Effectiveness of Pharmacotherapy for Body Dysmorphic Disorder: A Chart-Review Study

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Background: Research on the pharmacotherapy of body dysmorphic disorder (BDD) is limited. No placebo-controlled, continuation, maintenance, or discontinuation studies have been published. Only one augmentation study has been published.

Method: In this chart-review study of 90 patients with DSM-IV BDD treated for up to 8 years by the first 2 authors (K.A.P., R.S.A.) in their clinical practice, response to a variety of medications, including augmentation strategies, was assessed. The relapse rate with medication discontinuation was also determined.

Results: All subjects received a serotonin reuptake inhibitor (SRI), with 63.2% (55/87) of adequate SRI trials resulting in improvement in BDD symptoms; similar response rates were obtained for each type of SRI. Discontinuation of an effective SRI resulted in relapse in 83.8% (31/37) of cases. Response rates to selective SRI augmentation were clomipramine, 44.4% (4/9) of trials; buspirone, 33.3% (12/36) of trials; lithium, 20.0% (1/5); methylphenidate, 16.7% (1/6); and antipsychotics, 15.4% (2/13) of trials.

Conclusion: These findings from a clinical setting suggest that a majority of BDD patients improve with an SRI and that all SRIs appear effective. Certain SRI augmentation strategies may be beneficial. The high relapse rate with SRI discontinuation suggests that long-term treatment is often necessary. These preliminary findings require confirmation in placebo-controlled efficacy studies and effectiveness studies.

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Research on the pharmacotherapy of body dysmorphic disorder (BDD) is increasing but remains limited. Obtaining information about effective treatment is important, since this disorder is relatively common,[1–3] causes severe distress and functional impairment,[4–6] and is associated with a high suicide attempt rate and markedly poor quality of life.[5,7] In the dermatology[8,9] and psychiatry[10] literature, BDD has been said to be extremely difficult to treat.

Recent data suggest that BDD, including its delusional variant, may respond to serotonin reuptake inhibitors (SRIs) but not to other medications or electroconvulsive therapy. This evidence comes from case series in adults,[5,11,12] children,[13] and adolescents[13] as well as 2 open-label trials[14,15] of fluvoxamine in adults. In a 10-week open-label fluvoxamine study,[14] 10 of 15 patients improved, and in a 16-week open-label fluvoxamine trial,[15] 63% of 30 patients improved. A double-blind crossover study[16] (N = 40) recently reported that the SRI clomipramine was more effective than the non-SRI desipramine. Published reports of augmentation strategies are limited to 1 case series[17] in which buspirone augmentation of SRIs was effective in 6 (46%) of 13 patients.

Systematic research on the response of BDD to SRIs and SRI augmentation strategies is limited to the above studies, and no placebo-controlled, continuation, maintenance, or discontinuation studies of any type of pharmacotherapy have been published. Furthermore, the likelihood of response to a subsequent SRI after failure to respond to an initial SRI is unknown.

This chart-review study addressed a number of previously uninvestigated questions, including the following: (1) What are response rates to the different SRIs, and are there any predictors of response? (2) What is the response rate to a subsequent SRI after failure to respond to an initial SRI? (3) What is the relapse rate after SRI discontinuation? (4) What is the response rate with various SRI augmentation strategies? and (5) Is an SRI augmentation strategy more likely to be effective if the SRI trial was partially effective?

METHOD

Subjects

This chart-review study included 90 outpatients (69 adults [76.7%], 21 adolescents [23.3%]; 61.1% female [N = 55]; mean ± SD age = 30.1 ± 11.5 years; range, 12–65 years) with DSM-IV BDD. All patients were treated by the first (K.A.P.) or second (R.S.A.) author in their clinical outpatient practice at a private psychiatric hospital (mean duration of treatment = 1.8 ± 1.8 years; range, 0.1–8.4 years). Only medications that were begun by these authors outside of a treatment study were included in the present study, i.e., clinical trial data and data on medications begun during a clinical trial were excluded.

