Case Report

Electrophysiological Findings in Human Parechovirus-Associated Acute Flaccid Paralysis: A Case Report

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ABSTRACT

Objectives: This paper aims to describe the electrophysiological findings seen in a young boy diagnosed with human parechovirus-associated acute flaccid paralysis (AFP) in the Philippines.
Study design: Case report.
Setting: Hospital.
Subject: This is a case of a 1-year-old Filipino boy who presented with focal flaccid limb weakness following a prodromal illness, which then progressed asymmetrically and with respiratory paralysis. Lumbar puncture results were normal, nasopharyngeal swab showed enteroviral antigens, and human parechovirus (HPeV) was isolated in stool. Whole spine magnetic resonance imaging (MRI) initially demonstrated inflammatory changes in the C4-C6 cord levels, but was normal on repeat testing after five months. Following the set case definition, findings altogether led to diagnosis of enterovirus-related AFP.
Methods: Electromyography (EMG) and nerve conduction study (NCS) was conducted 6 months into the disease course.
Results: NCS of the upper limbs revealed normal sensory nerve action potential parameters. Compound motor action potentials were likewise normal except for apparent conduction blocks on the left proximal median and peroneal nerves. EMG showed active partial denervation and patchy evidence of reinnervation process on the tested limbs. These findings of a diffuse axonal motor neuropathy are compatible with neurophysiological changes seen in limited published studies on enterovirus-associated AFP.
Conclusion: Electrophysiological studies may be clinically useful as part of the multidisciplinary approach in diagnosing enterovirus-associated AFP. To our knowledge, this is the first electrophysiological description of parechovirus-associated AFP in Asia.

Keywords: human parechovirus, acute flaccid paralysis, non-polio enterovirus, nerve conduction study, electromyography

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Introduction

With the marked decline in the incidence of poliovirus worldwide and its eradication in some industrialized countries, Non-Polio Enteroviruses (NPEVs) including Coxsackievirus, Echovirus and newly discovered Enteroviruses, such as Enterovirus D68 (D68V) have become an emerging concern as a suspected cause of acute flaccid paralysis (AFP).[1,2] These viruses are known to have a worldwide prevalence and are associated with multiple clinical presentations including gastroenteritis, respiratory tract infections, aseptic meningitis, fever of unknown origin and flaccid paralysis, among others.[3-10]

Acute flaccid paralysis represents a wide spectrum of neuromuscular diseases, ranging from acute inflammatory motor polyneuropathy to hypo/hyperkalemic paralysis, poliomyelitis and polio-like infections.[7] Despite the consistent utilization of polio vaccine in many parts of the world, sporadic cases of acute paralysis similar to paralytic poliomyelitis are accounted by other enterovirus serotypes. In particular, a new emerging NPEV is being recognized among different countries as a cause of severe neurodevelopmental complications. In the late 2017, public health warnings were issued about a national epidemic of human parechovirus (HPeV) in young children.

The human parechovirus is a single-stranded, RNA virus from the Picornaviridae family, which includes enteroviruses and poliovirus. HPeVs were first isolated in 1956 and classified as enteroviruses (named echoviruses 22 and 23) but was only reclassified into a separate genus in 1996. EV and HPeV are clinically and genetically similar, with differences in the 5' non-translated region. Both viruses replicate in the gastrointestinal tract, are transmitted via the fecal oral route, and shed from the nasopharynx. Nucleic-acid detection, culture, serology, and antigen detection are the available diagnostic techniques. At present, nineteen genotypes (HpeV-1 to HpeV-19) have been identified based on their complete genome or viral protein 1 (VP1) sequences. HPeV1 and HPeV2 have been associated with mild gastrointestinal and respiratory symptoms, while HPeV3 has been associated with more severe clinical manifestations in the form of sepsis-like and CNS illnesses, particularly in neonates and

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infants. HPeV types 4-8 seem to cause disease similar to those associated with HPeV1 and HPeV2 infections. Young children, particularly infants less than 3 months old, are especially susceptible because of an immature immune system.[3, 9, 11-14]

