ENDOSCOPIC NO:YAG LASER THERAPY FOR WATERMELON ENDOSCOPIC NO. TAG LABER THERAPY FOR WATERMELON STOMACH. Craig Radford, M.D., David Ahlquist, M.D. https://documents.com/phistopher Gostout, M.D., Thomas Viggiano, M.D. Clinic, Rochester, MN 55905.
Watermelon stomach is a recently described wascular malformation which can cause significant visualing. Antrectomy has controlled blood loss in

bleeding. Antrectomy has controlled blood loss in bleeding. Intercepting has controlled blood loss several published cases. We report preliminary several passes of the endoscopic Nd:YAG laser in the treatment of five patients with severe anemia due to this condition. There were three women and two to this condition. There were three women and two men with a median age of 65 years (range 54-86). Despite iron and anti-ulcer therapy, the median hemoglobin was 6.7 g/dl (range 6.1-7.8), and four of the patients had required blood transfusions in the preceding six months (range 3.17 moits). the preceding six months (range 3-17 units). The median duration of anemia was 12 months (range 3-24 months). Each patient had typical vascular lesions in linear clusters distributed radially from the pylorus across the antrum. Colonoscopy revealed small sigmoid vascular malformations in one patient and was more unremarkable in the others. Laser therapy was administered with a median power of 40 watts (range 35-65) and a median energy per session of 4,626 joules (range 2,879–14,926). Initial treatment was accomplished in either one or two sessions; no complications were encountered. Iron was continued in all patients. In follow-up ranging from two to nine months, one patient with Child's C cirrhosis has required a blood transfusion. In the three patients followed for three months or longer, median hemoglobin at three months was 10.7 g/dl (range 9.7-11.9). We conclude that endoscopic laser treatment may offer an alternative to surgery for the treatment of bleeding due to watermelon stomach. Further studies are indicated to determine long-term effectiveness.

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CAMPYLOBACTER PYLORI IN DIFFERENT SOCIO-ECONOMIC GROUPS IN PERU. A. Ramirez-Ramos, M.D., O. Hurtado Munoz, M.D., C. Rodriguez Ulloa, M.D., R. Leon-Barua, M.D., G. Garrido Klinge, M.D., S. Recavarren Arce, M.D., R. Gilman, M.D., W. Spira, Ph.D., and J. Watanabe Yamamoto, M.D. Universidad Peruana Cayetano Heredia, Lima, Peru and The John Hopkins University, Baltimore, MA.

The purpose of the study was to determine if an association between the socio-economic level of Peruvian patients and their rate of colonization with <u>Campylobacter pylori</u> (CP). We studied two different groups of patients one who came to a public hospital attended by patients from the lower or lower middle class and the other a private clinic catering to the elite. All patients came to endoscopy because of symptoms associated with the upper gastrointestinal tract.

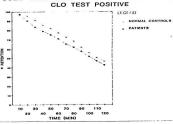
Patients were endoscoped and biopsies taken

from the upper and lower curvature of the antrum and stained with silver to detect Campylobacter-like organisms in the mucosa. In the public hospital population, 143 patients were examined and 105 (73%) found to be CP-positive. In the private 85 of 122 patients (69%) were clinic group,

Thus, there appears to be no significant difference in the rate of colonization with CP in patients coming from different socio-ecomic strata in a developing country.

GASTRIC EMPTYING OF SOLIDS IN PATIENTS WITH CAMPYLOBACTER PYLORI GASTRITIS: C Prakash, BJ Marshal, MW Plankey, R. Guerrant, and RW McCallum, M.D., F.A.C.G. University of Virginia, Charlottesville, VA 22908

Recent reports have shown a strong association between Campylobacter pylori (CP) and duodenal ulcer (DU)(95%), gastric ulcer (GU)(75%) and Gastritis(50%). The organism has been isolated from the antrums of all these patients. Since the antrum is instrumental in the emptying of solids from the stomach we wanted to determine whether: 1) CP colonization with antral gastritis impairs gastric emptying (GE) of solids and, 2) antral dysmotility could help explain the symptoms of dyspepsia. Patients presenting with dyspepsia including epigastric burning, indigestion, post-prandial epigastric pain, bloating, belching, heartburn, nausea, or vomiting were examined, and detailed questionnaires were completed. They underwent endoscopy with routine biopsies subsequently sent for histopathology, Gram's stain and culture. They also had a GE study utilizing chicken liver labeled with 99mTc-sulfur colloid mixed with beef stew.



SOLID MEAL GASTRIC EMPTYING RESULTS CLO TEST POSITIVE 2 hrs. of imaging with pts positioned supine under the camera. Results: 10 patients with positive CP cultures and appropriate symptoms were identified. Their GE were identified. Their Gl of solids (see graph) was similar to 12 normal subjects studied as controls. 1 patient with a bezoar had abnormally slow GE.

He also presented with halatosis and GE normalized after

treatment and clearance of CP.

Concl: CP does not affect GE of solids in the presence of antral gastritis. This suggest that mucosal inflammation (endoscopic redness and histologic infiltration with PMS) is a mucosal event without any apparent effect on muscular functions.

PROTECTION OF CAMPYLOBACTER PYLORI (CP) AGAINST ACID

SUSCEPTIBILITY BY UREA. C. Prakash, B. Marshall, L. Barrett, R. Guerrant, R. McCallum, FACG, University of Virginia School of Medicine, Charlottesville, VA 22908
CP colonization of the stomach is present in 30-60% of dyspeptic patients who undergo upper gastrointestinal endoscopy. CP is usually isolated by culture of a gastric biopsy, and in 20% of positive cases live covarians can biopsy, and in 20% of positive cases live organisms can be recovered from the gastric juice. However, the ability of the bacterium to survive in the range of acidity present in the stomach and the role of its urease in its survival is uncertain. Therefore, we examined the pH susceptibility of CP for comparison with campylobacter

jejuni (CJ) in the presence and absence of 5 mM urea.

A healthy 2-3 day growth of CP was harvested from a horse blood agar plate (BAP) into 3 ml normal saline at pH 7.0 to give a cloudy suspension. 1:10 dilutions were made in phosphate buffered saline at pH's 1.5 to 7.0 and, after 30 minutes at 27° C cuantitative cultures were after 30 minutes at 37° C, quantitative cultures were done on BAPs which were incubated for 3-5 days at 37° in a microaerophilic environment. Sheep BAPs at 42° C were used for CJ cultures. We found that while CP survived well to pH 4-4.5, it was reduced by 3-5 logs at

≤ pH 3.5.

CJ was similar in its acid susceptibility to CP. striking contrast, the addition of 5 mM urea completely protected CP but not CJ from acidic pH's down to 1.5. protected CP but not CJ from acidic ph's down to 1.5. This occurred in two ways: 1) by neutralizing the solution ph from  $\geq$  3.0 to  $\geq$  6.6 and 2) in addition, however, urea did not alter the highly acidic solution phs of 1.5, 2.0, or 2.5 but still fully protected the CP organism. These findings show that urea allows the organism to protect its "microenvironment" in highly acidic solutions as well as to provide a modest buffer in mildly acidic solutions. The high urease activity of CP minutes allowed the ability to survive in acid and mildly acidic solutions. The high urease activity of uniquely allows it the ability to survive in acid and thus may be an important prerequisite for colonization of the mammalian stomach.