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Cognitive functioning in medication-free obsessive-compulsive patients treated with cognitive-behavioural therapy

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ABSTRACT

So far it is still uncertain whether neuropsychological test results in obsessive-compulsive disorder (OCD) change after CBT and whether neuropsychological and clinical variables predict response to CBT. 60 medication-free patients hospitalised for severe OCD were treated with CBT including exposure and response prevention (ERP). Pre- and Post-treatment patients completed a cognitive test battery covering a broad range of neuropsychological and clinical variables. The Yale Brown Obsessive Compulsive Scale (Y-BOCS) was used to assess symptom severity. A healthy control group matched for age, gender, intelligence and education completed the same neuropsychological test battery, with similar test–retest intervals as the patients. Analyses showed significantly worse cognitive functioning in patients compared to healthy controls before treatment on many measures. After CBT patients had significantly improved test results regarding speed of information processing, verbal fluency, visuo-constructive functions and set shifting ability while controls did not perform better at the second testing. None of the neuropsychological and clinical variables assessed was significantly correlated with treatment outcome. These results indicate that cognitive dysfunctions in OCD are in part state-dependent and reversible after treatment. Moreover, our results suggest that CBT is successful even in those patients who exhibit cognitive dysfunctions and who are severely affected at the beginning of treatment.

1. Introduction

1.1. Cognitive behavioural therapy and neuropsychological performance

The efficacy of Cognitive Behavioural Therapy (CBT) in the treatment of obsessive-compulsive disorders (OCD) has been confirmed by numerous studies (see e.g. Abramowitz, 2006). So far, many therapy studies have focused rather one-sidedly on symptom-related outcome measures, e.g. the patients' score on the Y-BOCS in OCD treatment studies. In contrast to that CBT aims not only at a mere symptom reduction, but also a change in dysfunctional cognitions. Overton and Menzies (2005) found that change of dysfunctional cognitive patterns even occurs during ERP treatment without further cognitive therapy elements. Such alterations of dysfunctional thoughts, e.g. an increased tolerance of uncertainty after therapy, may bring about more cognitive flexibility and thus promote a better general cognitive functioning. On the other hand, neuropsychological impairments in untreated OCD might play an important role in the maintenance of symptoms. Untreated OCD might be associated with neuropsychological variables that render the cognitive system of OCD patients inflexible and less open to change, for example slower information processing resulting in slower reaction times (Soref, Dar, Argov, & Meiran, 2008), lower verbal fluency and impaired set-shifting (Rampacher et al., 2010). Even before OCD symptoms surface, these variables might increase the probability of inflexible dysfunctional beliefs and of overemphasising intrusive thoughts as Salkovskis' model (1985) proposes, and thus might contribute to the aetiology of OCD. With regard to cognitive impairments in OCD the literature shows heterogeneous evidence. Kuelz, Hohagen, and Voderholzer (2004) reviewed fifty studies examining cognitive impairment in OCD patients and concluded that mainly memory dysfunctions stemming from an impaired organisation during the encoding process as well as executive functions are commonly disturbed in these patients. A study by Rampacher et al. (2010) found delayed spatial recall and verbal fluency as well

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as impaired visual memory, visuo-motor speed and set shifting, visual organisation, and problem solving in OCD. According to a review by Markarian et al. (2010), OCD patients do not have memory impairments, but executive planning and organisational difficulties. The authors conclude that OCD patients apply unfavourable strategies when dealing with memory tasks, which indicates that their problems are due to processing deficits, not to traits. CBT interventions that aim at altering dysfunctional beliefs and behaviours might have a positive side effect on neurocognitive processes by generally increasing the flexibility of the cognitive systems (Kuelz et al., 2006). In line with this, neuroimaging data show that the brain activation in OCD changes after CBT, both in response to symptom provocation and in neuropsychological tasks (Nakao et al., 2005). In a previous study of our research group (Kuelz et al., 2006) it was found that OCD patients were impaired at baseline regarding measures of delayed nonverbal memory, organisational strategies, flexible, self-guided behaviour and speed related set shifting. After CBT they did no longer perform worse than controls. However, Vandborg et al. (2012) report in their systematic review that the evidence on whether neurocognitive functioning changes after CBT in OCD is still inconsistent. Their review reveals that while some studies report persisting neurocognitive impairments and interpret them as a trait of OCD, others found improvements on the same measures such as visuo-spatial functioning and set shifting. One reason for these inconsistencies might be sample sizes that are too small to detect small effects (Vandborg et al., 2012). Also, the nature of the applied treatment varied in the reviewed studies. Some used a combination of selective serotonin reuptake inhibitors (SSRI) and CBT, others SSRI or CBT treatment alone. To draw conclusions on the effect of CBT alone, it will be important to exclude SSRI medication in future studies. Although the mechanisms that are responsible for the positive effects of SSRI on OCD are not clear (Fineberg, Reghunandanan, Brown, & Pampaloni, 2013), SSRI might also ameliorate neurocognitive functioning in OCD (Herrera-Guzman et al., 2010, for example, present such evidence in depressive patients). If this is the case, studies combining SSRI and CBT cannot resolve the effects of either treatment on neurocognitive functioning and treatment outcome.

