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Odontogenic Herpes Zoster Infection: A Case Report and Review

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Abstract

Herpes zoster is a commonly encountered entity of which all clinicians should be aware. The diagnosis is most often considered and made based on a characteristic vesicular rash that presents in a unilateral dermatomal distribution and is usually accompanied by a painful neuritis. Not uncommonly, the pain presents a few days before the rash becomes evident and may even occur without the rash appearing. When this happens patients may be subject to further diagnostic testing seeking alternative diagnoses, as herpes zoster is known to mimic a variety of other non-cutaneous organ system entities. Although the thoracic and lumbar dermatomes are the affected most frequently, in approximately one-fifth of cases the cranial nerves are involved either singularly or in combination. Trigeminal nerve zoster is of particular concern as it poses a risk of developing into zoster ophthalmicus with subsequent keratitis and uveitis resulting in permanent vision impairment. Involvement of the second (maxillary) and third (mandibular) branches of this 5th cranial nerve are less common and may present with signs and symptoms of a primary dental process. The infrequency and unfamiliarity of herpes zoster odontogenic manifestations can lead to unnecessary investigation and treatment. Apropos such a case we review the epidemiology, pathophysiology, signs, and symptoms of odontogenic herpes zoster.

Keywords

Herpes Zoster, Trigeminal Nerve, Odontogenic

1. Introduction

Of the multiple dozens of identified herpesviruses, only eight members are

known to infect humans. The simian herpesvirus known as Herpes B virus geologically infects macaques but on rare occasions can infect exposed humans who may then go on to present with mucocutaneous lesions and/or encephalitis. Specific genetic and structural features of these double stranded DNA viruses afford them the ability to establish latency, which is then maintained throughout the infected individual's life. Based on their specific sites of latency, human herpesviruses are subdivided into three subfamilies. Varicella-zoster virus (VZV) along with herpes simplex type 1 (HSV-1) and type 2 (HSV-2), and herpes B virus constitute the Alphaherpesvirinae group with sensory ganglia neurons serving as their site of latency. The Betaherpesvirinae subfamily comprised of cytomegalovirus (CMV), human herpes virus 6 (HHV-6), human herpes virus 7 (HHV-7) and Gammaherpesvirinae, which includes Epstein-Barr virus (EBV) and Kaposi sarcoma-associated herpesvirus (KSHV or HHV-8) establish latency in mononuclear cells and lymphoid cells, respectively. With few exceptions, herpesviruses generally have overlapping modes of transmission. Infection can occur via direct human-to-human spread, either through saliva, sexual contact, *in Utero*, or during childbirth. CMV may also be transmitted by blood transfusion, organ donation, and post-partum breast feeding. VZV is unique in that it is the only herpesvirus that is spread by airborne transmission.

Herpes zoster infection is the result of reactivation of varicella-zoster virus that had established latency in dorsal root ganglia following primary varicella, generally known as chickenpox, infection. Colloquially referred to as *shingles*, derived from the Latin word "cingulum," which means belt or girdle, zoster affects ≥20% of individuals during their lifetime [1]. Although zoster infection may present at any age, it mainly affects senior adults and immunocompromised patients because cell-mediated immunity in these individuals is decreased. Most cases present with a unilateral dermatomal cutaneous vesicular rash most often involving the thoracic and lumbar dermatomes. Odontogenic zoster is estimated to occur in less than 1 in 50 cases. Almost always the rash is accompanied or preceded by an acute neuritis in the same dermatomal distribution. On rare occasions painful neuritis occurs without the development of skin lesions, a condition appreciated as *zoster sine herpete.* While less than 4% of persons will experience a second episode of zoster, approximately 25% to 50% of those over 50 years-old will continue to have some degree of pain, often for several months and at times debilitating, in the dermatomal distribution after the lesions have crusted. This condition is referred to as postherpetic neuralgia. Extracutaneous manifestations of zoster may include central nervous system (meningoencephalitis, encephalitis, granulomatous cerebral angiitis) and neuromuscular (transverse myelitis, Guillain-Barre syndrome) disorders as well as visceral disease such as pneumonitis and hepatitis [2]. Even though not thought of as visceral involvement, odontogenic and other oropharyngeal manifestations of herpes zoster, though rare, at times may be the initial zoster presentation prior to the development of the tell-tale vesicular eruption. The seldom occurrence, atypical

