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Borderline personality disorder traits are not associated with a differential change in global cognitive function during acute course ECT

1. Introduction

Borderline personality disorder (BPD) is characterized by persistent impairments in emotional regulation and interpersonal relationships [1]. It is highly comorbid with mood, anxiety, and substance use disorders, and when comorbid with major depressive disorder is associated with reduced remission rate for depressive symptoms [2]. Recent evidence has shown that electroconvulsive therapy (ECT) results in equivalent short-term improvement in depressive symptoms among patients who screen positive or negative for BPD traits [3]. Less is known, however, about whether the presence of BPD traits mediates the potential cognitive side effects of ECT. In order to characterize the risks and benefits of ECT treatments in patients with BPD traits, this study assesses changes in global cognitive function during acute course ECT among patients who screen positive or negative for BPD traits.

2. Methods

This is a retrospective cohort study of patients aged 16+ receiving ECT at a single center between April 2015 and May 2020. Patients were treated using a Mecta Spectrum 5000Q (Tualatin, OR) with methohexital as the default anesthetic agent (although etomidate, propofol, or ketamine could also be used) and succinylcholine muscle relaxant. Individualized seizure threshold was determined at the time of first treatment [4,5], and subsequent treatments were given a default schedule of three times weekly [6]. As part of routine care, patients were screened using multiple self-reported measures including the Quick Inventory of Depressive Symptomatology Self Report 16 item scale (QIDS; a scale of depression severity), the Montreal Cognitive Assessment (MoCA; a screening measure for global cognitive function) [7], and the McLean Screening Instrument for BPD (MSI-BPD; a 10-item screening instrument for BPD traits) [8].

For this study, all patients who completed the baseline screening scales and remained in ECT for at least 10 ± 1 treatments were reassessed using the MoCA, with an alternative version used to reduce practice effects. Patients were divided into two groups, with those answering "yes" to ≥7 MSI-BPI items defined as screening positive for BPD traits (BPD+); patients with MSI-BPD scores <7 were defined as screening negative for BPD traits (BPD-). In prior studies, a score of ≥7 on the MSI-BPD was associated with a sensitivity of 0.81 and a specificity of 0.85 for defining BPD vs. a structured clinical interview [8].

Baseline differences between treatment groups were analyzed using χ^2 tests for categorical variables and two-sided t tests for continuous variables. For the primary statistical analysis, the MoCA score at treatment #10 was analyzed using linear regression, with age, sex, diagnosis (major depressive disorder, bipolar affective disorder, other), initial treatment location (inpatient vs. outpatient), initial QIDS score, initial MoCA, and screening positive or negative for BPD as covariates. In a secondary analysis, we explored factors associated with decline in MoCA of ≥2 points during ECT, representing a clinically-relevant decline on that scale [9]. The binary outcome of ≥2 point decline in MoCA vs. < 2 point decline in MoCA (including patients with unchanged or improved scores) was analyzed using logistic regression, with age, sex, diagnosis (major depressive disorder, bipolar affective disorder, other), initial treatment location (inpatient vs. outpatient), initial QIDS score, initial MoCA, and screening positive or negative for BPD as covariates. Analyses were completed using GraphPad Prism (v 9, San Diego, California, USA).

3. Results

A total of 915 patients had MoCA data available at baseline and at treatment #10 ± 1. Of these, 154 (16.8%) were BPD + while 761 (83.2%) were BPD- (Table S1). These groups differed in baseline characteristics, with BPD + patients being younger (mean age 34.7 ± 12.4 for BPD + vs. 47.7 ± 17.1 for BPD-) and more likely to begin treatment as inpatients (68.8% for BPD + vs. 58.3% for BPD-). Baseline depression severity was higher for BPD + patients (QIDS 19.4 ± 4.1 for BPD + vs. 16.5 ± 4.8 for BPD-), but the two groups did not differ significantly in baseline global cognitive function (MoCA 26.3 ± 2.9 for BPD + vs. 25.8 ± 3.0 for BPD-), ECT parameters, or other demographics.

In a linear model of MoCA score at treatment #10, BPD traits were not associated with a differential change in MoCA (estimate −0.347, 95% CI -0.836 to 0.141; P = 0.163). Likewise, sex, age, diagnosis, and initial treatment location were not significantly associated with final MoCA (Table 1). In contrast, initial QIDS and baseline MoCA scores were both positively associated with the final MoCA score.

Abbreviations: BPD, borderline personality disorder; ECT, electroconvulsive therapy; QIDS, quick inventory of depressive symptomatology; MoCA, Montreal cognitive assessment; MSI-BPD, McLean Screening Instrument for BPD.

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Table 1
Linear model of MoCA at treatment 10 ± 1, with age, sex, diagnosis (major depressive disorder, bipolar affective disorder, other), initial treatment location (inpatient vs. outpatient), initial QIDS score, initial MoCA, and screening positive or negative for BPD traits as covariates. Bolded values are significant at the level of $p < 0.05$.

