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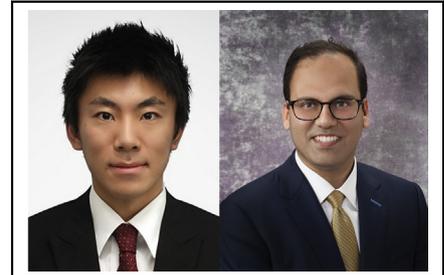


Commentary: Infective endocarditis of the aortic valve: External influences?

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Infective endocarditis (IE) can be a highly morbid condition, particularly when the infection is invasive, with valvular destruction and annular involvement. In-hospital mortality and 1-year mortality with IE have been reported to be 15% to 20% and 40%, respectively, despite surgical interventions.¹ A Society of Thoracic Surgeons study examined 34,905 patients who underwent valve surgery from 2011 through 2018.² The incidence of drug-induced and nondrug-induced IE had increased 2.7-fold and 1.4-fold during the study period, respectively. An aging population with comorbidities, increasing number of patients with implanted devices, and a rapid growth in intravenous drug use likely contributed to the trend.³ While public health strategies to manage the opioid crisis may have a downstream impact on the trends for IE, the only strategy recommended to prevent nonintravenous drug use–related IE in the American Heart Association/American College of Cardiology guidelines was antibiotic prophylaxis before a dental procedure.¹ In this setting, some studies have postulated the potential benefit of antiplatelet or anticoagulation to prevent IE.

In the December 2021 issue of *JTCVS Open*, Theys and colleagues⁴ examined their experience with IE to investigate whether antiplatelet or anticoagulation agents have a protective effect for IE. The study included 2 cohorts: (1) patients



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CENTRAL MESSAGE

The risk of developing infective endocarditis is maybe related to patient-related risk factors as opposed to the influence of antiplatelets and antiaggregants.

who underwent aortic valve replacement with a bioprosthetic valve (AVR cohort) between 2009 through 2019 and (2) patients with IE of the native aortic valve (NVE cohort) between 2007 through 2015.⁴ Patients who received mechanical valve were excluded. The AVR cohort consisted of 333 patients, and 16 (4.8%) developed IE over the median of 3.72 years. Fine-Gray analysis was adopted and demonstrated that “Fondaparinux or low-molecular-weight heparins” were significantly associated with the development of IE, contrary to their hypothesis (hazard ratio, 4.61; 95% confidence interval, 1.01-21.9). The NVE cohort included 137 patients. As there was no comparative cohort, the authors compared the relationship between NVE and with/without each antiplatelet or anticoagulation therapy. Patients who received vitamin K antagonists, dual antiplatelet therapy, novel oral anticoagulants, or “fondaparinux or low-molecular-weight heparins” were associated with a significantly greater incidence of IE than patients without any antiplatelet or anticoagulation. Considerably, those multivariable analyses only included age, sex, and year of surgery aside from antiplatelets and anticoagulants in the model.

Risk factors for IE include acquired degenerative valvular disease, congenital cardiac abnormality, immunosuppression, diabetes, hemodialysis, intravenous drug use, and implanted devices.⁵ Limited variables in the multivariable analyses certainly raise concern for uncaptured cofounders in this study. For instance, patients who have pacing leads at risk for developing IE are prone to receive antiplatelet or anticoagulation because of prothrombotic

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Disclosures: I.S. receives institutional research support from Abbott, Edwards, Medtronic, and AtriCure. These conflicts are unrelated to this article. T.O. reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

Received for publication Nov 14, 2021; revisions received Nov 14, 2021; accepted for publication Dec 8, 2021; available ahead of print March 16, 2022.

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JTCVS Open 2022;10:183-4
2666-2736

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<https://doi.org/10.1016/j.jtc.2021.12.013>

nature of foreign intravascular bodies. Patients who have diabetes with coronary artery disease may have taken antiplatelet therapy. Furthermore, antiplatelets or anticoagulants are frequently prescribed after valve replacements. The aforementioned guidelines recommended lifelong aspirin or vitamin K antagonist for 3 to 6 months after AVR with bioprosthetic valve (class 2a).¹ Discharge from a hospital with antiplatelets or anticoagulation is often observed for thromboembolic complications such as stroke and venous thromboembolism after valve surgery. The authors acknowledged that other factors might have impacted their findings.⁴ Whether a direct relationship between antiplatelet or anticoagulation and IE exists is very much unknown, and this study may lead to us asking more questions as opposing to drawing conclusions.

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