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Case Report

Diagnostic and Progression Particularities of a Patient with Amyotrophic Lateral Sclerosis (Flail Arm Variant): A Case Report

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Abstract: Amyotrophic lateral sclerosis (ALS) is a progressive neurodegenerative disorder characterized by degeneration of both upper and lower motor neurons, resulting in progressive muscle weakness, atrophy and functional decline. We present the case of a 69-year-old female patient with ALS, flail arm variant, who initially developed progressive weakness of the upper limbs in 2021, followed by dysarthria and dysphagia during disease progression. Neurological examination revealed marked bilateral upper limb amyotrophy, fasciculations, abolished upper-limb tendon reflexes and a positive Babinski sign on the left side. Electromyographic studies demonstrated chronic diffuse denervation and neurogenic changes consistent with motor neuron disease. Magnetic resonance imaging of the brain and cervical spine excluded alternative structural causes, while laboratory investigations did not identify other relevant abnormalities. Based on clinical findings and electrophysiological evidence, the diagnosis of ALS was established according to accepted diagnostic criteria. The patient received multidisciplinary management, including riluzole therapy, physical rehabilitation, kinesiotherapy and supportive care. This case highlights the diagnostic challenges associated with the flail arm variant of ALS and emphasizes the importance of early recognition, comprehensive electrophysiological evaluation, and multidisciplinary management in maintaining functional status and quality of life.

Keywords: amyotrophic lateral sclerosis, flail arm syndrome, motor neuron disease, electromyography, rehabilitation

1. Introduction

Amyotrophic lateral sclerosis (ALS), first described by Jean-Marie Charcot in 1869, is a progressive neurodegenerative disorder characterized by degeneration of both upper and lower motor neurons (UMN and LMN) [1].

The onset of the disease occurs in adulthood, most commonly through localized motor deficit in the limbs, predominantly distal, which spreads as the disease progresses. Symptoms usually appear after the age of 40 and become more pronounced as

patients age, with some patients also developing dementia, Parkinson's disease, or other neurodegenerative disorders. The progression of the disease is fatal, with death usually occurring approximately 3 years (on average 2-5 years) after diagnosis, the main cause of death being respiratory failure [1,2].

ALS is diagnosed clinically, but also based on diagnostic investigations, especially electromyography (EMG). The most commonly used are the El Escorial criteria and their revised variants (Airlie House, 1998; Awaji, 2008).

The essential clinical elements for diagnosis include:

1. Signs of upper motor neuron (UMN) involvement:
- osteotendinous hyperreflexia
- clonus
- Babinski/Hoffman sign
- spasticity
2. Signs of lower motor neuron (LMN) involvement:
- muscle weakness
- muscle atrophy
- fasciculations
- EMG: fibrillation potentials, fasciculations, high-amplitude motor units.

Symptoms must be progressive and not explained by another disease.

The affected anatomical regions are: the bulbar area (dysarthria, dysphagia, tongue fasciculations); the cervical area (upper limbs), the thoracic area (trunk), and the lumbosacral area (lower limbs) [3].

The classification according to the El Escorial criteria (1998) is as follows:

1. Definite ALS - UMN+LMN signs in at least three regions
2. Probable ALS - UMN+LMN signs in at least two regions, with the UMN rostral to the LMN
3. Probable ALS (supported by EMG): EMG denervation trace in at least two regions + clinical signs of UMN
4. Possible ALS: UMN+LMN in a single region or only UMN in at least 2 regions
5. Suspected ALS (rarely used) - limited signs that do not meet the above criteria.

The Awaji criteria (2008) consider EMG signs of denervation equivalent to clinical signs of LMN involvement, increasing the sensitivity of the diagnosis, especially in the bulbar and early forms [3, 4].

The most recent diagnostic criteria for ALS are known as the Gold Coast Criteria (GCC), proposed as a simplified and more sensitive alternative to the revised El Escorial and Awaji criteria:

- Progressive motor deficit, documented by history or repeated assessments, after a period of normal motor function;
- Signs of UMN or LMN involvement in one body region or
- Signs of LMN involvement in at least two regions (if UMN is not clearly present in all cases).

Other important elements in the diagnosis of ALS are represented by diagnostic investigations: EMG, nerve conduction studies, Magnetic Resonance Imaging (MRI), used especially to exclude other diagnoses. Genetic biomarkers may play an increasingly important role in the future, but are not included in all guidelines [5, 6].

