Volume 109, January 2019, Pages 1078-1084

Mechanistic role of cAMP and hepatocyte growth factor signaling in thioacetamide-induced nephrotoxicity: Unraveling the role of platelet rich plasma

Keshk, W.A.[a]Email Author, Zahran, S.M.[b]

[a] Medical Biochemistry Department, Faculty of Medicine, Tanta University, El-Geish Street, El-Gharbia, Tanta, Egypt

[b] Biochemistry Department, Faculty of Pharmacy and Drug Manufacturing, Pharos University, Alexandria, Egypt

Abstract:

Chronic kidney diseases occur as result of exposure to wide range of deleterious agents as environmental pollutants, toxins and drug. Currently, there is no effective protective therapy against renal damage, fibrosis and its sequel of end stage renal disease. Platelet-rich plasma (PRP) has a progressively gained consideration in wound healing, repair/regeneration of damaged tissues and conservation of organ function. However, its impact on thioacetamide (TAA) induced chronic renal damage has not been elucidated yet. So, the present study was carried out to evaluate the possible protective and regenerative effect of PRP against TAA induced renal damage and their potential underlying mechanism. PRP treatment improved redox state, renal function disturbed histologicl features; decreased monocyte chemo-attractant protein-1 (MCP-1) level; increased Peroxisome proliferator-activated receptor gamma co-activator-1α (PGC-1α) marker of mitochondrial biogenesis and metabolism; cyclic adenosine monophosphate (cAMP); hepatocyte growth factor (HGF) and autophagy protein beclin-1 level. In addition, PRP treatment decreased apoptosis and fibrosis as evidenced by decreased active caspase3 and α-SMA expression and immunoreactivity, respectively. In conclusion, PRP could potentially protect against TTA-induced chronic kidney damage by alleviating oxidative stress, improving, mitochondrial biogenesis, autophagy, disruption of the inflammatory, apoptotic and fibrotic response induced by TTA. © 2018 Elsevier Masson SAS

Reference: