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SREAT :- A RARE CASE OF RECURRENT ENCEPHALOPATHY

Neurology

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ABSTRACT

Background: SREAT (Steroid responsive encephalopathy associated with autoimmune thyroiditis) may follow a relapsing remitting course with recurrence of encephalopathy, seizures, psychiatric symptoms and abnormal gaits after initial response or while tapering down the steroids, the optimal duration of tapering regimen of corticosteroid therapy still remain uncertain. There is no guidelines yet on how to prevent the relapse in SREAT. Our case is notable for recurrent SREAT with multiple episodes of encephalopathy, seizure, and psychiatric symptoms followed by clinical improvement with steroid therapy. This case highlights the relapsing nature of SREAT and the challenges involved in long term management. **Case Presentation:-** A 49 year old female with recurrent episodes of, altered behavior, excessive sleepiness, memory disturbances. She would stop eating, her gait was changed, this would gone on for 3-4 days followed by seizure episodes. Following exclusion of infectious, metabolic, structural, neoplastic, and alternative autoimmune causes, SREAT had been diagnosed. The patient improved with intravenous Methyl prednisolone and recovered their mobility and sensorium within 9-10 days, however she experienced recurrent episodes after seven months. **Conclusion:** This case highlights the relapsing nature of SREAT and the difficulty of establishing the diagnosis in the presence of nonspecific neurological findings. There is a combination of matching clinical features, markedly high Anti TPO levels, supportive investigations, exclusion of differential diagnosis, and good response to corticosteroids supported the diagnosis. Recurrent disease may require prolonged immunotherapy and consideration of steroid sparing agents.

KEYWORDS

SREAT (Steroid Responsive Encephalopathy Associated with Autoimmune Thyroiditis), HE (Hashimoto's Encephalopathy), Autoimmune Encephalitis, Anti-TPO (Anti Thyroid Peroxidase), Corticosteroids, Rituximab

INTRODUCTION

Hashimoto's encephalopathy (HE) also called Steroid responsive encephalopathy associated with autoimmune thyroiditis (SREAT), is a rare brain disorder linked to thyroid autoimmunity. It was first reported in 1966 and the name SREAT was introduced later [1,2]. Patients may develop sudden or gradual confusion, memory disturbances, seizures, psychiatric symptoms, jerky movements, tremor, walking difficulty, or speech problems [3,7,8]. There is no specific test for HE. Thyroid function is often normal or only mildly low, and anti-TPO and anti-thyroglobulin antibodies, though often raised, are not specific and do not track with severity [2,3,7,9].

In 2016, Graus et al. included HE in a clinical framework for autoimmune encephalitis. It requires subacute encephalopathy with seizures, myoclonus, hallucinations, or stroke-like episodes plus reasonable exclusion of other causes [4]. These criteria were later refined by Mattozzi et al. [5]. HE can be over diagnosed. In a Mayo Clinic case series of 144 suspected cases, 73% turned out to have a non-immune diagnosis [6].

Corticosteroids are the main treatment and most patients improve[3,7,8]. Relapse is common, however, In a review of 251 cases, 91% improved neurologically, but 16% relapsed at least once [3]. Second-line options such as rituximab have been reported in difficult cases, including children [10].

Case Presentation

A 49-year old female from Delhi, a homemaker with no formal education, had a 20 year history of hypothyroidism treated with levothyroxine 25 µg daily without follow-up and she was non-compliant of medication. She had intermittent left ear discharge for 10–15 years (chronic suppurative otitis media). There was no history of diabetes, fever, rash, joint pain, recurrent oral ulcers or any substance abuse and no relevant family history. Before her illness she was alright.

Episode 1 (January 2026): Diagnosis of SREAT

The first symptom was excessive daytime sleepiness. She stopped waking at her usual time, had to be shaken awake and drifted back to sleep after meals. She developed a broad based swaying gait and bilateral upper-limb action tremor causing her to spill water and smear food around her face, requiring assistance with feeding. She became withdrawn, staring vacantly without eye contact. By day 5, she was bedbound and mute, with eye opening only and had a seizure with upward eye deviation and head turning followed by approximately 30 minutes of unconsciousness.

She was admitted to the Department of Medicine on 16 January 2026. On examination, GCS was 9/15 (E3V1M5), BP 138/72 mmHg, pulse 102/min, SpO_2 98%, and she was afebrile, drowsy and disoriented. Differential diagnoses include infectious meningoencephalitis, metabolic or toxic encephalopathy, structural or vascular disease, antibody-defined autoimmune encephalitis, paraneoplastic disease, and SREAT. Investigations are shown in Table 1.

