS
▶ 168 had at least 2 USS
▶ 114 had 6–12 monthly USS
▶ 95 (52%) had 6 monthly USS (recommended by AASLD)

48 (26%) received 6 monthly USS and AFP levels (current BSG recommendation).

Conclusion Surveillance of cirrhotic patients for HCC is currently suboptimal, with poor adherence to national guidelines. There is evidence that patients engaged initially but timing of subsequent USS and AFP teasting was erratic. Despite this, results are favourable in comparison with a large US study of cirrhotic hepatitis C carriers which demonstrated routine surveillance in only 12% of patients. Results did not differ widely from similar departmental audits carried out in the last 5 years. Suggestions for the future include routine 6 monthly postal invitation to screening with facilitated access to scans, together with education of both patients and treating clinicians regarding HCC risk. This should ideally be carried out within an established local surveillance scheme.

Competing interests None declared.

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PWE-284 | KHAT AS A POSSIBLE CAUSE OF DRUG INDUCED AUTOIMMUNE HEPATITIS; A CASE SERIES

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Introduction Khat is well recognised for its hepatotoxic effects. The exact mechanism by which it causes liver damage remains unknown. We report a series of patients with a history of khat use presenting with acute hepatitis, and review the potential role of khat in triggering auto immune hepatitis (AIH).

Methods The database at Sheffield Hospitals was searched for patients referred to the Hepatology department between 2005 and 2010 with liver problems and a history of khat use. They were then assessed for probability of having AIH using the revised criteria for diagnosis of AIH.[1]

Results Six patients presenting with acute hepatitis on a background of khat use were identified. All of the patients were male. Five of these patients were of Somali origin, while one patient was from Yemen. The age range of these patients was 24–57 years (mean 42.3 years). The patients were scored according to the revised autoimmune hepatitis criteria. They were given minus four (−4) for khat use on the scoring system due to its potential hepatotoxicity. Despite this, five out of six patients had a pre treatment score of 10 to 15 which placed them in the probable group for autoimmune hepatitis. The five patients that were in the probable group had at least a partial response to corticosteroids with a greater than 50% reduction in their ALT after 1-month of treatment. The patient that had scored negative for AIH (<10) showed the least improvement with prednisolone and continued to have raised liver enzymes after 1-year of treatment.

Conclusion The exact mechanism by which khat causes hepato-toxicity remains elusive. One possibility could be by triggering autoimmune hepatitis in a genetically susceptible individual. Further studies are needed to evaluate this phenomenon.[2][3]

Competing interests None declared.

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PWE-285 | CLINICAL BUT NOT HISTOLOGICAL FACTORS PREDICT LONG-TERM PROGNOSIS IN PATIENTS WITH BIOPSY PROVEN ADVANCED ALCOHOLIC LIVER DISEASE

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Introduction Alcoholic liver disease (ALD) is a significant and increasing threat to the health of the British population. It remains one of the commonest indications for liver transplantation and a leading cause of death. Despite this, the long term clinical course and predictive factors of survival in advanced ALD have not been well described. We aimed to identify factors that predict 15-year survival in out-patients with biopsy-proven advanced ALD.

Methods Patients attending clinic in our institution during early 1996 (n=134) with biopsy proven advanced (stage III or IV) ALD were followed-up for 15 years or until death or transplantation. At

baseline, clinical data including alcohol and smoking history, BMI and features of portal hypertension were recorded. Laboratory data (bilirubin, creatinine, sodium, PT, INR and platelet count) were collected. On biopsy, the presence of cirrhosis and histological features (fat severity, lymphocyte and neutrophil infiltration) were scored semi-quantitatively.

Results Median age was 51 (29−67) and the majority (72%) were male. All patients had a history of alcohol excess (>80 g/day for men, 50 g/d for women). Patients were followed until death (n=97; median 62 m), OLT (n=5; median 96 m) or are still alive (n=32; median 187 m). Overall, the 5, 10 and 15-year survival was 64, 40 and 26%, respectively. Patient baseline characteristics are shown according to outcome (Abstract PWE-285 table 1). In multivariate analysis age (p=0.01), smoking (p=0.01), persistent drinking (p<0.01) and serum albumin at baseline (p=0.02) were associated with significantly increased risk of death. No histological features correlated with prognosis.

