Indian Journal of
Neurology

Open Science Publications

Volume 7, Issue 1 - 2026

www.opensciencepublications.com

Paraneoplastic Adenocarcinoma Presenting as Bulbar-Predominant Motor Neuronopathy: A Reversible ALS Mimic in an Elderly Male

Case Report

Fatema Arsiwala[1], Akansha Joshi[1]*, Ashutosh Tiwari[1], Aarohi Doshi[2], Tushar Raut[2] and Jyotsna Oak[3]

[1]Department of General Medicine, Kokilaben Dhirubhai Ambani Hospital, Andheri, Mumbai, Maharashtra, India

[2]Department of Neurology, Kokilaben Dhirubhai Ambani Hospital, Andheri, Mumba Maharashtra, India

[3]Head of Department, General Medicine and Consultant Rheumatologist, Kokilaben Dhirubhai Ambani Hospital, Andheri, Mumbai, Maharashtra, India

***Corresponding author:** Akansha Joshi, Department of General Medicine, Kokilaben Dhirubhai Ambani Hospital Andheri, Mumbai, Maharashtra, India. E-mail Id: akankshajoshi710@gmail.com

Article Information: **Submission:** 07/02/2026; **Accepted:** 03/03/2026; **Published:** 05/03/2026

Abstract

Background: Bulbar-onset amyotrophic lateral sclerosis (ALS) is a fatal neurodegenerative disorder, characteristically non-reversible. In rare instances, paraneoplastic neurological syndromes (PNS) can present with ALS-like features. These immune-mediated conditions may show modest response to immunotherapy but typically require tumor therapy for meaningful recovery.

Case Presentation: A 75-year-old man presented with two months of progressive dysphagia, muscular twitching of the tongue, and weakness. Neurophysiology confirmed motor neuronopathy. PET CT revealed a carotid bifurcation mass and hypermetabolic rectosigmoid thickening. Biopsy of a cervical lymph node demonstrated moderately differentiated adenocarcinoma. Intravenous corticosteroids resulted in only partial improvement, but subsequent targeted radiotherapy to the primary malignancy led to marked and sustained recovery of bulbar and motor function.

Conclusion: This case highlights the diagnostic importance of identifying paraneoplastic motor neuronopathies in atypical, rapidly progressive bulbar presentations. ALS mimics may respond incompletely to corticosteroids but show more substantial benefit with targeted oncologic treatment.

Keywords: Amyotrophic Lateral Sclerosis; Paraneoplastic Neurological Syndrome; Motor Neuronopathy; Adenocarcinoma; Bulbar Dysfunction; Radiotherapy

Introduction

Motor neuron diseases, especially ALS, are progressive neurodegenerative conditions without disease-modifying therapy. Bulbar-onset ALS is characterized by early dysphagia and cranial nerve involvement and typically follows an inexorably downhill course. Crucially, ALS is not responsive to corticosteroids or immunotherapy.

Paraneoplastic neurological syndromes (PNS), though rare, may phenocopy ALS by producing motor neuronopathy in the context of malignancy. These conditions are immunologically mediated and may stabilize or improve following immunomodulation or tumor-directed therapy. We describe a case of adenocarcinoma-associated motor neuronopathy with bulbar predominance that showed only partial steroid responsiveness but much stronger sustained improvement with radiotherapy.

Case Presentation

History

A 75-year-old retired male presented to us with a two-month history of progressive dysphagia (initially to solids, later to liquids), frequent choking, excessive salivation, and weak cough. Over the same period, he developed generalized weakness and imbalance of gait, requiring support.

Two weeks prior to onset of dysphagia, he had a painful right cervical swelling and a dry cough. This was treated with antibiotics providing only temporary relief.

Past medical history: He was a known hypertensive for 10 years, and had recently been diagnosed with type 2 diabetes mellitus. He had no prior malignancy or neurological disorder.

