

Case Report

Primary Obsessive Slowness: A Complex Presentation and Treatment Difficulties

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Abstract: Obsessive slowness is described as a complex and disabling clinical syndrome that causes extreme slowness in performing tasks, with potential personal and functional impairment. It is a rare condition with a challenging differential diagnosis with obsessive-compulsive disorders, mental retardation and catatonia, and its existence as an independent syndrome is still debated by authors and not included by classification systems. Therefore, its treatment management is not well-defined and it still represents a clinical challenge for clinicians. Currently, the main proposal is a mix of antidepressant, antipsychotic, psychoeducation, psychotherapy and biological non-pharmacological interventions. Hereby, we describe a case of an 18-year-old male patient who presented debilitating slowness and severe impairment. Managing his treatment was particularly challenging for clinicians and was ultimately improved with escitalopram 30 mg/day combined with memantine 10 mg/day and amisulpride 400 mg/day.

Keywords: antidepressant; antipsychotic; case report; co-medication; differential diagnosis; obsessive-compulsive disorder; personalized treatment; psychopharmacology; tailoring; treatment



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1. Introduction

Obsessive-compulsive disorder (OCD) is one of the most common and severe psychiatric disorders. It presents a juvenile onset and a chronic-relapsing trend, clinically characterized by intrusive and disturbing thoughts (obsessions) and repetitive behaviors (compulsions) that individuals feel driven to perform [1]. Starting with the DSM-5, and confirmed in the DSM-5 TR, OCD was removed from the chapter on anxiety disorders, resulting in the creation of a specific chapter on OCD and related disorders. This Copernican change did not exclude the possibility of comorbidity or overlap between disorders, but it has restored an autonomous dignity to the OCD, with a nosologic revision reflecting the growth of neurobiological and treatment response data that distinguishes OCD from anxiety disorders such as panic disorder, generalized anxiety disorder or social anxiety disorder [2–4]. In fact, it very often occurs in comorbidity with other psychiatric disorders, including anxiety disorders, affective disorders, psychosis, sleep disorders and eating disorders, with consequences both on prognosis and on the general quality of life [1,5,6].

Psychopathologically, OCD is typically characterized by a number of recurring themes, including fears of illness and contamination, unwanted aggressive thoughts, sexual or religious taboo thoughts, the need for symmetry or cleanness and/or compulsions, such as excessive arranging, checking, counting, repeating or reassurance [1,7].

Furthermore, dysfunctional OCD is characterized by primary obsessive slowness, a psychopathological construct that some authors have considered as a derived condition of the clinical symptomatology. According to this theory, in fact, the slow performance of OCD patients may represent an epiphenomenon linked to a meticulous concern for the correct execution of each task or an intrusion of obsessive thoughts that can influence the speed with which cognitive functions are carried out [8–11]. The proposed alternative, on the other hand, considers that patients with OCD show a cognitive slowness relatively independent of those clinical factors. Data from the literature support the hypothesis, which states that patients with OCD are more likely to exhibit slowness in functional executive tasks subserved by frontostriatal circuits, rather than slowness resulting in a generalized deficit in time-mediated tests [12]. Finally, several studies question the validity of the first hypothesis, as a recent meta-analysis found that, when the OS component was controlled, only a small association with the severity of obsessive symptoms was observed [13], thus discrediting symptom-explanations based on slowness in OCD. However, although OS represents a crucial component of OCD clinical features, it has also been reported autonomously. Indeed, when OS appears so prominent that it overpowers the other clinical manifestations (e.g., obsessions and compulsions) it almost assumes a connotation of a transdiagnostic element, which is not just the OCD, but could, for example, also be identified in other psychiatric disorders or be described on its own [14,15].

OS is an extremely rare clinical syndrome characterized by a debilitating slowness of all movements and all voluntary adaptive activities [16]. It is a diagnosis of exclusion with great complexity in management [17].

We report the case of an adolescent male who manifested extreme slowness in all voluntary activities with catatonic signs, as well as initially transient psychotic signs. A diagnosis of obsessive-compulsive disorder (OCD), concomitant with obsessive slowness and nonspecific psychosis, was made. He had shown clinically significant improvement when anti-obsessional drugs were added in comedication with anti-psychotics that have already been prescribed with minimal response. To the best of our knowledge, this is the first case of primary OS describing the challenges in diagnosis and treatment of this clinical condition.

