ΠΡΟΣΚΕΚΛΗΜΕΝΕΣ ΞΕΝΟΓΛΩΣΣΕΣ ΑΝΑΚΟΙΝΩΣΕΙΣ ΕΛΛΗΝΩΝ ΕΡΕΥΝΗΤΩΝ

● DISTINCT PRESENCE OF CagA-POSITIVE STRAINS WITH HIGHER NUMBER OF EPIYA-C REPEATS IN THE FUNDUS VERSUS THE ANTRUM OF *H. PYLORI* INFECTED PATIENTS

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The EPIYA tyrosine phosphorylation sites are important determinants of CagA virulence presenting extensive variability in both the type and number, especially the EPIYA-C sites, among wild-type *H. pylori* of Western origin.

We have previously identified the occurrence of mixed infections by isogenic *H. pylori* strains with variable number of EPIYA-C repeats within the same patient and we aimed to assess whether they preferentially colonize distinct compartments of the gastric mucosa, namely the gastric antrum and fundus.

CagA and EPIYA status were determined by polymerase chain reaction (PCR) and sequencing in *H. pylori* isolates from 140 paired antral and fundic biopsies from 70 Greek adult patients. Clonal relations between strains were assessed by RAPD-PCR and MLST analysis of the housekeeping genes atpA, efp, mutY, ppa, trpC, ureI, vacA and yphC.

In all cases with the exception of one, paired isolates of antral and fundic origin were clonally related. In 59 patients, the same strain was isolated from both antrum and fundus (20 cagA-negative, 23 ABC, 11 ABCC and 5 ABC/ABCC isolates), whereas in 10 patients the fundus was colonized by cagA-positive *H. pylori* harboring more EPIYA-C repeats compared to the corresponding strains from the antrum. In conclusion, with regards to the CagA EPIYA status, the vast majority of individuals were found to be infected by the same *H. pylori* strain. Nevertheless, in approximately 15% of the patients, isogenic strains carrying more EPIYA-C repeats were identified, preferentially colonizing the gastric fundus, possibly reflecting a response to microenvironmental differences in acidity between the two gastric sites.

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● CagA AND VacA POLYMORPHISMS ARE ASSOCIATED WITH DISTINCT PATHOLOGICAL FEATURES IN *H. PYLORI*-INFECTED ADULTS WITH ULCER AND NONULCER DISEASE

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CagA and VacA polymorphisms define *Helicobacter pylori* virulence and may predict the development of severe gastric disease. We determined the variability of functional EPIYA tyrosine phosphorylation motifs in CagA and the isotypes for signal, intermediate and middle regions of VacA in Greek adults with duodenal (n=44) or gastric (n=21) ulcers and nonulcer dyspepsia cases (n=79) and assessed potential associations to the severity of histopathology.

EPIYA motifs were determined by polymerase chain reaction (PCR) and sequencing and VacA alleles by PCR. *cagPAI* functionality was assessed by interleukin (IL-8) secretion, whereas CagA translocation was confirmed by western blot detection of CagA, after antiphosphotyrosine immunoprecipitation of total protein lysates from *H. pylori*-infected AGS cells. Statistical analysis was pursued with multivariate logistic regression. Infection with *CagA-positive* strains carrying one EPIYA-C site was found to be an independent risk factor for gastroduodenal ulceration [odds ratio (OR):4.647, 95% confidence interval (CI):2.037-10.602], while the risk was 2-fold higher in mixed infections with isogenic strains harboring increasing EPIYA motifs. CagA species with more EPIYA-C repeats exhibited higher tyrosine phosphorylation rates but did not contribute to elevated IL-8 secretion, or to increased neutrophilic or mononuclear infiltration in the antrum. Increasing EPIYA-C repeats in CagA were associated with highly vacuolating vacA isotypes (s1/i1/m1 or m2). V*acAs1* allele was related to increased activity of chronic antral gastritis (OR: 3.319, 95% CI: 1.449-7.600) and the *vacA*i1 allele to greater chronic inflammatory infiltration (OR: 6.514, 95% CI: 2.298-18.878). In conclusion, CagA and VacA contribute to *H. pylori* infection in a coordinated manner, differentially affecting clinical phenotypes and the inflammatory response.

