Abstract

Parkinson’s disease is the commonly occurring neurodegenerative disorder that about 1 to 3% of the population was affected by it, mostly age above 65 years. The current study was conducted to evaluate the neuroprotective role of *Moringa oleifera* (MO) in the rotenone induced mouse model of PD. For this purpose, ethanolic leaf extract (EthMO) and aqueous leaf extract (AqMO) were prepared. The profiling was done using the HPLC in which quercetin and cinnamic acid were run as standards and all the three standards were found in the AqMO while EthMO contains many compounds other than the standards. Four animal groups were formed; control, rotenone treated, EthMO+Rotenone and AqMO+Rotenone. Batch I was run (n=16) with low dose of rotenone 1 mg/kg diluted in sunflower oil, injected intraperitoneally for consecutive 21 days, and MO (200 mg/kg) was given orally for 28 days. With this dose no behavioral changes observed, and the lipid peroxidation level did not change significantly as compared to control group. To induce PD, a chronic dose of rotenone used. Batch II was run (n=24) with rotenone 2.5 mg/kg, subcutaneously, in DMSO and further diluted in sunflower oil for 21 days with the same dose of MO i.e. 200 mg/kg, orally, for consecutive 28 days. After 28 days protocol, Parkinson’s disease symptoms were observed, using beam walk, pole climb down, open field, tail suspension, stepping tests and stride length measurements. The biochemical tests, such as lipid peroxidation (LPO) level, catalase (CAT), glutathione-S-transferase (GST) and reduced glutathione (GSH) activities were evaluated, and substantia nigra was also evaluated histologically using hematoxylin and eosin stain. The rotenone treated group replicate the PD symptoms such as limited movement in beam walk test, time to descend increased in pole test, less locomotory activity in open field test, immobility time increased and shorter stride length. The LPO level significantly increased while CAT, GSH and GST levels significantly decreased when compared to vehicle group. While the treatment groups reduced the rotenone induced deficits and showed improvement in the behavioral and enhanced the antioxidant activity. The histological study showed that in rotenone group the neurons shrunk and vacuolation increased with the %neurodegeneration of about 45% while the EthMO and AqMO showed less vacuolation and less %age neurodegeneration of about 25%. Collectively, current experiment data furnishes the protective behavior of MO on rotenone induced PD through antioxidant capacities.