A 72 year-old diabetic woman with herpes zoster paresis: a case report

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ABSTRACT

Background: Varicella-zoster (VZV) is an exclusively human pathogen. The primary infection typically occurs during childhood and causes varicella. As other members of the herpes viruses’ family, VZV is noninfectious in its latent form but can reactivate at a later time to form intact virions in the involved sensory neurons. These virions then migrate to the skin through axons, spread from cell to cell, and penetrate the epidermis.

Patient: A 72-year-old woman with history of diabetes mellitus and hypertension hospitalized because of urinary retention, weakness and paresthesia in right leg complicated with vesiculoulcerative lesions in sacral area with distribution to right buttock and vagina. Lumbar puncture confirmed inflammatory radiculopathy and showed aseptic meningitis. Treatment was commenced with acyclovir and prednisolone. Patient enjoyed healthy life thereafter.

Conclusion: Motor weakness in non-cranial nerve is one of the zoster complications that known as zoster paresis. Weakness begins suddenly 2-3 weeks after rash and progress to extremities. In the present case nerve involvement was detected 3 weeks after rash.

Keywords: Varicella-zoster; Paresis; Diabetes mellitus.

INTRODUCTION

Varicella-zoster is an exclusively human pathogen that infects 98% of adults in USA. Primary infection typically occurs in childhood period and causes varicella in viremic phase, cells infected with varicella access to epidermal cell and cause typical varicella rashes. Then virus enters to sensory neurons from mucocutaneous locations and through axon enters to postganglionic roots near spinal cord (via retrograde), where the virus established in the body of the nerve cells (1). Latent VZV with genomic copies concentration <10 in contaminated cell, affect 1-7% of sensory ganglionic neurons (2).

Furthermore, it is possible that in viremia period, affected posterior ganglionic roots take place.

Possibly the importance of viremia period were determined based on skin damages and the ability of VZV in localization in neuron ganglia during primary varicella infection (3).

Like other members of herpes virus family, VZV is not infectious in the latent period, but it can be activated in a later period of time, and then migrate to skin through axons, then spread from cell to cell, and penetrate to epidermis (4).

Complete clinical symptoms of zoster, can be diagnosed by pain, subsequently vesicular rashes appear near dermatomes. Main reason for activation of VZV is not determined and possibly different factors are involved in. The commonest
zoster complication is PHN (post herpetic neuralgia), which mostly provide painful condition, can be continued until months or years. Another important complication is ophthalmic zoster (5) that was seen in 10-25% of cases. Another rare complication is Ramsy-Hunt syndrome that in fact is a facial nerve paralysis with zoster vesicles in ear, hard palate or tongue (6).

Also zoster can cause autonomic dysfunction such as urinary retention and pseudo-obstruction of colon. Another focal complication is SZP (segmental zoster paresis). Neurological weakness is asymmetric and occurs in limb that previously affected by cutaneous eruption of zoster. In one retrospective study, it was seen at least in 3-5% of patients with cutaneous zoster, most of patients were in age more than 60 years old. Weakness appears in proximal muscles (via L2, 3, 4 or C5, 6, 7). The remission of motor function was seen in 75% of cases that usually occur within 1 to 2 years (7).

Secondary attack of VZV was seen in less than 5% of cases and occurs more commonly in immunocompromised hosts. Primary infection causes long immunity against varicella and protection against reactivation of virus depends on protection of cellular immunity (CMI) that is decreased with age and some disorders (HIV, malignancy) and immunosuppressive therapy (8).

**CASE PRESENTATION**

A 72-year-old woman with a 5-year history of hypertension and diabetes mellitus presented to our department with urinary retention, weakness and paresthesia in right lower limb. Meanwhile, she had vesicular eruptions on right sacral and buttock and outer right part of vagina. Before cutaneous eruptions occurred, she complained of pain and radicular burn. Then, stool incontinency was added. Her initial vital signs were all within normal limit except for oral temperature of 38.8°C. Physical examination of heart, lung and abdomen was normal, however, several eruptions with 1-3 mm ulcer in the right sacral and buttock area was noted.

