

243

MONOCYTE SUBPOPULATION COUNTS AND TNF ALPHA ASSOCIATIONS WITH CLINICAL OUTCOME POST ST ELEVATION MYOCARDIAL INFARCTION

A Ghattas,¹ E Shantsila,¹ A Devitt,² H Griffiths,² G Lip¹ ¹Centre for Cardiovascular Sciences University of B; ²Aston University

doi:10.1136/heartjnl-2013-304019.243

Background Monocytes are implicated in the initiation and progression of the atherosclerotic plaque contributing to its instability and rupture. Although peripheral monocytosis has been related to poor

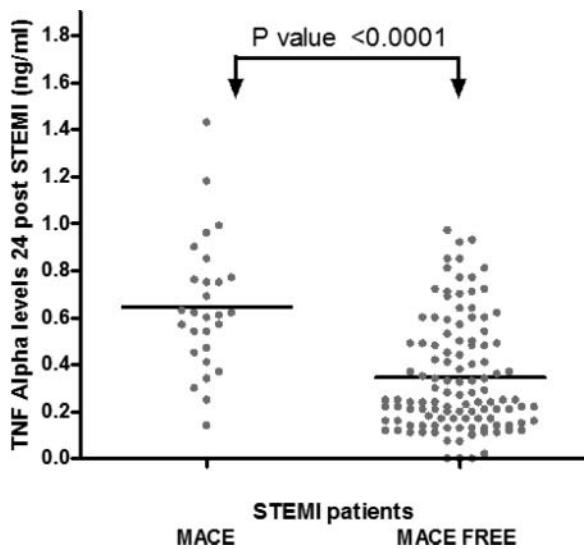


Figure 1 TNF α levels in STEMI patients with and without MACE.

clinical outcome post ST elevation myocardial infarction (STEMI), only scarce information is available of mechanisms of this association. Tumour necrosis factor alpha (TNF α) is a key cytokine in the acute phase inflammatory response, and it is predominantly produced by inflammatory macrophages. Little is known about TNF α association with circulating monocyte subpopulations post STEMI.

Method A total of 142 STEMI patients (mean age 62 ± 13 years; 72% male) treated with percutaneous revascularization were recruited with blood samples obtained within first 24 hours from the onset and on day 10-14. Peripheral blood monocyte subpopulations were enumerated and characterized using flow cytometry after staining for CD14, CD16 and CCR2 and were defined as: CD14++CD16-CCR2+ (Mon1), CD14++CD16+CCR2+ (Mon2) and CD14+CD16++CCR2- (Mon3) cells. Plasma levels of TNF α were measured by enzyme-linked immunosorbent assay (ELISA, Peptotec system, UK). Major adverse cardiac events (MACE), defined as recurrent STEMI, new diagnosis of heart failure and death were recorded at follow up, mean of 164 ± 134 days.

Results TNF α levels were significantly higher 24 hours post STEMI, compared to day 14 (paired t-test, $p < 0.001$) with day 1 levels weakly correlated with total monocyte count as well as Mon1 (Spearman's correlation, $r=0.19$, $p=0.02$ and $r=0.22$, $p=0.01$, respectively). There was no correlation between TNF α and Mon2 or Mon3 subpopulations. TNF α levels were significantly higher in patients with a recorded MACE ($n=28$, Mann-Whitney test, $p < 0.001$) (figure 1).

Conclusion In acute STEMI concentration of TNF α correlate with Mon1 subset count only and are higher in patients with MACEs. These data support the role of the 'classical' Mon1 subset in the inflammatory shift and negative outcome in STEMI. This also sheds light on the function of the newly described Mon2, which appears not to have an inflammatory role.