The impact of Helicobacter pylori eradication on peptic ulcer healing

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Authors' objectives
To investigate whether successful Helicobacter pylori (H. pylori) therapy affects peptic ulcer healing.

Searching
MEDLINE was searched from January 1988 to February 1997 for studies published in any language, using the search terms 'Helicobacter pylori', 'eradication' and 'ulcer healing'. The following journals were searched manually: American Journal of Gastroenterology, Gut, Gastroenterology, Scandinavian Journal of Gastroenterology, European Journal of Gastroenterology and Hepatology, Digestive Diseases and Sciences, Zeitschrift fur Gastroenterologie, and Alimentary Pharmacology and Therapeutics. All abstracts from major meetings during 1995 to 1996 that were published in Gastroenterology, and those from a workshop on gastroduodenal pathology and H. pylori (held at Edinburgh, 1995) were reviewed.

Study selection
Study designs of evaluations included in the review
The authors do not state that any specific study designs were included or excluded.

Specific interventions included in the review
H. pylori eradication.

Participants included in the review
Non-pediatric patients with duodenal (DU), gastric (GU) or peptic (PU) ulcers were included.

Outcomes assessed in the review
PU healing rates were assessed.

How were decisions on the relevance of primary studies made?
The authors do not state how the papers were selected for the review, or how many of the authors performed the selection.

Assessment of study quality
The authors do not state that they assessed validity.

Data extraction
The authors do not state how the data were extracted for the review, or how many of the authors performed the data extraction.

Methods of synthesis
How were the studies combined?
The studies were combined using Fisher's exact test, or the chi-squared test, depending on the number of patients. The odds ratios (ORs) were calculated with 95% confidence intervals (CIs). Statistical probabilities for correlations between PU and H. pylori eradication rates were calculated using the Spearman rank correlation coefficient.

How were differences between studies investigated?
The authors do not state how differences between the studies were investigated.
Results of the review

Sixty studies (with 66 treatment arms) comprising a total of 4,329 patients were included in the review.

H. pylori eradication improved ulcer healing by 15 to 20%, regardless of diagnoses (GU, DU or PU). The OR was 2.7 (95% CI: 1.3, 5.4, p<0.01) for GU, 5.1 (95% CI: 4.6, 6.5, p<0.0001) for DU, and 6.6 (95% CI: 4.1, 10.6, p<0.0001) for PU.

Gastric acid suppression by normal or high doses of antisecretory drugs combined with H. pylori treatment provides better results (11% difference) only if therapy fails. Normal and high doses of antisecretory drugs gave ORs of 4.4 (95% CI: 3.3, 5.8, p<0.0001) and 6.5 (95% CI: 3.8, 10.9, p<0.0001), respectively, for all ulcer types.

The results from prolonged acid suppression treatment (extending the initial eradication phase) revealed that only those patients who had prolonged acid inhibition in initially normal or high doses, and who had had eradication failure at the end of evaluation, had an increase in PU healing (approximately 15% difference). The subgroup of patients remaining infected, who had had eradication therapy with missing acid suppression, had no benefit from subsequent prolonged acid inhibition. If the infection was cured by using high-dose acid suppression (mainly dual therapy), ulcer healing was 13% lower in H. pylori-negative patients, if not supported by additional prolonged acid suppression. The ulcer healing rate, however, was still better (20% benefit) than in those patients in whom the infection was not cured.

PU healing rates were better in patients successfully treated for H. pylori, regardless of the time point considered (range: 13 to 25%).

There was a positive correlation between PU healing and H. pylori eradication (correlation coefficient, r=0.539, p<0.0001). This relationship also existed for studies with no prolonged acid inhibition after initial eradication (r=0.667, p<0.001).

Authors’ conclusions

Successful H. pylori eradication therapy accelerates PU healing even without concomitant acid suppression.

CRD commentary

The review focused on a well-defined question. The inclusion and exclusion criteria were reported.

The search was fairly narrow and could have been extended to include other databases, such as EMBASE, and an attempt to identify unpublished material from experts in the field. The validity of the included studies was not assessed and there was no restriction on the study designs included. No details of the individual studies were reported, for instance, the authors did not report how healing rates were defined in individual studies, or how long they followed patients for. The data from primary studies were combined, but tests for heterogeneity were not undertaken first. The authors stated that most of the included studies had a weakness in that they reported the positive effects on ulcer healing by eradicating H. pylori; however, concomitant acid suppression including antacids or bismuth compounds were not usually mentioned. The results of this review should be interpreted with caution in view of these limitations.

Implications of the review for practice and research

The authors state that effective H. pylori treatment therapy should be the standard for all associated PUs from the time of diagnosis onwards, not only to reduce ulcer recurrence but to achieve rapid PU healing in greater than 90% of the participants. They also suggest that prolonged acid inhibition for ulcer healing, with initial effective bacterial eradication, is justified only in large or complicated (e.g. bleeding) ulcers or if gastroesophageal reflux disease is present. Clinical trials that evaluate the impact of acid suppression, both during and after H. pylori eradication with antibiotics, are needed.

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