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Investigating the frequency of tick-borne infections in a case series of 37 youth diagnosed with pediatric bipolar disorder

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Introduction: This retrospective chart review examined 37 youth with pediatric bipolar disorder from a private practice in the Lyme-endemic state of New Jersey, expanding on findings from 27 previously reported cases to explore the potential contribution of tick-borne infections to disease etiology.

Methods: Diagnoses were based on DSM-IV-TR and DSM-V criteria using parent and child interviews, questionnaires, and school reports. Initial screening evaluated for possible PANDAS/PANS, with testing for Group A beta-hemolytic streptococcus, *Borrelia burgdorferi*, *Babesia*, *Bartonella*, and *Mycoplasma pneumoniae*. Lyme disease testing included ELISA, Western Blot (IgM/IgG), and immunoblots, interpreted per CDC guidelines. Other pathogens were assessed via IgM/IgG titers, anti-streptolysin O, anti-DNAase B, fluorescent *in situ* hybridization, and blood cultures. A positive diagnosis required both laboratory evidence and clinician confirmation.

Results: *Babesia* was detected in 51% (19/37), *Bartonella* in 49% (18/37), *Mycoplasma pneumoniae* in 38% (14/37), *Borrelia burgdorferi* in 22% (8/37), and Group A Streptococcus in 19% (7/37). Overall, 92% (34/37) had evidence of tick-borne exposure, with 81% (30/37) meeting both laboratory and clinical criteria.

Discussion: More than three-quarters of the cohort demonstrated confirmed tick-borne infections. Overlaps between bipolar disorder and tick-borne illness—such as immune dysregulation, chronic symptomatology, and responsiveness to treatments like minocycline and anti-inflammatory agents—support further exploration of infectious contributors to pediatric bipolar disorder. While limited by its single-practice retrospective design, these findings suggest that tick-borne pathogens may play a role in the pathogenesis of bipolar symptoms in youth, warranting larger, controlled studies.

KEYWORDS

pediatric bipolar disorder, tick-borne illness, lyme disease, *Babesia*, *bartonella*

Introduction

Recent decades have brought increased recognition of pediatric bipolar disorder (PBD) as a valid clinical diagnosis (1, 2). Concurrently, the rising incidence of tick-borne infections (TBIs) has prompted growing concern regarding their potential neuropsychiatric and neurocognitive sequelae (3, 4). Parallel to these developments, a substantial body of research has underscored the critical role of immune dysregulation

and inflammatory processes in the pathogenesis of a broad range of psychiatric conditions, including schizophrenia, major depressive disorder, bipolar disorder, anxiety disorders, obsessive-compulsive disorder (OCD), and attention-deficit/hyperactivity disorder (ADHD) (5–17). Some of the strongest evidence has been in the area of mood disorders, both depression and bipolar disorder. A wide array of infectious agents—including bacterial, viral, fungal, and parasitic pathogens—have been implicated as potential initiators of systemic and neuroinflammatory responses. Previous research has specifically identified associations between certain infectious agents—such as *Toxoplasma gondii*, and *Cytomegalovirus* (CMV)—and the development of bipolar disorder (18). Given the growing evidence linking immune activation to psychiatric symptomatology, elucidating the role of underlying infectious triggers represents an important area for ongoing investigation (19, 20).

Another relevant factor when discussing infections and behavioral/emotional change involves breakdown of the blood brain barrier, normally a gatekeeper and filter protecting the brain from toxic substances carried in the circulation. When compromised, pathogens and toxic substances as well as peripheral immune and inflammatory agents such as cytokines and chemokines normally excluded, can enter the central nervous system. Their entry can subsequently interfere with neuronal function, create neuroinflammation and change neurotransmission (21).

Support for the infection-immune hypothesis in childhood neuropsychiatric illness is seen in the clinical recognition of Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal infections (PANDAS) (22). It is a syndrome first described by Swedo and colleagues in 1998. The cardinal clinical presentation involves the abrupt onset or dramatic exacerbation of obsessive-compulsive disorder symptoms and/or multiple, complex or unusual tics in children, following infection with Group A beta-hemolytic Streptococcus (GABHS) infection. The course is relapsing and remitting and associated with neuropsychiatric symptoms. Subsequently, the broader diagnostic category of Pediatric Acute-onset Neuropsychiatric Syndrome (PANS) was introduced to describe cases of sudden-onset OCD or severely restricted food intake without requiring a documented preceding streptococcal infection (23). Like PANDAS, PANS can encompass a range of additional neuropsychiatric symptoms, including anxiety, emotional lability, irritability, oppositional behavior, aggression, behavioral/developmental regression, psychotic symptoms, sensory sensitivities, sleep disturbances and urinary disturbances, and can result in the development of arthritic joints and additional autoimmune diseases (24). Studies indicate that PANDAS and PANS are due to an autoimmune encephalopathic process in the basal ganglia (25).

