Results: NSE levels significantly increased after coronary angiography and intervention compared to baseline levels (22.03±27.70, 10.08±3.15 consecutively). Baseline characteristics were similar between groups (Table-1). Left ventricular ejection fraction in the SNI+ group was significantly lower than SNI- group (43.71±12.51%, 50.84±9.34%, p=0.002). Maximal CK-MB, Troponin-I, Syntax Score (SS), of SNI+ group were significantly higher than SNI- group (103.83±99.22, 51.92±78.33, p=0.006; 50.04±66.18, 19.18±30.50, p=0.002; 103.83±99.22, 51.92±78.33, p=0.006 and 50.04±66.18, 19.18±30.50, p=0.002 successively). SS and performing PCI were the independent predictors of SNI (p=0.009, OR=1.06, 95%CI=1.014-1.107, p=0.036, OR=4.262, 95%CI=1.097-16.56).
Conclusions: PCI and coronary artery lesion complexity may increase the risk of SNI in patients with acute coronary syndrome.

Table-1

Variable	SNI+	SNI-	p value
Age, year	62.68±9.33	64.01±11.27	0.566
Male n(%)	26 (83.8%)	49 (73.1%)	0.243
Body Mass Index, kg/m2	28.06±4.03	28.01±4.85	0.515
Hypertension, n(%)	10 (32.3%)	25 (37.3%)	0.627
Diabetes, n(%)	10 (32.3%)	24 (35.8)	0.730
Hyperlipidemia, n(%)	9 (28.1%)	22 (32.8%)	0.782
Smoking, n(%)	11 (35.5%)	20 (29.9%9	0.752
Atrial fibrillation, n(%)	2 (6.5%)	8 (11.9%)	0.404
Mean Arterial Pressure, mmHg	100.71±13.80	98.26±12.14	0.374
Percutaneous Coronary Intervention, n(%)	21 (67.7%)	30 (44.8%)	0.034
SYNTAX score	28.13±14.68	18.71±13.23	0.002
Fluoroscopy Time, min	6.37±4.23	4.70±4.16	0.069
estimated Creatinine Clearance, ml/min	103.65±30.51	99.10±34.47	0.530
Glucose, mg/dl	127.71±60.40	133.07±67.80	0.707
LDL, mg/dl	141.32±42.59	135.19±47.74	0.733
Total Cholesterol, mg/dl	201.21±66.84	205.85±55.17	0.718
Hemoglobin, g/dl	14.42±1.59	13.97±1.70	0.210
Mean Platelet Volume, femtolitre	8.27±1.51	8.21±0.93	0.797
Red cell Distribution Width, %	13.55±0.91	13.36±1.42	0.498
CK-MB, ng/ml	103.83±99.22	51.92±78.33	0.006
Troponin-I, ng/ml	50.04±66.18	19.18±30.50	0.002
Left Atrial diameter, mm	38.29±4.09	38.51±5.85	0.853
Left Ventricular End Diastolic Diameter, mm	49.90±5.83	49.30±5.03	0.600
Left ventricular End Systolic Diameter, mm	36.10±5.76	33.39±6.00	0.037
Ejection Fraction, %	43.71±12.51	50.84±9.34	0.002
Asendan Aorta diameter, mm	34.71±4.11	34.57±4.20	0.875

OP-012

Treatment of Coronary No-Reflow With Intracoronary Vasodilators Added to Intravenous Tirofiban

