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Management of dissociation in high-functioning autism adolescents

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Abstract

Background: Dissociation is an umbrella term for trans, depersonalization, derealization, and dissociative amnesia. Dissociation is usually seen in association with trauma, but it can arise in individuals with autism spectrum disorder (ASD) even in the absence of trauma. The interactions between sensory sensitivities, emotional dysregulation, and heightened stress responses in ASD may predispose patients to dissociative experiences, which significantly impair functioning and complicate psychiatric care.

Case presentation: We report the case of an 18-year-old female with high-functioning ASD, attention deficit hyperactivity disorder (ADHD), and generalized anxiety disorder who presented with recurrent dissociative episodes. These were characterized by emotional withdrawal, a subjective sense of detachment, and difficulties maintaining present awareness, particularly during stress and overstimulation. Her clinical history was notable for sensory sensitivities, learning difficulties, and a prior depressive episode. At presentation, she endorsed persistent sleep disturbance, fatigue, poor appetite, low self-worth, impaired concentration, and depressed mood. Current medications included duloxetine, lisdexamfetamine, brexpiprazole, and L-methylfolate.

Conclusion: Dissociation in ASD presents unique diagnostic and therapeutic challenges, particularly in non-trauma contexts. A combined psychosocial and pharmacological approach, tailored to individual symptom profiles, may optimize outcomes. This case underscores the importance of recognizing dissociation as a clinically significant phenomenon in ASD and calls for further study into targeted management strategies.

Keywords: Autistic spectrum disorder, dissociation, high function autism, neurodivergent disorder

Introduction

According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) [29], ASD is defined as a persistent deficit in social interactions and communications and at least 2 restricted or repetitive behaviors (rigid routines, intense interests, sensory abnormalities, or repetitive actions) [29]. These symptoms usually begin early in childhood which progressively impair their functioning, and most importantly, cannot be explained by other developmental disorders [30]. ASD was further redefined as a spectrum which also included sensory abnormalities like hypo and hyper reactivity [30]. The precise etiology of ASD remains unclear, but current research highlights the contributions of genetic and environmental factors to the development of autistic behaviors [11]. Environmental influences may interact with genetic predispositions, resulting in abnormal brain growth, altered neuronal development, and disrupted functional connectivity [11]. Recent increases in ASD prevalence are partly explained by enhanced clinical tests and diagnostic tools [25, 26].

Dissociation is a defense mechanism marked by an alteration in consciousness that causes a disconnection between the self and its components [2]. It may present as depersonalization, trance states, derealization, and dissociative amnesia, as well as identity inconsistencies [8]. Various psychiatric conditions can feature dissociation, including depersonalization / derealization disorder, unspecified dissociative disorder, dissociative amnesia, and dissociative identity disorder (DID) [1].

While trauma is the precipitating factor ^[9], dissociation is also associated with heightened stress and anxiety ^[3]. In individuals with autism spectrum disorder (ASD), the etiological factors are often amplified, contributing to a higher level of dissociation in this group ^[3]. Dissociation compromises psychological functioning and interferes with everyday activities ^[10]. Consequently, treatment approaches typically focus on enhancing coping mechanisms ^[10]. Research supports the conceptualization of dissociation as a coping strategy, given its role in preventing further elaboration of distressing emotional states ^[15].

This case report describes an 18-year-old high-functioning autistic adolescent who presented with recurrent dissociative episodes, experiences of emotional withdrawal, and detachment unrelated to trauma.

Case Presentation

An 18-year-old female with a history of high-functioning autism spectrum disorder (ASD), attention deficit/hyperactivity disorder (ADHD), and generalized anxiety disorder presented with recurrent dissociative episodes characterized by episodes of emotional withdrawal, a subjective sense of detachment from her surroundings, and difficulties maintaining present awareness, particularly during periods of overstimulation and stress.

Developmentally, she was born full term to a mother with type 2 diabetes mellitus managed by diet. There were no developmental delays noted, though she had difficulties with fine motor skills. Eye contact was not problematic, though she was noted to experience emotions with greater intensity than her peers. From early childhood, she demonstrated sensory sensitivities, including aversion to bathing, tooth brushing, and grooming.

Even though she had learning difficulties, she completed her education in a general education setting without individualized educational accommodations. She struggled with procrastination, impaired time management, and test-taking difficulties.

She reported a history of major depressive episode the previous year, characterized by emptiness, loneliness, and self-harm urges without suicidal attempts. In the months following her mother's cancer diagnosis, her anxiety escalated and dissociative symptoms intensified. She endorsed ongoing symptoms, including daily sleep disturbances, chronic fatigue, poor appetite, low self-worth, impaired concentration, and persistent depressed mood. She denied a history of mania or psychosis.

At presentation, she was taking duloxetine, L-methylfolate, lisdexamfetamine (Vyvanse), and brexpiprazole (Rexulti). Mirtazapine was part of her regimen, but a plan was made to taper it gradually due to limited benefit.

Discussion

The neurobiology of ASD is unique, evidenced by structural and connectivity differences in the brain, including alterations in cortical thickness (Ecker *et al.*, 2015) ^[31]. The neural dysfunction is brought on by the imbalance between excitatory glutamatergic and inhibitory GABAergic signaling, along with abnormalities in synaptic proteins ^[32], and these disruptions are thought to contribute to either hypersensitivity or hyposensitivity seen in ASD ^[33]. Despite accumulating evidence regarding the role of endogenous biomarkers in ASD pathophysiology, early detection of the disorder remains a significant challenge ^[12, 25, 26].