Currently, there is no cure for ALS, but some drugs (riluzole, edaravone) can slow the progression of the disease. Physical and occupational therapy, speech therapy, and essential respiratory and nutritional support are also crucial to prolonging the life of these patients. Psychological support and the presence of a multidisciplinary team play a crucial role in the care of these patients. There are ongoing studies on gene therapies, stem cells, and immunotherapy [7].

2. Case Presentation

A 69-year-old woman from an urban area, diagnosed with amyotrophic lateral sclerosis (ALS), flail arm variant, based on neurological and electrophysiological assessments, presented to the outpatient Rehabilitation Clinic with cervical, thoracic and lumbar spine pain, upper-limb motor impairment predominantly affecting the left side, dysarthria and impaired grasping and gait. The patient had a history of progressive bilateral brachial paresis, dysphagia and dysphonia since 2021. Following clinical evaluation, she was admitted to the Department of Physical and rehabilitation medicine for further investigations and specialized rehabilitation management. The patient was hospitalized from March 31 to April 11, 2025, during which she underwent pharmacological treatment and a comprehensive rehabilitation program.

Symptom onset was reported in May 2021 and was characterized by progressive weakness and tremor of both upper limbs, and reduced muscle strength in the neck musculature. The patient subsequently underwent multiple neurological evaluations, two MRI scans of the cervical spine showing multiple disc protrusions at the C4-C7 level, a craniocerebral MRI scan without pathological changes, and two EMG examinations describing lesions suggestive of motor neuron disease.

Electrophysiological studies performed in 2021 demonstrated chronic neurogenic changes including predominantly the upper-limb muscles, characterized by fasciculations, reduced recruitment patterns, and high-amplitude motor unit potentials, without evidence of conduction block or significant sensory nerve involvement. These findings were compatible with motor neuron disease (Table 1).

A follow-up EMG assessment performed in 2025 showed progression of the neurogenic process, with more prominent denervation changes and a reduction in compound muscle action potential amplitudes compared with the initial study. F-waves responses were infrequent, indicating reduced F-wave persistence. Sensory conduction studies remained essentially preserved, while motor conduction velocities did not demonstrated features suggestive of a demyelinating neuropathy (Table 1). The serial electrophysiological findings supported disease progression and were consistent with amyotrophic lateral sclerosis, flail arm variant (Table 1).

Table 1. Summary of electrophysiological studies performed in 2021 and 2025

Parameter	EMG 2021	EMG 2025	Interpretation
Fasciculations	Present	More extensive	Progressive LMN involvement
Neurogenic MUAPs	Present	More pronounced	Chronic denervation/ reinnervation
Recruitment pattern	Reduced	Further reduced	Motor unit loss
CMAP amplitudes	Mildly reduced	Further reduced	Disease progression
Sensory studies	Preserved	Preserved	Against peripheral neuropathy
Conduction block	Absent	Absent	Against multifocal motor neuropathy
Overall conclusion	Motor neuron disease	Progressive ALS pattern	Supports ALS diagnosis

MUAPs- Motor Unit Action Potentials; CMAP- Compound Muscle Action Potential

During the neurological examination, the patient was able to stand and walk only with assistance. Gait was characterized by anterior pelvic tilt and forward flexion of the head. Muscle hypotonia and atrophy were observed in the cervical paravertebral muscles, shoulder girdle muscles, deltoids, biceps, and triceps bilaterally as well as in the thenar and hypothenar eminences. Tenderness was noted on palpation of the paravertebral muscles and on percussion of the cervical, thoracic and lumbar spinous processes.

