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EFFICACY OF OMEPRAZOLE AND AMOXICILLIN TO ERADICATE HP

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Elaborate triple therapy schedules eradicate Helicobacter pylori (HP) in a high percentage of treated patients. However, a simple therapy regimen with low complications and adequate efficiency is not available up to now.

In five open clinical studies, 181 HP-positive patients with ulcer disease (n=162) or severe functional dyspepsia (n=19) were treated with 40 mg omeprazole (OME) and 4x500 mg amoxicillin (AMOX) suspension over one week (n=35, group I), 2x40 mg OME and 4x500 mg AMOX over one week (n=50, group II), 2x20 mg OME and 4x500 mg AMOX over two weeks (n=62, group III), 2x20 mg OME day 1-14 and 4x500 mg AMOX day 8-14 (n=22, group IV), or 2x20mg OME + 2x1 g AMOX (tablets) over two weeks (n=12, group V). The HP-status was evaluated pre-treatment and during the fifth week after discontinuation of study medication by an urease test, specific culture and histology after modified GIEMSA staining.

169 out of 181 patients completed the studies without contravening the protocols. The proportion of HP eradication was 61.3% in group I, 61.7% in group II, 82.8% in group III, 28.6% in group IV and 83.3% in group V. 15 out of 169 patients (8.9%) complained of side effects that led to therapy discontinuation in 4 cases (2.4%).

In conclusion, combined amoxicillin/omeprazole treatment eradicates HP in about 60% to 80% of patients. The best resultes were obtained with a two weekly schedule comprising 2x20 mg omeprazole and 4x500 mg or 2x1 g amoxicillin. Pretreatment with omeprazole endangers the efficacy of this novel therapy regimen.

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ENDOSCOPIC LIGATION OF FUNDIC VARICES. Glaciomar Machado, Ph.D., Universidade Federal do Rio de Janeiro, Brazil.

Gastric fundal varices represent a major limitation for sclerotherapy because the commonly used sclerosant agents frequently cause mucosal ulceration and severe bleeding when injected in such varices. In 1986 Stiegmann et al. reported a "new elastic band ligating device" to treat esophageal varices. From May to December,1991 we are using such device for the treatment of fundic varices. Twelve patients were treated, 8 males and 4 females agying 46 to 70 years and were grouped according to Child - Pugh classification: 1 patient in class A; 7 in B and 4 in C. Regarding etiology of portal hypertension, 7 patients presented with alcohol-related cirrhosis; 3 with post-hepatitis cirrhosis and 2 patients with chronic active hepatitis. Eleven out of 12 patients were treated electively (at least one previous episode of bleeding) and the remaining one was treated prophylactily (Child class C with large varices with more than 10 red spots signs). Nine patients presented with large size varices and 3 patients with medium. Small varices were not treated.Treatment includes repeated sessions on a 2 weeks interval and follow-up EGD at 30,90,180 days and every 12 months after conclusion of treatment. Varices disappeared in all patients and no complications were observed but small erosions on the sites where elastic bands were placed.

Although such results are optimistic, more data is necessary for confirmation of the efficacy and safety of this newer form of endoscopic therapy before firm conclusions can be drawn.

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HELICOBACTER PYLORI PREVALENCE IN AIDS. Benjamin J. Marano, Jr., M.D., Fred Smith, M.D., C. A. Bonanno, M.D., St. Vincent's Hospital & Medical Center of New York.

Helicobactor pylori is consistently reported with high prevalence in HIV negative patients with gastritis and active ulcer disease. This study is an evaluation of the prevalence of H. pylori in AIDS patients and the association with gastritis and active ulcer disease.

Seventy three AIDS patients referred for evaluation of gastrointestinal symptoms underwent upper endoscopy and gastric biopsy. Histologic gastritis was graded on hema-toxylin-eosin stain and H. pylori was identified by acridine orange stain. A single pathologist evaluated the biopsy specimens.

H. pylori was found in 15% (11 of 73) of AIDS patients. Histologic gastritis was evident in 94.5% (69 of 73) of the study group. H. pylori was identified in 15.9% (11 of 69) of biopsy specimens with histologic gastritis. The organism was more common in biopsy specimens with a higher grade of histologic gastritis. Endoscopic ulcerations were noted in eleven patients (seven gastric, four duodenal). H. pylori was present in 18% (2 of 11) of AIDS patients with active ulcerations.

The prevalence of H. pylori in AIDS patients with histologic gastritis and active ulcer disease is much lower than the prevalence previously reported for HIV negative patients with similar pathology. The low prevalence observed does not implicate H. pylori as the causal agent in most gastritis and ulcer disease in the AIDS population. Impaired acid secretion may reduce colonization of gastric mucosa and explain the low rate of H. pylori observed.

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H.PYLORI (HP) INFECTION IN THE SPOUSES OF INFECTED PATIENTS. *Barry J. Marshall, MD, FACG, Susie R. Hoffman, RN, Richard L. Guerrant, MD, Richard W. McCallum, MD, FACG.* University of Virginia, Charlottesville, VA 22908.

The prevalence of H.pylori in the United States is known to vary depending on country of birth, socioeconomic status, and race, but the mode of HP transmission is uncertain, with both fecal-oral and oral-oral transmission being likely. **AIM**: To determine the prevalence of HP in the spouses of patients and to correlate this with the HP status of the patient. **METHODS**: Patients who attended the gastroenterology clinic with their spouses had HP status determined by endoscopic biopsy and HP status of the spouse was determined by ^{14}C-urea breath test. Demographic data was collected on all subjects. Data was analyzed using S.A.S. Cochran-Mantel-Haenszel statistics. **RESULTS**: 115 patients were studied (58 F, 67 M), mean age 51 years, 5 patients were black, 100 white, and 10 others. Excluding black patients (who were all HP+ with HP+ spouses), 67% of the patients were HP+ as were 44% of their spouses. When the patient was HP+, the spouse was infected 54% of the time. When the patient was HP-, the spouse was infected 25% of the time. The relative risk of infection in the spouse was 3.4 (1.4-8.2) if the patient was HP+ ($p<0.006$), independent of age, sex, and race. If the patient was HP+, age did not affect prevalence of HP in the spouse, with 54% of spouses <40 years of age still being HP+. **CONCLUSIONS**: Most spouses of HP+ patients are also infected (54%). Spouses of HP- patients have HP prevalence (25%) similar to that seen in controls (blood donors). Spouses of HP+ patients tended to be infected regardless of age, suggesting that infection occurs soon after cohabitation.