She received IVMP 1 gm daily for five days, with GCS improving from 9/15 to 15/15 within 48 hours and altered sensorium, disorientation and psychotic symptoms resolving over the next 4–5 days.

She was discharged on 29 January 2026 on oral prednisolone with outpatient tapering, levothyroxine 25 µg, and levetiracetam 500 mg twice daily. She regained full independence in ADLs by the first week of February 2026. Contrast-enhanced MRI of the brain on 7 February 2026 was normal. Prednisolone was tapered and discontinued in April 2026, and no steroid-sparing agent was started.

Episode 2 (September 2026): Relapse After Steroid Withdrawal

About five months after stopping steroids, she again developed excessive sleepiness, made no eye contact with family members and gait abnormalities like first episode. After two days she had two GTCS at home while being helped back from the washroom, fell and

sustained a left scapular fracture, and was brought to hospital. on 8 September 2026 she was brought to emergency as she had a generalized tonic clonic seizure and during the seizure she fractured her left scapula.

She received IVMP from 10 to 14 September 2026 for 5 days followed by oral prednisolone 60 mg daily. Her sensorium improved, and within 4–5 days she was walking, conversing normally and back at baseline. Because this was her second episode, the neurology team started rituximab 1 gm on 25 September 2026 after premedication, with a second 1 gm dose planned at follow-up. Zoledronic acid 4 mg was given on 24 September 2026 for bone protection. At discharge on 25 September 2026, she was taking prednisolone 60 mg daily (to be reviewed for tapering after two weeks), cotrimoxazole double-strength three times weekly, calcium, weekly vitamin D_3, levothyroxine 50 µg, levetiracetam 500 mg twice daily and lactulose, with a sling for the scapular fracture. Follow-up has been planned in the autoimmune clinic, with a repeat contrast MRI of the brain and a DEXA scan.

Table 1. Key Investigations Across Episodes

Investigation	Result	Timing
Anti-TPO antibody	>1000 IU/mL	Jan 2026; Sep 2026
Thyroid function	Normal (euthyroid); no features of myxoedema or thyrotoxicosis	Jan 2026
Thyroid function	TSH 1.593, T3 2.09, T4 0.87 (within normal limits)	Sep 2026
CSF	0 cells; protein 198 mg/dL; glucose 68 mg/dL; ADA <3 U/L; viral meningoencephalitis panel negative	Jan 2026
CSF	2–4 cells; protein 125 mg/dL; glucose 65 mg/dL (blood 126); Gram stain, culture, VDRL, TPHA, CrAg, viral PCR negative; OCB absent; IgG index 0.35	Sep 2026
Neuronal surface antibodies (serum and CSF)	NMDAR, AMPAR1, AMPAR2, GABA-B receptor, LGI1, CASPR2: Negative	Jan 2026
Onconeural / intracellular synaptic antibodies	Amphiphysin, CV2/CRMP5, PNMA2 (Ma2/Ta), Ri, Yo, Hu, recoverin, SOX1, titin, Zic4, GAD65, Tr (DNER): Negative	Jan 2026
Systemic autoimmunity	ANA, ANCA, antiphospholipid antibodies Negative; C3 179, C4 59 (normal)	Jan and Sep 2026
Serum immuno-globulins	IgG 2829 (raised); IgA 246	Sep 2026
Infection screen	HIV, HBsAg, anti-HCV, VDRL non-reactive; procalcitonin negative; blood and urine cultures sterile; Cy-TB Negative	Sep 2026
Tumour screen	AFP 2.37, CEA 1.54, CA 19-9 and CA-125 normal; CECT thorax and abdomen Normal	Jan and Sep 2026
Metabolic and haematology	Renal and liver function, electrolytes, glucose, calcium, ammonia normal (Jan). Hb 12 g/dL, WBC 7030/µL, platelets 2.1 lakh/µL, urea 23, creatinine 0.7, Na 134, K 4.1, Ca 9.1, vitamin B12 309 pg/mL, vitamin D 36, HbA1c 5.7% (Sep)	Jan and Sep 2026
Brain imaging	CECT and MRI brain: no infarct, haemorrhage, mass, demyelination or features of limbic encephalitis or CNS vasculitis	Jan 2026
Brain imaging	CEMRI brain: normal apart from sphenoid sinusitis	Feb 2026
Brain imaging	NCCT head: normal	Sep 2026
EEG	Generalised slowing in the theta range, No epileptiform discharges	Sep 2026
Other	2D echo LVEF 55–60%; USG abdomen grade 1 fatty liver; chest X-ray left scapular fracture	Sep 2026

ADA, Adenosine deaminase, CrAg, Cryptococcal antigen, OCB, Oligoclonal bands, CEMRI, Contrast-enhanced MRI; NCCT, Non-contrast CT.