Parechovirus serotypes 1 and 3 are most commonly associated with human disease.[15] HpeV-1 genome caused an outbreak of AFP in Jamaica in 1986 while HPeV3 infection was first associated with AFP in a young girl in 2004.[3,16] There was only one documented case of HpeV in 2017 in a 4-year-old male presenting as flaccid paralysis of both upper and lower limbs in the United Kingdom.[17] In these patients neuroimaging, particularly MRI, showed spinal cord inflammation, root enhancement, and anterior horn cell involvement. Findings similar with parechovirus and poliovirus infections were the presence of long-segment T2 hyperintensity of spinal cord anterior horn cell, nerve root enhancement as well as anterior horn cell enhancement.

There are a few published reports regarding electrophysiological findings observed in patients with HPeV. Electrodiagnostic testing on a 6-year-old boy in Italy diagnosed with HPeV Type 6 (Pellegrinelli et al, 2018) revealed signs of an axonal motor neuropathy mostly affecting the lower limbs.[18] In a case series of twenty-nine patients who had EV-D68–associated acute flaccid myelitis (AFM) in Europe (Knoester et al, 2018), eleven underwent EMG-NCS which generally showed low amplitude compound muscle action potentials most often with normal conduction velocity without signs of sensory nerve conduction abnormalities.[10] In the said case series, EMG done on varying stages of the disease (ranging from 7 days to 3 months) revealed spontaneous muscle fiber activity in the affected muscles. A report on two Norwegian cases (Pfeiffer et al, 2015) exhibited similar findings of motor axonal involvement (reduced motor amplitudes) with sensory nerve sparing parameters on NCS.[19] Of these 2 patients, only one presented with an abnormal needle study consistent with an axonopathic process.

To date, there are no published reports or studies on cases of human parechovirus infection in Asia recorded. Furthermore, electrodiagnostic findings of HPeV have not been thoroughly described, although the clinical as well as radiologic description have been stated as having similarities with poliovirus infection. We describe in this first reported case the electrophysiological findings of HPeV-induced AFP in a young Filipino boy.

Case presentation

This is the case of a previously-well 15-month-old Filipino boy with complete immunization status, who developed fever, lethargy and dyspnea while on a trip to Spain. There he was admitted at the pediatric intensive care unit of a hospital and was admitted for 6 months before being transferred to the Philippines. While confined in Spain, the boy developed progressive respiratory distress prompting eventual mechanical ventilation. Magnetic resonance imaging (MRI) with contrast of the cervical spine cord revealed an inflammation on the C4 to C6 levels. The medical report from the source hospital stated that lumbar puncture results were normal, however the detailed cerebrospinal fluid (CSF) profile or findings were not included in the endorsed document. Nasopharyngeal swab revealed the presence of an enterovirus whereas stool analysis detected parechovirus. While enterovirus and parechovirus are genetically similar hence possibly the reason for the inconsistency in laboratory result reporting, an EV and HPeV coinfection cannot be entirely ruled-out given the limited test reporting. The parechovirus genotype was also not specifically mentioned in the medical report from the hospital in Spain, as with any mention of subtyping performed. A few days after admission, the patient was observed to have focal right upper limb weakness consequently progressing to involve all extremities. Poor oral feeding necessitated gastrostomy tube and button placement. Motor assessment revealed poor neck control and truncal balance with flaccid limbs and no neck stiffness reported. The initial clinical presentation of the patient was consistent with encephalitis and this condition was entertained early, but was ruled out after normal cranial computed tomography (CT) scan and electroencephalography (EEG) results. The features observed in the patient were consistent with the case definition of EV-related AFM.

The child underwent regular rehabilitation sessions (physical, occupational and speech therapies). He could be propped with support on both his wheelchair and an adapted stroller. Therapeutic standing up to one hour was performed in a pediatric inclined plane.

The child was transferred to a tertiary hospital in the Philippines after six months in Spain. EMG-NCS was done within the first month after transfer (Tables 1, 2 and 3). Repeat cervical spine MRI by this time did not show the previously seen spinal cord lesions. This may signify either an absence of active inflammation at the time of neuroimaging, or that the prior cervical cord damage was not permanent and may have resolved.

