

Disseminated herpes zoster ophthalmicus in an immunocompetent 8-year old boy

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Abstract

Varicella results from a primary infection with the varicella virus while herpes zoster is caused by a reactivation of a latent infection. Dissemination of herpes zoster is uncommon in immunocompetent individuals. Reports of disseminated herpes zoster in children are even less common than in adults. An unusual case of disseminated herpes zoster ophthalmicus in an 8-year old immunocompetent black boy is presented. He had a previous primary *Varicella zoster* virus infection at three years of age. In the current report, he presented during an on-going chicken pox outbreak and survived with no significant complications. A breakthrough varicella virus re-infection or a reactivation is possible, both of which could present as zoster. This case emphasizes the need for prevention of varicella virus infection through universal childhood immunization and effective infection control strategies in health care settings.

Introduction

Varicella zoster virus (VZV) is a neurotropic herpes virus that causes two distinct diseases. Varicella (chicken pox) results from a primary infection with this virus. However, *Herpes zoster* (HZ) is caused by reactivation of VZV resulting from a reactivation of a latent infection during which the virus migrates from the dorsal root ganglion along the neural pathways to the skin causing unilateral vesicular rash which usually is limited to a single or rarely two adjacent dermatomes.¹ It is said to be disseminated when there are more than 20 lesions outside the primary and adjacent affected dermatome.^{2,3}

Age is the most important risk factor for VZV reactivation. Hope-Simpson reported a 10 times increase in incidence in adults aged 80 to 89 years when compared with children less than 10 years.⁴ The most common denomina-

tor however with the advancing age especially from 50 years and above is the declining immunity to VZV.⁵ Varicella has been found to be a risk factor for developing HZ in children in the first year of life.⁶ Schamder *et al.*⁷ documented the incident rates of HZ twice in whites when compared to blacks. In a study of 205 Indian patients with HZ over a two years period, the maximum incidence occurred in the third and fourth decades of life while a minimum incidence was observed in the age group less than 10 years.⁸

Although some authors maintain that HZ has no sex predilection² some studies have found increased incidence in adult females.⁹ Other risk factors include, disease-related immune compromise as in malignancies, Human immunodeficiency virus (HIV) infection or iatrogenic immune suppression resulting from treatment interventions as steroid use or radiotherapy. HIV infection causes an eightfold increase in incidence.^{5,10}

Trigeminal ophthalmic zoster accounts for 15% of zoster infections. Incidence of HZ and relative frequency of ophthalmic zoster increases with age probably due to the decline in cell-mediated immunity.^{1,2,11} Dissemination occurs in 2-10% of immunocompromised hosts¹² but uncommon in immunocompetent individuals.² This report highlights the unusual case of *Herpes zoster ophthalmicus* (HZO) in a black immunocompetent child.

Case Report

A previously healthy 8-year old boy presented with a five-day history of right sided headache and a four day history of a vesicular rash on the ipsilateral forehead, eye lid and nose. There was associated pain and swelling of the areas involved. The rash later progressed to involve the ipsilateral side of the scalp while the facial swelling spread to the contralateral side of the face (Figure 1). Three days after the onset of the facial rash, a vesicular rash was noticed on the chest, back, upper and lower limbs. There was associated fever and generalized headache. Review of systems was negative. There was a past history of a febrile exanthematous illness suggestive of chicken pox at 3 years of age and his parents and siblings had similar illness at about the same time. There was no history of recurrent illnesses, intake of neither steroids nor cytotoxic drugs. A history of contact with individuals with varicella infection was uncertain but the child had been attending school before his illness. He presented during an ongoing chicken pox outbreak as evidenced by an increased number of cases observed within and outside the hospital setting.

At presentation, he was acutely ill looking,

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febrile with an axillary temperature of 39.6°C. He had coalesced pustulo-bullous crusting lesions on the right peri-orbital and frontal areas, extending upwards into the temporo-parietal region of the scalp on the ipsilateral side and downwards to the right nostril, in keeping with involvement of the ophthalmic branch of the trigeminal nerve. There was associated gross facial edema involving both periorbital areas and extending to the chin and submandibular region. The swelling on the right side of the face was crepitant, suggestive of subcutaneous emphysema. Both eyes were tightly shut with copious discharge. He had right-sided pre-auricular, posterior auricular, submandibular and cervical lymphadenopathy. His weight was 33.5 kg (90th percentile) and his height was 128 cm (50th percentile). He had disseminated lesions on the trunk and extremities except spared the soles and palms. There were umbilicated vesicles on erythematous base with some crusting of some of the lesions (Figure 2).

Other systems were essentially normal. A diagnosis of herpes zoster ophthalmicus with suspected secondary bacterial infection was made.

His hematocrit was 38%, the white cell count was normal at presentation. HIV antibody test was non-reactive. His random plasma

glucose was 123 mg/dL. Serology for varicella antibodies could not be carried out until 6 weeks after and was reported as positive for varicella immunoglobulin (Ig) G and negative for IgM. Viral cultures and polymerase chain reaction (PCR) were not available.

He was admitted and nursed in isolation and was managed by both the pediatric infectious diseases unit in conjunction with the ophthalmologists. He was tolerating oral fluids and feeds fairly well and commenced on oral acyclovir at 20 mg/kg five times a day (the intravenous form was not available), acyclovir eye ointment and drops, regular ocular toiletting with 0.9% saline and dexamethasone eye drops on account of chemosis observed on eye examination. A full eye examination was not possible at this point as it was difficult to prise the lids open. He received in addition, intravenous flucloxacillin, ceftriaxone and metronidazole on account of the bilateral periorbital cellulitis. Topical acyclovir was applied to the lesions on the frontal region.

