Letter to the Editor

Pathogen Burden and Hypertension: More Questions Than Answers

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To the Editor: We read with great interest the article “Association of Pathogen Burden and Hypertension: The Persian Gulf Healthy Heart Study” by Vahdat et al.1 Although the authors of this well-conducted study negate the association between pathogen burden and hypertension, we believe that some additional issues need to be clarified.

First, the role of periodontal pathogens, which have been widely investigated for their involvement in hypertension and atherosclerosis,2 is totally ignored by the authors. In particular, Porphyromonas gingivalis is currently accepted as a pathogen that possibly contributes to pathogen burden in atherosclerosis.3 Furthermore, there is accumulating cross-sectional and epidemiological evidence associating chronic periodontitis not only with elevated blood pressure levels but also with target organ damage.2 Thus, it would be really interesting to assess the coexistence of periodontal pathogens in the study’s population.

Second, the authors provide no data regarding hypertension stage, management, and control or target organ damage in their population. Assessment of atherosclerosis and vascular dysfunction would also provide additional insight into the contribution of pathogen to atherosclerosis.

Third, we should take into consideration that Helicobacter pylori, cytomegalovirus, and the coexistence of H. pylori and Chlamydia pneumoniae were significantly associated with hypertension after adjustment for traditional cardiovascular risk factors in this study. Therefore, one cannot refute the notion of pathogen burden involvement in hypertension based on the results of this study. The coexistence of H. pylori and C. pneumoniae, together with other bacterial or viral pathogens, may have been associated with hypertension.

In conclusion, the study by Vahdat et al. provides more questions than answers regarding the controversial issue of infectious burden in hypertension and atherosclerosis. Thus, prospective studies evaluating the coexistence of a number of pathogens that have been involved in atherosclerosis (C. pneumoniae, H. pylori, cytomegalovirus, herpes simplex virus, Porphyromonas gingivalis, Epstein–Barr virus, Hemophilus influenzae, Mycoplasma pneumoniae, influenza A virus, hepatitis C virus, human immunodeficiency virus) based on serological, histopathological, and molecular detection are warranted. Beyond that, the possible therapeutic role of antibiotics and or antivirals needs to be further investigated in well-designed interventional studies.

REFERENCES

