Oral and Maxillofacial Surgery Cases 1 (2015) 5–7

Contents lists available at ScienceDirect

Oral and Maxillofacial Surgery Cases

journal homepage: www.oralandmaxillofacialsurgerycases.com

Case report

A suspected dental cellulitis leading to diagnosis of both herpes zoster ophthalmicus and HIV

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ARTICLE INFO

Article history:
Received 10 July 2014
Received in revised form
24 November 2014
Accepted 26 March 2015

Keywords:
Herpes zoster
Ophthalmic nerve
HIV
Dental cellulitis
Post herpetic neuralgia
Immunosuppressive disease

ABSTRACT

Herpes zoster ophthalmicus and HIV are serious health problems. We report a case of a 37-year-old woman who presented to the Korle-Bu Teaching Hospital with dyspnea and facial cellulitis, and a diagnosis 5 days prior of dental cellulitis made at a district hospital. Investigations revealed that the facial cellulitis was secondary to herpes zoster infection involving the ophthalmic division of the left trigeminal nerve. The patient responded well to oral acyclovir but developed postherpetic neuralgia. During the course of treatment, she was also diagnosed to be HIV-1 positive and was referred for further management. This case represents a unique report in which the patient presented to the hospital with symptoms of cellulitis suggestive of underlying dental infection but was later diagnosed with both herpes zoster ophthalmicus and an underlying HIV infection. Atypical presentations of herpes zoster can occur in HIV/AIDS. Signs of herpes zoster infection with cellulitis should alert the clinician that the patient may have a possible underlying immunosuppressive disease. The population must be educated regarding the importance of early presentation and careful compliance with treatment as well as regular follow-ups.

1. Introduction

Herpes zoster (HZ) is an infection caused by reactivation of the varicella-zoster virus (VZV), also known as human herpes virus type 3. Studies in 1958 identified the virus causing HZ as identical to that which causes chicken pox [1]. VZV is a member of the family of herpesviridae, which includes the cytomegalovirus, herpes simplex, and Epstein-Barr viruses. Since the licensing of the varicella vaccine in the United States in 1995, the incidence of symptomatic varicella infection in childhood has been reduced by >80% [2,3]. Despite this, there are currently over 1 million cases of VZV in the United States each year with a lifetime attack rate of 30% [2,3]. The incidence of VZV increases with age, especially between the fifth and eight decades of life in the temperate climates of the United States and the UK [2,4,5]. This trend has also been observed in more tropical climates such as Thailand [6].

The varicella virus remains latent in the dorsal root ganglion of a nerve after initial infection [3,4]. When the virus is reactivated, it travels along the sensory nerve axon to cause shingles (HZ) [3]. Symptoms generally manifest along a single dermatome. The thoracic segments of the trunk are most commonly involved; however, divisions of the trigeminal nerve are affected in 10%-30% of cases [7,8]. Herpes zoster ophthalmicus occurs when a latent varicella-zoster virus in the trigeminal ganglion is reactivated to involve the ophthalmic division of the trigeminal nerve. The ophthalmic division is involved 20 times more frequently than the maxillary or mandibular division of the trigeminal nerve [2,7]. Ocular complications occur in an estimated 25%-30% of patients with herpes zoster ophthalmicus and include stromal and neurotrophic keratitis, anterior uveitis, scleritis, infectious retinitis, cranial nerve palsies, and postherpetic neuralgia. Corneal complications have been reported in 65% of patients [7].

HZ occurs more frequently in older individuals [2]. Increased incidence of zoster is also observed in immunosuppressed populations [9], including those receiving systemic steroid therapy, chemotherapy, and radiation that subsequently decrease the effectiveness of cytotoxic T cell–mediated immunity [2]. Risk

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http://dx.doi.org/10.1016/j.omsc.2015.03.001

factors for HZ include advanced age, diabetes, female gender, and recent trauma or psychological stress [2]. HIV-positive patients have a 15- to 25-fold greater prevalence of varicella zoster compared with the general population [2,7]. We present a unique case of herpes zoster ophthalmicus with its management that was initially suspected to represent a dental cellulitis and referred to the Korle-Bu Teaching Hospital (KBTH) for further management by the Oral and Maxillofacial Surgery Clinic.

