

Acquired Attentional Deterioration as a Diagnostic Pitfall in Child Psychiatry: A Pediatric Case of Possible Anti-GABABR Encephalitis With a Narrative Literature Review

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Case Report

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Abstract

Background:

Attentional complaints are common in child psychiatry and are often initially considered within a primary attentional-disorder framework. However, acute or subacute deterioration in attention, functioning, or emotional state may warrant diagnostic reassessment, particularly when accompanied by medical or neurological red flags. Pediatric anti- γ -aminobutyric acid-B receptor (anti-GABABR) encephalitis is extremely rare. This report describes a child initially evaluated for worsening attentional difficulties who was subsequently classified as having possible autoimmune encephalitis with serum anti-GABABR positivity, and provides a focused narrative review of reported pediatric cases.

Case presentation:

A 9-year-old girl had mild attentional complaints for approximately one year, with limited impact on academic functioning. During the month before admission, her inattention worsened substantially, accompanied by emotional changes and academic decline. She was admitted after an initial outpatient concern for a possible primary attentional disorder. After admission, electroencephalography (EEG) showed increased slow-wave activity, prompting reassessment of the clinical history and further neurological evaluation. A febrile episode approximately one month before admission was then identified. Brain magnetic resonance imaging (MRI) and routine cerebrospinal fluid (CSF) studies were unremarkable. Anti-GABABR antibodies were detected in serum, whereas CSF antibody testing was negative. The case was classified as possible autoimmune encephalitis with serum anti-GABABR positivity. Clinical and electroencephalographic improvement was observed after immunotherapy. A focused narrative review of 15 previously reported pediatric cases showed sparse and heterogeneous evidence, with variable psychiatric and neurological manifestations. Reported pediatric cases with isolated anti-GABABR antibodies and those with co-existing antibodies appeared to differ in clinical context, although current evidence is insufficient for prognostic stratification. Serum anti-GABABR positivity without CSF confirmation should be interpreted cautiously within the broader clinical context.

Conclusions:

This case illustrates that acute or subacute functional deterioration, rather than attentional symptoms alone, may warrant reassessment of an initial attentional-disorder formulation in child psychiatry. In this case, EEG slowing was a non-specific but clinically relevant clue that prompted neurological reassessment. Pediatric anti-GABABR encephalitis showed sparse and heterogeneous evidence, with variable psychiatric and neurological manifestations.

1 Introduction

Attentional complaints are common in child psychiatry and are usually assessed within attentional, neurodevelopmental, emotional, sleep-related, or psychosocial frameworks [1, 2]. In many children, such formulations are appropriate [1, 2]. However, acute or subacute worsening of attention, particularly when accompanied by functional decline or emotional and cognitive changes, may require the initial psychiatric formulation to remain provisional [3, 4]. In this setting, the course of illness can be as important as the symptom itself [3, 4].

Anti-GABABR encephalitis is a rare form of autoimmune synaptic encephalitis associated with antibodies targeting the B1 subunit of the GABAB receptor [5]. In adults, it has most often been described as limbic encephalitis with seizures, memory impairment, and behavioral changes [5–7]. Malignancy, particularly small cell lung cancer, has been reported in approximately 50–60% of adult cases, supporting a paraneoplastic association in a substantial proportion of patients [5–7]. Pediatric anti-GABABR encephalitis is far less frequently reported [8–10]. Earlier data suggested a detection rate of nearly 2% in children tested for autoimmune encephalitis-related antibodies [8], whereas more recent large-scale pediatric cohorts indicate a prevalence well below 1% among children with encephalitis or antibody-mediated autoimmune encephalitis [9]. Tumor associations have not been consistently reported in pediatric cases [8, 10, 11]. Overall, the published pediatric literature remains limited and clinically heterogeneous [8, 10, 11].

We report a child who was initially evaluated in a child psychiatry setting for worsening attentional difficulties and emotional symptoms. Non-specific EEG slowing prompted reassessment of the illness history and further neurological and immunological evaluation. The case was classified as possible autoimmune encephalitis with serum anti-GABABR positivity [3, 4]. We also present a focused narrative review of reported pediatric anti-GABABR cases to place the present case within the limited pediatric literature.

2 Case presentation

2.1 Patient information and presenting concerns

A 9-year-old girl was admitted to a child psychiatry service because of worsening attentional difficulties, anxiety and depressive symptoms, and declining school performance. According to her parents, she had had mild attentional complaints for more than one year, during which she could usually maintain attention in class for approximately 20 minutes and had only limited academic impairment. In the month before admission, her inattention became more prominent, interfered with schoolwork and homework completion, and was accompanied by labile mood and headache.

2.2 Relevant medical, developmental, family, and psychosocial history

At the initial assessment, there was no history of seizures, febrile seizures, altered consciousness, movement disorder, developmental regression, known neurological disease, or previous encephalitic illness. No clear family history of neurological or major psychiatric disorders was reported.

A febrile episode approximately one month before admission was not fully identified during the initial psychiatric assessment. After the EEG abnormality prompted review of the medical history, this episode was clarified as having preceded the marked worsening in attention, emotional symptoms, and learning performance. The temporal relationship was considered relevant to the diagnostic assessment, but was not interpreted as evidence of causality.

2.3 Initial psychiatric assessment and provisional formulation

At the outpatient visit, attention-deficit/hyperactivity disorder (ADHD) or a possible primary attentional disorder was considered because of the patient's long-standing attentional complaints, reduced classroom concentration, and recent decline in learning performance.

After admission, the Conners' Parent Rating Scale (CPRS) showed an elevated total score of 12, above the clinical cut-off of 10. The Integrated Visual and Auditory Continuous Performance Test (IVA-CPT) showed marked impairment in attentional performance, particularly sustained attention, with both auditory and visual sustained attention at PR = 1. Visual response control and visual attention were also impaired, with PR values of 7 and 3, respectively. The Swanson, Nolan, and Pelham Rating Scale-IV (SNAP-IV) did not reach the clinical range, with mean scores of 1.22 for inattention, 0.67 for hyperactivity/impulsivity, and 1.38 for oppositional defiant symptoms. The Screen for Child Anxiety Related Disorders (SCARED) total score was 38, above the cut-off of 25, and the Depression Self-Rating Scale for Children (DSRSC) score was 24, above the cut-off of 15.

