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Not All That Looks Like ALS Is ALS: Spectrum of Amyotrophic Lateral Sclerosis Mimics at a Tertiary Neuromuscular Center

ABSTRACT

Background: Amyotrophic lateral sclerosis (ALS) is a progressive motor neuron disorder diagnosed clinically in the absence of a definitive biomarker. Several neuromuscular and structural disorders can closely mimic ALS, leading to diagnostic errors with significant prognostic and therapeutic implications.

Objective: To analyze the spectrum, clinical characteristics, and key predictors of ALS mimic disorders among patients referred with suspected ALS at a tertiary neuromuscular center.

Methods: This retrospective observational study reviewed approximately 500 patients referred with a provisional diagnosis of ALS between January 2021 and December 2025. Clinical data, electrophysiology, neuroimaging, and relevant laboratory investigations were analyzed. Fifty-one patients with confirmed ALS mimic diagnoses and complete clinical data were included. Comparative analyses with published ALS epidemiologic parameters were performed using Student's t-test and chi-square/Fisher's exact tests. Multivariate logistic regression was used to identify predictors of ALS mimic diagnosis.

Results: The most frequent ALS mimics were Hirayama disease (29.4%), post-polio syndrome (23.5%), and adult-onset spinal muscular atrophy (23.5%). Patients with ALS mimics were significantly younger at symptom onset than reported ALS cohorts (19.1 ± 3.6 vs 58.7 ± 11.3 years; $p < 0.001$). Bulbar onset was absent in the mimic cohort ($p < 0.001$). Conduction block on electrophysiology and structural spinal abnormalities on MRI were significantly associated with mimic diagnoses. Multivariate analysis identified age <40 years, absence of bulbar onset, conduction block, and MRI structural abnormalities as independent predictors of ALS mimic disorders.

Conclusion: ALS mimics represent a substantial proportion of patients referred with suspected motor neuron disease. Recognition of clinical red flags, careful electrophysiological interpretation, and routine spinal imaging are essential to avoid misdiagnosis and identify treatable conditions.

Key words: Amyotrophic lateral sclerosis, ALS mimics, misdiagnosis, motor neuron disease

INTRODUCTION

Amyotrophic lateral sclerosis (ALS) is a relentlessly progressive neurodegenerative disorder characterized by degeneration of upper and lower motor neurons, leading to weakness, muscle wasting, bulbar dysfunction, and respiratory failure.[1] Despite well-established diagnostic criteria, ALS remains a clinical diagnosis with the help of El Escorial and Gold Coast criteria, as no single biomarker reliably confirms or excludes the disease.[2,3]

A wide range of neurological and systemic disorders may present with motor weakness resembling ALS, particularly in early stages. These conditions, collectively termed ALS mimic syndromes, differ markedly in prognosis and management and include inflammatory neuropathies, myopathies, hereditary motor neuron disorders, neuromuscular junction disorders, structural spinal pathologies, and metabolic conditions.4,5 Population-based studies suggest that 6–10% of patients initially suspected to have ALS are ultimately diagnosed with a non-ALS condition, many of which are treatable.[4]

Misdiagnosis of ALS carries profound consequences, including psychological distress, inappropriate prognostication, premature end-of-life counseling, and exclusion from disease-specific therapies for reversible disorders.6 Careful attention to clinical red flags, supported by targeted electrophysiology and imaging, can reliably distinguish ALS from its mimics.

This study analyzes the spectrum, frequency, and clinical characteristics of ALS mimics encountered at a tertiary neuromuscular referral center and highlights practical red flags to aid early and accurate diagnosis.

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METHODOLOGY

This retrospective observational study was conducted at the Department of Neurology, Bombay Hospital and Medical Research Centre, Mumbai, over five years from January 2021 to December 2025. Medical records of patients referred with a provisional diagnosis of amyotrophic lateral sclerosis (ALS) were systematically reviewed. Patient records tagged as suspected ALS were screened during the study period. Patients aged ≥18 years with complete clinical documentation and electrophysiological evaluation were included. Patients with incomplete clinical data or insufficient diagnostic evaluation were excluded. Among the screened cohort, patients with complete clinical and investigative data who were ultimately identified as ALS mimic disorders were included for detailed analysis.

