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Tooth Pain, Alveolar Bone Necrosis and Spontaneous Exfoliation of Multiple Teeth Following Herpes Zoster Infection of Trigeminal Nerve: A Case Report

Dr Manjeet Singh

(BDS, MDS) (Periodontology), MO Dental Civil Hospital Sarkaghat District Mandi Himachal Pradesh, India

Abstract: ***Herpes zoster (HZ) presents as cutaneous vesicular eruptions in the areas innervated by the affected sensory nerve and is usually associated with severe pain. Oral manifestations of herpes zoster appear when the maxillary or mandibular divisions of the trigeminal nerve are involved. Infection commonly occurs unilaterally and affects mostly middle aged or the elderly persons. Multiple complications have been reported following herpes zoster of the trigeminal nerve. The most common among them is the neuralgia. There are only a few reports of bony and dental complications following HZ infection. The aim of this article is to present a case of a 44 years old male patient affected by herpes zoster infection involving trigeminal nerve which led to necrosis of bone and exfoliation of teeth.***

Keywords: Herpes zoster, trigeminal nerve, neuralgia, alveolar bone loss, teeth exfoliation, VZV.

1. Introduction

Varicella-zoster virus (VZV) is a member of the herpes virus group. As all other viruses from this group, VZV can manifest itself as a recurrent infection. After entering the body and causing primary infection, varicella-zoster virus remains latent in the neurons of sensory ganglion, especially dorsal roots of ganglion of the spinal nerves and extramedullar ganglion of the cranial nerves. VZV infection is common in elder persons, immunocompromised or HIV positive individuals, and patients affected by malignant blood dyscrasias, malignant tumours, or undergoing immunosupressive therapy and radiotherapy. Varicella (chickenpox) is the primary infection of VZV and it is very common among children of both sexes. Herpes zoster (shingles) is the recurrent form of infection and occurs in the 3-5% of population, mainly among older individuals and immunocompromised. In 10% of HIV positive patients, HIV disease starts with herpes zoster infection in the oral cavity as an oral opportunistic infection. Reactivation of infection is infrequent in younger people and children. When branches of the trigeminal nerve are involved, lesions appear on the face, mouth, eyes and tongue.[1] The lesions are commonly found in the thoracic dermatome. The typical history is of itching, tingling & burning sensation followed by painful vesicular eruptions along the course of the nerve. Osseous changes related to HZ infection were first reported by Rose in 1908. Since then, only a few cases have been reported on this rare complication.[1-3]

2. Case Report

A 44 year old male patient, diagnosed with herpes zoster infection of face was referred from the Department of Dermatology to Department of dentistry for the diagnosis and treatment of painful lesions present intraorally and extraorally on left side. Patient presented with history of traumatic extraction of left mandibular first molar 15 days ago, which was followed by tingling and burning sensation on the left side of the face and continuous severe pain in upper and lower left teeth. This was followed 4-5 days later by appearance of multiple vesicles affecting skin of left face region from below the eye to the mandible. The vesicles ruptured spontaneously led to formation of ulceration and crustations, which was followed by unaesthetic scars on left lateral canthal area, zygoma, cheek, and left parasymphyseal area. Fig.1. Right side was not involved. The medical history was not significant for any serious disease or any long term medication. Physical examination revealed he was well built, nourished, and afebrile. Routine laboratory tests and investigations were carried out. All hematologic values were within normal limits, ELISA for HIV was negative.

Intraoral examination showed painful, multiple ulcerations covered by whitish Pseudomembrane on labial mucosa, buccal mucosa, tongue, labial vestibule and peritonsillar area of left side. Fig. 2,3. Intraoral and panoramic radiographs showed generalized moderate bone loss. Fig.4&5.The patient was already receiving Tab. Acyclovir 800mg 5 times a day. Patient was placed on amoxicillin 500mg and potassium clavulanate 125 mg three times a day and was instructed to rinse with 0.12% chlorhexidine digluconate. After 1 month patient reported with exfoliation of remaining left mandibular teeth spontaneously and grade II mobility in the maxillary left teeth, intraoral and extraoral lesions had resolved completely. Fig.6. Intraoral radiographs showed advanced alveolar bone loss in the regions of exfoliated teeth.Fig.7 and Fig.8.

