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Novel Oppositional Defiant Disorder 6 months after Traumatic brain injury in children and adolescents

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Abstract

Objective: To assess predictive factors of novel Oppositional Defiant Disorder (ODD) in the first 6 months following traumatic brain injury (TBI).

Methods: Children aged 5 to 14 years who had suffered a TBI were recruited from consecutive admissions to five hospitals. Testing of a biopsychosocial model that may elucidate the

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development of novel ODD included assessment soon after injury (baseline) of pre-injury characteristics including psychiatric disorders, adaptive function, family function, psychosocial adversity, family psychiatric history, socioeconomic status (SES), injury severity, and post-injury processing speed which may be a proxy for brain injury. MRI analyses were also conducted to examine potential brain lesions. Psychiatric outcome including that of novel ODD was assessed 6 months after the injury.

Results: While 177 children were recruited for the study, 134 children without pre-injury ODD or conduct disorder (CD) or disruptive behavior disorder, not otherwise specified (DBD NOS) returned for the 6-month assessment. Of the 134 children, 11 (8.2%) developed novel ODD and none developed novel CD or DBD NOS 6 months post-injury. Novel ODD was significantly associated with SES, pre-injury family functioning, psychosocial adversity, and processing speed.

Conclusion: The results show that an important minority of children with TBI developed ODD. Psychosocial and injury-related variables, including SES, lower family function, psychosocial adversity, and processing speed significantly increase risk for these outcomes.

Keywords

Pediatric traumatic brain injury; Oppositional Defiant Disorder; prospective longitudinal study

Introduction

TBI in children and adolescents is a major public health problem in the United States with over 837,000 TBI-related emergency department visits, hospitalizations, and deaths occurring amongst children 17 years old and younger in 2014 alone {1}. New-onset post-injury psychiatric disorders, also termed novel psychiatric disorders, occur commonly, and have been studied with regard to their biopsychosocial predictors or correlates {2-7}. The current investigation, informed by a biopsychosocial model {8}, is the first prospective study of a consecutively recruited sample of children with TBI that examines DSM-IV-TR {9} post-injury onset of oppositional defiant disorder (ODD), conduct disorder (CD), or disruptive behavior disorder, not otherwise specified (DBD NOS) assessed 6-months post-injury. The latter disorder would meet criteria for “other specified disruptive, impulse-control, and conduct disorder” in DSM-5.{10} Our approach was to study children with any of these new-onset disorders as a single group, “novel ODD or CD or DBD NOS”, because of anticipated low incidence and phenomenological similarities. However, as will be revealed, at 6-months post-injury there were no cases of novel CD or DBD NOS. Therefore, for simplicity’s sake, our outcome of interest is termed novel ODD.

There are only two prospective longitudinal psychiatric standardized-interview pediatric TBI studies that have investigated novel ODD or novel CD symptomatology. One of the studies examined post-injury ODD symptom counts and change in ODD symptom counts in consecutively hospitalized children with mild to severe TBI (n=50) over the first two years post-injury {11}. The other study investigated symptom counts and categorical diagnoses of novel ODD and novel CD in a referred sample of inpatient rehabilitation center patients with severe TBI (n=94) one-year post-injury {3}. Despite their different designs these studies had overlapping first post-injury year findings implicating psychosocial risk factors (e.g.,

SES, pre-injury family function, psychosocial adversity, pre-injury ODD symptomatology, pre-injury aggression and delinquency), overlapping comorbidities (e.g., emotional lability and/or personality change due to TBI, novel ADHD) {12-15} and only one of the studies {11} had a potential biological risk factor, a smaller bicaudate ratio identified on the day-of-injury CT scan in exploratory analyses. Neither study found a significant relationship of first-year post-injury ODD with the lowest post-resuscitation Glasgow Coma Scale (GCS) score {16} which is the primary acute measure of brain injury severity.

The literature of pediatric TBI and novel ODD symptomatology is limited in several respects. Among the limitations are that 1) there are only two relevant studies including only one that studied consecutively treated children presenting with TBI; 2) the sample sizes were relatively small (<100); and 3) there were minimal data on a relationship between novel ODD and brain injury indices, including neuropsychological measures known to be sensitive to brain injury. The current investigation was designed to address these limitations. We therefore attempted to replicate the findings of a relationship of pre-injury psychosocial variables and novel ODD in a larger sample of consecutively treated injured children. In addition, we planned to study the relationship of novel ODD to the neuropsychological domain of processing speed which has been shown to be sensitive to brain injury in children {17}, in children with developmental ADHD particularly with prominent inattentive symptoms {18, 19}, and also sensitive to the broader category of novel psychiatric disorder after mild TBI {20}.

