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Cognitive Behavioral Therapy for Obsessive Compulsive Disorder

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1. Introduction

Obsessive Compulsive Disorder (OCD) is a neuropsychiatric condition characterized by obsessions (defined as recurrent and persistent thoughts, impulses or images that are intrusive and inappropriate and cause marked distress) and compulsions (repetitive behaviors or mental acts driven by obsessions to reduce distress or prevent dreaded events [1]. OCD is the fourth most prevalent psychiatric disorder and was considered as one of the most debilitating disorders with a prevalence rate of 2.5 % in an individuals' lifetime [2, 3]. OCD is widespread among both adults and children [4]. OCD is considered a relatively common occurrence in childhood and adolescence where prevalence rates are between 1 to 3.6% [5-7]. As it is the case with other mood and anxiety disorders, females tend to be at a higher risk of developing OCD compared to males [8]. About one third to one half of adult OCD cases has its origins in childhood or adolescence [9, 10]. However, a recent meta-analysis found that pregnant women and post partum women have higher prevalence rates of OCD compared to the general population, with postpartum women at a much greater risk compared to the general female population [11]. Research has found that early onset is associated with males while late onset is associated with females [10].

It is worthwhile to mention that the new DSM- 5 released in May, 2013 has a number of changes to OCD and related disorders such as hoarding disorder and Body Dysmorphic Disorder (BDD). OCD is no longer an anxiety disorder, rather is has its own chapter in the DSM-5. The chapter includes other disorders, in addition to obsessive-compulsive disorder, such as Body Dysmorphic Disorder and trichotillomania (hair-pulling disorder), as well as two new disorders: hoarding disorder and excoriation (skin-picking) disorder [12].

The presence of co-morbidities is common in people with OCD. For example, some of the most common disorders associated with OCD are depression [13-15], anxiety [16-19], alcohol or substance misuse [20-22], BDD [23], or eating disorders [24]. People with OCD have a tendency for impulsive behavior (may include symptoms of childhood conduct disorder). They also show higher rates of suicidal attempts [25]. OCD is often considered a chronic condition and it significantly impairs various spheres of a person's life including social, emotional and somatic [25].

1.1. Etiology

The etiology of OCD is multifaceted. OCD is etiologically and phenomenologically heterogeneous [26]. For individuals with OCD, it has been indicated that there are abnormalities in the neurotransmitter; serotonin, which plays an important role in carrying nerve impulses from neurons to the receptor cells *via* the brain. The normal functioning of serotonin is impaired in patients with OCD as a result of blocked or impaired receptor sites in the brain [27]. Neurological aberrations could be associated with genetic mutations, while environmental factors can play a role in the expression of OCD symptoms [27]. The use of Clomipramine or Selective Serotonin Reuptake Inhibitors (SSRIs) has shown promising results in reducing symptoms of individuals with OCD [4, 28]. This indicates the involvement of the brain in the etiology of OCD [27, 28].

Although the same diagnostic criteria are used to diagnose OCD, its expression varies from person to person (i.e. onset, severity, obsessions and compulsions) [29]. Symptom dimensions are heritable in OCD [30]. It is imperative to pinpoint that the onset of OCD is linked to the interaction between both genetic and environmental correlates. This does not necessarily indicate that negative life events cause OCD, rather a number of studies have found that for people with biological or psychological vulnerability, negative events may trigger the onset of OCD [31, 32].

The identification of susceptible genes involved in the onset of OCD is challenging given the heterogeneity of the disorder. The use of symptom-based rather than disorder-based construct has reduced the heterogeneity of OCD [3]. vanGrootheest (2008) conducted a twin-study to explore the OC symptom dimension heritability [33]. The vanGrootheest study is pioneering in the field because it utilized population-based rather than OCD - sample of twin pairs. It identified rumination, contamination and checking; three OC symptom dimensions which shared discrepancy with OC behavior that was explained by both genetic (36%) and environmental factors (64%). However, certain genes influenced the contamination dimension and therefore, that dimension was independent. The use of family pedigrees is yet another approach to determine the heritability of the OCD trait [34]. In pedigree analysis, a high genetic loading was associated with symmetry and ordering symptoms. More interestingly, for symmetry, hoarding and contamination symptoms, correlations among independent sibling pairs were found [35, 36]. According to the OCD Collaborative Genetics Study, sibling-sibling association was associated with four factors; hoarding and taboo thoughts being the most prevalent [37, 38]. In families with a multigenerational history of OCD and hoarding, OC symptoms were found to have a higher rate of heritability compared to hoarding [39].

1.2. Symptoms and its Sub-types

Obsessive-Compulsive symptomatology is generally measured using the Yale-Brown Obsessive Compulsive Scale (Y-BOCS), which includes an assessment of the severity of the disorder and a checklist (YBOCS-CL) of the OC symptoms [40, 41]. This scale consists of 45 obsessions and 29 compulsions within 15 predefined symptom categories [40, 41]. A recent meta-analysis identified five OC symptom dimensions or factors: (i) symmetry and repeating; (ii) aggressive, sexual and religious obsessions; (iii) contamination and cleaning, (iv) aggressive obsessions and checking; and (v) somatic obsessions [42].

1.3. Diagnosis

According to the *DSM-IV* criteria, the symptoms of OCD should not be due to a general medical disease, chemical substance, or drug overdose [1]. OC symptoms are associated with several neurological lesions of the cortico-striatal-thalamic-cortical circuits resulting from the administration of drugs such as methylphenidate or cocaine (dopamine agonists) or after streptococcal infection [4]. Clinically speaking, OCD symptoms are associated with marked anguish and dysfunction. Subclinical OC symptoms are common and are noted during the path of normal development [4]. However, OCD can negatively affect a person's quality of life [43-45]. In some cases, diagnosis becomes unclear, when the symptoms are perplexed with those of Obsessive-Compulsive Personality Disorder [42]. Although, OCD symptoms vary from symptoms observed in other anxiety and mood disorders, they are an intrinsic component of other disorders such as autism and frontal lobe lesions [46]. Inappropriate diagnosis, under diagnosis and incompetent treatment are linked to tremendous direct and indirect costs [4].

2. Brief historical overview

Since the 4th century BC, OCD has been elucidated as "melancholia", a unique disease with certain mental and physical symptoms. These symptoms are attributed to the imbalance in the bodily fluids; blood, black bile, yellow bile, and phlegm [47]. Hippocrates defined all fears and depressions if lasting for a long time as symptoms of melancholia [47].

In the early Middle Ages in Europe, blasphemous, sexual or obsessive perceptions were attributed to satanic possession, which was believed to lead to compulsive behaviors [48]. Priests treated patients by expelling spirits from their bodies. However, in the 17th century, the concept of "religious melancholy" came to be known as the cause of several mental disorders including those with OCD-like symptoms [49].

Sigmund Freud, an Austrian psychiatrist, suggested that OC behavior is associated with unconscious conflicts [47, 50]. Freud's analytical conceptualization of "obsessive neurosis" was based on the idea that people develop OCD as a result of the difficulties in the anal psychosexual stage of development. Freud utilized psychoanalysis to treat OCD. There is no current evidence - based research that supports the use of traditional psychodynamic psychotherapy in the treatment of OCD [51]. This, in turn, gave rise to the behavioral mode of psychotherapy.

