Figure (abstract FRI-244)

lymphocytes (T reg), which balance the response and favor immunological tolerance.

We aim to evaluate if the accumulation of polymorphisms in the TLR genes decreases the oxidative response and the proportion of Treg cells in patients with cirrhosis.

Method: Prospective observational study in patients with cirrhosis and non-infected ascites. Serum ROS concentration was determined by ELISA and the blood Treg cell population was quantified by flow cytometry using a CD3+ CD4+ CD25+ CD127-FoxP3+ panel.

Results: We included 71 patients with a mean age of 60 ± 10 years. The main etiology was alcohol and the mean Child-Pugh was 9.2 ± 1.8 . Twenty-two patients (30.9%) were taking β -blockers and 25 (35.2%) PPIs. Seven patients did not show SNPs (9.9%), 33 showed 1 SNP (46.5%), 26 patients the combination of 2 SNPs (36.5) and 5 carried the 3 SNPs studied (7.1%). The concentration of ROS and the percentage of Treg cells in blood are shown in Figure 1. Patients without SNPs showed significantly higher ROS and Treg levels than those observed in patients with one or more SNPs. In addition, patients with 3 SNPs showed significantly lower levels in both variables compared with patients with 1 or 2. A positive correlation was observed between ROS concentrations and the percentage of Tregs in the sample studied ($r = 0.615$, $p = 0.001$).

Conclusion: Polymorphisms in TLR genes limit the immune response and its modulation. Therefore, they could hinder the clearance of translocated bacterial antigens in patients with cirrhosis and non-infected ascites.

FRI-245

Bacterial translocation during experimental cirrhosis coincidences with the infiltration of inflammatory monocytes in the gut

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Background and Aims: Patients with advanced cirrhosis are susceptible to bacterial infections. The majority of infections originate from the gut due to a failure of the intestinal barrier function and the local innate immune system to dampen the response towards translocated bacteria. Indeed, we have previously demonstrated that intestinal myeloid cells from decompensated cirrhosis patients feature elevated levels of inflammatory parameters that may aggravate intestinal barrier dysfunction. However, the mechanisms governing the intricate cross talk between host and microbe, remain incompletely understood. Here we aim to elucidate

the role and dynamics of inflammatory monocytes and macrophages during cirrhosis pathogenesis.

Method: C57BL/6J mice received subcutaneous carbon tetrachloride (CCl₄) injections (2ml/kg of CCl₄ 50% (v/v)) (n = 5–8) or vehicle only (n = 3–4) twice a week. Mice were sacrificed after 4-, 9-, 14-, or 19 weeks and the extent of liver fibrosis/cirrhosis was assessed histologically following Picrosirius Red staining. Additionally, intestinal samples (proximal- distal small intestine, and colon) were collected for flow cytometry and mesenteric lymph nodes (MLNs) and plasma were collected for bacterial culture and liver function tests respectively.

Results: Histological analysis confirmed advanced fibrosis and cirrhosis development at week 14 and 19 respectively. Bacterial translocation as evaluated by culture positive MLN's, occurred at week 19, although elevated levels of bacterial products such as LPS, were detected both at week 14 and week 19 of CCl₄ exposure. Flow cytometric analyses of lamina propria cells isolated from the small bowel and colon revealed a gradual decrease in the frequencies of MHC^{high}CD11b^{high}CX3CR1^{pos}CD64^{pos} resident macrophages. Conversely, intestinal samples from cirrhotic mice were enriched with MHCII^{int}CD11b^{int}CX3CR1^{pos}Ly6C^{pos} inflammatory monocytes and the latter observation coincided with the presence of bacterial products in the circulation. Moreover, infiltrating inflammatory monocytes expressed high levels of CCR2 implicating their responsiveness towards the chemokine microenvironment.

Conclusion: These studies reinforce the pathogenic role of monocytes in dysregulated responses to intestinal bacteria during cirrhosis. Further understanding of their importance during intestinal barrier dysfunction are ongoing and may lead to strategies aimed at interrupting cirrhosis-driven pathology.

FRI-246

Effectiveness of a practical nutritional intervention in advanced liver disease: A prospective study

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Background and Aims: Malnutrition is one of the most prevalent complications of advanced liver disease and is associated with poor clinical outcome. In mixed populations of malnourished patients, the benefits of nutrition therapy are evidenced by reduction in mortality, infections and hospital length of stay. However, cirrhosis-specific studies are limited. The aim of the present study was to evaluate the