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Social anxiety in body dysmorphic disorder

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Abstract

Although clinical impressions suggest that patients with body dysmorphic disorder (BDD) experience distress in social situations, social anxiety in BDD has received little investigation. This study examined social anxiety in 81 patients with BDD and change in social anxiety with pharmacotherapy. Subjects completed the Social Avoidance and Distress Scale (SADS) and were assessed with measures of BDD symptomatology. Participants in a placebo-controlled fluoxetine trial completed measures at baseline and endpoint. The mean SADS score was 1.3 SD units higher than nonclinical sample means but consistent with other clinical sample means. Social anxiety was significantly correlated with BDD severity. Greater depressive symptoms as well as comorbid avoidant personality disorder, but not comorbid social phobia, were also associated with higher SADS scores. Social anxiety did not improve more with fluoxetine than placebo, yet it improved significantly more in fluoxetine responders than in nonresponders. Understanding social anxiety in BDD has implications for reducing rates of misdiagnosis and treatment dropout.

Keywords

Body dysmorphic disorder; Social anxiety; Avoidance; Pharmacotherapy; Social phobia; Avoidant personality disorder

Introduction

While patients with body dysmorphic disorder (BDD) experience impairment across various life domains (Phillips, McElroy, Keck, Pope, & Hudson, 1993), social functioning may be particularly affected. Though data are limited, research suggests that individuals with BDD have significantly greater social anxiety than nonclinical controls (Veale, Kinderman, Riley, & Lambrou, 2003). Social anxiety, defined as anxiety due to a concern about how one will be perceived by others (Leary & Kowalski, 1995), includes anticipatory anxiety, cognitive/somatic symptoms in social situations, and avoidance behavior when distress is chronic. Though most commonly associated with social phobia (SP) and avoidant personality disorder (AVPD), social anxiety is a general construct thought to be present in various psychiatric conditions.

An individual's perception of his/her physical appearance has been linked to social anxiety; individuals who perceive themselves as unattractive (but do not necessarily have BDD) tend to have more social anxiety (Leary & Kowalski, 1995). Further, negative body image is related to greater social introversion (Archer & Cash, 1985) and public self-consciousness (Theron, Nel, & Lubbe, 1991).

Available data and clinical impressions suggest that avoiding social situations or enduring them with great distress is a prominent feature of BDD (Phillips et al., 1993). Veale et al. (1996)

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reported high scores on a self-report measure of social phobia and anxiety symptoms in 50 participants with BDD. Compared to individuals with an eating disorder and nonclinical controls, subjects with BDD (n = 51) reported more avoidance of activities due to self-consciousness about appearance (Rosen & Ramirez, 1998). In a study examining interpretation of ambiguous scenarios, subjects with BDD (n = 61) were more likely than controls or subjects with obsessive compulsive disorder to interpret social scenarios as threatening (Buhlmann et al., 2002).

Comorbidity studies point to an overlap between BDD and other conditions in which social anxiety is a prominent feature. Studies using structured interviews have reported a high lifetime prevalence of comorbid SP in BDD, e.g., 37% of 293 subjects in Gunstad and Phillips (2003). Wilhelm, Otto, Zucker, and Pollack (1997) showed a high prevalence of BDD (12%) among patients with a primary diagnosis of SP. Similarly, AVPD is the most common personality disorder in patients with BDD (Phillips & McElroy, 2000).

Only two published studies have examined change in social anxiety with BDD treatment. In the first, a chart-review study (n = 6) (Hollander et al., 1994), social anxiety decreased as BDD symptoms improved with fluvoxamine treatment. However, quantitative data on social anxiety were not reported. In a pilot study of cognitive behavioral therapy (CBT) in BDD (Veale et al., 1996), a difference in SP symptomatology (measured by self-report) was noted between, but not within, patients who received CBT (n = 9) versus those on a waiting list (n = 10).

