Helicobacter pylori Eradication and Its Effect on Iron Stores: A Reappraisal

To the Editor—Limitations in the design and analysis of the study by Gessner et al. [1] may have led them to the conclusion that “treatment of H. pylori infection in patients with isolated iron deficiency or mild anemia may not be appropriate.”

First, the study was conducted in a population with a high prevalence of clarithromycin- and metronidazole-resistant Helicobacter pylori strains, leading to a failure rate of 77% [2]. Indeed, a recently published article about this trial, also authored by Gessner et al., reported that, 2 months after the initiation of triple therapy for H. pylori eradication, only 34% of the children had cleared their infections [3], leading one to question the basis for their assertion that “Alaskan Natives from Western Alaska are an ideal population in which to evaluate [the H. pylori–iron deficiency relationship] hypothesis” (p. 538).

Second, the study included only children with iron deficiency, who made up only 38% of the children 7–11 years old, which thus limited the generalizability of the study to iron-deficient children, despite epidemiologic evidence suggesting that H. pylori–infected subjects have low serum ferritin levels even if they do not fall into the category of iron deficient [4–8].

Third, the analyses of Gessner et al. [1] compared a group of H. pylori–infected children who received triple therapy for H. pylori eradication with a group of children who received ferrous sulfate supplementation; as such, they could not really assess the effect of H. pylori infection on iron stores. Rather, they compared the efficacy of 2 treatment regimens. A more informative analysis would have been to compare the percentage change in levels of serum ferritin—an approach that has been used in epidemiologic studies [5, 8]. The data presented in their table 1 [1] show that the mean levels of serum ferritin for both the triple H. pylori eradication and the ferrous sulfate regimen was 6.2 μg/L. Their table 3 shows that, at 14 months, the mean levels of serum ferritin had increased to 8.5 and 7.8 μg/L in the intervention and control groups, respectively. The point estimates of the percentage changes were 37.1% and 25.8% in children who received triple H. pylori eradication therapy plus iron and iron only, respectively. In our opinion, these are not effects of a size to be easily dismissed. Importantly, the direct comparison with a group receiving neither triple H. pylori eradication therapy nor ferrous sulfate (a true control group) was omitted from the study.

Clearly, better designed and, probably, larger trials are needed to address the issue of whether a “seek and treat” approach to H. pylori infection results in improved levels of iron stores. Even Gessner et al., in a third report from this study [9], suggested that “H. pylori infection may be an important risk factor for iron deficiency and iron deficiency anemia among children in rural Alaska and possibly in other areas of the world where these conditions are highly prevalent” (p. e396) [9].

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Reply to Cardenas et al.

To the Editor—Cardenas et al. raise several important criticisms of our study [1]. As they note, after initial clarithromycin-based triple therapy, only 34% of children in the intervention group cleared their in-

References