Sars-Cov-2 Associated Acute Sensorimotor Axonal Neuropathy

Sars-Cov-2 İlişkili Akut Sensorimotor Aksonal Nöropati

Abstract:

The World Health Organization (WHO) announced a pandemic in December 2019 due to the SARS-CoV-2. In addition to typical findings, such as fever, fatigue and respiratory system symptoms, other system findings have also been described. In this article, we present a pediatric case, diagnosed with acute sensorimotor axonal neuropathy associated with SARS-CoV-2 to raise the awareness, that patients presenting with atypical symptoms may be infected with SARS-CoV-2.

Özet:

Dünya Sağlık Örgütü 2019 yılının Aralık ayında SARS-CoV-2 virüsüne bağlı pandemi ilan etti. Ateş, yorgunluk ve solunum sistem şikayetlerine ek olarak diğer sistem bulguları da tanımlandı. Bu makalede atipik şikayetlerle gelen hastaların SARS-CoV-2 ile enfekte olmuş olabileceğine dair farkındalığı artırmak amacıyla SARS-CoV-2 ilişkili akut sensorimotor aksonal nöropati tanısı alan bir pediatrik vaka sunulmuştur.

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Introduction

The World Health Organization (WHO) announced a pandemic started with pneumonia cases of unknown etiology, observed in Wuhan, China in December 2019 due to the SARS-CoV-2(1). SARS-CoV-2, a zoonotic virus, spreads very rapidly and has a high mortality rate. It has increased globally, causing a pandemic due to the extraordinary balance between contagiousness and mortality. SARS-CoV-2 isolated from human airway epithelial cells identified as a new member of the coronoviridae family, different from MERS-CoV and SARS-CoV (2).

Pandemic SARS-CoV-2 infection is characterized by fever, fatigue and respiratory system` findings such as cough, sore throat and respiratory distress. Less frequently, clinical symptoms such as loss of the sense of smell and taste, vomiting or diarrhea can be seen. Nervous system involvement is rare. In a study conducted with 214 patients in Wuhan, China; 78 (36,4%) of the patients had neurological symptoms involving the central and peripheral nervous system and musculoskeletal" system. Some of these patients did not have the typical symptoms associated with SARS-CoV-2. They only presented with neurological symptoms (3). In this article, a pediatric case diagnosed with acute motor-sensorial axonal neuropathy associated with SARS-CoV-2 is presented.

Case

A 15-year-old male patient presented to the emergency department with pain, numbness and loss of strength of the lower extremity. There was no remarkable feature in his medical story until a week ago. The patient was admitted to the emergency room with complaints of diarrhea, abdominal pain, loss of appetite and fatigue. He had household transmission. SARS-CoV-2 reverse-transcription polymerase chain reaction (RT-PCR) was positive. He was hemodynamically stable with normal oxygen saturation on room air and normal heart rate. His venous blood gas, white cell counts, renal and liver function parameters and ions were normal. After four days of hospitalization, the patient was discharged, as his symptoms regressed. Three days after the discharge, he presented to the emergency department again with new-onset pain, numbness and loss of strength in the left lower extremity. His vital signs were stable. There was a limitation of the dorsal flexion in the left foot in his neurological examination. The muscle strength examination showed weakness in only one extremity with Weakness of Medical Research Council (MRC) Muscle Scale 4/5 in his left lower limb. Aside from an absent left Achilles reflex, deep tendon reflexes, cranial nerve and sensory examination and laboratory findings were normal.

Spinal magnetic resonance imaging was normal. Electromyography and nerve conduction studies showed axonal type polyneuropathy in the bilateral lower extremities. His cerebrospinal fluid was negative for SARS-CoV-2. Intravenous immunoglobulin (IVIG) treatment was started on the patient. Loss of muscle strength in both lower extremities became prominent until the third day of hospitalization, and bilateral Achilles reflexes could not be obtained on the third day. After five days of IVIG treatment, muscle strength in both lower extremities increased gradually. At the end of two weeks of hospitalization, muscle strength in bilateral lower extremities were evaluated as 5/5 with MRC.

Discussion

Guillain Barre syndrome (GBS) is an immune-associated acute polyradiculopathy disease. The disease occurs on a genetic basis due to environmental factors such as infections, vaccination and aging. Many infectious agents such as Mycoplasma Pneumonia, Campylobacter Jejuni, Cytomegalovirus, Epstein Barr virus, Haemophilus influenza, Hepatitis viruses and Zika virus have been shown in the etiology.

