body response to *H. pylori* and first nonecardioembolic ischemic stroke. This finding is in accordance with recently published epidemiological studies and meta-analyses on coronary artery and cerebrovascular diseases.\[16\],[18],[20]\) According to Cremonini et al., association between *H. pylori* positivity, anti-CagA positivity and stroke is modest and seems higher with stroke due to large vessel disease.\[20\] Further studies that are presently ongoing and planned for the near future are expected not only the elucidation of the pathophysiology related to the association between chronic bacterial infection and atherosclerosis but evaluating whether antibiotic treatment may result in clinical benefit of the patient.

References


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Invited Comments

Well documented risk factors for acute ischemic stroke, including elevated blood pressure, diabetes mellitus, hyperlipidemia, heavy smoking, and ischemic heart disease do not fully account for the whole risk of stroke and it is frequently observed that stroke patients often do not present any of these predisposing conditions.

Recent studies have demonstrated that inflammatory parameters and chronic and acute infectious diseases have been considered to modify stroke risk independent of conventional risk factors. Increasing evidence has linked infections (especially respiratory and dental infections) to atherosclerosis, myocardial infarction, and stroke.\[12],[13\] On the basis of current knowledge, infection/inflammation, specific genetic predispositions, and traditional risk factors interact with each other and may cooperatively enhance the risk of stroke.\[11\] Therefore, a genetically determined strong response to inflammatory stimuli (e.g., acute or chronic infection) may be associated with an increased risk for stroke.

Infections may induce thrombosis and brain infarction by several mechanisms. Some mechanisms indicate that infections are related to large-artery atherogenesis. Other mechanisms suggest a prothrombotic state. Chronic infection may indirectly influence the risk of atherosclerosis and thrombosis by other pathways, such as: 1) changes in lipid metabolism, 2) immune-mediated mechanisms; 3) recurrent bacteremia which may induce platelet activation and a procoagulant state, 4) alterations in endothelial function, 5) spasm in vascular smooth muscle, and 5) atheroma instability and subsequent plaque rupture.

Infection with *Helicobacter pylori* is among the infections disease discussed in this respect. Recent results regarding *H. pylori* antibody titers and stroke reported an association be-
between atherothrombotic and lacunar stroke (but not with other stroke subtypes) and *H. pylori* seropositivity, as shown by the study published in the current issue of the Journal.\[^3\]\ How-
 ever, in a recent study, only increased antibody titers against cytotoxin-associated gene-A bearing strains of *H. pylori* were independently associated with cerebral ischemia, whereas *H. pylori* seropositivity in general was not.\[^4\]

The link between *H. pylori* infection and ischemic stroke requires further studies. *H. pylori* infection is a potentially curable disease and for this reason, the identification of this condition as a stroke risk factor may have important implications for the prevention of acute cerebrovascular accidents. If the potential role of *H. pylori* infection as a risk factor for stroke will be confirmed, antimicrobial treatment would have a role in the prophylactic strategies of cerebral ischemia, together with effective drug therapies based on antiplatelet agents, therapeutic anticoagulation or treatment with statins.

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References