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A DETAILED REVIEW ON COTARD'S SYNDROME

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ABSTRACT

Cotard's Syndrome is an infrequent neuropsychiatric condition. The main feature of this condition is 'nihilistic' delusion of one's own body which includes feeling of being dead or non-existing and also loss of body parts. It has been reported to be associated with various organic conditions in DSM IV. However, this disorder is itemized in fifth edition of Diagnostic of Statistical Manual of Mental Disorder (DSM V) and International Classification of Diseases and related health problems (ICD) as a specific disorder; Although it is important to ratify the syndrome because precise mechanism is present along with therapeutic consequences. In this paper, Cotard's syndrome is briefly reviewed with up to date knowledge about syndrome.

KEYWORDS: *Cotard's syndrome; Delusions; Nihilistic delusions; Review.*

INTRODUCTION

Cotard's syndrome also known as walking corpse syndrome, is a rare psychiatric illness in which the person involved has the mistaken illusion that he or she is dead and does not exist. It's been debated whether the syndrome is a distinct disorder or a symptom of other disorders. A hundred-patient cohort statistical analysis reveals that denial of self-existence occurs in 45 per cent of Cotard's syndrome cases; the other 55 per cent of patients presented with delusions of immortality.[1] Cotard's illusion falls into the category of somatic delusions, those which include body functions or sensations, according to the DSM-5 (Diagnostic and Statistical Manual of Mental Disorders, 5th Edition). While it is not a diagnostic factor in our current medical practice, information on Cotard 's syndrome and a clear approach to treating patients is useful.[2] Cotard's syndrome is a disorder of nihilistic delusion as the main symptom. The condition was known as ''délire des négations.''[3]

This review provides an up to date description of Cotard's syndrome, which was first identified more than a century ago. It covers historical aspects, etiology, classification and recent views on treatment and pathogenesis of this syndrome. The literature available is mostly in the form of case studies. As this condition is very rare, there is lacking of sample and prospective studies and as a result the literature is limited and present in the form of case studies.

Historical background

The first illustration about Cotard's syndrome was given by Jules Cotard (a French neurologist) in 1880. He presented the case of 'Madame X', a 43-year-old woman in a lecture. Jules Cotard found out symptoms such as anxiety, delusions of possession, tendency to suicidal thoughts, self-harm, hypochondriac thoughts of non-existence and delusions of destruction of several organs and body with thoughts of inability to die were present in patient.[4] He named it as "hypochondriac delusion", and some years later "delusion of negations." The "Cotard's syndrome" was named after his death.[5]

In 1882, Cotard officially called his case as "délire des négations." In 1884, Cotard reported a case of melancholia with nihilistic delusion with some additional features like denying the existence of other persons and delusions of external influence along with immobility and a patient's inability to visualize own child.[6] In 1887, he described his 11 cases into three categories: 8 cases as simple delusion of negation, 1 case as delusion of negation and 2 cases as delusion of negation associated with Persecutory delusions. He used a term 'lypémanie' for this condition which was introduced by Esquirol.[7] In 1888, Jules Cotard found that subjects with this anxious melancholy and hypochondriac delusions often were prone to use hyperbolic conditions in their criticism which he called 'delusions of enormity' (délire d'énormité).[8] It 1889 he died.

In 1893, Emile Régis coined the term "Cotard's Syndrome" which was made popular by Jules Séglas.[9] It 1896, Séglas assumed that Cotard's syndrome was an analogue to secondary paranoia.[10] Later to that Séglas classified nihilistic ideas. He classified syndrome according to origin (i.e. form and not to content) as psychosensorial, affective and motor types.[11,12]

In 1921, Tissot extracted two components from Cotard's syndrome i.e. effective and cognitive.[13] In 1968, Saavedra described 10 cases of Cotard's syndrome into three types: Depressive, Mixed and Schizophrenic cases[14] In 1995, Berrios and Luque did first evidence

based classification analysis of 100 cases and described three types: Psychotic depression (prominent melancholia), Cotard's syndrome type I (pure nihilistic delusions without affective symptoms) and Cotard's syndrome type II (nihilistic delusions with symptoms such as anxiety, depression and auditory hallucination).[1] In 1999, Yamada et al. described three stages which resulted into evolution of Cotard's syndrome: Germination stage, Blooming stage and chronic stage. These stages are explained in paper below.

