

Shingles Radiculoplexoneuropathy

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Abstract We report on a 59 year old man with background history of hypertension who presented to our clinic with vesicular rash of two days duration extending from the right deltoid region to the dorsum of the fingers. He was treated with Valacyclovir plus oral steroid and Tramadol after a clinical diagnosis of shingles; he represented one week later with worsening pain. At that time the vesicular rash were crusting and dry. He was diagnosed with post herpetic neuralgia and his pain medication dosage was adjusted. He represented again two weeks after the initial appearance of rash with complaints of weakness in the right upper extremity. Physical examination confirmed the weakness in the proximal and distal muscle groups of the right arm associated with weak hand and finger grips. He was then referred for electromyography and nerve conduction studies. The result showed the right ulnar motor response recording at abductor digit minimi (ADM) and first dorsal interosseous (FDI) had prolonged latency, reduced amplitude and slowed conduction velocity. The findings are most consistent with right radiculoplexoneuropathy affecting predominantly lower plexus. (EMG/NCS table). He was referred for physical therapy and regained full functions of the right upper extremity after 6 weeks of therapy. Segmental motor weakness secondary to Varicella Zoster infection is uncommon; as a result, clinicians may not suspect it and it may be confused with Parsonage-Turner syndrome, brachial plexus syndrome, cervical radiculopathy and myelopathy particularly if the rash is not obvious or absent leading to delayed diagnosis and or investigations.

Keywords: shingles, brachial plexopathy, motor weakness, varicella zoster

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1. Introduction

Varicella zoster virus (VZV) is a highly infectious virus that is present world wide and it is an exclusively human virus of the family alpha herpesvirus. VZV causes two clinically distinct infections. Initial infection result in chicken pox, characterized by vesicular eruptions at different stage of development [1,2]. Shingles also known as herpes zoster occurs as a result of reactivation of latent infection of VZV within the dorsal root ganglia and cranial nerve

VZV infection is usually limited to one or more dermatome particularly in immunocompetent host, however involvement of two or more dermatomes have been documented [2]. Post herpetic neuralgia is the most common complication in older patients with resulting prolong debilitating pain without adequate pain control at diagnosis. The prevalence of post herpetic neuralgia in those of over 60 years of age; informed the recommendation of the United States (US) Advisory Committee on Immunization Practices (ACIP) to offer varicella zoster vaccine to clients over 60 years of age, presenting to the clinic for care [3]. VZV (shingles) brachial plexus neuritis and motor weakness are rare complications of shingles in immunocompetent host [1]. In a review of 858 patient with herpes zoster, 0.9% of the patient developed motor neuropathy (uptodate).

Myelitis is a more frequent complication in HIV-infected patients and involves direct spread of VZV from the dorsal root ganglia centrally into the spinal cord [1,2,3]. Several reports describing zoster myelitis in HIV-infected patients in the absence of any rash have documented VZV DNA within spinal cord specimens at autopsy [1,2].

Once the rash of herpes zoster appears, the diagnosis is usually readily apparent. The other main agent to consider in the differential diagnosis is herpes simplex virus. VZV is mainly characterized by a painful sensory prodrome, dermatomal distribution, and lack of prior history of a similar rash. If the patient has had a similar vesicular rash in the same location, then recurrent zosteriform herpes simplex should be considered. Non-infectious etiologies to consider are contact dermatitis and Parsonage –Turner syndrome

2. Case Report

A 59 year old man with background history of hypertension who presented to our clinic with blistering painful vesicular rash of two to three days duration extending from the right deltoid region to the dorsum of the fingers. The rash was not associated with swelling, pruritus or weakness and he had no rash in other parts of the body. He also denies exposure to people with rash

and he was not sure of his vaccination status to Varicella Zoster.

Initial examination showed vesicular skin rash mainly at the back of the right arm, from the deltoid region, extending to the dorsum of the fingers. He was alert and oriented to person, place and time. He had no fever and his vital signs were stable. His motor exam was normal, power was 5/5 proximally and distally bilaterally in the lower and upper extremities. He had allodynia in the C7/C8 distribution, reflexes were 2+ bilaterally and symmetrically in the upper and lower extremities. His laboratory results showed normal electrolytes. His CBC showed WBC of 7.38, normal hemoglobin and platelets and his CRP was <5.0 mg/L. He was diagnosed with Varicella Zoster infection on clinical grounds and started on Valacyclovir for seven days, Tramadol and steroids.