All subjects met DSM-IV criteria for BDD: (A) preoccupation with an imagined defect in appearance; if a slight physical anomaly is present, the person's concern is markedly excessive; (B) the preoccupation causes clinically significant distress or impairment in social, occupational, or other important areas of functioning; and (C) the preoccupation is not better accounted for by another mental disorder (e.g., dissatisfaction with body shape and size in anorexia nervosa). Patients with delusional BDD were included in the study because available data suggest that the delusional and nondelusional forms of BDD are variants of the same disorder,[18] and DSM-IV allows them to be double coded. At the initial assessment, 38.0% (27/71) of subjects had appearance-related beliefs that were currently delusional. The most common current comorbid disorders at the baseline assessment were major depression (65.7% [46/70]), obsessive-compulsive disorder (OCD; 34.3% [24/70]), and social phobia (32.9% [23/70]).

Evaluation of Treatment Effectiveness

Information was obtained from charts on all pharmacotherapy received, including medication type, maximum dose, and trial duration. Effectiveness was rated at the beginning and end of each medication trial, and results are reported for individual medication trials. Only those trials that were considered "minimally adequate" were rated for effectiveness. Criteria for a minimally adequate trial were based on available literature as well as clinical experience, since empirical data on this issue are limited, especially for augmentation strategies. The following daily SRI doses were considered minimally adequate: fluvoxamine, 150 mg; fluoxetine, 40 mg; paroxetine, 40 mg; sertraline, 150 mg; and clomipramine, 150 mg. For augmentation strategies, minimally adequate daily doses were buspirone, 30 mg; olanzapine, 5 mg; risperidone, 2 mg; a chlorpromazine equivalent of 100 mg for typical antipsychotics; methylphenidate, 10 mg; and a therapeutic blood level of clomipramine (a minimum of 200 μg/L) or lithium (a minimum of 0.5 mEq/L). A minimally adequate trial duration for SRIs was 10 weeks and for augmentation strategies, 6 weeks, except that a minimum of 10 weeks was required for augmentation of a selective SRI (SSRI) with clomipramine or vice versa. SRI augmentation trials were rated for effectiveness only if the augmenting medication was added to an adequate SRI trial. Data are not reported for benzodiazepine augmentation because benzodiazepines were added at varying times during treatment; 18.7% of SRI trials and 19.7% of augmentation trials were accompanied by benzodiazepine use. Relapse following SRI discontinuation was assessed if the SRI was completely discontinued for a minimum of 1 week. Reasons for medication discontinuation were obtained.

Information was obtained on psychotherapy received. Subjects were determined to have received cognitive-behavioral therapy (CBT) if the treatment included exposure, response prevention, or cognitive techniques focusing on BDD symptoms.

Assessments

Baseline assessments. Subjects were evaluated at baseline with the following instruments:

- Structured Clinical Interview for DSM-III-R (SCID-P). This reliable and widely used instrument[19,20] obtained information on demographic characteristics and comorbidity.
- Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS-PL). This reliable, valid, and widely used diagnostic instrument for children and adolescents[21] was used instead of the SCID-P for all children younger than 12 years.
- BDD Diagnostic Module. Because the DSM-III-R SCID-P does not include BDD, this disorder was diagnosed with a reliable semistructured SCID-like diagnostic instrument based on DSM-IV criteria for BDD.[22]
- BDD Form. This semistructured instrument (K.A.P., unpublished) obtained additional data on demographics and the clinical characteristics of BDD, such as age at BDD onset.
- Brown Assessment of Beliefs Scale. This reliable and valid 7-item semistructured clinician-administered scale assesses delusionality (insight) both dimensionally and categorically.[23] Ratings are available for the 25 subjects (38 medication trials) assessed after the scale was developed. For the remaining subjects, the delusionality of appearance-related beliefs was assessed at the baseline evaluation using item 11 (insight) of the BDD-YBOCS (see below) after this scale was developed.

Ratings of medication effectiveness. The following ratings were done at the beginning and/or end of each medication trial:

Clinical Global Impressions Scale for BDD (BDD-CGI). This 7-point scale assesses global improvement or worsening of symptoms, with ratings ranging from "very much worse" to "very much improved."[24] Much or very much improvement (score of 1 or 2) was defined as improvement in BDD, and much or very much worse (score of 6 or 7) was defined as relapse of BDD. Except where indicated, reported rates of improvement with treatment or worsening with medication discontinuation are based on the BDD-CGI.

