

Herpes Zoster Duplex Bilateralis in Immuno-Competent Patients: Report of Two Cases

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ABSTRACT

Herpes Zoster is a common viral disorder, occurs due to reactivation of latent Varicella Zoster Virus (VZV) usually in adults or elderly patients, usually confined to a single dermatome. Herpes zoster duplex is a rare but well established entity which is simultaneous, occurring of herpes zoster at two different non contiguous dermatomes, can be unilateral or bilateralis. Here we are reporting two cases of herpes zoster duplex bilateralis, in case-1 lesions occurs in two different distant dermatomes while in case-2 it appeared in a single dermatome but both sides were involved. Both the patients were healthy immuno-competent male.

Keywords: HZDB, Immunity, Dermatome, Reactivation, Varicella zoster virus

INTRODUCTION

Herpes zoster, presents as unilateral, vesicular eruption, associated with prodrome of pain and burning [1], involving a single dermatome or adjacent same side dermatome usually in adults and elderly, occasionally in children.

In children and adolescents on primary infection, VZV, causes chicken pox, characterized by generalized vesicular eruptions all over the body, associated with fever, sore throat, pruritus, headache etc. After this episode the virus goes into latency for weeks to years, on reactivation causes herpes zoster which is different from chicken pox. Reactivation of latent VZV is related with decline in immunity which may be due to ageing [1,2], HIV/AIDS, radiotherapy, malignancy [3], immunosuppressive/chemotherapy [4], autoimmune disease [5], or even stress also.

Herpes zoster duplex, simultaneous reactivation of VZV involving two different dermatomes, of which Herpes Zoster Duplex Unilateralis (HZDU), occurs unilaterally in two non-contiguous dermatomes while in Herpes Zoster Duplex Bilateralis (HZDB) it may occur in same (symmetric) or different dermatomes (asymmetric) bilaterally [2,3]. Clinical appearance is similar to classical herpes zoster. VZV reactivation occurs ones in a lifetime and involves a single dermatome. VZV specific T-cell immunity is the most important factor responsible for VZV reactivation in otherwise healthy individuals.

VZV specific IgM antibody in blood can be detected only during active disease, not when the virus is in dormant phase. In research laboratories, Polymerase Chain Reaction (PCR) for VZV DNA and electron microscopy for virus particles can be done from exudates collected from a blister [2].

Throughout the world the incidence rate of herpes zoster ranges from 1.2 to 3.4 cases per 1,000 healthy individuals, increasing to 3.9–11.8 per year per 1,000 individuals among those older than 65 years [2,3,6].

Here we are reporting two cases of herpes zoster occurring simultaneously bilaterally in two different and same dermatomes respectively in immuno-competent male patients.

CASE-1

A 28-year-old man presented with multiple grouped vesicular, painful eruptions over right thigh anteriorly and posteriorly (T8-9) and over left side of abdomen and back (T12, L1-2) for last three days [Table/Fig-1,2]. Previously, the patient had prodromal pain in the areas of skin involvement. History suggestive of chicken pox in childhood can't be elicited. There was no past history of



[Table/Fig-1]: HZDB involving dermatome T8-9 of left side while dermatome T12, L1-2 of right side (anteriorly) **[Table/Fig-2]:** HZDB involving dermatome T8-9 of left side while dermatome T12, L1-2 of right side (posteriorly)

similar lesions, weight loss or diabetes mellitus/asthma/hypertension/malignancy. No history of any medication including immunosuppressive agents or history of chronic illness in last six months was present. He had no risk factors for HIV, and denied any previous unusual infections or family history suggestive of an immune defect. The patient was a farmer & had no history of any mental, emotional, or financial stress. He was non-alcoholic, non-smoker, and did not chew or snuff tobacco. Patient was healthy and having pain in the region of herpes zoster at both sites, with same intensity. The patient had no lymphadenopathy, chronic fever, cough etc. His sleep was disturbed due to pain in the lesions but he denied insomnia, prior to appearance of skin lesions. There was no history of trauma in immediate past. Systemic examination did not reveal any abnormalities. Family history was unremarkable and no consanguinity. After thorough clinical examination and history, the diagnosis of herpes zoster duplex bilateralis was presumed. Smears prepared from both sites, from the base of vesicle and stained with 1% aqueous solution of Toluidine blue 'O' showed multinucleate giant cells with faceted nuclei and homogenously stained 'ground glass' chromatin on Tzanck smear and intra-nuclear inclusions seen on Giemsa staining. Patient was advised test for HIV, CBC, and PBF for abnormal cells, blood sugar, chest radiograph, whole abdominal ultrasound, LFT and RFT. ELISA test for HIV was negative while other investigations were within normal limits. Tests for hepatitis A, B, C & VDRL were negative. Patient was treated with Tab acyclovir

