Review

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Cotard's Syndrome: A Review

Cotard Sendromu: Bir Derleme

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ABSTRACT

Cotard's syndrome is a rare neuropsychiatric condition characterized by nihilistic and existential delusions. First described by Jules Cotard in 1880, this syndrome most commonly emerges in the context of psychotic depression, although it may also be observed in association with other psychiatric conditions, including bipolar disorder and schizophrenia spectrum disorders. The clinical course typically progresses from an initial phase marked by hypochondriacal concerns and depressive symptoms to a more advanced stage in which nihilistic delusions—such as denial of one's own existence, body, or organs—become prominent. During this progression, the severity of delusional content may increase, resulting in a more complex clinical presentation. Epidemiological data indicate that Cotard's syndrome has a very low prevalence and is more frequently reported in middle-aged and older individuals, with a higher incidence among females. In addition to psychiatric disorders, the syndrome has also been associated with neurological conditions, including Parkinson's disease, Alzheimer's disease, epilepsy, and cerebrovascular lesions. Clinically, patients commonly present with depressive mood, excessive guilt, anxiety, derealization, depersonalization, and self-harm behaviors. The treatment approach primarily focuses on the management of the underlying psychiatric or neurological condition. Pharmacological interventions typically involve combinations of antidepressants and antipsychotics, while electroconvulsive therapy is considered an effective option in treatment-resistant cases, particularly in patients with severe depression and high suicide risk. Furthermore, psychotherapeutic interventions may serve a supportive role by enhancing insight and improving treatment adherence.

Keywords: Cotard's syndrome, nihilistic delusions, neuropsychiatric disorders

ÖZ

Cotard sendromu, nihilistik ve varoluşsal içerikli sanrılarla karakterize edilen nadir bir nöropsikiyatrik tablodur. İlk olarak 1880 yılında Jules Cotard tarafından tanımlanan bu sendrom, çoğunlukla psikotik depresyon bağlamında ortaya çıkmakla birlikte, bipolar bozukluk ve şizofreni spektrum bozuklukları gibi diğer psikiyatrik durumlarla da ilişkili olarak gözlenebilmektedir. Klinik seyir, genellikle hipokondriyak yakınmalar ve depresif belirtilerle başlayan bir süreçten, bireyin kendi varlığını, bedenini ya da organlarını inkâr ettiği nihilistik sanrıların belirginleştiği bir evreye doğru ilerleyebilmektedir. Bu süreçte sanrıların şiddeti artabilmekte ve klinik tablo daha karmaşık bir görünüm kazanabilmektedir. Epidemiyolojik veriler, sendromun oldukça düşük prevalansa sahip olduğunu ve özellikle orta ve ileri yaş gruplarında daha sık bildirildiğini, ayrıca kadınlarda daha yüksek oranlarda görüldüğünü göstermektedir. Bununla birlikte, Cotard sendromunun yalnızca psikiyatrik bozukluklarla sınırlı olmadığı; Parkinson hastalığı, Alzheimer hastalığı, epilepsi ve serebrovasküler lezyonlar gibi nörolojik durumlarla da ilişkili olabileceği bildirilmektedir. Klinik prezentasyon sıklıkla depresif duygudurum, yoğun suçluluk duyguları, anksiyete, derealizasyon ve depersonalizasyon gibi belirtilerle birlikte kendine zarar verme davranışlarını içermektedir. Tedavi yaklaşımı temelde altta yatan psikiyatrik ya da nörolojik durumun yönetimine dayanmakta; farmakoterapide antidepresan ve antipsikotik kombinasyonları yaygın olarak kullanılmaktadır. Dirençli olgularda elektrokonvülsif tedavi etkili bir seçenek olarak öne çıkmakta, özellikle ağır depresyon ve yüksek intihar riski bulunan hastalarda hızlı klinik yanıt sağlayabilmektedir. Ayrıca psikoterapötik yaklaşımlar, hastalığın seyrinde içgörü gelişimini destekleyerek tedaviye uyumu artırmada önemli bir tamamlayıcı rol üstlenmektedir.

