Neuronal stem cells are attractive tool to study the neurodegenerative disorders. In the present study, we have used the immortalized ReN (VM) cell line, which is derived from the human fetal midbrain region. The ReN cells were differentiated into Dopaminergic neurons and the colocalization of Calcium Calmodulin dependent protein kinases I and IV (CaMKs I and IV) was studied in normal (untreated) and 6-hydroxydopamine (6OHDA)-treated dopaminergic neurons. CaMK-I and IV play a crucial role in the synthesis of tyrosine hydroxylase (TH) which in turn stimulates the production of dopamine. The expression of TH was used as a marker of dopamine in dopaminergic neurons. Our results showed the presence of CaMK-I and IV in untreated dopaminergic neurons. However, the expression of both CaMKs-I and IV found to be reduced significantly following 6OHDA treatment which correlated with the increase in ROS activity followed by the apoptotic cell death. Our data clearly describes the involvement of both CaMKs-I and IV in normal as well as in 6OHDA-treated dopaminergic neurons. Furthermore, it explains the role of calcium-stimulated signalling pathways in Parkinson Disease. Our results support the notion that damage or loss of dopaminergic neurons following oxidative stress caused by 6OHDA treatment disrupts the neuronal calcium homeostasis pathway. While decreasing the calcium buffering capacity may contribute to the development of neurodegenerative processes.