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Cytokine-Based Regenerative Treatment for School Avoidance: A Case Study

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ABSTRACT

A 16-year-old girl presented to our clinic with school avoidance and reduced social motivation. Comprehensive clinical evaluation, including MRI, EEG, and neurological examination, revealed ADHD-like symptoms associated with paroxysmal EEG activity, spike thrombosis involving the middle cerebral arteries, and MRI findings suggestive of traumatic brain injury. The patient received cytokine-based regenerative treatment targeting epilepsy-related paroxysmal activity, ADHD-like symptoms, and traumatic brain injury. During follow-up, school avoidance improved, social motivation increased, and the patient resumed regular school attendance. To our knowledge, this is the first case report describing improvement in school avoidance following cytokine-based regenerative treatment in association with resolution of EEG abnormalities and MRA findings. These findings may offer a potential therapeutic perspective for school avoidance, which is conventionally managed primarily with cognitive behavioral therapy; however, they should be interpreted cautiously as a temporal association rather than evidence of causality.

Keywords: School Avoidance, Cytokine, Regenerative Medicine, Electroencephalography, Traumatic Brain Injury

INTRODUCTION

School avoidance is a heterogeneous condition in which children and adolescents experience persistent difficulty attending school despite no primary intention to be absent. It is influenced by psychological, developmental, familial, and school-related factors, including anxiety, depression, peer conflict, bullying, reduced motivation, and neurodevelopmental vulnerabilities such as attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (ASD) [1,2]. Increasingly, school avoidance is recognized not only as a behavioral or psychosocial problem but also as a condition that may involve neurobiological dysfunction, including impaired attention,

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emotional dysregulation, altered stress responses, sleep–wake rhythm disturbance, and reduced social motivation [2]. Comprehensive assessment from both psychosocial and medical perspectives is therefore important for understanding its pathogenesis and guiding individualized treatment [1].

Previous studies have suggested that neurodevelopmental disorders, particularly ADHD and ASD, are important risk factors for school avoidance [2]. Children with ADHD may have difficulties with sustained attention, impulse control, emotional regulation, and peer relationships, which can contribute to academic failure, interpersonal conflict, bullying, and reduced motivation to attend school [3]. Similarly, children with ASD often experience sensory hypersensitivity, social communication difficulties, intolerance of environmental change, and comorbid anxiety, all of which may increase school-related distress [4]. These findings indicate that school avoidance should not be regarded solely as a psychological or behavioral problem but may also reflect underlying neurodevelopmental vulnerability requiring comprehensive assessment and individualized intervention. EEG abnormalities may also reflect neurobiological dysfunction associated with school avoidance, including impairments in attention, emotional regulation, and cognition in children and adolescents [5,6].

Cognitive behavioral therapy (CBT)-based interventions, including graded exposure, parent involvement, and school reintegration, have shown benefits for improving attendance and emotional symptoms in school avoidance [7,8]. However, the evidence remains limited by small sample sizes and few rigorous randomized trials, and pharmacotherapy is generally used as an adjunct for comorbid anxiety or depression [8].

In the present case report, we describe a 16-year-old girl with school avoidance and reduced social motivation who exhibited ADHD-like symptoms accompanied by paroxysmal EEG activity, spike thrombosis involving the middle cerebral arteries, and MRI findings suggestive of traumatic brain injury. Following cytokine-based regenerative treatment, school avoidance and social motivation improved, and the patient resumed regular school attendance. Although causal inference cannot be drawn from a single case, this report may provide a clinically relevant perspective on school avoidance beyond conventional cognitive behavioral approaches.

METHODS

The cytokine and exosome cocktail formulation used in this study was designed and developed by Luis Carlos Aguilar Cobos at the Livant Neurorecovery Center, Mexico, as described previously [9-15]. Seven cytokine- and exosome-based formulations were administered in this case study (Table 1). Gabatrof (EPI), EPI, and OST SHELL were selected for their putative roles in supporting GABAergic neurogenesis and modulating cortical excitability through HGF, G-CSF, miR-124, progranulin, and calbindin-related mechanisms. Neurogen EP contained miR-124–enriched exosomes derived from young adult porcine brain, whereas Renotrof (23), SAL EXO MIX, and SAL100 contained HPLC-purified GDNF, omega-3 fatty acids, and salmon-derived exosomes. All formulations were administered sublingually three times daily.

Table 1. Cytokine and exosome formulations used in this study

Formulation	Main Components	Intended Clinical Target
Gabatrof (EPI), EPI	HGF, G-CSF, adiponectin, miR-124, progranulin, and calbindin-related components	Support of GABAergic mechanisms and modulation of cortical excitability
OST SHELL	High concentration of calbindin-28	Reduction of excessive glutamatergic excitation
Neurogen EP	Young adult porcine brain-derived exosomes enriched in miR-124	Support of cortical and frontotemporal function
Renotrof (23), SAL EXO MIX, SAL100	HPLC-purified GDNF, omega-3 fatty acids, and salmon-derived exosomes	Support of attention, motivation, and dopaminergic-related pathways

CASE DESCRIPTION

A 16-year-old girl from Gunma, Japan, presented to Ochanomizu Health and Longevity Clinic on March 26, 2024, with school avoidance, orthostatic hypotension, impaired concentration, and disruption of her daily rhythm. She had no significant past medical history and had been delivered vaginally without perinatal complications. She had received four doses of a COVID-19 mRNA vaccine, with the fourth dose administered on October 24, 2022. Beginning in November 2022, she developed difficulty waking in the morning, which progressively interfered with school attendance. In February 2023, she was diagnosed with orthostatic dysregulation at a local hospital. Her younger brother had been diagnosed with autism spectrum disorder; otherwise, there was no family history of psychiatric or neurological disorders.

Initial Assessment

The initial EEG assessment, recorded on March 26, 2024 (Table 1, Figure 1), showed clusters of paroxysmal activity composed primarily of sharp theta waves. These activities showed apparent propagation predominantly to the frontal and temporal lobes and, to a lesser extent, to the parietal and occipital lobes (Figure 2A, 2B). The findings suggested cortical hyperexcitability or multiple epileptiform foci associated with abnormal electrophysiological activity (Figure 2A, 2B). Flash visual evoked potentials demonstrated asymmetric responses in the frontal, temporal, and parietal lobes (Figure 3A). Dissociation was observed between the left and right anterior temporal leads (T3 and T4) and between the left and right posterior temporal leads (T5 and T6) (Figure 3A). In the attention test, task-related brain activity was markedly suppressed compared with the control response (Figure 3B). During the mental flexibility test using the Wisconsin Card Sorting System, a premature low-voltage brain response was observed, suggesting possible disorganization of ideational processing associated with cortical epileptiform activity (Figure 3C). P300 EEG responses to high-pitched sound stimulation exhibited asymmetric electrical activity across the frontal, central, temporal, parietal, and occipital regions (Figure 4A). Magnified P300 responses to low-pitched sound stimulation in the posterior temporal lobes demonstrated substantial dissociation between T5 and T6. The P300 peak was delayed to 650 ms in both posterior temporal leads (Figure 4C). P300 coherence analysis showed mildly decreased fluctuations in the F3, F4, and Cz leads, suggesting impaired inhibitory GABAergic circuitry in the frontal and central brain regions (Figure 4D).

