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Case Report

Neurobehavioral Changes Resulting from Recurrent Head Injuries

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Abstract

In recent years, there has been a significant focus on the potential link between traumatic brain injury (TBI) due to recurrent head injuries (RHI) and the development of Traumatic Encephalopathy Syndrome (TES), whichcould lead to chronic traumatic encephalopathy (CTE). Recurrent head injuries may cause neurobehavioral changes, significantly impacting an individual's cognitive and behavioral functioning. These changes can lead to various challenges, including difficulties in social interactions, work performance, and overall quality of life. CTE is a progressive disease characterized by Axonal injury tau neurofibrillary tangles (NFTs) and, in some cases, transactive response DNA binding protein 43 (TDP43). This case report discusses a patient who experienced auditory verbal hallucinations (AVH) and cognitive symptoms with behavioral issues due to recurrent RHI. The aim is to investigate this presentation further to examine the relationship between recurrent head injury (RHI) and chronic traumatic encephalopathy (CTE) and to understand the psychological impact of physical brain trauma. A 43-year-old white male with a history of RHI from multiple sports-related concussions was admitted to the psychiatric unit for cognitive difficulties with auditory verbal hallucinations and associated mood symptoms for the past four years, impacting both his sleep and productivity at work. This article will discuss the different types of auditory hallucinations seen in patients with RHI and CTE, along with exploring the evidence to support our determination due to repeated RHI, which led to symptomatology suggestive of CTE. Differential diagnoses will be considered, and evidence is provided to rule them out based on the patient's history. This case report underscores the diagnostic challenges associated with CTE, which can manifest with a spectrum of psychiatric symptoms encompassing mood, cognition, and behavior. The medical diagnosis of Chronic Traumatic Encephalopathy (CTE) in living patients is still a challenge, as effective biomarkers for this condition have yet to be discovered. Although research diagnostic criteria have been proposed, there is no definitive way to diagnose CTE in living patients. Therefore, further investigation is necessary to develop accurate diagnostic tools and effective treatments for CTE.

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1. Introduction

Traumatic brain injury (TBI) happens when an individual receives a significant blow to the head, potentially leading to brain dysfunction [1]. An estimated three million cases of TBI occur every year, with research indicating that as many as 24% of females and 38% of males sustain at least one TBI before reaching the age of twenty-five [2]. Traumatic Brain Injury (TBI) is an intracranial injury that occurs when an external force injures the brain [1]. TBIs can be classified into concussions, closed head injuries, skull fractures, penetrating injuries, hematomas, etc., all of which can lead to a breakdown of neuronal connections [1]. In addition to the potential long-term neurological consequences of TBI, such as headaches, seizures, and visual changes, potentially severe psychiatric manifestations such as depression, psychosis, and suicide have been linked to TBI. It is these presentations of TBI that are the focus of this paper. Achté et al., in a specific study in Denmark, observed 113,906 individuals and found a significant correlation between head injuries and bipolar disorder. The study also found that groups with a genetic predisposition to psychosis or a prior history of neurological pathology had an increased risk of psychosis following a TBI [3]. Fuji and Ahmed 2002 analyzed 69 case studies of psychotic disorder secondary to a TBI and noted that half of the individuals suffered from hallucinations, with auditory hallucinations being the most common [3].

This observed association between traumatic brain injury and psychiatric disorders supports our current understanding of Chronic Traumatic Encephalopathy (CTE), a progressive neurological disease. CTE is characterized by tau neurofibrillary tangles (NFTs) and, in some cases, transactive response DNA binding protein 43 (TDP43). These pathologic changes have shown a bias for perivascular and subcortical areas [4]. Currently, A provisional diagnosis of CTE can be made based on the history of repetitive trauma from contact-based sports along with symptoms of depressed mood, cognitive difficulties, and behavioral symptoms. A definitive diagnosis of the disorder can be made only on postmortem histopathological evidence of NFT. However, proposed research-based clinical criteria are beginning to be applied. These criteria require the presence of at least one core clinical feature with two supportive features for a minimum of twelve months. The core clinical features of Traumatic Encephalopathy Syndrome (TES) include depressed mood, cognitive difficulties, and behavioral issues such as aggression and violence. Supportive features include impulsivity, anxiety, apathy, paranoia, suicidality, headache, motor signs, documented decline in function for a minimum of one year, and delayed onset of clinical features several years after injury [5]. To accurately diagnose and treat traumatic encephalopathy syndrome, it is crucial to determine whether patients meet the criteria. Depending on the clinical criteria they meet, patients can be classified as possible Chronic Traumatic Encephalopathy (CTE), probable CTE, or unlikely CTE if they do not meet the criteria for TES [5].