Mental status examination revealed inattention and affective lability, without evidence of psychotic, manic, or obsessive-compulsive symptoms, tics, self-injurious behavior, or fluctuating consciousness. While these findings confirmed clinically relevant attentional dysfunction with emotional symptoms, they did not converge to support a straightforward ADHD diagnosis. The recent functional decline further suggested an altered illness trajectory rather than a chronic, stable attentional disorder. Crucially, although rating scales highlighted attentional and emotional difficulties, the SNAP-IV score did not reach the clinical threshold, and the subacute deterioration was inconsistent with the typical ADHD course. This discrepancy between the clinical presentation and standardized scale thresholds set the stage for diagnostic reassessment after the subsequent EEG abnormalities.

2.4 Timeline, EEG findings, and diagnostic reassessment

EEG was performed in the context of recent worsening of attention, emotional symptoms, and academic decline. The EEG showed increased slow-wave activity without epileptiform discharges. This finding was non-specific: it did not indicate a seizure disorder, establish an inflammatory or autoimmune etiology, or exclude a primary attentional disorder.

In the context of recent functional deterioration, however, the EEG abnormality made a purely psychiatric or attentional-disorder explanation less sufficient. It prompted clinicians to revisit the illness timeline and medical history. During this reassessment, a febrile episode approximately one month before admission was clarified. The combination of recent clinical deterioration, non-specific EEG abnormality, and retrospectively clarified febrile history broadened the differential diagnosis and led to further neurological and immunological evaluation. The diagnostic sequence is summarized in Fig. 1.

Figure 1 Timeline of clinical course and diagnostic reassessment.

This timeline illustrates the 26-month clinical course of the 9-year-old girl, from psychiatric admission for subacute attentional deterioration (Month 0) through sequential diagnostic assessments (EEG, MRI, CSF, serum antibodies) to immunotherapy and follow-up. The diagnostic turning point—including the revisited febrile prodrome and abnormal EEG findings—is highlighted, culminating in the detection of serum anti-GABABR antibodies (1:100; CSF-negative) and a diagnosis of possible autoimmune encephalitis. Clinical improvement following first-line immunotherapy is shown.

EEG, electroencephalography; CSF, cerebrospinal fluid.

2.5 Neurological and immunological evaluation

Further evaluation was performed after the EEG abnormality and history reassessment. Physical and neurological examinations revealed no abnormalities. Brain MRI showed no structural or inflammatory abnormalities. Chest computed tomography, electrocardiography, and ultrasonography of the thyroid, liver, gallbladder, kidneys, and uterus were unremarkable. Routine laboratory investigations, including complete blood count, coagulation profile, lipid profile, liver and renal function tests, thyroid function tests, sex hormone levels, blood-borne infection screening, and cytokine analysis, were within normal limits. No tumor was identified during initial screening.

Lumbar puncture showed normal intracranial pressure, cell count, protein concentration, and glucose level. Oligoclonal bands (OCB) were absent in paired CSF and serum samples. Serum anti-GABABR immunoglobulin G (IgG) antibodies were detected by cell-based assay (CBA) at a titer of 1:100, whereas CSF anti-GABABR antibody testing was negative. Serum tissue-based assay (TBA) showed weak reactivity at a titer of 1:10. Extensive screening for other autoimmune encephalitis-associated antibodies and paraneoplastic antibodies yielded negative results in both serum and CSF. The specific panel included NMDAR, AMPAR, GABAAR, LGI1, CASPR2, DPPX, IgLON5, GlyR1, DRD2, mGluR5, mGluR1, GABAAR α 1, GABAAR β 3, Neurexin3 α , AK5, and KLHL11, along with paraneoplastic markers Hu, Yo, Ri, CV2, PNMA2, Amphiphysin, Ma1, SOX1, Tr, Zic4, GAD65, PKC γ , Recoverin, and Titin. Infectious evaluation was also performed. Targeted testing for more than 100 pathogens, including *Mycoplasma*, *Chlamydia*, and *Mycobacterium tuberculosis*, was unremarkable. CSF metagenomic next-generation sequencing (mNGS) detected 5 reads of human herpesvirus type 1 (HSV-1).

2.6 Diagnostic assessment and classification

The diagnostic formulation was revised after psychiatric findings, illness trajectory, EEG results, neuroimaging, CSF studies, infectious testing, and antibody results were considered together. The initial concern for ADHD or a primary attentional disorder was understandable, given the patient's long-standing mild attentional complaints. However, it did not adequately account for the recent subacute attention deterioration, emotional symptoms, decline in learning ability, and EEG slowing.

Primary ADHD was considered less likely because the recent change in clinical course was inconsistent with a stable neurodevelopmental disorder, and SNAP-IV scores remained below the clinical threshold. Anxiety and depressive disorders were also considered, as both screening scores were elevated and these symptoms may have contributed to the attentional and learning difficulties. However, mood lability was the predominant emotional feature, and the

pattern, duration, and severity of the anxiety and depressive symptoms did not meet criteria for a primary anxiety or depressive disorder. Neither diagnosis adequately explained the subacute functional decline or the EEG abnormality.

Active herpes simplex encephalitis was also considered, given the preceding febrile episode and the detection of 5 HSV-1 reads in CSF mNGS. This diagnosis was not well supported by the overall findings: the patient had no rapidly progressive encephalitic course, seizures, impaired consciousness, focal neurological deficits, CSF pleocytosis, elevated CSF protein, or brain MRI abnormalities. The low-read HSV-1 result was therefore interpreted as being of uncertain clinical significance rather than evidence of active viral encephalitis. Post-herpes simplex encephalitis autoimmune encephalitis was also considered, but the absence of documented clinically overt herpes simplex encephalitis, a biphasic course, destructive or necrotic MRI lesions, and inflammatory CSF findings made this explanation less likely.

