Journal of Neuroimmunology 406 (2025) 578654

Contents lists available at ScienceDirect

Journal of Neuroimmunology

journal homepage: www.elsevier.com/locate/jneuroim

Short Communication

Association of ADHD symptoms, pain, and tics with anti-thalamus antibodies in cerebrospinal fluid

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ARTICLE INFO

Keywords:
Autoimmune
Inflammation
Brain
Tourette
Immunotherapy

ABSTRACT

Introduction: Complex mixed presentations of severe mental disorders (SMD) with treatment resistance pose major challenges in clinical practice. The role of novel neuronal antibodies in cerebrospinal fluid (CSF) is largely unexamined in this context.

Methods: A well-studied paradigmatic case of a 36-year-old female patient is reported.

Results: She presented with attention-deficit/hyperactivity disorder-like symptoms (including frequent sensory overload), severe pain (pre-diagnosed as fibromyalgia and somatoform pain disorder), and motor tics. In addition, she developed secondary depressive symptoms. Various psychopharmacological treatment attempts were unsuccessful or not tolerated. The diagnostic routine work-up with a wide range of blood tests, electroencephalography (EEG), routine magnetic resonance imaging (MRI), cerebrospinal fluid (CSF) analyses, and [^{18}F]fluorodeoxyglucose positron emission tomography revealed no clear pathological findings. Tissue-based assays using CSF material found strong immunoglobulin G antibody staining specifically directed against a cell population in the thalamus. Neurotransmitter measurements detected low GABA, glutamate and serotonin concentrations as well as high dopamine levels in the CSF. Different MRI-based analyses indicated no neurostructural alterations in the thalamus; however, left mesiotemporal volume loss was identified. The independent component analysis of the EEG showed left temporal theta waves, partly resembling spike-wave-complexes. Immunotherapy using high-dose steroids resulted in a partial improvement with subjectively reduced stimulus overload, intermediate disappearance of pain, and fewer tics. The improvement could not be objectified psychometrically/neuropsychologically. The mesiotemporal volume loss was no longer present. There were no relevant changes in further research MRI measurements of the thalamus including arterial spin labeling, diffusion tensor imaging, and diffusion microstructure imaging from pre to post-immunotherapy.

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https://doi.org/10.1016/j.jneuroim.2025.578654
Received 29 December 2024; Received in revised form 28 February 2025; Accepted 3 June 2025
Available online 4 June 2025

Discussion: Novel antibodies against strategic brain structures, such as the thalamus, might be associated with some complex SMD. Further immunopsychiatric research in this direction holds promise for a better understanding of similar patients.

1. Introduction

Complex mixed presentations of severe mental disorders (SMD) with treatment resistance remain major challenges in everyday clinical practice. A diagnostic work-up to exclude secondary causes could be of particular importance in these cases (Pollak et al., 2020; Howes et al., 2022). Antibody-mediated autoimmune encephalitis or autoimmune SMD could play a relevant causal role, as the clinical manifestations are defined by antibody effects against specific antigens (e.g., NMDA-R or LGI1) (Graus et al., 2016; Dalmau and Graus, 2018; Prüss, 2021; Endres et al., 2022a). The role of novel neuronal antibodies (Endres et al., 2022b) in cerebrospinal fluid (CSF) in complex mixed presentations of SMD is largely unexamined. Therefore, this article describes a paradigmatic patient with novel antibodies against thalamic structures.

