Comorbidity of Obsessive-Compulsive Disorder and Depression: Prevalence, Symptom Severity, and Treatment Effect

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Background: The goal of this study was to investigate the co-occurrence of depressive disorders in obsessive-compulsive disorder (OCD) and the effect of these disorders on combined pharmacologic and behavioral treatment for OCD.

Method: A retrospective chart analysis was performed on baseline ratings of 120 OCD patients and posttreatment ratings of 72 of these patients. For depressive symptoms, the Montgomery-Asberg Depression Rating Scale and the Self-Rating Depression Scale were applied; for obsessive-compulsive symptoms, the Yale-Brown Obsessive Compulsive Scale and the Maudsley Obsessive Compulsive Inventory were used; and for general anxiety symptoms, the Self-Rating Anxiety Scale, the Clinical Anxiety Scale, and the State-Trait Anxiety Inventory were given.

Results: One third of the OCD patients in our sample were found to be depressed. Symptom severity on OCD symptoms at baseline did not differ between depressed and nondepressed OCD patients; on general anxiety symptoms, the comorbid group was more severely affected. Both depressed and nondepressed OCD patients responded well to treatment, as reflected in assessments for depressive, obsessive-compulsive, and general anxiety symptoms. However, comorbid depression had a negative effect on treatment: depressed OCD patients showed less improvement than nondepressed OCD patients on most scales.

Conclusion: Depression frequently accompanies OCD and appears to affect treatment outcome negatively. While both groups of patients improved with combination treatment, the OCD-alone group had more improvement than the group that had comorbid depression.

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Clinical psychiatric conditions frequently show overlap between different psychopathologies. Clinical studies have demonstrated that different diagnoses may coexist. A large number of epidemiologic studies have shown a relationship between anxiety disorders and affective disorders.[1,2] Anxiety disorders rarely exist in isolation, with several studies reporting that over 90% of individuals with anxiety disorders have a lifetime history of other psychiatric problems.[3] Comorbidity of any of the anxiety disorders with depression is also very common: panic disorder with or without agoraphobia, social phobia, generalized anxiety disorder (GAD), and obsessive-compulsive disorder (OCD) display a higher than expected rate of co-occurrence with depressive disorders.[4,5] These studies give rise to speculations as to the etiology, pathophysiology, psychopharmacology, and behavioral treatment of both disorders. In the present study, we report on the comorbidity of OCD and depression in an outpatient population that was referred for treatment of OCD.

The magnitude of the comorbidity reported in the literature varies widely[1] and depends on methodology (study design and experience of interviewers), samples (community or treatment), and case definition (diagnostic criteria and thresholds). The term *comorbidity* should be used with caution and should be properly defined. However, in various studies, its use is often vague or erroneous. Maser and Cloninger[6] mention the large number of definitions that are commonly used. Ideally, *comorbidity* should be restricted to disorders and syndromes but is, in effect, also used to describe the co-occurrence of

symptoms. Another erroneous use derives from the nature of the current classification system in psychiatry. The *Diagnostic and Statistical Manual of Mental Disorders,* Fourth Edition (DSM-IV),[7] is intentionally designed to serve as a taxonomic instrument and therefore offers criteria for inclusion and exclusion, but pathognomonic signs in psychiatry are lacking, and a diagnosis is cumulatively made based on the presence of clusters of symptoms. Another confusing factor is the chronological appearance of diseases, since comorbidity can be present throughout a patient's lifetime, at any time in the past, or just at the moment that another disorder exists. Because of the absence of a theoretical basis in the classification system, careful use of DSM-IV is warranted in cases of comorbidity.

The coexistence of more than one psychiatric disorder at a time can also have both theoretical and practical implications. For example, theoretically, the high cooccurrence of more than one psychiatric disease may suggest shared pathogenic or genetic factors. If chronological patterns occur at the same time, certain causal links may be established or preventive action taken. Furthermore, practical clinical implications could be that course and response to treatment differ between patients with one or more diagnoses. For example, it has often been suggested that depressed panic disorder patients are more resistant to treatment than patients with panic disorder alone.[8]