2. Case Report

A 43-year-old white male with a prior history of ADHD, chronicback pain, and multiple sports-relatedconcussions leading to traumatic brain injury (TBI) sought psychiatric evaluation to address auditory verbal hallucinations, along with their associated mood symptoms. He reported that the hallucinations began four years ago and took the form of the voices of male and female family members. These voices were derogatory and criticizing in nature. His symptoms, although present for the past four years, had worsened in the past two weeks and were now disturbing his sleep and affecting his ability to function at work. Per his statement, the individual experienced a persistent, intrusive auditory phenomenon characterized by non-stop voices. These voices were most noticeable in moments of solitude and ambient noise. Notably, they were most pronounced in the presence of white noise or when the individual had inadequate sleep.

Regarding other psychiatric symptoms, the individual confirmed the absence of any suicidal or homicidal ideations and denied any use of illicit substances. He was taking oxycodone 30 mg every six hours as needed for chronic back pain. He was unsure about any family history of psychiatric disorders such as Schizophrenia or bipolar disorder. In the ED, he was noted to be pleasant and well-mannered, and his physical exam was unremarkable. A urine drug screen was negative for all substances, including oxycodone. He was admitted to the inpatient psychiatric unit later in the evening. Upon arrival, he was noted to be pleasant and cooperative, with mild to moderate anxiety and restlessness. An assessment of the patient's medical record yielded a previous admission four years prior for similar symptoms, resulting in a diagnosis of schizoaffective disorder and substance-induced psychotic disorder due to his history of cocaine and marijuana use. During the previous admission, he was started on Ad-

derall 20 mg for his ADHD symptoms and quetiapine 150 mg for psychosis. Adderall and quetiapine made him anxious and drowsy, respectively, so he discontinued both medications following discharge. He followed up with an outpatient psychiatrist for one year and was started on risperidone 1 mg, which partially improved his symptoms. At a later point, the timing of which is unclear, he discontinued all psychiatric treatment, including risperidone, because he felt it was no longer helping him.

The team performed a thorough history and evaluation of the patient, which showed a complex history. The patient suffered from multiple head injuries while playing football. When questioned in detail about these incidents, he stated that he could not fully recall every one of them but stated that he had 8 to 10 concussions in the past. However, he did mention that his last head injury occurred six years ago, and he lost consciousness for almost an hour, which necessitated a trip to the hospital for further evaluation. Within a year of this head injury, he was in a car accident that resulted in a whiplash injury, and he also lost consciousness and required hospitalization for evaluation and treatment. He had a CT scan done then, which was standard procedure.

In terms of his medical issues, the multiple injuries led to chronic back pain, for which he was prescribedoxycodone and fentanyl patches. He had back surgery related to his football injuries four years prior but was unable to recall the specific procedure. He noted that the hallucinations began shortly after this surgery. He was tapered entirely off oxycodone and fentanyl by the time he started experiencing hallucinations. He started using marijuana as well as cocaine two to three times per week in an attempt to alleviate his hallucinations. However, he reported that they were ineffective and that cocaine exacerbated his symptoms. At the time of his previous admission, he had not used marijuana or cocaine in sixty days. He had started retaking oxycodone as needed for pain but denied any use of fentanyl patches since the time his hallucinations started.

