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IS ERADICATION OF HELICOBACTER PYLORI PREVENTS RECURRENCE OF ULCER AFTER SIMPLE CLOSURE OF DUODENAL ULCER PERFORATION?

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Background: most patients with chronic peptic ulcer disease have helicobacter pylori (h.Pylori) infection. In the past, immediate acid reduction surgery has been strongly advocated for perforated peptic ulcer because of the high incidence of ulcer relapse after simple closure. Although H. Pylori eradication is now the standard treatment of uncomplicated and bleeding peptic ulcers, it's role in perforation remains controversial.

Study aim: The aim of this randomized study, is to determine whether eradication of H. Pylori could reduce the risk of ulcer recurrence after simple closure of perforated duodenal ulcer or not

Patients and methods: Of 80 patients suffering from acute peptic ulcer perforation, 65 (81%) were shown to be infected by H. Pylori (CLO test). Sixty-five positive patents were randomized to receive a course of triple anti-helicobacter therapy or a 4-week course of omeprazole alone. Follow-up endoscopy was performed 8 weeks, 16 weeks (if the ulcer did not heal at 8 weeks), and 1 year after hospital discharge for surveillance of ulcer healing and determination of H. Pylori status. The endpoints were initial ulcer healing and ulcer relapse rate after 1 year.

Results: Of 58 patients who did undergo follow- up endoscopy, 31 of the 32 patients in the first group (anti- Helicobacter group) and 4 of the 26 patients in the second group (omeprazole alone group) had H. Pylori eradicated, initial ulcer healing rates were similar in the two groups (90.6% versus 88.4%). After 1 year, ulcer relapse was significantly less common in patients treated with anti-Helicobacter therapy than these who received omeprazole alone (6.2% versus 34.6%).

Conclusions: We have found a high prevalence of H. Pylori infection in patients with perforated peptic ulcer. An immediate and appropriate H. Pylori eradication therapy for perforated peptic ulcers reduces the relapse rate after simple closure. Response rate to triple eradication protocol was excellent in hospital setting. Immediate acid- reduction surgery in the presence of generalized peritonitis is unnecessary.

Key words: Helicobacter pylori Duodenal ulcer Omeprazole Endoscopy Anti-Helicobacter pylori

INTRODUCTION

The long-term results of omental patch repair for perforated duodenal ulcer are unsatisfactory; a high incidence of ulcer recurrence has been repeatedly reported [1-5]. Some advocate immediate acid-reduction procedures in addition to repar of the ulcer as a preventive measure against subsequent ulcer relapse [6,7]. Immediate definitive surgery in selected patients is safe, without increasing the rate of perioperative complications or death [8]. However with recent advances in antiulcer medical therapy, fewer surgeons have acquired sufficient expertise in performing

the definitive operation. Because perforated peptic ulcer is often an emergency a simpler life-saving procedure suth as simple oversewing procedures either by an open or laparoscopic approach is an attractive option in many centers [9].

The recent rediscovery of Helicobacter Pylori has revolutionized the therapeutic approach to peptic ulcer disease. Eradication of H. Pylori heals most uncomplicated peptic ulcer and prevents relapse [10-12].

In the case of bleeding peptic ulcers, a short course of antibiotics eradicating H. Pylori is efficacious as maintenance acid-reduction medication in preventing recurrent ulcer haemorrhage [13]. Meeting of the European Helicobacter Pylori Study Group have recommended eradicatin of H. Pylori as the standard treatment for uncomplicated and bleeding peptic ulcers [14-15].

However, the association between H. Pylori and perforated duodenal ulcer is less well defined. The reported infection rates range widely, from 47% by serologic testing [16] to more than 80% in two recent biopsy-based studies [17-18]. Whether there is a causal relation between the bacterium and duodenal ulcer perforation is contraversial. We therefore performed a prospective randomized trial to determine whether eradication of H. Pylori could lead to sustained ulcer remission in patients who underwent only simple repair for doudenal ulcer perforation.

PATIENTS AND METHODS

Of eighty patients with clinical or radiologic sign of perforated peptic ulcer, 65 (81%) were shown to be infected by H. Pylori.

Another 7 patients were excluded from this study for several reasons (as 3 failed endoscopic examination, 2 patients refused to undergo intraoperative endoscopy, 2 patients do not follow up by endoscopy). So our study including 58 patients, and done in Sohag University Hospital from March 1999 to March 2001. Informed consent was obtained for surgical exploration, intraoperative per oral gastroscopy, and possible enrollment into the study if infected with H. Pylori.

Exclusion criteria were age older than 75% or younger than 16y, use of antibiotic or acid-suppressing medications within 4 weeks before admission, previous vagotomy or gastrectomy, pregnancy.