Recent case reports have documented children and adolescents who presented with a variety of psychiatric syndromes that appeared to be resistant to standard treatments, including bipolar disorder, schizophrenia, severe obsessive-compulsive disorder, restrictive eating behaviors, functional neurological symptom disorders, severe anxiety, and cognitive

regression (26–28). In these cases, the identification of potential underlying contributors—such as occult infections (e.g., group A streptococcal infections, tick-borne diseases, viral, fungal, and parasitic infections), micronutrient deficiencies, mycotoxin exposure, and gastrointestinal dysbiosis—followed by targeted interventions, was associated with marked clinical improvement.

Against this background, the clinical features of PANDAS/PANS overlap significantly with those observed in pediatric bipolar disorder (PBD), a mental illness characterized by episodic changes in mood, alterations in energy, sleep disruption, cognition, and behavior that impair functioning and can be associated with illness chronicity and significant lifetime morbidity (29).

This paper explores the potential role of tick-borne pathogens including species of *Borrelia*, *Bartonella*, *Babesia* and *Mycoplasma*, in the development or exacerbation of neuropsychiatric symptoms, particularly in the pediatric population.

The first of these illnesses is Lyme disease (LD), the most frequent vector-borne illness in North America and Europe. In the United States, it is caused by the bacteria *Borrelia burgdorferi* and estimated to be responsible for as many as 476,000 new cases each year (30, 31). Approximately 25% of new LD cases occur in youth ages 19 and under, which translates to approximately 119,000 youth/year (32). An Ixodes scapularis tick (the vector) that initiates the infection through its bite may or may not carry *Borrelia* bacteria, but it is also capable of injecting multiple other pathogens into its target. These can include *Babesia*, *Bartonella*, *Ehrlichia*, *Anaplasma*, *Mycoplasma* species, viruses and other noxious agents. Basically, the tick bite serves as a dirty needle for spread of potentially pathogenic materials.

Historically, discussion of symptoms caused by infectious diseases has focused on the more obvious physical manifestations rather than the effect on mental processes. Recognition that the immune system through mechanisms such as inflammation, immune dysfunction, breakdown of the blood brain barrier, and autoimmunity plays an important role in the development of psychiatric illness and not solely physical disorders, is a rapidly expanding area of scientific study (33). The medical literature refers to the association of microbes and mental illness usually in case reports, and only infrequently focuses on a specific psychiatric illness (20).

This article examines two diagnostic areas, PBD and TBIs, both of which are characterized by unclear symptoms, limited diagnostic methods, and frequent challenges in treatment. According to the American Academy of Pediatrics, around 1%–2% of all U.S. children under 18 have bipolar disorder (34). Other estimates have been higher and up to 5% if one includes the category of bipolar disorder not otherwise specified (35).

The childhood onset of bipolar disorder is associated with significant dysfunction and is a pernicious illness (36–38). It is also a highly heritable disorder. A review of family studies of mood disorders in relatives of individuals with early-onset bipolar disorder indicate these youth have a higher rate of bipolar disorder than relatives of later-onset case, indicating a larger genetic contribution in early-onset forms (39). The risk of

bipolar disorder (15%–42%) in first-degree relatives of children with bipolar disorder are consistently larger than the 8.7% estimate of recurrence risk of bipolar disorder in first-degree relatives of adult bipolar disorder cases (40). Genetics is felt to account for 60%–85% of the risk, leaving 15%–40% likely due to other factors. Additional influences include environmental, infectious, immune, and psychosocial factors (41). Multiple adversities early in life can more than double the child's risk of developing bipolar disorder (41). As noted by Post et al. higher genetic loading, increased incidence of childhood abuse and adversity, significant obesity, eating a high inflammatory diet, and limited healthcare access have been factors that contribute to the disorder being a greater problem in the U.S. compared to other countries (42–44).

PBD youth spend more than half (up to 2/3) of their lives struggling in a state of illness, having profound effects on development and the individual's future. It is also important to remember in terms of long-term morbidity that up to 1/3 of children and teens diagnosed with depression in the United States may be experiencing the early onset of PBD (45).

