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A CASE REPORT ON HERPES ZOSTER WITH TENIA CORPORIS

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ABSTRACT

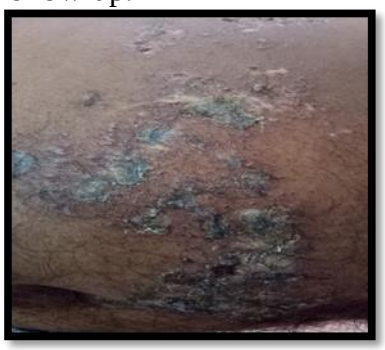
Herpes zoster is a clinical manifestation of the reactivation of latent varicella zoster virus infection. It is a cause of considerable morbidity, especially in elderly patients, and can be fatal in immunosuppressed or critically ill patients. The pain associated with herpes zoster can be debilitating, with a serious impact on quality of life, and the economic costs of managing the disease represent an important burden on both health services and society.Varicella zoster virus (VZV) is a DNA virus and a member of the alpha herpes viridae family, causing both primary and recurrent infection. Herpes zoster (HZ), commonly called shingles, is a distinctive syndrome caused by reactivation of VZV. This reactivation occurs when immunity to VZV declines because of aging or immune-suppression. HZ can occur at any age but most commonly affects the elderly population. HZ may affect any sensory ganglia and its cutaneous nerve. HZ causes pruritic, localized, vesicular rash which usually appears unilaterally in the distribution of one or more adjacent sensory nerves accompanied by neuropathic pain in the affected dermatome. This is a case report of HZ infection in a 60-year-old male patient who was managed with comprehensive medical treatment.[1]

INTRODUCTIO

Varicella zoster virus (VZV) has a high level of infectivity and has a worldwide prevalence. Herpes zoster is a re-activation of latent VZV infection. Herpes zoster (HZ) can occur at any time after varicella infection or varicella vaccination. But the incidence of zoster is less after varicella vaccination than after natural infection. The incidence of zoster increases with age, although children who had varicella during the first year of life (or *in utero*) are at increased risk of developing zoster. It is diagnosed clinically by unilateral vesicular eruption involving a dermatome or dermatomes. Varicella zoster virus (VZV) is a ubiquitous DNA virus belonging to the alpha herpes viridae family causing both primary and recurrent infection. Chicken pox (varicella) is a generalized primary infection.[2] After the primary infection, it remains latent in the cranial nerve, dorsal root, and autonomic nervous system ganglia along the entire neuraxis.

CASE REPORT: A 60-yeasr-old male patient reported to the department of

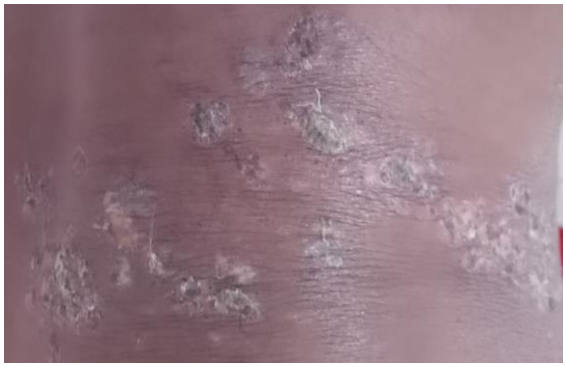
dermatology with the chief complaint of patient was apparently 4days back after patient developed pain over right side of the abdomen with simultaneously onset of multiple fluid filled vesicles at right side of abdomen, gradually involving lateral and post aspect of abdomen in 2days. Multiple fluid lesions are over anterior aspect at abdomen right side for $T_{7,}T_{8,}T_{9}$ dermatosses, Itching lesions with erythematous border, since 6months post inflammatory hyper pigmentation [tenia corporis]. Patient was previously treated with anti-fungal for 10days from a dermatologist in Kurnool. Patient was advised to have a bland diet, adequate hydration; Tab. acyclovir (800 mg) five times daily for 5 days, tab. levosis(10 mg) two times daily for 5 days, Inj.tramadol, tab.terbest 250mg for 5days, and topical application of calamine lotion on facial lesions 4–5 times daily were advised. Patient was recalled after 5 days. On first follow-up, extraoral and intraoral lesions had healed by 50%, healing was appreciated with the formation of dry scabs on extraoral lesions ,pain subsided by 30%; acyclovir was continued for 5 more days. On second follow-up, extraoral and intraoral lesions almost resolved, and acyclovir was discontinued in a tapering manner. The patient was advised regular follow-up.