2. Methods

A 36-year-old female patient, who gave her written informed consent for this case study, was investigated according to an established diagnostic protocol, including broad laboratory blood analyses, magnetic resonance imaging (MRI) on a 3 Tesla scanner, and electroencephalography (EEG) (Runge et al., 2023). This was supplemented by CSF analysis and [^{18}F]fluorodeoxyglucose positron emission tomography (FDG-PET). The T1-weighted 3D magnetization prepared rapid gradient echo (MPRAGE) MRI images were used for automated morphometry using VeoBrain software (https://www.veobrain.com/?page=veomorph; accessed February 27, 2025). The underlying methodology has been described in previous publications (e.g., Egger et al., 2020). EEGs were also analyzed using independent component analysis (ICA) (Endres et al., 2017). In addition, diffusion tensor imaging (DTI) with diffusion microstructure imaging (DMI) and arterial spin labeling (ASL) for measuring the cerebral blood flow within the thalamus were used as follow-up parameters. Relative ratios (pre/post immunotherapy values) were calculated to identify possible changes. In addition, the DMI data were compared with a healthy control group from the local Department of Neuroradiology (Hosp et al., 2024). The analyses were carried out via the NORA platform (https://www.nora-imaging.com/; accessed February 27, 2025) and have been described in detail previously (Hosp et al., 2024). Well-characterized neuronal antibodies against cell surface, intracellular, and glial antigens were analyzed using established assays (Endres et al., 2022b). Serum and CSF samples were examined for novel neuronal antibodies using tissue-based assays on unfixed murine brain tissue (Kreye et al., 2020) and for neurometabolic changes (especially of neurotransmitter levels) using liquid chromatography and mass spectrometry (Mandal et al., 2012; Endres et al., 2022c; Endres et al., 2024). Finally, a systematic PubMed literature search (on February 27, 2025) on the association between mental disorders and anti-thalamus antibodies (search strategy: "(antibod*) AND (thalamus OR thalamic) AND (ADHD OR pain OR tic OR depression OR mental disorder)") was performed.

3. Results

The patient suffered primarily from attention-deficit/hyperactivity disorder (ADHD)-like symptoms, severe pain, and motor tics. Her ADHD-like symptoms had been worsening for 4 to 5 years and included attention and concentration deficits, impulsivity, mood instability, and states of inner tension. Pain had been present for eleven years and had worsened over the last two years (with recurrent pain in different parts of the body). Fibromyalgia and somatoform pain disorder had previously been diagnosed. The patient also had recurrent headaches and migraine with aura. Motor tics with e.g. head turning had been observed for about a year and worsened under temporary medication with bupropion. Additionally, the patient was easily overstimulated, had trouble sleeping through the night and early awakenings, repeated deja vu experiences, and developed recurrent secondary depressive episodes. Several food intolerances (e.g., lactose) and allergies (e.g., hazelnut with a history of angioedema) had been observed over the past three years, and the patient has had recurrent abdominal pain and diarrhea (gastroscopy and colonoscopy remained currently unremarkable). There were no indications of celiac disease, rheumatoid arthritis, or porphyria. The test for a histamine intolerance (histamine in serum and diaminooxidase) revealed normal findings. Various treatment attempts with psychotherapy and psychopharmacological drugs (escitalopram, venlafaxine, duloxetine, bupropion, tianeptine, methylphenidate, quetiapine, lithium, and lamotrigine) were not convincingly successful earlier. Autonomic dysregulation under low doses of antidepressants (e.g., under 37.5 mg venlafaxine) was observed. Exocrine pancreatic insufficiency was suspected earlier and therefore substituted. The abdominal ultrasound, which was added because of twice minimally elevated lipase levels, did not reveal signs of acute or chronic pancreatitis. The family history revealed that her father had multiple sclerosis. The mother had ADHD symptoms and a non-specified rheumatological disease.

The diagnostic work-up, including different urine/blood tests and routine EEG, MRI, and CSF analyses, revealed no clear pathological findings (Table 1). The MRI showed a previously known pineal cyst (up to 4 mm in diameter) and a low tonsil extending up to 5 mm below the McRae line, with no evidence of CSF outflow obstruction. FDG-PET of the brain identified normal findings; whole body FDG-PET detected no evidence of malignancy. Tissue-based assays using CSF revealed strong bar-shaped IgG antibody staining, specifically against neurons in the thalamus (with the highest intensity). The neurotransmitter profile from the CSF revealed low GABA, glutamate, serotonin, and high dopamine concentrations. Urea was also increased in serum and CSF. Brain morphometry showed no changes in the thalamus. A comparison of the DMI data of the patient with an age- and sex-matched control group (age mean 28.8 years, SD ± 6.9 years, min. 21, max. 47) showed no altered microstructure of the thalamus. However, automated morphometry detected a reduction in left mesiotemporal volume. The ICA of the EEG showed distinct left temporal theta waves, which were partly spike-wave-like and aggravated during hyperventilation in the first EEG. The intermittent rhythmic delta/theta activity already dropped significantly in the second EEG (after discontinuation of methylphenidate and directly before steroids) and remained stably reduced after treatment.

