Disseminated Herpes Zoster in immunocompetent host: A rare case report and review of the literature.

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ABSTRACT:

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Disseminated cutaneous herpes zoster is uncommon in immunocompetent. Cutaneous dissemination 10-40% is occurring in immunosuppression, especially due to cell-mediated immunity (CMI) deficiency. Varicella zoster virus-specific CMI is required to halt the virus reactivation. Age-related decline of VZV CMI seems to be one of the most important risk factor for VZV reactivation and subsequent herpes zoster. Nevertheless disseminated herpes zoster in the elderly patient who has no immunosuppressive condition is uncommon. We report a case of elderly man with no apparent immunosuppressive condition presented with vesicular rash on T11 dermatome before generalized spreading of vesicular eruption in 3 days. The patient was treated successfully with intravenous acyclovir.

Key words: disseminated, elderly, herpes zoster, immunocompetent

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บทคัดย่อ :

สุพิชชา กมลรัตนกุล จิตติมา ฐิตวัฒน์ โรคงูสวัดระยะแพร่กระจาย ในผู้ป่วยภูมิคุ้มกันปกติ

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ภาควิชาอายุศาสตร์เขตร้อน คณะเวชศาสตร์เขตร้อน มหาวิทยาลัยมหิดล

โรคงูสวัดระยะแพร่กระจาย พบได้ 10-40% ของผู้ป่วยที่มีภาวะภูมิคุ้มกันบกพร่อง โดยเฉพาะในกลุ่มภูมิคุ้มกันชนิดเซลล์ผิดปกติ เนื่องจากร่างกายใช้ Varicella zoster virus-specific cell-mediated immunity ในการหยุดยั้งไวรัส ซึ่งภูมิคุ้มกันชนิดเซลล์จะลดลงตามอายุที่มากขึ้น โรคนี้พบได้น้อยในผู้ป่วยที่มีภูมิคุ้มกันปกติ โดยพบว่ามีการรายงานเพียง case report หรือ case series เท่านั้น คณะผู้นิพนธ์ได้นำเสนอผู้ป่วยสูงอายุ 1 ราย ที่ไม่มีภาวะภูมิคุ้มกันบกพร่องใดๆ มาด้วยโรคงูสวัดที่ด้านขวาของลำตัว ต่อมาผื่นได้แพร่กระจายไปทั่วร่างกายภายในเวลา 3 วัน ภายหลังจากการให้การรักษาโรคงูสวัดระยะแพร่กระจายด้วยยาacyclovir ชนิดฉีดเข้าเส้นเลือด ผู้ป่วยอาการดีขึ้น และหายเป็นปกติ

คำสำคัญ: งูสวัด, ระยะแพร่กระจาย, ภาวะภูมิคุ้มกันปกติ, คนสูงอายุ

Introduction

Disseminated cutaneous herpes zoster is uncommon in immunocompetent, although it has been described in immunocompromised patients[1]. We report a case of disseminated herpes zoster in elderly man who had absence of a known immunosuppressive condition. The patient was treated successfully with intravenous acyclovir.

Case report

A 63-year old man presented with a 2-day history of vesicular rash on right side of trunk and followed by the spread of vesicular eruption to face, chest, back and all extremities over 3 days. He also had fever and myalgia. Review of systems were negative. The patient had a history of chickenpox during childhood. He has been treated dyslipidemia with simvastatin 10 milligrams per day for 7 years. The patient has not been on immunosuppressive or other medications.

On examination, the patient was afebrile (36°C). He had multiple crusted papules on face trunk and limbs. Several fresh vesicles were noted on the face and chest wall. There was a cluster of vesicles and papules with crusting on the right side of trunk, extending along T11 dermatomal distribution. (Figure 1) There was no mucosal involvement. There was no lymphadenopathy. Other system examinations were unremarkable.