2. Case Presentation

Mr. X, an 18-year-old boy and 10th standard student, whose history is characterized by low mood, withdrawn behavior, psychomotor retardation, suspiciousness for 6 months, mutism and posturing for 8 to 10 h together in a day, also poor self-care, decreased sleep and appetite for 2 months. On examination, mutism, posturing, negativism, and stupor was present. BFCRS (Bush Francis Catatonia Rating Scale) score was 25, reduced to 17 following lorazepam per os testing. He was provisionally diagnosed with psychosis/severe depression with psychotic symptoms and started on escitalopram 10 mg/day along with lorazepam 2 mg per os three times daily. Since the treatment was not of much help in reducing catatonia further, he started on ECT (Electroconvulsive therapy). Then, the BFCRS decreased to 13 with ECTS (8 Bifrontal ECTs with 180 mc charge) in 10 days. Improvement in catatonia symptoms was noted. However, the patient continued to show amotivation. During the hospital course, as the effect stabilized after 3rd ECT, he started on a 10 mg tablet of olanzapine and increased to 20 mg/day in view of non-affective psychosis. He was found to be inactive and speaking occasionally after 3rd ECT, but improvement did not last long after stopping ECT. He was found to be mute most of the time. He asked his aunt to support him in daily activities. Positive reinforcement techniques were tried, but little cooperation was evident on his part. Cognitive-behavioral psychotherapy was also attempted but without any result due to the patient's lack of participation and collaboration, and it was therefore suspended after a few sessions. The patient was discharged on request after 1 month on olanzapine 20 mg, combined with fluoxetine 20 mg to 40 mg/day, given to stimulate behavioral activation. After 3 months, the patient again presented with traits of mutism, withdrawn behavior, posturing, and

negativism; however, he improved with regard to suspiciousness and self-care. Given the failure of the lorazepam and olanzapine trial in the last IP care, a trial of amisulpride 100 mg/day with zolpidem 10 mg/day was started. Improvements in catatonia symptoms were noted with zolpidem 30 mg/day (1/2 10 mg tablet 6 times daily, with the aim of covering the whole day with its short half-life) within 2 weeks, and the BFCRS score reduced to 4 points. However, he continued to have mild fear and was inhibited during the interview whenever amisulpride was increased to 400 mg/day. He was seen to be drowsy most of the time, so zolpidem was reduced to 10 mg/day within a span of 2 weeks. Subsequently, his catatonic symptoms started to worsen again. Therefore, treatment with memantine was started at this point of time in view of the catatonic symptoms, as he was not talking again. Through observations, it was noted that he clung to his aunt and would ask her to accompany him wherever he went. He did not want to touch food and asked his aunt to feed him. First, he gave no reason. However, after some days, he started expressing that if he ate with his own hand, something bad would happen to everyone and he would not process the same. He gradually began expressing that, if he spoke up, there would be bad consequences. Then, he would get distressed and cry if someone touched his one side of the body but not the other and kept asking, through gesturing, bystanders to touch the other side (for an example, other side of the shoulder or the other side of the cheek). He got stuck on doors, asking which foot to hold first, and would not proceed unless touched by someone and, because of this, kept asking people to touch him. He also told the reason for not eating with his own hand: food would accumulate only on one side of his stomach if he ate on his own. A differential diagnosis of Primary Obsessive Slowness was made.

2.1. Assessment

Routine investigations, including complete blood count, liver function test and renal function tests, were normal. A lorazepam-assisted interview was also done after an informed consent from his aunt. He was given 6 mg lorazepam in 300 mL in 0.05 mg/min speed (40 drops per minute). The patient was asked general questions about his school, friends, daily activities, hobbies, etc. He was not very fluent in the conversation, but he was trying to reply gradually. However, the interview was not free floating, despite multiple prompting. Hence, the interview was terminated and there were no complications after the lorazepam test. After the diagnosis of Primary Obsessive Slowness, the Y-BOCS checklist was tried for which patient was not cooperative.

