# Cotard Syndrome with Catatonia: Unique Combination

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#### ABSTRACT

Cotard syndrome is a rare psychiatric condition characterized by extreme nihilistic delusions. Catatonia though common, its combination with the Cotard syndrome is exceeding rare and more so the response with the pharmacotherapy as in our case. Since, both are found in organic conditions the importance of studying such a case is to understand the underlying neurobiologic determinants.

Key words: Catatonia, cotard syndrome, lorazepam challenge test

## INTRODUCTION

Cotard syndrome is named by the French physician Jules Cotard (1840-1889), who in 1882 named délire des negations, a condition where the patient considers non-existence of almost everything including self.<sup>[1]</sup> Though most commonly found in unipolar, bipolar depression and schizophrenia - it has also been found in organic conditions such as neurosyphilis, epilepsy, cerebrovascular accident, parietal lobe tumor, Parkinson's disease, and multiples sclerosis.<sup>[2]</sup>

Cotard syndrome is rare and described in terms of mostly case-reports. A latest systematic PubMed and Medline search found only 47 searches in combined English and all European languages. An attempt was made by Berrios *et al.* who systematically studied all case-reports till 1995 and tried to classify Cotard syndrome based on factor analytic methods.<sup>[3]</sup> Most commonly, it is a syndrome of mood congruent nihilistic delusions like delusion of being dead, guilt,

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and immortality seen in patients of severe depressive episode. Patients are mostly found in advanced state, in severe psychomotor retardation with impairment in biological functions requiring electroconvulsive therapy (ECT). Interestingly, contrary to their presentation, prognosis is good. Cotard syndrome is associated with other psychopathology such as Capgras delusion, lycanothropy and catatonia. [4-6] Here, we describe a case of Cotard syndrome with catatonia.

## **CASE REPORT**

A 45-year-old married female belonging to low socio-economic condition of rural background with a family history of psychosis in younger brother is the patient. Twenty years back she had a history of severe depressive episode without psychotic features with post-partum onset. It remitted completely after 6 months and thereafter, she had optimum functioning with no psychiatric symptoms. The current episode had onset about 2 years back precipitated by significant family conflict regarding the daughter's marriage. She gradually developed persistent and pervasive sadness, severe anhedonia, crying spells, worthlessness, hopelessness, and suicidal ideation. After 3-4 months of the onset of illness, for treatment she was taken to a nearby city (Jaipur). There they could not afford the treatment due to financial losses. Thereafter, family members noted that her speech output decreased further and she would only speak in

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a very soft and monotonous voice that her dead-body is in Jaipur. When others would confront her she would be insistent and even request them to get her buried. She would also deny food and water and would say that there is nothing inside her abdomen and body. She would have severe psychomotor retardation, would change her posture very infrequently and often stare at a particular direction for several minutes without blinking.

When she presented to us about 2 years after the onset of illness she was stuporous. There was no history suggestive of any organic illness. On admission, though, she was severely malnourished (weight 28 kg, body mass index - 11.7), her vitals were stable, and systemic examination were within normal limits. There was a score of 11 (mutism - 3, posturing - 2, immobility - 1, staring - 2, withdrawal - 3) on Bush Francis Catatonia rating scale (BFCRS). A lorazepam challenge test carried out with 2 mg of intravenous lorazepam showed dramatic improvement and BCFRS score decreased to 2.

Thereafter, she was started on lorazepam upto 8 mg parenterally in divided doses and her nutrition and hydration were maintained. Her routine investigations like hemogram, blood biochemistry including liver, kidney function tests, and electrolytes were within normal limits. Further investigations such as Electroencephalogram (EEG), magnetic resonance image of brain, HIV status, VDRL, thyroid level were done. Serum folate and vitamin B12 was advised, but could not be carried out because of lack of resources. Lack of memory deficits and a normal higher cognitive function ruled out dementia. She was diagnosed as a case of recurrent depressive disorder with currently severe depressive episode with the psychotic features as per International Classification of Diseases-10 (ICD-10). She was started on tablet sertraline up to 200 mg and olanzapine up to 10 mg and lorazepam was tapered gradually. There were no catatonic symptoms from 2<sup>nd</sup> week. Hamilton Rating Scale for Depression (HAM-D) decreased to 22 on the 2<sup>nd</sup> week and 16 after 4 weeks. There was also significant an improvement in her body weight (up to 40 kg) and overall functioning.

## DISCUSSION

We diagnosed this patient to have nihilistic delusions in the form of Cotard delusion after differentiating it from several related conditions like depersonalization and culturally mediated belief (the concept of life after death is very common in Hindu mythology). However, absence of any "as if" phenomenon and persisting with the belief not shared by other family members even on repeated confrontation substantiates our claim. It may

be argued that the catatonic features were part of the depressive stupor, but the dramatic improvement with lorazepam challenge test settles all controversy.

After ruling out organic causes by means of absence of any findings on general physical examination and normal investigations and other psychiatric illnesses, a mood disorder appears to be the most probable. Though, currently we have made a diagnosis of unipolar depression in the absence of any frank hypomanic episode yet a high probability of bipolarity (particularly bipolar disorder II) appears in view of post-partum onset, first episode depression with prominent psychomotor retardation, relatively quick improvement in severe depressive symptoms in current episode. This would be very useful in formulating her future medication plan like planning a mood-stabilizer.

To our knowledge this combination of Cotard syndrome and catatonia is very rare. Only a handful of such cases have been reported till date. [7,8] Management wise also our case is unique because most of the prior cases responded to ECT while only one case had responded with pharmacotherapy.<sup>[6]</sup> Thus, medications can also be effective in Cotard syndrome. Overall, apart from arousing interest this association of two clinical conditions can have neurobiological underpinnings as both are known to occur in organic conditions. Particularly remarkable has been the association of Cotard syndrome with right parietal lobe afflictions, which is responsible for concepts regarding self and insight.[9] Neuroimaging studies also show impairment in circuits in relation to the right parietal cortex in catatonia.[10] Thus, study of these common biological determinants can go a long way in unraveling etiology of such illnesses.

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**How to cite this article:** Basu A, Singh P, Gupta R, Soni S. Cotard syndrome with catatonia: Unique combination. Indian J Psychol Med 2013;35:314-6.

Source of Support: Nil, Conflict of Interest: None.

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