Copious experimental studies provided concrete evidence that oxidative stress and mitochondrial dysfunction are the basic etiology of Parkinson’s disease. Thus, to combat disease progression, hybrid drugs with superior pharmacological properties will be an effective way of treatment. Hence, the present study was carried out to investigate the neuroprotective properties of CNB-001. Results of insilico studies proved that CNB-001 satisfied all the properties to be taken over as a drug. Moreover, this study stands unique and proved that CNB-001 inhibited free-radical formation and protected dopaminergic neurons against toxic models of Parkinson’s disease. Invitro studies indicated that CNB-001 offers neuroprotection by its antioxidant, mitochondrial protective and anti-apoptotic properties. Furthermore, invivo results supported the therapeutic potential of CNB-001 by its ability to inhibit behavioral impairments, oxidative stress, mitochondrial deficits, inflammatory and apoptotic response and increasing the expression of TH, DAT and VMAT2 which might contribute to enhanced dopamine synthesis. Hence, owing to its pleiotropic mechanism of action, we speculate that further investigation on CNB-001 would be worth to bring out a potent therapeutic contender in treatment of PD.