Identifying specific genetic, infectious, and immune vulnerabilities has the potential to identify preventable causes of PBD. Therefore, elucidation of the connection of PBD and TBIs, has the potential to prevent and treat a great deal of illness and suffering.

Rationale for screening pediatric psychiatric patients for a variety of bacterial (including streptococcus bacteria), viral and parasitic pathogens which could be associated with PANDAS or PANS came from the author's observation of the presence of these infections in some very ill and ostensibly treatment resistant psychiatric patients.

The goal of the present study is to further examine the hypothesis that TBIs play a yet unrecognized role in the etiology of some cases of pediatric bipolar disorder (PBD). This retrospective chart review adds an additional 10 pediatric bipolar subjects to 27 youth previously reported on from this author's New Jersey child psychiatry practice [Greenberg, R. (2017). Infections and childhood psychiatric disorders: Tick-borne illness and bipolar disorder in youth. *Bipolar Disorder Open Access*, 3, 113. <https://doi.org/10.4172/2472-1077.1000113>] (46).

Methods

In this observational study, a retrospective chart review of 37 youth from a single Northeast U.S. practice in New Jersey, considered by the Center for Disease Control and Prevention as a Lyme and Babesia endemic state, was performed. Rates of exposure to tick-borne pathogens were examined in youth meeting criteria for Bipolar I or II by DSM-IV TR (2000) or DSM-V (2013) (47, 48). The primary difference between the editions is the addition of heightened energy as a criterion for mania in DSM-V, which did not create meaningful divisions between the cohorts. The first chart review of 27 children, conducted prior to the COVID-19 pandemic, used DSM-IV TR, while the second cohort, assessed post-pandemic, used DSM-5V

criteria for psychiatric diagnosis. Diagnostic evaluation was consistent across cohorts, with the first assessed in person and the second via a combination of telepsychiatry and in-person visits based on post-COVID practices. The approach utilized in this study was an aggregate data analysis of 37 cases.

All patients underwent a full assessment which included details of the presenting problem, pregnancy, birth and developmental history, school history, social history, first, second and third degree of family history of autoimmune disorders, neurological disorders, cardiovascular disease, medical illnesses, as well as mental disorders including depression, bipolar disorder, anxiety disorders, obsessive compulsive disorders, autism, schizophrenia, ADHD, eating disorders, alcohol abuse, substance abuse, suicide attempts or completed suicide. Inclusion criteria required that patients had received a diagnosis of PBD and had undergone laboratory testing for evidence of exposure to *Streptococcus species*, *Borrelia*, *Bartonella*, *Mycoplasma pneumoniae*, or *Babesia* spp. Exclusion criteria included patients with alternative primary psychiatric diagnoses and those who had not undergone pathogen testing.

Patient blood samples were evaluated for exposure to:

1. *Borrelia burgdorferi*, the causative agent of LD. Testing included Immunofluorescent Antibody testing (IFA), Enzyme-Linked Immunosorbent Assay (ELISA), Western Blot Immunoglobulins M and G antibody levels, immunoblots and culture techniques. Center for Disease Control and prevention criteria was used for diagnosis.
2. *Babesia*, *Bartonella*, and *Mycoplasma pneumoniae*, pathogens that are all potentially carried by ticks. IgM/IgG antibody titers and in some cases fluorescent *in situ* hybridization (FISH) testing were utilized.
3. *Group A Beta Hemolytic Streptococcal* bacteria. Even though ticks are not known to carry this type of bacteria, the information in this paper was obtained as part of a PANDAS/PANS workup. Streptococcal throat cultures, measurement of anti-streptolysin O titers and anti-strep DNAase B titers were reviewed. Having a PANDAS with comorbid TBI's may be important in illness presentation, course, and treatment.

Laboratories used: LabCorp, Quest Diagnostics, Mayo Medical, IgeneX Laboratory, Advanced Labs, and Galaxy Diagnostics.

Individuals with positive laboratory results were referred for medical evaluation. Clinical confirmation of tick-borne infections (TBIs) was conducted by physicians from diverse specialties, including pediatrics, family medicine, infectious disease, immunology, and rheumatology. All participating clinicians had prior experience with symptom pattern recognition and assessing treatment response in the context of TBIs. Final diagnoses were based on a comprehensive review of the patient's medical history, physical examination findings, and laboratory results.