To interpret prevalence rates of comorbid disorders, individual prevalence rates are needed. For OCD these figures have gradually grown over the years. For a long time, OCD was thought to be quite rare since people are apparently reluctant to request treatment due to fear or shame.[9] Recent epidemiologic studies have shown a 6-month prevalence rate of OCD of approximately 1%[10] and a lifetime prevalence rate of 2% to 3%,[2,11–13] which implies that OCD is much more common than formerly suggested. The lifetime prevalence rate for depressive disorders varies from about 8%[14] to 11.7%.[5]

As reported in several studies,[15] depression is the most frequent complication of OCD. Comorbidity rates in reported studies vary widely, from 19% to 90%.[16] This is largely due to methodological and semantic differences. Within this wide range, however, most epidemiologic studies show that about one third of OCD patients suffer from a lifetime depressive episode. In clinical populations, comorbidity rates are increasing to about two thirds.[17] Rasmussen and Eisen[9] found that one third of OCD patients suffer from concurrent depression at referral and two thirds suffer from lifetime depression. A possible explanation for the discrepancy between clinical and nonclinical studies may be that many OCD patients seek help only when depressed, as suggested by Black and Noyes.[15] As for chronology, it is reported that most often the onset of OCD is before that of depression (38%), whereas transition from depression to OCD occurs in only 11% of cases.[15]

As noted earlier, the therapeutic implications of a comorbid depressive disorder in OCD are still poorly understood. Comorbidity affects the outcome of behavioral versus pharmacotherapeutic interventions. For example, in OCD, severe depression may worsen the prognosis for behavioral treatments, whereas most studies suggest that pharmacologic treatment with selective serotonin reuptake inhibitors (SSRIs) is equally effective for OCD with or without concomitant depression.[18]

To better understand the previously mentioned wide range of reported comorbidity, to appreciate the limitations of these studies, and to gain insight into questions regarding the impact of treatment, we decided to conduct the following study in our clinical sample of OCD patients. We investigated the prevalence of depressive disorder as a comorbid diagnosis in a sample of OCD patients in our outpatient clinic, which specializes in anxiety disorders. *Comorbidity* was defined as the simultaneous coexistence of 2 syndromes (i.e., OCD and depressive disorder according to the DSM-IV criteria, not only the presence of subsyndromal symptoms). We also sought to assess the influence of a comorbid depression on treatment outcome for OCD.

Hypotheses were posed as follows: (1) as reported in other studies, point prevalence of depressive disorder in OCD will be in the range of 30% to 60%; (2) the presence of a comorbid depression will be reflected in more severe symptoms of OCD and, therefore, in higher scores on the Maudsley Obsessive Compulsive Inventory (MOCI) and the Yale-Brown Obsessive Compulsive Scale (Y-BOCS); and (3) the effect of combined psychopharmacologic and behavioral treatment for OCD will not differ between depressed and nondepressed OCD patients.

METHOD

We performed a retrospective chart analysis. One hundred twenty patients with DSM-IV Axis I OCD as their principal diagnosis were included for baseline (prevalence and severity) assessments; for 72 patients, posttreatment data were also available. Patients were referred for treatment to the Academic Anxiety Center Maastricht, the Netherlands, from 1995 through 1999. As part of a standard protocol, all patients were evaluated by means of a semistructured psychiatric interview, with special emphasis on anxiety and affective spectrum symptoms. A diagnosis was made according to DSM-IV criteria and confirmed by 2 experienced psychiatrists. Patients also received a physical examination and were additionally evaluated by means of the following standardized questionnaires and structured interviews: the Yale-Brown Obsessive Compulsive Scale (Y-BOCS)[19]; the self-rating Maudsley Obsessive Compulsive Inventory (MOCI)[20];

the Montgomery-Asberg Depression Rating Scale (MADRS)[21]; the Self-Rating Depression Scale (SDS)[22]; the Clinical Anxiety Scale (CAS)[23]; the Self-Rating Anxiety Scale (SAS)[24]; and the State-Trait Anxiety Inventory, state form (STAI-1).[25]

Patients were offered combined psychopharmacologic and behavioral therapy treatment after the diagnostic procedure was performed. Initially, in the first phase, patients received pharmacotherapy, i.e., a prescription for 1 of the current SSRIs, in increasing dosages for 10 to 12 weeks (up to effective clinical doses or maximum daily doses of 300 mg of fluvoxamine, 60 mg of fluoxetine, or 60 mg of paroxetine), with a visit for a global clinical assessment at our clinic every 2 to 4 weeks. In the second phase, behavior therapy was added, based on exposure and response-prevention intervention. The duration of the 2 phases was flexible, depending on individual clinical progression. For example, some OCD patients benefited from medication to such an extent that only a few sessions of behavior therapy were needed to stabilize the symptoms and to obtain satisfactory results, whereas other patients were more complicated in the sense that they needed more prolonged and sometimes extensive behavioral therapy. Discharge was planned by mutual agreement between patient, behavioral therapist, and psychiatrist. At discharge, the initial psychopathologic rating scales were repeated (posttreatment assessments).