All patients received fluid resuscitation; intravenous cefobid 1 gram was administered during induction of anesthesia. No other antibiotics or acid suppressing medication were prescribed before surgery. When duodenal ulcer perforated was confirmed by laparotomy, intraoperative flexible gastroscopy was performed to obtain biopsy samples of gastric antrum several samples were obtained one for urease test (campylobacter- like organism test "CLO test") 2 for gram stain and culture, and 2 in 10% buffered formalin for histologic examination.

Patients were considered to be H. Pylori positive if any one of the following results was found a positive CLO test plus gram-negative helical bacteria in the smear or positive CLO test result plus helical microorganism in histologic section of gastric biopsy samples

Gastrectomy or other definitive acid reduction procedures were considered only if patients had large perforation (more than 1 cm in diameter) not suitable for simple over-sewing of the perforation or perforation associated with hemorrhage or obstruction, and such patients not included in our study.

Perforation less than 1 cm in diameters were repaired by simple omental patch repair. After closure of the perforation, thorough peritoneal lavage with worm saline was performed before closure of the abdominal incision. After surgery, intravenous cefobid (1 gram) was continued every 12 hours for 3 days and intravenous omeprazole 40 mg/day was given until the patient resumed eating on oral diet. Only H. Pylori- positive patients who had undergone patch repair were eligible for randomization trial. After resuming an oral diet, patients were randomly assigned to one of the two treatment options

First group or eradication group in which patients receiving triple eradication protocol, or 1 week course of oral antibacterial treatment (metranidazole 500 mg two times daily, amoxicillin 500 mg three times daily) plus 4 weeks of omeprazole (20 mg twice daily) was prescribed. Second group or control group in which patients were given a 4-week course of omeprazole alone.

Endoscopy was scheduled 8 weeks after randomization. Biopsy samples were again obtained from the gastric antrum to determine the patient's H. Pylori status (Fig. 1).

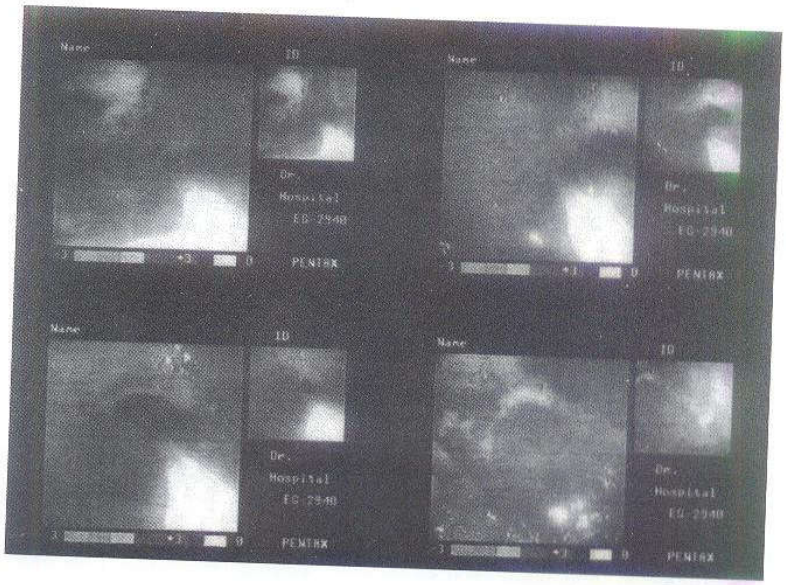

Fig. (1): Biopsy samples obtained from gastric antrum.

All patients with complete ulcer healing confirmed on scheduled endoscopy were then interviewed every 3 months for symptomatology. Maintenance acid-suppression agents were not prescribed during the follow up period. Repeated endoscopic examination was performed whenever patients were symptomatic. All patients were invited for follow up endoscopy at 1 year for ulcer surveillance and determination of H. Pylori status.

RESULTS

From March 1999 to March 2002, 80 patients (66 men and 14 woman), mean age of 49.3 years (SD=16.2) were confirmed to have duodenal ulcer perforation by laparotomy. Sixty-five patients (81%), out of 80 patients were infected with H. Pylori. So this 15 patients were excluded from our study, other 7 patients were excluded for different reasons as 3 cases failed endoscopic examination, 2 patients refused to undergo intraoperative endoscopy, last 2 patients excluded also as long term follow up was not possible for them. Of the remaining 58 patients, 32 were allocated to anti- Helicobacter therapy and 26 to omeprazole alone.

The two groups were evaluated in age, sex, ratio, smoking habit, use of non-steroidal anti-inflammatory drugs, severity of peritoneal contamination and method of repair (Table 1).