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Oxidative status of red blood cells, neutrophils, and platelets in paroxysmal nocturnal hemoglobinuria.

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Abstract

OBJECTIVE: Paroxysmal nocturnal hemoglobinuria (PNH) is an acquired stem-cell disorder associated with intravascular hemolysis and thrombosis. Hemolysis is caused by the hypersensitivity of PNH-red blood cells (RBC) to complement-mediated lysis due to deficiency in the surface glycosyl phosphatidylinositol-anchored antigens, CD55 and CD59. Thrombosis may be related to the platelet tendency to undergo hyperactivation. We previously suggested that hemolysis and thrombosis in other hemolytic anemias are related to oxidative stress. In the present study, we assessed the oxidative status of blood cells in PNH and tested the potential protective effects of antioxidants.

MATERIALS AND METHODS: Blood samples were obtained from 11 PNH patients and 11 normal control donors. Flow cytometry was used to measure oxidative stress markers in conjunction with the PNH immunophenotype.

RESULTS: Results indicated that abnormal, CD55/CD59-negative, RBC, neutrophils, and platelets are under oxidative stress. Their intracellular reactive oxygen species, membrane lipid peroxides, and external phosphatidylserine were higher and their reduced glutathione was lower than CD55/CD59-positive cells of the same patient or cells of normal controls. PNH-RBC were hypersensitive to an oxidative insult (e.g., hydrogen peroxide) and their oxidative status increased following interaction with complement, prior to hemolysis. Antioxidants reduced this hemolysis as well as activation of PNH platelets.

CONCLUSION: We propose that oxidative stress mediates the symptoms of PNH and suggest that antioxidants might be considered as a therapeutic modality.

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