He stated that while his symptoms were intensifying, he began experiencing a sense of paranoia towards his wife, frequent mood swings, and anger outbursts without triggers, along with difficulty sleeping. When questioned about his ADHD, the patient reported that he did not receive a diagnosis for attention-related issues during his childhood. However, following a whiplash injury, he was later diagnosed with difficulties in attention, concentration, and planning. The patient endorsed mood swings along with impulsivity at times. He denied any other symptoms of mania or hypomania at this time. Although he experienced auditory hallucinations and mild paranoia, he did not exhibit any other symptoms of psychosis. The patient received a score of 26 on the Montreal Cognitive Assessment. He lost two points in attention and concentration and two in visuospatial executive function. His score has been consistent throughout his hospital stay.

The patient's history of multiple head injuries prompted the team to order an MRI of the brain without contrast, which showed a probable punctate area of old blood in the left anterior frontal lobe. An EEG did not indicate any abnormal changes. Neurology was consulted for further workup. The neurology team agreed with the MRI findings but stated that it does not need any intervention as it has been chronic. However, they were agreeable to run biomarkers in the context of his presentation to rule out the possibility of traumatic encephalopathy in the context of his multiple head injuries. The patient declined to undergo the recommended cerebrospinal fluid (CSF) level evaluation to assess the possibility of traumatic encephalitis. Furthermore, the patient expressed financial constraints, preventing him from obtaining the recommended positron emission tomography (PET) or FMRI scan. The patient declined any history of dementia in the family.

The patient was started on risperidone 1 mg twice a day since he had responded well to this medication in the past. Due to his mood instability, the patient was also started on Divalproex sodium 500 mg twice a day. He was prescribed mirtazapine as needed for sleep. Over the course of the patient's admission, risperidone was slowly titrated up to 2 mg twice a day. The patient did not respond well to mirtazapine, so he was switched to doxepin 25 mg, which was more effective for sleep. The patient was observed to be experiencing mood symptoms. However, it was determined that neither Bipolar I nor Bipolar II disorder were viable diagnoses, as there were no manic or hypomanic episodes present. A mood questionnaire was administered, and collateral history was taken from the patient's family members to confirm the absence of these symptoms. His previous diagnosis of substance-induced psychosis was deemed unlikely in this case, given his negative urine drug screen.

The patient's history of multiple concussions during his twenty-year football career and a motor vehicle accident, followed by a delayed onset of auditory hallucinations, mood instability, and paranoia, led the team to strongly consider a diagnosis of traumatic encephalitis syndrome and possible chronic traumatic encephalopathy (CTE). The patient was discharged after a six-day hospital stay and advised to attend outpatient follow-up appointments. During his discharge, the patient mentioned improvement in his mood and denied having any suicidal ideations or paranoia. Although he still experienced mild hallucinations, he had learned coping techniques from the therapy team during his hospital stay to manage them. After being discharged from inpatient care, the patient did not comply with the recommended follow-up protocol with the outpatient treatment team.

3. Discussion

Auditory hallucinations are "sensory perceptions of hearing in the absence of an external stimulus" [6]. They can manifest in various disorders, thought disorders, mood disorders, major and minor neurocognitive disorders, epilepsy, delirium, focal brain lesions, cerebral tumors, and infections, including viral encephalitis [7] and, in most cases, are unwanted, uninten-

tional, and intrusive. It is noteworthy that auditory verbal hallucinations (AVHs) can manifest as a consequence of withdrawal or intoxication from certain substances, such as amphetamines, alcohol, and cocaine. It is important to note that these AVHs can be experienced without apparent pathology [8]. In the realm of auditory hallucinations, two primary classifications exist: verbal and non-verbal. When an auditory hallucination contains a language component, it is called a verbal hallucination. These verbal hallucinations can manifest in either simple or complex forms. A misconception regarding hallucinations is that these are unique or primarily seen in patients with schizophrenia. According to a study by Daalman et al. (2011), lower frequency of AVHs, earlier age of onset, positive content of the voices, and a higher degree of control over them predicted the absence of a psychotic disorder in 92% of cases. Additionally, the harmful content of the voices alone predicted the presence of psychosis in 88% of cases [8].

