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Omega-3 Fatty Acids Could Alleviate the Risks of Traumatic Brain Injury – A Mini Review

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ABSTRACT

Traumatic brain injury (TBI) is an acquired brain trauma that occurs when any sudden trauma/injury causes damage to the brain. TBI is characterized by tissue damage and imbalance in the cerebral blood flow and metabolism. It has been established through laboratory experiments that the dietary supplementation of omega-3 fatty acids (FAs) could reduce the oxidative stress developed in brain due to TBI. The inclusion of omega-3 FA in diet could normalize the levels of brain-derived neurotrophic factor (BDNF), and thus, it could restore the survival of neuronal cells. BDNF improves the synaptic transmission by regulating synapsin 1 and cyclic adenosine monophosphate (cAMP) response element binding protein. The brain tissue analysis of TBI models supplemented with omega-3 polyunsaturated fatty acids (PUFAs) showed significantly reduced lipid peroxidation, nucleic acid and protein oxidation, thereby promoting neuronal and glial cell survival. Thus, omega-3 FA intake could be considered as a therapeutic option to reduce the secondary neuronal damages initiated by TBI.

Key words: Brain trauma, Neuronal damage, Omega-3 fatty acids, Oman, Traumatic brain injury

INTRODUCTION

Traumatic brain injury (TBI) is an acquired brain trauma that occurs when any sudden trauma/injury causes damage to the brain. An external mechanical force could displace the brain inside the skull, causing injury against the dura or neurocranium. Even the blood vessels in brain and nervous tissues could get disrupted. The impact of trauma ranges from mild to moderate to severe TBI.[1] The mortality indices vary from 1% in mild TBI to 2-5% in moderate TBI and up to 30-50% after suffering a severe head injury. According to the report of Centers for Disease Control and Prevention, about 50,000 individuals die every year from traumatic brain and twice this number suffer from permanent disabilities in the US.[2] The treatment opportunities are much limited in reversing the effects of TBI. It has been reported that omega-3 fatty acids (FAs) are effective natural agents in reducing the neuronal damage, and reducing the brain oxidative stress.

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FATAL SECONDARY EFFECTS OF TBI

The initial stages of cerebral injury after TBI are indicated by tissue damage and imbalance in the cerebral blood flow and metabolism. These irregular patterns can lead to increased accumulation of lactic acid due to anaerobic glycolysis, with increased membrane permeability causing more fluid retention and edema formation.[3] As a result of these events, there is depletion of ATP reserves, as the cellular energy requirements become inadequate with the anaerobic glycolysis. This ATP depletion, in turn, disrupts the membrane ion pumps. Therefore, the secondary stage of the TBI is initiated, which is characterized by the depolarization of terminal membrane leading to the release of excitatory neurotransmitters like glutamate. The Ca^{2+} and Na^{+} influx becomes high due to the impaired ion pumps. The excess Ca^{2+} causes activation of lipases, proteases, and lipid peroxidases, and produces free FA and free radicals. Moreover, the activated caspases and endonucleases initiate the degradation of biomembranes and the nucleosomal DNA. This eventually leads to programmed cell death of neuronal cells. The oxidative stress formed in response to TBI generates superoxides, NO, H_2O_2, and peroxynitrite. These events lead to the peroxidation of cellular structures, proteins, and vascular systems in the brain, and the cleavage of DNA, and finally inhibition of electron transport chain (ETC) in the mitochondria. All these factors sum up for the apoptos is.[3-5] All the above factors are given in Figure 1 as a flowchart. The therapeutic options are very much limited in reversing the impairments initiated as a result of TBI.

