Protection from Preeclampsia

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To the Editor: We appreciated the attentive and interesting paper by Huai *et al*.[1] on the protective effects of pravastatin on preeclampsia (PE)-like effects in mouse models. We would like to remind that PE is the first cause of illness and death for pregnant women and their children worldwide, the highest prevalence being in South America. Hence, every measure capable of preventing or averting the full-blown eclampsia is very welcomed. In mice, *Helicobacter pylori* infection exerted a negative effect upon pregnancy; in humans, Eslick *et al*.[2] observed fetal intrauterine growth restrictions in newborns from mothers infected with *H. pylori*. This bacterium was also shown to be an independent risk factor for cerebral ischemia of atherothrombotic origin. Therefore, we reasoned that the cerebral ischemia typically occurring in PE could be associated with the infection by this pathogen. Indeed, we and others found it to be the case;[3] in Turin, Italy, 90.6% of fetal intrauterine growth restrictions occurred in newborns from PE mothers carrying pathogenic strains of *H. pylori* with an odd ratio of 54.97 (95% confidence interval: 9.24–326.88), compared to noncytotoxin-associated gene A (cagA) strain-infected mothers.[4] These strains are characterized by the presence of the *cagA* coding for the CagA protein that is endowed with an increased inflammatory potential. For example, the infection in mouse by CagA-expressing strains induces a Th1-type response even at the level of the endometrium. Such phenomenon would downregulate the secretion of cytokines produced by Th2 immunocytes, which enable fetal survival. Not surprisingly, South American countries have a very high prevalence of both PE and *H. pylori* infection, whose long-term consequence, i.e., gastric cancer, still is the first cause of cancer death in Peru and Colombia, the second in Mexico and Brazil, as well as in Japan. Referring to possible causes of ischemia, *H. pylori* was shown to bind von Willebrand factor and to interact with glycoprotein Ib to induce platelet aggregation;[5] not only, but both in mice and in humans, *H. pylori* infection causes platelets activation and that of endothelial cells. All of the above is present in the preeclamptic state that could eventually lead to the full-blown eclampsia. We believe that it would be prudent to add *H. pylori* in the workup of pregnancy tests.

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Conflicts of interest

There are no conflicts of interest.

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