Case Report

Herpes Zoster with Oral Manifestations: A Case Report

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ABSTRACT

A case of herpes zoster limited to the left palate and gingiva in the absence of concurrent skin lesions or predisposing systemic disease is presented. Because of the relative rarity of isolated mucous membrane lesions of this viral disease, the tendency for intraoral vesicles to break down and ulcerate early was seen. Cytological smear and immunological testing confirmed the diagnosis.

INTRODUCTION

Herpes Zoster (HZ) is an acute infectious viral disease of an extremely painful and incapacitating nature which is characterized by inflammation of dorsal root ganglia, or extramedullary cranial nerve ganglia, associated with vesicular eruptions of the skin or mucous membranes in areas supplied by the affected sensory nerves. Varicella-zoster virus (VZV) is the agent causing chickenpox, the common childhood infection. Following resolution of chickenpox, VZV lies dormant in the spinal dorsal root ganglia until reactivation results in herpes zoster (shingles). "Shingles" is a syndrome characterized by a painful, unilateral vesicular rash usually restricted to a dermatomal distribution. At times, especially in the immunosuppressed patient, the infection may spread and produce severe systemic illness with involvement of multiple visceral organs and multiple dermatomes (disseminated zoster).[1,2,3,4]

CASE REPORT

A 40-year-old female presented with multiple, pruritic blistering eruptions of two days duration in her mouth associated with fever. The lesions were associated with severe pain along with difficulty in chewing food. Family history was non contributory.

Cutaneous examination revealed no signs of any lesions on face and other parts of the body. The patient was febrile (100ºF) and had multiple enlarged, discrete, nontender lymph nodes measuring about 0.5 cm to 1cm in the left submandibular region. Intra orally, multiple ulcers with irregular borders and pseudomembranous covering were present limited to the left maxillary labial mucosa, palatal mucosa and gingival not crossing the midline (Fig. 1,2). Fetor oris was present.

Cytological smears stained with H&E on microscopic examination showed altered, acantholytic epithelial cells and few multinucleated giant cells. Serology for HIV and syphilis was nonreactive. Immunologic studies showed antibodies against herpes zoster. The patient was diagnosed to have herpes zoster and was treated with oral acyclovir 800 mg 5 times a day for 7 days along with NSAIDS, in addition to the supportive nutritional supplements. Complete resolution occurred without sequelae.

DISCUSSION

Exactly why VZV reactives from latency is not fully understood. However, VZV-specific cell-mediated immunity has

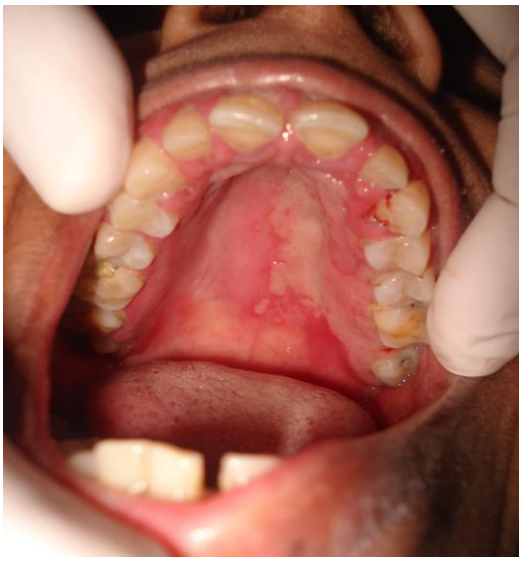

FIG 1: *showing intraoral involvement of HZ on palate*

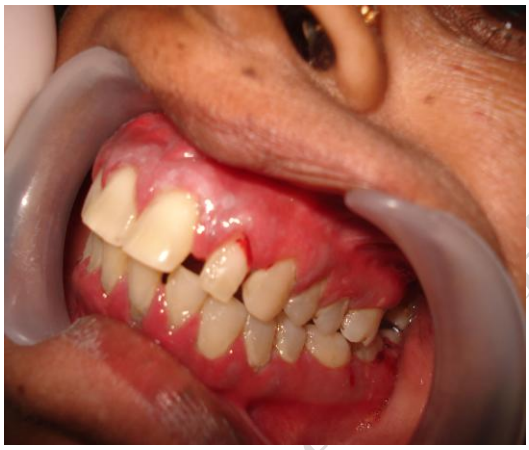

FIG 2

FIG 2: *showing gingival involvement of HZ*

shown to be a major factor in determining reactivation of VZV. Cell-mediated VZV-specific immunity decreases with age and in patients with certain malignancies. These groups have much higher rates of herpes zoster This supports the concept of an important role for cell-mediated immunity in the pathogenesis of VZV infection.[1,2]

VZV reactivation causes inflammation in the dorsal root ganglion accompanied by hemorrhagic necrosis of nerve cells. The result is neuronal loss and fibrosis. The distribution of the rash corresponds to the sensory fields of the infected neurons within a specific ganglion. The anatomic location of the involved dermatome often determines the specific manifestations.[1,2,3,4]

The primary physical finding is a rash in a unilateral dermatomal distribution; the rash may be erythematous, vesicular, pustular, or crusting, depending on the stage of disease. The initial rash is typically "herpetic" in appearance: small vesicles grouped on an erythematous base. It has been described as "dew drops on a rose petal." Bilateral rash is rare. Zoster lesions occur simultaneously and remain in congruent stages of healing. Lesions on the tip of the nose signify involvement of the nasociliary nerve; this finding mandates slit-lamp examination with fluorescein stain to look for the dendritic corneal lesions of herpetic keratitis.[1,2,6]

Oral manifestations results from trigeminal nerve involvement. The oral lesions are extremely painful and they rupture to leave areas of eruptions. Herpes zoster occurring during tooth formation causes pulpal necrosis and internal root resorption. In the present case HZ affected only the oral cavity with lesions mainly involving the palate and gingiva.[4,6,7]

Zoster-associated complications include neurological components such as Gullian-Barre Syndrome, encephalitis, myelitis, Ramsey - Hunt Syndrome and Horner's syndrome. Generally, ocular complications involve ulcerations, hemorrhage, conjunctivitis and optic neuritis. Additionally, cutaneous scarring and bacterial infections are common occurrences.[8]

Patients with herpes zoster usually experience pain. Antiviral and steroid therapy provides relatively minor relief of pain, and narcotic analgesics are often needed. Initial therapy may include nonsteroidal anti-inflammatory drugs (NSAIDs).The goals of antiviral therapy are to decrease pain, to promote healing of skin lesions, and to prevent or reduce the severity of postherpetic neuralgia. Acyclovir and the newer antivirals valacyclovir and famciclovir have been shown to be effective if given within 48-72 hours of the

appearance of the rash. The newer agents have better bioavailability and do not need to be given as frequently. Outcomes studied have included time to crusting of skin lesions, duration and severity of acute pain, and duration and incidence of postherpetic neuralgia.[9,10]

Acyclovir has been the most studied and widely recommended, but in a blinded, randomized comparison trial, valacyclovir was shown to be superior to acyclovir. The trial included more than 1100 patients with uncomplicated zoster who were 50 years or older. Adverse effects were similar in both groups.

The duration of antiviral treatment in studies has varied from 7-21 days. Based on current literature, for immunocompetent patients, acyclovir for 7-10 days or a 7-day course of the newer agents is appropriate. Longer courses may be needed in the immunocompromised patients.

The addition of corticosteroids has been evaluated in patients treated with acyclovir. The benefit of steroids included accelerated healing of lesions and more rapid resolution of acute pain. [9,10]

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