After a multidisciplinary case discussion, the patient was offered a treatment trial with a steroid pulse (500 mg methylprednisolone for five days with oral tapering over approximately one month; parallel psychopharmacotherapy was not prescribed at that time). Long-term immunotherapy was not carried out. Immunotherapy using steroids resulted (approximately one month after starting methylprednisolone) in a partial subjective improvement with reduced sensory overload, temporary disappearance of pain, and fewer tics. As a result of the treatment, the patient was able to attend public events again and managed to move into a new apartment, as she had planned for a long time. However, the improvement could not be objectified (see Fig. 1 and Table 1). Psychometrically, only the Beck Depression Inventory relevantly improved. Neuropsychological testing of attentional performance did not improve convincingly. The mesiotemporal volume loss was no longer observed (the follow-up MRI was performed already

Table 1

All diagnostic findings. Conspicuous findings are marked in bold. *Approximately 1.5 weeks after ending of the steroid pulse therapy. **Follow up after approximately one month. Abbreviations: ADHD, attention-deficit/hyperactivity disorder; ANAs, antinuclear antibodies; ANCAs, anti-neutrophil cytoplasmic antibodies; APAs, antiphospholipid antibodies; AQP4, aquaporin-4; ASL, arterial spin labeling; BDI-II, Beck's Depression Inventory II; CAARS, Conners' Adult ADHD Rating Scales; CBF, cerebral blood flow; CLIA, chemiluminescence immunoassay; CCP, cyclic citrullinated peptide; CMV, cytomegalovirus; CRP, C-reactive protein; CSF, cerebrospinal fluid; CT, computed tomography; DMI, diffusion microstructure imaging; DNaseB, deoxyribonuclease B; DSM-IV, Diagnostic and Statistical Manual of Mental Disorders; DTI, diffusion tensor imaging; EBV, Epstein-Barr virus; ECG, electrocardiography; EEG, electroencephalography; ERG, electroretinography; FDG-PET, [^{18}F]fluorodeoxyglucose-positron emission tomography; GAD65, glutamic acid decarboxylase 65-kilodalton isoform; HAV, hepatitis A virus; HBV, hepatitis B virus; HCV, hepatitis C virus; HIV, human immunodeficiency virus; IgA/G/M, immunoglobulin A/G/M; IRDA, intermittent rhythmic delta activity; MOG, myelin oligodendrocyte glycoprotein; MRI, magnetic resonance imaging; MRZ, antibody indices against measles, rubella, and varicella zoster virus; OCT, optical coherence tomography; PCR, polymerase chain reaction; ref., reference; SD, standard deviation; TB, tuberculosis; TG, thyroglobulin; TPO, thyroid peroxidase; TSH, thyroid-stimulating hormone; V, volume; VZV, varicella zoster virus; WBC, white blood cell.

