CLINICAL IMAGE

FACIAL RASH WITH EYE SWELLING

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INTRODUCTION

A 60-year-old male presents to the office for a painful rash on his forehead and scalp associated with periorbital swelling. Initially, the patient noticed a slight headache on his head's right parietal aspect, described as aching and constant. Two days later, he developed blister-like lesions on his right upper forehead and scalp. These lesions subsequently spread to his face and nose, causing his right eye to become swollen (Figures 1 and 2). He denies any associated vision changes, but his eye was swollen shut and he noted that his conjunctiva became erythematous. The patient had obtained a shingles vaccination 10 years ago and recalled having chickenpox as a child around the age of seven. The visual acuity check was within normal limits. He denied headache, fever, trauma, known sick contacts or previous episodes of similar presentation.

FIGURE 1:

Swollen eye and rash, left side

FIGURE 2:

Swollen eye and rash, straight on

QUESTIONS:

1. What is the most likely diagnosis?

A. Bacterial Conjunctivitis

B. Contact Dermatitis

C. Herpes Simplex Keratitis

D. Herpes Zoster Ophthalmicus

E. Ramsay Hunt Syndrome

2. What is the preferred initial treatment?

A. Acyclovir

B. Amoxicillin

C. Clotrimazole

D. Prednisone

E. Reassurance

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3. What is the most likely causative agent?

A. Dimorphic Fungi

B. Double-Stranded DNA

C. Gram-Negative Bacilli

D. Gram-Positive Cocci

E. Positive-sense RNA

ANSWERS:

1. What is the most likely diagnosis?

Correct Answer:

D) Herpes Zoster Ophthalmicus

The lesions are present in a dermatomal distribution, along with the ophthalmic division of the trigeminal nerve. The patient's rash is vesicular, and there is also ocular involvement consistent with herpes zoster ophthalmicus.[1] Contact dermatitis is incorrect because the patient had no known exposures to irritants or allergens. The patient also has ocular involvement, which is not necessarily caused by contact dermatitis.[2] Herpes simplex keratitis is incorrect because the rash is presenting in a dermatomal distribution. Herpes simplex keratitis can cause a corneal infection and may cause periocular lesions, but this patient's rash expands beyond this region. Reactivation of the varicella-zoster virus also causes Ramsay Hunt syndrome.[2] The vesicular rash on the ear or in the mouth may be accompanied by a facial nerve palsy and hearing loss.[3] Bacterial conjunctivitis would typically present with conjunctival erythema, discharge, irritation, eyelid crusting and periorbital swelling.[2]

2. What is the preferred initial treatment?

Correct Answer:

A) Acyclovir

The preferred treatment is acyclovir. It is recommended to begin an antiviral medication- such as acyclovir, valacyclovir or famciclovir - within 72 hours of onset.[4] Treatment has been shown to speed the resolution of skin lesions, reduce viral shedding and decrease the incidence of dendritic and stromal keratitis as well as anterior uveitis.[4] Amoxicillin would be used for a bacterial infection and topical clotrimazole for a fungal infection. Corticosteroids will be indicated if there are acute retinal necrosis symptoms, including blurred vision and pain in the eye.[4]

3. What is the most likely causative agent?

Correct Answer:

B) Double-Stranded DNA

The diagnosis is herpes zoster ophthalmicus and is caused by the varicella-zoster virus. Herpes zoster is an enveloped virus that contains double-stranded DNA.[5] Gram-negative bacilli is incorrect, with examples of gram-negative bacilli include Escherichia coli, Pseudomonas aeruginosa and Bacteroides fragilis.[5] Gram-Positive cocci would include Staphylococcus aureus, Streptococcus pyogenes and Streptococcus pneumoniae.[5] These organisms will not cause a vesicular skin reaction such as this. Positive-sense RNA viruses include those of the Picornaviridae family and Flaviviridae family.[5] These viruses will not cause this presentation. Picornaviridae viruses include diseases such as Poliovirus, Coxsackievirus and Rhinovirus. Flaviviridae viruses include Yellow fever virus, West Nile virus, Dengue virus and Zika virus.[5] Dimorphic fungi include Candida albicans, Blastomyces dermatitidis and Histoplasma capsulitis.[5] DNA viruses replicate in the nucleus using host cell polymerases, except poxviruses.[5] Many DNA viruses, including HSV can establish latent infections and later reactivate.[5] In contrast, RNA viruses replicate in the cytoplasm, except for orthomyxoviruses and retroviruses.[5]