On the 3rd month of hospital stay, he experienced episodes of desaturation with cyanosis, and suffered a cardiac arrest but was revived and started on antiseizure medications due to post-arrest focal seizures. Video electroencephalogram (VEEG) revealed abnormal interictal electroencephalography (EEG) patterns due to diffuse cerebral dysfunction, while cranial CT scan showed hydrocephalus ex vacuo. The VEEG and cranial CT scan results likely signify neuropathological complications from brain hypoxia. Multiple tests done over the succeeding months documented other co-existing medical conditions such as poor left hemi-diaphragmatic excursion by ultrasound, right hip subluxation with neuromuscular thoracolumbar levoscoliosis by radiographs, and osteopenia by skeletal survey. The presence of levoscoliosis coupled with the immaturity of the hip, lack of weightbearing

Table 1. Sensory nerve conduction study done within the first month after transfer

Nerve	Peak latency (ms)	Amplitude (μV)	Conduction velocity (m/s)
I. Median nerve			
Left median (palm – wrist)	0.5	82.0	71.0
Right median (palm – wrist)	ND	ND	ND
Normal values for age[1]	≤ 0.9	≥ 12.7	49.7 ± 6.4
II. Ulnar nerve			
Left ulnar (5th digit – wrist)	0.5	16.1	60.0
Right ulnar (5th digit – wrist)	ND	ND	ND
Normal values for age[1]	≤ 0.8	≥ 8.5	56.7 ± 4.8
III. Sural nerve			
Left sural	1.1	22.0	44.0
Right sural	ND	ND	ND
Normal values for age[1]	≤ 1.3	≥ 5.1	48.5 ± 8.7
IV. Superficial peroneal nerve			
Left superficial peroneal	1.4	28.0	46.0
Right superficial peroneal	ND	ND	ND
Normal values age[2]	-	21.90 ± 8.58	48.13 ± 6.24

[1]Reference: Jabre JF, Pitt MC, Smith R. Deriving pediatric nerve conduction normal values in the very young (< 3 years). Clin Neurophysiol. 2020;131:177-182

[2]Reference: Verma S, Pochiraju S. Superficial fibular nerve sensory nerve conduction study in children. Muscle Nerve. 2016;54:808-9.

ND, not done due to intravenous attachments

and abnormal muscle forces around the affected hip likely contributed to the asymmetrical (right) hip subluxation. Orthopedic intervention was done through an application of a hip abduction brace for 2 months. Mechanical ventilation was also continued and episodes of desaturation with cyanosis were addressed. His overall condition continuously deteriorated and he ultimately went into cardiopulmonary arrest leading to his demise 2 years after the onset of symptoms.

Electrodiagnostic findings

EMG-NCS was done (with the patient awake) after transfer. Electrophysiological findings demonstrated normal NCS on the left median, left ulnar, left sural and left superficial peroneal nerves (Table 1). Sensory conduction study of the same nerves on the right was not done due to technical difficulties (i.e., multiple intravenous attachments, positional problems).

Compound motor action potential (CMAP) parameters were likewise normal on all the nerves tested (left median, left ulnar, left common peroneal and left tibial nerves) except for reduced amplitudes of the left median nerve and left peroneal nerve by > 20% upon proximal stimulation (Table 2). Motor NCS of the right upper extremity nerves was not done due to technical difficulties. F-wave latencies of the left median, left ulnar and both tibial nerves were within normal limits.

Needle EMG study revealed increased insertional activities with abnormal spontaneous potentials in the form of positive sharp waves (PSW) and fibrillations in the right biceps (musculocutaneous nerve, C5-C6), left gastrocnemius (tibial nerve, S1) and left tibialis anterior (peroneal nerve, L4), indicating an active denervation process (Table 3). These muscles together with the left rectus femoris and right medial gastrocnemius exhibited markedly reduced recruitment with long-duration polyphasic motor unit action potentials (MUAPs).