Sample size estimation was performed using the single-proportion formula ($n = Z^2pq/d^2$), assuming an expected ALS mimic prevalence of 10%, a 95% confidence level, and an absolute precision of 11%, yielding a minimum required sample size of 60 patients. The final sample met this requirement.

Demographic characteristics, referral details, clinical features (including pattern of weakness, upper and lower motor neuron signs, bulbar involvement, sensory symptoms, pain, fatigability, and disease course), and results of diagnostic investigations were extracted from the medical records. Investigations included electromyography and nerve conduction studies, MRI of the brain and spine, relevant laboratory testing, genetic testing, and neuromuscular junction studies where clinically indicated. Final diagnoses were established by experienced neuromuscular neurologists based on clinical, electrophysiological, radiological, and genetic findings.

Statistical analysis was performed using descriptive and inferential methods. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, and categorical variables were expressed as frequencies and percentages. Comparative analyses between the ALS mimic cohort and published epidemiologic parameters of ALS were performed using Student's t-test for continuous variables and chi-square or Fisher's exact tests for categorical variables. Variables demonstrating clinical relevance were entered into a multivariate logistic regression model to identify independent predictors of ALS mimic diagnosis. A p value <0.05 was considered statistically significant.

OBSERVATIONS AND RESULTS

A total of 51 patients with complete clinical and investigative data were included in the final analysis from an initial cohort referred with a provisional diagnosis of amyotrophic lateral sclerosis (ALS). The cohort showed a marked male predominance with 43 males (84.3%) and 8 females (15.7%), yielding a male-to-female ratio of 5.4:1. The mean age at presentation was 37.0 ± 19.1 years (median 40 years, range 2.5–78 years), while the mean age at symptom onset was 19.1 ± 3.6 years. Upper-limb–predominant weakness was the most common presenting pattern, observed in 25 patients (49.0%), followed by lower-limb involvement in 15 patients (29.4%), and combined upper- and lower-limb weakness in 10 patients (19.6%).

The spectrum of ALS mimic disorders identified in the cohort included Hirayama disease (n = 15, 29.4%), post-polio syndrome (n = 12, 23.5%), and adult-onset spinal muscular atrophy (n = 12, 23.5%), which together constituted most cases. Less frequent diagnoses included multifocal motor neuropathy with conduction block (n = 4, 7.8%), ventral cervical cerebrospinal fluid collection due to duropathy (n = 3, 5.9%), inclusion body myositis (n = 3, 5.9%), and spinal bulbar muscular atrophy (n = 2, 3.9%). Figure 1 demonstrates the spectrum of ALS mimics in our cohort

When compared with published epidemiologic data of ALS, patients with ALS mimics were significantly younger at symptom onset (19.1 ± 3.6 vs 58.7 ± 11.3 years; Student's t-test, p <0.001). Bulbar onset was not observed in this cohort, whereas approximately 25–30% of ALS cases present with bulbar symptoms at onset, a highly significant difference (Fisher's exact test, p <0.001).

Electrophysiological and radiological abnormalities provided additional clues to alternative diagnoses. Conduction block on nerve conduction studies was identified in 4 patients (7.8%), a finding rarely seen in ALS (Fisher's exact test, p = 0.04). Structural abnormalities on MRI, including dynamic cervical cord compression and ventral cerebrospinal fluid collections, were observed in 7 patients (13.7%) and were significantly associated with ALS mimic diagnoses (chi-square test, p = 0.006). Sensory involvement was uncommon in this cohort and did not significantly distinguish ALS mimics from ALS populations (p = 0.21).

