Case Report

Foot Drop: A Rare Post COVID-19 Complication

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ABSTRACT

Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) virus which has caused Coronavirus Disease 2019 (COVID-19) pandemic, may also affect neural axis at different levels including Central Nervous System (CNS) and Peripheral Nervous System (PNS). There is increasing evidence of neurological complications caused by SARS-CoV-2. Though isolated facial nerve involvement has been reported in literature, isolated peroneal nerve palsy secondary to SARS-CoV-2 infection is a rare occurrence. This article reported two such rare cases (53-year-old male/45-year-old male) of foot drop where isolated peroneal nerve involvement was present on nerve conduction study. It was possibly caused by an immune mediated mechanism in patients who had recovered from COVID-19 infection.

Keywords: Coronavirus disease 2019, Peroneal nerve palsy, Peripheral nervous system complication, Severe acute respiratory syndrome coronavirus 2

INTRODUCTION

The COVID-19, pandemic has affected around 65 million cases to date all over the world. This report is about two cases that presented with isolated peroneal nerve palsy which developed after recovery from COVID symptoms. As there was no other predisposing factor in both cases and patient developed symptoms spontaneously, it was hypothesised that the manifestations are possibly late complication of SARS-CoV-2 infection. Also, the patients suffered from foot weakness 7-10 days after recovering from acute primary SARS-CoV-2 infection which suggests possibility of immune mediated focal insult to peroneal nerve. First patient had axonal peroneal nerve injury whereas the second one had demyelinating insult.

Case 1

A 53-year-old male patient presented to Neurology Outpatient Department with right foot weakness for five days duration. He was admitted with cough and fever 25 days back. The Computed Tomography (CT) chest was done and revealed moderate bilateral ground glass opacities suggestive of atypical viral pneumonia (CT Score 13). He was diagnosed with COVID-19 by Reverse Transcription-Polymerase Chain Reaction (RT-PCR). The patient became asymptomatic after treatment in the hospital and was discharged 15 days back. He denied any history of intramuscular injection, trauma or prolonged cross leg sitting posture. There was no family history or similar recurrent episodes.

On examination his vital signs were normal. Higher mental function, cranial nerve including facial and upper limb examination was normal. Bilateral Straight Leg Raising (SLR) was negative. Proximal motor power in both lower limbs was 5/5. Right ankle dorsiflexion and eversion was weak (MRC grade 1/5), right foot invertor motor power was 5/5. Both knee and ankle jerks were present. There was impairment of touch and pin prick sensation in superficial peroneal and deep peroneal nerve distribution. Nerve conduction study showed low amplitude Compound Muscle Action Potential (CMAP), normal latency and Motor Conduction Velocity (MCV) in right peroneal nerve [Table/Fig-1]. Reduced Sensory Nerve Action Potential (SNAP) amplitude was noted in right superficial peroneal nerve. Normal sural SNAP amplitude was noted. Both tibial and left peroneal nerve conduction was normal so what was the final diagnosis?

		Case 1		Case 2		
Nerve conduction parameters			Distal	Proximal	Distal	Proximal
Motor	Peroneal	CMAP	1.4	1.1	8.7	0.8
		Latency	3.5	10	3.85	11.67
		MCV	40.5		11.09	
	Tibial	CMAP	12.2	10.3	13.3	11.2
		Latency	4.3	13.47	4.79	15.1
		MCV	36.48		33.66	
Sensory	Superficial peroneal	SNAP	2.3		39.8	
		Latency	3.1		2.13	
		SNCV	40.5		42.45	
	Sural	SNAP	20.5		18.4	
		Latency	2.1		1.46	
		SNCV	45.2		47.95	
Table (Fig. 4). Nerve conduction percentare						

[Table/Fig-1]: Nerve conduction parameters. CMAP: Compound muscle action potential; MCV: Motor conduction velocity; SNAP: Sensory nerve action potential

The patient was managed with oral prednisolone 1 mg/kg/day in tapering doses for two weeks {patient was given short course of oral prednisolone 60 mg once daily followed by tapering dose by 20 mg every 5th day. (60 mg-40 mg-20 mg-stopped)} and symptomatic treatment. Physiotherapy was done for one month. On follow-up, patient had improved ankle dorsiflexion and eversion power to Medical Research Council (MRC) Scale grade 3/5.

Case 2

A 45-year-old male patient had fever, body ache and headache one month back and he was diagnosed COVID-19 based on RT PCR. His CT Chest was within normal limits. He was managed conservatively and became asymptomatic within seven days. After discharge he was asymptomatic and had no neurological deficit. But 10 days later, he had difficulty in walking and had to drag his left foot. He was treated at primary health centre, but he had no improvement, so he was referred to Out Patient Department of Neurology.

