iMedPub Journals www.imedpub.com

DOI: 10.21767/2471-8041.100109

2018

Vol.4 No.2:73

The Photobiomodulatory Therapy and ILIB in the Repair of Encephalic Cisterns and Progressive Cognitive Restoration in a Patient with Traumatic Brain Injury

Juliano Abreu Pacheco^{1*} and Letícia Mello Bezinelli²

¹Centro Educacional Don André ArcoVerde, Valença, Ribeirão Preto, Hospital Albert Einstein, São Paulo, Intensive Therapy, Unibrati, São Paulo, Hospital Câncer Ribeirão Preto, Brazil

²Dentistry College of University of São Paulo, Fundecto-USP, São Paulo, Brazil

***Corresponding author:** Juliano Abreu Pacheco, Odontologist graduate from Centro Educacional Don André Arco Verde, Valença, Postgraduate in Implantodology USP, Ribeirão Preto, Postgraduate in Hospital Dentistry in Hospital Albert Einstein, São Paulo, Master degree in Intensive Therapy, Unibrati, São Paulo, Researcher in Hospital Câncer Ribeirão Preto, Brazil, Tel: +55 16 3878-9700; Fax: +33(0)383 646 158; E-mail: coepacheco@gmail.com

Received: May 08, 2018; Accepted: May 20, 2018; Published: May 22, 2018

Citation: Pacheco JA, Bezinelli LM (2018) The Photobiomodulatory Therapy and ILIB in the Repair of Encephalic Cisterns and Progressive Cognitive Restoration in a Patient with Traumatic Brain Injury. Med Case Rep Vol.4 No.2:73.

Abstract

This case study aims to present the process of neuropsychological rehabilitation of a patient during the acute post-trauma period, and incisive results, through the complementary (complementary) intervention of photobiomodulatory laser therapy in muscle areas of the craniofacial and photo-parenteral region by ILIB (Intravascular Laser Irradiation of Blood). These procedures contributed in a short period of time to the neuropsychological rehabilitation of this patient who had suffered severe cranioencephalic trauma and evolved with cognitive and behavioral alterations that had a significant impact on their autonomy.

Keywords: Neurology; Cranioencephalic trauma; Cognitive level; Facial muscles; Low level laser; ILIB; Health sciences; Dentistry; Speech-language pathology; Physiotherapy; Medical

Introduction

Neuropsychological Rehabilitation can be defined [1] as an active process of education and training, focused on the appropriate management of acquired cognitive alterations. This way aims at obtaining the best physical, mental and social potential of the individual, so that he can reintegrate to the old one or adapt to the new social environment. Therefore, neuropsychological rehabilitation [2] has as one of the main functions, the total adaptation of the cognitive, communicative and behavioral functioning of patients with functional alterations following neurological damage, which we will exemplify in this study, regarding post-TBI (Traumatic brain injury). It should be noted that during the rehabilitation process of the affected patient [3,4], the clinical reasoning of this one, corroborates for the maintenance of the total or partial preserved functions, which will determine the teaching

of compensatory strategies, acquisition of new skills and adaptation to permanent losses. According to Tate et al. [3] cognitive rehabilitation refers to any intervention strategy or technique, which makes clients or patients and their respective families able to coexist, manage, overcome, reduce or accept cognitive deficits caused by brain injuries. It should be added that neuropsychological rehabilitation and cognitive rehabilitation are analogous and aim to improve cognitive abilities, emphasizing the emotional, psychosocial, behavioral and physical aspects that may be deficient after brain injury.

Case Study

This case study aims to present the role of laser therapy and ILIB in the process of neuropsychological rehabilitation of a patient during the acute post-traumatic brain injury.

The patient was TLV, 48 years old, male, with an initial diagnosis of severe TBI; Subarachnoid hemorrhage; Hemoventricle; Diffuse axonal injury, Mild ectasia (dilatation) of the ventricular system. The treatment of the medical staff (Hospital Santa Mônica/GO) consisted of Amantadine hydrochloride 100 mg/2x per day, Hidantal 100 mg/3x per day, B complex 1x per day, and physical therapy in two daily periods.

In agreement with the responsible physician of the neurology and relatives, the patient was treated associatively with the low power laser (3 J/cm²-red/infrared) in muscle groups (a): epicranic, temporomandibular, temporal temporal fascia (deep and superficial lamina), occipitofrontal, procerus, corrugator of the eyebrow, lifter of the lip and nose wing, lifter of the upper lip, orbicularis of the mouth, mentally, zygomatic major, masseter, to decrease pain, inflammatory, and the evolution of cognition (Figures 1 and 2). The five sessions protocol was performed by punctuating the cited muscles for 10 seconds and ILIB Technique (variable time/5-15 minutes) during the five-day uninterrupted period (Figure 3).