The following two hypotheses consistent with the existing literature were examined: 1.) Novel ODD will be significantly correlated with psychosocial adversity measures (SES, pre-injury psychosocial adversity score, pre-injury family function). 2.) Slower processing speed, a sensitive marker of brain damage, measured as soon as possible after TBI (baseline assessment), will be significantly associated with novel ODD independent of the presence of pre-injury ADHD. In related fashion, we hypothesized that processing speed, as a marker of brain damage, would be significantly associated with injury severity measured by the GCS. 3.) Given the rare nature of prospective longitudinal psychiatric studies of pediatric TBI, we performed exploratory analyses focused on the relationship of novel ODD with demographic variables (age, sex), other psychosocial variables (pre-injury adaptive function, family psychiatric history, pre-injury ADHD, pre-injury lifetime psychiatric disorder), comorbid novel internalizing psychiatric disorders (novel anxiety disorder and novel depressive disorder), and other injury variables (GCS, frontal lobe white matter/network lesions).

Methods

Recruitment:

In this study, 177 participants, who suffered a TBI between 1998 and 2003, were recruited between the ages of 5 and 14 from admissions to three academic medical centers in Texas (University of Texas, Houston; Baylor College of Medicine, Houston; University of Texas, Dallas); Rady Children's Hospital in San Diego, California; and The Hospital for Sick Children in Toronto, Canada. All hospitals recruited children with mild-to-severe TBI except in San Diego, where only complicated mild-to-severe TBI patients were included in the study. Children with preexisting autistic disorder or schizophrenia, intellectual disability,

and injury due to child abuse or penetrating-missile injury were not included in the study. In San Diego only, children were excluded if they had preexisting ADHD. Because parents/guardians of children were not required to answer eligibility questions before deciding to participate in the study, data regarding the number of children approached, the proportion eligible for recruitment, and participation rate of those who were eligible for recruitment are missing. As required by the Institutional Review Boards, all children signed assent or consent forms to participate in the study, and their legal guardians provided informed consent. Demographic information, pre-injury psychosocial variables and injury indices for participants studied at the 6-month follow up are shown in table 1.

Measures

PSYCHOSOCIAL ASSESSMENTS

Psychiatric Outcome (Novel ODD) and Psychiatric Predictor and Mediator Variables—Our outcome psychiatric measure of novel ODD as well as several other potential pre-injury psychiatric predictor variables (pre-injury ADHD, pre-injury lifetime psychiatric disorder), and concurrent novel psychiatric disorder mediator variables (novel anxiety disorder, novel depressive disorder) were derived using the *Schedule for Affective Disorders and Schizophrenia for School-Age Children, Present and Lifetime Version (K-SADS-PL)* {21} and *Neuropsychiatric Rating Schedule (NPRS)* {22}, DSM-IV-TR psychiatric diagnoses were made. To record pre-injury diagnoses, these interviews were carried out at baseline (after resolution of posttraumatic amnesia) and were repeated six months post-injury to record any new diagnoses that may have developed. The K-SADS-PL, developed to make diagnoses in both children and adolescents based on DSM-IV-TR criteria, is a semi-structured, integrated parent/child interview. While the NPRS is structured similarly to the K-SADS-PL, it is more specific in that it assesses for personality change due to TBI. The last author trained all of the interviewers, Master's- and Ph.D.- level clinicians, in a pre-study workshop and a mid-study workshop using videos of his research interviews and written vignettes. Four sites had a child psychiatrist supervising the assessments, and one site had a child psychologist. In addition to this supervision, the last author reviewed written summaries organized by the interviewer and also held monthly teleconferences with the interviewers to discuss the cases. The study was centered on the main questions, which were regarding present and lifetime symptoms and timing of the onset of these symptoms in relation to the TBI. Novel ODD was recorded if the child had no pre-injury disorder but later developed ODD after the injury. Novel ODD could also occur in circumstances where the child developed the disorder but had a different pre-injury psychiatric disorder such as generalized anxiety disorder or ADHD.

