CASE REPORT

A Case of Treatment-resistant Depression and Body Dysmorphic Disorder: The Role of Electroconvulsive Therapy Revisited

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ABSTRACT

Body dysmorphic disorder is a common, often disabling condition, and is frequently comorbid with major depressive disorder. Selective serotonin reuptake inhibitors constitute first line set of somatic interventions but the management of refractory patients remains challenging. Electroconvulsive therapy, an often highly beneficial treatment for medication resistant-depression, is not considered an effective therapeutic alternative for treatment refractory body dysmorphic disorder. Here we present a 50-year-old woman with body dysmorphic disorder and comorbid major depressive disorder who remained incapacitated and suicidal despite several trials with selective serotonin reuptake inhibitors and antipsychotic medication. Depressive and dysmorphic symptoms appeared to resolve with electroconvulsive therapy, and remission was sustained for two months. Electroconvulsive therapy has an important place in the management of treatment-resistant depression associated with body dysmorphic disorder, and, in select cases, may be effective for dysmorphic symptoms as well.

INTRODUCTION

Body dysmorphic disorder (BDD) is a relatively common,[1] distressing, often disabling condition characterized by excessive concerns about perceived defects in physical appearance that are either nonexistent or grossly exaggerated by the patient. Delusional beliefs and absence of insight are considered particularly incapacitating manifestations of this disorder.[2] Although currently classified in the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition*

(*DSM-5*)[3] with obsessive compulsive disorder, BDD may be closely associated with major depressive disorder (MDD) as well.[4] Suicide risk is of serious concern in BDD[5] and may be heightened in patients with comorbid MDD,[6] lack of insight, and dysmorphic delusions.[7] Selective serotonin reuptake inhibitors (SSRIs) and cognitive behavior therapy are effective in BDD,[8,9] but treatment options for refractory patients are limited.[10] Although a small number of case reports suggest that BDD may improve with ECT, this modality is generally not considered an effective therapeutic alternative.[9,10] Here, we present a patient with treatment-refractory MDD and BDD in whom both conditions appeared to have resolved with ECT.

CASE REPORT

Our patient was a 50-year-old, single, unemployed, white woman with BDD referred to our unit for ECT treatment for comorbid depression and suicidality. The current episode began almost two years previously when she became concerned that the shape of her eyes was distorted by movements of the bony structure of her orbits. She soon began avoiding mirrors so as not to be confronted with this apparently grotesque alteration in appearance. She quit work to avoid embarrassment and became socially isolated, but compulsively sought reassurance from family members about her appearance. A major depressive episode supervened after roughly one month, marked by depressed mood with anhedonia, feelings of helplessness and hopelessness, and poor energy, concentration, and appetite. She subsequently made six suicide attempts, two of which required intubation. Treatment with a variety of SSRI medications (sertraline, clomipramine, and venlafaxine) alone or with neuroleptics (olanzapine, thiothixine, and aripiprazole) was unsuccessful, and she failed a four-month trial of fluoxetine titrated to 60mg daily, thiothixine, and aripiprazole 20mg daily prior to commencing ECT.

The pre-ECT assessment found our patient to be profoundly depressed with dysmorphic delusions, delusions of reference, and plans to commit suicide by shooting herself. Routine medical evaluations, assays for N-methyl-D-aspartate receptor (NMDA) antibodies and Lyme's disease antigens, and a magnetic resonance imaging (MRI) scan of the brain, were all negative. After the patient consented to ECT, a course of 11 bifrontal treatments was administered under general anesthesia (propofol 100–120mg intravenously [IV]) with muscle relaxation (succinylcholine 100mg IV) using the Spectrum device (stimulus parameters: 50-60Hz; 63.2J to 125J). Seizure duration ranged between 20 and 35 seconds. Fluoxetine had been discontinued before initiating ECT, but aripiprazole 20mg daily was maintained throughout. Lithium carbonate was started after the eighth ECT, omitting the dose on evenings preceding ECT days and administering morning dosages after treatment. Marked clinical improvement was noted after the fifth ECT treatment, and the patient was free of both depressive and dysmorphic symptoms at the end of her course of treatment. ECT was well tolerated with the exception of one self-limited episode of post ictal confusion after the 11th ECT. She was discharged to another institution on lithium carbonate 900mg at bedtime (lithium level: 0.8meq/L), venlafaxine 225mg daily, and olanzapine 15mg at bedtime. We subsequently learned that dysmorphic symptoms recurred two months later, despite medication adherence, and that the depression that followed after an additional month led to another serious suicide attempt.

DISCUSSION

Diagnostic considerations. The diagnosis of BDD is strongly supported in this case. The patient's preoccupation with a nonexistent deformity was of delusional intensity and impelled her to repeatedly seek reassurance from family members. Ensuing delusions of reference caused her to quit work and to shun social interactions. Her assiduous avoidance of mirrors also is of diagnostic importance—her reflected image reinforced the distressing conviction that she was deformed—and may have been a manifestation of the visual processing abnormalities said to be associated with BDD.[11,12] Symptoms of major depressive episode (MDE) were equally evident, manifested by profoundly depressed mood, anhedonia, poor appetite, utter hopelessness, and dangerous suicidality.

