AU InforMed

Volume 22 Number 8 (Issue 342) Monday, December, 9 2024

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Key Inforbits

- What is OCD?
- Common Comorbidities of OCD
- Managing OCD
- Emerging Studies

What is OCD?

History: Through most of recorded history, obsessive compulsive disorder (OCD) was described with religious connotations, and was not described in a non-religious manner until the 19th century, when French psychiatrists first began to distinguish it as a "monomania" or *folie du doute* (madness of doubt).[1] As the French psychiatrists focused on the condition as an emotional disorder, German psychiatrists began to focus study of OCD as a disorder of thinking, with neurologist Wilhelm Griesinger (1817-1868) describing 3 cases of *Grubelnsucht*, or ruminatory/questioning illness. The origin of the term "obsessive-compulsive disorder" comes from translational differences. German psychiatrist Westphal used the term *Zwangsvorstellung* (compelled presentation) to describe obsessions, and this was translated as "obsession" in the UK and "compulsion" in the US, with OCD being the compromise. After the condition was named, the leading theories became those of unconscious… id impulses and the demands of conscience and reality."[2]

Fig 1- Wilhelm Griesinger. From wikipedia.com

Pathology: The exact mechanism that causes OCD has not been determined, though disordered serotonergic and dopaminergic function, especially in the cortico-striato-thalamo-cortical (CSTC) circuit.[3] One of the main sources of evidence for the role that serotonin, especially the 5-HT_{2A} receptor, plays is the effectiveness of selective serotonin reuptake inhibitors (SSRI) in reducing symptoms. Dopamine dysregulation is implicated in OCD due to the presence of neurological symptoms such as tics appearing in some patients. Additionally, dopamine agonists such as pramipexole (Mirapex) often worsen OCD symptoms, and augmentation with antipsychotics has been shown to be effective in patients who achieve partial response to an SSRI, indicating that both systems are disordered. The CSTC circuit is implicated due to its role in regulating self-control, altered activity during PET (positron emission tomography), SPECT (single-photon emission computerized tomography), and other neuroimaging studies, and the effectiveness of neuromodulation therapy.[4]

Epidemiology: About 1-3% of people are estimated to have OCD, with this figure being fairly consistent across different cultures. Recent studies have also examined the prevalence of subclinical OCD, though these figures vary significantly from 2-19%. Due to the difficulties patients with these symptoms experience in work, school, activities of daily living (ADLs), and social interactions, the estimated cost of OCD is over $8 billion per year. Many studies have examined the difference in rates of OCD between male and female patients, but these results frequently conflict, with some finding increased incidence in one sex, and others finding equal distribution.[5]

Fig 2. Symptoms of OCD. From NeurodivergentInsights.com

Symptoms: Separated into two categories: Obsessions and Compulsions. They can exist together or independently.

Diagnosis: The DSM-5 criteria for a positive diagnosis of OCD are:[7]

1. Presence of obsessions, compulsions, or both
    a. Obsessions: Persistent or recurrent thoughts that the patient considers intrusive, unwanted, and causing anxiety or stress, which the patient attempts to ignore, suppress, or neutralize with some other thought or action.
    b. Compulsions: Repetitive behaviors or mental acts that the patient feels like they must do as response to an obsession or according to rules that must be applied, that are aimed at preventing or reducing anxiety, stress, or a dreaded situation, that are not realistically connected to the situation, or are excessive.

2. The obsessions or compulsions are time-consuming (e.g., take more than 1 hour per day) or cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
3. The obsessive-compulsive symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.
4. The disturbance is not better explained by the symptoms of another mental disorder…

The DSM-5 states that the following specifications should be made:
- Level of insight: from good-fair insight (understands that beliefs are probably not or may not be true) to poor insight (thinks OCD beliefs are probably true) to absent insight/delusional beliefs (completely convinced that OCD beliefs are true).
- Tic-related: does the individual have a current or past history of a tic disorder?

