CASE REPORT

Cotard's Syndrome with Epilepsy and Frontal Atrophy: A Rare Case Highlighting the Neuropsychiatric and Cultural Interface

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Received on: 19 October 2025; *Accepted on*: 26 December 2025; *Published on*: XX XXXX XXXX

Abstract

Cotard's syndrome is a rare neuropsychiatric condition characterized by nihilistic delusions in which individuals deny the existence of their body, organs, or self. Although often associated with severe depression or psychosis, neurological abnormalities can also contribute. We report on a 26-year-old woman with epilepsy and mild frontal-lobe atrophy who presented with delusions of death and "soul–body separation." Her symptoms resolved completely with combined olanzapine and fluoxetine therapy. This case is distinctive for the coexistence of Cotard's syndrome with structural brain pathology and its culturally rooted phenomenology within the Indian context, highlighting the interaction between neural self-representation networks and cultural narratives in the formation of delusional beliefs.

Keywords: Case report, Cotard's syndrome, Cultural psychiatry, Epilepsy, Frontal lobe atrophy, Nihilistic delusions.

Indian Journal of Private Psychiatry (2025): 10.5005/jp-journals-10067-0209

Introduction

Cotard's syndrome, first described by Jules Cotard in 1880, is a rare but severe disorder marked by delusions of negation, anxious melancholia, and occasionally delusions of immortality or enormity.[1,2] While popularly reduced to the belief of being dead or non-existent, Cotard's original "delusion of negations" encompassed a far broader phenomenological spectrum, including feelings of damnation, dissolution, and transformed existence.[2]

Current evidence regarding Cotard's syndrome is based mainly on case studies, and therefore, no clarity can be obtained about the various aspects of the syndrome, such as prevalence and pathogenesis.[2] Although traditionally associated with psychotic depression, the syndrome has also been reported in the context of epilepsy, traumatic brain injury, and stroke.[3,4] Neuropsychiatric research implicates dysfunction in fronto-temporo-parietal networks governing self-representation and emotional integration. Disruption of these circuits—through structural lesions or epileptic activity—can distort the integration of interoceptive and affective information, leading to experiences of depersonalization and self-negation.[5,6]

In India, metaphysical dualism between *atma* (soul) and *sharir* (body) is deeply embedded within spiritual belief systems. Consequently, delusional experiences of self-loss are often expressed as "soul–body separation" rather than simple non-existence. This case highlights the neuropsychiatric underpinnings of Cotard's syndrome while illustrating its culturally specific manifestation in the Indian context.

Case Description

A 26-year-old married woman from rural Rajasthan presented with a 4 months history of progressive restlessness, irritability, insomnia, and a culturally nuanced nihilistic delusion. She belonged to a Hindu family in a rural farming community where traditional beliefs in the dualism of *atma* (soul) and *sharir* (body) are widely held. She repeatedly stated, "Mhare shareer ri aatma alag ho gayi hai" ("my soul has separated from my body"), insisting that she was already dead but her body continued to exist like a corpse. She believed her body was actively decaying ("mharo *sharir* sadd riyo hai" – "my body is rotting") and emitting a foul odor that others could perceive, although no such odor was detectable. She also reported derealization, perceiving her surroundings as unreal. These symptoms caused marked distress and socio-occupational dysfunction.

She had a 5 years history of generalized tonic-clonic seizures, initially poorly controlled due to medication nonadherence, but currently stable on phenytoin 100 mg twice daily, with her last seizure 9 months prior. There had no history of substance use, head trauma, developmental delays, prior psychiatric hospitalizations, or family psychiatric history. Mental status examination revealed dysphoric mood, restricted affect, and an increased rate of speech with pressured quality, likely reflecting psychomotor agitation and frontal disinhibition, possibly secondary to the documented cortical atrophy.

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How to cite this article: Bhasin EK, Sharma I, Seervi A. Cotard's Syndrome with Epilepsy and Frontal Atrophy: A Rare Case Highlighting the Neuropsychiatric and Cultural Interface. Ind J Priv Psychiatry 2025;x(x):xx–xx.

Source of support: Nil

Conflict of interest: None

Patient consent statement: A written informed consent was obtained from the patient for the publication of details, which can include photographs and/or videos and/or case history to be published in any printed/online journals.

She expressed prominent nihilistic and guilt-based delusions with intact orientation and preserved cognition but severely impaired insight and judgment. Intellectual disability was ruled out based on her educational attainment (primary level), intact premorbid functioning, including household management and farming activities, and preserved cognitive abilities on mental status examination. Physical and systemic examinations were normal. Routine laboratory tests, thyroid profile, and serum electrolytes were within normal limits. Electroencephalography (EEG) showed normal background activity without epileptiform discharges. Magnetic resonance imaging (MRI) of the brain revealed bilateral medial frontal lobe atrophy.

The patient was diagnosed under the ICD-11 classification with secondary mood syndrome (6E62) with psychotic symptoms, attributable to epilepsy and a structural brain disorder (frontal lobe atrophy).[7] Cotard's syndrome represents the phenomenological presentation within this diagnostic framework.

