Prognostic Value of C-Reactive Protein in Unstable Angina

To the Editor:

We appreciate the work by Ferreirós et al1 in confirming the strong prognostic value of C-reactive protein (CRP) in unstable angina. However, we believe that some issues should be raised. First, the authors did not exclude patients with Braunwald type C unstable angina and they did not provide an explanation of this choice. Class C unstable angina has a strong prognostic role in prospective studies.2,3 Confirming this fact, in their Table 1, the time between prior acute myocardial infarction (AMI) and the occurrence of unstable angina was not specified, and the group of patients with raised CRP (>1.5 mg/dL) had a higher, yet not statistically significant, prevalence of prior AMI. In fact, the pathophysiological meaning of a raised CRP titer is quite different in a patient recovering from a recent AMI than in a patient with unstable angina without previous myocardial necrosis.

Moreover, in the Results and Discussion sections, the authors did not emphasize the prognostic value of CRP in refractory angina alone. Instead, several prospective studies considered, in the outcome analysis, either major cardiac events (AMI/death) alone4 or minor (recurrence of angina) and major cardiac events evaluated as different end points.5

Pierfrancesco Agostoni, MD
Niguarda Ca’ Granda Hospital
20162 Milan, Italy

Giuseppe G.L. Biondi-Zoccai, MD
S. Raffaele Scientific Institute
20132 Milan, Italy


Response

We thank Drs. Agostoni and Biondi-Zoccai for their comments regarding our study about the independent predictive value of elevated levels of C-reactive protein (CRP) in unstable angina.1 They raise the possibility that our study results could be explained by the inclusion of patients with Braunwald type C unstable angina. We agree that elevated CRP levels could be more difficult to interpret in this group of patients because of the increase in CRP secondary to the acute phase inflammatory reaction generated by recent myocardial infarction.

However, it is unlikely that this factor could have influenced our results, because only 3 patients in our study had a diagnosis of Braunwald type C unstable angina (2 in the derivation and 1 in the validation set). Moreover, the category “prior acute myocardial infarction” in Table 1 did not represent patients diagnosed with Braunwald type C unstable angina but only those with a history of an old (>2 months) myocardial infarction.

Regarding their concern about a separate analysis of the end points (major, death or acute myocardial infarction; minor, refractory angina), Table 3 shows the association between a CRP level >1.5 mg/dL and the 90-day rate of events. In this table, the relationship between the level of CRP, either at admission or discharge, and the 90-day rate of refractory angina can be easily deducted by subtracting the death/acute myocardial infarction rate from the death/acute myocardial infarction/refractory angina rate. In a Cox regression model, a predischarge CRP level >1.5 mg/dL remained the strongest independent marker for identifying those patients who would present with refractory or recurrent angina requiring hospitalization during the 90-day follow-up (hazard ratio, 6.1; 95% confidence interval, 3.2 to 10.8; P<0.001). The data from the last regression analysis were not shown in the original article because of space limitations.

Ernesto R. Ferreirós, MD
Carlos P. Boissonnet, MD
Rodolfo Pizarro, MD
Pablo F. García Merletti, MD
Gianni Corrado, MD
Arturo Cagide, MD
Oscar O. Bazzino, MD
Servicio de Cardiologia
Hospital Italiano de Buenos Aires
Buenos Aires, Argentina

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Pierfrancesco Agostoni and Giuseppe G.L Biondi-Zoccai

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