Depersonalization/derealization disorder: Epidemiology, clinical features, assessment, and diagnosis

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INTRODUCTION

Depersonalization/derealization disorder (DDD) is characterized by the persistence or recurrence of depersonalization and/or derealization that cause clinically significant distress or impairment in the presence of intact reality testing [1].

DDD has a prevalence of approximately 2 percent and is associated with significant morbidity, but often goes undetected or misdiagnosed, leading to delays in treatment.

This topic discusses the epidemiology, pathogenesis, clinical manifestations, course, and diagnosis of DDD. Our approach to selecting among treatments for DDD is discussed separately. Individual medications and psychotherapies for DDD are also discussed separately. (See "Approach to treating depersonalization/derealization disorder" and "Depersonalization/derealization disorder: Psychotherapy".)

DEFINITIONS

Depersonalization — Depersonalization is a persistent or recurrent feeling of detachment or estrangement from one's self. An individual experiencing depersonalization may report feeling like an automaton or as if in a dream or as if watching himself or herself in a movie. Depersonalized individuals may report the sense of being an outside observer of their mental

processes or their body. They often report feeling a loss of control over their thoughts, perceptions, and actions.

Derealization — Derealization is a subjective sense of detachment or unreality regarding the world around them (eg, individuals or objects are experienced as unreal, dreamlike, foggy, lifeless, or visually distorted).

EPIDEMIOLOGY

Prevalence — Transient experiences of depersonalization and derealization are very common in the general population [2]. In a nonurban United States sample, the one-year prevalence of depersonalization symptoms was approximately 20 percent [3]. In a large normative sample of German adolescents, 12 percent reported clinically significant depersonalization [4]. Approximately half of college students in another United States study reported experiencing an episode of depersonalization in the past year. Adolescents and young adults appear to be at the highest risk for reporting such symptoms, possibly because their sense of self is still fluid and developmentally unstable.

Transient depersonalization symptoms, which may be primary or secondary to another condition, are more common than depersonalization/derealization disorder (DDD). Studies from several countries including the United States, Canada, United Kingdom, Turkey, and Germany have shown a lifetime prevalence for DDD in the range of 0.8 percent to 2.8 percent [5-7], comparable to that of other psychiatric disorders such as schizophrenia, bipolar disorder, and obsessive-compulsive disorder.

Risk factors — DDD has been found to be equally common in males and females in clinical samples [8,9]. In up to half of cases there is no readily identifiable proximal precipitant [8]. Common risk factors for DDD include the following:

- Acute and chronic trauma or severe stress.
- Psychiatric conditions, most commonly anxiety or depressive disorders – Chronic depersonalization and derealization symptoms meeting diagnostic criteria for the disorder have also been reported to begin during severe or prolonged episodes of a mood or anxiety disorder, which may destabilize the sense of usual expected self and in itself act as a powerful internal stressor [8]. This pattern occurs in approximately one-third of all patients with DDD. Typically, in this scenario, the depersonalization and derealization symptoms set in at some point of escalation of the original disorder, but persist or worsen

on a long-term basis after the precipitating disorder has spontaneously remitted or been treated, taking on an independent course, a “life of its own.”

- Alexithymia – Additionally, there is evidence of elevated rates of alexithymia in those suffering from DDD, with nearly half of participants demonstrating high or moderate alexithymia in one study [10]. This difficulty in identifying and processing emotions is likely not simply an outcome of the disorder, but is a risk factor for its genesis, as alexithymic individuals have greater difficulty with the emotional processing of traumatic and stressful events.

- Fearful attachment style – There is a high prevalence of insecure attachment in DDD, with a preponderance of fearful attachment style [11].

- Substance use disorder.