Analysis of MRI data acquired on March 26, 2024, revealed increased volume of the frontal and temporal lobes, as shown in the three-dimensional reconstructions from the left lateral view (Figure 5A) and right superolateral view (Figure 5C). MRI also demonstrated fibrotic adhesion of the arachnoid membrane along the cerebral falx and over both parietal lobes, as indicated by red asterisks in Figures 5A–C. The patient had participated in school-based kendo for approximately 18 months, and the MRI findings were considered potentially consistent with kendo-associated concussion. A two-dimensional coronal section of the T1-weighted MRI image showed a mild increase in white matter volume and narrowing of both lateral ventricles, as indicated by the red asterisk and the red arrow in Figure 5D.

As shown in Figure 7, virtual endoscopic imaging also demonstrated intraluminal material within the right and left middle cerebral arteries, with apparent attachment to the endothelial surface of the left middle cerebral artery (Figure 7B). Based on their morphology and vascular location, these lesions were considered compatible with spike thrombosis, as previously described [16].

Based on electrophysiological and morphological evaluations, the patient was considered to have ADHD-like symptoms potentially associated with multiple contributing factors, including traumatic brain injury, genetic vulnerability, and angiitis associated with spike thrombosis.

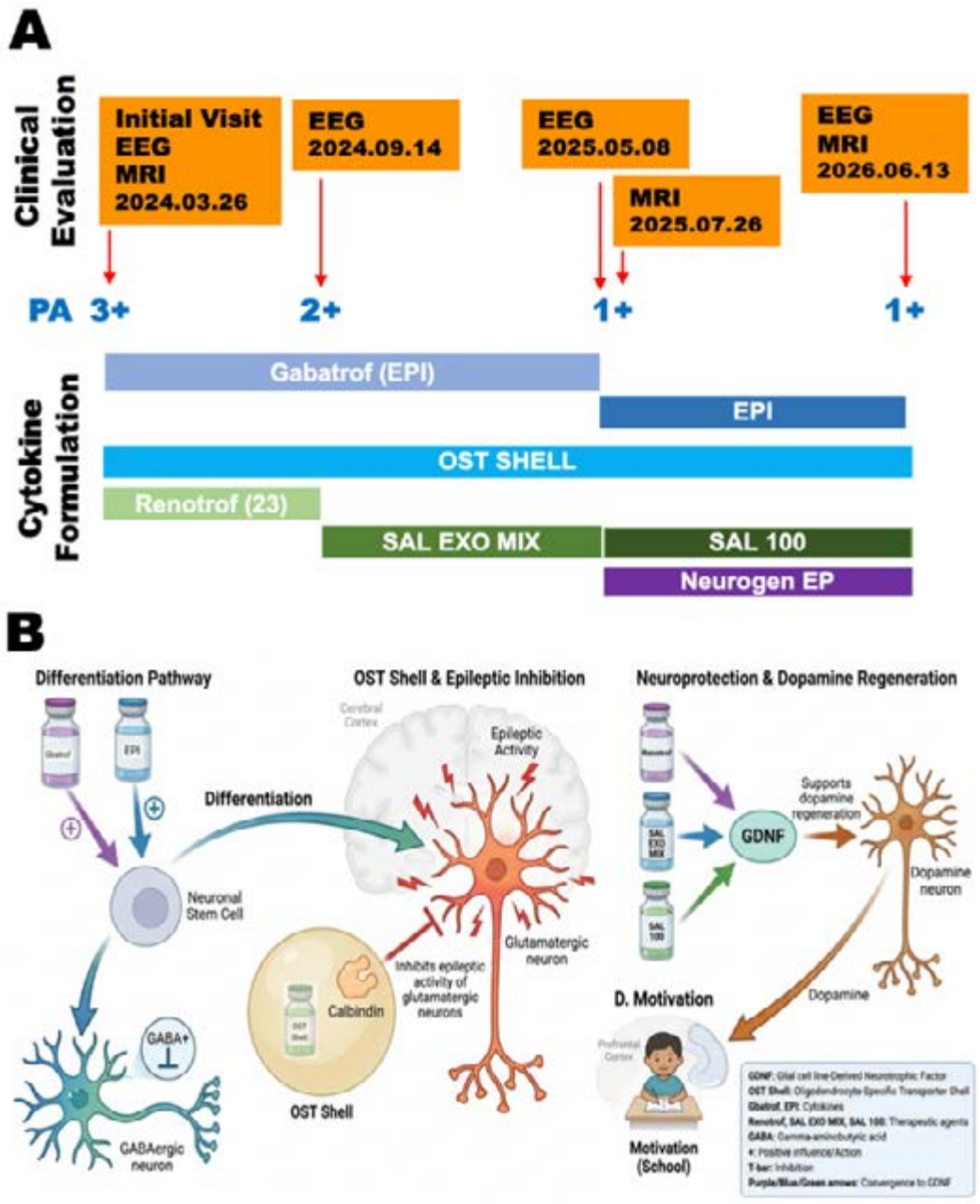

Figure 1.

A. Clinical evaluations are shown over the course of cytokine-based therapy. Paroxysmal activity (PA) on resting electroencephalography (EEG) is indicated. The cytokine formulations administered during treatment are shown below.

B. The pharmacological functions of the cytokines used in this case study are illustrated. The figure was created using Figurelabs.ai.

