Case Report

Postinfectious (Varicella Zoster) Myositis and Mixed Polyneuropathy

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Abstract

We present a case of a 21-year-old male with weakness of all four limbs after primary episode of chicken pox. Nerve conduction studies and magnetic resonance imaging dorsal spine with whole spine and brain screening confirmed polyneuropathy and myositis, respectively. He responded well to intravenous steroids, with complete reversal of symptoms. This case shows that primary varicella zoster infection is a sufficient stimulus to drive antibody generation and precipitate clinical complications.

Keywords: Mixed polyneuropathy, myositis, varicella zoster

Introduction

Chicken pox is a viral infection which presents with fever and exanthematous rash which is extremely contagious. It is caused by a neurotropic human herpesvirus, the varicella zoster virus. The virus has the quality of remaining latent after the primary infection in the spinal and cranial ganglia and may be reactivated at a later stage in a state of immunocompromise to present as herpes zoster.

Neurological complications caused by chicken pox include cerebellar ataxia, encephalitis, transverse myelitis, aseptic meningitis, Guillain–Barre syndrome, meningoencephalitis, ventriculitis, optic neuritis, postherpetic neuralgia, herpes zoster ophthalmicus, delayed hemiparesis, peripheral motor neuropathy, myositis, cerebral angiitis, Reye's syndrome, and facial palsies.[1-3] However, these are rare and are seen in only 0.01%–0.03% cases.[4]

As per our knowledge, there has been no case which reported both polyneuropathy and myositis occurring simultaneously postvaricella. We present a case of 21-year-old male who developed both polyneuropathy and myositis at the same time after an episode of chicken pox.

Case Report

A 21-year-old unmarried male, mechanic by occupation, presented with a complaint of weakness and myalgia of all four limbs for 1 month. He was apparently well 1 month back when he suffered from fever with rash which was diagnosed as varicella zoster. Fever and rash recovered with treatment in a week. However, 7 days after the onset of rash, he developed pain, tenderness, and weakness in his left forearm followed by left hand muscles, so that the patient had difficulty in grasping objects with his left hand. 4–5 days later, he developed similar complaints in his right forearm and hand. 2–3 days after this, the patient developed pain and tenderness in both his thigh and calf muscles along with weakness of both lower limbs such that he had difficulty in walking and standing up from sitting position. At this time, he was investigated outside. His complete blood count (CBC), rheumatoid factor titer, serum uric acid, and serum calcium levels were normal. Urine examination revealed 6–8 pus cells/hpf and calcium oxalate crystals. He was given analgesics, but did not improve. 4–5 days after this, he presented to our hospital with the same complaints which had worsened in intensity. By this time, he had also developed difficulty in dorsiflexion of both feet. There was no bowel and bladder involvement and no history

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of headache, neck muscle rigidity, nasal regurgitation, altered speech, involuntary movements, diplopia, facial weakness, or asymmetry, but 4–5 days after admission, he developed mild dysphagia which was more for solids than liquids.

The patient gave a history of hepatitis B surface antigen-positive status detected in 2009. He had a family history of his sister having similar fever with rash 1 week after he had developed the same. She was also diagnosed and treated as varicella zoster and had recovered completely with no sequelae.

On examination, his pulse was 108/min and regular and adequate in volume in the right radial artery and blood pressure was 128/90 mmHg in the left arm in supine position. He had nonpitting edema over both forearm muscles and calf muscles with superficial warmth and tenderness [Figure 1].

Respiratory, cardiovascular, and per abdomen examination were unremarkable. On central nervous system examination, the higher mental function and cranial nerve examination revealed no abnormality. There was no muscle wasting, and tone in all four limbs was also normal. Power in all four limbs was 3+/5. Coordination could not be tested due to weakness. The knee and ankle jerks of both limbs were exaggerated, whereas the deep tendon reflexes of the upper limb, the jaw jerk, and the gag reflex were normal. There was loss of fine touch on dorsal (DL) aspect of both feet, whereas the rest of the sensory system examination was unremarkable. Babinski sign was negative in both the feet. A provisional diagnosis of postinfectious myositis or postinfectious neuropathy was kept in mind and the patient was investigated further on these lines.