Noted was the family history of bipolar disorder and depression in the family history of the probands, which included siblings, parents and grandparents, given the issue of heritability.

Results

Characteristics of the group are seen in Table 1.

The mean age of the 37 subjects was 7.9 years with 27% (10/37) females and 73% (27/37) males. Diagnostically using DSM IV TR and DSM V 57% (21/37) met the criteria for bipolar disorder I and 43% (16/37) were considered bipolar disorder II.

In only three cases of the 37 was there a history of a known tick bite, making this exposure a previously unknown occurrence in at least 31 of the 34 cases (91%) that were positive on testing.

Given that the data was from a private practice and not designed as a clinical study, the timing of the TBI testing relative to the bipolar diagnosis depended upon clinician judgment.

Of note 92% (34/37) of PBD youth were positive on blood testing for TBI exposure. Since this was a case series, there was no standardized protocol for evaluating tick-borne illness (TBI). Over 10 different clinicians conducted assessments based on the recommendation of the author or patient's existing provider or parental choice. Evaluations relied on clinician experience, general medical guidelines, and pattern recognition, reflecting real-world variability in care. Despite this heterogeneity, 88% (30/34) of lab-positive patients were clinically confirmed as having a TBI. Of the four-remaining lab-positive cases two were

considered negative by clinician evaluation while the other two families did not wish to seek a consultation. Discordant cases were recorded but not adjudicated.

Several individuals tested positive for more than one pathogen, which is why the cumulative percentages for each pathogen total 179% (Table 2). Specifically, 59% of those with positive results (20/34) had multiple tick-borne infections. All 34 patients with positive testing were referred for clinical evaluation; in two cases, parents declined further assessment. Additionally, two cases were not confirmed as positive after physician review. Overall, clinical evaluation—including history, physical examination, and laboratory results—confirmed TBIs in 81% of the total sample (30/37). Two patients had begun treatment for TBIs prior to receiving a PBD diagnosis. Observing that several newly diagnosed bipolar patients had TBIs led to extending testing to older patients with established PBD, with positive results occurring up to 12 years after the initial psychiatric diagnosis.

Discussion

In this study greater than 75% of the PBD sample showed evidence of one or more TBIs on testing and clinical exam. At present there is an ongoing controversy about the significance of test results in TBIs, especially LD (49–51).

Although LD is the most studied of the various TBIs, unlike in diabetes mellitus where the accuracy of the testing is very good, standard TBI testing has been considered no better than a coin toss. Emphasis on clinical judgment has been utilized to make the proper diagnosis. Better techniques, especially those focusing on direct detection of the pathogen (*Borrelia burgdorferi*) rather than indirect techniques are sorely needed. Present indirect techniques which are mainly based on demonstrating the presence of antibodies in the blood or other body fluids are subject to a variety of limitations, including using an immune based test to identify a pathogen that is itself immunosuppressive (*Borrelia burgdorferi*, *Bartonella*) (52, 53). Newer techniques such as FISH testing provide direct proof of the presence of the pathogen. In this case series, a diagnosis of TBI was confirmed only when both laboratory testing and clinical assessment were positive. This dual-confirmation approach was implemented to minimize bias that could arise from relying solely on either laboratory results or clinical judgment in isolation. It should be noted in general that not 100% of individuals with a streptococcal infection mount a positive immune response. Also, testing for a streptococcal infection through indirect antibody testing may be negative if there are co-occurring TBIs that have compromised the immune system's ability to make antibodies or if there is baseline immune dysfunction.

What factors are common to both bipolar disorder and TBIs?

1. Immune dysfunction plays a role in some individuals with bipolar disorder and in those with chronic TBIs e.g., LD/*Bartonella* (14, 52, 53):

TABLE 1 Characteristics of the pediatric bipolar disorder sample (N = 37).

Characteristic	Result
Sex	n = 10 females (27%); n = 27 males (73%)
Mean age (years) at first visit	7.9 (range 2.75 y.o.–16 y.o.)
Age range at PBD diagnosis (years)	3.3–16 y.o.
History of known tick bite	n = 3 (8%)
PBD ^a subtype	PBD I: n = 21 (57%); PBD II: n = 16 (43%)
+FH ^b in 1st to 3rd generation relatives (sibs, parents and grandparents) ^c	
Bipolar disorder	n = 12
Depression	n = 25

^aPBD = pediatric bipolar disorder.

^bFH = family history.

