Please note: Dr. Madias has reported that he has no relationships relevant to the contents of this paper to disclose.

REFERENCES

1. Cahill TJ, Baddour LM, Habib G, et al. Challenges in infective endocarditis. J Am Coll Cardiol 2017;69:325–44.

2. Patrono C, Rocca B. Type 2 diabetes, obesity, and aspirin responsiveness. J Am Coll Cardiol 2017;69:613–5.

3. Chan KL, Dumesnil JG, Cujec B, et al., Investigators of the Multicenter Aspirin Study in Infective Endocarditis. A randomized trial of aspirin on the risk of embolic events in patients with infective endocarditis. J Am Coll Cardiol 2003;42:775–80.

4. Veloso TR, Oechslin F, Que YA, Moreillon P, Entenza JM, Mancini S. Aspirin plus ticlopidine prevented experimental endocarditis due to Enterococcus faecalis and Streptococcus gallolyticus. Pathog Dis 2015;73:ftv060.

5. Veloso TR, Que YA, Chaouch A, et al. Prophylaxis of experimental endocarditis with antiplatelet and antithrombin agents: a role for long-term prevention of infective endocarditis in humans? J Infect Dis 2015;211:72–9.

REPLY: Aspirin for the Prevention of Infective Endocarditis?

Dr. Madias proposes a potential role for aspirin in the prevention of infective endocarditis (IE). The role of platelets in the pathogenesis of IE is established: in addition to enabling bacterial adhesion, activated platelets lead to formation of neutrophil extracellular traps, which enmesh platelet-bacterial aggregates together to facilitate vegetation formation (1). Furthermore, platelets contribute to the formation of multilayered biofilms. However, platelets also have protective effects through release of platelet microbicidal proteins and activation of the immune response.

Findings in preclinical models reflect these dual functions. In a rabbit model of experimental streptococcal IE, platelets have been shown to restrain development of early IE (2). In a rat model of staphylococcal and streptotoccal IE, Veloso et al. (3) demonstrated that platelet inhibition using aspirin and ticlopidine could prevent formation of vegetations. Notably, this required dual agent antiplatelet inhibition with both aspirin and ticlopidine; either drug alone had no significant effect.

Clinical data to support aspirin for prophylaxis of IE are currently lacking. Given the scale and cost of conducting a randomized controlled trial, we agree that analysis of existing cohorts of "at-risk" groups would be valuable and might provide supportive evidence. In fact, a prospective series addressing precisely this question in patients with bioprosthetic heart valves has recently been proposed (4). Finally, the risks of aspirin prophylaxis need to be taken into account, and depending on the population, dose and nature of the dental/surgical intervention, might be associated with an increase in bleeding complications.

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REFERENCES

1. Jung C-J, Yeh C-Y, Shun C-T, et al. Platelets enhance biofilm formation and resistance of endocarditis-inducing streptococci on the injured heart valve. J Infect Dis 2012;205:1066–75.

2. Sullam PM, Frank U, Yeaman MR, Tauber MG, Bayer AS, Chambers HF. Effect of thrombocytopenia on the early course of streptococcal endocarditis. J Infect Dis 1993;168:910–4.

3. Veloso TR, Que Y-A, Chaouch A, et al. Prophylaxis of experimental endocarditis with antiplatelet and antithrombin agents: a role for long-term prevention of infective endocarditis in humans? J Infect Dis 2015;211:72–9.

4. Veloso TR, Que Y-A, Moreillon P, Entenza JM. Reply to Eisen and McBryde. J Infect Dis 2015;212:674–5.