11:45

IS PTCA OF ONE MAJOR CORONARY ARTERY WITH THE CONTRALATERAL VESSEL OCCLUDED SAFE AND EFFECTIVE ?

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Attempted angioplasty (PTCA) of either a dominant right (RCA) or left anterior descending (LAD) coronary artery when the contralateral vessel is occluded may trigger overwhelming LV dysfunction and hemodynamic collapse. We evaluated the risk vs benefit of PTCA of a critical stenosis (>70%) of RCA (n=52) or LAD (n=143) in 195 patients with the contralateral coronary artery occluded. Left ventricular function (LVF) was normal (38%), mildly (34%), moderately (22%), or severely (6%) compromised in the study group. Data collected included periprocedure angiography and clinical results as well as both immediate and follow-up clinical status, repeat PTCA, coronary artery bypass graft surgery (CABG), acute myocardial infarction (AMI), and death. Immediate and late outcome (33±18 months) of RCA PTCA and LAD PTCA was compared with 216 pts having PTCA of both the RCA and LAD and 195 pts who had coronary surgery (CABG), matched for lesion, LVF, age (mean, 60 years), sex (75% male) and study period. Results: In the study group there were 7 emergency (3.6%) and 10 elective CABG , 1 AMI (0.5%), and 1 death (0.5%) in the hospital. After discharge, there were 33 elective CABG, 9 AMI, and 9 deaths. There were not more deaths or AMI in the study group compared to the CABG or PTCA control groups, either periprocedure or after discharge. Emergency CABG was necessary more frequently with PTCA of RCA (9.6%) than LAD (1.4%) (p=.02). The study group had more elective CABG before (p=.02) and after discharge (p=.004) than the control PTCA group.
Conclusions: 1) Dilating the LAD when the RCA is occluded is as safe as CABG; 2) Dilating the RCA when the LAD is occluded leads to more emergency CABG than LAD PTCA with RCA occlusion; 3) CABG did not improve the survival rate or the occurrence of AMI more than PTCA, both in the hospital and during 3 year follow-up.

10:45

ASPIRIN USE BEFORE ONSET OF MYOCARDIAL INFARCTION FAILS TO REDUCE SHORT TERM MORTALITY.

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Clinical data of 4,049 participants in the Halifax County WHO MONICA project, consecutively admitted to CCU in 1984-86, were reviewed to determine the influence of acetylsalicylic acid (ASA) use before admission in patients diagnosed as definite myocardial infarction (MI) by MONICA criteria, on short term mortality. Definite MI was found in 1,072 of the patients. Their mean age was 59.9 years (±9.9), 73.8% were males, 51.9% had a history of coronary heart disease (CHD) and 9.6% were taking ASA at the time of admission. Within the 28-day follow-up period there were 13 deaths (12.6%) in the 103 patients taking ASA before admission, and 110 deaths (11.4%) in the 969 patients not on ASA before admission.

Multivariate logistic regression analysis was performed to test the influence of ASA use before and during the hospital admission, on 28 day mortality. We controlled for age, sex, CHD history, diabetes, chest pain symptoms (whether absent, atypical, or typical for MI), congestive heart failure on admission (CHF), CK enzyme rise, and stroke history. ASA use before presentation did not contribute independently in a positive or negative direction to 28 day mortality (95% confidence limits for odds ratio CL= 0.552, 3.225). ASA administered during the hospital phase was significantly associated with 28-day mortality reduction (CL= 0.28, 0.84).

Our results indicate that over 90% of this study group with a high prior CHD prevalence did not take aspirin before CCU admission for MI and that pre-admission use of ASA did not influence short term mortality. The observed beneficial effect of ASA in the acute MI setting is consistent with previous studies.

Tuesday, March 5, 1991
10:30AM–12:00NOON, Room 214, East Concourse
Acute Myocardial Infarction: Therapies

10:30

SUSTAINED EFFECT OF CAPTOPRIL ON THE ATTENUATION OF INFARCT EXPANSION FOLLOWING ACUTE MYOCARDIAL INFARCTION.

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In a double blind study 99 patients (82 male, aged 40-75) were randomly assigned to receive captopril/placebo within 24 hrs of acute myocardial infarction and continue treatment for 1 year. There were no differences in clinical and echocardiographic variables between the groups at baseline except for an excess of previous MI in the placebo group 13/50 v 2/49, p=0.002. After 12 months treatment LV end-diastolic volume index (EDVI) had increased 8.4(1.9)ml/m^2 in the captopril group vs 19.0(2.6) ml/m^2 with placebo, p=0.002. LV end-systolic volume index (ESVI) had increased by 5.4(1.3)ml/m^2 in the captopril group vs 14.7(2.3)ml/m^2 with placebo, p=0.001. The resulting mean volume indices were significantly lower in the captopril group: EDVI 76.9(3.0)ml/m^2 vs 89.5(3.1)ml/m^2, p=0.005, ESVI 47.0(3.8)ml/m^2 vs 59.9(3.4)ml/m^2, p=0.005, and LV ejection fraction was higher in the captopril group: 40.0(1.6)% vs 34.0(1.6)%, p=0.01. In the sub-group with anterior MI, anterior segment length (ASL) increased by 4(3)mm in the captopril group vs 17(4)mm with placebo, p=0.009, resulting in a significantly shorter mean ASL with captopril: 91(5)mm vs 107(4)mm, p=0.002. In the sub-group with infero-posterior MI, the PSL increased by 4(2)mm in the captopril group vs 13(3)mm with placebo, p=0.03. These changes indicate that captopril limits infarct expansion and LV dilatation for at least up to 1 year after MI.

11:00

PILOT STUDY OF COMBINED ADMINISTRATION OF RIDOGREL AND rt-PA IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION (AMI)

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The tolerance, safety and pharmacodynamics of a combined treatment of 100 mg rt-PA with 50 mg (group I, n=3), 150 mg (group II, n=3), 300 mg (group III, n=8) and 450 mg (group IV, n=3) of Ridogrel (R), a thromboxane ($Tx)A_2$ synthase inhibitor/prostaglandin endoperoxide receptor antagonist, were studied and compared to 100 mg rt-PA + 1,000 IU/h heparin (group V, n=8) in patients with AMI. No major bleeding occurred despite bleeding times > 9 min in 9/17 R-treated patients. Coronary patency at 5-10 days was obtained in 14/14 R-treated patients. Serum TxB_2 levels decreased to 1% and 3% of baseline ($p < 0.001$) 45 min and 12 hours after R. 6-keto-$PGF_{1\alpha}$ and PGF_2 levels increased 3-fold ($p < 0.001$) and PGE_2 levels 25-fold ($p < 0.001$) in R-treated patients (groups I-IV), suggesting a reorientation of the prostaglandin synthesis towards endothelial antiaggregatory prostaglandins. The C_{50} of the synthetic endoperoxide analogue U46619 for inducing ex vivo platelet aggregation increased from 0.5 µM at baseline to 4.0 µM with 150-450 mg R (groups II-IV).

We conclude that R is sufficiently safe and pharmacologically effective to justify further clinical investigation of its potential as an adjuvant treatment to accelerate and sustain coronary artery thrombolysis with rt-PA in patients with AMI.