Subjects

The data of 120 consecutive OCD patients referred to our center from 1995 to 1999 were included in the study for baseline ratings. For 72 patients, posttreatment assessments were also available. Data from this group were analyzed for treatment effect.

Patients were included if a diagnosis of OCD was made as the principal diagnosis per DSM-IV Axis I criteria. Because of the nature of our clinic, even in comorbid cases, the reason for referral was OCD. The exclusion criterion for the present study was missing data at baseline.

Analysis

All statistics were performed with Statistical Package for the Social Sciences (SPSS-PC), version 10. Student t test for independent samples was used to test differences between depressed and nondepressed OCD subgroups at baseline and after treatment. A t test for paired samples was used to test differences before and after treatment for the 72 completers. A chi-square test was applied to test the proportion of males and females having a depressive episode. Chi-square tests were also used to compare the distribution of depressives in the group of completers and noncompleters. Hypotheses regarding improvement were analyzed using a multivariate analysis of variance (MANOVA).

RESULTS

Baseline Values

Demographics. The majority of the patients in the study had been referred by their family practitioner; some had been referred by psychiatrists. Of the 120 subjects we assessed at baseline, 40 (33%) were male and 80 (67%) were female. Nineteen (40%) of the 48 patients who were assessed only at baseline were men, and 29 (60%) were women (nonsignificant [NS]). Of the 72 patients who also received a posttreatment assessment, 21 (29%) were men and 51 (71%) were female (NS).

The mean ± SD age of our 120 baseline subjects was 37.8 ± 12.1 years. The mean age of the women was 37.1 ± 11.7 years; for the men it was 39.3 ± 12.8 years (NS). The depressed patients were 37.6 ± 11.3 years of age, and the nondepressed were 37.9 ± 12.6 years of age (NS). The mean age of the completers was 39.2 ± 14.0 years, and that of the noncompleters was 37.0 ± 10.7 years (NS).

Prevalence. To address our first hypothesis on the prevalence of a comorbid depressive disorder, all 120 patients were considered. Of these 120 baseline patients with OCD as the principal Axis I diagnosis, 44 patients (37%) also met DSM-IV criteria for depressive disorder at the time of referral (point prevalence). The distribution in the group that had only 1 assessment was 19 (40%) with comorbid depression and 29 (60%) without depression; in the posttreatment group, 25 patients (35%) were depressed at baseline, whereas 47 patients (65%) were not (chi-square, NS).

Notably, the gender distribution in the comorbid subgroup, as well as in the completer and noncompleter subgroups, was decidedly female. It appeared that (for all 120 baseline subjects) only a minority of male OCD patients suffered from comorbid depression—only 6 (15%) of 40 compared with 38 (48%) of 80 of the female OCD patients ($\chi^2 = 12.12$, $p = .001$). For the 72 completers, only 2 of 21 men were depressed, versus 23 of 51 women ($\chi^2 = 8.30$, $p = .006$).

Severity. To answer the second hypothesis on symptom severity, we looked at the mean scores for the 120 baseline subjects and at the subgroups of completers and noncompleters. Mean scores on baseline assessment scales are reported in Table 1. The severity of symptoms at intake for all 120 patients differed for depressed and nondepressed OCD patients on the general anxiety (CAS, $p < .001$; SAS, $p = .001$; and STAI-1, $p < .001$) and depression measures only (MADRS, $p < .001$; SDS, $p < .001$), not for the specific OCD measurements (MOCI [NS] and Y-BOCS [NS]). The severity of initial symptoms across the subgroups of completers and noncompleters differed only on the Y-BOCS ($p = .035$), with the important finding that the noncompleters showed a lower mean value. Within the group of depressed OCD

Table 1. Baseline Scale Scores for Completers Versus Noncompleters Among Patients With Obsessive-Compulsive Disorder (OCD)