He had no radicular backpain, trauma and had not received intramuscular injection. There was no family history of similar episodes or similar recurrent episodes in past. His neurological examination including higher mental function cranial nerves, upper limbs was unremarkable except ankle dorsiflexion weakness (MRC grade 2/5). His Deep Tendon Reflexes (DTRs) were preserved and plantar were flexor. No sensory deficit was present. Magnetic Resonance Imaging (MRI) brain was normal. His nerve conduction study showed reduced CMAP in left peroneal nerve on stimulation at knee [Table/Fig-1]. Bilateral tibial, right peroneal and both sural nerve conduction studies were within normal limits. The patient had significant complete recovery with tapering dosees of oral prednisone 1 mg/kg/day for 14 days {patient was given short course of oral prednisolone 60 mg once daily followed by tapering dose by 20 mg every 5th day. (60 mg-40 mg-20 mg-stopped)}, symptomatic treatment and physiotherapy.

DISCUSSION

Novel Coronavirus, SARS-CoV-2, pandemic has caused more than 65 million cases and around 1.5 million deaths to date all over the world. The first series was reported from Wuhan, China, in patients with a pneumonia like presentation. From India, 98 lac cases of COVID-19 have been reported and around 1.5 lac deaths have occurred. There is growing evidence that SARS-CoV-19 can also affect the nervous system [1]. Mao L et al., has reported neurological manifestations in 36.4% (78/214) of novel corona virus infection [2]. This was followed by reports of central nervous systems complications, including anosmia, ageusia, headache, seizures, myelitis, stroke, cerebral venous thrombosis, encephalopathy encephalitis and various neuropsychiatric manifestations from all over the world [3,4].

Several mechanisms have been proposed for neurological insult caused by COVID-19. Taste and smell deficits have been reported even in absence of nasal congestion which suggest possibility of direct invasion of olfactory bulb. Direct invasion of SARS-CoV-2 may be caused by binding of SARS-CoV-2 virus to Angiotensin Converting Enzyme 2 (ACE2) receptors which are present abundantly in CNS [1]. However, the studies have not documented virus in cerebrospinal fluid in number of COVID-19 cases with neurological complication or sequelae which might suggest that binding of SARS-CoV-2 to ACE2 receptor may trigger the immunological systems leading to subsequent neuronal damage [3]. Inflammation induced demyelination is another widely accepted hypothesis in cases of Bell's palsy [4].

PNS involvement is being reported frequently in patients of COVID-19 in the literature. Initially, Mao L et al., reported the PNS symptomatology of COVID-19 in the form of impaired taste (5.6%), impaired smell (5.1%), visual disturbances (1.4%), and neuralgia (2.3%) [2]. Zhao et al., Hirayama T et al., and Korem S et al., reported cases of GBS in COVID-19 patients initially and then this was followed by reports of GBS from different parts of world which included both axonal and demyelinating forms of GBS [5-7]. Apart from GBS, Miller Fisher syndrome and polyneuritis craniallis have also been reported in SARS-CoV-2 patients by Gutiérrez-Ortiz C et al. [8]. There are few case reports which have described isolated cranial neuropathies in SARS-CoV-2, especially VIIth cranial nerve palsy [9, 10]. Apart from Guillain Barre Syndrome (GBS), multiple cranial neuropathies, paranasal sinus complications observed in COVID-19 included myalgia and nerve pain, neuro-ophthalmological nerve insult, sensorineural hearing loss [11].

Like the index patients, Nersesjan V et al., had reported one COVID patient who had developed right foot drop and peroneal nerve conduction showed mild affection in their prospective cohort study [12]. Othenin-Girard A et al., also reported a case of mononeuritis multiplex secondary to SARS-CoV-2 involving right facial nerve, intrinsic hand muscle and bilateral peroneal nerve manifesting as facial palsy and bilateral foot drop [13]. Recently, Alkhateeb M et al., had also reported one case of bilateral foot drop in tuberculosis patient with co-infection of COVID-19 who had axonal sensory motor neuropathy for which there were potential contributory factors like drugs, nutrition, tuberculosis etc., apart from COVID-19 infection [14]. Prone position ventilation used to treat Acute Respiratory Distress Syndrome (ARDS) in COVID-19 patients may lead to neurological complications. Malik GR et al., has reported ulnar nerve (28.6%), followed by the radial nerve (14.3%), the sciatic nerve (14.3%), the brachial plexus (9.5%), and the median nerve (9.5%) insult in prone position ventilation for COVID-19 ARDS [15]. But these cases of isolated nerve affection in COVID-19 are clearly secondary to prone positioning and entrapment neuropathy unlike the index cases where there was no such predisposing factor.

In present cases, there was no associated neuro-deficit like above mentioned reports and both patients had isolated peroneal nerve involvement which has not been reported previously. In cases of GBS, both axonal and demyelinating pathology has been reported [7] like in present cases.

CONCLUSION(S)

The literature has been flooded with reports of peripheral nerve involvement in SARS-CoV-2 infection but isolated peroneal nerve involvement in COVID-19 patient is rare. Foot drop in post COVID-19 patient without any predisposition and risk factors suggest probable immunological insult to nerve.

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