Socioeconomic Status—The *Four-Factor Index* measured SES {23}. Scores from this index result from a formula that accounts for the educational and occupational levels of both the mother and father. The scores range from 8 to 66, with a higher score representing a higher SES.

Family Function—The *Family Assessment Device General Functioning Scale (FAD)* was used to measure global family functioning {24}. The child's primary caretaker completed

this scale, consisting of 12 questions, each on a 4-point scale, with lower scores representing healthier family functioning. Scores in families of nonclinical, psychiatric, and medical probands were 1.89 (.43), 2.27 (.51), and 1.89 (.45) respectively {24}.

Psychosocial Adversity—The *Psychosocial Adversity Measure* used was very similar to that used in a seminal study of pediatric TBI {7}. The areas of adversity assessed were: (1) child not living with biological or adoptive parents, (2) sibship of at least four children or a person-to-room ratio exceeding 1, (3) family difficulties leading to admission of the child into local authority care, (4) maternal ‘‘malaise inventory’’ score of 7 or more, (5) paternal criminality, and (6) father or mother with an unskilled or semiskilled job. For each area, a score of 1 was given for adversity, and 0 for no adversity.

Family Psychiatric History—The *Family History Research Diagnostic Criteria* interview was done by trained research assistants {25, 26}. In the interview, at least one parent for each child answered questions that were aimed at documenting the presence and severity of psychiatric disorders in the child’s first-degree relatives. Scores range from 0 to 3 with increasing severity.

Adaptive Function—The *Vineland Adaptive Behavior Scales* were used to measure adaptive functioning {27}. This assessment, done with the child’s primary caretaker, is a nondirective interview that accounts for the kinds of behaviors a child displays in his or her environment and then provides an overall adaptive-behavior composite standard score (mean +/− standard deviation is 100 +/− 15).

NEUROPSYCHOLOGICAL ASSESSMENT

The *Wechsler Intelligence Scale for Children (3rd Edition)* Coding and Symbol Search subtests were used to measure processing speed {28}. In the Coding subtest, children are required to transcribe the correct geometric designs below numbers guided by a key. The number of symbols transcribed correctly in 2 minutes was measured. The Symbol Search subtest required the child, when presented with target stimuli, to check a “yes” or “no” box as quickly as possible to indicate whether or not the target or targets appeared among the presented stimuli (45 total trials). The Symbol Search score was the number of correct responses minus the number of errors completed in 2 minutes. A scaled Processing Speed score was obtained and averaged for both subtests.

NEUROLOGICAL ASSESSMENTS

The *Glasgow Coma Scale* (GCS) was used to assess the severity of the children’s brain injuries {16}. The GCS, which is the standard measure of brain injury severity, has three different score ranges: severe, moderate, and mild. The score ranges for each classification, respectively, are: 3-8, 9-12, 13-15.

MRIs (1.5T) were conducted in most of the subjects about 3 months after their injuries. The procedure consisted of a T1 volumetric spoiled gradient-recalled echo (1.5 mm slices) and fluid-attenuated-inversion recovery sequences (3 mm slices), which were obtained in coronal and sagittal planes based on a research protocol followed by all sites. At each

site, a neuroradiologist coded the different lesions from the multiple-slice, hard-copy films. Anatomical location was coded from a list of brain structures, among which were white matter, cortical gray matter (frontal, temporal, parietal, occipital), and subcortical gray matter (thalamus, basal ganglia) {12}. Because expert neuroradiologists coded the lesions, and volumetric analyses were not done, images were not registered and tissue types were not segmented.

Statistical Analyses—To test the relationship of 6-month novel ODD with the hypothesized continuous and categorical predictors, logistic regression univariable analyses were conducted. The association between processing speed and injury severity (GCS) was assessed via Pearson's correlation coefficient and tested using the t-test of correlation. To shed light on the relative importance of variables significantly associated with novel ODD, a stepwise logistic regression analysis was performed with ODD as the dependent variable. The independent baseline predictors were included in the model using backwards model selection with a $p<.015$ inclusion criterion using the likelihood ratio test. Statistical significance was considered at level $\alpha=0.05$. All tests were two-sided. The analyses were conducted in SPSS.

