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# Anti-Glutamic Acid Decarboxylase-Associated Autoimmune Encephalitis Presenting as Presumed Herpes Simplex Viral Encephalitis in a Patient With Type 1 Diabetes Mellitus: A Case Report

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## Abstract

A 68-year-old man presents with acute confusion, fevers, and new-onset seizures on the background of established type 1 diabetes mellitus (T1DM) and chronic Hashimoto's thyroiditis. An MRI scan identifies right-predominant multifocal T2/FLAIR hyperintense lesions involving the temporal, frontal, and parietal lobes without diffusion restriction. The patient was treated for presumptive herpes simplex virus encephalitis (HSE) with empiric antiviral therapy and clinically improved. Cerebrospinal fluid (CSF) subsequently returned strongly positive for anti-glutamic acid decarboxylase (anti-GAD) antibodies, with a negative extended viral panel. He was discharged home but re-presented two months later with progressive seizures, confusion, memory impairment, and interval progression of his MRI T2/FLAIR lesions, continuing to deteriorate despite broad-spectrum antimicrobial therapy. A diagnosis of anti-GAD-associated autoimmune encephalitis (AE) was established, and he made a rapid neurological recovery following initiation of immunosuppressive therapy. This case explores the diagnostic challenge of identifying anti-GAD-associated AE in the context of pre-existing T1DM, where elevated anti-GAD antibodies are not atypical, and highlights the importance of maintaining a broad differential in patients with autoimmune polyglandular disease presenting with encephalopathy.

**Categories:** Neurology, Internal Medicine, Allergy/Immunology
**Keywords:** anti-gad 65 encephalitis, anti-gad antibodies, autoimmune encephalopathy, autoimmune limbic encephalitis, herpes virus encephalitis

## Introduction

AE comprises a heterogeneous group of disorders in which the host immune system targets central nervous system (CNS) self-antigens [1]. With increasing clinical recognition and improved diagnostic testing, AE has been established as a leading cause of encephalitis, with an incidence comparable to cases attributable to viral infections [2,3]. The clinical spectrum of AE is broad, reflective of the diversity of the underlying antigenic targets, many of which are involved in critical neurophysiological processes [3]. Diagnosis and management are further complicated by the broad list of differentials with overlapping clinical features that require very different treatment strategies, including infectious, malignant and metabolic aetiologies [3].

Glutamic acid decarboxylase (GAD) is an intracellular enzyme expressed in both neurons and pancreatic beta cells [4]. It plays a key role in the decarboxylation of glutamate to gamma-aminobutyric acid (GABA), the principal inhibitory neurotransmitter of the central nervous system [5]. Anti-GAD antibodies are associated with a spectrum of neurological disorders that include a rare form of AE characterised by seizures, memory loss, and behavioural disturbance [2,6-10]. The clinical and radiographic features of anti-GAD-associated AE overlap considerably with other forms of both autoimmune and infectious encephalitis [11], with diagnosis often dependent on identification of elevated anti-GAD antibodies in serum and CSF. This diagnostic reliance on serology is complicated by the presence of anti-GAD antibodies also being a recognised feature of non-neurological autoimmune conditions such as T1DM [9,12], which not infrequently coexist in patients with anti-GAD-associated AE [9].

We report a case of anti-GAD-associated AE in an adult male with multiple autoimmune and renal comorbidities initially managed as presumptive HSE. Early improvement with aciclovir was followed by clinical and radiological relapse, which then improved following treatment with immunosuppression. This case illustrates the difficulty of interpreting anti-GAD serology in patients with co-existent autoimmune endocrine disease, and highlights features that should prompt early consideration of a primary autoimmune CNS pathology.

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## Case Presentation

A 68-year-old man with a background of T1DM, end-stage renal disease on peritoneal dialysis and Hashimoto's disease presented to the emergency department with acute-onset confusion, fevers and new focal and generalised seizures. He had no prior history of neurological disease or cognitive impairment and was functionally independent at baseline, living at home with his family and able to self-administer his peritoneal dialysis.

A lumbar puncture performed on admission showed mildly elevated CSF protein, elevated glucose and minimal pleocytosis. A CSF viral polymerase chain reaction (PCR) panel was negative. An MRI brain on admission showed right-predominant cortical and subcortical T2/FLAIR hyperintensities involving the temporal, inferomedial frontal and parietal lobes, without involvement of the hippocampus or amygdala. Given the clinical and radiological findings, HSE was considered the most likely diagnosis, and he was treated with IV aciclovir empirically. Within 24 hours, his confusion had significantly improved, and his seizures abated, following which both serum and CSF samples were found to be strongly positive for anti-GAD antibodies. Given his rapid improvement with antiviral therapy, these were assumed to be pre-existing in the context of his known T1DM, and in the context of ongoing clinical improvement, he was discharged with plans for outpatient neurologist follow-up. No repeat viral PCR was done prior to discharge.

