
CASE REPORT & REVIEW

***Herpes zoster* of the trigeminal nerve third branch: a case report and review of the literature**

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Abstract

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Literature review and case report A literature review of *Herpes zoster* of the trigeminal nerve is

presented. Included are differential diagnosis and treatment modalities that will enable the dental practitioner to identify and manage this disease. A case report is provided to amplify this timely information.

Keywords: acyclovir, *Herpes zoster*, shingles, trigeminal, *zoster sine herpete*.

Introduction

Herpes zoster of the trigeminal nerve is a disease that falls within the diagnostic purview of all dentists and dental specialists. A thorough knowledge of this disease will prevent unnecessary and delayed treatment for the patient. During the prodromal stage of this disease in particular, the only presenting symptom may be odontalgia; this may prove to be a diagnostic challenge to the clinician who is not familiar with *H. zoster* of the trigeminal nerve (Millar & Troulis 1994).

The objectives of this paper are (i) to present a brief review of *H. zoster*, (ii) to present the three classical diagnostic stages of *H. zoster* infections, (iii) to review unusual complications of trigeminal *H. zoster* infections, (iv) to present treatment modalities for the different stages of the infection, (v) to present a differential diagnosis, (vi) to highlight the role of the endodontist in diagnosis and management of *H. zoster* of the trigeminal nerve, and (vii) to present a case report.

Background

The *Varicella zoster* virus is responsible for two common infectious diseases: chicken pox (*Varicella*) and shingles (*H. zoster*). Chicken pox is the primary infection. After the initial infection, the virus remains dormant until there is a reactivation that may occur decades later. The subsequent reactivation is *H. zoster*.

Chicken pox and *H. zoster* are contagious and will spread via direct contact with an infected person. The transmission can also occur through inhalation of airborne respiratory secretions. The virus infects the cells of the respiratory tract or conjunctival epithelium and is carried through the body via the blood stream and lymphatic system. It is then spread from the capillary epithelium to the epidermis where viral replication destroys the basal cells. The virus then remains in a latent stage in perineural satellite cells of the dorsal nerve root ganglia. The person with *H. zoster* remains in a contagious state from 2 days before the appearance of a rash, and remains contagious until all lesions are crusted with no detectable drainage from the sites (Rubin & Farber 1995).

Zoster infections do occur many decades later in about 20% of the population. In the immunologically

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impaired host, *zoster*, infections may occur within a year or two following the initial *varicella* infection. The incidence of *H. zoster* does increase with age (Ragozzino *et al.* 1982).

Herpes zoster may affect any sensory ganglia and its cutaneous nerve (Strommen *et al.* 1988). Most of the infections affect dermatomes of T-3 to L-2 but about 13% of patients present with infections involving any of the three branches of the trigeminal nerve (Millar & Troulis 1994).

Zoster-associated complications include neurologic components such as Guillian–Barre Syndrome, encephalitis, myelitis, Ramsey–Hunt Syndrome (Bell 1985) and Horner's syndrome (Poole *et al.* 1997). Generally, ocular complications involve ulcerations, haemorrhage, conjunctivitis and optic neuritis. Additionally, cutaneous scarring and bacterial infections are common occurrences.

Three diagnostic stages of *Herpes zoster* infections

Patients with *H. zoster* infections usually progress through three stages: (i) prodromal stage, (ii) active stage (also called acute stage), and (iii) chronic stage (Strommen *et al.* 1988, Carmichael 1991). However, some patients do not develop symptoms of all stages. Some patients do not form vesicular eruptions of the active stage, but do develop pain restricted to a dermatome, and this has been termed *zoster sine herpette* which makes proper diagnosis more difficult (Barrett *et al.* 1993).

The prodromal syndrome stage presents as sensations described as burning, tingling, itching, boring, prickly or knife-like occurring in the skin over the affected nerve distribution. It is believed that these sensory changes are a result of degeneration of nerve fibrils from viral infection activity. This usually precedes the rash of the active stage by a few hours to several days (Strommen *et al.* 1988, Carmichael 1991, Millar & Troulis 1994). The patient may present with an odontalgia that may be the only prodromal symptom (Barrett *et al.* 1993, Law & Lilly 1995).

