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Decreased glutathione levels in acute myocardial infarction.

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Abstract

Although experimental studies have demonstrated that reduced glutathione (GSH) is involved in cellular protection from deleterious effects of oxygen free radicals (OFRs) in ischemia and reperfusion, there are controversial data on the correlation between the levels of erythrocyte GSH and the ischemic process. To clarify, we determined the erythrocyte GSH levels in 21 patients with acute myocardial infarction (AMI), aged 39-70, who were not given thrombolytic therapy and 21 age- and sex- matched healthy controls. Samples of blood were taken on days 1, 3, 5 and 7 from AMI patients and on the same days from the controls. The GSH levels of patients with AMI were significantly depressed by 11.5% as compared to the controls on the second day after infarction (7.44 +/- 1.71 vs 8.41 +/- 1.54 U/gHb $p < 0.05$). Although the total mean of GSH levels for all days was lower (3.8%) in patients than in the controls, this finding did not reach statistical significance (7.41 +/- 1.71 vs 7.71 +/- 1.27 U/gHb, ns). There was no correlation between the erythrocyte GSH levels and cardiac enzyme concentrations, infarct localization, hemodynamic status according to Killip classification and the frequency of ventricular arrhythmias. This preliminary work suggests that depressed GSH levels may be associated with an enhanced protective mechanism to oxidative stress in AMI. Measurements of erythrocyte GSH can be helpful in the estimation of oxidative stress in the course of AMI. However, further research must be done to determine the primary scavenger in AMI by analyzing all the enzymes and substrates involved in the endogeneous system that controls the effects of OFRs.

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