presentation, and unfamiliarity of this zoster manifestation may influence the treating clinician to consider and empirically treat alternative diagnoses. Apropos such a case we review the epidemiology, pathophysiology, signs, and symptoms of odontogenic herpes zoster.

2. Case Summary

A 79-year-old immunocompetent woman presented to a local medical clinic complaining of history of right ear discomfort, gingival pain, and oral mucosa lesions. Initially, she recalled experiencing a burning sensation in the right commissure of the mouth, which she attributed to consuming a hot meal. The following day, she empirically began treatment with amoxicillin for a presumed dental infection. Over the course of the following 2 days, vesicular lesions appeared in the right corner of the mouth and around the mandibular area associated with increasing right ear and facial pain. These worsening symptoms prompted her to visit her local hospital emergency department where she was treated with analgesics with minimal pain relief. She presented to our clinic for further evaluation 5 days after symptom onset. She denied having fever or other systemic manifestations and other than having asthma and hyperlipidemia, her past medical history was unremarkable. Physical examination was significant for right facial edema, erythema, and multiple vesicular lesions in the right mandibular area associated with tenderness to palpation. Intraoral inspection showed multiple ulcerative and purulent lesions (**Figures 1(a)-(c)**). Over the next 2-days there was an increase in facial edema, erythema, and pain as well as the progression of vesicle formation, some of which began to crust over, all in a unilateral dermatomal pattern (**Figure 1(d)**). A maxillofacial computerized tomographic scan (CT) with contrast was performed demonstrating diffuse asymmetric inflammatory changes adjacent to the 2nd right upper molar with surrounding a periapical area of lucency along with a 2 cm fluid collection adjacent to area of inflammation characteristic of small abscess (**Figure 2**). Her antimicrobial regimen was changed to parenteral ampicillin-sulbactam for suspected dental abscess. Subsequently, consultation with an Infectious Diseases specialist was obtained, where upon a diagnosis of herpes zoster was suggested. Lacking access to varicella-zoster nucleic acid assay (PCR) or direct fluorescent antibody stain tests, a standard serologic immunologic assay measuring both VZV IgM and IgG antibodies was obtained. While the IgM antibodies were undetectable, based upon the significantly elevated level of IgG antibodies, 2986 (<135, negative; ≥165, positive) and considering her having had a history of childhood varicella infection, a diagnosis of odontogenic herpes zoster infection was made clinically. Treatment with valacyclovir 1000 mg and amoxicillin-clavulanate 500 mg every 8 hours for 7 days was provided. Corticosteroids were not included in the treatment of this patient's symptoms. Evaluation by an oral maxillofacial surgeon found no indication for a dental surgical procedure. One month after presentation, her cutaneous lesions and dental symptoms had resolved, however, she

