Defining myocardial infarction: not too late for common sense or consistency?

A recent consensus statement from a joint committee of the European Society of Cardiology and the American College of Cardiology (Joint European Society of Cardiology/American College of Cardiology Committee, 2000) has proposed a new biochemical definition for acute myocardial infarction. This definition is based on the measurement of cardiac troponins T and I rather than the widely used creatine kinase (CK) and the isoenzyme CK_MB. The implications of implementing this proposal require careful examination.

THE REDEFINITION

The greater sensitivity and specificity of cardiac troponins over CK and CK_MB are well established (Apple et al, 1997; Wu et al, 1999). The value of troponin estimation in risk stratification of acute coronary syndromes is also well documented (Hamm et al, 1992; Antman et al, 1996; Lindahl et al, 1996).

These advantages make the general use of troponin assays vital in the evaluation of patients presenting with chest pain thought to be of cardiac origin. However, the consensus document proposes that any elevation of troponin beyond the 99th centile of the reference range, occurring in the appropriate clinical context, should be defined as myocardial infarction. Thus patients presenting with an acute coronary syndrome with any troponin release, however small, should now be given a diagnosis of myocardial infarction.

This applies to interventions as well as naturally occurring events; troponin release following percutaneous coronary angioplasty should be recorded as myocardial infarction.

CONCERNS ABOUT THE NEW DEFINITION

A number of questions must be asked about this proposition. The first is whether this was indeed a consensus; not all collaborators at the consensus meeting would agree (Tunstall Pedoe, 2001). The second, setting aside for the moment whether it is desirable, is how is it planned to implement this very important diagnostic shift? It is not clear.

The implications for patients, epidemiologists, clinicians, and for public policy, are substantial. It is one matter to identify concerns in a consensus statement, it is quite another to leave resolution of these complex problems in the hands of individual clinicians.

THE IMPACT ON NUMBERS OF INFARCTIONS

The consensus statement recognizes that the proposed new definition of myocardial infarction with troponin will result in substantial increases in the apparent incidence of infarction. The scale of the increase is very substantial.

In the author's hospital the conventional biochemical confirmation of infarction is used: a CK value greater than twice the upper limit of the reference range applied in the appropriate clinical context. From an examination of 1 months' data using CK and troponin I (a single troponin I taken >12 hours after onset of symptoms, and several CK assays), the redefinition of infarction using the recommendations of the consensus statement would increase the number of infarctions by 150%. Even with a cut-off point of ten times the threshold level the number of infarctions would increase by 70% (JS Birkhead, data on file, 2001).

THE IMPACT ON PATIENTS

A diagnosis of myocardial infarction is a heavy burden, both in terms of employment and insurance. We have little or no control over the views or understanding of insurers or employers. Many employers are very anxious about employing someone with a previous history of myocardial infarction; should we increase their numbers by 150%? Altering the perceptions of patients and the general public to this diagnostic shift will be very difficult, and cannot happen quickly.

THE IMPACT ON LONGITUDINAL SURVEYS

Presently there is wide variation in which markers are used in the diagnosis of myocardial infarction. More than 90% of hospitals in England use CK, and about 60% CK_MB. Only 50% were using troponin assays in mid 2000, and some of these were near-patient tests of limited applicability (Birkhead, 2001). Nevertheless some hospitals have already moved to the sole use of troponin for the biochemical confirmation of infarction.

In some cases this has been encouraged by clinical biochemistry departments with tight budgets, although the cost of diagnostic biochemical investigations is minuscule in relation to the total inpatient cost of managing acute coronary syndromes. Even when troponin is the sole diagnostic assay there is a lack of diagnostic uniformity. Some colleagues have accepted the consensus definition as it stands, while others use a locally determined multiple of the threshold level to confirm infarction (personal communications, 2001). Meanwhile the majority of clinicians continue to use the conventional definition of infarction using elevation of CK greater that twice the upper limit of normal.

In those hospitals relying on troponin the incidence rates for myocardial infarction will rise and case fatality rates will fall when compared with those adhering to the conventional definition. National longitudinal mortality trends will soon become uninterpretable. The national myocardial infarction audit project (MINAP), which analyses the performance of hospitals on the audit targets set for the management of myocardial infarction in the *National Service Framework for Coronary Heart Disease* (Department of Health 2000), will be unable to provide comparable analyses of case fatality without diagnostic consistency.

Given that the potential impact of the consensus proposal is substantial, is the reasoning behind it sound? The consensus document is not persuasive:

'The change in definition of myocardial infarction seems reasonable, because it has been definitively shown that any amount of cardiac damage, as detected by cardiac troponins, implies a worsened long term outcome for the patient.'

This statement makes a strong case for the routine use of troponin to assess patients suspected of having an acute coronary syndrome, but makes no case for a change of diagnostic label from what might previously have been labelled unstable angina to one of myocardial infarction. There may be a satisfying tidiness to this reductive approach, but it is not helpful. This criticism is not just a semantic quibble. Insufficient thought has gone into the consequences for clinicians, epidemiologists or patients of applying a label of myocardial infarction in this way.

The consensus statement goes further than simply making a strong recommendation for the use of troponin. It also discourages the continuing use of CK for the routine diagnosis of infarction 'because of the wide tissue distribution of the enzyme'. However, the consensus document supports the use of CK for epidemiological or scientific purposes when combined with another marker such as troponin. The need for consistency of diagnostic criteria in longitudinal studies cannot be simply limited to a few research centres, but must continue wherever the outcome of myocardial infarction is examined.

The combination of CK and troponin provides allows a rational categorization of patients with acute coronary syndromes (without ST elevation) as proposed in a recent guideline (British Cardiac Society and Royal College of Physicians, 2001). In essence this guideline recommends that elevation of CK beyond twice the upper limit of the reference range with an elevated troponin should be considered as non Q wave infarction (or non ST elevation infarction), and elevation of troponin without CK elevation beyond twice the upper limit of the reference range as unstable angina. This allows a broad but nonetheless clinically useful distinction between patients likely to have plaque rupture, with distal microembolization (unstable angina) and occlusive coronary events (myocardial infarction). The implication is that CK should continue to be used in tandem with troponin wherever acute coronary syndromes are managed.

'When I use a word it means just what I choose it to mean, — neither more nor less', said Humpty Dumpty. Cardiologists must not adopt the same inconsistent approach to the definition of acute myocardial infarction. It is important for patients and clinicians alike that consistency is achieved. This should be allied to some common sense in the clinical definition of the term myocardial infarction. HM

KEY POINTS

- The consensus statement proposes a redefinition of myocardial infarction based on any release of cardiac troponin.
- The importance of universal use of troponin in the assessment of acute coronary syndromes is unquestioned; the need to redefine myocardial infarction on this basis is less clear.
- Introduction of this proposal will result in a substantial increase in the number of patients having a label of myocardial infarction, with considerable implications for employment and insurance prospects.
- The public health and epidemiological impacts of this proposal remain unresolved.
- Some clinicians have already abandoned use of creatine kinase in confirming myocardial infarction, so that there is no longer any diagnostic consistency for this important diagnosis. The implications for longitudinal and comparative outcome studies are substantial.
- The universal use of troponin in conjunction with creatine kinase offers a helpful compromise.

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Conflict of interest: Dr JS Birkhead is the clinical lead for the national audit of myocardial infarction (MINAP). The project is sponsored by the National Institute for Clinical Effectiveness.

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