Chronic depersonalization and derealization has been reported in as many as one-third to one-half of individuals experiencing chronic traumatic or highly stressful situations. Examples of these situations include (see 'Role of trauma' below):

- Childhood abuse or neglect
- Unexpected death of a loved one
- Growing up with a seriously mentally ill parent or caregiver
- Sexual orientation/identity conflicts in adolescence and young adulthood
- Serious relationship or work stressors

Comorbid conditions — Based on clinical samples, DDD appears to have high rates of comorbidity with other psychiatric disorders. As an example, in a study of 117 United States adults with DDD [8], the lifetime prevalence of comorbid disorders was as follows:

- Unipolar depression, 73 percent
- Any anxiety disorder, 64 percent
- Obsessive-compulsive disorder, 21 percent
- Avoidant personality disorder, 23 percent
- Borderline personality disorder, 21 percent

PATHOGENESIS

Although the pathogenesis of depersonalization/derealization disorder (DDD) has not been established, it is generally assumed that the syndrome becomes manifest in individuals who have an inherent dissociative diathesis combined with psychological and/or chemical stressors.

Role of trauma — Acute depersonalization and derealization are quintessential responses to acute trauma (such as a motor vehicle accident) or other life-threatening experiences [12,13], speculated to be hardwired into the brain so that a sense of distance and detachment from the traumatic event can facilitate surviving and negotiating the event without experiencing overwhelming, disorganizing emotion (ie, “going through the motions”) [12,13]. However, acute depersonalization and derealization symptoms occurring in the above circumstances typically clear within minutes, hours, or days, and do not become chronic. Chronic depersonalization and derealization symptoms qualifying for the disorder are more common among persons experiencing chronic traumatic or highly stressful situations such as verbal or emotional abuse or neglect during childhood [10,14].

Other traumatic antecedents include growing up with a parent or caregiver who had severe mental illness, traumatically struggling with sexual orientation or identity, and experiencing the unexpected death or suicide of a family member or close friend. Sexual abuse and physical abuse are less common antecedents of DDD than emotional maltreatment but clearly occur (see above). Numerous other chronic life stressors (interpersonal, financial, or occupational) have been associated with onset or exacerbation of DDD. Examples include loss of a relationship or divorce, or job loss, or social ostracization. It should be noted, however, that acute, short-lived traumas or stressors can trigger the onset of the disorder as well. (See 'Risk factors' above.)

The traumas reported by DDD patients are generally of a different nature than those typically reported by patients with other dissociative disorders such as dissociative identity disorder [10,14], centering on emotional maltreatment rather than sexual abuse. (See "Dissociative identity disorder: Epidemiology, pathogenesis, clinical manifestations, course, assessment, and diagnosis".)

In a cumulative sample of 93 adults with DDD compiled across several research studies, application of established clinically significant childhood maltreatment cutoff scores for the Childhood Trauma Questionnaire revealed that 63 percent of participants had experienced at least one form of maltreatment, predominantly emotional abuse and/or neglect (55 percent). Additionally, physical neglect was endorsed by 40 percent, physical abuse by 27 percent, and sexual abuse 20 percent [15].

Organic precursors — Common organic risk factors for depersonalization and derealization include seizures, mild to moderate head injury, brain tumors, and sleep apnea [16]. In such cases the diagnosis of the disorder is not made, as it requires the exclusion of such “organic” underpinnings.

Substance use is known to precipitate chronic depersonalization in some people [17]. (See 'Differential diagnosis' below.)

These individuals can thus be conceptualized as having a neurobiological or genetic vulnerability to the onset of chronic depersonalization and derealization (ie, DDD), after drug use. A large survey study has shown that the characteristics and course of the condition do not differ between drug-induced and non-drug-induced cases [17].

Neurobiology — Several neurotransmitter systems, brain regions, and functional circuits have been associated with depersonalization and derealization symptoms with some consistency and are summarized below [18].

- **NMDA (N-methyl-D-aspartate) system** – NMDA receptors are widely distributed in the cortex, hippocampus, and amygdala, and mediate associative processes. The NMDA antagonist ketamine induces a profound dissociative state in healthy subjects that is distinct, both phenomenologically and in the implicated brain pathways, from the psychotomimetic effects of ketamine [19,20]. Ketamine has complex pharmacodynamics and effects on other neurotransmitter systems, including Kappa opioid receptors that could relate to the acute induction of depersonalization and derealization [21].

