Abstract
Trumatic brain injury (TBI) is one of the serious neurodisorders commonly caused by car accidents and sports. Preventive measures are highly recommended to reduce the risk and number of TBI cases. After TBI, the primary injury to the brain initiates a secondary injury process that spreads via multiple molecular mechanisms in the pathogenesis of TBI. Many studies in animals using cDNA microarray hybridization technique have shown differential regulation of 86 genes (mainly transcription factors, signal transduction genes and inflammatory proteins) which take part in the physiological and pathological response to TBI. Genetic polymorphisms which involve interleukin-6 (such as -174G>C and -572G>C) and haem oxygenase -1 may influence the inflammatory effects seen after TBI. In addition, genes regulating the vascular responses including the hypoxia-inducible factor-1 and 2 (HIF1&2) genes activate follow ing cerebral ischemia. Moreover, Apolipoprotein epsilon and P53 genes regulate the neuronal response to TBI. Also, there are three isoforms of the enzyme catechol-o-methyltransferase (COMT) encoded by 3 genetic polymorphisms (COMT Val/Val, COMT Val/Met, and COMT Met/Met) which regulate catecholamines activity falling TBI. A greater understanding of the genetics could aid in the prediction of outcomes and could be targeted for treatment strategies.

Keywords: Catecholamines, Car Accident, Ischemia, Traumatic Brain Injury.