The nosological status of BDD has endured a peripatetic history.[13] First included in the third edition of the *DSM* (*DSM III*) as a somatoform disorder almost a century after its initial description, BDD is currently classified as part of the obsessive compulsive disorder (OCD) spectrum. Comorbidity with MDD, as evidenced by our case, is particularly common,[6] and it has been postulated that the two conditions frequently share overlapping mechanisms, based on evidence that improvements in depressive and dysmorphic symptoms are intercorellated in a large proportion of patients having this comorbidity.[4] In our patient, the simultaneous resolution of depressive and dysmorphic symptoms is consistent with this model, whereas the course of symptomatology, in which the emergence of dysmorphic beliefs consistently preceded the advent of depressive episodes, would accord with the view that BDD represents a distinct construct, separate from MDD.[14] We therefore applied *DSM-5* diagnostic criteria to our patient as follows: BDD with absent insight/delusional beliefs, and—because the patient's delusions were more convincingly imputed to BDD than to depression—MDD, otherwise specified.

Treatment. Whereas SSRIs are the preferred choice of somatic treatments for BDD and have been reported to be effective for BDD-related delusions and comorbid depression with suicidal ideation[8,10,15] ECT was administered in our case to manage depression that failed to respond to a variety of medication combinations that included SSRIs. It is advised to first treat BDD with SSRIs for 12 to 16 weeks, increasing medication to maximum allowable dosage, and to provide additional similarly prolonged trials with different SSRIs for initial treatment failures.[10] A more variegated sequence of medications of shorter duration is recommended for MDD.[16] Hence our patient was considered to have failed the four-month treatment with fluoxetine according to both guidances, and her depressive episode *per se* was classified as medication refractory on the basis of previous failures. However, we have not been able to establish that the length of these earlier treatments was in accord with BDD recommendations. It is possible, therefore, that her condition may have responded to an additional prolonged treatment with SSRI medication. Nevertheless, the severity of her depressive symptoms warranted urgent consideration of ECT.

It is likely that the resolution of BDD symptoms and the remission of the MDE were both due to the therapeutic effect of ECT. A carry-over effect of fluoxetine cannot be ruled out definitively but is improbable as there had been no response to this medication during the four months preceding ECT. Fluoxetine treatment had been discontinued prior to ECT, and the only medication administered concomitantly was aripiprazole—not considered beneficial for BDD. Moreover, the two-month, symptom-free interval that followed was sufficient for the index episode of BDD to be considered in remission according to recent research.[17]

ECT has generally not been found to be effective for BDD. Although improvement in comorbid depressive episodes with ECT may be robust, BDD usually persists regardless.[9] To our knowledge, a specific therapeutic effect on BDD symptoms from ECT administered to manage comorbid depression has only been previously reported in two patients.[18,19] ECT was beneficial for dysmorphic delusions in a third report on a patient with schizophrenia.[20] As was the case here, the patients in the previous reports did not respond to SSRIs, but, in contradistinction to our patient, had significant medical antecedents. Carrol et al[18] reported remission of BDD and depression with ECT following unsuccessful treatment with sertraline and haloperidol in a 34-year-old woman with a history of encephalopathy secondary to meningitis. Readministration of ECT was effective when symptoms recurred five months later. The temporal sequence of dysmorphic and depressive symptoms was not specified in that report. In the second patient,[19] a 24-year-old man with seizure disorder and unilateral cryptorchidism, BDD was present for eight years before the onset of MDD. Neither condition responded to SSRI treatment but both improved with ECT. The patient remained in remission six months later. Here the antecedence of BDD, much more pronounced than in our patient, suggests that dysmorphic symptoms constituted an independent construct rather than a secondary manifestation of depression.[14]

CONCLUSION AND RECOMMENDATIONS

ECT has an important place in the management of treatment-resistant depression associated with BDD and, in select patients such as ours, may be effective for dysmorphic symptoms as well. It is currently not possible to identify such patients. However, BDD is said to be underestimated,[21] and useful indications for ECT may emerge as the ascertainment of BDD in depressed populations becomes more refined. Previous studies establishing the efficacy of ECT in MDD may have unknowingly included and successfully treated a significant number of patients with BDD, given the elevated comorbidity of these two disorders. Interestingly, clinical symptoms that have been associated with BDD, such as somatization, hypochondriasis, and neurotic depression,[13,14,20] have been reported to predict poor response to ECT except in a subgroup with particularly severe depression.[22–25] It is possible that the construct of BDD itself—not specifically assessed in these reports—may similarly moderate the effectiveness of ECT in depression.

With these current limitations in mind, we suggest the following approach regarding the use of ECT for the depressed patient with comorbid BDD:

1. SSRIs are generally the preferred class of somatic treatment, even in the presence of dysmorphic delusions.
2. ECT should not be avoided in depressed patients for whom this treatment would be otherwise indicated simply because of the co-occurrence of BDD—particularly when there is a history of previous response to ECT.
3. The assessment of the depressed patient referred for ECT should include careful screening for dysmorphic symptoms.
4. Dysmorphic symptoms commonly persist after the remission of depressive episodes and should be reevaluated during intermorbid states.

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