Within 24 h of oral acyclovir treatment, new skin lesions ceased to appear. He improved remarkably over the next week (Figure 3). The pustules became ulcerated and gradually healed. Fever and facial edema gradually resolved. He was able to open both eyes and had no problems with vision. He was discharged home with acyclovir ointment application after having completed a week of systemic acyclovir use and antibiotics. Follow-up at six weeks showed residual ptosis of the left eye (Figure 4A) without evidence of Bell's palsy or other neurological findings that may be in keeping with a complication including Ramsay-Hunt or Trigeminal herpes zoster. A slit-lamp examination of the eye did not reveal any significant corneal scarring and ophthalmoscopy did not show any intraocular lesion (Figure 4B). Visual acuity was normal.

Discussion

Dissemination of HZ is rare in immunocompetent individuals. The 8-year old boy in the present report was HIV negative, was not on immunosuppressive medications and did not have any obvious malignancy. Even in adults, reports of this complication of herpes zoster are rare in immunocompetent individuals.¹³⁻¹⁷ Reports of disseminated HZ in children are even less common than in adults. One of such reports in the literature involved two Indian children. The first case was malnourished while the second was an 11-year old child without any documented immunodeficiency but had a background history of atopy.¹⁸

There are many complications of HZ and the possibility of multi-organ involvement of the disease should be kept in view when there is

disseminated cutaneous disease. Apart from dissemination to the skin, HZ may disseminate to involve the central nervous system, lungs, liver and kidneys.^{19,20} HZO is a complication of HZ with the involvement of the trigeminal nerve. Involvement of the nasociliary branch is indicated by vesicles at the tip of the nose (Hutchinson's sign), the risk of eye involvement is 76% and cutaneous extension could occur² as was the case in the present report. Ophthalmologic assessment is necessary to identify complications. Our patient had chemosis at presentation and further ophthalmic assessment subsequently revealed ptosis of the affected side though there was no significant intraocular complication. In a study of the disease spectrum of HZO, ophthalmic features reported included lid edema, ptosis, cicatricial lid deformities, sclerokeratitis, peripheral ulcerative keratitis, neuroparalytic keratitis, keratouveitis with concomitant glaucoma, secondary bacterial keratitis and superficial punctate keratitis with dry eye, optic neuritis, and trochlear nerve palsy.²¹

Accurate clinical diagnosis of HZ can be made with the distinctive clinical features especially after the rash appears.⁵ However to differentiate it clearly from other conditions such as herpes simplex and impetigo among

others, laboratory tests may be necessary. Such tests include Tzank smear, direct fluorescent antibody staining of VZV-infected cell scraping from the base of the lesion, PCR and serology. A positive serological IgG test result with a positive IgM indicates a recent infection while a positive IgG with a negative IgM indicates past exposure. Negative results do not rule out varicella infection as it may depend on when the specimen was drawn. In the present case, the serological test was carried out six weeks after the onset of the rashes, resulting



Figure 3. One week after treatment.



Figure 1. At presentation, pustulo-bullous lesions on the right periorbital and frontal areas with facial edema and subcutaneous emphysema.



Figure 2. Umbilicated vesicular lesions on the trunk.



Figure 4. A) Residual ptosis of the right lid and scarring on the frontal region at follow up after six weeks; B) Eye examination revealed no significant corneal or intraocular residual manifestations.

from lack of access and high cost of laboratory logistics typical in many resource poor settings. IgG was positive but IgM antibodies were negative and might have waned at this point. It remains unclear then what the true situation was in the child in the present report. The explanation could be first; a reactivation of the infection which occurred five years earlier manifesting as disseminated herpes zoster ophthalmicus and second; an exogenous primary new infection from the current outbreak presenting as zoster. Johnson *et al.*²² reported a case of re-infection involving a young, immunocompetent physician with documented childhood disease and recently confirmed serum IgG antibodies but became re-infected with VZV after exposure to an immunocompetent patient with localized herpes zoster. Schade *et al.*,²³ in their observation reported a re-infection with wild type viral strain, from the molecular typing of the isolate, resulting in breakthrough varicella infection presenting as herpes zoster in a child who had been previously immunized. As a result of this observation, it was recommended that an analysis of VZV strains from patients who present with herpes zoster during an outbreak of chickenpox would be valuable. The VZV strain analysis was not carried out in the present case, as the technology was unavailable. Varicella infection has a high secondary attack rate of up to 90%,²⁴ transmission occurring through direct contact with the lesions or droplets from respiratory fluids or from the vesicular lesions. Both the primary infection and the herpes zoster are contagious however the risk of transmission is reported to be less in herpes zoster infection.²⁴ Varicella needs to be included in the routine immunization schedule for children. Universal varicella immunization in the US in the mid 1990s has led to a significant decline in the hospitalizations and reduction in varicella outbreaks. The issue of universal varicella vaccination in children has become a topic of debate as opponents of this strategy argue that as it provides temporary protection of unknown duration, which is at best 70-90%, long-term immunity is compromised-shifting chickenpox to a more vulnerable adult population. With increased HZ-related morbidity as well as the adverse effects of both the varicella and HZ vaccines reported, hence, the cost effectiveness of these vaccines is in question.²⁵ Nosocomial transmission of varicella to other patients and staff has been well documented and can be life threatening.²² Though none was recorded in this case, it is equally important to prevent transmission in health care settings. One of the infection control strategies to prevent varicella in health care settings includes evidence of immunity to varicella with varicella immunization or laboratory confirmation of the antibodies.

Conclusions

A case of disseminated HZO in an 8-year old immunocompetent child with previous primary VZV infection is presented. He was seen during a chicken pox outbreak. A breakthrough VZV re-infection or a reactivation is possible, both of which could present as zoster. This case emphasizes the need for prevention of VZV infection through universal childhood immunization and infection control strategies to be put in place in health care settings.

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