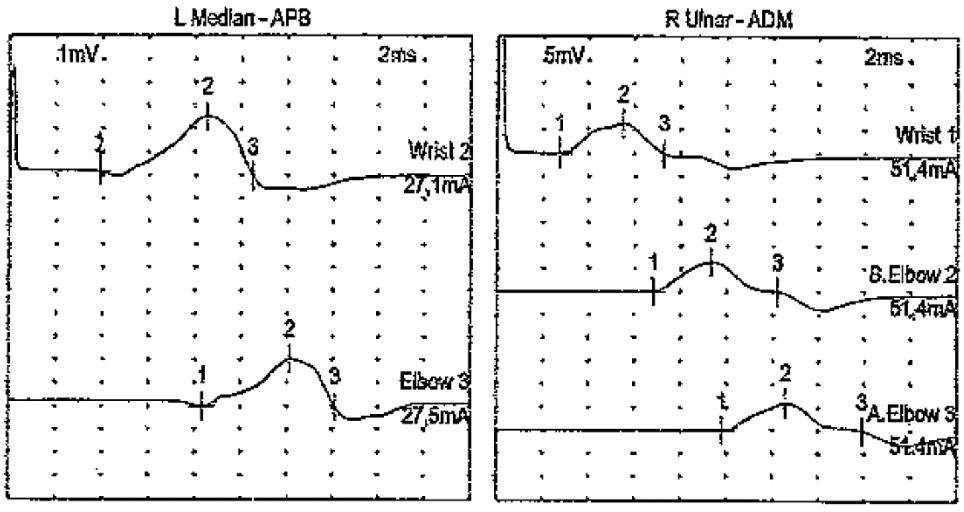
Muscle strength of the arm flexors, abductors, and extensors was graded 3/5 bilaterally, according to the Medical Research Council (MRC) scale, accompanied by reduced handgrip strength. The biceps, triceps and brachioradialis deep tendon reflexes were absent bilaterally. A positive Babinski sign was present on the left side. Coordination testing was limited by severe upper limb weakness. The patient exhibited dysarthria. Sphincter function was preserved. The patient was alert and oriented to time and place.

As far as the laboratory tests are concerned, they had normal values, except for the erythrocyte sedimentation rate, which was 42 mm per hour.

The rheumatological examination did not reveal symptoms specific to connective tissue disease. For the differential diagnosis, biological tests were performed: Lactate Dehydrogenase (LDH), Creatine Kinase (CK), and myositis immunoblot tests, which had normal values.

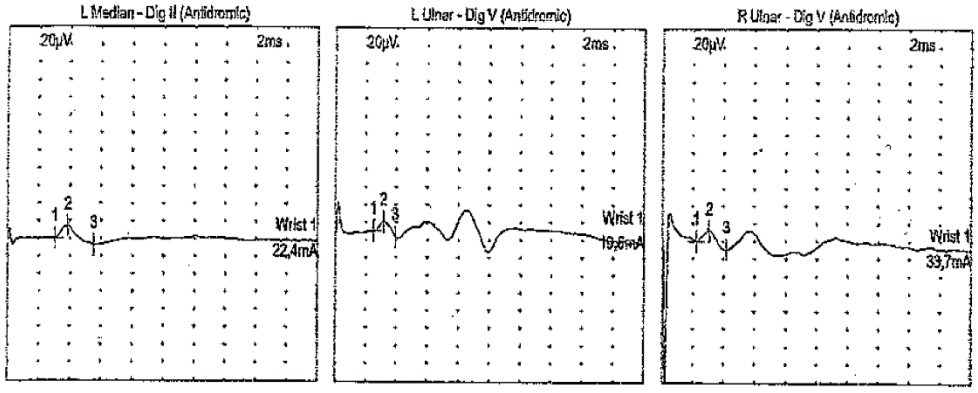
Motor nerve conduction studies demonstrated reduced compound muscle action potential (CMAP) amplitudes, particularly in the median and ulnar nerves, reflecting progressive loss of functional motor units. As illustrated in Figure 1, motor conduction velocities remained relatively preserved and no conduction block was identified, supporting a motor neuron disorder rather than a demyelinating neuropathy.