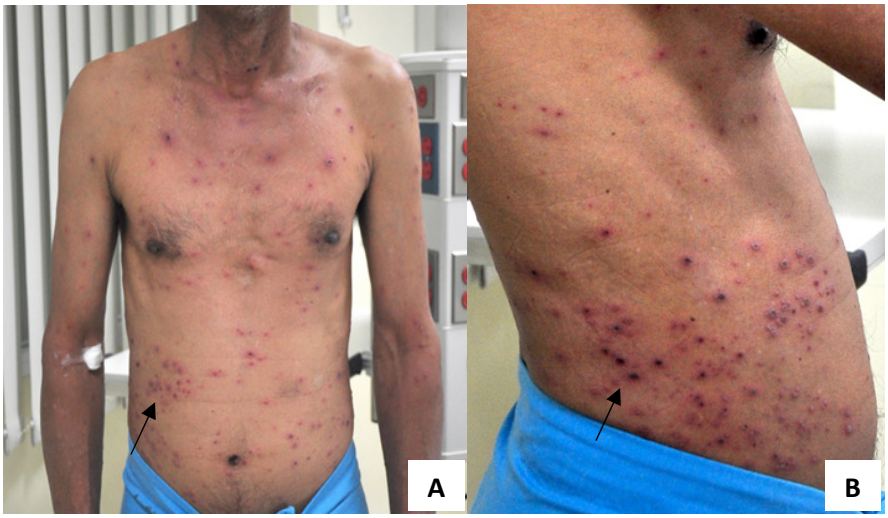

Figure 1 Disseminated herpes zoster. **(A)** Generalized vesicles and crusted papules with **(B)** concentration along T11 dermatome (arrow).

Tzank smear from lesional skin showed multinucleated giant cells and some acantholytic cells. Skin biopsy from fresh vesicle on the trunk revealed an intraepidermal vesicle that contains necrotic keratinocytes, acantholytic cells and multinucleated giant cells. The keratinocytes surrounding this vesicle shows intracellular edema (ballooning degeneration), margination of neucleoplasm, necrotic keratinocytes and multinucleated giant cells. The dermis shows a superficial perivascular lymphocytic infiltrate with neutrophil. (Figure 2)

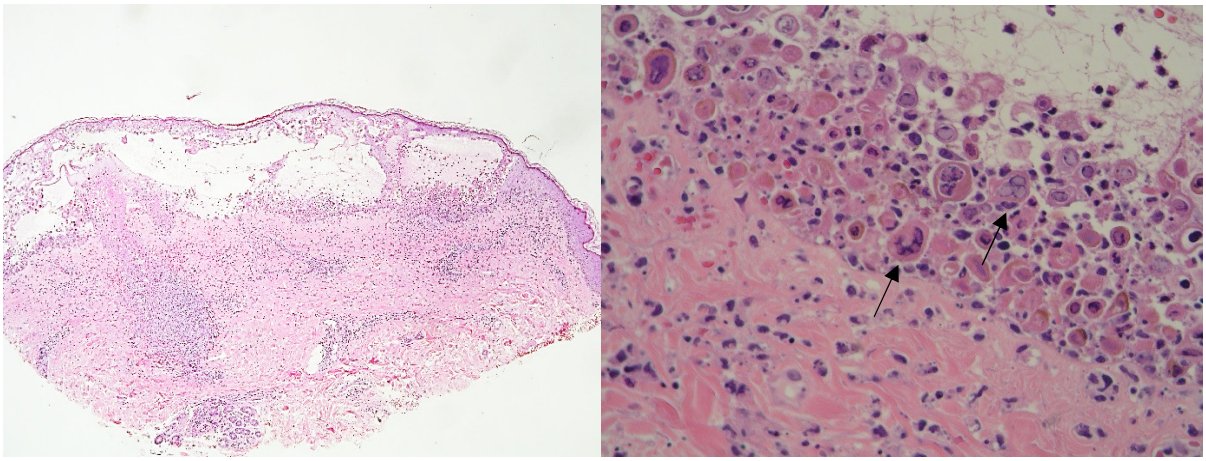

Figure 2 Histopathologic features of herpes zoster. Intraepidermal vesicle that contains multinucleated giant cells (arrow), necrotic keratinocytes and some acantholytic cells (**A**x40. **B**x100)

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Complete blood count, peripheral smear, routine biochemistry, blood glucose, liver function tests, and chest x-ray were normal. Serology for Human Immunodeficiency Virus (HIV) was negative. Serum Varicella zoster antibody both Immunoglobulin G (IgG) and Immunoglobulin M (IgM) done on day8 after the onset of rash were positive (the titers were not obtained).

The patient was treated with intravenous acyclovir 500 mg every 8 hours. In the next 72 hours, no new vesicles were founded and his clinical status improved. Intravenous acyclovir was sustained to six days then the patient was discharged with oral acyclovir 800 mg 5 times daily for ten days. One week follow-up, his skin lesion improved, leaving with hyperpigmented scars. At 6 months of follow-up, he denied any serious illnesses.

