those who had a dose reduction to 40mg (80/40mg, -6.2 [n=13]). In addition, a majority of these participants had an AIMS response after 48 weeks of treatment (40mg, 53.7%; 80/40mg, 53.8%). In the 1506 population, the percentage of participants who had a CGIS-TD score ≤2 (rating of "normal, not at all ill" or "borderline ill") at Week 12 was 63.6% (7/11) in the 40mg group and 30.8% (4/13) in the 80/40mg group. Data from Weeks 24 to 60 of 1506 were limited by the small sample sizes (<10 participants each in 40mg or 80/40mg group at each of these visits).

CONCLUSIONS: Based on these analyses and results from published studies, VBZ 40mg may be an effective long-term option for some TD patients. Dose reductions from 80 to 40mg, if necessary, did not appear to compromise long-term benefit.

Funding Acknowledgements: This study was sponsored by Neurocrine Biosciences, Inc.

124
Reframing the Approach to the Diagnosis and Treatment of Borderline Personality Disorder in Adolescents

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BACKGROUND: Using the same DSM-5 criteria as in adults, BPD in adolescents is defined as a 1-year pattern of immature personality development with disturbances in at least 5 of the 9 domains listed in the DSM-5. BPD can now be reliably diagnosed as young as 13 using one of several standardized clinician, or self-rated diagnostic instruments. Unfortunately published US and Canadian positions regarding pharmacological treatment have been, With regard to evidence-based studies, pharmacological treatment is not recommended and, if ultimately required, should be limited to second-generation antipsychotics. Fortunately, the last decade s extensive advancements in brain-mapping have provided more clarity about the various brain dysfunctions underlying the symptoms/traits presenting in BPD, providing new opportunities to address these primarily Fronto-Limbic dysfunctions neuropharmacologically and potentially, significantly ameliorate. Thus, in turn, likely enhancing the effectiveness of the newer available therapies.

OBJECTIVES: The current study explores the feasibility of more effectively managing BPD symptoms/traits with a unique medication protocol consisting of two medications; an anticonvulsant (oxcarbazepine) and a dopaminergic (amantadine HCl), without use of an antipsychotic medication.

METHODS: Subjects were 147 females, ages 13-16, with the diagnosis of BPD treated with the described medication protocol in a residential facility. Positive outcome was described as achievement and maintenance of greater than 50% improvement from baseline admission state of functioning for 1 year. They were discharged when stable and having achieved greater than 50% improvement from baseline. Outpatient prescribers were requested to be compliant with the treatment protocol. However, some were non-compliant, substituting antipsychotic medication instead. Care givers were surveyed at 6 months and 1 year to determine whether their child was maintaining greater than 50% improvement.

RESULTS: The percent maintaining greater than 50% improvement was calculated for those whose caregivers reported continuation of the medications as prescribed, versus those whose prescribers changed the medications to the Community Standard. Of those compliant with the medication protocol, 61 of 86 (71%) maintained >50% improvement. Of those moved to the Community Standard approach, 19 of 61 (31%) maintained >50% improvement. Using Chi Square analysis, there was a significant relationship between maintenance of improvement and medication protocol compliance. Chi Square, Fisher's exact test = $p<0.001$.

CONCLUSION: The results indicate that, for adolescents 1 year post-discharge from residential treatment for BPD, continuation of the above described medication protocol provides significantly higher rates of maintenance of achieved symptom improvement. Further controlled studies are needed.

Funding: None.

127
Successful Treatment of Major Depressive Disorder with Moclobemide After Recurrent Hyponatremia Induced by Multiple Antidepressant Classes

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ABSTRACT: Background: Antidepressant-induced hyponatremia/syndrome of inappropriate antidiuretic hormone (SIADH) can cause significant morbidity and mortality. Antidiuretic hormone release due to stimulation of central serotonin 5HT1C, 5HT2 and α-1 adrenergic receptors is thought to cause this adverse effect (Spigset, 1995). Evidence on which antidepressants are more likely to cause hyponatremia is inconsistent (Coupland, 2011;

https://doi.org/10.1017/S1092852920000437 Published online by Cambridge University Press

Leth-Moller, 2016). Owing to its uncommon use, there is limited and conflicting data on the risk of hyponatremia with Moclobemide, a reversible inhibitor of monoamine oxidase A (Mercier, 1997; Mazhar, 2019). There are few reports of hyponatremia induced by multiple antidepressant classes in the same patient.

