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Anais

ELEVATED MALONALDEHYDE (MDA) IN SICKLED ERYTHROCYTES AS INDICATIVE BY THE LIPID PEROXIDATION IN SICKLE CELL ANEMIA

VANUSA MANFREDINI; L.L. LAZZARETTI; I. H. GRIEBELER; A. P. SANTIN; S. C. WAGNER; S. M. CASTRO; M. C. R. PERALBA; M. S. BENFATO

Sickle cell anemia (SCA) is a molecular blood disorder that is caused by a single point mutation in one of the genes encoding hemoglobin. In sickle hemoglobin (HbS), the normal $\beta 6$ glutamic acid residue is replaced by valine (GAG to GTG) mutation at codon. The disease is characterized by a variety of clinical problems including oxidative stress. We evaluate 55 patients: 25 (HbAA), 20 (HbAS) and 10 (HbSS) from different ages (1-60 years old) selected from Centro de Apoio ao Portador de Anemia Falciforme (CAPAF-RS). Ten SCA patients and twenty trait patients were identified and characterized by ion-exchange high-performance liquid chromatography (HPLC). All patients were not detectably deficient in red cell glucose-6-phosphate dehydrogenase (G6PD). Patients were selected who had not been transfused or in hemolytic crisis for at least 3 months prior to study and did not smoke. In this work, we measured the main antioxidants enzymes: catalase (CAT), glutathione peroxidase (GPx) and superoxide dismutase (SOD) in healthy controls, trait and sickled cell erythrocytes and characterized the membrane damage in terms of lipid peroxidation as indicated by the lipid peroxidation potential, accumulation of malonaldehyde (MDA) by HPLC. Our data show that trait patients had significant higher CAT levels than healthy controls. On the other hand, sickle cell patients had elevated levels of GPx and SOD. Sickled erythrocytes had higher peroxidation potential and higher levels of endogenous MDA than normal erythrocytes. Supported by: CNPq Brazil