On discharge, he continued to experience mild ongoing confusion and a functional decline from his premorbid baseline, now requiring substantial family assistance for his peritoneal dialysis. He reported slowly progressive forgetfulness and new-onset prosopagnosia. His family reported gradually worsening behavioural changes and disruption of his sleep-wake cycle. Two months following his initial discharge, he developed acutely worsening confusion and recurrence of focal aware seizures, manifesting primarily as unilateral left perioral twitching, and re-presented to the emergency department.

On examination, he was alert but confused and unable to follow commands. There was no hypertonia, hyperreflexia, or ataxia. He was febrile but had no features of meningism. He had diabetic ketoacidosis, suspected to be in the context of missed insulin doses. His blood sugar level was 42 mmol/L (3.5-7.7mmol/L), his pH on a venous blood gas was 7.17 (7.3-7.4), and his blood ketone level was 4 mmol/L (< 1.5 mmol/L). A presumptive diagnosis of recurrent infective encephalitis was made, and he was commenced on empirical antimicrobials. Despite broad antimicrobial cover and rapid biochemical resolution of his diabetic ketoacidosis, he deteriorated over the ensuing days. He developed refractory focal status epilepticus with worsening confusion and a reduced level of consciousness, ultimately requiring intubation and vasopressor support in the intensive care unit (ICU).

### Investigations

Key laboratory investigations are included below (Table *1*). Serum immunoglobulins, kappa/lambda free light chain ratio, and peripheral blood flow cytometry were within normal limits. Serum HSV-1 and varicella zoster IgG were positive on both admissions, consistent with prior exposure rather than acute infection. Anti-GAD antibodies remained strongly positive in both CSF and serum. An extended CSF and serum AE and onconeuronal antibody panel was negative, including NMDAR, LGI1, CASPR2 and AMPAR antibodies. Serum anti-GAD antibodies were assessed by tissue-based indirect immunofluorescence assay, with subsequent line blot assay for confirmation. Serum anti-GAD antibody quantification was not performed. CSF anti-GAD antibodies were assessed by line blot assay, with subsequent quantification performed by an enzyme-linked immunosorbent assay (ELISA). CSF anti-GAD quantification was not performed on the first admission.

A heavy metal screen, vasculitis panel and serum infectious screen, including syphilis serology and an interferon gamma release assay, were negative on the second admission. Thyroid-stimulating hormone and thyroxine levels were within normal limits on both admissions. Anti-thyroid peroxidase antibodies were 1444 kIU/L (< 115 kIU/L) and anti-thyroglobulin antibodies were 147 kIU/L (< 34 kIU/L) on the second admission, unchanged from premorbid values in the context of his known Hashimoto's thyroiditis (Table *1*).

| Investigation | First Admission | Second Admission | Reference Range |
|---|---|---|---|
| Serum | | | |
| Anti-GAD antibodies | Strongly positive | Strongly positive | - |
| Cerebrospinal fluid | | | |
| Protein | 0.67 g/L | 0.41 g/L | 0.19-0.65 g/L |
| Glucose | 6.1 mmol/L | 5.4 mmol/L | 2.5-4.5 mmol/L |
| Red blood cells | 0 x $10^6$/L | 0 x $10^6$/L | 0 x $10^6$/L |
| Mononuclear cells | 3 x $10^6$/L | 1 x $10^6$/L | < 5 x $10^6$/L |
| Polymorphonuclear cells | 1 x $10^6$/L | 0 x $10^6$/L | 0 x $10^6$/L |
| Viral polymerase chain reaction panel | Negative | Negative | - |
| *Neisseria meningitidis* DNA | Negative | Negative | - |
| *Streptococcus pneumoniae* DNA | Negative | Negative | - |
| Cryptococcal antigens | Negative | Negative | - |
| Anti-GAD antibodies | Strongly positive | 136 IU/mL | < 5 IU/mL |
| RT-QuIC | Not completed | Negative | - |
| Protein 14-3-3 | Not completed | Negative | - |
| Cytology | Not completed | No malignant cells | - |

**TABLE 1: Key investigation results.**

The viral polymerase chain reaction panel included enterovirus RNA, herpes simplex virus 1 and 2 DNA, varicella zoster DNA and parechovirus RNA. Blood glucose levels had normalised by the time of lumbar puncture on the second admission.