2.2. Case Conceptualization

We conceptualized the case as OCD and primary obsessive slowness with psychosis. Although there was a limitation on the part of the patient, from whom we were not able to obtain much, given his muteness, we still managed to describe the OCD through the observations and narrations of the assistants (apart from the psychotic symptoms that presented during the initial course of the illness). We have also noted improvements in the patient following the administration of an adequate dose of anti-obsession medication.

2.3. Course of Treatment and Assessment of Progress

Fluoxetine supplementation was stopped, given the absence of significant responses. It was therefore started with escitalopram 10 to 30 mg/day as anti-obsessional dosages with existing memantine 10 mg/day and amisulpride 400 mg/day. The patient showed improvement in social interactions, verbal communication and also started taking part in ward activities including singing and dancing. The patient did not report any psychotic symptoms, was psychoeducated about OCD and ERP was tried considering the symptoms that he had. He was, subsequently, moved to the residential rehabilitation center for environmental modification.

2.4. Complicating Factors

The fact that there was little information we could elicit from the patient because of his recurrent catatonic symptoms, and that only observation and information could be relied upon, was a challenge in the diagnosis. Nonresponse to different agents to treat catatonia made it difficult to grasp the underlying pathology.

3. Treatment Implications of the Case

OCD may present as Primary Obsessive slowness, characterized by debilitating slowness in all voluntary adaptive movement and activities. In addition, an OCD patient with poor insight has delusional beliefs that are obsessive in nature, but the treatment mainly considered consists of anti-obsessional drugs with a reduced presence of anti-psychotic doses of neuroleptics. The fact that the patient showed improvement with anti-obsessional drugs and low doses of anti-psychotics may have worked as dose escalation for OCD. In this regard, escitalopram was prescribed, exceeding the classical antidepressant dose of 20 mg per day, in an attempt to obtain a greater anti-obsessive effect and always on the basis of evidence-based considerations, with strict cardiological monitoring to avoid QTc prolongation [18]. Furthermore, catatonia or catatonia-like pictures, due to primary obsessive slowness of OCD, might not improve with the conventional medicines of catatonia such as lorazepam or ECT. Furthermore, ECT was used relatively early in the case presented, before attempting further pharmaceutical trials with antidepressants, antipsychotics, benzodiazepines or their combination. This choice was dictated by the poor response to treatment, the clinical severity of the case and the availability of ECT and, anyway, it was supported by literature data even in the early stages of therapeutic management [19–21]. Though zolpidem and memantine were helpful initially, they were not sustained. On the other hand, amisulpride was prescribed because of its substantial affinity for 5-HT₇ and GHB receptors, apart from D₂ and D₃ presynaptic and postsynaptic receptors [22–24]. The use of stimulants was also considered but not carried out due to cost and lack of availability. Hence, difficulties in diagnosing primary obsessive slowness makes it nearly impossible to select an anti-obsessive agent leading to nonresponse or extremely delayed response as in our case. Solid, evidence-based data about primary obsessive slowness are haphazard and there is a need to increase clinicians' awareness on this topic. In a similar, previously published case [25], a 21-years-old male responded to a combination of behavioral therapy (thinking habit and exposure) and pharmacotherapy (fluoxetine and thyroxine), although authors highlighted diagnostic difficulties and management issues similarly to our case.

4. Discussion

OS is a rare and damaging clinical condition, first described in 1974 [26] and still far from to be deeply understood [15]. To date, there are only sporadic case reports describing this condition. Therefore, solid evidence-based data is still missing [27]. The main hypothesis to explain the phenomenon was that, although patients with OS had OCD, the motor symptoms were not related to the presence of obsessions or compulsions that could be expected to cause motor slowness (such as mental checks and rituals), but they were instead related to a separate and "primary phenomenon". In the case that we have reported, we confirm this hypothesis, placing the diagnosis of OS in comorbidity with OCD. This derives from two main elements: the first, because to date, OS is not considered an autonomous disorder by international classification systems and, secondly, because the existing literature and the proposed assessment go in the same direction.

OS causes significant impairments at the level of the individual's personal, social and occupational functioning, resulting in a significant reduction in the quality of life of the subject. However, debates remain open about whether the SO is truly a distinct and primary entity or may, for phenomenological reasons, be the expression of a number of other conditions [11,16,28].

