
Erythrocytic phosphatidyserine exposure and hemostatic alterations in β-thalassemia intermediate patients.

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Abstract

INTRODUCTION: Hypercoagulable state is one of the common findings in beta-thalassemia intermedia (β-TI), particularly in splenectomized patients, with infrequent blood transfusion. Abnormality of the red blood cells (RBC) membrane due to oxidative damage is suggestive of possible etiologies. Membrane lipid peroxidation increases the exposure of phosphatidyserine (PS) that plays a role in the activation of coagulation factors V and X, subsequently initiating thrombosis. Our aim of this study was to find the probable correlation of the alteration of the PS on the RBC outer membrane with the hypercoagulable state in the β-TI patients.

MATERIALS AND METHODS: Our cross-sectional study was conducted on 39 splenectomized β-TI patients and 38 age-matched healthy controls. The mean age was 37 years. Analysis of the PS exposure on the RBCs was performed by fluorescein isothiocyanate (FITC) conjugated AV protein. Measurement of the coagulation factors V, X, and antithrombin III (AT-III) was performed. We also checked the D-dimer levels. Analysis was performed by SPSS16.

RESULTS: Fluorescence of FITC-Annexin V labeling on patients RBCs were higher than healthy controls (2.8 ± 2.2%) of the patients versus (0.4 ± 0.18%) in the control group and was statistically significant (P < 0.05). Mean levels of factor X and AT-III of the patients as compared with the control group decreased and showed significant difference (P < 0.05).

CONCLUSIONS: Circulation of thalassemic RBCs which abnormally possess PS on RBC membrane outer surface, suggests the possibility of the gradual consumption of the coagulation factors in the presence of a chronic coagulability state.

KEYWORDS: Antithrombin III; D-dimer; Erythrocytic phosphatidyserine; Factor V; Factor X; Hypercoagulability state; Thalassemia intermedia

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