55

THE MORTALITY OF IN-HOSPITAL PEPTIC ULCER PERFORATION

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Despite the benefit of prophylactic treatment against stress ulcers occurring in ill patients (pts), perforated ulcer disease occurring in hospitalized patients continues to be an important problem. The clinical course of 19 patients treated for in-hospital peptic ulcer perforation was compared to that of 41 pts who presented to the hospital during the same interval with a perforated ulcer. Pts were evaluated regarding previous history of ulcer disease, use of ulcerogenic substances, preperforation ulcer prophylaxis and the type of operative therapy employed. The severity of illness was evaluated by an acute physiologic score (APS).

	In-Hospital Perf.		Prehospital Perf.	
	Patch Closure	Ulcer Op.	Patch Closure	Ulcer Op.
No. Pts.	12	7	16	25
Prev. Ulcer Hx. (%)	33	43	31	48
Preperf. Ulcerogenic Substance (%)	33	57	63	60
Preperf. Ulcer Prophylaxis (%)	17	0	19	20
APS (Mean ± SEM)	14 ± 5	17 ± 10	9 ± 7	8 ± 6
Mortality (%)	50	57	13	12

Pts operated on for ulcer perforation occurring in the hospital were more ill and had a significant delay in diagnosis (3.3 ± 7 days) vs. prehospital perforation pts (1.1 ± 1 day). No significant differences in outcome could be discerned between pts treated with patch closures or definitive ulcer operations. In-hospital ulcer perforation is associated with a high mortality. Greater attention to stress ulcer prophylaxis in hospitalized patients is indicated.

56

HOW DOES THE ENDOSCOPIST DECIDE WHICH PATIENTS SHOULD BE BIOPSIED FOR H.PYLORI? C.G. Antonescu, MD, B.J. Marshall, MD, FACG, H.F. Frierson, MD, R.L. Guerrant, MD, R.W. McCallum, MD, FACG, University of Virginia, Charlottesville, VA 22908.

H.pylori (HP) causes chronic gastritis, a common histologic finding in patients with dyspepsia. **THE AIM** of this study was to search for endoscopic correlates of histologic gastritis in a large group of patients in whom HP status had been determined. **METHODS**. All patients referred to our Dyspepsia Clinic between 1987 and 1990 were entered. The initial findings were recorded by the endoscopist at the time of the procedure. For analysis, each patient was included only once and the grading system was based on the most severe endoscopic lesion. At endoscopy 4 mucosal biopsies (2 antrum, 2 body) were taken for histology and 1 for culture. The diagnosis of HP gastritis was made if the culture was positive or CLO were present on biopsy and there was associated active chronic gastritis. Statistical analysis was done by Fisher's exact test. **RESULTS**. Of 211 patients enrolled, 73 were HP-, 138 were HP+. The age distribution was similar in the two groups and 50% were males. Endoscopy was normal in 83 (61% HP+), while in 128 (68% HP+) at least one abnormality was found on endoscopy. Abnormal esophageal findings in 24 patients were equally distributed in the HP- and HP+ groups. Of the 68 patients with a gastric abnormality, 70% were HP+ and in these, antral ulcerations were significantly more common (11/48) than in HP- group (1/20) ($p<0.01$). There was no significant difference in the prevalence of either a "red stomach" or minor erosions in the HP+ vs. HP- group. Of 36 patients with duodenal lesions, 27 were HP+ (75%). All 9 patients with duodenal ulcerations were HP+ ($P<0.05$). Endoscopic duodenitis or erosions were not clearly associated with HP (70% HP+). **CONCLUSIONS**. 1. In patients without actual peptic ulceration, the presence or absence of H.pylori (or histological gastritis) cannot be predicted by the endoscopic appearance of the mucosa. 2. It is not logical to select patients for biopsy on the basis of gastric erythema or minor gastric erosive disease. 3. The endoscopist should routinely biopsy the gastric mucosa in dyspeptic patients with a normal EGD.

57

OPTIMIZATION OF MEDIA FOR THE LONG-TERM CULTURE OF GASTRIC EPITHELIAL CELLS FROM THE PATIENT WITH MENETRIER'S DISEASE. Calin Antonescu, MD, Jerzy Sarosiek, MD, Barry Marshall, MD, FACG, Hannah Anderson, Joyce Hamlin, MD, and Richard McCallum, MD, FACG. Departments of Internal Medicine and Diabetes Research Center, University of Virginia, Charlottesville, VA 22908.

INTRODUCTION: A few previous reports have described the in vitro growth of heterologous gastric mucus cells, but reproduction of techniques has been difficult. The availability of a fully differentiated gastric mucus cell line would be important in creating an in vitro model of H.pylori infection. In Menetrier's disease, the normal distribution of specialized cells in the glands of the body of the stomach is variably replaced by hyperplastic mucin secreting cells. **AIMS**: To define a technique which would allow us to maintain perpetual growth and attachment of a primary gastric mucus cell line (Menetrier's cells). **METHODS**: The growth of epithelial cells was maintained in 24 well- tissue culture plates in 5% CO_2. To enhance the attachment of gastric epithelial cells, tissue culture wells covered with extracellular matrix components (laminin or collagen) were developed. **RESULTS**: Optimal growth was maintained in media containing: 50% DMEM, 50% F-12 Nutrient Mixture (Ham) supplemented with 10% FBS and 1% PN/SM. Pentagastrin (5μg/ml) and Epidermal Growth Factor (0.1 μg/ml) improved growth slightly. Gastric epithelial cells obtained from a full thickness endoscopic biopsy and maintained over 24 weeks in culture still showed strong morphological resemblance to mucus cells, and remained mitotically active with continuous secretion of mucus in the culture medium. However, a continuous monolayer was not yet obtained. **CONCLUSIONS**: 1. Gastric mucosal cells obtained from the biopsy of a patient with Menetrier's disease have well preserved proliferative activity in vitro, perhaps related to their very high mitotic activity in vivo. 2. Since morphology and function of these epithelial cells are preserved throughout their long-term maintenance in culture, this model may be suitable for the study of host cell-bacteria interaction.

58

DOES SMOKING, ALCOHOL OR NSAIDS AFFECT THE PRESENCE OF HELICOBACTER PYLORI? Bashar M. Attar, M.D., F.A.C.G., Benjamin T. Go, M.D., Frank Kocka, M.D., Cook County Hospital, Hektoen Institute for Medical Research and Rush University Medical Center, Chicago, Illinois.

Helicobacter pylori (HP) has been implicated in the pathogenesis of Type B chronic gastritis. The purpose of this prospective study was to investigate the role of smoking, alcohol or NSAIDs in HP-associated gastritis. 246 consecutive patients underwent elective upper endoscopy. They were interviewed beforehand for any history of smoking, alcohol or NSAIDs use and these data were not revealed to the endoscopists. Antral biopsies were done and the presence of HP was determined by a positive urease test, culture and histology.

Of the 246 patients, 147 gave a history of active smoking, alcohol or NSAIDs use (Group A). Histologic evidence of chronic active gastritis (CAG) was seen in 78% compared to 65% of patients who gave a negative history to smoking, alcohol or NSAIDs use (Group B). The incidence of HP positivity was similar in the two groups showing CAG (69% vs 70%), but significantly lower compared when CAG is absent (44% and 31%) respectively. In patients where smoking, alcohol and NSAIDs use were seen together in the same patient, CAG was seen in 90%, all of which were positive for HP.

Conclusion: Antral gastritis by itself is a major predisposing factor for the presence of HP regardless of other associated factors. Smoking, alcohol or NSAIDs use increase the incidence of gastritis, and thus indirectly the presence of HP infection.