A contrast computed tomography (CT) angiogram of the brain on second presentation showed no acute pathology. A 32-channel scalp routine EEG on day three of the second admission while intubated demonstrated a diffusely suppressed background rhythm with intermittent low-voltage sharp waves in the central region, in keeping with a severe encephalopathy. An MRI brain on the same day showed multiple new subcortical predominant T2/FLAIR hyperintense lesions in the right temporal lobe and insula, bilateral frontal and left parietal lobe, without involvement of the hippocampus or amygdala and without diffusion restriction (Figure *1*). A malignancy screen involving neck, chest, abdomen, and pelvis CT and a testicular ultrasound was unremarkable.

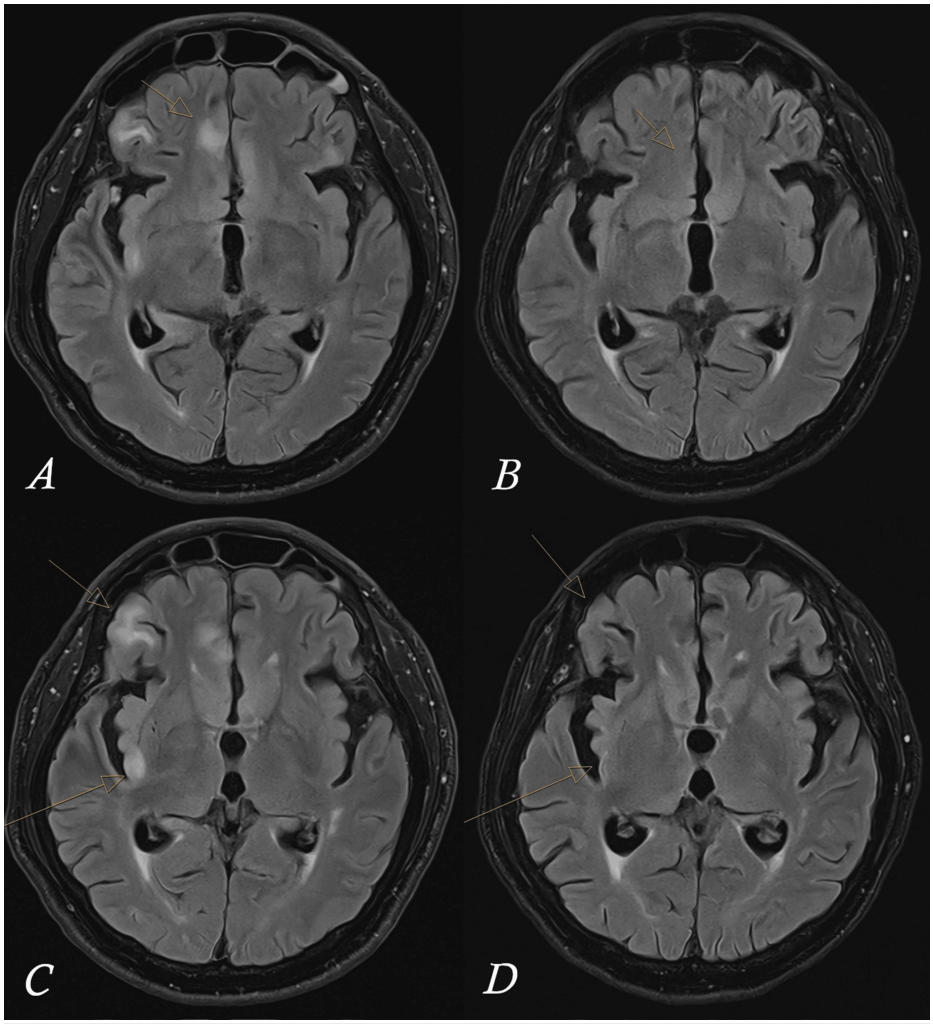



**FIGURE 1: Axial T2/FLAIR sequence MRI brain.**

Axial T2/FLAIR sequence MRI brain before (A and C) and 2 weeks after (B and D) immunosuppressive treatment. Panels A and B demonstrate the right inferomedial frontal lobe hyperintense lesion, while panels C and D demonstrate the hyperintensities in the right insula cortex.