Common Comorbidities of OCD

OCD is often associated with intense anxiety related to the obsessions and/or compulsions of the disorder itself, however, it can also be a disorder that manifests outside of OCD symptoms. Treatment for anxiety does not typically differ from what is used to treat OCD. Social and generalized anxiety are the most common of the anxiety disorders associated with OCD. Depending on the obsessions and compulsions, a person may feel anxiety for a multitude of reasons that may hinder their ability to socialize, maintain a career, or even care for themselves appropriately. Along with anxiety and OCD, major depressive disorder can occur in some patients due to the extreme levels of hopelessness and helplessness caused by the OCD symptoms.[8] Another common comorbidity with OCD are eating disorders that could stem from a person's hand hygiene, food contamination, body dysmorphia, just to name a few.[9]

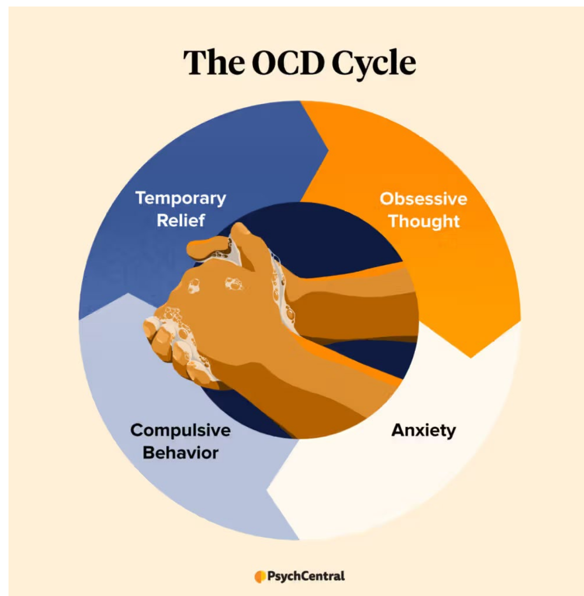


Fig 3- the OCD Cycle. From: PsychCentral.com

Managing OCD

Pharmacologic Treatment:

- Selective Serotonin Reuptake Inhibitors (SSRIs): first-line treatment option
  - The serotonin hypothesis demonstrates that certain receptor types can exacerbate OCD thus inhibitors of serotonin may help to diminish symptoms.[10,11]
  - Common side effects include:
    - sexual dysfunction
    - nausea
    - vomiting
    - dry mouth
    - dizziness

- ➔ Tricyclic antidepressants, Anafranil (clomipramine): first-line treatment option.[10,11]
    - ◆ Thought to work similarly to SSRIs and may work in patients who did not achieve a response to SSRIs
    - ◆ Common side effects include:
        - weight gain
        - dry mouth
        - dry eyes
        - increased heart rate
        - urinary retention
- ➔ Second-line treatment: augmentation with second-generation antipsychotics.[10,11]
    - ◆ Risperidone
- ➔ Duration:
    - ◆ 10 - 12 weeks at maximally tolerated dose

Non-pharmacologic Treatment:

- ➔ Cognitive Behavioral Therapy (CBT):
    - ◆ Exposure and Response Prevention (ERP):
        - As depicted below, ERP is a form of CBT that is used to systematically introduce the person to increasing levels of stress related to their OCD symptoms to help decrease the level of anxiety when subsequent encounters occur. The example can be linked to a "fear of contamination" or "need for cleanliness" by beginning with a singular dirty plate and gradually increasing the articles to build a person's tolerance to their distress.