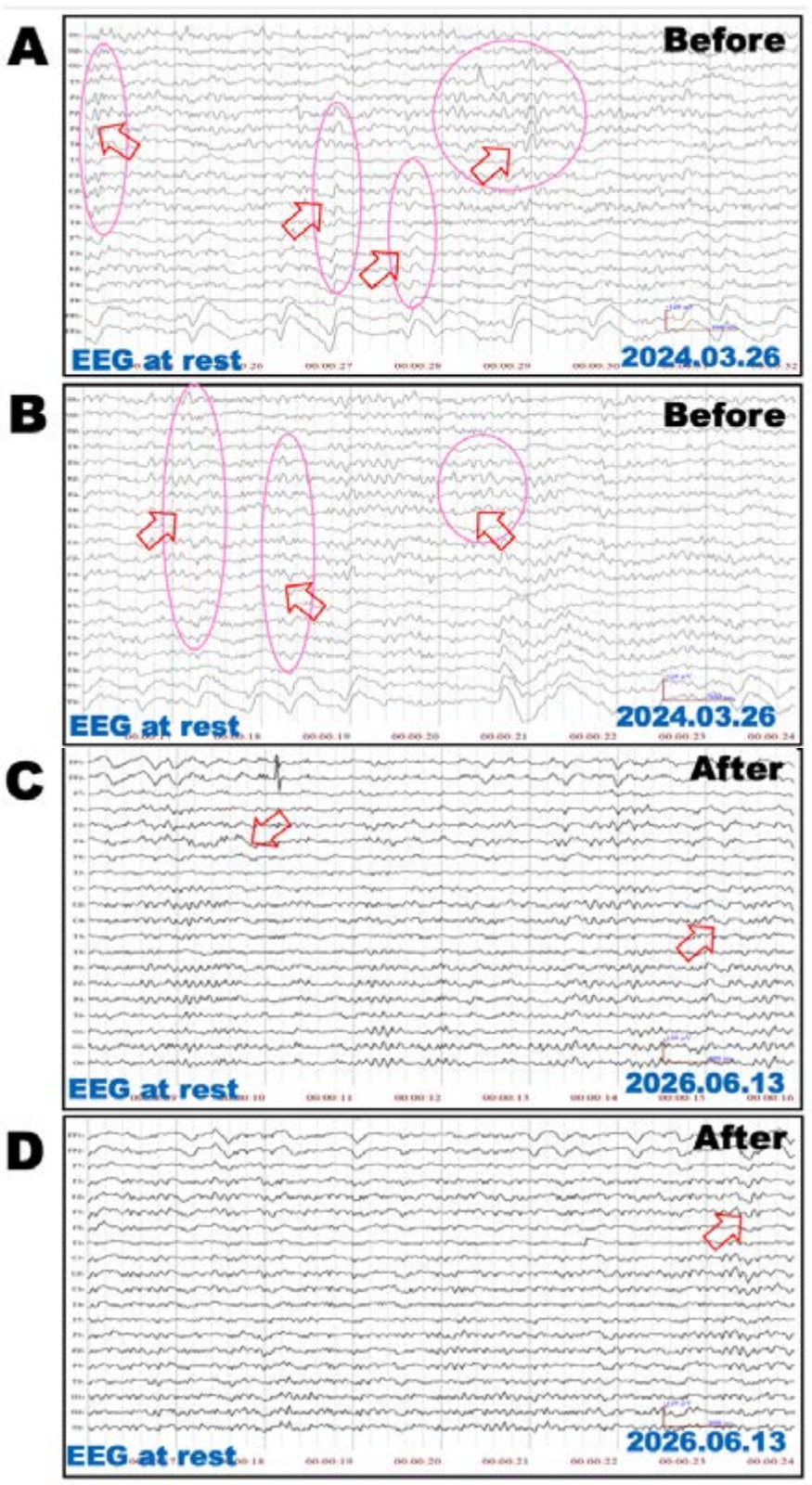

Figure 2.

A and B: Resting electroencephalography (EEG), recorded before cytokine-based treatment on March 26, 2024, showed clusters of paroxysmal activity composed primarily of sharp theta waves. Red arrows indicate the sharp theta-wave activity, and red circles indicate the apparent propagation of paroxysmal activity predominantly to the frontal and temporal lobes and, to a lesser extent, to the parietal and occipital lobes.

C and D: Electroencephalograms (EEGs) were recorded following the administration of cytokine-based therapeutic interventions. The presence of sharp theta waves is indicated by the red arrows.

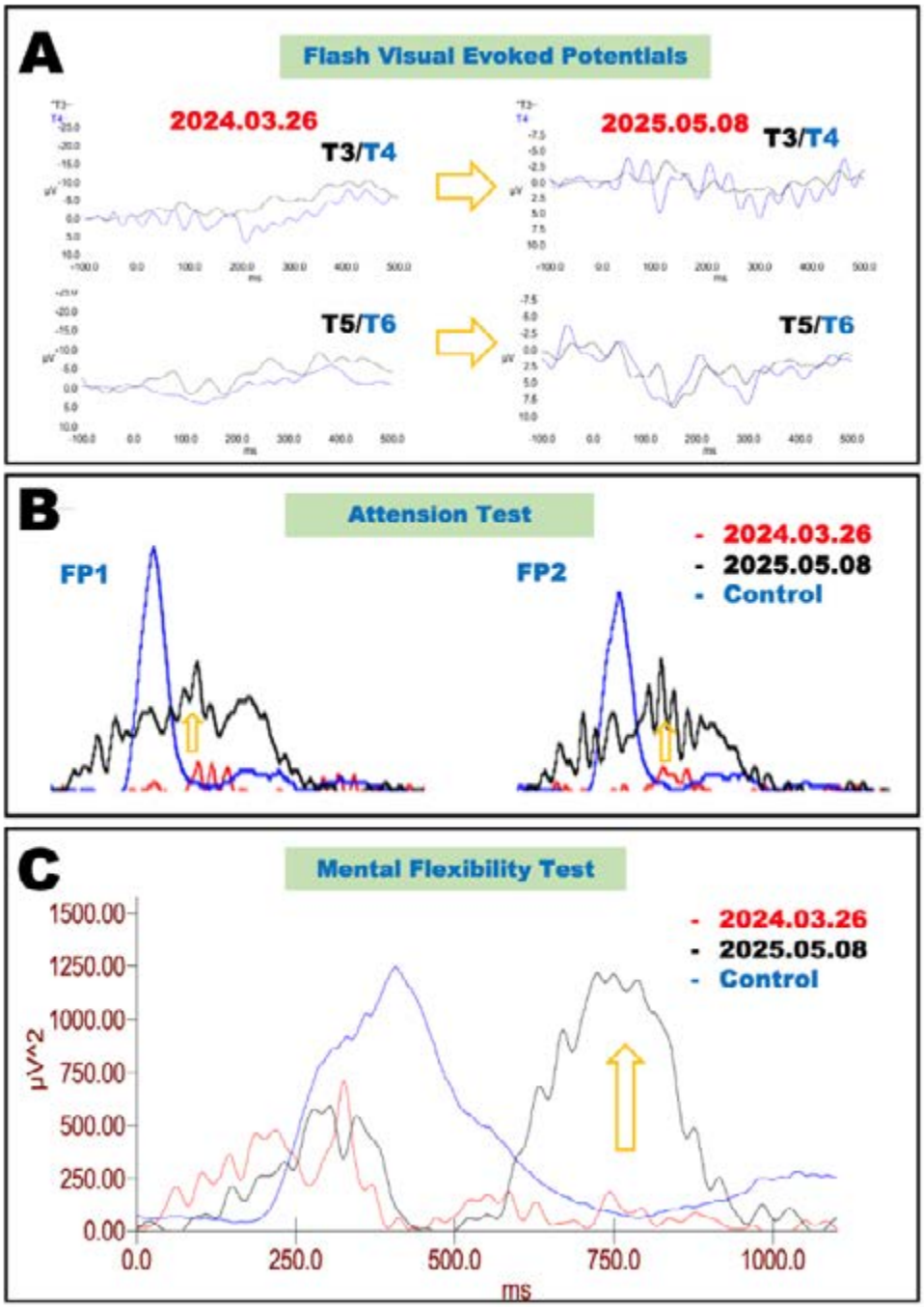

Figure 3.

A. Flash visual evoked potentials obtained before and after cytokine-based treatment. In the left panel, asymmetric responses were observed in the frontal, temporal, and parietal lobes before treatment, with dissociation between the left anterior temporal lead (T3) and the right anterior temporal lead (T4), as well as between the left posterior temporal lead (T5) and the right posterior temporal lead (T6). These dissociations appeared to improve after cytokine-based treatment, as shown in the right panel.