- **Endogenous cannabinoid system** – Cannabis has been experimentally shown to induce depersonalization in healthy volunteers, with a pronounced component of temporal disintegration, and particular brain regions have been implicated [22]. Cannabinoids block NMDA receptors at sites distinct from other noncompetitive NMDA antagonists [23] and therefore their dissociative effect may be partly mediated via NMDA antagonism, as well as by the endogenous cannabinoid system.

- **Kappa opioid agonists** – The illicit drug salvia, which acts as a selective kappa opioid agonist, has been found to precipitate chronic depersonalization [17,24]. Nonselective antagonists such as naloxone, naltrexone, and nalmefene, which have varying affinity for the kappa receptor, have been reported to diminish dissociative symptoms.

- **Serotonin agonists** – Hallucinogens act as agonists at serotonin 5HT2A and especially 5HT2C receptors. Experimental challenge studies with the partial 5HT2A and C agonist m-CPP have demonstrated the induction of depersonalization in a mixed group of social phobia, borderline personality disorder, and obsessive-compulsive disorder participants [25], the induction of flashbacks and dissociative symptoms in a subgroup of posttraumatic stress disorder patients [26], and the induction of dissociation in healthy volunteers [27].

- **Autonomic hypo-reactivity** – There is some evidence for autonomic hypo-reactivity in DDD [28,29].

- **Hypothalamic-pituitary-adrenal (HPA) axis** – The HPA axis has been investigated in DDD with conflicting findings. While one study reported nonsignificantly lower basal salivary cortisol in DDD subjects compared to healthy ones [30,31].

- **Neuroimaging** – Neuroimaging studies show evidence for the following in DDD:

  - Sensory integration occurring in the temporoparietal junction, in particular in the right hemisphere, has been implicated in out-of-body experiences [32]. Altered activity in this area has been shown by positron emission tomography scans [33].

  - Functional magnetic resonance imaging studies have implicated hypoactivation of the limbic system, in particular the insula, driven by heightened prefrontal inhibition, in the hypoemotionality and overthinking of DDD [34-36].

  - Specific brain regions are involved in the experience of dissociation, such as the retrosplenial cortex [37]. Additionally, evidence suggests that the sense of self and one's physical location within a spatial environment is processed in the posteromedial cortex [38]. Trauma-related derealization has been identified as involving increased ventromedial prefrontal cortex activation and its decreased resting-state connectivity with the cerebellum and orbitofrontal cortex [39].

  - Deficient representation of visceral-afferent neural signals at the brainstem level [40].

  - Evidence of less effective arousal suppression under cognitive stress [41] and a higher sympathetic tone [42].

  - Distinct brain correlates for anxiety, depression, somatization, and dissociation symptoms [43].

  - Grey matter alterations [44,45].

  - Heightened activation in several brain areas associated with self-processing [46].

The relationship between genes and DDD is not known. An analysis of proband-based family history data in 117 subjects with DDD found a heritability rate of only 5 percent [8].

Psychotherapeutic models — Conceptual models for DDD have been developed from cognitive-behavioral and psychodynamic perspectives; these are described separately. (See

"Depersonalization/derealization disorder: Psychotherapy", section on 'Psychodynamic therapy' and "Depersonalization/derealization disorder: Psychotherapy".)

CLINICAL MANIFESTATIONS

Patients experiencing depersonalization and derealization often have great difficulty putting their impalpable experiences into words. Unfamiliarity with these symptoms may lead individuals to fear that they may be taken for "crazy" or will become crazy. This is especially common in those raised in homes with mental illness, those prone to hypochondriasis and ruminations, and those who fear that a drug ingestion has caused irreversible brain damage. Clinicians' typical focus on comorbid symptoms such as mood and anxiety often contributes to underdiagnosis or misdiagnosis of depersonalization/derealization disorder (DDD).

Symptoms, although quite specific, may sound vague or metaphorical rather than clear subjective experiences, such as "feeling dead," "everything is unreal," "I don't feel I'm the one doing anything," "it's like I'm watching a movie," etc. The symptoms are typically extremely distressing, at times crippling, and are associated with major morbidity and some mortality. The affectively flattened and robotic demeanor that these patients often demonstrate can also fool the clinician into not recognizing the extreme emotional pain of the condition. It is not uncommon, for example, for patients to wonder whether it would really matter if they died as they already feel dead. Suicide attempts are associated with DDD; however, given the high rates of comorbid depression and other psychiatric disorders in DDD, the relationship between suicidality and DDD remains unclear [8,17]. (See 'Comorbid conditions' above.)