Serum antibodies, immunological markers and serologies	
Anti-thyroid antibodies (against TPO, TG and TSH-receptor)	Negative
ANAs (*on HEp-2 cells*), ANCAs (*on EthOH- /formalin-fixed neutrophils*), APAs	Negative
Rheumatoid factor/anti-CCP	Normal
Anti-gliadin IgG/IgA and transglutaminase IgA antibodies	Negative
Complement factors (C3, C4)	Normal
IgG, IgM, and IgA levels	Normal
IgE	***164 IU/ml*** (ref.: 0-100 IU/ml)
CRP	<3 mg/l (ref.: <5 mg/l)
Anti-streptolysin-O	231 IU/ml (ref.: <300 IU/ml)
Anti-DNaseB	***322U/ml*** (ref.: <200 U/ml)
Serology for Lyme disease or lues	Negative
Serologies for CMV, EBV, HAV, HBV, HCV, HIV and tuberculosis	Negative
Paraneoplastic IgG antibodies against intracellular antigens (Immunoblot)	Negative
GAD65 antibodies using CLIA	Normal
Well-characterized neuronal IgG cell surface antibodies	Negative
Anti-MOG/AQP4-IgG antibodies	Negative
Tissue based assay on unfixed murine brain tissue (Prof. Prüss; Charité Berlin, Germany)	***Non-specific ANA pattern***
Cerebrospinal fluid	
White blood cell count	4/µL (ref.: <5/µL)
Protein concentration	300 mg/L (ref.: <450 mg/L)
Albumin quotient	4.5 (ref.: <7.7)
IgG-index	0.58 (ref.: <0.7)
Oligoclonal bands in serum/CSF	Negative/negative
Local IgG/IgA/IgM synthesis	None (ref.: < 10%)
Well-characterized neuronal IgG cell surface antibodies	Negative
Tissue based assay on unfixed murine brain tissue (Prof. Prüss; Charité Berlin, Germany)	***Strong, bar-shaped IgG antibody staining, specifically against neurons in the thalamus***
Neurometabolic measurements from serum and cerebrospinal fluid	
Neurometabolic measurements (*under medication with methylphenidate 10 mg and promethazine 20 mg)*	***Reduced glutamate*** (1.8±0.2 µM; ref. range: 26-40 µM), ***low serotonin*** (0.11±0.06 µM; ref. range: 0.82±0.48 µM), ***low GABA*** (0.079±0.013 µM; ref. range: 0.1270 ± 0.0052 µM), and ***elevated dopamine*** (13.6±1.1 nM; ref. range: 0.04–4.5 nM) in CSF; ***elevated urea*** in plasma (16 mM; ref. range: 1.8-7.1 mM) and in CSF (12.5 mM; ref. range: 3.0-6.5 mM)
Urine	
Porphyria diagnostics	Unremarkable

MRI of the brain	
Visual inspection	***Previously known pineal cyst (up to 4 mm in diameter) and a low tonsil up to 5 mm below the McRae line***, with no evidence of CSF outflow obstruction
Automated morphometry	***Left mesiotemporal volume loss before immunotherapy; no longer detectable in the follow-up MRI (approx. 1.5 weeks after discontinuation the steroid pulse and during oral tapering)***
Research MRI approaches pre- and post-immunotherapy*	
Cerebral blood flow (CBF) using arterial spin labeling (ASL)	CBF thalamus mean - relative ratio t1/t2: 0.966745607
Diffusion tensor imaging (DTI)	Fractional anisotropy thalamus mean - relative ratio t1/t2: 1.008616693 Axial diffusivity thalamus mean - relative ratio t1/t2: 1.004637787 Mean diffusivity thalamus mean - relative ratio t1/t2: 1.00385705