She was initially managed with intramuscular haloperidol 5 mg and promethazine 25 mg for acute agitation, followed by oral risperidone (titrated to 5 mg/day) and fluoxetine (20 mg/day) targeting the underlying depressive symptoms, including dysphoric mood, insomnia, anhedonia, and guilt. Due to extrapyramidal side effects and limited improvement, risperidone was replaced by olanzapine (titrated to 15 mg/day) while continuing fluoxetine. Over 3 weeks, she demonstrated progressive recovery of mood, sleep, and insight, with complete remission of nihilistic delusions. She remained clinically stable at follow-up on phenytoin and the olanzapine-fluoxetine combination.

Discussion

This case exemplifies secondary mood syndrome with psychotic features, manifesting as Cotard's syndrome at the intersection of epilepsy-related cortical dysfunction, frontal atrophy, and affective disturbance. The bilateral medial frontal lobe atrophy in our patient likely disrupted self-referential processing networks and executive control mechanisms, explaining the coexistence of dysphoric mood with psychomotor agitation and pressured speech, a pattern characteristic of frontal-variant mood disorders rather than classical retarded depression.[4,5]

The patient's Hindu cultural background, with its traditional beliefs in *atma-sharir* dualism, provided the specific phenomenological framework through which her nihilistic delusions were expressed. Rather than simply denying the existence of the soul, she articulated a culturally coherent belief in its dissociation from the body, with the soul departing while the body remained as a decaying vessel. This case demonstrates how cultural schemas shape the form and content of delusions in neuropsychiatric disorders, highlighting the importance of recognizing culturally shaped narratives for diagnostic accuracy and therapeutic engagement.

Theoretically, Cotard's syndrome reflects a dissolution of the bodily self, arising from psychosensory disintegration of self-awareness and right-hemispheric dysfunction, leading to perceptual and somatosensory feelings of unreality, which, coupled with reasoning impairments, result in beliefs of non-existence.[6] This aligns with modern models positing Cotard's syndrome as a disorder of bodily self-representation within fronto-temporo-limbic networks.[4,5] In our patient, frontal atrophy combined with epileptic cortical instability likely disrupted these networks, while her cultural background determined the specific delusional narrative.

Based on a factor analysis of 100 cases, Berrios and Luque[8] classified the condition into three subtypes: Psychotic depression, characterized by melancholia and nihilistic delusions; Cotard type I, consisting of pure nihilistic delusions without affective features; and Cotard type II, presenting with anxiety, depression, and auditory hallucinations. Huarcaya-Victoria et al.[9] recently demonstrated that Cotard's syndrome comprises three major symptom clusters: Psychotic-depressive, delusive-hallucinatory, and mixed-anxious. Our patient's presentation, dominated by nihilistic and guilt-related delusions with dysphoria, aligns with the psychotic-depressive factor.

Symptom evolution in Cotard's syndrome often follows three stages: Germination (hypochondriasis and cenesthopathy), blooming (fully developed nihilistic delusions), and chronic (systematized delusional content).[10] The patient's clinical trajectory from vague somatic preoccupations and pervasive despair to a fully crystallized belief in nonexistence mirrors this triphasic progression, underscoring the dynamic evolution of Cotard's phenomenology in the context of affective and neurocognitive dysfunction.

Several treatment modalities were reported to improve the symptoms of Cotard's syndrome, from pharmacotherapy, mainly consisting of antipsychotics and antidepressants, to electroconvulsive therapy.[11] Management requires addressing both psychiatric and neurological contributors. Continued anticonvulsant therapy with phenytoin maintains seizure control and reduces cortical excitability. The treatment rationale combined dopaminergic blockade (olanzapine) to address psychotic symptoms with serotonergic enhancement (fluoxetine) to target the affective substrate. This synergistic approach, acting across D_2 and 5-HT_2A/2C receptors while enhancing serotonergic transmission, addressed both delusional content and underlying mood disturbance.[12]

Electroconvulsive therapy remains highly effective for severe, treatment-resistant cases or when complicated by catatonia or nutritional deficiency.[13] Our patient's complete remission with pharmacotherapy alone reinforces observations that integrated medication management can achieve full recovery in selected presentations.

Conclusion

Cotard's syndrome represents a case that illustrates a convergence of neurobiological dysfunction, psychopathological distortion, and culturally mediated self-concept. In this patient, epilepsy and mild frontal lobe atrophy likely disrupted neural circuits involved in self-referential processing. At the same time, deeply ingrained beliefs in soul–body dualism shaped the specific delusional content. The patient's complete recovery with olanzapine–fluoxetine therapy, in the absence of electroconvulsive treatment, underscores the potential efficacy of pharmacological management in selected cases. Clinicians should remain alert to culturally influenced expressions of self-negation and consider underlying neurological contributions when evaluating Cotard's syndrome as a complex disorder of self-awareness, rather than a unitary delusion, thereby fostering more integrative and person-centered management approaches.

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