Alcoholics (155 vs 45, $p = 0.47$), and family h/o CAD (43 vs 14, $p = 0.13$). All the risk factors of CAD were equally matched between two groups thereby removing the confounding factors if any that may lead to biased results. The serum uric acid levels in cases were significantly higher than in control group (7.12 ± 2.04 vs 5.6 ± 1.02 mg/dl, $p < 0.0001$). The correlation of serum uric acid with CAD was positive ($r = 0.35$, $p < 0.0001$). A cut off point of 7.4 mg/dl has been made by ROC analysis for the presence or absence of CAD with sensitivity of 44.5% and specificity of 98.8%. The correlation of serum uric acid with severity of CAD was weak. The factors which were found to be statistically significant with univariate analysis were analysed by logistical regression for the independent predictors of CAD. Age, sex, diabetes, RDW CV, and serum uric acid were found to be independent predictors of CAD.

Conclusion: Serum uric acid can be used as a marker of CAD for its presence or absence but not with severity of CAD.

Role of low serum bilirubin as a marker of coronary artery disease – A single center study

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Background: Serum bilirubin is having protective effect, as an antioxidant with capacity to remove reactive species of oxygen. Studies have suggested that an increased bilirubin level promotes protection against atherosclerosis. We have evaluated the level of serum bilirubin in patients with coronary artery disease and its correlation with severity of lesion as assessed by modified Gensini score on angiography.

Methods: A total of 540 patients have been enrolled in the study. Patients with diagnosis of acute coronary syndrome, stable angina, history of typical angina, and Treadmill test positive were enrolled in the study. All patients were confirmed to have normal liver and renal function and were taken written consent for coronary angiography. The severity of lesion on angiography was assessed by modified Gensini score. Based on angiography results, the patients have been divided into CAD and control group.

Results: The number of patients in CAD group were 380 and in control group were 160. Mean age of presentation in control group was 53.60 ± 10.47 yrs compared to controls 49.56 ± 10.01 yrs ($p < 0.0001$). Males were 75.26% in CAD group and 55.62% in controls (p value is 0.1). The serum bilirubin was significantly lower in cases than in controls (0.66 ± 0.28 mg/dl vs 0.79 ± 0.30 mg/dl, $p < 0.0001$). There was a significant difference between other parameters assessed, i.e. RDW CV (14.46 ± 0.74 vs 13.72 ± 0.85, $p < 0.0001$), RDW SD (45.61 ± 4.42 vs 44.41 ± 5.68, $p = 0.0086$), serum uric acid (7.10 ± 2.06 mg/dl vs 5.96 ± 1.96 mg/dl, $p < 0.0001$). Univariate analysis have been done followed by multivariate logistic regression analysis for assessing the independent risk factors for CAD. Serum bilirubin, RDW CV, sex, age, and diabetes were found to be independent predictors of CAD (Table2). Serum bilirubin negatively correlated with severity of CAD ($r = -0.2121$, $p < 0.0001$). The cut off value of serum bilirubin based on Receiver Operating Characteristic curve (ROC) analysis was 0.7 mg/dl for presence or absence of CAD with sensitivity of 60.3% and specificity of 58.1%. There is no significant decrease between low serum bilirubin in smokers compared to non-smokers both in cases and control groups.

Conclusion: Serum bilirubin is negatively correlated with presence and severity of CAD.

Correlation of red cell distribution width with the severity of coronary artery disease – A single center study

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Introduction: Red cell distribution width (RDW) is a measure of the variability in the size of circulating erythrocytes, commonly utilized in the differential diagnosis of anemia. Increased RDW reported to be a marker associated with the presence and adverse outcomes of various cardiovascular diseases. RDW relation with the severity of CAD is limited by small sample size in previous studies.

Objectives: The aim of the present study is to prospectively evaluate the severity of coronary artery disease on angiography as assessed by Gensini score with RDW in patients admitted to Osmania General Hospital, Hyderabad.