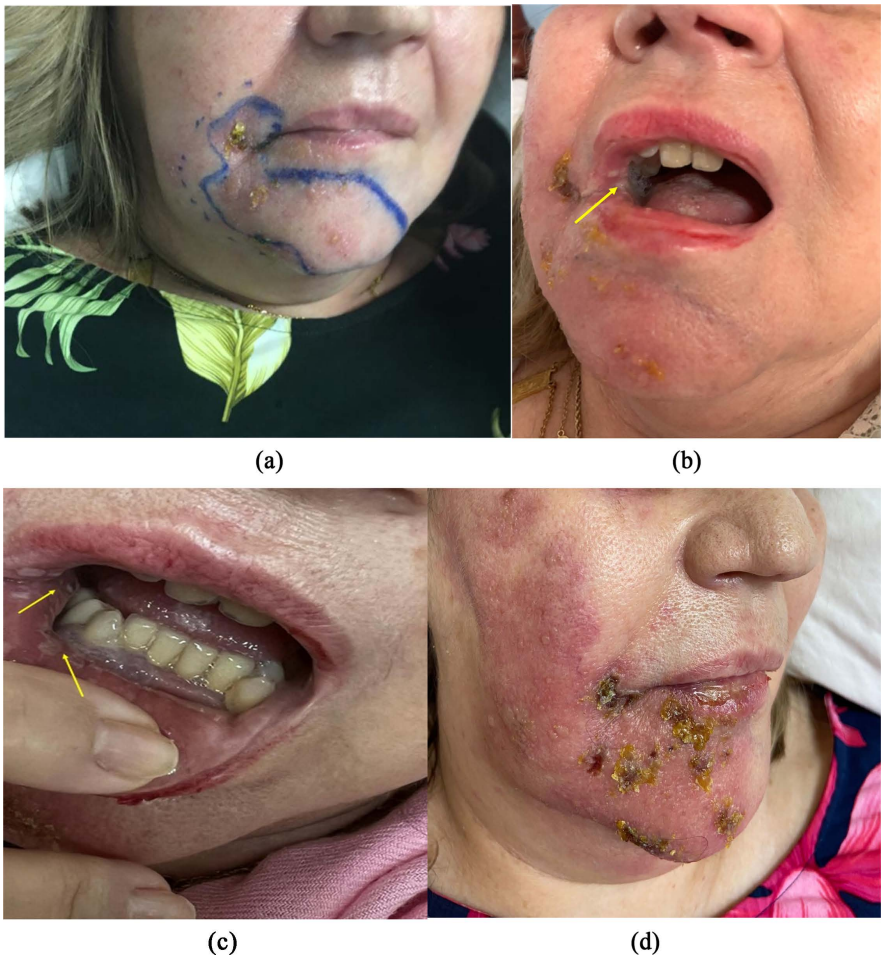

Figure 1. (a) Right facial edema, erythema, and multiple vesicular lesions in the right mandibular area. (b) & (c) Multiple intraoral ulcerative and purulent lesions (yellow arrow). (d) 48 hours after initial presentation increase in facial edema and erythema, progression of vesicles and crust formation.

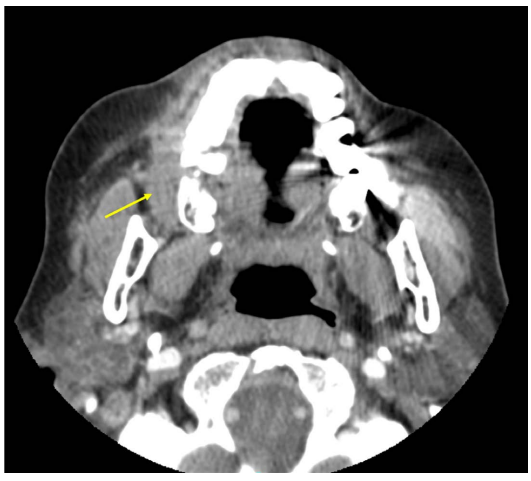

Figure 2. Maxillofacial CT with contrast—Diffuse asymmetric inflammatory changes adjacent to the 2nd right upper molar with surrounding a periapical area of lucency along with a 2cm fluid collection adjacent to area of inflammation characteristic of small abscess (yellow arrow).

continues to have mild residual right facial and ear pain.