Two factor analytic studies of the core symptomatology have reported five discrete symptom clusters common in patients with DDD [47,48]:

- Unreality of self, defined as feeling detached from one's physical body, mind and thoughts
- Perceptual alterations, which encompasses visual, tactile, and somatosensory distortions
- Emotional numbing, characterized by blunted affect, pain and volition
- Anomalous subjective recall, which consists of disrupted experiences of time and related imagery
- Alienation from surroundings, which comprises symptoms of derealization

COURSE

Depersonalization/derealization disorder (DDD) can have an episodic, relapsing/remitting, or chronic course. In a series of 117 cases of patients with DDD, approximately one-third of individuals had an episodic course; one-third had a continuous course from time of onset; and one-third initially had an episodic course that over time became continuous [8].

Many patients with chronic depersonalization and derealization experience marked impairment in occupational, social, and personal functioning. In a study comparing 223 patients with DDD with a sample of patients with depressive disorders without comorbid depersonalization/derealization, the two groups differed markedly in clinical presentation and course. DDD patients were younger, significantly more male, had a longer illness duration, earlier age of onset, and tended to show greater functional impairment [49].

Available research suggests that onset is most commonly in late adolescence or early adulthood, with onset of the disorder rarely occurring as late as the fourth or fifth decade of life [8]. Onset may be very sudden or insidious. Most patients with DDD are initially treated for secondary anxiety and mood disorder symptoms; the primary nature of the DDD may not be recognized until later. Once chronic depersonalization has set in, new traumatic or stressful events or episodes of other illness may lead to exacerbations of the symptoms.

Depersonalization or derealization that follows acute traumatic or stressful experiences or intoxication is often transient and remits spontaneously [3]. In our clinical experience, depersonalization accompanying mood or anxiety disorder episodes commonly remits with timely treatment of these conditions, but runs the risk of becoming more chronic and difficult to treat over time. In some cases, dissociation persists long after the initial precipitants and can become more refractory to treatment.

ASSESSMENT AND DIAGNOSIS

Assessment — We undertake a diagnostic assessment including detailed psychiatric history, medical history, and mental status examination and cursory physical examination in all individuals with suspected depersonalization/derealization disorder (DDD). A thorough medical and neurologic evaluation is not routinely necessary; however, we conduct these evaluations in all patients presenting with atypical symptoms or illness course, of older age, or a general medical status or history that raises concerns about the differential diagnosis. (See 'Differential diagnosis' below.)

We obtain brain imaging (eg, computed tomography or magnetic resonance imaging) and ambulatory electroencephalogram (EEG) in cases of diagnostic ambiguity or in the presence of

atypical symptoms (eg, blackouts, unexplained subtle motor or sensory symptoms, intermittent episodes that are difficult to correlate with any precipitants), seizures, or a family history of epilepsy. For example, we obtain brain imaging, EEG, and basic metabolic panel in individuals.

- Age of 40 or greater; onset is unusual at this age (see 'Risk factors' above)
- Focal findings on neurologic examination
- History of head injury, seizure disorder, or any brain lesions
- History of any medical illness that could be associated with altered brain structure and function (eg, autoimmune disease)
- Suspicion of sleep apnea
- Suspicion of Lyme disease or other infectious processes
- Family history of neurologic or related diseases
- Any atypical symptoms such as numbness, tingling, or head pressure sensations

We obtain urine toxicology screening if history or examination suggests the possibility of a substance use disorder, although patients with DDD will typically refrain from any further substance use once the disorder sets in and even become phobic of such drug use [17].

Rating scales — We typically use the Dissociative Experiences Scale (table 1). The scale has several items pertaining to depersonalization/derealization experiences whereas the endorsement of other items pertaining to amnesia and identity alteration should be essentially very low or absent [50-52]. More specific to the disorder, the Cambridge Depersonalization Scale is a self-report questionnaire comprised of 29 items which rate both frequency and duration of depersonalization/derealization experiences (table 2). A total score of 70 has been shown to reliably differentiate DDD patients from those with various mood, anxiety, or neurologic disorders [53].