The active stage is characterized by the emergence of the rash that may be accompanied by generalized malaise, headache, low-grade fever and sometimes nausea. The rash progresses from erythematous papules and oedema to vesicles in 12 to 24 h and finally progresses to pustules within 1 to 7 days. The pustules begin to dry with crust formations that fall off in 14 to 21 days, leaving erythematous macular

lesions that result in hyperpigmented or hypopigmented scarring. In severe cases, areas of epidermis and variable amounts of dermis may be lost due to haemorrhagic necrosis (Strommen *et al.* 1988, Carmichael 1991). Intraoral lesions usually appear after the cutaneous rash. Pain and dysaesthesia during the active stage are reported to be minimal when the rash is most active. However, there is a return of pain during the crusting and scale phase of the active stage but this pain subsides as the rash and scales clear (Strommen *et al.* 1988).

The chronic pain syndrome stage is termed post herpetic neuralgia (PHN). PHN is defined as pain lasting beyond the period of healing of the active skin lesions. This has been described as pain lasting 1 to 3 months after the skin lesions have cleared but may in fact last for years and decades (Strommen *et al.* 1988, Rowbotham & Fields 1989a, Carmichael 1991). PHN pain has been described as pain consisting of three distinct components: (i) a constant, usually deep pain; (ii) a brief recurrent shooting or shocking tic-like pain; and (iii) a sharp radiating dysaesthetic sensation evoked by very light touching of the skin, termed allodynia (Rowbotham & Fields 1989b).

Differential diagnosis

Definitive diagnosis often involves a process of elimination, with several likely aetiologies in the differential diagnosis. A differential diagnosis should include trigeminal neuralgia, maxillary sinusitis, periodic migrainous neuralgia, myocardial pain, atypical facial pain and Munchausen's syndrome (Drinnan 1987). Thus, the dental practitioner should be familiar with the classical features of various diseases that can produce facial pain, but should always be aware that not every patient will report every feature of the condition. Further, it is prudent to remember that not all preliminary diagnosis by patients are correct, and although a patient may be convinced a problem is 'dental', he or she may be incorrect (Drinnan 1987).

The diagnosis of *H. zoster* is clear when the prodromal symptoms are present and the dermatomal vesicular rash is present. A diagnostic challenge is created when the vesicular rash does not occur, as in *zoster sine herpette*. Culturing may be required to confirm a *H. zoster* infection in these patients (Barrett *et al.* 1993). Careful history and a thorough dental examination usually rule out other pathology (Strommen *et al.* 1988, Carmichael 1991, Millar & Troulis 1994).

Unusual complications of *Herpes zoster* infections of the trigeminal nerve

The clinical manifestations of *H. zoster* infections are variable and can be demonstrated beyond the region of the head and neck. These manifestations can range from meningoencephalitis to superficial skin changes (Carmichael 1991). It is important to remember that any part of the central nervous system can be affected. Rare infections have been reported to affect many other organs including the brain, heart, liver gastrointestinal tract, urinary tract and the joints of the musculoskeletal system (Strommen *et al.* 1988). These occurrences can be found in individuals with some degree of immunosuppression or systemic illness (Strommen *et al.* 1988). In patients with HIV infection, *H. zoster* may be an early symptomatic finding (Carmichael 1991). Dental complications reported include devitalization of teeth, tooth exfoliation, internal root resorption, tooth neuralgia and osteonecrosis (Millar & Troulis 1994, Sigurdsson & Jacaway 1995).

While ocular involvement is the most common trigeminal nerve infection (50% of all trigeminal occurrences), it is critical to remember that Hutchins' sign (cutaneous *zoster* lesions of one side of the tip of the nose) is pathognomonic of V2 (maxillary) branch involvement (Millar & Troulis 1994). One of the most serious and rare complications of *H. zoster* of the trigeminal nerve is the presence of spontaneous tooth exfoliation and necrosis of the mandible. In these cases the course of *zoster* infection is unremarkable, with pain and vesicular eruption along the mucosa and cutaneous membranes innervated by the trigeminal nerve. However, upon resolution of the acute/active phases, the patient suffers spontaneous exfoliation of the teeth on the affected side and also osteonecrosis. The contralateral side of the patient's jaw remains within normal limits and, as with most of these cases, there are no predisposing factors of localized pathosis (Muto *et al.* 1990).

