PHOTO ROUNDS

A young girl with blisters on her forehead

5-year-old girl came into the office complaining of severe burning and tingling sensation of her right forehead. She'd had fever, chills, myalgia, and a relentless headache for 3 days. The morning of her appointment, a few "bumps" and water-filled blisters began to appear on the right side of the forehead; the lesions then started to multiply and grow (FIGURE). The patient's mother expressed concern over the rapid development of the lesions, which were accompanied by marked edema of the forehead and right evelid. The mother indicated that no one else in the family was affected at the time of presentation.

The child appeared ill and pale at the time of presentation, but there was no history of immediate antecedent illness or any drug intake prior to the current eruption. Her growth and development were in the lower normal range. The child had a history of recurrent bacterial infections; she was not vaccinated for varicella. There was, however, a personal (and family) history of varicella when the child was about 3 years old.

On physical examination, there were multiple vesicles and bullae that varied in size from 2 mm to 1 cm in diameter on the right side of the forehead; there were areas of dried yellowish serous exudates limited to the right eyebrow. The forehead and periorbital areas were edematous with underlying erythematous skin. The entire eruption appeared to be restricted to the right upper part of the face, extending from the eyelid and medial canthus up to the frontal scalp. On close examination, several vesicles and crusts were present on the right side of the nasal tip. Serology for HIV was negative.

What is your diagnosis?

How would you treat?

FIGURE

Vesicles and bullae on forehead



This 5-year-old had significant edema of the forehead and periorbital areas. The entire eruption was restricted to the right upper part of the face, and extended from the eyelid and medial canthus to the frontal scalp. Note the presence of vesicles and crust on the nasal tip.

Khashayar Sarabi, MD Loma Linda University Medical School, Loma Linda, Calif

Abdulhafez Selim, MD, PhD King Fahd University of Petroleum and Minerals, Dhahran, Saudi Arabia

Amor Khachemoune, MD, CWS

Ronald O. Perelman Department of Dermatology, New York University School of Medicine, New York

amorkh@pol.net

FEATURE EDITOR

Richard P. Usatine, MD University of Texas Health Science Center at San Antonio

FAST TRACK

Without antiviral treatment, patients have a 50% chance of eye complications such as iritis and keratitis

Diagnosis: Herpes zoster ophthalmicus

This case of herpes zoster ophthalmicus (HZO) was unusual—not because of the way the patient presented, but because of her age. This condition is rarely seen in children, though it is not uncommon in adults—specifically, patients who are 60 years of age and older.¹⁻⁴

Herpes zoster ophthalmicus, which refers to the involvement of the ophthalmic branch of the trigeminal nerve or the fifth cranial nerve, usually manifests with a typical vesicular or bullous eruption of the forehead, often in unilateral fashion. The lesions of herpes zoster (shingles) are similar to those of varicella (chickenpox) but in herpes zoster, painful unilateral vesicular eruptions are usually limited to one dermatome. (If hematogenous dissemination occurs, more than 20 vesicles will form in skin areas away from the affected dermatome.¹)

Patients typically present with fever, headache, and abnormal sensations that precede the development of cutaneous lesions by a few days. The eruptions may be pustular and hemorrhagic initially, and within 10 days evolve into crusts.^{5,6}

Differential diagnosis: Pain sets HZO apart

In its classical presentation, HZO does not pose a diagnostic challenge for practitioners well-versed in skin disease management. The clinical differential diagnosis of herpes zoster will include other causes of blisters or vesicular eruptions of autoimmune etiologies, viral infections, or hypersensitivity reactions.

The most common blistering diseases that may be mistaken for zoster include herpes simplex, contact dermatitis, erythema multiforme, and cellulitis. The prodromal stage and the characteristics of pain usually set herpes zoster apart from the other diagnoses.

Beware of complications

Eye complications. HZO may result in paralytic ptosis, conjunctivitis, keratitis, cataracts, glaucoma, retinitis, and optic neuritis and atrophy.^{2,5,7}

Herpetic lesions on the tip of the nose are believed to herald ocular involvement and may precede typical dermatomal eruption of HZO.² Such cutaneous involvement along the distribution of the nasociliary nerve is called Hutchinson's sign. It should prompt a complete ophthalmologic evaluation, as it did with our patient.

Neurologic complications. The most common complication of herpes zoster infection in general is postherpetic neuralgia (PHN), which results in severe pain that persists for months—or years—after the skin lesions have completely healed. Patients over 70 have a 70% chance of developing PHN.^{4,7}

Neurological complications such as encephalitis, myelitis, and Guillain-Barré syndrome have also been reported.

Other complications. Other complications include secondary bacterial infections, pneumonitis, and polyradiculitis.

