The Role of Interleukin 10 in the Associations between Migraine and Helicobacter pylori Infection

To the Editor

We read with great interest the recent contribution by Faraji et al (1). They reported that Helicobacter pylori (HP) eradication significantly improved the clinical symptoms of migraine, measured with the MIDAS score and speculated that inflammation, oxidative stress, nitric oxide imbalance, or virulence of CagA-positive HP strains might be possible factors between the associations between migraine and HP infection (1).

However, we would like to add another possible mechanism on the beneficial effect of HP eradication on migraine. Munno et al. reported that high interleukin (IL)-10 levels were seen in 52.2% of the 23 patients with migraine and 10 showed a subsequent decrease in IL-10 after the treatment with sumatriptan, suggesting IL-10 might be involved in the pathogenesis of migraine (2).

Also, Hida et al (3) showed that CagA positive HP infection was associated with the increase in IL-10 mRNA expression and Haeberle et al (4) demonstrated that when peripheral blood leukocytes from either infected or uninfected donors were stimulated with live HP, IL-10 levels increased 2- to 5-fold, compared to cells stimulated with medium alone. Furthermore, Windle et al (5) found that IL-10 production by lamina propria lymphocytes was significantly higher in the HP positive group after stimulation with HP-NAP ($P < 0.05$) than in the HP negative group.

Therefore, there is a possibility that IL-10 stimulated by HP might play an important role in the severity of migraine symptoms in patients with HP infection and eradication of HP could relieve the symptoms. However, further studies are necessary to elucidate the exact molecular role of IL-10 and signaling pathway in the pathogenesis of migraine related to HP infection.

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