
CASE REPORT**Autonomic-predominant longitudinally extensive acute transverse myelitis associated with autoimmune thyroid disease***Manjunath S^{1*}, Pankaj Singhai¹, Pramod Krishnan²**¹Department of Internal Medicine, ²Department of Neurology,
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Abstract

Longitudinally Extensive Transverse Myelitis (LETM) is most commonly associated with neuromyelitis optica spectrum disorders, infections, or systemic autoimmune diseases. Association with autoimmune thyroid disease is rare and poorly understood. We report a 47-year-old man who presented with unexplained sweating, dysesthesia, severe constipation, and acute urinary retention due to acute LETM extending from T2 to T9. Extensive evaluation excluded infectious, vascular, and common systemic autoimmune causes. He had hypothyroidism and he tested positive for anti-thyroid peroxidase and anti-thyroglobulin antibodies with high titres. The patient showed marked clinical improvement following treatment with high-dose steroids. This case highlights autoimmune thyroid disease as a potential immune trigger for acute LETM and dysautonomia preceding sensory or motor symptoms in LETM can be misdiagnosed as thyrotoxicosis.

Keywords: LETM, Transverse myelitis, Hashimoto thyroiditis, Autoimmune thyroiditis, Autonomic nervous system

Introduction

Longitudinally Extensive Transverse Myelitis (LETM), characterized by a spinal cord lesion extending over three or more vertebral segments on MRI, represents a severe subtype of inflammatory myelopathy with motor or sensory abnormalities at presentation. Although autonomic disturbances including bladder, bowel, and cardiovascular dysfunction are almost always present during the disease course, dysautonomia as the initial presentation is distinctly uncommon [1].

While Neuromyelitis Optica Spectrum Disorders (NMOSD), Myelin Oligodendrocyte Glycoprotein (MOG) Antibody-associated disease, infections, and systemic autoimmune diseases like systemic lupus erythematosus, Sjogren's syndrome and sarcoidosis account for most cases of LETM, a subset remains idiopathic [1,2]. Autoimmune thyroid disease has been sporadically reported in association with central nervous system demyelination, but spinal cord involvement is rare and poorly understood. Coexis-

tent autoimmune thyroid disease (AITD), including both Grave's disease [3] and autoimmune thyroiditis [2,4,5], has been documented in a sparse but expanding set of case reports, illustrating temporal and clinical associations with demyelinating myelitis especially NMOSD. Such cases underscore the importance of comprehensive serologic evaluation for Aquaporin-4, MOG, and systemic autoantibodies in patients presenting with LETM, as early diagnosis and tailored therapy are critical determinants of outcome [6]. Case reports on autoimmune thyroiditis associated transverse myelitis even have noted high risk of relapse and recurrence of myelitis in those cases although sample size had been very low to predict causation [5].

Case report

A 47-year-old man presented with 15 days of unexplained episodic sweating and generalized weakness. This was followed by progressive mid-

thoracic back pain and severe constipation unresponsive to laxatives. Four days prior to admission, he developed abnormal pricking sensations over the upper trunk, followed by acute urinary retention requiring catheterization.

There was no antecedent fever, respiratory illness, vaccination, or gastrointestinal infection. He denied limb weakness, visual symptoms, diplopia, gait imbalance, or sensory loss in the extremities. Initial evaluation and management elsewhere focused on cardiogenic and anxiety-related causes of hyperhidrosis, and was unrewarding.

Past medical history

He had been suffering from type 2 diabetes mellitus with nephropathy and non-proliferative diabetic retinopathy on oral hypoglycemic agents and basal insulin, hypothyroidism on levothyroxine, immune thrombocytopenia (treated previously with steroids and azathioprine, currently on romiplostim), and recently diagnosed hypertension.

Examination

On examination, he was afebrile but tachycardic with labile blood pressure. Neurological examination revealed intact cranial nerves and normal motor strength in all limbs. Sensory examination demonstrated patchy reduction in thoracic sensation (right greater than left) with marked dysesthesia. Deep tendon reflexes were globally reduced, with a unilateral extensor plantar response on the left. Prominent autonomic features included cold, clammy upper limbs, bowel pseudo-obstruction, and persistent urinary retention. Fundus examination showed features of non-proliferative diabetic retinopathy.

Investigations

Complete hemogram showed neutrophilic leucocytosis otherwise with normal hemoglobin and platelet count. Magnetic resonance imaging (MRI)

of the spine revealed a long segment of T2 hyperintensity involving the anterior third and central portions of the spinal cord extending from T2 to T9, without compression, hemorrhage, or vascular flow voids. Figure 1 shows the sagittal section of MRI of the thoracic spine depicting long segment T2 hyperintensity involving the anterior third of the spinal cord from T2-T9. Figure 2 shows T2 hyperintensity in the anterior spinal cord at the third vertebral body level in the axial section of spine MRI. Brain MRI showed only mild age-inappropriate cerebral and cerebellar atrophy without demyelinating lesions or optic neuritis. Cerebrospinal fluid analysis demonstrated 4 lymphocytes/mm³ with normal protein, glucose and adenine deaminase levels. There was no evidence of infection; bacterial cultures and Mycobacterium tuberculosis polymerase chain reaction were negative. Testing for CSF oligoclonal bands, IgG index, aquaporin-4 and MOG antibodies were negative as well.

Inflammatory markers, including C-reactive protein, were normal. Autoimmune testing including anti-nuclear antibodies, antineutrophil cytoplasmic antibodies, angiotensin-converting enzyme levels, and infectious serologies were negative. Serum creatinine phosphokinase was mildly elevated (597U/L). Evaluation for systemic malignancy and metabolic causes was unremarkable except for recent fluctuations in serum Thyroid Stimulating Hormone (TSH) level. Evaluation revealed euthyroid state with markedly elevated anti-thyroid peroxidase (TPO) antibody (284 IU/mL) and anti-thyroglobulin antibody titres suggestive of autoimmune thyroiditis.

