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DISSEMINATED HERPES ZOSTER – A CASE SERIES.

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ABSTRACT

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Herpes Zoster (HZ) is caused by varicella zoster virus (VZV) due to reactivation of latent virus from dorsal root ganglia or cranial nerve ganglia. This occurs in up to 15% of individuals following varicella infection, frequently in adults, and the latent period varies from several weeks to years. [1]

Disseminated herpes zoster is defined as at least 20 vesicles in multiple dermatomes. In particular, it has been reported mainly in patients with immunological defects who are at increased risk for visceral involvement of VZV infection, which may affect vital organs such as the brain, liver and lungs. These patients are at increased risk for diagnostic delay and morbidity in the absence of high clinical suspicion for disseminated HZ. [2]

Human immunodeficiency virus infections, chronic infections, primary immunodeficiency syndrome, and immunosuppressive therapy are important causes of disseminated herpes zoster. Herein, we report a case series of disseminated herpes zoster infection.

Key words: Disseminated Herpes zoster, Varicella zoster virus, Acyclovir, Immunocompromised state.

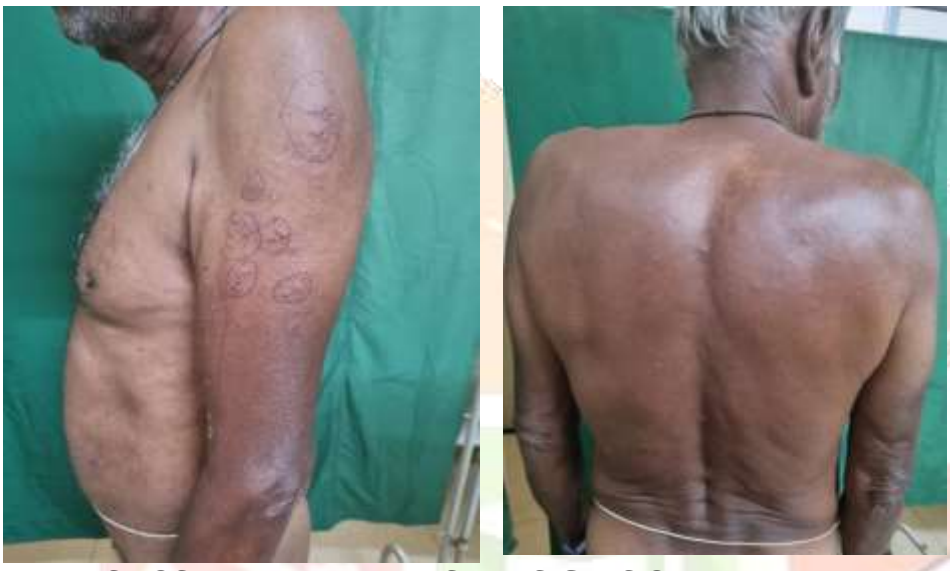
Introduction

Herpes zoster (HZ) or shingles is an acute vesico bullous cutaneous infection with dermatomal distribution caused by reactivation of varicella zoster virus an alpha group of herpes virus predominantly in adults. Extensive cutaneous dissemination has been reported in immune compromised individuals. However, its presence has also been documented in immune competent individuals.[3,4,5]

The estimated average incidence is 2-4/1000 per year which increases to 10/1000/ year at the age of 80 years.[6] Herpes zoster clinically presents as painful grouped vesicles on erythematous base restricted to one or two dermatomes , 75% of patients have pre-eruptive pain which may be constant or intermittent and can precede the rash by 3–4 days and sometimes longer. Most patients describe the pain as a deep “burning”, “throbbing”,or “stabbing” sensation .the lesions evolves through stages of papule, vesicle, pustule and crusting. The main risk factor for HZ is advanced age, most commonly affecting elderly patients. It is hypothesized that a physiological decline in varicella-zoster virus (VZV)–specific cell-mediated immunity among elderly individuals triggers reactivation of the virus within the dorsal root ganglion and similarly in immune compromised individuals, including those with human immunodeficiency virus (HIV) infection, due to suppression of T cells immune to VZV, as well as immunosuppressed transplant recipients , chemotherapy patients .[7] Secondary complications of HZ infection such as postherpetic neuralgia, bacterial superinfection progressing to cellulitis) lead to increased morbidity .The patients of disseminated herpes zoster are at risk for visceral involvement, which may affect vital organs such as the brain, liver and lungs.[6]