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● ARE FIRST DEGREE RELATIVES OF GASTRIC CANCER PATIENTS AT AN INCREASED RISK FOR GASTRIC CANCER? A META-ANALYSIS

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Background: *Helicobacter pylori* is believed to predispose to gastric cancer by inducing precancerous changes, i.e., atrophy and intestinal metaplasia (IM). First-degree relatives of patients with gastric cancer might be at an increased risk of developing gastric cancer. However, this evidence is based on scattered individual studies.

Aims: The aim of this study was to examine the risk of first-degree relatives developing gastric cancer by meta-analyzing all relevant studies.

Methods: Extensive English language medical literature searches for human studies were performed up to the end of April 2009. Inclusion and exclusion criteria were identified and in eligible studies data on three parameters, i.e., *H. pylori* prevalence, atrophy, and IM, were extracted. Pooled estimates [odds ratio (OR) with 95% confidence intervals (CI)] were obtained using either the fixed or random-effects model as appropriate.

Results: Out of 149 initially identified studies, seven studies, from various countries, fulfilling the inclusion criteria, examined the risk of first-degree relatives developing gastric cancer (n = 1095) in comparison to controls (n = 1248). For *H. pylori* prevalence, the pooled OR with 95% CI was 1.90 (1.30-2.79) and the test for overall effect Z was 3.34 (p=.001). The respective values for atrophy and IM were 4.14 (2.55-6.71), Z= 5.67, (p=.00) and 2.67 (1.87-3.81), Z= 5.41 (p=.0001) respectively.

Conclusion: First-degree relatives of patients with gastric cancer are at an increased risk of developing gastric cancer. Consequently upper gastrointestinal endoscopy with *H. pylori* detection and prophylactic eradication of the infection should be offered to such individuals.

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● INTESTINAL METAPLASIA OF THE GASTRIC CARDIA IS MORE FREQUENT IN GERD PATIENTS WITH FAMILY HISTORY OF GERD

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Aim: To define the effect of family history of gastroesophageal reflux disease GERD(FH) in the severity of carditis and intestinal metaplasia of the gastric cardia in GERD patients.

Patients-Methods: One hundred and twenty patients with FH(-) GERD (mean age 60±15 years, 73 male) and 120 FH(+) (mean age 59±15, 72 male) after gastroscopy with biopsies started on omeprazole 20 mg twice a day for 1 year plus 10-day *H. pylori* eradication regimen if *HP*(+). Finishing treatment, we repeated endoscopy with biopsies, on omeprazole and ^{13}C-urea breath test, off omeprazole if *HP*(+). The Sydney classification system was used for carditis/intestinal metaplasia. Stat: X2.

Results: Cardiac mucosa was identified in 111 (93%) FH(+), 109 (91%) FH(-) patients (p=.64); carditis in 81 (68%) FH(+), 71 (59%) FH(-) patients (p=.18), not correlated with *H. pylori*. Intestinal metaplasia of gastric cardia was found in 50 (42%) FH(+), 34 (28%) FH(-) patients (p=.0001) [*HP*(+): 23(19%) FH(+), 11 (9%) FH(-) patients (p=.007); *HP*(-): 27 (23%) FH(+), 23 (19%) FH(-) patients (p=.01)]. Thirty-two FH(+) and 29 FH(-) patients eradicated *H. pylori*. Of them 13 FH(+) and 18 FH(-) patients present no carditis in follow-up endoscopy. Seventy (58%) FH(+) and 53 (44%) FH(-) patients presented carditis (p<.001). During follow up the severity of intestinal metaplasia increased by 0.4±0.1 grades in FH(+) patients while remained unchanged in FH(-) patients (p<.001). Five (4%) FH(+) and no FH(-) patients developed low-grade dysplasia (p=.02). Carditis regression was more frequent after *H. pylori* eradication.

