A CRITICAL REVIEW ON OBSESSIVE-COMPULSIVE DISORDER

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ABSTRACT
Obsessive-compulsive disorder is a frequent, chronic, costly, and disabling mental disorder that presents in several medical settings, but is under-recognized and undertreated. For many years, obsessive-compulsive neurosis was perceived as a disorder that provided an important window on the mechanisms of the unconscious mind. Nowadays, obsessive-compulsive disorder is viewed as a good example of a neuropsychiatric disorder, mediated by pathology in specific neuronal circuits, and responsive to specific pharmacotherapeutic and psychotherapeutic interventions. In the present article we have discussed various causes, pathophysiology and management of Obsessive-compulsive disorder.
1. INTRODUCTION:

Obsessive-compulsive disorder (OCD) is a enervating, severe mental disorder where a person constantly feels the need to check things, perform certain rituals or have certain thoughts repeatedly. Due to this, a person’s daily life is negatively affected.[1] It usually occurs in late adolescence or early adulthood and if left under-recognized and untreated, has a chronic progression. It affects about 2.3% of individuals at some point in their life.[2] The cause is not known but there seems to be some genetic components with both identical twins affected more often than both non-identical twins. The other reason may be following infection. Risk factors include an antiquity of child abuse or other stress inducing event.[1] Rating scales such as the Yale–Brown obsessive compulsive scale can be used to evaluate the severity.[3] Other disorders which have similar symptoms include anxiety disorder, major depressive disorder, eating disorders, tic disorders, and obsessive–compulsive personality disorder.[2] Nutritional deficiencies may also contribute to OCD and other mental disorders.[4]

1. Causes

1.1. Genetics

There appear to be some genetic components with identical twins who are affected more often than non-identical twins.[2] In cases where OCD develops during childhood, there is a much stronger familial connection in the disorder than cases in which OCD develops later in adulthood. In general, genetic factors account for 45–65% of the variability in OCD symptoms in children diagnosed with the disorder.[5] Latest evidence supports the likelihood of a heritable susceptibility for neurological development favoring OCD.[6]

A mutation has been found in the human serotonin transporter gene, hSERT, in unrelated families with OCD.[7]

1.2. Infections

Rapid onset of OCD in children and adolescents may be caused by a syndrome connected to Group A streptococcal infections which causes inflammation to the basal ganglia. Such cases are grouped within a set of clinical conditions called pediatric autoimmune neuropsychiatric disorders associated with streptococcal infection (PANDAS).[8]

2. Pathophysiology of OCD

Brain scans of individuals with OCD have shown that they have different patterns of brain activity than people without OCD and that different functioning of circuitry within a certain part of the brain may cause the disorder. Neurotransmitter dysregulation, mainly serotonin and dopamine, may also contribute to OCD.[9] Independent studies have
consistently found unusual dopamine and serotonin activity in various areas of the brain in individuals with OCD. These can be defined as dopaminergic hyper function in the prefrontal cortex (mesocortical dopamine pathway) and serotonergic hypo function in the basal ganglia.\textsuperscript{[10-12]} Glutamate dysregulation has also been the focus of recent research.\textsuperscript{[13,14]} Although its role in the disorder's etiology is yet unclear.

Individuals with OCD exhibit increased grey matter volumes in bilateral lenticular nuclei, extending to the caudate nuclei, with decreased grey matter volumes in bilateral dorsal medial frontal/anterior cingulate gyri.\textsuperscript{[15,16]} These findings contrast with those in people with other anxiety disorders, who exhibit decreased (rather than increased) grey matter volumes in bilateral lenticular/caudate nuclei, while also decreased grey matter volumes in bilateral dorsal medial frontal/anterior cingulate gyri.\textsuperscript{[16]} Orbitofrontal cortex over-activity is mitigated in individuals who have successfully responded to SSRI medication, a result believed to be caused by elevated stimulation of serotonin receptors 5-HT2A and 5-HT2C.\textsuperscript{[17]}

3. Management

The first-line treatments for OCD are Behavioral therapy (BT), cognitive behavioral therapy (CBT) and medications. Psychodynamic psychotherapy may help in managing some facets of the disorder.

