

CASE REPORT

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Can dissociative amnesia be a residual symptom of prolonged complex post-traumatic stress disorder?

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Abstract

Background Dissociative amnesia, a disorder characterized by impairments in multiple memory areas, is frequently associated with trauma. Complex post-traumatic stress disorder (CPTSD) is marked by mood dysregulation, negative self-concept, and impaired interpersonal relationships, in addition to the classic symptoms of post-traumatic stress disorder (PTSD). The relationship between CPTSD and dissociative amnesia, as well as whether CPTSD should be considered a dissociative subtype, remains uncertain in the literature. Individuals diagnosed with CPTSD tend to exhibit higher levels of dissociative symptoms than those diagnosed with PTSD.

Clinical presentation We present the clinical report of a 42-year-old male who, after a car accident, exhibited core symptoms of PTSD along with symptoms of self-organization disorders. While these symptoms persisted, the patient developed dissociative amnesia years after the trauma. Neuroimaging studies, psychometric tests, reviewed hospital records, and clinical interviews were conducted to speculate on the differential diagnosis of organic psychiatric conditions and potential diagnoses. The possible relationship between dissociative amnesia and complex post-traumatic stress disorder was examined.

Conclusion This case demonstrates the complexity of differentiating dissociative amnesia from organic conditions. Discussing the possible shared mechanisms between CPTSD and dissociative amnesia could contribute to a better understanding of both conditions.

Keywords Trauma, Mental disorder, Psychiatry, Amnesia, Complex post-traumatic stress disorder, Dissociation, Dissociative amnesia

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Introduction

The World Health Organization's (WHO) 11th Classification of Diseases (ICD-11, 2022) introduced complex post-traumatic stress disorder (CPTSD) as a significant update to mental and behavioral disorders. ICD-11 PTSD is defined by three symptom clusters: re-experiencing the trauma in the present, avoiding traumatic reminders, and a persistent sense of threat. CPTSD includes these symptoms along with three additional clusters: emotional dysregulation, negative self-concept, and disturbances in relationships, collectively termed "disorders of self-organization" (DSO) [1].

Dissociation, recognized as a key feature of the response to psychological trauma [2], generally refers to failures in integrating biopsychosocial experiences [3]. Trauma-related dissociation can manifest as a wide range of psychoform and somatoform symptoms, including amnesia, flashbacks, identity dissociation, and psychogenic loss of sensory or motor functions. These symptoms are common in individuals with PTSD and CPTSD [4]. This report presents a patient with dissociative amnesia and CPTSD symptoms developing years after the trauma.

Case presentation

A 42-year-old married man with two children, living in Trabzon, presented with memory loss. He had been working in iron and aluminum joinery before his symptoms appeared. He struggled to recognize loved ones and remember much of his past. Prior to the onset of amnesia, the patient experienced fever, abdominal pain, and nausea, but these symptoms resolved quickly. The next day, while at work, he was unable to perform familiar tasks and had to ask his child for help. He remembered his family, but details like his wedding day, the birth of his children, and other significant life events were lost. He knew that his parents had passed away, but he could not remember why, where, or when. He remembered some of his close relatives but did not recognize others. For example, of his three uncles, he only remembered the one who had died; the others seemed as if he was seeing them for the first time in his life. Of the five friends he had been close to before the incident, he could only remember two. His memory was selective, recalling some relatives but not others, and he could only remember a car accident in 2012.

Following the onset of memory loss, he temporarily forgot how to drive and struggled with basic tasks. Over time, he relearned how to drive, but occasionally forgot he could. The patient also left his job and took up lumberjack work, which he had never done before. His wife had to monitor him closely, as he often forgot daily events. In the evening, he would forget what he had talked about in the morning and what he had done throughout the day.

The patient would either tell his wife what he planned to do in the near future or write it down on small pieces of paper. Because he would forget to take his insulin and his meal times, he kept track of them by making daily notes. He could remember and complete most of the work on his own, but he kept forgetting to change the machine's program. There was no family history of dementia, and he had no history of substance abuse or childhood trauma. He had recently experienced a significant decrease in sales in his business and had frequent arguments with his wife due to economic problems. In fact, he had been experiencing difficulties in his interpersonal relationships for a long time. The patient had a history of intensive care and splenectomy following a traffic accident in 2012. He was driving the car at the time of the accident, and besides himself, his wife and children were also present. The others sustained injuries that could be treated with simple medical intervention. After the accident, he began experiencing flashback episodes, sensitivity to reminders of the event, irritability, mood changes, recurrent nightmares about the accident, and accompanying insomnia. Initially, he was prescribed mirtazapine 15 mg/day, but he spontaneously discontinued the treatment and neglected follow-up care. After a while, he started experiencing additional outbursts of anger and fluctuations in affect. His tolerance threshold towards those around him seemed to have decreased. His attitude towards his wife and children changed. He stopped seeing his close friends, felt insecure around people, and was unable to establish intimacy with them. He blamed himself for not being able to rebuild his life after the accident and thought he was a burden to his loved ones. In 2013, the patient attempted suicide by hanging twice, two months apart. In 2014, a combined treatment with sertraline and mirtazapine was started with the diagnoses of PTSD and depressive disorder for his ongoing symptoms. In trauma-focused supportive psychotherapy, the focus was on making the relationship between his current complaints and past traumatic experiences more visible, and on addressing the negative effects of these experiences on him. The aim was for the patient to gain awareness of his current situation, make sense of his experiences, and pave the way for the construction of a new, non-stigmatized identity. After the treatment, there was a significant decrease in trauma-related flashback episodes, sensitivity to trauma reminders, and recurrent nightmares with an accident theme. However, irritability, mood changes, sleep problems, negative self-perception, and emotional dysregulation persisted. The patient continued to attend follow-up visits for a short period before discontinuing them. He did not seek medical support again until the most recent period of complaints. During this time, there was a significant decline in his social and occupational functioning.

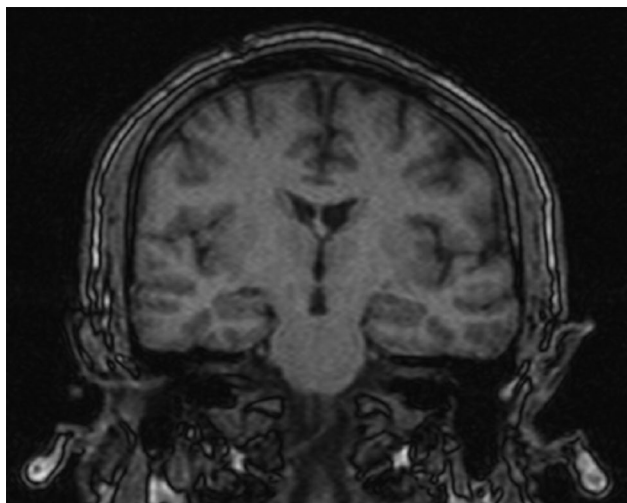


Fig. 1 T1 weighted brain MRI scan

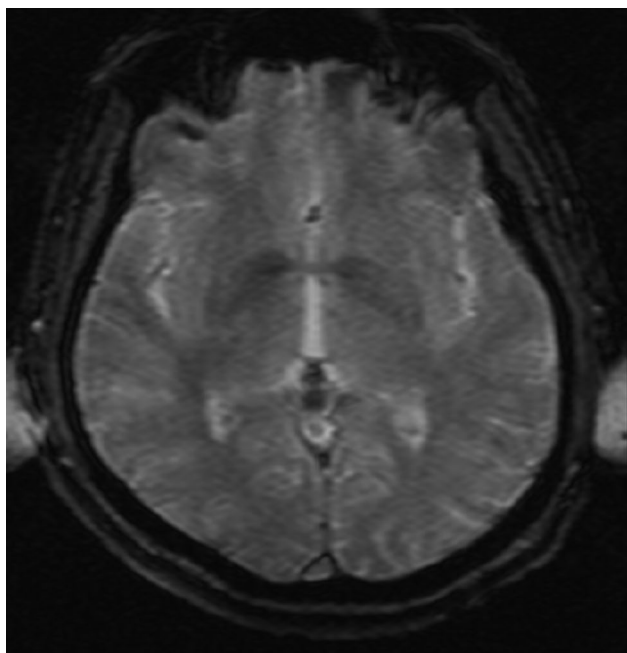


Fig. 2 T2 weighted brain MRI scan

Audiovisual Number Sequence Test, Stroop Test, Clock Drawing, Proverb Interpretation and Binary Similarities Subtests were administered to evaluate attention and executive functions; Auditory Verbal Learning Test, Benton Test, Bender Gestalt Test were administered to evaluate memory; MMPI was administered to evaluate personality traits and psychopathology. According to the results of neuropsychological tests, mental flexibility, abstract thinking, semantic and phonemic fluency were intact, while focused attention, attention maintenance and response inhibition skills were impaired. According to the MMPI test, the patient was severely depressed with anxiety and agitation and may have borderline

personality traits. No significant pathological findings were found in neuroimaging studies (Figs. 1 and 2) (brain CT, brain MRI and brain PET CT), dementia markers (HIV, syphilis, hepatitis, CBC, biochemistry, urine tests), toxicity markers and rheumatologic markers, which were performed with the recommendations of the neurology department to exclude organic etiology. The patient with known comorbidities of hypertension and diabetes mellitus was consulted to cardiology and endocrinology departments. As a result of the follow-up and treatment process in both departments, it was concluded that no pathology was detected that could explain the patient's current picture.

Discussion

When evaluating the patient's current symptoms in terms of memory types, it is evident that autobiographical memory is largely impaired. Semantic memory remains partially intact, while procedural memory is impaired concerning some acquired skills. Recall, recognition, and retrieval related to short-term memory also showed lower-than-expected performance. Two critical issues arise: the organic-psychiatric distinction of amnesia and the relationship between PTSD and amnesia. The onset of amnesia following symptoms of infection, the patient's anxiety due to memory loss, and the features of both anterograde and retrograde amnesia support an organic pathology. However, there are reports suggesting that people with dissociative amnesia rarely exhibit anterograde memory impairment [5, 6]. Additionally, a comprehensive evaluation for organic causes, particularly the possible presence of an infection, did not reveal any significant findings, and these symptoms have not recurred. Factors suggestive of psychiatric pathology include a normal neurologic examination, absence of significant findings in neuroimaging studies, mood changes and long-term psychiatric treatment, absence of impairment in other cognitive areas affecting daily functioning, and the reversibility of some memory-related symptoms [7].

In understanding dissociative amnesia, it is helpful to comprehend the current categorization of memory based on dimensions of time and content [8]. However, memory types do not have distinct boundaries. For instance, there are variations in the processing of public semantic information. When public event information is linked to personal experiences (i.e., has autobiographical meaning), it is incorporated into autobiographical memory and is thus more vulnerable to autobiographical memory impairments [9]. Autobiographical -semantic memory is typically preserved in selective or more restricted cases of dissociative amnesia. However, as observed in our case, it may present with severe (generalized) retrograde dissociative amnesia, marked by combined deficits in episodic-autobiographical memory and autobiographical-semantic

memory. Additionally, autobiographical-semantic memory components may deteriorate in conditions like evolving mental amnesia or neurodegenerative dementias [10]. In our case, the amnesia may eventually progress into an early neurodegenerative disorder, which was not detectable through neuro-psychological and imaging studies at the time of assessment, but could become evident later. Procedural skills tied to another memory domain can often be relearned, though emotional and motivational factors may affect this process [11, 12]. Symptoms and disease progression also vary, which might indicate a heterogeneous disorder. Furthermore, accompanying clinical features differ across cultural groups [13]. Each of these aspects may contribute to the variation in the clinical manifestations, triggers, and course of dissociative amnesia. The condition may present with clinical profiles resembling organic pathologies. It has also been posited that there is a continuum, perhaps even a fundamental similarity, between organic and psychogenic amnesia, rooted in a shared brain mechanism underlying both phenomena [14]. The findings of mixed physical and psychological antecedents draw attention to the similarities between dissociative amnesia and transient global amnesia (TGA), now recognized as a legitimate neurological disorder, moving towards bridging “the traditional gap between neurological and psychiatric approaches in defining a disease process.” In TGA, a psychologically or environmentally triggered situation can cause sudden somatic changes at the brain level (such as vasospasms or metabolic stress), which may later result in changes in the CA1 sector of the hippocampus. Similar mechanisms might also apply to dissociative amnesia, potentially leading to the chronicization of this condition [15]. These findings underscore the need for a comprehensive evaluation of amnesia through a multidimensional and interdisciplinary approach.