B. Attention-test EEG responses obtained before and after cytokine-based treatment. In the left panel, task-related brain activity recorded before treatment on March 26, 2024, was markedly suppressed, as indicated by the red line, compared with the control response, indicated by the blue line. The right panel shows the corresponding response after cytokine-based treatment on July 13, 2026, indicated by the black line.

C. EEG responses during the mental flexibility test using the Wisconsin Card Sorting System, obtained before and after cytokine-based treatment. In the left panel, a premature low-voltage brain response was observed before treatment on March 26, 2024, compared with the control response, indicated by the blue line. The right panel shows the corresponding response after cytokine-based treatment on July 13, 2026, indicated by the black line. The yellow arrow indicates the premature low-voltage response.

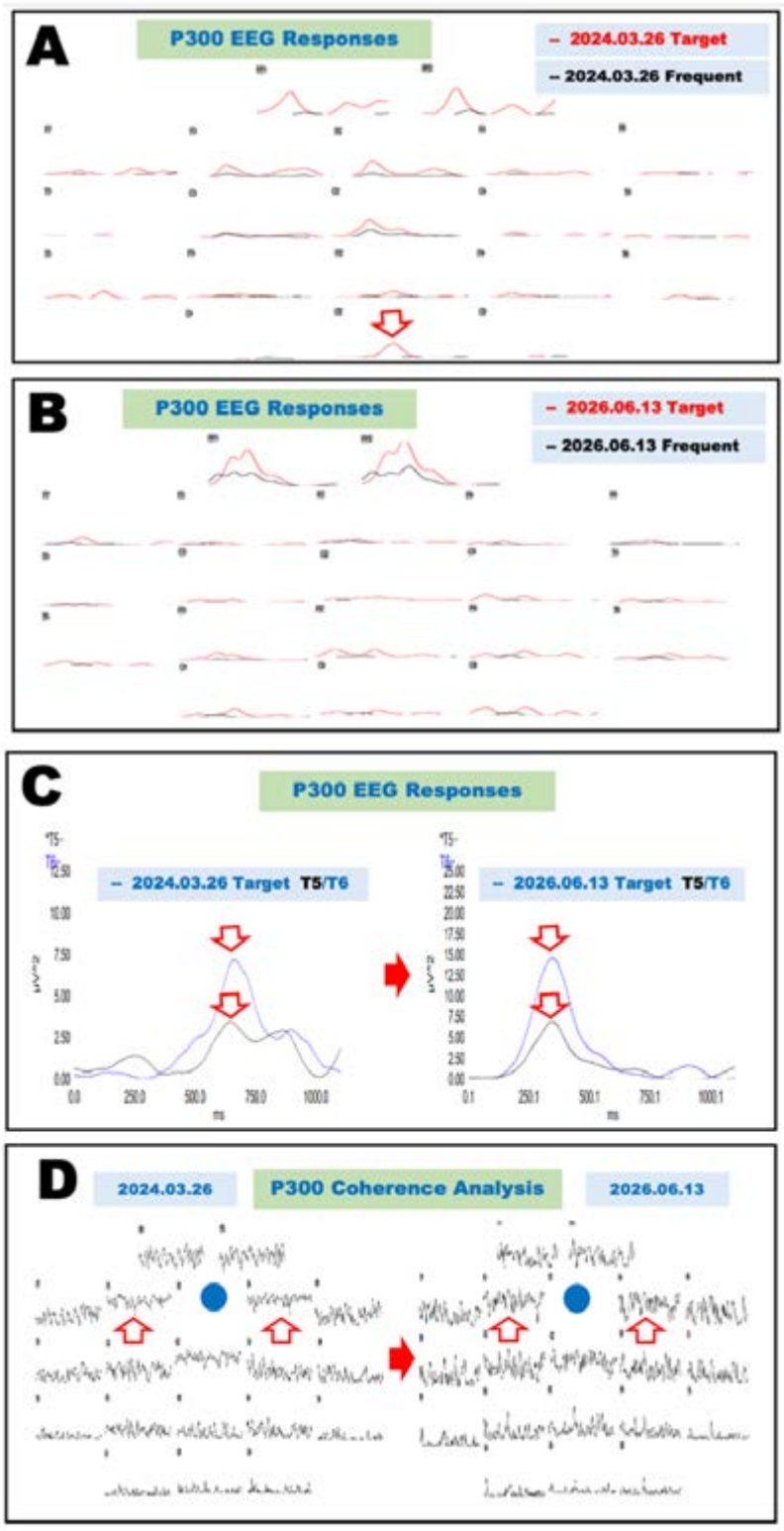

Figure 4.

A. P300 EEG responses during cytokine-based treatment. Before treatment, on March 26, 2024, responses to frequent stimuli (low-pitched sounds) are shown in black, and responses to target stimuli (high-pitched sounds) are shown in red.

B. P300 EEG responses after cytokine-based treatment on July 13, 2026.

C. Magnified P300 EEG responses from T6 (right posterior temporal electrode; blue) and T5 (left posterior temporal electrode; black) before treatment on March 26, 2024 (left panel), and after treatment on July 13, 2026 (right panel).

D. P300 coherence analysis during cytokine-based treatment. Neural network connectivity at the frontal electrodes (F3 and F4, red arrows) showed lower fluctuation before treatment on March 26, 2024 (left panel), and increased connectivity after treatment on July 13, 2026 (right panel).