Figure no 1



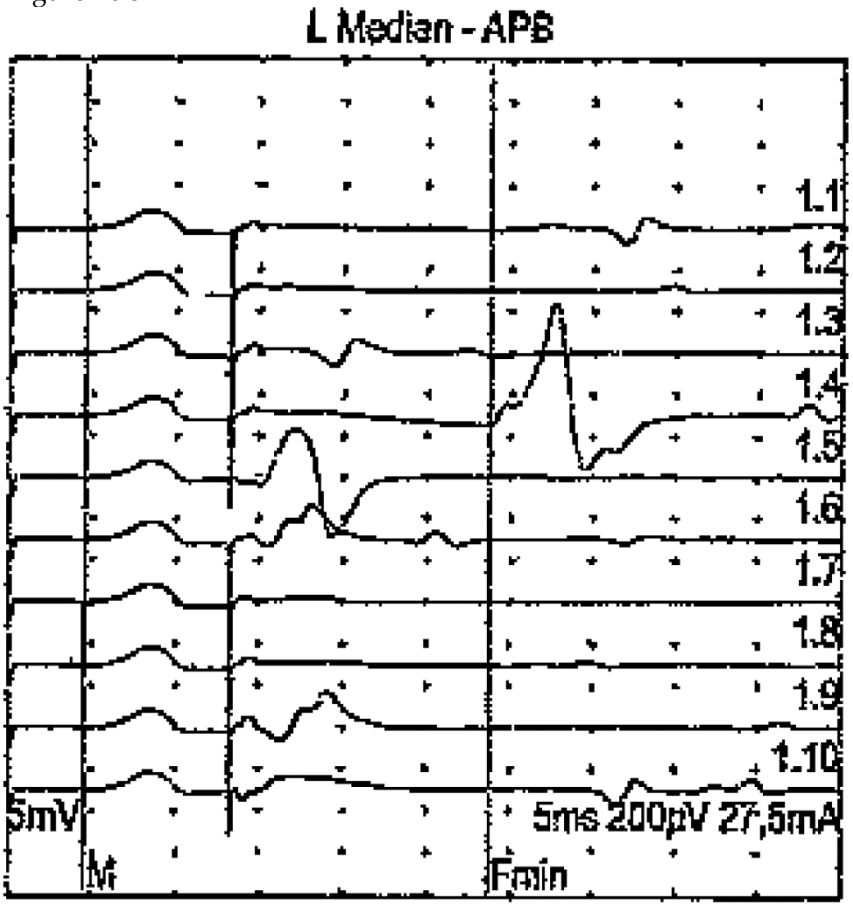
Sensory nerve conduction studies showed sensory nerve action potentials. Figure 2 illustrates representative sensory conduction recordings, which support the absence of clinically significant sensory neuropathy and are consistent with the diagnosis of ALS.

Figure no 2



Repetitive nerve stimulation was performed as part of the electrophysiological assessment. As shown in Figure 3, the findings did not suggest a primary neuromuscular junction disorder and were interpreted in the patient's established motor neuron disease.

Figure no 3



Rehabilitation Management

The patient participated in a 12-day multidisciplinary rehabilitation program during hospitalization. The rehabilitation protocol consisted of daily kinesiotherapy sessions (30-45 minutes, 5 days/week), individualized according to the patient's functional status and fatigue tolerance. The exercise program included active and active-assisted

range of motion exercises for the upper and lower limbs, stretching exercises to prevent contractures, postural training, balance exercises and low-intensity functional training focused on activities of daily living.

Particular attention was paid to energy conservation strategies and avoidance of excessive muscular fatigue. Exercise intensity was maintained at a moderate level, and sessions were interrupted whenever signs of overexertion occurred. Respiratory exercises and breathing control were also incorporated to maintain respiratory muscle function.

Physical therapy modalities were used primarily for symptomatic management. Transcutaneous electrical nerve stimulation (TENS) and interferential current therapy were applied according to standard rehabilitation protocols for pain relief and reduction of cervical and shoulder discomfort associated with muscle weakness and postural imbalance. Neuromuscular electrical stimulation was applied to selected muscle groups with the objective of maintaining muscle trophicity and supporting residual motor activity. Therapeutic massage was performed to reduce muscle discomfort and improve overall well-being.

The rehabilitation objectives were to preserve mobility, maintain functional independence, prevent secondary musculoskeletal complications and improve quality of life.

At discharge, the patient reported a clinically significant reduction in cervical and shoulder pain (VAS score decreased from 6 to 3). Functional independence improved, as reflected by an increase of Barthel Index from 65 to 75 points. Upper-limb muscle strength remained stable during hospitalization, while a modest improvement in shoulder mobility was observed. The patient required less assistance for activities of daily living and reported improved comfort during routine tasks (Table 2). These findings support the beneficial role of rehabilitation in symptom management and functional maintenance in patients with amyotrophic lateral sclerosis.

