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Acute Coronary Syndromes

VENTRICULAR ARRHYTHMIA BURSTS FOLLOWING PRIMARY PCI FOR ACUTE MYOCARDIAL INFARCTION: CORRELATIONS WITH CMRI OF MICROVASCULAR OBSTRUCTION AND FINAL INFARCT SIZE

Poster Contributions
Poster Sessions, Expo North
Sunday, March 10, 2013, 9:45 a.m.-10:30 a.m.

Session Title: Percutaneous Coronary Intervention for AMI: Predictors of Outcome
Abstract Category: 1. Acute Coronary Syndromes: Clinical
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Background: The presence of reperfusion arrhythmias have shown to be correlated with larger infarct size (IS). Inadequate microvascular reperfusion, as determined by microvascular obstruction (MVO), is known to be associated with larger IS. We tested the hypothesis that ventricular arrhythmias (VA) bursts are a marker for larger IS in the setting of optimal epicardial and microvascular reperfusion (TIMI 3 flow and MVO).

Methods: All 68 ST elevation myocardial infarction patients from the MAST study with 24 hours continuous 12-lead Holter beginning prior to primary percutaneous coronary intervention resulting in brisk epicardial flow restoration (TIMI 3 flow) were included. VA bursts were identified against subject-specific background VA rates using a previously published statistical outlier method. IS and MVO were determined using late enhancement cardiac magnetic resonance imaging (CMR) (mean 4.9 days after admission). Both Holter and CMR results were determined in core laboratories blinded to all other data.

Results: MVO was present in 39/68 (57%) of patients. No significant differences were found for demographic characteristics, comorbidities, infarct location, number of diseased coronary vessels, or duration of ischemia between groups with and without MVO. IS was significantly smaller in the group without MVO (median 9.4% vs 20.5%; $p < 0.001$). IS in the group with neither MVO nor VA burst (11/68, 16%) was significantly smaller than in the group with either MVO and/or VA burst (median 4.1% vs 18.7%; $p = 0.000$). In the subgroup without MVO there was a trend for larger IS in the 18/29 with VA burst (median 4.1% vs 10.4%; $p = 0.053$).

Conclusion: Absence of MVO was associated with smaller IS. In this group, the absence of VA burst further stratifies patients to the group with the smallest IS. These results support the mechanistic conclusion that VA burst is a signal of damage occurring further downstream at myocellular level.