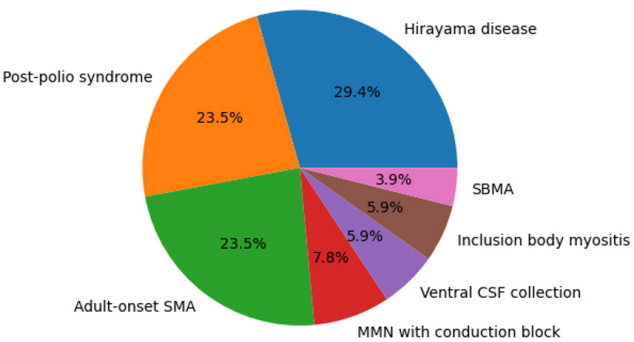

Figure 1: The spectrum of ALS mimics in the cohort (n=51)

Multivariate logistic regression analysis identified several independent predictors of ALS mimic diagnosis. Age <40 years emerged as the strongest predictor (OR 17.2, 95% CI 5.8–51.4, p <0.001), followed by absence of bulbar onset

(OR 8.9, 95% CI 3.0–26.3, p <0.001). Conduction block on electrophysiology (OR 6.8, 95% CI 1.3–33.1, p = 0.02) and structural abnormalities on MRI (OR 5.4, 95% CI 1.6–17.8, p = 0.006) were additional independent predictors. Sensory involvement was not included in the multivariate model due to lack of variability within the cohort. Figure 2 represents several independent predictors after multivariate logistic regression analysis.

Predictor	Odds Ratio	95% CI
Age <40 years	17.2	5.8–51.4
Absence of bulbar onset	8.9	3.0–26.3
Conduction block	6.8	1.3–33.1
MRI structural abnormality	5.4	1.6–17.8

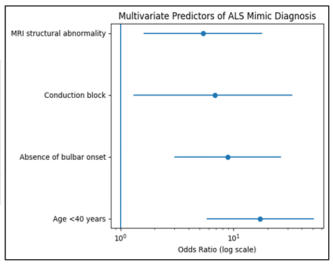

Figure 2: Several independent predictors after multivariate logistic regression analysis.

DISCUSSION

The findings of this cohort highlight several key clinical features that distinguish ALS mimic disorders from ALS. Patients with ALS mimics were significantly younger and lacked bulbar onset, both of which are well-recognised red flags reported in previous studies of ALS mimics. Like prior series, focal anterior horn cell disorders and hereditary motor neuron diseases constituted most mimics, with Hirayama disease, post-polio syndrome, and spinal muscular atrophy accounting for most cases in our cohort.[7,8] Previous literature has also emphasised the diagnostic value of electrophysiological findings such as conduction block and structural abnormalities on spinal imaging in identifying treatable ALS mimics. Our results are consistent with these observations, demonstrating that younger age at onset, absence of bulbar involvement, conduction block, and structural MRI abnormalities independently predicted ALS mimic diagnoses.[7] Interestingly, sensory involvement—often considered a useful distinguishing feature—was uncommon in our cohort, suggesting that many ALS mimics may present as pure motor syndromes and highlighting the importance of careful pattern recognition and targeted investigations in differentiating these disorders from ALS.[4,5]

Hirayama Disease (n=15, 29.4%)

In our clinical series, Hirayama disease emerged as an important mimic of lower motor neuron–predominant motor neuron disease, particularly in young individuals presenting with distal upper limb weakness. Most patients were young males in the second to third decades of life, with a clear male predominance. The typical clinical presentation consisted of insidious onset weakness and wasting of the distal upper limb muscles, most commonly affecting the C7–T1 myotomes. In many cases the weakness began unilaterally and later involved the contralateral hand to a milder degree.[9] Tremor of the fingers during action was a frequent accompanying feature and often drew attention to the problem early in the course. Sensory examination was uniformly normal, and lower limb involvement was absent. In several patients, relative sparing of the brachioradialis muscle was noted, producing the characteristic pattern of distal forearm wasting. The tempo of progression was usually slow and tended to stabilize over time, which further distinguished these patients from classical amyotrophic lateral sclerosis.

