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Prevalence of *Helicobacter pylori* and Antibiotic Resistance in An Aboriginal Population in Canada's Arctic: Preliminary Results from the Aklavik *H. pylori* Project

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Background: The rate of gastric cancer in the Northwest Territories Aboriginal population is higher than the rest of the Canada. The aims of this study were to determine the *Helicobacter pylori* (Hp) prevalence and antibiotic resistance pattern in this community. **Methods:** As part of a community health project focused on Hp risks, including urea breath test (UBT) screening, residents (age ≥ 9 yrs) of Aklavik, a remote town of 620 inhabitants in the Northwest Territories of Canada, were invited to undergo endoscopy in February 2008. An on-site unit was assembled in the local health centre to perform upper gastrointestinal endoscopy with gastric biopsies for Hp culture and antibiotic susceptibility. Positive Hp cultures were tested using the E-test method for susceptibility to metronidazole, clarithromycin, amoxicillin, ciprofloxacin, tetracycline, nitrofurantoin, and rifampin. **Results:** Hp prevalence was 55% among the 240 residents who had a UBT. Of all eligible participants, 35% (n = 194) underwent endoscopy and biopsies for culture, with UBT-positive residents more likely to consent to endoscopy. The mean (\pm SD) age of the participants was 40 (\pm 17) years and 59% were female. Hp culture results were available from 170; 117 (69%) were Hp positive. Antibiotic resistance was found in 33% (n = 33) of 99 Hp-positive cultures tested. Resistance to only metronidazole, clarithromycin, or amoxicillin was present in 23% (n = 23), 4% (n = 4), 0% (n = 0), respectively. There were no cases of resistance to rifampin and tetracycline. Multi-drug (≥ 2) resistance was present in 6 cases (6%). **Conclusion:** There is a high prevalence of Hp infection with relatively low levels of antibiotic resistance and occasional multi-drug resistance to antibiotics among residents in this Canadian arctic hamlet. A randomized control trial is being conducted to determine the optimal treatment to cure Hp infection in this community.

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Impact of Efficacies of Triple Therapy Using Lafutidine Plus Amoxicillin-Metronidazole for Proton Pump Inhibitor-Amoxicillin-Clarithromycin Treatment Failures for *Helicobacter pylori* Infection

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Background: The failure of first-line anti-*Helicobacter pylori* (*H. pylori*) therapies consisting of a proton pump inhibitor (PPI), amoxicillin and clarithromycin is increasing due to primary antibiotic resistance. In Japan, only the following regimen: A 7-day course of proton pump inhibitor-amoxicillin-metronidazole is recommended as second-line *H. pylori* therapy and covered by the national health insurance. Lafutidine is H₂-receptor antagonist with gastroprotective action through capsaicin-sensitive afferent neurons and relatively inexpensive compare to PPI. And there is a little evidence available on eradication therapy including H₂-receptor antagonists compared to PPI based therapy. **Aim:** To assess the efficacy and safety of second-line eradication using the H₂-receptor antagonist lafutidine as a substitute for PPI sodium rabeprazole. **Methods:** Fifty-one patients who failed in first-line *H. pylori* eradication using PPI-amoxicillin-clarithromycin were randomly assigned to either second-line therapy including amoxicillin and metronidazole: A 7-day course of sodium rabeprazole 10 mg b.i.d., amoxicillin 750 mg b.i.d., and metronidazole 250 mg b.i.d. (rabeprazole group); or a 7-day course of lafutidine 10 mg t.i.d., amoxicillin 750 mg b.i.d., and metronidazole 250 mg b.i.d. (lafutidine group). Eradication was assessed for each group by 13C urea breath test at least 6 weeks after completing eradication therapy. Drug susceptibility test was performed using 40 strains in pretreatment to amoxicillin, clarithromycin and metronidazole. **Results:** Prior to second-line *H. pylori* eradication, the rate of resistance to clarithromycin was high at 82.5% (33/40). Similarly, resistance to metronidazole was low at 2.5% (1/40); however, no amoxicillin-resistant strains were found. The eradication rates for both lafutidine group and rabeprazole group were high at 96% (25/26) and 100% (25/25), respectively. **Conclusions:** Lafutidine plus metronidazole-amoxicillin as second-line therapy provided a high eradication rate and safe treatment similar to sodium rabeprazole-amoxicillin-metronidazole therapy. Lafutidine-based eradication therapy is therefore considered significant for patients whom proton pump inhibitor are unsuited and also anticipated to reduce health-care costs in *H. pylori* eradication.

