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Unravelling Mitochondrial Dysfunction in Rheumatoid Arthritis patients

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Abstract

Rheumatoid arthritis (RA) is a chronic, inflammatory, autoimmune disease associated with systemic, extra-articular and articular effects, causing permanent disability, early morbidity; making the patient compromised with a worldwide prevalence of 0.8%, commonly effecting women with a rate of 0.7% in India. With improved and developing therapeutics, this disease needs special focus for improved diagnosis and better treatment. The hyperactivity of immune cells is responsible for pathogenesis and progression of the disease. This study unravels the changes in mitochondria of RA patients which may be a potential reason for abnormal functioning of immune cells against self-antigens and occurrence of the disease. In this study we examine the following aspects of mitochondrial functions in the peripheral blood mononuclear cells (PBMCs) of patients and their paired control samples: 1) Change in mitochondrial membrane potential (MMP); 2) mitochondrial mass; 3) mitochondrial superoxide and 4) ATP levels. Patients satisfying the 2010 ACR/EULAR classification criteria for RA diagnosis were enrolled in this study. PBMCs of RA patients and controls were collected by differential gradient centrifugation. MMP, mass and superoxide levels were measured using respective commercially available dye using flow cytometry. ATP levels were measured by lysing equal number of cells from patients and controls using ATP measurement kit. In our case control cohort, we found a significant decrease in MMP ($p<0.005$) in PBMCs of RA patients where the change in mitochondrial mass was insignificant. The mitochondrial superoxide levels were found to be significantly low ($p<0.05$) in PBMCs of RA patients with significantly low ($p<0.005$) total cellular ATP as compared to controls. Our results indicate reduced potential and mitochondrial superoxides with decreased total cellular ATP. Reduced potential will disturb proper functioning of mitochondria in PBMCs which may affect most important function of mitochondria to produce ATP and various other functions. Results depict dysfunction in basic mitochondrial activities which may be a reason for abrupt functioning of immune cells, leading to autoimmunity in RA patients.

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