Results

Occurrence

Of the original 177 children, 11 were excluded from the analyses because of their pre-injury ODD (n=7 including 3 resolved), CD (n=2), and DBD NOS (n=2) precluded them from developing a novel ODD/CD/DBD NOS. Returning children (n=134) of the remaining 166 eligible children (80.7%) were assessed 6-months post-injury. There was no difference between the returning children and those who did not return with respect to age at injury, sex, SES, race, psychosocial adversity, pre-injury family function, injury severity, pre-injury lifetime psychiatric disorder, pre-injury anxiety disorder, pre-injury depressive disorder, and pre-injury ADHD. Those lost to follow up had significantly lower pre-injury adaptive function standard score (89.6 +/− 18.8; n=28 versus 96.1 +/− 14.4; n=126; t=−2.0; df=152; p=.045) and significantly lower baseline post-injury processing speed standard score (90.5 +/− 18.4; n=24 versus 99.5 +/− 19.1; n=115; t=−2.1; df=137; p=.036). Eleven of the 134 children (8.2%) developed novel ODD. There were no cases of CD or DBD NOS and therefore we shall hereafter refer to the novel disorder of interest as novel ODD rather than novel ODD/CD/DBD NOS.

Psychosocial and Neuropsychological Correlates of Novel ODD

Table 2 shows the relationship of psychosocial variables and novel ODD. Logistic regression analyses demonstrated that SES (OR=0.900; 95%CI [0.846, 0.958]; p<.0005), pre-injury family function (OR=1.117; 95%CI [1.016, 1.228]; p=.024), and psychosocial adversity score (OR=2.128; 95%CI [1.217, 3.720]; p=.008) were significantly associated with novel ODD. These results support hypothesis 1 which predicted novel ODD to be significantly associated with psychosocial variables.

Processing speed assessed at the first post-injury assessment, within 2 weeks after injury, was significantly associated with novel ODD (OR=0.959; 95%CI [0.922, 0.998]; p=.031) (Table 2). Hypothesis 2, which predicted that the significant association of novel ODD with processing speed would be independent of the presence of developmental (pre-injury) ADHD, was tested. A backward stepwise likelihood ratio logistic regression analysis with novel ODD as the dependent variable and processing speed and pre-injury ADHD as independent variables was conducted. The regression produced a significant final model (χ^2=4.64; *df*=1; *p*=.031) which included processing speed (Wald χ^2=4.23; *df*=1; *p*=.040) supporting hypothesis 2. The bivariate correlation of processing speed and GCS (injury severity) was significant (Pearson's r=.37; n=134; p<.0005).

As planned, a backward stepwise likelihood ratio logistic regression was conducted with novel ODD as the dependent variable and the independent variables were comprised from baseline assessment measures that were associated with novel ODD in univariable analyses at the p<.15 level (SES; psychosocial adversity score; pre-injury family function; processing speed standard score). The regression produced a significant final model (χ2=22.469; *df*=2; *p*<0.0005) which included lower SES (Wald χ2=9.178; *df*=1; *p*=.002) (OR=0.850; 95%CI [0.766, 0.944]; *p*<0.0005) and lower WISC3: Processing Speed Scale Score (Wald χ2=4.146; *df*=1; *p*=.042) (OR=0.944; 95%CI [0.892, 0.998]; *p*=.023). This is notable given the known generally significant relationship of processing speed with SES {29}.

Exploratory Analyses

The planned exploratory analyses with respect to novel ODD are shown in table 3. Novel ODD was not significantly related to demographic variables (age, gender, race), family psychiatric history, pre-injury lifetime psychiatric disorder, pre-injury ADHD, novel anxiety disorder, and injury variables (GCS score, presence of a frontal lobe white matter lesion on MRI). There was a trend noted with regard to the association of pre-injury adaptive function and novel ODD (Wald χ2=3.116; df=1; p=.078; OR=0.955; 95%CI [0.908, 1.005]; p=.065). There was also a trend association found between novel depressive disorder and novel ODD with 2/11 (18%) children with novel ODD exhibiting novel depressive disorder versus 4/123 (3%) children with no novel ODD exhibiting novel depressive disorder (Wald χ2=3.992; df=1; p=.046; OR=6.444; 95%CI [1.036, 40.088] p=.073).