Based on the comprehensive evaluation, the case was ultimately classified as possible autoimmune encephalitis associated with serum anti-GABABR positivity. This diagnostic impression was primarily driven by the subacute onset of neuropsychiatric deterioration, the presence of a non-specific EEG abnormality, serum anti-GABABR IgG positivity via cell-based assay, and weak reactivity in the serum tissue-based assay, in the context of otherwise unexplained symptoms. However, we acknowledged the limitations in diagnostic certainty. The classification was constrained by negative CSF anti-GABABR antibody testing, a normal brain MRI, unremarkable routine CSF profiles, and the uncertain significance of the low-read HSV-1 result. Consequently, a definitive diagnosis of anti-GABABR encephalitis could not be established. Given the possible autoimmune etiology and the lack of a better-supported alternative diagnosis, a trial of immunotherapy was initiated.

2.7 Therapeutic intervention

After classification as possible autoimmune encephalitis with serum anti-GABABR positivity, empirical first-line immunotherapy was initiated. The patient received intravenous methylprednisolone at 20 mg/kg/day for 3 consecutive days, together with intravenous immunoglobulin (IVIG) at 12.5 g/day for 3 days.

No stimulant, antidepressant, antipsychotic, or other specific psychotropic medication was initiated during the acute treatment period. Antiviral therapy was not administered. After intravenous treatment, the patient was transitioned to oral prednisone at 30 mg/day, with a planned gradual taper over 4 months.

2.8 Follow-up and outcomes

Following immunotherapy, the patient was monitored clinically and with serial EEG recordings. One week after treatment initiation, headache and emotional symptoms resolved, and attentional function improved clinically. Repeat EEG demonstrated improved background activity, although the tracing remained borderline abnormal.

During outpatient follow-up, oral prednisone was gradually tapered. At the 4-month follow-up, the patient remained clinically improved, prednisone was discontinued, and repeat EEG showed normalization of background activity. No recurrence of attentional deterioration, emotional symptoms, headache, seizures, impaired consciousness, or abnormal movements was observed during this period.

At the final telephone follow-up 26 months after discharge, the parents reported that the patient's attention and academic performance had returned to the pre-illness baseline. According to parental report, she remained symptom-free and medication-free. No objective post-treatment neuropsychological reassessment or serial anti-GABABR antibody titers were obtained. Thus, while the temporal association between immunotherapy and subsequent clinical and EEG improvements was supportive of the diagnosis, it was not considered confirmatory.

2.9 Patient and family perspective

A formal written patient or family perspective was not collected. During follow-up, the patient's parents reported that her attention and academic performance had returned to the pre-illness baseline. This information was recorded as part of clinical follow-up rather than as a structured patient-reported outcome.

3 Focused narrative review of reported pediatric anti-GABABR cases

3.1 Search approach and case selection

To place the present case in the context of reported pediatric anti-GABABR encephalitis, we performed a focused literature search up to July 2026. The databases searched were PubMed, Web of Science, Embase, Scopus, Cochrane Library, China National Knowledge Infrastructure (CNKI), Weipu, and Wanfang. Search terms included "GABABR," "gamma-aminobutyric acid receptor," "GABAergic," "anti-GABABR encephalitis," "autoimmune encephalitis," and "pediatric cases." Articles published in English or Chinese were considered. Reports were reviewed if they described patients younger than 18 years with anti-GABABR encephalitis and provided extractable clinical information. Case reports, case series, and studies containing pediatric cases were considered. Reports without pediatric cases or without usable clinical data were excluded from the summary.

We identified 15 previously reported pediatric cases of anti-GABABR encephalitis. The present case was described separately as the index case. For contextual comparison, the index case was also displayed alongside previously reported cases in Table 1, but literature-derived descriptive summaries and case-level descriptive comparisons were based on previously published cases unless explicitly stated otherwise. Among the 15 previously reported cases, 13 had individual-level clinical data. Of these, 9 isolated anti-GABABR cases were summarized at the case level in Table 1, and 4 cases with coexisting neural autoantibodies were presented in Table 2. Two cases were available only as aggregate data; because individual-level links between antibody profile, tumor status, treatment, and outcome could not be determined, these cases were described qualitatively and were not included in case-level descriptive comparisons.

3.2 Overview of included pediatric cases

Among the 15 previously reported pediatric cases, the age range was approximately 3 to 18 years, with 10 males and 5 females. Onset was not uniformly reported, although available descriptions included acute or subacute presentations. The reported clinical features were heterogeneous. Psychiatric or behavioral symptoms and cognitive changes were described in several cases, including emotional or behavioral disturbance, memory impairment, disorientation, or attentional difficulties. Neurological manifestations were also reported, including seizures, impaired consciousness, movement symptoms, headache, and fever. Ancillary findings varied across reports, including EEG, MRI, CSF, and antibody testing results. Overall, the pediatric cases showed both psychiatric and neurological manifestations, without a consistent clinical pattern. These data provide clinical context for a rare pediatric condition, but they should be interpreted descriptively. The present case is discussed separately as the index case and is used only for contextual comparison unless explicitly stated.

3.3 Clinical manifestations: psychiatric and neurological features

The previously reported pediatric cases included both psychiatric and neurological features. Psychiatric or behavioral changes were described in several reports, and cognitive symptoms included memory impairment, disorientation, and attentional difficulties. In the present case, worsening attention and emotional symptoms led to admission to a child psychiatry service. In the published pediatric cases, however, the type and prominence of psychiatric or cognitive symptoms varied. Neurological features were also reported, including seizures, impaired consciousness, movement symptoms, headache, and fever or febrile prodrome. Movement symptoms included ataxia, dystonia, or opsoclonus in some cases. These findings were not present in all patients, and the level of clinical detail varied across reports. Overall, psychiatric, behavioral, or cognitive symptoms were present in some pediatric cases, but they were not specific.

