

A Next Step Practice of Infection as a Trigger for Cardiovascular Diseases



To the Editor:

In their recently published article “Infection as a cardiovascular trigger: associations between different organ system infections and cardiovascular events,” Sebastian and colleagues indicated the importance of infection as a trigger for cardiovascular events, reporting a gradient of decreased magnitudes of association with longer latent periods and cardiovascular events across the majority of infection types.¹ Another novelty of this study was the potentially minimal risk of statistical confounding factors, brought by a case-crossover analysis, reinforcing the reliability of the results of this study.

As they concluded, some infection may require cardiovascular prophylaxis. For instance, coronavirus disease is notorious for its tendency to cause coagulopathy. Some expert societies recommend pharmacologic prophylaxis of venous thromboembolism for all hospitalized patients.² In contrast, however, a study by Gafter-Gvili et al, recently published in *The American Journal of Medicine*, indicated that routine prophylaxis of venous thromboembolism did not reduce either mortality or venous thromboembolism,

but increased major bleeding. In the subanalysis of active infection in their study, 16.4% of the patients with venous thromboembolism prophylaxis passed away, whereas the mortality rate of patients without venous thromboembolism prophylaxis was 6.4%.³

The study undertaken by Sebastian and colleagues did not confirm the clinical benefit of cardiovascular disease prophylaxis in individuals with infection. Therefore, more detailed investigations are warranted to assess the risk-benefit balance and clarify the concrete methods of anticoagulation in infected patients, possibly leading to better clinical practices and improvement of prognosis in critical patients.

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