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 0.82%, & CR-BE 64%. 97% with no baseline histology HGD, 27% IMC & 3% LGD. Mean length baseline BE 5.6cm (1–20).

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 endothermy 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)
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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, Retino 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, Adiponecit, Retinol Binding Protein) were compared. Pre and post values (T riglycerides, LDLc, HDLc, ALT, HOMA-IR, TNF-alfa, Leptin, Adiponectin, Retinol Binding Protein, HBA1c, Serum necro-inflammatory NAFLD and NASH fibrotic score were analysed at 5, 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 and 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
CCL25 expression is upregulated in liver injury suggesting that CCL25 mediates recruitment of Plasmacytoid Dendritic Cells (pDC) to the liver. CCL25 expression is upregulated in liver injury suggesting that CCL25 mediates recruitment of Plasmacytoid Dendritic Cells (pDC) to the liver. CCL25 expression is upregulated in liver injury suggesting that CCL25 mediates recruitment of Plasmacytoid Dendritic Cells (pDC) to the liver. CCL25 expression is upregulated in liver injury suggesting that CCL25 mediates recruitment of Plasmacytoid Dendritic Cells (pDC) to the liver.