3.4 Ancillary findings: EEG, MRI, CSF, and antibody profiles

Ancillary findings were inconsistently reported across the previously published pediatric cases. Among the 9 previously reported cases with isolated anti-GABABR antibodies, interpretable EEG data were available in 4 patients. One showed isolated slow-wave activity, one showed slow-wave activity with epileptiform discharges, and two had normal EEG findings. When the present case was considered for contextual comparison, the isolated-antibody cases with interpretable EEG data increased to 5, including 2 cases with isolated slow-wave activity. In the small number of cases with co-existing neural autoantibodies, EEG abnormalities were reported in all 4 patients, including generalized slowing with epileptiform discharges or focal spike-and-slow-wave complexes in some cases.

Brain MRI findings also varied. Among the 9 previously reported isolated-antibody cases, MRI data were available in 6 patients. Three had abnormal findings, including limbic T2/FLAIR hyperintensities in 2 patients and bilateral thalamic and basal ganglia involvement in 1 patient. The remaining 3 patients with available MRI data had unremarkable findings. The present case also had unremarkable brain MRI findings. Among cases with co-existing neural autoantibodies, MRI abnormalities were reported in all 4 patients, often with multifocal involvement beyond the limbic system.

CSF findings were available for only part of the reported cases. In the 9 previously reported isolated-antibody cases, routine CSF data were reported in 5 patients; 3 had abnormalities such as mild pleocytosis, elevated protein, or increased intracranial pressure, whereas 2 had normal CSF profiles. The present case also had an unremarkable routine CSF profile. In the co-existing antibody group, CSF data were available in 3 of 4 patients, and all 3 had reported abnormalities, including pleocytosis or elevated protein.

Antibody testing showed variable serum–CSF patterns. Among the 6 previously reported isolated-antibody cases with paired serum and CSF antibody data, 3 were positive in both serum and CSF, 2 were positive only in serum, and 1 was positive only in CSF. In 3 additional previously reported isolated-antibody cases, only serum positivity was reported. Including the present case, the ratio of serum+/CSF- among isolated cases with paired data increased from 2/6 to 3/7. Co-existing neural autoantibodies in the published pediatric cases included anti-NMDAR, anti-GABAAR, and anti-CASPR2 antibodies. Serum–CSF discordance was reported in some cases, but the meaning of serum-only positivity differed across reports and required interpretation within the individual clinical context.

3.5 Descriptive comparison of reported cases by antibody profile

Among the 13 previously reported pediatric cases with individual-level data, 9 involved isolated anti-GABABR antibodies and 4 involved co-existing neural autoantibodies. The present case was displayed alongside these cases in the summary table for contextual comparison, but it was not included in the literature-derived case-level comparison unless explicitly stated. This comparison was used to organize the available reports according to antibody profile and was not intended to define clinical subtypes.

In the 9 previously reported isolated anti-GABABR cases, psychiatric, behavioral, or cognitive symptoms were reported, including memory impairment, disorientation, and attentional difficulties. Seizures and impaired consciousness were each reported in 3 cases. No tumor was reported in this group. All 9 patients received first-line immunotherapy; 2 also received second-line treatment. Complete recovery was reported in 8 cases and partial recovery in 1 case. When the present case was included for contextual comparison, the isolated-antibody group comprised 10 cases. The present case showed attentional and emotional symptoms. No tumor was identified during initial screening; brain MRI and routine CSF findings were unremarkable; anti-GABABR antibodies were detected in serum but not in CSF. The patient received first-line immunotherapy and showed clinical and EEG improvement during follow-up.

The 4 cases with co-existing neural autoantibodies included anti-NMDAR, anti-GABAAR, and anti-CASPR2 antibodies. All were reported in young male children. Psychiatric symptoms and seizures were each reported in 3 cases, and impaired consciousness and movement symptoms were each reported in 2

cases. MRI and EEG abnormalities were reported in all 4 cases, while CSF abnormalities were reported in all 3 cases with available CSF data. Second-line immunotherapy was used in 3 cases, and 1 fatal outcome was reported.

3.6 Aggregate-only reports

Two previously reported pediatric anti-GABABR cases from Kang et al. were available only as aggregate data and were not included in the case-level descriptive comparison by antibody profile. They were retained in the review because they provided information on severe pediatric presentations, although individual-level links among antibody profile, tumor-related findings, treatment escalation, and outcome could not be determined.

Both patients were male, aged 88–118 months. One had acute onset and the other had subacute onset. Seizures were reported as the initial manifestation in one child and behavioral symptoms in the other. During the disease course, both children had seizures, impaired consciousness, memory deficits, and fever. Status epilepticus (SE), behavioral symptoms, speech disorder, sleep disturbance, involuntary movements, and autonomic dysfunction were each reported in one of the two cases.

Both cases had abnormal ancillary findings. Brain MRI was abnormal in both patients, with thalamic involvement reported in both and additional cortical, insular, basal ganglia, or other lesions reported in one. EEG abnormalities were present in both, including slow-wave activity and epileptiform discharges. CSF pleocytosis was reported in both cases, whereas elevated CSF protein was not reported.

Antibody and tumor-related data were also provided only in aggregate form. One patient had anti-GABABR antibodies detected in both serum and CSF, while the other had serum-only positivity. One patient had co-existing anti-CASPR2 antibodies. A tumor-related finding was reported in one anti-GABABR case, but individual-level details were not available. The article reported that both children received methylprednisolone and intravenous immunoglobulin, and one received rituximab (RTX) after insufficient response to first-line treatment. Both required pediatric intensive care. Final outcomes differed, with one complete recovery and one death.

4 Discussion

4.1 Main findings

This report describes a process of diagnostic reassessment in child psychiatry. The clinical issue was not attentional symptoms alone, but acute or subacute functional deterioration in a child with pre-existing mild attentional complaints. In this case, non-specific EEG slowing prompted clinicians to revisit the illness timeline, clarify a febrile episode approximately one month before admission, and pursue further neurological and immunological evaluation. The case was classified as possible autoimmune encephalitis with serum anti-GABABR positivity. The accompanying focused narrative review places this case within the limited pediatric anti-GABABR literature, which remains sparse and clinically heterogeneous.

