

## Whether inflammatory disorders matter for long-term prognosis after myocardial infarction?

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**Introduction:** In the acute period of myocardial infarction (MI) inflammatory disorders of blood are registered, but data of prognostic significance of these disorders is multiple-valued.

**Purpose:** Research of manifestation of the inflammatory response in patients who suffered MI, and estimation of its prognostic significance.

**Materials and methods:** 772 patients with myocardial infarction were examined. Prospective follow-up from 1 to 7 years was performed.

**Results:** The death rate during all years of follow-up was 14,2 % of all included in the study. 61% of patients died suddenly, but 26% of them died as a consequence of the progression of chronic cardiac insufficiency. In patients died suddenly lower level of lymphocytes in the first 24 hours:  $1,30 \pm 0,47 * 10^9/l$  vs  $1,80 \pm 0,73 * 10^9/l$ , ( $p = 0,03$ ) was registered. In patients who died due to heart failure progression, authentically registered higher leukocytosis in the first 24 hours, in comparison to survived ones, reached  $13,83 \pm 6,00 * 10^9/l$  (vs  $11,9 \pm 3,12 * 10^9/l$ ;  $p = 0,005$ ), but to 5th day in the compared groups leukocytes levels had practically the same values ( $7,36 \pm 1,89 * 10^9/l$  vs  $7,47 \pm 1,99 * 10^9/l$ ; accordingly  $p = 0,8$ ). In the compared groups the number of lymphocytes, expressing CD 95 did not differ authentically, but in died abruptly patients this index was rather lower.

There were not authentic differences detected among groups of dead and survived patients, in terms of interleukins 1 $\beta$ , 2, and 6, whereas TNF- $\alpha$  was almost twice as high in patients who died due to CHF progression. Studying inflammatory markers were not included in the number of independent indexes, connected with the risk of death when conducting multivariate regressive Cox-analysis.

**Conclusion:** In our opinion, inflammatory factors were displaced from the prognostic model for assessing the risk of death, both sudden and due to heart progression, by more powerful structural-functional predictors.