2. Presentation of case

A 37-year-old woman presented to the district hospital with progressive left facial swelling of 5 days duration. She reported working as a trader and being married with 4 children. She denied any significant history of tobacco or alcohol use. There were no identified drug allergies or use of medications, including herbal preparations. Her medical history indicated a rash that was not present at the time of initial presentation, nor was it well enough described to provide any substantive information to the differential diagnosis.

At the district hospital, examination revealed a fully conscious, breathless, acutely ill-looking female with significant pain and facial swelling extending to the lateral aspect of her left neck. No vesicles were noted at this time. Ptosis of the left eyelid was present. She was mildly pale with no evidence of jaundice, cyanosis, or clubbing. Her cardiovascular status included a regular pulse rate of 100 bpm and blood pressure of 90/70 mm Hg. Heart sounds were present with no murmurs, and her chest was clinically clear. Owing to a suspicion of an underlying dental infection, an initial diagnosis of dental cellulitis was made. The patient was given the narrow-spectrum beta-lactam antibiotic flucloxacillin (500 mg 4 times daily), ibuprofen (400 mg 3 times daily), and Funbact-A cream (Bliss GVS Pharmaceutical Co., Mumbai, India), a topical formulation that contains (1) a synthetic antifungal, clotrimazole 1.0%, (2) a topical corticosteroid, betamethasone dipropionate 0.05%, and (3) a broad spectrum antibiotic, neomycin sulfate 0.5%. She was then referred to the KBTH for further management by the Oral and Maxillofacial Surgery clinic.

On presentation to the KBTH, 5 days after presentation at the district hospital, the patient described a burning sensation and severe pain on the left side of her face. Vesicles had appeared along her hairline in the region of distribution of the left ophthalmic division of the trigeminal nerve, apparently after she had braided her hair 5 days prior. Rupturing of the vesicles occurred shortly after initial formation, resulting in crusting. The patient experienced difficulty in breathing, as well as fever and chills. She was unable to open her eyes bilaterally with increased restriction in the left eye. Extraoral examination showed facial cellulitis with severe pain along with ulceration and crusting affecting the left ophthalmic division of the trigeminal nerve (Figure 1A). There were also several vesicles present in the affected area.

Differential diagnosis was highly suggestive of herpes zoster ophthalmicus, although panfacial cellulitis secondary to use of herbal preparations, cavernous sinus thrombosis, and dentoalveolar infection were also considered. Management of the patient included investigations of full blood count, sickling test, blood urea and electrolytes, creatinine, and Retroscreen for HIV infection. Results of the laboratory investigations included hemoglobin 11.9 g/dL (normal, 12-16 g/dL), platelets 225×10^9/L (normal, 150-400 $\times 10^9$/L), and white blood cell count 4.4×10^9/L (normal, 4.3-10.8 $\times 10^9$/L). Retroscreen was positive for HIV-1 infection, and a written referral was made to the fevers unit for further management.

Oxygen via face mask and intravenous (IV) fluids were administered. Medications given were as follows: tablet acyclovir 800 mg 5 times daily for 7 days, IV Flagyl (Eskay Therapeutics, Accra, Ghana) 500 mg every 8 hours for 48 hours, IV gentamicin 80 mg every 8 hours for 48 hours, IV dexamethasone 8 mg stat., followed by 4 mg every 8 hours for 48 hours, suppository diclofenac 100 mg twice daily for 5 days, vitamin B complex 1 tablet daily for 2 weeks, tablet Metran (Daewon Pharmaceutical Companies. Ltd, Busan, South Korea) 1 twice daily for 2 weeks, and Neo Hycolex eye drops (Tobinco Pharmaceutical Ltd., Accra, Ghana).

The patient gradually improved and continued treatment as an outpatient under care of an ophthalmologist. One month later, the patient returned for follow-up from the fevers unit with a CD4 baseline of 183 cells/μL (normal, 500-1200, AIDS <200), complaining of loss of vision in her left eye and persistent pain in the affected area consistent with the diagnosis of postherpetic neuralgia (Figure 1B). This further confirmed the diagnosis of herpes zoster ophthalmicus. At that time, she had not yet begun antiretroviral treatment. The patient was seen 7 months later with healing but extensive scarring of the forehead, eyelid, and cornea (Figure 1C) with persistent loss of vision in the eye. She continued review with the ophthalmologist and was on antiretroviral therapy. At her follow-up appointment 2 years later, she had complete loss of vision in the left eye and hopes for corneal implant.