Conclusions: 1) Intestinal metaplasia of the cardia, but not carditis is more frequent in GERD FH(+) patients. 2) Carditis regresses less frequently after *H. pylori* eradication in FH(+) GERD patients. 3) Intestinal metaplasia of the cardia worsens and dysplasia develops more rapidly in FH(+) GERD patients despite high-dose omeprazole treatment.

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● CARDITIS CAN PARTIALLY REGRESS AFTER *H. PYLORI* ERADICATION BUT NOT PROTON PUMP INHIBITOR TREATMENT

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Aim: To define the effect of *Helicobacter pylori* eradication and antireflux treatment in carditis and intestinal metaplasia of the gastric cardia.

Patients-Methods: Two hundred and forty patients with gastroesophageal reflux disease (GERD) (mean age 59±15years, 145 male) and 240 controls without GERD (mean age 58±17years, 138 male) after gastroscopy with biopsies were started on omeprazole 20 mg twice daily for 1 year plus 10-day *H. pylori* eradication regimen if *H. pylori* positive. Finishing treatment, we repeated endoscopy with biopsies, on omeprazole and performed ^{13}C-urea breath test, off omeprazole if *H. pylori* positive. The Sydney classification was used for carditis/intestinal metaplasia.

Results: Cardiac mucosa was identified in 180 (75%) controls, 220 (92%) refluxers ($p<.001$); carditis in 102 (43%) controls, 152 (63%) refluxers ($p<.001$) [*H. pylori* positive: 50 (21%) controls, 76 (32%) refluxers ($p=.007$); *H. pylori* negative: 52 (22%) controls, 76 (32%) refluxers ($p=.01$)]. Intestinal metaplasia of the cardia was found in 45 (19%) controls, 84 (35%) refluxers ($p<.001$). Forty one controls with carditis and 61 refluxers successfully eradicated *H. pylori*. Of them 29 controls, 31 refluxers present no carditis in the follow up endoscopy. After 1 year, 73 (30%) controls, 123 (51%) refluxers presented carditis ($p<.001$). During follow up there was no change in the severity of intestinal metaplasia, carditis regressed in 71% ($n=29$) *H. pylori*-positive controls who eradicated *H. pylori*, 51% ($n=31$) refluxers; it increase by 0.5±0.1 grades in controls, 0.3±0.1 in refluxers who persisted *H. pylori*, while remained rather unchanged in *H. pylori*-negative patients and controls. Five refluxers developed dysplasia.

Conclusions: 1) Both carditis and intestinal metaplasia of the gastric cardia are more frequent in GERD patients. 2) Carditis but not intestinal metaplasia can regress less frequently in *H. pylori*-positive patients than controls after *H. pylori* eradication. 3) High dose omeprazole treatment has little effect on carditis.

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● INFECTION WITH *HELICOBACTER PYLORI* IS ASSOCIATED WITH AN ATHEROGENIC PROFILE: DYSLIPIDAEMIA AND MODIFICATION OF INFLAMMATORY INDICES

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Introduction: *Helicobacter pylori* (*HP*), a Gram-negative, spiral, microaerophilic bacterium, has been associated with a variety of intestinal and extraintestinal disorders, including atherosclerosis. The results, however, originating from related studies are controversial.

Aims & Methods: The objectives of the study were to determine the frequency of *HP* infection among 120 ischaemic heart disease (IHD) patients and 114 otherwise healthy individuals and examine the effect of the infection on lipids, coagulation, inflammatory indices and determinants of homocysteine levels. IgG and IgA antibody titers against *HP* were calculated by means of an enzyme-linked immunosorbent assay (Enzygnost, Dade Behring Marburg GmbH, Germany). Subjects suffering from diseases – or under medication - that could alter the levels of the tested parameters, were excluded from the study. The study participants were classified into *HP*-seropositive and seronegative, sex, age, body mass index, diabetes and smoking-matched subgroups. Lipids - total cholesterol, low and high density lipoproteins (LDL, HDL) and triglycerides - coagulation parameters - platelet count, mean platelet volume, prothrombin time, partial thromboplastin time, fibrinogen and D-dimers - inflammatory indices - white blood cell count, C reactive protein, serum amyloid A, ceruloplasmin, complement factors C3 and C4 - and components of myocardial remodelling - brain natriuretic peptide - were determined in all study participants (expressed as mean±SEM).

