7.7 MENTAL HEALTH COMORBIDITY IN PEDIATRIC BIPOLAR DISORDER AND DMDD: IMPACT OF DMDD INTRODUCTION?

Yasmina Saade, MD, The Johns Hopkins University School of Medicine, yasminasaadeh@gmail.com; Robert L. Findling, MD, MBA; Prethibha George, PhD; Xiaofeng Zhou, PhD; Phillip B. Chappell, MD, MBA

Objectives: The aim of the study was to describe mental health comorbidities in youth diagnosed with either bipolar disorder (BPD) or DMDD before and after the introduction of the DMDD diagnosis.

Methods: This was a longitudinal, retrospective cohort study with data extracted from the 2008-2018 Optum® electronic health records. Youths aged 10 to <18 years, with ≥6 months of enrollment before cohort entry, were included. Concomitant BPD and DMDD diagnoses (on the same visit) were exclusionary criteria. The 10 most common mental health comorbid diagnoses were included. We performed analyses for the periods 2008 to 2013 (prior to DMDD introduction) and 2016 to 2018. Continuous data were analyzed as mean and SD. Dichotomous and categorical data were computed as counts and percentages of patients in different categories.

Results: Both BDP and DMDD were highly comorbid conditions (proportions ranging from 80.0% to 97.3%). Patients with DMDD had an even greater level of comorbidity than patients with BPD. ADHD and depressive and anxiety disorders were the most comorbid conditions in both groups. The most frequently comorbid conditions in BPD were ADHD (49.4% pre-DMDD, 47.8% post-DMDD), depressive disorders (33.3% pre-DMDD, 50.1% post-DMDD), and anxiety disorders (21.4% pre-DMDD, 47.7% post-DMDD). The most comorbid conditions in DMDD were ADHD (71.7%), depressive disorders (70.1%), and anxiety disorders (50.2%). Compared to youth with DMDD, youth with BPD had higher proportions of PTSD and substance use disorders. Substantially higher proportions of ADHD, ODD, conduct disorder, intermittent explosive disorder, depressive disorders, and autism spectrum disorder were observed in the DMDD cohort.

Conclusions: Mental health comorbidities were frequent in pediatric BPD and even more so in DMDD. ADHD and depressive and anxiety disorders were the most frequent comorbidities among patients with either BPD or DMDD, although different patterns of comorbidity were otherwise observed in these 2 groups. The introduction of a DMDD diagnosis led to an increase in comorbid diagnoses in patients aged 10 to <18 years. Additional studies addressing these comorbidities in the setting of BPD and DMDD are needed.

DMDD, BRD, CM

Supported by Pfizer Inc.

http://dx.doi.org/10.1016/j.jaac.2020.08.128

7.8 NEURAL CORRELATES OF REWARD PROCESSING DISTINGUISH HEALTHY YOUTH AT FAMILIAL RISK FOR BIPOLAR DISORDER FROM YOUTH AT FAMILIAL RISK FOR MDD

Akua F. Nimarko, BS, Stanford University, animarko@stanford.edu; Adina S. Fischer, MD, PhD; Kelsey E. Hagan, PhD; Kayla E. Carta, BA; Aaron J. Gorelik, BS; Ananya Nrusimha, BA; Jaskanwaljeet Kaur, BS; Nicholas S. Rodriguez, BA; Manpreet K. Singh, MD, MS

Objectives: Youth at familial risk for bipolar disorder (BD) and MDD have separately been found to have dysfunctional reward processing, which is also a core feature of BD and MDD. However, it is yet to be understood how youth at familial risk for BD differ from youth at familial risk for MDD with respect to reward processing.

Methods: We examined reward processing differences among the healthy offspring of a parent with BD (n = 40, "BD-risk"), the healthy offspring of a parent with MDD (n = 41, "MDD-risk"), and youth without any personal or family psychopathology (n = 45, "HC"), with a mean age of 13.09 +/- 2.58 years and 56.3% being female. All participants completed the Monetary Incentive Delay (MID) functional neuroimaging task. To examine group differences in neural activation during the anticipation and outcome of monetary gain and loss, region of interest (ROI) analyses were conducted using anatomically defined thalamus, ventrolateral prefrontal cortex (VLPFC), and putamen seeds (Bonferroni corrected $p = 0.017$). Whole-brain voxel-wise group differences were also examined ($z > 3.1$; family-wise error [FWE]-cluster corrected $p < 0.05$). All analyses included age, sex, and subthreshold depression and mania severity as covariates.