Electrophysiological studies consistently demonstrated chronic motor axon loss confined to the lower cervical segments, most commonly C7, C8, and T1, without evidence of generalized anterior horn cell involvement or sensory nerve abnormalities.[10] Magnetic resonance imaging of the cervical spine played a decisive diagnostic role. Neutral-position imaging was sometimes unrevealing, but flexion MRI demonstrated the typical features of Hirayama disease in most patients, including forward displacement of the posterior dural sac and widening of the posterior epidural space in the lower cervical region, often accompanied by focal lower cervical cord flattening or anterior horn signal changes.[11] Recognition of this characteristic clinical–radiological pattern is critical, as Hirayama disease is a benign and potentially stabilizing condition that can closely resemble early motor neuron disease but has a distinctly different prognosis and management strategy.

Red Flags – How It Differs from ALS

Key distinguishing features from ALS include:

- young age at onset,
- focal distal upper limb involvement,
- absence of upper motor neuron signs,
- lack of bulbar or respiratory involvement, and
- early disease stabilization.

The presence of characteristic flexion MRI findings and a benign, non-progressive course strongly favor Hirayama disease over ALS.

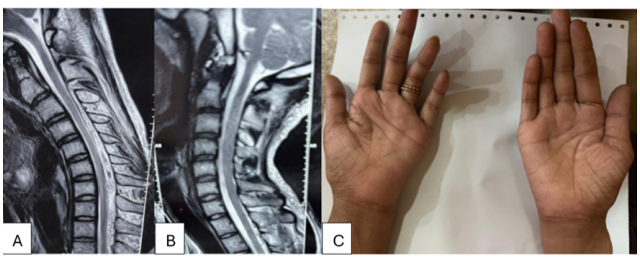

Figure 3: Cervical spine MRI representing dynamic compression and flattening of the spinal cord in flexion view (A) in Hirayama disease, secondary to anterior displacement of the posterior dura which is relieved on extension view (B). (C) is demonstrating the reverse split sign seen in Hirayama disease with atrophy of Hypothenar muscles instead of thenar muscles,

Spinal Muscular Atrophy (n=12, 23.5%)

Among the ALS mimic disorders identified in our cohort, hereditary lower motor neuron syndromes consistent with spinal muscular atrophy (SMA) constituted an important subgroup. The clinical profiles observed in these patients—characterized by pure lower motor neuron involvement, absence of upper motor neuron signs, and a slowly progressive or static course—are well recognized features that distinguish hereditary motor neuron disorders from classical ALS.[12] Although several patients initially presented with patterns of weakness that raised concern for motor neuron disease, the absence of bulbar involvement, the presence of longstanding symptoms, and electrophysiological evidence of chronic anterior horn cell degeneration without widespread active denervation helped differentiate these conditions from ALS.

Genetic confirmation of SMN1 deletions in a subset of patients further supported the diagnosis of 5q spinal muscular atrophy, while other cases likely represented related hereditary lower motor neuron syndromes.[13] These findings underscore the importance of considering hereditary motor neuron disorders in the differential diagnosis of suspected ALS, particularly in younger patients or those with atypical disease trajectories. Recognition of these conditions has become increasingly important in the modern era, as disease-modifying therapies targeting SMN protein deficiency have significantly altered the prognosis of SMA, making early and accurate diagnosis critical.[14]

Red Flags – How It Differs from ALS

Key distinguishing features from ALS include:

- Younger age at onset with very slow progression
- Pure lower motor neuron involvement without upper motor neuron signs
- Symmetric proximal muscle weakness predominance
- Absence of bulbar, cognitive, or respiratory involvement in most adult cases
- Positive genetic testing confirming SMN1 mutation
- Long survival and non-fatal course

These features, particularly pure LMN involvement with genetic confirmation and slow progression, strongly favor spinal muscular atrophy over ALS.

