Hyperglycemia and No-Reflow Phenomenon in Acute Myocardial Infarction

Dr. Iwakura and colleagues in a recent issue of the *Journal* (1) conclude that, from a retrospective analysis of 146 patients, hyperglycemia might be associated with impaired microvascular function after acute myocardial infarction. The investigators describe an independent relation between elevated blood glucose levels on admission and no-reflow phenomenon after primary angioplasty of the infarct-related artery. They also state that this relation was independent of HbA1c level or diabetes mellitus (DM), because no differences existed in HbA1c levels or in the incidence of DM in both the no-reflow group and the reflow group.

We have some considerations about these results. First, the investigators defined hyperglycemia by the optimal cutoff to differentiate the patients showing no reflow with a receiver-operating characteristic curve analysis. By using this cutoff in the same cohort they found a relation between hyperglycemic patients and no-reflow phenomenon. The researchers have generated an interesting hypothesis, but new studies in another patient population still have to prove the validity of this cutoff. Furthermore, we are interested in details with regard to the methods used to determine blood glucose and HbA1c levels. No information was reported as to whether the blood glucose level was measured with point-of-care laboratory measurements or whether their method has been validated in critically ill patients. Clinically relevant differences in blood glucose levels have been observed in patients with shock (2), and there may be an influence of acidosis and hematocrit values (3). Finally, the absolute differences of 0.3% in HbA1c and 13% in incidence of DM were not statistically related to the no-reflow phenomenon, but this may be due to the small number of patients. The investigators found that 45.3% of the patients with hyperglycemia were diabetic versus 9.9% in patients without hyperglycemia (p < 0.0001).

The hypothesis that acute hyperglycemia (i.e., hyperglycemia at admission) is associated with the no-reflow phenomenon is intriguing. We, however, hypothesize that this relation is not independent of chronic hyperglycemia (i.e., elevated HbA1c level and/or DM). Therefore, new clinical studies have to determine the effect of hyperglycemia on no-reflow and the impact of metabolic regulation.

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REFERENCES


REPLY

We thank Dr. Zijlstra and colleagues for their interest in our work and for their comments. We agree with them that further study is preferable to prove the validity of the present cutoff point of blood glucose for prediction of the no-reflow phenomenon. We measured blood glucose in whole arterial blood samples with reflectance glucometer at the bedside. The measurements of glucose in whole blood are predictably 10% to 15% less than those in plasma, and arterial blood glucose is higher than venous glucose. Although capillary blood glucose measurement could be inaccurate in patients with severe hypotension, whole-blood glucose measurement has been reported to be accurate even in critically ill patients (1) (also see Ref. 1 in Dr. van der Horst’s letter). Moreover, only 7 (4.8%) of 146 patients had blood pressure <100 mm Hg. Hematocrit might also affect blood glucose measurement, especially in the whole-blood sample. However, the effects of hematocrit could be important only at high hematocrit levels, such as >55% (2), and no patients in our study had such a high hematocrit. Thus, we believe that the effects of blood pressure or hematocrit on blood glucose measurement could be negligible in our study.

It is important to question whether chronic hyperglycemia also affects the development of the no-reflow phenomenon. In our previous report based on the 199 patients with anterior acute myocardial infarction, none of whom were enrolled in our recent study, we did not find that diabetes mellitus predicts the no-reflow phenomenon independently (3), as in our recent study. We also performed multivariate logistic analysis again and found that blood glucose level could be an independent predictor of the no-reflow phenomenon (p = 0.008, odds ratio [95% confidence interval] = 1.03 [1.01 to 1.06]) even among the 105 patients without diabetes. Thus, we concluded that acute hyperglycemia in itself is related with the no-reflow phenomenon independently from chronic hyperglycemia. Akbari et al. (4) also reported that acute hyperglycemia impairs vasoilation in microcirculation even in healthy subjects.

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doi:10.1016/S0735-1097(03)00473-X

REFERENCES