Discussion

To the authors' best knowledge, this is the first reported case of AFP caused by human parechovirus in a pediatric patient in the Philippines. In the study by Apostol et al in 2012, a total of 790 NPEVs were isolated from the stool specimen of children < 15 years of age presenting with acute flaccid type of paralysis.[1] Only 47 serotypes of NPEV strains were identified using neutralization test and molecular typing, and there was no mention of an HPeV serotype. They concluded that multiple patterns of circulation of plural NPEV serotypes may have existed in the Philippines over 17 years.

The features observed in the patient are consistent with the case definition of EV-related AFM comprised of the following key components: (1) Acute onset of focal limb weakness, (2) MRI showing spinal cord lesion largely restricted to the grey matter spanning 1 or more spinal segments and (3) Detection of EV-D68 in a respiratory, fecal, blood or CSF specimen using a validated Polymerase Chain Reaction (PCR) assay for EVs in general and subsequent sequencing and typing.[10]

This patient's NCS yielded normal sensory transmission and normal motor amplitude values except for attenuated proximal left median nerve and left peroneal nerve amplitudes by > 20% (stimulated supramaximally), which likely signify conduction block due to axonal loss. The left ulnar motor amplitude dipped by 17% upon stimulation at the elbow

Table 2. Motor nerve conduction study done within the first month after transfer

Nerve	Distal latency (ms)	Amplitude (mV)	Conduction velocity (m/s)	F-waves minimal latency (ms)
I. Median nerve				
Left median (wrist)	2.1	4.0	-	17.0
Left median (elbow)	-	2.7	43.0	-
Right median (wrist)	ND	ND	-	ND
Right median (elbow)	-	ND	ND	-
Normal values for age[1]	<2.5	>3.5	≥ 35	≤ 21
II. Ulnar nerve				
Left ulnar (wrist)	2.0	6.3	-	17.2
Left ulnar (elbow)	-	5.2	52.0	-
Left ulnar (above elbow)	-	5.6	63.0	-
Right ulnar (wrist)	ND	ND	-	ND
Right ulnar (elbow)	-	ND	ND	-
Right ulnar (above elbow)	-	ND	ND	-
Normal values for age[1]	< 2.5	≥ 2.5	≥ 40	≤ 17
III. Common peroneal nerve				
Left common peroneal (ankle)	1.9	2.9	-	-
Left common peroneal (knee)	-	2.0	52.0	-
Left common peroneal (above knee)	-	2.5	54.0	-
Right common peroneal (ankle)	2.2	2.1	-	-
Right common peroneal (knee)	-	2.1	45.0	-
Right common peroneal (above knee)	-	ND	ND	-
Normal values for age[1]	≤ 3.5	≥ 1.5	≥ 35	-
IV. Tibial nerve				
Left tibial (ankle)	2.5	9.9	-	24.5
Left tibial (knee)	-	9.7	47.0	-
Right tibial (ankle)	2.4	10.8	-	22.7
Right tibial (knee)	-	11.2	45.0	-
Normal values for age[1]	≤ 3.0	-	≥ 30	≤ 26

[1]Reference: Kang PB. Pediatric nerve conduction studies and EMG. In: Blum AS, Rutkove SB, editos. The Clinical Neurophysiology Primer. Totowa, NJ: Humana Press Inc; 2007: 369-89.
ND, not done due to intravenous attachments

Table 3. Electromyography findings done at a tertiary hospital in the Philippines after transfer

Muscle	Insertional activity	Abnormal spontaneous activity[1]			Motor unit action potentials
		Fibrillations	Fasciculations	PSW	
A. Upper limbs					
Right biceps	Increased	2+	0	2+	No activated motor units, severely reduced recruitment with paucity of motor unit remodeling
B. Lower limbs					
Right gastrocnemius (medial head)	Normal	0	0	0	Reduced recruitment; large motor units with increased duration, polyphasia, and evidence of motor unit remodeling
Left rectus femoris	Normal	0	0	0	
Left tibialis anterior	Increased	2+	0	1+	Severely reduced recruitment; large motor units with increased duration, polyphasia, and evidence of motor unit remodeling
Left gastrocnemius (medial head)	Increased	2+	0	2+	Severely reduced recruitment; large motor units with increased duration, polyphasia, and evidence of motor unit remodeling