3. Discussion

HZ infection is produced by the reactivation of the latent varicella-zoster virus from the dorsal root ganglia of sensory nerves. Major risk factors for HZ include immunosuppression and decreased cell-mediated immunity. Other risk factors include an elderly population and those with poor nutrition. For any

Figure 1. (*A*) Initial presentation. (*B*) One month after presentation. (*C*) Seven months after treatment.

population that includes these risk factors, the practitioner should have a heightened sense of awareness toward the possibility of herpes zoster ophthalmicus [1,2,7]. Approximately 9% of patients with HZ, of which there are over 1 million per year in the United States, have complications involving the eye [2,3,8].

This case represents a unique report in which the patient presented to the hospital with symptoms of cellulitis suggestive of underlying dental infection and was referred to an oral and maxillofacial surgeon for management. After presentation to the oral surgeon, the patient was diagnosed with both herpes zoster ophthalmicus and an underlying HIV infection. HIV is a serious health problem, and HZ can be the initial presentation of the disease [1,2].

HZ begins with a prodromal stage of flu-like illness with fatigue, low-grade fever, and chills that last up to 1 week before the characteristic skin lesions appear. Sixty percent of patients complain of pain [2]. Erythematous macules then appear along the involved dermatome. Over several days, they progress into papules and vesicles and later pustules that rupture and crust, taking several weeks to heal. HIV-positive patients may have generalized vesicular rash and become ill 1-2 weeks after the onset of the disease.

Herpes zoster ophthalmicus can present with extraocular and ocular manifestations. Infection and inflammation secondary to HZ can affect virtually all adnexal, ocular, and orbital tissues [2,7,9,10]. The skin manifestations of herpes zoster ophthalmicus involve $\geq$1 branches of the ophthalmic division of the trigeminal nerve. These include the supraorbital, supratrochlear, lachrymal, infratrochlear, and external nasal (a branch of the nasociliary) branches. Because the nasociliary branch innervates the cornea, the most serious ocular involvement occurs if this branch is affected. Eyelids are also commonly affected [2,7]. Patients can develop blepharitis, and this can lead to secondary bacterial infection, eyelid scarring, marginal notching, loss of eyelashes, trichiasis, and cicatricial entropion. Scarring and occlusion of the lachrymal puncta or canaliculi can occur. Ptosis secondary to edema and inflammation may also be present. Some of these signs were observed in this case with inflammation of the patient's eyelids and subsequent crusting, retraction, and deformity of her left eyelid (Figure 1C). Unlike this case, most patients will have vesicular lesions that resolve with minimal scarring.

Some of the complications that can appear in the course of herpes zoster ophthalmicus are corneal neovascularization and scarring resulting in poor vision. Other complications are neurotrophic ulcer with perforation, secondary bacterial or fungal infection and secondary glaucoma from uveitis and steroid treatment. Necrotizing interstitial keratitis, postherpetic neuralgia, and loss of vision from optic neuritis or chorioretinitis can also occur [2,7,9].

Herpes zoster ophthalmicus can be successfully managed with systemic antivirals (acyclovir 800 mg 5 times per day for 1 week to 10 days) [7], sometimes in combination with tricyclic antidepressants to inhibit the infectious inflammatory component of the associated pain [2]. Antiviral agents may decrease the severity and duration of the pain if given early in the course of the illness. Antibiotics should also be given if secondary bacterial infection of the vesicles has occurred. For acute retinal necrosis, the acyclovir may be administered intravenously (1500 mg/d divided into 3 doses) for 7-10 days with long-term oral acyclovir follow-up ($\sim$14 weeks of 800 mg 5 times daily) [7,10]. Unfortunately, oral acyclovir has little effect on the incidence, severity, or duration of postherpetic neuralgia.

4. Conclusion

HIV and HZ are serious health problems. The initial presentation of the disease in this case was mistakenly a dental cellulitis. Complications and atypical presentations of HZ are common in populations with a high burden of HIV/AIDS. Recognition of the early signs of HZ allows for early clinical diagnosis and intervention. In rural settings where access to health services is difficult, the general population should be educated regarding the importance of early presentation and careful compliance with treatment as well as regular follow-ups.

Consent

Written informed consent was obtained from the patient for publication of this case report and the accompanying image. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Conflict of interest

The authors do not have any conflicts of interest to disclose.

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