3. Discussion

Varicella-zoster virus is a DNA virus which primarily causes chicken pox and when reactivated causes shingles or herpes zoster.[4] Trigeminal nerve involvement is usually unilateral[5,6] and limited to a single division, more often the first (ophthalmic).[7] Oral manifestations appear when the second

(maxillary) or third (mandibular) trigeminal divisions are affected. Frequently, the intraoral lesions are associated , as in this case, with cutaneous lesions affecting the corresponding area innervated by the affected sensory nerve.[3]

Patients with HZ infections usually progress through three stages: (1) prodromal stage, (2) active stage (also called acute stage), and (3) chronic stage. However, some patients do not develop symptoms of all the stages. Some patients do not form vesicular eruptions of the active stage, but pain develop restricted to a dermatome, and this has been termed ***Zoster sine herpete*** which makes proper diagnosis more difficult. 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. This usually precedes the rash of the active stage by a few hours to several days. The patient may present with an odontalgia that may be the only prodromal symptom. The active stage is characterized by the emergence of the rash that may be accompanied by generalized malaise, headache, low grade fever and some times nausea. The rash progresses from erythematous papules and edema to vesicles in 12 to 24 hours and finally progresses to pustules with in 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. 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.[8] The intraoral vesicles scattered and surrounded by an erythematous zone, soon become ulcerated and covered by a white pseudomembrane.[9] The patient may have general malaise, and lymphadenopathies may be present in the submandibular region. Spontaneous exfoliation of teeth in the area innervated by the affected nerve has been reported. Some authors believe that this is an early event occurring during the first 2 weeks of the infection, while others consider this to be a late complication that will occur between the 3rd to 12th weeks after onset. Loss of teeth is due to alveolar bone necrosis and/or to necrosis of the periodontal ligament.

The mechanisms by which the HZ infection leads to the alveolar bone necrosis are not well known. It has been suggested that preexisting pulpal or periodontal inflammatory conditions or surgical procedures performed in the site of a zoster infection have the potential to contribute to more destructive alveolar bone necrosis. Our patient did not have pulpal pathologic conditions in the affected teeth and, although he had gingivitis and early lesions of chronic periodontitis that may have contributed to the severity of the destruction, the case may be that, as it has been suggested previously, a vasculitic component related to the HZ infection contributed to an obliteration of the vessels supplying the left mandible. Wright et al. postulated that the necrosis could be the result of ischemia related to an infarction of vessels supplying the teeth. Direct invasion of blood vessels by virus spreading from adjacent cranial nerves and segmental granulomatous vasculitis, associated with HZ infection, with multifocal infarcts in the brain and spinal cord have been reported. Therefore, it has been suggested that considering the close anatomical relationship between virus infected fifth cranial nerve branches and blood vessels, this vasculitis component may contribute to the infarction of the vessels. Probably an ischemic problem with these characteristics would result in more than one tooth affected.[3] Pre-existing pulpal and periodontal infection are said to contribute further, to the above mechanism of tooth exfoliation. Our patient had chronic periodontitis, which could have further contributed towards exfoliation of teeth[4].

Following primary infection, the virus is latent in the neurons of the sensory ganglia and reactivates itself as a consequence of immunodeficiency. VZV affects neighboring neuron ganglia and it might affect several branches of the nerve. Viruses spreading through sensory parts of the second and third branch of the trigeminal nerve, lead to the pathological changes in the oral cavity. The viral presence further leads to the acantholysis in the prickle cell of the epithelium and formation of the vesicles. Because of the subtle overlying layer, vesicles rupture rapidly, leaving erosions. VZV damages peripheral nerves through demineralization, leading to sclerosis and degeneration.[1]

Definitive diagnosis often involves a process of elimination. A differential diagnosis should include trigeminal neuralgia, maxillary sinusitis, periodic migrainous neuralgia, myocardial pain, atypical facial pain and Munchausen`s syndrome.The diagnosis of HZ is clear when the prodomal symptoms are present and the dermatomal vascular rash is present. A diagnostic challenge is created when the vascular rash does not occur, as in zoster sine herpete.[8] Diagnosis is made on the basis of clinical manifestations and subjective symptoms, presence of the viral antigens as well as presence of antibodies against VZV. The best laboratory diagnostics are PCR and direct VZV identification in the cell culture of human fibroblasts. Serological findings are helpful in recurrent VZV infections and show increased IgM, ten days after eruptions and increased IgG and IgA four days after the eruptions. Serological tests which reveal antibody titers might be useful in immunocompromised patients.[1] 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, histopathological examination being generally non-specific and not pointing towards the diagnosis of herpes zoster infection. Histopathological findings of the necrotizing bone are considered non-specific, but may show areas of bone necrosis and mixed inflammatory infiltrate. Recommended management for the patient with HZ should include:

Isolation: Patients with HZ infections are contagious to the persons at risk. This includes neonates, non-immune persons, pregnant women, and immune-compromised patients. The contagion is transmitted as varricella zoster virus (chicken pox). HZ patients remain contagious until crusting and scaling have taken place.

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. Ointments may be used after the acute phase to soften and remove adherent crusts.