He represented one week later with worsening pain. At this time the vesicular rash were crusting and dry. He was diagnosed with post herpetic neuralgia and started on Gabapentin. His Tramadol dosage was adjusted upward. He represented again two weeks after the initial appearance of rash with complaints of weakness in the right upper extremity. Physical examination confirmed the weakness in the proximal and distal muscle groups of the right arm associated with weak hand and finger grips.

He was then referred to Neurology for electromyography and nerve conduction (EMG/NCV) studies with the result showing right ulnar motor response recording at abductor digit minimi (ADM) and the first dorsal interosseous (FDI) had prolonged latency, reduced amplitude and slowed conduction velocity. The findings are most consistent with right radiculoplexoneuropathy affecting predominantly lower plexus (Table 1 -Table 5).

Table 1. Motor Summary Table

Stim Site	NR	Onset (ms)	O-P Amp (mV)	Neg Area (mVms)	Neg Dur (ms)	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)
Right Median Motor (APB)										
Wrist		4.8	6.5	27.34	7.03	Wrist	APB	4.8	7.0	
Elbow		9.4	6.4	27.69	7.66	Elbow	Wrist	4.6	25.5	55
Right Radial Motor (EIP)										
Forearm		2.7	4.3	22.51	9.69	Forearm	EIP	2.7	7.0	
Elbow		5.5	3.9	18.97	9.53	Elbow	Forearm	2.8	14.0	50
Ab. Elbow		7.7	3.6	17.40	9.06	Ab. Elbow	Elbow	2.2	11.0	50
Spiral Groove		10.0	3.5	16.71	9.53	Spiral Groove	Ab. Elbow	2.3	11.5	50
Right Ulnar Motor (ADM)										
Wrist		4.1	0.6	2.23	8.44	Wrist	ADM	4.1	7.0	
B Elbow		15.9	0.1	0.46	12.19	B Elbow	Wrist	11.8	24.5	21
A Elbow		17.2	0.1	0.53	12.34	A Elbow	B Elbow	1.3	10.0	77
Median Elbow	NR									
Right Ulnar (FDI) Motor (FDI)										
Wrist		4.7	1.8	5.36	5.78	Wrist	FDI	4.7	7.0	
B Elbow		15.3	0.6	3.66	12.50	B Elbow	Wrist	10.6	22.5	21
A Elbow		18.3	0.5	1.87	6.72	A Elbow	B Elbow	3.0	10.0	33
Left Median Motor (APB)										
Wrist		4.4	13.3	49.83	6.41	Wrist	APB	4.4	7.0	
Elbow		9.2	13.0	50.00	6.72	Elbow	Wrist	4.8	26.0	54
Left Radial Motor (EIP)										
Forearm		2.8	4.6	27.84	11.25	Forearm	EIP	2.8	7.0	
Elbow		4.7	4.7	25.64	9.69	Elbow	Forearm	1.9	10.0	53
Ab. Elbow		7.3	4.6	24.24	9.53	Ab. Elbow	Elbow	2.6	14.5	56
Spiral Groove		9.2	4.2	21.12	9.84	Spiral Groove	Ab. Elbow	1.9	11.0	58
Left Ulnar Motor (ADM)										
Wrist		2.5	11.2	37.29	6.56	Wrist	ADM	2.5	7.0	
B Elbow		7.2	10.7	35.94	6.72	B Elbow	Wrist	4.7	24.5	52
A Elbow		9.5	10.4	35.01	6.56	A Elbow	B Elbow	2.3	12.0	52

Table 2. Anti Sensory Summary Table

Stim Site	NR	Peak (ms)	O-P Amp (µV)	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)
Right Median Anti Sensory (2nd Digit)								
Wrist		3.7	15.8	Wrist	2nd Digit	2.6	13.0	50
Right Radial Anti Sensory (Snuffbox)								
Forearm		2.5	10.0	Forearm	Snuffbox	1.8	10.0	56
Right Ulnar Anti Sensory (5th Digit)								
Wrist	NR			Wrist	5th Digit		11.0	
Left Radial Anti Sensory (Snuffbox)								
Forearm		2.2	27.3	Forearm	Snuffbox	1.6	10.0	63

