EFFECTS OF ACUTE ALUMINUM CHLORIDE EXPOSITION ON BLOOD PRESSURE, VASCULAR REACTIVITY AND OXIDATIVE STRESS IN RATS

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Aluminum is abundantly distributed in our environment and this exposure is inevitable nowadays. This metal can be accumulated in several organs and has been associated with osteopenia, anemia and neurological disorders. However, the effects of aluminum on cardiovascular system remains unclear. We investigate the effects of acute exposure to aluminum chloride (AlCl3) on blood pressure, vascular reactivity and oxidative stress. Male Wistar rats were divided into groups: Untreated: vehicle (ultrapure water, ip) and AlCl3: single dose of AlCl3 (100 mg/kg ip). Systolic and diastolic blood pressure was assessed in anesthetized rats through cannulation of carotid artery immediately after aluminum injection and after one hour of exposure. AlCl3 concentration was measured in serum; malondialdehyde levels (MDA), superoxide dismutase (SOD) and catalase (CAT) activities were analysed in plasma. Aorta reactivity was done in isolated organ bath and concentration-response curves to acetylcholine, sodium nitroprusside and phenylephrine were performed in presence and absence of endothelium, nitric oxide synthase inhibitor (L-NAME), non selective COX inhibitor (indomethacin), NADPH oxidase inhibitor (apocynin), potassium channels blocker (TEA) and SOD. Results were expressed as mean±SEM and analyzed by t test or two-way ANOVA followed by post-hoc Bonferroni (P<0.05). One hour of AlCl3-exposure was able to increase Al serum concentration (Untreated: 67.0 ± 10.8 vs AlCl3: 147.7 ± 25.0* µg/L), MDA levels and CAT activity, on the contrary Al reduced SOD activity. Acute exposure did not change blood pressure however decreased vasoconstrictor response to phenylephrine (Emax - Untreated: 114.5±1.4 vs AlCl3: 91.0±4.3*-%KCl), with increased negative endothelial modulation, ROS production from NADPH oxidase mainly superoxide anions. Aluminum exposure did not changes the endothelium-dependent and independent relaxation. Thus, a single exposure to AlCl3 is able to promote vascular dysfunction. Increased oxidative stress and nitric oxide pathway activation are potential mechanisms of the current dysfunction.

Key-Words: Aluminum, cardiovascular system, oxidative stress.

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