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Neuroprotective effect of carvedilol against aluminium induced toxicity: possible behavioral and biochemical alterations in rats

Anil Kumar, Atish Prakash, Samrita Dogra

Pharmacology Division, University Institute of Pharmaceutical Sciences, UGC Center of Advanced Study, Panjab University, Chandigarh-160014, India

Correspondence: Anil Kumar, e-mail: kumaruips@yahoo.com

Abstract:
Aluminium, is a trace element available in the Earth's crust naturally and has a toxic potential for humans. It has been suggested as a contributing factor in the pathogenesis of Alzheimer's disease. β-Adrenoceptor blocking agents (β-blockers) have been established as therapeutics for the treatment of patients with hypertension, ischemic heart diseases, chronic heart failure, arrhythmias and glaucoma. Over the years, however, β-blockers have been associated with an incidence, albeit low, of central nervous system (CNS) side effects. In addition, noradrenergic receptors play a modulatory role in many nerve functions, including vigilance, attention, reward, learning and memory. Therefore, the present study has been designed to explore the possible role of carvedilol, an adrenergic antagonist against aluminium chloride-induced neurotoxicity in rats. Aluminium chloride (100 mg/kg) was administered daily for six weeks that significantly increased cognitive dysfunction in the Morris water maze and oxidative damage as indicated by a rise in lipid peroxidation and nitrite concentration and depleted reduced glutathione, superoxide dismutase, catalase and glutathione S-transferase activity compared to sham treatment. Chronic aluminium chloride treatment also significantly increased acetylcholinesterase activity and the aluminium concentration in brain compared to sham. Chronic administration of carvedilol (2.5 and 5 mg/kg, *po*) daily to rats for a period of 6 weeks significantly improved the memory performance tasks of rats in the Morris water maze test, attenuated oxidative stress (reduced lipid peroxidation, nitrite concentration and restored reduced glutathione, superoxide dismutase, catalase and glutathione S-transferase activity), decreased acetylcholinesterase activity and aluminium concentration in aluminium-treated rats compared to control rats ($p < 0.05$). Results of this study demonstrated the neuroprotective potential of carvedilol in aluminium chloride-induced cognitive dysfunction and oxidative damage.

Key words:
aluminium, Alzheimer's disease, oxidative stress, carvedilol, neuroprotection