Neurological examination revealed right inferior limb force of 4/5 in proximal and 5/5 in distal area while knee reflexes were=0 in right and ++ in left side. Nerve conduction studies (NCS) were normal but compound muscle action potentials (CMAP) decrease in femoris rectus muscles, adductor longus, and iliopsoas with fibrillation, while sharp positive waves (PSW) and active denervation evidence was noted in paraspinal muscles. The diagnosis of inflammatory radiculopathy was added and radiculopathy due to disk was reported. MRI revealed lumbosacral discopathy (L5-S1 and L4-L5).

Based on the abovementioned features, a diagnosis of zoster paresis was proposed.

Lumbar puncture analysis revealed the following: WBC number, 120/mm³ with lymphocyte predominance (90%), sugar 60 mg/dl with simultaneous blood sugar level of 120mg/dl and protein value of 40 mg/dl.

PCR-DNA for both VZV and HSV was negative, but blood CSF IgG anti-VZV was positive and IgG anti-HSV was negative. Meanwhile, serum IgG and IgM anti-VZV was positive.

She received acyclovir (500mg/Q8h/IV), prednisolone (20mg/daily/orally), gabapentin (300mg/Q8h/orally), and vitamin B1 (300mg /daily/orally). Insulin was prescribed to control blood sugar. One week following the commencement of therapy, weakness of inferior limbs was decreased, and on the 18th and 25th day of hospitalization her stool incontinency and urine retention were resolved, respectively. Finally, she was discharged with good general condition and enjoys normal healthy life thereafter.

**DISCUSSION**

Nearly 3% of zoster patients that are admitted in hospital have one or more predisposing factors. Our
patient had two predisposing factors, DM and aging.

Zoster can sometimes cause weakness in motor
system as called zoster paresis (9,10). Suddenly
weakness occurs 2-3 weeks after appearing rash. It
may involve superior or inferior limbs and even
involvement of diaphragm was reported. It seems
that weakness of segmental zoster happen by spinal
and cord invasion with VZV. Because of disturbance
in autonomic nervous system, urinary
retention and stool incontinency may occur (11).
Although prognostic recovery of neurologic
function in our patient was good, it was variable in
some reports.

It must be noted that diagnosis of zoster is often
clinically based and commonest localized
complication in differential diagnosis of zoster
eruptions are rashes caused by HSV, like eczema
herpetiform from (12).

In our patient anti-VZV IgG was positive in
CSF, however, VZV PCR analysis was negative.
This could be in part explained by delay in
neurologic complication.

Appearing of new vesicle or herpes zoster
complication may be symptoms of continuing virus
amplification. In our patient, with regard to
appearing neurologic (myelitis) complication and
CSF disturbances, treatment with intravenous
acyclovir was started.

In a clinical trial, 3-week corticosteroid with
dose decline was compared with acyclovir (alone)
and showed no significant elimination in chronic
pain (13), however, adding corticosteroid revealed
useful for therapy of acute pain.

It must be noted that providing effective and
immune vaccine for zoster, create opportunity for
decreasing this disease and its complication (8).

REFERENCES


2. Wang K, Lau TY, Morales M, Mont EK, Straus SE.
Laser-capture microdissection: refining estimates of the
quantity and distribution of latent herpes simplex virus 1
and varicella-zoster virus DNA in human trigeminal
ganglia at the single-cell level. J Virol. 2005; 79:14079-
87.

3. Gershon AA, Larussa P, Steinberg S. The varicella
vaccine clinical trials in immunocompromised

4. Arvin A. Aging, immunity, and the varicella zoster

5. Scott FT, Leedham-Green ME, Barrett-Muir WY. A
study of zoster and the development of postherpetic
S24-30.


7. Merchut MP, Gruener G. Segmental zoster paresis of
369-75.

8. Dworkin RH. Recommendations for the management
of herpes zoster. Clin Infect Dis. 2007;44(Supp1 1):S3-
S4.

9. Braverman DL, Ku A, Nagler W. Herpes zoster
78:880-2.

10. Vincent KD, Davis LS. Unilateral abdominal
distension following herpes zoster outbreak. Arch


12. Dworkin RH, Johnson RW, Breuer J, Gnann JW,
management of herpes zoster. Clin Infect Dis. 2007; 44

chemical exposures and risk of herpes zoster. Environ