Post-injury Outcome for Children with Pre-injury ODD/CD/DBD NOS

Because the effect of TBI on children with pre-injury ODD/CD/DBD NOS is of interest to clinicians and researchers, these data are provided. Three of the 4 children with unresolved pre-injury ODD continued to manifest ODD, although the ODD of 1 of the children remitted partially. The fourth child with unresolved pre-injury ODD did not return for the 6-month assessment. Two of the 3 children with resolved pre-injury ODD remained free of ODD at the 6-month assessment, and the third child did not return for the assessment. The pre-injury CD of one child resolved, and the second child with pre-injury CD did not return. Similarly, pre-injury DBD NOS of one child resolved, and the second child with pre-injury DBD NOS did not return for the 6-month assessment.

Discussion

The main findings from this study are that new-onset ODD, also called novel ODD, occurs in the first 6 months after TBI in children and adolescents, and that it appears to have rather robust biopsychosocial clinical correlates which generally coincide with but expand findings from the very few related previous studies. Specifically, novel ODD occurred in 8% of children aged 5-14 years at the time of injury and was significantly correlated with pre-injury psychosocial risk factors (low SES, psychosocial adversity, low family function), and injury severity-associated slow processing speed measured in the early weeks after TBI.

The incidence of novel ODD was similar to that reported at the 12-month follow up of a sample of patients treated consecutively at a rehabilitation center (8% versus 9%). This compatible finding is remarkable given the important differences in the studies. The respective differences between the current study and the earlier study include 1) consecutively hospitalized patients for TBI versus patients with TBI consecutively treated at a rehabilitation center; 2) range of severity mild to severe versus severe TBI only; and 3) use of impairment criteria to define ODD versus using symptom counts without impairment criteria. An important difference between the studies was that the present study found no cases of novel conduct disorder while the earlier study found the rate of novel conduct disorder to be 8%. The reason for this difference is unclear although we think it is most likely related to methodological differences in applying impairment criteria.

The association of novel ODD with pre-injury psychosocial variables (hypothesis 1) is a consistent characteristic across all related studies {3}. The current study found novel ODD was significantly associated with lower pre-injury SES, higher pre-injury psychosocial adversity, and lower pre-injury family function. Novel ODD in the inpatient rehabilitation sample was significantly associated with psychosocial adversity in univariable analyses; however, in that study, only pre-injury special education status was significant in multivariable analyses {3}. Our earlier study of consecutively hospitalized children with mild to severe TBI examining ODD symptoms post-injury rather than novel ODD found that total ODD symptoms 6-months post-injury were significantly related to pre-injury family function, pre-injury ODD symptom count, and SES in a regression analysis {11}. A closer comparison of our earlier study with the current study was the examination of change in ODD symptom count from pre-injury to 6-months post-injury which was significantly associated with only SES in a regression analysis {11}.

Consistent with hypothesis 2, novel ODD was associated with slower processing speed. This association remained significant following a regression analysis that controlled for the presence of pre-injury ADHD. This is the first time that a significant neurocognitive association of novel ODD has been demonstrated in a pediatric TBI cohort, and this finding is not surprising given neurocognitive differences in children with and without ODD in uninjured cohorts {30}. The finding is intriguing because processing speed was significantly correlated with brain injury severity (GCS score) and has been shown in other studies to be sensitive to brain injury {31}. Therefore, it may be that relatively crude clinical measures of injury severity (GCS score) and macroscopic lesions on structural imaging, neither of which were significantly related to novel ODD, are less sensitive than this neurocognitive

measure in reflecting brain damage. Regression analysis demonstrated that novel ODD was significantly and independently associated with both processing speed and SES and therefore the finding could not be attributed to the known association between processing speed and SES {29}. Therefore, these findings may underscore the role of psychosocial variables (e.g., SES) and biological variables (e.g., brain injury-related slower processing speed) in the presentation of novel ODD at 6-months post-injury. It is this biological variable that is a new finding because in neither of the previous studies was novel ODD, ODD symptoms, or change in ODD symptoms at 6-months post-injury related to severity of injury {3, 11}. Nevertheless, one of the earlier studies {11} found a significant negative correlation of change in ODD symptoms and the "bicaudate ratio" recorded from the day-of-injury CT scan, and this was presumed to reflect brain parenchymal edema and a degree of ventricular compression which may be associated with eventual damage to frontal lobe structures and connections possibly implicated in the pathophysiology of ODD.