Medical Case Reports ISSN 2471-8041

Vol.4 No.2:73

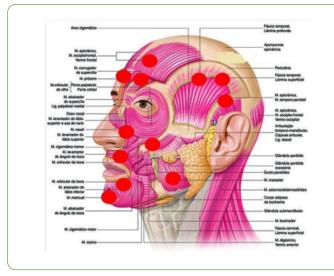


Figure 1 Muscle groups.



Results

The results were measured by the Rancho los Amigos Cognitive Levels Scale [5-17], represented by the state of agitation or inappropriateness and functional capacity, divided into ten levels, which assign values to the different levels of brain function, according to the patient's reaction to external stimuli. In this study, the patient reached an evolution from level 1 to level 3, and reestablishment of brain cisterns (axial section) in the image examination (Computerized Tomography), according to the **Figures 4 and 5**.

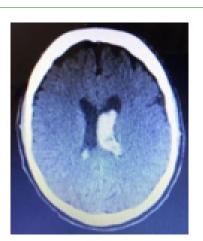


Figure 4 TC axial time 1/CDI-GO.

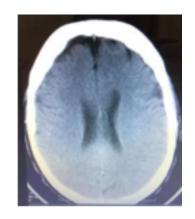


Figure 5 TC axial time 2/CDI-GO.

Discussion

In this study, brain damage following traumatic brain injury (TBI) [5] is caused by an aggression or by a high-intensity acceleration or deceleration of the brain within the skull. This alteration compromises the structural and functional process of the scalp, skull, meninges, or encephalon of its vessels. Cranioencephalic lesions can be classified according to their mechanism, morphology and severity. As for the mechanism of injury is classified as closed or penetrating. Closed TCE is associated with automobile collisions, falls and aggressions and will permeate the study of this one that was provided by cyclical accident. The neurological impairment due to TBI is nonspecific as to the possible lesions that cause it. However, the Glasgow Coma Scale (GCS) guides the degree of change that will determine the functional prognosis after ECT [6] and is obtained by observing three parameters: ocular opening, verbal response and motor response [7], being classified in the first six hours after the trauma in mild (GCS 14-15), moderate (GCS 9-13) and severe (GCS 3-8), through the author [6]. In severe TBI, patients are not able to obey orders even after stabilization, the diagnosis must be rapid so that this victim can have a more qualified treatment thus having a better prognosis [8]. It may be associated with a mortality rate of 30%

2018

Vol.4 No.2:73

to 70% [9] and recovery of survivors is marked by severe neurological sequelae and a poor quality of life. In the imaging examination, the diagnosis is complemented, through CT (Computerized Tomography) that suggests the compromise of certain cranial structures/functions, among them, Subarachnoid Hemorrhage; Hemoventricle; Diffuse axonal injury and ectasia of the ventricular system. The classification for HSAt (traumatic subarcenoid hemorrhage) was proposed by Fukuda [10] in his comparative study of late ischemic damage caused by HSAt in relation to that caused by ruptured aneurysms. Subdivided into 3 types: Type 1: Focal HSA in 1 or 2 cisterns. Type 2: Diffuse laminar or thick in 1 cistern + HSA in another topography. Type 3: Diffuse thick or thick in 2 or more cisterns. [8] Diffuse Axonal Injury (LAD) involves axonal injury in the cerebral hemispheres, corpus callosum and brainstem. Refers to loss of consciousness for more than six hours, associated with TBI, without metabolic disturbance or visible expansive lesion on the tomography that justifies the condition. It can be observed in mild, moderate or severe cranial trauma, resulting in axonal edema and disconnection.

According to Khuman [11], the initial lesion of the brain includes deterioration to neuronal cells, glia and vascular structures, leading to other secondary lesions that may still cause greater corruption to the brain tissue, through multiple, molecular mechanisms. Secondary lesions may be due to inflammation, glutamate excitotoxicity, cell necrosis, glial proliferation, mitochondrial dysfunction, apoptosis, and oxygen free radical production and diffuse axonal injury [12-15]. These pathological alterations result in changes in synaptogenesis, dendritic remodeling and neurogenesis in the cortical and limbal regions of the hippocampus, prefrontally [16]. It is suggested that the intervention of the LIB in the muscular structures stimulated the fascias in a complementary way potentiating the drug effect and reducing the inflammatory process in the hemoventrículos, besides the systemic function ILIB to stimulate the photoenteral route in the process of apoptosis without corrupting the organ nocientemente and activation of new cellular synapses [16] in the compromised lobe.

