

# A Disabled Army Veteran with Severe Traumatic Brain Injury and Chronic Suicidal Ideation

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A 63-year-old disabled Army veteran was involuntarily admitted to our inpatient facility after asking his sister to buy him antifreeze so that he could kill himself. He had been living with his sister, and he required total care for his activities of daily living because he was a paraplegic (requiring the use of a wheelchair). The patient endorsed command auditory hallucinations to kill himself that were congruent with his own motivations to commit suicide because he was tired of living with the crippling physical and mental deficits resulting from the prior trauma. Many years after an honorable discharge from his

Army service, he was the victim of a horrific assault by a group of assailants that resulted in multiple injuries, including severe head trauma and third-degree burns after being set on fire with gasoline. Follow-

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ing the assault, he was in a coma for many weeks and was intubated during much of that time. Months after his recovery and discharge from the intensive care unit, he began to experience overt psychotic symptoms including visual and auditory hallucinations with pronounced paranoia, anxiety, and severe depressive symptoms including chronic suicidal ideation.

Since the attack, this patient had multiple psychiatric admissions, including several in the few months preceding admission to our facility. Prior to admission to our facility, the patient had been diagnosed and treated for schizoaffective disorder and had been prescribed multiple psychiatric medications

(combinations of antipsychotics and antipsychotics), including olanzapine, mirtazapine, divalproate, and, most recently, monthly injections of paliperidone palmitate, none of which provided any amelioration of his mood or psychosis symptoms. He confessed that he had in the past resorted to the use of heroin, methamphetamine, cocaine, and alcohol as a means of self-medication to ameliorate his symptoms but was currently in full sustained remission from substance abuse. His chronic suicidal ideation, which had increased in frequency over the years, was the reason for his multiple inpatient psychiatric admissions.

Thorough medical testing completed in our facility did not suggest anything abnormal. Tests showed normal results for serum alpha-fetoprotein, vitamin B12, folate, human immunodeficiency virus, hepatitis, and syphilis serology (using the rapid plasma regain test and Venereal Disease Research Laboratory test), urine toxicologic screen, blood alcohol, complete blood count, general chemistry, and lipid panel. Endocrine function tests for pituitary, thyroid, adrenal glands, and the hypothalamus were also unremarkable. Brain imaging including magnetic resonance im-

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aging and computed tomography scan showed patchy hypoattenuation suggestive of hypoperfused cortical tissue as well as cerebellar and mammillary body volume loss, likely as a result of ischemia consequent to his severe traumatic brain injury (TBI).<sup>1</sup>

The patient had pronounced symptoms meeting criteria for recurrent and severe major depressive disorder, such as depressed mood, hopelessness, suicidal ideation, aberrations in sleep patterns, low appetite, severe anxiety, and anhedonia. In addition, within a system of delusional disorganized thinking and bizarre visual and auditory hallucinations we were also able to discern symptoms not seen in other purely psychotic patients. Specifically, he endorsed recurrent, involuntary, and intrusive distressing memories of the traumatic event, dissociative reactions such as flashbacks, avoidance of strangers, and intense distress. He also vocalized feelings of guilt and shame as well as persistent fear, mistrust, and anger. His trauma-related alterations in arousal and reactivity had, during the years since the assault, worsened to include severe mood swings and self-destructive behavior culminating in numerous suicide attempts. His hypervigilance was notable particularly in the context of group settings and often included a highly reactive startle response and with concomitant anxiety and paranoia. The patient maintained that he had chronic sleep disturbances as a result of his hyperarousal in the context of ongoing visions and

## DIAGNOSIS

### Posttraumatic Stress Disorder

The patient presented here is a case of a noncombat veteran who developed posttraumatic stress disorder (PTSD) symptoms after an assault. He later developed psychotic symptoms and was then diagnosed with schizoaffective disorder. Upon a closer investigation however, we were able to isolate significant PTSD symptoms from what appeared to be a fixed system of psychotic hallucinations.

The patient was given citalopram, due to the known efficacy of selective serotonin reuptake inhibitors in PTSD and its low level of P450 interactions, as well as its relatively benign side-effect profile.<sup>2</sup> The antipsychotic olanzapine was switched to oral asenapine at a dose of 5 mg at night (because olanzapine was deemed not to be effective). Depakote was continued at a nightly dose of 1,000 mg and gabapentin was decreased to 300 mg 3 times a day due to oversedation. The citalopram was titrated to a dose of 40 mg daily and was tolerated well. His other medications included amlodipine at 10 mg daily, two tablets of docusate/senna twice a day, hydrochlorothiazide at 12.5 mg daily, and lisinopril at 40 mg daily.

As part of his recovery, daily group therapy was instituted along with elements of cognitive-behavioral therapy and intensive supportive therapy to help him cope with his cognitive disso-

nance. Over a 2-week inpatient period, his mood and other symptoms stabilized and began to show signs of improvement, including an improved positive affect. His suicidal ideation that was a daily occurrence decreased to such an extent that he rarely mentioned taking his life during his therapy sessions and in rare conversations with his fellow patients. Importantly, he began to manifest a sense of future-oriented thinking. Having attained a measure of stability with a significant attenuation of his symptoms, he was discharged and transferred to another facility closer to his sister's place of residence.

## DISCUSSION

PTSD affects approximately 6.8% of people in the United States.<sup>3</sup> Complex trauma increases risk for developing PTSD. Many PTSD patients suffer from comorbid psychiatric conditions such as depression, anxiety, substance use, and TBI. With PTSD and TBI alone, there is an overlap of cognitive symptoms that are difficult to appropriate to any single condition. These may also change or evolve with time, resulting in the PTSD diagnosis being overlooked, particularly in the case of severe TBI.