	Completers (N = 72)				Noncompleters (N = 48)				Total (N = 120)			
	OCD With Depression (N = 25)		OCD Without Depression (N = 47)		OCD With Depression (N = 19)		OCD Without Depression (N = 29)		OCD With Depression (N = 44)		OCD Without Depression (N = 76)	
Scale (range)	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
MADRS (0–60)	21.0	11.3	11.9	6.8	23.0	7.1	9.5	7.1	21.9	9.6	11.0	7.0
SDS (20–80)	58.2	9.0	49.9	9.4	58.2	6.0	46.4	10.6	58.2	7.8	48.6	9.9
MOCI (0–34)	18.5	6.9	19.8	4.9	19.0	6.3	16.6	7.2	18.7	6.6	18.6	6.0
Y-BOCS (0–40)	32.7	6.6	31.8	5.8	30.9	7.1	27.4	7.0	31.8	6.8	30.2	6.6
STAI-1 (20–80)	62.3	11.5	52.8	12.5	60.5	11.6	48.7	15.3	61.5	11.5	51.2	13.7
CAS (0–24)	12.3	4.6	8.9	4.8	15.2	5.9	7.1	4.9	13.6	5.4	8.2	4.9
SAS (20–80)	49.3	9.1	45.5	9.5	51.1	7.1	41.3	9.1	50.1	8.3	43.9	9.5

Abbreviations: CAS = Clinical Anxiety Scale, MADRS = Montgomery-Asberg Depression Rating Scale, MOCI = Maudsley Obsessive Compulsive Inventory, SAS = Self-Rating Anxiety Scale, SDS = Self-Rating Depression Scale, STAI-1 = State-Trait Anxiety Inventory, Y-BOCS = Yale-Brown Obsessive Compulsive Scale.

Table 2. Treatment Effect in Completers (N = 72)

	OCD With Depression (N = 25)				OCD Without Depression (N = 47)				MANOVA (p Value)			
	Baseline		Posttreatment		Baseline		Posttreatment					
Scale (range)	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Posttreatment	Time	Group	Time by Group
MADRS (0–60)	21.0	11.3	6.4	7.8	11.9	6.8	3.7	4.4	NS	< .001	.002	.029
SDS (20–80)	58.2	9.0	45.2	11.1	49.9	9.4	36.6	9.4	.001	< .001	< .001	NS
MOCI (0–34)	18.5	6.9	13.2	7.0	19.8	4.9	8.1	6.8	.004	< .001	NS	.002
Y-BOCS (0–40)	32.7	6.6	14.9	10.5	31.8	5.8	9.2	6.6	.022	< .001	NS	NS
STAI-1 (20–80)	62.3	11.5	45.7	14.9	52.8	2.5	36.1	11.8	.004	< .001	< .001	NS
CAS (0–24)	12.3	4.6	5.0	4.4	8.9	4.8	4.3	5.8	NS	< .001	NS	NS
SAS (20–80)	49.3	9.1	40.8	9.4	45.5	9.5	32.6	7.4	< .001	< .001	.003	NS

Abbreviations: CAS = Clinical Anxiety Scale, MADRS = Montgomery-Asberg Depression Rating Scale, MANOVA = multivariate analysis of variance, MOCI = Maudsley Obsessive Compulsive Inventory, NS = nonsignificant, SAS = Self-Rating Anxiety Scale, SDS = Self-Rating Depression Scale, STAI-1 = State-Trait Anxiety Inventory, Y-BOCS = Yale-Brown Obsessive Compulsive Scale.

patients, there were no differences on assessments between completers and noncompleters. Interestingly, however, within the nondepressed OCD patient group, it was the completers who had significantly higher scores on the Y-BOCS (p = .017) and MOCI (p = .049).

Posttreatment Values

To test the third hypothesis on treatment effect, the data of the 72 completers were analyzed (Table 2). Mean ± SD treatment duration did not differ significantly between the groups: depressed OCD patients were discharged after 29 ± 9 weeks, and nondepressed patients after 28 ± 13 weeks (NS). Although the mean scores on the MADRS (Figure 1) were not significantly worse for the depressed patients compared with the nondepressed patients posttreatment, on the SDS (Figure 2), the formerly depressed patients still showed significantly more severe (p = .001) symptoms. Posttreatment scores on the Y-BOCS (p = .02) (Figure 3) and MOCI (p = .004) (Figure 4) were worse for the depressed OCD patients, although on these scales depressed and nondepressed patients were equally affected at baseline. Also, symptoms on 2 of the 3 general anxiety measures (SAS, p < .001; STAI-1, p = .004) were worse for the depressed OCD patients (Table 2).

