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LETTER TO THE EDITORS

Uncertainty about our own beliefs: the co-occurrence of obsessive and psychotic phenomena

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The presence of obsessive-compulsive symptoms (OCS) in schizophrenia-spectrum disorders (SSD) has been described since the beginning of modern psychiatry.[1] The co-occurrence of obsessive-compulsive disorder (OCD) and SSD exceeds what would be expected by chance and is much higher than the prevalence of each disorder alone in the general population (1-2%). Around 25% of patients with schizophrenia suffer from OCS and about 12% meet the criteria for OCD,[2] giving rise to hypotheses of shared etiopathogenetic mechanisms, including common neurobiological and genetic underpinnings. Several studies have shown that OCS are associated with more clinically severe SSD and a higher risk of transition to psychosis in at-risk mental states.[1] Furthermore, the timing of OCS onset (whether pre-psychotic, concurrent, or post-psychotic) appears to be linked to distinct clinical trajectories.[1] Family studies have provided additional support, showing that first-degree relatives of individuals with schizophrenia have a higher prevalence of OCS/OCD, and conversely, that relatives of individuals with OCD are more likely to present SSD.[1] Polygenic risk score analyses have revealed overlapping genetic risk between OCD and schizophrenia, which further supports a partially shared genetic architecture.[3] This association may also be relevant to treatment even in patients without apparent comorbidity. A recent study comparing cognitive behavioral therapy response and polygenic risk scores found that individuals with higher polygenic risk scores for schizophrenia were less responsive to cognitive behavioral therapy.[4]

Nevertheless, the overlap of catatonic, psychotic, affective, and obsessive phenomena makes the clinical picture difficult to grasp. This overlap further reinforces the "lost in translation" problem between neurobiological substrates and therapeutic targets, as symptom boundaries in clinical practice are often fuzzy. We have conducted neurobiological studies on poorly defined clinical phenotypes while treating overlapping conditions such as SSD + OCD as simple comorbidities. But what if this apparent overlap actually points to a distinct clinical entity with unique therapeutic responses? In this context, clinical phenotypes described with sufficient granularity to bridge phenomenology and pathogenetic mechanisms are needed.[5] This demands a commitment to clinical detail – a move "back to the future."

Contemporary classifications accept OCD with little or no insight and without resistance, features that are traditionally described in psychotic disorders, with low insight occurring in roughly 20% of OCD patients.[6] This group of patients typically has severe symptomatology and greater comorbidity with schizotypal disorder, which is genetically linked to SSD. However, the overall clinical picture is similar to OCD, with good-to-moderate insight, and insight may improve with treatment.[1] Moreover, the diagnostic overlap between OCD and SSD appears to have been emphasized over the past three decades, with a recent study reporting that over one-third of patients in specialized OCD clinics have comorbid SSD.[7] But the differences between OCS in OCD and SSD remain poorly understood. Most studies have focused merely on presence or severity, overlooking clinical features often evident in detailed interviews, such as insight, resistance to obsessions and compulsions, and their temporal relationship to other symptoms or antipsychotic treatment. The conspicuous emergence of OCS during the course of SSD raises questions about their nature. Patients with OCD show a relatively higher risk of diagnostic conversion to schizophrenia, as do their offspring, which suggests shared genetic vulnerability.[1]

From a neurodevelopmental standpoint, OCD may be positioned along a gradient that spans from continued insight to fading self-agency to SSD. This association may reflect shared changes in sensory-motor predictive mechanisms that emerge during neurodevelopment. Recent models suggest that impaired corollary discharge signals play a pathophysiological role. This would compromise the integration between motor commands and sensory feedback, thereby reducing the sense of agency over thoughts and actions while preserving the sense of ownership. Within this framework, OCD may arise from a partial loss of sense of agency, while more profound disturbances in both sense of agency and sense of ownership underlie SSD.[8]

From a neurobiological perspective, Wang et al.[9] demonstrated that patients with SSD comorbid to OCD showed unique alterations not seen in OCD or schizophrenia alone, which suggests a dynamic, bidirectional relationship between obsessive-compulsive and psychotic symptoms. This lends support to the concept of schizo-obsessive disorder as a distinct entity. The literature indicates that antipsychotics, especially clozapine, may induce or exacerbate OCS, which suggests the involvement of serotonergic pathways in this phenomenon. Patients with SSD treated with clozapine often have a longer illness duration, which seems to contribute to this association, as OCS are more prevalent in chronic psychosis than in first-episode patients. De novo onset of OCS during clozapine treatment has been reported in up to 28% of patients, while pre-existing symptoms can be exacerbated in nearly 20%.[10] Schirmbeck & Zink[10] further emphasize that clozapine-induced OCS tend to persist over time and correlate with both dose and treatment duration. This points to a complex interaction

between individual genetic vulnerability, the pharmacodynamic profile of specific second-generation antipsychotics, and the longitudinal course of SSD.

Past systematic reviews have highlighted important limitations that we aim to address.[2] First, inconsistent reporting across studies has made it difficult to assess the role of relevant variables on OCS, such as antipsychotic and antidepressant medication. Second, some studies have distinguished obsessions from psychotic symptoms while others have not, contributing to definitional inconsistencies – especially in differentiating poor insight obsessions from delusions or stereotyped behavior. Finally, the emphasis on psychotic populations (at-risk mental state, first episode psychosis, schizophrenia) over OCD samples in dimensional analyses has made cross-condition comparisons challenging, while the lack of data on the temporal relationship between OCD and psychosis has left an important gap in understanding disease trajectories.

To begin addressing these issues, we are currently conducting a systematic review and meta-analysis (PROSPERO PROTOCOL: CRD420251016912) aimed at updating and expanding upon previous evidence regarding the co-occurrence of OCD and schizophrenia-spectrum disorders. Our review protocol is grounded in dimensional and developmental perspectives and includes transdiagnostic phenomena such as insight, resistance, and temporal onset. By synthesizing data across the putative continuum of obsessive and psychotic symptoms, this effort seeks to offer a more nuanced mapping of the schizo-obsessive interface and to provide a platform for future translational studies.

However, clinical trials specifically designed for this population are also needed. Patients with comorbid SSD and OCD are often excluded from randomized clinical trials, which contributes to a significant evidence gap. As a result, clinicians are left to make therapeutic decisions based on fragmented data or extrapolations from studies that systematically omit the very patients who may present the most complex diagnostic and therapeutic challenges.

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The authors declare no conflicts of interest.

Data availability

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