CASE REPORT-1

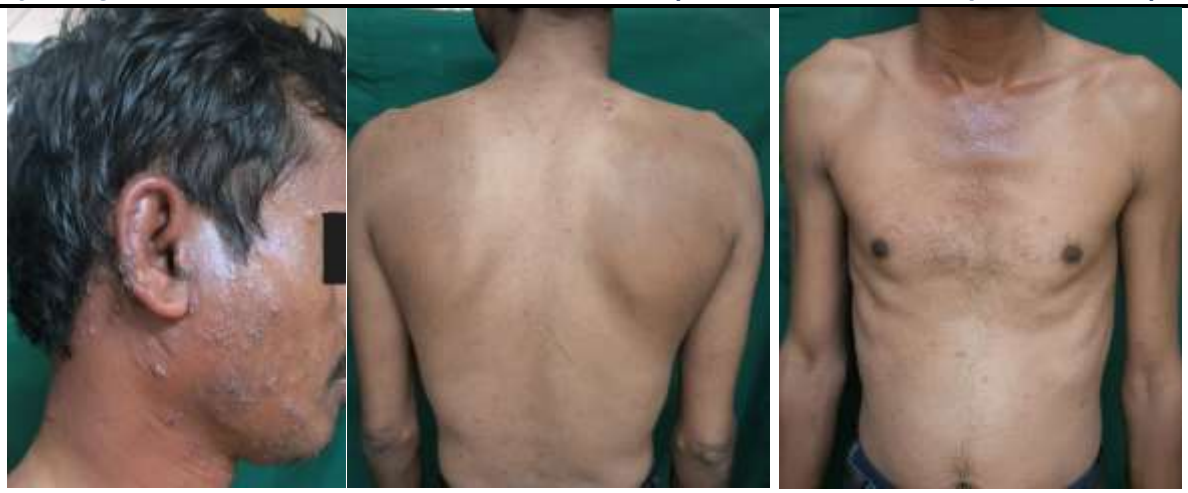
A 75 year old male patient visited our hospital with complaints of stabbing pain and grouped erythematous vesicles on the left arm, neck and left upper back, which started approximately 10 days ago. On examination, the lesions were distributed along the left C5 and C6 dermatomes with more than 20 vesicles scattered predominantly over the trunk, with multi-dermatomal involvement. Patient was a known case of type 2 diabetes mellitus on oral hypoglycemics with poor glycemic control. Patient was hospitalized and on further investigation, complete blood count, liver function test , renal function test were within normal limits. The serological tests for HIV, Hepatitis-B, Hepatitis-c were negative. FBS, PPBS, HBA1C were abnormally high . Tzanck smear from base of vesicle showed multinucleated giant cells .The patient was treated with intravenous injection of Acyclovir 10 mg/kg TID for 5 days , pregabalin tablet 75 mg/day, .Nortriptyline tablet, 10 mg/day, insulin and oral hypoglycemic drugs . The patient recovered from disseminated HZ after 3 weeks.



HERPES ZOSTER WITH DIFFUSE VESICLES ON TRUNK.