Providers often know to screen for PTSD in context of mild combat-related TBI. We would, however, like to draw attention to severe TBI cases (which by definition requires a diminishment or loss of consciousness; namely a Glasgow Coma Scale [GCS] score of 3-8) that can and do harbor significant PTSD symptoms.<sup>4,5</sup> Assessing for

PTSD symptoms in light of severe TBI is important for two reasons:

1. The *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition (*DSM-5*)<sup>6</sup> has removed the requirement for a patient's response: namely, "intense fear, helplessness, or horror," during the trauma. In other words, the patient can have a severe TBI, be unconscious or near unconscious, and still suffer the consequences of PTSD as a result of the trauma.

2. Assessing and recognizing symptoms of PTSD allows for treatment applications such as psychotherapy and a wider range of psychopharmacology that are usually not applicable in patients otherwise diagnosed solely with severe TBI.

The *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition, text revision (*DSM-IV-TR*)<sup>7</sup> criteria originally required that "The person's response involve[d] intense fear, helplessness, or horror" (ie, an awareness that would be unlikely in a state of impaired consciousness and impossible during an unconscious state as is the case in severe TBI). As stated before, severe TBI by definition requires a GCS of between 3 and 8. The *DSM-IV TR* "response" criterion no longer exists in *DSM-5*.<sup>6</sup> This, therefore, paves the way toward considering PTSD even within the context of severe TBI where there may not be such a "response."

It had traditionally been assumed that PTSD cannot develop after severe TBI because of the pervasive loss of consciousness that occurs after a severe TBI precludes encoding of the traumatic experi-

ence.<sup>8</sup> However, numerous case studies and cohort studies have described PTSD after severe TBI.<sup>9-11</sup> This is in spite of the fact that these patients suffer from lengthy periods of amnesia to the extent that they do not recall details of the trauma. Some of the proposed theories to explain PTSD in the context of patients who have lost consciousness during the trauma include the following:

1. Fear-conditioning: namely extreme sympathetic arousal resulting in the release of stress neurochemicals such as norepinephrine and epinephrine, mediating a consolidation of the memory.<sup>12,13</sup>

2. Memory reconstruction, in which trauma patients reconstruct traumatic memories as a representation of what occurred during the period of impaired consciousness.<sup>14-16</sup>

3. Post-amnesia resolution, in which patients experience the aftermath of the trauma in the field or while being treated, including fear for their safety, severe pain, or anguish at their disability.<sup>17</sup>

TBI can result from a variety of mechanisms, including hypoxia, as was the case with this patient. Imaging also revealed loss of brain volume, probably as a result of his ischemic injury. Additionally, it has been suggested that PTSD itself causes regions of the cortex to lose brain volume through gray matter structural damage. This has been shown in a recent study.<sup>18</sup>

The capacity to regulate the fear reaction may be impaired after TBI if the neural networks involved in the regulation of anxiety are damaged, as was demonstrated in this

patient's symptoms.<sup>19</sup> In addition, neuronal damage, as had occurred in this patient, has been noted to be both a predictor of increased vulnerability of acquiring PTSD as well as a presumed result of stress.

TBI is difficult to distinguish from PTSD. The two disorders have some characteristics in common, and the difference between the symptoms of TBI—including problems with memory, attention, concentration, headaches, sleep disturbances, irritability, and depression—and those of PTSD can be subtle.

The patient's auditory and visual hallucinations can be hypothesized along two fronts. First, that his psychosis was a result of TBI. A recent meta-analysis supports an increased risk of schizophrenia after TBI, with a larger effect in those with a genetic predisposition to psychosis.<sup>20</sup> Second, that his psychotic experiences were related components of a multifaceted form of psychopathology that encompassed his PTSD. Vivid flashbacks of PTSD can be similar to, or the same as, hallucinations, and the intense fear and re-experiencing symptoms of PTSD can be similar to a psychotic delusion. There is accumulating evidence that people who have primary PTSD may experience psychotic symptoms without meeting criteria for schizophrenia, a mood disorder, or other psychotic spectrum disorder. Thus, a multifaceted and simultaneous treatment of the PTSD, including psychotherapy and psychopharmacology, appears to be the more efficacious route<sup>21</sup> than just focusing on the patient's

psychosis through the utilization of psychopharmacology.

### CONCLUSION

PTSD and TBI can coexist because brain injuries may be sustained in traumatic experiences. The recent changes to *DSM-5* now obligate clinicians to look for evidence of PTSD symptoms even in cases of severe TBI.<sup>6</sup> Integral to the discussion are the similarities between the symptoms accompanying each condition. Despite the overlap, some symptoms such as re-experiencing and avoidance are characteristic of PTSD and not the effects of TBI, whereas persistent disorientation and confusion are more specific to TBI (neurocognitive effects) than to PTSD. Questions worth considering are the potential treatment benefits of PTSD not applied in severe TBI, and that this oversight may indeed be occurring routinely because of a clinical approach still based on *DSM-IV-TR* criteria as well as the similarities of the symptoms.

Our hope is that clinicians are receptive to symptoms of PTSD within the context of severe TBI because of the increased availability of treatment choices for PTSD, particularly in light of the magnitude of PTSD cases, which far outnumber cases of severe TBI alone, and the concomitant allocation of resources, both governmental and private, in ongoing research for improved treatment toward ameliorating symptoms. Examples

of treatment that someone with TBI would ordinarily not receive whereas a patient with a PTSD diagnosis may benefit from include cognitive processing therapy, prolonged exposure therapy, and eye movement desensitization and reprocessing.

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