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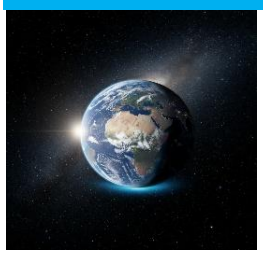

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Autoimmune Encephalitis Associated with Endocrine Dysfunction: A Case Report

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Abstract
Autoimmune encephalitis is a rare immune-mediated neurological disorder characterized by brain inflammation associated with autoantibodies targeting neuronal surface or synaptic antigens. It frequently presents with rapidly progressive psychiatric symptoms, cognitive impairment, seizures, and autonomic instability, often mimicking primary psychiatric or infectious conditions. The disease course may be further complicated by concomitant endocrine dysfunction, requiring interdisciplinary management.
We report the case of an 18-year-old woman with a history of autoimmune encephalitis and secondary hormonal abnormalities who developed recurrent psychotic decompensation. At the age of 15, she was hospitalized due to acute behavioral deterioration, hallucinations, confusion, and autonomic symptoms. Diagnostic evaluation revealed influenza A infection and inflammatory-demyelinating lesions in the corpus callosum and left cerebral hemisphere on MRI. Laboratory findings showed elevated antithyroid antibodies and hypothalamic amenorrhea, confirming endocrine involvement. Combined antiviral, immunomodulatory, and corticosteroid therapy resulted in significant clinical and radiological improvement.

After two years of remission, the patient was readmitted with severe psychotic symptoms, including agitation, delusions, and behavioral disorganization. Neuroimaging demonstrated a stable post-inflammatory lesion without signs of active inflammation. Treatment required individualized polypharmacotherapy, with careful monitoring of extrapyramidal and endocrine adverse effects.
This case highlights the complex interaction between autoimmune neuroinflammation, hormonal dysregulation, and psychiatric manifestations. Early recognition, comprehensive immunological and endocrine evaluation, and long-term multidisciplinary follow-up are essential to optimize outcomes and reduce relapse risk in autoimmune neuropsychiatric syndromes.

Keywords:

autoimmune encephalitis, Endocrine disruption, autoimmune disease, Psychotic symptoms, neurology

Introduction

Autoimmune encephalitis (AE) represents a group of immune-mediated inflammatory disorders of the central nervous system, characterized by diverse mechanisms and clinical presentations. Understanding these mechanisms is crucial for accurate diagnosis and selecting the appropriate

treatment. Broadly, AE can be divided into two major subtypes based on the antibody targets. The first group involves paraneoplastic disorders with antibodies directed against intracellular antigens, such as anti-Hu, anti-Ma/Ta, anti-CV2, and anti-GAD. These are typically associated with underlying malignancies, have a poor prognosis, and are driven by T-cell–mediated neuronal injury, which often results in limited therapeutic response. The second group includes antibodies targeting neuronal cell-surface antigens such as anti-NMDA, anti-VGKC, anti-GABA, and anti-AMPA, which are usually non-paraneoplastic, show better outcomes, and frequently manifest with limbic or psychiatric symptoms [1, 2].

The estimated annual incidence of encephalitis of any cause is approximately 2–3 per 100,000 in Northern Europe, which is about half that of newly diagnosed multiple sclerosis (4–8 per 100,000 per year). While 40% of encephalitis cases are infectious and another 40% remain idiopathic, up to 20% are immune-mediated. Among these, anti-NMDA receptor encephalitis accounts for around 4% and VGKC-complex antibody–positive cases for 3% [3].

Autoimmune mechanisms frequently extend beyond the nervous system, involving systemic and endocrine dysfunction. Systemic autoimmune diseases, such as rheumatoid arthritis, systemic lupus erythematosus, and systemic sclerosis, share standard pathogenic mechanisms of immune dysregulation, including loss of tolerance to self-antigens, genetic predisposition, hormonal influence, and environmental triggers [4]. Autoimmune thyroid diseases (AITD), including Graves' disease and Hashimoto's thyroiditis, are the most prevalent organ-specific autoimmune disorders, affecting 2–4% of women and up to 1% of men worldwide [5, 6]. Involvement of the hypothalamic–pituitary axis may also occur, as in autoimmune hypophysitis, which can be primary or secondary to systemic autoimmunity. Lymphocytic hypophysitis (LYH) is the most frequent subtype, although mixed histopathological forms are common [7].