Table 3. Comparison Summary Table

Stim Site	NR	Peak (ms)	O-P Amp (μ V)	Site1	Site2	Delta-P (ms)
Right Median/Ulnar Palm Comparison (Wrist - 8cm)						
Median Palm		2.7	41.0	Median Palm	Ulnar Palm	
Ulnar Palm	NR					
Left Median/Ulnar Palm Comparison (Wrist - 8cm)						
Median Palm		2.5	91.6	Median Palm	Ulnar Palm	0.9
Ulnar Palm		1.6	80.5			

Table 4. F Wave Studies

NR	F-Lat (ms)	L-R F-Lat (ms)
Right Median (Mrks) (Abd Poll Brev)	32.81	

Table 5. EMG

Side	Muscle	Nerve	Root	PSW	Fibs	Fasc	Poly	Amp	Dur	Ins Act	Activa	Recrt	Comment
Right	Deltoid	Axillary	C5-6	Nml	Nml	Nml	None	Nml	Nml	Nml	Nml	Nml	
Right	Triceps	Radial	C6-7-8	Nml	Nml	Nml	None	Nml	Nml	Nml	Nml	Nml	
Right	1stDorInt	Ulnar	C8-T1	3+	2+	Nml	None	Nml	Incr	Incr	SevDecr	SevDecr	
Right	C8 Parasp	Rami	C8	Nml	Nml	Nml	None	Nml	Nml	Incr	Nml	Nml	

3. Discussion

We have described a case of a delayed segmental motor weakness in the right upper extremity secondary to brachial radiculoplexoneuropathy caused by VZV infection in an immunocompetent patient.

Post herpetic neuralgia (PHN) is the most common complication in older patients with resulting prolong debilitating pain even with pain control. The prevalence of post herpetic neuralgia in those of over 60 years with VZV infection, informed the recommendation of the Advisory Committee on Immunization Practices (ACIP) to offer varicella zoster vaccine to clients over 60 years presenting to the clinician office for care [3,4].

Motor neuropathy is not a common complication of Varicella zoster infection in immunocompetent persons. In a review of 858 patient with herpes zoster, only 0.9% of the patient developed motor neuropathy [3]. Other published reports have estimated about 3 percent of all patient with VZV infection developed segmental zoster paresis [5,6,7]. The onset of motor paresis is usually at the time of presentation with rash and pain; but delayed presentation of motor weakness as in our patient has been described [8,9,10,14,15]. In our patient his right upper extremity weakness started approximately two weeks after the onset of the rash.

VZV myelitis is a more frequent complication in HIV-infected patients and involves direct spread of VZV from the dorsal root ganglia centrally into the spinal cord [1,2]. Zoster myelitis in HIV-infected patients in the absence of any rash; usually is associated with documented VZV DNA within spinal cord specimens at autopsy [1]. Our patient is HIV negative and he has no history of malignancy or of being immunocompromised. However there have been several report of recurrent myelopathy caused by VZV in immunocompetent adults [4,5,6,16].

The pathophysiologic mechanism of segmental zoster motor weakness is thought to be secondary to reactivation of VZV within the dorsal root ganglion and direct spread

or extension to adjacent anterior root. [8,11,14]. VZV infection is generally limited to one or more dermatomes particularly in immunocompetent host, however involvement of two or more dermatomes have been documented.

Most patients recover fully without residual weakness after treatment as in this case but there are reports in the literature that recovery may be unsatisfactory in as many as a third of the patients. [10,12].

Choi et al [8] suggested doing an MRI of the brachial plexus in addition to electrophysiological studies; because MR images will more accurately reflect the functional impairment than EMG/NCS. We did not think an MR of the brachial plexus was necessary in our patient, because the EMG/NCS findings were consistent with the clinical diagnosis and the patient was improving with treatment.

4. Conclusion

Segmental motor weakness secondary to Varicella Zoster infection is uncommon; as a result, clinicians may not suspect it and it may be confused with Parsonage-Turner syndrome, brachial plexus syndrome, cervical radiculopathy and myelopathy particularly if the rash is not obvious or absent leading to delayed diagnosis and or investigations.

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