
Cisplatin, or cis-diamminedichloridoplatinum(II), (CDDP) is a broad-spectrum antineoplastic chemotherapeutic agent with a potent efficacy against several malignancies. Its main clinical antineoplastic therapy-limiting adverse effect is nephrotoxicity, where the developments of effective nephroprotectors are needed. Therefore, the present study aimed to investigate the nephroprotective and antifibrotic potential of ceftriaxone (CTX) against CDDP-induced toxicity. Male Wister rats were treated with saline or CTX (100 or 200 mg kg\(^{-1}\) bw) an hour before CDDP administration (1 mg kg\(^{-1}\) bw). All the treatments were intraperitoneally administered twice weekly for consecutive 10 weeks. Twenty-four hours after last CDDP dose, blood samples were collected, then the animals were euthanized and their kidneys were isolated for measurements. CDDP significantly increased serum uric acid, urea, and creatinine contents. Toxicopathic changes showed that CDDP induced marked tubulointerstitial damage, overexpressed fibrogenic factors alpha-smooth muscle actin (alpha-SMA) and transforming growth factor-beta1 (TGF-beta1), and down expressed cellular proliferating biomarker bromodeoxyuridine (BrdU). CTX pretreatment, particularly 200 mg/kg bw, improved the renal function biomarkers; histoarchitecture; and alpha-SMA, TGF-beta1, and BrdU expressions. It could be concluded that CTX is endowed with antifibrotic properties and could be, therefore,
used as adjuvant therapy to improve CDDP-induced nephrotoxicity. Further clinical researches are necessary to evaluate whether CTX may exhibit a new therapeutic choice for treating renal fibrotic diseases.