Diffusion microstructure imaging (DMI)	Free water/CSF (V-CCF) fraction thalamus mean - relative ratio t1/t2: 1.000890372 Volume fraction outside of axons or dendrites (V-extra) thalamus mean - relative ratio t1/t2: 1.024537832 Volume fraction within neuronal processes (i.e., axons and dendrites; V-intra) thalamus mean - relative ratio t1/t2: 0.96690012
Diffusion microstructure imaging (compared with controls)	
Diffusion microstructure imaging (presented are the z-scores compared with healthy female controls (age mean 28.8 years, SD 6.8, min. 21, max. 47)	V-CSF / V-extra / V-intra t1: -1.73 / 1.18 / -0.49 t2: -1.74 / 1.06 / -0.32
EEG	
Visual analyses	No intermittent/generalized slowing, no epileptic activity
Independent component analysis	Distinct ***left temporal theta waves*** that were partly spike wave-like and aggravated during hyperventilation in the first EEG; the intermittent rhythmic delta/theta activity already dropped significantly in the second EEG (after discontinuation of methylphenidate and directly before steroid treatment) and remained stable reduced after steroid treatment
FDG-PET	
Brain	Normal cerebral glucose consumption
Whole body	No evidence of malignancy
Ophthalmological tests	
Optical coherence tomography (OCT)	***Micro-papilla right and left***
Electroretinography (ERG)	Normal
Psychometric testing (initial before steroids/follow-up after steroids**)**	
BDI-II (for depressive symptoms)	40/27
ADHD-Checklist (for ADHD symptoms)	52/56
Bell-Score (for chronic fatigue symptoms)	30/50
Premonitory Urges for Tics Scale-Revised (for tic symptoms)	17/18
Gilles de la Tourette Syndrome - Quality of Life Questionnaire (for quality of live in patients with tics)	55%/40%
CAARS t-values (for ADHD symptoms):	
A. Inattention/memory problems	78/87
B. Hyperactivity/restlessness	57/59
C. Impulsivity/emotional instability	75/81
D. Problems with self-concept	60/76
E. DSM-IV Inattention Symptoms	89/87
F. DSM-IV Hyperactivity-Impulsivity Symptoms	64/64
G. DSM-IV ADHD Symptoms Total	81/79
H. ADHD Index	75/81
Internal medicine examinations	
Gastroscopy and colonoscopy	No gastritis, inconspicuous ileocolonoscopy
Sonography of the abdomen	No evidence of liver parenchymal damage, ***very slightly inhomogeneous pancreatic parenchyma***
Liver and pancreas values	***Lipase slightly elevated in 2 of 14 examinations with a maximum value of 69 U/l*** (ref.: 13-60 U/l); ***liver values (GOT and GPT) slightly elevated under steroid treatment*** (with normalization in the course)
IgG4 levels	Normal (0.229 g/l; ref.: 0.030-2.0 g/l)
Elastase in faeces	Normal (under substitution; > 500 µg/g; ref.: >200 µg/g)
Calprotectin in faeces	Normal (38 mg/kg; ref.: <50 mg/kg)

