2006 DDW *H. pylori* abstracts

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The number of abstracts submitted to DDW relating to *H. pylori*, especially ulcer disease, continues to decline, paralleling the worldwide decrease in prevalence of the infection. Nevertheless there were some interesting and important presentations which I will mention.

H. PYLORI DYSPEPSIA AND SCREENING

Debate still surrounds the effectiveness of *H. pylori* eradication compared to empiric PPI treatment in the management of dyspepsia. Delaney (*Gastro* 2006 Volume 130 (Supplement 2) A-38) presented efficacy and cost effectiveness data for *H. pylori* eradication. Noting that previous reports evaluating management of dyspepsia suggest treatment of H. pylori is less satisfactory but more cost effective than endoscopy, he compared 1-year efficacy and cost of *H. pylori* eradication and treatment with PPIs. Analyzing two studies enrolling 571 patients he showed that eradication was more effective for treating dyspepsia but not heartburn than PPIs. The calculated monetary benefit in favor of *H. pylori* treatment was $6.12.

Hansen (*Gastro* 2006 Volume 130 (Supplement 2) A-37) provided five year follow up information on a large cohort of middle aged individuals randomized to screening and treatment of infection or to a control group. Quality of life was assessed at baseline and one and five years. 5-year data were available for 11,000 subjects (17.5% Hp+). He observed only a modest benefit on dyspeptic symptoms and related health care cost in the Hp+ group compared to Hp- and controls. He concludes in a population with low Hp prevalence, screening has only a modest clinical and economic impact.

Ford (*Gastro* 2006 Volume 130 (Supplement 2) A-459) provides 10- year follow up of 8400 individuals (age 50-59) enrolled in a *H. pylori* screening and treat program. A total 140 deaths occurred - 64 related to cancer (2 UGI cancers both in Hp- subjects). Ischemic heart disease accounted for 31 deaths and was increased in Hp+ subjects. He also observed a trend toward increased ischemic heart disease mortality following *H. pylori* treatment. He concludes that *H. pylori* infection confers an increase all cause mortality and cautions that treatment may actually increase cardiac related deaths.

Yaghoobi (*Gastro* 2006 Volume 130 (Supplement 2) A-400) conducted a meta analysis of studies to access if *H. pylori* treatment affected GERD symptoms. Four randomized control trails and 4 cohort studies in duodenal ulcer patients (endoscopy was GERD endpoint) and 3 randomized control and one cohort dyspepsia study (endoscopy was GERD end point) and 5 dyspepsia randomized control trials (heartburn GERD endpoint) were analyzed. *H. pylori* did not appear to increase GERD after treatment in dyspepsia patients but did increase esophagitis (1.5 -2 fold) in duodenal ulcer patients. The increase esophagitis was only seen in cohort and not randomized controlled trials.

H. PYLORI TREATMENT AND RECURRENCE

Primary treatment for *H. pylori* infection has variable success depending on such things as patient compliance, duration and type of treatment regimen and antibiotic resistance. A number of papers at *DDW* 2006 addressed treatment regimens, especially retreatment.

A systematic review conducted by Gisbert (*Gastro* 2006, Volume 130 (Supplement 2) A-571) concluded that 10 day levofloxacin, amoxicillin and a proton pump inhibitor is the favored retreatment regimen. It is better than a 7- day regimen and more effective and better tolerated than quadruple therapy.

Miehlke (*Gastro* 2006, Volume 130 (Supplement 2) A-93) reported the efficacy of a novel moxifloxacin treatment regimen in individuals with dual imidazole and macrolide resistant *H. pylori*. Once daily doing for seven days with esomeprazole (40 mg), moxifloxacin (400 mg) and rifabutin (300 mg) was 80 % effective in eliminating dual resistant organisms which offers support for its use after multiple other regimens have failed.

H. pylori recurrence rates, especially true re-infection, are low. Gisbert (*Gastro* 2006, Volume 130 (Supplement 2) A-572) presented a five year follow up of 1,000 patients treated for *H. pylori* infection. Therapies they received were classified as low or high efficacy and individuals had a urea breath test 4 to 8 weeks following initial treatment and then yearly. *H. pylori* cure rates were high 94.7% and 90.7% at one and five years respectively. Recurrence was 2.6% per patient year. Most recurrences occurred in the first year which suggests they were actually recrudescent infections not true re-infection. Those who had evidence of infection at 1-year tended to be younger and had been treated with a low efficacy regimen.

H. PYLORI TRANSMISSION IN CHILDREN

H. pylori infection is acquired through fecal/oral or oral/oral mechanisms during early childhood.

Kori (*Gastro* 2006, Volume 130 (Supplement 2) A-459) addressed the prevalence and transmission of *H. pylori* infection using stool antigen testing of children > 2 months of age attending day care centers in Israel. Through questionnaires he assessed family origin, size and crowding. Of 316 children enrolled in the study (mean age 18.9 months), 78 (24.7%) were infected with *H. pylori*. Prevalence of infection increased with age from 7.1% to 31% to 35% in children < 12 months of age, 12 to 24 months of age and > 24 months of age respectively. He concluded that 25-30% of children in day care facilities in Israel are infected with *H. pylori* and the prevalence of infection increases with age.

Ford (*Gastro* 2006, Volume 130 (Supplement 2) A-460) accessed the influence of sibling number and birth order on *H. pylori* infection in 4,000 participants of a community screening program. *H. pylori* prevalence was 20% in those with no siblings and as high as 63% in those with seven or more siblings. The prevalence increased with > 3 siblings. Birth order and other factors such as shared beds and bathrooms, lack of indoor toilets and father's socioeconomic status also appeared to influence the prevalence of *H. pylori* infection.

H. PYLORI VARIANCE FACTORS

Last year at *ECOS I* presented data suggesting that *H. pylori* Dup-A gene was associated with duodenal ulcer in Japan and Korea and reduced gastric cancer risk in Japan, Korea and Columbia. Attempting to confirm these observations Queiroz (*Gastro* 2006, Volume 130 (Supplement 2) A-145) evaluated 487 H. Pylori strains isolated from Brazilian patients. Isolates came from children (38 DU; 100 gastritis) and adults (125 DU; 143 gastritis; 81 gastric cancers). He noted that 99% of the strains in children tested positive for the Dup-A gene while approximately 87% of adults had Dup-A positive strains and most were without disease association. He concluded that Dup-A + strains are frequent in Brazil and are not necessarily associated with an adverse clinical outcome. It is likely that there are regional differences in Dup-A gene prevalence as is the case with other putative virulence markers.