Role of salivary cardiac troponin I in acute myocardial infarction

Patients presenting in the emergency department (ED) with chest discomfort or other symptoms suggestive of acute myocardial infarction (AMI) or unstable angina are at priority for triage and risk stratification. Their treatment is on priority basis and is time bound to have favourable outcome. The goal of door-to-needle time for patient with AMI subjected to fibrinolysis is less than 30 min, while those subjected to primary percutaneous coronary intervention (PCI), the door-to-balloon time is less than 90 min. The therapeutic decision is mostly independent of serum cardiac biomarkers levels, as classical symptoms of chest discomfort and a ST segment elevation in ECG are sufficient to start specific therapy in most of cases. This is important as there is a crucial role of time to therapy in AMI management. Mirzaii-Dizgah and Riahi had estimated serum and salivary cardiac troponin-I (cTnI) at 12 and 24 h after AMI. Firstly, such a delayed estimation of cardiac biomarkers is of no therapeutic advantage, as primary PCI or fibrinolysis has to be started at the earliest in ED. A serial cardiac biomarker estimation following fibrinolytic therapy can support the patency of infarct related artery, if patient is not undergoing recommended coronary angiography within 24 h of fibrinolytic therapy. The details about reperfusion therapy received by 30 enrolled patients and coronary angiography findings, if performed are not documented in the present study. Therefore, it would not be useful to estimate these cardiac biomarkers at 12 and 24 h after AMI, as there is no therapeutic or prognostic advantage of these, especially when it is not estimated at baseline. Secondly, the salivary cTnI levels are quite low (in ng/l) in comparison to serum cTnI (in mg/l), and a correlation between the two is moderate, therefore, salivary cTnI cannot be as sensitive and specific as serum cTnI is, for the diagnosis of AMI. A similar value of stimulated salivary cTnI levels at 12 and 24 h following AMI and in healthy controls, rules out any practical utility of salivary cTnI in the management of AMI. Moreover, the normal range of salivary cTnI in healthy population is not known, hence it may not be of use in AMI management. Therefore, salivary cTnI measurement perhaps cannot be used as a substitute for serum cTnI, but can be an additive with serum cTnI for the diagnosis. The potential role of salivary cTnI as a point-of-care testing to detect AMI needs to be tested in further studies.

Rajesh Vijayvergiya
Department of Cardiology
Advanced Cardiac Centre
Postgraduate Institute of Medical Education & Research
Chandigarh 160 012, India
rajeshvijay999@hotmail.com

References
