2:45

848-4 | Angiographic Validation of Bedside Markers of Coronary Reperfusion in Acute Myocardial Infarction; Can Unnecessary Emergency Angioplasties be Reduced? A Prospective Study

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Background: Infarct-related arteries which fail to recanalize after thrombolytic therapy may benefit from rescue PTCA while conservative treatment is recommended in reperfused vessels. Non-invasive detection of reperfusion might be helpful to decide which pts need angiography.

Methods: In a prospective angiographic study, the value of 7 ECG and clinical markers of reperfusion were analyzed in 233 pts with acute myocardial infarction. All pts had a 12-lead ECG on admission and immediately before angiography. Non-invasive markers before angiography were correlated to flow: Analysis A: TIMI 2–3 vs TIMI 0–1 flow; Analysis B: TIMI 3 vs TIMI 0–2 flow. Outcome is expressed as positive (P) and negative (N) predictive values (PV).

Results:

Predictive values of bedside markers of reperfusion

	Prevalence (%)	PPV A	PPV B	NPV A	NPV B
Decrease ST-segment elevation >50%	30	85	66	75	87
Decrease chest pain	41	78	57	80	89
Increase chest pain	0	48	31	57	71
AIVR	7	94	59	61	73
Ventr. fibrillation	6	62	46	58	72
Increase in ventricular ectopy	16	73	55	62	73
Terminal negative T-wave, >0.1 mV	21	78	39	65	79

Conclusions: Bedside markers (especially decrease of ST segment elevation) are of value to assess reperfusion. However, differentiation between TIMI 2 and TIMI 3 flow is not possible.

3:00

848-5 | The Impact of Achieving Predetermined Intravascular Ultrasound Criteria on Angiographic Restenosis After Coronary Angioplasty: An Analysis of the Guide II Trial

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The Guide II trial is a multicenter prospective trial in which PTCA or DCA was performed using angiographic guidance followed by blinded intravascular ultrasound (IVUS) documentation. The purpose of this analysis was to determine the impact of achieving predetermined IVUS criteria on angiographic restenosis. A total of 223 pts who had angiographic follow-up at 6.8 ± 1.2 months were included. Angiographic and IVUS measurements are shown in the table below.

	Restenosis n = 96	No Restenosis n = 127	P value
Pre angio ref. (mm)	2.98 ± 0.68	3.12 ± 0.92	ns
Pre angio MLD (mm)	0.97 ± 0.32	1.01 ± 0.44	ns
Post angio MLD (mm)	2.19 ± 0.48	2.24 ± 0.61	ns
Post angio %DS	28.7 ± 8.77	32.77 ± 9.08	0.002
Post IVUS MLD (mm)	1.92 ± 0.72	2.28 ± 0.88	0.001
Post IVUS LCSA (mm²)	4.3 ± 1.6	5.1 ± 1.8	0.0007
Post IVUS % plaque area	72.14 ± 11.2	59.09 ± 13.6	< 0.0001

The post-procedure IVUS criteria studied were: MLD >2.0 mm, %PA ≤65% and mean lumen reference >3.5 mm. These criteria were met in 103/223 (46%) of pts. The overall restenosis rate was 96/223 (43%). However, lesions in which all IVUS criteria were met had a restenosis rate of 23% compared to a restenosis rate of 60% for lesions in which non of the above criteria were met (p < 0.0001).

Conclusions: Achievement of predetermined IVUS criteria after PTCA is associated with a relatively low restenosis rate.

3:15

848-6 | Impact of Abciximab and Stents on Outcomes and Economics in a Community Hospital

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To determine the effect of abciximab and stents on interventional practice

at a community hospital, events and economics were evaluated for 3,758 consecutive, elective procedures from 1/95–6/97.