Post-Polio Syndrome (n = 12; 23.5%)

In our clinical series, post-poliomyelitis motor neuron syndromes represented an important diagnostic consideration in patients presenting with late-life lower motor neuron weakness. Most patients had a clear history of paralytic poliomyelitis in early childhood, followed by a long period of clinical stability extending over several decades. New neurological symptoms appeared typically in the fourth to seventh decades of life and consisted predominantly of progressive weakness, fatigue, and functional decline affecting previously involved limb muscles. The weakness was usually asymmetric and localized to the lower limbs, although in some patients, additional neurological disorders such as radiculopathy, polyneuropathy, or cerebrovascular disease complicated the clinical picture. Bulbar symptoms and clear upper motor neuron signs were generally absent.[15]

Electrophysiological studies consistently demonstrated very chronic anterior horn cell degeneration with large motor unit potentials and reduced recruitment, reflecting longstanding reinnervation following the original poliomyelitis. In most cases, the abnormalities were electrically inactive, without the widespread active denervation typical of amyotrophic lateral sclerosis. Neuroimaging was mainly useful in excluding structural causes such as spinal compression. These findings highlight that progressive weakness occurring decades after poliomyelitis often reflects post-polio motor neuron decline rather than a primary degenerative motor neuron disease. However, rarely, ALS may develop independently in individuals with prior poliomyelitis.[16]

Red Flags – How It Differs from ALS

Key distinguishing features from ALS include:

- Definite past history of paralytic poliomyelitis
- Very long interval (decades) between initial illness and new weakness
- Weakness confined to previously affected muscles
- Prominent fatigue and musculoskeletal pain
- Absence of upper motor neuron signs
- Much slower progression and relatively benign prognosis

A clear history of prior poliomyelitis and segmental lower motor neuron weakness with prolonged stability strongly favor post-polio syndrome over ALS.

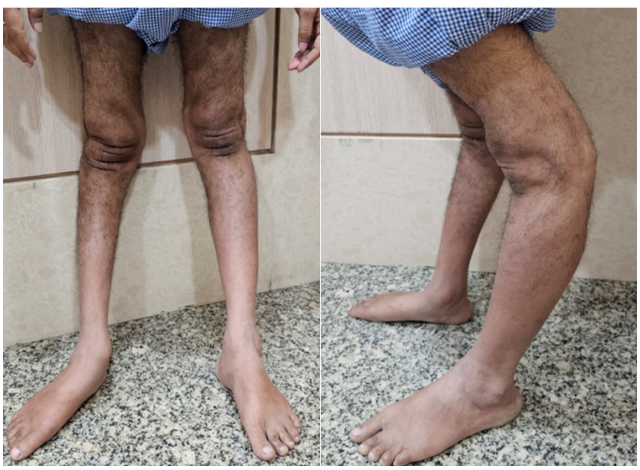

Figure 4: Clinical photograph showing asymmetric lower limb muscle wasting and weakness in a patient with post-polio syndrome

Multifocal Motor Neuropathy with Conduction Block (n = 4, 7.8%)

Multifocal motor neuropathy (MMN) represented another important ALS mimic in our cohort. This immune-mediated motor neuropathy typically presents with asymmetric distal limb weakness without sensory involvement, a pattern that

can closely resemble lower motor neuron–predominant ALS. However, several clinical and electrophysiological features help distinguish MMN from motor neuron disease. The absence of upper motor neuron signs, the presence of motor conduction block on nerve conduction studies, and occasional supportive findings such as anti-GM1 antibodies or focal nerve enlargement on imaging strongly favor MMN over ALS. Recognition of this entity is particularly important in clinical practice because, unlike ALS, MMN is potentially treatable, with many patients demonstrating significant clinical improvement following intravenous immunoglobulin (IVIg) therapy. Early identification of MMN therefore has major therapeutic implications and underscores the importance of careful electrophysiological evaluation in patients presenting with focal lower motor neuron syndromes suggestive of ALS.[17–19]

Red Flags – How It Differs from ALS

Key distinguishing features from ALS include:

- Asymmetric weakness in individual peripheral nerve distribution
- Presence of motor conduction block on nerve conduction studies
- Absence of upper motor neuron signs
- No bulbar or respiratory involvement
- Normal sensory examination despite peripheral nerve pathology
- Marked clinical improvement with IVIg therapy

The presence of conduction block and treatment responsiveness strongly favors MMN over ALS, making it one of the most important and treatable ALS mimics.