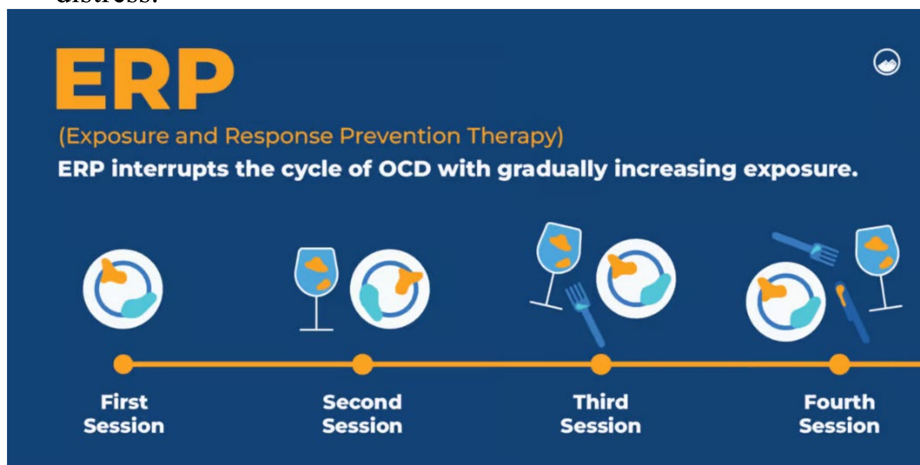


Fig 4-Exposure and response prevention therapy. From sandstonecare.com

        - This type of therapy is also geared towards learning how to not engage with compulsions once an obsession takes hold.
        - Stanford Medicine reports ~50% of patients would benefit from ERP alone, 20-30% are resistant to ERP and 20% drop out before any benefit is seen. They also report 25% do not show lasting benefit at follow-up visits.[10]
    - ◆ Acceptance and Commitment Therapy (ACT):
        - A form of CBT focused on helping individuals accept their obsessions and emotions associated with the discomfort rather than trying to eliminate or control the obsessions.[11]

Emerging Studies

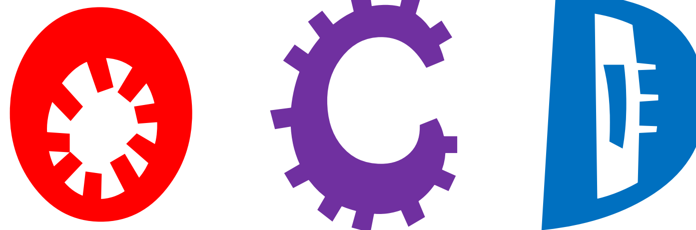
Fig 5-Research Graphic. From Freepik.com

Current OCD guidelines have not been updated since 2007 which leave significant room for improvements and advancements. One such avenue is the effect of glutamate and its role in OCD. The International OCD Foundation discusses glutamate in relation to OCD because of its excitatory effect on the brain which may contribute to OCD symptoms. Through MRI imaging and CSF testing investigators across the world have found that patients with OCD have a higher concentration of glutamate exposure to the brain.[13] Though this does not represent causation, it does open the door to further research!

Treatment possibilities with the glutamate hypothesis include: Namenda, N-acetylcysteine, Riluzole, and even ketamine. The International OCD Foundation and Dr. Carolyn Rodriguez from NIH Record discusses ketamine in the use of MDD as well as OCD. Dr. Rodriguez has researched ketamine over the years to determine its effects on OCD symptoms and has found a positive correlation between the two regarding symptom reduction.[13]

Summary

It is essential to raise awareness for OCD because of its hidden nature that can often lead to those affected feeling hopeless and alone. Through raising awareness those unaffected by OCD will have a better understanding of the condition that allows an opportunity to offer support and encouragement to those in need.



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<u>The last "dose"</u> …

“We suffer more often in imagination than in reality.”

- Seneca the Younger, Stoic philosopher 4 BCE - 65 CE

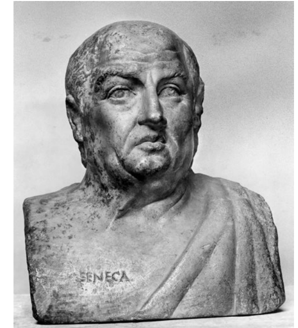

Fig 6- bust of Seneca the Younger. From britannica.com

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