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Mini Review

Head Injury Treatment (HIT): Perspectives and Comments

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Abstract

Behaviors vary significantly from subject to subject when considering traumatic brain injury (TBI)/ chronic traumatic encephalopathy (CTE) with different clinical/behavioral findings involving genetic factors contributing to autism, OCD tendencies, inhibitions and altered with sensory sensitivities which may impact treatment. Novel recommendations were implemented during a case study of an adult male with blunt force induced acute head injury with altered recall of sense memory attributes to initiate past recognitions, music, literature, and visual arts. Subject responded well with certain medications in the recovery from brain injury including: Doxylamine succinate Acetaminophen, Dextromethorphan, Phenylephrine and Cannabidiol (CBD) but performed poorly with Ibuprofen, Fentanyl, Celecxib, Chlorpromazine, Alprazolam (a Fetanyl derivative), Acetyl fentanyl, and Beta-hydroxy-3-methylfentanyl, Tramadol hydrochloride, and Naproxen. These medication responses may indicate possible changes in drug metabolism due to differences in liver enzymes that break down medications and may require pharmacogenetics testing ostensibly prior to treatment.

Keywords: Traumatic Brain Injury (TBI); Chronic Traumatic Encephalopathy (CTE); Head Injury; Brain Injury; Genetic Testing; Brain Scan; Behavior

Introduction, Background and Discussion

Interventions and evaluation were undertaken including brain scans, neurology evaluation and rest with increased staged structural exercise in our reported subject in order to recover from head injury [1]. Alterations in diet and an increase in fats and supplements including ginger, dark chocolate, sugar, reishi mushrooms, lion's mane mushrooms, green tea, honey, ghee, sage, turmeric, and maple syrup at the subject's request were also incorporated. Weight gain issues were found, suggesting additional nutritional alternatives including ketosis and fatty acid diet supplements that would seem to promote brain health were included, such as: oleic acid and erucic acid (which is prepared from olive oil and rapeseed oil, 1-part glycerol trierucate, as the triacylglycerol forms of oleic acid and erucic acid) and medium-chain triglyceride oil.

Risk factors often contribute to head injury and recovery can be identified [2]. with new genetic testing technology, especially next-generation sequencing (NGS) of DNA collected from the patient using disease specific gene panels or other contributing factors, e.g.

neurodevelopment or functions that could cause autism[3,4]. Other diagnostic tests include brain imaging: PET [5] brain scans, CT, and MRS that allow for characterizing the location and type of brain injury impacting TBI/CTE severity and recovery. Identification of risk factors and source triggers associated with head injury and response may present as PTSD, hypervigilance, acute savant presentation or synaesthesia, lapse in memory, MDD, and anxiety. These may be attributable to predisposing gene variants important to characterize and understanding for the subject with a head injury [4].

A prime target of our recent observation and case study of brain repair in the CNS is neuroplasticity impacted by neurodevelopmental genes. Developmental disorder research and treatment have uncovered that chromatin remodeling, cell proliferation, migration, synaptic networks, long-term potentiation and pharmacogenetics are targets of autism spectrum disorder where hundreds of genes have been identified as playing a role [3,4,6]. These processes are critical for brain plasticity and recovery from injury. Deleterious or damaging variants in identified neurodevelopmental genes could greatly impact brain function, response to injury and repair after TBI. Outcome measures during stages of recovery may be helpful. Disruption of processes in the area(s) of neuron plasticity are characteristic of childhood neurological disorders caused by mutations in neurodevelopment functional genes as proposed including: MECP2, FMR1, TSC1, TSC2, UBE3A, and NF1 as examples that impact dendritic spines and synapse morphology and repair. When these genes are mutated then classical conditions such as Rett, fragile X, tuberous sclerosis 1 and 2, Angelman syndrome, and neurofibromatosis type 1 can present, and all conditions are at risk for autism or similar clinical presentation. Additionally, the TOR1A gene influences cerebellum synaptogenesis, while SHANK3 modulates the expression of receptors for both AMPA and NMDA, and in turn impact plasticity, long-term potentiation and autism [3,4]; hence, could improve head injury susceptibility and recovery. Additionally, the developmental genes are responsible for neuron survival and migration by assisting growth cone formation.