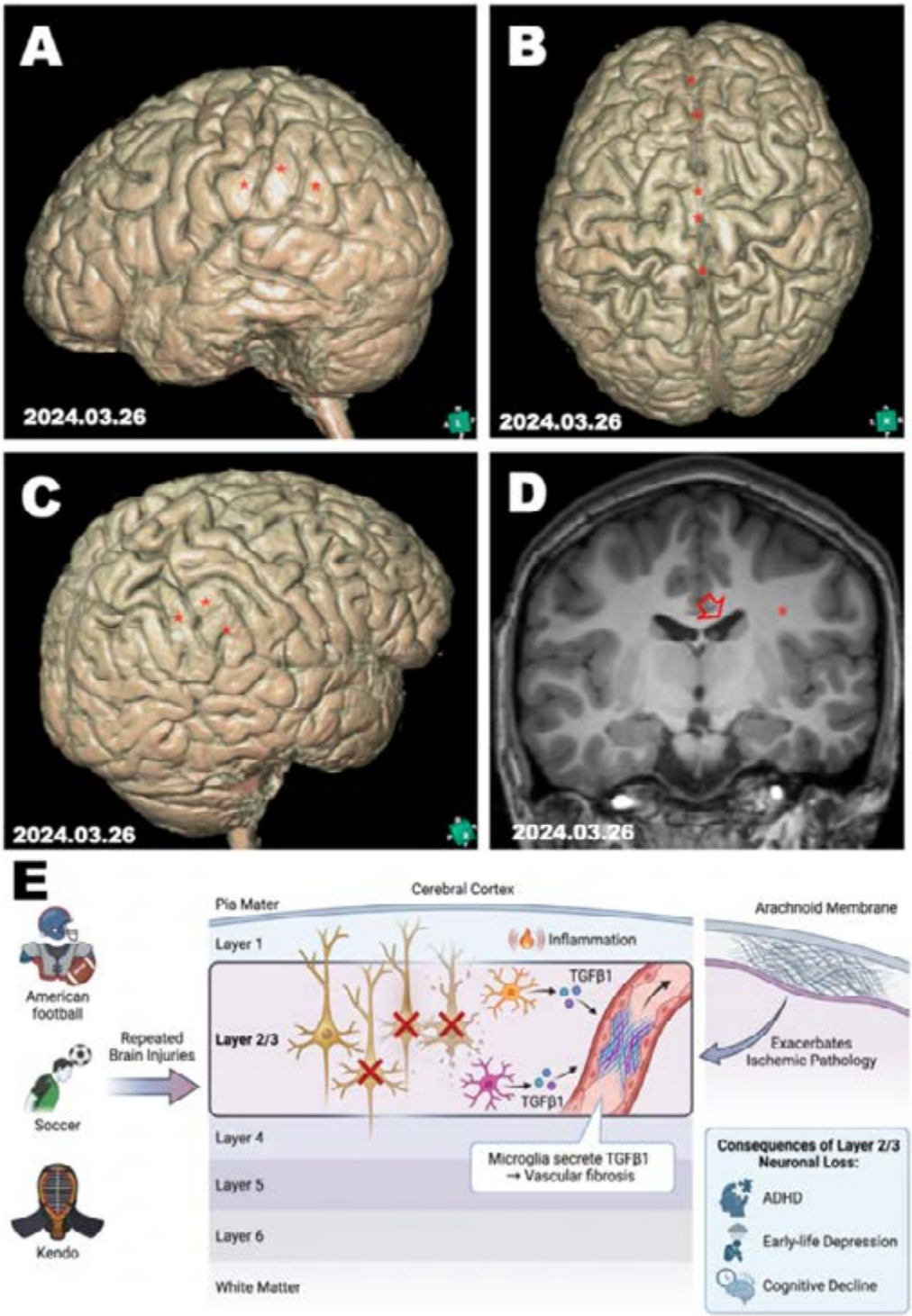

Figure 5.

A–C: MRI was performed on March 26, 2024, before cytokine-based treatment. A three-dimensional brain reconstruction was generated in silico from T1-weighted MRI images acquired as 1-mm sagittal slices using Expert INTAGE software. Panels A–C show the left lateral, horizontal, and right superolateral views, respectively.

D: A coronal section of a T1-weighted MRI image is shown.

E: Schematic representation of traumatic brain injury pathology. Panel E was generated using Figurelabs.ai.

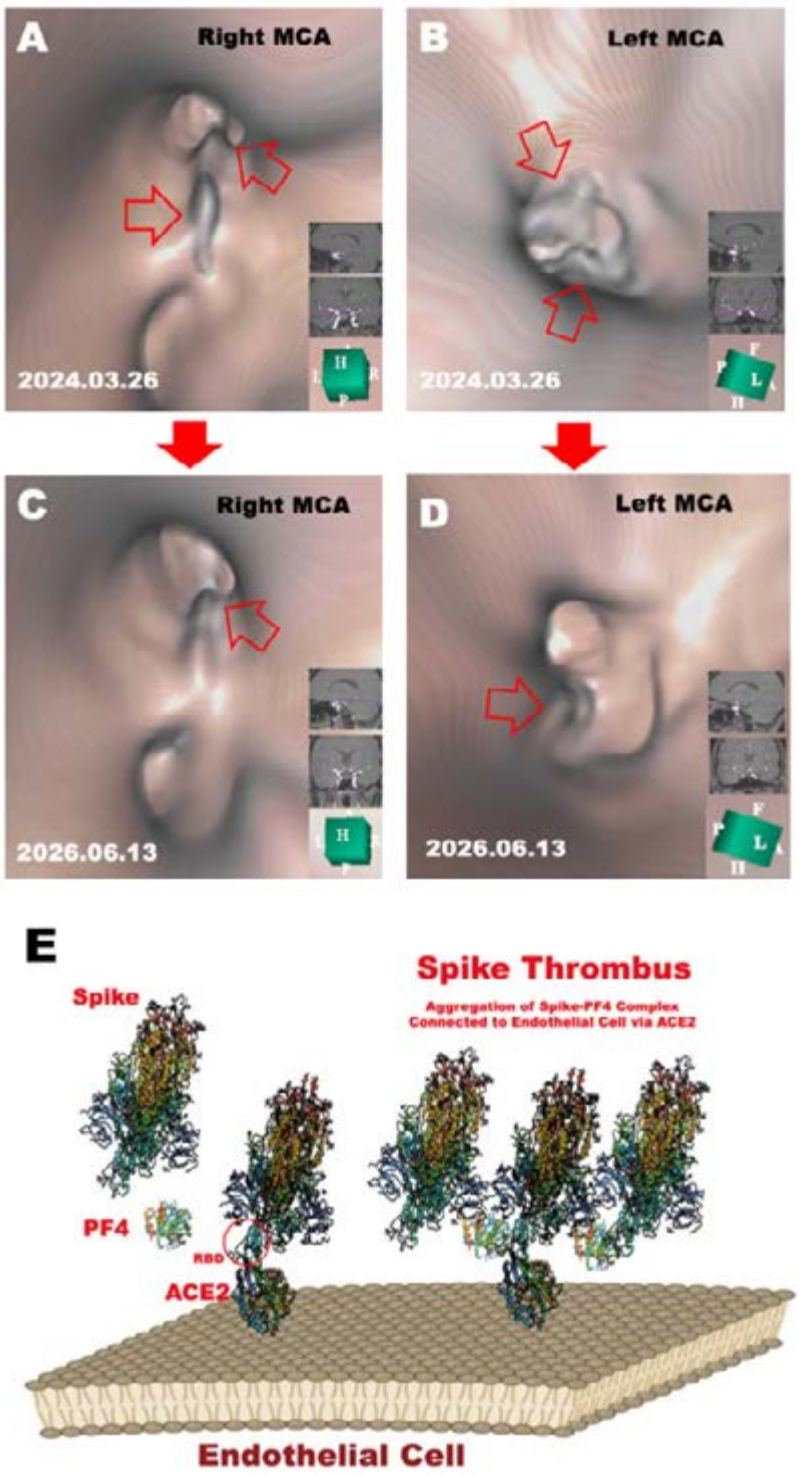

Figure 6.

A–B: Endoscopic in silico views of thrombosis within the right (A) and left (B) middle cerebral arteries, indicated by red arrows, recorded on March 26, 2024, before cytokine-based treatment.

C–D: Endoscopic in silico views of thrombosis within the right (C) and left (D) middle cerebral arteries, indicated by red arrows, recorded on June 13, 2026, after cytokine-based treatment.