3. Discussion

Cranial nerve dermatomal involvement by varicella zoster virus (VZV) comprises about 20% of the dermatomes affected, with the trigeminal nerve representing most of the occurrences [3]. A recent study by Tsau *et al.* retrospectively studied 330 patients with cranial nerve involvement by herpes zoster over a period of 10 years and determined that the trigeminal nerve represented 57.9% of all cases, followed by the facial nerve (52.1%), and vestibulocochlear nerve (20.0%) [4]. The ophthalmic and maxillary branches of the trigeminal nerve are sensory branches, while the mandibular branch is both sensory and motor. Multiple studies have determined that trigeminal nerve zoster generally occurs in one sensory nerve dermatome, but two branches of the trigeminal nerve involvement have also been reported [4]. The V_1 ophthalmic branch of the trigeminal nerve is affected about 20 times more frequently compared to its V2 (maxillary) and V3 (mandibular) branches [5]. Herpes zoster ophthalmicus may lead to dangerous sequela for the patient affected if timely recognition and treatment do not occur. Hutchinson's sign, which clinically manifests as vesicles on the tip of the nose, signifies involvement of the nasociliary branch of the ophthalmic nerve and should prompt an immediate ophthalmologic evaluation. The maxillary (V_2) and mandibular (V_3) division of the trigeminal nerve are rarely affected by VZV. Furthermore, simultaneous involvement of all three branches of the trigeminal nerve has also been reported in immunocompetent patients [6]. Facial cranial nerve zoster has a typical presentation with appearance of vesicular lesions in the auricle, external auditory canal and oral cavity and is also associated with facial nerve palsy characterized by ipsilateral facial weakness. This clinical presentation, known as Ramsay Hunt Syndrome (RHS), reflects the involvement of the facial sensory and motor nerves.

In a retrospective study by Peitersen *et al.*, 4.5 percent of all peripheral facial nerve palsies were caused by herpes zoster [7]. Vestibulocochlear nerve involvement in RHS clinically manifests with hearing loss, vertigo and tinnitus. Dr. Hunt hypothesized that these features can be explained by the proximity of the geniculate ganglion to the vestibulocochlear nerve within the bony facial canal [8].

Our comprehension of the sophisticated function of the cranial nerves and their respective dermatome involvement aids us in better understanding the associated symptomatology triggered by herpes zoster infections. In our case, the patient attributed the prodromal symptoms (burning sensation of the lower half of her face) to an odontogenic cause resulting in treatment with antibiotics for a presumed tooth infection, thus delaying her diagnosis.

Odontogenic (dental) pain is a common presenting complaint leading the patient to seek care in dental offices, primary care clinics, and emergency departments. In most instances this pain is secondary to dental caries, pulpitis, perico-

ronitis, periodontitis, dentine sensitivity or dental trauma [9]. Given the high prevalence of these typical causes of odontogenic pain, herpes zoster is not often a diagnosis of initial consideration [10]. There are multiple published case reports of prodromal herpes zoster pain initially misdiagnosed as a primary dental condition with subsequent identification of the correct diagnosis when classical features of zoster reactivation become evident later during the disease [10] [11] [12] [13]. Anatomic and radiographic findings mimicking other more customary odontogenic conditions have also been noted in the dental and endodontic literature [12] [13]. Viral infections such as intraoral VZV have been implicated in the formation of periapical abscesses, though the precise relationship between these two is yet to be elucidated. In one study by Ferreira, *et al.*, using PCR technology, VZV was detected in 9% of apical abscesses sampled. Moreover, 56.5% of samples were positive for any herpesvirus, reaffirming the role that these viruses play in the pathogenesis of dental infections [14].

Because pulpitis, pericoronitis, and apical periodontitis are frequently encountered entities in the practice of dentistry and endodontics, it is predictable that patients with prodromal orofacial herpes zoster may undergo unnecessary nonsurgical and surgical interventions to treat one of these more common conditions [10] [15] [16].

4. Conclusion

The diagnosis of odontogenic zoster infection may be challenging in patients who present atypically, as the prodromal symptoms closely mimic those encountered with dental infections, thereby resulting in the prescribing of unnecessary antibiotics instead of appropriate antiviral therapy. Expanding our differential diagnoses by considering uncommon causes of odontogenic pain, such as herpes zoster, prevents delay in diagnosis, avoids unnecessary and costly diagnostic procedures, and may decrease the likelihood of lasting sequelae such as postherpetic neuralgia.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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