	Gender	AIP I/2	AIP Diagnosis Date	IBD Diagnosis	IBD Diagnosis Date	Serum IgG4	Tissue IgG4	Pancreatic Resection	IBD surgery
Patient 1	F	AIP 1	2011 (age 71)	CD	2012 (age 72)	High	+ pancr + colon	Distal pancreatectomy	Sigmoid resection
Patient 2	M	AIP 2	2012 (age 34)	UC	2012 (age 34)	Normal	- maj pap	No	No
Patient 3	M	AIP 1	2010 (age 72)	UC	1992 (age 54)	N/A	+ pancr	Whipple	Colectomy
Patient 4	M	AIP 2	2010 (age 28)	UC	2010 (age 28)	Normal	+ maj pap + colon	No	No
Patient 5	F	AIP 2	2011 (age 28)	UC	2009 (age 26)	High	- maj pap + colon	No	No
Patient 6	F	AIP 2	2012 (age 32)	UC	2009 (age 29)	Normal	- maj pap	No	No
Patient 7	M	AIP 2	2011 (age 20)	UC	2012 (age 21)	Normal	+ maj pap	No	No

AIP = autoimmune pancreatitis; maj pap = major papilla

Mo1248

Rates and Predictors of Endoscopic and Clinical Recurrence After Primary Ileocolic Resection for Crohn's Disease

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Background: Most patients with Crohn's disease (CD) will undergo at least one intestinal resection during the course of their disease. Postoperative endoscopic recurrence is reported to occur in 60-80% of patients within 1 year. The utility of postoperative medical prophylaxis and the treatment of mild endoscopic recurrence continue to be debated in the literature. Aims: To document rates and predictors of endoscopic and clinical recurrence after primary ileocolic resection for CD in a large, tertiary academic center. Methods: We performed a retrospective chart review on patients who underwent primary ileocolic resection for CD between 1991 and 2010 at our institution. Only patients with clinical follow-up including postoperative endoscopic assessment at our institution were included in our analysis. Details of preoperative disease behavior, medication history, surgical pathology, and postoperative endoscopic, radiologic, and clinical course were recorded. Endoscopic recurrence (ER) was defined using the Rutgeerts score (RS) where RS ≥ 1 indicated ER. Clinical recurrence (CR) was defined as symptoms of CD with endoscopic or radiologic evidence of neo-terminal ileal disease. Results: 171 patients met inclusion criteria (median age at diagnosis 22 years, 50% male; 19% current smokers). 66 patients (39%) received postoperative medical prophylaxis. The cumulative probability of ER (RS ≥ 1) at 1, 2 and 5 years was: 29%, 51% and 77%, respectively. In a multivariate model, the only independent predictors of ER were the absence of postoperative medical prophylaxis (HR 1.5; 95% CI, 1.1-2.2; P = 0.03) and penetrating disease behavior (HR 1.5; 95% CI, 1.0-2.1; P = 0.05). In patients not receiving postoperative medical prophylaxis, perianal disease was associated with ER (HR 1.7; P = 0.05). The cumulative probability of CR at 1, 2 and 5 years was: 8%, 13% and 27%, respectively. In a multivariate model, the only independent predictors of CR were perioperative smoking (HR 2.3; 95% CI, 1.3-4.0; P < 0.01) and preoperative upper gastrointestinal tract involvement (HR 4.0; 95% CI, 1.8-8.3; P < 0.001). There was a higher rate of clinical recurrence in patients with RS-2 compared to RS-1 on endoscopy (HR 2.5; 95% CI, 1.2-5.9; P = .02). Conclusions: Rates of early ER in this cohort are less than previously reported in the literature. Postoperative medical prophylaxis did decrease the likelihood of ER while certain phenotypes of CD appear to increase the risk of developing ER and CR. The majority of patients with ER did not develop CR, which may reflect the increasing use of postoperative endoscopic surveillance that can facilitate early treatment of ER prior to the evolution of clinical symptoms.

Survival free of endoscopic recurrence after primary ileocolic resection for Crohn's disease.

Mo1249

The Anti-TNF Agent Infliximab Reverses Inflammatory Sarcopenia in Crohn's Disease