The present study examined social anxiety and its correlates in patients with BDD. To our knowledge, this is the first investigation to examine pharmacotherapy effects on social anxiety in BDD using a standard measure. This topic is important because social anxiety may compound other functional impairment and deter patients from seeking treatment. Based on the literature and our clinical experience, we hypothesized that patients with BDD would have high levels of social anxiety, compared to normative data, and that greater social anxiety would be related to more severe BDD, more severe depressive symptoms, and greater delusionality of appearance-related beliefs. We were also interested in the relationship between social anxiety and comorbid SP and AVPD. Finally, we hypothesized that social anxiety would improve more with fluoxetine than placebo.

Method

Participants

Subjects were 81 outpatients with current DSM-IV BDD. Fifty-seven (70%) were female, and the mean age was 31.5 years (SD=10.7, range = 17–62). Subjects were referred from various sources to a BDD clinical research program where they all entered BDD pharmacotherapy studies. Sixty-six (81%) subjects were randomized in a double-blind, placebo-controlled trial of fluoxetine, and 15 (19%) participated in an open-label citalopram study. The pharmacotherapy studies, described elsewhere (Phillips, Albertini, & Rasmussen, 2002; Phillips & Najjar, 2003), had standard exclusion criteria, including lifetime schizophrenia or bipolar disorder, current or recent substance use disorder, or clinically significant suicidality. Written informed consent was obtained from all subjects, and the studies were approved by the hospital Institutional Review Board.

Measures

The Social Avoidance and Distress Scale (SADS; Watson & Friend, 1969), one of the most widely used self-report measures of social anxiety, assessed avoidance and subjective distress about social interactions. True/false items assess distress and discomfort in social situations (e.g., "I am seldom at ease in a large group of people"), and deliberate avoidance of social

situations (e.g., "I try to avoid situations which force me to be very sociable"). Total scores range from 0 to 28, with higher scores indicating greater social anxiety. The SADS has good internal consistency (KR-20 = .94), convergent and discriminant validity, and sufficient test-retest reliability (r = .68) (Leary, 1991).

Diagnoses were established using the Structured Clinical Interview for DSM-III-R (SCID-P; Spitzer, Williams, Gibbon, & First, 1992). Since the SCID-P did not include BDD, BDD was diagnosed with a reliable semi-structured SCID-like instrument based on DSM-IV criteria (Phillips, Atala, & Pope, 1995). Patients diagnosed with comorbid SP had to endorse a marked and persistent fear of social situations (in which embarrassment may occur) that was independent of appearance concerns. The Structured Clinical Interview for DSM-III-R Personality Disorders (SCID–II; First, Spitzer, Gibbon, & Williams, 1995) assessed Axis II disorders.

Current BDD severity was assessed with the reliable and valid clinician-rated Yale-Brown Obsessive Compulsive Scale Modified for BDD (BDD-YBOCS; Phillips et al., 1997). Scores range from 0 to 48; higher scores indicate more severe BDD. Response to treatment is defined as a 30% or greater decrease in BDD-YBOCS score. This response threshold has been associated with ratings of much or very much improved on the Clinical Global Impressions Scale (see Phillips, Dwight, & McElroy, 1998), so it reflects clinically significant improvement in global BDD symptoms, including distress and impairment. The delusionality (i.e., insight) of appearance-related beliefs (e.g., "I look deformed") was evaluated with the reliable and valid clinician-administered Brown Assessment of Beliefs Scale (BABS; Eisen et al., 1998), which assesses delusionality dimensionally (higher scores indicate greater delusionality) and categorically, based on an empirically derived cut point. Scores range from 0 to 24. The clinician-administered 17-item Hamilton Depression Rating Scale (HAM-D; Hamilton, 1960) assessed current severity of depressive symptoms.

Procedures

The SADS and other measures were completed at baseline before initiation of pharmacotherapy, while patients were free of all psychiatric medications. While citalopram participants completed measures at baseline only, fluoxetine participants repeated assessments at study termination. Of the 66 randomized, 59 participants (31 active drug, 28 placebo) completed the 12-week fluoxetine trial. Seven participants (three active drug, four placebo) withdrew during the trial for the following reasons: lost to follow-up (n = 3), no longer wished to participate (n = 2), and worsening of symptoms (n = 2). Two participants (one active drug, one placebo) who completed the fluoxetine trial did not complete endpoint SADS ratings. Comparisons between 57 subjects who completed endpoint SADS ratings and seven noncompleters indicated no significant differences in demographics (age, gender), and baseline SADS, BDD-YBOCS, BABS, and HAM-D.