SARS-CoV-2 is a neuro-invasive virus similar to SARS and MERS viruses. Angiotensin Converting Enzyme 2 (ACE2), which is found in the skeletal system, nervous system and many other systems, is thought to be responsible for neuroinvasion via direct and indirect mechanisms (4). Murine models suggest that the presence of spike glycoprotein in addition to hemagglutinin increases the neuroinvasive properties of betacoronavirus (5).

Four pediatric cases were presented in a systematic review of 73 cases, published by Rumeileh et al. in July 2020. These cases were evaluated as the classic sensorimotor variant with the paresis developed following mild classic covid symptoms similar to our case (6).

It is known that the pandemic SARS-CoV-2 is associated with many neurological conditions such as febrile convulsion, infantile spasm, multiple sclerosis, acute demyelinating encephalomyelitis and Guillain Barre syndrome. In addition to systemic complaints, SARS-CoV-2 should be considered in patients presenting with neurological symptoms.

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Nerve/Sites	Rec. Site	Onset Lat ms	Peak Lat ms	NP Amp μV	PP Am μV	Segments	Distance mm	Velocity m/s
R Median- Digit II (Antidromic)								
Wrist	Dig II	2.66	3.39	26.9	45.0	Wrist-Dig II	140	53
R Median- Digit II (Antidromic)								
Wrist	Dig II	2.92	3.75	33.0	56.3	Wrist-Dig II	145	50
R Ulnar- Digit V (Antidromic)								
Wrist	Dig V	2.50	3.28	17.7	36.9	Wrist-Dig V	115	46
L Ulnar- Digit V (Antidromic)								
Wrist	Dig V	2.92	3.65	28.1	18.2	Wrist-Dig V	125	43
R Sural- Ankle (Calf)								
Calf	Ankle	3.54	3.96	12.4	5.5	Calf- Ankle	120	34
L Sural- Ankle (Calf)								
Calf	Ankle	3.49	4.06	15.1	10.2	Calf- Ankle	120	34

Nerve/Sites	Muscle	Latency ms	Amplitude mV	Amp %	Duration ms	Segments	Distance mm	Lat Diff ms	Velocity m/s
R Median- APB									
Wrist	APB	3.23	10.3	100	6.30	Wrist APB	70		
Elbow	APB	6.96	11.7	114	7.03	Elbow-Wrist	225	3.75	60
L Median- APB									
Wrist	APB	3.49	13.1	100	6.35	Wrist APB	70		
Elbow	APB	7.76	12.6	95.9	6.09	Elbow-Wrist	225	4.27	53
R Ulnar- APB									
Wrist	ADM	2.45	8.7	100	7.50	Wrist ADM	70		
Elbow	ADM	6.72	7.3	83.5	7.34	B.Elbow-Wrist	240	4.27	56
L Ulnar- APB									
Wrist	ADM	2.66	6.3	100	6.87	Wrist ADM	70		
Elbow	ADM	6.67	6.0	96.3	6.46	B.Elbow-Wrist	230	4.01	57
L Peroneal-EDB									
Ayak bileği	EDB	4.58	2.0	100	6.51	Ayak bileği-EDB	80		
Fib head	EDB	11.46	2.0	96.5	6.72	Fib head-Ayak bileği	310	6.87	45
Pop fossa	EDB	15.83	1.8	88.7	10.05	Pop fossa-Fib head	140	4.38	32
R Peroneal-EDB									
Ayak bileği	EDB	4.32	3.0	100	6.09	Ayak bileği-EDB	80		
Fib head	EDB	11.93	2.4	80.1	6.56	Fib head-Ayak bileği	305	7.60	40
Pop fossa	EDB	14.74	2.5	83.6	6.61	Pop fossa-Fib head	110	1.81	39
L Tibial- AH									
Ankle	AH	4.90	4.7	100	10.68	Ankle- AH	80		
Pop fossa	AH	13.59	3.5	74.5	13.33	Pop fossa-Ankle	405	8.70	47
R Tibial- AH									
Ankle	AH	5.47	2.7	100	4.90	Ankle- AH	80		
Pop fossa	AH	14.95	2.2	81.6	5.00	Pop fossa-Ankle	400	9.48	42

F Wave

Nerve	F min ms
R Peroneal- EDB	30.99
R Tibial- AH	24.95
L Peroneal- EDB	40.57
L Tibial- AH	35.63