Myocardial Infarction and Infarction

1100-41

An Angiographic Risk Score Integrating Both Epicardial and Tissue Level Perfusion Before and After Facilitated Percutaneous Coronary Intervention in Acute MI
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Background: Both epicardial and tissue level perfusion have been related to clinical outcomes in the setting of acute myocardial infarction (AMI). Although myocardial reperfusion therapy and the performance of additional steps to rescue percutaneous intervention may alter clinical outcomes after thrombolytic administration, Objectives & Methods: The goal of this study was to develop a simple, broadly applicable method that integrates epicardial and tissue level perfusion both before and after PCI to arrive at a single angiographic risk score (ARS) in patients undergoing PCI after thrombolysis. The angiographic risk score is the arithmetic sum of the TIMI Flow Grade (0-3) before and after PCI (total possible score of 0-12). This risk score was evaluated in patients from the LIMIT AMI trial of IPA monotherapy vs fibrinolytic therapy. Arterial vessel size was assessed using a previously validated computer algorithm (QGS). Data from patients with an angiographic risk score in the lowest group (%d)-6 had a risk of 30 day death or MI of 9.3% (5/44), whereas those with an ARS of -7% had a risk of 1.3% (7/598) (p=0.04). There were no deaths or recurrent MIs among patients with a risk score greater than 10. Likewise, larger SPECT infarct sizes were observed among patients with an ARS of -0.6 (22.6± 20.4%, n=59) compared to patients with an ARS of 7-12. (12.3± 13.4%, n=71, p=0.001). In a second analysis, data from patients who did not undergo PCI was incorporated by using the final TIMI Flow Grade and the final TIMI Myocardial Perfusion Grade on diagnostic angiography instead of the post PCI values, and similar results were seen: the risk of 30 day death or MI was 11.7% (11/ 94) for ARS of -0.6, whereas it was 4.2% (6/143) for ARS of 7-12. SPECT infarct sizes were larger for ARS of 0-4 (21.0± 19.0, n=98) vs ARS of 7-12 (11.8± 15.2, n=127, p=0.0001). Conclusions: The angiographic risk score integrating epicardial and tissue level perfusion before and after PCI or at the end of diagnostic cardiac catheterization to arrive at a single risk estimate that is associated with infarct size and 30 day death or MI. Failure to achieve an ARS of -6 is associated with a doubling of infarct size.

1100-42

Myocardial Perfusion in Patients With Non-ST- Segment Elevation Acute Coronary Syndrome Assessed With Venous Contrast Echocardiography
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Non ST segment elevation acute coronary syndrome (NSTEACS) is a dynamic condition and the underlying pathophysiology is currently thought to consist not only of atherothrombotic plaque rupture and superimposed thrombosis of an epicardial coronary artery but also microvascular obstruction to flow in the microcirculation, microvascular steal from the epicardial arteries to microvascular endothelium. We therefore sought to compare venous myocardial contrast echocardiography (VMCE), a new bedside technique allowing for exclusively imaging myocardial vasculature, to Sestamibi SPECT (S), angiographic and biochemical findings in patients with NSTEACS.

60 patients (women, n=15, age 65±11y) with typical anginal chest pain at rest or during minimal physical activity plus either transient EKG changes (without ST segment elevation) or elevated Troponin T (>0.3 µg/l) on presentation were included in the study. All patients underwent VMCE, SPECT, ICA and coronary angiography within 1 hour after presentation. Sestamibi 740 MBq (87%) had myocardial contrast defects on VMCE. Concordance with respect to presence of myocardial defects on SI was 88% (0.74). 161 patients (43%) had TIMI Flow <3, <2 and 0 in 17 patients (10%) had a contrast defect on VMCE. In 170 patients (66%) had elevated Troponin I. 34/60 patients (57%) had TIMI Flow 3; 26/34 patients (76%) had a contrast defect on VMCE and 14/34 patients (41%) had elevated Troponin I. All of these patients had a contrast defect on VMCE and 17/26 patients (65%) had elevated Troponin I. These data suggest that in the absence of reperfusion, infarct size is smaller (rather than larger) in the elderly. Further studies will clarify if this hypothesis is correct, the distribution of culprit vessels in non ST-segment acute coronary syndromes(ACS) will be expected to be relatively even. Methods: We retrospectively analyzed angiograms from our cath lab database to identify the culprit vessel in 166 pts with ST-elevation MI and in 134 others without ST-elevation ACS. Results: In pts withheld ST-elevation MI, the CFX was uncommonly the culprit vessel (12%), compared to LAD (43%) or RCA (46%) involvement (p=0.001). In pts with non-ST-elevation ACS, the distribution of culprit vessels was more even and in fact the CFX was most frequently the culprit vessel (40%), whereas the LAD was responsible in 28% of pts and the RCA in 32% of cases (CFX vs LAD or RCA, p=NS). Concordance of non-ST-elevation ACSs with TIMI flow 3 was expected to be relatively even. Conclusion: These data suggest that in the absence of reperfusion, infarct size is smaller (rather than larger) in the elderly. Further studies will clarify if this hypothesis is correct, the distribution of culprit vessels in non ST-segment acute coronary syndromes(ACS) will be expected to be relatively even. Methods: We retrospectively analyzed angiograms from our cath lab database to identify the culprit vessel in 166 pts with ST-elevation MI and in 134 others without ST-elevation ACS. Results: In pts withheld ST-elevation MI, the CFX was uncommonly the culprit vessel (12%), compared to LAD (43%) or RCA (46%) involvement (p=0.001). In pts with non-ST-elevation ACS, the distribution of culprit vessels was more even and in fact the CFX was most frequently the culprit vessel (40%), whereas the LAD was responsible in 28% of pts and the RCA in 32% of cases (CFX vs LAD or RCA, p=NS). Concordance of non-ST-elevation ACSs with TIMI flow 3 was expected to be relatively even. 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