approximately 1.5 weeks after ending of the steroid pulse therapy). There were no relevant changes in research MRI measurements including ASL, DTI, and DMI of the thalamus from pre- to post-treatment with relative ratios ranging from a minimum of 0.97 to a maximum of 1.02. DMI showed no relevant changes compared to the age- and sex-matched control group also after treatment. Approximately three months after starting methylprednisolone, the patient reported (as sustained positive effects) hardly any tics, reduced sensory overload, and

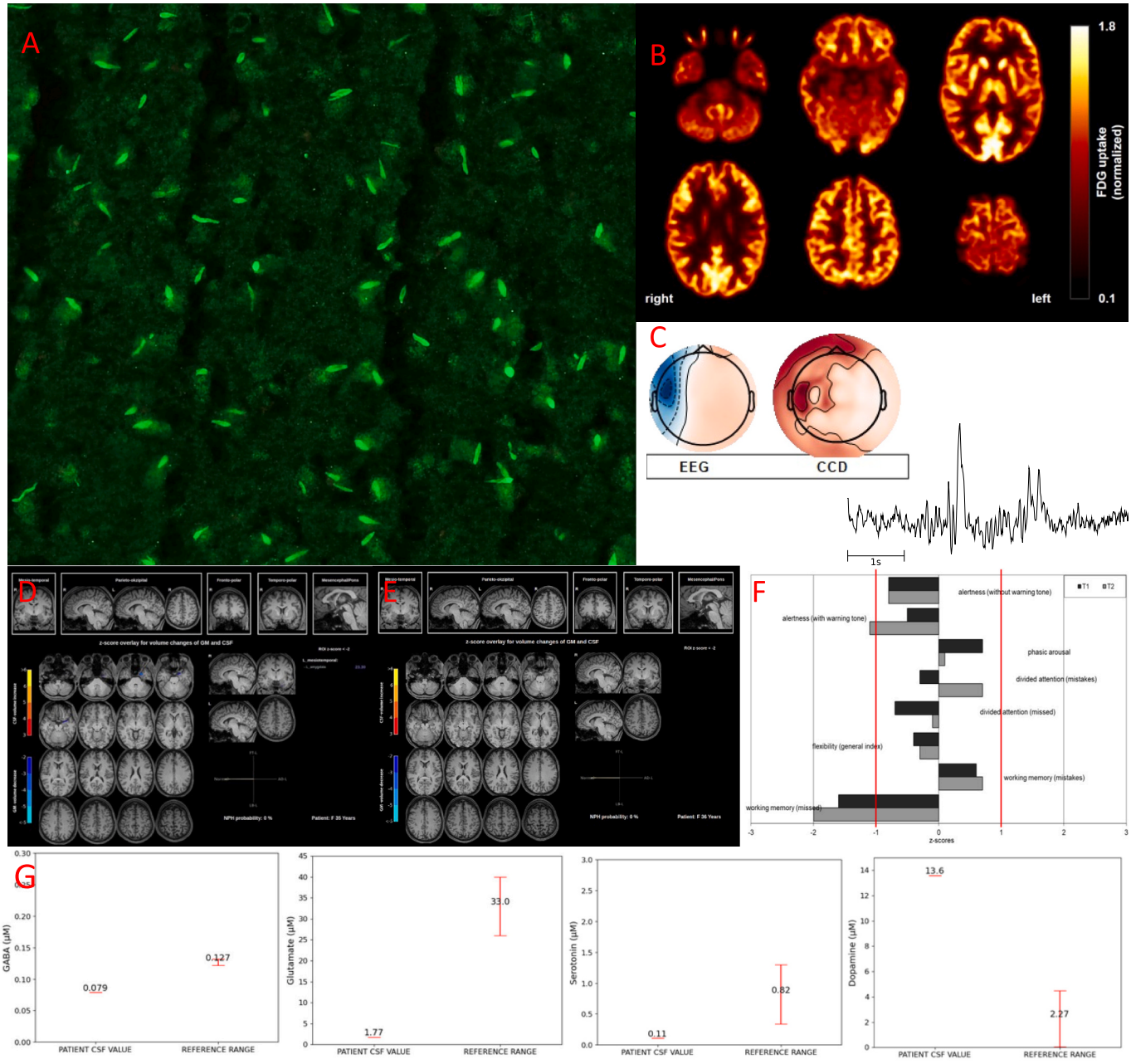

Fig. 1. Diagnostic findings in the presented patient.

A) Tissue-based assays on unfixed murine brain tissue using cerebrospinal fluid showed a strong bar-shaped immunoglobulin G antibody staining against a cell population in the thalamus.

B) [^{18}F]fluorodeoxyglucose positron emission tomography (FDG-PET) revealed normal cerebral glucose consumption. The panel depicts transaxial slices of FDG-PET with normalization of the uptake to the whole-brain parenchyma.

C) The independent component analysis of the electroencephalography (EEG) showed distinct left temporal theta waves, partly spike-wave-like, aggravated during hyperventilation (HV) (EEG: EEG topography; CCD: Cortical current density in a sphere model; beside: characteristic time series of this component after HV).

D) In the research magnetic resonance imaging (MRI) analyses with automated morphometry, a left mesiotemporal volume loss was identified (https://www.veobrain.com/?page=veomorph; accessed February 27, 2025).

E) The mesiotemporal volume loss was no longer detectable in the follow-up MRI (approx. 1.5 weeks after discontinuation of the steroid pulse and during oral tapering).

F) Testing for attentional performance pre- (t1) and post (t2) immunotherapy. Follow-up testing takes place approximately one month after steroid pulse treatment and after complete discontinuation of oral steroids. The presented z-scores (reference: −1 to +1) identified no convincing improvement.

G) The neurometabolic testing (presented are only the neurotransmitter results) from CSF detected low GABA, low glutamate, low serotonin and high dopamine levels within the cerebrospinal fluid (under medication with methylphenidate 10 mg and promethazine 20 mg).

affective stabilization.

The systematic literature search for related publications revealed 196 results. However, no comparable cases with ADHD, pain, or tics, and anti-thalamus antibodies could be identified.

