Cotard Syndrome: "I'm Dead, So Why Do I Need to Eat?"

To the Editor: First described in 1880, Cotard syndrome is characterized by anxious melancholia, ideas of damnation or rejection, insensitivity to pain, and nihilistic delusions concerning one's own body or existence.[1] It is becoming more accepted that Cotard syndrome is a symptom of an underlying disorder—it is not listed as a specific disorder in *DSM-IV* or *-5*.[1] The symptoms and course of Cotard syndrome vary from ideas of spontaneous destruction of organs, body, and soul to the complete denial of one's existence. Subsequently, some patients may cease to eat and may have increased tendency toward self-mutilation or suicidal behavior.

A literature search in June 2015 with PubMed using keyword *Cotard* yielded 90 results with a predominance of case reports. In 1995, a retrospective factor analysis of 100 cases in the literature by Berrios and Luque[2] led to the first evidence-based classification of 3 forms of Cotard syndrome. The most common symptoms reported in subjects were depression (89%), nihilistic delusions concerned with the body (86%) and existence (69%), anxiety (65%), guilt (63%), hypochondriacal delusions (58%), and delusions of immortality (55%). They found that there were no differences between men and women on underlying diagnostic categories and an increased likelihood of development with age. The first form extracted from their study describes a psychotic depression with prominent melancholia but few nihilistic delusions. Cotard type 1 is the second form, identifying a more prominent delusional component over depression. Cotard type 2 is the third form, identifying the presence of a mix of anxiety, depression, and auditory hallucinations.[2] While Cotard syndrome was first described over a decade ago, the literature remains scarce, with the continued need for informative case reports.

Case report. Our patient, Ms A, is a woman in her 50s with a history of anxiety and psychosis, a past medical history of quadriplegia and tracheostomy, and a family history of depression. She presented to the emergency department having had 1 week of an altered mental status, worsening shortness of breath, and sputum production for several days. She was admitted for metabolic encephalopathy secondary to sepsis and hyponatremia. Further history revealed the patient was in her normal state of health until 1 week prior to admission when she began stating, "I am dead." She subsequently stopped eating, attempted to remove her tracheostomy, and refused medications. Ms A's caregiver indicated that prior to her decline, the patient had been made aware of financial trouble. Initially, Ms A was poorly oriented and unable to carry a conversation. Her encephalopathy improved with initial treatment of hyponatremia and sepsis; however, nihilistic delusions persisted. Ms A indicated that command-type auditory hallucinations told her she was dead, that she had died as an infant, and that her family had died. She also expressed helplessness and negative cognitive distortions of inability to provide support for her family due to her condition. Voices communicated she was evil and that she had lost her bank account and home. Her practice of Buddhism and the principle of karma appeared to influence these voices. Ms A admitted to ongoing anxiety about finances and health insurance. Throughout hospitalization, she clearly and consistently denied suicidal ideations or thoughts of self-harm.

On the basis of history and examination, unspecified anxiety disorder (*DSM-5* F41.9) and unspecified schizophrenia spectrum and psychotic disorder (*DSM-5* F29) with Cotard delusion were diagnosed. Hyponatremia was felt to be secondary to syndrome of inappropriate secretion of antidiuretic hormone (SIADH) (*ICD-10* E22.2) and primarily treated with demeclocycline, salt tablets, and fluid restriction. Sepsis resolved upon addressing pneumonia with antibiotics. During this hospitalization, the patient was given a trial of risperidone and mirtazapine, which resulted in minimal improvement. This hospitalization was the first time Ms A had exhibited characteristics of Cotard syndrome, which led to a retrial of aripiprazole monotherapy and remission of Cotard delusion. Prior to this hospitalization, the patient was treated for auditory hallucinations and anxiety with aripiprazole, escitalopram, quetiapine, olanzapine, and risperidone, although noncompliance most likely interfered with efficacy. Notably, frequent reassurance by social work about finances and psychotherapy were of benefit, as Ms A began to identify elements that were in opposition with her delusion—she began to believe she was alive. The elements identified included her drive to eat, her medical condition, the feeling of her heartbeat, and the reassurance of others. The patient was discharged with in-home psychotherapy to further address anxiety and cognitive distortions.

This case highlights characteristics of Cotard syndrome with the goal of providing insight into a seemingly rare syndrome. Cotard delusion has been compared to Capgras delusion, a delusion in which familiar people are replaced by imposters. Specifically, studies into the role of premorbid personality characteristics suggest that patients with an internal attribution style (commonly co-occurring with depression) may be predisposed to develop Cotard delusion.[3] Conversely, patients with an external attribution style (commonly co-occurring with paranoia) may be predisposed to develop Capgras delusion.[3] Studies have also suggested that these bizarre psychiatric syndromes may be related to parietal brain dysfunction.[4] Specifically, dysfunction may lie in the information-processing subsystem in which face and body recognition are associated with an affect of familiarity. This hypothesis suggests that derealization and depersonalization occur when the normal feeling of familiarity is absent.[3] However, the majority of computed tomography neuroimaging studies have found no gross structural changes in patients with Cotard delusion.[5] A few investigations have associated Cotard syndrome with multifocal brain atrophy and enlargement of lateral and third ventricles, which may indicate a role for frontotemporoparietal circuitry in the pathophysiology of Cotard syndrome.[1]

The literature review reveals that there have been several successful pharmacologic treatments studied for Cotard syndrome—including aripiprazole, amitriptyline, duloxetine, fluoxetine, paroxetine, olanzapine, sulpiride, lithium, and combination strategies with selective serotonin reuptake inhibitors, tricyclic antidepressants, and antipsychotics.[1,6] Electroconvulsive therapy has also been found to be beneficial in the treatment of Cotard syndrome with psychotic depression.[7] By using the classification system by Berrios and Luque,[2] we determined our patient's presentation in this case report meets criteria for Cotard type 2 form given a more prominent delusional component over depression. It is possible that aripiprazole monotherapy was effective in the remission of Cotard delusion in our patient due to the stabilizing effects on dopaminergic and serotonergic systems. Aripiprazole reduces psychotic symptoms as a partial D_2 agonist by lowering the dopaminergic neurotransmission in the

mesolimbic pathway while enhancing the dopaminergic activity in the mesocortical pathway. Aripiprazole also quite likely improved depressive symptoms by serving as a partial 5-HT_{1A} agonist and 5-HT_{2A} antagonist. A case report by De Berardis et al[6] demonstrates another example of a patient with Cotard type 2 form successfully treated with aripiprazole monotherapy.

Current studies remain limited as the syndrome is relatively rare, and to substantiate a hypothetical relationship, large cohort or cross-sectional studies are needed. Further research is needed to explore the pathogenesis of Cotard syndrome and possible methods of treatment, including the role of aripiprazole in improving Cotard delusion with concurrent anxiety and depressive symptoms.

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