Several cognitive behavioral theories have been developed to understand and target symptoms' progression and maintenance in people with OCD. The following sections will discuss the various cognitive behavioral theories.

2.1. Mowrer's two stage theory

Also known as the two-factor theory, Hobart Mowrer proposed a strictly behavioral drive-based theory in 1947 to explain avoidance in entirely behavioral terms [52]. Mowrer's theory merged the learning principles of classical and operant conditioning and explains fear acquisition and the vicious cycle of rituals. Based on classical conditioning, phobias are the result of an association between a neutral stimulus (conditioned stimulus; CS) and feared stimulus (unconditioned stimulus; UCS) [53]. Mowrer's theory supports the concept that a CS provokes fear when it is recurrently presented with an UCS. The neutral stimulus is any mental event and/or physical object while the avoidance of the stimulus reduces anxiety [53]. People with OCD react to the neutral stimulus with the same fear level associated with the unpleasant event. Furthermore, it is through the generalization process that fear and avoidance can extend to other places or situations, which remind the individual of the trauma, triggering the same fear response. The persistence of behavioral avoidance leads to further distress. The theory also explains how rituals are maintained. Dollard and Miller (1950) further adopted Mowrer's theory to explain the progress and maintenance of fear/anxiety and avoidance in OCD [54].

In a series of experiments, Rachman and colleagues showed that obsessions increase obsessional distress while compulsions reduce distress levels [55, 56]. It was this concept that defined a functional relationship between obsessions and compulsions, defining OCD in *DSM-III* and its successors [1]. Recently, many studies explored the relationship between OCD and other anxiety disorders, and reconsidered the validity of the diagnostic classification of OCD [26, 57]. The core psychopathology of OCD is grounded on "preoccupation" and "repetitive behaviors", a result of cognitive and motor failure, which overlap with obsessive-compulsive and related disorders (OCRDs), recently included in DSM-5 and ICD-11 [58].

Further, Roper et al. (1973) noted that obsessions and compulsions were maintained to overcome associated anxiety. In addition, results showed that the urge to perform a particular activity disappeared within a 15-180 minute period if the patient did not engage in the activity [56]. The repetition of the exercises reduced the anxiety and urges to carry out these activities; the phenomenon being defined as "habituation" and formed the basis of EX/RP therapy. In 1970s, clinical trials added more support to the efficacy of exposure and ritual prevention (EX/RP) therapy in reducing OC symptoms, thus becoming one of the first choices for the treatment of OCD [59, 60].

2.1.1. EX/RP Therapy — Efficacy and limitations

In more than 70% of the cases, individuals treated with OCD responded to EX/RP therapy more efficiently compared to individuals who were on SSRIs [61]. However, the model has some limitations. About 20- 30% of individuals with OCD drop-out or refuse treatment. This is evident in cases with severe OCD or low insight level [62]. In addition, EX/RP had little or

no benefit in helping people with pure obsessions. Furthermore, the model did not explain why OCD sufferers become distressed by aggressive or sexual thoughts, without having witnessed or committed such acts [63].

Cognitive theory and therapy was developed as a result of the limitations of the behavioral and ERP theories and therapies. Cognitive techniques focus on distorted thoughts and dysfunctional beliefs along with highlighting the role of cognitions in the origin and maintenance of symptoms [64]. The addition of cognitive techniques to ERP has been effective in the treatment of patients who presented with pure obsessions or obsessive ruminations [60]. Also, it led to the development of the “cognitive behavioral theories”, which is now considered as the gold standard treatment for OCD [65].

2.2. Cognitive theories

Several cognitive theories emerged to understand the development and maintenance of OCD symptoms. According to Rachman and de Silva (1978), obsessions of a disturbing nature are a normal occurrence in people with and without OCD [66]. However, individuals with no OCD do not give importance to such thoughts and they, as a result, disappear easily, although they can play a critical role in transforming normal unpleasant thoughts into obsessions [66]. This section will focus primarily on two theories: Foa and Kozak’s, and Salkovskis’ theory.

2.2.1. Foa and Kozak’s theory

According to Foa and Kozak, OCD is distinguished by two erroneous cognitions: (i) individuals with OCD have a cognitive vulnerability for the overestimation of the probability of danger in safe situations; (ii) they exaggerate the severity of the consequences of negative events [67].

2.2.2. Salkovskis theory

Salkovskis proposed an improved cognitive model in which responsibility was considered a critical issue for the onset of obsessions [68, 69]. He proposed five basic assumptions that are characteristic of individuals with OCD: (i) Thoughts and actions are the same; (ii) causing harm is the same as not preventing harm; (iii) despite difficult events, personal liability for harm continues; (iv) not engaging in harm related rituals is the same as having the intention to harm; (v) controlling one’s thoughts is a personal obligation. Salkovskis’s theory proposes that patients’ catastrophic appraisals of obsessions lead to an increased sense of responsibility [70]. Such responsibility beliefs tend to cause increased anxiety and end with compulsions to exert control over obsessions [70]. Obsessions tend to persist as long as the distorted and blasphemous beliefs persist, and decline when such thoughts are enfeebled.

Rachman (2002) proposed a counterpart to this theory to explain the origins of compulsions [71]. According to Rachman (2002), scrutiny of rituals occurs as inflated thoughts of responsibility lead to compulsions aimed at reducing any possible damage. Scrutiny, as a result, leads to continuous scrutiny to eliminate doubt and possible risk [71].

However, several theorists suggested that 6 other belief domains could be associated with the origin and maintenance of OCD symptoms, including (i) inflated responsibility [68]; (ii) over-importance of thoughts [72]; (iii) extreme concern about the importance of controlling one's thought [73, 74]; (iv) overestimation of risk [75]; (v) intolerance of uncertainty [75]; and (vi) perfectionism [76].

Freeston et. al (1996) indicated various common features in patients who benefited from cognitive interventions and they include: (i) placing high value over the incidence of obsessions; (ii) believing that obsessions determine the patient's real character; (iii) equating thoughts and actions; and (iv) believing that the presence of a thought increase the possibility of its occurrence. Several cognitive techniques were added to ERP therapy, leading to the adoption of "Cognitive Behavioral Therapy (CBT)" [77].

Cognitive Therapy: Efficacy and Limitations: Cognitive therapy is efficient in the treatment of patients with predominant obsessions, and those with obsessions and compulsions [72]. Cognitive therapy can benefit patients with poor insight or those with dysfunctional beliefs, suggesting the use of cognitive therapy prior to EX/RP therapy to reduce anxiety and improve adherence to EX/RP exercises. Cognitive therapy has not shown to be effective in patients who significantly display obsessions of sexual, aggressive or destructive content. However, the use of cognitive therapy alone has not been established, giving rise to cognitive behavior therapy [73].

3. Cognitive Behavioral Therapy (CBT)

The CBT model for the treatment of OCD is brief and structured, and it aims to help patients cope with OCD symptoms. This is typically done through the utilization of behavioral as well as cognitive strategies. Some of these strategies involve exposure to fearful stimuli, response prevention, challenging dysfunctional erroneous beliefs, psycho-education, and home exercises such as symptom monitoring and exposure exercises [79].