Results: Higher levels of LDL were recorded in *HP* positive (115.6±4.157 mg/dL) compared to *HP* negative individuals (102.2±2.991 mg/dL) ($P < 0.01$). Moreover, *HP* positive subjects tended to exhibit lower HDL levels (46.44±1.349 mg/dL) as compared to those found in the seronegative qroup (42.72±1.414 mg/dL), although this difference reached only a marginal statistical significance ($P=0.06$). An upregulation of ceruloplasmin became evident in the seropositive (26.82±0.5181 mg/dL) compared to the seronegative (25.36±0.4866 mg/dL) participants ($P=0.041$). In addition, *HP* positive subjects tended to exhibit lower C3 levels (112.4±2.341 mg/dL) compared to the *HP* negative group (117.7±1.793 mg/dL) ($P=0.076$).

Conclusion: While the changes in the levels of lipids are suggestive of an *HP*-induced dyslipidaemic background, complement activation and the upregulation of ceruloplasmin are indicative of an associated inflammatory, oxidative disequilibrium. Our results show that *HP* infection may predispose to atherosclerosis by inducing a multivariate atherogenic condition.

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● PATIENTS WITH GASTRO ESOPHAGEAL REFLUX DISEASE AND RESPIRATORY MANIFESTATIONS DON'T PRESENT LUNG FUNCTION DISORDERS DURING EXERCISE TEST

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Introduction: Gastroesophageal reflux disease (GERD) has been associated with a variety of pulmonary manifestations but it is unclear if gastroesophageal reflux causes any abnormality in pulmonary function. Ergometry is a specialized method which is used to evaluate the respiratory function during exercise.

Aims & Methods: The aim of this study was to reveal any abnormality of pulmonary function in patients with GERD and respiratory symptoms. We evaluated 34 patients with GERD (age 21-63, 24 men) and extraesophageal respiratory symptoms (wheezing and/or cough) before therapy and after twelve weeks treatment with double dose omeprazole. No patient presented abnormal spirometry. Ergometry was performed in all patients at baseline and after completion of 12 week treatment. The following ergometric parameters: VO2rest, VO2max, VCO2rest, VCO2max, O2 - puls rest, O2 - puls max, HR (heart rate) rest, HRmax, PETCO2rest, PETCO2max, VE/VCO2 SLOPE were recorded pre-treatment and post-treatment.

Results: Twenty four patients (70.6%) had esophagitis (grade I-IV), 16 patients had hiatal hernia (47.1%) and in 13 patients (38.2%) *Helicobacter pylori* was positive. Ergometric parameters were within normal limits in all patients, no patient presented abnormality during exercise. Twenty eight patients were reevaluated. No improvement in any ergometric parameter posttreatment was observed despite remission of esophageal and extraesophageal symptoms in all patients. No statistically significant difference was observed pre and post-treatment, between older and younger than 40 year old patients, smokers and non smokers, *Hp*(+) and *Hp*(-) patients and those with and without hiatal hernia and esophagitis.

Conclusion: Patients with GERD and respiratory manifestations and normal spirometry present no disordes during ergometry (exercise test). Also no alteration in ergometric values post-treatment neither difference in ergometric values according to age, smoking, *Hp* status, presence of esophagitis or hiatal hernia were observed.

Table 1.