Abciximab	PTCA – N = 1368	PTCA + N = 209	Stent – N = 1488	Stent + N = 633
Death%	1.0	0.7	0.7	0.8
Re-PTCA%	0.8	0.4	0.1*	0.2*
CABG%	3.2	0.7*	1.6*	0.6*
Composite% 5.0	1.9*	2.4*	1.3*	
Bleed%	0.0	0.7	0.8	0.8
LOS day	4.1	3.6	3.9	3.5*
Hosp Cost	$7,483	$8,499*	$10,615*	$12,256*

Composite = death + MI + Re-PTCA + CABG, * p ≤ 0.01 vs. PTCA.

Use of abciximab correlated with increasing lesion complexity (ACC/AHA). Higher hospital costs associated with abciximab or stents compared to PTCA alone were due to increased procedural expenses (equipment and drugs). Logistic regression identified prior PTCA (odds ratio [OR]: 1.8, CI 95%: 1.1–2.7) and use of abciximab (OR 2.3, CI 1.3–4.2) or stents (OR 2.2, CI 1.5–3.2) as predictors of the absence of major events in hospital (p < 0.01). Cox regression identified diabetes (OR 1.3, CI 1.1–1.6), multivessel CAD (OR 1.7, CI 1.5–2.0) and absence of stent (OR 1.3, CI 1.1–1.5) as predictors of repeat revascularization within one year (p < 0.01). Thus, despite greater in-hospital cost, abciximab and stents significantly improve outcomes of interventional practice.

849 | Myocardial Contrast Echocardiography: Assessment of Coronary Physiology

Tuesday, March 31, 1998, 2:00 p.m.–3:30 p.m.
Georgia World Congress Center, Room 256W

2:00

849-1 | Structural Mechanisms of Impaired Flow Reserve in Collateral Dependent Myocardium: Insights From Intramyocardial Blood Volume Measurement Using Harmonic Imaging

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Coronary collaterals (coll) maintain resting perfusion to a chronically occluded bed, but are frequently unable to augment flow during stress. The microvascular level (capillary recruitment vs arteriolar vasodilation) at which coll flow reserve impairment resides is unclear. Because videointensity values during myocardial contrast echo (MCE) indicate predominantly capillary blood volume, we used MCE to determine the microvascular site regulating coll responses to pharmacologic stress. An ameroid constrictor was placed around the left anterior descending artery (LAD) in 9 dogs (Day 0) to cause gradual occlusion (occ) and LAD coll growth over 6 weeks (wks). LAD risk bed was defined on Day 0 with intravenous (iv) MRX115 and gated harmonic imaging. MCE was performed after 6 wks: at rest, with iv dobutamine, and adenosine. Flow was measured with radioactive microspheres, and LAD and circumflex (LCX) peak intensity (PI) were derived from MCE images.

Stage	LAD intensity	LAD flow (ml/min/g)	LCX intensity	LCX flow (ml/min/g)
Day 0 occlusion	15 ± 5	0.2 ± 0.1	56 ± 9	1.6 ± 0.2
6 wks occlusion: Rest	47 ± 7†	1.0 ± 0.1†	53 ± 7	1.0 ± 0.1
Dobutamine 40 ug/kg/min	47 ± 4	1.9 ± 0.5*	63 ± 7*	3.6 ± 0.3*
Adenosine 0.4 mg/kg/min	52 ± 6	2.2 ± 0.3*	73 ± 6*	5.8 ± 0.5*

* p < 0.01 vs Rest †p < 0.001 vs day 0

Resting LAD flow and PI increased from Day 0 to 6 wks. With stress, LAD coll flow increased less than LCX flow. With stress, despite LAD hyperemia, LAD PI was unchanged, whereas LCX PI increased. Thus, impaired coll reserve manifests as lack of video intensity change in the coll bed, even with coll hyperemia. These data suggest that capillary volume in the coll-dependent bed is already maximal at rest, and that coll hyperemia, albeit blunted, is mediated by larger resistance vessels not primarily imaged by MCE. By distinguishing capillary from total coronary blood volume, MCE can yield insight into coll regulation.