Results: Relative to MDD-risk and HC, BD-risk had increased activation of the thalamus during anticipation of monetary loss ($F_{2,123} = 4.58$; $p = 0.01$) and decreased activation of the VLPFC during outcome of monetary gain ($F_{2,117} = 4.65$; $p = 0.01$). There were no significant group differences in putamen activation. Whole-brain analyses showed that BD-risk had significantly reduced activation in the left lingual gyrus compared to MDD-risk and HC during anticipation of monetary reward ($z > 3.1$; $p < 0.001$). There were also significant group activation differences in the right cerebellar crus II during outcome of monetary gain ($z > 3.1$; $p = 0.003$). Post hoc analyses demonstrated decreased activation in the right cerebellar crus II in BD-risk ($p < 0.001$), less so in MDD-risk ($p < 0.02$), but not in HC ($p = 0.15$).

Conclusions: Aberrant thalamic, VLPFC, lingual, and cerebellar activation during reward processing may distinguish familial risk for BD from those at familial risk for MDD and represent a unique marker of early vulnerability while processing rewards.

ADOL, BRD, DDD

Supported by NIMH Grant K23-MH085919

http://dx.doi.org/10.1016/j.jaac.2020.08.129

7.9 OFFSPRING OF PARENTS WITH BIPOLAR DISORDER ARE CHARACTERIZED BY PSYCHOPATHOLOGICAL AND NEUROPHYSIOLOGICAL BUT NOT NEUROCOGNITIVE ABERRATIONS

Martin Brunovsky, MD; Jiri Horacek, MD; Michaela Viktorinova, PsyD; Antonin Sebela, MD; Adam Kalina, MD; Markéta Mohaplova, PsyD; Michal Goetz, MD, PhD, Motol University Hospital, michal.goetz@lfmotol.cuni.cz

Objectives: Despite the fact that it is possible to trace, retrospectively, the beginning of bipolar disorder (BD) in childhood or adolescence in many patients, the pediatric form of BD is unexplained and controversial. Therefore, it is important to investigate the neurobiological alterations underlying the BD endophenotype in childhood. We investigated brain intracortical activity and functional connectivity together with clinical and cognitive characteristics in a sample of offspring of parents with BD (BDO).

Methods: Thirty-five children (aged 8-17 years) of parents with BD (BDO group) and 35 age- and gender-matched healthy control (HC) children were assessed using clinical measures of psychopathology and a battery of neuropsychological tests. Resting-state, scalp-derived EEG was recomputed into current source densities using exact low-resolution brain electromagnetic tomography (eLORETA) to elucidate between-group differences in source activity and lagged cortical connectivity in 8 frequency bands. In addition, event-related potential (ERP)-P300 latency and the N200/P300 peak-to-peak amplitude between BDO and HCs were compared.

Results: The BDO group manifested significantly higher lifetime psychopathology compared to HCs, but there was no between-group differences in the N200 and P300 components of the cognitive ERP that corresponded to the absence of any differences between the groups in a wide range of neuropsychological domains. Compared to HCs, the BDO subjects showed significantly decreased alpha-1 sources in bilateral temporal gyri and subgenual cingulate and decreased beta-3 and gamma sources in the middle and posterior cingulate as well as in the mediofrontal areas. Moreover, the BDO group had significantly weaker alpha-1 lagged phase synchronization between the left and right temporal cortex and stronger beta-3 lagged phase synchronization between the right frontal and temporal areas.

Conclusions: This is the first EEG study to document reductions of intracortical activity and alteration in functional connectivity in offspring of BD parents. The reduced beta-3, gamma, and alpha-1 current sources together with changes in the frontal cortex might reflect the early neurophysiological trait markers of BD. It also emphasizes the importance of understanding a