4.2 Diagnostic reassessment of attentional complaints in child psychiatry

The initial clinical suspicion of ADHD or a primary attentional disorder at the outpatient visit was reasonable, given the patient's long-standing mild attentional complaints and recent learning difficulties [1, 2]. However, objective testing after admission yielded mixed results that challenged a straightforward ADHD formulation. While CPRS and IVA-CPT indicated marked attentional impairment, the SNAP-IV scores remained below the clinical cutoff for inattention and hyperactivity/impulsivity. The presence of clinically significant anxiety and depression symptoms, coupled with recent functional decline, further suggested that a primary psychiatric diagnosis alone was insufficient.

This diagnostic reassessment was catalyzed by the EEG findings. Although the increased slow-wave activity without epileptiform discharges was non-specific [3, 4]—it could not confirm autoimmune encephalitis, nor could it exclude a primary attentional disorder—its occurrence in the context of subacute functional deterioration, emerging emotional symptoms, and declining academic performance prompted a critical re-evaluation of the illness trajectory. Specifically, the recognition of a febrile episode approximately one month prior to admission broadened the diagnostic horizon and led to targeted neurological assessment, lumbar puncture, and autoantibody testing [3, 4]. This temporal relationship did not establish causality, but it prompted a shift from a primary psychiatric formulation to a medical diagnostic reassessment.

Ultimately, the case was classified as possible autoimmune encephalitis with serum anti-GABABR positivity [3, 4]. Diagnostic certainty was limited by the absence of CSF antibody positivity, lack of inflammatory CSF changes, and normal brain MRI. Therefore, the central clinical issue extended beyond isolated attentional deficits: it was the combination of recent functional decline followed by an abnormal EEG that prompted detailed history-taking and comprehensive neurological workup. This case does not support routine autoimmune encephalitis screening in all children with attentional problems. Rather, it underscores that subacute or rapidly progressive functional decline, new-onset emotional or cognitive changes, a preceding febrile illness, or non-specific EEG abnormalities should serve as red flags that prompt clinicians to reconsider acquired medical or neurological causes. In such contexts, a careful review of illness trajectory—not merely symptom checklists—is essential to avoid misattributing organic encephalopathy to primary psychiatric disorders.

4.3 Pediatric anti-GABABR encephalitis: sparse and heterogeneous evidence

Reports of pediatric anti-GABABR encephalitis remain limited [8–11]. Most clinical descriptions of anti-GABABR encephalitis are derived from adult cohorts, where seizures and tumor associations are more frequently reported [5–7, 12]. In children, the available literature consists mainly of individual case reports and small case summaries [8, 10, 11]. Reported pediatric cases involve both neuropsychiatric and neurological manifestations, including behavioral or psychiatric symptoms, cognitive impairment, seizures, movement abnormalities, and impaired consciousness, with considerable variation across cases (Tables 1–3).

In the available pediatric reports, cases with co-existing neural autoantibodies appeared clinically more complex than those with isolated anti-GABABR antibodies (Table 3). This is broadly consistent with reports from autoimmune encephalitis cohorts in which multiple neural autoantibodies have been associated with greater disease severity or poorer outcomes [13, 14]. In pediatric anti-GABABR encephalitis, however, the evidence remains too limited for prognostic interpretation (Table 3).

Severe outcomes should also be considered within this heterogeneous pediatric literature. Fatal outcomes were reported in 2 previously reported pediatric cases, but this finding should be interpreted descriptively rather than as a mortality estimate because the reports were few, heterogeneous, and inconsistently followed (Tables 1–3). Adult anti-GABABR encephalitis has been associated with substantial mortality and a stronger tumor association, particularly with small cell lung cancer, but adult cohort data are not directly comparable with pediatric case reports [5–7, 12]. The available pediatric evidence only indicates that both favorable outcomes and severe or fatal courses have been reported (Tables 1–3).

4.4 Antibody testing, serum–CSF discordance, and diagnostic uncertainty

Serum–CSF discordance in anti-GABABR antibody testing is not uncommon. Among 6 previously reported isolated anti-GABABR cases with paired results, 2 were serum-positive/CSF-negative, 3 were positive in both specimens, and 1 was positive only in CSF (Table 3). Three additional cases had serum positivity reported without corresponding CSF data (Table 3). A separate cohort of 22 patients with paired plasma and CSF results showed an even more striking distribution: 8 were positive only in plasma, 9 in both specimens, and 5 only in CSF [12].

These observations are consistent with recommendations to test both serum and CSF in patients with suspected autoimmune encephalitis [3, 4, 15], but they do not establish an anti-GABABR-specific diagnostic rule. A serum-positive/CSF-negative result should therefore be interpreted cautiously and in relation to the clinical presentation and other investigations [3, 4]. In the present case, normal brain MRI and routine CSF findings did not exclude autoimmune encephalitis [3, 4], but, together with negative CSF anti-GABABR testing, they limited confidence in attributing the presentation specifically to anti-GABABR antibodies. The serum result was therefore interpreted alongside the subacute illness course, EEG findings, and exclusion of alternative causes rather than as a stand-alone basis for diagnosis [3, 4].

4.5 Tumor association, febrile history, and etiological uncertainty

Tumor association is common in adult anti-GABABR encephalitis, particularly with small cell lung cancer [5–7]. In the initial series, tumors were identified in 7 of 15 patients, including small cell lung cancer in 5. Subsequent cohorts reported small cell lung cancer in 10 of 20 patients and tumors in approximately 66% of 32 patients, most commonly small cell lung cancer [5–7]. Tumors have been reported far less often in children [8, 10, 11]. Across the available pediatric literature, only one tumor-related finding was recorded. Because the relevant data were reported in aggregate, the affected patient, tumor type, and pathological confirmation could not be determined (Table 3) [8].