## Differential diagnosis

HSE was the initially favoured diagnosis on first admission owing to the acute onset of confusion, fever and seizures, temporal lobe lesions on MRI and rapid clinical improvement following antiviral therapy. The negative CSF viral PCR was attributed to early sampling and the positive anti-GAD in serum, and CSF was assumed to be pre-existing in the context of known T1DM. Interpretation of anti-GAD was complicated by the result returning positive only following the clinical improvement attributed to antiviral treatment. In retrospect, the absence of diffusion restriction and sparing of the amygdala and hippocampus were atypical for HSE, and the strongly positive CSF anti-GAD antibodies may have suggested an underlying autoimmune process.

On second admission, recurrent infective encephalitis was suspected, given the fevers, acute deterioration and relative immunocompromise secondary to T1DM and end-stage renal disease. This was considered less likely following return of the negative CSF viral panel and failure to improve following antimicrobial therapy, at which point the leading differential became AE either de novo or as a post-infectious phenomenon.

Features in keeping with an anti-GAD-associated AE on the second admission included the subacute progressive cognitive and behavioural changes, focal seizures, temporal lobe MRI lesions and the strongly positive CSF anti-GAD antibodies. Seizure-related changes may have contributed to some of the MRI lesions, though they were thought unlikely to account for them entirely or for the cognitive and behavioural decline between admissions. The diagnosis was further supported by the clinical and radiological improvement after immunosuppressive treatment.

An underlying CNS lymphoproliferative disorder was weighed unlikely following normal serum immunoglobulins, peripheral flow cytometry, reassuring CT and testicular ultrasound and negative CSF cytology. Steroid-responsive encephalopathy associated with autoimmune thyroiditis was also considered as a differential but favoured to be less likely given the stable anti-thyroid antibody titres, euthyroid state and markedly elevated CSF anti-GAD antibody. Creutzfeldt-Jakob disease was entertained given the rapidly progressive cognitive decline but was considered unlikely following negative RT-QuIC and 14-3-3.

### Treatment

On admission to the ICU, renally adjusted empiric antimicrobial cover was commenced with intravenous (IV) ceftriaxone 2 g twice daily, IV benzylpenicillin 1.2 g four times daily and IV aciclovir 375 mg daily. Diabetic ketoacidosis was managed with an IV insulin infusion as per local protocol, with biochemical resolution within 12 hours of treatment. Antiseizure therapy was escalated to IV valproate 750 mg twice daily, IV levetiracetam 1000 mg twice daily, and a continuous IV midazolam infusion, with seizure resolution by day two of admission. Despite this, he remained ventilator-dependent without improvement in his level of consciousness. IV methylprednisolone was commenced on day seven of his admission, at a dose of 1 g daily for five days, with intravenous immunoglobulin (IVIG) 130 g divided over 4 doses commenced on day 10.

The patient's neurological status began to improve on day 10 following commencement of IVIG. Seizures did not recur, and he was safely extubated on day 13. Repeat MRI brain on day 20 of his admission demonstrated marked improvement of the previously identified T2/FLAIR hyperintensities (Figure *1*). Rituximab 1 g IV was added on day 30 after multidisciplinary team discussion, given the severity of the initial presentation, with two doses two weeks apart and a plan to continue six-monthly for 24 months. Following his course of methylprednisolone, he was continued on oral prednisolone 70 mg until day 30, at which point it was reduced to 40 mg with a plan for further reduction by 5 mg every two weeks. The IVIG was planned to continue at 70 g monthly for 24 months.

His inpatient course following discharge from ICU was complicated primarily by highly labile blood sugar levels in the setting of his ongoing corticosteroid therapy. With regular specialist Endocrinology input, this gradually improved as steroids were weaned, though it remained a barrier to performing an inpatient fluorodeoxyglucose positron emission tomography (FDG-PET) scan for further investigation of underlying malignancy. He also developed localised herpes zoster across his right T4 dermatome on day 35 of his admission, necessitating a delay in his second rituximab dose by two weeks and a further course of renally adjusted antiviral therapy in the form of aciclovir 375 mg IV daily for seven days followed by valaciclovir 1000 mg daily for seven days. The steroid and IVIG schedules continued unchanged. Despite these complications and significant deconditioning following his critical illness, his function improved gradually with regular physiotherapy, and by discharge he was mobilising independently with a single-point stick, although he continued to require assistance with his regular peritoneal dialysis. He was discharged on day 50 with plans for an outpatient FDG-PET for further screening for an underlying malignancy.