The pathophysiology of auditory hallucinations is complex, and several competing hypotheses exist. Several areas of the brain are thought to play a role in the generation of auditory hallucinations. Jadri et al. (2011) demonstrated that AVHs are associated with increased activity in the frontotemporal areas involved in speech generation and speech perception, as well as within the medial temporal lobe, which is involved in verbal memory. This study supports evidence from functional neuroimaging in other studies that auditory hallucinations result from the abnormal activation of cortical areas involved in auditory processing. Dierks et al. (1999) demonstrated the role of the transverse gyrus of Heschl, where the primary auditory cortex is located, in auditory hallucinations. They found that the same areas of the transverse temporal gyrus activated during hallucinations are activated by tones and verbal auditory stimuli [8].

The patient, in this case, reported experiencing what is best described as functional hallucinations, in whichthe verbal hallucination is triggered by and co-occurs with a real auditory stimulus. These rare hallucinations have been reported in cases of schizophrenia and other psychotic disorders, but little is known about their diagnostic importance and effective treatments. In order to determine the etiology of the patient's functional auditory hallucinations, it is essential to take into consideration his entire history, as well as his associated symptoms of mood disturbance and paranoia. According to the patient, he sustained multiple concussions and a back injury during his twenty-year football career. This historical aspect makes a compelling case for traumatic brain injury as the etiology.

3.1. Traumatic Brain Injury

A study by Sachdev et al. (2001) compared patients who developed schizophrenia-like psychosis (SLP) following brain trauma and found that psychoses had a mean latency of 54.7 months following injury, were usually gradual in onset, and had a subacute or chronic course. Prodromal symptoms such as bizarre behavior and social withdrawal were commonly seen, and depression was present at onset in one-half of the subjects. Paranoid delusions and hallucinations were common, while formal thought disorder, catatonia, and negative symptoms occurred less frequently.

Hallucinations were most often auditory (84%), and the majority (55%) were voices commenting on the patient. Only 22.2% of patients demonstrated flattened affect, avolition, or asociality. A family history of psychosis was the most crucial predictor of SLP following traumatic brain injury, with a duration of loss of consciousness also being significant [9]. The psychoses demonstrated by the patients in the Sachdev et al. case share several features with the psychosis seen in this case, namely auditory hallucinations, paranoid delusions, and agitation/aggression. It proved difficult to assess risk factors in the patient presented in this case, as his psychiatric family history was not apparent. Depression, psychosis, and suicide are severe psychiatric manifestations of TBIs and can be highly disruptive, if not devastating, to a patient's life.

3.2. Substance-Induced Psychotic Disorder

Cocaine can produce numerous psychiatric symptoms, including agitation, paranoia, hallucinations, and suicidal or homicidal behavior. The patient's history, however, indicates that the auditory hallucinations preceded his cocaine use, suggesting an alternative etiology. Additionally, the patient reported that he had not used cocaine in sixty days before his subsequent admission.

3.3. Complex Partial Seizures

Some cases of Complex partial seizures (also called temporal lobe epilepsy) present predominantly withbehavioral changes and may be misdiagnosed as a psychiatric disorder. Temporal lobe seizures typically include a prodromal event, or aura, which deja vu, hallucinations, illusions, mood changes, depersonalization, or oral-facial movements can characterize. Following the aura, individuals may experience compulsive thoughts, automatisms in conjunction with a fixed stare, hallucinations, illusions, and affective symptoms. While the patient, in this case, experienced paranoia and mood lability in the formof angry outbursts, his history did not suggest any distinct ictal experiences or periods of post-ictalconfusion. Furthermore, an EEG was performed during the patient's previous admission four years ago and the recent one from this admission was within normal limits. Although a normal EEG does not entirely rule out a seizure disorder, a regular study makes a diagnosis of temporal lobe epilepsy more unlikely.

3.4. The Link Between Traumatic Brain Injury, Recurrent Head Injury and CTE

In light of increasing criticism of the American National

Football League (NFL) and several associated lawsuits, there has been a significant focus on the potential link between TBI and the development of chronic traumatic encephalopathy (CTE) [10]. There is a known association between head injuries in athletes and the eventual development of CTE. Several former NFL football players, including the well-known athletes Mike Webster and Junior Seau, have exhibited behaviors consistent with these criteria and have shown clear evidence of CTE on autopsy.