4. Discussion

The case of a treatment-resistant patient with ADHD-like symptoms, pain, motor tics, and secondary depression with anti-thalamus IgG antibodies in the CSF, and left temporal MRI/EEG pathologies was presented.

A systematic literature search identified no similar cases with anti-thalamus antibodies.

From a clinical perspective, the complex neuropsychiatric mixed symptoms with ADHD-like symptoms, pain, tics, as well as treatment resistance and psychotropic drug intolerance with autonomic dysregulation, as well as the positive family history for autoimmune diseases (in both parents), could be interpreted as "red flag" symptoms for an autoimmune cause (Herken and Prüss, 2017; Endres et al., 2020; Pollak et al., 2020). In particular, complex SMDs that deviate from standard diagnostic criteria (i.e., as found in the International Classification of Diseases in the eleventh revision or the Diagnostic and Statistical Manual of Mental Disorders in the fifth edition) or that are diagnosed with multiple comorbid disorders, as observed in this patient, might suggest a secondary cause. Such patients often do not respond sufficiently to conventional treatment approaches, potentially increasing the risk of treatment resistance (Howes et al., 2022). Anti-thalamic antibodies have not been detected in any other patient groups studied with the same method, including schizophreniform, affective, and obsessive-compulsive disorders (Endres et al., 2022b; Pankratz et al., 2023), and may be associated with the reported symptoms. The abnormalities in GABA, glutamate, serotonin, and dopamine levels in the CSF might be neurometabolic consequences of the antibodies. However, some changes (especially the high dopamine levels) could also be associated with the medication taken during the time of lumbar puncture (at the time the patient was still taking methylphenidate, which was later stopped, so that the immunotherapy was carried out without concomitant medication) or by other influencing factors. The high urea levels (with normal renal markers such as creatinine) could be a consequence of the catabolic metabolic situation with starvation and dehydration.

From a pathophysiological perspective, the specific target antigen and the functional implications of the antibodies detected against the thalamus remain unclear. As the thalamus is "strategically" located in the diencephalon and integrates signals from many areas of the central nervous system, dysfunction of the thalamus caused by antibodies could influence brain activity on a "global" scale (Shine et al., 2023). The thalamus 1) generates waking-state EEG rhythms (Feige et al., 2005; Bailey and Joyce, 2015), 2) connects thalamic neural circuits to both cortical and subcortical areas (Bailey and Joyce, 2015), which show significant differences in ADHD and correlations with ADHD symptom severity, particularly in the thalamocortical dorsal attention, somatomotor, and default mode networks (Hong, 2023), 3) serves as a relay for the transmission of nociceptive signals to the cerebral cortex, thereby playing a key role in pain perception (Yen and Lu, 2013), and 4) consistently exhibiting volumetric alterations in patients with primary tic disorder (Wan et al., 2021). In cases of secondary tic disorders resulting from lesions, there is converging functional connectivity to a cortical-subcortical network that includes the thalamus (Ganos et al., 2022). Correspondingly, the thalamus is a frequent target for deep brain stimulation in managing tic disorders (Baldermann et al., 2016). Hence, the involvement of the thalamus is evident across all clinical symptoms observed in the presented patient, including ADHD-like symptoms, pain, and tics. More broadly, the thalamus acts as a crucial hub within the cingulo-opercular network (Dosenbach et al., 2008), also known as the action-mode network (https://osf.io/preprints/psyarxiv/2vt79), which is implicated in mental or cognitive states requiring action. Although speculative at this point, this functional attribution potentially encapsulates the multifold symptomatology of this case. Left mesiotemporal changes observed in research MRI using automated morphometry – which were no longer detectable after immunotherapy – might have developed secondarily due to disturbed cortico-limbo-thalamo-cortical circuits (Kamali et al., 2023) or weaker antibody effects against the temporal brain.

