## 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 $\alpha$ 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 hepatotoxicity 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