His CBC showed slightly raised total leukocyte counts, but was otherwise normal. His urine examination, serum electrolytes, renal function tests, total protein/albumin, sickling, rheumatoid arthritis factor, and thyroid function tests were all normal. His liver function test showed normal bilirubin, but the serum glutamic oxaloacetic transaminase and serum glutamic pyruvic transaminase levels were raised slightly; C-reactive protein was also slightly raised, whereas serum calcium was reduced. Vitamin D3 levels were significantly low and Vitamin B12 levels were also on the lower side. Vitamin B12 and Vitamin D3 supplementations were given to the patient, but he did not show any significant improvement, rather his symptoms increased in intensity as the time passed. Hence, a cerebrospinal fluid (CSF) examination, nerve conduction study/electromyography (NCS/EMG), and magnetic resonance imaging (MRI) DL spine with whole spine and brain screening were done. The CSF examination was unremarkable, but the NCS/EMG was suggestive of mild demyelinating pure motor neuropathy and the MRI-DL spine with whole spine and brain screening showed the possibility of inflammatory myositis [Table 1]. The patient was given tablet acyclovir and intravenous corticosteroids for 5 days. With this, he showed a significant improvement in symptoms (probably due to myositis responding to steroids) with reduction of pain, tenderness, and swelling over the muscles with reduced difficulty in walking and dorsiflexion of the feet [Figure 2].

After 5 days, he was discharged on oral corticosteroids and acyclovir with advice of physiotherapy. There was a gradual improvement after discharge, and on follow-up after 30 days, he was able to walk with support.

Discussion

Myositis is defined as an inflammation of voluntary muscles, which presents with pain, tenderness, swelling, and weakness of voluntary group of muscles. Infectious myositis may be due to wide variety of pathogens including bacteria, viruses, parasites, and fungi. Bacterial myositis presents as focal muscle infection, whereas viruses and parasites tend to cause diffuse disease with generalized myalgias and multifocal myositis.[5] Viruses are reported as the most common etiologies of nonbacterial infectious myositis cases in the U.S. and the rest of the developed world. It may be caused by a variety of viral agents, including influenza (the most common etiology), parainfluenza, enteroviruses (coxsackievirus and echovirus), adenovirus, severe acute respiratory syndrome-coronavirus, HIV, herpes viruses (varicella, herpes simplex, Epstein–Barr, and cytomegalovirus), parvovirus B19, dengue, and West Nile virus.[6,7] Although it results in spontaneous recovery in most of the cases, complications such as rhabdomyolysis, myoglobinuria, acute renal failure, cardiac arrhythmias, and compartment syndrome which could be potentially dangerous have also been associated with significant morbidity.[8]

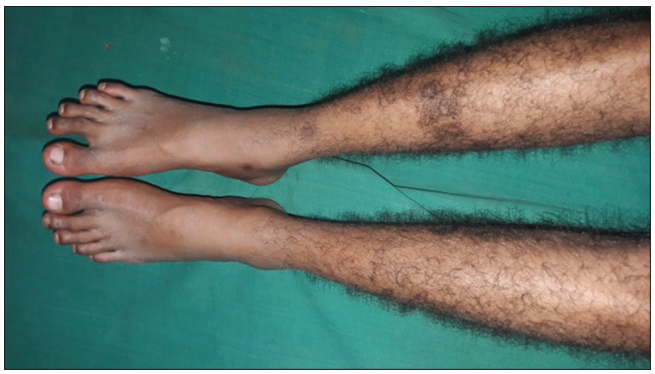

Figure 1: Swelling over the calf muscles and feet before starting corticosteroids

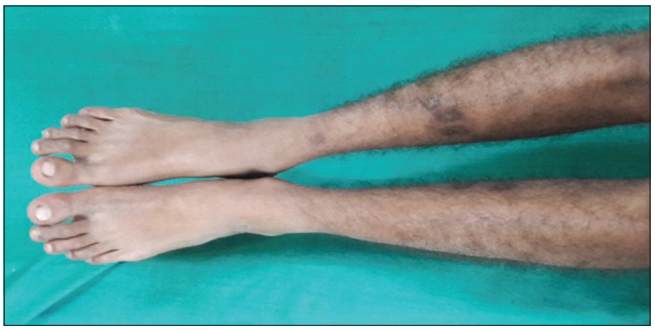

Figure 2: Swelling over the calf muscles and feet decreased after 5 days of intravenous corticosteroid therapy