His examination revealed the following:

- Cranial nerves: Left LMN facial palsy, fibrillations in the tongue with mild deviation, reduced gag reflex, intact palatal elevation, muscular fasciculations in the upper back
- Motor: Tone normal; strength – right upper limb 4+/5, left upper limb 5/5, both lower limbs 4+/5.
- Reflexes: Upper limb reflexes 2+, knees 1+, right ankle absent.
- Gait: Broad-based, required assistance. No ataxia or sensory deficits

Investigation findings are outlined in (Table 1).

Table 1: Results of imaging studies and hematological investigations

Investigation	Findings
EMG/NCV	Widespread LMN involvement including bulbar and limb muscles
MRI / MRA	Mass encasing right carotid bifurcation with bilateral ICA atherosclerosis and A2 stenosis
PET-CT	Hypermetabolic thickening of rectosigmoid colon with pericolic fat stranding
Sigmoidoscopy	Non-specific colitis
Cervical node biopsy	Moderately differentiated adenocarcinoma consistent with metastatic GI primary IHC: ● Luminal cells: Ckit, CK7, p40, p63 positive ● Basal cells: S100, Calprotectin positive ● Ki-67 proliferation index: 30%
Immunology / Serology	AChR Antibody: 0.74 (Borderline but non-significant) MuSK: negative ANA: negative ANCA PR3 & MPO: negative ASCA: 73.43 units (elevated) Anti Hu antibody positive
Other	Hemoglobin: 10.6 g/dL WBC: 7800 per microL Platelets: 177000 per microL Sodium: 129 mmol/L Potassium: 3.88 mmol/L CRP: 134 mg/L Creatinine: 0.24 mg/dL ESR 53 mm/hr Urine routine: Protein +, glucose ++ Echocardiogram EF 55%, no structural abnormalities

Table 2: Neurological examination findings

System	Findings
Cranial Nerves	Left LMN facial palsy Tongue fasciculations with mild deviation Reduced gag reflex Intact palatal elevation
Motor	Tone: normal Strength ● Right ○ upper limb 4+/5 ○ lower limb 4+/5 ● Left ○ upper limb 5/5 ○ lower limbs 4+/5
Reflexes	Upper limb reflexes: 2+ Lower Limb reflexes: ● Bilateral Knee reflexes: 1+ ● Right ankle reflex: absent
Sensory	No sensory deficits
Gait	Broad-based gait, required assistance No ataxia

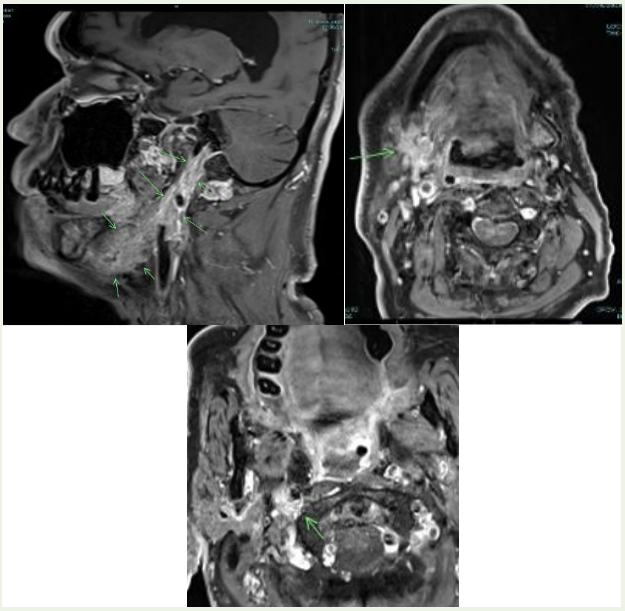

Figure 1: MRI Brain: Mass encasing right carotid bifurcation.