Discussion

Herpes zoster results from the reactivation of Varicella zoster virus (VZV), an exclusively human double-stranded DNA virus of the Herpesviridae family. The virus spreads from the epidermis to sensory nerve endings, then enters a lifelong latent state within dorsal root ganglia. Under unknown conditions, the VZV reactivates and travels along the nerve axon to the skin to cause a localized vesicular rash in a dermatomal distribution, called herpes zoster[1]

Disseminated cutaneous zoster is defined as greater than 20 vesicular lesions outside the primary and directly adjacent dermatomes[1]. Cutaneous dissemination 10-40%[2] is occurring in immunosuppression, especially due to deficiency of the CMI, for instance HIV, hematological malignancy, renal transplantation or chemotherapy and hardly occurs in immunocompetent patient that has been discussed as only case reports.[3-12]

Varicella zoster virus-specific cell-mediated immunity (CMI) is required to halt the virus reactivation. Age-related decline of VZV CMI seems to be one of the most important risk factor for VZV reactivation and subsequent herpes zoster. Nevertheless disseminated herpes zoster in the elderly patient who has no immunosuppressive condition is uncommon. Humoral immunity does not perform to defend against reactivation of VZV as antibodies levels are preserved throughout all age groups. Correspondingly, patients with humoral immunity deficiency are not more risk to severe varicella infections.[13, 14]

In addition to the clinical, biopsy of the skin lesions may provide definitive evidence of herpes virus infection but cannot distinguish between herpes simplex, herpes zoster and primary varicella. Infected epithelial cells exhibit acantholysis, ballooning degeneration, multinucleate giant cells and acantholysis of the

epidermis.[15] From reviewing case reports, most patients (95.12%) with disseminated zoster firstly presented with a localized herpes zoster in one or several dermatomes then generalized distributed rash occurred 1-12 days after the initial dermatomal rash which is different from chicken pox in adult.[6-8,10,11,16] In immunocompetent host, most common involving dermatome is cranial nerve followed by thoracic. Most frequent complications of cutaneous dissemination are bacterial superinfection then post herpetic neuralgia that is not different from immunocompromised host.[16]

Extracutaneous involvement were reported in 3 case, 2 cases had aseptic meningitis, 1 case had Ramsay-Hunt syndrome. There are no significant differences in clinical manifestation, outcome and mortality between immunocompromised and immunocompetent host.[16] Disseminated zoster cases typically are treated with intravenous acyclovir 10 mg/kg every 8 h for 5–7 days. Other medication are oral acyclovir, oral valacyclovir. All patients with reported outcomes recovered. (Table 1)

Table 1 Reported cases of disseminated herpes zoster in immunocompetent host

Age (yrs)	Sex	Initial Dermatome	Extracutaneous involvement	Interval before dissemination (days)	Treatment	Outcome
37[8]	F	CN V	None	5	N/A	Recovered
58[8]	M	CN V	None	6	N/A	Recovered
72[8]	F	C1 to C3	None	4	N/A	Recovered
54[8]	F	CN V	None	3	N/A	Recovered
43[12]	M	Right T8	None	2	N/A	N/A
50[12]	M	Right thigh	None	1	N/A	N/A
79[10]	M	Left L3	None	N/A	Oral acyclovir 800 mg 5 times daily	Recovered
37[9]	M	Left T2	Aseptic meningitis	N/A	Intravenous acyclovir therapy (10 mg/kg every 8 h)	Recovered
24[17]	M	Left T5	Aseptic meningitis	3	N/A	Recovered
39[3]	M	Right T6	None	9	Oral valacyclovir then IV acyclovir	Recovered

Age (yrs)	Sex	Initial Dermatome	Extracutaneous involvement	Interval before dissemination (days)	Treatment	Outcome
29[18]	M	CN VII	Ramsay-Hunt	6	None	Recovered
25[18]	M	CN V	None	7	None	Recovered
40[18]	F	N/A	None	5	None	Recovered
69[7]	M	Right CN V	None	2	IV acyclovir 800 mg q 8 hr for 6 day then oral acyclovir 800 mg 5 times daily for 16 days	Recovered
97[4]	F	Left CN V3		4	Oral valacyclovir for 7 days	Recovered
79[5]	M	Left T7,8	None	N/A	IV acyclovir	Recovered
80[5]	M	Right shoulder	None	N/A	IV acyclovir	Recovered
71[5]	M	Left T8	None	N/A	IV acyclovir	Recovered

Conclusion

Elderly patients should be recognized as a group in whom the risk of dissemination is higher than the average immunocompetent host. Early diagnosis and aggressive treatment with intravenous acyclovir can reduce morbidity and severity of complications.

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