Pain: Mild to moderately strong analgesics, such as acetaminophen, codeine and non-steroidal anti-inflammatory agents are effective. However, these analgesics are notoriously ineffective for the chronic post- herpetic neuralgia phase.

Anti-viral drug therapy: Once the diagnosis of HZ infection has been determined, anti-viral therapy must be swift and precise. Acyclovir has been the drug of choice for number of years. Recently, newer forms of anti-viral drugs have been developed specifically to address the acute stage of HZ (Famiciclovir) and for use in immunocompetent patients (Valacyclovir).

Post herpetic neuralgia: standard analgesic narcotic combinations are not effective in patients with post herpetic neuralgia (PHN). The treatment for PHN pain includes the topical use of capsaicin cream, transcutaneous nerve stimulation, topical anaesthetics, injected local anaesthetics and low dose amitriptyline . No single treatment is universally effective for all PHN patients.

4. Conclusion

Multiple factors must be considered by the dentist in the diagnosis and management of cases of herpes zoster. Initially, because the nature of 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 attack, 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. This unfortunate circumstance can occur when no vesicles appear or their presence is mistaken for aphthous stomatitis. A second situation facing by the dentist is the fact that a herpetic attack of the trigeminal nerve can cause pulpal necrosis. 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. Likewise, previous episode of herpes 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 herpes zoster often than others, the dentist must be constantly altered to the possibility of this differential diagnosis. Herpes zoster infection may lead to extensive osteonecrosis and exfoliation of the teeth in the area innervated by the affected nerve. Awareness of the potentially debilitating complications is necessary among the clinicians to prevent further complications.

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Figures

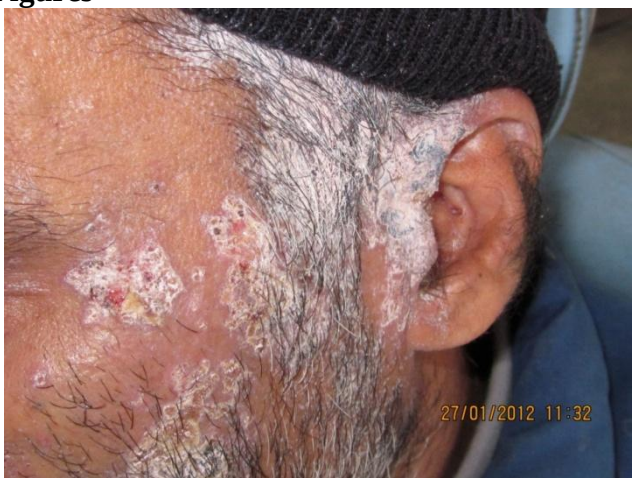

Figure 1: Herpes zoster lesions and scars on left lateral canthal, zygoma, and cheek area

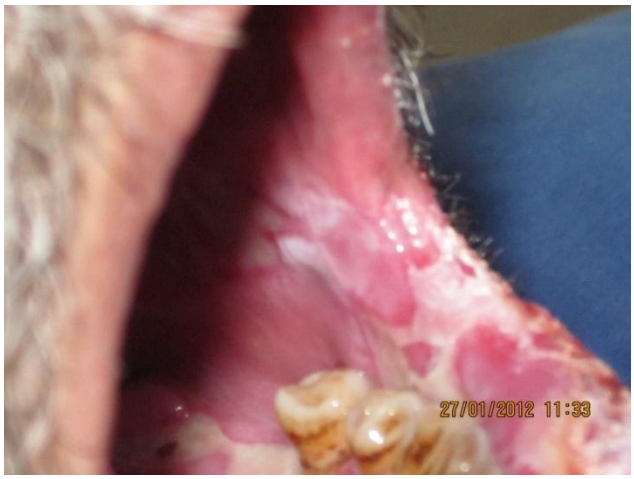

Figure 2: Herpes zoster lesions on labial mucosa, buccal mucosa, and left labial vestibule