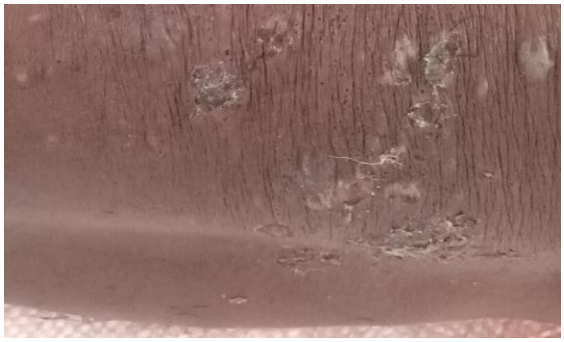
Multiple vesicles & lessions

DISCUSSION: HZ of the trigeminal nerve is a disease that falls within the diagnostic purview of all dentists and dental specialists. In 1892, Von Bokay[3]first suggested the relationship between the etiologies of varicella and HZ. The first suggestion was made by Garland and Hope-Simpson[4] that HZ is caused by reactivation of latent virus acquired during varicella. Initially, the lesions in the affected dermatome are clustered nerve. Most of the infections affect dermatomes of T3 to L2, however, approximately 13% of the patients present with infections involving any of the three branches of the trigeminal nerve[5].A diagnosis of HZ is made by detailed clinical examination and confirmed by lab diagnosis by doing a Tzanck smear of scrapings from the floor of the vesicles that reveal multinucleated giant cells on direct microscopy. The other methods are by direct fluorescent antibody tests, presence of high or rising titers to VZV, or by culture studies.[6] Direct fluorescent monoclonal antibody test or detection of serum specific IgM by the indirect fluorescent antibody method is also used to confirm HZ. Ideally, in childhood HZ, lymphocyte counts, CD4/CD8 ratio, and serum immunoglobulin levels have also to be estimated to rule out undetected concurrent immunosuppression. The severity of clinical manifestations of HZ is mainly dependent on the age of the child and CD 4 count even though HIV serology is positive as seen in our study where the CD 4 count (immune status) was above 350/cumm. HIV infection may not lead to AIDS by taking care of one's immune system from the beginning by changing lifestyle such as fresh air, protected water, balanced diet, sound sleep and positive attitude. Malnutrition is one of the earliest and major complications of HIV infection and a significant factor in advanced disease. If we prevent NAIDS (nutritionally acquired immunodeficiency syndrome), we can postpone AIDS though they are HIV reactive. HZ follows a prodromal, active and chronic stage. [7]Classically it presents with a prodrome of mild-to-moderate burning or tingling (or in some cases numbness) in the skin of a given dermatome, often

associated with fever, headache, general malaise and stomach upset. Similar findings were reported in both our cases, however, stomach upset was not reported. In a duration of about 48–72 h from the prodrome, there is development of a unilateral erythematous, maculopapular rash along the dermatome, which eventually develops into a vesicular lesion, this represents the active stage.[8]



FIRST FOLLOW UP



SECOND FOLLOW UP

TREATMENT:

Treatment options are based on the patient's age, immune status, duration of symptoms, and presentation. Several studies indicate that antiviral medications decreased the duration of symptoms and the likelihood of post herpetic neuralgia, especially when initiated within 3 days of the onset of rash. In children between the age group of 2 and 12 years, who are otherwise healthy, oral acyclovir need not be prescribed. An important study by Kubeyinje suggested that the use of acyclovir in healthy young children with zoster is not clearly justified, especially *in situ* ations of limited economic resources.[9]Acyclovir has been the drug of choice; given in a dosage of 800 mg four times a day for 10 days. Recently, newer forms of antiviral drugs have been developed, specifically to address the acute stage of HZ (Famciclovir) and for use in immune- competent patients (Valacyclovir). In the former, the dosage is 500 mg every 8 h for 7 days; for the latter it is 1 g three times daily for 7 days. Acyclovir (800 mg, 5 times/day for 7–10 days) shortens the duration of viral shedding, inhibits the formation of new lesions, hastens healing and reduces the severity of acute pain. Variable benefits are recorded with respect to reduction in the frequency and duration of PHN.[10]

The first line of therapy in childhood HZ is oral acyclovir, given at a dose of 20–40 mg/kg body weight, four times a day. Patients with HIV infection are at risk of developing severe illness from either varicella or zoster. Progressive primary varicella, a syndrome with persistent new lesion formation and visceral dissemination, may occur in HIV-infected patients and may be life threatening. Though many studies have been done in adult HIV patients, so far there are only few case reports of childhood HIV patients acquiring zoster.[11]

CONCLUSION:

Herpes zoster is an uncommon disease in childhood. Varicella in early childhood is a risk factor for herpes zoster either in immunocompromised or immunocompetent children. Childhood zoster occurs in either healthy or underlying immunodeficient children. The appearance of herpes zoster in a young child does not always imply an underlying immunodeficiency or malignancy. But the identification of herpes zoster with or without immunodeficiency is of prime importance for the treatment and prognostic point of view and should be considered in the differential diagnosis of

vesicular eruptions. The prognosis was generally good both in healthy and in HIV-reactive children with CD 4 count above 350/cumm in our study.

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