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Peripheral Blood Monocyte Subset Assessment in Non–ST-Segment Elevation Myocardial Infarction Is Required

We read with great interest both the research paper by Tsujioka et al. (1) and the editorial comment by Shantsila and Lip (2) outlining the role of monocytes in myocardial infarction (MI). We agree that understanding the role of different monocyte population subsets during acute coronary syndromes (ACS) might provide important clinical information; however, much more work is required, and there is a need to express some cautionary notes. An important limitation in the paper by Tsujioka et al. (1) and others referenced by Shantsila and Lip's editorial (2) in considering the kinetics of monocyte chemoattractant protein (MCP)-1 release (3) are the small sample sizes (n = 36 and n = 23 for MI, respectively). Thus, interpretation must be restrained. Our data from 216 patients with ACS do not support the association of the early release kinetics of MCP-1 (the ligand for chemokine [C-C motif] receptor 2 [CCR-2]). Thus, the idea that it is related to the "prompt up-regulation of CCR-2 expressing CD14+CD16- monocytes" might not be correct (2). In our larger ACS population, we found no time-dependent increase in MCP-1 measurement during the first 12 h after the onset of symptoms (4). In addition, Tsujioka et al. (1) present data indicating that there was an increase in the $CD14^{+}CD16^{+}$ subgroup (these cells lack CCR-2; refer to Figs. 3B and 4D in Tsujioka et al. [1]) in that at admission the levels were lower in the MI group as compared with the stable angina group, with subsequent peak levels in the MI group significantly higher (1). However, this cell subpopulation did not correlate with myocardial salvage (1). Moreover, the data are from patients with ST-segment myocardial infarction (STEMI), which tend to be larger infarctions. Data in a more diverse group, including those with smaller events diagnosed with cardiac troponin—which is recommended by the guidelines groups (5)—would be important to see whether differences exist between those groups (1).

If monocyte subgroups are important in non-STEMI, they might prove extremely useful in deciding on the timing of invasive intervention. Non-STEMI patients benefit from early intervention but not necessarily immediate intervention (6). Perhaps data related to monocyte activity and expression would help to differentiate those who might benefit immediately from those who can wait for some hours. Such considerations will require detailed information on the effects of invasive interventions on monocyte subgroups as well as whether serial monitoring is required for risk stratification. These are important issues, which when addressed will shed additional light on the role of monocytes and their laboratory use in risk stratification for patients presenting with ACS.

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Reply

We are grateful to Drs. Kavsak and Jaffe for their valuable comments and suggestions to our study (1). They pose a question regarding the kinetics of monocyte chemoattractant protein (MCP)-1 release. They found no time-dependent increase in MCP-1 measurement during the first 12 h after the onset of symptoms in 216 patients with acute coronary syndromes (2). On the contrary, Matsumori et al. (3) have