Considering the rarity of this syndrome, one of the main problems related to its management is the correct identification and diagnosis by the clinicians, together with the suggestive therapeutic management. This was also one of the problems encountered in

the case we described, where clinicians, in addition to having to resolve the diagnostic doubt, asked themselves the question of which treatment to follow in light of the lack of international guidelines on the subject. The controversial nature of the diagnosis of OS is also not helpful in clarifying the true epidemiology of the disorder, with the possibility that it may, therefore, be strongly under-identified and under-diagnosed.

A recent literature review identified a total of 58 articles using the term OS, and 15 articles were identified with sufficient clinical information for 77 patients [27]. Another disorder with which OS enters the differential diagnosis is catatonia, with which it shares profound difficulties in initiating voluntary action, slow movements, poor language production, motor perseverations and abnormal postures [27,29].

In addition, some neurological diseases associated with OS symptoms, such as Down syndrome and autism spectrum disorder, are also associated with catatonic symptoms [30–32]. Therefore, we can speculate that the two disturbances may, in fact, constitute a spectrum with OS representing a whip-like form of catatonia [33]. We can assume that a confounding element in the evaluation of the OS has been the lengthy use of the word “primary” [26]. The significance of the addition of the term “primary” lies in describing a separate and distinct disease entity, but, in the specific case of OS, we believe it goes beyond the meaning that the authors have attributed to it. It is much more likely that the goal was to discriminate the symptoms described for OS from the more common symptoms of OCD or obsessions and/or compulsions, even severe ones, such as causing a secondary ideomotor slowness. In fact, even reading the first case reports, one notes that the intent was always to describe a peculiar and particularly accentuated slowness in patients already diagnosed with OCD and, apparently, it was unrelated to the obvious obsessive-compulsive rituals [27].

In this sense, if we consider OS as a rare and peculiar symptom, which can present itself in OCD, rather than considering it an autonomous entity, we can better understand the numerous case reports given over time on this topic. In fact, many cases have been described of patients with OCD and various comorbidities, who presented a marked severity of OS [14,25,28,32,34].

Thus, OS is certainly a rare, peculiar clinical condition that is difficult to diagnose and manage, very disabling for patients and not always clearly related to evident OCD. Sometimes, there may be a misdiagnosis of catatonia or a genetic syndrome or neurological condition, but the etiopathogenesis appears more similar to OCD. In light of these considerations, it is evident that a greater study of this psychopathological condition is necessary and still missing. Therefore, the goal of our work is to shed light on this disorder in order to increase the evidence-based information available to clinicians and researchers.

5. Conclusions

On some occasions, diagnosing a case can be a real challenge when the patient has selective mutism or mutism, and there is little information from the patient and caregivers, but a careful behavioral analysis can be helpful in the diagnosis and case management. Primary obsessive slowness may be of such a type that it puts the clinician in a dilemma to choose between the psychotic spectrum, the neurotic spectrum or the interphase of both.

