DISSEMINATED HERPES ZOSTER IN AN IMMUNOCOMPETENT ELDERLY

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ABSTRACT

Herpes zoster characteristically presents with a prodrome of burning pain followed by an outbreak of vesicles distributed unilaterally within a single dermatome. Disseminated cutaneous herpes zoster has been described in persons with immunosuppression due to human immunodeficiency virus (HIV), haematological malignancy or chemotherapy and can be diagnosed when 20 or more vesicles develop within a week of typical herpes zoster infection. A case of 72-year-old male patient is presented here with disseminated cutaneous zoster in the absence of any known immunosuppression.

KEYWORDS

Herpes Zoster, Disseminated, Immunosuppression, Dermatome, Elderly.

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INTRODUCTION

Herpes zoster is a disease where grouped vesicobullous lesions appear along the ipsilateral one or two dermatome and is accompanied by pain. This disease is caused by the reactivation of the varicella-zoster virus (VZV), which may be dormant in the sensory ganglia of the cranial nerve or in the dorsal root ganglia after a previous varicella infection. A disseminated herpes zoster infection can be diagnosed when 20 or more vesicles develop systemically within a week of typical herpes zoster infections.[1] Disseminated cutaneous herpes zoster has been described in persons with immunosuppression due to human immunodeficiency virus (HIV), haematological malignancy or chemotherapy. However, it is uncommon to see dissemination of zoster in healthy individuals.[2] In this case report, we describe the clinical course of an elderly patient who presented with disseminated cutaneous zoster in the absence of any known immunosuppressive condition.

CASE REPORT

A 72-year-old male presented to our OPD with complaints of lesions and burning type of pain over left upper limb since 5 days. He also complained of lesions developing simultaneously over trunk, face and neck over same and also other side of body. There was history of fever, headache and ear ache. No history of blood transfusion, any drug intake, insect bite or any sexually transmitted disease could be elicited. Patient gives history of chicken pox in the childhood. No other comorbidity present. On examination, multiple grouped vesicles few of them haemorrhagic over erythematous base present over left upper limb, chest [Figure 1a]and back [Figure 1b] with few crusted lesions involving cervical 8 and thoracic 1 (C8 and T1) dermatomes. Multiple i.e. more than 20 discrete vesicles were present altogether over both sides of neck, and face, [Figure 2a & 2b] and trunk. [Figure 3] Patients ELISA for HIV was negative. Patient was diagnosed clinically as disseminated herpes zoster without immunosuppression. Patient was treated with tab acyclovir 800 mg five times a day for 7 days, analgesics and topical antibiotic and he responded with resolution of lesions as well as pain.

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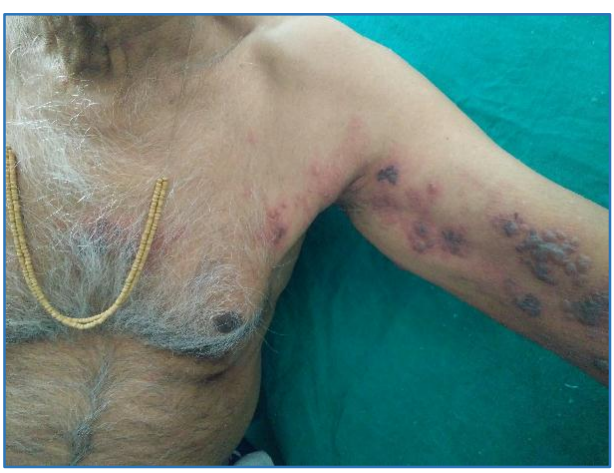

Fig. 1a: Multiple grouped vesicles few of them hemorraghic over left upper limb and chest

