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OPISTHORCHIS FELINEUS INFECTION MIMICING SCLEROSING CHOLANGITIS.

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Gabriele M. BIRKENFELD*, DISCO R.**. II. Medical Clinic*, Department of Microbiology**, Technical University, Munich, Germany. Medical history: A 29 year old female from Tomsk, Siberia, presented with a longterm history of ubiquitous abdominal pain and weight loss of ten kilogram within the last year. She also reported to be allergic to many substances. Course: Physical examination was normal, so were the laboratory results with the exception of a moderate hypereosinophilia. This led to parasitological stool examinations; numerous eggs of *Opisthorchis felinus* were found (picture 1). Abdominal ultrasound exposed alterations of the gallbladder that reminded of polyposis (2). MRC revealed an irregular common bile duct (3). ERC showed the typical picture of intra- and extrahepatic sclerosing cholangitis (4), but the curettage and sampling of these solid, constricting bile duct masses produced undestroyed, long *O. felinus* parasites (5). Therapy and follow up: Praziquantel (orally given, 25 mg/kg tid, for two days) led to the patient's well being and regain of weight. Allergic reactions did not reoccur. Conclusion: *Opisthorchis felinus* infection can mimic sclerosing cholangitis. In a patient from an endemic area, who has hypereosinophilia and PSC-resembling alterations of the bile ducts, parasitosis should be excluded.

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HUMAN OESOPHAGOSTOMIASIS: A HISTOMORPHOMETRIC STUDY OF 13 NEW CASES OF NORTHERN GHANA.

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Oesophagostomiasis is an infrequently described and recognised parasitic infection in humans caused by *Oesophagostomum bifurcum*. Although the disease is most often found in northern Togo and Ghana, sporadic cases have been described in other parts of Africa such as Uganda, Ivory Coast, Sudan, Kenya and Ethiopia. Infection occurs by the ingestion of food contaminated with L3 larvae. These larvae penetrate the intestinal wall, especially the colon. Some of these larvae develop into young adult worms and return to the bowel lumen. Other larvae however develop into immature worms, which fail to settle in the lumen, forming abscesses in the bowel wall and causing pathology. In the literature 105 human cases have been described, often originating in the northern regions of Ghana and Togo. The present study was performed to evaluate 13 new cases originating in the northern part of Ghana (7 female and 6 male patients, age between 2 and 60 years). Histopathologically, the patients could be divided into two groups: the first group showed a multinodular disease, the second group presented with a single, nodular mass. In the first group, abscesses were seen throughout the colonic wall. The mean size of the cavities was 4.3 ± 0.7 mm. There was no relation between the size and the localisation in the colonic wall. Abscesses in male patients were significantly larger than in female patients. There was no relationship with the age of the patient. In the second group, histopathological examination showed a thin-walled cyst with very limited inflammation. These cysts represented older lesions, encapsulated in the mesentery. In conclusion, in this study we present 13 new cases of human oesophagostomiasis. The abscess formation was found to be organ-specific, age-independent and gender-related, producing a more intense tissue reaction in male patients.

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ULCERATIVE COLITIS: AN INVESTIGATION OF THE POTENTIAL ROLE PLAYED BY $\alpha^E\beta_7$ +VE T-CELLS.

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Background: The aetiology and pathological mechanisms of mucosal damage in ulcerative colitis (UC) remain areas of controversy. Activated lymphocytes are likely to play an important role in both the induction and the final pathway of colonic inflammation and epithelial cell damage. In particular, it has become clear that intra-epithelial lymphocytes (IEL) expressing the surface integrin $\alpha^E\beta_7$ (CD103) have cytotoxic potential. The only known ligand for $\alpha^E\beta_7$ is e-cadherin, a homophilic cell-adhesion molecule restricted to epithelial cells. It is our hypothesis that the $\alpha^E\beta_7$ /e-cadherin interaction may play a vital role in the initial disruption of bowel wall structure leading to the onset of UC and may contribute to the tropism of inflammatory damage in this disease. The objective of this study was, therefore, to assess the expression of $\alpha^E\beta_7$ on IEL *in situ* in patients with UC. Materials & Methods: Pinch biopsies were obtained from patients with UC and normal controls undergoing routine colonoscopy. The samples were snap-frozen and stored at -80°C . Using a standard immunoperoxidase technique, $6\mu\text{m}$ acetone fixed cryostat sections were stained with mouse monoclonal anti- $\alpha^E\beta_7$ and anti-CD8 (Dako UK) primary antibodies and counter-stained with Mayers haematoxylin. A Kontran VIDAS Image Analysis System was used to measure the length of the epithelium and $\alpha^E\beta_7$ +ve IEL were counted and expressed as a number of cells per mm

of epithelium. Results: $\alpha^E\beta_7$ +ve IEL were present in all normal colonic samples (4.48 ± 0.22 cells/mm of epithelium). Mean $\alpha^E\beta_7$ +ve IEL/mm epithelium values were 9.6% higher in colonic samples from UC patients than normal controls. Significant inter-individual variation was seen, however, with the highest $\alpha^E\beta_7$ +ve IEL numbers being seen in patients with active disease. Conclusions: 1) Reliable *in situ* staining of IEL for $\alpha^E\beta_7$ was demonstrated. 2) No suppression of $\alpha^E\beta_7$ +ve IEL infiltrates was seen in patients with active or inactive colitis in comparison to normal controls. 3) There appears to be a positive association between the degree of $\alpha^E\beta_7$ +ve IEL infiltrate and disease activity. 4) These findings are compatible with $\alpha^E\beta_7$ IEL playing an active role in the inflammatory process in UC.

