Results 630 patients have outcomes recorded. We report on 370 who have completed treatment. 81% male, mean age 68 years (40–91). Patient’s underwent mean 2.5 ablations (1–6) during protocol. 70% baseline histology HGD, 27% IMC & 3% LGD. Mean length baseline BE 5.6cm (1–20). At 12 months CR-HGD was 87% patients, CR-D 82%, & CR-BE 64%. 97% with no dysplasia at 12 months remain disease free at most recent follow up (median 18 months, range 2–68). Kaplan Meier statistics predict CR-D is durable at 5 years with 88% remaining disease free. Logistic regression demonstrate each extra 1 cm of BE reduces chances of attaining CR-D by 15.7% (OR 1.156, SE 0.0003) & for each extra RFA treatment likelihood of CR-D increases by 31.7% (OR = 0.683, SE 0.95, CI 0.52–0.89, p = 0.0006). Progression to invasive cancer at 12 months is 2.7%. Symptomatic strictures requiring dilatation occurred in 9% after treatment.

Conclusion End of protocol CR-D is encouraging at 83% & successful eradication appears durable. Patients with shorter segment BE respond better & multiple treatments are more likely to achieve CR-D. Our data represent real life outcomes of integrating novel endotherapy into demanding endoscopy service commitments

Disclosure of Interest None Declared

REFERENCES

Liver symposium: impact of clinical research in hepatology

OC-053 CURCUMIN, ANTI-OXIDANT AND PIOGLITAZONE THERAPY WITH INCLUSION OF VITAMIN E IN NON ALCOHOLIC FATTY LIVER DISEASE-A RANDOMIZED OPEN LABEL PLACEBO CONTROLLED CLINICAL PROSPECTIVE TRIAL (CAPTIVE)
doi:10.1136/gutjnl-2013-304907.052

Introduction NAFLD is a global clinical challenge which progresses to cirrhosis and liver cancer. Defective transport of free fatty acids and mitochondrial dysfunction lead to explosion of a series of free radicals, apoptosis, up regulated cytokines and fibrogenesis ultimately causing cirrhosis and cancer. Curcumin is a pan-antioxidant with anti-inflammatory, anti-apoptotic, anti-microbial, and anti-fibrogenic properties. This study evaluates the role of curcumin in NAFLD to progression of NASH

Methods Eighty patients (n = 80) with mean BMI 29%, NAFLD score 0.66, NASH fibrotic score 0.53, HOMA IR 3.8, ALT 58, LDLc 143, HDLc 29, Triglyceride 186 and Adipokines (leptin, Adiponectin, Retinol Binding Proteins) were divided into Group A-(n = 20) pioglitazone 15mg, Group B-(n = 20) vitamin E, Group C-(n = 20) curcumin (all the three above groups received placebo), and Group D (n = 20) vitamin E plus curcumin. Pre and post values (Triglycerides, LDLc, HDLc, ALT, HOMA-IR, TNF-alfa, Leptin, Adiponectin, Retinol Binding Protein, HBA1c, Serum necro-inflammatory NAFLD and NASH fibrotic score were analysed at 3, 6, and 12 months. Diet and exercise were left unchanged. Daily alcohol content was less than 30 grammes

Results Group A-Minimal changes on ALT, Hba1c, HOMA, lipids, no changes in TNF-alfa, adipokines, lipid profile and necro-inflammatory score and/or NASH fibrosis score. Group B and Group C had modest changes in ALT, lipid profile, Hba1c and HOMA; while no changes in adipokines, necro-inflammatory score and fibrotic score. Group D had significant changes in all scores particularly the adipokines and small improvements in fibrotic score. All patients tolerated the medications well

Conclusion This study postulates the effects of Curcumin plus vitamin E in NAFLD may prevent NASH with a modest anti-fibrotic effects and necro-inflammatory score; with impressive changes in adipokines levels. Additive effects of Curcumin with vitamin E has significant effects on Serum lipids and insulin sensitivity. Unavailability of Pre and post liver biopsy was the limitation A large control trial needs to validate.

Disclosure of Interest None Declared

OC-054 HEPATIC EXPRESSION OF CCL25 MEDIATES RECRUITMENT OF PLASMACYTOID DENDRITIC CELLS TO LIMIT LIVER INJURY
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Introduction Ectopic CCL25 expression in the liver after liver injury protects against liver injury in a mouse model. In connective tissue diseases, aberrant CCL25 expression is associated with liver injury and inflammation. We hypothesized that CCL25 mediates recruitment of plasmacytoid dendritic cells (pDCs) to limit liver injury.

Results We found hepatic CCL25 expression in mice and human liver injury models. In the mouse model, human pDCs were recruited into the liver and expressed high levels of CCL25 in a time-dependent manner. In human patients, liver biopsies showed high levels of CCL25 expression in the liver, which correlated with the degree of liver injury. Furthermore, in vitro studies using human pDCs showed that CCL25 expression was induced by liver injury and enhanced the recruitment of human pDCs to the liver.

Conclusion These findings suggest that CCL25 plays a critical role in the recruitment of pDCs to limit liver injury in both experimental and human liver injury models. Further studies are needed to evaluate the clinical relevance of CCL25 in liver injury.

Disclosure of Interest None Declared
Introduction Primary Sclerosing Cholangitis (PSC) is an incurable chronic immune mediated biliary disease that occurs in individuals with IBD. We have previously reported aberrant expression of the gut chemokine, CCL25 in the human PSC liver and the recruitment of CCR9 expressing gut derived T cells. In order to gain further insights into the consequences of aberrant CCL25 expression in the liver in PSC, we induced CCL25 in the murine liver and assessed biliary inflammation in-vivo.