Conclusion

The Low Intensity Laser (LBI/ILIB) used for global patient recovery is considered beneficial [14,15] in a variety of different modalities due to its photobiomodulatory effect, and the most important transcranial laser therapy for TBI is related to neuronal repair and neurogenesis, not only in the formation of new brain cells, but also in synaptogenesis [16], which is the formation of new connections between existing brain cells. And in this study, after the photobiomodulatory intervention, a cognitive and functional restoration took place that conditioned a scientific-literary interpretation ratifying the possible activation of brain cells by the repeated processes of neurogenesis and synaptogenesis, potentiated, in a supportive way, by the photobiomodulator protocol.

References

- De Toni PM, Romanelli EJ, De Salvo CG (2005) A evolução da neuropsicologia: da antiguidade aos tempos modernos. Psicol Arg 23: 47-55.
- Gronwall D (1989) Behavioral assessment during the acute stages of traumatic brain injury, Assessment of the behavioral consequences of head trauma. New York, USA. Alan R Liss Inc 19-36.
- Tate R, Strettles B, Osoteo T (2003) Enhancing outcomes after traumatic brain injury: A social rehabilitation approach, Neuropsychological rehabilitation: Theory and practice. Lisse: Swets and Zeitlinger Publishers, The Netherlands pp: 37-170.
- Xu J, Rasmussen IA, Lagopoulos J (2007) Dissertação de lesão axonal difusa em lesão cerebral traumática grave visualizada usando difusão de alta resolução Na imagem de tensor. J Neuro trauma 24: 753-65.
- Gennarelli T (1983) Cerebral concussion and diffuse brain injuries. In: Cooper P, (ed). Head Injury, Philadelphia: Williams & Wilkins, USA.
- Marshall LF, Marshall SB, Van Berkum CM, Eisenberg HM, Jane JA, et al. (1991) Uma nova classificação de traumatismo craniano com base em tomografia computadorizada. J Neurosurg.
- Elizabeth G (2016) Dra em Ciências (EEUSP), pós-graduada em Administração Hospitalar (UNAERP): e Saúde do Adulto Institucionalizado (EEUSP), especialista em Terapia Intensiva (SOBETI), Estudo de caso, Lesão axonal difusa.
- 8. Brain Trauma Foundation (2000) American Association of Neurological Surgeons.
- Hemorragia subaracnóidea traumática: aspectos clínicos, radiológicos ecomplicações (2008) Traumatic subarachnoid hemorrhage: clinical and radiological aspects and complications. Fonte J bras neurocir 19: 31-36.
- Ogasawara K, Tomitsuka N, Kobayashi M, Komoribayashi N, Fukuda T et al. (2006) Stevens-Johnson syndrome associated with intravenous acetazolamide administration for evaluation of cerebrovascular reactivity. A case report. Neurol Med Chir 2: 1.
- 11. Khuman J (2012) A terapia de luz laser de baixo nível melhora os déficits cognitivos e inibe a ativação microglial após o impacto cortical controlado em camundongos. Neurotrauma 29: 408-417
- 12. Blennow K, Hardy J, Zetterberg H (2012) The neuropathology and neurobiology of traumatic brain injury. Neuron 76: 886-899.
- Kabadi SV, Faden AI (2014) Neuroprotective Strategies for Traumatic Brain Injury: Improving Clinical Translation. Int J Mol Sci 15: 1216-1236.
- 14. Sindi S, Mangialasche F, Kivipelto M (2015) Avança na prevenção da doença de Alzheimer. F1000Prime Rep 7: 50
- Bellou V, Belbasis L, Tzoulaki I, Evangelou E, Ioannidis JP, et al. (2016) Fatores de risco ambientais e doença de Parkinson: uma análise geral das meta-análises. Parkinsonismo Relat Disorder 23: 1-9.
- Kaplan GB, Vasterling JJ, Vedak PC (2010) Brain derived neurotrophic factor in traumatic brain injury, post-traumatic stress disorder, and their comorbid conditions: Role in pathogenesis amd treatment. Behav Pharmacol 21: 427-437.
- 17. http://www.fonoemneuro.com/saiba-mais/traumatismocranioencefalico/