Diagnostic Assessment

Once infectious, metabolic, toxic, structural, neoplastic and antibody-defined autoimmune causes had been excluded, the patient fulfilled all six Graus criteria for Hashimoto encephalopathy (Table 2) [4]. She also met the additional features emphasised by Mattozzi et al, subacute onset and an anti-TPO titre above 200 IU/mL [5]. Her diagnosis does not rest on antibody titre alone. It is supported by objective findings: subacute onset, seizures and ataxia, raised CSF protein on two occasions, generalised theta slowing on EEG, and a rapid, reproducible, measurable response to corticosteroids (GCS 9→15 within 24 hours in January, and return to baseline within 4–5 days in September).

Table 2. Graus et al. (2016) Criteria for Hashimoto Encephalopathy Applied to this Patient

Criterion [4]	Findings in this patient	Met
1. Encephalopathy with seizures, myoclonus, hallucinations or stroke-like episodes	Subacute encephalopathy with GTCS in all three episodes, visual hallucinations and exaggerated auditory startle	Yes
2. Subclinical or mild overt thyroid disease	Known hypothyroidism, clinically euthyroid with normal TSH/T3/T4 at both admissions; goitre	Yes
3. Brain MRI normal or with non-specific abnormalities	MRI normal (incidental sphenoid sinusitis only)	Yes
4. Serum thyroid antibodies (TPO and/or thyroglobulin)	Anti-TPO >1000 IU/mL on two occasions	Yes
5. Absence of well-characterised neuronal antibodies in serum and CSF	Cell-surface, onconeural and intracellular synaptic panels negative in serum and CSF	Yes
6. Reasonable exclusion of alternative causes	Infectious, metabolic, toxic, structural, neoplastic and systemic autoimmune causes excluded	Yes

DISCUSSION

SREAT is a diagnosis of exclusion, and thyroid antibodies alone are not specific. Although our patient had anti-TPO levels >1000 IU/mL, the diagnosis was supported by her subacute and relapsing encephalopathy, seizures, ataxia, abnormal EEG, negative neuronal antibodies in serum and CSF, and exclusion of infectious, metabolic, structural, and neoplastic causes [3,6,9]. Her presentation with excessive sleepiness, tremor, gait disturbance, mutism, behavioural changes, and seizures demonstrates the broad clinical spectrum of SREAT and its potential to mimic psychiatric illness [6,7,8].

The most important feature of this case was the relapsing course, with two episodes over approximately seven months despite a marked response to corticosteroids. Relapse has been reported in SREAT, with Laurent et al. reporting recurrence in 16% of 251 cases [3]. Recurrent disease may require prolonged corticosteroid therapy and consideration of steroid-sparing immunotherapy [3,7,8]. In our patient, repeated episodes following previous steroid treatment raised concern for steroid-dependent or recurrent disease.

Although there are no standardized guidelines for maintenance therapy [7,8], immunosuppressive agents including azathioprine, mycophenolate mofetil, intravenous immunoglobulin, and rituximab have been reported in recurrent or steroid-resistant cases [3,7,8,10]. In view of the multiple relapses and need for repeated corticosteroid therapy, Rituximab had been given as steroid-sparing therapy in this patient. This case highlights the importance of recognizing relapse early and considering longer term immunotherapy in patients with recurrent SREAT.

CONCLUSION

SREAT should be considered in patients with subacute neuro-psychiatric symptoms, seizures, fluctuating consciousness, or movement abnormalities with evidence of thyroid autoimmunity. It is particularly important when symptoms are initially attributed to a primary psychiatric disorder and do not respond to antipsychotics. Although corticosteroids often produce a marked response, relapse may occur after treatment withdrawal. Recurrent disease may require

prolonged steroid therapy and consideration of steroid sparing immunotherapy such as Rituximab.

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