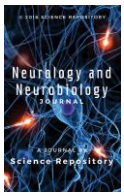

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Case Report and Review of the Literature

Repeat Traumatic Brain Injury Complications with Localized Dissociative Amnesia of Twenty-Two Years

Madeline Aulisio[1], Sandro Pasagic[2], Amanda C. Glueck[2], Timothy J. Ainger[2] and Dong Y. Han[2,3,4] *

[1]Health and Exercise Science Program, Transylvania University, USA
[2]Department of Neurology, University of Kentucky College of Medicine, USA
[3]Department of Neurosurgery, University of Kentucky College of Medicine, USA
[4]Department of Physical Medicine & Rehabilitation, University of Kentucky College of Medicine, USA

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ABSTRACT

A 53-year-old male with history of traumatic brain injury (TBI) presented with localized dissociative amnesia following a second head injury. While memory loss due to TBI is present in the literature, presentations of this type are very rare. Although fully independent in activities of daily living, this patient demonstrated severe deficits in visual memory and processing speed upon neurocognitive evaluation. Effort testing was unremarkable. Increased awareness and study of memory loss following TBI can contribute to enhanced understanding and improved care for patients experiencing these deficits. The authors present this unique case's profile, clinical history, and discuss their findings.

Introduction

Traumatic brain injury (TBI) has been implicated in the development of a broad spectrum of pathologies including deficits in cognitive abilities such as executive function and memory [1-3]. Such deficits may manifest within the first 24 hours after trauma and can lead to severe, lasting decrements in both functional capabilities and quality of life [4-6]. Memory deficits have been previously characterized in the TBI literature; however, most have described prospective memory impairment or other types of memory loss. To our knowledge, few if any studies describe a case of localized dissociative and retroactive amnesia (as opposed to diffuse retroactive amnesia) that was not later attributed to dissociative identity disorder. However, determining the etiology of these disorders remains difficult and controversial [7, 8]. This case report details a patient with TBI who is able to participate in activities of daily living while suffering from long-term episodic memory loss not attributable to dissociative identity disorder. This is atypical and unique because a damaged long-term episodic memory is typically associated with significantly higher functional impairments.

Case Report

I Background

A 53-year-old male with 15 years of education and a history of TBI presented in 2018 with an inability to recall 22 years of long-term episodic memories. He was alert and near baseline functioning during clinical presentation, but neuropsychological testing revealed several deficits.

II Methods

Clinical history was recorded in conjunction with administration of neuropsychological assessments. Patient presented with a history of a single, developmentally unremarkable infant febrile seizure and unremarkable adolescence with no history of special needs education. He previously worked as a police officer and sustained TBI and polytrauma in 1996 when he was assaulted on the job. He was able to return to the police force after recovering to near baseline functioning, but a self-reported heart attack during vision surgery and subsequent

**Correspondence to: Dong Y. Han, Professor of Neurology, Neurosurgery, Physical Medicine & Rehabilitation, University of Kentucky College of Medicine, 740 S. Limestone, J400, Lexington, Kentucky, 40536, USA; Tel: 8593235661; Fax: 8593235943; E-mail: d.han@uky.edu*

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development of double vision led to his retirement from the police force under hazard duty related reasons in 1998. The patient was able to continue employment as a manager of retail and service stores until an esophagogastroduodenoscopy (EGD) in 2018.

While under anaesthesia, the patient may have had a possible seizure episode. Upon waking from anaesthesia in a confused state, he suffered a fall and subsequent blow to the head. Immediately after the incident, the patient believed he was still a police officer in 1996, was unable to recognize familiar people, and suffered from headaches and a host of cognitive deficits. He continues to re-learn significant people and events, but his condition is complicated by a complex medical history that also includes obstructive sleep apnea, exotropia of the right eye, asthma, heart murmur, high cholesterol, hypertension, hyperthyroidism, minimal alcohol consumption, and previous tobacco use. The patient's medication history included amlodipine besylate 2.5 mg, atorvastatin calcium 80 mg, carvedilol 25 mg, mometasone formoterol 100-5 mcg/act, gabapentin 300 mg, levothyroxine sodium 50 mcg, montelukast sodium 10 mg, and albuterol 108 mcg/act. His family history included cardiovascular disease, glaucoma, and malignant neoplasms but was not significant for dementia or neuropsychological disorders.

Although unemployed, patient lived with his wife and adult children and was fully independent in all activities of daily living including driving, finances, taking medications, cooking, shopping, bathing, and cleaning. He and his wife reported that he was functionally back to near-baseline (following the recovery from the 1996 TBI) but continued to describe a significant amount of long-term episodic amnesia and reported anxiety and poor quality of sleep due to the headaches.

Neuropsychological testing in 2019 revealed intact visuospatial skills but notable deficits in other domains. The Montreal Cognitive Assessment v8.2 (MoCA) demonstrated a score of 20/30, below the expected range for the patient's demographic background. Mild deficits in short-term memory recall in verbal domains and severe visual memory deficits (scoring <0.1 percentile) were present. Language, attention, and executive skills displayed variations with mild deficits in their subdomains. Processing speed was also severely impaired (scoring <0.1 percentile). However, the patient's IQ was consistent with his premorbid baseline, suggesting against a functional intellectual decline. Additionally, results revealed from effort measures indicated that the patient's condition is likely an actual presentation of a neurocognitive disorder.

Discussion

The literature suggests that memory deficits are one of the most frequent long-term sequelae of TBI, making it highly likely that patient's TBIs contributed to his memory loss [9, 10]. The literature has indicated that significant memory deficits can be present across varying states of recovery, even in cases of mild TBI; however, typically reduced recall for episodic memory has been more associated with severe TBI [11, 12]. Therefore, it is difficult to elucidate the exact mechanism(s) responsible for it. Additionally, while not a primary etiological concern, his current medications and comorbidities may confound his condition. Amlodipine besylate and montelukast sodium are known to induce headaches [13, 14]. Gabapentin has been shown to induce cognitive decrements, including executive function and acute memory loss [15]. Atorvastatin has also been implicated in bouts of confusion and memory loss [16]. Patient also has obstructive sleep apnea, which as has been shown to contribute to cerebral microvascular damage and loss of episodic verbal memory [17]. All of these factors combined may have contributed synergistically to exacerbate the TBI sequelae. However, the extent of influence that medications and comorbidities had in this patient's case is difficult to extrapolate, especially in the context of his long-term episodic memory loss.

Further limitations of this case report include possible confounds due to previous psychological evaluation in 2018, confounding with practice effects in certain measurement domains. Despite these limitations, this case report describes a very atypical presentation of TBI-induced retroactive amnesia and contributes to the growing literature of memory loss following TBI, potentially enhancing knowledge and improving care for patients.

Funding

None.

Conflicts of Interest

None.

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