When OCD symptoms are mild and are not associated with any co-morbidity, CBT is usually brief lasting between 13-20 sessions [80]. Initially, CBT is held on a weekly basis, or twice a week until some improvements are observed. As the condition improves, sessions are spaced. Behavioral exposure techniques help to achieve habituation to fear while cognitive techniques target dysfunctional beliefs, especially for patients with a preponderance for obsessions [47]. The following sections will describe the process of CBT, which include patient assessment followed by psycho-education, ERP and cognitive exercises, discharge and relapse prevention.

3.1. Patient assessment

A detailed psychiatric and psychological assessment is solicited from patients to rule out differential diagnosis and any associated disorders [81]. In some cases, specific brain imaging examinations may be included to rule out organic causes. The Yale-Brown Obsessive Compulsive Scale (Y-BOCS) can be utilized to acquire a comprehensive overview of the OCD

symptoms. The effectiveness of CBT is reduced when OCD is accompanied by other co-morbid conditions such as severe anxiety or depression, psychosis and addiction [82].

3.2. Initiation of CBT

Psychoeducation: Once OCD diagnosis has been confirmed, the therapist utilizes psychoeducation mini lessons to educate the patient about the nature of OCD, the CBT model of treatment, vicious maintenance cycles and the role of avoidance and fear in maintaining the disorder. In addition, patients will be educated about helpful and unhelpful coping strategies, escape and avoidance, exposure therapy, the development of fear hierarchy and principles of exposure. Given the crucial role of homework, patients and their families are introduced to the role of homework within the therapy framework [83]. Engaging family members in this stage is crucial, given the challenge family members face in changing their daily routines and responding to questions with reassurances to accommodate the patient [84].

Motivation: Despite the growing body of research supporting the use of EX/RP in the treatment of OCD, it is estimated that about 25% of patients with OCD refuse EX/RP [85]. This refusal may be associated with the increased exposure to anxiety, which is an intrinsic part of the treatment [86]. Lack or low level of motivation is especially problematic for pediatric patients with OCD, and is associated with negative therapeutic outcomes. Therefore, interweaving motivational interviewing techniques within the therapy framework helps patients decrease their ambivalence about treatment. A recent study found that using the motivational interviewing within therapy sessions proved helpful in facilitating speedy improvement and minimizing the burden for families [87].

3.2.1. Elaboration of list of hierarchy

The initial task in CBT is to work with patients to create a list of anxiety provoking situations that targets main OCD symptoms. The Y-BOCS-CL is frequently used by clinicians to create a comprehensive list. Anxiety provoking situations will be ranked by patient according to the level of anxiety it creates starting from 1 (mild anxiety) to 4 (high level of anxiety). To increase the patient's self-efficacy, exposure exercises start with the least anxiety provoking situation [88].

Assessment measures: To monitor the progress of patients throughout the therapeutic process, it is crucial to utilize assessment measures at various points during treatment. One of the widely used sensitive scales is the Yale-Brown Scale, which is a valid and reliable instrument for assessing the severity of obsessive-compulsive disorder [40, 41]. The Yale-Brown Scale is composed of 10 questions; 5 to assess obsessions and 5 to assess compulsions. The scale assesses severity from (0- no symptom, to 4 –severe symptoms). A maximum of 40 points can be obtained for patients with severe symptoms [40, 41]. Another sensitive tool is the revised version of the Obsessive Compulsive Inventory. Its usefulness has been proven as a diagnostic tool for screening patients with OCD [89].

3.2.2. *Exposure and response prevention exercises (EX/RP)*

In Vivo exposure (involves the direct confrontation with the fearful stimulus) is the most common form of exposure. Utilizing the already developed hierarchy list, the patient along with the therapist initiate therapy by choosing a situation that evokes a medium level of anxiety. Therapy sessions are focused on generalizing and extending gains to other situations through designing homework exercises. It is crucial to discuss with the patient that while confronting anxiety may increase anxiety, over time, anxiety will dissipate. Response prevention increases anxiety level, but it gradually declines. That is why response prevention reduces the need to engage in compulsions [61]. The main effect of EX/RP exercises is the instant increase of anxiety in the session, which reaches its peak during the first exercise and decreases or even disappears during the next 15-180 minutes [90].

3.3. **Cognitive exercises**

The initiation of cognitive exercises occurs when the patient have mastered EX/RP exercises and is content with identifying OC symptoms. The patient should be able to make a distinction between obsessions and normal thoughts. In these exercises, various parameters including unpleasant thoughts, their background and emotive, physiological and behavioral concerns are documented. Patients are taught the classifications of these beliefs based on their domains. Cognitive techniques include the recognition and documentation of unpleasant thoughts; Socratic questioning; downward arrow; and pie of responsibility [69].

3.4. **Ending CBT and relapse prevention**

Once OCD symptoms have significantly improved, spacing sessions are proposed and CBT is ceased. Given that OCD is a chronic disorder with high relapse rates [91], both therapists and patients work to prevent relapse through creating relapse prevention plans. Booster sessions help in monitoring the maintenance of therapeutic gains [92].

4. **Cognitive behavioral psychotherapy adaptation for children and adolescents with obsessive compulsive disorder**

Childhood OCD is linked with severe commotion in social and academic functioning, comorbid emotional and behavioral complications and family difficulties [93]. Cognitive-Behavioral Therapy (CBT), especially exposure and response prevention (ERP) is the pivotal therapy used for OCD in adults [94]. Exposure encompasses useful confrontation of objects or situations that trigger obsessions; response prevention includes refraining from activities that reduce anxiety generated by obsessions [93]. Initially, ERP was considered more helpful for adults compared to children and adolescents.

However, since the mid-1990s, research proved ERP to be viable, safe and effective for both children and adults [95]. However, not many children and adolescents receive CBT for various factors, one being lack of trained clinicians. Interestingly, clinicians find the research-driven-

treatment methodology not practical [93]. Furthermore, even though OCD in children is more prevalent, variations in dimensions between adults and children (age, maturity, language development..etc) obfuscate the application of CBT for children. Children are not able to recognize or label their obsessions or fear responses. In certain cases, a child's responses tend to mislead adults into believing that the child's behavior is willful [96]. One of the challenges facing the child therapist is children's lack of understanding of OCD symptoms. High anxiety levels during ERP makes children resistant to engage in therapy. EX/RP homework exercises are challenging to children given the aversion towards homework. This in turn makes parental supervision crucial for a successful treatment [93]. Criticism and conflicts within the family negatively affect therapy [97]. Dropout rates are associated with the rigidity in applying treatment protocols, suggesting the need to assess developmental issues, and consequently adopting treatment plans tailored to meet each child's needs [93].

Recently, CBT protocols have been developed for children. These protocols typically include psycho-education in age-appropriate language, and cognitive strategies to cope with anxiety, and the use of reinforcement contingent strategies within the family environment [98]. Parents' active involvement in the child's treatment increases the effectiveness and long-term gains from the treatment [99]. A sensitive protocol for CBT in children was recently developed and is considered a flexible and practical guide for clinicians; RIDE Up and Down the Worry Hill [100, 101].

4.1. RIDE up and down the worry hill

The abbreviation RIDE and the metaphor of riding a bicycle Up and Down the Worry Hill were designed to describe basic CBT principles in a child-friendly manner [100, 102].

