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Short Commentary

Transcranial Photobiomodulation Add-On Therapy to Antiepileptic Drugs in Pediatric Epilepsy

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Abstract

Transcranial Photobiomodulation (tPBM) monotherapy with a wavelength of 808 nm attenuated Pentylenetetrazole (PTZ)-induced seizures in peripubertal rats. Furthermore, tPBM add-on therapy to low-dose Valproic Acid (VPA) yielded synergistic effects on PTZ-induced seizures while offset effects were shown when tPBM add-on therapy was applied to high-dose VPA. With the development of the optimal protocols of the tPBM add-on therapy to antiepileptic drugs such as VPA, synergistic therapeutic effects are expected to present in the prevention and treatments of pediatric epilepsy in the future.

Introduction

Transcranial Photobiomodulation (tPBM) [1] is the application of Photobiomodulation (PBM) transcranially and noninvasively. The mechanisms of PBM started with the absorption of photons by mitochondrial cytochrome c oxidase [2] and resulted in the promotion of ATP synthesis and multiple neuroprotective effects [3]. The regularly used red to near-infrared light with a wavelength range 610-1064 nm for tPBM was owing to the penetration characteristics of photons through the skull and brain tissues. By far, tPBM with a wavelength 808 nm [3,4] and 780 nm [5] had shown antiepileptic effects on rats. Tsai et al., [3] first reported that when tPBM with a wavelength of 808 nm was served as monotherapy, tPBM attenuated Pentylenetetrazole (PTZ)-induced Convulsive Status Epilepticus (CSE) in peripubertal rats by attenuating hippocampal neuronal damages [3], neuroinflammation [4], apoptosis of Parvalbumin-Positive Interneurons (PV-INs), astrogliosis, microgliosis [4] and preserving the integrity of peri-somatic inhibitory neuronal networks of PV-INs surrounding principal cells in the hippocampus of peripubertal rats [3].

tPBM with a wavelength 808 nm could also serve as add-on therapy in pediatric epilepsy. Tsai et al., [6] further demonstrated in the paper entitled "Transcranial photobiomodulation add-on therapy to valproic acid for pentylenetetrazole-induced seizures in peripubertal rats" that when tPBM was added to low-dose Valproic Acid (VPA), it yielded synergistic effects in shortening the latency to mild seizures while offset effects were presented in increasing the duration of moderate-to-severe seizures when tPBM was added to high-dose VPA. tPBM add-on therapy to low-dose VPA could reduce the demand of VPA dosage and this reduction would lessen side effects of VPA including hepatotoxicity [6].

The sequential arrangement of the experiments that tPBM was administered 30 min after VPA injection and immediately prior to PTZ injection [6] implied the future application of CSE prevention. Certainly, optimization of experimental protocols such as shortening the interval of VPA and tPBM, adjusting the dosage of VPA, duration or power density of tPBM, might bring out better synergistic seizure prevention effects. Furthermore, not only for "prevention", tPBM add-on to VPA for "treatment" might be feasible after onset of pediatric seizures or CSE. Further compatible experiments with sequential design for that tPBM add-on to VPA after the onset of seizures are needed.

The Antiepileptic Drugs (AEDs) used in the above-mentioned study of tPBM add-on therapy was valproate, which belongs to the second-line AEDs for pediatric CSE. tPBM add-on therapy to the second-line AEDs for pediatric CSE such as phenobarbital, lacosamide, levetiracetam and phenytoin might exert synergistic effects. Further experiments are yet to be explored.

Refractory Status Epilepticus (RSE) refers to the condition that seizure activity persists after administration of first- and second-line AEDs. Intravenous continuous infusions of anesthetics such as propofol, midazolam, or barbiturates to cause medically induced coma are critical treatment means for RSE. Nevertheless, when status epilepticus persists or recur 24 h or more after the administration of general anesthesia or recurrence of the withdrawal of general anesthesia, the condition was considered as Super Refractory Status Epilepticus (SRSE) [7]. tPBM add-on to general anesthetics for the treatments of pediatric RSE and SRSE might be feasible as well. Such tPBM add-on strategies might reduce the demand of general anesthesia so as to reduce the side effects of anesthetics.

In summary, tPBM as monotherapy attenuates PTZ-induced seizures, and tPBM as add-on therapy to low-dose VPA yielded synergistic seizure prevention effects while offset effects were presented when tPBM added to high-dose VPA. Further optimization and adjustment of sequential arrangements of tPBM add-on therapy for seizure/CSE prevention and treatments are crucial. Furthermore, tPBM add-on to anesthetics for treatment of RSE or SRSE might be feasible. More basic laboratory research and clinical studies or clinical trials are needed to lay the foundation on future clinical application of tPBM add-on therapy for pediatric epilepsy.

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