The pediatric evidence is too sparse to support a firm conclusion about paraneoplastic association. Although the pattern described in adult cohorts has not been demonstrated in children, the small number of cases and incomplete reporting do not establish that pediatric anti-GABABR encephalitis is uniformly non-paraneoplastic. Age-appropriate tumor screening may therefore be considered on an individual basis, particularly in severe, atypical, or clinically complex presentations [4–7].

Febrile or infection-like symptoms were described in some pediatric cases, including the present patient (Tables 1–3) [8, 10, 11]. In this case, the febrile episode helped clarify the illness timeline after the EEG abnormality prompted review of the medical history. The temporal sequence, however, does not establish infection as a trigger. Infection-related immune activation, molecular mimicry, and bystander activation have been proposed as links between infection and autoimmunity more broadly [16], but these mechanisms have not been demonstrated in pediatric anti-GABABR encephalitis [8, 10, 11].

4.6 Clinical implications for child psychiatrists

For child psychiatrists, attentional complaints alone do not justify routine autoimmune encephalitis testing [3, 4, 17]. Reassessment becomes more relevant when the clinical course is not well explained by a primary psychiatric or neurodevelopmental formulation [3, 4]. Acute or subacute functional decline, altered mental status, cognitive change beyond inattention, focal neurological signs, new movement abnormalities, unexplained seizures, rapidly evolving severe psychiatric symptoms, or a relevant febrile history may prompt neurological reassessment [3, 4]. When supported by the overall clinical context, neurological consultation and targeted investigations may include EEG, brain MRI, CSF studies, and paired serum–CSF autoimmune encephalitis antibody testing [3, 4, 15]. These features should broaden the differential diagnosis rather than serve as a checklist mandating autoimmune testing [3, 4, 17].

4.7 Limitations

This study has several limitations. First, the index case was classified as possible autoimmune encephalitis with serum anti-GABABR positivity, not definite anti-GABABR encephalitis. Second, post-treatment neuropsychological testing and serial anti-GABABR antibody titers were not available, limiting objective assessment of cognitive recovery and antibody dynamics. Third, this was a narrative literature review. The number of reported pediatric cases was small, reporting was heterogeneous, follow-up was incomplete, and two cases were available only as aggregate data. These limitations preclude phenotype definition, subgroup classification, or prognostic stratification.

5 Conclusions

This case illustrates that acute or subacute functional deterioration, rather than attentional symptoms alone, may warrant reassessment of an initial attentional-disorder formulation in child psychiatry. In this case, EEG slowing was a non-specific but clinically relevant clue that prompted neurological reassessment. Pediatric anti-GABABR encephalitis showed sparse and heterogeneous evidence, with variable psychiatric and neurological manifestations.

Abbreviations

AK5

Adenylate kinase 5

AMPA

α -amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor

CASPR2

Contactin-associated protein-like 2

CBA

Cell-based assay

CBZ

Carbamazepine

CNKI

China National Knowledge Infrastructure

CPRS

Conners' Parent Rating Scale

CSF

Cerebrospinal fluid

CV2

Collapsin response mediator protein 5 (also termed CRMP5)

DPPX

Dipeptidyl-peptidase-like protein 6

DRD2

Dopamine D2 receptor

DSRSC

Depression Self-Rating Scale for Children

EEG

Electroencephalography / electroencephalogram

FLAIR

Fluid-attenuated inversion recovery

GABA

γ -aminobutyric acid

GABABR

γ -aminobutyric acid-B receptor

GABAAR

γ -aminobutyric acid-A receptor

GlyR1

Glycine receptor 1

HSV-1

Herpes simplex virus type 1

IgG

Immunoglobulin G

IgLON5

Immunoglobulin-like cell adhesion molecule 5

IV

Intravenous

IVA-CPT

Integrated Visual and Auditory Continuous Performance Test

IVIG

Intravenous immunoglobulin

KLHL11

Kelch-like protein 11

LE

Limbic encephalitis

LEV

Levetiracetam

LGI1

Leucine-rich glioma-inactivated 1 protein

mGluR1 / mGluR5

Metabotropic glutamate receptor 1 / 5

mNGS

metagenomic next-generation sequencing

MMF

Mycophenolate mofetil

MRI

Magnetic resonance imaging

NMDAR

N-methyl-D-aspartate receptor

OCB

Oligoclonal band(s)

PLEX

Plasma exchange

PM2 / PNMA2

Paraneoplastic antigen Ma2 / paraneoplastic Ma antigen

RTX

Rituximab

SCARED

Screen for Child Anxiety Related Disorders

SE

Status epilepticus

SNAP-IV

Swanson, Nolan, and Pelham Rating Scale-IV

TBA

Tissue-based assay

WBC

White blood cell(s)

Declarations

Ethics approval and consent to participate

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Sichuan Provincial People's Hospital (Approval Number: 2025-269). Informed consent to participate in the study was obtained from the patient's legal guardians (parents).

Consent for publication

Written informed consent for publication of the clinical details was obtained from the patient's legal guardians. The manuscript has been de-identified to protect the patient's privacy.

Data Availability Statement

The datasets generated and/or analyzed during the current study are not publicly available because they contain individual patient clinical information protected by confidentiality and ethical approval, but are available from the corresponding author on reasonable request. The extracted data from previously reported pediatric anti-GABABR cases supporting the narrative review are included in Tables 1–3 of this manuscript.

Competing interests

The authors declare no competing interests.

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Author contributions

JM C conceptualized and designed the study, collected the data, carried out the statistical analysis, interpreted the data, and drafted the manuscript. J C, J L, P W, LY L, B Z collected the data. Y H revised the manuscript. Y Z conceptualized and designed the study and revised the manuscript. All authors contributed to the article and approved the submitted version.