The "Worry Hill" explains the relationship between exposure and habituation. It explains the gradual increase in anxiety levels as a result of exposure to the fearful stimuli. Anxiety levels continue to increase as exposure to fear continues. The fear will reach the top peak in the Worry Hill. This is when autonomic habituation starts and anxiety declines. However, if the child surrenders to or escapes fear, habituation is interrupted and obsessions are strengthened [102].

The four-step RIDE acronym (Rename, Insist, Defy, Enjoy) comprises the steps that either the child or adolescent should take to complete the Worry Hill [100, 102]. The RIDE was developed to streamline EX/RP for children and adolescents and raise persistence of anxiety until habituation occurs. RIDE includes cognitive (externalizing, distancing) and behavioral (Controlling obsessions, exposure and self-reinforcement) techniques. Coaching is provided in each of the four steps followed by therapist modeling, behavioral rehearsal, repeated practice and reinforcement, until the individual has grasped each step [102]. The following section will discuss each of the four steps.

4.1.1. R: Rename the thought (That's OCD talking, not me!!)

This is the first step and comprises recognition of OCD thoughts as separate and different from the child's rational self [101, 102]. The method of externalizing OCD has been employed by

Schwartz (1996) [103] with adults and March et al (1994) [98] with children and has been found to be effective.

4.1.2. I: Insist that YOU are in charge (I'm in charge. I choose NOT to believe OCD!)

The second step allows the child to use the power of choice [101, 102]. Insisting that oneself is in charge helps build self-confidence and strength needed to embark on exposure.

4.1.3. D: Defy OCD-Do the OPPOSITE of what it wants (I will RIDE up the worry hill and stick it out until i can coast down)

This step involves ERP, specifically a change in behavior [101, 102]. Response prevention involves exposure to the obsession and associated fear response without engaging in compulsions. This escalates anxiety till it reaches the peak, and once habituation is set, the anxiety starts to decline. With continuous practice, anxiety habituates at a faster rate.

4.1.4. E: ENJOY your success — Reward yourself (I did it! I BEAT OCD! I can do it again)

This is the final step and lets the child review his/her success and take due credit for effort [101, 102].

The Worry Hill represents a universal metaphor as children, adolescents and even adults can relate to the conception of riding a bicycle up a hill and makes it easier to understand the principle behind CBT. The implementation of CBT in both children and adolescents occurs in four sequential phases, which will be discussed in the following section.

4.2. Implementation of CBT in children and adolescents

The four phases are focused on specific goals and aims to develop various skills in every phase. The severity of the disorder determines the number of psychotherapy sessions. In some cases, a total of 6 sessions are required, whereas in others, an average of 10-20 sessions is required.

4.2.1. Phase 1 — Biopsychosocial assessment and treatment plan

This phase lays the foundation for effective treatment and takes from one to three sessions, each session equaling 50-minutes. This assessment is designed to be culturally sensitive and comprehensive to include an appreciation of OCD symptoms in a holistic manner. This involves joint efforts amongst the therapist, parent, child and other concerned personnel. The aim is to develop an individualized child-centered treatment plan. The use of clinical interviews, self-report inventories and behavioral observations are important at this stage [101].

4.2.2. Phase 2 — Building treatment readiness

This phase emphasizes planned and active preparation for treatment. Although a critical phase, it tends to be ignored, leading to frequent treatment failures. This phase requires one to three sessions and includes stabilization of any family crisis and the use of effec-

tive communication skills to increase awareness about treatment and builds therapeutic rapport [101].

4.2.3. Phase 3 — *The RIDE up and down the worry hill*

This phase comprises of 4 to 15 sessions and consists of separate as well as joint sessions with both child and parents. It is during this stage that the child participates in EX/RP [101].

4.2.4. Phase 4 — *After the RIDE*

As the final step of the treatment, it is initiated when the child and parents have mastered the RIDE and the RALLY effectively, respectively as well as when there is a decline in the OCD symptoms [101].

5. Efficacy studies of the cognitive behavioral psychotherapy and pharmacology for OCD

In a systematic review, Foa and Kozak (1996) found that about 83% of patients with OCD included in trials (n=330), showed a minimum of 30% improvement in their symptoms compared to pre-treatment measures. These trials utilized exposure and response prevention techniques [104].

When exploring long-term outcomes of patients with OCD who utilized CBT, a review of 17 studies indicated that about 76% of patients continue to show improvements in their OCD symptoms at an average of about two years post treatment [104]. In addition, CBT was shown its effectiveness not only in the treatment of OCD but also other mental health disorders such as depression and generalized anxiety disorder as evidenced by a review of 16 meta-analyses [105].

On the other hand, Gava et.al (2007) conducted a systematic review of eight randomized trials comparing the therapeutic outcomes of patients receiving a form of cognitive behavioral therapy versus patients who were treated as usual [106]. Treatment as usual included no treatment, waiting list or usual care. Results from 214 patients indicated that cognitive-behavioral therapy is associated with more significant decline in OCD symptoms compared to treatment as usual [106]. Amelioration of 50-70 % of OCD symptoms were noted for patients with OCD involved in the above mentioned trials. Therapeutic outcomes did not differ among the variations of cognitive behavioral therapy. In addition, results showed no difference in the clinical outcomes for patients with OCD who were assigned to individual or group psychotherapy sessions.

Various randomized clinical studies were conducted to control for confounding variables such as the presence of co-morbid disorders, OCD severity, age, just to name a few. However, Franklin et al. (2000) compared the results of 4 randomized controlled trials (EX/RP) with treatment outcomes for 110 clinical adult patients receiving the same treatment

modality on a paid outpatient service [107]. Results showed that clinical patients showed significant improvement in their OCD symptoms that were comparable to patients in randomized trials [107].

Many factors have been identified to help reach favorable clinical outcomes [108]. In session (in vivo exposure) have been found to be associated with better clinical outcomes compared to exposure techniques done by patient alone at home [108]. Tremendous reductions in symptoms is associated with complete response prevention, as opposed to partial response prevention [108].

CBT is indicated for patients with OCD with various levels of symptom severity. It is worthwhile to mention some of the considerations when evaluating a patient's suitability for this treatment modality which include

1. An understanding of the treatment module and rationale, given that the implementation of CBT requires a good grasp of main concepts that are practiced by the patient as homework. Therefore, the utilization of CBT with people who are cognitively challenged may be difficult.
2. Low insight level for the OCD symptoms poses a challenge for patients and therapists. Low insight is associated with reduced engagement in exposure techniques, thus rendering the use of cognitive techniques essential to increase insight levels for patients [109]. In addition, individuals with predominantly hoarding symptoms have showed lower responses to CBT [104].
3. Patient's preference: CBT is preferred by patients with no co-morbid conditions, mild to moderate OC symptoms, or those with a preference for psychotherapy over medications [110]. Furthermore, CBT is the preferred choice for pregnant women [110]. CBT is highly effective for patients who did not or partially respond to treatment with psychotropic medications [111, 112]. However, in some cases both medications and CBT are preferred [113]. However, some cases warrant the use of medications such as the presence of severe OCD symptoms, low psychological insight, and co-morbidities [113]. Respecting the patient's choice of his/her preferred treatment modality, after discussing the pros and cons of each, is pivotal and is linked to increased adherence to treatment [114].
4. The presence of co morbid conditions such as depression and anxiety along with OCD reduces the efficacy of CBT techniques [108].
5. Negative emotional involvement by family members was found to be associated with higher treatment dropout rates. Hostility was a consistent factor associated with early dropout rates and negative treatment outcome [115].