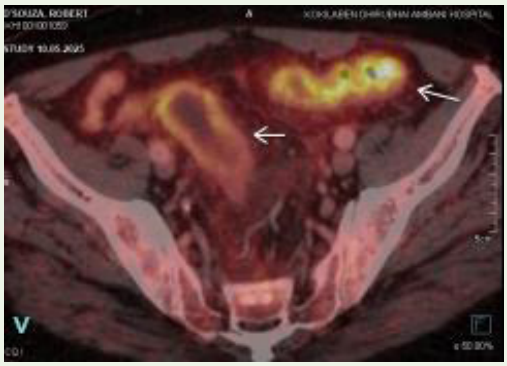

Figure 2: PET CT: Hypermetabolic thickening of rectosigmoid colon.

Citation: Arsiwala F, Joshi A, Tiwari A, Doshi A, Raut R, et al. Paraneoplastic Adenocarcinoma Presenting as Bulbar-Predominant Motor Neuronopathy: A Reversible ALS Mimic in an Elderly Male. Indian J Neurol. 2026;7(1): 163.

Clinical course

Given high aspiration risk, an NG tube was inserted. He was administered intravenous methylprednisolone (1 g/day × 5 days). This led to modest improvement in swallowing and energy, but deficits persisted.

Following confirmation of adenocarcinoma, the patient underwent targeted radiotherapy to the suspected rectosigmoid primary and regional nodes. In the weeks after radiotherapy, his bulbar function, swallowing, and gait improved significantly, and he was able to resume partial oral feeding. This dual pattern suggested an immune-mediated, paraneoplastic motor neuronopathy, partially steroid-responsive but substantially improved only after oncologic treatment.

The patient was followed up for 12 months after completion of radiotherapy. At 3 months post treatment, he demonstrated marked improvement in bulbar function, with significant reduction in dysphagia and aspiration episodes, allowing removal of the nasogastric tube and gradual resumption of oral feeding. Tongue fasciculations diminished, facial weakness improved, and limb strength improved to near-baseline (Medical Research Council grade 5-/5 proximally and distally). By 6 months, he was independently ambulatory without support and had regained functional swallowing with only mild residual fatigue on prolonged speech. At the 12-month follow-up, neurological status remained stable without progression of motor neuron signs. There was no emergence of upper motor neuron features. This sustained neurological stabilization and functional recovery over one year strongly supports a paraneoplastic etiology rather than classical ALS, which would be expected to show relentless progression.

Discussion

Bulbar-onset amyotrophic lateral sclerosis (ALS) is classically characterized by relentless progression, mixed upper and lower motor neuron involvement, and absence of meaningful response to immunotherapy or oncologic treatment. In contrast, paraneoplastic neurological syndromes (PNS) are immune-mediated disorders that may mimic ALS but differ fundamentally in pathophysiology, prognosis, and therapeutic responsiveness.

Several features in this case strongly favored a paraneoplastic motor neuronopathy over classical ALS. The temporal profile was subacute, progressing over weeks rather than months to years. Neurological findings were predominantly lower motor neuron in distribution, without upper motor neuron signs, and remained non-progressive over 12 months of follow-up. Anti-Hu antibody positivity provided serological evidence of a high-risk paraneoplastic antibody associated with malignancy-related neurological syndromes. There was a clear temporal association between neurological onset and the diagnosis of metastatic adenocarcinoma. Most importantly, the patient demonstrated partial responsiveness to corticosteroids and marked, sustained improvement following definitive tumor directed radiotherapy, an outcome incompatible with the natural history of classical ALS.

This clinical trajectory is in keeping with published reports: immunotherapy providing only transient stabilization in paraneoplastic motor neuron disease, [1] partial improvement or stabilization in a subset of patients with immune modulation,[2] resolution of motor neuronopathy following resection of colon adenocarcinoma,[3] and reversible ALS-like presentations where recovery followed treatment of the underlying malignancy.[5,6] Thus, our case confirms that tumor-directed therapy, such as surgery, chemotherapy, or radiotherapy, are often decisive in achieving sustained neurological recovery.

