of underweight (7/9 (78%)), healthy (28/46 (61%)), overweight (14/23 (61%)) and obese (6/12 (50%)) $p=0.642$. Patients with $>5\%$ weight loss (13/101 (13%)) or suboptimal HGS (56/101 (55%)) were significantly more likely to be at nutrition risk using IBD-NST, MUST and SGA. Patients with suboptimal MAMC (10/66 (15%)) were not significantly more likely to be at nutrition risk using IBD-NST or MUST.

Conclusion The IBD-NST identifies more patients with high nutrition risk and places less importance on BMI than SGA or MUST. Finally, we confirm that BMI is a poor indicator of HGS in IBD supporting a reduced importance in identifying nutrition risk. Further work is underway to test the repeatability of the IBD-NST.

PTH-136 INFLAMMATORY BOWEL DISEASE PATIENTS EXPRESS HIGHER LEVELS OF CIRCULATING GALECTINS-1 AND -3 THAN HEALTHY PEOPLE

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Introduction Non-invasive biomarkers currently available in clinic in (i) accurately diagnosing IBD and (ii) in determining disease activity in established IBD all show poor sensitivity and substantial limitation. In this study, we investigated whether levels of serum galectins, a family of 15 galactoside-binding proteins, could be used as potential non-invasive biomarkers for IBD diagnosis and for determining the disease activity.

Methods A single centre prospective cross-sectional study was conducted in 168 IBD patients (97 CD and 71 UC) and 40 healthy controls. Disease activity was assessed using Harvey-Bradshaw Index (HBI) for CD and simple clinical colitis activity index (SCCAI) for UC. Active disease was defined as HBI >5 and SCCAI >2 . Serum levels of galectins-1, -2, -3, -4, -7 and -8 was analysed by ELISA and compared with that in active and non-active IBD patients and healthy controls.

Results Serum levels of galectins-1 and -3, but not galectins-2, -4, -7 and -8, were significantly higher in patients with CD ($p=0.0001$ and $p=0.0003$ for galectin-1 and -3, respectively) and UC ($p=0.0002$ and $p=0.0001$ for galectin-1 and -3, respectively) as compared to healthy controls. Twenty seven (27.8%) CD patients and 41 UC patients (57.8%) had clinically active disease. None of the serum galectins including galectins -1 and -3 showed significantly different in patients with active UC or CD compared to patients in clinical remission. Serum CRP was significantly higher in patients with active UC (mean $\pm$ SD, 7.3 ± 12.5 mg/L) compared to those in remission (4.8 ± 14.5 mg/L, $P=0.047$). Similarly, serum CRP was higher in patients with active CD (4.6 ± 8.2 mg/L) compared to those in remission (10.2 ± 12.1 mg/L, $P=0.047$).

Conclusions Serum levels of galectins-1 and -3 were significantly higher in IBD patients compared to healthy people and this difference however could not separate active from inactive IBD.

PTH-137 PHOSPHATE STUDIES: IMPORTANT CHANGES IN PHOSPHATE HOMEOSTASIS AND BONE METABOLISM AFTER IV IRON

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Introduction Iron deficiency anaemia (IDA) is the most common extraintestinal complication of inflammatory bowel disease (IBD), and IV iron is commonly used to treat IDA in IBD. Of the two commonly used IV iron preparations, ferric carboxymaltose (FCM) and iron isomaltoside IIM, there is evidence that FCM can cause hypophosphatemia due to FGF23 mediated renal phosphate wasting, which has been associated with osteomalacia; the comparative effects of IIM are unknown.

Methods In two identical, open-label trials, 245 adults with IDA randomly received (1:1) IIM (1 $\times$ 1000 mg) or FCM (2 $\times$ 750 mg; 1 week apart). We compared the incidence, severity and duration of hypophosphatemia, and effects on fractional phosphate urinary excretion (FEPO₄), FGF23, parathyroid hormone (PTH), vitamin D (VitD), and bone turnover biomarkers in blood and urine.

Results In a pooled analysis, the incidence of hypophosphatemia <2 mg/dL was markedly higher in the FCM vs. IIM group (74.4% vs. 8.0%, $p<0.0001$). Hypophosphatemia persisted at day 35 in 43.0% of FCM-treated patients compared to 0.9% of IIM-treated patients ($p<0.0001$). Severe hypophosphatemia ≤ 1 mg/dL occurred in 11.3% of FCM patients compared to 0.0% of IIM-treated patients ($p<0.0001$). FCM infusion significantly increased intact FGF23 compared to IIM ($p<0.0001$): FGF23 after FCM rose from 49.9 pg/mL at baseline to 149.5 pg/mL one day post-infusion; and to 327.9 pg/mL by day 8. The corresponding figures for IIM were 59.9 pg/mL at baseline, 58.3 pg/mL by day 1 and 66.9 pg/mL by day 8. Compared to treatment with IIM, FCM significantly: increased FEPO₄; decreased serum 1,25-(OH)₂ vitD; decreased ionized calcium; and increased PTH, which persisted through day 35. Post-FCM changes were accompanied by significant increases in total and bone specific alkaline phosphatase that persisted through day 35. Correction of IDA was comparable between the two treatments. Significantly fewer adverse drug reactions occurred with IIM than with FCM. Serious or severe hypersensitivity reactions occurred in 0.8% in the IIM group and 1.7% in the FCM group.

Conclusion Compared to IIM, FCM-induced high rates of FGF23-mediated hypophosphatemia, which was frequently