**MON-P061**

**GLUCAGON-LIKE PEPTIDE-2 MODULATES THE EFFECTS OF AUTOPHAGY ON INTESTINAL ADAPTATION IN A RAT MODEL OF SHORT BOWEL SYNDROME**

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**Rationale:** To explore the molecular mechanism of glucagon-like peptide-2 modulating the effects of autophagy on intestinal adaptation in a rat model of short bowel syndrome.

**Methods:** The rats underwent massive bowel resection (80% small bowel resection) to imitate the clinical condition of SBS caused by IRI. The expression of autophagy at different time points postoperatively through combined methods of electron microscopy (EM), polymerase chain reaction (PCR) and Western Blotting, and a variety of autophagy markers including LC3, p62, Atg5 and Beclin-1 were observed, the expression of apoptosis through the method of Western Blotting by detecting the apoptosis-related protein PARP and Caspase 8 was analysed.

**Results:** The number of autophagc body within 24 hours after MBR was greater than the sham-operated group, and the number decreased significantly at postoperative 72 hours. The expression of autophagy marker including LC3, Atg5 and Beclin-1 were significantly increased within postoperative 24 hours on the level of mRNA. Among them, the level of Beclin-1 was the most significantly reduced. While the expression of p62 significantly increased after the treatment at this time point. The rate of transition from LC3 I to LC3 II and the expression of LC3 II were reduced significantly after the treatment of GLP-2 at postoperative 24 hours. Meanwhile, the levels of Atg5 and Beclin-1 were also reduced at this time point. However, the levels of P62 decreased significantly after the treatment. The expression of autophagy increased significantly after the treatment of GLP-2 at postoperative 72 hours.

**Conclusion:** The intervene of GLP-2 initiated at postoperative 24 hours is the optimal time for early intestinal rehabilitation a rat model of massive bowel resection.

**Disclosure of Interest:** None declared.

**MON-P062**

**PREVALENCE OF INTESTINAL FAILURE ASSOCIATED LIVER DISEASE (IFALD) IN ADULTS ACCORDING TO DIFFERENT INDICES**

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**Rationale:** A comprehensive and unique definition of IFALD is not yet available. The aim of this study was to assess the prevalence of IFALD according to indirect biochemical and instrumental indices of liver disease.

**Methods:** Cross sectional study carried out in a single medical center, on adult outpatients on Home Parenteral Nutrition (HPN) for Chronic Intestinal failure (CIF) due to benign disease, with no liver disease due to a known cause. The followings were collected: anagraphic and anthropometric data, CIF mechanism, underlying disease, HPN characteristics, biochemistry and liver ultrasound (US). Criteria for IFALD diagnosis: ‘general criteria’: alkaline phosphatase and gamma-glutamyl transferase >1.5 ULN plus US signs of liver steatosis; cholestasis: direct bilirubin >0.3 mg/dL; steatosis at biochemistry: AST/ALT ratio (AAR) <1; steatosis at US; indices of fibrosis (APRI, Forns, NFS, FIB-4). Statistic: median, chi-squared test.

**Results:** A total of 117 patients were included (M 44%, age 52 yrs). Prevalence (%) of IFALD according to the individual index: ‘general criteria’, 7; cholestasis, 27; steatosis-AAR, 51; steatosis-US, 44; fibrosis: APRI, 10; Forns, 24; NFS, 3; FIB-4, 20. Patients with all positive indices, 0; patients with no positive index, 15. Prevalence of positive APRI (P < 0.01) or FIB-4 (P < 0.03) was greater in females; positivity of Forns (P < 0.001), NFS (P < 0.001) and FIB4 (P < 0.002) increased with age category; prevalence of cholestasis was greater in patients with jejunostomy or entero-cutaneous fistulas (P < 0.05).

**Conclusion:** In adults, indirect indices of liver disease indicate that IFALD is mainly represented by liver steatosis, whereas cholestasis and hepatic fibrosis may affect about one fourth of patients. Sex, age and CIF mechanism might play a role in the positivity of IFALD indices.

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**MON-P063**

**ASSOCIATION BETWEEN PRESARCOPENIA, SARCOPENIA AND BONE MINERAL DENSITY IN PATIENTS WITH CHRONIC HEPATITIS C**

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