Troponin 1 in acute myocardial infarction

To the Editor

We have read with interest the published article "Pilot study of Cardiac Troponin I (Tn1) in patients with acute myocardial infarction unstable angina" by Selim and Hamouda[1] in your issue of the Saudi Med J 2002; 23: 526-528. We would like to offer the following comments. 1) We were concerned by the authors' conclusion that "Cardiac Tn1 levels are not helpful in the initial management of patients with acute myocardial infarction". The role of Troponin is now well established in the diagnosis, management and prognosis of acute coronary syndrome. For the authors to come to their conclusion from a retrospective study of a very small number of patients with MI (13) is disturbing, and conveys a wrong message. Myocardial infarction (MI) is a prevalent disease in KSA. Many general and specialist hospitals admit up to 6-10 cases of MI daily. Hence it was a surprise to read a small retrospective review of medical records on 16 patients admitted over a 2-year period. The number of patients reviewed is too small for statistical analysis, which might have enhanced the study. 2) Moreover, according to the authors, the first positive result of Tn1 was available within a mean time of 16.66 ± 20.8 hours from admission. Blood should have been taken for the triaging of chest pain in the emergency room (ER) and not be delayed until the patient is admitted to the intensive care unit or cardiac care unit. We are aware of the importance of the overall clinical scenario, in the diagnosis of acute coronary syndrome. However, if one awaits the initial results of cardiac markers for that length of time to determine the diagnosis of acute coronary syndrome, it defeats the purpose of having the test, and becomes pointless, as the window of opportunity to commence thrombolytic therapy would have been missed. 3) Guidelines on diagnosis and management of MI are available in the literature such as by European Society of Cardiology and American College of Cardiology. The authors made no reference to the new Consensus Document of the Joint European of European Society of Cardiology/American College of Cardiology Committee for the Redefinition of Myocardial Infarction. The consensus was published before the date of submission of the authors' article.[2] As a matter of fact, 7 out of 9 references the authors cited were before 1994. The Consensus Document states that, "Myocardial infarction is diagnosed when blood levels of sensitive and specific biomarkers, such as cardiac troponin and the MB fraction of creatine kinase (CK-MB), are increased in the clinical setting of acute ischemia. The most recently described and preferred biomarker for myocardial damage is Cardiac Troponin (cTn1) (I or T), that has nearly absolute myocardial tissue specificity, as well as high sensitivity. If cTn1 assays are not available, the best alternative is CK-MB (measured by mass assay.' In clinical trials, as in clinical practice, measurement of cTn1 T or I is preferred over measurement of CK-MB, as well as total CK and other biomarkers, for the diagnosis of MI. It seems to us unscientific and unreasonable to contradict a guideline approved by the European Society of Cardiology and the American College of Cardiology by a pilot study on 16 patients in a very prevalent disease like MI. Reports of local experience are useful, but such reports, even 'pilot studies', have to be valid. 4) The authors use a Troponin level of >2 ng/l as being 'significantly positive' without indicating if its is first generation or 2nd immuno assay (which is more sensitive and specific) The guideline indicated that "an increased value for cTn1 should be defined as a measurement exceeding the 99th percentile of a reference control group. Reference values must be determined in each laboratory by studies using specific assays with appropriate quality control, while the currently available analyses demonstrates no threshold below which elevations of Troponins are harmless and without negative implications for prognosis. Thus, any other definition of MI would involve an arbitrary setting of limits for an abnormal troponin and would be open to criticism and considerable debate.[2] 5) The authors stated that half of the patients with MI do not exhibit electrocardiogram (ECG) changes. The fact is that about 50% of patients with MI do not exhibit segment in electrocardiogram elevation, but display other or nondiagnostic ECG changes.[3] 6) They affirm that serial determination of Tn1 is not cost effective. This is in contradiction to many other reports, which are much larger.[4] 7) There are many reports that have addressed the relationship between Tn1 concentration and severity of coronary artery disease.[5]

For the benefit of those managing chest pain, and for the sake of the patients we treat, we are submitting this letter "Troponin I in Acute Myocardial Infarction" for your consideration for publication in your Journal, as a comment to the above article. We urge the editor to refer to this comment wherever this article is cited, so as not to mislead general practitioners and residents in training, with regards to the role of Troponin in acute coronary syndrome.

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Reply from the Author

First we wish to thank Dr. Kinsara and Prof. George for their interest in the above titled paper, the items enumerated are noted. We would like to emphasize that our paper was a pilot study in a 330 beds tertiary

hospital in the North Eastern province of the Kingdom. Thus, only a few patients are included in the study (16 cases with confirmed acute myocardial infarction (AMI) out of 32 cases with coronary heart disease were admitted through ER over a period of 2.5 years.

However, this study highlighted the insufficient predictive value of cTn1 as a biomarker for the initial diagnosis of AMI. Of course, our results did not reach the level of statistical comments due to the limited number of cases. However, we wish that the conclusions drawn from this pilot study will serve as a starting point for others to plan a larger, more detailed, prospective, randomized and controlled studies correlating the cTn1 values with severity of the cardiac disease. We also hope that future studies can be carried out in a major referral or in a tertiary hospital. We shall be very interested to know about the results of similar studies in the future for the main objective of getting more input for better patients' management with AMI.

May I also add that this particular study had been accepted for presentation at the 2002 National Cardiovascular Health Conference in United States of America – proving that the study really draws the attention of other health care practitioners.

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