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Cellular biochemistry and innate immunity: Insight into the pathogenesis of Rheumatoid arthritis

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Rheumatoid arthritis (RA) is an autoimmune disorder affecting majorly the joints. The pathogenesis of RA is still elusive. Biochemical investigations have long been treated as a valuable tool for the prognosis but not the diagnosis of the disease. Present work has been undertaken to investigate the role of intra-cellular biomolecules in the pathogenesis of RA. Peripheral blood samples of RA patients (n=20) and healthy volunteer (n=20) with ethically approved inclusion and exclusion criteria were obtained. The intracellular levels of Superoxide dismutase (SOD), Catalase (CAT), Glutathione Peroxidase (GSH-Px), Malondialdehyde (MDA), reduced Glutathione (GSH), reactive oxygen species (ROS) and the percentage population of Natural Killer (NK) cells were enumerated in the red blood cells (RBCs) and peripheral blood mononuclear cells (PBMCs). The intracellular oxidative stress has been found elevated in the patients. The NK cell number were found lessened and negatively correlated with levels of MDA and ROS while positively correlated with levels of SOD, CAT, GSH-Px and GSH. The disease severity was also found to be positively correlated with elevated oxidative stress and negatively with the NK cell number. The results explain the effect of oxidative stress on the innate immunity of the RA patients. The innate immunity of the patients goes down in the oxidatively stressed patients. Diminished NK cell population might be the result of lesser ATP production because of elevated oxidative stress. Further research in this direction may un-reveal newer horizons in autoimmunity research and treatment. The present study was supported by the CSIR, India.

Biography

Ashish Aggarwal is doing his Ph.D in Panjab University.

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