It is notable that while distal (family) psychosocial measures such as SES, psychosocial adversity, and family function were significantly related to novel ODD, the only proximal (child) pre-injury psychosocial variable that even approached significance was pre-injury child adaptive functioning. It will be of interest in longer-term follow up of this and other cohorts whether pre-injury adaptive function as a measure of behavioral "reserve" akin to the concept of "cognitive reserve" {13, 32} will be predictive of later or chronic novel ODD outcome.

Exploratory analyses of novel ODD and comorbid novel psychiatric disorders found a trend association with novel depressive disorder limited possibly by insufficient power. There was no association with novel anxiety disorder. However, as we have noted in previous reports from this cohort that focused on personality change due to TBI {12, 13} and review of the literature, there is extensive agreement across existing studies with regard to comorbidity of novel ODD or ODD symptoms and emotional lability captured categorically with the diagnosis of Personality change due to TBI or continuously with specific questionnaire scales {3}. There is similar agreement across studies, including previous novel ADHD-focused reports from the same cohort studied here {14, 15}, regarding the association of novel ODD or ODD symptoms and novel ADHD or ADHD symptoms {3, 11}. This is not surprising given that emotional lability, ADHD, and ODD are typically related in non-TBI samples {33}. Despite these significant comorbidities, novel ODD and personality change following TBI at 6-months post-injury, as well as novel ODD and novel ADHD 6-months post-injury have incomplete overlap with regard to their respective statistically significant clinical correlates {2, 12, 15, 34-37}. Specifically, personality change following TBI at 6-months post-injury is related to severity of injury and dorsal frontal lobe lesions, but not to psychosocial variables. Furthermore, novel ADHD or change in ADHD symptoms are often related to indices of injury severity or specific lesions such as orbitofrontal gyrus lesions or putamen lesions, in addition to psychosocial variables {15, 34, 35}.

There were several limitations in study methodology that are important to acknowledge. First, we did not include a non-brain related injury control group to compare to the TBI group. Without this control group, it is difficult to establish a causal pathway between brain injury in children and development of ODD. Second, we did not directly test

interrater reliability for psychiatric diagnoses within and across testing sites. However, there were specific procedures of quality control and training as described in the Methods section to mitigate this issue. Third, image analyses did not include volumetric or tissue-segmentation measurements. Fourth, sample attrition was approximately 20% with children who had lower post-injury baseline processing speed and lower pre-injury adaptive function being less likely to return for their 6-month assessment. These variables were associated respectively with a significant and trend level association with novel ODD and therefore it is possible that our findings would have been even more robust had they participated. However, the participants and non-participants were no different on multiple demographic variables such as age, sex, race, SES, pre-injury psychosocial adversity, pre-injury family function, pre-injury psychiatric status, and injury severity. Fifth, diagnoses were determined using the DSM-IV-TR, the version that was current at the time of the study, rather than DSM-5; however the classification of Oppositional Defiant Disorder, including meeting at least four of eight criteria to qualify for ODD, did not change between the two versions aside from minor semantic differences {38}. Sixth, potential variability in the natural history of post-injury treatment-seeking by the families of participants could influence outcome. Finally, this study is limited to only measuring the impact of TBI at 6 months post-injury as opposed to multiple time points as some other studies have done. It is unclear from this report taken in isolation as to the persistence or lack thereof of the TBI impact on novel ODD outcome noted here.

There are several notable strengths of this study. Perhaps most importantly, this study fills a gap in the literature in that it is the only prospective TBI study of novel ODD to use a semi-structured psychiatric assessment to make a diagnosis that requires clinical judgment to document impairment. The breadth and depth of assessments were extensive and included interview assessments of adaptive functioning, family psychiatric history, and psychopathology. In addition, this study accounts for pre-injury diagnoses assessed by semi-structured interviews in all study participants. The documentation of pre-injury diagnoses is vital for measuring novel psychiatric outcomes. Furthermore, expert neuroradiologists coded the lesions to ensure accurate brain imaging results, despite lesion correlates being a negative finding. Finally, this study examined children with TBI ranging in severity from mild to severe versus severe TBI only, making the results more generalizable to a wider pediatric TBI population.

