Sir,

Markers of myocardial necrosis have gone through a long journey since 1950s. The cardiac biomarkers (CB) of past include total creatine kinase (CK) activity, aspartate aminotransferase activity (AST), lactate dehydrogenase (LDH) and LD1/LD2 ratio while the current CB are CK-MB, myoglobin, CKMB isoforms, cardiac troponin (cTn) I and T. The introduction of heart-type fatty–acid binding protein (H-FABP), high sensitive troponin (hs Troponin) and ST2 has led to a better prognosis, early and more precise diagnosis of myocyte injury and apoptosis.

High sensitive troponin scores over contemporary cTn assays in detection of cTn in healthy individuals and gives a more precise definition of what is normal (= 99 percentile). These titres can differentiate acute from chronic cardiac myocyte necrosis. cTnT and cTnI levels are not detected in healthy subjects and the 99th percentile is very low (eg-0.04 to 0.5 micrograms/L). Therefore, most assays were imprecise at this low level, so it was recommended to raise the definition of myocardial infarction (MI) at which a specific assay has a coefficient of variation of 10% or less. cTnI is less associated with false positives in population with chronic kidney disease (CKD) and therefore is more preferred CB than cTnT in such a clinical setting.

The heart type fatty acid binding protein (H-FABP) occurs in nine different isoforms and have a half life of several days. FABPs are transport proteins that carry lipophilic molecules like eicosanoids and retinoids, fatty acids across the membranes. It is released after myocardial death within 6 hours and is not cardiac specific like myoglobin. It can help in much earlier diagnosis as it can be detected earlier than myoglobin or troponin. It is released to smaller extend in mammary glands, skeletal muscle, some parts of brain, distal tubular cells of kidney and placenta.

Myoglobin is found in skeletal and cardiac muscle and can be used to pick up early acute MI. It rises 2-4 hours after beginning of infarction, peaks at 6-12 hours and normalises by 24-36 hours. Myoglobin is not very cardio specific and may require serial rapid myoglobin assay every 1-2 hours which can increase its specificity and sensitivity. Therefore, a rise of 25-40% over 1-2 hours strongly suggests an acute MI. Except for MI; myoglobin increases in renal failure, rhabdomyolysis, shock, vigorous exercise, open heart surgery and progressive muscular dystrophy. It does not increase in patients with non cardiac chest pain, cardiac catheterisation and mild to moderate exercise and in heart failure without myocardial infarction.

The ST2 cardiac biomarker measured by immunoassay is a member of interleukin 1 receptor family and has two isoforms which are soluble form (sST2) and membrane bound form (ST2 L). In patients with acute coronary syndrome (ACS), ST2 value of above 35 ng/ml have a three times higher risk of cardiovascular death and new heart failure at 30 days and at one year the relative risk of 2.3 for adverse outcomes. The results complement other biomarkers and are not influenced by age, gender, body mass index and impaired renal function.

In patients with low-intermediate likelihood of ACS, combination of markers of myocardial injury (cTnT or hs TnT) with markers of myocardial stress like N-terminal pro-B type natriuretic peptide (NT-proBNP) and newer mid-regional pro-atrial natriuretic peptide...
(MR-pro ANP) improves diagnostic accuracy and increases the negative predictive value (NPV) for excluding ACS. These have well been correlated with cardiac computed tomography (CT) and presence of regional wall motion abnormality (RWMA) on echocardiogram.3

References

