Significance of ST-segment Elevations in Posterior Chest Leads (V_7 to V_9) in Patients With Acute Inferior Myocardial Infarction

We read with much interest the article by S. Matetzky et al. (1). It is a valid study emphasizing the value of posterior chest lead (V_7 to V_9) in early identification of patients with larger inferior myocardial infarction (IMI) exhibiting more benefit from effective thrombolysis.

We also performed 16 lead ECGs (12 leads, V_7 V_8 V_9 and V_4 R) in a series of 66 first IMIs admitted within six hours of chest pain (2). Like Matetzky et al. we observed significantly lower radionuclide left ventricular ejection fraction, higher peak creatine kinase levels and more frequent 12 lead-ECG pattern of posterior wall extension when ST elevation was greater than 0.05 mV in lead V_9. Unlike the authors we did not observe any difference in the in-hospital clinical course. We explained the observation by the fact that right ventricular infarction (RVI) was significantly more frequent in our control group. In spite of the important role of RVI in IMI (3), the authors did not record V_4 R and they did not discuss the possible influence of RVI on the prognosis while right coronary artery was more frequently involved in their control group (63% and 90%, p < 0.003).

They also did not discuss the balance of other early 12 lead-ECG prognostic markers (4) among the groups, which could have influenced the results.

We believe that independent prognostic value of posterior chest leads (V_7 to V_9) in IMI has to be assessed in a multivariate analysis combining initial 16 lead ECG variables and clinical predictors of events before recommending its systematic use in IMI.

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References

Better Patency With SK Than With tPA?

We read with interest the comments of Peto and Collins. However, we must disagree with the interpretation that the results in 13 patients treated with SK at 6 h after pain onset are the sole basis for the main claims about the findings of our study. In fact, rather than focusing on small patient subsets, we chose to perform multivariate analysis of the probability of early patency as a logistic function of the agent used, the time to treatment and their potential interaction. Indeed, the results of this analysis on the whole cohort of 481 patients identified a negative effect of treatment on the patency probability for SK but not for accelerated tPA. A similar negative correlation between the time from symptom onset to treatment and the patency rate has been reported by other investigators with other “non-fibrin-specific agents” than streptokinase (1,2). Finally, our report was not intended nor presented as a comparison of the clinical benefit of streptokinase versus accelerated tPA, which has been the subject of intense discussion elsewhere (3), but rather as a mechanistic analysis.

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