Yale-Brown Obsessive Compulsive Scale Modified for Body Dysmorphic Disorder (BDD-YBOCS). This reliable and valid semistructured scale assesses current BDD severity with 12 clinician-administered items.[25] Items are rated on a scale of 0 (no symptoms) to 4 (extreme symptoms). In this study, only the first 3 items of the scale (which yield a total score of 0 to 12) were rated because information for these items was available in the patients' charts. These 3 items correspond to the DSM-IV diagnostic criteria for BDD and have good interrater reliability.[25]

Psychiatric Status Rating Scale for Body Dysmorphic Disorder (BDD-PSR). PSRs are disorder-specific, reliable, and valid global ratings of disorder severity.[26–28] A PSR for DSM-IV BDD was adapted from PSRs for mood and anxiety disorders.[26–28] The BDD-PSR is a 7-point scale that reflects whether BDD symptoms are currently "in episode" (i.e., meet full DSM-IV criteria for BDD; score of 5 to 7), in partial remission (score of 3 or 4), or in full remission (score of 1 or 2). The BDD-PSR has good interrater and test-retest reliability (ICC = 0.95 and 0.81, respectively) and good convergent validity (K.A.P., unpublished data).

Global Assessment of Functioning (GAF). The GAF is a widely used 90-item global measure of symptom severity and psychological, social, and occupational functioning.[29] Scores range from 0 to 90, with lower scores denoting more severe illness and poorer functioning.

Data Analysis

Split-plot factorial analyses of variance were computed for all SRI trials combined and all augmentation trials combined to identify medication, time (baseline vs. endpoint values), and interaction effects on each outcome measure (BDD-YBOCS, BDD-PSR, and GAF). The F value was computed for all SRI and augmentation trials from valid pairwise data only; because of some missing data, the number of pairwise trials that compare baseline and endpoint may be less than the total number of treatment trials. If the overall F value for all SRI trials or all augmentation trials was significant, F values were computed for individual medications. For analyses that yielded F values at $p < .10$, the effect size (partial η^2) for each medication was computed to obtain the degree of relationship with each outcome measure. A large association is generally considered as partial $\eta^2 = 0.14$ or greater[30] (d equivalent = 0.80). Power was calculated for analyses of change on the BDD-YBOCS, BDD-PSR, and GAF; power of 0.80 or higher is considered adequate. Correlations between selected variables were examined using the Pearson product-moment correlation coefficient.

RESULTS

All 90 subjects received psychotropic medication, with a total of 228 medication trials (mean ± SD = 2.6 ± 2.1; range, 1–10 trials per subject) and 174 adequate trials (mean ± SD = 2.0 ± 1.7; range, 0–7 adequate trials per subject) received. All subjects received an SRI (1.6 ± 1.1; range, 1–6 SRI trials per subject). Of the 78 subjects with an SRI trial initiated by the investigators, 80.8% (63/78) received at least one adequate SRI trial (1.4 ± 1.1; range, 0–5 adequate trials per subject). SRI augmentation was received by 44.4% (40/90) of subjects (2.2 ± 1.3; range, 1–5 trials per subject), and 43.3% (39/90) of subjects received at least one adequate SRI augmentation trial (2.0 ± 1.2; range, 1–5 adequate trials per subject).

Table 1 shows doses, trial durations, and outcomes for adequate SRI trials. Effect sizes on the BDD-YBOCS ranged from 0.44 (for sertraline) to 0.79 (for fluvoxamine), and on the BDD-PSR, they ranged from 0.45 (for sertraline) to 0.83 (for fluvoxamine). Effect sizes on the GAF ranged from 0.51 (for clomipramine) to 0.71 (for fluvoxamine). (Effect sizes for all of these analyses were large; power for analyses of all SRIs combined and for fluoxetine was greater than 0.90, for analyses of fluvoxamine it was greater than 0.65, and for all other analyses it was less than 0.55.) Improvement on the BDD-CGI occurred with 63.2% (55/87) of adequate SRI trials, with similar response rates for each type of SRI. On the BDD-PSR, 17.5% (11/63) of adequate SRI trials resulted in full remission and 31.7% (20/63) in partial remission. SRI trials were as effective in delusional as in nondelusional subjects (54.5% [18/33] vs. 75.7% [28/37], $\chi^2 = 3.5$, df = 1, $p = .06$). Two of 5 venlafaxine trials and 0 of 2 nefazodone trials were effective. (Three additional nefazodone trials were discontinued early because of worsening or persistence of severe symptoms.)