Management

Routinely, patients with *H. zoster* are seen by their physicians for treatment. However, the dentist and the dental specialist are often involved in the initial diagnosis of this disease and therefore must be familiar with its management. Recommended therapy should include (i) patient isolation, (ii) local management of skin lesions, (iii) control and elimination of pain, (iv) limitation of the extent, duration, and severity of the disease with antiviral agents and (v) treatment of post

herpetic neuralgia (Strommen *et al.* 1988, Carmichael 1991).

Isolation: Patients with *H. zoster* infections are contagious to persons at high risk. This includes neonates, nonimmune persons, pregnant women and immuno-compromised patients. The contagion is transmitted as *Varicella zoster virus* (chicken pox). Herpes zoster patients remain contagious until crusting and scaling have taken place (Strommen *et al.* 1988, Carmichael 1991).

The skin: Management usually encompasses the application of open wet dressings followed by lotions. Gauze or a face cloth soaked in cool water and applied to the rash area for 30 min three to six times a day is recommended. However, occlusive ointments should be avoided. Creams and lotions containing corticosteroids are not recommended. Ointments may be used after the acute phase to soften and remove adherent crusts (Strommen *et al.* 1988).

Pain: The acute pain of *H. zoster* infections can be reduced during the prodromal and the acute phases. Mild to moderately strong analgesics, such as acetaminophen, codeine and nonsteroidal anti-inflammatory agents are effective. However, these analgesics are notoriously ineffective for the chronic post-herpetic neuralgia (PHN) phase (Strommen *et al.* 1988, Carmichael 1991).

Anti-viral drug therapy: Once a diagnosis of *H. zoster* infection has been determined, anti-viral therapy must be swift and precise. Acyclovir has been the drug of choice for a number of years. Acyclovir is the first stage drug for many reasons, the primary being that it has been proven to decrease drastically the duration and severity of the *H. zoster* infection in the acute phase, if treatment is started within 48 h of the onset of the rash (Strommen *et al.* 1988, Carmichael 1991, Millar & Troulis 1994). Acyclovir is also beneficial in treating *zoster* infections, compared to some other anti-viral drugs, because it has a much higher rate of phosphorylation in *herpes* infected cells because of the activity of *herpes* specific (thymidine) kinase. Acyclovir also proves to be less toxic than other anti-viral drugs (Strommen *et al.* 1988). While dosage regimens range from discipline to discipline, 800 mg four times a day for 10 days remains the standard of care. Research has shown that dosages of up to 800 mg five times a day have been given with even more promising results (Wood *et al.* 1996).

Recently newer forms of anti-viral drugs have been developed specifically to address the acute stage of *H. zoster* (Famciclovir) and for use in immunocompetent



Figure 1 Herpes zoster rash, left chin area.



Figure 2 Herpes zoster rash, left zygoma area.



Figure 3 Periapical radiograph of mandibular centrals, left mandibular lateral and left mandibular canine.



Figure 4 Residual pigmented scarring of herpes zoster.

patients (Valacyclovir). In the former, the dosage is 500 mg every 8 h for 7 days; for the latter it is 1 g three times daily for 7 days. After undergoing rapid biotransformation to the active compound, penciclovir, Famciclovir is phosphorylated by viral thymidine kinase in HSV-1, HSV-2 and VZV-infected cells to a monophosphate form; this is then converted to penciclovir triphosphate and competes with deoxyguanosine triphosphate to inhibit HSV-2 polymerase. In essence the *herpes* viral DNA synthesis/replication is selectively inhibited. To date there are no known studies that have compared the reduction of pain in patients taking these newer drugs versus Acyclovir. Likewise no significant drug interactions have been noted (Gill & Wood 1996).