Other Psychotherapy Modalities: Among all psychosocial interventions and techniques, exposure and response prevention, has led to better clinical outcomes for patients with OCD [116]. Given the absence of randomized clinical trials to investigate the effectiveness of other psychotherapeutic modalities in psychotherapy [106], CBT is considered as the gold *standard for the treatment of OCD*.

6. Psychotropic medications or CBT?

The utilization of CBT is associated with better clinical outcomes compared to the use of Serotonin Reuptake Inhibitors. Although Clomipramine (CMI) was considered as the first line drug therapy associated with significant improvements in OCD symptoms, [117], the randomized trial conducted by Foa et al. (2005) showed that clinical outcomes did not differ for the two control groups (EX/RP alone, and EX/RP plus Clomipramine) [61]. In addition, research has indicated that D-cycloserine has shown to produce favourable clinical outcomes when augmented with CBT [118]. Specifically, D-cycloserine helped to reduce response times after exposure exercises [119].

7. Conclusion

Over the last 40 years, EX/RP has been recommended as first choice treatment for OCD, with CBT as an alternate. More work is needed to enhance CBT programs and make it sensitive to the population needs. Future studies need to explore the treatment responses for specific sub-types of OCD patients such as “washers” and “checkers”. Such understanding will help in designing sensitive treatment plans for the various sub-types of OCD. Also, future studies need to incorporate patients’ populations with co-morbid conditions as they tend to be excluded from such studies, thus making it difficult to know the efficacy level for CBT in this patient population.

Abbreviations

OCD	Obsessive Compulsive Disorder
CBT	Cognitive Behavioural Therapy
DSM	Diagnostic and Statistical Manual of Mental Disorders
BDD	Body Dysmorphic Disorder
SSRIs	Selective Serotonin Reuptake Inhibitors
YBOCS	Yale Brown Obsessive Compulsive Scale
YBOCS-CL	Yale Brown Obsessive Compulsive Scale Checklist
CS	Conditioned Stimulus
UCS	Unconditioned Stimulus
ICD	International Classification of Disorders
EX/RP	Exposure and Response Prevention
CMI	Clomipramine

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References

- [1] American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4th ed. Washington, DC: American Psychiatric Association; 1980.
- [2] Karno M, Goldin JM, Sorenson SB et al. The epidemiology of obsessive-compulsive disorder in five US communities. *Archives of General Psychiatry*, 1988; 45: 1094-99.
- [3] Katerberg H, Delucchi KL, Stewart SE, Lochner C, Denys D, Stack DE, Andresen JM, Grant JE, Kim SW, Williams KA, den Boer JA, van Balkom A, Smit JH, van Oppen P, Polman A, Jenike MA, Stein DJ, Mathews CA, Cath DC. Symptom Dimensions in OCD: Item-Level Factor Analysis and Heritability Estimates. *Behav Genet*, 2010; 40(4): 505-517.
- [4] Stein DJ. Obsessive-compulsive disorder. *The Lancet*. 2002; 360: 397-405.
- [5] Flament MF, Whitaker A, Rapoport JL, Davies M, Berg CZ, Kalikow K, Sceery W, Shaffer D. Obsessive compulsive disorder in adolescence: An epidemiological study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 1988; 27: 764-771.
- [6] Valleni-Basile LA, Garrison CZ, Jackson KL, Waller JL, McKeown RE, Addy CL, Cuffe SP. Frequency of obsessive-compulsive disorder in a community sample of young adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, 1994; 33: 782-791
- [7] Zohar H, Ratzoni G, Pauls DL, Apter A, Bleich A, Kron S, Rappaport M, Weizman A, Cohen DJ. An epidemiological study of obsessive-compulsive disorder and related disorders in Israeli adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, 1992; 31: 1057-1061.

- [8] Pigott TA. Obsessive-compulsive disorder: symptom overview and epidemiology. *Bull Menninger Clin*, 1998 ;62(4 Suppl A): A4-32.
- [9] Kolada JL, Bland RC, Newman SC. Obsessive-compulsive disorder. *Acta Psychiatrica Scandinavica Supplementum*, 1994; 376: 24–35.
- [10] Noshirvani HF, Kasvikis Y, Marks IM, Tsakirisa F, Monteiro WO. Gender-divergent aetiological factors in obsessive-compulsive disorder. *British Journal of Psychiatry*, 1991; 158: 260–263.
- [11] Russell EJ, Fawcett JM, Mazmanian D. (2013). Risk of obsessive-compulsive disorder in pregnant and postpartum women: a meta-analysis. *Journal of Clinical Psychiatry*, 2013; 74 (4):377-85.
- [12] Grohol, J. DSM-5 changes, Obsessive –Compulsive and Related Disorders. *Psych Central*, 2013. <http://pro.psychcentral.com/2013/dsm-5-changes-obsessive-compulsive-and-related-disorders/004404.html> (Accessed on September 11 2013).
- [13] Abramowitz, J.S. Treatment of obsessive-compulsive disorder in patients who have comorbid major depression. *Journal of Clinical Psychology*, 2004; 60, 1133–1141.
- [14] Abramowitz, J.S., Schwartz, S.A., Moore, K.M. et al. Obsessive-compulsive symptoms in pregnancy and the puerperium: a review of the literature. *Journal of Anxiety Disorders*, 2003; 17: 461–478.
- [15] Apter A, Horesh N, Gothelf D, et al. Depression and suicidal behavior in adolescent inpatients with obsessive compulsive disorder. *Journal of Affective Disorders*, 2003; 75: 181–189.
- [16] Biederman J, Petty C, Faraone SV, et al. Moderating effects of major depression on patterns of comorbidity in patients with panic disorder. *Psychiatry Research*, 2004; 126, 143–149.
- [17] LaSalle, V.H., Cromer, K.R., Nelson, K.N., et al. Diagnostic interview assessed neuropsychiatric disorder comorbidity in 334 individuals with obsessive compulsive disorder. *Depression and Anxiety*, 2004; 19, 163–173.
- [18] Nestadt, G., Addington, A., Samuels, J., et al. The identification of OCD:related subgroups based on comorbidity. *Biological Psychiatry*, 2003; 53, 914–920.
- [19] Welkowitz, L.A., Struening, E.L., Pittman, J., et al. Obsessive-compulsive disorder and comorbid anxiety problems in a national anxiety screening sample. *Journal of Anxiety Disorders*, 2000; 14, 471–482.
- [20] Abram KM, Teplin LA, McClelland GM, et al. Comorbid psychiatric disorders in youth in juvenile detention. *American Journal of Psychiatry*, 2003; 60:1097–1108.
- [21] Bakken K, Landheim AS, Vaglum P. Primary and secondary substance misusers: do they differ in substance-induced and substance-independent mental disorders? *Alcohol and Alcoholism*, 2003; 38, 54–59.

