Abstract P094 Figure 1 ZO-1 immunohistochemistry of ileum showed ZO-1 was downregulated in NAR naïve group. Albumin infusion restored ZO-1 expression

albumin supplementation (figure 1). Hepatic expression of TLR4 and associated pathways: Cirrhotic NAR animals had greater hepatic TLR4 expression which was reduced by albumin administration. Hepatic TLR4 gene array confirmed the activation of TLR4 dependent pathways in the cirrhotic NAR animals, which was abrogated by albumin infusion.

Conclusion NAR animals have significantly greater liver injury, increased sensitivity to LPS and mortality which is prevented by albumin administration. Our data show for the first time that the mechanism of the protective effect of albumin is consequent upon restoration of gut junctional proteins and reduction of hepatic TLR4 expression.

P095 IMPLEMENTATION OF DECOMPENSATED CIRRHOSIS DISCHARGE BUNDLE: A UNIVERSITY HOSPITAL EXPERIENCE

Muhammad Omar Saeed*, Muhammad Zuhaïd, Krishan Bountra, Unitt Esther, Nwe Ni Than. *UHCW, Coventry, Coventry, UK*

10.1136/gutjnl-2021-BASL.103

Background Decompensated liver cirrhosis is a frequent reason for admission to acute medical and gastroenterology units. Over the last two decades, a significant rise in the prevalence of liver cirrhosis in the UK has been noted, with the major culprits being alcohol related liver diseases, hepatitis B & C, and non-alcoholic obesity related disease. It has been observed that re-admissions to the hospital are common following discharge of the patients with decompensated liver cirrhosis. In order to improve the quality of discharge and reduce the re-admissions a decompensated discharge bundle has been developed by BSG and BASL. We aimed to assess the practice in our hospital against BSG/BASL guidelines and the impact by the implementation of the said discharge bundle.

Methods All those patients who were admitted with decompensated cirrhosis were included for data collection. Standard Quality Improvement model was adopted using two PDSA cycles. In cycle 1, discharge letters of 40 patients were assessed retrospectively against the decompensated cirrhosis discharge bundle tool kit during the months of January, February and March 2021. In cycle 2, there was re-assessment of discharge letters for 40 patients during the months of April, May and June to look for any change or improvement.

Results In cycle 1, it was noted that only 20% of the decompensated cirrhotic patients had weight, urea and electrolytes, diuretic dose adjustment and communication with the patients regarding future plans recorded on the discharge letters. Hence, the bundle was introduced by displaying the awareness posters in the Gastro ward and discussed with the junior doctors in the board round. Additionally, emails were sent to

doctors of gastro unit regarding the discharge bundle introduction.

There was a significant improvement of results in cycle 2, where 60% of the patients with decompensated cirrhosis had the above mentioned parameters documented in the discharge letters respectively.

Conclusion There were inconsistencies in the discharge letters when assessed during cycle 1 and the documentation was sub-optimal. However, with the introduction of discharge bundle in the hospital has led to a significant improvement in the discharge letter documentations when compared against the decompensated cirrhosis discharge bundle in cycle 2. In order to get much better results and to continue the improvement, we would consider the incorporation of the bundle in the Trust E-Library and include in the junior doctor inductions.

P096 THE POTENT UREASE INHIBITOR FLUROFAMIDE EFFECTIVELY SUPPRESSES AMMONIA PRODUCTION BY THE COLONIC MICROFLORA

^{1,2}Saeed Motamedi, ¹Robert Nelson, ²Val Edwards-Jones, ¹Shaun Greer*. ¹Wrightington, Wigan and Leigh Teaching Hospitals NHS Foundation Trust, Wigan, UK; ²Manchester Metropolitan University, Manchester, UK

10.1136/gutjnl-2021-BASL.104

Background Ureolysis by colonic microorganisms gives rise to a significant fraction of the portal vein ammonia load; colonic urease inhibition would therefore be a logical approach to reducing systemic ammonia in patients with hepatic encephalopathy. This was trialled in the 1960's in a small number of patients, using the urease inhibitor acetohydroxamic acid (AHA); disappointing clinical results led to the approach being abandoned. However, AHA is not a particularly potent inhibitor of urease; more potent inhibitors have since been developed, one of which is flurofamide. It is possible flurofamide might be more effective in this context, but no relevant work has been reported; we have conducted an initial laboratory study into the effects of flurofamide on ammonia production by the colonic microflora, in particular the flora of the right colon.

Methods Patients attending for routine colonoscopy who did not have inflammatory bowel disease or change of bowel habit were approached to take part. At colonoscopy, 50 mls sterile water was introduced into the right colon, and aspirated back into a polyp trap. These washings were transported to the laboratory for processing within 2 – 4 hours; a mechanical cell count was performed, and the samples were incubated for 22 hours in urea-containing culture medium with various concentrations of flurofamide, under both aerobic and anaerobic conditions. Following incubation, ammonia and urea levels in the medium were measured.

Results As expected, colonic washings produced ammonia when cultured in urea containing medium, although ammonia production did not correlate directly with the number of organisms in the inoculum. Lower concentrations of flurofamide had variable effects on ammonia production, but higher concentrations reduced levels to an average of less than 5% of the ammonia generated in the absence of flurofamide (figure 1). Results from aerobic and anaerobic cultures were very similar; there was no evidence for flurofamide toxicity. Urea levels were difficult to interpret due to assay variability and the high baseline level of urea