Of those subjects who failed an initial adequate SRI trial, 42.9% (6/14) responded to at least one subsequent adequate SRI trial, and 43.5% (10/23) of subsequent adequate SRI trials received by these subjects were effective. Among responders to an initial SRI who were subsequently treated with a different SRI, 92.3% (12/13) of subsequent trials also led to improvement.

Table 1. Doses, Trial Duration, and Outcome for SRIs[a]

Variable	All SRIs (85 trials)[b]	Fluoxetine (38 trials)	Paroxetine (14 trials)	Fluvoxamine (6 trials)	Sertraline (12 trials)	Clomipramine (15 trials)
Maximum dose, mg/d, mean ± SD	...	66.7 ± 23.5	55.0 ± 12.9	308.3 ± 49.2	202.1 ± 45.8	203.3 ± 52.5
Trial duration, wk, mean ± SD	38.3 ± 33.8	35.8 ± 27.1	51.7 ± 47.3	79.4 ± 51.3	36.5 ± 20.7	24.1 ± 16.3
BDD-CGI score at endpoint, mean ± SD	2.3 ± 1.1	2.3 ± 1.2	2.4 ± 0.7	2.3 ± 0.5	2.6 ± 1.1	2.3 ± 1.2
BDD-YBOCS						
F [ES (N)][c]	57.7‡ [0.60 (39)]	51.9‡ [0.75 (17)]	6.9 [0.77 (3)]	14.7** [0.79 (5)]	5.5* [0.44 (8)]	6.4* [0.62 (5)]
Baseline	8.6 ± 2.0	8.3 ± 1.9	9.0 ± 1.9	7.0 ± 2.5	9.5 ± 1.6	9.6 ± 1.9
Endpoint	4.6 ± 3.5	3.7 ± 3.2	6.0 ± 2.3	5.3 ± 2.7	6.9 ± 3.8	3.2 ± 3.7
BDD-PSR						
F [ES (N)][c]	69.8‡ [0.55 (57)]	49.5‡ [0.66 (26)]	5.0* [0.56 (4)]	16.0** [0.83 (6)]	7.31† [0.45 (10)]	10.0** [0.53 (10)]
Baseline	5.9 ± 0.7	5.8 ± 0.6	5.8 ± 0.6	5.4 ± 0.9	6.2 ± 0.6	5.8 ± 0.9
Endpoint	4.1 ± 1.7	3.7 ± 1.7	5.0 ± 0.9	4.8 ± 1.3	5.1 ± 1.4	3.8 ± 2.0
GAF						
F [ES (N)][c]	77.5‡ [0.56 (60)]	60.9‡ [0.70 (26)]	5.6** [0.55 (8)]	9.8** [0.71 (4)]	7.8** [0.53 (8)]	10.2† [0.51 (11)]
Baseline	46.5 ± 9.9	46.9 ± 8.7	45.7 ± 9.0	54.2 ± 9.0	43.2 ± 10.1	45.4 ± 13.1
Endpoint	62.4 ± 17.6	57.2 ± 17.4	64.5 ± 17.0	64.0 ± 15.9	52.3 ± 18.0	57.4 ± 16.6

[a]Abbreviations: BDD-CGI = Clinical Global Impressions Scale for Body Dysmorphic Disorder, BDD-PSR = Psychiatric Status Rating Scale for Body Dysmorphic Disorder, BDD-YBOCS = Yale-Brown Obsessive Compulsive Scale Modified for Body Dysmorphic Disorder, GAF = Global Assessment of Functioning, SRI = serotonin reuptake inhibitor.
[b]The number for all SRI trials is 85 because 2 citalopram trials were excluded from this table.
[c]A split-plot factorial analysis of variance, (F) SRI by time (baseline-endpoint), was computed across all groups for each outcome measure (BDD-YBOCS, BDD-PSR, GAF). No differences were found between groups; however, time effects were found within groups. Follow-up repeated measures analyses of variance were performed to determine individual group time effects. Effect sizes (ES), partial eta squared, are provided as an estimation of each effect's strength. Number of paired trials (N) is indicated where there were missing data. Results are presented as F [ES (N)] where F = the F statistic, ES = effect size (partial eta squared), N = number of trials. The F value was computed from valid pairwise data only and may not represent the baseline and endpoint mean ± SD.
*p ≤ .10.
**p ≤ .05.
†p ≤ .01.
‡p ≤ .001.