A major limitation is that neurostructural explanatory models are still very speculative. The symptoms could have developed completely independently of the antibodies. The diagnostic findings required for autoimmune encephalitis or autoimmune psychosis, which indicate clear neuroinflammation (e.g., pleocytosis in CSF), were not fulfilled (Graus et al., 2016; Pollak et al., 2020). While subjective clinical improvement was observed following immunotherapy, this improvement could not be confirmed in psychometric and neuropsychological tests. Therefore, the therapeutic effects may also be attributed to placebo effects. The case illustrates that subjective improvements following immunotherapy may not be objectively measurable with standard questionnaires or neuropsychological tests, underscoring the need of having corresponding objective follow-up parameters in similar cases. In general, effects of immunotherapies may be difficult to detect on the individual patient level and this should therefore foster the initiation of clinical trials. Neurotransmitter levels should not be overinterpreted in the context of the complex neurochemical system with potential different external influences. Further research efforts should aim to identify the exact target antigens (e.g., using immunoprecipitation with mass spectrometry approaches) and the functionality (e.g., by injecting the purified antibodies into mouse brains with subsequent behavioral experiments) of such anti-thalamic antibodies in similar constellations (Prüss, 2021).

In summary, novel antibodies directed against strategic brain structures, such as the thalamus, might be associated with some complex SMD. Further immunopsychiatric research in this direction is necessary and promising.

Ethics/consent for publication

The patient gave her signed written informed consent for this case report to be published.

CRediT authorship contribution statement

Dominique Endres: Writing – original draft, Visualization, Supervision, Methodology, Funding acquisition, Data curation. **Katharina von Zedtwitz:** Writing – original draft, Methodology, Investigation, Formal analysis, Data curation. **Alexander Rau:** Writing – review & editing, Methodology, Formal analysis. **Bernd Feige:** Writing – review & editing, Software, Methodology, Formal analysis. **Hansjörg Mast:** Writing – review & editing, Investigation. **Alexander Maier:** Writing – review & editing, Investigation, Data curation. **Marco Reisert:** Writing – review & editing, Software, Methodology. **Kathrin Nickel:** Writing – review & editing, Methodology, Investigation, Data curation. **Joachim Brumberg:** Writing – review & editing, Visualization, Methodology, Investigation. **Tobias Boettler:** Writing – review & editing, Formal analysis. **Cornelia Glaser:** Writing – review & editing, Investigation, Data curation. **Nils Venhoff:** Writing – review & editing, Supervision, Investigation. **Horst Urbach:** Writing – review & editing, Supervision, Formal analysis. **Juan C. Baldermann:** Writing – review & editing, Validation. **Katharina Domschke:** Writing – review & editing, Supervision. **Ludger Tebartz van Elst:** Writing – review & editing, Supervision. **Luciana Hannibal:** Writing – review & editing, Software, Methodology, Formal analysis. **Harald Prüss:** Writing – review & editing, Visualization, Supervision, Methodology, Investigation, Formal analysis. **Simon J. Maier:** Writing – original draft, Supervision, Methodology.

Funding

The article processing charge was funded by the Baden-Wuerttemberg Ministry of Science, Research and Art and the University of Freiburg in the funding programme Open Access Publishing. DE is funded by the German Research Foundation (419859038) and the German Ministry of Education and Research (01GM2208, CONNECT-GENERATE). BF and LTvE were funded by the German Research Foundation (419859038).

Declaration of competing interest

KD: Member of the Neurotorium Editorial Board, The Lundbeck Foundation. LTvE: Advisory boards, lectures, or travel grants within the last three years: Roche, Eli Lilly, Janssen-Cilag, Novartis, Shire, UCB, GSK, Servier, Janssen, and Cyberonics. All other authors declare no potential conflicts of interest.

Acknowledgements

We would like to thank the patient for the opportunity to publish this case study. DE and LTvE are members of the Immuno-NeuroPsychiatry Network of the ECNP. KN and JB were funded by the Berta-Ottenstein-Programme for Advanced Clinician Scientists, Faculty of Medicine, University of Freiburg. AR was supported by Berta-Ottenstein-Programme for Clinician Scientists, Faculty of Medicine, University of Freiburg.

Data availability

This is a case report. All necessary data can be found in the paper.

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