We find self-report questionnaires to be helpful in affirming the diagnosis of DDD. This may be especially useful for clinicians who are not as extensively familiar with dissociative symptoms.

Diagnostic criteria — DDD is diagnosed using the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (DSM-5-TR) diagnostic criteria [1]:

- A. The presence of persistent or recurrent experiences of depersonalization, derealization, or both:
  - 1. **Depersonalization** – Experiences of unreality, detachment, or being an outside observer with respect to one's thoughts, feelings, sensations, body, or actions (eg, perceptual alterations, distorted sense of time, unreal or absent self, emotional and/or physical numbing).
  - 2. **Derealization** – Experiences of unreality or detachment with respect to surroundings (eg, individuals or objects are experienced as unreal, dreamlike, foggy, lifeless, or visually distorted).
- B. During the depersonalization or derealization experiences, reality testing remains intact.
- C. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
- D. The disturbance is not attributable to the physiological effects of an illicit substance, medication, or another medical condition (eg, seizures).
- E. The disturbance is not better explained by another mental disorder such as schizophrenia, panic disorder, major depressive disorder, acute stress disorder, posttraumatic stress disorder (PTSD), or another dissociative disorder. (See 'Differential diagnosis' below.)

Depersonalization and derealization that follow traumatic or stressful experiences or intoxication commonly remit spontaneously. Our clinical experience suggests that it may not be useful or effective to diagnose and treat DDD unless the symptoms continue for a month or possibly longer. The DSM-5-TR diagnostic criteria for DDD do not specify a minimal duration of symptoms [8].

Differential diagnosis — A detailed psychiatric history and mental status examination can distinguish DDD from other mental disorders and medical conditions. Depersonalization or derealization can present as a symptom of other mental disorders. We differentiate these by the following symptoms or symptom patterns:

- **Schizophrenia or other psychotic spectrum disorder** – Individuals with DDD have intact reality testing whereas in individuals with schizophrenia or other psychotic spectrum disorder reality testing is typically not intact. Additionally in prodromal psychotic syndromes, perceptual alterations are often accompanied by magical ideation-type

interpretations of the symptoms whereas this is not seen in DDD. For example, a person with DDD may feel like they are a zombie, but knows they are not a zombie. A study comparing perceptual alterations and magical ideation in patients with DDD and healthy volunteers found that people with the disorder only had elevated perceptual, but not cognitive, distortions [54]. (See "Schizophrenia in adults: Clinical features, assessment, and diagnosis".)

- **Panic disorder** – While both panic disorder and DDD may present with symptoms of derealization or depersonalization, individuals with panic disorder have recurrent unexpected surges of intense fear or discomfort that reaches a peak within minutes and are associated with symptoms such as palpitations, sweating, trembling, chest pain or dizziness. These are not typically seen in DDD. (See "Panic disorder in adults: Epidemiology, clinical manifestations, and diagnosis".)

- **Major depressive disorder** – We differentiate DDD from depressive disorder by the presence of depressed mood, diminished interest or pleasure in most or all activities, changes in sleep pattern (insomnia, hypersomnia), fatigue, feelings of worthlessness or guilt, and psychomotor changes that are typically seen in major depressive disorder. These symptoms are not typically seen in DDD.

- **Acute stress disorder** – While both DDD and acute stress disorder may have dissociative symptoms, in acute stress disorder the symptoms last for three days to one month and are associated with symptoms such as intrusions, avoidance of distressing memories or external reminders of the traumatic event, and symptoms of altered arousal (eg, sleep disturbance, hypervigilance, exaggerated startle reflex.) These are not commonly seen in DDD. (See "Acute stress disorder in adults: Epidemiology, clinical features, assessment, and diagnosis".)

- **Posttraumatic stress disorder** – We distinguish PTSD from DDD by the presence of intrusion symptoms (ie, flashbacks, persistent avoidance of stimuli associated with the traumatic event), and symptoms of altered arousal that are commonly seen in PTSD. (See "Dissociative aspects of posttraumatic stress disorder: Epidemiology, clinical manifestations, assessment, and diagnosis".)