	Pre treatment	Post treatment	p
VO2max (l/min)	1.803	1.950	0.43
VCO2 max (l/min)	1.733	1.89	0.29
O2-puls max (ml/beat)	12.22	14.70	0.15
HR max (bpm)	150.40	162.21	0.092
PETCO2max (mmHg)	38.08	38.21	0.27
VE/VCO2 SLOPE	23.81	22.67	0.87

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● *HELICOBACTER PYLORI* INFECTION AND BONE MINERAL DENSITY IN POSTMENOPAUSAL WOMEN

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Introduction: Cytokines regulate bone turnover and influence the pathogenesis of skeleton disorders, such as osteoporosis which affects approximately 30% of postmenopausal women. *Helicobacter pylori* (*HP*) infection increases the systemic levels of inflammatory cytokines such as, IL1, IL6 which may affects bone metabolism, leading to an elevated risk for developing osteoporosis.

Aims & Methods: The aim of this study was to investigate the possible relationship between *HP* infection and bone mineral density (BMD) in postmenopausal women. We examined 126 postmenopausal women aged 45-65 for serum antibodies against *HP* using ELISA test and determined BMD, T-score and Z-score of both lumbar spine and hip by dual energy X-ray absorptiometry (DEXA). Several biochemical parameters of bone metabolism such as calcium (Ca), phosphorus (P), parathyroid hormone (PTH) and 25(OH)D3 were also measured in serum. Postmenopausal women were divided into two groups in relationship to serum positive antibodies against *HP*. Group A consisted of 58 *HP*-ve and Group B consisted of 68 *HP*+ve postmenopausal women.

Results: There was no significant difference between groups in age, gender, body mass index (BMI), smoking, underlying disease, and/or concomitant medications. Moreover, no significant difference in lumbar spine and hip BMD, T-score, Z-score, Ca, P, PTH and 25(OH)D3 levels was observed between the two groups (Table 1).

Table 1.

Parameters[a]	Group A	Group B	P
BMD (LS)	1.087±0.122	1.079±0.145	>0.5
T-score/Z-score (LS)	-1.53±1.37 / -1.43±1.24	-1.55±1.28 / -1.48±1.31	>0.5
BMD (hip)	1.017±0.127	1.023±0.121	>0.5
T-score/Z-score (hip)	-1.56±1.15 / -1.53±1.05	-1.53±1.19 / -1.59±1.11	>0.5
Ca (mg/dl)	9.38±1.15	9.23±1.35	>0.5
P (mg/dl)	3.08±1.22	3.16±1.15	>0.5
PTH (pg/ml)	53.6±22.1	51.7±22.6	>0.5
25(OH)D3 (nmol/l)	52.6±22.6	58.5±30.7	>0.5

[a]Values are mean±SD.

CONCLUSION: *HP* infection did not have any impact to BMD and other parameters of bone metabolism in Greek postmenopausal women.

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● DELAYED POSITIVITY OF RAPID UREASE TEST: IS THERE ANY CLINICAL SIGNIFICANCE?

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Background-Aim: Rapid urease tests (RUT) are widely used because of their simplicity and reliability. It is not uncommon though, the tests to render late (≥72 hours) positivity, although initially negative (≤24 hours). It is not clear if it is due to a false positive result or either slow production of urease from other urease-positive bacteria or it is related to less densely populated gastric mucosa with *Helicobacter pylori*.

Patients and Methods: One hundred and thirty-six patients (44 male, 92 female), endoscoped for dyspepsia, heartburn or anaemia, were studied. Exclusion criteria included current or recent (≤4 weeks) use of proton pump inhibitors, H_2 antagonists and/or antibiotics and the presence of active upper gastrointestinal bleeding. A double gel-based RUT (Hut-Test AstraZeneca GmbH) and histology from both the antrum and corpus (two specimens) were performed. RUT tests were assessed at 24 hours (early) and 72 hours (delayed) and were compared with histology.

Results: Eighty-four of 136 (62%) of patients had either positive (30 of 84) or negative (54 of 84) results to both (early and delayed) RUT readings. In 80 of 84 (95%) histology was in concordance with RUT, while in the rest four of 84 (5%) there was discrepancy between RUT and histology. Fifty-two of 136 (38%) presented delayed positivity of RUT test, with the early reading being negative. In 18 of 52 (34%) [18/136 (13%) of total] histology results were in concordance with the delayed positive reading, confirming the presence of *H. pylori*, while in the rest 34 of 52 (66%) histology was negative for *H. pylori*.