Methods To clarify the functional role of CCL25 expression in the liver, we generated a murine liver specific knock-in of CCL25 expression and tested the effects on immune mediated cholangitis using the Ova-Bil model of antigen driven biliary injury. Immune cell phenotyping and isolation were performed using flow cytometry. Liver injury was assessed by ALT measurements and histopathology. pDC function was assessed ex-vivo in co-culture with naive transgenic TCR T cells.

Results Ova-Bil x CCL25KI mice developed significantly less liver injury than wt Ova-Bil controls. Flow cytometry revealed increased numbers of CCR9+ pDCs in the Ova-Bil x CCL25KI livers. CCR9+ x Ova-Bil mice developed significantly worse liver injury compared to wt Ova-Bil controls and severely lacked pDCs in the liver. Adoptive transfer of wt pDCs to CCR9+ x Ova-Bil mice rescued the phenotype and reduced the degree of liver injury comparable to wt Ova-Bil controls. In vitro studies demonstrated the ability of liver-derived pDCs to induce regulatory T cells in a retinoic acid dependent manner as a possible mechanism by which CCR9+ pDCs are able to control liver injury.

Conclusion Aberrant expression of CCL25 in the liver enhances recruitment of CCR9+ pDCs and appears to be an attempt to limit the extent of hepatic inflammation in PSC. Regulatory effects of CCR9+ pDCs appears to be at least in part mediated through the expansion of hepatic regulatory T cells.

Disclosure of Interest None Declared

REFERENCE

Endoscopy symposium: endoscopy in the management of obesity

**OC-055** **A NOVEL TECHNIQUE FOR FULL THICKNESS LARAPENDOSCOPIC EXCISION OF COLONIC LESIONS: AN EXPERIMENTAL PILOT STUDY**

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Introduction Introduction of a National Bowel Cancer Screening Program in England has resulted in an increasing number of patients diagnosed with endoscopically irresectable colonic polyps. A significant proportion of these patients is referred for hemicolectomy and is subject to a significant risk of morbidity and mortality. Therefore, a less invasive treatment option is required and to address this, we modified a previously reported full thickness laparoscopic excision (FLEX) technique.

Methods Surgery was performed in five 70-kg pigs. A simulated colonic polyp was created by endoscopic injection of Spot™ and the clearance margin delineated by circumferential placement of mucosal argon plasma coagulator (APC) marks. Full thickness excision of the colonic wall, including the lesion, was achieved by endoscopic placement of prototype BraceBars (BBs). The everted section was excised using a linear laparoscopic stapler placed below the BBs. The first pig was terminated immediately and others were sacrificed 8 days after surgery.

Results The median procedure duration, defined from placement of mucosal APC marks to specimen excision, was 26 min (range 20–31 min). All excised specimens contained three pairs of BBs, included the APC marks and had a median diameter of 5.1 cm (range 4.5–6.3 cm). Postoperative recovery in survival animals was uneventful. Post-mortem evaluation demonstrated well-healed resection sites with no evidence of intra-abdominal infection or inadvertent organ damage. Endoscopic evaluation of anastomoses at post-mortem demonstrated a widely patent lumen without evidence of stenosis at excision sites. Histological examination of the anastomoses showed primary closure by mucosal abutment and regeneration, with repair and restoration of submucosal continuity.

Conclusion This proof-of-concept study has demonstrated the feasibility and safety of a novel full thickness colonic excision technique that is now ready for translation as an alternative to hemicolectomy. The excision size will accommodate most colonic polyps that currently come to surgery. Accurate placement of endoscopic BBs ensures complete excision, reducing the risk of residual disease and recurrence, while laparoscopic overview avoids collateral damage. The ability to preserve mesenteric vasculature and colonic length is likely to result in less morbidity and mortality, better functional outcomes and the approach should reduce treatment costs.

Disclosure of Interest None Declared

**OC-056** **LONG-TERM TRENDS IN COMORBIDITY AND RISK SCORES AND THEIR INFLUENCE ON OUTCOMES OF UPPER GASTROINTESTINAL BLEEDING**

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Introduction The prevention of upper gastrointestinal bleeding (UGIB) can be facilitated by understanding the changes in environmental and socio-pathological factors; these might not become obvious in short-term studies. We, therefore, aimed to study the changes in comorbidity and risk scores and their influence on the outcomes of UGIB over a 14-year period.

Methods We analysed the clinical characteristics of all patients presenting with UGIB to a single institution, 1996–2010. The Charlson’s comorbidity and the complete Rockall scores were analysed, together with patients’ drug use and 30-day mortality. Trends with time were assessed using logistic regression analysis with year of presentation as a continuous predictor variable. Regression coefficients were expressed as odds ratios (OR), representing the relative change in odds of death or other binary dependent variables over a time interval of one year.

Results A total of 2669 patients were included. The Charlson score increased significantly with time (P < 0.001), the odds of a high (3+) score increasing at a relative rate of 4.4% a year (OR = 1.044, 95% CI 1.022–1.065). No significant trend with time was noted for age (P = 0.09), haemoglobin level (P = 0.47) or Rockall score (P = 0.94). The overall 30-day mortality was 4.9% and this showed no relationship with age, sex or Charlson score increasing at a relative rate of 4.5% per year (OR = 0.953 (0.914–0.997); P = 0.038). Trends in the prevalence of taking potentially damaging and protective drugs are shown in Table-1, below. The rise in use of aspirin, other anti-thrombotic drugs and SSRI’s with pro-UGIB activity was paralleled by a rise in the use of PPIs [protective activity] and beta-blockers, ACE inhibitors, and statins [being able to affect mortality.

OC-054 Hepatic Expression of CCL25 Mediates Recruitment of Plasmacytoid Dendritic Cells to Limit Liver Injury

D Reid, V Lai, C Weston, T Vo, M Peters, D Adams and B Eksteen

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