Conclusion We have identified a subset of PBAD subjects with high triglycerides and fasting FGF19 levels comparable to healthy individuals. The post prandial rise in FGF19 suggests no defect in the response of FGF19 synthesis in this subset. It may instead be caused by impaired BA absorption due to reduced ASBT expression which is also manifested as high serum triglycerides. PBAD may be a heterogenous condition with more than one underlying key abnormality.

Disclosure of Interest None Declared.

OC-028 SMALL BOWEL CANCER IN THE UK

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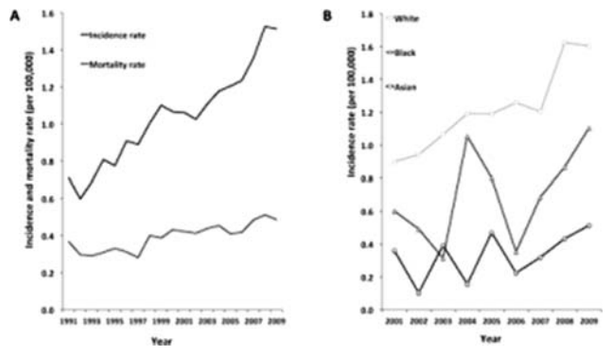
Introduction Small bowel cancer (SBC) is uncommon worldwide and accounts for only 5% of all gastrointestinal (GI) malignancies despite the small bowel forming 75% of the GI tract. [1] Our understanding is limited by its rarity, insidious course, difficult assessment and late diagnosis, coupled with multiple histological subtypes. We aim to review the trend of SBC in the UK over the last two decades.

Methods The national UK Association of Cancer Registries (UKACR) database identified SBC patients diagnosed from January 1991 to January 2009. We retrospectively reviewed and carried out a descriptive analysis of SBC incidence rates with respect to gender, age, ethnicity (as per UK Census 2001) and socio-economic status (as per UK Indices of Deprivation 2004, 2007 and 2010) and mortality rates.

Results The registry identified 11,872 patients, 53.6% male and 46.4% female, who were diagnosed at a singular peak mean age of 67 years over the study period. The overall incidence of SBC increased from 0.71 to 1.51 per 100,000 from 1991 to 2009 with mortality increasing simultaneously but to a lesser extent (Figure 1A). SBC was 1.5 times more common in males than females. They were most frequently located at the duodenum (57.5%, n = 7860) where incidence almost tripled (0.24 to 0.63 per 100,000), and less frequently at the jejunum (12.1%) and ileum (30.4%) where incidence approximately doubled (0.07 to 0.11 and 0.14 to 0.33 per 100,000 respectively). The incidence in white patients was 1.5 times higher than black patients and 3 times higher than in Asian patients over the period 2001 to 2009 (Figure 1B). SBC incidence was unchanged with respect to socio-economic status.



Abstract OC-028 Figure 1 (A) SBC incidence and mortality rates, 1991 to 2009. (B) SBC incidence by ethnic group, 2001 to 2009

Conclusion The incidence of SBC in the UK has increased over the last two decades with little improvement in mortality rates. It is most common in males in their 6th decade and in the proximal small intestine, which is in keeping with current literature. However, the higher incidence in white patients is in contrast to the geographical variation seen in both United States SBC and UK colorectal cancer data. A more comprehensive understanding of the natural history, environmental and genetic predisposition is needed to allow for potential patient stratification, more efficient diagnosis and treatment and thus improving its poor prognosis.

REFERENCE

1 Ross *et al*. *British Journal of Cancer*. 1991;63:143–145

Disclosure of Interest None Declared.

Liver section free papers

OC-029 RIFAXIMIN IS EFFICACIOUS IN THE TREATMENT OF CHRONIC OVERT HEPATIC ENCEPHALOPATHY: A UK LIVER MULTI-CENTRE EXPERIENCE

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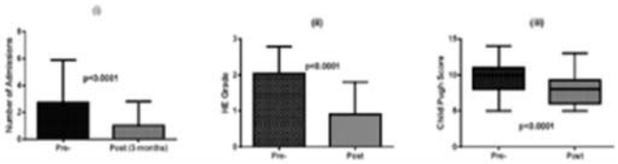
Introduction Rifaximin-α is a non-absorbable antibiotic increasingly being used for the secondary prevention of recurrent overt hepatic encephalopathy (HE) in the UK. The therapeutic mechanism of rifaximin has yet to be elucidated, with reduction in gut ammonia production postulated. We undertook a UK multicentre retrospective audit of patients receiving rifaximin therapy for HE in 4 hospitals, two of which are liver transplant units, with the aim of assessing tolerability, impact on HE/liver disease severity and hospitalisation rates.

Methods Patient demographics, concurrent therapy, Child Pugh, MELD, UKELD and number of hospital admissions were collected 3 months prior to initiation of rifaximin therapy and then 3 months following treatment.

Results 170 patients were identified (mean age 57yrs±12; 68% male) over the period 05/2010–03/2013. Three month post treatment outcome data were available for 73 patients (43%); 53 patients (31%) died during the 3 month follow up period. Average duration of treatment was 79 ± 121 days, with therapy well tolerated in 97.6% of patients. 74% were taking concomitant lactulose with 23.5% on rifaximin monotherapy. No cases of *Clostridium difficile* infection were reported.