Statistical analysis

Pearson product-moment and point-biserial correlation coefficients examined relationships between selected variables. Between-group differences for selected continuous variables were examined with t-tests. Change in SADS scores with fluoxetine versus placebo was tested using analysis of covariance (ANCOVA), with baseline SADS as the covariate. Based on the ANCOVA, an effect size (f) was calculated. Additional post-hoc analyses of treatment data were not corrected for possible Type I error because they were preliminary and exploratory. All tests were two tailed with α set at .05.

Results

Table 1 shows means and standard deviations for baseline SADS and symptom variables. On average, the sample had high social anxiety, moderate to severe BDD, moderate to severe depression, and poor insight. Based on the BABS, 32 (41%) patients were delusional at baseline.

As predicted, social anxiety was positively correlated with BDD symptom severity (r(79) = .33, p = .002) and severity of depressive symptoms (r(79) = .36, p = .001). However, contrary to our hypothesis, social anxiety was not correlated with delusionality (r(76) = .11, p = .356), and SADS scores of delusional (M = 19.8, SD = 7.5) and nondelusional (M = 19.3, SD = 7.9) patients did not differ, t(76) = .24, p = .813.

Thirty-two percent (n = 23) of subjects met criteria for current SP, and 37% (n = 27) met lifetime criteria. SADS scores were not correlated with the presence of either current (r(70) = -.05, p = .679) or lifetime (r(71) = -.07, p = .550) SP. In addition, SADS scores were similar for patients with and without current SP (M = 18.7, SD = 9.1 versus M = 19.5, SD = 7.3; t(70) = -.42, p = .679).

Forty-four percent (n = 28) of the fluoxetine study participants met criteria for AVPD. SADS scores were correlated with the presence of AVPD (r(62) = .48, p < 001). Patients with AVPD (M = 24.8, SD = 2.7) had higher SADS scores than those without this personality disorder (M = 18.5, SD = 7.4), t(47) = 4.70, p < .001.

At the end of the fluoxetine trial, as previously reported, there was a treatment effect for fluoxetine on BDD severity (F(1,64) = 16.5, p < .001, f = .35) (Phillips et al., 2002). Despite this effect, there was no difference in the endpoint SADS ratings of the fluoxetine (n = 30) and placebo (n = 27) groups, controlling for baseline SADS score (F(1,54) = .61, p = .437, f = .08; fluoxetine: M = 16.7, SD = 9.3; placebo: M = 17.6, SD = 8.7). Both groups showed significant improvement in social anxiety pre- to post-treatment. However, in post-hoc analyses, among participants in the active drug condition, fluoxetine responders (n = 17) had significantly lower SADS scores at the end of the trial than fluoxetine nonresponders (n = 13), controlling for baseline SADS (F(1,27) = 5.81, p = .023, f = .31; responders: M = 12.4, SD = 9.3; nonresponders: M = 22.5, SD = 5.4). Among all fluoxetine study participants, change in SADS score was highly correlated with change in BDD severity (r(55) = .64, p < .001).

Discussion

Results suggest that patients with BDD experience high levels of social anxiety. The mean baseline SADS score of 19.8 (SD = 7.7) is 1.3 SD units higher than the mean of 9.1 (SD = 8.0) for the normative sample (n = 205) reported by the scale authors (Watson & Friend, 1969), indicating a higher magnitude of avoidance and distress about social interaction in BDD relative to nonclinical samples. Similarly, in Veale et al. (2003), 107 participants with BDD scored approximately 1.5 SDs higher than nonclinical controls on both the Social Phobia Scale and the Social Interaction Anxiety Scale. However, BDD does not appear to notably differ from other clinical samples in terms of social anxiety. The SADS score for our sample is consistent with SADS scores reported in Turner, McCanna, and Beidel (1987) for agoraphobia without panic (M = 18.4, SD = 10.2), generalized anxiety disorder (M = 20.5, SD = 7.8), obsessive-compulsive disorder (M = 20.5, SD = 8.4), and SP (M = 22.7, SD = 7.1). These data indicate that high levels of generalized social anxiety are not specific to SP and are in fact present across a number of disorders, including BDD.