Abstract PWE-285 Table 1   Characteristics of cohort according to 15-year survival

Characteristic	Alive n=32	Dead n=102	OR (95% CI)	p Value
Age*	48 (44−54)	53 (47−58)	1.08 (1.02 to 1.13)	0.01
Male gender	18 (56.3)	78 (76.5)	2.53 (1.10 to 5.83)	0.03
Smoker	12 (50.0)	72 (72.7)	2.67 (1.07 to 6.65)	0.04
Subsequent abstinence	13 (41.9)	17 (16.8)	0.28 (0.12 to 0.687)	0.01
Ascites	2 (7.1)	20 (20.8)	3.42 (0.75 to 15.64)	0.11
HE	1 (3.6)	4 (4.3)	1.20 (0.13 to 11.20)	0.87
Prev features PHT	8 (26.7)	44 (44.0)	2.16 (0.88 to 5.32)	0.09
Albumin*	44 (42−47)	39 (32−44)	0.83 (0.75 to 0.92)	0.01
Child's Pugh	9 (9−10)	10 (9−12)	1.26 (1.19 to 2.59)	0.01

Conclusion In out-patients with biopsy-proven advanced ALD, clinical but not histological factors determine prognosis. Age, persistent alcohol intake, smoking habit and serum albumin are independent poor prognostic factors. Abstinence from alcohol and smoking cessation should be the priorities in the long-term management of ALD.

Competing interests None declared.

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Introduction The identification of fibrosis in patients with NAFLD is important for ascertaining prognosis and stratifying patients for emerging therapeutic interventions. Use of both direct marker panels (liver matrix components) and indirect marker panels (simple biochemical tests) have been described for the detection of fibrosis in NAFLD. The aim of this study was to compare the performance of direct and indirect serum marker panels in the detection of fibrosis in NAFLD as compared with liver biopsy.

Methods From two centres, 177 patients were recruited and underwent percutaneous liver biopsy. Fibrosis staging was assessed using Kleiner criteria by two senior liver-histopathologists. Serum at the time of biopsy was used to calculate six indirect marker panels of fibrosis (APRI, BAAT, BARD, Cirrhosis discriminate score, NAFLD fibrosis index and Fib4). These panels were compared with the ELF Test (HA, TIMP1, PIIINP) and HA alone. Diagnostic accuracy was assessed using receiver operating characteristic curves which were compared using the method of DeLong.

Results The distribution of fibrosis stages in the cohort were as follows: F0 39.5% (n=70), F1 19.2% (n=34), F2 17.5% (n=31), F3 13.6% (n=24), F4 10.2% (n=18). While ELF and HA alone had the best performance overall, the ELF test was better than HA in its ability to discriminate minimal fibrosis (p=0.02) and cirrhosis (p=0.06). All indirect serum markers tested had significantly worse performance than ELF in the detection of cirrhosis (p<0.05).

Conclusion In patients with NAFLD, direct serum marker panels have superior performance compared to indirect marker panels allowing superior stratification and prognostication. The performance of HA alone is enhanced by the addition of PIIINP and TIMP1.

Competing interests None declared.

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Introduction Hepatic encephalopathy (HE) is a neuropsychiatric state which may complicate cirrhosis following the accumulation of toxic substances that cross the blood-brain barrier and affect brain function. Cirrhosis or portal-systemic shunting results in the accumulation of substances in the blood that may undergo alveolar gas exchange to be excreted in the breath. The aim of this work was to

Abstract PWE-286 Table 1

Test	F0 vs F1−4 AUROC (95% CI)	F0−1 vs F2−4 AUROC (95% CI)	F0−2 vs F3−4 AUROC (95% CI)	F0−3 vs F4 AUROC (95% CI)
Fib4	0.692 (0.614 to 0.771)	0.783 (0.715 to 0.851)	0.846 (0.755 to 0.937)	0.846 (0.754 to 0.938)
APRI	0.708 (0.631 to 0.784)	0.728 (0.652 to 0.803)	0.814 (0.707 to 0.921)	0.813 (0.706 to 0.921)
NAFLD FS	0.673 (0.594 to 0.753)	0.755 (0.684 to 0.826)	0.856 (0.782 to 0.931)	0.856 (0.781 to 0.930)
CDS	0.584 (0.501 to 0.668)	0.588 (0.501 to 0.675)	0.634 (0.491 to 0.778)	0.631 (0.487 to 0.775)
BARD	0.644 (0.560 to 0.728)	0.616 (0.532 to 0.700)	0.730 (0.602 to 0.858)	0.733 (0.606 to 0.860)
BAAT	0.600 (0.515 to 0.684)	0.660 (0.577 to 0.743)	0.522 (0.377 to 0.668)	0.517 (0.372 to 0.663)
HA	0.716 (0.641 to 0.791)	0.804 (0.739 to 0.868)	0.915 (0.862 to 0.968)	0.911 (0.856 to 0.965)
ELF	0.759 (0.689 to 0.828)	0.813 (0.747 to 0.879)	0.942 (0.902 to 0.981)	0.937 (0.896 to 0.979)