DISCUSSION

There are one million estimated cases of herpes zoster in the U.S. each year, with approximately 50% occurring in individuals 50 years and older.[6] Only 10–20% of these infections are diagnosed as herpes zoster ophthalmicus, with an overall lifetime risk of herpes zoster ophthalmicus of 1%.[7] Herpes zoster is a very prevalent infection and is also known as shingles, a reactivation of the varicella-zoster virus; it is part of the herpes virus family.[8] In those that have previously been infected with the virus, it remains latent in the dorsal root ganglia. When the virus is reactivated, it typically results in a vesicular eruption along a specific dermatome that does not cross the midline. There can also be associated symptoms, including a viral prodrome of flu-like symptoms, such as fever and the disease's early stages. Postherpetic neuralgia is a common residual side effect of the varicella-zoster virus. This is a pain in the corresponding infected dermatome for months to years after the infection and appears to be due to damage of the nerve root.[9]

Herpes zoster ophthalmicus occurs when the varicella-zoster virus is reactivated from the trigeminal nerve, specifically the ophthalmic division.[10] Herpes zoster ophthalmicus typically has a prodromal period including fatigue and fever that appears up to one week before the rash appears.[11] There is a pain in the distribution of the ophthalmic nerve in 60% of patients.[12] It presents as fluid-filled vesicles that are unilateral, presenting over five to seven days.[9] Seventy-one percent of individuals with herpes-zoster ophthalmicus develop ocular complications.[13] Examples of ocular complications include corneal disease, uveitis, scleritis and ocular motor palsies.[13] If there are vision changes, then there should be a referral to an ophthalmologist for further evaluation.[14] It is important to check patients for Hutchinson's sign, which includes vesicles on the tip or side of the nose. When these vesicles are present, there is a high risk of ocular complications because of the shared innervation of the nasociliary branch of V1.[14]

Herpes zoster is typically diagnosed based on clinical assessment, but there are confirmatory tests such as immunofluorescence, immunoperoxidase staining and serological diagnosis. The U.S. Centers for Disease Control and Prevention (CDC) states that

polymerase chain reaction (PCR) is the most useful test when using laboratory testing to diagnose herpes zoster.[15] The specimen's ideal sampling is obtained by swabbing unroofed vesicular lesions and scabs from the lesions.[15] Samples are recommended to be collected in the first days of illness.[16] Clinical diagnosis is usually enough to diagnose, but laboratory methods, such as PCR, are useful in situations in the absence of a rash.[15] Clinical judgment is an effective method of diagnosis of herpes zoster. The rash presents unilaterally, which helps physicians make the diagnosis. In a study with 272 participants, clinical diagnosis was confirmed in 91% of patients with signs and symptoms of herpes zoster.[17]

The vaccine for herpes zoster can be effective in preventing the reactivation of the virus and a reduction in prodrome. The CDC recommendation is that healthy adults aged 50 or older should receive two doses of the Shingrix® vaccine separated by two to six months.[18] In a case study of 266 herpes zoster patients and 362 matched controls, the vaccine was 54% effective at preventing herpes zoster in persons age 60 and older over three years following vaccination.[19] There is also a reduction in the severity of prodromal discomfort as well as post herpetic neuralgia in patients who experience a herpes zoster event after having a vaccination.[19]

Herpes zoster ophthalmicus infections should be treated within 72 hours of the rash onset using acyclovir, valacyclovir or famciclovir.[4] Early treatment improves skin lesions' healing time, reduces viral shedding and decreases keratitis and anterior uveitis incidence.[4] Without antiviral treatment, half of the patients with herpes zoster ophthalmicus will develop an eye disorder.[20] Treatment with antivirals reduces the percentage of patients with eye disorders from 50% to 20–30%.[20] A patient who has Hutchinson's sign, visual complaints or an unexplained red eye indicates referral to an ophthalmologist.[20] Patients who develop uveitis or keratitis may require a topical corticosteroid such as prednisolone. If the patient has increased intraocular pressure, the patient should receive a topical corticosteroid and aqueous suppressant.[21]

AUTHOR DISCLOSURES:

No relevant financial affiliations. Lindsay Tjiattas-Saleski, DO, MBA, FACOEP, is a member of the ACOFP Editorial Committee. If the authors used any personal details or images of patients or research subjects, written permission or consent from the patient has been obtained. This work was not supported by any outside funding.

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