

Increased erythrocyte glutathione peroxidase activity and serum tumor necrosis factor-alpha in HIV-infected patients: relationship to on-going prothrombotic state.

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A condition of oxidative stress, due to perturbation of oxidant/antioxidant balance, has been suggested to play a role not only in the pathogenesis of human immunodeficiency virus (HIV) infection, but also in the promotion of a thrombophilic condition. Because various hemostatic dysfunctions usually considered as risk factors for thrombotic events were reported in HIV infection, this study was undertaken to investigate whether the oxidative phenomenon could promote a prothrombotic state in such condition.

Erythrocyte glutathione peroxidase (GSH-Px), the major free-radical scavenger enzyme, and serum tumor necrosis factor-alpha (TNF-alpha) were evaluated in 33 consecutive HIV-infected out-patients and 35 matched HIV-negative healthy controls at a distance of any acute episode. Thrombin generation was explored by measuring the plasma levels of prothrombin fragment 1 + 2 (F1 + 2), whereas fibrin degradation products (D-dimer) and plasminogen activator inhibitor (PAI-1) activity were evaluated as indices of plasmin activity and fibrinolytic derangement. The anticoagulant pathway was investigated by measuring the plasma levels of antithrombin and protein C. Erythrocyte GSH-Px activity and serum TNF-alpha were significantly higher in HIV-infected patients when compared to controls. F1 + 2, D-dimer, and PAI-1 activity were increased in HIV-infected patients by comparison with controls. Normal antithrombin, but decreased protein C, was instead detected in HIV-infected patients. In the latter patients, serum TNF-alpha negatively correlated with both erythrocyte GSH-Px activity and plasma D-dimer. On the other hand, a positive correlation was shown between F1 + 2 and D-dimer and between D-dimer and GSH-Px activity. Furthermore, a trend toward increasing levels of GSH-Px with increasing PAI-1 activity was reported. These findings suggest a relationship between erythrocyte oxidative stress and the hypercoagulable condition during HIV infection.