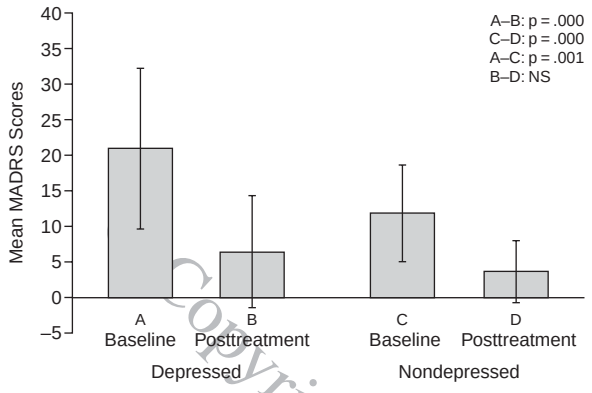
Treatment Effect

Treatment proved to be effective on all measures (Table 2). Paired-sample t tests showed significantly lower mean scores on the posttreatment assessments (p < .001). The repeated-measures MANOVA also revealed significant time effect on all measures for both depressed and nondepressed OCD patients. Differences between the depressed and nondepressed groups were statistically significant for measures on the MADRS, SDS, STAI-1, and SAS, but not statistically significant on the CAS, MOCI, and Y-BOCS (p values in Table 2). MANOVA tests showed a significant time-by-group interaction effect on the MADRS and the MOCI, suggesting that nondepressed patients improved significantly more than depressed ones on these scales. For the other measurements, there were no significant interaction effects. Thus, on these scales, both subgroups improved to a similar extent.

DISCUSSION

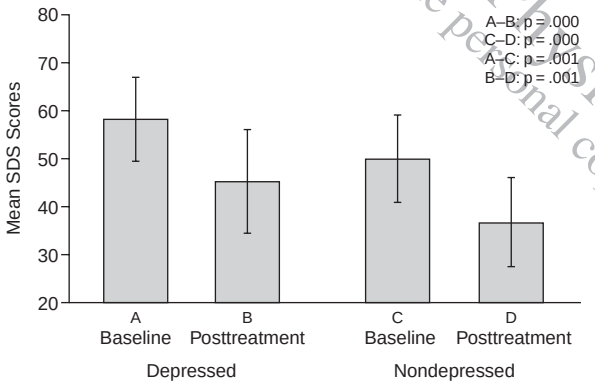
As for our first hypothesis, which defined comorbidity as coexistence of 2 separately defined psychiatric disorders according to full DSM-IV criteria, we found a point prevalence of depressive disorder of 37% in our clinical sample of OCD patients. This was well within the range

Figure 1. Mean MADRS Scores at Baseline and Posttreatment for Depressed (N = 25) and Nondepressed (N = 47) OCD Patients



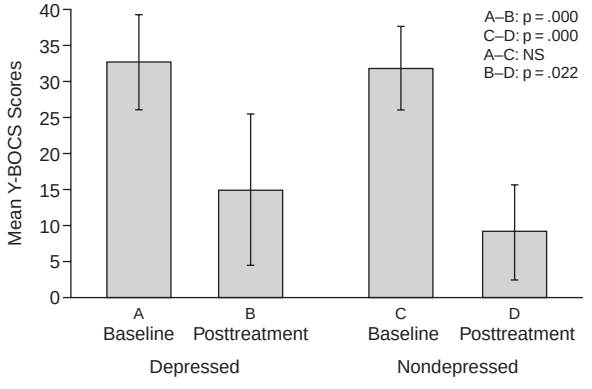
Abbreviations: MADRS = Montgomery-Asberg Depression Rating Scale, NS = nonsignificant, OCD = obsessive-compulsive disorder.