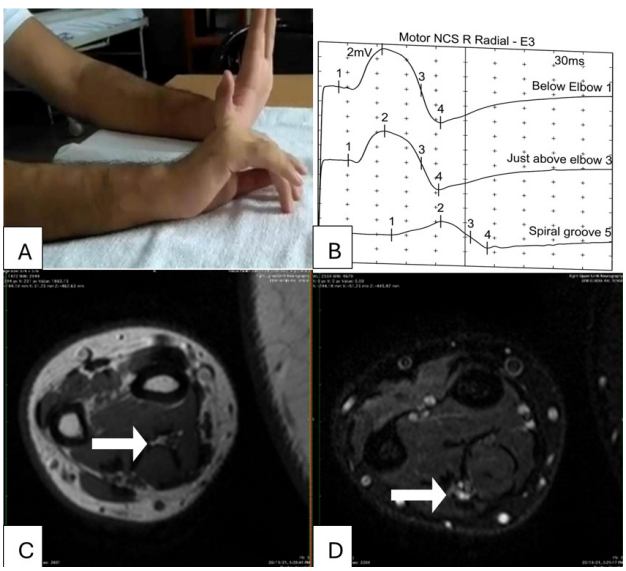

Figure 5: (A) - Showing right wrist drop and (B) - Conduction block across the spiral groove segment of the right radial nerve, affecting the extensor indicis muscle. (C and D) Showing T1 and STIR axial sections of right forearm demonstrating Radial nerve thickening and enhancement in a patient with right MMNCB

Ventral Longitudinal Intraspinal Fluid Collection (VLISFC) / Ventral CSF Fluid Collection (n = 3; 5.9%)

Ventral cervical cerebrospinal fluid (CSF) collections represent an underrecognized structural cause of lower motor neuron syndromes that can closely mimic ALS. These lesions are thought to arise from dural defects or spinal CSF leaks, resulting in ventral epidural CSF accumulation and chronic compression or stretching of the cervical spinal cord and nerve roots. The resulting dysfunction of anterior horn cells across multiple cervical segments can produce a clinical picture of bibrachial weakness or cervical-predominant motor involvement that may initially resemble motor neuron disease. However, several features help distinguish these conditions from ALS, including segmental involvement confined to the cervical region, absence of upper motor neuron signs, and characteristic structural abnormalities on spinal MRI. Recognition of this entity is particularly important because it represents a potentially treatable cause of motor neuron dysfunction, with stabilization or improvement reported following surgical repair of the dural defect or management of the underlying CSF leak. These observations emphasize the need for careful spinal imaging in patients presenting with atypical lower motor neuron syndromes, as structural duropathies may masquerade as ALS in clinical practice.[20,21]

Red Flags – How It Differs from ALS

Key distinguishing features from ALS include:

- Structural abnormality visible on spinal MRI
- Predominantly bibrachial lower motor neuron weakness
- Symptoms of intracranial hypotension (orthostatic headache)
- Absence of progressive generalized spread typical of ALS
- Potential stabilization or improvement after surgical repair

The presence of a ventral epidural CSF collection on MRI and potential reversibility strongly favors VLISFC over ALS, highlighting the importance of spinal imaging in suspected motor neuron disease.

