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Snips from the Journals

Aetiology, classification, and treatment of obsessive compulsive disorder: an update

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Obsessive-compulsive disorder (OCD) is the fourth most common mental disorder with a lifetime prevalence of 2-3%, however, it remains to be underdiagnosed and undertreated (1).

The National Comorbidity Survey Replicated (NCS-R) conducted in the United States showed that 90% of participants with lifetime OCD met diagnostic criteria for another lifetime disorder, the most common comorbidity being anxiety disorders (75.8%), followed by mood disorders (63.3%), impulse control disorders (55.9%), and substance misuse disorders (38.6 %). The commonly comorbid anxiety disorders include panic disorder (20%), agoraphobia without panic (7.8%), specific phobia (42.7%), social phobia (43.5)%, generalized anxiety disorder (8.3%), post-traumatic stress disorder (19.1%) and separation anxiety disorder (37.1%) (1).

Internal Classification of Diseases 11th revision (ICD-11) describes the phenomenology of obsessions and compulsions and reflects the current knowledge of the cardinal symptoms of the disorder better than ICD-10 (2,3).

The ICD-11 Working Group on obsessive-compulsive or related disorders (OCRD) conducted thorough reviews and looked into the evidence base for the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM 5), before recommending changes to the existing ICD-10 guidelines for obsessive-compulsive disorder (4,5).

Changes that have been made in the ICD -11 include: the removal of the obsessive-compulsive disorder from the neurotic, stress-related and somatoform disorders chapter and moving it under a new chapter of "obsessive-compulsive or related disorders (OCRD)", with body dysmorphic disorder, olfactory reference disorder, hypochondriasis, hoarding disorder, and body-focussed repetitive behaviour disorder. All the above diagnostic entities are reported to share the core feature of unwanted thoughts and related repetitive behaviours, as well as similar genetic, physiological, or biochemical abnormalities (6). It is hoped that an OCRD grouping will prompt clinicians to better recognize, assess, and treat these conditions and will stimulate research based on a more accurate conceptualization of these serious disorders and achieving the objective of the World Health Organization in improving global mental health and reducing the associated disease burden (7).

In addition, the ICD-11 has removed the OCD subtypes described in ICD-10, which are predominantly obsessional thoughts or ruminations, predominantly compulsive acts, and mixed obsessional thoughts. It is mentioned that this was done due to the lack of clinical utility, and lack of predictive validity of these subtypes for treatment response (6). Instead, three OCD sub-categories have been introduced in ICD-11 based on the level of insight and these are; obsessive compulsive disorder with fair to good insight, obsessive compulsive disorder with poor to absent insight and obsessive compulsive disorder, unspecified (3).

Repeated factor analysis have consistently supported a four or five-factor model for obsessive-compulsive symptomatology that includes contamination, harmful thoughts, forbidden thoughts, symmetry, and hoarding dimensions (8,9). The working group recommended that the definition of obsessions in OCD be broadened to capture the range of experiences described in the literature, to better represent the phenomenology of compulsions in OCD and describe a functional relationship between obsessions and compulsions in ICD-11.

Further, in ICD-11 the hierarchical rule of giving diagnostic primacy to depression over OCD has been removed in order to improve the detection of OCD better than when ICD-10 is used (3).

New insights into the influence of "maternal effects" on the risk of OCD in the offspring (10).

OCD is a polygenic disorder with many identified risk loci of small effect size, with additive genetic effects accounting for 40% of the variance, while some subtypes having a higher genetic component and earlier onset of OCD with tics (11). In a pioneering study, Mahjani et al., have demonstrated that the combination of, maternal genotype and, maternal environment, described as the

"maternal effects" increases the risk of developing OCD in the offspring. (10).