Methods: A total of 576 consecutive patients who underwent coronary angiography after diagnosis of ACS (STEMI/NSTEMI), CSA or presence of angina like chest pain and/or positive treadmill test were enrolled in the study. Patients were divided into two groups based on the results of coronary angiography CAD group ($n = 438$) and control group ($n = 138$). The clinical information of the enrolled patients including the classical risk factors of CAD, RDW CV, and RDW SD were analyzed to identify their relationship to CAD. Severity of CAD was evaluated by Gensini score and its relationship to RDW was also analyzed.

Results: The mean age of presentation in CAD group was 53.64 ± 10.36 yrs whereas in controls was 49.4 ± 9.73 yrs ($p < 0.0001$). Male:female ratio in the overall study was 2.42:1. Patients with angiographic CAD had significantly elevated RDW CV levels compared with the controls [(14.59 ± 1.04)% vs (13.6 ± 0.68)%, $p < 0.0001$]. The RDW SD levels were also significantly elevated compared with the controls [(45.78 ± 4.76) vs (40.77 ± 3.01), $p < 0.0001$]. A significant positive correlation between RDW CV, RDW SD, and Gensini score was noted ($r = 0.32$, $p < 0.0001$) ($r = 0.43$, $p < 0.0001$), respectively. In multivariate logistic regression analysis, RDW was demonstrated to be an independent predictor for angiographic CAD (OR = 4.17, 95% CI 3.05–5.69, $p < 0.0001$). In receiver operating characteristic curve (ROC) analysis, an RDW value of 14.3% was identified as an effective cut off point in diagnosing CAD with a sensitivity of 58.9% and specificity of 84.8%. Among the classical CAD risk factors analyzed in CAD group, those with hypertension were $n = 287$, 65.5%, h/o smoking ($n = 243$, 55.47%), diabetes ($n = 189$, 43.15%), alcohol intake ($n = 189$, 43.15%), and family history noted in 61 (13.92%) patients.

Conclusions: RDW is an independent predictor of CAD and severity of coronary stenosis, suggesting that it might be a readily available marker for prediction and severity of CAD.

Delayed presentation of patients with acute myocardial infarction in CCU of Chittagong Medical College Hospital, Chittagong, Bangladesh

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Background: Delay between the onset of symptoms and hospital presentation is a critical factor in determining the management strategy and subsequent outcome.

Objective: To identify predictors of late presentation in patients with AMI and target interventions for those at high risk of late presentation.
Methods: In our cross-sectional study we prospectively analyzed a cohort of 1032 AMI patients for 1 year (from August, 2014 to May, 2015). Demographic factors, clinical characteristics, perception of health and access to health care were compared between early (within 12 h of symptom onset) and late presenters (>12 h of symptom onset). Bivariate comparison and multivariate logistic regression was done to identify independent predictors of late presentation. Odds ratio and 95% confidence intervals were calculated directly from the estimated regression coefficient.
Result: Our cohort was an average of 53 ± 10 years old. Of the total 1032 patients 384 (37.2%) were early presenters and 648 (62.8%) were late presenters. Average age of early presenters was 48.25 ± 12.71 and that of late presenter was 58.02 ± 13.76. Mean time interval between onset of symptom and presentation to hospital were 6.85 ± 8.06 h (range 1.5–12 h) in early and 37.88 ± 25.13 h (range 13–120 h) in late presenters respectively. Bivariate comparison found that having STEMI (late 64.2% vs. early 78.1%, $p = 0.02$) and ≥1 anginal episode over past 4 weeks (late 51.9% vs. early 33.3%, $p = 0.004$) were associated with early presentation. Multivariate analysis showed older age ≥65 years (OR 1.5 95% C.I. 1.3–1.8), living alone (OR 1.6 95% C.I. 1.4–1.7), traveling long distance ≥50 miles (OR 1.3 95% C.I. 1.1–1.6), reporting one or more anginal episodes over past 4 weeks (OR 1.3 95% C.I. 1.1–1.6) and misinterpreting chest pain as PUD (OR 3.5 95% C.I. 2.6–4.9) were associated with late presentation.
Conclusion: A significant majority patients with AMI were late presenters. Having a STEMI was independently associated with early presentation. Misinterpreting chest pain as of peptic origin was responsible for the delay in the majority. Reporting ≥2 anginal episodes in 24 h prior to hospital admission was also independently associated with late presentation. Patient education, appropriate utilization of existing resources and use of tele-electrocardiography that allows transmission of ECG signal to a medical control officer may decrease late presentation and improve outcome.

