Delayed complications after traumatic brain injury (TBI) include more clinical entities; among the most severe are cognitive impairment, depression and tardive post-traumatic epilepsy. Their development is related to many factors, among which the type and severity of TBI are essential but also the reactivity of the brain tissue to complex injury is extremely important, including the role of neurotrophic factors. The data related to pathogenesis of tardive post-traumatic epilepsy are discussed and their implications related to possible pathways for its potential prevention.