# CATATONIA IN OBSESSIVE COMPULSIVE DISORDER

# K.JAGADHEESAN , S.HAQUE NIZAMIE & ANUPAM THAKUR

### **ABSTRACT**

Catatonia occurs in a wide range of neuropsychiatric conditions. Among the psychiatric disorders, occurrence of catatonia has rarely been documented in obsessive-complsive disorder. Given the paucity of reports, we report two cases of obsessive compulsive disorder that presented as catatonia.

Key words: Catatonia, obsessive-compulsive disorder, phenomenology, neurobiology

Catatonia, a syndrome characterized by several motor abnormalities, occurs in a wide range of psychiatric, medical and neurological conditions (Gelenberg, 1976; Barnes et al., 1986; Shiloh et al., 1995; Ahuja, 2000). Of the psychiatric conditions, catatonia or catatonic signs have seldom been documented in association with obsessive compulsive disorder (OCD). Following a detailed literature search, we have found only two reports mentioning catatonia as a presenting feature of OCD. In an early report, Blacker (1966) indicated the occurrence of catatonic signs such as mutism, negativism and waxy flexibility in a patient with long standing OCD. In another report, Hermesh et al., (1989) described two patients of OCD presenting with prominent catatonic features viz., mutism, negativism, and posturing. For the rarity of this entity and its obvious implications for the management and conceptual issue, we report two cases of catatonia in OCD.

### **CASE REPORTS**

Patient No.1: An 18-year-old female was brought to our outpatient clinic with a history of episodes

of mutism, negativism and posturing. According to the family members, she had been having fear of dirt and contamination, excessive concern over cleanliness and washing and bathing rituals over the last three years. Whenever her rituals were prevented, she used to feel sad and made suicidal attempts when she could not tolerate anxiety associated with the intense fear of dirt and contamination and during one such event she burnt her upper body. In addition, a year later, she also started getting transient episodes of possessions attacks and visual loss. Later, in the last six months, she developed episodes of mutism, negativism, maintaining sitting postures with both hands kept in between her thighs and unresponsiveness.

For catatonic signs, she received injection lorazepam 2-mg/day thrice daily and within two days, all these signs disappeared. During subsequent interviews, she was noted to have depressed affect, obsessions of dirt and contamination, obsessional doubts that whether she could do an act or not, washing compulsion, magical thinking, ambitendency. She explained that she had experienced a great distress and anxiety, felt confused and lost awareness when

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her obsessional doubts that whether she could do an act or not became intolerable. Furthermore, the episodes of ambitendency, mutism, negativism, posturing and dissociative symptoms were correlated with intense psychic anxiety that emerged consequent to obsessions. She was diagnosed with OCD with unspecified psychosis (catatonia). Although her symptoms worsened with the combination of clomipramine (200 mg/day) and risperidone (3 mg/day), subsequently she improved well with a combination therapy for six weeks including clomipramine (200 mg/day), thioridazine (200 mg/day) and buspirone (20 mg/day).

Patient No.2: A 24 year-old male, having a family history of alcohol dependence syndrome in his brother, was brought with the following history. Having remained withdrawn and poorly interested in work for six months, patient developed episodes of odd posturing characterized by standing continuously on one leg for several hours with the belief that if he puts his other foot on ground, serious harm would happen to his brother and sister-in-law, extreme slowness of movement and stiffness of limbs. In addition, he also began spending 15-30 minutes before taking each step while walking, spending more time during bathing and contemplating the probable response in his head whether it is 'right' or 'wrong' several time before doing anything from eating or answering simple questions.

His first psychiatric examination revealed slowness, irritable affect, negativism, compulsive counting and magical thinking and absence of delusions and hallucinations. With the diagnosis of catatonic schizophrenia, treatment was initiated with electroconvulsive therapy (ECT). He did not improve with a course of ECT (Six) and several adequate antipsychotics trials rather his catatonic symptoms worsened and he additionally developed gegenhalten, persevarative speech, stereotypy and blocking. Because subsequent interviews consistently revealed, in addition to catatonic features, anxious affect, obsessional ideas, compulsive checking, magical thinking and ambivalence, his diagnosis was changed to obsessive-compulsive disorder with unspecified

psychosis (catatonia), and treatment was initiated with amitriptyline (250 mg/day) and augmenting agent lithium (900 mg/day). Though his symptoms responded inadequately to the above treatment, subsequently he improved to the second trial of ECT (Thirteen) and a combination of imipramine (150 mg/day) and trifluperazine (20 mg/day).

#### DISCUSSION

Both patients of this report had features of both obsessive-compulsive disorder and catatonic signs. In line with earlier observations (Blacker, 1966; Hermesh et al., 1989), patients of this report had commonly observed catatonic signs. None of these patients had features suggestive of major psychoses such as delusions or hallucinations. Further, depressive symptoms were noted only in patient 1, and these symptoms appeared after the onset of OC symptoms. In addition to catatonic signs, patient 1 had developed dissociative symptoms. Occurrence of such kind of symptoms in a patient with OCD is not a new phenomenon given that a similar presentation has been reported earlier (Grabe et al., 1999). It rather indicates the progression of OCD and worsening of subjective anxiety. Although OC symptoms became prominent later, for social withdrawal and declined interest in work that started earlier, the second patient initially received the provisional diagnosis of catatonic schizophrenia. Notably, his catatonic signs improved when an antidepressant was added. Response to a drug cannot be a reliable indicator of diagnosis, however, in the absence of core symptoms of a major psychiatric disorder, this drug response maybe considered as supplementary evidence to justify the diagnosis of OCD with unspecified psychotic disorder rather than catatonic schizophrenia. Because international classificatory systems, ICD-10 (World Health Organization, 1992) and DSM-IV (American Psychiatric Association, 1994), allow the diagnosis of catatonia exclusively under major psychoses, we could not make a single diagnosis that allows the diagnosis of both OCD and catatonia together.

Based on the existing neurobiological

## **CATATONIA IN OCD**

evidences, occurrence of catatonia in OCD seems. possible. First, a common brain substrate, frontal lobes, has been found involved in both OCD (Khanna, 1988; Jenike et al., 1996; Fitzgerald et at., 1999; Stein, 2000) and catatonia (Cummings. 1985; Joseph, 1990). Second, both in OCD and in catatonia, involvement of dopaminergic and GABAergic systems have been recognized. Catatonia is reported to occur in hyper as well as hypodopaminergic states (Menza & Haris, 1989). mediated **GABA** reduction hyperdopaminergic state, particularly in basal ganglia, is a proposed mechanism to explain benzodiazepines' effectiveness in catatonia (Rosenbaugh & Mazurek, 1991), Serotonin exerts inhibitory control over dopamine in all the brain regions including striatum (Kapur & Remington, 1996), hence, dopaminergic hyperactivity is expected to occur in conditions associated with serotonergic system hypofunction. OCD is one such condition that is associated with serotonergic system hypofunction (Charney et al., 1998). In addition, evidences (Fitzgerald et al., 1999) indicating the beneficial roles of antipsychotics and benzodiazepines also hint at a possibility of hyperdopaminergic state in the subcortical structures in OCD could have resulted in catatonia.

The clinical relevance of this report is of two folds. Firstly, it suggests that catatonia maybe manifestation of severe OCD. Secondly, it underscores that an appropriate treatment of OCD is essential to manage and prevent the occurrence of catatonia. Although rare, patients of OCD presenting with catatonia maybe investigated to understand the neurobiological aspects of OCD in detail.

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