^cDetailed family history was absent in 2 adopted youth in the sample but in one case it was known that both parents had bipolar disorder and a history of substance abuse. The applicable information was included in the above statistics.

TABLE 2 Prevalence of different infections among 37 patients with pediatric bipolar disorder^a.

Pathogen	Number of positive cases	Percentage positive
<i>Babesia</i>	19	51%
<i>Bartonella</i> spp.	18	49%
<i>Mycoplasma pneumoniae</i>	14	38%
<i>Borrelia burgdorferi</i> (LD)	8	22%
Group A beta-hemolytic <i>Streptococcus</i>	7	19%

^a14 individuals tested positive for 1 pathogen; 13 were positive for two pathogens; and 7 showed evidence of exposure to 3 pathogens.

2. Minocycline, a tetracycline antibiotic with anti-inflammatory effects has shown some efficacy in treating both disorders (54, 55).
3. Both disorders can be chronic and often marked by intermittent exacerbations and remission of the symptoms. This chronicity can make these illnesses harder to treat and is associated with significant morbidity and mortality (3, 56, 57).
4. Both bipolar disorder and chronic Lyme disease (LD) are associated with depression, suicidal ideation, and increased risk of suicide, with early onset conferring greater long-term morbidity and mortality (58, 59). Notably, a large Danish study found that individuals who contracted LD before age 10 and experienced depression had a higher risk of suicide attempts later in life compared to those without LD or those who contracted it after age 10 (60).
5. Adults with bipolar disorder who present with rapid cycling or mixed states often demonstrate the greatest resistance to standard treatments (56, 61). Children with PBD frequently exhibit these features and can be challenging to stabilize (62). Could treatment resistant mood symptoms be better explained by the presence of underlying unrecognized infections such as *Borreliosis*, *Bartonellosis* or *Babesiosis*?
6. Elevated proinflammatory cytokine levels which can occur in both illnesses have the potential to affect multiple monoamines and other neurotransmitters important in mood disorders (63–65).
7. Non-steroidal anti-inflammatory agents such as celecoxib and omega 3 fatty acids have been shown to have a possible role in the treatment of bipolar disorder, depression, and LD (66–68).
8. Both illnesses appear to be multisystem disorders (69–72).
9. Sleep dysfunction, cognitive dysfunction, symptoms worsening with stress, are all intimately linked to both disorders (73–82).
10. Increased rates of autoimmune disorders seen in some studies as associated with bipolar disorder, and autoimmune mechanisms (e.g., molecular mimicry) are suspected to play a role in LD pathology (83–88).

The diagnosis of PBD is more common, and bipolar disorder in general has a younger age of onset, in the U.S. compared to many European countries (42). In America, 1/4 of adults with bipolar disorder experience the onset of illness before age 13, and 2/3 before age 19. In many European countries only 1/3 of adults with bipolar disorder have their disorder begin by age 19. These differences suggest that the predominance of *Borrelia burgdorferi* in the U.S., compared with *Borrelia afzelii* and *Borrelia garinii* in Europe, may contribute to the earlier onset and higher prevalence of PBD among American youth. *Borrelia burgdorferi* is known to trigger a stronger inflammatory response than the European strains. These differences suggest that the predominance of *Borrelia burgdorferi* in the U.S., compared to *Borrelia afzelii* and *Borrelia garinii* in Europe, may in part contribute to the earlier onset and higher prevalence of PBD among American youth (89, 90).

Another factor to consider is that all four of the major tick-borne pathogens, *Borrelia burgdorferi*, *Babesia*, *Bartonella* and *Mycoplasma pneumoniae* have been found to be capable of

getting past the blood brain barrier and causing CNS illness (91–94).

Both *Borrelia burgdorferi* and *Babesia* are considered endemic and are reportable infections in New Jersey. Lyme disease, caused by *Borrelia burgdorferi* is transmitted by the blacklegged tick (*Ixodes scapularis*). New Jersey consistently ranks among the states with the highest reported cases, coming in third nationally in 2023 according to the New Jersey State Health Assessment Data (95). Since 2004, the incidence of Lyme disease in New Jersey has more than doubled, rising from 36.1 cases per 100,000 population to 77.8 per 100,000 in 2023 (96, 97).

In comparison to LD, both *babesiosis* and *bartonellosis* remain less extensively studied in humans, and knowledge of their potential long-term effects is limited. The incidence of babesiosis, a parasitic infection that has a preference for invasion of erythrocytes, has increased markedly in New Jersey over the past two decades. In 2004, only 38 cases were reported statewide, whereas 406 cases were documented in 2023 (95, 96).

