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Neurology

A Rare Case of Longitudinally Extensive Transverse Myelitis Associated with Isolated Hyperparathyroidism

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Abstract | **Case Report**

Longitudinally extensive transverse myelitis (LETM) is typically associated with autoimmune, paraneoplastic, or infectious etiologies. We report a rare case of LETM in a middle-aged male with persistently elevated intact parathyroid hormone (iPTH) levels, normal calcium-phosphorus profile, and vitamin D deficiency, with complete recovery after corticosteroid therapy and vitamin D correction. This case raises the possibility of a rare parainflammatory association between isolated hyperparathyroidism and spinal cord demyelination.

Keywords: Transverse myelitis, Hyperparathyroidism, Vitamin D deficiency, LETM, Demyelination.

INTRODUCTION

LETM is defined as spinal cord inflammation extending over three or more vertebral segments. It is most commonly seen in conditions such as neuromyelitis optica spectrum disorder (NMOSD), MOG antibody disease (MOGAD), sarcoidosis, paraneoplastic syndromes, and infections. However, in a proportion of cases, no definitive cause is identified even after comprehensive evaluation. Hyperparathyroidism, particularly the normocalcemic variant, is not a widely recognized cause of LETM. Normocalcemic primary hyperparathyroidism (NPHPT) is characterized by elevated iPTH levels with consistently normal serum calcium and is often underdiagnosed. While NPHPT has been implicated in systemic and neuropsychiatric symptoms, its role in demyelinating spinal cord disease remains unclear. We report a unique case of LETM with persistently elevated iPTH and low vitamin D, but with normal calcium and phosphorus levls, in a patient who demonstrated full clinical recovery with steroids and vitamin D therapy.