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Tables

Table 1 Previously reported pediatric cases with isolated anti-GABABR antibodies and the present case for contextual comparison.									
	Sex	Age(years)	Comorbidity	Prodromal symptom	Symptom	CSF analysis	Brain MRI	EEG	Anti-GABABR
1	F	9	N	Fever	Attention deficit, depression, anxiety, mood instability, headache	Normal	Normal	Increasing in slow wave activity	Serum(+, 1:100) CSF(-)
2	F	16	N	N	Seizure, delirium, psychiatric symptoms	NA	NA	NA	Serum(+, 1:1920) CSF(NA)
3	F	16	N	NA	LE(no detail)	NA	NA	NA	Serum(+, NA) CSF(NA)
4	M	18	N	N	Memory deficit, indifference, psychomotor agitation, declining mental status, disorientation, cognitive decline Refractory insomnia	WBC 10×10 ⁶ /L Protein:39 mg/dl	Normal	Normal	Serum(+, NA) CSF(+,NA)
5	M	11	Congenital language retardation	N	Seizures, psychiatric symptoms, impaired consciousness, speech disorder, sleep disorder, involuntary movements	Intracranial pressure: 195mmH ₂ O	T2/FLAIR: Abnormal signals in the right hippocampus	NA	Serum(+, NA) CSF(+,NA)
6	M	11	Japanese encephalitis during convalescent phase	Fever, headache	Fever, headache, irritability, aggressive behavior	NA	T2/FLAIR: Hyperintensities in the bilateral thalamus and basal ganglia	NA	Serum(-) CSF(+,NA)
7	F	11	N	N	Seizure, SE, Psychiatric symptoms	WBC 30×10 ⁶ /L	T2/FLAIR: Abnormal signals in the left hippocampus	Background slowing + left temporo-occipital polymorphic δ/θ rhythms	Serum(+, 1:32) CSF(+, 1:32)
8	M	16	NA	NA	Mental and behavioral abnormalities	NA	NA	NA	Serum(+, 1:32) CSF(-)
9	F	13	N	N	Behavioral abnormality, recent memory loss, language disturbances, breathing difficulty	Normal	Normal	Normal	Serum(+, NA) CSF(-)
10	F	8.5	N	N	Mental and behavioral abnormalities: inattention, sleep disturbances, headache and dizziness, involuntary limb movements	Normal	Normal	Spike-and-slow waves	Serum(+, 1:10) CSF(NA)

CBZ, carbamazepine; CSF, cerebrospinal fluid; EEG, electroencephalogram; F, female; FLAIR, fluid-attenuated inversion recovery; IVIG, intravenous immunoglobulin; LE, limbic encephalitis; LEV, levetiracetam; M, male; MMF, Mycophenolate Mofetil; MRI, magnetic resonance imaging; N, No; NA, not available; PLEX, plasma exchange; RTX, rituximab; SE, status epilepticus; WBC, white blood cell

Note: The first row represents the index case of this study, included for contextual comparison; the subsequent 9 rows are previously reported isolated anti-GABABR cases. The reference for the index case is this report.

Table 2 Previously reported pediatric anti-GABABR cases with co-existing neural autoantibodies.

	Sex	Age (years)	Comorbidity	Prodromal symptom	Symptom	CSF analysis	Brain MRI	EEG	Anti-GABABR	Treatment
1	M	3	Anti-GABA _A R encephalitis	N	Confusion and lethargy, opsoclonus, dystonic movements of the tongue, ataxia, chorea affecting the limbs and trunk, complex partial seizures	WBC 154×10 ⁶ /L Protein:59 mg/dl	Multifocal T2/FLAIR hyperintensities in the brainstem and cerebellum with involvement of the basal ganglia and hippocampus	Generalised slow waves and epileptiform discharge	Serum(+, NA) CSF(+,NA)	steroids+ IVIG+ Intensive supportive care+ seizure management
2	M	7	Anti-NMDAR encephalitis after herpes simplex encephalitis	Fever	Seizure, psychiatric symptoms	NA	FLAIR hyperintensities in the bilateral frontal and temporal lobes, insula, hippocampus, thalamus and localized atrophy	Increasing in slow wave activity and epileptiform discharge	Serum(+, 1:10) CSF(+,1:1)	Steroids+ Cyclosporin
3	M	NA	CVT and Anti-NMDAR encephalitis	Headache	Mood instability, abdominal pain, Seizure	WBC 90×10 ⁶ /L, slightly increased immunoglobulin	T2/FLAIR: hyperintensity in the right temporal lobe	Spike and slow waves in the right temporal and occipital region	Serum(+,1:10) CSF(+,1:1)	IVIG+Steroids+ RTX + anticoagulation therapy
4	M	6	Anti-CASPR2 encephalitis	NA	Fever, impaired consciousness, sleep disorder, movement disorders, irritability, autonomic symptoms	WBC 214×10 ⁶ /L, Protein: 59.3mg/dl	Lesions in O/P cortex, thalamus, globus pallidus, hippocampus and cerebral peduncle	Slow wave background	Serum(-) CSF(+,NA)	Steroids+ IVIG+ RTX

CBZ, carbamazepine; CSF, cerebrospinal fluid; CVT, Cerebral venous thrombosis; EEG, electroencephalogram; FLAIR, fluid-attenuated inversion recovery; IVIG, intravenous immunoglobulin; M, male; MRI, magnetic resonance imaging; N, No; NA, not available; RTX, rituximab; WBC, white blood cells.

Table3 Descriptive summary of previously reported pediatric anti-GABABR cases according to antibody profile.

Data are presented as n/N, where N is the number of patients with available data for each parameter. NA, not applicable/not reported. CSF, cerebrospinal fluid; EEG, electroencephalography; MRI, magnetic resonance imaging; WBC, white blood cell.

