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CASE REPORT

Herpes zoster induced alveolar necrosis in an immunocompetent patient

Veeranna Guledgud Mahima*, Karthikeya Patil, Hanasoge Srivathsa Srikanth
Department of Oral Medicine and Radiology, J.S.S Dental College and Hospital, Karnataka, India

ABSTRACT

Background: Herpes group of viruses give rise to vivid manifestations in the human body. Herpes zoster infection and reactivation results in characteristic vesiculo-bullous-ulcerative lesions of the face and the oral cavity. Herpes zoster induced alveolar bone necrosis is a rare manifestation and very few cases reports are available in the literature.

Patient: A 60-year-old male patient presented with a complaint of an ulcer over the lower lip since a month. Based on the clinical features, a diagnosis of herpes zoster of mandibular division of right trigeminal nerve inducing alveolar necrosis with spontaneous exfoliation of teeth was proposed.

Conclusion: Herpes zoster infection is a rather common viral infection. The manifestations of this infection can range from subtle to extensive lesions such as alveolar bone necrosis and exfoliation of teeth. When such widespread expression of the infection is noted the clinician should investigate to determine an underlying immunocompromised status.

Keywords: *Herpes virus; Zoster; Alveolar necrosis; Spontaneous exfoliation of teeth.*
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INTRODUCTION

Herpes group of viruses give rise to vivid manifestations in the human body. Oral cavity also shows many of these manifestations, some of which are quite rare.

Herpes zoster infection and reactivation results in characteristic vesiculo-bullous-ulcerative lesions of the face and the oral cavity. Less is known about the involvement of the hard tissues of the oral cavity such as teeth and alveolar bone.

Herpes zoster induced alveolar bone necrosis is a rare manifestation and very few cases reports are available in the literature. Most often this extensive manifestation is noted in immunocompromised subjects and rarely in immunocompetent individuals.

CASE PRESENTATION

A 60-year-old male patient presented with a complaint of an ulcer over the lower lip since a month. The patient had noticed the development of fluid filled blisters on the right side of the face including the lower lip, one month back preceded by burning sensation over the same side of the face. The blisters had ruptured and healed completely in the next 15 days except for the ulcer that formed on the lower lip. In the following fortnight patient noticed mobility of his lower right posterior teeth

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Reprint or Correspondence: Hanasoge Srivathsa Srikanth, MDS. Department of Oral Medicine and Radiology, J.S.S Dental College and Hospital, Mysore-570015, Karnataka, India.
E-mail:Srikanth_vathsa2000@yahoo.com

which exfoliated spontaneously in two days without causing much discomfort.

Medical and surgical histories were unremarkable. Examination of the skin of the face on the right side revealed multiple, irregular hyper-pigmented and hypo-pigmented scars scattered over the temple, malar surface, para-symphysis region, abruptly stopping at the midline. The vermilion of the lower lip showed a large, solitary ulcerative defect with irregular borders having granulation tissue in the floor surrounded by hypo pigmented area. The ulcer was non tender and non-indurated (figure 1).

Figure 1. Large ulcerative lesion on the lower lip

Intraoral examination revealed the mandibular right alveolar process to be denuded of the soft tissue from the central incisor up to the first molar region. The sockets of the corresponding teeth were filled with food debris (figure 2). The oral mucosa did not show any other abnormality. A routine blood investigation showed values within normal limits. Patient was non-reactive for HIV 1 & 2. (ELISA and Western blot). A panoramic radiograph showed only the empty teeth sockets on the right side of the mandible with no sequestrum (figure 3).

Panoramic radiograph also showed a solitary well defined, unilocular radiolucency, located below the right inferior alveolar canal, which was radiographically diagnosed as developmental salivary gland depression (Stafne's cyst).

Based on the above features a diagnosis of herpes zoster of mandibular division of right trigeminal nerve inducing alveolar necrosis with spontaneous exfoliation of teeth was arrived at. Treatment included alveolectomy and reconstruction of the lower lip defect under general anesthesia. The patient tolerated the procedures well and the recovery was uneventful.

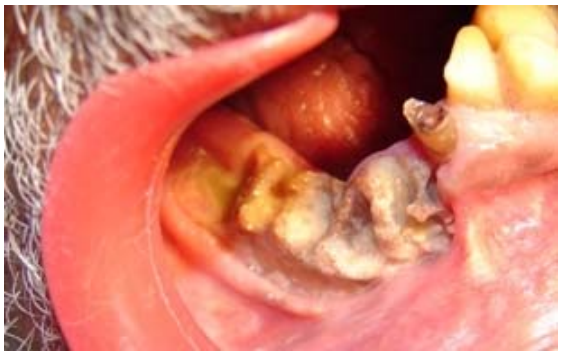

Figure 2. Mandibular alveolus with food debris

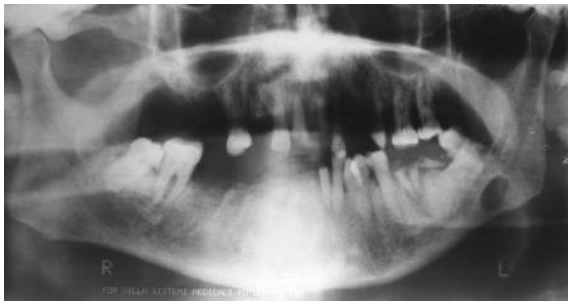

Figure 3. Panoramic radiograph showing alveolar bone without sequestra

DISCUSSION

Varicella-zoster virus is a DNA virus which primarily causes chicken pox and when reactivated causes shingles or herpes zoster (1). The characteristic feature of this virus is indefinite