Anahtar sözcükler: Cotard sendromu, nihilistik sanrılar, nöropsikiyatrik bozukluklar

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Introduction

Cotard's syndrome is a rare neuropsychiatric disorder first described in 1880 by French neurologist Jules Cotard. In a case involving a 43-year-old female patient who claimed that her organs were absent and she consisted only of skin and bones, Cotard characterized this condition as "délire des négations" (delusion of negation). In the first evidence-based classification of Cotard's syndrome, the condition was divided into three subtypes according to its clinical features: psychotic depression, Cotard's Type I, and Cotard's Type II (mixed type). Patients in the psychotic depression group predominantly present with melancholic symptoms, whereas in the group defined as Cotard's Type I, only nihilistic or hypochondriacal delusions are observed, in the absence of depressive or anxious symptoms and hallucinations. Cotard's Type II (mixed type) represents a more complex clinical presentation characterized by prominent anxiety, depression, auditory hallucinations, delusions of immortality, nihilistic delusions, and suicide attempts (Berrios et al. 1995a).

Yamada et al. (1999) evaluated the clinical course of Cotard's syndrome in three stages. The first stage, referred to as the "germination stage," is characterized by hypochondriasis and depressive mood. During this period, somatic complaints are observed, typically accompanied by depressive symptoms. In the second stage, termed the "blooming stage," the characteristic features of the syndrome fully emerge; anxiety, depression, nihilistic delusions, and beliefs of immortality may co-occur during this stage. The final stage, the "chronic stage," is marked by the persistence of symptoms and may progress in two distinct forms over time: the depressive type and the paranoid type. In this stage, depressive symptoms may gradually remit, while delusions may persist in a stable and enduring manner.

Cotard's syndrome has not been classified as an independent diagnosis in either the DSM-5 or the ICD-10. In the DSM-5, the syndrome is generally considered within the framework of psychotic delusional content (APA 2013, WHO 2023). However, findings from the literature indicate that, despite the absence of a distinct category within diagnostic systems, Cotard's syndrome constitutes a clinically well-defined and specific syndrome. The aim of this review is to examine the contemporary literature on the historical development, clinical features, etiopathogenesis, and neurobiological foundations of Cotard's syndrome; to evaluate diagnostic challenges, differential diagnosis, and treatment approaches from a comprehensive perspective; and to provide recommendations for the clinical management of the syndrome in light of current evidence.

This narrative review was conducted through a structured search of the PubMed, Scopus, and Google Scholar databases. The search included combinations of the keywords "Cotard syndrome," "Cotard delusion," "nihilistic delusion," "etiopathogenesis," and "treatment." Articles published in English up to 2025 were reviewed. Additional relevant studies were identified through manual screening of reference lists of the retrieved articles.

Epidemiology

Epidemiological studies and case series indicate that Cotard's syndrome has a very low prevalence and poses considerable challenges in terms of differential diagnosis. It has been reported that the prevalence of the syndrome is below 1% in older adults, increasing to up to 3% among elderly individuals with severe depression. In psychotic patients, however, the prevalence has been found to remain below 1% (Bott et al. 2016). In their study examining 479 patients on the psychotic spectrum, Ramirez Bermudez and colleagues (2010) identified a prevalence rate of 0.62% for the syndrome. Similarly, in another study conducted by Stompe and Schanda (2013) involving 346 patients with schizophrenia, the prevalence was reported as 0.87%. In a systematic review by Takahashi et al. (2020) that evaluated published cases of Cotard's syndrome between 1984 and 2018, most of the 102 analyzed cases fell within the 50–79 age range. They also examined psychiatric diagnoses and identified depression in 63 patients, bipolar disorder in nine, schizophrenia in nine, dementia in five, and other psychiatric disorders or conditions with an organic etiology in nine. Furthermore, demographic analyses revealed that the proportion of female patients was higher than that of male patients.

Clinical Features and Etiopathogenesis

Cotard's syndrome is characterized by symptoms such as anxious melancholia, nihilistic delusions, and beliefs of immortality. Importantly, the nihilistic delusions observed in Cotard's syndrome are not limited to the belief of being dead; rather, several forms of negation have been described in the literature, including denial of one's own existence, bodily organs or functions, the external world, and even abstract or metaphysical concepts such as God or the soul (Dieguez 2018). The various forms of negation described in Cotard's syndrome are summarized in Table 1. In one study, the most frequently reported symptoms of Cotard's syndrome were depressive mood (89%), bodily nihilistic delusions (86%), delusions related to existence (69%), anxiety (65%), delusions of guilt (63%), beliefs of immortality (55%), and hypochondriacal delusions (58%) (Berrios et al. 1995a). These findings demonstrate that the syndrome manifests not only with psychotic features but also with prominent affective and somatic symptoms. Consistent with this view, recent systematic reviews of reported cases suggest that the psychopathology of Cotard's syndrome is heterogeneous and may involve several symptom clusters, including psychotic depression, delusional-hallucinatory features, and mixed presentations (Huarcaya-Victoria et al., 2020).