Case Presentation

Child and Adolescent Psychiatry Admission

The patient, a 15-year-old girl, was admitted to the Child and Adolescent Psychiatry Department due to a rapid and severe deterioration in mental status. According to the patient's mother, symptoms had begun approximately one week earlier and included increasing weakness, loss of appetite, and upper respiratory tract infection symptoms. Over subsequent

days, the patient developed behavioral disturbances, anxiety, withdrawal, and perceptual distortions. She reported unpleasant odors, spoke incoherently, refused to eat or drink, and displayed paranoid behavior, warning her mother not to consume a beverage she believed to be poisoned.

During hospitalization in the psychiatric unit, there was further deterioration in both mental and somatic condition. The patient developed fever, psychomotor agitation, confusion, and disturbances in orientation to time, place, and self. Due to progressive deterioration in consciousness and suspected organic etiology, she was transferred to the Neurology and Pediatrics Department for further evaluation and management of suspected encephalitis.

Neurology Department Admission

Upon admission to the Neurology Department, the patient was in serious general condition, minimally responsive, and required intermittent physical restraint and passive oxygen therapy. Neurological examination revealed neck stiffness, increased muscle tone, preserved tendon reflexes, and no focal neurological deficits. Physical examination showed pharyngeal hyperemia, a coated tongue, and diminished vesicular breath sounds with rales over both lungs. The liver and spleen were not enlarged.

During the early hospitalization period, multiple venous blood gas analyses were performed, revealing variable parameter values (Table 1).

pH	**pCO2 [mmHg]**	**pO2 [mmHg]**	**HCO3- [mmol/L]**	**BE [mmol/L]**	**tCO2 [mmol/L]**	**SatO2[%]**	**Lactate[mmol/L]**
7.3	40.3	67.6	19.4	6	18	91.1	1.1
7.39	38.8	52.8	22.8	-1.4	21	86.5	1.1
7.45	43.7	53.5	29.9	5.5	24.8	85.4	1.4
7.42	50.6	36.4	32	7.4	32	62.2	was not performed

Table 1. Venous blood gas
Note: pH - blood acidity, pCO_2 - partial pressure of carbon dioxide (mmHg), pO_2 - partial pressure of oxygen (mmHg), HCO_3^- - bicarbonate concentration (mmol/L), BE - base excess (mmol/L), tCO_2 - total carbon dioxide (mmol/L), $SatO_2$ - oxygen saturation (%), Lactate - blood lactate level (mmol/L)

During hospitalization, numerous immunological and serological tests were performed, including: ANA and ANCA tests (both screening and profile), IgG index determination (IgG

and albumin in CSF and serum), analysis of oligoclonal bands in PMR and serum, assessment of onconeuronal and antineural antibodies, as well as tests confirming the presence of antibodies against Borrelia burgdorferi (IgG and IgM in CSF) and testing for lupus anticoagulant. All results have been documented as electronic medical documentation (EDM) scans. Additionally, screening for HIV-1 and HIV-2 was performed during hospitalization, with negative results (Table 2).

Test	**Specimen**	**Result**	**Reference Range**
HIV 1/2 antibodies	Serum	Non-reactive	Non-reactive
Anti-thyroidantibodies	Serum	Elevated	<60 IU/mL
Antinuclearantibody (ANA) screening test	Serum	Positive	Negative (≤1:40 or ≤1.0 U)
Antinuclearantibody (ANA) profile	Serum	Ro-52 and AMA-M2 antibodiesd etected	Ro-52 and AMA-M2: normallyabsent
Antineutrophilcytoplasmicantibody (ANCA) screening test	Serum	Positive	Negative (< 20 U/mL)
Antineutrophilcytoplasmicantibody(ANCA) profile	Serum	Positive	Negative
Immunoglobulin G (IgG) index	Serum + cerebrospinalfluid (CSF)	Positive	0.00 - 0.70 (normal; ≥ 0.70 indicatesintrathecal IgG synthesis)

Table 2. Note: Reference ranges correspond to commonly accepted clinical laboratory standards.

Abbreviations: ANA – antinuclear antibodies; ANCA – antineutrophil cytoplasmic antibodies; AMA-M2 – antimitochondrial antibodies type M2; CSF – cerebrospinal fluid; IgG – immunoglobulin G; IgM – immunoglobulin M.

Microbiological and virological examinations performed during hospitalization revealed the findings summarized in Table 3. Cerebrospinal fluid collected by lumbar puncture demonstrated growth of Staphylococcus species (coagulase-negative, methicillin-sensitive strain) and a single Gram-negative bacillus of undetermined species. Gram-stained preparations showed both Gram-positive cocci and Gram-negative bacilli. The result was interpreted as a probable contamination of the specimen during collection, as growth was detected after approximately one day of incubation. A nasopharyngeal swab obtained during the early hospitalization period confirmed the presence of influenza A virus, while tests for influenza B

and respiratory syncytial virus (RSV) were negative. Blood cultures revealed no bacterial or fungal growth, effectively excluding bacteremia. A urine culture collected later during hospitalization demonstrated single colonies of Candida non-albicans yeasts, considered to represent colonization rather than infection, as no clinical symptoms of urinary tract infection were observed.