CBT was received by 26.4% (19/72) of subjects and accompanied 28.6% (20/70) of adequate SRI trials (4 subjects had 2 SRI trials each); SRI response rates were similar for trials that were and were not accompanied by CBT (63.2% vs. 67.3%, respectively; p = .74). Supportive or insight-oriented psychotherapy was received by 43.2% (32/74) of subjects and accompanied 44.1% (30/68) of adequate SRI trials; SRI response rates were similar for trials that were and were not accompanied by psychotherapy (67.7% vs. 67.6%, p = .99). SRI response was significantly positively associated with the following baseline variables: milder BDD symptoms (r = 0.25, df = 69, p = .04), the presence of current OCD (r = 0.24, df = 69, p = .05) or social phobia (r = 0.24, df = 69, p = .04), and a lesser degree of delusionality (r = 0.24, df = 69, p = .05). No association was found between SRI response and the presence of current major depression, duration of BDD, or gender.

Discontinuation of an effective SRI resulted in relapse of BDD in 83.8% (31/37) of cases. Reasons for discontinuation were the patient's desire to be medication free (37.8% [14/37]), side effects (29.7% [11/37]), miscellaneous (13.5% [5/37]), cost (8.1% [3/37]), and unknown (10.8% [4/37]).

Table 2 provides information on doses, trial durations, and outcome for adequate SRI augmentation trials. Response rates for augmenting medications were clomipramine, 44.4% (4/9); buspirone, 33.3% (12/36); lithium, 20.0% (1/5); methylphenidate, 16.7% (1/6); and antipsychotics, 15.4% (2/13). Effect sizes were large for all analyses except for the clomipramine BDD-YBOCS analysis, which was medium to small (power was greater than 0.80 for only analyses of buspirone and all SRIs combined). Augmentation was more likely to be effective when the augmenting agent was added to a partially effective SRI as opposed to an ineffective SRI (40.5% [15/37] vs. 18.2% [6/33]; $\chi^2 = 4.0$, df = 1, p = .04). Response rates were similar for augmentation trials that were and were not accompanied by CBT (20.0% vs. 29.2%, p = .43) or psychotherapy (29.5% vs. 24.0%, p = .87). Of those subjects who failed an initial SRI augmentation trial, 16 received a total of 24 subsequent augmentation trials with the same SRI, 33.3% (N = 8) of which were effective.

Three fourths (74.7% [56/75]) of subjects responded to at least one adequate SRI or augmentation trial; 25.3% (19/75) failed all treatment trials that they received.

DISCUSSION

Our finding that SRIs are often effective for BDD, and that all SRIs appear effective, extends published data on the efficacy of fluvoxamine and clomipramine. The effect

Table 2. Doses, Trial Duration, and Outcome for Augmenting Agents[a]

Variable	All Augmentation (76 trials)	Buspirone (36 trials)	Atypical Antipsychotics (9 trials)	Typical Antipsychotics (6 trials)	Methylphenidate (6 trials)	Lithium (5 trials)	Clomipramine (9 trials)
Maximum dose, mg/d, mean ± SD	...	56.5 ± 15.2	...	...	31.7 ± 6.8	825.2 ± 567.6	128.3 ± 76.4
Trial duration, wk, mean ± SD	30.7 ± 54.8	34.2 ± 72.5	16.7 ± 13.4	23.5 ± 26.2	47.8 ± 49.7	32.9 ± 42.2	41.3 ± 47.2
BDD-CGI score at endpoint, mean ± SD	3.2 ± 1.0	3.1 ± 1.0	3.4 ± 1.0	3.7 ± 0.5	3.2 ± 1.2	3.2 ± 0.8	3.1 ± 1.2
BDD-YBOCS							
F [ES (N)][b]	6.3‡ [0.16 (47)]	12.6† [0.39 (21)]	2.9 [0.32 (7)]	NA[c] (2)	2.7 [0.47 (4)]	4.0 [0.67 (3)]	0.4 [0.02 (4)]
Baseline	7.0 ± 3.0	6.0 ± 2.9	8.9 ± 2.9	5.0 ± 1.4	6.0 ± 3.1	6.5 ± 2.7	7.1 ± 2.3
Endpoint	5.9 ± 3.5	4.8 ± 3.1	7.8 ± 3.5	4.0 ± 1.4	4.5 ± 3.1	6.3 ± 3.5	6.3 ± 3.1
BDD-PSR							
F [ES (N)][b]	8.3‡ [0.16 (59)]	12.1† [0.32 (27)]	3.4 [0.30 (4)]	NA[c] (4)	2.0 [0.40 (4)]	1.0 [0.33 (3)]	0.6 [0.17 (5)]
Baseline	5.3 ± 1.2	4.9 ± 1.1	5.6 ± 1.3	5.3 ± 1.0	5.0 ± 1.2	5.0 ± 0.8	5.8 ± 1.1
Endpoint	4.7 ± 1.5	4.2 ± 1.5	5.1 ± 1.7	5.3 ± 1.0	4.3 ± 1.7	4.7 ± 1.2	5.4 ± 1.1
GAF							
F [ES (N)][b]	15.3‡ [0.23 (68)]	13.0† [0.32 (29)]	2.5 [0.24 (4)]	2.6 [0.39 (5)]	3.2 [0.39 (4)]	2.9 [0.49 (4)]	1.3 [0.31 (5)]
Baseline	49.9 ± 15.4	54.9 ± 13.6	42.8 ± 14.2	45.8 ± 14.9	57.5 ± 12.9	50.0 ± 13.2	37.0 ± 19.6
Endpoint	54.4 ± 18.0	61.1 ± 15.7	45.6 ± 16.4	47.2 ± 16.8	65.2 ± 12.4	52.0 ± 13.4	47.2 ± 15.3