- [22] Fals-Stewart W, Angarano K. Obsessive-compulsive disorder among patients entering substance abuse treatment: prevalence and accuracy of diagnosis. *Journal of Nervous and Mental Disease*, 1994; 182, 715–719.
- [23] Frare F, Perugi G, Ruffolo G, *et al.* Obsessive-compulsive disorder and body dysmorphic disorder: a comparison of clinical features. *European Psychiatry*, 2004; 19, 292–298.
- [24] Jordan J, Joyce PR, Carter FA, *et al.* Anxiety and psychoactive substance use disorder comorbidity in anorexia nervosa or depression. *International Journal of Eating Disorders*, 2003; 34, 211–219.
- [25] Hollander E, Greenwald S, Neville D, Johnson J, Hornig CD, Weissman MM. Uncomplicated and comorbid obsessive-compulsive disorder in an epidemiological sample. *Depress Anxiety*, 1997; 4: 111–19.
- [26] Matsunaga H. Current and emerging features of obsessive-compulsive disorder—trends for the revision of DSM-5. *Seishin Shinkeigaku Zasshi*, 2012; 114(9): 1023–30.
- [27] Aouizerate B, Guehl D, Cuny E, Rougier A, Bioulac B, Tignol J, Burbaud P. Pathophysiology of obsessive-compulsive disorder: a necessary link between phenomenology, neuropsychology, imagery and physiology. *Prog Neurobiol*, 2004; 72(3): 195–221.
- [28] The Clomipramine Collaborative Study Group. Clomipramine in the treatment of patients with obsessive-compulsive disorder. *Arch Gen Psychiatry*, 1991; 48(8): 730–8.
- [29] Mataix-Cols D, Pertusa A, Leckman JF. Issues for DSM-V: how should obsessive-compulsive and related disorders be classified? *American Journal of Psychiatry*, 2007; 164:1313–1314.
- [30] Pauls DL. The genetics of obsessive compulsive disorder: a review of the evidence. *Am J Med Genet C Semin Med Genet*, 2008; 148C:133–139.
- [31] Gothelf D, Aharonovsky O, Horesh N, *et al.* (2004) Life events and personality factors in children and adolescents with obsessive-compulsive disorder and other anxiety disorders. *Comprehensive Psychiatry*, 45, 192–198.
- [32] Khanna S, Rajendra PN, Channabasavanna SM. Life events and onset of obsessive compulsive disorder. *International Journal of Social Psychiatry*, (1988b); 34: 305–309.
- [33] vanGrootheest DS, Boomsma DI, Hetttema JM *et al.* Heritability of obsessive-compulsive symptom dimensions. *Am J Med Genet B Neuropsychiatr Genet*. 2008; 147B: 473–478.
- [34] Alsobrook IJ, Leckman JF, Goodman WK *et al.* Segregation analysis of obsessive-compulsive disorder using symptom based factor scores. *Am J Med Genet*, 1999; 88: 669–675.

- [35] Cullen B, Brown CH, Riddle MA et al. (2007). Factor analysis of the Yale-Brown obsessive compulsive scale in a family study of obsessive-compulsive disorder. *Depress Anxiety*;24:130–138.
- [36] Chacon P, Rosario-Campos MC, Pauls DL et al. Obsessive compulsive symptoms in sibling pairs concordant for obsessive compulsive disorder. *Am J Med Genet B Neuropsychiatr Genet.* 2007; 144B:551–555.
- [37] Hasler G, Pinto A, Greenberg BD et al. Familiality of factor analysis-derived YBOCS dimensions in OCD-affected sibling pairs from the OCD Collaborative Genetics Study. *Biol Psychiatry*, 2007; 61:617–625.
- [38] Pinto A, Greenberg BD, Grados MA et al. Further development of YBOCS dimensions in the OCD collaborative genetics study: symptoms vs. categories. *Psychiatry Res*, 2008;160:83–93.
- [39] Mathews CA, Nievergelt CM, Azzam A et al. Heritability and clinical features of multigenerational families with obsessive compulsive disorder and hoarding. *Am J Med Genet B Neuropsychiatry Genetics*, 2007; 144:174–182.
- [40] Goodman WK, Price LH, Rasmussen SA, Mazure C, Fleischmann RL, Hill CL, Heninger GR, Charney DS. The Yale-Brown Obsessive Compulsive Scale. I. Development, use, and reliability. *Arch Gen Psychiatry*, 1989; 46(11):1006-11.
- [41] Goodman WK, Price LH, Rasmussen SA, Mazure C, Delgado P, Heninger GR, Charney DS. The Yale-Brown Obsessive Compulsive Scale. II. Validity. *Archives of General Psychiatry*, 1989; 46(11): 1012-6.
- [42] Bloch MH, Landeros-Weisenberger A, Rosario MC, Pittenger C, Leckman JF. Meta-analysis of the symptom structure of obsessive-compulsive disorder. *American Journal of Psychiatry*, 2008; 165(12): 1532-42.
- [43] Leon AC, Portera L, Weissman MM. The social costs of anxiety disorders. *British Journal of Psychiatry*, 1995; 166 (Suppl 27), 19–22.
- [44] Antony MM, Roth D, Swinson RP, et al. Illness intrusiveness in individuals with panic disorder, obsessive-compulsive disorder, or social phobia. *Journal of Nervous and Mental Disease*, 1998; 186, 311–315.
- [45] Koran, L.M. Quality of life in obsessive-compulsive disorder. *Psychiatric Clinics of North America*, 2000; 23, 509–517.
- [46] Hollander E. Obsessive-compulsive related disorders. Washington, USA: American Psychiatric Press, 1993.
- [47] Allison A. A Brief History of Obsessive Compulsive Disorder: Ideas about OCD, a serious mental illness, have evolved from ancient notions of melancholia and possession through to Freud's psychoanalysis and modern neurochemistry. *Melancholia*,