Our findings of an association between BDD severity and social anxiety are consistent with previous studies linking negative body image to social anxiety (Leary & Kowalski, 1995) and

introversion (Archer & Cash, 1985). That social anxiety in BDD is strongly associated with comorbid AVPD but not related to the presence of comorbid SP is most likely due to both the structure of DSM criteria for these disorders and lack of specificity in the SADS. Since the SADS taps general social inhibition, prominent in AVPD, one would expect a higher magnitude of social anxiety in those with both BDD and AVPD. However, patients with and without comorbid SP differ in the kind, but perhaps not the magnitude, of social anxiety experienced, with the comorbid group experiencing social anxiety independent of appearance concerns. The lack of a hypothesized association between delusionality and social anxiety is likely due to a narrow range of BABS scores, with only 10% of participants in the excellent, good, or fair insight range.

Social anxiety did not improve more with fluoxetine than with placebo treatment and the effect size was small. Participants showed improvement in social anxiety regardless of treatment condition, suggesting the possible therapeutic effect of attending frequent pharmacotherapy visits and interacting with study staff. Social anxiety did improve significantly more in fluoxetine responders than in nonresponders, with a medium to large effect size. It should be noted that, among all participants in the fluoxetine trial, improvement in BDD severity was significantly correlated with improvement in social anxiety. However, it is unclear to what extent improvement in BDD may have led to decreased social anxiety, to what extent fluoxetine may have directly reduced social anxiety in responders, or both.

Our study has several limitations. Power for treatment analyses was limited. Due to our efficacy study's standard inclusion and exclusion criteria, our sample may not be representative of individuals with BDD in the community or other clinical settings. Also, subjects were recruited from a clinic that specializes in BDD, so the results may not extend to nonspecialty settings. Furthermore, since our sample consisted mostly of women, findings may not generalize to men with BDD. Finally, the generalizability of our findings may be limited by the fact that this was a treatment seeking sample. Because the evaluation and treatment process involves social interaction, individuals with BDD who do not seek treatment may be even more socially avoidant than the current sample.

Because the fluoxetine trial was only 12 weeks, longer treatment studies with follow-up phases are needed to determine whether social anxiety would improve more with fluoxetine than placebo over the longer term. Research comparing the effects of CBT versus pharmacotherapy, and their combination, on social anxiety in BDD is also needed.

It is not clear whether a causal relationship exists between social anxiety and the occurrence or severity of BDD; that is, whether high levels of social anxiety contribute to BDD's development and maintenance, whether BDD causes high levels of social anxiety, or whether the two covary with some other more primary etiologic factor. The authors' impression is that for some patients, high levels of social anxiety may contribute to BDD's onset and may contribute to symptom exacerbation. Consistent with this line of reasoning, Wilhelm et al. (1997) report preliminary data in which the onset of primary SP in patients with comorbid BDD preceded the onset of BDD by 12.6 years. Conversely, many patients with BDD report that appearance preoccupation itself causes significant secondary social anxiety and avoidance. It is important for clinicians to be aware that patients with BDD have high levels of social anxiety and avoidance and that, in our clinical experience, BDD may for this reason be misdiagnosed as SP or lead to dropout from treatment. Additional studies – in particular, longitudinal studies and studies of pathoetiology – are needed to examine the clinically and theoretically important interface between BDD and the construct of social anxiety.

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Table 1Means and standard deviations for baseline SADS and symptom variables

	n	Range	M	SD
SADS	81	1–28	19.8	7.7
BDD-YBOCS	81	21–44	31.2	5.6
HAM-D (17-item)	81	3–40	20.0	8.0
BABS	78	7–24	17.4	4.4

Note: Sample sizes differ due to missing data. SADS, Social Avoidance and Distress Scale; BDD-YBOCS, Yale-Brown Obsessive Compulsive Scale Modified for BDD; HAM-D, Hamilton Depression Rating Scale; BABS, Brown Assessment of Beliefs Scale.