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Objective: We prospectively assessed the management and short term prognosis of no-reflow phenomenon in a tertiary referral hospital.
Methods: We included 46 patients with ST-segment elevation acute myocardial infarction (STEMI) and occurrence of no-reflow phenomenon after primary percutaneous coronary intervention. They were all received intravenous tirofiban and then randomized into one of the 3 groups: intracoronary adenosine (n=16), intracoronary verapamil (n=15) or serum physiologic as placebo (n=15). Intracoronary drugs were administered after stent implantation. Thrombolysis In Myocardial Infarction (TIMI) frame counts were used to assess coronary flow.
Results: Groups were similar in terms of age, sex, STEMI localisation, diabetes, hypertension, mean reperfusion time. Hemotologic and biochemical blood parameters and procedural properties were also similar between groups (stent size and length, diameter and number of balloon(s) and stent(s) used). Verapamil plus tirofiban therapy had significant effect in restoring impaired coronary blood flow, decreasing TIMI frame count from 73±44 to 52±48 (p=0.024). However, adenosine and serum physiologic administration were not found to be so effective in decreasing TIMI frame count (from 81±35 to 71±46, p= 0.084; from 74±32 to 71±37, p=0.612; respectively). In-hospital and 6-month survival rates were similar among groups.
Conclusion: Tirofiban plus verapamil restored the impaired coronary blood flow more effectively than tirofiban plus adenosine and tirofiban only. Although both verapamil and adenosine have multiple effects on coronary circulation and even on platelet aggregation, verapamil as a calcium channel blocker, not only relieves microvascular spasm but also reduces calcium influx into ischemic cells, relieves cellular edema and restores calcium homeostasis. It may be hypothesized that these additional features put verapamil ahead of other drugs and may explain the better results we obtained with verapamil in comparison to adenosine.

OP-013

A Simple Index to Predict Adverse Clinical Outcome Associated with Acute Myocardial Infarction in Primary PCI Era

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Background: The major determinant of final infarct size for a given coronary occlusion is the size of myocardium that the artery perfuses. Defining the initial area-at-risk (AAR) for infarction has major clinical implications since it permits an accurate estimate of myocardial salvage provided by reperfusion therapies. We proposed a new index 'Relative Importance Index (RII)' to predict potential infarct size in patients with anterior MI.
Aim: The aim of the study is to assess the predictive role of RII for reduction in systolic function and its relation to adverse clinical outcomes.
Methods: One Hundred twenty-three acute anterior MI patients with their first acute coronary syndrome incident were consecutively and prospectively enrolled to the study. Patients with a clinical history of congestive heart failure, valvular heart disease, and previous coronary revascularization were excluded. All patients underwent primary percutaneous coronary intervention (PCI) for revascularization. Angiographic exclusion criteria were 1) pre-procedural TIMI flow ≥2 in the infarct related artery, 2) chronic total occlusion of other arterial territory 3) any visible collateral flow to infarct related artery 4) diffuse disease at proximal segments of coronary arteries that precludes defining reference segment. Coronary diameters were measured with quantitative coronary analysis program. RII was calculated by dividing culprit segment diameter to the sum of diameters of LAD, Cx, and RCA at their proximal segments (Figure 1). Troponin I (TnI) concentration at 72 hour was chosen as a serological estimate of infarct size. We evaluated 1-month follow up rates of major clinical endpoints (MCE), which is defined as death, non fatal MI, stroke, and new congestive heart failure. Left ventricular EF (LVEF) at 1st month was chosen as an index for systolic function.
Results: RII was significantly and negatively correlation LVEF (r=-0.65, p<0.001) (Figure 2). As RII of culprit lesion increased there was tendency to end up with lower EF. Likewise, RII was significantly correlated with 72 h TnI (r=0.48, p<0.001). Patients were dichotomized according to median value of RII (median RII=0.30) (Table 1). Supra-median RII was associated with lower EF and higher incidence of composite MACE. The mortality (12.9% vs. 6.6%), non-fatal MI (6.5% vs. 3.3%), and new CHF (12.9% vs. 3.3%) rate in supra-median RII group trend higher but they did not reach statistical significance. An RII >0.30 had a 88% sensitivity and 60% specificity (ROC area 0.82, p<0.001, CI [0.73-0.90]) for predicting severe LV dysfunction (LVEF <30%) (Figure 3).
Conclusion: A Simple index derived from coronary angiography at time of primary PCI can predict LV systolic function loss and adverse clinical outcome in patients with acute anterior myocardial infarction.