GIH-05 Randomised controlled study of rabeprazole, levofloxacin and rifabutin triple therapy versus quadruple therapy as second-line treatment of *Helicobacter pylori* infection

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Background and aims: To test the efficacy of rabeprazole, levofloxacin and rifabutin triple therapy versus rabeprazole-based quadruple therapy for the treatment of *Helicobacter pylori* (*H. pylori*) infection after failure of one or more courses of *H. pylori* eradication therapy.
Methods: One hundred and nine patients who failed previous *H. pylori* eradication were randomised to receive 1) rabeprazole 20mg twice daily, rifabutin 300mg and levofloxacin 500mg once daily for 7 days (RRL) or 2) rabeprazole 20mg twice daily, metronidazole 400mg thrice daily, bismuth subcitrate 120mg and tetracycline 500mg four times daily for 7 days (quadruple). Endoscopy with biopsy for CLO test, histology and culture was performed before treatment. Post-treatment *H. pylori* status was determined by ^{13}C-urea breath test. Metronidazole, clarithromycin and amoxycillin resistance was defined as MIC of >8 ug/ml, >2 ug/ml and >2 ug/ml respectively.
Results: The clarithromycin resistance rate (79% versus 21%, P<0.001) and metronidazole resistance rate (89% versus 40%, P<0.001) was significantly higher in patients who have taken these antibiotics in their previous *H. pylori* treatment compared to those who have not. Intention-to-treat and per protocol *H. pylori* eradication rate were 91% / 91% for the RRL group and 91% / 92% for the quadruple group. For patients with double resistance to metronidazole and clarithromycin, the eradication rates were 85% (17/20) in the RRL group and 87% (13/15) in the quadruple group. Compliance was greater than 95% for both regimens.
Conclusion: Rabeprazole, levofloxacin and rifabutin based triple therapy and rabeprazole-based quadruple therapy were equally effective as a second-line treatment of *H. pylori* infection.

GIH-06 The role of cigarette smoking and its interaction with cyclooxygenase-2 in acute ulcerative colitis in mice

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Introduction: Epidemiological studies show that smokers are more likely to have Crohn's disease but are less susceptible to ulcerative colitis. Our previous study indicated that passive cigarette smoking exacerbated the Crohn's disease induced by 2,4,6-trinitrobenzene sulfonic acid in rats. However, no direct evidence had been found regarding the protective effect of cigarette smoking on ulcerative colitis. Besides, cyclooxygenase-2 (COX-2) is strongly associated with inflammatory bowel disease for its inhibition could either potentiate or attenuate colitis. The aim of the present study is to evaluate the role of cigarette smoking on ulcerative colitis induced by dextran sulfate sodium (DSS) in mice and to determine whether COX-2 takes part in the inflammatory process in this model.
Methods: Acute ulcerative colitis was induced in balb/c mice by giving 5% DSS in drinking water for 7 days. The mice were simultaneously exposed to passive 0% or 2% cigarette smoke (CS) once daily for the same period of time. Colonic tissues were assessed for the inflammatory parameters and COX-2 expression in these animals.
Results: DSS in drinking water given for 7 days increased colon weight when compared with the control. Histology findings revealed that there were infiltration of inflammatory cells and loss of glands in the colonic mucosa. An increase in myeloperoxidase (MPO) activity, a marker for neutrophils infiltration, was also observed. Both immmunohistochemistry and Western immunoblotting indicated that COX-2 was induced in the colonic tissues with ulcerative colitis. It was accompanied with an induction of the cellular proliferation. On the other hand, passive CS exposure neither affected the degree of inflammation nor the MPO activity in these animals. However, both COX-2 protein expression and cellular proliferation was increased in colonic tissue in mice with CS exposure alone and synergistically induced in mice when combined with DSS.
Conclusion: Passive cigarette smoking did not influence the severity of acute colonic inflammation induced by DSS though it further provoked COX-2 expression and cellular proliferation in colitis animals. Furthermore, COX-2 was induced by CS exposure alone but did not accompany with colitis formation. All these findings indicated that COX-2 was not directly involved in the initiation of inflammation in this acute ulcerative colitis model. However, whether the induction of cigarette smoke on proliferation exerts beneficial effect on ulcerative colitis is undefined and requires further study.