lesions can be addressed by this technique thereby reducing the cost, as a single stent is sufficient and also, the amount of metal at the bifurcation is reduced, thereby reducing the incidence of restenosis. To conclude TAP is a safe, easy, and cost effective technique that can be used to address most of the bifurcation lesions.

On treatment platelet reactivity in post percutaneous coronary intervention patients on thienopyridine drugs: A six-month outcomes study

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Background: In the present era of percutaneous coronary intervention (PCI) dual antiplatelet therapy plays a pivotal role. However there exists inter individual variability in response to antiplatelet therapy. Thus, monitoring antiplatelet therapy becomes vital and may identify poor responders who would benefit from the change in therapy.
Objectives: We sought to identify the efficacy of thienopyridine antiplatelet drugs in post PCI patients, by using AggreGuide A-100 Platelet Aggregometer, point of care device developed to monitor platelet aggregation using laser scattering technique.
Methods: This prospective, single-center study, includes patients who received thienopyridine antiplatelet drugs after PCI during May 2014–January 2015. In these patients, platelet aggregation inhibition was evaluated from the whole blood sample using AggreGuide A-100 and expressed in terms of platelet activity index – PAI. Patients were labelled as poor responders (high on treatment platelet reactivity) if the PAI was more than 5. Genetic polymorphisms of ABCB1 gene were studied in Clopidogrel group.
Results: A total of 405 patients were enrolled in this study. Among them 109 (26.9%) were female and 296 (83.1%) were males, 108 (26.7%) underwent primary PCI and 297 (83.7%) underwent elective PCI. At the discretion of treating physician 221 (54.6%), 111 (27.4%), and 73 (18%) patients received Clopidogrel, Prasugrel, and Ticagrelor, respectively. It is found that therapy was ineffective (PAI > 5) in 73 (16%) patients of which 52, 8, and 5 were in Clopidogrel, Prasugrel, and Ticagrelor groups, respectively. Cumulatively, there was high PAI in females and those who underwent primary PCI. In the Clopidogrel group those with additional Cilostazol were having low PAI ($p < 0.05$). Bleeding complications were observed in 3, 9, and 3 patients of Clopidogrel, Prasugrel, and Ticagrelor groups, respectively. Four patients in Clopidogrel group had reinfarction. Antiplatelet therapy was optimised in those with high PAI.
Conclusion: It is found that there is high resistance to antiplatelet drugs (especially Clopidogrel), and thus monitoring individual's platelet reactivity and optimising therapy based on it, should become a new standard-of-care for patients on antiplatelet therapy.

Electrocardiogram: A simple tool to predict angiographic localization of coronary lesions in STEMI patients

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Background: Rapid risk stratification of the patient with acute chest pain is essential to select the best management. Although culprit lesions in STEMI cluster in the proximal coronary arteries, their relationship to bifurcations and curvatures, where blood flow is disturbed, is unknown.
Objective: We investigated the value of the ECG at first medical contact to determine location and size of the ischemic myocardial area and thereby severity of risk. We also hypothesized that (a) culprit lesions are mostly localized to areas with disturbed flow, e.g. distal to bifurcations and curvatures and (b) the distribution of culprit lesions in the left coronary artery (LCA) and right coronary artery (RCA) and resulting infarct size are related to the location of bifurcations and curvatures.
Methods: In patients with ST elevation myocardial infarction (STEMI), ECG findings were correlated with the coronary angiogram. Using ST-segment deviation patterns the location of the coronary culprit lesion was predicted and also the size of the myocardium at risk. By quantitative coronary angiography, the distances from the vessel ostium, major bifurcations, and major curvatures to the culprit lesion were measured in 104 patients.
Results: Correct coronary culprit artery identification was possible in 96% of the patients and this was most accurate in localization of culprit lesion in the proximal part of the coronary arteries. Culprit lesions were located within 20 mm of a bifurcation in 77% of patients and closer to the bifurcation in the LCA compared with the RCA (10.13 ± 7.09 vs 18.49 ± 17.16 mm, $p = 0.01$). Of RCA culprit lesions, 53% were located within 30 mm of a major curvature. Compared with those in the RCA, culprit lesions in the LCA were located more proximally (19.28 ± 10.27 vs 28.77 ± 20.65 mm, $p < 0.01$) and were associated with larger myocardial infarctions as assessed by CK-MB (139.5 ± 139.4 vs 89.2 ± 93.9 unit/L, $p = 0.03$), and lower ejection fractions ($39.58 \pm 7.0\%$ vs $45.16 \pm 8.75\%$, $p = 0.001$).
Conclusions: Admission ECG gives adequate insight regarding the infarct related artery and the location of the lesion in the culprit vessel. In patients with STEMI, culprit lesions are frequently located immediately distal to bifurcations and in proximity to major curvatures where disturbed flow is known to occur. This supports the role of wall shear stress in the pathogenesis of STEMI

Serum uric acid as a marker of coronary artery disease – A single center study

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Background: Uric acid is the end product of nucleotide metabolism. Serum uric acid when elevated is diagnostic of gout and is a stress marker in hypertension. The correlation of serum uric acid with coronary artery disease has been evaluated though in small number cohort. Present study aims to correlate the serum uric acid with the severity of CAD and as a marker of CAD.
Materials and methods: A total of 470 patients consecutively have been enrolled into the study. The patients with already a diagnosis of gout, on long term use of thiazides and those responsible for elevated serum urate levels were excluded from the study. The patients enrolled underwent coronary angiography after informed and written consent. Based on the results of CAG the patients were divided into two groups, CAD ($n = 350$) and control group ($n = 120$). The severity of CAD was assessed by modified Gensini score.
Results: Mean age of presentation in CAD group was 53.72 ± 10.38 yrs and in controls was 49.60 ± 9.82 yrs ($p < 0.0001$). Males in total cohort were 331 (70.43%). Hypertensives 226 vs 82 ($p = 0.54$), diabetics (161 vs 30, $p = 0.60$), smokers (209 vs 48, $p = 0.64$),