investigate the use of breath analysis as a non-invasive and simpler means of diagnosing HE, cirrhosis and harmful drinking.

Methods A bespoke breath-sampling device was used to sample one litre of breath through adsorbent tubes from patients with alcohol-related cirrhosis with (n=11) and without HE (n=25), non-alcoholic cirrhosis without HE (n=19), harmful drinkers without cirrhosis (n=7), inflammatory respiratory disease (n=18), and healthy controls (n=15). Compounds trapped on these tubes were released via thermal desorption and analysed by gas chromatography mass spectrometry for separation and detection. Multivariate discriminant analysis was used to identify volatile organic compounds to differentiate patients according to disease status and build models for disease classification.

Results Models based on the presence or absence of volatiles were tested in the patient groups. HE was correctly classified in 91.0% of patients with alcoholic cirrhosis. Patients with cirrhosis could be discriminated from those without cirrhosis with 100% accuracy in drinkers. In patients without clinical signs of HE, alcohol was correctly predicted as the underlying cause of cirrhosis in 82.6% of patients and non-alcoholic causes of cirrhosis were correctly determined in 84.2% of patients. Non-alcoholic cirrhosis, alcoholic cirrhosis, and harmful drinking could also be discriminated from healthy controls with a sensitivity of 99.5%, 97.1% and 100%, respectively.

Conclusion Breath volatiles can be used to aid the diagnosis of HE, cirrhosis, and harmful levels of drinking, therefore breath testing may offer a means to detect liver conditions non-invasively at earlier and more treatable stages.

Competing interests None declared.

REFERENCE

**PWE-288** SPONTANEOUS BACTERIAL PERITONITIS: PREVALENCE ON ADMISSION TO A TERTIARY CENTRE AND SUBSEQUENT OUTCOME
doi:10.1136/gutjnl-2012-302514d.288

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Introduction Spontaneous bacterial peritonitis (SBP) is a sinister complication of cirrhosis associated with poor survival (approximately 38% at 1 year). However, a diagnosis of SBP does not represent, in its own right, an indication for liver transplantation in the UK under current listing criteria.

Methods We sought to investigate the prevalence and subsequent mortality in patients with an admission diagnosis of SBP. We retrospectively identified 366 consecutive cases admitted with ascites to our liver unit during the calendar year 2009. Of these 271 (74%) patients underwent diagnostic paracentesis at time of admission and were included for further analysis.

Results Of the 271 cases, 26 (9.6%) were diagnosed with SBP at admission on the basis of an ascitic fluid white cell count above 250 cells/mm³ (25 cases). Patients with positive cultures but no white cell response were only included if symptomatic (one case). Alcohol was the underlying aetiology in 17 cases (65.4%) and clinical presentations were as follows: routine paracentesis (14 cases), associated pleural effusion (four cases), variceal haemorrhage (three cases), abdominal pain (two cases), jaundice (two cases), encephalopathy (one case). Aseptic culture on two patients grew gram-negative bacilli, both resistant to quinolones being used for prophylaxis. One aseptic culture grew *Lactobacillus* spp. and one a mixed growth of *Enterococcus faecalis* and gram-positive cocci. Three patients (11.5%) died during the index admission. Three patients (11.5%) had successfully undergone liver transplantation and were alive 6 months after admission. Overall mortality at 6 months from an admission diagnosis of SBP was 50%.

Conclusion SBP is not uncommon in cirrhotic patients with ascites, can often present silently and is associated with high mortality. Resistance to standard quinolone prophylaxis and isolation of gram-positive bacteria are more recent phenomena in this group of patients. All cirrhotics admitted with significant ascites should undergo diagnostic paracentesis to exclude SBP and assessment for liver transplantation must be an urgent consideration in appropriate candidates. Listing criteria may need to be revised to include SBP as a standard indication.

Competing interests None declared.