Table 1: Laboratory reports of the patient

	December 18, 2018	December 20, 2018
Hb (g %)	15.7	14
PCV (%)	44.9	37.3
MCV (fl)	71.5	72.9
MCH (pg)	25	26
MCHC	35	35.7
Total RBC count (million/µL)	6.28	5.12
RDW (%)	19	18.3
TLC (cells/cumm)	13,600	10,300
Platelets (lacs/cumm)	2.61	2.37
Urine analysis	Normal	
RBS (mg/dl)	80	
FBS (mg/dl)	108	
Serum (Mmol/L)		
Sodium	136	
Potassium	4.2	
Chloride	104	
Urea (mg %)	14	
Serum creatinine (mg %)	0.6	
Total proteins (g %)	6.2	
Albumin (g %)	3.0	
Globulin (g %)	3.2	
Albumin/globulin ratio	0.94	
ESR (mm)	35	
Sickling	Negative	
Serum bilirubin (mg%)	0.8 (direct - 0.3, indirect - 0.5)	
	December 18, 2018	**December 22, 2018**
SGPT (IU/L)	168	84
SGOT	287	149
HIV, HBsAg; HCV (rapid)	Negative	
HbsAg (ELISA)	Negative	
CPK total (U/L)	45	1214
CPK-MB (U/L)	158	
LDH (U/L)	824	
RA factor (IU/ml)	<10	
CRP (mg/L)	17.26	
Serum calcium (mg %)	8.5	
TSH (µIU/ml)	4.28	
Vitamin B12 (pg/ml)	321	
Vitamin D3 (ng/ml)	11	
CSF examination (December 21, 2018)		
Physical appearance	Clear	
Sugar (mg/dl)	73	
Protein (/dl)	62	
ADA (U/L)	4	
LDH (U/L)	77	
Total count (cells/cumm)	5	
Lymphocytes (%)	90	
Ependymal cells (%)	10	
Gram stain	No pus cells seen, no organisms seen	
ZN stain	Acid-fast bacilli not seen	
ECG	Sinus tachycardia, normal axis, no ST-T segment changes	
USG abdomen pelvis	NAD	
Chest X-Ray PA view	NAD	

Contd.....

Table 1: Contd...

	December 18, 2018	December 20, 2018
MRI DL spine with whole spine screening with brain screening	Diffuse STIR hyperintense signal involving bilateral psoas muscle, iliacus, paraspinal, gluteus, and intercostal muscles, suggestive of edema, possibility of inflammatory myositis likely	
NCS/EMG report (December 21, 2018)	MNCS: There is increased DL, normal CMAP, CV noted in the right median and right tibial nerves. Reduced CMAP, normal DL and CV noted in the right peroneal nerve	
	F wave: Normal F wave minimum latency over all tested nerves	
	SNCS: Sensory conduction parameters were normal over all tested nerves	
	Needle EMG: EMG was done over the right vastus lateralis and right gastrocnemius muscles. No spontaneous activity was observed. MUAP analyzed were of normal duration, amplitude, and phases. Interference pattern was complete	
	Conclusion: EP study suggestive of mild demyelinating pure motor neuropathy	
NCS/EMG report (December 28, 2018)	Mixed predominantly demyelinating sensory motor polyneuropathy involving all 4 limbs	

Hb: Hemoglobin, PCV: Packed cell volume, MCV: Mean corpuscular volume, MCH: Mean corpuscular hemoglobin, MCHC: MCH concentration, RBC: Red blood cell, RDW: Red cell distribution width, TLC: Total leukocyte count, RBS: Random blood sugar, FBS: Fasting blood sugar, ESR: Erythrocyte sedimentation rate, SGPT: Serum glutamic-pyruvic transaminase, SGOT: Serum glutamic-oxaloacetic transaminase, HbsAg: Hepatitis B surface antigen, HCV: Hepatitis C virus, CPK: Creatine phosphokinase, CPK-MB: Creatinine phosphokinase-MB fraction, CRP: C-reactive protein, RA: Rheumatoid arthritis, LDH: Lactic dehydrogenase, TSH: Thyroid-stimulating hormone, CSF: Cerebrospinal fluid, ADA: Adenosine deaminase, ZN: Ziehl–Neelsen, ECG: Echocardiogram, USG: Ultrasound sonography, PA: Posteroanterior, MRI-DL: Magnetic resonance imaging distal latency, STIR: Short-TI Inversion Recovery, MNCS: Motor nerve conduction study, CMAP: Compound motor action potential, CV: Conduction velocity, SNCS: Sensory nerve conducting study, NCS: Nerve conduction study, EMG: Electromyography, EP: Evoked potential

Many explanations have been suggested as the pathogenic basis for these complications such as direct viral invasion[9] or immune-mediated allergic mechanism. However, allergy-mediated injury has been reported to be the cause in most pathologic studies.[10] However, most of the times, the neurological sequelae are in relation to reactivation of latent disease, whereas those following primary infection are rare.[11]

This case presented with a combination of polyneuropathy and myositis, that too following the primary infection. It progressed to dysphagia also, but responded well to intravenous steroids. Hence, this case brings to notice that neurological complications postvaricella can occur even following primary infection and that one should keep high index of suspicion for detecting these as they are reversible with treatment without any residual involvement.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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