The clinical presentation frequently includes depressive mood, guilt, anxiety, and nihilistic delusions (Berrios et al. 1995b). These features further support the association between the syndrome and mood disorders. In advanced cases, self-harm behaviors, suicidal ideation, or suicide attempts may also be observed (Barradas et al. 2023). In the psychiatric literature, Cotard's syndrome has most frequently been associated with major depressive disorder, bipolar disorder, and schizophrenia spectrum disorders. Depression represents the most common diagnostic context in which the syndrome is observed, and numerous case reports and clinical observations support this association (Basu et al. 2013, Bott et al. 2016, Takahashi et al. 2020). However, Cotard's syndrome is not confined to depressive psychotic states; it may also emerge in primary psychotic disorders such as schizophrenia, particularly in the form of nihilistic delusions related to bodily representation (Kuppili et al. 2017).

Table 1. Types of negations reported in Cotard's syndrome

Category of Negation	Description	Examples
Personal existence	Denial of one's own existence or belief that one is dead	"I do not exist", "I am already dead", "I have disappeared"
Body and bodily integrity	Denial of the presence or integrity of the body or specific organs	Claims that the brain, heart, stomach, or internal organs are absent; beliefs that the body is empty or decomposed
Bodily functions	Denial of physiological processes or internal sensations	Belief that one does not need to eat, drink, or breathe
External world	Denial of the existence of people, objects, or the surrounding environment	"The world no longer exists"
Metaphysical or abstract concepts	Denial of metaphysical entities	Claims that God, the soul, or moral order does not exist

Although the etiopathogenesis of Cotard's syndrome has not been fully elucidated, it is thought that various neurobiological and psychological mechanisms may play a role. Early conceptualizations interpreted this condition primarily as a combination of severe depression and nihilistic delusions, often accompanied by anxiety and related affective symptoms (Berrios et al. 1995b). However, it remains unclear whether the syndrome is primarily related to delusional themes per se or to the intense feelings of devastation and helplessness that emerge during certain depressive episodes. Patients experiencing these psychotic phenomena may report a sense of bodily loss accompanied by psychomotor retardation, as well as a profound state of melancholia (Mashayekhi et al. 2016).

From a neuropsychological perspective, delusion formation has been conceptualized as a disturbance in both perceptual interpretation and higher-order reasoning processes. Classical psychopathological descriptions emphasize the role of delusional perception, in which a normal percept acquires an abnormal meaning for the patient. In addition, Conrad's stage model proposes that delusions may develop through a

sequence beginning with a delusional mood (trema), followed by the emergence of new meanings (apophany) and eventual consolidation of a delusional belief system. Contemporary cognitive models further highlight abnormalities in information processing and reasoning biases, including a tendency toward premature conclusions and altered attributional styles. For example, individuals with delusional beliefs may exhibit a "jump-to-conclusions" reasoning style, forming firm beliefs based on limited evidence. In this context, delusion is not considered a single homogeneous disturbance of thinking but rather an umbrella term encompassing multiple abnormalities of cognition and interpretation (Oyebode 2015). Within this framework, Cotard's syndrome may be understood as a specific manifestation of delusional thinking in which disturbances in self-related perception and cognitive evaluation contribute to the emergence of nihilistic beliefs concerning the body or existence.