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Regression of Atrophy in Patients with Atrophic Body Gastritis Following *Helicobacter pylori* Treatment: Occurrence and Predictor Factors

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Background: Atrophic body gastritis (ABG) is considered a condition predisposing to gastric neoplasia. Hp infection is present in the vast majority of ABG patients. In ABG patients with Hp infection, the effect of Hp cure with respect to the potential regression of atrophic damage is still debated. **Aim:** To evaluate the occurrence and to identify predictor factors for the body atrophy regression in ABG patients following Hp eradicating treatment. **Methods:** 186 ABG patients (F=135; median age 51 [18-78] years) were included. ABG diagnosis was based on hypergastrinaemia and histological presence of body atrophy. Hp infection was diagnosed by histology (Giemsa) and serology (IgG ELISA). All patients were treated for Hp infection (bismuth-based triple regimen) and underwent regular endoscopic follow-up investigations. Gastroscopy with antral (n=3) and body (n=3) biopsies was performed and histology evaluated according to the updated Sydney System and OLGA System at baseline and at follow-up (median follow-up of 3 [0.5-16.5] years). **Results:** After Hp eradication treatment, body atrophy reversed in 51 (27.4%) patients (median age 46 [18-77] yrs). In 44 (86.3%) of these patients the regression of body atrophy was observed at 6-12 months after treatment. The comparison between ABG patients with and without regression of body atrophy showed, respectively, the following significant differences: female gender (84% vs 68%, p=0.02), gastrinemia [185 (40-1650) pg/ml vs 500 (40-2500) pg/ml, p<0.0001],

pepsinogen I levels [26 (3-176) ng/ml vs 10 (3-84) ng/ml, p=0.0003], parietal cells antibodies (58% vs 81%, p=0.0042), pernicious anaemia (5.8% vs 32.8%, p=0.0001), iron deficiency anaemia (72.5% vs 51.1%, p=0.012), presence of metaplastic atrophy (37% vs 80%, p<0.0001). Grade 4 and Stage 1 of OLGA system showed a significantly higher prevalence in ABG patients with regression of atrophy than in the others (p<0.002). Logistic regression identified as predictor factors for regression of body atrophy: pernicious anaemia (OR= 0.11, 95%CI 0.02-0.71), metaplastic atrophy (OR=0.14, 95%CI 0.05-0.42), Grade 1 (OR=0.18, 95%CI 0.04-0.74), Stage 1 (OR= 3.6, 95%CI 1.3-9.95) and parietal cells antibodies (OR 0.25, 95%CI 0.09-0.71). **Conclusion:** Treatment of Hp infection may reverse body atrophy in 27% of ABG patients. This subset of ABG patients is characterized by the absence of indicators of severe gastric atrophic damage (pernicious anaemia, metaplastic atrophy, parietal cells antibodies) and high-grade gastric mucosa inflammation. An accurate initial clinical and histological assessment of ABG patients allows to identify those who may benefit from eradication treatment.

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The Green Tea Catechin Epigallocatechin-3-Gallate Inhibits *H. pylori*-Induced and TNF α -Induced IL-8 Transcription and IL-8 Secretion in Gastric Epithelial Cells