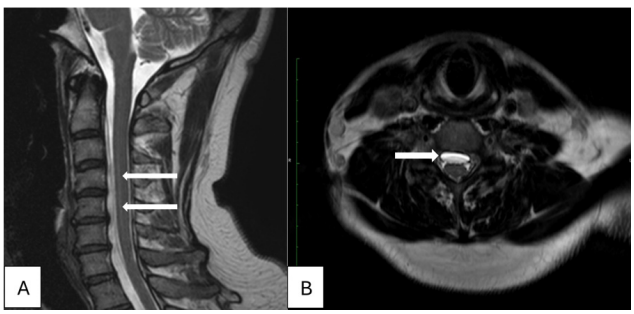

Figure 6: Ventral cervical cerebrospinal fluid (CSF) collection causing anterior spinal cord compression. (A) Sagittal T2-weighted MRI of the cervical spine demonstrating a ventral epidural CSF collection (arrows) displacing the spinal cord posteriorly. (B) Axial T2-weighted image confirming the ventral CSF collection anterior to the spinal cord (arrow), consistent with a duropathy-associated ventral CSF leak, a structural cause of lower motor neuron syndrome that may mimic ALS

Inclusion Body Myositis (n = 3; 5.9%)

Inclusion body myositis (IBM) represented another important ALS mimic within our cohort, particularly in older patients presenting with progressive limb weakness. The characteristic pattern of preferential involvement of the quadriceps and deep finger flexor muscles is a key clinical feature that helps distinguish IBM from motor neuron disease. However, early in the course, the presence of asymmetric weakness, muscle wasting, and occasional neurogenic features on electrophysiology may lead to diagnostic confusion with ALS. Additional clues favoring IBM include mildly elevated creatine kinase levels, dysphagia, and the selective pattern of muscle involvement, which contrasts with the more diffuse motor neuron degeneration seen in ALS. Definitive diagnosis relies on muscle biopsy demonstrating endomysial inflammation and rimmed vacuoles or inclusion bodies, which confirm the myopathic nature of the disorder. Recognition of IBM as a potential ALS mimic is clinically important, as the disease follows a slowly progressive course with preserved upper motor neuron function, and its management and prognosis differ substantially from those of ALS.[22,23]

Red Flags – How It Differs from ALS

Key distinguishing features from ALS includes:

- Predominant involvement of finger flexors and quadriceps (selective pattern)
- Myopathic rather than neurogenic pathology on biopsy
- Mild CK elevation
- Absence of upper motor neuron signs
- Very slow progression over many years
- Dysphagia without widespread LMN and UMN involvement

The presence of selective muscle involvement and confirmatory muscle biopsy findings strongly favors inclusion body myositis over ALS.

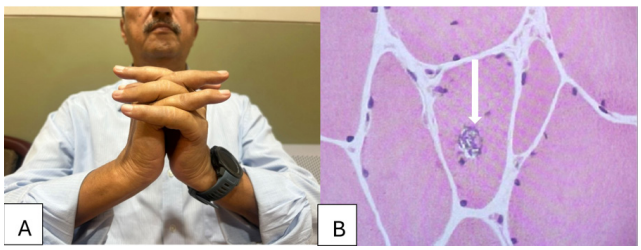

Figure 7: (A) Demonstration of finger flexor weakness. The patient is asked to flex the fingers and grip the opposite hand tightly; weakness of the finger flexors is evident, a characteristic clinical finding in inclusion body myositis. (B) Muscle biopsy (H&E stain) showing a rimmed vacuole (arrow) within a muscle fiber, a histopathological hallmark of inclusion body myositis.

Spinal Bulbar Muscular Atrophy (Kennedy Disease) (n = 2; 3.9%)

Kennedy syndrome, or spinal and bulbar muscular atrophy (SBMA), represented a rare but important hereditary motor neuron disorder that may mimic ALS in our cohort. This X-linked condition typically presents with slowly progressive lower motor neuron weakness involving bulbar and limb muscles, often leading to initial diagnostic consideration of motor neuron disease. However, several clinical features help distinguish SBMA from ALS. The presence of endocrine manifestations such as gynecomastia, testicular atrophy, or other signs of androgen insensitivity, along with sensory nerve involvement on electrophysiological studies, strongly favors SBMA. In contrast to ALS, the disease course is slowly progressive with preservation of upper motor neuron function, and genetic testing demonstrating CAG repeat expansion in the androgen receptor gene provides definitive confirmation. Recognition of Kennedy syndrome as an ALS mimic is clinically important because it carries a more benign prognosis and distinct genetic implications, including the need for appropriate counseling and family screening.[24,25]

Red Flags – How It Differs from ALS

Key distinguishing features from ALS include:

- X-linked inheritance affecting only males
- Presence of sensory neuropathy
- Endocrine features such as gynecomastia and testicular atrophy
- Pure lower motor neuron involvement without upper motor neuron signs
- Very slow progression with prolonged survival
- Genetic confirmation of androgen receptor mutation

The presence of sensory involvement and endocrine abnormalities strongly favors SBMA over ALS, making genetic testing essential in suspected cases.