Ongoing research is dedicated to elucidating the mechanism by which acute brain injury can lead to the development of neurofibrillary tangles, as the link is purely correlational at this time. Several pathways have been implicated in the link between acute brain injury and CTE, including a stress response by the endoplasmic reticulum, glutamate toxicity leading to mitochondrial failure, and the activation of pro-inflammatory microglia [4]. As previously discussed, currently, CTE can only be diagnosed postmortem through pathological evidence of NFTs.

The proposed TES diagnostic criteria include five general criteria, three core clinical features, and ninesupportive features. The general criteria were selected based on prior literature to maximize sensitivity over specificity. Family members retrospectively reported core clinical features at a minimal rate of 70% among pathologically confirmed CTE cases. The nine supportive features were selected to increase specificity based on clinical features described in the CTE literature [5].

The research diagnostic criteria describe four TES subtypes: (1) a behavioral/mood variant occurring in younger patients; (2) a cognitive variant, which occurs later in life; [3] a mixed variant; and [4] a TES dementia form [5]. Increasing emphasis is being placed on understanding the pathophysiology of CTE and developing a method for premortem diagnosis based on specific biomarkers [11]. One potential diagnostic tool explored is the development of radiopharmaceuticals that could label phosphorylated tau (p-tau). This would allow for the detection of p-tau by positron emission tomography (PET). Using PET in conjunction with magnetic resonance imaging (MRI) modalities, such as structural MRI, functional MRI (fMRI), and diffusion tensor imaging (DTI), is a particular area of interest [9]. Improved diagnostic methods could lead to the development of targeted therapies. Because CTE is diagnosed by postmortem pathology, a definitive diagnosis of CTE cannot be made in this patient.

The patient has a medical history that includes multiple head injuries during his twenty-year football career and involvement in a motor vehicle accident. He experienced delayed onset of symptoms such as mood instability, cognitive dysfunction, paranoia, auditory hallucinations, and brief substance use disorder. The patient displayed more than one core clinical feature and more than two supportive features, meeting the diagnostic criteria for traumatic encephalopathy syndrome [5]. The medical team carefully considered the possibility of chronic traumatic encephalopathy (CTE).

4. Conclusions

This report details a complex case in which no definitive diagnosis can be made at this time due to an inability to diagnose CTE pre-mortem. However, the patient meets the proposed clinical criteria, making a diagnosis of Possible chronic traumatic encephalopathy very likely. As a result of financial constraints, thepatient was unable to receive the necessary biomarkers, FMRI, and PET scans required to assist in diagnosing the potential presence of Probable CTE. In the past twenty years, there has been a significant increase in detecting repetitive concussions in athletes. Raising awareness about TBI, recurrent head injuries, chronic traumatic encephalopathy (CTE) and adopting preventive measures against head injuries, particularly in high-risk sports and activities, is of paramount importance. It is crucial to prioritize the safety of individuals engaged in such activities and to take proactive steps toward promoting a culture of safety and well-being. Further research into the complicated pathophysiology of CTE and efforts to develop in vivo diagnostic methods, such as PET and MRI, is imperative, along with making it accessible to the public. It is anticipated that the progression of diagnostic modalities in the future will pave the way for the creation of effective and targeted therapies to aid individuals who suffer from CTE.

Abbreviations

TBI	Traumatic Brain Injury
RHI	Recurrent Head Injury
TES	Traumatic Encephalopathy Syndrome
CTE	Chronic Traumatic Encephalopathy
NFT	Neuro Fibrillary Tangle
TDP 43	Trans Active Response DNA Binding Protein 43
AVH	Auditory Verbal Hallucinations
ADHD	Attention Deficit Hyperactivity Syndrome
PET	Positron Emission Tomography
fMRI	Functional Magnetic Resonance Imaging
EEG	Electroencephalogram

Disclosures

Human subjects

Consent was obtained or waived by all participants in this study. This study was approvedby WVU Institutional Review Board issued approval 2110443979.

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All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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Julie Bosley: Resources, Validation, Writing – review & editing

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Conflicts of Interest

The authors declare no Conflicts of Interest.

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