The most common aetiology was alcohol 90/170 (53%) with 25 (28%) actively drinking . 36 patients (21%) were transplanted during the audit period. The predominant HE phenotype was episodic overt (67%), with persistent overt featuring in 20% and the Parkinsonian phenotype in 6%.

Admission data were available for 143/170 (84%) patients with a total of 444 admissions in the 3 months prior to therapy (average admission length 23 ± 25 days). The hospitalisation rate per patient fell significantly from 2.7 ± 3.2 to 1.0 ± 1.8 admissions in the 3 months following initiation of therapy (p <



Abstract OC-029 Figure 1

0.0001) [Figure i]. HE grade improved significantly following therapy ($p < 0.0001$) [Figure ii].

Child Pugh score fell significantly following therapy ($p < 0.0001$) [Figure iii], as did MELD and UKELD scores: 15 ± 7 vs. 13 ± 5 ($p < 0.03$) and 55 ± 6 vs. 51 ± 5 ($p < 0.02$), respectively. This is noteworthy as the MELD score does not include HE as a parameter and is based on bilirubin, INR and creatinine.

Conclusion Our UK multi-centre experience is that rifaximin is well-tolerated and an efficacious treatment for the secondary prevention of HE. Rifaximin significantly reduced both hospital re-admission rates after 3 months treatment, impacting significantly on the NHS resource burden of HE, and reduced overall liver disease severity raising the possibility that its therapeutic effect may extend beyond reducing gut ammonia production.

Disclosure of Interest V. Patel Grant/research support from: Norgine (UK) Ltd., J. Orr Grant/research support from: Norgine (UK) Ltd., J. Sturgeon: None Declared, Z. Habtemariam: None Declared, H. Preedy: None Declared, P. Richardson Consultant for: Norgine (UK) Ltd., R. Aspinall Consultant for: Norgine (UK) Ltd., M. Hudson Consultant for: Norgine (UK) Ltd., D. Shawcross Grant/research support from: Norgine (UK) Ltd., Consultant for: Norgine (UK) Ltd.

OC-030 EFFECTIVE STRATIFICATION OF HEPATOCELLULAR CARCINOMA RISK IN PRIMARY BILIARY CIRRHOSIS: RESULTS OF A MULTI-CENTRE INTERNATIONAL STUDY

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Introduction Hepatocellular carcinoma (HCC) is an important but infrequent outcome in primary biliary cirrhosis (PBC). Improved risk evaluation is an important goal for stratified surveillance.

Methods Risk-factor analysis of the 'Global PBC Study Group' comprising 15 centres across North America and Europe spanning >40-years follow-up was performed using Cox proportional hazards model, logistic regression and Kaplan-Meier estimates (SPSSv21).

Results Of 3546 patients with PBC (med. follow-up 8.6 yrs; IQR 4.4–14.1), 131 developed HCC. Excluding those who developed HCC within 12 months of PBC diagnosis (n = 23), median time to HCC was 12.7 yrs (6.9–16.8) and subsequent survival 1.1 yrs. (0.2–2.7). At diagnosis, factors associated with HCC development were male gender (adj. HR: 4.4;1.4–12.2, p = 0.014) and thrombocytopenia (adj. HR: 4.5;1.4–14.8, p = 0.012). Use of ursodeoxycholic acid per-se was not associated with future risk of HCC, but stratification of risk by biochemical response at 12 months was effective by Rotterdam (adj. HR: 8.9;2.1–37.3, P = 0.003), Paris-I (adj. HR: 7.6;2.0–29.0, p = 0.003) or Toronto criteria (HR: 5.6;1.6–18.8, P = 0.006). Five (4.6 vs. 0.2%) and 10-year (13%vs.1.9%) HCC incidence was significantly increased for biochemical non-responders ($p = 2.2 \times 10^{-9}$), and by multivariate analysis non-response remained the only significant risk factor.

Conclusion Our uniquely powered cohort allows robust demonstration that 12-month biochemical non-response is associated with an increased risk of developing HCC in PBC. Routine surveillance in those achieving biochemical response is unlikely cost-effective.

Disclosure of Interest None Declared.

OC-031 RELAXIN MODULATES CIRRHOSIS-INDUCED RENAL VASCULAR ENDOTHELIAL DYSFUNCTION

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10.1136/gutjnl-2014-307263.31

Introduction Hepatorenal syndrome (HRS) is a feared complication of cirrhosis characterised by intense renal vasoconstriction. The pathophysiology remains unclear, pharmacotherapy is limited and mortality is high. We investigated vascular responsiveness and the pathogenesis of renal vasoconstriction in models of advanced rat cirrhosis. Additionally,we determined the mechanism of action of the vasoactive peptide relaxin (RLN), previously shown to increase renal blood flow (RBF) in experimental cirrhosis (Snowdon V *et al*., *BSG* 2013).

Methods We induced cirrhosis,reduced RBF and renal dysfunction in male SD rats by carbon tetrachloride (16 wk) or bile duct ligation (4 wk). Arteries from renal (renal, segmental, interlobar) and splanchnic circulation were isolated for functional assessment using wire myography. qPCR array for vasoactive signalling genes,