Interpreting the patient’s amnesia findings in favor of dissociative symptoms raises the question of whether this is a coincidental case. The patient’s inability to recall autobiographical memories—characteristic of dissociative amnesia—and inability to suppress recollection of the traumatic event—typical of PTSD—suggest that the two conditions may overlap [16]. Given the patient’s long-term PTSD symptoms, this possibility is strengthened. The presence of psychiatric comorbidities, emotional dysregulation, impulsive and self-destructive behaviors, depersonalization, and derealization, along with the absence of borderline features in past history, support the diagnosis of CPTSD. The sudden nature of the trauma, as opposed to cumulative trauma, and the absence of identity crisis, fugue, and alteration point toward a dissociative type of PTSD [17]. Furthermore, the presence of symptoms grouped under DSO, including emotional dysregulation, negative self-concept, and disturbances in

relationships, reinforces the PTSD diagnosis. Participants with CPTSD show significantly greater impairment in functioning compared to those with PTSD, and CPTSD has been reported to involve higher levels of dissociative symptoms compared to PTSD [18, 19].

Traumatic events not only increase the risk of PTSD but also depression. Trauma-induced depression, which meets the criteria for major depressive disorder (MDD), can stem directly or indirectly from trauma. Non-adherence to treatment regimens, as observed in our case, is a common phenomenon in mood disorders like PTSD and MDD [20]. Understanding the predictors of non-adherence is crucial for clinicians to identify high-risk patients and address the underlying causes of nonadherence. Important case-specific predictors include younger age (under 40 years), patient beliefs, illness severity, treatment-related side effects, and a poor therapeutic alliance [21]. Younger patients may doubt the efficacy of medication, view it as dangerous, or feel that they can control their illness independently. Weak insight and patient beliefs about their treatment and illness have been shown as negative predictors of medication adherence in various mood disorders. In the long term, side effects of antidepressants—such as sexual dysfunction, weight gain, and anticholinergic symptoms—are among the most common reasons for treatment discontinuation in MDD patients [22]. Adverse events remain the most frequently reported reason for early discontinuation within the first three months [23]. The doctor-patient relationship plays an essential role in this process, as factors like inadequate patient education, inappropriate medication prescriptions, insufficient dosages, communication style, and lack of follow-up care remain within the physician’s control [24].

PTSD can influence treatment adherence through various mechanisms stemming from PTSD-specific behavioral and cognitive symptoms. For instance, avoidant behaviors, characteristic of PTSD, may cause patients to avoid medications that evoke thoughts of their mortality or, more specifically, their initial trauma [25]. This avoidant tendency could partly explain the link between PTSD and medication non-adherence [26]. PTSD has also been associated with a perceived lack of personal control over illness [27] and a foreshortened sense of the future [28, 29]. Consequently, patients might neglect preventive medication due to a fatalistic belief that such interventions will have little impact and are thus not worth pursuing. Additionally, a sense of futility—believing that the future holds no potential for positive experiences—may further contribute to medication non-adherence. Moreover, cognitive impairments frequently seen in PTSD [30, 31] are linked to difficulties in adherence. For example, cognitive dysfunctions associated with PTSD may lead to unintentional non-adherence; in a recent study, nearly

half of PTSD patients reported routinely forgetting to take their medication [32].