CSF - cerebrospinal fluid; RSV - respiratory syncytial virus. All cultures were performed under standard aerobic conditions. Results were interpreted in the clinical context and according to established microbiological criteria. The findings indicate no evidence of bacteremia or clinically significant infection. The detection of Staphylococcus species (coagulase-negative) and a single Gram-negative bacillus in CSF was considered most likely due to specimen contamination during collection.

Magnetic resonance imaging studies of the central nervous system showed CLOCCs (cytotoxic changes of the corpus callosum) and hyperintense foci in the periventricular white matter of the left hemisphere of the brain (Figure 1). A follow-up study showed regression of corpus callosum changes and a stable periventricular focus. An MRI performed revealed a 12 × 5 × 10 mm hyperintense area at the level of the left lateral ventricle, with no diffusion restriction or contrast enhancement. Brain structures appeared normal, with symmetrical ventricles and normal subarachnoid spaces. A 5 mm pineal cyst and a small polyp in the left maxillary sinus were also noted. The findings suggest a stable, likely post-inflammatory, demyelinating, or vasogenic lesion in the left hemisphere of the brain. A chest CT showed frosted glass parenchymal thickenings and small subpleural nodules, consistent with viral-related inflammatory changes.

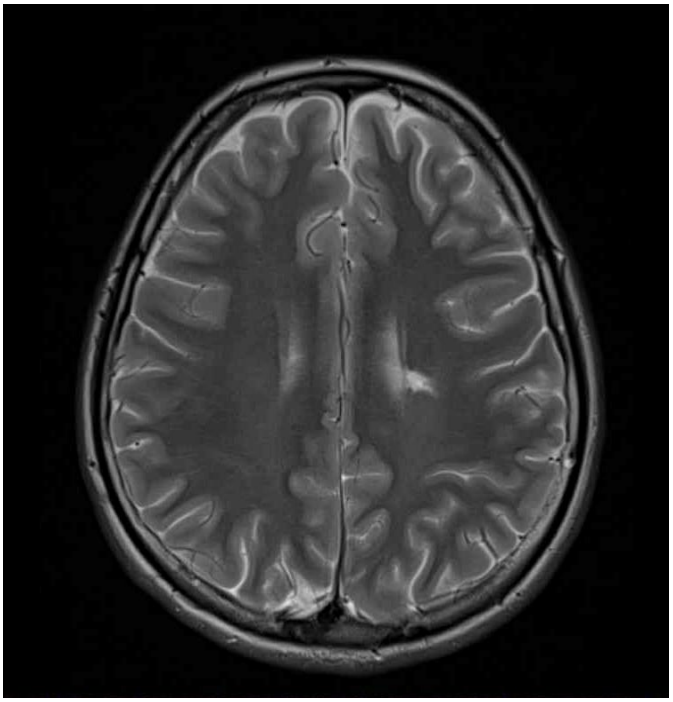

Figure 1. CT scan of the brain

An abdominal ultrasound examination performed demonstrated a liver of normal size and homogeneous parenchymal echogenicity, with a focal area of increased echogenicity in segment IV, consistent with possible focal fatty infiltration. No other focal hepatic lesions were observed. The bile ducts and gallbladder appeared normal, without dilation or wall thickening. A small amount of free fluid (up to 13 mm) was visualized in the right iliac fossa, most likely representing reactive changes secondary to inflammation or infection. Both kidneys were mildly enlarged (approximately 13 cm) with preserved corticomedullary differentiation and slightly increased cortical echogenicity, findings suggestive of transient functional or mild inflammatory/metabolic alterations. No evidence of pelvicalyceal stasis or calculi was noted. The urinary bladder was smooth-walled and adequately filled, and the ureters were not dilated.

Echocardiographic evaluation demonstrated normal cardiac anatomy and preserved left ventricular systolic function, with an estimated ejection fraction of approximately 70%. Chamber sizes and wall thickness were within normal limits, with no evidence of ventricular hypertrophy or dilation. Valve morphology and flow velocities were normal, showing no signs of stenosis or regurgitation. The continuity of the interventricular septum was maintained, and there were no findings suggestive of intracardiac shunts or hemodynamically significant anomalies. A thin echo signal was observed at the site of the foramen ovale, consistent with a

small (approximately 3 mm) patent foramen ovale, without evidence of significant left-to-right shunt. The right ventricular outflow tract and pulmonary artery demonstrated normal flow patterns and velocities, excluding pulmonary hypertension. No pericardial effusion was detected. Overall, the echocardiographic findings indicated normal cardiac structure and function, with no abnormalities requiring further cardiological intervention.