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TREATMENT OF SEVERE CROHN'S DISEASE (CD)-USING RIFABUTIN-MACROLIDE-CLOFAZIMINE COMBINATION: RESULTS AT 30-37 MONTHS.

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Mycobacterium paratuberculosis (Mp) is probably the best candidate for a microbial cause of Crohn's Disease (CD). Growing evidence suggests that prolonged antimicrobial combination therapy can reverse CD in a proportion of patients. We commenced a prospective trial in 12 patients with severe CD 30-37 months ago and reported encouraging interim findings¹. AIM to report longer-term observations in this same cohort treated with triple macrolide-based antimycobacterial therapy. METHODS-Patients failing maximum CD therapy were commenced prospectively on a bid combination of rifabutin (R=450mg/d), clarithromycin (C=750mg/d), and clofazimine (CF=2mg/kg). Azathioprine was terminated while 5-ASA and steroids were tapered off then ceased. Progress was monitored by clinical response, colonoscopy/biopsy, cross-sectional U/S, haematology, and Harvey-Bradshaw activity index. RESULTS In the 12 patients (7m;19-48y) follow-up data are available for 29-37 months since start of therapy. Initially 10/12 patients obtained near-complete control of CD on antimycobacterial therapy alone but in spite of treatment 3 reverted to active CD and went back to immunosuppressants. Complete clinical, colonoscopic and histologic remission was achieved in 6/12 with 2/6 remaining disease-free off drugs 5-7m and 1 relapsed at 6m. Remainder continue well on drugs. Clinical remission with histologic inflammation is present in 3/12. Ileal strictures became colonoscopically and histologically normalised in 4/5; extensive pseudopolyp crops regressed completely (n=1), deep colonic ulcers healed to longitudinal scars (n=1), defunctioning ileostomy was closed at 11m in 1. Clofazimine ileal staining is visible in all those treated. Harvey-Bradshaw index in those with long-term remission fell from 16.3 to 1.5 ($p < 0.001$). Adverse effects such as arthralgia, leucopenia and dry skin appear to resolve after 6-8m of therapy. All develop suntan pigmentation. CONCLUSIONS: 1) Complete reversal of severe CD can be achieved with prolonged combination R-C-CF antimycobacterial therapy in >50%. 2) Initial improvement with relapse on treatment suggests development of resistance. 3) Ideal duration for this therapy is unknown. 4) Perhaps Quad therapy will result in fewer relapses. 5) These results support a causal role for *M. paratuberculosis* in CD. Borody TJ et al. Gastroenterology 1998;114:A938

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INFLIXIMAB THERAPY FOR CROHN'S DISEASE: THE CROHN'S AND COLITIS CENTER OF NEW JERSEY EXPERIENCE.

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Aim: To assess our clinical experience with the use of infliximab (anti TNF- α) in 29 patients with Crohn's disease (CD). Methods: Since October 1998, 29 patients received a total of 60 infliximab (5mg/kg) IV infusions. Patient demographics, concurrent medications, duration of disease, disease characteristics and Crohn's Disease Activity Index (CDAI) were assessed. Post infusion side effects were also assessed. Results: Mean age of patients was 38.8 years (range 19-72), 21 female, 8 male. All patients had required systemic steroids in the past. 18 were on 6-MP, 1 on azathioprine, 1 on cyclosporine, & 1 on cyclic antibiotics. All were on 5-ASA. Twenty patients received infusion for acute exacerbation or non-responsive disease & ten for fistulous CD. Of patients with active CD, 15 had single infusion and 5 multiple infusions. 15 of 20 had subjective and objective improvement with reduction of CDAI (mean 45.7%). Mean time to response was 5.5 days. Mean duration of response was 10 weeks with first infusion, 9 weeks with second infusion and 8 weeks with third and fourth infusion. Ten patients had a series of 3 infusions for fistulous disease at 0, 2 & 6 weeks. Six of 7 with external fistulas had complete cessation of drainage. One had significant improvement but continued to have minimal drainage. One of three patients with internal fistulas had symptomatic improvement with mean reduction of CDAI of 59%. Seven patients experienced side effects; 4 were minor, mainly headache and chills. Two had exacerbation of asthma symptoms, and one had a Lupus like reaction. Conclusions: 75% of patients with active CD had a significant subjective and objective improvement with infliximab. Six of 7 patients with external fistulae (86%) had complete cessation of drainage from their fistulae, with the 7th patient having a significant reduction in drainage. One of three patients with internal fistulae had symptomatic improvement with mean reduction of CDAI of 59%.