E: The RBD of the spike protein from COVID-19 (PDB, 6VXX) can bind the PF4 protein (PDB, 1F9Q) secreted from activated platelets [17]. The RBD of the spike protein also binds to the ACE2 receptor (PDB, 7VXM) in endothelial cells. The molecular structure of the spike protein is a trimer of spike peptides, in which one spike molecule has three RBD domains that can bind either PF4 or ACE2. Spike and PF4 proteins can aggregate and form filamentous thrombi that become attached to endothelial cells via the ACE2 receptor in the outer membrane of endothelial cells.

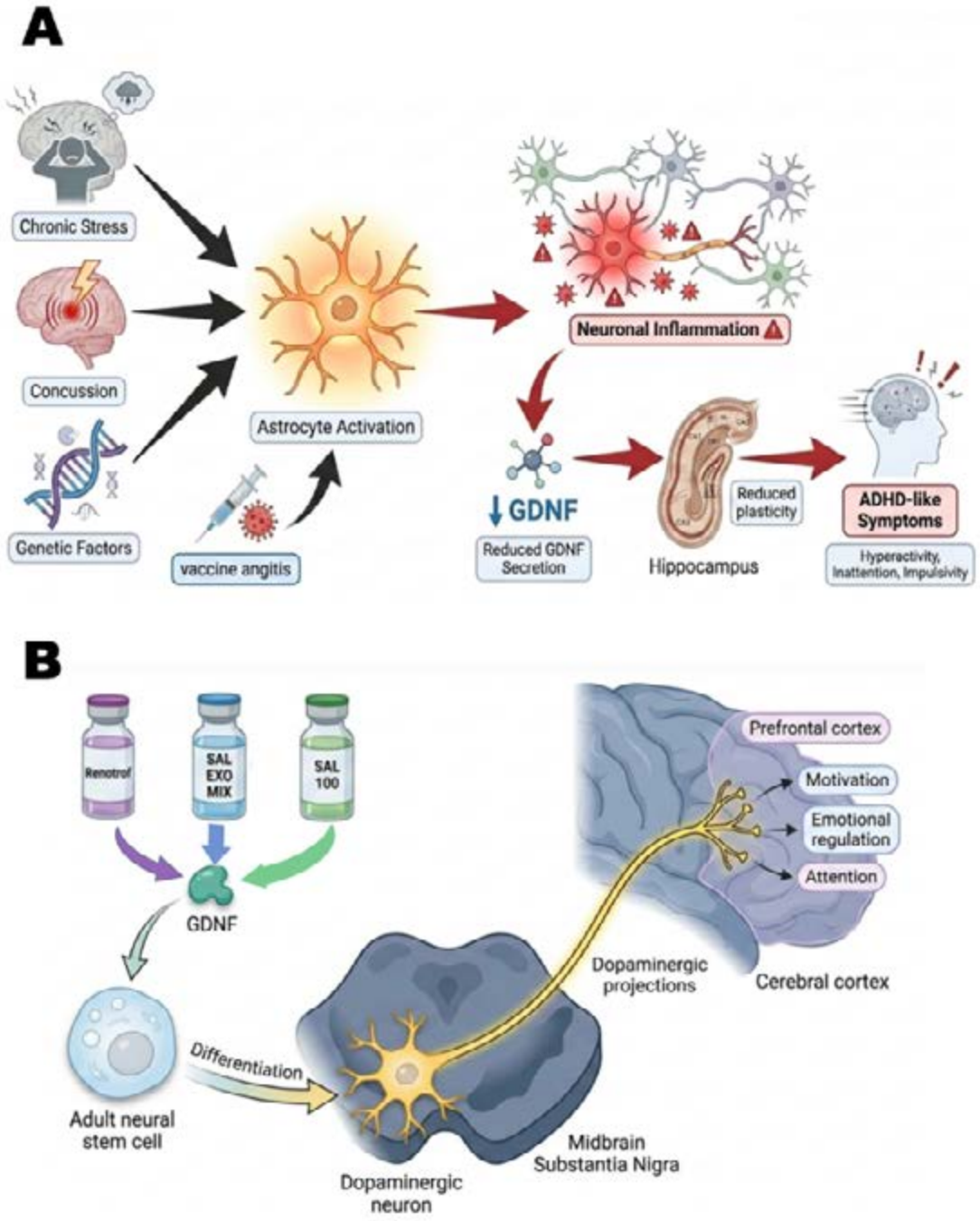

Figure 7.

A: Pathogenesis of ADHD-like symptoms. Chronic stress, brain concussion, genetic factors, and vaccine-associated angiitis activate astrocytes and induce neuronal inflammation, which subsequently decreases GDNF expression and causes hippocampal atrophy and ADHD-like symptoms.

B: Cytokines, Renotrof, SAL EXO MIX, and SAL 100 contain porcine GDNF, which induces adult stem cells in the substantia nigra of the midbrain and enhances the differentiation of dopaminergic neurons projecting to the prefrontal cortex. The prefrontal cortex plays physiological roles in behavioral control, including motivation, emotional regulation, and attention.

Cytokine-Based Treatment and Follow-Up

Based on these findings, a cytokine-based therapeutic regimen was selected. Gabatrof (EPI) (x6) 4.0 ml three times daily and OST SHELL (x3) 2.0 ml three times daily were prescribed with the aim of reducing paroxysmal brain activity observed on resting EEG. Renotrof (23) (x6) 1.0 ml three times daily was also prescribed with the aim of supporting attention and mental flexibility (Figure 1A).

A second EEG evaluation performed on September 14, 2024 (Table 2), showed reduced paroxysmal activity and improved responses in the attention and mental flexibility tests. Renotrof (23) was subsequently replaced with SAL EXO MIX (x3) 2.0 ml three times daily (Figure 1A).

A third EEG evaluation performed on May 8, 2025 (Table 2), showed a further reduction in paroxysmal activity, along with improved responses in the attention and mental flexibility tests (Figure 3B, 3C). Dissociation of flash visual evoked potentials between the right and left temporal leads was markedly reduced, suggesting possible functional improvement in cortical neuronal networks (Figure 3A).

A fourth EEG evaluation performed on June 13, 2026 (Table 2), showed improvement in asymmetric P300 responses across the frontal, central, temporal, and parietal lobes (Figure 4B). Furthermore, the hyperexcitable P300 response at the Oz lead observed on March 26, 2024 (indicated by red arrow in Figure 4A), was substantially attenuated by July 13, 2026 (Figure 4B). By July 13, 2026, the delayed P300 peak had improved, with peak latency near 300 ms in both the T5 and T6 leads (Figure 4C). P300 coherence analysis showed increased fluctuations and greater apparent neuronal connectivity in the frontal lobe (Figure 4D). Virtual endoscopic imaging also demonstrated apparent substantial resolution of spike thrombosis in the right and left middle cerebral arteries (Figure 6C, D).

Four months after the initiation of cytokine-based treatment, physical symptoms such as dizziness improved, followed by a gradual recovery of motivation. By 10 months after treatment initiation, the patient was attending school regularly and had joined the drama club. At the fourth evaluation on June 13, 2026, the patient no longer exhibited symptoms of school avoidance.

