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Dhara Dave

NEW THERAPEUTIC STRATEGIES FOR THE TREATMENT OF BRAIN INJURY

Tutor Associate Professor, Candidate of Medical Sciences Vauchok A. V.
Department of Pharmacology
Belarusian State Medical University, Minsk

Traumatic brain injury (TBI) is an injury that affects how the brain works. Nearly 2 million TBIs occur every year resulting in 50,000 deaths and 76.5 billion dollars in medical costs. It causes a long-lasting effect on brain. Nowadays, there has been many research going on new therapeutic strategy for treatment of brain injury. TBI patients are more likely to develop stroke, even Alzheimer's disease or other disorders later in life. Disturbance in blood brain barrier which are cerebral endothelial cells with special structures can lead to damage to the cerebral microvasculature and to the nearby brain tissue.

New therapeutic strategy that is repurposed pharmacological interventions leading to improvement of injury in brain includes use of statins, angiotensin II receptor type I antagonists, phosphodiesterase type 5 inhibitors and growth factor treatment is being used. Even infusion of erythropoietin hormone, hyperbaric oxygen therapy is new steps for the angiogenesis process in human brain after injury. Many experiments have been taken under considerations of experimental injury or stroke, it has been concluded that statins have ability to induce neurogenesis, neuroprotection, neuroplasticity and angiogenesis in ischemic border zone. But, the dose is taken in optimal lower concentration as it shows proangiogenic effect but a slight higher dose can convert statin into antiangiogenics. Statins have a dose-context-dependent modulatory effect on angiogenesis of brain vessels. Similar to statins, angiotensin II receptor type 1 antagonists are used. But the use of this antagonists usually leads to high risk of lung cancer in treated patients as it increases the expression of vascular endothelial growth factor (VEGF) and Fibroblast growth factor (FGF), so the use of this antagonist cannot be chosen for the treatment. Besides it, phosphodiesterase type 5 inhibitors are considered to be the important determinant of angiogenesis process by promoting atonal remodelling and therefore leading to recovery. The most importantly the growth factor treatment enhances the angiogenesis, neurogenesis and functional recovery is observed. The reason behind it was associated with increase of both VEGF and BDNF expression. The use of erythropoietin was discontinued due to increase in number of haemorrhage combined with tissue-type plasminogen. Usually, the erythropoietin increases the matrix metalloprotein which maintains the integrity of the CNS vasculature.

In conclusion, angiogenesis occurs after stroke and can be controlled by pharmacological means and also by non-pharmacological means. These strategies usually helps angiogenesis along with neuronal plasticity in the lesions hemisphere is the brain.