Alternative explanations

Although our patient demonstrated widespread LMN involvement consistent with a paraneoplastic motor neuronopathy, two features, that is the presence of a carotid bifurcation lesion and an LMN facial palsy, could suggest direct tumor infiltration of cranial nerves. Local compressive or infiltrative effects remain an important differential diagnosis in patients with focal adenopathy.

Paraneoplastic etiology remains more likely in our case rather than an isolated local invasion due to the following several points:

1. The distribution of LMN signs extended beyond the cranial territory of the cervical lesion, involving limb muscles and bulbar regions simultaneously.
2. The modest but clear response to corticosteroids suggested an immune-mediated component, which would not be expected with purely infiltrative pathology.
3. Most importantly, the marked and sustained functional recovery after targeted radiotherapy to the primary malignancy and nodal disease is characteristic of paraneoplastic neurological syndromes, in which definitive oncological therapy often results in improvement.

A limitation of this report is the absence of a comprehensive paraneoplastic antibody panel (Hu, CV2, KLHL11, amphiphysin, etc.), which might have provided further serological support. Nonetheless, the clinical trajectory—rapid progression, partial steroid responsiveness, and dramatic oncological treatment response—supports a paraneoplastic etiology over ALS or local infiltration alone.

Conclusion

This case highlights adenocarcinoma-associated paraneoplastic motor neuronopathy mimicking bulbar-onset ALS. Unlike ALS, such cases may show partial steroid responsiveness and more substantial recovery with targeted oncological therapy. Clinicians should suspect PNS in rapidly progressive bulbar syndromes, as timely recognition and combined immune- and tumor-directed therapy may dramatically alter prognosis.

Rapidly progressive bulbar ALS-like presentations in elderly patients should raise suspicion for paraneoplastic syndromes.

Partial response to corticosteroids can occur, but sustained recovery usually requires oncological treatment.

Direct tumor infiltration should be considered, but widespread LMN involvement supports a systemic immune-mediated mechanism.

Citation: Arsiwala F, Joshi A, Tiwari A, Doshi A, Raut R, et al. Paraneoplastic Adenocarcinoma Presenting as Bulbar-Predominant Motor Neuronopathy: A Reversible ALS Mimic in an Elderly Male. Indian J Neurol. 2026;7(1): 163.

Timely recognition can significantly alter prognosis in ALS mimics.

Patient Consent and Ethics

Written informed consent for publication was obtained from the patient's next of kin. Institutional ethics approval was waived as per local policy for single case reports.

References

1. Liu X, Yang H, Liu Q, et al. (2019) Paraneoplastic motor neuron disease: a retrospective analysis of clinical characteristics. Chin Med J (Engl) 132: 1233-1239.
2. Yang Z, HE L, Ren M, Lu Y, Meng H, et al. (2022) Paraneoplastic Amyotrophic Lateral Sclerosis: Case Series and Literature Review. Brain Sci 12: 1053.
3. Yamashita S, Kimura A, Takahashi T, et al. (2007) Paraneoplastic motor neuron disease associated with colon adenocarcinoma. Intern Med 46: 1941-1944.
4. Darnell RB, Posner JB (2003) Paraneoplastic syndromes involving the nervous system. N Engl J Med 349: 1543-1554.
5. Evans BK, Fagan C, Arnold T, Dropcho EJ, Oh SJ (1990) Reversible paraneoplastic motor neuron disease associated with renal carcinoma. Neurology 40: 960-962.
6. Mandel-Brehm C, Dubey D, Kryzer TJ, O'Donovan BD, Tran B, et al. (2019) Kelch-like Protein 11 Antibodies in Seminoma-Associated Paraneoplastic Encephalitis. N Engl J Med 381: 47-54.

Citation: Arsiwala F, Joshi A, Tiwari A, Doshi A, Raut R, et al. Paraneoplastic Adenocarcinoma Presenting as Bulbar-Predominant Motor Neuronopathy: A Reversible ALS Mimic in an Elderly Male. Indian J Neurol. 2026;7(1): 163.