TCTAP A-139
Endothelial Dysfunction and Inflammation: Band Master of the Orchestra of Slow Coronary Flow Phenomenon
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Background: Slow flow of contrast without significant lesion in coronary arteries can manifest as life threatening arrhythmia and sudden death. But, its responsible mechanism is still unknown. We here want to find the most influential mechanism of slow coronary flow by analyzing previously proposed theories in a single randomized controlled study.

Methods: Among the approximately 3200 Acute Coronary Syndrome or Unstable Angina patients who had consecutively done coronary angiogram from 15th October, 2012 to 15th April, 2013, 100 patients (58 ± 10 years; 45 male) with neither slow coronary flow nor significant lesion were randomized as Control group and another 100 patients (57.1 ± 10 years; 81 male) were also randomized from 212 patients who were found as slow coronary flow with normal angiographic finding as Slow Coronary Flow (SCF) group. Tortuosity status (whenever there was less than 125 degree angled three or more turns in slow coronary flow affected artery or two or more normal coronary arteries), demographic parameters and investigations including cardiac enzymes, lipid profiles, platelet and plaque parameters, Viscosity parameters, Echocardiographic parameters, Inflammatory parameters and Uric Acid were recorded and compared within two groups.

Results: Patient gender, High Density Lipoprotein, Hemoglobin, Hematocrit and Uric Acid had strongest strength of correlation (all r=0.00 and p=0.31) with SCF group while Tortuosity was weakly correlated with SCF group (p=0.002, r=-0.213). Moreover, only Uric Acid (p=0.000, CI=2.244-13.642) and Tortuosity (p=0.000, CI=2.264-14.142) were left as top independent determinant factors for Slow Coronary Flow after multivariable logistic regression. After ROC curve analysis Uric Acid got the highest Area Under Curve (AUC=0.720) with the best cut off point of 286 μmol/L. Sensitivity=69%, Specificity=69% followed by Tortuosity. Uric Acid was further correlated with not only mean TIMI frame count (p=0.000, r=0.291) but also number of affected artery (p=0.000, r=-0.268).

Conclusion: Uric Acid was found as the most determinant factor of slow coronary flow followed by Tortuosity in our study. Uric Acid also plays a role in both inflammation and endothelial dysfunction. Therefore, Inflammation and Endothelial Dysfunction may be the most influential mechanism of slow coronary flow phenomenon.

Figure 1. Study Design Flow Chart

Figure 2. Possible Mechanism of Slow Coronary Flow Phenomenon Based On Endothelial Dysfunction And Inflammation

TCTAP A-140
Manual Vacuum Thrombectomy (MVT) Combined with Endovascular Angioplasty of the Infarct-related Coronary Artery (IRA) for the Management of STEMI Patients After Pre-hospital Systemic Thrombolytic Therapy (TLT)
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Background: The study of effectiveness and safety of manual vacuum thrombectomy (MVT) from the IRA in STEMI patients after pre-hospital systemic TLT.

Methods: 242 patients with STEMI after pre-hospital TLT with signs of thrombus (TCTAP A-140 to TCTAP A-142) were divided into 2 groups: in Group (n=121) standard percutaneous coronary interventions (PCI) were complemented by MVT; in Group 2 (n=121) only standard PCI were performed. Baseline clinical, history and coronary angiographic parameters were similar for both groups. The average time interval between the onset of angina pain and the PCI was 5.8 hours. Vast majority of patients from both groups (>75%) had blood flow in the IRA TIMI-3, with MBG >3 in 61.9% and 48.7% in both groups, respectively. LV EF was 48±6.5% in Gr. 1 and 49±7.5% in Gr. 2 (p=0.15). Complete ST resolution was seen in 54.6% and 44.6% of cases, respectively. Hospital mortality was 0.8% and 1.6%, MACE occurred in 1.6% and 2.5% of cases, respectively (p=0.05).

Results: Mid-term (7.8 ± 1.2 months) mortality in Gr. 1 was 1.6%, and in Gr. 2 2.2% (p=0.05). No cases of IRA occlusion were seen. In-stent restenosis occurred in 14.8% of cases in Gr. 1 and in 20.6% in Gr. 2. Significant increase of LV EF was seen in both groups - 54.9 ± 5.5% and 54.3 ± 5.2%, respectively.

Conclusion: Vacuum thrombectomy is safe and does not increase total duration of myocardial ischemia and the rate of MACE and allows to evacuate thrombotic mass from the IRA. The combination of this method with interventional procedures permits to achieve fuller restoration of the blood flow in the microcirculatory bed of the IRA in STEMI patients after pre-hospital systemic TLT.

TCTAP A-141
Does Intracoronary Administration of Metabolic Cytoprotector Mexicor During Rescue PCI Limit Angioplasty of the Infarct-related Coronary Artery (IRA) for the Management of STEMI Patients After Pre-hospital Systemic Thrombolytic Therapy (TLT)

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Background: The study of cardioprotective action of intracoronary metabolic cytoprotector Mexicor (methylhexylpiridinol succinate), after recanalization of IRA in STEMI patients.

Methods: 253 patients (average age - 56 ± 7 years) with acute occlusion of the proximal or middle segment of the LAD and absent antegrade blood flow (TIMI 0) underwent successful recanalization of IRA within the first 6 hours after the onset of AMI. Prior to angiography, all patients were randomized into 2 groups. Patients from Group I (n=126) received intracoronary Mexicor (0.2 g). Patients from Group II (control, n=127) did not receive intracoronary cytoprotector. Baseline clinical, historical and angiographic data were not significantly different in the studied groups. Intracoronary administration of a special solution containing the diluted agent was performed through a special microcatheter during 10 min. simultaneously with myocardial reperfusion. Blood samples for markers of cardiomyocytes injury (Troponin I, myoglobin) were taken during recanalization of IRA, in 12 and 24 hours after the procedure. On day 10 of the disease all patients underwent control ventriculography, and also were recommended to have in-hospital control examination in 6 months.

Results: In-hospital course of the disease was rather uneventful, 1 patient (0.8%) died in Gr. I and 3 (2.3%) – in Gr. II. Average values of Troponin I at 12 hours after the procedure in Grs. I and II were 311 ± 47 and 632 ± 39 ng/ml, respectively (p=0.05). In the long-term after the procedure, in average – in 6.8 ± 0.7 months, the survival in Gr. I was 96.8%, in Gr. II - 87.6% (p=0.05). Baseline clinical indices in both groups were not significantly different. The increase of LV EF in Grs. I and II was 9.2 ± 5.1% and 4.1 ± 8.2%, respectively (p=0.05). We also noted a significantly better dynamics of contractility on infarct-related segments of the LV in Gr. I in comparison with Gr. II (p<0.05).

Conclusion: Our study suggests that intracoronary administration of metabolic cytoprotector Mexicor limits reperfusion injury of the myocardium and contributes to the preservation of structural and functional integrity of cardiomyocytes after antegrade blood flow restoration in IRA within the first hours after the onset of AMI.