

References

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Reply

We appreciate the comments made by Goodman with regard to our recent study and would like to clarify some points mentioned by him. Goodman suggests a discrepancy between our findings and those of Eisenberg et al. (1). However, there are a few important differences between the two studies.

Eisenberg et al. (1) classified the patients according to Q wave appearance (on admission, 0 to 3 h, 3 to 24 h and 24 h to discharge and patients with non-Q wave myocardial infarction) and showed the importance of the time of Q wave appearance after thrombolytic therapy. Matetzky et al. classified the patients according to eventual electrocardiographic (ECG) pattern at two time points: 24 h and before discharge and demonstrated Q wave regression in a substantial number of patients with a Q wave myocardial infarction early (24-h ECG) after thrombolytic therapy and late Q wave appearance in a few patients.

Although the proportion of patients with a non-Q wave myocardial infarction at the end of the first 24 h in the study of Eisenberg et al. (1) (16%) was similar to that in the study of Matetzky et al. (2) (20%), it was substantially smaller than that on the discharge ECG (28%) in the Matetzky et al. study, where the dynamic changes in the Q waves throughout the hospital period were manifested. Thus, the two studies looked at the same phenomenon but from a different standpoint.

Eisenberg et al. (1) did not compare, as did Matetzky et al. (2), patients with a Q wave and non-Q wave myocardial infarction, but grouped together patients with Q wave appearance within 3 h ("early group") and those with Q wave appearance beyond the first 3 h and with non-Q wave myocardial infarction ("delayed group") and compared these two groups.

Contrary to the remarks of Goodman with regard to the study of

Eisenberg et al. that "the early and 1-year prognosis among the non-Q and Q wave groups was similar," when the patients in the study of Eisenberg et al. are grouped on the basis of the presence or absence of Q waves, important differences existed, resembling the differences that Matetzky et al. had shown. Patients with a Q wave myocardial infarction compared with those with a non-Q wave myocardial infarction had higher peak creatine kinase levels (1,251 to 1,081 vs. 661), a higher incidence of heart failure (13% vs. 3%) and a higher in-hospital mortality rate.

Goodman points toward another potential discrepancy between the two studies—the rate of Q wave appearance after the first 24 h: 1.5% in the study of Eisenberg et al. versus 5% in the study of Matetzky et al. However, whereas Eisenberg et al. reported the rate of at least one new Q wave appearance, Matetzky et al. reported the rate of patients moving from non-Q wave to Q wave myocardial infarction, which is something different. Moreover, this discrepancy might represent a higher rate of late reocclusion among the patients of Matetzky et al., where more successful thrombolysis might be anticipated as a result of earlier thrombolytic therapy (within 4 vs. 6 h) and administration of recombinant tissue-type plasminogen activator to all patients compared with treatment with streptokinase in 27% of the patients in the study by Eisenberg et al.

In conclusion, the two studies are complementary rather than contradictory, and both add important information to the published data:

1. The timing of Q wave appearance after thrombolytic therapy carries important prognostic information independent of the later natural history of the Q waves.

2. In postthrombolytic patients, the dichotomization for Q and non-Q waves is still important for risk stratification and should be determined according to the discharge ECG.

We thank Goodman for commenting on our report and hope that we have clarified the points raised in his letter.

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