Microalbuminuria in the early phase of ST-elevation myocardial infarction: beyond the methodologic issue

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To the Editor. We thank Dr Berton *et al.* for their interesting comments [1] that give us the opportunity to further discuss intriguing features of the prognostic role of microalbuminuria in patients with ST-elevation myocardial infarction (STEMI).

First, we discuss the methodologic issues. In our paper [2], microalbuminuria was measured on the first day of admission (in the overnight urine collection) and was defined as ranging from 20 to 200 μg/min, as previously described [3]. Discrepancies between our investigation and previous ones can be related mainly to two factors. First, previous studies [4–7] were performed in heterogeneous populations of patients with myocardial infarction with and/or without hypertension, patients with and without diabetes, and those submitted either to thrombolysis or mechanical revascularization or not revascularized. Therefore, in-hospital mortality rate exhibited a wide range [from 4.5% (two) to 7.11% (four) and 7.2% (seven)]. Second, population selection criteria were different. We enrolled STEMI patients with a history of hypertension and without previously known diabetes who account for part of the STEMI population in the real-world scenario. The clinical significance of a biohumoral marker is known to be strictly linked (and has to be linked) to the patients' characteristics and admission diagnosis [8]. In fact, although microalbuminuria has been shown to be a useful tool to predict illness severity and outcome in critically ill adult patients admitted to an ICU [9,10], in 962 patients undergoing elective cardiothoracic surgery, preoperative microalbuminuria was not associated with most early adverse outcomes and information on preoperative microalbuminuria did not improve the accuracy of the additive EuroSCORE [11].

We agree with Dr Berton *et al.* that serial measurements of a biohumoral factor can add information on its clinical significance and pathophysiologic mechanisms. In fact, we recently investigated [12] the patterns of temporal variations of procalcitonin (PCT) throughout intensive cardiac care unit (ICCU) course in patients with cardiogenic shock following STEMI and documented a progressive reduction in PCT values only in patients who survived. However, when aiming at a early risk stratification, one measurement may be enough (i.e. admission glycemia) [13].

Finally, we discuss the statistical analysis. In our paper, at logistic regression analysis, nonsignificant variables among those inserted as candidates in the initial model were dropped by means of both backward and forward selection. Indeed, the backward method, using a probability less than 5% to insert an 'already present' variable in the logistic model and less than 10% to remove it [as default setting in SPSS (SPSS Inc., Chicago, Illinois, USA)] is less rigorous than the forward method, in which a 'new' variable can be inserted into the model only if it is related with outcome at a probability level less than 5%. Our intention was to ascertain whether a variable in the final backward model was related with ICCU death enough to remain in the final model also when the analysis was conducted in a more 'rigorous' way. Both methods were used with the same set of candidate variables, as indicated by the term 'stepwise'.

In conclusion, we agree with Dr Berton *et al.* that the bedside cardiologist should interpret data obtained in clinical studies with an 'acute mind'. However, this interpretation should not rely only on methodologic issues, but necessarily on the population selection criteria, treatments and end points (i.e. early vs. late mortality). Only results from studies with similar characteristics (methods, population studies, outcomes) should be compared.

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