REFERENCE
**PWE-290**

ACTIVE ALCOHOL CONSUMPTION INDUCES FUNCTIONAL IMMUNE PARESIS BUT PARADOXICALLY PROMOTES ENDOXIN TOLERANCE IN THOSE WITH ADVANCED ALCOHOL-RELATED CIRRHOSIS

doi:10.1136/gutjnl-2012-302514d.290

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**Introduction**

Patients with alcohol-related cirrhosis (ARC) are particularly prone to infection which is frequently a precipitant of organ failure and death. Neutrophil dysfunction has been reported in patients with ARC. Sepsis and associated endotoxemia occur in approximately 40% of hospitalised patients. Neutrophil TLRs are able to sense pathogens and induce inflammatory responses but whether active alcohol drinking is protective or detrimental in this context remains unknown. The aim of this study was to characterise the neutrophil phenotype, TLR2/4/9 expression and plasma cytokine profile in patients with ARC who were abstinent (n=15) compared to those who were drinking (n=16), split by MELD score.15 compared to healthy controls (n=12).

**Methods**

Neutrophils isolated from patients with ARC were studied ex vivo at baseline, and following 2-h stimulation with lipopolysaccharide (LPS) and the bacterial degradation protein fMLP. Neutrophil phenotype and TLR expression were determined using flow cytometry. Intracellular cytokine production pre- and post-stimulation will be measured by CBA.

**Results**

The severity of cirrhosis (MELD15) or abstinent/active drinking status did not significantly impact on resting CD16, CD11b, TLR2 and 9 expression. TLR 2/9 expression on exposure to LPS and fMLP showed downregulation in those who were active alcohol-drinkers compared to the baseline. Conversely TLR4 expression was upregulated in the abstinent group with MELD >15; an effect significantly abrogated by the effect of active drinking (p=0.004). Active alcohol consumption did not however change TLR4 expression in those with MELD.

**Conclusion**

Active alcohol consumption was shown to downregulate functional TLR2/9 expression resulting in immunoparesis with potential susceptibility to Gram-positive infection. However, active alcohol consumption was shown to abrogate the increased TLR4 responses to endotoxin stimulation seen in the abstinent ARC cohort with MELD >15. This implies that alcohol paradoxically promotes endotoxin tolerance and may act as a potential protective mechanism against Gram-negative insults in those with advanced cirrhosis.

**Competing interests** None declared.

**REFERENCES**


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**Introduction**

Non-alcoholic fatty liver disease (NAFLD) is becoming a common cause for secondary care referral. While simple steatosis does not lead to liver related morbidity, non-alcoholic steatohepatitis (NASH) may lead to cirrhosis and hepatocellular carcinoma. Currently, non-invasive imaging including ultrasoundography (US) may reveal fatty infiltration. Many patients attend secondary care for US, in whom NAFLD may be identified incidentally. While the true prevalence and burden of NAFLD in the community remains unclear, patients attending for USS in whom NAFLD is identified could provide the opportunity to reduce disease burden by monitoring for any complications. The aims of this study were to assess the true prevalence and burden of NAFLD in the community.

**Methods**

A special computer-based NAFLD screening was performed in 1840 patients attending for US at Chase Farm Hospital, London. The computer compared the ultrasound image (US) to a database of 50 images (CDI and US) with clinical history. Patients were divided into 3 groups: (1) No liver lesions; (2) Steatosis; (3) NAFLD.

**Results**

An estimated 17% of the patients attending for US could have NAFLD. The prevalence of NAFLD increases with age in both men and women (p<0.05). The prevalence of NAFLD is higher in men (18.17%) compared to women (13.83%). The prevalence of NAFLD increases with BMI (p<0.05). The prevalence of NAFLD is higher in obese patients (20.47%) compared to normal weight patients (14.18%).

**Conclusion**

This study is among the first to attempt to screen an unselected patient population for NAFLD. The study is limited because the data is obtained from a single hospital in London and the prevalence of NAFLD could be higher in other regions of the UK. Further studies are required to determine the true prevalence of NAFLD in the community.

**Competing interests** None declared.

**REFERENCES**