Traumatic Brain Injury and their Possible Therapeutics

Domenico De Berardis*

Department of Mental Health, Institute of Psychiatry, Portugal

Commentary

Traumatic brain injury (TBI) is the leading cause of morbidity, disability, and mortality in all age groups. Over 50 million people worldwide su er from TBI each year. As of 2005, approximately 3.17 million TBI survivors su er from post-traumatic complications ranging from neurological and psychosocial problems to long-term disability. Huge spending on clinical management of TBI patients and the associated socio-economic problems place a heavy burden

action of methylprednisolone, but it is thought to be incorporated into the structure of the lipid bilayer, hardening the cell membrane and thereby limiting the mobility of lipid peroxy radicals. In particular, methylprednisolone should be given at optimal concentrations in the early stages of CNS injury to ensure maximum anti-in ammatory and neuroprotective e ects. Methylprednisolone was previously included in a randomized, placebo-controlled trial called CRASH in 2004. A large sample size of more than 10,000 TBI patients was enrolled in the study with a 2-week follow-up period. Nevertheless, with increasing mortality, the results were undesirable. In fact, rats treated with methylprednisolone also showed a signi cant increase in neuronal apoptosis in the hypothalamus, pituitary gland, and hippocampus [7].

ese are associated with memory and learning disabilities. Primary damage to the TBI is primarily irreversible. e resulting damage occurs, progresses, and has access to therapeutic intervention over months to years. Because the delayed phase of this injury involves numerous events such as excitotoxicity, apoptotic cell death, inhibition of axonal regeneration, neuro in ammation, and oxidative stress, the development of e ective treatment strategies has been multiple over time. You need to target the mechanism. e availability of a depot system for regulated and sustained delivery of therapeutic agents that can enter cells by penetrating the plasma membrane is clearly an additional bioavailability of existing drugs will allow improvement. More importantly, it o ers the opportunity to explore the therapeutic potential of new compounds for drug-worthy targets. In fact, this therapeutic approach has been applied to the treatment of many neurodegenerative diseases such as Alzheimer's disease, Huntington's disease, and Parkinson's disease.

e feasibility of this strategy in the treatment of TBI has not yet been demonstrated, but events during secondary injury of TBI that require continued availability of bioactive therapeutic agents at non-cytotoxic concentrations. Looks promising because of the slow progress of. TBI has become a major health and socio-economic problem worldwide, imposing a signi cant health burden on modern societies