In conclusion, clinically-significant novel ODD occurs as a post-injury complication in a small (8%) but important proportion of children and adolescents who were consecutively hospitalized for mild to severe TBI. Novel ODD was significantly associated with pre-injury psychosocial risk factors (lower SES, higher psychosocial adversity, lower family function) as well as injury-severity associated slower processing speed documented in the first-weeks post-injury. A key implication of our biopsychosocial risk factor findings is that children who are at higher risk for developing novel ODD may be identified soon after injury and surveilled for the purposes of mitigating this specific adverse outcome.

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38. DSM-IV to DSM-5 Oppositional Defiant Disorder Comparison. Rockville, MD: Substance Abuse and Mental Health Services Administration; 2016. p. Table 18.

Table 1.

Data of Demographic, Psychosocial, And Injury Variables Among Children Assessed 6-Months After Traumatic Brain Injury (n=134)

Demographic Variables		
Age at Injury (mean years +/− SD)	10.15	2.83
Gender: male, N (%)	92	68.7
Socioeconomic Status (mean +/− SD)	37.60	12.61
Psychosocial Variables		
Pre-injury Lifetime Psychiatric Disorders, N (%)	35	26.1
Pre-injury Vineland Adaptive Behavior Composite Score (mean +/− SD)	96.10	14.43
Pre-Injury Family Assessment Device Score (mean +/− SD)	1.63	.50
Injury Variables		
Glasgow Coma Scale Score (Lowest Post-resuscitation) (mean +/− SD)	10.78	4.23
Glasgow Coma Scale Score, N (%)		
3-8	51	38.1
9-12	17	12.6
13-15	66	49.3

Legend: SD = standard deviation

Table 2.

Psychosocial and Neuropsychological Correlates of Novel ODD

	Novel ODD N=11			No Novel ODD N=123			OR	95% CI	p
Socioeconomic Status (mean +/− SD)	24.0	10.6		38.8	12.1	n=121	0.900	0.846, 0.958	<.0005
Pre-injury Family Functioning (mean +− SD)	1.99	.63	n=10	1.59	.47	n=116	1.117	1.016, 1.228	.024
Pre-injury Psychosocial Adversity score (mean +/− SD)	1.64	1.36		.75	.92	n=118	2.128	1.217, 3.720	.008
Baseline Processing Speed standard score (mean +/− SD)	86.7	18.0	n=9	100.6	18.4	n=106	0.959	0.922, 0.998	.031

Legend: The values are expressed for children with novel ODD (n=11) and for children with no novel ODD (n=123) unless otherwise indicated due to missing data.

Table 3.

Relationship of Demographic, Family Psychiatric History, Adaptive Function, Psychiatric Diagnoses, and Injury Variables with Novel ODD

	Novel ODD N=11			No Novel ODD N=123			OR	95% CI	p
Age at Injury mean (SD)	9.3	2.7		10.2	2.8		0.890	0.710, 1.116	NS
Sex (male), N (%)	9	82%		83	67%		0.461	0.095, 2.234	NS
Race									NS
White, N (%)	5	45%		69	56%		1		
Hispanic, N (%)	4	36%		23	19%		2.400	0.594, 9.702	NS
Black, N (%)	1	9%		22	18%		0.627	0.070, 5.661	NS
Asian, N (%)	1	9%		3	2%		4.600	0.402, 52.693	NS
Other, N (%)	0	0%		6	5%		-	-	NS
Family Psychiatric History mean (SD)	1.45	1.13		1.04	1.06	n=104	1.328	0.745, 2.366	NS
Pre-injury Adaptive Functioning mean (SD)	88.3	14.5	n=10	96.8	14.3	n=116	0.955	0.908, 1.005	.065
Pre-injury Lifetime Psychiatric Disorder, N (%)	3	27%		32	26%		0.938	0.234, 3.752	NS
Pre-injury ADHD, N (%)	3	27%		20	16%		0.518	0.126, 2.122	NS
Novel Anxiety Disorder, N (%)	1	9%		10	8%		0.885	0.103, 7.635	NS
Novel Depressive Disorder, N(%)	2	18%		4	3%		6.444	1.036, 40.088	.073
Injury Variables									
Glasgow Coma Scale score mean (SD)	10.2	4.6		10.8	4.2		0.965	0.836, 1.114	NS
Frontal White Matter Lesion, N (%)	2	18%		25	22%	n=113	0.782	0.159, 3.856	NS