Associated Conditions and Comorbidities

Cotard's syndrome has also been reported in association with several neurological and neurosurgical conditions, including subdural hematoma, intracranial tumors, and subcortical strokes (McMurtray et al. 2014, Perez et al. 2014, Gonçalves et al. 2016, Taib et al. 2022). Moreover, cases have been described in which Cotard's syndrome co-occurs with rare or complex clinical conditions. For example, in a patient presenting with acute behavioral changes, the case was initially evaluated as Cotard's syndrome; however, following the development of epileptic seizures during hospitalization, the patient was subsequently diagnosed with anti-NMDA receptor encephalitis (Ramirez-Bermudez et al. 2010). In a case report presented by Gonçalves and Tosoni (2016), a patient with multifocal glioblastoma in the right temporoparietal region, who was later diagnosed with Grade IV glioblastoma, developed Cotard's symptoms, which resolved following treatment with oral dexamethasone. In addition, the development of nihilistic delusions during the post-ictal period has been reported in patients with focal epilepsy (Gil et al. 2019). These findings suggest that the underlying mechanisms of Cotard's syndrome may be more complex than previously assumed.

Cotard's syndrome may also occur concurrently with other rare syndromes. In this context, a case reported in the literature emphasized that, prior to establishing a diagnosis of Cotard's syndrome, asomatognosia—characterized by denial of a part of one's body and more commonly associated with neurological disorders—should be considered in the differential diagnosis (Fonseca et al. 2013, Örüm et al. 2020). In another example suggesting a neurological substrate underlying such syndrome combinations, Cotard's and Capgras syndromes co-occurred following a left temporoparietal ischemic cerebrovascular event (Sottile et al. 2015). Additionally, Ansari and colleagues (2025) reported the concurrent occurrence of Cotard's syndrome and auto-hemophagia, a rare form of self-injurious behavior. This case underscores that Cotard's syndrome is not limited solely to psychotic delusional content but may also be accompanied by rare and destructive self-harm behaviors, highlighting the need to consider a broad spectrum of psychopathology in clinical evaluation.

The similarity between Cotard's syndrome and other misidentification syndromes, such as Capgras syndrome, suggests that impairments in face recognition, self-integrity, and affective processing may also contribute to this clinical presentation (Barradas et al. 2023). Within this framework,the relationship between Cotard's and Capgras syndromes has also been discussed within cognitive neuropsychiatric models of delusion formation. While Capgras syndrome is characterized by the denial of the identity of familiar individuals—patients recognize a face perceptually but experience an absence of the normal affective sense of familiarity—Cotard's syndrome involves a more global form of nihilistic denial directed toward one's own body or existence. From a neurocognitive perspective, this distinction has been interpreted in relation to the extent and localization of neural disconnection. In Capgras syndrome, the disruption has been proposed to involve a relatively selective disconnection between face recognition processes within the ventral visual pathway and limbic structures responsible for affective tagging of familiar stimuli. In contrast, Cotard's syndrome has been hypothesized to reflect a broader disturbance in affective integration across perceptual systems, leading to a generalized loss of emotional resonance with internal and external experiences. Such models suggest that the qualitative difference between the two syndromes—the misidentification of others in Capgras and the nihilistic denial of self in Cotard—may be

partly related to differences in the scope of neural network dysfunction underlying affective and perceptual processing (Phillips 2002). These interpretations are also consistent with cognitive neuropsychiatric accounts of delusion formation. In this context, Debruyne and Audenaert (2012) describe the two-factor model, according to which an anomalous perceptual or affective experience constitutes the first factor, whereas an impairment in belief evaluation represents a second factor that allows the delusional belief to persist despite contradictory evidence.

Collectively, these cases suggest that Cotard's syndrome may not represent an isolated phenomenon but rather a multifaceted clinical entity arising from disrupted connectivity among large-scale neuronal networks and shared cognitive-biological mechanisms.

Neuroimaging Findings

Although numerous case reports and case series have been published describing structural and functional brain alterations observed in the context of Cotard's syndrome emerging in the background of neuropsychiatric disorders, comprehensive and integrative explanations regarding its underlying neurobiological mechanisms remain limited. Early neuroimaging studies suggest that individuals with Cotard's syndrome may exhibit distinctive structural brain abnormalities (Parks et al. 2014, Shokrgozar et al. 2016). In an FDG-PET study conducted by Charland-Verville and colleagues (2013), marked hypometabolism within the bilateral frontoparietal cortical network was identified in a 48-year-old patient with Cotard's syndrome. The affected regions included the precuneus, anteroposterior cingulate cortex, mesiofrontal cortex, posterior parietal and dorsolateral frontal lobes, and the right temporoparietal junction. In contrast, hypermetabolic activity was reported in the cerebellum, brainstem, and bilateral thalamus. Similarly, in a case reported by Özkan and Çalıyurt (2016), a 48-year-old patient with schizophrenia who exhibited treatment-resistant Cotard's symptoms demonstrated widespread hypometabolism in the fronto-parieto-temporal associative cortex related to the default mode network (DMN), as well as in the cerebellum and left frontal operculum; additionally, marked hyperactivity was detected in the basal ganglia. Collectively, these findings underscore the difficulty of defining the syndrome based on a consistent and universal neuroanatomical marker aligned with neuroimaging data.