[a]Abbreviations: BDD-CGI = Clinical Global Impressions Scale for Body Dysmorphic Disorder, BDD-PSR = Psychiatric Status Rating Scale for Body Dysmorphic Disorder, BDD-YBOCS = Yale-Brown Obsessive Compulsive Scale Modified for Body Dysmorphic Disorder, GAF = Global Assessment of Functioning.
[b]A split-plot factorial analysis of variance, (F) augmentation by time (baseline-endpoint), was computed across all groups for each outcome measure (BDD-YBOCS, BDD-PSR, GAF). No differences were found between groups; however, time effects were found within groups. Follow-up repeated measures analyses of variance were performed to determine individual group time effects. Effect sizes (ES), partial eta squared, are provided as an estimation of each effect's strength. Number of paired trials (N) is indicated where there were missing data. Results are presented as F [ES (N)] where F = the F statistic, ES = effect size (partial eta squared), N = number of trials. The F value was computed from valid pairwise data only and may not represent the baseline and endpoint mean ± SD.
[c]No F value can be computed owing to equal variances.
†$p \leq .01$.
‡$p \leq .001$.

sizes for all SRIs were large. Of note, a substantial percentage of subjects who failed an initial SRI responded to a subsequent SRI, suggesting that sequential SRI trials may be beneficial for unresponsive patients. Patients who responded to an initial SRI were highly likely to respond to a subsequent SRI.

Because SRI treatment more often resulted in partial remission than full remission, the effectiveness of SRI augmentation strategies is of interest and importance. Augmentation has particular appeal when trying to avoid the relapse that could occur when switching to another SRI. It is notable that augmentation was more successful when patients had partially responded to an SRI, as was found in the only published augmentation study in BDD.[17] It is unclear whether this improvement was due to the augmentation agent itself or to increased efficacy of the SRI over time, although patients had received an SRI for a mean duration of 1.2 ± 1.5 years (range, 0.2–8.2 years) and a median duration of 8.0 months before an augmenting agent was added, suggesting that the augmenting agent may have been largely responsible for further improvement in BDD symptoms.

All augmentation trials combined resulted in a large effect size. The effect size with buspirone augmentation was large, and the response rate (33.3%), although somewhat lower than in a previous smaller study,[17] suggests that this strategy may be worth trying, especially given buspirone's good tolerability. For other augmentation agents, calculation of effect sizes was compromised by the small number of treatment trials. In terms of response rate, clomipramine augmentation of an SSRI or vice versa appeared most effective. This combination was generally well tolerated but should be used with caution, given the relatively narrow therapeutic index of clomipramine and the potential for SSRIs to increase blood clomipramine levels; blood clomipramine levels should be carefully monitored when using this approach.