OBJECTIVE: To add to the literature on risk of hyponatremia with Moclobemide and other antidepressants.

METHODS: We report a case of hyponatremia sequentially induced by multiple different antidepressant classes who was treated with Moclobemide with no recurrence of hyponatremia. We review existing literature on antidepressant-induced hyponatremia.

RESULTS: A 67-year-old man with a history of hypertension, dyslipidemia and gout was first diagnosed with major depressive disorder at age 50 after presenting with pervasive depressed mood, anhedonia, insomnia, poor concentration and feelings of worthlessness. Investigations found no medical causes of depression. His depression remitted on Venlafaxine 75mg/day with no hyponatremia induced. During a second depressive episode 4 years later, his serum sodium (Na) dropped from a normal baseline to 122mmol/L after Venlafaxine was restarted. He appeared euvolemic on physical examination. Investigations found no other causes of hyponatremia and were consistent with SIADH, which was attributed to Venlafaxine. His depression later remitted on Mirtazapine 30mg/day with no hyponatremia induced. During his third depressive episode at age 67, he developed hyponatremia (serum Na 123mmol/L) a week after restarting Mirtazapine. His clinical picture was consistent with SIADH. He later developed hyponatremia after initiating the following antidepressants sequentially: Fluvoxamine, Agomelatine, Nortriptyline, Bupropion. Hyponatremia resolved with fluid restriction and cessation of the implicated antidepressant each time before the next was initiated. He eventually tolerated Moclobemide 300mg/day with no recurrence of hyponatremia.

CONCLUSIONS: Agomelatine, Nortriptyline and Bupropion are reported to have a low risk of hyponatremia but were implicated in this case. Venlafaxine and Mirtazapine did not cause hyponatremia when first taken but were implicated when restarted after a period of cessation, underscoring the idiosyncratic nature of antidepressant-induced hyponatremia. Moclobemide can be considered for depressed patients with recurrent antidepressant-induced hyponatremia. Serum Na should be regularly monitored in patients taking antidepressants who are at high risk of hyponatremia.

128
Factors Associated with Cost Savings Following Use of a Pharmacogenetic Assay in Individuals with Mood and Anxiety Disorders

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ABSTRACT: Background: In a study conducted in the database of a large commercial healthcare insurer, we previously demonstrated that use of a commercial pharmacogenetic assay for individuals with mood disorders was associated with decreased resource utilization and cost in the 6 month period following use compared to propensity-score matched controls. We conducted a post hoc analysis to understand variables associated with high cost savings.

METHODS: The results and methods of the initial study have previously been described. Cases were individuals with mood and anxiety disorders who received a commercial pharmacogenetic assay (Genomind, King of Prussia PA) to inform pharmacotherapy. 817 tested individuals (cases) with mood and/or anxiety disorders were matched to 2745 controls. Overall costs were estimated to be $1,948 lower in the tested group. The differences were largely the result of lesser emergency room and inpatient utilization for cases. In the present analysis, cost difference for cases compared to their matched controls was rank ordered by decile. High cost savers were arbitrarily defined a priori as the top 20% of savers. Using multivariable modeling techniques, an ordinal logistic regression model was generated in which baseline or follow-up variables were statistically tested for independent associations with high, low, and no cost savings.

RESULTS: 606 (74%) of cases were net cost savers compared to their controls (cost difference <0). High cost savers (n=121) saved on average $10,690 compared to their matched controls. They were statistically more likely to have been diagnosed with bipolar disorder (n=33/121) than low cost savers (n=57/485) or non-savers (n=31/211), and had a lower Charlson Comorbidity index. High cost savers had fewer mean number of antidepressants in the baseline period (mean=3.16) compared to non-savers (3.73) but more than low cost savers (2.72) ($p<0.05$ across groups). In a multivariable model, bipolar, count of antidepressants, outpatient visits, and inpatient visits were statistically associated with being a high cost saver; antidepressant count and all-cause inpatient and outpatient visits in the baseline period were inversely associated with cost savings.

https://doi.org/10.1017/S1092852920000437 Published online by Cambridge University Press