Figure 2. Mean SDS Scores at Baseline and Posttreatment for Depressed (N = 25) and Nondepressed (N = 47) OCD Patients



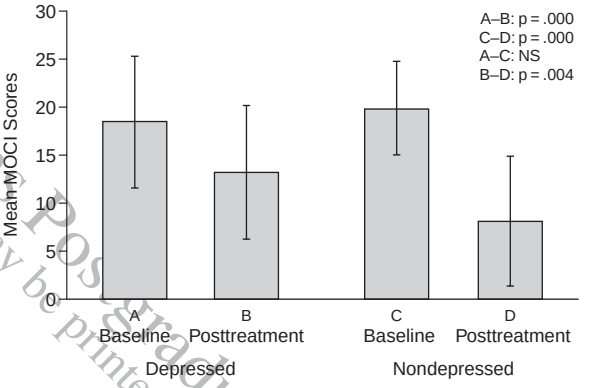
Abbreviations: OCD = obsessive-compulsive disorder, SDS = Self-Rating Depression Scale.

Figure 3. Mean Y-BOCS Scores at Baseline and Posttreatment for Depressed (N = 25) and Nondepressed (N = 47) OCD Patients



Abbreviations: NS = nonsignificant, OCD = obsessive-compulsive disorder, Y-BOCS = Yale-Brown Obsessive Compulsive Scale.

Figure 4. Mean MOCI Scores at Baseline and Posttreatment for Depressed (N = 25) and Nondepressed (N = 47) OCD Patients



Abbreviations: MOCI = Maudsley Obsessive Compulsive Inventory, NS = nonsignificant, OCD = obsessive-compulsive disorder.

we expected on the basis of earlier cited clinical studies.[15,26] However, it did not support the assumption that the majority of OCD patients only seek help when depressed, since 37% is not much higher than most population-based epidemiologic studies[17] have reported for concurrent depression. The mean MADRS score for the total group of depressed OCD patients in the present study appeared rather low (mean ± SD = 21.9 ± 9.6), reflecting depressive disorder of only moderate severity. Moderate depression would seldom be a reason to seek help, since depressive symptoms are easily accepted as a rather normal reaction to impairment by OCD, whereas severe levels of depression interfere more with daily life and affect the quality of life to a greater extent.

It was interesting to find that there was a significant difference in gender distribution in relation to comorbid depression. There were more females with comorbid OCD and depression in the ratio of 3.2:1 in our sample. This was higher than the known (epidemiologic) ratio of 2:1 female:male ratio for depressive disorder alone. A higher ratio for depression in OCD has, to our knowledge, not been reported in other clinical studies and should be interpreted with caution. In the first place, there are reported general differences in treatment-seeking behavior between males and females. Secondly, few studies report on gender differences in OCD in relation to depressive disorder, and in these the additional risk for females did not exceed the reported epidemiologic gender distribution for depressive disorder. Female relatives of OCD patients seem to have a higher risk of developing depressive disorder (but not OCD) than male relatives.[27] Another study by Castle et al.[28] explicitly addressed gender differ-

ences in OCD and found that more female OCD patients were taking antidepressant medication or had a past history of treatment for a depression than male OCD patients, but the distribution was in line with the gender ratio for depression alone.

Our second hypothesis, that OCD patients with comorbid depression tend to show more severe symptomatology than nondepressed OCD patients, could not be confirmed for scores on the specific OCD rating scales Y-BOCS and MOCI. Depressed patients, however, did show more severe symptoms on the general anxiety scales CAS, SAS, and STAI-1. Unfortunately, these scales are known to measure not only specific symptoms of anxiety disorders but also nonspecific feelings of stress and show overlap with depressive symptoms.[29] As could be expected, the specific depression assessments MADRS and SDS showed significantly higher scores for the depressed patients. So, the most interesting finding regarding symptom severity was that baseline values of the specific OCD rating scales MOCI and Y-BOCS did not differ significantly between depressed and nondepressed OCD patients. There are at least 2 possible explanations for this. The first possibility is that OCD and depressive disorder are 2 separate diagnostic entities,[30] and thus no correlation between depression and OCD scales would be expected. A second explanation could be that the scores on the MOCI and Y-BOCS in our sample were quite high from the start, so there was not much room for significant variation (ceiling effect) and presumably more suffering could be reflected only in the other, more general anxiety-related scales. In any case, with regard to symptom severity, we can conclude that comorbid, depressed OCD patients showed more severe (general) anxiety and depressive symptoms than nondepressed OCD patients, but their baseline level of OCD symptoms was the same.

