Mean and standard deviation age was higher in Females (45.6 ± 15 years) versus Males (44.1 ± 14 years) $p = 0.005$. Age adjusted differences in Females versus Males were EPA (0.23 ± 0.23% vs. 0.26 ± 0.20%, $p = 0.0001$), DHA (3.54 ± 1.36 vs. 3.73 ± 1.37, $p = 0.0001$), Omega-3 index (3.76 + 1.44 vs. 3.99 + 1.47), $p = 0.0001$, AA (0.36 + 0.13 vs. 0.36 + 0.13, p = NS), and EPA/AA (0.70 ± 0.64 vs. 0.79 ± 0.66, $p = 0.0002$). 46.4% of females and 39.1% of males had a blood omega-3 index <3.5% ($p < 0.0002$), and 73.7% of females and 66.3% of males ($p < 0.0001$) had an EPA/AA <0.75 suggesting increased CVD risk which was evident significantly more in women compared to men.

Conclusion: A large proportion of the Saudi population may be at increased CVD risk due to low blood levels of omega-3 fatty acids and low EPA/AA blood level ratio. Females may be at higher risk than males. Future research will investigate the relationship of blood omega-3 blood levels and CHD incidence in the Saudi population.

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22. Joint effect of acute myocardial infarction on lipoprotein(a), C- reactive protein and markers of haemostasis

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Objectives: An issue of considerable interest is the relative contribution of each component of atherogenesis to CAD risk. An integrated approach encompassing the relationship between non traditional risk factors and coagulation assembly needs further studies.This study aimed at an integrated approach to study the joint changes in lipoprotein(a) [Lp(a)], high sensitivity C-reactive-protein (hsCRP) and haemostatic parameters in patients with acute myocardial infarction(AMI) and their correlation with clinical characteristics in these patients.

Methods: This study was conducted at the departments of physiology, emergency medicine and cardiology. Serial blood samples were collected from 50 patients with AMI on admission to the hospital (1st sample), after 3–4 days (post revascularization, 2nd sample) and at 2–3 months of follow up (3rd sample) and control group of 22 healthy subjects. The relationship of these markers were also studied with the presence and severity of CAD assessed by vessel and Gensini scoring. Blood samples were analyzed for lipids, Lp(a), hsCRP, fibrinogen, total(TFPI-T) & free(TFPI-F) tissue factor pathway inhibitor, plasminogen activator inhibitor-1 (PAI-1), and tissue-plasminogen-activator (t-PA).

Results: Lp(a), hsCRP, fibrinogen, PAI-1, TFPI-T and TFPI-F were significantly higher in AMI compared to control subjects. Lp(a) significantly increased in 2nd sample by 19.5% ($P < 0.05$) and 3rdsample (15.14%). hsCRP levels increased significantly by 82.6% ($P < 0.001$) in 2nd sample and dropped in 3rd sample (−39.4%). Among markers of hemostasis PAI-1 was the only marker which was significantly high in the first sample and dropped in successively by −37.8 & −57.2% respectively. Lp(a), hsCRP and TFPI-T were significantly related to Gensini scores.

Conclusions: Acute myocardial infarction affects Lp(a), hsCRP and hemostatic markers significantly. Lp(a), hsCRP and TFPI-T related significantly to CAD severity. While fibrinogen, PAI-1 and TFPI-F were related to the presence of CAD but there was no relationship with its severity.

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23. Does the maximum allowable contrast dose (MACD) predict the risk of contrast induced nephropathy (CIN) in patients with chronic kidney disease (CKD)

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Background: CIN is associated with high in-hospital mortality. Some studies recommend the utilization of the MACD formula to guide safe contrast dosing, however the evidence supporting use of this measure is limited.

Objective: The purpose of this study was to determine if MACD is helpful in predicting the risk of CIN in patients with CKD.

Methods: 8670 patients who underwent coronary angiography in our center with or without Percutaneous Coronary Intervention (PCI) (2008–2013) were included. Patients with CKD ($n = 144$) were selected. Patients in shock, on intra aortic balloon pump, on prophylactic hemofiltration or on dialysis were excluded.

Creatinine was measured 48–72 h post procedure. T-test, Chi-Square and multiple regression were used to compare those patients who developed CIN and those who did not develop CIN. CIN was defined as an increase in serum creatinine by $\geqslant$25% or 0.5mg/dL from baseline within 48–72 h after contrast exposure.

Results: CIN occurred in 28 patients (19.4%). Only 8 (5.6%) of the 144 patients exceeded MACD and 2 of these patients developed CIN. The use of biplane angiography explains the lower contrast dose. For this reason the impact of exceeding MACD could not be evaluated. Primary PCI was associated with CIN ($p = 0.012$; OR 5.1)).

Conclusion: Overall it is best to limit contrast dose to the extent possible as this is a known risk factor, however MACD is not a useful variable in a risk model for predicting CIN in our population. Primary PCI was the only predictor of CIN in our population.

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