1.2. Relation between neuropsychological function and therapy outcome

A broad range of prognostic factors for therapy outcome of CBT has been identified during the past years, including biological, individual, social and clinical dimensions (Clark, 2004). Several positive predictors for successful treatment have been found such as a predominance of compulsions, low comorbidity, low levels of depression, lack of magical thinking, high compliance and a stable social network. (Rufer, Fricke, Moritz, Kloss, & Hand, 2006; Abramowitz, 2006; Shavitt et al., 2006; Mataix-Cols, Marks, & Greist, 2002). Several studies have been conducted to investigate the relevance of neuropsychological factors as predictors of therapy outcome but results are inconsistent. While Moritz et al. (2005) did not find significant differences in measures of neuropsychological impairment between responders and non-responders to CBT, others reported significant relations between difficulties in a problem solving task or semantic fluency and negative therapy outcome (Cavedini et al., 2002; Sieg, Leplow, & Hand, 1999). Flessner et al. (2010) found that executive functioning deficits are associated with poorer responses to CBT in children with OCD. These conflicting findings might be due to three different factors. Firstly, the methods applied to record neuropsychological status varied considerably in the different studies so that certain findings could not be replicated in others. Secondly, treatment methods also differ between CBT, mere behaviour therapy (BT) and pharmacological intervention or a mixture of these. Thirdly, most of the sample sizes were too small to detect a possible effect of neuropsychological impairment as a predictor for therapy outcome. To avoid confounding effects it appears reasonable to investigate possible effects of neuropsychological parameters on therapy outcome in patients who are medication-free and exclusively treated with CBT. Moreover, we consider it necessary to broaden the screening for possible neuropsychological predictors in order to increase the chances of detecting relevant variables.

In the present study our first aim was to investigate neuropsychological performance before and after treatment in a large, not medicated sample exclusively treated with CBT. We were interested in neurocognitive variables that are potentially related to increased flexibility of the cognitive system and have been shown to be associated to OCD in former studies. More specifically, we focused on speed of information processing (Soref et al., 2008), verbal fluency, visuo-spatial functioning and set-shifting (Rampacher et al., 2010; Kuelz et al., 2004, 2006).

A second focus of our study was to analyse whether these neuropsychological variables are related to therapy outcome in patients treated exclusively with CBT. In addition to neuropsychological variables we also addressed the relevance of clinical patient variables for successful CBT, such as the level of depression and symptom severity (see 2.2.4.).

2. Method

2.1. Subjects

OCD subjects were recruited from the Department of Psychiatry and Psychotherapy of the University Hospital in Freiburg and from the Department of Psychiatry of the University Hospital in Lübeck. All OCD subjects were inpatients in a specialised unit for the treatment of OCD. Before admission, patients were diagnosed by an experienced clinician according to DSM IV criteria. Patients with substantial neurological impairment, head injury, substance abuse, current or previous psychotic episodes, current Major Depression and age under 18 or over 65 years were excluded from the study. Subjects consisted of 66 patients with a primary OCD diagnosis who did not receive any kind of psychopharmacological treatment throughout the study. In case of a prior intake of psychopharmacological medication, the medication had been withdrawn at least four weeks prior to the study. Six patients dropped out before concluding therapy (9.1%), resulting in a sample of 60 patients with an average age of 32.5 years (mean=M)+/−8.4 (standard deviation=SD) and consisting of 35 female and 25 male participants. All subjects were screened for co-morbid disorders on axis I and II. 10 subjects (16.7%) fulfilled the criteria for a co-morbid diagnosis on axis I and 19 were diagnosed with a co-morbid personality disorder (31.7%). Presence of a personality disorder was not correlated with outcome (see Section 3.2) and thus not considered as a covariate. Subjects reported an average duration of OCD of $M=10.9$ years +/−SD=8.4 and, on average, remained in treatment for 13 weeks (+/−5.7). At the time of admission, the total Y-BOCS score was $M=25.3$ (+/−SD=4.6).

Forty healthy control subjects were recruited through newspaper advertisements and/or personal contact. In total, 39 healthy subjects completed both testings. The average age of the 22 female and 17 male subjects was 28.2 years +/−7.6. There were no significant differences between patients and controls regarding age, sex, educational level and general intelligence. For the normal adults additional exclusion criteria were: evidence for personal and family lifetime history of axis I disorder or history of treatment for a psychiatric disorder that was verified by a clinical interview by an experienced psychiatrist.

Before being included in the study, patients were informed about the procedure and goal of the study and informed consent was obtained.

2.2. Design

Patients were tested in a prospective longitudinal design at two measurement points. The first assessment was conducted immediately after admission and before starting the treatment (T0) while the second measurement took place before discharge after 12 weeks of exposure sessions (T1). Each patient was treated by an experienced clinician, trained in CBT for obsessive-compulsive disorders. Therapeutic intervention was based on a manualised treatment (Lakatos & Reinecker, 2007) containing an initial phase in which the patients received psycho-education and developed an individual working model that explained the obsessive-compulsive behaviour on a micro- and macro level. In the following treatment