Mahjani et al., studied a cohort of 822,843 individuals of which 7184 (0.87%) were diagnosed with OCD. The relative recurrence risk (RRR) of OCD was found to be consistent with the maternal effects in OCD risk architecture. The RRR for paternal half-siblings was 1.084, while the RRR for maternal half-sibling was 1.849. The generalized linear mixed model (GLMM) showed that females to be at 1.26 times higher risk relative to male individuals (95% CrI =1.21-1.31) in developing OCD, and offspring of older mothers to be 1.14 times higher risk for OCD (95% CrI =1.05-1.23) than offspring of younger mothers (10).

Early detection is both challenging and critical in patients with OCD (12).

Fineberg et al., highlight the importance of early diagnosis of OCD given its generally early onset, the associated academic, social and occupational impairment and secondary depression that leads to the high disease burden (12).

They report two other features seen among patients with OCD, i.e., ego-dystonia and poor insight as reasons that patients with this disorder do not seek help, making the early detection challenging and critical. Therefore, Fineberg et al., recommend, adoption and validation of a model for OCD, that focuses on addressing environmental and biological aetiological targets as well as primary, secondary and tertiary prevention, in order to change the course of illness in a positive manner.

However, they also highlight the unavailability of outcome predictors and therefore the importance of keeping in mind the potential harms of early intervention in the prodromal stages.

Exposure and response prevention (ERP) is superior to other treatments, in reducing OCD symptomatology (13).

Ferrando and Selai conducted a systematic review and a metanalysis exclusively on randomized controlled trials that included patients above the age of 18 years with a primary diagnosis of OCD as per the International Classification of Disorders or the Diagnostic and Statistical Manual of Mental Disorders(13). They assessed the difference in intervention effect by comparing within group changes in pre-treatment and post-treatment scores. They report that there was a greater reduction in the pre-treatment to post-treatment OCD symptoms, as measured via the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS), in patients who received ERP than in any of the 'other' groups.

Song et al., in their systematic review and meta-analysis looked into the effectiveness of ERP in treating OCD and factors that may influence the outcome report that ERP had a definite effect on OCD (g=0.37), and this effect was significant when the control condition was placebo (g=0.97) or drugs (g=0.59) (14). They also report that both the therapist-controlled and self-controlled exposure and total response prevention can improve OCD symptoms. In addition, compared with the control group, ERP reduced depression (g= 0.15) and anxiety symptoms (g = 0.23) in patients with OCD. Meta-regression results showed that the longer the length of sessions, the better the treatment effect (t=2.41, p=0.022) and that the number of treatment hours (number of sessions per week, the total number of treatment sessions and the length of the treatment) did not moderate treatment outcome.

About 40-60% of patients with OCD respond poorly to serotonin reuptake inhibitors (SRIs) (15).

Van Roessel et al., in a recent review mention that patients with a younger age of onset of the illness, longer duration of untreated illness, severe symptoms as well as those with symptoms related to symmetry/ordering or hoarding have poorer response to SRIs than patients who do not have the above features(15). They also mention that patients with neurocognitive deficits may also show a poor response to the above agents. However, they emphasize that when pharmacological treatments are considered, the SRIs remain first-line option for all patients including the above groups. In their review, they discuss emerging evidence looking at various pathophysiological pathways which involve monoamines and glutamate, as well as anti-inflammatory agents in treating patients with refractory OCD symptoms. They mention that, out of these alternative interventions, dopaminergic agents have the best evidence to date, but highlight that these agents have a complex long-term safety profile. The evidence base for glutamatergic agents such as ketamine, lamotrigine, pregabalin, and topiramate seems to be less robust than for the SRIs and the dopamine agents, although their safety profiles may be better. They also mention that the use of anti-inflammatory agents such as celecoxib, naproxen and octagam 5% (intravenous immuno-globulins) and immunomodulatory agents in OCD but report that the evidence base for these are currently very limited.

Van Roessel et al., highlight the importance of clear communication between patients and clinicians, and that it should include the rationale for pharmacological agents, their risks, benefits, alternative treatments, and highlight

that the prescriber should be familiar and be comfortable with the interventions offered.

Statement of contribution

MH and AW contributed equally to the selection of articles and writing the manuscript. Both authors have approved the final version.

Conflicts of interest

None declared.

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