Figures

Feature	Isolated anti-GABABR antibody reports (n = 9)	Co-existing neural autoantibody reports (n = 4)
Age range (years)	8.5-18	3-7
Sex	4 male / 5 female	4 male / 0 female
Prodromal fever or headache	1/7 with available data	2/3 with available data
Clinical Features		
Psychiatric symptoms	8/9	3/4
Cognitive impairments (memory/attention)	3/9	NA
Seizures	3/9	3/4
Impaired consciousness	3/9	2/4
Fever	1/9	1/4
Sleep disorder	3/9	1/4
Speech disorder	1/9	NA
Headache	2/9	NA
Movement disorders	2/9	2/4
Autonomic dysfunction	0/9	1/4
Breathing difficulty	1/9	NA
Abdominal pain	NA	1/4
Opsoclonus	NA	1/4
Tumor reported	0/9	0/4
Paraclinical Findings		
CSF analysis		
Elevated intracranial pressure	1/5with available data	NA
Pleocytosis	2/5 with available data	3/3with available data
WBC count ($\times 10^6/L$)	10 - 30	90 - 214
Elevated protein	1/5with available data	2/3with available data
Protein level (mg/dL)	Mild elevation (39)	Significant elevation (up to 59)
Brain MRI findings		
Normal	3/6with available data	0/4
Abnormal	3/6 with available data	4/4
Multifocal lesions	1/6with available data	3/4
EEG findings		
Normal	2/4 with available data	0/4
Abnormal	2/4 with available data	4/4
Antibody Profiles		
Serum + CSF Positivity	3/6	3/4
Serum Positivity+ CSF Negative	2/6	0/4
Serum Negative+ CSF Positivity	1/6	1/4
Serum-only without CSF data	3/9	0
Presence of co-existing antibodies	0/9	4/4
Treatment & Outcomes		
First-line immunotherapy	9/9	4/4
Second-line immunotherapy	2/9	3/4

Feature	Isolated anti-GABABR antibody reports (n = 9)	Co-existing neural autoantibody reports (n = 4)
Complete recovery at follow-up	8/9	3/4
Mortality	0/9	1/4

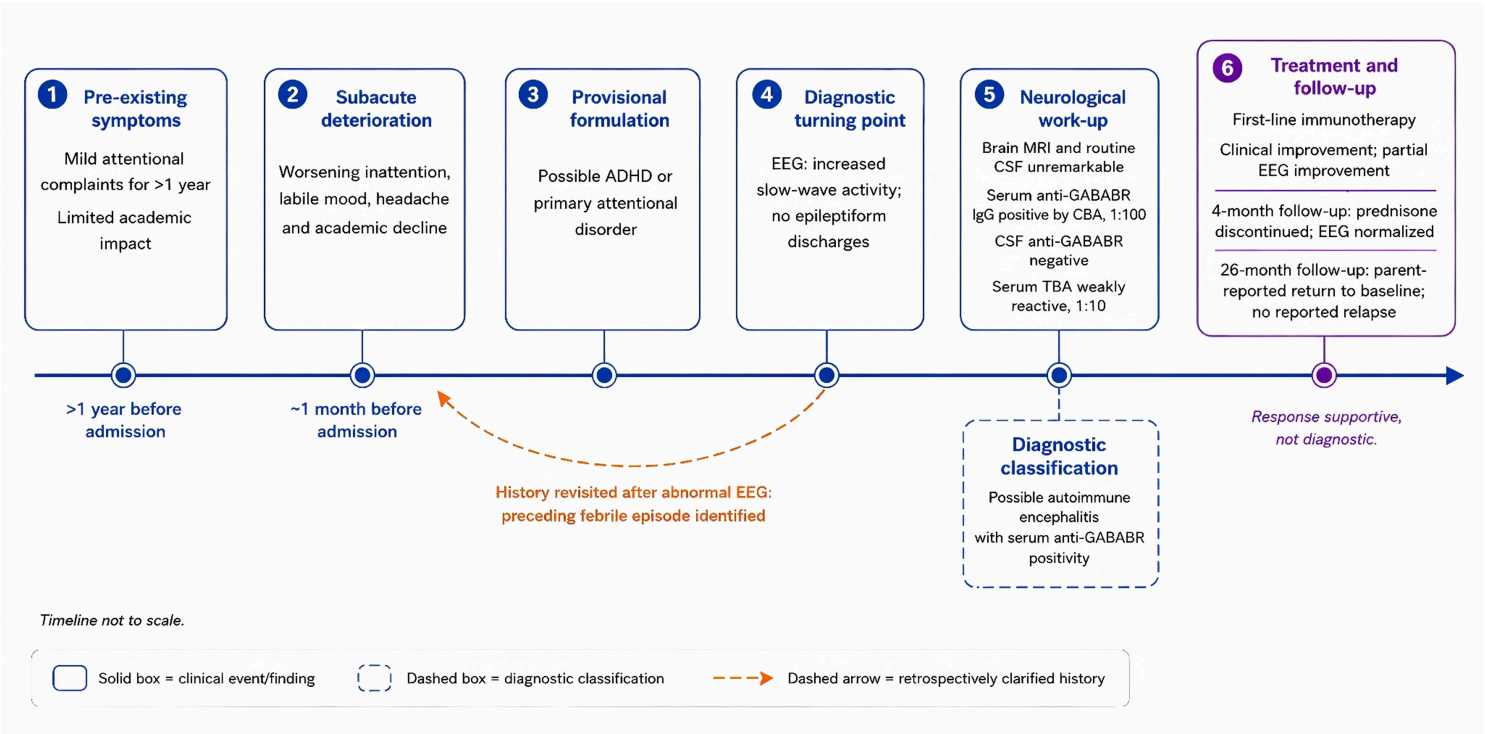


Figure 1

Timeline of clinical course and diagnostic reassessment.

This timeline illustrates the 26-month clinical course of the 9-year-old girl, from psychiatric admission for subacute attentional deterioration (Month 0) through sequential diagnostic assessments (EEG, MRI, CSF, serum antibodies) to immunotherapy and follow-up. The diagnostic turning point—including the revisited febrile prodrome and abnormal EEG findings—is highlighted, culminating in the detection of serum anti-GABABR antibodies (1:100; CSF-negative) and a diagnosis of possible autoimmune encephalitis. Clinical improvement following first-line immunotherapy is shown.

EEG, electroencephalography; CSF, cerebrospinal fluid.