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A MODEL TO PREDICT THE DEVELOPMENT OF POUCHITIS.

J Hinojosa, I Bau, B Martinez, J Garcia, Armengol, JR Moles, JV Roig, P Nosi, J Primo, J Fernandez. Gastroenterology Unit. Surgery Departament and Histopathology Departament. Hospital Sagunto. (*)Hepatogastroenterology Departament. Hospital La Fe. Valencia Spain.

Abdominal colectomy with ileal pouch -anal anastomosis (IPAA) has become the standard surgical treatment for ulcerative colitis (UC). The reservoir mucosa can experience morphological changes (total and subtotal microvilli atrophy and colonic metaplasia) that evoke the colonic mucosa. These changes are in turn associated with changes at mucin composition (colonic sulphomucin in place of ileal sialomucin). pouchitis and the expression of "colonic" antigens. Is nor defined if at the terminal ileon of the patients with UC there are changes at morphology. mucin histochemistry. in macrophages subpoblations and in the immunoglobulins.

AIM: Is to determine if the changes at morphology. mucin histochemistry. immunohistochemistry of the terminal ileon section (localized at the piece of colectomy that was realized after IPAA for UC). the presence of p-ANCA. the HLA status. and the availables clinical variables (previous of colectomy). predict the posterior development of pouchitis.

PATIENTS AND METHODS: Were evaluated the colectomy pieces of 20 patients (12 woman/8 man: 43.3 y) with proctocolectomy and IPAA. All the pieces include 5 cm of terminal ileon (macroscopicaly normal) that were studied using routine histology [acute (IA). chronic (CI) and global (GI) inflammation. the degree of villi atrophy and crypt cell hyperplasia]. mucosal morphometry [calculating the degree of villi atrophy (AVI) and crypt cell hyperplasia]. mucin histochemistry [to distinguish between sialomucin (ileal) and sulphomucin (SF. colonic)] and a study of immunohistochemistry (to determine lymphocytes B.T. macrophages. CD8. IgA and IgG). The determination of p-ANCA was realized by means of indirect immunofluorescence and the HLA by the linfocitoxicity test. For the diagnosis of pouchitis were utilised the Sandborn index. The following characteristics were documented:age.sex.onset of symtoms. disease duration.anatomic location of the disease (universal and extense colitis, proctitis and proctosigmoiditis). evolutive pattern (acute. continue chronic. remitente chronic). weight loss. smoking. extraintestinal manifestations. the flares number of the disease and the treatment with previous costicosteroids at the colectomy. Assesment of the predictive value of pouchitis was performed using stepwise logistic regression analysis (forward LR).

RESULTS: After perfomer stepwise regression logistic analysis were founded three predictive variables of pouchitis development

VARIABLES	B	P
HLA-DR2	-34,9135	0,0002
Weight loss	12,6377	0,0129
Intermitente chronic	-22,4523	0,0048
Constant	10,5078	

The ecuation proposed was: Z=10,5078 - 34,9135 (HLA-DR2) + 12,6377 (Weight loss) - 22,4523 (Intermitent chronic). Probability $P=1/(1+e^{-Z})$

CONCLUSIONS: Although the number of patients is little, the algorithm proposed predicts correctly 92.31% of the pouchitis episodes. The validation of these study is required.

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MORPHOLOGIC AND IMMUNOHISTOCHEMISTRY CHANGES IN THE TERMINAL ILEON OF PATIENTS WITH ULCERATIVE COLITIS: RELATIONSHIP WITH p-ANCA, HLA-DR AND DEVELOPMENT OF POUCHITIS.

J Hinojosa, I Bau, B Martinez, J Garcia-Armengol, J Ferrando, JV Roig, JR Molès. Gastroenterology Unit. Surgery Department and Histopathology Department, Hospital Sagunto, Valencia. Spain.

Abdominal colectomy with ileal pouch anal arastomosis (IPAA) has become the standard surgical treatment for ulcerative colitis (UC). The most frequently observed long-term complication IPAA is acute or chronic inflammation of the ileal reservoir, called pouchitis. The etiology of pouchitis is uncertain. Multiple observations suggest that pouchitis represents a recurrence of UC. Is not defined if at the terminal ileon (TI) of the patients with UC there are changes at morphology, mucin histochemistry, in macrophages subpopulations and in the immunoglobulins. Some studies have indicated that p-ANCA expression is increased in pouchitis but others have not, another authors were unable to demostrate a relationship between ANCA and HLA class-II genes in patients with UC.

AIM: 1) to determine the presence the changes in morphology, mucin histochemistry and immunohistochemistry in the section of the terminal ileon in the piece of colectomy after IPAA for UC; 2) to study if there is relationship between p-ANCA, HLA status, characteristics morphologic/ immunohistochemistry of de IT and development of pouchitis. **METHODS:** The pieces of colectomy of 20 patients (12 woman/8 man; 43.3 y; follow-up mean is 61.6 months) with proctocolectomy and IPAA are evaluated. All the pieces include 5 cm of terminal ileon (macroscopically normal) that are studied using routine histology [acute (AI), chronic (CI) and global (GI) inflammation], mucosal morphometry [calculating the degree of villi atrophy (AVI) and crypt cell hyperplasia], mucin histochemistry[to distinguish between sialomucin (ileal) and sulphomucin (SF, colonic)] and study immunohistochemistry [to determine lymphocytes B, lymphocytes T, macrophages, CD8, IgA and IgG]. The same study have been realized in the IA pouch. The determination of p-ANCA was realized by means of indirect immunofluorescence and the HLA-DR by the lymphocitoxicity test. For the diagnosis of pouchitis the Sandborn index is utilised. Data are expresed as mean (DS) or median (QIR).