As for the third hypothesis on treatment effect, we were able to confirm that both depressed and nondepressed OCD patients profit from the combined treatment offered for OCD. Both groups improved significantly during therapy with a reduction of more than 50% on initial scores on the MADRS, Y-BOCS, and CAS. The scores on the SDS, MOCI, STAI-1, and SAS also showed significant decreases of between 25% and 50%. It is interesting to note that the measurements that show the best improvement are investigator-rated scales, whereas the self-rating instruments show a smaller range of improvement. The phenomenon that clinician-scored measures often show more improvement than do the self-administered questionnaires has been described before.[31]

Although the baseline scores for the depressed subgroup did not differ on the OCD measures, after treatment the depressed OCD patients scored significantly higher on the Y-BOCS and MOCI than did the nondepressed OCD patients, reflecting more remaining symptoms. Also, on the general self-report anxiety scales SAS and STAI-1, after treatment the depressed patients still had higher symptom severity than the nondepressed ones. This finding, that the residual scores on several scales posttreatment were higher for the depressed subgroup, did point to the fact that a combined occurrence of obsessive-compulsive and affective symptoms showed more resistance to treatment or complicated treatment. Treatment duration, however, was similar in both groups.

The frequent co-occurrence of depression in OCD has formerly led to hypotheses about OCD being an affective variant.[32] This is partly supported by the therapeutic benefit of antidepressant agents for OCD.[33] Initial studies mention that levels of depression at the outset are important for the final outcome.[34] However, later meta-analytic studies have shown that the beneficial effects of antidepressants such as clomipramine and of SSRIs on OCD symptoms are unrelated to the presence of depressive symptoms,[18,33,35] which again undermines this hypothesis and implies another relationship between the 2 disorders. Hollander et al.[36] state that depressive symptoms interfere with the response to both psychopharmacologic and behavioral treatments of OCD. Also, Foa[37] reported behavioral therapy outcome to be negatively influenced by the presence of (severe) depressive disorder.

Montgomery[38] argues that depression seen in OCD should be regarded as part of OCD and not as a separate disorder of major depression, for depressive symptoms in OCD can, in his view, be expected to respond only to effective antiobsessional treatments and not to noradrenergic antidepressive agents. Delgado and Moreno[30] emphasize the different etiology and pathophysiology of OCD and depression, as implicated by tryptophan depletion studies and neuroimaging. Although there may be some shared elements or contributions of neural networks, there is no complete overlap. One may generally conclude from these pharmacologic, depletion, and imaging data that, although linked, OCD and major depression are 2 different psychopathologic entities that, if diagnosed carefully, require different therapeutic strategies. These data support the notion that it is more difficult to treat 2 psychiatric disorders at a time than 1. This is partly confirmed by the present study, as more severe symptoms remained in the initially depressed patient group than in the nondepressed group.

Some shortcomings of this study must be mentioned. First, the nature of the study imposed restrictions on the interpretation of our findings, as retrospective studies always do, even though we used a semistructured interview and DSM-IV criteria and always required diagnostic agreement from 2 clinicians. Second, as described above, some of our rating instruments were self-rated scales and, as such, they were more liable to subjective interpretation and were less reliable. Third, we had posttreatment data on only 72 of the 120 patients we examined at baseline. We were unable to trace all subjects for

various reasons. Patients sometimes ended their therapy prematurely if there was no clear or rapid effect; others were doing well after treatment and did not show up for posttreatment assessments. What we do know, however, is that the demographics and clinical values at baseline did not differ between the group of completers and the patients we assessed only at baseline. Thus, it was not specifically the most depressed or severely ill patients who were lost to follow-up; on the contrary, for example, within the nondepressed subgroup, the completers showed more severe symptoms at baseline assessments.

At the same time, one should see the above shortcomings in relation to the strengths of this study, which are the clear-cut definition of comorbidity and its naturalistic nature. Defining comorbidity as current, coexisting DSM-IV–defined syndromes is one of the most reliable assessments of comorbid conditions. Patients' reports of past lifetime events have never been proven to be very reliable; the only way to counter this is to do longitudinal research on large patient groups, and this design has its own drawbacks. An advantage of naturalistic studies like this one is, in our opinion, that the results are directly derived from general clinical practice, which is relevant for patients in the clinical setting. Diagnosing comorbid conditions is of great importance since they obviously have an impact on treatment outcome.

Drug names: clomipramine (Anafranil), fluoxetine (Prozac and others), fluvoxamine (Luvox and others), paroxetine (Paxil).

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