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CASE STUDY

HERPES ZOSTER OF TRIGEMINAL NERVE AFTER DENTAL EXTRACTION: A CASE REPORT

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ABSTRACT

Herpes Zoster also known as Shingles is an acute viral infection which is an extremely painful and incapacitating ailment. It results from the reactivation of the varicella zoster virus. The triggering factors for the onset of an attack of Herpes Zoster include some form of immuno suppression. The diagnosis of Herpes Zoster can be made on proper medical history and a thorough clinical examination. During the prodromal stage, the only presenting symptom may be odontalgia, which may prove to be a diagnostic challenge for the dentist. Dentist must be familiar with the presenting signs and symptoms of patients experiencing the prodromal manifestations of herpes zoster of the trigeminal nerve. Here is the case report of a female patient affected by Herpes Zoster infection which followed after extraction of a lower first molar.

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INTRODUCTION

Herpes zoster (HZ) is an acute viral infection caused by reactivation of varicella zoster virus (VZV). Following the primary varicella infection (chickenpox), typically in childhood, VZV establishes latency in dorsal root or cranial nerve ganglia. (Owotude *et al*., 1999) Reactivation of VZV, although uncommon, results in its spread from the ganglion to the corresponding dermatome(s), producing neurocutaneous signs and symptoms – HZ or shingles. (Schmader and Dworkin, 2008) Herpes zoster of the trigeminal neuralgia is a disease that falls within the diagnostic purview of all dentist and dental specialist. During the prodomal stage of this disease in particular,the only presenting symptom may be odontolgia; this may prove to be a diagnostic challenge to the clinician which is not familiar with Herpes zoster of the trigeminal nerve.(3) Other diagnoses in the early stages of symptoms may include irreversible pulpitis, acute periapical periodontitis, or even acute sinusitis.

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Prompt management is required, especially in immunocompromised individuals, to prevent complications, which may cause significant morbidity. (Patil *et al*., 2013) The practicing dentist must be familiar with the presenting signs and symptoms of patients experiencing the prodromal manifestations of HZ of the trigeminal nerve.

Case Report

A 55 yr old female patient reported to the department of oral medicine and radiology with chief complaint of pain in lower right back tooth region since 1 week. No prior medical or dental history of any disease revealed by patient. The patient was housewife from a rural household, vegetarian in diet, often used homemade remedies for dental treatment. On intra oral examination, right mandibular 1st molar was carious/ grossly decayed with slight tenderness on percussion. An additional diagnosis of generalized periodontitis was also made with poor oral hygiene of patient. The patient reported back to our OPD after 5 days of extraction with complain of painful blisters on right side of the face. On clinical examination, widespread vesicles and crusting ulcerations mostly over the lower lip, right side of cheek, extending till the periauricular region was seen. (Figure 1) No cranial nerve neuropathies were noted, with

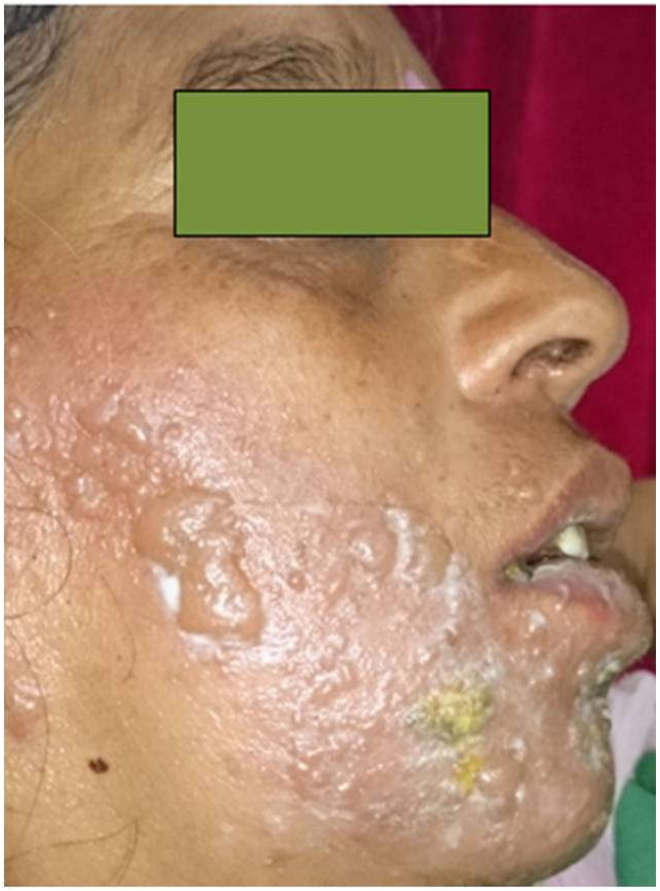

Fig. 1. Extraoral photograph: Unilateral distribution with widespread vesicles and crusting ulcerations mostly over the lower lip, right side of cheek, extending till the periauricular region