3.1. Therapy

The specific method used in BT/CBT is called exposure and ritual prevention. It involves teaching the person to intentionally come into contact with the situations that trigger the obsessive thoughts and fears ("exposure"), without carrying out the usual compulsive acts accompanied with the obsession ("response prevention"), thus gradually learning to tolerate the discomfort and anxiety associated with not performing the ritualistic behavior. At first, for example, someone might touch something only very mildly "contaminated" such as a tissue that has been touched by another tissue that has been touched by the end of a toothpick that has touched a book that came from a "contaminated" location, such as a school (exposure). The "ritual prevention" is not washing it. Another example might be leaving the house and checking the lock only once (exposure) without going back and checking again (ritual prevention). The person fairly quickly habituates to the anxiety-producing situation and learns that their anxiety level drops considerably; they can then progress to touching something more "contaminated" or not checking the lock at all-again, without performing the ritual behavior of washing or checking.\textsuperscript{[18]}

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Exposure ritual/response prevention (ERP) has a strong evidence base, and it is reflected the most effective treatment for OCD.\textsuperscript{[18]} However, this claim has been suspected by some researchers who complain the quality of many studies.\textsuperscript{[19]}

It has usually been accepted that psychotherapy in combination with psychiatric medication, is more effective than either option alone. Though, more recent studies have shown no difference in consequences for those treated with the combination of medicine and CBT versus CBT alone.\textsuperscript{[20]}

### 3.2. Medication

Most frequently used medications are the selective serotonin reuptake inhibitors (SSRIs). Clomipramine, a medication belonging to the class of tricyclic antidepressants seems to work as effectively as SSRIs but has a greater rate of side effects.\textsuperscript{[21]}

SSRIs are a second line therapy of adult obsessive compulsive disorder (OCD) with mild functional impairment and as first line therapy for those with moderate or severe impairment. In children, SSRIs can be considered as a second line treatment in those with moderate-to-severe impairment, with close observations for psychiatric adverse effects. SSRIs are efficacious in the treatment of OCD; people treated with SSRIs are about twofold as likely to respond to treatment as those treated with placebo.\textsuperscript{[22]} Efficacy has been demonstrated both in short-term (6–24 weeks) treatment trials and in discontinuation trials with durations of 28–52 weeks.\textsuperscript{[23]}

In 2006, the National Institute of Clinical and Health Excellence (NICE) guidelines recommended anti-psychotics for OCD that does not improve with SSRI treatment.\textsuperscript{[24]} For OCD the evidence for the atypical antipsychotic drugs riserpidone and quetiapine is tentative with insufficient evidence for olanzapine.\textsuperscript{[25]} A 2014 review article described two studies that indicated that aripiprazole was "effective in the short-term" and found that "there was a small effect-size for risperidone or anti-psychotics in general". However, the study authors found "no evidence for the effectiveness of quetiapine or olanzapine in comparison to placebo.\textsuperscript{[24]} Although quetiapine may be useful when used in addition to an SSRI in treatment-resistant OCD, these drugs are often poorly tolerated, and have metabolic side effects. None of the atypical antipsychotics seem to be useful when used alone.\textsuperscript{[26]}

### 4. RESEARCH

The naturally occurring sugar inositol has been proposed for management for OCD.\textsuperscript{[27]} μ-Opioids viz., hydrocodone and tramadol may improve OCD symptoms.\textsuperscript{[28]} Administration of opiate treatment may be contraindicated in individuals simultaneously taking CYP2D6 inhibitors such as fluoxetine and paroxetine.
Current research is devoted to the therapeutic potential of the agents that affect the release of the neurotransmitter glutamate or binding to its receptor. These include riluzole, memantine, gabapentin, N-acetylcysteine, topiramate and lamotrigine.\textsuperscript{[14]}

5. CONCLUSION

OCD is a severe, debilitating and chronic mental disorder. If OCD is not effectively treated, most patients have clinically significant disability, with symptoms that wax and wane over time. Even though many advances have already been made in treatment of obsessive-compulsive disorder, in the future a better understanding of the pathogenesis of OCD will hopefully lead to further extension of the present range of treatments, including advances in psychopharmacology, psychotherapy, and other modalities of intervention.

6. REFERENCES:


