

Bilateral Finger Drop: A Rare Presentation of Amyotrophic Lateral Sclerosis

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Abstract: *The clinical hallmark of Amyotrophic lateral sclerosis (ALS) is the combination of upper and lower motor neuron signs. Finger drop is an uncommon sign, characterized by severe weakness of finger extensors with relatively preserved power in finger flexors, wrist extensors and wrist flexors. It has been described in posterior interosseous neuropathies, cervical radiculopathy, Guillain-Barre' Syndrome, multifocal motor neuropathy and myasthenia gravis. Here we describe bilateral Finger drop as a presentation of ALS in a young male.*

Keywords: finger drop, amyotrophic lateral sclerosis

1. Introduction

Finger drop is a clinical entity characterized by selective weakness of finger extensors with preserved wrist extension and flexion. It has been commonly associated with posterior interosseous neuropathies, cervical radiculopathy Guillain-Barre' Syndrome (GBS), and rarely in multifocal motor neuropathy (MMN) and myasthenia gravis.¹⁻³ We describe bilateral Finger drop as an uncommon presentation of Amyotrophic Lateral Sclerosis (ALS) in a young male.

2. Case

A 22 years old male presented with insidious onset gradually progressive weakness of both hands for three years. He first noticed difficulty in straightening his right index finger while playing carrom-board. Slowly he started experiencing similar difficulty with other fingers, which progressed to complete inability to straighten all his fingers, however there was no difficulty in performing fine work (breaking chapaties, buttoning) and he could make a fist and grip objects strongly. Over next few days he also noticed similar complaints in the left hand. He also noticed thinning of both forearms and hollowing of his hands. There was no history suggestive of weakness in proximal upper limbs, neck muscles, lower limbs, cranial nerves or bladder bowel involvement. He denied worsening in cold, fluctuations, twitching, neck pain, radiating pains in limbs, parasthesias or any sensory loss. There was no history of exposure to toxins/heavy metals, drug intake or illicit substance abuse.

General physical and systemic examination was normal. Examination of the cranial nerves was normal except brisk jaw jerk and gag reflex. Motor system examination revealed

wasting of bilateral forearms (both dorsal and ventral aspects), hypothenar muscles and dorsal interossei, but there were no fasciculations observed. There was prominent bilateral finger drop with fingers flexed at metacarpophalangeal (MCP) joints and interphalangeal (IP) joints and very minimal extension possible (Fig 1). Muscle tone in upper and lower limbs was normal. The periscapular muscles, shoulders fixators, elbow flexors, forearm supinators were strong. Elbow extension (MRC 3/5), wrist extension (MRC 4/5), wrist flexion (4-/5), finger extensors (MRC 2/5) were weak. Bilateral thenar and hypothenar muscles were all weak, but fingers flexors (FDS and FDP), and lumbricals were strong. Lower limb, neck and truncal muscles were all strong. Deep tendon jerks were all brisk, finger flexor reflex was present with bilateral flexor plantar response. Sensory and cerebellar examination was normal.

The pattern of involvement in our patient did not conform to involvement of any particular nerve or root distribution. In view of pure motor weakness, with significant wasting and brisk deep tendon jerks in the wasted segments, a clinical possibility of Amyotrophic Lateral Sclerosis was considered.

Routine hematological and biochemical parameters were normal. His HIV 1 and 2 were negative by ELISA. CSF was clear in appearance, and showed 5 cells, all lymphocyte, sugar and proteins were normal. IgM to GM1 antibodies was negative. Conduction studies showed normal CMAP, distal latencies and conduction velocities in bilateral Median, Ulnar, Common Peroneal and Posterior Tibial Nerves. Radial CMAP amplitudes were reduced bilaterally with evidence of conduction block and preserved distal latencies and velocities. Electromyography results are shown in Table 1.

