CHANGES IN ERYTHROCYTE DEFORMABILITY IN ALZHEIMER’S DISEASE: A ROLE FOR NITRIC OXIDE SYNTHASE

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Background: Until few years ago, most studies of Alzheimer’s disease (AD) investigated the effects of this syndrome in the CNS. Only recently, several evidences suggest that many abnormalities in vascular could be responsible for the AD. In particular, the reduced deformability of erythrocytes is one of the most suspected events associated to vascular abnormalities in AD. At this regard NO was proposed to be a regulatory factor of RBC mechanical properties since inhibitors of endogenous NO synthesis induce decreased erythrocyte deformability.

Methods: The present study aimed at investigating the effects of amyloid beta peptide in the alterations of RBC rheologic behaviour by NO. Human erythrocytes were incubated with amyloid beta peptide in the presence and absence of NO donors. After incubation, erythrocyte deformability and acetylcholinesterase (AChE) activity, a marker of erythrocyte membrane integrity, were evaluated.

Results: Preliminary studies showed that in human erythrocytes, eNOS (erythrocyte nitric oxide synthase) activity is affected following to 24 h exposure to amyloid-beta peptide. Concurrently, amyloid beta peptide significantly reduced erythrocyte deformability and Ache activity, whereas the NO donors, were able to reverse the effects of amyloid beta peptide.

Conclusions: These results provide support that the hemorheological abnormalities found in AD patients may be explained by the amyloid mediated effects on eNOS linked-pathway that could result in a decrease of erythrocyte deformability. These events could contribute to the vascular alterations associated with AD disease.