CASE REPORT-2

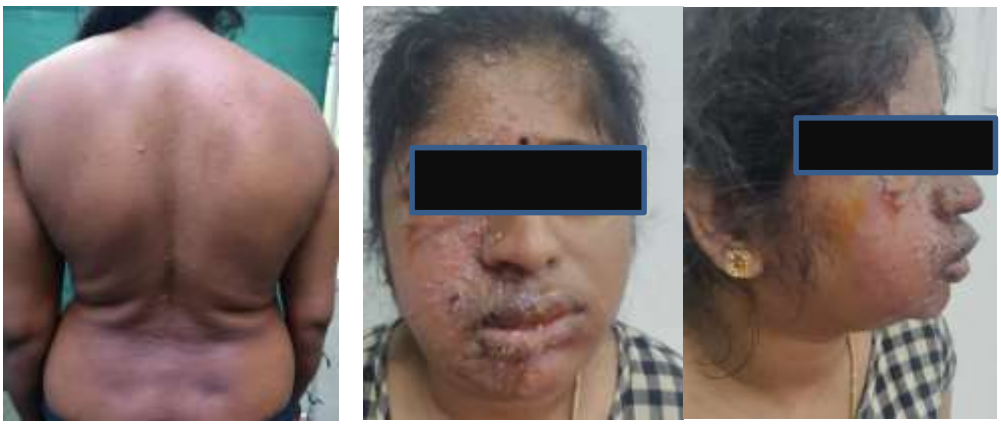
A 35 year old male patient presented with painful grouped vesicles on right side of face since 5 days and on examination grouped vesicles on erythematous base noted on the right C2,C3 dermatomes and along the distribution of mandibular branch of trigeminal nerve. The patient also had > 20 scattered vesicles over the trunk upper limb with multi dermatomal distribution . At the initial onset of the symptoms, the patient had been diagnosed with herpes zoster infection at a local clinic and was taking oral antiviral agents for 2days with no improvement in the symptoms. According to his premedical history he had no co-morbidities, patient was hospitalized and was with investigated complete blood count, liver function test and renal function test were normal .Serological test for, Hepatitis-B, Hepatitis-c, were negative. FBS, PPBS, HBA1C was normal and serology for HIV was positive. There were no other abnormal observations in other tests . Tzanck smear from the base of vesicle showed multinucleated giant cells and neutrophils . Patient was started on injection Acyclovir 10mg/kg I.V TID for 7 day,along with tablet pregabalin, 75 mg/day and nortriptyline, 10 mg/day. The lesions resolved completely after 10 days and the patient was discharged after initiating HAART .



HERPES ZOSTER GROUPED VESICLES ON RIGHT SIDE OF FACE WITH DISCRETE SCATTERED VESICLE ON THE TRUNK .

CASE REPORT-3

A 26 year old female patient visited our hospital complaining of stabbing pain and fluid filled lesions on right side of face which started 5 days ago .On examination grouped vesicles on erythematous base noted on the right ophthalmic, maxillary ,mandibular branch of trigeminal nerve dermatome , with right-sided facial palsy.the ophthalmic examination revealed superficial corneal erosion of the right eye. There was no history suggestive of comorbidities.On investigating her complete blood count, liver function test and renal function test were normal. Serological test for HIV, Hepatitis-B, Hepatitis-c were negative. FBS, PPBS, HBA1C, were abnormally high with 2+ sugar in urine which was newly detected. Tzanck smear from the base of vesicle showed multinucleated giant cells. The patient was hospitalized and IV acyclovir (10 mg/kg TID) was initiated with oral Gabapentine 300 mg TID, pregabalin,75 mg/day and moxifloxacin eye drops. The patient continued to have new eruptions for 2 days after admission and the new vesicles appeared over the trunk which was more than 20 in number with multidermatomal, bilateral involvement. . The patient was treated with insulin and oral hypoglycemics for type 2 diabetes and a short course of steroids for Ramsey hunt syndrome. The lesions of HZ healed completely after 2 weeks but recovery with residual facial palsy recovered after 6 months. .



HERPES ZOSTER WITH GROUPED VESICLE ON RIGHT SIDE OF FACE WITH DEVIATION OF ANGLE OF MOUTH TO LEFT WITH DIFFUSE VESICLES ON TRUNK

DISCUSSION

Disseminated cutaneous herpes zoster (DCHZ) is defined by the presence of more than 20 vesicles beyond the primary or adjacent dermatomes. VZV usually remains dormant in the sensory ganglion after primary infection. VZV-specific cell-mediated immunity is very important to prevent reactivation of the primary infection. Thus, this complication of zoster has been predominantly reported in individuals with underlying immunosuppression (especially in T-cell deficiency) such as HIV, malignancy, chemotherapy or immunosuppressive therapy, bone marrow transplant recipients and immunological disorders. The incidence is reported to be as high as 10% – 40% with severe cutaneous and visceral disseminated disease.[8] Healthy immune competent individuals affected with dissmenated HZ have been rarely reported in the literature .[3]The virus spreads through interconnections between ganglia or through hematogenous spread. The patients afflicted with HIV have multiple recurrences of HZ and the recurrence may involve the same, contiguous, or distant dermatomes. The complications like postherpetic neuralgia ocular complications , Ramsay Hunt syndrome, secondary bacterial infection, meningoencephalitis, motor paralysis, pneumonitis, hepatitis, aseptic meningitis, peripheral motor neuropathy, Guillain-Barré syndrome, transverse myelitis, Cardiac arrhythmias, Urinary retention and hematuria can occur.[9]