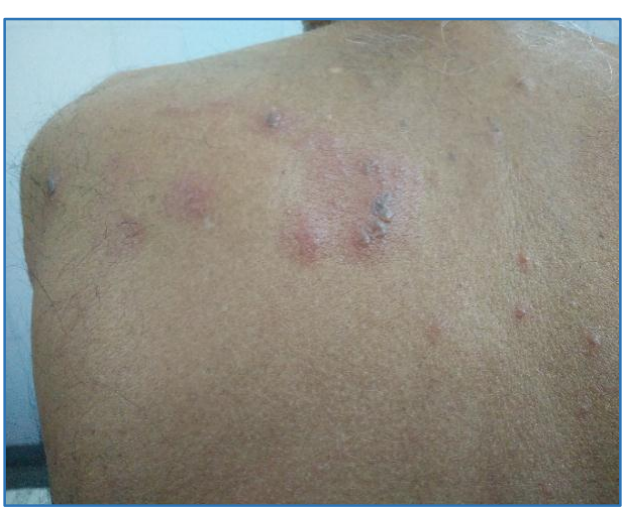

Fig. 1b: Over back

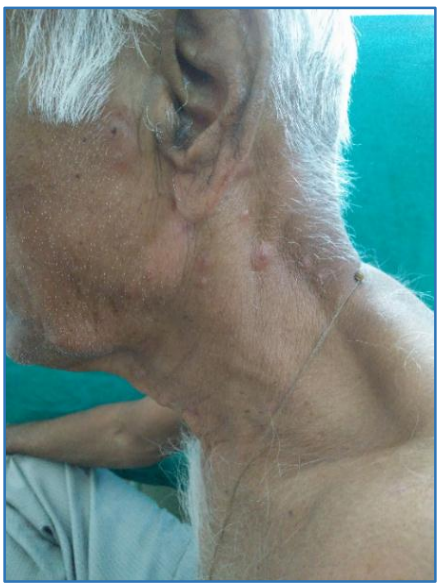

Fig. 2a: Discrete papulovesicular lesions over left side of neck and face

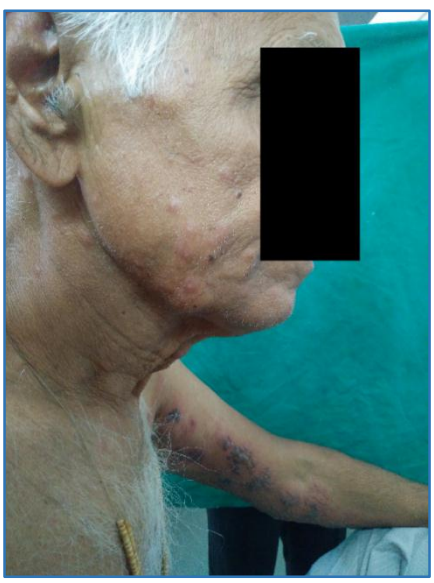

Fig. 2b: Right side of neck and face

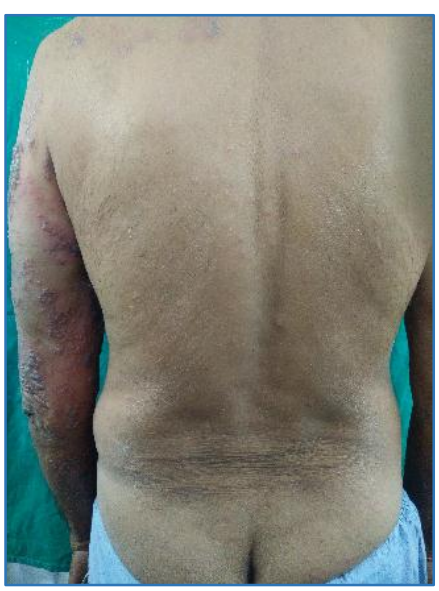

Fig. 3: Multiple discrete vesicles over trunk

DISCUSSION

Human herpes virus 3 (HHV-3) of the Herpes viridae family is an exclusively human virus that causes chickenpox as primary infection and varicella zoster (Also referred to as shingles) on reactivation.[3] Varicella most commonly strikes children, typically causing a self-limited, vesicular eruption. Triggering factors for reactivation leading to herpes zoster include trauma, sunburn, exhaustion, immunosuppression, irradiation or immunodeficiency virus infection.[4]

Single serotype of a virus can have varying presentations depending on the immune status of the host.[5]Zoster characteristically presents with a prodrome of burning pain followed by an outbreak of vesicles distributed unilaterally within a single dermatome.[6] A few lesions (typically less than 20) can normally appear adjacent to the affected dermatome. More extensive skin involvement of several adjacent dermatomes is called multi-dermatomal zoster, whereas spread to a non-adjacent dermatome is known as zoster duplex unilateralis or bilateralis.[6] Disseminated herpes zoster has been defined as more than 20 vesicles outside the area of the primary and adjacent dermatomes.[7]