RESULTS: Histology, mucosal morphometry and mucin histochemistry: a) TI-UC vs Control: CI (1.2 vs 1, p<0.02), CIG (2 vs 1,p<0.0003), AIV (0.66 vs 0.77, p<0.001), and %SF (2.8 vs 1, p=0.13); b) TI-UC vs IA Pouch: There are significative differences in CI (1 vs 2,p<0.05), AI (0 vs 1.4, p<0.001), CIG (2 vs 4, p<0.05), AVI (0.66 vs 0.49, p<0.05) and %SF (4.3 vs 2.8, p<0.001) between TI-UC and IA pouch.

Study immunohistochemistry: The contenue of IgA, IgG and lymphocytes B in the *IT of UC* is superior to the *IT control* (p<0.005); there aren't differences in the macrophages and CD8 populations (p=NS). *IT Control vs IT pouch IA:* There only are differences in the value of macrophages, lymphocytes B and CD8 (p<0.05). IT-pouchitis vs IT-non-pouchitis: there aren't significative differences in the hystology, mucosal morphometry and mucin histochemistry. The contenue of lymphocytes T, CD8 and macrophages in the *IT-pouchitis* is superior to *IT-non-pouchitis,* but the differences aren't significant. p-ANCA/HLA: p-ANCA are positives in 11 patients (55%) and negatives in 9 patients (45%). The 36.6% of patients with p-ANCA (+) (4 patients) are also HLA-DR2 (+) and only one patient with p-ANCA (-) expres HLA-DR2. Two patients with p-ANCA (+) (18.2%) and three patients with p-ANCA (-) (33.3%) expres HLA-DR4, respectively. During the follow-up, 7 patients have developped pouchitis (35%) and only two patients were p-ANCA (+) (28.6%) Only one patient with p-ANCA (+) and HLA-DR2 (+) have develop pouchitis. Of the 9 patients with p-ANCA (-), four developped pouchitis but only one of them expres HLA-DR2.

CONCLUSIONS: *1)* The IT of patients with UC is different (histology, morphometry, mucin and immunohistochemistry) of the IT of patients without UC; these alterations are independents of the posterior development of pouchitis *2)* There aren't relathionship between positivity of p-ANCA, HLA-DR2 status and pouchitis.

G4081

ARE THERE *HELICOBACTER PYLORI* ON GASTRIC MUCOSA IN SUDDEN INFANT DEATH SYNDROME (SIDS)?

G.Y. Ho, H.M. Windsor, C.P. Pattison*, G.G Vergara*, B.J. Marshall, Departments of Medicine and Gastroenterology, University of Western Australia and U.Mo-KC School of Medicine, Kansas City*, Missouri.

INTRODUCTION: The cause of SIDS is unknown but its epidemiology parallels that of *H. pylori* in Western Countries; i.e. declining incidence and association with lower socio-economic status. Since apparent *H. pylori* have been seen in histological sections from some SIDS cases (Gastroenterol 1997; 112:A254), it is important to know the prevalence of *H. pylori* in SIDS.

AIM: Using molecular identification methods, to determine the prevalence of *H. pylori* in SIDS and compare this with the prevalence in non-SIDS infants.

METHODS: At the UKMC, infants with a post mortem diagnosis of SIDS were studied consecutively and classed as SIDS vs. Non-SIDS. At post mortem, samples of gastric mucosa and trachea were fixed in formalin, and mounted in paraffin. Laboratory studies were performed blind as to other results and the clinical findings. When sectioning tissue blocks, the microtome blade was cleaned between each case. After staining with H&E and toluidine blue, sections were examined under oil for the presence of curved or spiral organisms consistent with *H. pylori.* In addition, four 10 μM sections from each were de-waxed in xylene, digested with proteinase K, and the DNA amplified using primers developed by Kawamata (Biochem. Biophys. Res. Comm. 1996) which target a 314 bp segment of the urease A gene. After amplification, DNA was spotted onto a nylon membrane and hybridized with a digoxigenin labeled 130bp probe which detects a sequence internal to the 314 amplicon. To validate the PCR probe, we confirmed that it reliably detected *H. pylori* in control sections with a histological grading of 1+, 2+ and 3+ organisms but was negative on gastric biopsies from HP-neg patients as well as from skin and from muscle.

RESULTS: Adequate sections of gastric mucosa and trachea were obtained from 22 SIDS infants. Most gastric specimens exhibited considerable autolysis so that that the luminal epithelial cell layer could not be identified. In half the cases bacteria were seen but these did not resemble *H. pylori.* None of the specimens produced a band on the initial PCR or a signal from the dig. Probe although control lanes reacted well. The experiment was repeated with the same negative result.

CONCLUSION: In this study we were unable to confirm that gastric bacteria seen in cases of SIDS were *H. pylori.*

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CORTISOL SUPPRESSION AFTER ORAL DELIVERY OF BUDESONIDE IN pH-MODIFIED RELEASE CAPSULES.

G.Hochhaus[1], M.Wagner[2], H.W.Möllmann[2], A.Tromm[2], A.C.Möllmann[2], S.Homrighausen[2], J.Barth[2], M.Krieg[2], [1]University of Florida, Gainesville, USA, [2]University of Bochum, Germany.

Local delivery of glucocorticoids for the therapy of inflammatory bowel disease (IBD), including Crohn's disease attempts to achieve pronounced local effects with reduced systemic side effects. The design of pH modified release capsules seems to be promising as it allows the specific release of budesonide at a pH of 6.4, a pH realized in the colon close to the disease area. Aim of this study was to investigate cortisol suppression observed after different doses of