Table 2. Dates, evaluations, and main findings in this study

Date or Interval	Evaluation or Treatment	Main Finding or Clinical Change
March 26, 2024	Initial EEG, MRI, and vascular imaging	Paroxysmal EEG activity, asymmetric evoked responses, MRI findings suggestive of traumatic brain injury, and vascular abnormalities interpreted as spike thrombosis
September 14, 2024	Second EEG evaluation	Reduced paroxysmal activity and improved attention and mental flexibility responses
May 8, 2025	Third EEG evaluation	Further reduction in paroxysmal activity and improved cortical network responses
June–July 2026	Fourth EEG and vascular follow-up	Improved P300 responses, increased frontal connectivity, apparent vascular improvement, and resolution of school avoidance symptoms

DISCUSSION

In this case study, a 16-year-old girl presented with school avoidance and reduced social motivation and underwent comprehensive clinical evaluation, including MRI, EEG, and neurological examination. The findings were compatible with ADHD-like symptoms associated with paroxysmal EEG activity, vascular abnormalities interpreted as spike thrombosis involving the middle cerebral arteries, and MRI findings suggestive of traumatic brain injury. The patient received cytokine-based regenerative treatment selected to address paroxysmal EEG activity, ADHD-like symptoms, and suspected traumatic brain injury. During follow-up, school avoidance improved, social motivation increased, and the patient resumed regular school life. These observations indicate a temporal association between cytokine-based regenerative treatment, improvement in clinical symptoms, and changes in EEG and vascular imaging findings; however, they should not be interpreted as evidence of causality.

To our knowledge, this is the first case report describing improvement in school avoidance following cytokine-based regenerative treatment in association with reduced epileptiform EEG activity and improved MRA findings. These findings may offer a hypothesis-generating therapeutic perspective for school avoidance, which is conventionally managed primarily with cognitive behavioral therapy.

Epileptiform Brain Activity as a Potential Contributor to School Avoidance

In the present case, brain MRI revealed findings interpreted as consistent with sports-related traumatic brain contusion, together with vascular abnormalities interpreted as spike thrombosis involving the middle cerebral arteries [16]. These findings raise the possibility that traumatic and vascular mechanisms contributed to the patient's neurobiological profile. The paroxysmal activity observed on resting EEG may have reflected ischemic or irritative cortical changes related to peripheral cerebral vascular abnormalities, sports-related trauma (Figure 5B), or both (Figure 6). Notably, occipital hyperexcitability on the initial P300 EEG recording at the Oz lead (Figure 4A), together with asymmetric abnormalities on flash-evoked visual evoked potentials (Figure 3A), was not readily explained by traumatic injury alone. In addition, reduced responsiveness in the attention and mental flexibility tests at the initial examination suggests that ADHD-like symptoms may have contributed to school avoidance (Figure 8). Given the patient's family history of autism spectrum disorder and the symmetrical recovery of EEG findings in the attention test between the initial examination and recovery phase (Figure 3B), a genetic contribution to the ADHD-like symptoms remains possible.

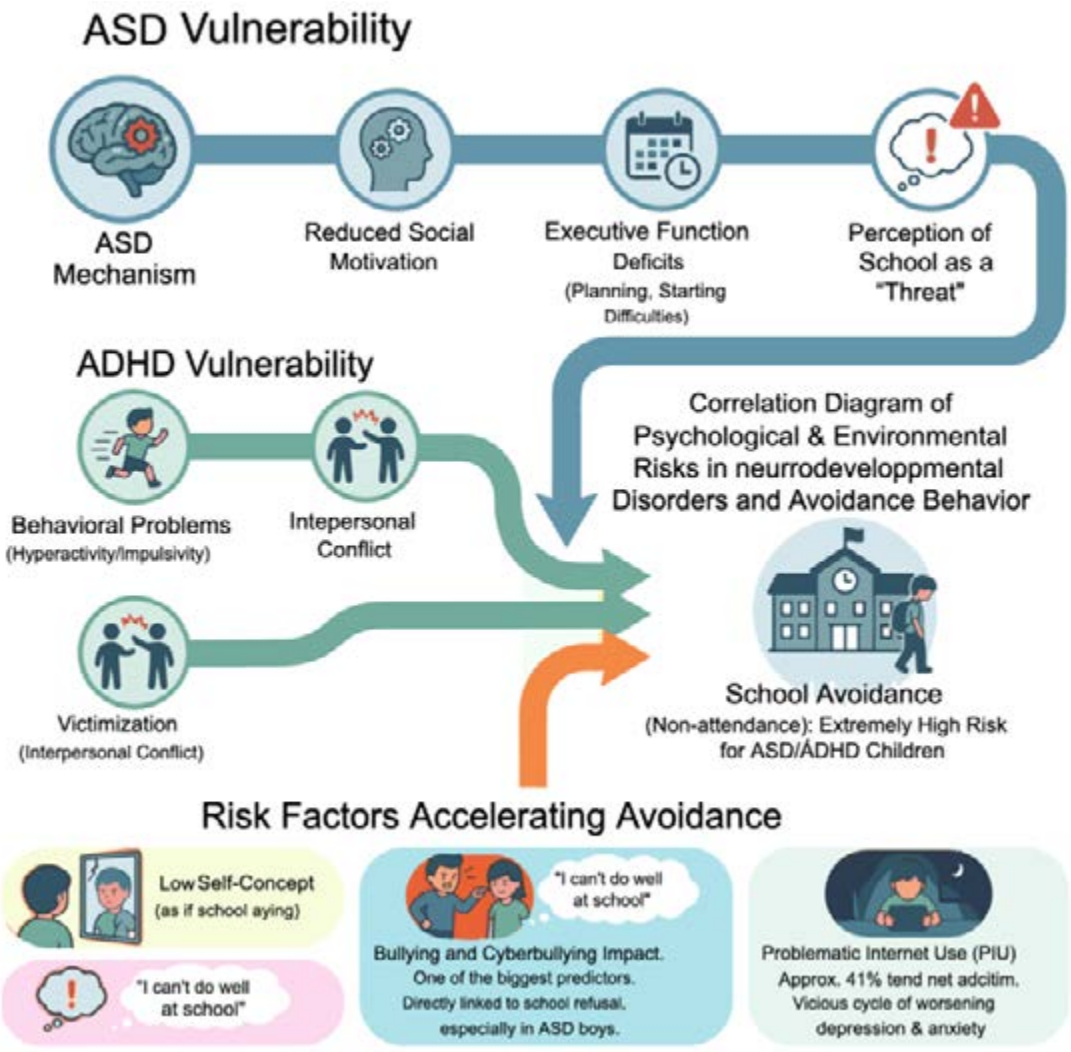

Figure 8. Correlation diagram illustrating psychological and environmental risk factors associated with neurodevelopmental disorders and avoidance behaviors.