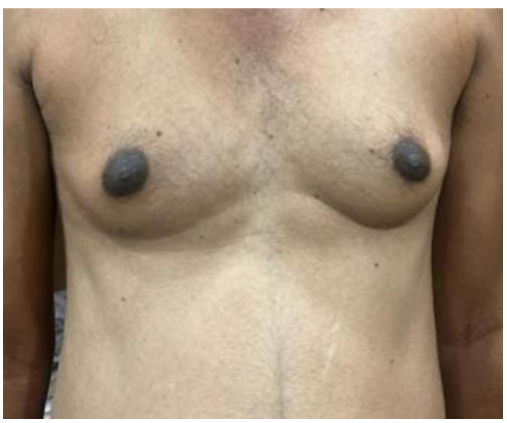

Figure 7: Patient with SBMA having Gynecomastia

CONCLUSION

ALS mimics constituted a significant proportion of patients referred with suspected motor neuron disease at our tertiary neuromuscular center. Hirayama disease, hereditary motor neuron disorders, and post-polio syndrome were the most common mimics encountered.

Recognition of clinical red flags, careful electrophysiological interpretation, and routine use of spinal imaging are essential to avoid misdiagnosis. Importantly, a substantial number of ALS mimics are potentially treatable or have a benign course, underscoring the need for systematic evaluation before confirming a diagnosis of ALS. Table 1 and 2 summarize the red flags and features associated with ALS mimics.

Table 1: Clinical red flags favoring ALS mimic diagnosis

Red flag feature	**Diagnostic implication**
Young age of onset	Suggests hereditary or structural disorder
Sensory involvement	Excludes classic ALS
Fluctuating or stepwise progression	Suggests NMJ or inflammatory etiology
Slow progression with early plateau	Seen in Hirayama disease
Pure LMN phenotype	Consider SMA, MMN, post-polio
Conduction block on NCS	Suggestive of MMN
Elevated serum creatine kinase	Suggests myopathy
Structural abnormality on MRI spine	Indicates compressive or CSF-related pathology
Endocrine features (gynecomastia)	Suggests SBMA
Past history of poliomyelitis	Suggests post-polio syndrome

Table 2: ALS mimics: clinical profile, red flags, and learning points

ALS mimic	**n (%)**	**Key clinical features**	**Key red flags**	**One-line learning point**
Hirayama disease	15 (29.4)	Distal upper limb weakness, young males, slow progression	Young age, distal weakness, flexion MRI changes	Always obtain flexion MRI in young patients with distal upper limb amyotrophy
Spinal muscular atrophy	12 (23.5)	Slowly progressive LMN weakness, familial	Pure LMN signs, early onset	Pure LMN syndromes are not ALS until genetics are excluded
Post-polio syndrome	12 (23.5)	Late worsening weakness after poliomyelitis	Remote polio history	Past poliomyelitis history prevents ALS misdiagnosis
MMNCB	4 (7.8)	Asymmetric distal weakness	Conduction block on NCS	Conduction block excludes ALS
Ventral CSF collection	3 (5.9)	LMN-predominant cervical weakness	MRI ventral CSF	Structural spine disease must be ruled out
Inclusion body myositis	3 (5.9)	Finger flexor, quadriceps weakness	Selective muscle involvement, ↑CK	Patterned weakness suggests myopathy
SBMA	2 (3.9)	Bulbar symptoms, tremor	Gynecomastia, sensory involvement	Endocrine signs exclude ALS

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