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Cyclophosphamide-induced oxidative stress in Platelets: Effect of N-acetylcysteine in vitro

Background: Therapeutic drugs are capable of causing oxidative stress (OS) during their course of action while undergoing metabolism. Platelets are present in whole blood microenvironment and are susceptible to the presence of free radicals. Cyclophosphamide is a chemotherapeutic drug capable of generating high reactive oxygen species and thereby, induces OS in platelets. It is also implicated in thrombocytopenia. Antioxidants can mitigate the OS and attenuate the damage caused by these radicals. N-acetylcysteine (NAC), a thiol compound is a potent scavenger of reactive oxygen and nitrogen species (ROS and RNS). This study investigates the influence of NAC on platelets during cyclophosphamide-induced OS.

Methods: Platelets isolated from the whole blood of male *Wistar* rats were categorized into four groups (n=5): Controls, Drug (cyclophosphamide-treated), NAC (NAC-treated), and Drug+NAC (NAC preincubated and cyclophosphamide treated). Antioxidant defenses, oxidative stress, and platelet functions were analyzed.

Results: Superoxide dismutase, catalase, glutathione, and protein sulfhydryls increased and ROS decreased in Drug+NAC compared to Drug. ATP secretion and aggregation increased in Drug+NAC compared to Drug.

Conclusion: N-acetylcysteine was capable of augmenting the endogenous antioxidant defenses in platelets and also, scavenged the ROS efficiently. It protects the protein thiols from oxidative damage. It improved platelet responsiveness to collagen by ameliorating ATP secretion and aggregation of platelets. Therefore, NAC has proved to be beneficial to platelets during cyclophosphamide-induced OS. This study lays the foundations for the potential application of NAC in animal models of OS.

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