Post herpetic neuralgia: Standard analgesic narcotic combinations are not effective in patients with post herpetic neuralgia (PHN). The pain of PHN is because of injury to the central nervous system, and therefore, pain is unlikely to respond to any analgesic-narcotic combination (Strommen *et al.* 1988). The treatment for PHN pain includes the topical use of capsaicin cream (Zostrix), transcutaneous nerve stimulation, topical anaesthetics, injected local anaesthetics and low dose amitriptyline (Elavil) (Carmichael 1991). Strommen *et al.* (1988) offer an indepth review of the use of antidepressants and neurolytics in the pain management of PHN. No single treatment is universally effective for all PHN patients (Carmichael 1991).

Role of the endodontist in diagnosis and management

Multiple factors must be considered by the endodontist in the diagnosis and management of these cases.

Initially, because the nature of the pain reported by the patient might be considered as being of tooth origin, a thorough subjective and objective assessment of the patient is essential. Leaving out any facts, especially a history of past herpetic attacks, or failing to do a thorough assessment will often lead to the erroneous performance of root canal treatment with no subsequent cessation of patient symptoms. For the unsuspecting clinician, this situation often leads to additional nonsurgical or even surgical root canal treatment. This unfortunate circumstance can occur when no vesicles appear or their presence is mistaken for aphthous stomatitis (Bell 1985). In the latter situation, confirmation can usually be made by the anatomical location of the lesions.

A second situation facing the endodontist is the fact that a herpetic attack of the trigeminal nerve can cause pulpal necrosis (Gregory *et al.* 1975, Goon & Jacobsen 1988). The causative agent in this situation is the varicella-zoster that may be harboured in the ganglion cells some time after a primary varicella infection. Because the dental pulp contains terminal nerve endings, it can be speculated that the reactivated virus travels the length of the nerve and infects the pulpal vasculature, leading to infarction and death. These possibilities highlight the need to do a thorough medical/dental history along with extensive pulp testing in those cases that present with constant, intense, burning, hot discomfort of the skin that increases with any stimulus and may include sharp, stabbing pains as well. If the pain is limited to the distribution of the affected nerve and lesions are present the diagnosis is reasonably straightforward. Likewise, previous episodes of *H. zoster* infection often leave scars, making subsequent diagnosis easier. Finally, with the increasing geriatric population and their desire to maintain their teeth, and knowing that individuals over the age of 70 years are affected by *H. zoster* more often than others, the endodontist must be constantly alerted to the possibility of this differential diagnosis.

Case report

A 59-year-old Caucasian female currently on oestrogen therapy and with a history of removal of a brain tumour in 1984 presented to the Baylor College of Dentistry in early February 1997. Her chief complaint was a severe throbbing toothache pain radiating to her ear and chin. A rash was noted on her left chin. She could pinpoint her toothache to the mandibular left canine. She stated there were

sensations of itching and burning in the skin 2 days prior to the appearance of the rash. The rash was localized to the left chin (Fig. 1) and was also localized over the left zygomatic arch in front of the ear (Fig. 2). She was taking over-the-counter analgesics that did not control her pain.

Pulp and periapical diagnostic testing in conjunction with a radiographic examination, revealed that the mandibular left canine responded normally to all tests and that no periradicular involvement was noted (Fig. 3). Incipient recurrent decay was noted adjacent to the existing composite restorations. All adjacent teeth in both arches on the left side were also tested and all were found to be within normal limits. The patient had generalized chronic periodontitis.

After careful diagnostic testing of the patient's dentition, the history of the skin lesions was reviewed. The skin lesions appeared to follow the sensory distribution of the third branch of the trigeminal nerve on the left side of the face. The isolated lesion over the cheek near the ear was believed to follow the sensory distribution of the auricular-temporal nerve. Very small vesicles were present. No intraoral lesions were noted on this visit. A diagnosis of *H. zoster* was made and confirmed by the oral pathology department and a dermatologist in the stomatology clinic. The patient was referred to her primary care physician for treatment.

The patient returned to the dental clinic for follow-up evaluation of her condition the next day. Pustules had begun to appear and intra-oral lesions were noted. One lesion was located in the floor of the mouth and one lesion was noted lingual to the retromolar pad. In late February 1997, the patient returned to the clinic to have the mandibular left canine restored. The tooth responded normally to all diagnostic tests. In mid-March 1997 the patient presented for other dental treatment. At this time it was noted that the rash had healed but residual pigmented scarring was present. The patient was symptom free (Fig. 4).

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