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Letters

TO THE EDITOR

Stent Fracture and Hypersensitivity

What Happens First?

Mori el al. (1) hypothesized that stent fracture accelerates release of metal ions leading to a focal hypersensitivity reaction and stent thrombosis. Although reasonable, we propose an opposite theory regarding the relationship between stent fracture and hypersensitivity.

Kounis syndrome (KS) is an allergic acute coronary syndrome induced by exposure to drugs, food, stent metallic components, and other triggers. Three major variants of KS have been described: types I, II, and III. KS type III (the least common, at 5.1%) includes patients with stent thrombosis/restenosis secondary to an allergy, possibly to the metallic ions. Mast cell degranulation and inflammatory mediators release is triggered by antigen-antibody reaction on the surface of the mast and basophil cells, and activation of the complement system. This stimulates a coagulation cascade with platelet aggregation and collagen fibers deposition around the implant to form a dense, acellular, and neointimal hyperplasia causing stent thrombosis/restenosis (2). Although unproven pathologically, KS had been previously reported with bare-metal stents (3).

Hoshi et al. (4) reported a patient with KS caused by metal allergy and resulting in coronary aneurysm. Coronary angioscopy and optical coherence tomography showed malapposed stent struts at the sites of positive remodeling, whereas multiple interstrut hollows and neointimal hyperplasia covering stent struts were observed in multiple areas. This supports our theory that hypersensitivity happens first and if progressed may result in-stent cavitations and subsequent fracture.

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RESEARCH LETTER:

Cangrelor Use in Cardiogenic Shock

A Single-Center Real-World Experience

Cardiogenic shock complicates approximately 5% of contemporary cases of acute coronary syndrome and is associated with an adverse prognosis, especially early after percutaneous coronary intervention (PCI) (1). Perturbations in hemodynamics, together with impaired gut absorption of oral antiplatelet therapies, may contribute to excess risk of early stent thrombosis (ST) in cardiogenic shock. Cangrelor, a parenteral, fast-acting, reversible $P2Y_{12}$ inhibitor, may possess favorable pharmacological properties in this high-risk cohort. Although the 3 phase 3 CHAMPION (Cangrelor versus Standard Therapy to Achieve Optimal Management of Platelet Inhibition) trials (2) supported cangrelor's regulatory approval for use across a spectrum of PCI, these experiences excluded patients in cardiogenic shock. As such, the relative safety and tolerability of cangrelor in these patients are presently unknown.

In this single-center experience from a large tertiary-care center, we report patterns of use and periprocedural outcomes in patients in clinical shock who received cangrelor. Shock was adjudicated by 2