In immunocompromised hosts, zoster has a disseminated presentation rather than the classic dermatomal distribution as the body cannot suppress the virus and reactivation of zoster can present as a widespread rash.[5] However, disseminated herpes zoster in otherwise healthy persons who are not on immunosuppressive therapy or have no underlying malignancy is rare Butare reported.[2] (Table I)

Sl. No.	Age (Yrs.)	Sex	Side	Primary Dermatome Involved	Treatment
1.(Our case)	72	Male	Left	C7,8	T Acyclovir 800 mg 5 times a day for 7 days
2.	37[8]	Male	Left	T2, then generalized	Acyclovir 10 mg/kg, IV, three times a day for 7 days
3.	39[9]	Male	Right	T6, then generalized	Acyclovir 10 mg/kg, IV, three times a day for 7 days
4.	67[10]	Female	Right	Lower extremity, trunk, and buttocks	Acyclovir 800 mg, orally, 5 times daily for 7 days
5.	79 y[10]	Male	left	L3	Acyclovir 800 mg, orally, 5 times daily for 7 days
6.	70[10]	Female	left	Maxillary division of trigeminal nerve	Acyclovir 800 mg, orally, 5 times daily for 7 days

7.	75 y[1]	Male	Left	C2	Acyclovir 1500 mg, IV, daily for 7 days
8.	69[2]	Male	Right	Ophthalmic division of trigeminal nerve	Acyclovir 800 mg, IV, 8th hrly for 6 days, then acyclovir 800 mg, orally, 5 times daily for 16 days
9.	97[11]	Female	Left	Mandibular division of trigeminal nerve	Valacyclovir, orally, for 7 days

Table 1: Cases of disseminated cutaneous herpes zoster in immunocompetent

The reactivation of VZV is seen to be connected to the decrease in VZV-specific T-cell immunity, and with an increase in age, the cell mediated immunity is weakened, thus there is a higher incidence rate of herpes zoster and the possibility of it developing into post herpetic neuralgia in elderly.[12] In immunocompromised, the incidence rate of disseminated herpes zoster infections is very high, at 10-40% in patients with decreased immunity, especially in patients with HIV infections, haematological malignancies, and those who are undergoing chemotherapy, since they have a decreased number and decreased activity of T lymphocytes.[1] VZV lies dormant extensively along the sensory ganglia, so herpes zoster infections can occur on any part of the body. When it occurs along several dermatomes, as in disseminated herpes zoster infections, the virus can be spread through interconnections between several ganglia or haematogenous spreading.[1]The classical presentation does not cause any difficulty in diagnosis. In doubtful cases, tzanck smear can be made which shows multinucleated giant cells but does not differentiate from herpes simplex infections. Direct fluorescent antibody test and viral cultures are helpful to differentiate from herpes simplex.[13]

Our patient presented with characteristic skin findings of disseminated cutaneous herpes zoster and was diagnosed clinically. Significant age related depression in cellular immunity could have contributed to the dissemination of herpes zoster. Elderly patients should be recognized as a group in whom the risk of dissemination is higher than the average immunocompetent host.[2] Patients with cutaneous dissemination of VZV are at risk of infection of visceral organs, particularly lungs, liver and brain. Other complications include corneal ulceration and post herpetic neuralgia.[7] The important goals of therapy are to lessen the severity and duration of pain associated with the disease. Antiviral therapy certainly hastens the resolution of lesions decreases acute pain and helps in preventing post herpetic neuralgia especially in elderly patients.[13] Acyclovir 800 mg five times daily for 5 days, valacyclovir 1g TID for 5 days and famciclovir 500 mg TID for 7 days are the available antiviral drugs. Analgesic helps to relieve the pain. Therefore, identification and aggressive treatment of disseminated herpes zoster infection in elderly immunocompetent hosts is important.

CONCLUSION

Disseminated herpes zoster is a potentially serious infection that can present in the absence of immunosuppression. Early diagnosis and aggressive treatment with acyclovir can reduce morbidity and severity of complications.

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