

Helicobacter pylori and cobalamin deficiency

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We have read with interest Maktouf et al.'s letter about the etiological role of deficiencies of cobalamin or folate in megaloblastic anemia.¹ In this large prospective series, they investigated 478 patients with megaloblastic changes in bone marrow smears and reported the demographic, biological and clinical findings in cobalamin-deficient patients and patients with pernicious anemia. Out of 439 patients, 430 (98%) expressed low serum cobalamin levels and a pure cobalamin deficiency was detected in 424 patients. They reported that megaloblastic anemia was almost exclusively due to cobalamin deficiency. It is well known that identification of the underlying cause is important in the diagnosis of cobalamin deficiency that is usually attributed to malabsorption. Carmel *et al.*'s found that patients with food-cobalamin malabsorption and low levels of serum cobalamin had a higher seroprevalence of *Helicobacter pylori* (*H pylori*) infection.^{2,3} The association between *H pylori* infection and food-cobalamin malabsorption suggests that gastritis induced by *H pylori* infection predisposes to a more severe form of food-cobalamin malabsorption. In Kaptan *et al.*'s study, upper gastrointestinal endoscopy documented *H pylori* infection in 77 (56%) of 138 patients with cobalamin deficiency.⁴ Although Maktouf *et al.* also reported *H pylori* positivity in 5 (11.6%) of 43 patients, they evaluated only 43 of 430 patients for *H pylori* and did not report how evaluated their patients for *H pylori*.¹ It has also been shown that *H pylori* is a causative agent in the development of adult cobalamin deficiency, and eradication of *H pylori* infection alone may correct cobalamin

levels.⁴ It may be speculated that association of cobalamin deficiency and *H pylori* infection is coincidental, but restoration of anemia and the cobalamin deficient state in a significant group of patients via eradication therapy is strongly suggestive of this gram-negative rod's role in the pathogenesis. If the microorganism could be eradicated, patient does not need lifelong cyanocobalamin replacement therapy.

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