Epidemiology

It is difficult to draw specific data concerning the epidemiology of Cotard's syndrome as the majority of reports were focused on single event, and few studies have been reported.[15] In Hong Kong the case study of 349 Chinese psychogeriatric patients was done. Out of which two patients were found to have Cotard's syndrome. The syndrome is found in patients with extensive depression (prevalence of 3.2%) than in normal condition (prevalence of 0.57%).[16]

It is illustrated that with an increasing age, the probability of development of Cotard's syndrome also increases. The mean age was found to be 52. However, it occasionally described in children.[17]

The study published in 2007 reviewed all cases of Cotard's syndrome since then and found mean age for development of disease was 47.7 years; But the risk of development of bipolar disorder was nine times higher for teenagers and young adults (under 25 years) when compared to adults with syndrome. [18] Also the study conducted in 1991 found that, there is slight prevalence of syndrome in women as compared to men.[14]

Etiology and Pathogenesis

Cotard's syndrome have been identified in subjects across a fairly wide spectrum of organic brain illness and psychiatric disorders.[19] Also some papers suggest that, in neurological disorders like brain tumours, epilepsy, right subdural haemorrhage, traumatic brain injury, migraine, insular atrophy, arteriovenous malformations and Laurence-Moon-Bardet-Biedl syndrome, Cotard's syndrome has been recognised.[20-23] Multiple sclerosis[24] and movement disorders like Parkinson's disease[25] may also associated with Cotard's syndrome. Finally, Cotard's syndrome described in different parts of dementias.[26]

The clinical presentation of Cotard's syndrome, derived from an analysis of 100 cases, consists mainly of depressive mood (89%), nihilistic delusions concerning one's existence

(69%), anxiety (65%), delusions of guiltiness (63%), delusions of immortality (55%) and hypochondriac delusions (58%).[1]

Symptoms

The main symptoms were found to be nihilistic delusions and hallucinations. As mentioned above, depressive mood, anxiety and hypochondriac delusions are also some major symptoms. The hypochondriac delusions include delusion of loss of body organs, destruction of entire body, soul and complete denial of existence. Mainly, patients experience angst which results into sensation of physical destruction.[27] These sensations are anguishing, which in case results into impetuous self-harming actions by patient.[28] In the case reported in 2007 self-mutilation of nose was mentioned.[29] Also symptoms like analgesia, mutism, guilt and despair go hand in hand with primary delusions.

Diagnosis

A proposal to stage Cotard 's Syndrome was made by Yamada et al. in three stages:

1. **Germination stage:** characterized by hypochondriacal symptoms, development of depression and suicidal ideation.
2. **Blooming stage:** Symptoms such as anxiety, negativism and immortality delusions are developed.
3. **Chronic stage:** Persistent depressive emotional impairment and background depressive symptoms (paranoid type).

The bodily perceptions of Cotard patients can be differentiated in terms of differential diagnosis into two forms: Anosognosia and asomatognosia. Anosognosia means denial of an illness whereas asomatognosia means unconsciousness of body part. Hence, the diagnosis is completely based on observational parameters as patient denies his/her illness. As per the Feinberg et al. patients with right hemisphere lesions show these kind of symptoms.[30] Alternatively, Cotard's patients appear to perversely understand the presence of their organs, but appear baffled about them which confirms delusional characterization.[31]

Neurobiology of cotard's syndrome

There are large number of case studies and various series of case events available, which tried to acquire neurobiological supporting (cause) of Cotard's syndrome. However, there are currently only a few detailed examples of cases which show potential neurobiological cause and hence no systemic classification was found.[32] Many neuro-imaging reports indicate no

significant structural changes in the brain in Cotard 's syndrome.[33] However, earlier neuroimaging studies identified cerebral atrophy, lateral ventricles dilatation or enlargements of interhemispheric scissures , as well as lesions of the left temporoparietal lobe and the right temporal lobe in Cotard's patients as compared to controls.

De Risio et al. studied cotard patients using pivotal SPECT (single photon emission computed tomography) and identified that in a cotard patients there is considerable decrease in striatal dopamine D2 receptor binding as compared to frontal cortex. Also, there is decrement in left uptake as compared to right side of cortex. The uptake percentage decrement was found to be -4.92%. After three months of treatment decrement was reduced to -1.42%.[34]

The Charland-Verville et al. using FDG-PET (fluorodeoxyglucose – positron emission tomography) identified that there is hypo-metabolism in the frontoparietal cortical network and hyper-metabolism in the cerebellum, brainstem and bilateral thalami. The hypo-metabolism is generally seen in precuneus, antero-posterior cingulate cortices, mesiofrontal cortex, posterior parietal and dorsolateral frontal lobes and right temporoparietal junction.[35]

Chatterjee and Mitra proposed with a medical examination of a 65-year-old Cotar's syndrome woman who had severe insular atrophy at MRI that, there is eminent role of the insular cortex (IC) in interoception abnormalities in Cotard's syndrome.[36]

In a 19-year-old cotard patient with psychotic and catatonic symptoms, a recent FDG-PET study also documented significant hypo-metabolism in insular cortices and occipital lobes along with prominent frontotemporal hyper-metabolism. That changed significantly after treatment. Though the patient was still experiencing abnormal body sensations.[37]

The role of IC in Cotard's syndrome pathophysiology is consistent with other neurobiological theories that associate Cotard's syndrome with dysfunctions in brain areas dedicated to the elaboration and integration of body sensations, such as premotor cortex and temporoparietal junction, considered to be process body possession, self-location in the world and self-oriented environmental perspective.[38]

Alexithymia is traditionally defined as inability to understand one's own feelings or unable to explain them.[39] However, latest theories envisage alexithymia as a complex interception disorder involving abnormalities of the anterior cingulate cortex and the insular cortex.

