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Fototerapija u liječenju depresije nakon traumatske ozljede mozga

/ Light Therapy in the Treatment of Depression Following Traumatic Brain Injury

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Uvod: Patofiziologija traumatske ozljede mozga dijeli se na primarnu ozljedu, kao posljedicu izravnog mehaničkog djelovanja sile na moždano tkivo, i na sekundarnu ozljedu, koja se razvija postupno nakon inicijalne traume te je dostupna terapijskim intervencijama. Mehanizmi uključuju ekscitotoksičnost, oksidacijski stres, oštećenje krvno-moždane barijere, edem, ishemiju i neuroinflamaciju, uz kroničnu aktivaciju mikroglije i astrocita te pojačano otpuštanje proupalnih citokina TNF-α i IL-6. Nakon traumatske ozljede mozga poremećaji spavanja razviju se u 40 – 50 % i depresija u 30 – 40 % pacijenata. Za dugotrajne poremećaje raspoloženja i spavanja važni su i psihosocijalni čimbenici. Fototerapija ostvaruje antidepresivni učinak preko fotoosjetljivih retinalnih ganglijskih

Introduction: The pathophysiology of traumatic brain injury is divided into primary injury, as a consequence of direct mechanical force acting on brain tissue, and secondary injury, which develops gradually after the initial trauma and is amenable to therapeutic intervention. The mechanisms include excitotoxicity, oxidative stress, blood–brain barrier disruption, edema, ischemia, and neuroinflammation, with chronic activation of microglia and astrocytes and increased release of the proinflammatory cytokines TNF-α and IL-6. After traumatic brain injury, sleep disorders develop in 40–50% and depression in 30–40% of patients. Psychosocial factors are also important for long-term mood and sleep disturbances. Phototherapy exerts an antidepressant effect via intrinsically photosensitive retinal ganglion cells (ipRGCs), which contain the photopigment mela-

stanica (ipRGC), koje sadržavaju fotopigment melanopsin. Impulsi se duž živčanih puteva prenose do suprahijazmatske jezgre hipotalamusa te do drugih moždanih struktura uključenih u regulaciju cirkadijalnog ritma i raspoloženja, perihabenularne jezgre talamusa i lateralne habenule epitalamusa. Prikaz: Pacijentica A.A. (56 g) psihijatrijski se liječi nakon što je 2002. stradala u prometnoj nesreći, u kojoj je zadobila politraumu s ozljedom glave, a u našoj ambulanti godinu dana pod dijagnozom organskog afektivnog poremećaja. MR mozga pokazuje posttraumatsku leziju desno temporalno i hipokampalno. Neuropsihologijskim testiranjem registriraju se kognitivna i bihevioralna odstupanja organske etiologije smetnji, dok je na planu ličnosti i ponašanja naglašena depresivna simptomatologija i promjene osobnosti frontalnog tipa. Primjenom mirtazapina i klonazepama regulira se spavanje i reducira tjeskoba, no zaostaju sniženo raspoloženje, bezvoljnost, zaboravljivost i povremena iritabilnost. Bolesnica se uključi u fototerapiju u jutarnjem terminu tijekom četiri tjedna, a kliničko poboljšanje u obliku stabilnijeg raspoloženja i osnaženih voljnih dinamizama prati se i ocjenskim ljestvicama HAMD-17 i MADRS. Zaključak: Sve veći broj dokaza pokazuje da poremećaji raspoloženja i spavanja mogu perzistirati i više od 20 godina nakon oštećenja moždanog parenhima. Jutarnja fototerapija niskorizičan je i dobro podnošljiv pristup u liječenju depresije kao neuropsihijatrijske posljedice traumatske ozljede mozga.

nopsin. Impulses are transmitted along neural pathways to the suprachiasmatic nucleus of the hypothalamus and to other brain structures involved in the regulation of circadian rhythm and mood, including the perihabenular nucleus of the thalamus and the lateral habenula of the epithalamus. Case report: Patient A.A. (56 years) has been in psychiatric treatment since being injured in a traffic accident in 2002, in which she sustained polytrauma with head injury, and has been followed in our outpatient clinic for one year with a diagnosis of organic affective disorder. Brain MRI shows a post-traumatic lesion in the right temporal and hippocampal regions. Neuropsychological testing reveals cognitive and behavioral deviations consistent with an organic etiology, while at the level of personality and behavior there is pronounced depressive symptomatology and frontal-type personality change. Treatment with mirtazapine and clonazepam regulated sleep and reduced anxiety, but low mood, lack of motivation, forgetfulness, and occasional irritability persisted. The patient was included in morning phototherapy sessions over four weeks, and clinical improvement in the form of more stable mood and strengthened volitional dynamics was monitored with the HAMD-17 and MADRS rating scales. Conclusion: A growing body of evidence shows that mood and sleep disorders can persist for more than 20 years after damage to brain parenchyma. Morning phototherapy is a low-risk and well-tolerated approach in the treatment of depression as a neuropsychiatric consequence of traumatic brain injury.