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Introduction: *H. pylori* *cag* pathogenicity island (PAI) positive strains activate *interleukin-8* (IL-8) transcription and IL-8 secretion in gastric epithelial cells of relevance to the enhanced gastric mucosal neutrophilic responses and gastric carcinogenesis. Epidemiological studies suggest epigallocatechin-3-gallate (EGCG), a natural product from green tea, may have a role in prevention of gastric atrophy and gastric cancer in Asian populations. The aim of this study was to investigate if *H. pylori*-induced IL-8 transcription and IL-8 secretion in gastric epithelial cells was inhibited by EGCG. **Methods:** *H. pylori* (NCTC11637, *cag* PAI positive and G50 *cag* PAI negative strains) were co-cultured for 4 hrs with L5F11 gastric epithelial cells, a cell line stably transfected with IL-8 gene promoter fused to a luciferase reporter gene. TNF α (10 ng/ml) was used as a positive control. IL-8 transcription was evaluated by assessing luciferase activity. EGCG (50-200 μ M) was pre-incubated for 1 hr with L5F11 cells before co-culture with TNF α or *H. pylori*. Cultures of L5F11 cells were undertaken for 18 hrs to examine culture supernatants for IL-8 protein by ELISA following stimulation. **Results:** TNF α (10 ng/ml) and *H. pylori* (strain NCTC11637 but not strain G50) significantly stimulated IL-8 transcription in L5F11 cells at 4 hrs post-stimulation (p < 0.001, n = 10). EGCG (100 μ M) significantly inhibited (p < 0.01, n = 10) *H. pylori*-induced IL-8 transcription and EGCG (200 μ M) significantly inhibited TNF α (p < 0.01, n = 10) and *H. pylori* (p < 0.001, n = 10) induced IL-8 transcription 4 hrs post-stimulation. IL-8 secretion by L5F11 cells was significantly (p < 0.001, n = 3) increased by *H. pylori* strain NCTC11637 (but not G50) at 18 hrs after co-culture. Similarly, TNF α significantly (p < 0.01, n = 3) stimulated IL-8 secretion. EGCG at 100 μ M significantly inhibited (p < 0.01, n = 3) *H. pylori* (NCTC11637 strain)-induced IL-8 secretion 18 hrs post-infection. TNF α -induced IL-8 secretion was significantly inhibited (p < 0.01, n = 3) by 200 μ M EGCG. **Conclusions:** *H. pylori*-induced IL-8 transcription and IL-8 protein secretion in L5F11 cells is significantly inhibited by EGCG in a dose-dependent fashion. Green tea consumption as an inhibitor for *H. pylori*-induced neutrophilic responses to *cag* PAI positive *H. pylori* strains and TNF α inflammatory gastric responses might have a role in prevention of gastric carcinogenesis.

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A New 5 Minute Rapid Urease Test Is Superior to the CLO Test in the Diagnosis of H Pylori Infection

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BACKGROUND: In clinical practice the only limitation of the Rapid Urease Test (RUT) is the time reading (from 1-24 hours). The delay is associated with costs (generation of report, holding the patient from discharge for the result of RUT, etc.). **AIM:** To assess the accuracy of a new ultrafast test, approved in the European Union, to be read in 5 minutes compared to other commercially available RUTs. **METHODS:** A total of 375 consecutive untreated patients with upper GI symptoms were enrolled (m/f:146/229, age-range: 18-85 yrs). All underwent 13C-urea breath testing and endoscopy. At endoscopy, biopsy samples were obtained for: histology (n=2), culture (n=1) and one biopsy each for 3 rapid urease tests (UFT-300, ABS Srl, Cernusco Milan, Italy, Pyloritek, Serim Labs, Elkhart IN and CLOtest, Kimberley-Clark, Roswell GA). Tests were read at the time points approved by regulatory authorities (CLO test: 24 hours; Pyloritek: 1 hour; UFT-300: 5 minutes). **RESULTS:** Patients were considered infected with *H. pylori* if both histology and UBT were positive or if culture alone was positive (in accordance with European Guidelines). 160 out of 375 (42.7%) patients were infected with *H. pylori*. The sensitivity, specificity, predictive values and likelihood ratios of RUTs are shown in Table 1. Within the first minute, 85% of infected samples had turned positive with the UFT-300, 40% with the Pyloritek and 5% with the CLO test. **CONCLUSION:** 1. While all rapid urease tests are highly specific, sensitivity is variable and is superior with the Pyloritek and Ultrafast tests. 2. An endoscopic diagnosis of *H. pylori* infection is now possible in 5 minutes or less and may streamline flow through endoscopy units.

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