Endocrine consultation

During an endocrinology consultation conducted while the patient was still hospitalized in the Neurology Department, a 15-year-old girl diagnosed with suspected encephalitis was evaluated. Laboratory testing revealed elevated concentrations of antithyroid antibodies, despite a negative family history for autoimmune or thyroid diseases. On physical examination, the thyroid gland was non-palpable and not enlarged. Indicative ultrasound evaluation showed a thyroid gland of normal size, with homogeneous echotexture and normal echogenicity, without sonographic features suggestive of autoimmune thyroiditis. Secondary amenorrhea was noted, and the hormonal profile indicated hypothalamic etiology. Further evaluation with magnetic resonance imaging of the pituitary gland was recommended to exclude possible hypophysitis.

In-Hospital Management

The patient, hospitalized at the Department of Neurology and Pediatrics of Infectious Diseases and Pediatric Neurology, received multiple medications due to a diagnosis of encephalitis.
Antiviral treatment included acyclovir and oseltamivir (Ebilfumin), while cefotaxime (Biotaxime) and vancomycin were administered as antibiotics following confirmation of influenza A virus infection. To modulate the immune response, human immunoglobulins (Ig Vena, 2 g/kg body weight) were given, along with pulsotherapy using methylprednisolone (Solu-Medrol). Psychiatric management involved aripiprazole (Arpixor). Supportive care encompassed electrolyte supplementation (Kalium, Natrium chloratum, Optilyte), parenteral nutrition (Kabiven, Benelyte), oxygen therapy, anti-swelling agents (Corhydron, Furosemide Kabi), anti-inflammatory and antipyretic medications (Dexaven, Paracetamol B. Braun), anticoagulant prophylaxis (Clexane), and inhaled treatments (Ventolin, Budixon Neb). Throughout hospitalization, the patient's condition steadily improved, neurological symptoms diminished, and brain MRI findings regressed. After completing intensive treatment, further pediatric neurological care and outpatient follow-up were recommended.

The patient was discharged home in stable general condition, with further diagnostic and therapeutic recommendations and instructions for continued outpatient follow-up.

Psychiatric Department Admission

The patient, an 18-year-old woman, was admitted to the Psychiatric Department on an emergency basis, without a medical referral, in accordance with Article 22, paragraph 1a of the Mental Health Protection Act, due to a sudden deterioration in mental state. The episode was characterized by psychomotor agitation, behavioral disorganization, distractibility, and the occurrence of self-referential, persecutory, and religious delusions, accompanied by tension and brief episodes of verbal aggression towards her mother. The onset of symptoms was acute and occurred after approximately two years of relative emotional and cognitive stability following a systemic, likely post-viral encephalomyelitis with renal failure, which had been treated times before.

During the patient's psychiatric hospitalization, a comprehensive diagnostic evaluation was performed, including a contrast-enhanced brain MRI. The imaging revealed a persistent lesion in the periventricular white matter of the left cerebral hemisphere, measuring approximately 12 × 5 × 10 mm. The lesion appeared hyperintense on T2- and TIRM-weighted sequences, without contrast enhancement, diffusion restriction, or other pathological features. Its appearance remained unchanged compared with previous imaging studies. This finding was interpreted as a non-progressive lesion, most likely of post-inflammatory, demyelinating, or vasogenic origin.

A neurological consultation confirmed the lesion's stability and determined that no specific neurological intervention was indicated at this time, recommending continued clinical and radiological monitoring.

Additional laboratory findings included hyperprolactinemia, tachycardia, and signs of unspecified systemic connective tissue involvement.

In-Hospital Management

Neuroleptics and adjunctive medications have been used in psychiatric treatment, including Clopixol, Haloperidol, Zolpidem (zolpidem), Clonazepam, Gabapentin (pregabalin),

Bisoprolol, Oxazepam, Bromocriptine, Neorelium, Propranolol, Benztropine, and Lorazepam. Due to intolerance to haloperidol (extrapyramidal symptoms and galactorrhea), the therapy was modified, improving the mental state, partially equalizing mood and drive, and reducing delusional and anxiety symptoms.