In contrast, *Bartonella species*, gram-negative intracellular bacteria, are not currently designated as reportable infections in New Jersey, and their transmission by *Ixodes* ticks remains controversial. Despite this, *Bartonella* is often considered a potential co-infection in individuals undergoing diagnostic testing for tick-borne diseases. The Columbia University Lyme and Tick-borne Diseases Research Center note that “the evidence for ticks as vectors of *Bartonella* organisms is circumstantial but fairly strong” (98). Importantly, a growing body of evidence suggests that *Bartonella* infection may be associated with serious neuropsychiatric and cerebrovascular outcomes, including psychosis, bipolar disorder, schizophrenia, stroke, depression, and anxiety highlighting the need for further research into its clinical and public health impact (26, 99–103).

The generalizability of these findings is limited by several factors. Data were drawn from a single private psychiatric practice located in a Lyme and *Babesia* endemic region, making it unclear whether similar results would be observed in youth with other psychiatric disorders or in non-endemic areas. Retrospective chart reviews in general are limited by incomplete documentation, lack of standardized assessments, temporal ambiguity, and small sample size. Referral bias is also possible given the practice’s specialization in pediatric bipolar disorder (PBD), tick-borne infections (TBIs), and PANS/PANDAS. Diagnostic consistency was not standardized, and the use of multiple consultants added variability. In addition, neither psychiatric nor TBI specialists were blinded, as both were aware of clinical diagnoses and laboratory results, introducing potential bias. This non-consecutive case series was further subject to selection bias, since inclusion depended on timing and participants’ willingness to participate with different recommendations. Assessments conducted both before and after the COVID-19 pandemic may have influenced results, and unmeasured factors—such as reluctance to provide blood samples—could also have affected interpretation. Given the small sample size and lack of a control group, formal statistical comparisons were not feasible; the data are descriptive and cannot establish causality.

The primary aim was to identify a potential signal—the high frequency of TBI exposure among youth with PBD—that warrants further investigation. Future studies should use prospective designs with psychiatric control groups, standardized diagnostic tools, and systematic data collection to improve reliability and clarify temporal or causal links between pediatric bipolar disorder and tick-borne infections.

Despite these limitations, the study suggests that infections may act as environmental triggers in genetically or immunologically vulnerable individuals. This aligns with prior work implicating infection and immune-related factors in the pathogenesis of bipolar disorder.

Clinically, TBIs should be considered in youth with treatment-resistant psychiatric symptoms, particularly those consistent with PBD. Infections can exacerbate inflammation, compromise blood-brain barrier integrity, and reduce treatment effectiveness, sometimes necessitating targeted interventions. In families with a history of bipolar disorder, infections may act as triggers, revealing underlying genetic vulnerability.

Given the findings of this study, clinicians should consider TBIs in patients whose symptoms are refractory or only partially responsive to standard treatments. Certain infections may provoke inflammatory and immune responses that interfere with conventional therapies, and blood-brain barrier disruption may further contribute to treatment resistance, influencing the choice of interventions such as antibiotics capable of CNS penetration. In cases of PANS or PANDAS presenting with severe, treatment-resistant mood symptoms, assessing the patient's family history for bipolar disorder is important, as infection may unmask genetic susceptibility.

If confirmed, these associations underscore the significance of tick-borne pathogens—including *Borrelia*, *Bartonella*, *Babesia*, and *Mycoplasma* species—as potentially modifiable risk factors in pediatric psychiatric illness. Early recognition and treatment could meaningfully reduce the long-term burden of mental illness in vulnerable children.

Data availability statement

The original contributions presented in the study are included in the article/Supplementary Material, further inquiries can be directed to the corresponding author.

Ethics statement

Ethical approval was not required for the study involving humans in accordance with the local legislation and institutional requirements. Written informed consent to participate in this

study was not required from the participants or the participants' legal guardians/next of kin in accordance with the national legislation and the institutional requirements. This study involved a retrospective evaluation and audit of patient records by the treating physician. No identifiable personal information was included in the analysis or publication. This study did not undergo IRB review as all analyses were based on existing records and conducted in compliance with applicable data protection laws and institutional policies.

Author contributions

RG: Writing – original draft, Writing – review & editing.

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Conflict of interest

The author declares that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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