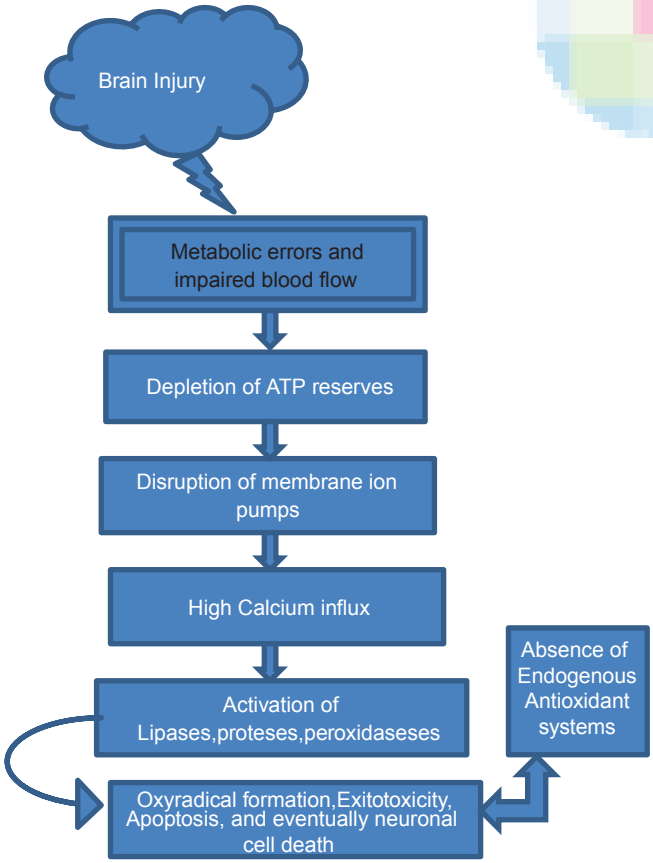

Figure 1. Schematic representation of possible damage occurring after TBI

IMPORTANCE OF OMEGA-3 FAs

Natural products and their active nutrients/components often cause possible improvement in most of the brain-related damages.[6] Omega-3 FAs are polyunsaturated fatty acids (PUFAs) having double bond at the third position from the end of the FA chain. They are highly essential for maintaining the membrane fluidity and are present in all tissues, but are rich particularly in retina, brain, and spermatozoa. There are three important physiological omega-3 FAs available, including alpha linolenic acid (ALA), eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA). Dietary omega-3 FAs are the precursors for the FA components of neuronal membrane and are essential for maintaining the structural balance of cell membranes, signaling, neurotransmission, and modulation of enzymatic activity.[7] Inadequate intake of omega-3 FAs results in inefficiency in learning and memory.[8,9] Brain tissue analysis of TBI models supplemented with omega-3 PUFAs showed significant reduction in the lipid peroxidation and nucleic acid and protein oxidation.[10]

POSSIBLE EFFECTS OF OMEGA-3 FAs IN RESCUING THE TBI SURVIVORS

As described earlier, brain injury may cause severe neuronal damage and consequent impairment in memory, learning, and motor coordination, and supplementation of omega-3 FAs might be able to reduce the neuronal damage. Neural trauma is usually associated with irregular phospholipid metabolism of neuronal membrane.[11,12] Omega-3FAs are the major constituents of the membrane phospholipids, which suggests that supplementation of PUFAs could help in reducing the irregular phospholipid metabolism that occurs during neuronal damage. Supplementation of DHA could enhance the gene expression, release of neurotransmitters, and restoration of synaptic activity, which are all deleteriously affected by the TBI.[13,14] The striatal dopamine levels reduced due to TBI could be normalized with the oral administration of fish oil.[15] The PUFAs are capable of inhibiting arachidonic acid catabolism and maintaining the levels of cytokines, and have a strong antioxidant potential, and thus, they are effective as strong anti-inflammatory factors.[16] Ying *et al*, from Pinnilla's group have manifested that induced Omega-3 deficiency during gestation enhances the susceptibility to TBI in rat pups.[17] Pinnilla's lab has also studied the effects of Omega -3FA supplementation and the action of progesterone in early life in the fluid percussion injury induced TBI rat models. They have demonstrated that Omega-3FA and progesterone are good candidates in counteracting the neuronal plasticity impairments.[18] A recent report showed that omega-3 FA intake could normalize the levels of brain-derived neurotrophic factor (BDNF), synapsin 1, and cyclic adenosine monophosphate (cAMP)-responsive element binding protein, reduce the oxidative damage, and improve memory and learn-

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