

2:30

CATHETERIZATION/RESCUE ANGIOPLASTY FOLLOWING THROMBOLYSIS (CRAFT) STUDY: ACUTE MYOCARDIAL INFARCTION TREATED WITH RECOMBINANT TISSUE PLASMINOGEN ACTIVATOR VERSUS UROKINASE

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To assess potential differences in safety and efficacy, 408 patients (pts) with symptoms of acute myocardial infarction (AMI) < 6 hrs and diagnostic ST elevation were randomized to intravenous recombinant tissue plasminogen activator (rt-PA), 100 mg, vs urokinase (UK) 1.5 million IU bolus + 1.5 million IU infusion (90 min). Pts were catheterized at 90 min (baseline) and rescue PTCA was suggested for pts with residual TIMI 0-1 flow. Aspirin and heparin were initiated before the initial cath and continued until a 7-10 day cath. Two hundred six (206) pts received rt-PA and 202 pts received UK. Ninety-minute arterial patency was 126/199 (63%) for rt-PA and 105/198 (53%) for UK (p=0.042). Acute PTCA was performed in 45 (23%) rt-PA pts and 63 (32%) UK pts. Patency leaving the cath lab was 168/199 (84%) for rt-PA and 166/198 (84%) for UK. Angiographic reocclusion during hospitalization was documented in 26/180 (14%) rt-PA pts vs 25/170 (15%) UK pts. Changes from baseline to follow-up left ventricular ejection fraction (EF) and infarct zone wall motion (IZWM) were not different between groups, although IZWM did significantly improve in the UK group.

Group(n)	EF (%)		IZWM (SD/chord)	
	Base-line	Follow-up	Base-line	Follow-up
rt-PA (153)	47.9	48.3	-2.9	-2.8
UK (151)	47.6	47.6	-2.9	-2.6 (p<0.05)

Thirty-day mortality was 12/206 (6%) for rt-PA and 12/202 (6%) for UK. Six-month mortality was 13/206 (6%) for rt-PA and 16/202 (8%) for UK. Major bleeding complications were 8% for rt-PA and 6% for UK. Intracranial hemorrhage occurred in 6 (3.0%) rt-PA pts and in one (0.5%) UK pt (p=0.122). Thus, when combined with rescue PTCA, either UK or rt-PA given within 6 hrs of acute MI results in similar effects on regional and global LV function and survival, with a trend toward more serious complications with rt-PA.

2:45

LESS SEVERE STENOSIS AFTER TREATMENT WITH SARUPLASE WHEN COMPARED TO STREPTOKINASE IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION: RESULTS OF QUANTITATIVE ANGIOGRAPHY.

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In a randomized, double blind study (PRIMI trial) 80 mg of intravenous saruplase (also known as recombinant pro-urokinase; S; 198 pts) was compared with 1.5 Mio IU of i.v. streptokinase (Sk, 203 pts). Treatment was given within 4 hours after onset of symptoms in pts with a first myocardial infarction. Coronary angiography was performed at 60 min, at 90 min, and 24 hours after the start of the infusion. Patency at 60 min was 71% after S and 48% after Sk (p = 0.001), and at 90 min 72% after S vs. 64% after Sk (p = 0.09). The severity of the stenosis was assessed quantitatively using the Coronary Angiography Analysis System. The diameter stenosis (DS) was calculated as well as the area stenosis (AS), the latter with the use of videodensitometry.

	DIAMETER STENOSIS(%)			AREA STENOSIS(%)		
	S	SK	p	S	SK	p
60 MIN	61	74	0.004	93	100	0.002
90 MIN	57	62	ns	92	92	ns
24 HRS	55	51	ns	83	80	ns

In the subset of pts with successful thrombolysis and an open infarct related artery at the 90 min angiogram median DS further decreased from 54% at 90 min to 52% at 24 hours, and median AS decreased from 86% to 82%. **CONCLUSIONS:** After intravenous administration of saruplase the severity of the stenosis in the infarct related artery is less severe than after i.v. streptokinase. A further decrease in the severity of the stenosis is observed from 90 min to 24 hours.

3:00

REGIONAL CORONARY BLOOD FLOW AFTER REPERFUSION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

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Previous animal studies have shown that, following release of a prolonged coronary artery occlusion, coronary blood flow to the affected area is lower than normal, known as the no flow phenomenon. To assess the regional coronary blood flow after reperfusion in patients with acute myocardial infarction (AMI), we studied 7 human subjects with AMI undergoing emergency PTCA and chronic angiography. Regional coronary blood flow velocity (CBFV) was measured using a 3F coronary Doppler catheter positioned in the proximal segment of the infarct artery (IA) and the non-IA, immediately after successful PTCA and during chronic angiography. Maximal hyperemia was produced by intracoronary infusion of papaverine. Coronary flow reserve (CFR) was calculated as the ratio of hyperemic CBFV/resting CBFV. **Results:** In the IA, resting CBFV after reperfusion was not different from that during chronic angiography (15.2 ± 7.8 cm/s vs 13.4 ± 5.6 cm/s) but hyperemic CBFV was significantly lower after reperfusion (18.1 ± 9.3 cm/s vs 30.3 ± 10.3 cm/s, $p < 0.001$). Compared with the non-IA, CFR in the IA was significantly attenuated after reperfusion (1.2 ± 0.3 vs 2.5 ± 0.6 , $p < 0.001$) but not different during chronic angiography (2.3 ± 0.3 vs 3.1 ± 0.8).

Conclusion: Reperfusion of the IA restored coronary blood flow without hyperemia and CFR was abolished. Therefore, coronary vasodilators may redistribute coronary blood flow from the IA to the non-IA, resulting in the no flow phenomenon.

3:15

FIBRINOLYTIC THERAPY FOR UNSTABLE ANGINA - A DOUBLE BLIND PLACEBO-CONTROLLED TRIAL WITH ALTEPLASE

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In patients with acute unstable angina (UA) subtotal thrombotic coronary occlusion has been reported. We studied 80 consecutive pts (56 male, mean age 61 yrs) with acute rest angina (>15 mins) and ST segment depression of >1 mm in any ECG lead. Twenty-nine pts had had a previous myocardial infarction. All pts received oral aspirin and IV heparin and were randomised to Group A (GpA) - alteplase (100mg) and Group B (GpB) - placebo, given over 3 hrs. Within 3 days of admission, coronary arteriography in 37/40 pts from each Gp showed similar significant coronary lesions (>50% luminal stenosis) - 8 pts with single, 14 with double and 14 with triple vessel disease in GpA compared with 7 pts with single, 13 with double and 13 with triple vessel disease in GpB. Five pts had no coronary stenoses (1 GpA, 4 GpB) and there were 4 left main stem stenoses (1 GpA, 3 GpB). The ischaemia related vessel was patent (TIMI 2 or 3) in 31/37 (84%) of pts in GpA and 29/37 (78%) in GpB. MI occurred in 26 pts in GpA (13 non-Q wave, 13 Q wave) and 24 pts in GpB (19 non-Q wave, 5 Q wave). Thirty pts did not develop MI. The left ventricular ejection fraction was 48% (range 17-69%) in GpA and 56% (range 31-81%) in GpB. Eight pts in each Gp required PTCA and 4 pts in GpA and 5 pts in GpB required CABG during hospitalisation. There were 7 deaths (4 in GpA and 3 in GpB). Thus for pts with UA (rest pain) receiving aspirin and heparin, alteplase did not significantly improve patency in the ischaemia related vessel. The requirement for PTCA and CABG was similar in both Gps.