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References

1. Goodman, W.K.; Grice, D.E.; Lapidus, K.A.B.; Coffey, B.J. Obsessive-Compulsive Disorder. *Psychiatr. Clin. N. Am.* **2014**, *37*, 257–267. [[CrossRef](#)] [[PubMed](#)]
2. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders: DSM-5*, 5th ed.; American Psychiatric Pub: Washington, DC, USA, 2013.
3. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition—Text Revision (DSM-5-TR)*; American Psychiatric Pub: Washington, DC, USA, 2022.
4. Phillips, K.A.; Stein, D.J.; Rauch, S.L.; Hollander, E.; Fallon, B.A.; Barsky, A.; Fineberg, N.; Mataix-Cols, D.; Ferrão, Y.A.; Saxena, S.; et al. Should an obsessive-compulsive spectrum grouping of disorders be included in DSM-V? *Depress. Anxiety* **2010**, *27*, 528–555. [[CrossRef](#)] [[PubMed](#)]
5. De Filippis, R.; Aloï, M.; Bruni, A.; Gaetano, R.; Segura-Garcia, C.; De Fazio, P. Bipolar disorder and obsessive compulsive disorder: The comorbidity does not further impair the neurocognitive profile. *J. Affect. Disord.* **2018**, *235*, 1–6. [[CrossRef](#)] [[PubMed](#)]
6. Gaetano, R.; de Filippis, R.; Segura-Garcia, C.; De Fazio, P. Impact of Bipolar Disorder and Obsessive-Compulsive Disorder comorbidity on neurocognitive profile: A mini-review. *Psychiatr. Danub.* **2020**, *32*, 346–350. [[CrossRef](#)] [[PubMed](#)]
7. Husted, D.S.; Shapira, N.A.; Goodman, W.K. The neurocircuitry of obsessive-compulsive disorder and disgust. *Prog. Neuro-Psychopharmacol. Biol. Psychiatry* **2006**, *30*, 389–399. [[CrossRef](#)]
8. Simón-Martínez, V.; Laseca-Zaballa, G.; Lubrini, G.; Periañez, J.A.; Martínez Álvarez, R.; Torres-Díaz, C.V.; Martínez Moreno, N.; Álvarez-Linera, J.; Ríos-Lago, M. Cognitive deficits and clinical symptoms in patients with treatment-refractory obsessive-compulsive disorder: The role of slowness in information processing. *Psychiatry Res.* **2021**, *304*, 114143. [[CrossRef](#)]
9. Galderisi, S.; Mucci, A.; Catapano, F.; D’amato, A.C.; Maj, M. Neuropsychological Slowness in Obsessive-Compulsive Patients. *Br. J. Psychiatry* **1995**, *167*, 394–398. [[CrossRef](#)]
10. Roth, R.M.; Baribeau, J.; Milovan, D.L.; O’Connor, K. Speed and accuracy on tests of executive function in obsessive-compulsive disorder. *Brain Cogn.* **2004**, *54*, 263–265. [[CrossRef](#)]
11. Veale, D. Classification and Treatment of Obsessional Slowness. *Br. J. Psychiatry* **1993**, *162*, 198–203. [[CrossRef](#)]
12. Riesel, A.; Kathmann, N.; Klawohn, J. Flexibility of error-monitoring in obsessive-compulsive disorder under speed and accuracy instructions. *J. Abnorm. Psychol.* **2019**, *128*, 671–677. [[CrossRef](#)]
13. Abramovitch, A.; McCormack, B.; Brunner, D.; Johnson, M.; Wofford, N. The impact of symptom severity on cognitive function in obsessive-compulsive disorder: A meta-analysis. *Clin. Psychol. Rev.* **2019**, *67*, 36–44. [[CrossRef](#)] [[PubMed](#)]
14. Mittal, A.K.; Majumder, P.; Agrawal, A.; Sood, M.; Khandelwal, S.K. Early Onset Obsessive Compulsive Disorder with Obsessive Slowness: A Case Report and Demonstration of Management. *Indian J. Psychol. Med.* **2013**, *35*, 407–409. [[CrossRef](#)] [[PubMed](#)]
15. Takeuchi, T.; Nakagawa, A.; Harai, H.; Nakatani, E.; Fujikawa, S.; Yoshizato, C.; Yamagami, T. Primary obsessional slowness: Long-term findings. *Behav. Res. Ther.* **1997**, *35*, 445–449. [[CrossRef](#)]
16. Ratnasuriya, R.H.; Marks, I.M.; Forshaw, D.M.; Hymas, N.F.S. Obsessive Slowness Revisited. *Br. J. Psychiatry* **1991**, *159*, 273–274. [[CrossRef](#)] [[PubMed](#)]