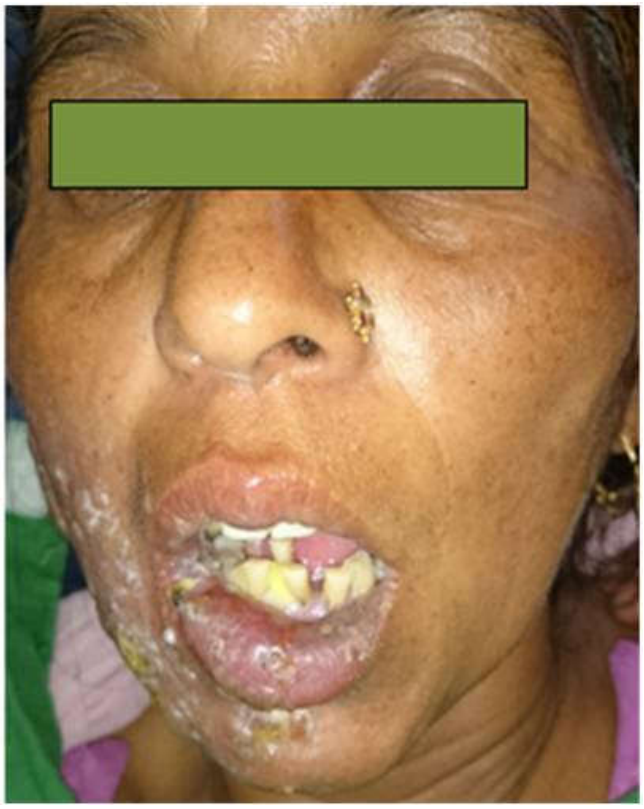

Fig. 2. Intra and extra oral photograph: showing unilateral vesicles, erythema, involving lower lip, labial mucosa, right side of face

all other nerves being grossly intact. Intraorally, vesicular eruptions, erythema and areas of ulcerations were noted unilaterally over the distribution of the maxillary and mandibular nerves, involving the labial and buccal mucosa. (Figure 2). A diagnosis of herpes zoster was made. To substiante the diagnosis, further questioning about past medical history revealed patient had positive history of varicella zoster infection in childhood. A provisional diagnosis of HZ of the trigeminal nerve was made and he was referred to dermatologist for further management. Dermatologist further confirmed the diagnosis of HZ of the trigeminal nerve and started acyclovir (ACV) course of treatment.

DISCUSSION

Herpes Zoster has an estimated life time incidence of 10–20% and gets reactivated with some form of immunosuppression. Herpes Zoster infection is common in elder persons, HIV positive individuals, and patients affected by malignant blood dyscrasias or malignant tumours or undergoing immune suppressive therapy and radiotherapy. (Patil *et al*., 2013) A Case of Herpes Zoster affecting the trigeminal nerve is reported here. This case highlights the importance of a thorough dental history and examination in patients with odontalgia. In those presenting with atypical ondontalgia, HZ should be considered in the differential diagnosis. With an ever increasing number of elderly and immunocompromised patients attending the dentist, the dental professional can expect to encounter an increasing number of HZ patients. The practicing dentist must be familiar with the presenting signs and symptoms of the patients experiencing the prodromal manifestations of HZ of the trigeminal nerve. Furthermore, appropriate decision making and referral to the concerned specialist seems to be utmost importance as seen in this case. Reactivation of VZV may occur spontaneously or when host defenses are compromised. Increasing age, physical trauma (Schmader and Dworkin, 2008) (including dental manipulation), (West, 1970) psychological stress, (Owotude *et al*., 1999; Schmader and Dworkin, 2008) malignancy, radiation therapy, and immunocompromised states including transplant recipients, immunomodulatory therapy, and HIV infection (Buchbinder *et al*., 1992) are predisposing factors for VZV reactivation. The case reported here, as well as others with increased age, physical trauma (Thomas and Hall, 2004) (including dental manipulation), (West, 1970) and psychological stress exhibit an impaired host immune response which may contribute to the development of HZ. (Wung *et al*., 2005) Involvement of the second and third branches of the trigeminal nerve results in vesicular lesions in oral cavity. Patients with HZ may progress through three stages: Prodromal stage, active stage (also called acute stage), and chronic stage. (Strommen *et al*., 1988)