PSW, positive sharp waves
[1]Grading of abnormal spontaneous activity: 0 = None; 1+ = Persistent single runs in > 1 second in 2 areas; 2+ = Moderate runs > 1 second in three or more areas; 3+ = many discharges in most muscle regions; 4+ = Continuous discharges in all areas of the muscle

and 11% above the elbow, which likely resulted from a submaximal stimulation at the elbow, and not fulfilling the description for a conduction block. The needle EMG study evoked abnormal insertional activities in the form of abnormal spontaneous potentials (PSW and fibrillations) along with varying degrees of reduced recruitment patterns of increased duration, some polyphasic MUAPs, and paucity of motor units remodeling in the right biceps, left tibialis anterior and left gastrocnemius. EMG findings seemed to demonstrate electrophysiological evidence of a diffuse motor axonal polyneuropathy consistent with what was observed on the study by Pellegrinelli et al.[18] Fibrillation potentials develop as motor axons degenerate, while the appearance of motor unit potential of large amplitude and long duration are indicative of reinnervation as a result of compensatory collateral sprouting thus remodeling of the motor units.[20-22]

The general finding of low CMAP amplitudes observed on children from the case series by Knoester et al,[10] and Pfeiffer et al,[19] was however not observed in our patient. This is surprising since axonal injury within the nerve bundle will typically result in a dropout of axons actually stimulated and consequently a reduction in the amplitude of the motor action potential. Neurogenic MUAP characteristics (such as seen in this case) and attenuated CMAP amplitudes on NCS (not seen in this case) are stereotypical for a motor axonal polyneuropathy, but can also be expected in an anterior horn cell (AHC) involvement. However, an AHC condition seems less likely to be the circumstance here because of the presence of conduction blocks observed in the patient's left median and peroneal nerves using supramaximal stimulation. Furthermore, flaccid paralysis in the background of anterior horn cell loss would be expected to produce much lower CMAP amplitudes (but depending on severity), contrary to the mostly-normal values observed in this patient. It is possible that in this patient, recovery may have begun on an existing motor axonal neuropathy, with the primary mechanism of early recovery being collateral reinnervation, with resolution of conduction block and nerve regeneration occurring later.[20]

While the spinal cord lesion seen on MRI (C4-C6) may explain the fibrillations and PSW seen on the right biceps (a muscle innervated at the corresponding levels affected) and even the conduction block observed on the left proximal median nerve (stimulated supramaximally), it is unlikely to cause MUAP changes seen on the lower limbs. Furthermore, even if cervical transverse myelitis (TM) as a likely sequela was entertained earlier in the course, a thorough investigation seems to discourage this as it appeared that the patient did not manifest with a rapidly progressing paraparesis or bilateral signs and symptoms, had no identifiable sensory level (cannot be tested), and had no CSF pleocytosis, which are all significant clinical features in the diagnostic criteria of TM.

This report has its own limitations. Electrophysiological evaluation was not done earlier in the course of the disease; hence we cannot definitively conclude that the normal CMAP parameters seen in this patient at the time of testing indeed point to signs of ongoing recovery as postulated. Moreover, repeat testing was not done so we cannot objectively determine if the changes identified will improve over time as might be seen in some cases of motor axonal polyneuropathy, or persist indefinitely as seen in patients with AHC involvement. Lastly, only a limited number of muscles were tested (especially for the upper limbs). More sites tested on needle study might provide more information to support our inferences.

Conclusion

The electrophysiological profile of non-poliomyelitis AFP remains poorly defined despite well-established epidemiological and clinical parameters. After extensive literature search, the authors believe that this is the first electrophysiological description of AFP caused by human parechovirus in a pediatric patient in Asia, revealing changes suggestive of a diffuse motor axonal neuropathy. This report illustrates the importance of including electrophysiology as part of the multidisciplinary approach in the diagnosis of enterovirus-induced flaccid paralysis, especially in situations when clinical judgment is inexplicit.

Disclosure

The authors disclose no potential conflicts of interest, including all relevant financial interests in any company or institution that might benefit from the publication.

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