- 2013; 1514. <http://suite101.com/article/brief-history-of-obsessive-compulsive-disorder-a70602>. (Accessed on 29th August 2013).
- [48] Aardema F, O'Connor K. The menace within: obsessions and the self. *International Journal of Cognitive Therapy*, 2007; 21: 182-197.
- [49] Berrios GE. Obsessive Compulsive Disorder: Its conceptual history in France during the 19th Century. *Comprehensive Psychiatry*, 1989; 30(4): 283-95.
- [50] Cordioli AV, Vivan A. Cognitive-Behavioral Therapy of Obsessive-Compulsive Disorder, Standard and Innovative Strategies in Cognitive Behavior Therapy, Dr. Irismar Reis De Oliveira (Ed.) 2012. ISBN: 978-953-51-0312-7, InTech, Available from: <http://www.intechopen.com/books/standard-and-innovativestrategies-in-cognitive-behavior-therapy/cognitive-behavioral-therapy-of-obsessive-compulsive-disorder>. (Accessed on 28th August 2013).
- [51] Jenike MA. Obsessive-compulsive disorder: efficacy of specific treatments as assessed by controlled trials. *Psychopharmacology Bulletin*, 1993; 29(4): 487-99.
- [52] Mowrer OH. Stimulus response theory of anxiety. *Psychology Review*, 1939; 46:553–565.
- [53] Foa EB. Cognitive behavioral therapy of obsessive-compulsive disorder. *Dialogues in Clinical Neuroscience*, 2010; 12(2): 199-207.
- [54] Dollard J, Miller NE. *Personality and Psychotherapy; an Analysis in Terms of Learning, Thinking, and Culture*. New York, NY:McGraw-Hill, 1950.
- [55] Roper G, Rachman S. Obsessional-compulsive checking: experimental replication and development. *Behav Res Ther*. 1976; 14:25–32.
- [56] Roper G, Rachman S, Hodgson R. An experiment on obsessional checking. *Behav Res Ther*. 1973; 11(3): 271–277.
- [57] Stein DJ, Fineberg NA, Joseph Bienvenu O, Denys D, Lochner C, Nestadt G, Leckman JF, Rauch SL, Phillips KA. Should OCD be classified as an Anxiety Disorder in DSM-V? *Depression and Anxiety*, 2010; 27: 495-506.
- [58] American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders (Fifth ed.)*. Arlington, VA: American Psychiatric Publishing, 2013, pp. 5–25.
- [59] Marks I, Hodgson, R, Rachman, S. Treatment of chronic obsessive-compulsive neurosis by in-vivo exposure – a two year follow-up and issues in treatment. *The British Journal of Psychiatry*, 1975; 127: 349-364.
- [60] Foa E, Goldstein A. Continuous exposure and complete response prevention in the treatment of obsessive-compulsive neurosis. *Behavioral Therapy*, 1978; 9(5): 821-829.
- [61] Foa E, Liebowitz M, Kozak M, Davies S, Campeas R, Franklin M, Huppert J, Kjesterned K, Rowan V, Schmidt A, Simpson H, Tu X. Randomized, placebo-controlled trial of exposure and ritual prevention, clomipramine, and their combination in the treat-

- ment of obsessive-compulsive disorder. *The American Journal of Psychiatry*, 2005; 162(1); 151-161.
- [62] Stanley MA, Turner SM. Current status of pharmacological and behavioral treatment of obsessive-compulsive disorder. *Behavior Therapy*, 1995; 26, 163–186.
- [63] Antony M, Purdon C, Summerfeldt L. Psychological treatment of obsessive compulsive disorder: Fundamentals and Beyond. American Psychological Association. Washington D.C., USA, 2007.
- [64] Wright JH, Basco MR, Thase ME. Learning cognitive-behavior therapy. An illustrated guide. American psychiatric publishing Inc: Washington DC, 2006.
- [65] Roth A, Fonagy P. What works for whom? A critical review of psychotherapy Research (2nd ed.). London: Guilford Publications, Inc, 2005.
- [66] Rachman D, de Silva P. Abnormal and normal obsession. *Behaviour Research and Therapy*. 1978; 16(4): 233-248.
- [67] Foa EB, Kozak MJ. Treatment of anxiety disorders: implications for psychopathology. In: Tuma AH, Maser JD, eds. *Anxiety and the Anxiety Disorders*. Hillsdale, NJ: Lawrence Erlbaum Associates, 1985; 421–452.
- [68] Salkovskis P. Obsessional-compulsive problems: a cognitive-behavioral analysis. *Behavior Research and Therapy*. 1985; 23(5): 571-583.
- [69] Salkovskis P, Forrester E, Richards C. Cognitive-behavioural approach to understanding obsessional thinking. *The British Journal of Psychiatry*. 1998; 173(35): 53-63.
- [70] O'kearney R. Responsibility Appraisals and Obsessive-Compulsive Disorder: A Critique of Salkovski'S Cognitive Theory. *Australian Journal of Psychology*. 1998; 50(1): 43-47.
- [71] Rachman S. A cognitive theory of compulsive checking. *Behaviour Research and Therapy*, 2002; 40(6): 625-639.
- [72] Frost RO, Steketee G. (Eds.). *Cognitive approaches to obsessions and compulsions: Theory, assessment, and treatment*. Amsterdam: Pergamon Press, 2002.
- [73] Purdon C, Clark DA. The need to control thoughts. In R. O. Frost & G. Steketee (Eds.), *Cognitive approaches to obsessions and compulsions: Theory, assessment, and treatment* (pp. 29–44). Amsterdam: Pergamon Press, 2002.
- [74] Thordarson DS, Shafran R. Importance of thoughts. In R. O. Frost & G. Steketee (Eds.), *Cognitive approaches to obsessions and compulsions: Theory, assessment, and treatment* (pp.15–28). Amsterdam: Pergamon Press, 2002.
- [75] Sookman D, Pinard G. Overestimation of threat and intolerance of uncertainty in obsessive compulsive disorder. In R. O. Frost & G. Steketee (Eds.), *Cognitive ap-*

- proaches to obsessions and compulsions: Theory, assessment, and treatment (pp. 63–90). Amsterdam: Pergamon Press, 2002.
- [76] Frost RO, Novara K, Rhéaume J. Perfectionism in obsessive compulsive disorder. In R O. Frost & G. Steketee (Eds.), *Cognitive approaches to obsessions and compulsions: Theory, assessment, and treatment* (pp. 91–106). Amsterdam: Pergamon Press, 2002.
- [77] Freeston M, Rhéaume J, Ladouceur R. Correcting faulty appraisal of obsessional thoughts. *Behavior Research and Therapy*, 1996; 34(5-6): 433-446.
- [78] Abramowitz J, Taylor S, McKay D. Obsessive-compulsive disorder. *Lancet*, 2009; 374(9688): 491-499.
- [79] Whittal M. Land, McLean PD. CBT for OCD: The rationale, protocol and challenges. *Cognitive behavioral practice*, 1999; 6: 383-396.
- [80] Koran L, Hanna G, Hollander E, Nestadt G. Practice guideline for the treatment of patients with obsessive-compulsive disorder. *The American Journal of Psychiatry*, 2007; 164(7): 5–53.
- [81] Swedo SE, Leonard HL, Gravy M et al. Pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections: clinical description of the first 50 cases. *American Journal of Psychiatry*. 1998; 155: 264-71.
- [82] Raffin A, Fachel J, Ferrão Y, de Souza F, Cordioli A. Predictors of response to group cognitive-behavioral therapy in the treatment of obsessive-compulsive disorder. *European Psychiatry*, 2009; 24(5): 297-306.
- [83] Houghton S, Saxon D. An evaluation of large group CBT psycho-education for anxiety disorders delivered in routine practice. *Patient education and counseling*, 2007; 68: 107-110.
- [84] Calvocoressi L, Lewis B, Hariis M, Trufan S, Goodman W, McDougle C, Price L. Family accommodation in obsessive-compulsive disorder. *The American Journal of Psychiatry*, 1995; 152(3): 441-443.
- [85] Franklin ME, Foa EB. Cognitive behavioral treatments for obsessive-compulsive disorder. In J. M. Gorman (Ed.), *A guide to treatments that work* (pp. 339–357); New York: Oxford University Press, 1998.
- [86] Maltby N, Tolin DF. A Brief Motivational Intervention for Treatment-Refusing OCD Patients. *Cognitive Behavior Therapy*, 2005; 34(3): 176-184.
- [87] Merlo LJ, Storch EA, Lehmkuhl HD, Jacob ML, Murphy TK, Goodman WK, Geffken GR. Cognitive Behavioral Therapy Plus Motivational Interviewing Improves Outcome for Pediatric Obsessive–Compulsive Disorder: A Preliminary Study. *Cognitive Behaviour Therapy*, 2010; 39(1), 24-27.
- [88] Salkovskis PM, Richards C, Forrester E. Psychological treatment of refractory obsessive compulsive disorder and related problems, in *Obsessive Compulsive Disorder:*

contemporary issues in treatment (personality and clinical psychology series). Edited by Goodman, WK, Rudorfer, M.V., Mahwah, N.J, Lawrence Earlbaum Associates, Inc, 2000, pp.201-221.