Although the findings have not yet been integrated into a systematic classificatory framework, existing studies suggest that the neurobiological basis of Cotard's syndrome may involve functional suppression in higher-order cognitive regions—particularly within structures of the DMN—responsible for self-awareness, the perception of bodily integrity, and the integrative interpretation of internal experiences. In this context, structures involved in emotional and somatosensory processing, such as the basal ganglia and thalamus, have been proposed to exhibit secondary hyperactivation (Basu et al. 2013, Barradas et al. 2023). Cotard's syndrome should not be conceptualized merely as a singular phenomenon defined by nihilistic delusions; rather, it may be understood as a multidimensional clinical entity reflecting a complex disruption in the interactions among self-perception, bodily integrity, emotional awareness, and cognitive processes. In particular, functional disconnection among the DMN, insula, frontoparietal networks, and the limbic system provides a neurobiological framework that may underlie the development of Cotard's syndrome symptomatology. Within this framework, disruptions in the subjective sense of existence are increasingly associated with both affective dysregulation and the failure to integrate bodily experiences (Ramirez-Bermudez et al. 2010). Accordingly, the assessment of Cotard's syndrome requires moving beyond a classical psychiatric approach toward a comprehensive evaluative model that incorporates neuroimaging, neuropsychological testing, and processes related to body perception.

Assessment and Treatment Approaches

The first step in the management of Cotard's syndrome should involve a comprehensive clinical evaluation encompassing both psychiatric and medical assessment. If an underlying medical or neuropsychiatric condition is identified, the primary intervention should be directed toward that condition; in life-threatening situations, a rapid and effective treatment strategy must be implemented. For instance, in severe cases where food refusal has developed, enteral nutrition may be required until oral intake is re-

established (Bott et al. 2016). Once these critical conditions have been stabilized, therapeutic interventions specifically targeting Cotard's syndrome may be initiated. Subsequently, treatment typically involves the use of psychotropic medications, including antipsychotics, antidepressants, benzodiazepines, and anticonvulsants.

Table 2. Summary of treatment approaches reported in Cotard's syndrome

Treatment Category	Therapeutic Approach	References
Pharmacological Treatment		
Antipsychotic monotherapy	Risperidone	Mendez et al. 2011
Combination pharmacotherapy	Escitalopram + antipsychotics; Valproic acid augmentation; Lithium augmentation	Soultanian et al. 2005, Couto et al. 2021
Antipsychotic + psychotherapy	Olanzapine + psychotherapy	Sottile et al. 2015
Mood stabilizer + Cognitive Behavioural Therapy + antipsychotic	Valproic acid + Cognitive Behavioural Therapy (addition of quetiapine)	Bott et al. 2016
Treatment in neurological disorders	Quetiapine	Solla et al. 2015
Typical antipsychotics	Haloperidol	Morgado et al. 2015
Antipsychotic + antidepressant + anxiolytic	Aripiprazole + Venlafaxine + Diazepam	De Berardis et al. 2010, Huarcaya-Victoria et al. 2016
Dopaminergic augmentation	Pramipexole	Maruo et al. 2016
Non-pharmacological Treatment		
Electroconvulsive therapy		Barradas et al. 2023, Örüm et al. 2020
Neuromodulation	Transcranial Magnetic Stimulation	Barradas et al. 2023

Antipsychotic monotherapy has been reported to be effective in numerous cases of Cotard's syndrome. For example, treatment with risperidone alone has proven to be sufficient in many cases (Mendez et al. 2011). In some instances, augmentation with agents such as escitalopram, valproic acid, or lithium has been associated with improved treatment response (Soultanian et al. 2005, Couto et al. 2021). In a case where Cotard's and Capgras delusions co-occurred, olanzapine administered in combination with psychotherapy resulted in complete remission of symptoms (Sottile et al. 2015).

