# *Helicobacter pylori* infection and complicated peptic ulcer

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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.

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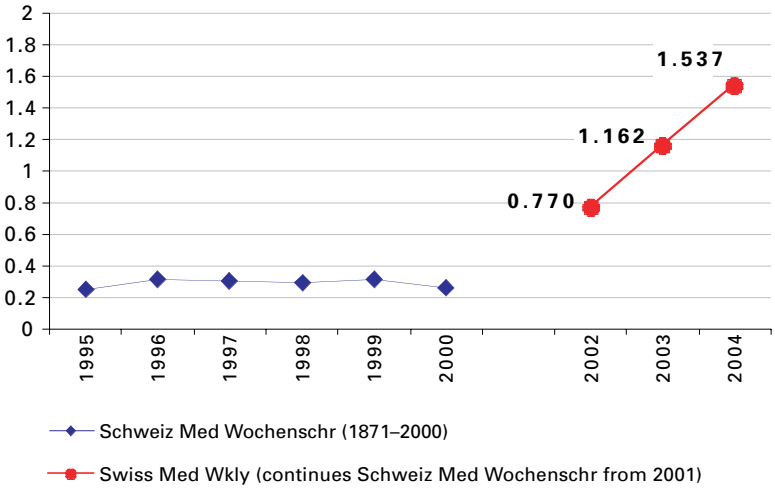


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