- [89] Foa E, Huppert J, Leiberg S, Langner R, Kichic R, Hajcak G, Salkovskis P. The Obsessive Compulsive Inventory: Development and validation of a short version. *The Psychological Assessment*, 2002; 14(4): 485-495.
- [90] Hupper JD, Roth DA. Treating obsessive-compulsive disorder with exposure and response prevention. *The behavior Analysis today*, 2003; 4(4): 66-70.
- [91] Braga D, Cordioli A, Niederauer K, Manfro G. Cognitive-behavioral group therapy for obsessive-compulsive disorder: a 1-year follow-up. *Acta Psychiatrica Scandinavica*, 2005; 112(3): 180-186.
- [92] Bell J. Relapse Prevention in the Treatment of OCD. International OCD Foundation, 2012. http://www.ocfoundation.org/eo_relapse.aspx. (Accessed on 01 September 2013).
- [93] Wagner AP. Cognitive-Behavioral Therapy for Children and Adolescents with Obsessive-Compulsive Disorder. *Brief Treatment and Crisis Intervention*, 2003; 3(3): 291-306.
- [94] Marks, I. Behavior therapy for obsessive compulsive disorder. A decade of progress. *Canadian Journal of Psychiatry*, 1997; 42: 1021–1026.
- [95] March J, Franklin M, Nelson A, Foa, E. Cognitive-behavioral psychotherapy for pediatric obsessive-compulsive disorder. *Journal of Clinical Child Psychology*, 2001; 30: 8–18.
- [96] Rapoport J, Inoff -Germain G, Weissman M, Greenwald S, Narrow W, Jensen P, et al. Childhood obsessive-compulsive disorder in the NIMH MECA study. Parent vs. child identification of cases. *Journal of Anxiety Disorders*, 2000; 14: 535–548.
- [97] Hibbs E, Hamburger S, Lenane M. Determinants of expressed emotion in families of children and adolescents. *Journal of Child Psychology and Psychiatry*, 1991; 32: 757–770.
- [98] March J, Mulle K, Herbel B. Behavioral psychotherapy for children and adolescents with obsessive-compulsive disorder: An open trial of a new protocol-driven package. *Journal of the American Academy of Child and Adolescent Psychiatry*, 1994; 33: 333–341.
- [99] Waters T, Barrett P, March JS. Cognitive-Behavioral family treatment of childhood obsessive-compulsive disorder. *American Journal of Psychotherapy*, 2001; 55(3): 372–387.
- [100] Wagner AP. *What to do when your child has obsessive-compulsive disorder: Strategies and solutions*, 2002; Rochester, NY: Lighthouse Press.

- [101] Wagner AP. Treatment of OCD in children and adolescents: A cognitive-behavioral therapy manual, 2003; Rochester, NY: Lighthouse Press.
- [102] Wagner AP. Up and down the Worry Hill: A children's book about obsessive-compulsive disorder, 2000; Rochester, NY: Lighthouse Press.
- [103] Schwartz JM. Brain lock. New York: Harper-Collins, 1996.
- [104] Foa EB, Kozak MJ. Psychological treatment for obsessive-compulsive disorder. In: Long-term treatments of anxiety disorders, Mavissakalian MR, Prien RF (Eds), American Psychiatric Press, Inc, Washington, DC 1996. p.285.
- [105] Butler AC, Chapman JE, Forman EM, Beck AT. The empirical status of cognitive-behavioral therapy: a review of meta analyses. *Clin Psychol Rev*, 2006; 26(1): 17-31.
- [106] Gava I, Barbui C, Aguglia E, et al. Psychological treatments versus treatment as usual for obsessive compulsive disorder (OCD). *Cochrane Database Syst Rev*, 2007; CD005333.
- [107] Franklin ME, Abramowitz JS, Kozak MJ, Levitt JT, Foa EB. Effectiveness of exposure and ritual prevention for obsessive-compulsive disorder: randomized compared with nonrandomized samples. *J Consult Clin Psychol*, 2000; 68(4): 594-602.
- [108] Abramowitz JS, Foa EB. Does comorbid major depressive disorder influence outcome of exposure and response prevention for OCD? *Behav Ther*, 2000; 31:795.
- [109] Foa EB, Abramowitz JS, Franklin ME, Kozak MJ. Feared consequences, fixity of belief, and treatment outcome in patients with obsessive-compulsive disorder. *Behav Ther*, 1999; 30:717.
- [110] Flosnik DL, Khin EK. Cognitive-Behavioral Therapy for Obsessive-Compulsive Disorder in Pregnancy. *Psychiatric Annals*, 2012; 42(7): 269-271.
- [111] Tolin, D. Maltby, N., Diefenbach, G., Hannan, S., Worhunsky, P. (2004). Cognitive behavioral therapy for medication nonresponders with obsessive-compulsive disorder: a wait-list-controlled open trial. *Journal of Clinical Psychiatry*, 2004; 65(7): 922-931.
- [112] Tenneij, N., van Megen, H, Denys, D., Westenberg, H. (2005). Behavior therapy augments response of patients with obsessive-compulsive disorder responding to drug treatment. *Journal of Clinical Psychiatry*, 2005; 66(9): 1169-1175.
- [113] Stein DJ, Denys D, Gloster AT, Hollander E, Leckman JF, Rauch SL, Phillips KA. Obsessive-compulsive disorder: diagnostic and treatment issues. *Psychiatr Clin North Am*, 2009; 32(3): 65-85.
- [114] Deacon BJ, Abramowitz JS. Patient's perception of pharmacological and cognitive-behavioral treatments for anxiety disorders. *Beh Ther*, 2005; 36:139.
- [115] oa EB. Failure in treating obsessive-compulsives. *Behav Res Ther*, 1979; 17:169.

- [116] Abramowitz JS. Effectiveness of psychological and pharmacological treatments for obsessive-compulsive disorder: a quantitative review. *J Consult Clin Psychol*, 1997; 65:44.
- [117] Fernandez-Cordoba, E., Lopez-IborAlino J. Monochlorimipramine in mental patients resisting other forms of treatment. *ActasLusoEspNeurolPsiquiatr*, 1967; 26:119–147.
- [118] Chasson GS, Buhlmann U, Tolin DF, et al. Need for speed: evaluating slopes of OCD recovery in behavior therapy enhanced with d-cycloserine. *Behav Res Ther* 2010; 48:675.
- [119] Kushner MG, Kim SW, Donahue C, et al. D-cycloserine augmented exposure therapy for obsessive-compulsive disorder. *Biol Psychiatry* 2007; 62:835.

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