An important discussion about CPTSD is its similarity to borderline personality disorder (BPD), a DSM-5 diagnosis. One notable similarity is emotional reactivity. Although anger, suicidal, and self-harming behaviors—considered part of emotional reactivity—are more central to BPD, our case does not exhibit changes in identity and relationships typical of BPD. In BPD, individuals often alternate between extremely positive (idealization) and extremely negative (devaluation) views of themselves and others. In contrast, CPTSD is characterized by a persistent and consistent negative self-concept and a deep distrust of others [33]. In our case, the central feature of CPTSD is “hypervigilance to harm,” whereas BPD is marked by “hypersensitivity to perceived abandonment.” [34].

Structural dissociation theory proposes that trauma disrupts the integration of various lived experiences into the personality system, with more complex trauma leading to more severe dissociation [3]. This theory posits that dissociation is an “important under-recognized feature” of CPTSD and predicts that CPTSD involves more severe dissociation than PTSD [35]. According to this theory, individuals with complex trauma-related disorders, especially CPTSD, experience a dissociation of their personality into distinct parts. This structural dissociation can result in various secondary symptoms, including flashbacks, nightmares, amnesia, altered states of consciousness, and phobic avoidance of dissociated emotions. Trauma survivors may use dissociation to avoid overwhelming emotions or experiences, which is highly associated with CPTSD symptoms such as emotional dysregulation [36]. From this perspective, the patient’s dissociative amnesia occurring years later may be a consequence of the long-term effects of DSO symptoms.

Conclusion

Traumas are experiences that can profoundly impact individuals’ mental health and trigger psychological conditions such as PTSD and CPTSD. Both conditions are associated with severe symptoms that impair functioning and can significantly affect a person’s life. While CPTSD and PTSD both result from traumatic experiences, they represent distinct psychological conditions with different origins, symptoms, and impacts. This case report contributes to the literature by highlighting dissociative amnesia in a patient with residual CPTSD symptoms. The theoretical and nosological significance of dissociation in CPTSD invites further discussion on whether CPTSD could be conceptualized as a dissociative disorder or if a dissociative subtype of CPTSD exists. It also suggests that CPTSD as defined in ICD-11 may provide a more descriptive case formulation than DSM-5, which

offers a broader conceptualization of PTSD with some overlapping symptoms of CPTSD. The report supports empirical evidence that CPTSD’s core features are clinically and conceptually distinct from PTSD [18, 37, 38]. Additionally, it highlights the complexity of distinguishing dissociative amnesia from organic conditions. This study aims to understand that dissociative amnesia varies in presentation, indicating it as a heterogeneous disorder that requires comprehensive evaluation. It explores how untreated or inadequately treated PTSD can evolve into CPTSD, which manifests with more severe impacts on psychosocial functioning, alongside dissociative amnesia throughout the clinical course. The study emphasizes the importance of recognizing that CPTSD has its own unique dynamics, necessitating the development of different treatment strategies and addressing the factors that complicate treatment adherence and its management. A potential shortcoming is the absence of a structured scale to assess clinical symptoms during the patient’s evaluation. It should be noted that, since this is a case report, the generalizability of the findings is limited. Therefore, additional supporting research is needed to establish the inferences made within a cause-and-effect context. Further investigation into the common mechanisms between PTSD and dissociative amnesia, as well as the role of dissociation in the assessment and management of CPTSD symptoms, is warranted. Exploring CPTSD symptoms in the context of dissociative amnesia by mental health professionals may enhance healthcare outcomes.

Abbreviations

| | |
|--------|---|
| CPTSD | Complex Posttraumatic Stress Disorder |
| PTSD | Posttraumatic Stress Disorder |
| DSO | Disorders of Self-Organization |
| MMPI | Minnesota Multiphasic Personality Inventory |
| BPD | Borderline Personality Disorder |
| WHO | The World Health Organization |
| ICD-11 | 11th Classification of Diseases |
| MMD | Major Depressive Disorder |
| TGA | Transient Global Amnesia |

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İzzet Çağrı Metin and Selman Yıldırım wrote the main manuscript text and Aykut Karahan prepared Figs. 1 and 2. All authors reviewed the manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethical approval

The patient provided consent for the case report through an informed consent form.

Competing interests

The authors declare no competing interests.

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