dormancy in the regional ganglia, after the primary infection (1). Herpes zoster commonly involves the branches of trigeminal nerve in the head and neck region, the ophthalmic and maxillary branches more commonly than the mandibular branch (1). The virus can get reactivated due to underlying causes such as an immunocompromised status secondary to HIV infection, hematologic malignancies, diabetes mellitus or cytotoxic chemotherapy and irradiation (2) as well as following stress and local trauma (3). Though an underlying cause for reactivation of the latent virus has been found in most of the cases (3,4), a few are found in otherwise healthy individuals. The proposed mechanism for reactivation in such cases is old age with deteriorating immune response (4).

During reactivation, the viruses travel along the nerve branches and reach the skin and the mucosa to cause painful vesicles that rupture, crust and heal (4,5). The skin lesions heal with either hypo- or hyper-pigmentation (6). Osseous and dental manifestations such as devitalized teeth, internal resorption, abnormal development of permanent teeth, spontaneous exfoliation of teeth and necrosis of maxilla and mandible have been reported (5).

Pathogenesis of osteonecrosis is not well understood. The proposed hypothesis includes: local vasculitis caused by extension of inflammation to the blood vessels leading to infarction of the vessels and necrosis of bone (2,5); infection of the terminal branches of trigeminal nerve supplying the periosteum and periodontium (2,5); and denervation of bone (3,4,6). The first two mechanisms of alveolar necrosis have received more acceptances (5).

The commonest age groups affected by herpes zoster-induced alveolar necrosis are the fourth through sixth decades. No gender or jaw preponderance has been observed (1). Among the forty reported cases of herpes zoster-induced alveolar necrosis, only about ten patients had no detectable underlying immunocompromised state (1). In the present case, there was no identifiable immunocompromised status, except for old age. Exfoliation of the teeth is brought about by co-infection of the periodontium with herpes virus which hasten the periodontal breakdown by causing reduction in periodontal defense, inducing vasulitis and altering blood flow (3,7). Pre-existing pulpal and periodontal infection are said to contribute further, to the above mechanism of tooth exfoliation (5). Our patient had chronic periodontitis, which could have further contributed towards exfoliation of teeth.

Clinically patients will often report with a history of low grade fever, tingling, pruritis of the skin followed by occurrence of classic vesicles and bullae that rupture to leave crusts healing in 7-14 days (7). Following the appearance of skin or the oral lesions the osseous lesions occur. A mean duration of 21 days from the onset of the skin lesions has been described for the occurrence of alveolar necrosis and teeth exfoliation (7). Although, herpes simplex virus can cause skin lesions identical to herpes zoster, manifestations like exfoliation of teeth and alveolar necrosis do not occur. Also, zoster lesions when occur are classically unilateral and abruptly stop at the midline. Skin lesions of impetigo can superficially resemble zoster lesions, but they neither occur unilaterally nor cause teeth exfoliation or alveolar necrosis (6).

Tooth exfoliation in herpes zoster is considered by a few authorities as the earliest sign of osteonecrosis (7). On the contrary, a few report it as a late complication, occurring between 3 to 12 weeks after the onset of skin lesions (3). Back to our case, exfoliation of teeth occurred approximately 15 days following the onset of skin lesions.

Osteonecrosis clinically appears as denudation of bone with exposure of teeth sockets (2,4,6,7). IOPAR and Panoramic radiographs may show empty teeth sockets and sequestrations of necrotizing bone (5,6,8) and rarely a “moth-eaten” appearance of the underlying bone (2). Radiograph

in the present case only showed empty teeth sockets.

Recently, numerous laboratory diagnostic methods have been developed for the diagnosis of herpes zoster infection. These include dot-blot hybridization and polymerase chain reaction and direct staining of cytologic smears with fluorescent monoclonal antibodies for VZV. Along with these, histopathologic examination of the necrotizing alveolar bone has been proposed (6).

There were a few hurdles in the present case; non availability of such advanced diagnostic aids such as PCR; no active skin lesions for direct staining technique; and finally, histopathologic examination being generally non-specific and not pointing towards the diagnosis of herpes zoster infection (4,5).

Histopathologic findings of the necrotizing bone are considered non-specific, but may show areas of bone necrosis and mixed inflammatory infiltrate (5). Treatment for herpes zoster induce alveolar necrosis includes local debridement and alveolectomy/sequestrectomy (5). Clinical presentation of herpes zoster is often very classic and the diagnosis is essentially clinical (6).

In conclusion, herpes zoster infection is a rather common viral infection. The manifestations of this infection can range from subtle to extensive lesion such as alveolar bone necrosis and exfoliation of teeth. When such widespread expression of the infection is noted the clinician should investigate to determine an underlying immunocompromised status. What the clinician needs to be sentient about is the possibility of an overt response in otherwise healthy individuals as well. Early intervention is essential to prevent secondary bacterial infection of the necrotizing bone which may further complicate the condition.

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