17. Gowda, G.S.; Sadh, K.; Singh, A.; Bada Math, S. Obsessive slowness mimicking catatonia in an adolescent—A case report. *J. Obsessive. Compuls. Relat. Disord.* **2017**, *13*, 14–17. [[CrossRef](#)]
18. Shim, G.; Park, H.Y.; Jang, J.H.; Kim, E.; Park, H.Y.; Hwang, J.Y.; Kim, S.N.; Jang, G.-E.; Kwon, J.S. What is the optimal dose of escitalopram for the treatment of obsessive-compulsive disorder? A naturalistic open-label study. *Int. Clin. Psychopharmacol.* **2011**, *26*, 284–290. [[CrossRef](#)]
19. Maletzky, B.M. The First-line Use of Electroconvulsive Therapy in Major Affective Disorders. *J. ECT* **2004**, *20*, 112–117. [[CrossRef](#)]
20. Lloyd, J.R.; Silverman, E.R.; Kugler, J.L.; Cooper, J.J. Electroconvulsive Therapy for Patients with Catatonia: Current Perspectives. *Neuropsychiatr. Dis. Treat.* **2020**, *16*, 2191–2208. [[CrossRef](#)]
21. Sienaert, P.; Dhossche, D.M.; Vancampfort, D.; De Hert, M.; Gazdag, G. A Clinical Review of the Treatment of Catatonia. *Front. Psychiatry* **2014**, *5*, 181. [[CrossRef](#)]
22. Glatard, A.; Guidi, M.; Delacrétaz, A.; Dubath, C.; Grosu, C.; Laaboub, N.; von Gunten, A.; Conus, P.; Csajka, C.; Eap, C.B. Amisulpride: Real-World Evidence of Dose Adaptation and Effect on Prolactin Concentrations and Body Weight Gain by Pharmacokinetic/Pharmacodynamic Analyses. *Clin. Pharmacokinet.* **2020**, *59*, 371–382. [[CrossRef](#)]
23. Arora, M.; Banal, R.; Praharaj, S.K.; Mahajan, V. Amisulpride Augmentation in Acute Catatonia. *Am. J. Ther.* **2017**, *24*, e381–e385. [[CrossRef](#)] [[PubMed](#)]
24. Katsioulis, G.E.; Argyraki, M.; Ekonomopoulou, M.T.; Mourelatos, C.; Iakovidou-Kritsi, Z.; Mourelatos, D. Typical versus atypical antipsychotics: Genotoxicity and cytostaticity testing. *Integr. Cancer Sci. Ther.* **2020**, *7*, 1–6. [[CrossRef](#)]
25. Singh, G.; Sharan, P.; Grover, S. Obsessive slowness: A case report. *Indian J. Psychiatry* **2003**, *45*, 60–61. [[PubMed](#)]
26. Rachman, S. Primary obsessional slowness. *Behav. Res. Ther.* **1974**, *12*, 9–18. [[CrossRef](#)]
27. Ganos, C.; Kassavetis, P.; Cerdan, M.; Erro, R.; Balint, B.; Price, G.; Edwards, M.J.; Bhatia, K.P. Revisiting the Syndrome of “Obsessional Slowness”. *Mov. Disord. Clin. Pract.* **2015**, *2*, 163–169. [[CrossRef](#)]
28. Hymas, N.; Lees, A.; Bolton, D.; Epps, K.; Head, D. The neurology of obsessional slowness. *Brain* **1991**, *114*, 2203–2233. [[CrossRef](#)]
29. Gjessing, L.R. A review of periodic catatonia. *Biol. Psychiatry* **1974**, *8*, 23–45.
30. Wing, L.; Shah, A. Catatonia in autistic spectrum disorders. *Br. J. Psychiatry* **2000**, *176*, 357–362. [[CrossRef](#)]

31. Jap, S.N.; Ghaziuddin, N. Catatonia Among Adolescents With Down Syndrome. *J. ECT* **2011**, *27*, 334–337. [[CrossRef](#)]
32. Charlot, L.; Fox, S.; Friedlander, R. Obsessional slowness in Down's syndrome. *J. Intellect. Disabil. Res.* **2002**, *46*, 517–524. [[CrossRef](#)]
33. Fontenelle, L.F.; Lauterbach, E.C.; Telles, L.L.; Versiani, M.; Porto, F.H.; Mendlowicz, M.V. Catatonia in Obsessive-compulsive Disorder: Etiopathogenesis, Differential Diagnosis, and Clinical Management. *Cogn. Behav. Neurol.* **2007**, *20*, 21–24. [[CrossRef](#)] [[PubMed](#)]
34. Clark, D.A.; Sugrim, I.; Bolton, D. Primary obsessional slowness: A nursing treatment programme with a 13-year-old male adolescent. *Behav. Res. Ther.* **1982**, *20*, 289–292. [[CrossRef](#)]