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INTRODUCTION: Muscle wasting or sarcopenia arising from chronic inflammation can be found in 60% of Crohn's disease (CD) patients¹. The transcriptional protein NF-Kb reduces muscle formation through MyoD transcription² and increases muscle breakdown by proteolysis³. As TNF is a potent activator of NF-Kb, and the anti-TNF agents infliximab (IFX) prevents NF-Kb activation, we assessed CD subjects treated with IFX for gains in muscle volume and strength. METHODS: We conducted a prospective, cohort study in CD subjects aged 18-70 presenting with an acute disease flare (Crohn's Disease Activity Index (CDAI) >220 and elevated C-reactive protein (CRP)). Subjects were instructed not to vary diet or activity and concomitant medications were kept stable. At week 0 (pretreatment), week 12 and week 21 of IFX induction regimen, we assessed (1) MRI volume of the quadriceps femoris at the anatomical mid-thigh; (2) maximal concentric quadriceps contractions strength in each leg at three specific speeds of contraction; (3) physical activity by validated instrument (IPAQ); (4) Three-day food record of intake and composition (food-weighting method); (5) Serum levels of cytokines TNFa, and IL6. RESULTS: 19 subjects (58% female, mean age 33.2 $\pm$ 10.7 y) were recruited based on preliminary power calculations. Mean baseline CDAI was 358 $\pm$ 79 and body mass index was 24 $\pm$ 4.8. IFX therapy led to significant progressive gains in muscle volume and muscle strength (Table). Muscle volume gain correlated with male gender. Significant gains in muscle strength were noted at each speed of contraction in both legs and were unrelated to age or gender. There were no significant differences in physical activity level throughout the study period, nor significant differences in calorie, protein and total fat intake. Serum TNFa fell significantly from pretreatment levels, as expected. CONCLUSION: We demonstrate for the first time that the anti-TNF agent IFX reverses inflammatory sarcopenia. Besides direct anti-inflammatory properties, IFX may have muscle protective effects, and may reduce time to return to work and improve quality of life. References:(1) Schneider et al. Inflamm Bowel Dis 2008; 14: 1562 - 8. (2) Guttridge et al. Science 2000; 289 (5488): 2363-6. (3) Cai et al. Cell 2004; 119 (2): 285-98.

Muscle Volume cm3	Week 0	Week 12		Week 21	
Right thigh	1505 $\pm$ 557.6	1607 $\pm$ 585.8	p=0.012	1569 $\pm$ 588.5	p=0.010
Left thigh	1478 $\pm$ 518.7	1565 $\pm$ 568.4	p=0.042	1534 $\pm$ 584.3	p=0.038
Muscle Strength Nm					
Right 300 /second	184.8 $\pm$ 96.64	192.3 $\pm$ 110.3	p=0.525	213.6 $\pm$ 112.8	p=0.002
Right 600/ second	183.5 $\pm$ 116.4	210.9 $\pm$ 108.5	p=0.008	216.9 $\pm$ 123.6	p=0.002
Right 900/ second	139.4 $\pm$ 54.4	162.8 $\pm$ 58.18	p=0.016	157.7 $\pm$ 66.4	p=0.014
Left 300/ second	166.5 $\pm$ 93.42	190.8 $\pm$ 99.28	p=0.007	199.5 $\pm$ 98.06	p=0.006
Left 600/ second	172.8 $\pm$ 103.5	190.6 $\pm$ 114.7	p=0.060	210.3 $\pm$ 109.9	p=0.002
Left 900/ second	128.5 $\pm$ 55.89	153.0 $\pm$ 65.65	p=0.009	155.5 $\pm$ 60.35	p=0.004

Mo1250

Cytomegalovirus (CMV) and Epstein Barr Virus (EBV) Specific ELISPOT Assays Detect Frequent Reactivation of CMV and EBV in Inflammatory Bowel Disease

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Objective: Patients suffering from inflammatory bowel disease (IBD) are often immunosuppressed rendering these patients susceptible to the reactivation of latent viral infections such as CMV or EBV. However, reliable non-invasive tools to detect viral reactivation are missing, especially since reactivation mainly occurs in the gut mucosa and not necessarily results in viremia. We hypothesized that the detection of CMV- and EBV-specific CD8 effector T cells should allow the distinction between dormant and reactivated viral infection. Methods: Peripheral blood was obtained from 26 IBD patients (16 Ulcerative Colitis UC, 10 Crohns Disease CD) and age-matched female and male healthy human donors after informed consent. Disease parameters were recorded and Colitis or Crohns disease activity indices determined. Peripheral blood mononuclear cells (PBMC) were isolated and single cell resolution ex vivo ELISPOT measurements of CMV- and EBV-antigen triggered release of Interferon- γ (IFN- γ), Granzyme B (GzB) and Perforin (PFN) were employed to identify actively ongoing T cell responses towards these viruses. Detection of effector T cells, delineated by GzB and PFN production, determined antiviral activity and was correlated with disease activity indices and immunohistochemistry staining of tissue samples where available. Results: CMV- and EBV-specific T cells were found to be in an activated state in a significant number of IBD patients; in particular during flares of disease (EBV reactivity was detected in 6 of 16 UC and 5 of 10 CD patients, CMV reactivity in 6 of 16 UC and 3 of 10 CD patients). No reactivity was found in healthy controls (p<0.01). While CMV reactivity was associated with greater disease activity with significantly less CMV reactive patients being in clinical remission (p<0.05), EBV reactivity was not associated with active disease (remission rates 33% vs. 43% for EBV reactive/nonreactive, respectively). Additionally, reactivity against CMV was associated with longer disease duration in CD (p<0.01) but not in UC (ns) while EBV reactivity was not associated with disease duration. Interestingly, PCR analysis on serum or immunohistochemistry staining of tissue samples frequently failed to detect CMV during flares. A correlation between the CMV immune response and current treatment with azathioprine was not observed. Conclusion: Our data support the importance of CMV and EBV infection in IBD. Single cell resolution ex vivo ELISPOT measurements of IFN- γ , GzB and PFN are a reasonable non-invasive tool to detect viral reactivation in IBD that might contribute to the intestinal inflammation. Immunological monitoring of viral reactivation might provide rational patient-tailored therapeutic options of immunosuppressive and/or antiviral treatment to ameliorate the course of disease.