markers have been identified to be associated with increased incidence of cardiovascular complications and death in patients with ACS and increased severity of coronary artery lesion. Among these biomarkers some have emerged to be potentially useful in early risk assessment of patients with ACS, such as CRP, B type natriuretic peptide, troponin T/II, interleukin 6, and white blood cell (WBC) count and among these WBC count is the simplest. Although total leukocyte count is an established prognostic marker of ACS, there are limited studies for differential neutrophil count and prognosis in ACS.

Methods: 202 consecutive patients admitted with a diagnosis of ACS were evaluated by history and physical examination. Venous blood was drawn from all the patients at the time of admission and was analysed for CK-MB, Troponin T and Total leukocyte count and differential leukocyte count. The neutrophil counts were assigned into three categories, N1 (<70%), N2 (70-90%) and N3 (>90%). Coronary angiography was carried out within 24 hours of admission.

Results: The Neutrophil count ranged from 45% to 98%. The median neutrophil count was 78%; Smokers had a higher neutrophil count than non-smokers. Neutrophil count also tended to be higher in patients who died within 7 days (P<0.001). The development of new CHF or shock was associated with a higher Neutrophil count (P<0.001). Patients with a closed infarct-related artery on angiography (TIMI grade 0 or 1 flow) had a higher neutrophil count than did patients with an open artery (P<0.001). The presence of angiographically apparent thrombus was associated with a higher Neutrophil count than those without thrombus. (82%, n=28 versus 68%, n=72) (p<0.01)

Conclusion: The results of the present study confirm previous observations that relate elevated WBC count to adverse clinical outcomes in patients with ACS and further explore the pathophysiology that underlies this relationship. In addition to the worse clinical outcome, reduced patency and greater thrombus burden seen in patients with an elevated Neutrophil count, these patients had poorer downstream microvascular perfusion as assessed with TIMI perfusion grade. It is possible that this impaired myocardial perfusion reflects neutrophil–mediated endothelial dysfunction and microvascular plugging, as described in animal models of ischemia-reperfusion.

Single nucleotide polymorphisms associated with myocardial infarction in patients from Western India: A genome wide association study


Sir H. N. Medical Research Society, Sir H. N. Hospital and Research Centre, Mumbai, India

Background: Myocardial infarction (MI) is leading cause of death worldwide. Moreover, in India the average age of acute myocardial infarction (AMI) has reduced to 50 indicating it as a grave public health concern. The objective of our study was to identify genomic variants associated with AMI in patients from Western India in genome wide association study (GWAS) and validating the identified single nucleotide polymorphisms (SNPs), using high-throughput DNA microarray analysis.

Methods: Initially, 48 AMI patients and 48 controls were screened for SNPs using Illumina human CVD55K beadchip, containing approximately 50,000 human SNPs probes. The identified SNPs were then further validated by genotyping additional 188 patients and 196 controls using custom based Illumina’s Veracode Gold-enGate Genotyping Assay. PLINK software was used to perform statistical analysis.

Results: On normalization and filtration of the preliminary microarray data, 98 SNPs of 94 genes were identified associated with AMI (odds ratio range of 1.84-8.85, p value 0.0486 to 0.00334). Eight of these 98 SNPs reproduced association (p<0.05). The genes associated with some SNPs encoded for the proteins linked with blood coagulation, innate immunity, troponin complex and inflammatory pathways.

Conclusion: The study identified 8 SNPs associated with AMI which may increase the susceptibility towards the disease in patients from Western India.

T peak – T end interval: Marker for arrhythmic events at 30 days following ST elevation myocardial infarction

Subrangshu Dey, John Roshan Jacob, Bobby John

Department of Cardiology, Christian Medical College, Vellore, India

Objectives: We aimed to analyze the effect of reperfusion of infarct related artery on the TpTe interval determined on the surface 12 lead ECG. We also studied the association of Major adverse cardiac events (MACE) with repolarization abnormality on the ECG.

Methods: Patients with new onset STEMI treated with thrombolysis or primary/ rescue PCI were included. Digital ECGs at 50 mm/sec speed and 20 mm/mV gain filtered at 0.50–150Hz were taken before and after reperfusion therapy. TpTe interval was measured in leads with limited ST-segment deviation. All patients were followed up at 30 days.

Results: From June 2013 to December 2013, total of 216 patients were included of which 183 were males (85.1%). The mean age was 54.86 years (range 24–80 years). One hundred and thirteen patients underwent primary PCI (52.3%), 57 lysis (26.4%) and remaining 46(21.3%) rescue PCI. Thirty day Mortality was 5.1 % (11 patients). The pre TpTe interval was 84.50ms (IQR 80 - 100 ms) and the mean TpTe intervals reduced following intervention; primary PCI (74.93ms, p <0.001), thrombolysis (72.76, p<0.001) and rescue PCI groups (72.86 ms, p<0.004). Of the 216 patients, 210 were followed up at 30 days. Six patients were lost to follow up. Eleven patients died (5.1%) patients died. Pre TpTe interval more than 100 ms predicted (OR 13.21, 95% CI 1.16 – 150.57) increased risk of arrhythmias (Ventricular Tachycardia and Ventricular Fibrillation). However, it did not predict mortality at 30 days (OR < 1.4, 95% CI – 0.28 – 6.84). After adjusting for established risk factors, TpTe interval difference (pre – post) was found to be significantly associated with duration of chest pain and Killip class.

Conclusion: The TpTe interval was significantly reduced after reperfusion therapy. Pre-reperfusion TpTe predicted the risk of arrhythmias at 30 days. However, it did not predict subsequent all-cause mortality and heart failure at 30 days.