Conclusions: It seems that RUT turns to render a true late (≥72 hours) positivity in about 15% of patients tested for *H. pylori*.

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● MULTICENTER STUDY ON THE TREATMENT OF *H. PYLORI* INFECTION WITH HIGH DOSE ESOMEPRAZOLE, AMOXICILLIN AND METRONIDAZOLE IN CHILDREN INFECTED WITH DOUBLE-RESISTANT STRAINS

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Background: The increasing number of pediatric patients with multiresistant *Helicobacter pylori* strains creates urgent need for evaluation of treatment regimens. Second-line antibiotics like tetracycline, chinolons or bismuth are not released for children. However, in vitro resistance to metronidazole may be overcome by a high dose and prolonged intake.

Objective: Prospective multicenter study on eradication rate and side-effects of a high dose triple therapy in pediatric patients with culture-proven double resistance.

Methods: In this investigator-initiated open treatment trial, including several European countries, 58 *H. pylori*-infected patients (15 kg) with proven resistance to metronidazole and clarithromycin were prospectively included. Therapy including amoxicillin (~75 mg/kg), metronidazole (~25 mg/kg) and esomeprazole (~1.5 mg/kg) was given twice daily for 2 weeks. Success of therapy was monitored by a ^{13}C-urea breath test at 6 and 24 weeks after treatment. Primary outcome parameter was the eradication rate at 6 weeks.

Results: Of 58 children included, follow-up data were available until May 2009 from 50 patients after 6 week and from 34 after 24 weeks follow up. Eradication rates after 6 weeks were 81% (per protocol) and 66% (intention to treat analysis), respectively: No serious side-effect occurred.

Conclusions: A high dose 2-week therapy with amoxicillin, metronidazole, and esomeprazole is a well-tolerated treatment option in children infected with a double-resistant *H. pylori* strain. Eradication rate is good when drugs were taken according to the protocol. However, compliance in this patient group was difficult to achieve.

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● A NEW SECOND-LINE SEQUENTIAL REGIMEN FOR *HELICOBACTER PYLORI* ERADICATION BASED ON LEVOFLOXACIN

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Introduction: A sequential regimen with three antibiotics has been proved to be very effective as a first-line treatment for *H. pylori*.

Aims & Methods: The aim of the study was to evaluate the efficacy of a second-line sequential regimen with levofloxacin (LEVO) and to compare it with the 10-day concomitant regimen with LEVO. 56 patients, who failed to eradicate *H. pylori* with the standard triple therapy were randomized to receive either a 10-day treatment with omeprazole (OME) 20 mg b.d., amoxicillin (AMO) 1 gr b.d. and LEVO 500 mg b.d. (group A) or a sequential 10-day treatment as follows: a) from day 1 to 5: OME 20 mg b.d. and AMO 1 gr b.d. b) from day 6 to 10: OME b.d. and LEVO b.d. (group B). *H. pylori* eradication was confirmed with a negative breath test performed at least 4 weeks after treatment end.

Results: Overall, *H. pylori* was eradicated in 75% (42/56) patients in intention-to-treat (ITT) analysis, whereas the per-protocol (PP) cure rate was 82% (42/51). The 2 groups were comparable concerning age, sex, consumption of tobacco, alcohol and NSAIDs, and percentage of peptic ulcer disease cases. Success rates were 72.4% (21/29) (95% CI: 52.8-87.2%) and 77.8% (21/27) (95% CI: 57.7-91.4%) in groups A and B respectively, in ITT analysis and 80.8% (21/26) (95% CI: 60.7-93.5%) and 84% (21/25) (95% CI: 63.9-95.5%) respectively, in PP analysis ($P > 0.05$, NS). No significant differences were observed between the 2 groups concerning symptoms resolution and adverse effects.

Conclusion: A new sequential regimen containing omeprazole, amoxicillin and levofloxacin is effective and safe in *H. pylori* eradication as a second-line treatment. Moreover, it is more simple and less expensive compared to the usually prescribed concomitant regimen.

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