Management: Antivirals and pain meds

Patients with HZO have a 50% chance of having eye complications (iritis and keratitis) without antiviral treatment.^{2,5,6} Therefore, treatment is recommended for all HZO patients.

Antivirals ASAP

Start antiviral drugs within 72 hours of clinical presentation, and when new lesions are still appearing on the skin, to achieve optimal effect. Acyclovir, famciclovir, and valacyclovir are the antivirals of choice.⁶ The usual dosages for adults are: acyclovir (800 mg orally 5 times per day for 7–10 days), valacyclovir (1000 mg orally 3 times daily for 7 days) and famciclovir (500 mg orally 3 times daily for 7 days). The suggested dose of acyclovir for children is 10 to 20 mg/kg/dose qid for 5 days; not to exceed 800 mg per day.^{2,5,6}

Preventing postherpetic neuralgia

The role of systemic corticosteroids in the prevention of PHN, decreasing duration and severity of the acute symptoms in the initial days of herpes zoster infection, remains controversial.⁸

Immunosuppressed individuals may be treated with acyclovir, interferonalpha, and vidarabine. In this population the live, attenuated vaccine is safer and is preferred to the varicella zoster immunoglobulins (VZIG).^{1,6,8,9}

PHN can be reduced by treating the patient within the first 24 hours of symptom onset. Pain usually resolves within 3 months in 50% of patients and within 1 year in 75% of patients.^{1,6,7}

Pain therapies for PHN

Therapeutic approaches to the management of PHN include topical anesthetic creams (lidocaine plus prilocaine), capsaicin, and oral medications such as tricyclic antidepressants (amitriptyline and desipramine), carbamazepine, and gabapentin, as well as nerve blocks.^{6,7}

Gloves and handwashing are key for caregivers

Individuals who have not had a confirmed varicella infection should avoid contact with those who have shingles unless a varicella zoster virus antibody is satisfactory and shows immunity.^{6,7}

Gloves should be worn when touching the lesions or infectious drainage, and hands should be washed after glove removal.⁵⁻⁹

Patients with disseminated disease require hospitalization with airborne precautions, which include a negative air pressure room and ensuring that caregivers wear N95 respirators.

What put our patient at risk?

In the case of our young patient, low immunity may have played a role in her contracting HZO. She had a history of recurrent bacterial infections and suffered from generally poor health.

She was referred to an ophthalmologist, who did not find any dendritic corneal ulcer or other complications on weekly follow up. The patient was successfully managed with oral acyclovir 10 mg/kg/dose for 5 days, and she responded well to treatment. We also prescribed acyclovir eye ointment, and local wet compresses with good outcome.

Correspondence

Amor Khachemoune, MD, CWS, Ronald O. Perelman Department of Dermatology, New York University School of Medicine, 530 First Avenue, Suite 7R, New York, NY 10016; amorkh@pol.net

Acknowledgments

The authors thank Dr Shahbaz Janjua for his assistance in the preparation of this manuscript.

Disclosure

No potential conflict of interest relevant to this article was reported.

References

- Trent JT, Kirsner RS. Herpesvirus infections and herpetic wounds. *Adv Skin Wound Care* 2003; 16:236–243.
- Zaal MJ, Volker-Dieben HJ, D'Amaro J. Prognostic value of Hutchinson's sign in acute herpes zoster ophthalmicus. *Graefes Arch Clin Exp Ophthalmol* 2003; 241:187–191.
- Binder NR, Holland GN, Hosea S, Silverberg ML. Herpes zoster ophthalmicus in an otherwisehealthy child. J AAPOS 2005; 9:597–598.
- 4. Gebo KA, Kalyani R, Moore RD, Polydefkis MJ. The incidence of, risk factors for, and sequelae of herpes zoster among HIV patients in the highly active antiretroviral therapy era. *JAIDS* 2005; 40:169–174.
- 5. Liesegang TJ. Herpes zoster virus infection. *Curr Opin Ophthalmol* 2004; 15:531–536.
- 6. McCrary ML, Severson J, Tyring SK. Varicella zoster virus. *J Am Acad Dermatol* 1999; 41:1–14.
- 7. Cohen JI, Brunell PA, Straus SE, Krause PR. Recent advances in varicella-zoster virus infection. *Ann Intern Med* 1999; 130:922–932.
- Santee JA. Corticosteroids for herpes zoster: what do they accomplish? Am J Clin Dermatol 2002; 3:517–524.
- Oxman MN, Levin MJ, Johnson GR, et al. A vaccine to prevent herpes zoster and postherpetic neuralgia in older adults. *N Engl J Med* 2005; 352:2271–2284.

FAST TRACK

Low immunity may have played a role in our young patient contracting herpes zoster ophthalmicus