Table 2. Functional assessment before and after the rehabilitation program

Clinical Parameter	Admission	Discharge
Pain intensity (VAS, 0-10)	7	3
Barthel Index (0-100)	65	75
MRC score- shoulder abductors (right)	3/5	3/5
MRC score- shoulder abductors (left)	3/5	3/5
MRC score- elbow flexors (right)	4/5	4/5
MRC score- elbow flexors (left)	4/5	4/5
Active shoulder flexion (right)	90^0	100^0
Active shoulder flexion (left)	95^0	105^0
Walking ability	Independent	Independent
Activities of daily living	Partial assistance required	Reduced assistance required

VAS- Visual Analogue Scale; MRC- Medical Research Council Muscle Strength Scale.

3. Discussion

This case illustrates the diagnostic evolution of a patient with the flail arm variant of amyotrophic lateral sclerosis, a regional phenotype characterized by predominant proximal upper-limb weakness and atrophy [8,9]. The patient initially presented with

progressive bilateral brachial weakness, cervical pain, dysarthria, and dysphagia, resulting in multiple neurological evaluations and repeated investigations over several years. The clinical presentation required exclusion of alternative diagnoses, including cervical myelopathy, inflammatory neuromuscular disorders, and other motor neuron disease mimics [10,11]. The final diagnoses of ALS was supported by the combination of upper and lower motor neuron signs and characteristic electrophysiological findings [12].

A particularly instructive aspect of this case is the availability of serial electromyographic assessments performed over an extended period. The initial studies demonstrated chronic neurogenic changes, while follow-up examinations documented progressive denervation and reduction of motor response amplitudes. The preservation of sensory conduction studies and the absence of conduction block supported a motor neuron disorder rather than a peripheral neuropathy [13,14,15]. These longitudinal findings provided objective evidence of disease progression and contributed to diagnostic confidence.

The flail arm variant accounts for a minority of ALS cases and is generally associated with a slower progression than classical ALS [16,17]. Although no disease-modifying treatment can halt disease progression, multidisciplinary management remains essential [12,18]. In the present case, rehabilitation interventions, including kinesiotherapy, physical therapy, and supportive care, contributed to pain control, maintenance of mobility and preservation of functional independence. Although rehabilitation does not alter the neurodegenerative process in ALS, it remains an essential component of patient management. In the present case, the rehabilitation program focused on symptom control, preservation of mobility, prevention of contractures and maintenance of functional independence. The combination of kinesiotherapy, therapeutic massage, TENS, interferential current therapy and neuromuscular electrical stimulation contributed to improved comfort and daily functioning despite ongoing disease progression.

This case highlights the importance of early recognition of regional ALS phenotypes, serial electrophysiological monitoring and comprehensive rehabilitation strategies in the long-term management of affected patients.

The educational value of this case does not reside solely in the identification of the flail arm phenotype, which is a recognized ALS variant, but in the prolonged diagnostic evolution and longitudinal clinical follow-up. The patient initially presented with symptoms that could be attributed to several neuromuscular and cervical spine disorders, resulting in repeated investigations and serial electrophysiological assessments over a four-year period.

4. Conclusions

Despite all current studies and research, the prognosis for ALS is reserved, with an average survival time of 3-5 years from the onset of symptoms. However, there are cases with a slower progression, particularly forms with isolated UMN or LMN involvement. Factors associated with a better prognosis include: spinal onset, younger age, good response to non-invasive ventilation, and adequate nutrition. In conclusion, ALS is diagnosed clinically and with EMG, after excluding diseases that can mimic it. Multidisciplinary management is essential for maintaining quality of life and prolonging survival.

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Tables and figure legends

Table 1. Summary of electrophysiological studies performed in 2021 and 2025

Table 2. Functional assessment before and after the rehabilitation program

Figure 1. Representative motor nerve conduction studies performed during follow-up evaluation. Motor conduction velocities were largely preserved, without evidence of conduction block or electrophysiological features suggestive of demyelinating neuropathy. Progressive reduction in compound muscle action potential amplitudes was observed, reflecting ongoing motor neuron loss and denervation consistent with ALS.

Figure 2. Sensory nerve conduction studies of the median and ulnar nerves. Sensory responses were largely preserved, supporting the absence of a clinically significant sensory neuropathy and favoring a motor neuron disorder.

Figure 3. Repetitive nerve stimulation study of the left median nerve. The findings were interpreted in the context of motor neuron disease and were not suggestive of a primary neuromuscular junction disorder. The overall electrophysiological evaluation supported the diagnosis of ALS.

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