Characteristic signs of oral HZ are the presence of unilateral vesicles that break rapidly, leaving small ulcers. On skin and lips, vesicle ruptures can result in erosions covered by pseudomembranes and haemorrhagic crusts which were also seen in our patient. By the end of second or third week the crusts and pseudomembranes disappear with eventual healing of the vesicular lesions. (Miller and Troulis, 1994; Strommen *et al*., 1988) The prodromal stage presents as sensations (described as burning, tingling, itching, boring, prickly) occurring in cutaneous distribution of the dermatome and is believed to represent viral degeneration of nerve fibrils. (Miller and Troulis, 1994) During this period, if branches of the trigeminal nerve are affected, odontalgia and pulpal necrosis may be seen. Furthermore, these symptoms may present up to 1 month before the acute mucocutaneous lesion, and pose

significant diagnostic difficulties. The active stage is characterized by the emergence of the rash which is nearly always accompanied by systemic upset. The characteristic skin rash progresses from erythematous papules and edema to vesicles and finally to pustules within 1-7 days which dry and crust and are exfoliated over 2-3 weeks, leaving erythematous macular lesions that may scar. The chronic stage is only seen in approximately 10% of all patients with HZ, and is termed post-herpetic neuralgia (PHN) within one to three months of healing of VZ lesions and is characterised by pain, paresthesia, hypoesthesia, or allodynia and can persist for months and years and is a significant cause of morbidity. Other complications include neurological disorders, and ophthalmologic, cutaneous, and visceral complications; (Schmader and Dworkin, 2008) immunocompromised individuals with HZ exhibit a significantly higher rate of complications. Periapical lesions, root resorption, tooth exfoliation, and alveolar osteonecrosis have also been reported in association with HZ infection.

However, some patients donot develop symptoms of all stages. Some patients donot form vesicular eruptions of the active stage, but do develop pain restricted to a dermatome, and this has been termed zoster sine herpete which makes proper diagnosis more difficult.[10] Surprisingly, pain is reported to subside when the rash is most active; however, it returns during the crusting and scale phase until the rash clears. It is during the active or "eruptive" phase that HZ is most contagious and could pose a significant cross-infection risk. Definitive diagnosis often involves a process of elimination, with several likely aetiologies in the differential diagnosis. A differential diagnosis should include trigeminal neuralgia, periodic migrainous neuralgia, atypical facial pain, herpes simplex. Thus, the dental practioner should be familiar with the classic 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. A 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 spine herpete. Culturing may be required to confirm a H.zoster infection in these patients. Careful history and a thorough dental examination usually rule out other pathology.

Management

Although HZ is a self-limiting condition and resolution is usually complete, treatment is indicated in some cases to reduce the acute symptoms of pain and malaise, to limit the spread and duration of the skin lesions, and to prevent complications. 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. On cutaneous lesions 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. The acute pain of H.Zoster infections can be reduced during the prodomal and the acute phases. Mild to moderately strong analgesics, such as acetaminophen, codein and non steroidal anti-inflammatory agents are effective. However, these analgesics are notoriously ineffective for the chronic postherpectic neuralgia (PHN) phase. (Strommen et al., 1988) 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. The treatment for PHN pain includes the topical use of capsaicin cream (zostrix), transcutaneous nerve stimulation, topical anaesthetics, injected local anaesthetics and lowe dose amitryptilline (Elavil). Once a diagnosis of H.zoster infection has been done, anti viral therapy is recommended. Acyclovir has been the drug of choice, 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 hours of the onset of the rash. (Strommen et al., 1988; Barrat et al., 1993) The dose regimen range from discipline to discipline, 800mg four times a day for 10 days remains the standard of care. Research has shown that dosages of up to 800mg five times a day have been given more promising results. Recently, other antiviral agents such as valaciclovir and famciclovir have been developed to overcome the low oral bioavailability of Acyclovir and its limited and less predictable effect in preventing the development of post-herpetic neuralgia, as well as to provide a more favorable dosage regime (Miller and Troulis, 1994) In the present case, dental treatment related stress and increased age would be the contributing factors for the reactivation of the varicella zoster; however, as the patient reported at an earliest stage to the dentist, the case was suspected for HZ and was referred to the dermatologist, where it was managed with antiviral drugs. The dentist plays an important role in identification of such cases at an early stage that would help in the early diagnosis and prompt treatment, thus decreasing the duration and complication of such viral infection.

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