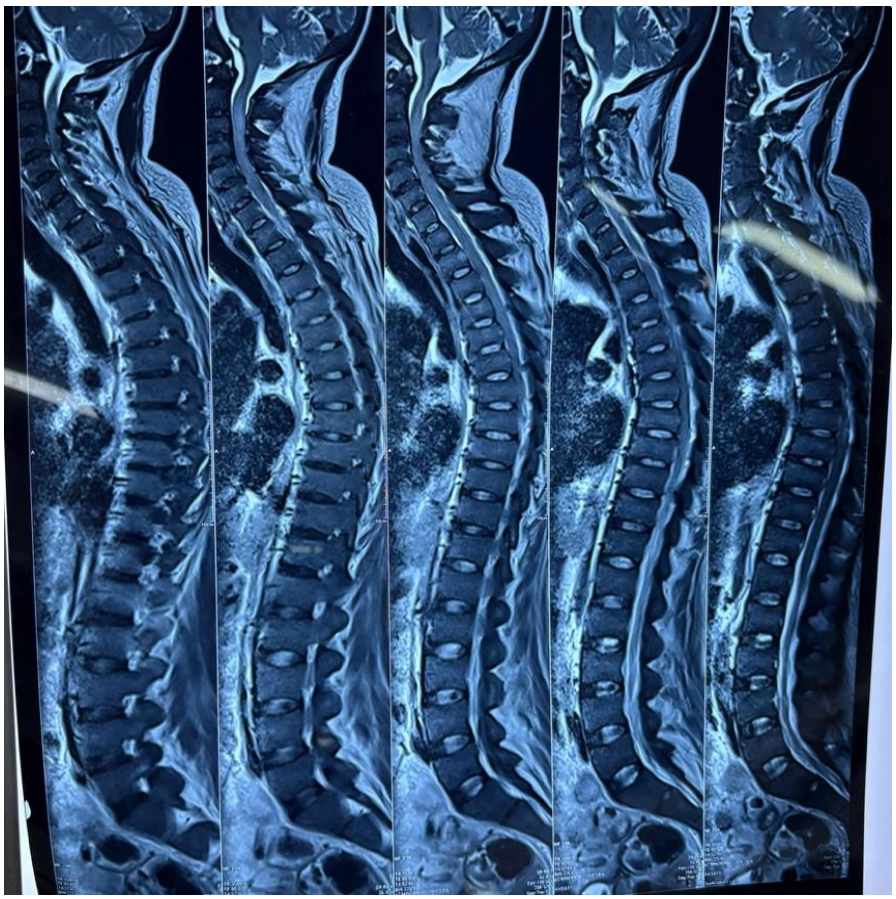
CASE PRESENTATION

A 44-year-old diabetic male presented with progressive asymmetric weakness over six months. Weakness began in the left upper limb, followed by the right upper limb, and then involved the left lower limb. The patient had a prior traumatic amputation of the right lower limb and used a prosthetic leg. Over time, he developed urinary retention, incontinence, and constipation. Neurological examination revealed muscle power of 4/5 in both upper limbs and 3/5 in the left lower limb. Deep tendon reflexes were brisk, and the left plantar response was extensor. A well-defined sensory level was noted at the T4 dermatome. No cranial nerve or cerebellar signs were present. MRI of the spine showed a longitudinally extensive T2hyperintense lesion spanning the cervicothoracic spinal cord, without gadolinium enhancement. Visual evoked potentials showed mild bilateral P100 latency prolongation. Cerebrospinal fluid analysis, autoimmune and paraneoplastic panel (ANA, ENA, AQP4, MOG), vasculitis markers, and infective screen (HIV, TB PCR, VDRL) were all negative. Chest imaging and serum ACE levels ruled out sarcoidosis. Biochemical workup revealed a persistently elevated intact parathyroid hormone (iPTH) of 574 pg/mL, with normal serum calcium, phosphorus, and arterial blood gas. Serum 25(OH) vitamin D was significantly low. Ultrasound neck showed no parathyroid enlargement. Bone marrow biopsy was normal. The endocrinology team diagnosed normocalcemic primary hyperparathyroidism in the setting of severe vitamin D deficiency. The patient was treated with intravenous methylprednisolone (1 g daily for 5 days), followed by tapering oral prednisolone, and

concurrent vitamin D and calcitriol supplementation. Significant clinical improvement occurred over the next 10–14 days, including regained lower limb strength and bladder control.

Table 1: Summary of Clinical and Laboratory Investigations Reference Range

Domain	Investigation	Finding	Comment
Hematology	Complete blood count	Within normal limits	
ESR / CRP	Normal		
Biochemistry	Serum Calcium (corrected)	9.2 mg/dL	Normal (8.5–10.5 mg/dL)
Serum Phosphorus	3.6 mg/dL	Normal (2.5–4.5 mg/dL)	
Serum Magnesium	Normal		
Serum Creatinine / Urea	Normal		
Endocrinology	Intact PTH	574 pg/mL	↑ (Reference: 15–65 pg/mL)
25(OH) Vitamin D	7 ng/mL	↓ Severe deficiency (<10 ng/mL)	
ABG (pH, HCO_3^-, anion gap)	Normal	No metabolic acidosis	
Fasting glucose / HbA1c	Normal / Controlled diabetic		
Immunology	ANA, dsDNA, ENA panel Negative		
AQP4-IgG (NMO), MOG-IgG	Negative		
Paraneoplastic panel Negative Vasculitis workup Negative (ANCA, C3/C4) Infectious Screening HIV, VDRL, HBsAg, TB- Negative			



MRI showing T2 T2WI & STIR images against C2 through C6 vertebrae with mild cord expansion wichout any post contrast enhancement

DISCUSSION

LETM typically raises suspicion for NMOSD or MOGAD. However, when common autoimmune or infectious causes are excluded, rare metabolic contributors should be considered. To our knowledge, no published literature has established a direct link between isolated hyperparathyroidism and transverse myelitis. Parathyroid hormone is known to cross the blood-brain barrier and may influence neuronal excitability and neuroinflammation. Animal studies suggest PTH can modulate microglial activity and cytokine profiles in the CNS. Additionally, vitamin D deficiency is a well-known risk factor in autoimmune demyelination such as multiple sclerosis and NMOSD. In this case, the resolution of symptoms after steroid therapy and vitamin D replacement suggests a parainflammatory or immune-mediated mechanism, potentially triggered or unmasked by metabolic dysfunction. Normocalcemic hyperparathyroidism is often subclinical, and the concurrent vitamin D deficiency may have precipitated a neuroinflammatory cascade. The absence of calcification or structural lesions on MRI further supports a reversible inflammatory etiology. While CNS manifestations in hyperparathyroidism (such as encephalopathy or basal ganglia calcification) have been reported, spinal cord demyelination is exceedingly rare. This case highlights the potential for underrecognized metabolic contributors to LETM and expands the differential diagnosis in seronegative cases.

CONCLUSION

We present a rare case of LETM associated with persistently elevated iPTH and vitamin D deficiency, with normal calcium-phosphorus profile, and complete steroid responsiveness. This case supports including metabolic panels such as vitamin D and parathyroid hormone in the evaluation of seronegative LETM. Correction of such abnormalities, alongside immunotherapy, may contribute significantly to clinical improvement and prevent misdiagnosis.

Ethics and Consent to Participate Declarations
Ethics and Consent to Participate declarations: not applicable.

Competing Interest Declaration
The authors declare that they have no competing interests.

Data Availability Declaration
Data Availability: All data supporting the findings of this study are available within the article. No additional source data are required.

Consent to Publish Declaration
Consent to Publish declaration: not applicable.

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Funding declaration: not applicable.

Declarations

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Short Title: LETM with Hyperparathyroidism

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