The results for antipsychotic augmentation are surprising, given that 38% of BDD subjects were currently delusional. However, the number of trials was small, limiting the conclusions that can be drawn. Although SRI augmentation with lithium or methylphenidate was effective for BDD in only a minority of cases, these approaches significantly improved severe depressive symptoms in a number of patients. These preliminary augmentation results suggest that the treatment response of BDD may differ somewhat from that of OCD[31] as well as nonpsychotic and psychotic depression,[32,33] disorders that have been hypothesized to be related to BDD.[34]

An intriguing result is that delusional subjects were as likely as nondelusional subjects to respond to SRIs, consistent with previous reports on BDD.[11,13,15,16] This finding

suggests that psychosis is a heterogeneous construct, with some types requiring antipsychotics and other types (e.g., delusions in BDD) responding to SRIs alone. However, our correlational analysis indicated that a lesser degree of delusionality was significantly associated with better SRI response; this finding contrasts with results from a previous study[16] in which the more delusional the patient was, the more he or she improved with clomipramine treatment. Additional studies using more rigorous methodology are needed to further investigate this issue.

Given preliminary evidence supporting the efficacy of CBT for BDD,[35,36] it is interesting that receipt of CBT was not associated with a higher response rate to medication. It is less surprising that concomitant insight-oriented or supportive psychotherapy was not associated with a higher medication response rate, since available data, while limited, suggest that this treatment is generally ineffective for BDD.[5] Our patients usually received psychotherapy as an adjunctive treatment that was intended, for example, to help them cope with BDD symptoms or address problematic personality traits or stressful life situations.

The high relapse rate with SRI discontinuation is notable, especially given that the analysis included SRIs that were discontinued for as briefly as 1 week. The relapse rate is similar to that seen in a number of other often-chronic psychiatric disorders, such as OCD.[37] This preliminary finding suggests that long-term SRI treatment is often needed to maintain improvement. It is our clinical impression that response of BDD is usually maintained or even further enhanced over time with continuation of effective medication, although this hypothesis requires investigation.

This study has a number of limitations. Most importantly, it was an uncontrolled chart-review study in which ratings were obtained retrospectively, raters were not blind to the treatment received, and some data were missing. Due to missing data, the number of subjects included in analyses varied for different variables, which may have influenced effect sizes. Also because of missing data and the small sample size, the study had limited power to assess the effectiveness of certain treatments (e.g., certain augmentation strategies). Another consequence of missing data is that we did not include analyses of response of comorbid disorders (which were common) versus that of BDD, leaving unanswered the question of whether improvement in BDD might be secondary to that of comorbid conditions (although data from other studies suggest that this is unlikely to be the case).[13,15] Furthermore, the charts contained inadequate data to address certain questions, such as time to relapse after medication discontinuation.

An additional limitation of the study is that the doses and durations used for an adequate medication trial may not have been optimal, since dose-finding studies have not been done in BDD, and the minimum duration of an adequate trial is not known with certainty (for example, the choice of 10 weeks for a minimally adequate SRI trial may have been too brief and thus resulted in an underestimation of SRI response rates). In addition, the generalizability of the study findings may be limited by the fact that patients were treated in an academic private hospital setting by clinicians with expertise in BDD. On the other hand, the fact that subjects were treated in a clinical setting and, unlike in efficacy studies, were not excluded because of the presence of prominent suicidality, comorbid disorders such as substance abuse or dependence, or other factors may make the results more generalizable.

Despite these limitations, this study—the first to investigate the response of BDD to a variety of pharmacotherapeutic approaches—suggests that a majority of patients treated in a clinical practice setting improve, although improvement is often partial. More methodologically rigorous treatment studies are clearly needed. Particularly needed are placebo-controlled studies of both single agents and augmentation strategies, continuation and maintenance treatment studies, placebo-controlled discontinuation studies, and studies of combined pharmacotherapy and psychotherapy. Although the mean SRI doses used in this study were fairly high, dose-finding studies are needed to ascertain the most effective SRI doses for BDD. Such studies will provide more definitive information on the treatment of this relatively common, distressing, and often-disabling disorder.

Drug names: desipramine (Norpramin and others), fluoxetine (Prozac and others), fluvoxamine (Luvox and others), methylphenidate (Ritalin and others), nefazodone (Serzone), olanzapine (Zyprexa), paroxetine (Paxil), risperidone (Risperdal), sertraline (Zoloft), venlafaxine (Effexor).

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