The neurological condition synesthesia was introduced in our reported case study during childhood [1], particularly the subject having an identifiably different relationship with sensory input from the environment prior to head injury. This neurological condition may follow post-traumatic brain injury as well as be observed by several professionals and from the subject's own reports. But, it may be an outcome for some patients following head injury and should be evaluated. Those with synesthesia are not a different class of people but simply have more explicit experiences, as noted by Ward and Simner [7] with a more extreme manifestation of what all individuals experience. For example, asking a question to synaesthetes such as 'What color is A?' would not be a question for those without synesthesia but a question synaesthetes can answer. Synaesthetes have a window into perception and can pair more common letters with brighter colours or higher pitched notes with lighter colours.

Furthermore, our reported patient potentially illustrates genetic markers that may predispose to a different genetic background than most and includes autism spectrum disorder, with or without synesthesia, and may indicate those who are more susceptible to TBI/CTE with variable outcomes. The role of neurogenesis on brain functioning may be relevant to repair of head injury, recovery and outcome via ischemic damage as neonates or in the pediatric context that may relate to genetics. It is agreed that quiescent stem cells exist early in development that are called into action in the face of injury. Studies show that lab animals' aging brains have quiescent stem cells that are kept in a dormant state by inflammatory signals and antagonism of the Wnt pathway. In signaling recovery, microglia cells in the brain play an active role in repairing damage in the CNS including TBI [8] and thus impact recovery and response following a head injury. Immune cells are responsible for clearance of debris during post injury; remodeling occurs along with neurogenesis, angiogenesis and oligodendrogenesis, along with remyelination during the healing process which can be variable from person to person. Furthermore, microglia can play a detrimental role through inflammation or neurotoxic cytokines as well as documented polarization of microglia and M2-like cells that aid in repair processes. An enhancing turnover of microglia either through genetic depletion and replacement or pharmacologic manipulation considerably enhances recovery after TBI through increased neurogenesis mediated by an IL6 immune response requiring more studies in TBI [8,9].

Strain-dependent differences in the inherent capacity for functional recovery after central nervous system (CNS) injury are known with findings highlighting axon growth within the inflammatory response thereby mediating recovery processes. Research on four mouse strains with genetic backgrounds with induced contusion injury to the spinal cord showed better recovery of function in C57Bl/10, B10.Pl research mice, relative to C57Bl/6 or BALB/c44. Spinal cord injury with substantial increase in axonal growth via the lesion area was observed in 129 × 1/SvJ animals and showed an association decreased by chronic inflammatory response relative to C57Bl/645. With fewer macrophages in the lesion of 129 × 1/SvJ animals, more neurons and astrocytes were generated, levels of laminin proved higher, and chondroitin sulfate proteoglycan (CSPG) was lower, suggesting a role in neural development and scar formation [9,10].

A third spinal cord injury reported for 129 × 1/SvJ mice displayed significant corticospinal axon extension relative to C57Bl/625 mice. Axon generation regrowth was enhanced in both strains on a Nogo−/− background which was reflected in an in vitro study of dorsal root ganglia neurite outgrowth with more macrophages found in the lesions of the C57Bl/6 animals. This information could have significance for human research. Hence, two mouse strains with differentially expressed genes were associated with neurite growth, synapse formation, inflammation, and immune response, providing information that may be helpful in human research. Furthermore, oxidative stress studies on research

animal neuronal cultures have revealed strain differences in innate neuronal response reflecting the ability to adapt more efficiently to inflammation [11,12]. but more animal research is needed and its impact on TBI in humans.

In addition, those with neurodevelopmental disturbances such as autism spectrum disorder and synesthesia may have a highly structured, non-random relationship between particular combinations of phonemes rather than graphemes, influenced by a number of fine-grained phonemic property orderings, allophone and phoneme [13,14]. These uncommon experiences have no modern frame of reference and thus fall under the label of spectrum disorder. This misrepresentation of experiences inhibits the full understanding of individuals as we reported and focused on the subject [1] as suggested in the literature [15].