Alexithymia is a possible neurobiological viaduct that could be used to nestle Cotard-like symptoms[40] While a thorough investigation into the alexithymia in prevalence of Cotard's syndrome-complicated psychiatric disorders is needed, the above mentioned theory may explain the prevalence of Cotard's syndrome particularly in affective and psychotic disorders with severe symptoms frequently associated with psychosomatic manifestations.

Also, a few studies offer thorough analysis of neuropsychological examination. Much of the research is performed using the tasks of face recognition. A general deficiency in all forms in face processing is proposed, with no proof that face recognition failure may be due to the lack of familiar information. Usually natural identification of emotional facial expressions is observed. [41]

Treatment

Cotard's syndrome has a stronger prognosis in the sense of psycho-organic diseases than in the case of depressive disorders, which may respond to antipsychotic treatments and persist as treatment-resistant symptoms.[14] Through and wide, Cotard's syndrome therapies are generally based on a detailed analysis of the underlying condition. A large number of studies detailing successful therapies have been published.

A large portion of the studies conclude that the most effective treatment for Cotard's syndrome is electroconvulsive therapy (ECT), including patients who do not respond to the usual first and even second and third line treatments i.e. in treatment refractory cases. [14] Different trials of SPECT and FDG-PET confirmed the success of ECT, not just in the treatment of symptoms but also in the alleviation of certain neurobiological alterations. For instance, Petracca et al. and Caliyurt et al. identified all brain perfusion changes were partly restored after ECT in their SPECT studies.[33,42] Moreover, after 14 ECT sessions, Ozkan and Caliyurt identified full resolution of DMN (default mode network) hypometabolism and basal ganglia hyperactivity in a 48-year-old schizophrenic patient with treatment-resistant Cotard's symptoms.[43] Furthermore, a partial restoration in frontotemporal hyper-metabolism is observed in 19-year-old patient after six sessions of ECT.[37]

Apart from ECT, several reports also suggested using pharmacological medications for treatment of Cotard's syndrome.

Monotherapy of tricyclic and SSRI (selective serotonin reuptake inhibitor) antidepressants have been shown to be effective against Cotar's syndrome.[44-46] Also, antipsychotics like clozapine, sulpiride, olanzapine and aripiprazole are utilizing successfully to treat Cotard's syndrome in psychotic and neurologic patients. [47-50] Moreover, lithium has been identified as having an impact in the treatment of CS in bipolar patients.[45] Whereas carbamazepine is used in post-seizure Cotard's syndrome.[51] Also, nomifensine (dopamine reuptake inhibitor) is used against Cotard's patients.[52]

The majority of cases report combination strategies with different classes of medicines used in Cotard's syndrome treatment which includes antipsychotics, antidepressants, benzodiazepines and mood stabilizers. The useful combinations are haloperidol and clomipramine, risperidone and fluoxetine, haloperidol and mirtazapine, aripiprazole and escitalopram, risperidone and citalopram, risperidone and sertraline, amitriptyline and clomipramine, clozapine and fluvoxamine and imipramine, quetiapine and venlafaxine, olanzapine and escitalopram and lorazepam.[52]

According to Enoch, ECT tends to have more positive effects on Cotard's syndrome in affective disorders than in psychotic disorders, making it the most accepted treatment.[14]

CONCLUSION

This detailed review has focused on the genesis, history and treatment of Cotard's syndrome and presented in a simple and understandable language. At present, Cotard's syndrome falls under the category of 'somatic delusions' in fifth edition of Diagnostic and Statistical Manual of Mental Disorders (DSM V) and in International Statistical Classification of Diseases and Related Health Problems (ICD) of World Health Organization. Also, reports in successive decades recognised symptoms of Cotard's syndrome arise in the context of different neuropsychiatric diseases.

Though the syndrome is fairly rare, greater work is required to explain the mechanisms of pathophysiology which will support this condition. ECT therapy is found more prominent in the treatment when compared to other pharmacological medications. Apart from case studies, more literature is required in order to determine exact cause and other possible medications and treatments for Cotard's syndrome.

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