Helicobacter pylori infection and complicated peptic ulcer

B. Papaziogas, T. Pavlidis, T. Papaziogas
2nd Surgical Clinic of the Aristotle University of Thessaloniki, Greece

We have read the article of Metzger et al. [1], “Prevalence of Helicobacter pylori infection in peptic ulcer perforations”, with great interest and would like to congratulate the authors on their detailed and accurate work. They report a relatively high incidence of H. pylori infection in patients operated on for perforated peptic ulcer (73%). All of their patients received postoperative eradication therapy which showed excellent results on long-term follow-up [1].

Some years ago we studied the prevalence of H. pylori infection in perforated peptic ulcers [2]. Our study prospectively included 35 patients with perforated peptic ulcers who underwent surgery in our department in the period 1997–1998. They comprised 30 men and 5 women with a mean age of 55.6 years. We found that the overall incidence of H. pylori infection was 54% (19/35 patients), as confirmed by mucosal biopsy and serological testing, a figure lower than that reported by Metzger et al. [1].

24/35 patients had a known history of chronic peptic ulcer; none of these patients with a known ulcer had received eradication therapy in the past, and 62.5% (15/24) were positive at the time of perforation. On the other hand, only a small proportion of the patients with perforation of an acute ulcer were found to be positive for H. pylori infection (3/11, 27%). The incidence of H. pylori infection among patients receiving NSAID was 33%, significantly lower than that reported by Metzger et al (8/11, 72%) [1]. This could be explained by the fact that in our study none of the patients receiving NSAIDs had a history of chronic ulcer.

In 27/35 patients simple closure of the ulcer was performed. The remaining 8 patients required, apart from closure of the ulcer, gastroenterostomy due to pyloric stenosis. All of these patients (8/8) were positive for H. pylori. All patients received eradication therapy postoperatively. On follow-up 12-38 (mean 25) months after surgery the incidence of ulcer relapse was extremely low (1/35, 2.8%).

We would be reluctant to state that “there is an evident association between H. pylori infection and acute perforated ulcers” [1]. The infection rate ranges from 0 to 90% and depends heavily on the characteristics of the population studied. Moreover, there are studies which show no significant difference between perforated and non-surgical ulcers [3], thus suggesting that the pathogenesis of perforation may be independent of H. pylori infection, particularly in the absence of chronic ulceration.

At all events, we agree that eradication therapy is justified in every H. pylori-positive patient with a perforated peptic ulcer, since most studies show that after such therapy the incidence of ulcer relapse is very low. This is confirmed by the authors’ excellent results.

Correspondence:
Dr. B. Papaziogas
Bl. Gabriilidis str. 29
546 35 Thessaloniki/ Greece
e-mail: papaziog@med.auth.gr

References
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