Risk factors and angiographic profile of young patients presenting with ST Elevation Myocardial Infarction

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Introduction: Acute coronary syndrome is a major health problem and accounts for a large proportion of the total number of hospitalizations all over the world. The incidence of coronary artery disease (CAD) in the young has been reported to be 12–16% in India. Contrary to other population, CAD in young Asian Indians has a poorer prognosis because of extensive atherosclerosis and multivessel disease.
Aims and objectives:

1. To assess the risk factors and angiographic profile of young patients ≤45 years of age presenting with ST Elevation Myocardial Infarction.
2. To find the association of risk factors with angiographic profile in these patients

Materials and methods: In this prospective observational study, 206 patients with diagnosis of ST Elevation Myocardial Infarction and age ≤45 years were included. Risk stratification was done as per conventional (male sex, family history of CAD, lifestyle, hyperlipidemia, hypertension, diabetes, tobacco in any use, and obesity) and novel Lipoprotein(a) (LPa) risk factors. Coronary angiography (CAG) was done in all patients. Correlation of these risk factors with severity of CAD was done.
Result: 94.2% of total patients were male and 5.8% were female.72.3% had active lifestyle and 27.7% had sedentary lifestyle. 3.9% had positive and 72.1% had negative family history of CAD. 53.4% had normal BMI, 35% were overweight, 7.3% were obese and 4.4% were underweight. 12.1% were diabetic, 11.61% were hypertensive, 13.6% were dyslipidemic, 67.5% were smoker/using tobacco in any form, 29.1% were alcoholic and, 35.4% had high LPa (≥30 mg/dl). 70% of the patients had Anterior wall ST Elevation Myocardial Infarction and Left Anterior Descending (LAD) artery was the culprit artery in majority (68%) of the patients. Out of 206 patients 77% patients had critical CAD and 23% patients had non-critical CAD on coronary angiography. In majority of the patients (around 54%) had single vessel disease (SVD), and double vessel disease and triple vessel disease was present in 12% and 11% of the patients respectively and 23% had non-critical CAD.
All female ($n = 12/206$) had critical CAD. This value was very significant ($p - 0.04$) from statistic point of view. None of the other risk factors were significantly affecting the severity of CAD. On univariate analysis patients with hypertension (SVD/DVD/TVD – 40%/20%/40%) and dyslipidemia (SVD/DVD/TVD – 44%/12%/4%) had significant triple vessel disease with significant p value of 0.001 and <0.001.none of the other risk factors had statistically significant difference in severity of lesion. On multivariate analysis none of these variables were positively affecting the CAD severity.
Conclusion: In patients with critical CAD mean age of the patient in our study was 39.64 years. The mean age of those with non-critical CAD was 37.87 years. While there is talk of increasing CAD in females, our study shows that in younger individuals presenting with acute ST elevation MI male predominance continues. Diabetes, dyslipidemia and, hypertension in our country is talked of a very important cause of CAD. However in our study of over 200 young patients DM, hyperlipidemia and, hypertension were present in very few patients. Among the various risk factors, however diabetes mellitus was very important risk factors. In our population LPa has been often highlighted as a novel risk factor, however our study shows that in most of patient of this younger cohort LPa was mostly normal. Additionally CAD was equally distributed in normal and high LPa group of patients. When presence of higher level of LPa was correlated with risk factors, only female sex was positively correlated to CAD. Smoking is an another risk factor for CAD. Majority of younger patients of myocardial infarction studied were smoker, however the severity of CAD was not correlated to the smoking status. In our study patients who were thrombolysed had less critical CAD as compared to those who were not thrombolysed possibly because the thrombolytic lysed the clot and the residual disease was non-significant disease. Criticality of CAD in our study was not correlated with left ventricular dysfunction and critical CAD was equally present in both those with LV dysfunction and normal LV function.159 out of 206 patients with 77% had critical CAD in our study. More than half of these patients had SVD. In terms of arterial involvement SVD was present in more than half of the patients and LAD was the commonest culprit artery.