Gastroenterology and Hepatology From Bed to Bench.

SHORT CUT

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Ahmad K, Fatemah F, Mehri N, Maryam S. ***Probiotics for the treatment of pediatric Helicobacter Pylori infection: a randomized double blind clinical trial.*** *Iran J Pediatr 2013 February;23(1):79-84*

Sub-optimal treatment compliance and consequential emerging antimicrobial resistance may contribute to the failure of eradication regimens for Helicobacter Pylori (H. pylori) infection, particularly in the pediatric population. This double-blind, randomized, placebo controlled study of 66 H. Pylori positive children aged 3-14 years (mean age 9.09 years) demonstrated that the addition of probiotics to a standard *H.Pylori* treatment regimen significantly improved *H.Pylori* eradication (p=0.04) whilst reducing treatment related side effects including nausea/vomiting (p=0.02) and diarrhea (p=0.039). Probiotic supplementation may improve treatment compliance by reducing the associated side effect burden.

Rácz I et al. ***Mucosal healing effect of mesalazine granules in naproxen-induced small bowel enteropathy.*** *World J Gastroenterol 2013 February 14;19(6)889-896*

Small bowel enteropathy, increasingly detected by video capsule endoscopy, is an important complication of non-steroidal anti-inflammatory drug therapy however preventative measures remain unexplored. This single centre, non-randomized, open label, uncontrolled pilot study investigated the effect of a four week course of mesalazine in 10 patients with mild or moderate-severe naproxen-induced small bowel enteropathy defined by Lewis index score (LIS) determined on video capsule endoscopy. Patients continued naproxen (1000 mg/d) and were treated with omeprazole (20 mg/d) whilst mesalazine (3 x 1000 mg/d) therapy occurred. The severity of small bowel enteropathy improved following mesalazine therapy in the 7 patients with initial moderate-severe disease (mean LIS: 1615±672 (pre-mesalazine) and 1064±424 (post-mesalazine), p=0.033). Although the authors enthusiastically conclude that mesalazine significantly reduces mucosal damage in moderate-severe naproxen-induced enteropathy this must be considered in the context of the small sample size and lack of a control group.

PhilipsonCW etal. ***The role of peroxisome proliferator-activated receptor γ in immune responses to enteroaggregative Escherichia Coli infection.*** *PLoS one 2013;8(2):1-11*

The mucosal immune response to Enteroaggregative Escherichia Coli (EAEC) infection, an emerging cause of gastroenteritis and diarrhoea worldwide, is poorly understood particularly in immunocompromised individuals. This interesting study explored the role of peroxisome proliferator-activated receptor (PPAR) γ, an immune modulator, in the mucosal response to EAEC infection in nourished and malnourished mice. In each cohort the effect of PPARγ modification by genetic deletion in T cells or pharmacological antagonism in wild type (WT) mice was investigated.

EAEC infected mice with PPARγ null T cells had reduced weight loss and enhanced recovery, determined by histological analysis of colonic specimen at 5 or 14 days post infection, compared with nourished or malnourished WT mice. In conjunction, interleukin (IL) 6 and tumour necrosis factor (TNF) α expression was significantly increased at day 5 post infection in malnourished mice with PPARγ deficient T cells compared with the WT ($p<0.05$). In nourished mice, PPARγ deficiency led to an increase in colonic IL-17 expression and TH17 response compared with all other groups ($p<0.05$).Infected mice treated with pharmacological PPARγ

antagonism expressed significantly higher colonic levels of pro-inflammatory cytokines(IL-1β, IL-6, CXCL1, MCP-1, TGFβ, IL-17 ($p<0.05$) and CCL20 ($p<0.0001$))and enhanced colonic EAEC clearance compared with untreated infected mice. The weight loss and colonic EAEC clearance benefits of PPARγ blockade were abolished by IL-17 neutralisation.
In addition to offering new in vivo informationabout colonic mucosal immune defence, this study identifies PPARγ antagonism as a novel therapeutic intervention for EAEC infection.

Mekaroonkamol P et al. ***Repeat colonoscopy's value in gastrointestinal bleeding.*** *World J Gastrointest Endosc 2013February 16;5(2):56-61*

This retrospective review of 139 patients investigated the clinical value of colonoscopy repeated within three years of the index procedure and for the same indication between 2000 and 2010.Colonoscopies completed for colorectal cancer screening/surveillance were excluded. New findings influencing clinical care were found in 25 out of 123 repeat endoscopies performed for lower gastrointestinal bleeding (20.33%) and 2 out of 7 performed for abdominal pain (28.57%). Only repeat colonoscopies performed within one year of the index procedure in patients with lower gastrointestinal bleeding were associated with a significantly increased detection rate of new lesions (odds ratio of repeat colonoscopy at 1-2 years and 2-3 years compared with 0-1 years were 0.09, 95% CI 0.01-0.74, p=0.025 and 0.26, 95% CI 0.09-0.72, p=0.010 respectively). This study highlights the utility of repeat colonoscopy, particularly in the first year after the index procedure, for recurrent lower gastrointestinal bleeding.

Vargas V et al. ***Surgically induced weight loss by gastric bypass improves non alcoholic fatty liver disease in morbid obese patients.*** *World J Hepatol 2013 December 27;4(12): 382-388*

The impact of bariatric surgery on non-alcoholic fatty liver disease (NAFLD) in obese patients is poorly understood however may represent a potential therapeutic option. This prospective, single blinded study of 26 morbidly obese patients (body mass index>40 kgm^{-2}) undergoing weight loss surgery (roux-en-Y gastric bypass with a modified Fobi-Capella technique) compared paired liver specimens biopsied at surgery and 16 ± 3 months postoperatively. The percentage of excess weight loss was 72.1% ± 6.6%. The prevalence of non-alcoholic steatohepatitis, diagnosed on biopsy, reduced from 96.1% at index biopsy to 15.3% at follow up ($p<0.001$). Marked histopathological improvements in the presence of steatosis ($p<0.001$), ballooning degeneration ($p<0.001$), Mallory bodies ($p=0.005$), glycogen nuclei ($p=0.001$), lobular inflammation ($p<0.001$), portal inflammation ($p<0.05$) and fibrosis ($p<0.001$) were visualised at follow upbiopsy. The authors conclude that restrictive/mildly malabsorptive bariatric surgery significantly improves NAFLD in morbidly obese patients.

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