- **Other dissociative disorders** – Individuals with DDD do not have distinct personality states as in dissociative identity disorder. Individuals with DDD do not have an inability to recall important autobiographical information as in dissociative amnesia. (See "Dissociative identity disorder: Epidemiology, pathogenesis, clinical manifestations, course, assessment, and diagnosis".)

- **Personality disorders** – Individuals with personality disorders or DDD may manifest symptoms such as depersonalization or derealization. However, in personality disorders, the enduring pattern of inner experience or behavior is manifested by changes in cognition, affect, interpersonal functioning, or impulse control. The behaviors are inflexible and pervasive across a broad range of personal and social situations, stable and of long duration, and lead to significant distress or impairment in psychosocial functioning. (See "Borderline personality disorder: Epidemiology, pathogenesis, clinical features, course, assessment, and diagnosis".)

- **Other medical or neurologic conditions** – Chronic depersonalization and derealization may less commonly result from a medical or neurologic condition (eg, temporal lobe epilepsy, mild to moderate brain trauma) [55,56], or be secondary to a substance use disorder (most commonly marijuana, hallucinogens, ketamine, and salvia) [17,57]. Rare cases have been seen with vestibular pathology or sleep apnea as the underlying cause. (See 'Assessment' above and "Focal epilepsy: Causes and clinical features", section on 'Seizure semiology' and "Cannabis use disorder: Clinical features, screening, diagnosis, and treatment" and "Vestibular neuritis and labyrinthitis".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Dissociative disorders".)

SUMMARY

- **Introduction and definitions** – Depersonalization/derealization disorder (DDD) is characterized by the persistence or recurrence of depersonalization and/or derealization that cause clinically significant distress or impairment in the presence of intact reality testing (see 'Assessment and diagnosis' above):

  - Depersonalization is a persistent or recurrent feeling of detachment or estrangement from one's self
  - Derealization is a subjective sense of detachment or unreality regarding the world around them

- **Epidemiology** – Transient experiences of depersonalization and/or derealization are very common, while DDD has a prevalence of approximately two percent. DDD has high rates

of comorbidity with depression and anxiety disorders as well as avoidant and borderline personality disorders. (See 'Epidemiology' above.)

- **Role of trauma** – While the pathogenesis of DDD has not been established, chronic depersonalization and derealization symptoms qualifying for the disorder are more common among persons experiencing chronic traumatic or highly stressful situations such as verbal or emotional abuse or neglect during childhood.

- **Organic precursors** – Common organic risk factors for depersonalization include seizures, mild to moderate head injury, and substance use disorder. Several neurotransmitter systems, brain areas, and functional circuits have been associated with depersonalization with some consistency in research studies. (See 'Pathogenesis' above.)

- **Clinical manifestations** – Studies of patients with DDD suggest that five discrete symptoms clusters commonly appear in the disorder (see 'Clinical manifestations' above):

  - Unreality of self
  - Perceptual alterations
  - Emotional numbing
  - Anomalous subjective recall
  - Alienation from surroundings

- **Assessment** – We undertake a diagnostic assessment including detailed psychiatric history, medical history and mental status examination and cursory physical examination in all individuals with suspected DDD. A thorough medical and neurologic evaluation is not routinely necessary; however, we conduct these evaluations in all patients presenting with atypical symptoms or illness course, of older age, or a general medical status or history that raises concerns about the differential diagnosis. (See 'Assessment and diagnosis' above.)

- **Differential diagnosis** – We differentiate DDD from other disorders that may present with symptoms of depersonalization/derealization. These include (see 'Differential diagnosis' above):

  - Schizophrenia or other psychotic spectrum disorders
  - Panic disorder
  - Major depression
  - Acute stress disorder
  - Posttraumatic stress disorder
  - Other dissociative disorders

- Personality disorders
- General medical conditions such as temporal lobe epilepsy, mild to moderate brain trauma, brain tumors
- Substance use disorder: marijuana, hallucinogens, ketamine, and salvia

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