Dissociation can occur in Autistic spectrum patients even in the absence of trauma history, reflecting the complex and constant interplay between the sensory processing challenges, stress regulation, and frank neurodevelopmental differences ^[3]. A comprehensive review by Reuben and Parish in 2022 discussed how dissociative symptoms in ASD can be linked to a number of risk factors, such as heightened anxiety, emotional dysregulation, disability stigma, and shame, rather than trauma exposure alone ^[3].

Genetic and biological associations with dissociation have been identified in numerous studies ^[26]. Monoamine-related genes (5-HTT, COMT), neuropeptide receptors (OXTR), and neural plasticity factors (BDNF) have been reported, showing linkages with trauma and other associated symptoms, including dissociation ^[26]. These findings are further supported by clinical evidence indicating that serotonergic medications demonstrate limited efficacy in treating dissociation ^[4]. In addition, the trans-experienced group exhibited elevated levels of dopamine and norepinephrine ^[4]. Despite some inconsistencies across studies, these results can inform the development of future pharmacological interventions.

There is no certified regime for ASD with dissociation ^[5]. Although dissociation is presented with many other psychiatric issues, the autistic population's detachment needs more delicate plans and follow-up ^[23]. The mainstream of dissociation management is divided into two aspects: acute management and long-term risk modification ^[22]. For the early stage, a proactive approach for the removal of dissociation is most important ^[22]. This is based on the result that a longer duration of symptoms is related to polypharmacology and aggressive management. And the next step, prevention of relapse, is needed to focus on, because symptoms shift, and it's a recurrent relapse ^[22].

Psychotherapy is a cornerstone of dissociative disorder treatment and is the primary care pathway ^[27]. However, the various types of dissociative disorders warrant different treatment plans and a full evaluation of what kind of dissociative disorder is needed ^[5]. For dissociative amnesia, removal from stress or threatening situations, administration of benzodiazepines, barbiturates, as well as psychotherapeutic hypnosis are often taken into management strategies ^[5]. In the case of dissociative fugue, drug-assisted therapy, hypnosis, and psychodynamic psychotherapy are integral to its therapeutic treatment ^[5, 19]. For depersonalization, therapeutic approaches include self-hypnosis, systematic desensitization, relaxation techniques, psychodynamic psychotherapy, cognitive behavioral therapy, and controlled dissociation, among a plethora of treatments ^[5, 27]. Trance-based dissociation approaches are relatively simple, implementing emotional awareness and concrete assistance ^[5].

Another vital approach within treatment for dissociation is grounding skills ^[28]. Grounding skills help patients regain focus from disturbing emotional sensations and shift their attention to a more present state ^[28]. Methods of cognitive awareness and sensory awareness are common in the grounding of dissociative patients ^[5].

Limited prevalence and intertwined comorbidities make studies of pharmaceutical approaches for dissociation challenging. Conventionally, medication is recommended for co-occurring psychiatric manifestations like anxiety and depression, not focusing on dissociation ^[14]. A textbook introduced evidence that a selective serotonin reuptake

inhibitor (SSRI), tricyclic antidepressant (TCA), and monoamine oxidase (MAO) inhibitor affected reducing underlying mood instability and anxiety^[18]. Additionally, mood stabilizers, beta blockers, clonidine, and benzodiazepines have been employed to alleviate trauma-related and comorbid symptoms^[7]. Benzodiazepines, commonly used to reduce anxiety, have demonstrated a significant reduction in comorbid anxiety symptoms, but are cautioned to increase dissociative symptoms^[7].

Despite no medication directly treating dissociation, pharmacological agents such as Paroxetine and Naloxone have shown promising clinical results as the pharmacotherapy group saw a significant reduction in dissociative symptoms compared to the placebo group^[6]. Among these medications, lamotrigine is the most well-studied. The first RCT of outpatient settings, conducted in 2011, reported that lamotrigine reduced dissociative symptoms by 50% compared to placebo^[13]. This study gained recognition because the participant exhibited depersonalization without any other comorbidities^[13]. Naloxone, an opioid receptor antagonist, has been reported to have a moderate effect on dissociative symptoms in Borderline Personality Disorder in a previous case report^[17]. Then, physicians and researchers should pay more attention to not only the detachment feeling but also its comorbid psychosocial aspect simultaneously.

Similar case presentations have been presented, such as a 13-year-old boy with ASD who presented paroxysmal episodes of wandering during stressful situations. Emotionally distressing events such as dentist appointments, arguments, and loud noises often prompted these dissociative fugue episodes. While his episodes were characterized by disorientation and inability to convey his attentions, he never wandered into dangerous situations and reported no harmful self-inflicted behavior. Unlike our case presentation, he heard auditory hallucinations of a male voice ordering him to hurt himself and others^[19]. Aside from this case presentation, additional presentations of dissociation in pediatric ASD include a presentation of a 17-year-old male with 6 weeks of dissociative amnesia^[21] and a 15-year-old female who underwent a continuous 6-day episode in which she assumed a new identity and displayed behaviors and actions she had never partaken in beforehand^[20]. These cases show the diverse ways in which dissociation presents in ASD.

Our case adds value by demonstrating dissociation in a high-functioning adolescent with ASD in the absence of trauma, emphasizing the need for increased clinical awareness and tailored management based on individuals.

Conclusion

Dissociation is a common symptom in neurodivergent disorders like ASD. Usually, dissociation is associated with trauma, although it may occur in settings with no trauma exposure. Though the underlying mechanism of dissociation remains unclear, the consensus on treatment is a multidisciplinary approach: Psychosocial and biological treatment.

Based on a strong rapport and education, cognitive and behavioral modulation with social factor management is needed to sustain a stable condition. Along with psychotherapy, appropriate medication for comorbid psychiatric symptoms can be beneficial. Further studies are needed to better characterize its clinical features, establish

evidence-based interventions, and guide long-term management in neurodivergent populations

Disclosure

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Conflict of Interest

None.

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