800 mg five times a day for seven days and other symptomatic management. After seven days lesions started to become dry and intensity of pain was also reduced. After 10 months follow-up patient is still healthy and having mild episodic burning sensation 2-3 times a day over the affected area.

CASE-2

A 55-year-old male presented with painful grouped vesicular eruptions over thoracic area (T4) both right and left half [Table/Fig-3,4] in a full belt pattern of four days duration. He gave history of prodromal pain and burning sensation in the same segment two days before the eruption. Neither history suggestive of chicken pox, nor of any previous significant chronic illness was there. No past history of similar illness in the patient and family members. Drug history was not significant as far as immunosuppressive drugs or drugs for chronic illnesses are concerned. No history of high risk behavior as the transmission of HIV is concerned. The patient is a farmer with no psychosocial or financial stress and not having habits of tobacco chewing, smoking or drinking alcohol. Systemic examination didn't show any significant abnormal finding. Local clinical examination of the lesions present bilaterally suggests diagnosis of herpes zoster duplex bilateralis which was confirmed by Tzanck smear and histo-pathological examination. Test for HIV-1 & 2 was negative. Patient's CBC, blood sugar, LFT, RFT were within normal limits. He was treated with acyclovir 800 mg five times a day for seven days and other symptomatic management. After seven days his pain was reduced and episodic burning pain was there for which he was advised tab methylcobalamine and gabapentin with analgesic on as and when required basis. After three months of follow-up patient is still healthy.



[Table/Fig-3]: HZDB involving dermatome T-4 bilaterally (anteriorly)

[Table/Fig-4]: HZDB involving dermatome T-4 bilaterally (posteriorly)

DISCUSSION

Herpes zoster occurs due to a decline in varicella specific immunity, commonly affects thoracic dermatomes & ophthalmic division of trigeminal nerve. In our case-1 thoracic and lumbar (T8-9 & T12, L1-2) dermatomes were involved while in case-2 thoracic dermatomes (T4) were affected bilaterally. Herpes zoster duplex bilateralis a rare clinical variant of herpes zoster characterized by simultaneous reactivation of VZV at two non-contiguous dermatomes bilaterally but that is very rare in immuno-competent patients.

Our both the cases were immuno-competent healthy males which came to department of dermatology within the time-period of 10 months and up to our knowledge it is the first study presenting two cases of herpes zoster duplex bilateralis simultaneously in which case 1 is asymmetrical and the case 2 is the symmetric one.

Age distribution worldwide in cases of herpes zoster duplex bilateralis ranges from 3- 77 years [Table/Fig-5]. Our both the cases were neither in childhood age group nor the elder age group as case-1 is 28-year-old and case-2 is 55-year-old.

Herpes zoster duplex bilateralis is an atypical presentation of herpes zoster that is usually found in patients with compromised immunity or in patients with advancing age but reverse to this our both the patients were immuno-competent.

