

P196. OXIDANT IMBALANCE IN THALASSEMIA PATIENTS

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Thalassemia is a hereditary blood disease. Beta (β)-thalassemia is an autosomal recessive and also one of the most common genetic diseases in worldwide that is caused by a point mutation on β -globin gene which is localized on short arm of chromosome 11 as a cluster. In other words, this disease is characterized by malfunctions during the globin chain synthesis of hemoglobin synthesis process. Unbalanced globin chain synthesis is the major cause of low level hemoglobin production leading to anemia. In iron deficiency anemia, sensitivity against oxidants of erythrocytes increases and life expectancy is shortened. Oxidative stress is caused by the increase of free radicals and it creates disorder in metabolism due to damage in biological macromolecules. Multifactorial mechanisms facilitate oxidative damage in thalassemia because of free, unpaired, unstable globin subunits create superoxide and hydroxyl radicals. Hydroxyl radical, excessive oxidizing free radical, leads to protein aggregation and hydroxylation of DNA. Moreover, it causes event such as decreasing in the deformability with membrane skeleton impairment, premature aging of the erythrocyte with antigenic changing, increasing in the rigidity, peroxidation of membrane lipids and losing in intracellular K^+ with deterioration of cation exchange. As to thalassemia patients, increased free radicals cause damage to the tissue in patients with suffering from iron overload via frequent blood transfusions and iron accumulation leads to production of toxic oxygen radicals.

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