A Lady with Left Sided Ophthalmic Nerve Distribution Rash…….A Clinical Diagnosis!!!!
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Abstract
A 26-year-old lady was admitted with a sudden onset of lesions that started on the left side of the face. Past medical history revealed a mild atopic eczema for several years. Inspection showed unilateral infection of the upper half of the face. The lesions consisted of erythematous macules with scaling. Examination of the oral cavity was uneventful, whereas cervical lymph nodes were slightly enlarged. Lesional pruritus was mild. The case is discussed in this report.

WHAT IS YOUR DIAGNOSIS?
Herpes Zoster infecting the ophthalmic division of Trigeminal Nerve

HERPES ZOSTER OPHTHALMICUS….A CONCISE REVIEW
INTRODUCTION
Varicella-zoster virus (VZV) is a member of the Herpesviridae family. It is the etiologic agent of varicella (chickenpox), the primary infection, and herpes zoster, the reactivation. Herpes zoster ophthalmicus involves the tissues innervated by the ophthalmic division of the trigeminal nerve and accounts for 10-25% of all cases of shingles. The sequelae of herpes zoster ophthalmicus can be devastating and include chronic ocular inflammation, visual loss, and debilitating pain.

>PATHO-PHYSIOLOGY
The frequency of dermatologic involvement in herpes zoster is similar to the centripetal distribution of the initial varicella lesions. This pattern may suggest that the latency arises from the contiguous spread of the virus during varicella viremic phase from infected skin cells to sensory nerve endings with subsequent ascent to the ganglia. It also may suggest that the ganglia are infected hematogenously during the viremic phase of varicella and that the frequency of the dermatome involvement in herpes zoster reflects the ganglia most often exposed to reactivating stimuli.

In immunocompetent patients, specific antibodies (immunoglobulins G, M, and A) appear more rapidly and
reach higher titers during reactivation (herpes zoster) than during the primary infection.

The appearance of the cutaneous rash due to herpes zoster coincides with a profound VZV-specific T-cell proliferation. Interferon-alpha production appears with the resolution of herpes zoster. The patient has a long-lasting, enhanced, cell-mediated immunity response to VZV.

**TREATMENT**

Treatment of acute herpes zoster ophthalmicus is optimal if started within 72 hours of rash onset.

Oral antiviral drugs (e.g. Famiciclovir 500 mg 3 times/d [tid], valacyclovir 1 g 3 times/d [tid], or acyclovir 800 mg 5 times/d for 7 d).

Tricyclic antidepressants nortriptyline, amitriptyline, or desipramine 25 mg, adjust up to 75 mg at bedtime (qhs) for several weeks if needed (to inhibit acute and prolonged postherpetic neuralgia [PHN]).

Additionally topical corticosteroids, antibiotics, cycloplegics and antivirals can also be used. Glaucoma medications; as necessary for keratitis, iritis, or glaucoma.

Treat late PHN with tricycles antidepressants (as listed above) and/or capsaicin ointment daily (qid) or 4 times/day (qid) or lidocaine patches. Neurontin 30-600 mg by mouth (PO) tid and/or OxyContin SR 10-20 mg PO 2 times/day (bid) with topical medications similar to those used in acute disease.

In a small study by Kanai et al, lidocaine 4% ophthalmic drops were administered to 24 patients with ophthalmic post-herpetic neuralgia in a crossover manner. A significant reduction in eye and forehead pain was observed in patients who were administered the lidocaine ophthalmic drops. Analgesic onset was noted via a visual analog scale within 15 minutes after administration and persisted for a median of 36 hours (range, 8-96 h).

**References**
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