Table 1:

Muscle	Insertional activity	Spontaneous Activity	MUAP Duration	Phases	Amplitude	Recruitment
Lt Triceps	Normal	Fasciculations	increased	normal	increased	Incomplete
Lt Brachioradialis	Normal	Fibrillations +	increased	Polyphasia	increased	Incomplete
Lt Ext Carpii Radialis Longus	Normal	Fasciculations	increased	normal	increased	Incomplete
Lt Ext Carpii ulnaris	Normal	Fasciculations Fibrillations +	increased	Polyphasia	increased	Incomplete
Lt TA	Normal	nil	normal	normal	normal	complete
LT Quadriceps	Normal	nil	normal	Polyphasia	increased	incomplete
RT FDI	Normal	Fasciculations	normal	normal	increased	incomplete
Lt Deltoid	Normal	nil	normal	normal	increased	incomplete
Rt TA	Normal	nil	normal	normal	increased	incomplete

MRI brain did not show any structural abnormality. MRI Cervical spine showed bilateral T2W hyperintensities involving the corticospinal tracts suggestive of Amyotrophic Lateral Sclerosis (Fig 2a,2b). Thus, based on the presence of both upper and lower motor neuron signs (clinical and EMG) in cervical segments and clinical upper motor neuron signs with lower motor neuron signs on EMG in lumbosacral segments, our patient was diagnosed as clinically probable lab supported ALS (El Escorial Criteria).

3. Discussion

Amyotrophic lateral sclerosis (ALS) is a clinically heterogeneous degenerative disease of the motor neurons characterized by combination of upper and lower motor neuron signs. The peak incidence of the disease is in the 6th and the 7th decade⁴. Most cases are sporadic, familial ALS occurring in 5–10%⁵. The clinical hallmark of ALS is the coexistence of muscle atrophy, weakness, fasciculations, and cramps (caused by lower motor neuron degeneration), together with hyperactive or brisk deep tendon reflexes, pyramidal tract signs, and increased muscle tone (due to corticospinal tract involvement). Muscle weakness usually begins in a focal area asymmetrically, first spreads to contiguous muscles in same region before involving other regions⁶. Onset of weakness is more commonly in the upper limbs than lower limbs. Weakness of hands usually presents as difficulty in turning keys, buttoning, turning knobs and gripping objects. Less common clinical presentation includes pseudoneuritic presentation, monomelic presentation; hemiplegic and flail arm variants⁷. Presentation with finger drop has however not been reported.

Our case had bilateral finger drop, but there was evidence of involvement of hypothenar dorsal interossei and thenar muscles as well. Thus, a selective involvement of posterior interosseous nerve could not be the underlying cause. Monomelic amyotrophy affecting both upper limbs were also considered, but it is a disorder of the lower motor neuron and the MRI picture was suggestive of a degeneration of pyramidal tracts.

Pure motor involvement and the conduction block (> 50% fall in the CMAP amplitude between the distal and proximal stimulation) in bilateral radial nerves in with normal distal latencies and conduction velocities lead to the speculation of MMN, which again is a peripheral nerve disorder. The MRI was suggestive of central involvement, but as it is a treatable disorder, we got the IgM to GM1 evaluated which was negative. This is probably explained by the fact that a

difference in amplitude has been described in patients with ALS and may possibly result from phase cancellation and mild slowing of motor conduction velocity⁶.

The diagnosis of ALS in our patient was based on clinical and electrophysiological findings full filling clinically probable lab supported ALS (El Escorial Criteria)⁸. The other supporting feature in our patient was the presence of symmetric T2W and FLAIR hyperintensities in the anterolateral columns of the spinal cord, an uncommon sign which has been characteristically described in patients with ALS and is due to a due to degeneration of the corticospinal tracts⁹. MRI should be considered in the investigation of suspected cases of ALS.

To the best of our knowledge our case is unique, as there is no case of ALS presenting with Finger Drop as yet reported in the literature.

Conflict of interest: nil

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Images



Figure 1: Bilateral Finger drop with flexion at MCP joints and hyperextension at the wrist.



Figure 2: MRI Cervical Spine (Axial view) T2 image showing hyperintensities involving bilateral corticospinal tracts.