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STATE-OF-THE-ART PAPER

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The Role of Monocytes in Angiogenesis and Atherosclerosis

Anthony S. Jaipersad, Gregory Y. H. Lip, Stanley Silverman, Eduard Shantsila

Monocytes are intimately involved in tissue damage and repair, and an imbalance of these processes may have detrimental consequences for plaque development and stability. Monocytes are comprised of distinct subsets identified by cell surface markers; these subsets have distinct functional characteristics, and this heterogeneity may be relevant to the angiogenic processes in atherosclerosis. Jaipersad and colleagues present an overview of the available evidence supporting a role for monocytes in angiogenesis.

VIEWPOINT

VIEWPOINT

The Disconnect Between Guidelines, Appropriate Use Criteria, and Reimbursement Coverage Decisions

Richard I. Fogel, Andrew E. Epstein, N. A. Mark Estes III, Bruce D. Lindsay, John P. DiMarco, Mark S. Kremers, Suraj Kapa, Ralph G. Brindis, Andrea M. Russo

Recently, the American College of Cardiology Foundation, in collaboration with the Heart Rhythm Society, published appropriate use criteria (AUC) for implantable cardioverterdefibrillators (ICDs) and cardiac resynchronization therapy (CRT). These criteria were developed to critically review clinical situations that may warrant implantation of an ICD or CRT device. The writing committee recognized that some of the scenarios that were deemed as "appropriate" or "may be appropriate" were discordant with the clinical requirements of many payers, including the Medicare National Coverage Determination (NCD). To charge Medicare for a procedure that is not covered by the NCD may be construed as fraud. Discordance among the guidelines, the AUC, and the NCD causes clinicians to have the difficult dilemma of trying to do the "right thing" for their patients, but recognizing that the "right thing" may not be covered by the payer or insurer.

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INTERVENTIONAL CARDIOLOGY

Bleeding and Cardiac Mortality in the HORIZONS-AMI Trial

Gregg W. Stone, Tim Clayton, Efthymios N. Deliargyris, Jayne Prats, Roxana Mehran, Stuart J. Pocock

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

SOCS1 Prevents Graft Arteriosclerosis

Lingfeng Qin, Qunhua Huang, Haifeng Zhang, Renjing Liu, George Tellides, Wang Min, Luyang Yu

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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