Letters to the Editor

Sensitivity and Long-Term Prognostic Value of Cardiac Troponin I Increase Shortly after Percutaneous Transluminal Coronary Angioplasty

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To the Editor:

I was very interested in the article by Attali *et al.* recently published in *Clinical Cardiology*. In this article, the authors examine the role of cardiac troponin I (cTn-I) increase after coronary angioplasty (PTCA). In the introduction, the authors state that only one previous study investigated the value of cTn-I in this particular setting. The cited article is the one published by Hunt *et al.* in 1991.¹ In that particular study, the method employed for the detection of cTn-I had a much lower sensitivity compared with currently available immunoassay. In fact, the cut-off level was set at 6 ng/ml, compared with the presently used 0.3 ng/ml. Based on these methodologic assumptions, the authors concluded that myocardial damage does not occur after uncomplicated PTCA. We had a chance to collect an experience of our own utilizing the Stratus II fluorometric enzyme immunoassay (Dade Behring, Inc., Newark, Del., USA). In our patients, we compared CK MB mass, cardiac troponin T (cTn-T) and cTn-I after PTCA.²⁻⁴ The data allowed us to conclude that cTn-I is more sensitive than both CK-MB mass and cTn-T for the detection of periprocedural myocardial damage. Moreover, we observed that in patients who have evidence of myocardial injury, the absolute change in serum cTn-I correlates strongly with the absolute change in CK MB mass, suggesting that CK MB mass increase, although within the range of normality, bears similar information compared with that of cTn-I. One additional finding was that coronary stenting is associated with a significantly higher frequency of positive cTn-I compared with conventional balloon angioplasty.³ This latter finding, obtained in a small series of patients, was recently confirmed in a larger series by Shyu *et al.* using cTn-T.⁵

Thus, from all these data it appears reasonable to accept the concept that periprocedural minor myocardial damage is a biochemical entity with still ill-defined boundaries.

In particular, the prognostic value of increased troponins after PTCA remains unknown. In the study by Abdelmeguid *et al.*,⁶ 4,863 patients were examined in order to detect the negative prognostic value of increased CK MB after PTCA, while the study by Antmann *et al.*⁷ required as many as 1,404 patients with unstable coronary syndromes to show the negative prognostic value of a serum cTn-I > 0.3 ng/ml.

From these data, it appears clear that none of the presently available series concerning troponins and PTCA has the power to answer the following question: "Is minor myocardial damage after PTCA a marker for subsequent adverse coronary events?"

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Authors' reply:

We thank Dr. Luigi La Vecchia very much for his relevant and interesting comments about our paper recently published in *Clinical Cardiology* concerning the sensitivity and the long-term prognosis value of cardiac troponin I (cTn-I) increase after a successful coronary angioplasty (PTCA). We completely agree with his main messages. Indeed, we believe similarly that, first, the periprocedural minor myocardial damage is an entity with still ill-defined boundaries and that, second, its prognostic value is still unknown. The results observed in the study by Abdelmeguid *et al.*¹ regarding the negative prognostic value of CK MB after PTCA and those in the study by Antmann *et al.*² concerning the negative prognostic value of cTn-I in unstable angina favor the possible usefulness of cTn-I measurement in prognosis after PTCA. But we are not exactly in the same situation: in the former, the marker is different and in the latter, the disease is different. In any case, it is correct that our series does not have the power to answer the question of the

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