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Research Report

The impact of depression on the treatment of obsessive–compulsive disorder: Results from a 5-year follow-up ☆

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ABSTRACT

Background: Many OCD patients present with comorbid conditions, and major depression is one of the most frequent comorbidities observed. OCD patients with comorbid depression exhibit functional disability and poor quality of life. However, it is unclear whether depressive symptoms are predictive of treatment response, and debate remains whether they should be targeted in the treatment of comorbid patients. The current study aimed at assessing the predictive value of depression and OCD symptoms in the long term outcome of OCD treatment.
Methods: In the current study, relations between OCD and depressive symptoms were systematically investigated in a group of 121 OCD patients who received 16 sessions of behavior or cognitive therapy either alone or with fluvoxamine.
Results: Depression (either as a continuous or categorical variable) was not predictive of treatment response in any of the treatment modalities for up to 5 years of follow-up. Changes in OCD symptoms largely predicted changes in depressive symptoms but not vice versa.
Limitations: Subsequent to participation in the RCT, almost two-thirds of the participants received some form of additional treatment (either pharmacological or psychological), and as a result, it is impossible to determine interaction effects with additional treatment received after the trial.
Conclusions: Treatment of OCD with comorbid depression should focus on amelioration of OCD symptoms. When OCD treatment is successful, depressive symptoms are likely to ameliorate as well.

1. Introduction

Once considered a rare phenomenon, epidemiological studies have found obsessive–compulsive disorder (OCD) to be the fourth most common psychiatric disorder (Myers et al., 1984), with prevalence rates between 1.5 and 3% in the general population (Bebbington, 1998; Stein et al., 1997). OCD is highly debilitating, and the World Health Organization has found it to be among the 10 most disabling medical conditions (Angst et al., 2004; Murray and Lopez, 1996).

OCD is usually not present as a single diagnosis as more than half the patients present with at least one other DSM-IV diagnosis (Steketee and Barlow, 2004). OCD patients with comorbid psychiatric diagnoses suffer from poorer quality of life and functioning than OCD patients without comorbity

☆ The present research was conducted at this location.

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(Huppert et al., 2009). Particularly striking is the relation between OCD and depression given that as many as 50% of patients present with comorbid depression (Crino and Andrews, 1996; Hong et al., 2004; Nestadt et al., 2001). Interestingly, depression was found to mediate most of the impact of OCD symptoms on functional disability in OCD patients (Storch et al., 2010), particularly in the presence of comorbid diagnoses (Huppert et al., 2009). Furthermore, depression levels were found to contribute to increased occupational disability of OCD patients (Mancebo et al., 2008). These findings stress the importance of reducing depressive symptoms in the treatment of OCD patients.

Cognitive behavior therapy (CBT) either in the form of behavior therapy (BT, consisting of exposure and response prevention) or cognitive therapy (CT) is the most efficacious psychological treatment of OCD (Rosa-Alcázar et al., 2008). However, data pertaining to the impact of depression on CBT treatment of OCD is inconsistent (for a review see: Kempe et al., 2007). In some studies, comorbid depression did not affect CBT results (McLean et al., 2001; Stewart et al., 2006; Storch et al., 2010), whereas in other studies patients with mood disorders (dysthymia or major depression) tended to respond partially to treatment (Keijsers et al., 1994; Raffin et al., 2009; Steketee et al., 1999). Research findings of combination treatment consisting of CBT and pharmacological treatment yielded more consistent results demonstrating depression levels do not affect treatment outcome (Steketee et al., 2000; Tolin et al., 2004). Some observations can be made about existing research of the influence of depression on CBT for OCD. First, in some studies, depressed patients were excluded (Keeley et al., 2008). Second, depression is measured in some studies as a continuous variable and in others as a categorical variable (as a comorbid condition). Third, influence of BT and CT has not been directly studied apart, whereas some researchers (Abramowitz, 2004) have suggested that CT may influence both conditions whereas BT might have more specific influence on OCD symptoms only, since the skills learned in CT are applicable to both disorders. Fourth, direction of influence — whether changes in depression symptoms affect changes in OCD symptoms or vice versa, has not been investigated. Fifth, the long term influence of depressive symptoms on OCD has scarcely been investigated. Sixth, effects of depression on psychological treatment of OCD (either with or without medications) have not been investigated, whereas these treatment modalities might affect depression differentially.

Therefore, the aim of the present study was to systematically assess the long term effect of depressive symptoms on OCD symptoms in the treatment of OCD. We used data from a large RCT trial in which 121 OCD patients were treated with BT or CT either with or without fluvoxamine.

We were particularly interested in the following lines of investigation:

(1) Do levels of depressive symptoms (depression as a continuous or categorical variable) affect treatment and follow up effects of BT or CT, with or without fluvoxamine?
(2) Do changes in depressive symptoms cause changes in OCD symptoms or vice versa, in the treatment of OCD with BT or CT with or without fluvoxamine?

2. Methods

2.1. Participants

The study was approved by the VU-University, Medical Centre Ethical Review Committee (Amsterdam, the Netherlands). All patients had participated in one of two 2-sited randomized controlled trials (RCTs), which were originally set up to compare the effectiveness of CT and BT for the treatment of OCD (van Balkom et al., 1998; van Oppen et al., 1995a, 2005). We combined data from these RCTs as the inclusion criteria, recruitment process, measures, and treatment protocols were all identical. Moreover, all patients were treated at the same outpatient clinics in the Netherlands (in Delft and Amsterdam) by the same therapists during the same period. Furthermore, identical assessments methods and measurement intervals were used in both studies. All patients who participated in this study were treated at two psychiatric outpatient clinics specializing in the treatment of anxiety disorders. Subjects were recruited by referrals from general practitioners, mental health agencies and newspaper announcements.

Participants were 121 individuals (71 females; 58.7%), between 19 and 64. All participants were diagnosed with primary OCD according to DSM-IIIR criteria using the Anxiety Disorder Interview Schedule-Revised (DiNardo et al., 1983). Many participants had an additional Axis-I disorder (44.6%) and most (67.8%) had received treatment in the past for OCD. Table 1 presents demographic and clinical measures for the entire sample.

Participants were excluded from the present study if they (a) solely reported obsessions (b) had suicidal tendencies (c) had organic brain disease (d) had past or present psychosis (e) had psychoactive substance use disorder (f) had a severe medical disorder (g) were receiving psychological treatment elsewhere and (h) had been treated with either behavior or cognitive therapy or treatment with fluvoxamine in the 6 months preceding baseline.

2.2. Treatments

The present study included 4 treatment conditions: cognitive therapy (CT), behavior therapy (BT), CT plus Fluvoxamine, and BT plus Fluvoxamine. All treatments were 16 weeks long. Therapists in psychological treatments were clinical psychologists who had ample experience in the use of cognitive and behavioral techniques and psychiatrists or psychiatry residents administered Fluvoxamine. Fluvoxamine dosage started at 50 mg per day, and in the absence of disturbing side effects, was increased to a maximum of 300 mg per day after 3 weeks of treatment. After 3 weeks Fluvoxamine remained at a constant level and no changes in dosage were made. Further details regarding the various treatments can be found elsewhere (van Oppen et al., 1995a; van Balkom et al., 1998; van Oppen et al., 2005).

2.3. Procedure

Participants were screened at pre-treatment by an experienced psychiatrist or clinical psychologist using a Dutch version of the standardized Anxiety Disorder Interview Schedule (DiNardo et al., 1983). Following the interview, participants