## Discussion

Anti-GAD-associated AE represents a rare but increasingly recognised manifestation of the spectrum of neurological disorders associated with anti-GAD antibodies [2,6-10], with a prevalence of approximately 2 per 100,000 [2] and a median age of onset of 25-45 years [7]. It typically presents with subacute onset of seizures, amnesia, cognitive impairment, confusion, behavioural changes and psychiatric features [8,13], and is associated with an underlying malignancy in 26% of cases [14]. Neuroimaging characteristically demonstrates T2/FLAIR hyperintensities affecting the medial temporal lobe, including the hippocampus and amygdala [15], with CSF testing and serology showing high titres of anti-GAD antibody [7,9].

A key diagnostic challenge that has emerged from the broader literature is differentiating between AE and HSE, due to the significant clinical and radiological overlap between these syndromes [16]. HSV encephalitis is also associated with T2/FLAIR lesions affecting the medial temporal lobes, though less typically the basal ganglia, and can present with significant overlap of the clinical features associated with AE [16,17]. The incomplete resolution of symptoms following antivirals seen in this case is frequently observed in HSE and was not thought of as atypical in this context [18]. The negative initial CSF viral panel is also an established phenomenon for HSE, often reflective of sampling within the first four days of infection [17,19]. The negative PCR two months later is similarly difficult to interpret, as HSV PCR is typically negative by this point following antiviral therapy [20].

One feature of this presentation which in retrospect may have alluded to a primary anti-GAD-associated AE was the high CSF anti-GAD titres. Although elevated anti-GAD antibodies are a well-recognised feature of T1DM [12], very high titres in serum and CSF are more characteristic of anti-GAD neurological disorders than isolated endocrinopathies [21]. Anti-GAD antibodies are quantified by ELISA originally designed for use in T1DM, which has superseded the previously used radioimmunoprecipitation assays [22]. An ELISA-based CSF anti-GAD concentration exceeding 100 IU/mL, or serum concentrations greater than 10,000 IU/mL, have been suggested as a threshold warranting further investigation for the presence of an anti-GAD neurological syndrome [9,21,22]. The use of an antibody index (AI) has also been proposed as a means of differentiating true intrathecal antibody synthesis from passive diffusion of antibody from the periphery into the CSF

[23,24]. While titre quantification and the use of an AI may be promising discriminatory tools in patients with co-existing T1DM, it should be acknowledged that these are not always readily available in the clinical setting and the need to act rapidly may initially preclude awaiting this level of diagnostic nuance. Emerging methods of antibody detection, including aptamer-based assays [25], and methods of assessing fluid biomarkers of disease activity [26] may in future expand our ability to differentiate anti-GAD neurological syndromes from their mimics.

The complexities in this case mean that the precise sequence of events is difficult to determine with certainty, even in retrospect. HSE is an established trigger for subsequent AE, with the putative mechanism involving virally induced disruption of the blood-brain barrier leading to release of neuronal antigens, precipitating a secondary autoimmune response [27]. Despite the negative CSF HSV results, the timing of the test results means that it remains possible that the index presentation did represent HSE, with anti-GAD-associated AE developing subsequently, causing the deterioration leading to the second admission. Alternatively, both conditions may have co-existed from the outset, with the autoimmune component only becoming clinically dominant following resolution of the infective process. In retrospect, a repeat viral PCR prior to the first-admission discharge or performance of an AI to support likely intrathecal antibody synthesis would have assisted in delineating these theories.

The evidence base for the treatment of anti-GAD-associated AE remains limited to observational studies and case series. The use of corticosteroids and IVIG as induction agents and the use of rituximab as second-line therapy are reflective of current practice [28]. While immunotherapy is effective at least initially in most cases [21], long-term response rates remain variable [29,30], which partly underpinned the rationale for prolonged immunosuppression in this case. The consequences of this approach must be carefully considered. Anti-GAD neurological disorders share a significant epidemiological overlap with T1DM [9], a population in which prolonged steroid therapy can cause marked glycaemic instability. The risk of infection is a further concern, especially in individuals with pre-existing immunocompromise, with infection-associated treatment interruptions risking clinical relapse.

## Conclusions

This case illustrates the significant diagnostic complexity inherent to anti-GAD AE, particularly in those with pre-existing T1DM. Markedly elevated CSF anti-GAD titres should prompt consideration of an anti-GAD neurological disorder, even in the presence of co-existing T1DM. Differentiating between HSE and AE in the acute setting is difficult, and a low threshold for empirical immunotherapy should be maintained in patients with potential features of AE who do not respond to antimicrobial therapy.

## Additional Information

### Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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