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STATE-OF-THE-ART PAPER

VIEWPOINT

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CLINICAL RESEARCH

INTERVENTIONAL CARDIOLOGY

Stone and colleagues sought to determine whether the reduction in cardiac mortality in patients undergoing primary percutaneous coronary intervention with bivalirudin compared with unfractionated heparin plus a glycoprotein IIb/IIIa inhibitor in the HORIZONS-AMI (Harmonizing Outcomes With Revascularization and Stents in Acute Myocardial Infarction) trial can be fully attributed to reduced bleeding. Bivalirudin resulted in lower 3-year rates of major bleeding (6.9% vs. 10.5%) and cardiac mortality (2.9% vs. 5.1%). The 3-year cardiac mortality was reduced in bivalirudin-treated patients with major bleeding (5.8% vs. 14.6%) and without major bleeding (2.6% vs. 3.8%). In a fully-adjusted multivariable model accounting for major bleeding and other adverse events, bivalirudin was still associated with a 43% reduction in 3-year cardiac mortality. The reduction in cardiac mortality with bivalirudin in ST-segment elevation myocardial infarction patients can only partly be attributed to the prevention of bleeding.

CORONARY SURGERY

Qin and colleagues sought to determine the role of suppressor of cytokine signaling 1 (SOCS1) in graft arteriosclerosis (GA). SOCS1, a negative regulator of cytokine signaling, is highly expressed in endothelial cells (ECs) and may prevent endothelial inflammatory responses. Coronary arteries with GA, with atherosclerosis, or without disease were collected for histological analysis. SOCS1 knockout mice or vascular endothelial SOCS1 transgenic mice were used in an aorta transplant model of GA. A dramatic, but specific, reduction of endothelial SOCS1 was observed in human GA and atherosclerosis specimens. SOCS1 deletion in mice resulted in basal EC dysfunction. Further studies confirmed the importance of SOCS1 in preventing GA progression by preserving endothelial function and attenuating cytokine-induced adhesion molecule expression in vascular endothelium.

Editorial Comment: Roel A. de Weger, p. 30

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