them precludes them from the benefit of early revascularization.

Serum levels of the ST2 (IL-1 receptor family) to predict mortality and clinical outcome in acute myocardial infarction

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Background: ST2 gene is a member of IL-1 receptor family. ST2 can be in membrane receptor form, or soluble form (in serum). Normally whenever there is a cardiac insult, fibroblasts produce IL-33, which binds to ligand on cardiomyocytes, triggering cardio protective anti-apoptotic cascade. However soluble ST2 reduces activity of interleukin-33-dependent protective signaling axis by inhibitory binding to IL-33.
Hence increased serum ST2 levels may indirectly indicate increased cardiac myocyte apoptosis, in presence of an insult, and hence increased chances of death and heart failure (HF). Thus it can be used as a prognostic marker.
Aim: To assess the prognostic value of serum ST2 levels with respect to cardiovascular death and HF in patients presenting with acute MI.
Objective: Whether ST2 levels are correlating or not with 180 day mortality in patients with acute myocardial infarction.
Whether ST2 levels correlate with clinical heart failure at 30 days, and readmission within 180 days.
Methodology: The study was a non randomized observational prospective study which was carried out in patients attending the emergency department/CCU Amrita Institute of Medical Sciences, Kochi with STEMI.
Serum ST2 estimation (admission and at 48–72 h) along with echo, clinical data was recorded at admission. Patient was followed up at end of 30 days and 6 months. Admission ST2 levels were correlated with mortality, heart failure, and readmission.
Results: Total of 85 patients, with a mean age of 61.3 ± 11.3 years' with 78.6% males were studied for a median follow-up period of 10.7 months (300 days) during which 13 (15.5%) patients died. Mean ST2 at admission was 225.8 ± 50.7 in patients who died during the study period while it was 137.9 ± 93.4 for the rest of patients (p value < 0.01). It also showed significant correlation with onset of heart failure during follow up (198.2 ± 72.1 vs 131.3 ± 94). ST2 levels correlated inversely with ejection fraction measured at follow up, however No statistically significant correlation was observed between LA size, LVEDD, or diastolic dysfunction with ST2 levels.
Conclusion: Serum levels of ST2 are a reliable predictor of mortality and clinical outcome in patient's with acute MI.

Retrospective analysis of risk factors, angiographic characteristics in young patients with acute coronary syndrome and comparison with older patients with acute coronary syndrome

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Objectives: This study examined the coronary risk factors, angiographic characteristic in young (<40 years) patients with ACS compared with that in older patients (>60 years).
Methods: 192 patients with ACS aged less than 40 years and 200 patients with ACS aged more than 60 years were included in this study. Coronary risk factors and angiographic characteristics were evaluated in young ACS patients and compared with that in older patients.
Results: Smoking was prevalent in all age group. In this study there is no significant difference between young patients and older patients. Family history of premature coronary artery disease was more frequent in young patients. Hypertension and diabetes was more frequent in older patients. Higher body mass index (BMI) was more in younger patients. Serum total cholesterol (TC), triglycerides (TG), LDL-C were higher in young patients. Serum HDL-C was lower in young patients. Mean hsCRP value was 4.92 ± 1.68 mg/L in young patients compared to mean hsCRP value 4.39 ± 1.86 mg/L (p value < 0.0001). Young ACS patients often had angiographically normal coronary arteries, nonobstructive disease and single vessel disease than older patients (p value < 0.05).
Conclusion: Family history of CAD, higher BMI, higher TC, TG, LDL-C, higher hsCRP are more frequent with young ACS patients. Smoking as a risk factor was frequent in both groups. Angiographically normal coronary arteries, nonobstructive disease, single vessel disease are more frequent in younger patients.

Rapid progression of CAD in young

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Introduction: CAD is a major cause of death worldwide. The risk of CAD in Indians is 3–4 times higher than white Americans, 6 times higher than Chinese and 20 times higher than Japanese. Indians are prone as a community to CAD at much younger age. CAD is less frequent in adult younger than 40 years than in elderly adults but is of increasing clinical interest in young adults because of the potential of premature death and long term disability.
Case report: A 32 years old male patient presented to us with chest pain, sweating and ghabrahat of 2 h duration. He was smoker and alcoholic. He had positive family history of premature coronary artery disease. On examination pulse rate was 100 per minute, blood pressure was 140/80 mmHg, chest was clear and other physical examinations were normal. ECG was done which showed ST segment elevation in precordial leads. Diagnosis of evolving acute anterior wall myocardial infarction was made. 2D-echo showed no regional wall motion abnormality (RWMA) and normal left ventricular ejection fraction. He was thrombolysed with injection streptokinase. After thrombolysis ECG settled but post myocardial infarction angina persisted. So coronary angiography was done which showed plaque in proximal and mid left anterior descending artery (LAD). Medical management was planned for the patient. He was in regular follow up after discharge. During follow up period, he again had chest pain two times with no fresh ECG changes. TMT was done which was negative for reversible myocardial ischemia. His medications were boost up.
After a period of three months he again presented to us with severe chest pain and sweating. ECG showed no new changes and troponin I was negative. 2D-ECHO showed no RWMA and left ventricular function was normal. CAG was done which showed 90% stenosis in proximal to mid segment. PTCA + stenting was done. After angioplasty and stenting patient was pain free and stable.
Discussion: First coronary angiogram of this patient showed only plaque in proximal to mid LAD (reanalyzed LAD). Second coronary angiogram showed 90% stenosis in proximal to mid LAD. Second episode of ACS in this patient is likely due to rupture of vulnerable atherosclerotic plaque in proximal LAD coronary artery. He had