Post-discharge Pharmacotherapy and Recommendations

It was recommended to continue psychiatric treatment at the Mental Health Clinic and further neurological, endocrine, and internal medicine follow-up.
Specific recommendations for pharmacotherapy have been made (including Clopixol 25 mg 1-0-1, Pregabalin 150 mg 1-0-1, Corectin ½-0-0, and Bromocriptine 2.5 mg 1-0-1). The recommendations include avoiding benzodiazepines, alcohol, and psychoactive substances, as well as temporary contraindications to driving and working in high-risk conditions.

After achieving clinical improvement, the patient was discharged to the family on with recommendations for further outpatient follow-up and provision of 24-hour home care.

Discussion

This case illustrates the complexity of diagnosing and managing autoimmune encephalitis in a young patient with overlapping neurological, psychiatric, and endocrine abnormalities. The coexistence of autoimmune inflammation, post-infectious sequelae, and hormonal dysregulation created a multifactorial clinical picture that required continuous interdisciplinary collaboration. The initial episode, associated with influenza A infection, likely triggered an aberrant immune response leading to neuroinflammation and subsequent demyelinating lesions. Such infection-induced autoimmune activation has been described as a potential mechanism linking viral exposure with secondary encephalitic and endocrine manifestations [1, 2, 3].

The recurrence of psychotic symptoms after a period of neurological stability underscores the chronic and relapsing nature of autoimmune encephalitis. Persistent endocrine dysfunction manifested as hypothalamic amenorrhea and hyperprolactinemia further complicated treatment, as hormonal imbalance can both reflect and exacerbate central nervous system inflammation. This overlap between autoimmune thyroid disease and autoimmune encephalitis has been well documented, emphasizing shared immunopathological pathways involving both neural and endocrine autoantigens [4, 5, 6]. Psychiatric pharmacotherapy posed an additional challenge:

the need to balance symptom control with the risk of metabolic and extrapyramidal side effects, particularly in the context of hormonal sensitivity.

This case highlights the critical importance of early immunological testing, hormonal assessment, and neuroimaging in patients presenting with acute psychiatric or cognitive disturbances. Timely initiation of immunotherapy, followed by coordinated psychiatric and endocrine management, can prevent long-term neuropsychiatric sequelae. Ultimately, sustained collaboration between neurology, psychiatry, endocrinology, and internal medicine is essential for achieving clinical stabilization, preventing relapse, and improving overall prognosis in complex autoimmune neuropsychiatric disorders [1, 2, 7].

Conclusion

Autoimmune encephalitis should be recognized as a disorder that extends beyond the nervous system, often involving complex interactions with endocrine and psychiatric functions. The presented case demonstrates how immune-mediated inflammation can lead not only to neurological and cognitive symptoms but also to hormonal dysregulation and behavioral disturbances. Identifying these multisystemic connections is crucial for accurate diagnosis and timely initiation of immunotherapy, which may significantly influence prognosis and recovery. Effective management of such patients requires an integrated, interdisciplinary approach that combines neurological, psychiatric, and endocrine care. Long-term follow-up, including neuroimaging, hormonal evaluation, and autoantibody monitoring, remains essential to prevent relapse and to detect subtle residual dysfunction. Awareness of these overlapping mechanisms can improve clinical outcomes and contribute to a more holistic understanding of autoimmune neuropsychiatric syndromes.

Disclosure:
The authors declare that they have no financial or non-financial conflicts of interest that could be perceived as influencing the interpretation of the research findings or the content of this manuscript. This work was conducted indepedently without any external funding or support.

Author's contribution

Conceptualization: Wiktoria Marszał; **Methodology:** Wiktoria Marszał; **Software:** Sandra Czyż; **Validation:** Wiktoria Marszał; **Formal analysis:** Sandra Czyż, Wiktoria Marszał; **Investigation:** Sandra Czyż; **Resources:** Sandra Czyż, Wiktoria Marszał; **Data curation:** Sandra Czyż; **Writing – original draft:** Wiktoria Marszał; **Writing – review & editing:** Wiktoria Marszał, Sandra Czyż; **Visualization:** Sandra Czyż; **Supervision:** Sandra Czyż; **Project administration:** Wiktoria Marszał

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Not applicable

Informed Consent Statement

Our work did not involve direct human subject research or obtaining their consent for participation in the study.

Data Availability Statement

As a review paper, our work does not present new data or analyses. Therefore, there are no specific databases or data availability to report. The information and findings presented in this review are based on previously published studies, which can be accessed through their resective sources as cited in the reference section.

Conflicts of Interest Statement

The authors declare that there are no significant conflicts of interest associated with this research work.

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