	Estimate	CI	p
BPD+	−0.347	−0.836 to 0.141	0.163
Sex (male)	−0.194	−0.548 to 0.160	0.283
Age	0.00257	−0.00830 to 0.0134	0.643
Diagnosis			
MDD	0.359	−0.505 to 1.22	0.415
BPAD	0.457	−0.454 to 1.37	0.325
Location (outpatient)	0.167	−0.187 to 0.522	0.354
Baseline QIDS	0.0415	0.00420 to 0.0789	**0.0292**
Baseline MoCA	0.443	0.383 to 0.502	**<0.0001**

In order to specifically examine those patients who demonstrate a clinically-significant worsening in MoCA score with ECT treatment, we conducted a logistic regression on the binary outcome of a ≥2 point decline in MoCA vs. no such decline. In total, 319 patients demonstrated a decline in MoCA of ≥2 points (Fig. 1). In this logistic model, only a higher baseline MoCA score was associated with a higher odds of MoCA decline of ≥2 points (aOR 1.34, 95% CI 1.26 to 1.43; P < 0.0001). BPD traits were not significantly associated with the odds of having a MoCA decline ≥2 points (Table S2).

4. Discussion

Among 915 patients presenting for ECT and then completing a 10 treatment course, screening positive for BPD traits was not associated with a differential change in global cognitive function, as measured by the MoCA. Patients in this sample had a mean reduction in MoCA of 0.5 over 10 treatments, a magnitude of change that is lower than the minimal clinically important difference of 1.22–2.15 that has been determined among stroke patients [9]. In our model only baseline cognition and depression severity were associated with final MoCA, with demographic parameters and treatment location not showing a significant association. Screening positive for BPD traits was likewise not associated with a differential change in MoCA, and so these results do not support the idea that BPD + patients are more likely to demonstrate a differential change in global cognitive function. Moreover, BPD traits were not associated with a higher odds of experiencing a clinically significant decline in MoCA score with ECT treatment.

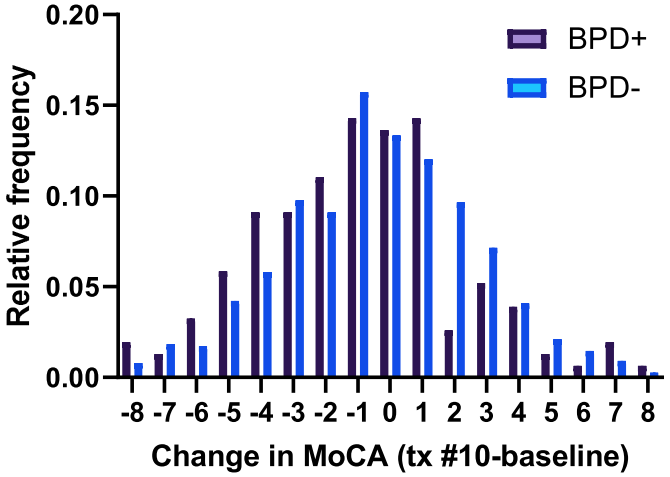

Fig. 1. Histogram of change in MoCA over the course of 10 ECT treatments, stratified by BPD+ (purple) and BPD- (blue). Negative numbers represent reduction in MoCA over the course of ECT treatment, and positive numbers represent improvement in MoCA over the course of treatment. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

Strengths of this study include large sample size, consistent assessment of subjects, and consistent treatment methods over the study period. The chief limitation is the use of a single screening tool, the MoCA, as the assessment of global cognitive function. This tool has not been specifically designed for ECT, and it may not optimally track changes in relevant cognitive parameters with treatment. In particular, it does not assess autobiographical memory loss following ECT that is among the most distressing complications noted from treatments [10]. Whether these side effects differ in BPD+ and BPD-patients requires further study. Additionally, the MoCA has not previously detected changes in global cognitive function on a group level among patients receiving ECT [11]. Moreover we are unable to assess the cognitive changes in patients who discontinue treatment before the follow-up MoCA at treatment #10 and thus the estimation of MoCA change is subject to survivorship bias. Some patients may have discontinued treatment because of adverse cognitive effects, and the calculated change in MoCA will not reflect those cognitive changes among those patients without follow-up MoCA. As rates of continuation in ECT differ based on BPD + or BPD-status [12], this introduces an additional cofounding effect. Furthermore, assignment to BPD+ and BPD-groups is based on a screening instrument and not a structured clinical diagnosis of borderline personality disorder. As a result, some of those BPD + patients may not meet full criteria for borderline personality disorder, and so these results may not generalize to patients with formal personality disorder diagnoses. Finally, as we only assess cognitive outcomes following treatment #10, we are unable to assess the long-term cognitive or depressive outcomes in patients, and how these may be modulated by BPD status. Existing data suggests that even 8 days out from treatment the response to ECT is lower in BPD patients [13], and it is unknown whether groups diverge more significantly during longer follow-up.

5. Conclusion

Among 915 patients receiving 10 acute course ECT treatments, screening positive for BPD traits was not associated with a differential change in global cognitive function, as measured by the MoCA, nor were BPD traits associated with a higher odds of experiencing a clinically significant MoCA decline.

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Declaration of competing interest

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.brs.2022.04.006.

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