Vaccination as a Potential Precipitating Factor

In this case, dizziness, the earliest clinical manifestation, developed shortly after administration of the fourth dose of a COVID-19 mRNA vaccine. This temporal relationship raises the possibility, but does not establish, that vaccination may have been one of several precipitating factors for subsequent neuropsychiatric symptoms associated with school avoidance [18]. Vascular abnormalities interpreted as spike thrombosis were identified at the origins of the bilateral middle cerebral arteries, suggesting that broad cerebral territories may have been vulnerable to thromboembolic or inflammatory vascular events. By June 13, 2026, these lesions had almost completely resolved, suggesting a reduced likelihood of ongoing vascular contribution to paroxysmal activity.

Sport-Related Traumatic Brain Injury as A Potential Contributor to The Pathology

In the present case, three-dimensional MRI demonstrated arachnoid fibrotic changes over the brain surface (Figure 5B). Repeated head impacts during school-based kendo may have produced concussive forces and mechanical friction between the cerebral falx and cerebral hemispheres, potentially contributing to these findings. Consistent with this interpretation, sport-related brain injury has been associated with neuroinflammation, vascular fibrosis, ADHD, early-life depression, and cognitive decline as shown in Figure 5E [19,20].

Cytokine-Based Treatment for Brain Pathology Associated With School Avoidance

In the present case, resting-state EEG demonstrated paroxysmal activity with seizure-like propagation, suggesting abnormal cortical excitability that may have contributed to the behavioral phenotype of school avoidance. MRI findings were suggestive of cerebral contusion, whereas MRA demonstrated vascular abnormalities interpreted as spike thrombosis. On this basis, the abnormal EEG activity was considered potentially associated with neuropathological changes related to recurrent cerebral contusion and microthromboembolism. Both traumatic and microvascular pathologies have been reported to promote neuroinflammatory processes [21,22].

GABAergic neurons are particularly vulnerable under ischemic and inflammatory conditions, and loss or dysfunction of these inhibitory interneurons has been documented in such pathological states [23]. Gabatrof (EPI) was administered with the aim of supporting regeneration or functional restoration of GABAergic neurons. Gabatrof (EPI) contains high concentrations of hepatocyte growth factor (HGF) and granulocyte colony-stimulating factor (G-CSF), both of which have been reported to act on neural stem cells and promote differentiation toward a GABAergic neuronal lineage in experimental contexts [24].

OST SHELL was administered concomitantly with the proposed aim of reducing epileptiform activity by attenuating excessive glutamatergic excitation through its calbindin-D28k content [25]. EPI is a cytokine cocktail containing HGF, G-CSF, adiponectin, miR-124, progranulin, and calbindin and is used in epilepsy-related pathology. Because the EEG examination on May 8, 2025, showed a reduction in paroxysmal activity, Gabatrof (EPI) was replaced with EPI.

Renotrof (23), SAL EXO MIX, and SAL100 were administered with the aim of supporting social motivation. These cytokine- and exosome-based formulations contain glial cell line-derived neurotrophic factor (GDNF), which has been reported to promote dopaminergic neuronal differentiation from adult stem cells in the midbrain substantia nigra in experimental models (Figures 1B and 7B) [26]. In this case, initially reduced EEG responsiveness during the attention test improved after cytokine-based treatment, accompanied by apparent recovery of social motivation and resumption of school attendance.

NEUROGEN EP contains porcine brain-derived exosomes that are proposed to reduce cortical excitability, stimulate frontotemporal lobe function, and activate cytokine-mediated signaling in neurons of the brain and spinal cord. It has been reported to support cognitive function and brain development in prior reports [27]. In the present case, NEUROGEN EP was administered to help maintain the observed behavioral improvement.

No adverse effects of cytokine-based treatment were observed during the follow-up of the present case.

Neuroinflammation, Reduced GDNF, and Social Motivation

In this case, sports-related concussion, possible vaccine-associated vascular abnormalities, ASD-related genetic vulnerability, and school-related stress may have

contributed to EEG abnormalities through mechanisms involving astrocyte activation, chronic neuroinflammation, and reduced astrocyte-derived GDNF secretion (Figure 7A). Reduced GDNF may impair hippocampal plasticity and nigrofrontal dopaminergic pathways, potentially contributing to reduced social motivation, impaired attention, and ADHD-like symptoms (Figure 7) [28]. Because these factors may be present in some children with school avoidance, EEG and MRI evaluation may help identify potentially treatable neuropathology in selected cases.

ASD and ADHD Vulnerability in School Avoidance

Since 2000, reported diagnoses of ASD and ADHD have increased markedly in Japan and the United States [29]. In children with ASD, reduced school-related motivation and impaired executive function may cause school experiences to be perceived as threatening, thereby increasing vulnerability to school avoidance (Figure 8) [30]. In children with ADHD, hyperactivity, impulsivity, and peer-related difficulties may increase vulnerability to bullying and subsequent school avoidance [31]. Although behavioral interventions remain central, medical evaluation may be considered when clinical findings suggest underlying neurological or physiological contributors. Neuroregenerative approaches remain investigational in this context but may warrant further study as potential complements to behavioral therapy.

Limitations

This report has several important limitations. First, it describes a single patient and cannot establish causality or generalizability. Second, the interpretation of EEG, MRI, and vascular imaging findings should be considered exploratory. Third, spontaneous improvement, psychosocial factors, school-related changes, and placebo effects cannot be excluded. Controlled studies are needed to determine whether cytokine- or exosome-based interventions have reproducible safety and efficacy in patients with school avoidance.

Clinical Implications

This case supports a hypothesis-generating combined care model in which behavioral therapy and medical evaluation are integrated according to each patient's clinical profile. Behavioral approaches should continue to address self-esteem, peer relationships, bullying, avoidance behavior, internet dependence, and gradual school reintegration. When EEG or MRI identifies potentially treatable neuropathology, cytokine- or exosome-based therapy may be considered as a complementary strategy, with careful monitoring and recognition that efficacy and causality cannot be established from a single case.

CONCLUSION

A 16-year-old girl presented with school avoidance and reduced social motivation. Comprehensive clinical evaluation, including MRI, EEG, and neurological examination, suggested ADHD-like symptoms associated with paroxysmal EEG activity, vascular abnormalities interpreted as spike thrombosis involving the middle cerebral arteries, and MRI findings suggestive of traumatic brain injury. The patient received cytokine-based treatment selected to address paroxysmal EEG activity, ADHD-like symptoms, and suspected traumatic brain injury. During follow-up, school avoidance improved, social motivation increased, and the patient resumed regular school attendance. To our knowledge, this is the first case report describing improvement in school avoidance following cytokine-based regenerative treatment in association with improvement in EEG abnormalities and MRA findings. These findings may offer a hypothesis-generating therapeutic perspective for school avoidance, which is conventionally managed primarily with cognitive behavioral therapy; however, they should be interpreted cautiously as a temporal association rather than evidence of causality, and further studies are required to evaluate safety, efficacy, and generalizability.

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Ethical Approval of Studies and Informed Consent

Written informed consent was obtained from the patient.

Conflict of Interest

The authors have no conflicts of interest.

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