Herpes Zoster Ophthalmicus is a serious condition occurring in 8–56% of all HZ cases with involvement of frontal branch of ophthalmic division of the trigeminal nerve presenting with hyperemic conjunctivitis , eviscerates, and lid droop can occur. The vesicular lesions on the tip and side of the nose are associated with a high risk of ocular involvement (Hutchinson's sign)[10] indicating involvement of the nasociliary branch of the trigeminal nerve, which also innervates the eye. Ocular complications include keratitis , dendritiform ulcers, iritis, conjunctivitis, and acute retinal necrosis

Ramsay Hunt syndrome is due to reactivation of latent VZV residing within the geniculate ganglion , presents as a triad of ipsilateral facial paralysis, ear pain, and vesicles in the auditory canal and auricle. The involvement of the nervus intermedius or its geniculate ganglion impairs the taste sensation from the anterior two-thirds of the tongue and alter salivation.
It is generally considered as polycranial neuropathy with the involvement of V, IX, and X cranial nerves. VZV also invades the 8th cranial nerve causing tinnitus, vertigo, and hearing impairment.[11]

Aseptic meningitis, peripheral motor neuropathy, meningitis, Guillain-Barré syndrome, and transverse myelitis can occur. Bacterial infections caused by Staphylococcus and Streptococcus are common. Cardiac arrhythmias can occur. Rarely, urinary retention and hematuria can occur with the involvement of sacral dermatomes. Immuno-compromised patients including HIV patients and transplant recipients are at more risk for all these complications.

A recombinant varicella zoster virus (VZV) envelope glycoprotein E (gE) antigen lyophilized component vaccine advised in elderly to decrease incidence and complication s of herpes zoster infection.[12] CDC recommends 2 doses of Shingrix vaccine 0.5ml intramuscular injection separated by 2–6 months for immuno competent adults aged 50 years and older. CDC recommends 2 doses of RZV to prevent shingles in adults aged ≥19 years who are or will be immunodeficient or immunosuppressed because of disease or therapy. The second dose of RZV should typically be given 2–6 months after the first dose .

Treatment for disseminated herpes zoster with antiviral therapy should be initiated within 72 hours of clinical presentation.. Acyclovir is given as an intravenous infusion (10 mg/kg every 8 hours) for 5- 7days or until crusting of vesicles. Acyclovir can cause reversible renal insufficiency by causing crystal-induced obstructive nephropathy. Nausea, vomiting, diarrhea headache and hallucinations are the other frequent side effects . Infrequent adverse effects (0.1–1% of patients) include agitation, vertigo, confusion, dizziness, edema, arthralgia, sore throat, constipation, abdominal pain, hair loss, rash, weakness. Rare adverse effects (<0.1% of patients) include coma, seizures, neutropenia, leukopenia, crystalluria, anorexia, fatigue, hepatitis, Stevens-Johnson syndrome, toxic epidermal necrolysis, encephalopathy and/ or anaphylaxis.[13]

Systemic steroid therapy is ineffective in preventing post herpetic neuralgia Furthermore, it could potentially increase the risk of secondary bacterial skin infection. However, steroids are effective in Ramsay Hunt syndrome and along with antivirals in VZV-induced cranial nerve palsies.

Our patienst presented with the characteristic skin findings of disseminated cutaneous herpes zoster and an immunocompromised state . They were successfully treated without any severe complications within a time frame similar to the pathogenesis and regression of general acute herpes zoster infections.

CONCLUSION.

All the three patients with disseminated HZ were treated adequately and development of systemic complications was prevented. Though, all the patients had profound suppression of immune system, 2 patients had a recovery time similar to immunecompetent individuals.

It is necessary for clinicians to recognize the spectrum of atypical presentations of herpes zoster and to adequately perform appropriate diagnostic or confirmatory test among at-risk patients with impaired immune function. Effective prevention and treatment modalities for VZV infection among immunocompromised patients is critical because, the morbidity associated with complications of VZV infection is substantial .

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