Current protocols may take months to years to identify in its full form, affecting vocabulary acquisition initiated and guided by learned linguistic and conceptual education with rehabilitation. Trigger phonemes appear with corresponding semantic association between the word expression and thought. Innate connections from the perceptual system relay to another, if the other sense is damaged as in the cases of autism spectrum disorder and TBI/CTE, a novel association may be established to make communication of the subject's experience timelier. If this is the case it is possible to influence symbolic/conceptual level of representation to interpret the highest level of communication acceptable to current societal norms. In other words, by addressing the damaged brain and allowing recovery space to be minimally influenced by non-helpful stimuli, we can induce the best-case scenario for treatment and recovery protocol in those with TBI. This information can be applied in the clinical setting impacting services rendered by physical therapy, rest, manipulation, exercise routines, timing and stimulation impacting rehabilitation and recovery of those with head injury requiring more research for optimal recovery.

Perspectives in cognitive neuroscience vary as do philosophies. What is most often agreed upon is the effects of priming and framing on individuals without the tools to navigate the complex experience. Sensations uncovered and disturbed from trauma can produce the best results when recognized and distractions are cleared to avoid unhelpful stimuli: i.e., media overload. A person with both TBI/CTE and features of autism including synesthesia may have a slower recovery time naturally due to neurogenetic factors and response to environmental influences, some recognized and understood while other factors are not clearly understood. When synapse and neuron overwork or rearranged from excessive stimuli, the damaged area with a slower rate of deciphering information is lost in the framing and priming aspect of the treatment scenario [16]. The reasoning is similar to children under the age of 5 years with developing brain processes underway that need mirroring and correction to appropriate the acceptable behavior of that which is in their environment. If a child is exposed to unacceptable behaviors, they will mirror and correct those behaviors to become their current norm of behavior.

Involuntary, automatic, and highly consistent protocols will establish this mechanism to protect the person with a head injury from having to waste valuable time sorting through the harangue of media over stimuli, requiring successful input from physical therapy and rehabilitation with proper and timely interventions. Crossing of the senses where a stimulus received in one sense gives rise to an experience in another is a common phenomenon that needs to be addressed for each individual with a head injury as responses may vary from person to person depending on factors just described. First, there are many apparent variants involving qualities within a single sense, inducing stimuli or inducers that are not restricted to conventional sensory input, meaningful units, numerals and words for relearning that can be used as part of rehabilitation. Meaningful quality for the subject being addressed in turn triggers the experience. Cognitive association, alphanumeric personification, letters and numerals are paired with consistent symbols that reflect the personality traits often associated with communication. Some individuals have weakened skills and thus different responses to types and levels of stimuli that may or may not be helpful during recovery from head injury.

Perceptual quality is experienced individually per subject and needs to be observed and diagnosed by an educated practitioner [17]. Apparent prototypical hearing triggered by spoken language have recently been shown to be triggered by abstract cognitive representations, rather than purely perceptual processing [18]. A unified notion of a condition is being argued that has disparate and isolated qualities. This area of research is in its infancy and many questions presently remain unanswered as normal or abnormal human variation does exist. Triggering stimuli presented as spoken language, grapheme and phoneme are in fact distinct variants, each with their own underlying systems used to communicate, which can operate regardless of the input mode of the triggering stimulus [19]. The range of viable terminology used is variable and simply reflects the lack of terminological agreement throughout the current literature and as a barrier for measuring response and success to treatment for head injury.

This identification would remove the current homogeneity of rehabilitation in head injury protocol and allow a more precise diagnosis of individual differences in functional recovery in the CNS in animals and human subjects that further impact prognosis, treatment, and outcomes depending on each patient's neurological development, skills, and abilities prior to head injury. In vitro modeling of cells and use of organoid cultures in humans, along with complete-organism studies, may help to identify genes and test their interactions and networks with advanced genetic methods that manifest individual variation in recovery from such injuries of the brain.

Conclusion

In summary, advanced genetic testing to identify genes involved

with brain development and function that may contribute to brain damage and response to treatment should be undertaken with more research needed. Brain imaging with CT/MRS/MRI/PET scans taken over time per patient would be needed as well to judge degree of brain injury and response to treatment during the recovery phase superimposed with neurology/behavior/psychiatry assessments to judge success. Research will point the way towards the development of new individual therapeutic approaches Currently, study of neurogenesis has failed to demonstrate proliferating cells in the adult CNS, while some researchers have demonstrated their presence; discrepancies can be attributed to the newness of this area of research. More work is needed using advanced treatment tools for assessment and response to treatment to improve treatment outcomes and measures to enhance quality of life in those with head injury.

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Conflict of Interests

No conflict of interests.

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