DERMATOMAL AND DISSEMINATED VARICELLA ZOSTER LESIONS IN A HIV INFECTED INDIVIDUAL (A Case Report)

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Introduction

Varicella infection is known to manifest in two recognized forms. Chicken pox is usually seen in young, non-immunised individuals; whereas herpes zoster, the other form due to reactivation of the dormant virus, occurs in adults. A depression in the level of cell-mediated response has been implicated [1]. Various studies have shown herpes zoster to be an early indicator for the development of acquired immunodeficiency syndrome (AIDS) in high risk individuals [2,3]. In the context of the AIDS pandemic, herpes zoster in an individual who is below 60 years, and at risk, demands that a concomitant HIV infection be ruled out. In a study from Central Africa, the attack rate of herpes zoster in patients with HIV infection was found to be 11%, and more significantly, 91% of individuals who had herpes zoster were HIV sero-positive [3].

Herpes zoster also manifests in a disseminated form. This has been reported in American patients with AIDS [3,4]. It occurs in 6-26% of immunosuppressed patients, 4-11 days after the onset of localized zoster [5]. We report an interesting case of dermatomal and disseminated varicellar zoster in an immunocompromised African patient when dissemination occurred two weeks after the healing of the dermatomal rash.

CASE REPORT

A 26-year-old African male from southern African region presented with a painful rash of 2 days duration over the left side of the chest. There was a prodrome of malaise, slight headache, itching, and mild pain over the affected part. Examination revealed circumscribed vesicular lesions, with an erythematous background, typical of herpes zoster, affecting T8-T10 dermatomes on the left hemithorax. Leading questions excluded a history of acute sero-conversion illness. The patient however gave a history suggestive of gonococcal urethritis three years ago. There were no other associated clinical markers of HIV infection in the form of oral candidiasis, oral hairy leucoplakia, seborrhoeic dermatitis, pyoderma, superficial fungal infections, or lymphadenopathy. There were no genital ulcers or warts and systemic examination of the patient was essentially normal. He was managed symptomatically and nursed in isolation. The rash healed without any complications in 2 weeks.

Remaining asymptomatic for a further 2 weeks thereafter, he returned with a generalized rash. This rash was pleomorphic, exhibiting multiple maculopapules and vesicles. They were superficial, centripetal in distribution, more on the face, trunk, upper arms and thighs (Fig). This episode was unaccompanied by any prodrome or fever. Patient was closely monitored but managed symptomatically. The rash subsided in 5 days with no complications. There was mild scarring which rapidly faded within a month.

Blood examination revealed WBC 5100/cumm; lymphocytes 34.4%, monocytes 4.9%, granulocytes 60.7%, basophils 0.2%; RBC 5.37 million/cumm; Hb 14.4 g/dL; Hct 44.2; MCV 82.4 mm³; MCH 26.8 pg; MCHC 32.5%; RDW 14.2 mm³; Plt 133 million/cumm. Absolute lymphocyte count was 1754/cumm. Urine examination revealed mild albuminuria which disappeared after a week. Though the Mantoux tests reading was 12 mm, radiogram chest was normal. Liver function tests were within normal limits. HIV screening by ELISA technique was positive, which was confirmed by Western Blot

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Fig. 1 : Disseminated varicella zoster lesions in a HIV infected individual.

test. CD4/CD8 counts could not be done due to lack of facilities.

Discussion

For over a century, clinical association between varicella and herpes zoster has been recognized. Herpes zoster usually follows a reactivation of the latent viruses [1,4]. Other modes like an exogenously acquired infection [5,6], and reinfection with varicella-zoster virus, which could be silent or clinically apparent, are also described [5,7]. Reactivation of herpes zoster is seen in 10-20% of patients with HIV infection. This reactivation syndrome of varicella-zoster virus is an indication of modest decline in immune function and is often the first clinical indication of immune deficiency in the individuals at risk [2]. The lesions may extend over several dermatomes and frank dissemination may also be seen occasionally.

Our patient presented with a dermatomal rash of herpes zoster, which had subsided completely after 14 days. Thereafter, as if under remission, he presented with a disseminated form resembling chicken pox 2 weeks after recovery from shingles. He had remained totally asymptomatic in the intervening period. Interestingly, this interval between the two episodes of rash fits in with the established incubation period of chicken pox. Probably the immunocompromised state of the patient resulted in viraemia that was sustained, and after its initial

manifestation as a dermatomal involvement, was followed by a disseminated form later on.

Herpes zoster in an immunocompromised state following HIV infection is not that severe as seen in other immune deficiency states, where the mortality has been rated 6-17% [3,5]. Our patient became asymptomatic quite early, within 5 days of the disseminated illness, and no complications were detected except mild and asymptomatic proteinuria that was self-limiting. He was not given any antiviral therapy and was managed conservatively.

Herpes zoster remains one of the earliest premonitory disease heralding the onset of AIDS within a span of 8 years in the HIV sero-positives [2]. It should be realized that when only 10% of the general population above 15 years of age remain unexposed to a primary infection, varicella infection, in an adult from an AIDS endemic region, is highly suggestive of an underlying immunodeficient state. The dermatomal, and rarely the disseminated form, may be encountered, which will usually require only a conservative management support by counselling.

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