In a more complex case involving a patient with a history of head trauma and schizophrenia, treatment consisted of valproic acid in combination with cognitive behavioral therapy (CBT); quetiapine was subsequently added to the regimen, and the patient achieved complete remission within one month (Bott et al. 2016). In a case involving Parkinson's disease, Cotard's syndrome symptoms were reported to follow a fluctuating course associated with the early wearing-off effect of levodopa, and psychiatric symptoms improved following the addition of quetiapine (Solla et al. 2015). Furthermore, the side effect profiles of antipsychotic agents represent an important consideration in clinical management; extrapyramidal side effects have been reported in cases treated with typical antipsychotics such as haloperidol (Morgado et al. 2015). These findings suggest that pharmacological approaches in the treatment of Cotard's syndrome should be individualized, taking into account the characteristics of the underlying disorder, clinical severity, and treatment response.

In numerous cases, Cotard's syndrome has been observed within a clinical presentation characterized by severe depressive symptoms. In such situations, the combined use of antipsychotics with antidepressants and anxiolytics has been most effective. For example, in a case involving major depressive disorder with catatonia, the combined administration of aripiprazole, venlafaxine, and diazepam served as an effective treatment (De Berardis et al. 2010, Huarcaya-Victoria et al. 2016). In another case presented by Maruo and colleagues (2016), pramipexole, a presynaptic dopamine agonist, was added to the treatment regimen of a patient with treatment-resistant major depression. This intervention was associated with a significant reduction in both depressive symptoms and nihilistic delusions.

The efficacy of electroconvulsive therapy (ECT) has been particularly supported in cases where nihilistic delusions are resistant to treatment. Studies have demonstrated that ECT rapidly yields positive outcomes, especially in cases of Cotard's syndrome characterized by high suicide risk and severe depressive symptoms (Örum et al. 2020, Barradas et al. 2023). In addition, neuromodulation techniques such as transcranial magnetic stimulation (TMS) have been considered among alternative treatment options in some cases (Barradas et al. 2023). Overall, the main treatment approaches described in the literature are summarized in Table 2.

Conclusion

Cotard's syndrome is a rare psychiatric condition that may lead to serious clinical consequences. Its delusional content, particularly the presence of nihilistic and existential themes, may profoundly affect patients' adherence to treatment and overall quality of life. The literature reviewed in this study demonstrates a strong association between the syndrome and both mood disorders and psychotic disorders. In cases that develop in the context of major depression, bipolar disorder, and schizophrenia, the combined use of antidepressants and antipsychotics is often preferred. In treatment-resistant cases, electroconvulsive therapy (ECT) emerges as one of the most effective therapeutic modalities and, in certain patients, may accelerate symptomatic improvement when used adjunctively with pharmacotherapy. Nevertheless, considering factors such as the feasibility of ECT and the requirement for informed patient consent, the importance of individualized treatment planning must be emphasized. Various psychotherapeutic approaches also play a supportive role, particularly in facilitating the development of insight.

From a clinical perspective, early recognition of Cotard's syndrome is essential, as the presence of nihilistic delusions may lead to severe consequences such as food refusal, poor treatment adherence, and an increased risk of suicide. Clinicians should therefore maintain a high index of suspicion in patients presenting with severe depressive symptoms, psychotic features, or unusual beliefs concerning bodily integrity and existence. Careful differential diagnosis is also necessary to distinguish Cotard's syndrome from other neuropsychiatric conditions, including neurological disorders, delirium, and misidentification syndromes. Given the heterogeneity of its clinical presentation, treatment planning should be individualized and based on the underlying psychiatric or neurological condition, with particular attention to suicide risk assessment and appropriate safety management.

Given the limitations of the existing literature, which is largely based on case reports, large-scale prospective studies would contribute substantially to the development of evidence-based treatment guidelines. Following diagnosis, the implementation of a rapid and comprehensive treatment approach is critical to support both clinical and functional recovery. Further research is needed to elucidate the etiological underpinnings and determine optimal therapeutic strategies for this rare clinical entity, which remains insufficiently understood in the literature, presents the risk of severe outcomes such as suicide, and often necessitates diverse treatment approaches.

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