S. No.	Age/sex	Dermatomes	Immuno-competency	Symmetry	Ref.
1.	72/F	Facial palsy-L Peroneal palsy-R	No	No	[1]
2.	3/F	T2-4-B/L	No	Yes	[2]
3.	49/F	T4	No	Yes	[3]
4.	64/F	Ophth-R, C4-L	No	No	[4]
5.	52/F	T2-3 R, T8-L	No	No	[5]
6.	24/M	T-8 B/L	Yes	Yes	[6]
7.	45/M	Maxillary-L/T9	Yes	No	[7]
8.	73/F	L:T9,10 R:L2,3,S3	Yes	No	[8]
9.	70/M	C4,T2-R, L1,L2-L	No	No	[9]
10.	61/M	T2,T3-R, C5,T1-L	Yes	No	[10]
11.	23/M	Ophthalmic-bl	Yes	Yes	[11]
12.	4/F	T5-L, T7,T8-R	Yes	No	[12]
13.	21/M	T2	No	Yes	[13]
14.	67/F	L4,L5-R, T7,T8-L	Yes	No	[14]
15.	64/F	T8-L, L4-R	No	No	[15]
16.	37/F	T3-R, T11-L	No	No	[16]
17.	24/F	Eye-L, Thigh-R	Yes	No	[17]
18.	10/M	L2-4-L, C4-6-R	No	No	[18]
19.	67/M	V3-R, V1-2-L	No	No	[19]
20.	16/F	V2-R, T4-7- L	Yes	No	[20]
21.	5/F	T5-R, T2-L	Yes	No	[21]
22.	22/F	T7-R, S3-L	No	No	[22]
23.	47/F	T7-R, L3,4-L	Yes	No	[23]
24.	76/F	V1-R, V3-L	Yes	No	[24]
25.	77/M	T6-8-R, S3,4-L	No	No	[25]
26.	36/M	V1-R, Iliopigial -L	Yes	No	[26]
27.	55/F	L1,2,3&S3,4-R, L1,2 & S3,4-L	No	Yes	[27]
28.	60/M	V1-B/L	Yes	Yes	[28]
29.	39/F	T8-B/L	No	Yes	[29]
30.	75/M	V1-B/L	No	Yes	[30]
31.	30/M	T-10 -B/L	No	Yes	[31]

[Table/Fig-5]: Clinico-epidemiological overview of reported cases of herpes zoster duplex bilateralis

Varicella Zoster Virus (VZV) is a neurotropic human herpes virus. After causing chicken pox, it remains latent for decades in cranial nerves, dorsal root and autonomic nervous system ganglia. It is the immune system which maintains the virus in latency, but as the function of immune system compromised which may be due to ageing, immunosuppressive diseases or drugs the virus gets the opportunity to get reactivated and results in herpes zoster. Reactivation of varicella zoster virus is usually confined to a single dermatome and for a single episode, in spite of latent viral genomes that is present in many peripheral sensory ganglia. This suggests the significant role of local factors, e.g., the number of viral copies in cell or a local trauma to nerve root ganglion. Reactivation of the VZV is thought to be due to the waning of VZV-specific T-cell immunity as one age which is not present in our case-1. Involvement of single dermatome in herpes zoster is hypothesized due to local booster immunity from the initial dermatome involvement but in case of herpes zoster duplex bilateralis it may be a possibility that virus may spread from one dermatome to other or simultaneous involvement of more than one dermatome as it occurs in both the patients. The occurrence of VZV reactivation bilaterally in the absence of systemic immuno-compromised condition makes this extremely rare phenomenon.

In both the cases the diagnosis was supported by clinical picture and cytology findings. Tzanck smear from both the lesions were

conclusive. Due to financial constraints viral culture and antibody studies could not be done. However in our patients it was not clear whether the reactivation occurred independently in two separate ganglia as in case-1 or if it was caused by viral spread from one half to another as in case-2. Incidentally both the patients are still healthy even after 10 months in case-1 and three months in case-2 with no additional herpes zoster or any other significant infection or any symptoms indicative of immuno-compromised state.

These cases emphasizes the need for further studies and observations regarding the mechanism for VZV reactivation as it can occur in two remote dermatomes without any systemic immuno-suppression.

CONCLUSION

Though HZDB, a rare variant of HZ, a well recognized and reported condition worldwide, present in immuno-compromised and immuno-competent patients but significantly it is more common in patients with compromised immune system. As our both cases indicates possibility of role of local factors and deny effect of immunity on reactivation of VZV and simultaneously these two uncommon cases raises the question regarding the pathogenesis of reactivation of VZV simultaneously at two different dermatomes or from one dermatome bilaterally and indicates towards the requirement of further research to solve the same and role of local factors and immunity.

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FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Jul 15, 2015**

Date of Peer Review: **Aug 11, 2015**

Date of Acceptance: **Oct 22, 2015**

Date of Publishing: **Dec 01, 2015**