## Protective Efficacy of Moringa oleifera against Oxidative Ovarian Damage and Reproductive Failure in Female Rats Caused by Cyclophosphamide

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Abstract : Cyclophosphamide (CP), an antineoplastic drug, has been found to induce reproductive damage. It is essential to develop approaches aimed at safequarding ovarian tissue integrity in women experiencing reproductive toxicity as a result of chemotherapy. The current study was conducted to assess the impact of an extract derived from Moringa oleifera (M. oleifera) leaves on ovarian damage produced by CP. A total of 32 female Wistar Albino rats, which were in a healthy cycling state, were randomly separated into 4 groups, with every group contains 8 rats. The first group was administered intraperitoneal (i.p.) saline. The second group was administered a solitary intraperitoneal dosage of cyclophosphamide (200 mg/kg). The third one received M. oleifera extract (150 mg/kg orally) for 20 days, followed by i.p. of CP on the last day of the experiment. The fourth group received M. oleifera extract (250 mg/kg orally) for 20 days, followed by i.p. of CP on the last day of the experiment. Hormonal assessments, including luteinizing hormone (LH), estrogen (ES), and follicle-stimulating hormone (FSH), were performed 24 hours after CP administration. In addition, evaluating the antioxidant status and inflammatory response against CP. Moreover, conducting detailed histopathological and ultra-structural pictures of the ovary. Our findings reported that rats intoxicated with CP exhibited elevated levels of FSH, LH, malondialdehyde (MDA), tumor necrosis factor-alpha (TNF-α), and a decrease in E<sub>2</sub>, and glutathione (GSH) levels. Pre-treatment with M. oleifera extract (250 mg/kg orally) ameliorated the disturbance in hormonal changes, oxidative stress indices, and the levels of pro-inflammatory mediators. Also, the histopathological and ultra-structural pictures of the ovaries were improved significantly in rats. In conclusion, M. oleifera extract possesses a significant protective role against CP-induced acute reproductive toxicity via modulating the values of FSH, LH,  $E_2$  and quenching the release of reactive oxygen species and inflammatory mediators in female rats.

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