Differential diagnosis

The differential diagnoses included NMOSD, MOG-associated disease, spinal cord ischemia, infectious myelitis, paraneoplastic myelopathy, and idiopathic autoimmune transverse myelitis. The

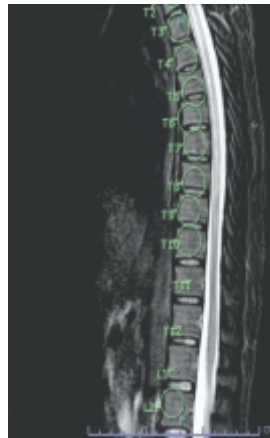


Figure 1: Sagittal section of MRI of the thoracic spine depicting long segment T2 hyperintensity involving the anterior

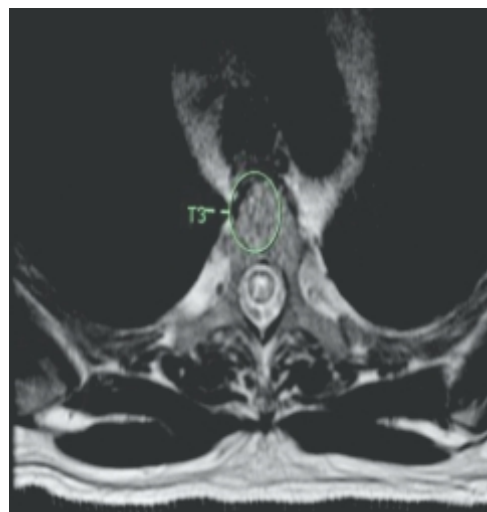


Figure 2: T2 hyperintensity in the anterior spinal cord at the third vertebral body level in the axial section of spine MRI

absence of optic neuritis, normal CSF inflammatory markers, preserved motor function, and steroid responsiveness favored an immune-mediated process distinct from classical NMOSD.

Treatment and outcome

The patient was treated with intravenous methylprednisolone (1 g daily for 5 days), followed by an oral steroid taper. Supportive management included bowel decompression, bladder catheterization, pain management, physiotherapy, optimization of

glycemic control and empirical antibiotics. Within one week, the patient demonstrated marked improvement in dysesthesia, sweating episodes, bowel function, and bladder control. He was discharged on a tapering course of oral corticosteroids along with gabapentin for neuropathic pain management, with advice for close outpatient follow-up. At discharge, he was ambulatory and hemodynamically stable; however, urinary catheterization was maintained due to persistent bladder dysfunction.

Discussion

This case illustrates the association between Hashimoto thyroiditis and LETM, presenting predominantly with autonomic dysfunction rather than motor deficits. While autonomic involvement is well described in spinal cord disease, its presentation as the primary clinical manifestation is uncommon and may lead to diagnostic delay. Selective involvement of the anterior horn and central cord, as seen in this patient, may explain the early dysautonomia and relative preservation of motor strength. Thyroid autoantibodies are generally considered markers of systemic immune dysregulation rather than direct mediators of central nervous system injury. However, emerging evidence suggests that autoimmune thyroid disease may predispose to immune cross-reactivity through molecular mimicry or by promoting a proinflammatory cytokine milieu [3,4].

Notably in our case, CSF inflammatory markers were minimal, and MRI showed anterior horn and central cord involvement, raising differential considerations including ischemia. However, anti-TPO antibodies in high titres, past history of immune thrombocytopenia, clinical evolution and steroid responsiveness supported an immune-mediated process. Mamootil & Grewal (2022) described similar acute transverse myelitis in a 73-year-old female with a known history of Hashimoto's thyroiditis [7]. Much like our case, their patient achieved a favorable prognosis following rapid diagnostic confirmation and a regimen that included corticosteroids. While the direct association of Hashimoto's thyroiditis with LETM or NMOSD is infrequent, Sharma *et al.* (2022) highlighted that presence of anti-TPO antibodies correlates with a more prolonged and severe course of myelitis [8]. The striking dissociation between

extensive MRI abnormalities and minimal cerebrospinal fluid inflammation suggests a compartmentalized immune process, potentially antibody-mediated or driven by innate immune activation within the spinal cord. Ohira *et al.* (2024) noted positive thyroid antibodies in transverse myelitis due to the "molecular mimicry" of epitopes between thyroglobulin and myelin [9]. Anti-TPO antibodies can form inflammatory immune complexes and modulate the immune response against myelin basic protein. This observation challenges the assumption that significant CSF pleocytosis or protein elevation is necessary for active inflammatory myelopathy.

Compared to the existing case reports, our case stands out due to its unusual initial presentation with severe dysautonomia preceding traditional motor or sensory symptoms; but it strongly aligns with the broader consensus that anti-TPO positivity may precipitate, modulate, or worsen antibody-driven inflammatory attacks on the spinal cord. It emphasises the need for testing for thyroid autoantibodies in cases of LETM, when classical conditions causing LETM are excluded, especially when a patient has associated thyroid disease.

Conclusion

This case expands the clinical spectrum of LETM and highlights the need for awareness of dysautonomia as an early sign of myelopathy. Autoimmune thyroid disease may serve as a predisposing immune trigger, even in the absence of classical neuroimmunological markers. Recognition of autoimmune thyroid disease as a potential trigger for LETM is crucial, especially when common etiologies are excluded.

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