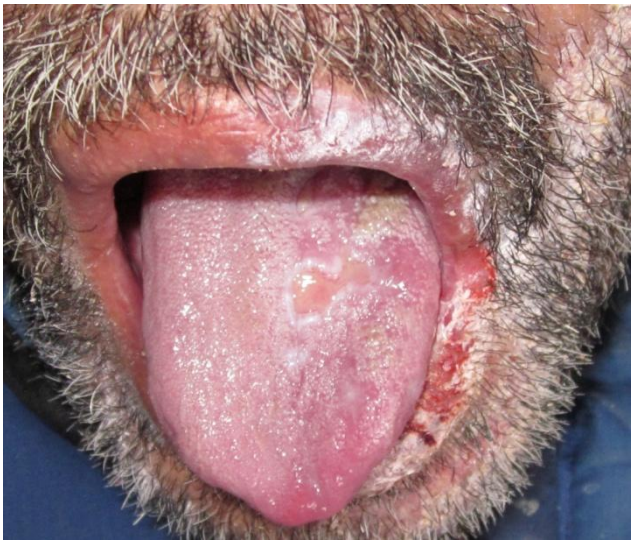

Figure 3: Herpes zoster lesions on tongue

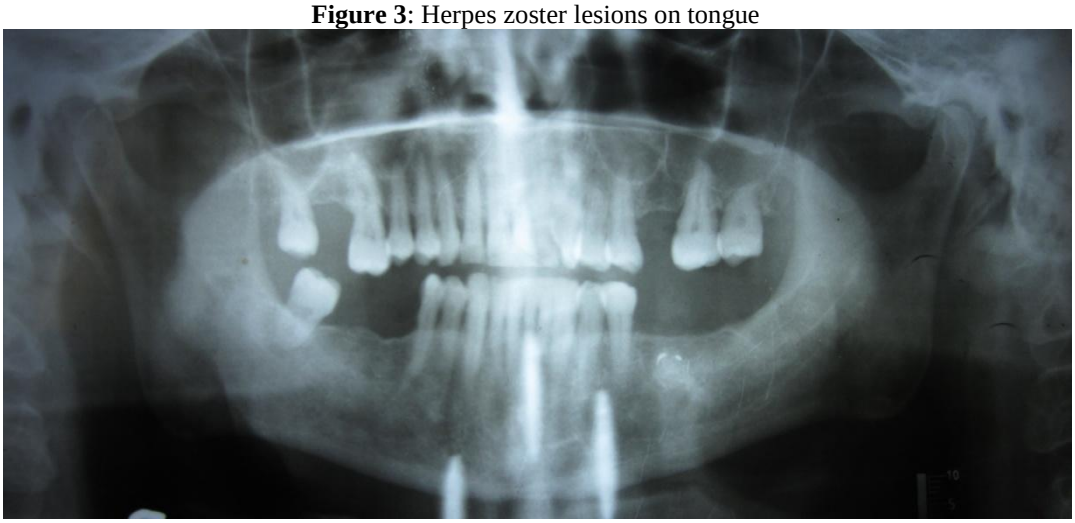

Figure 4: OPG view showing generalized moderate bone loss.

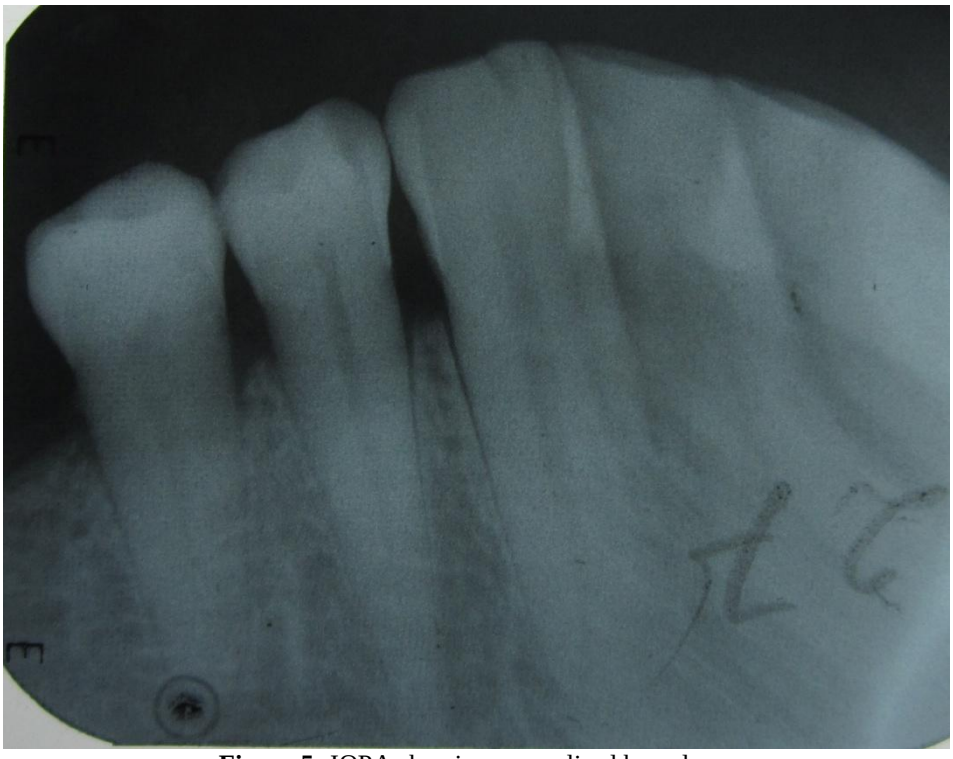

Figure 5: IOPA showing generalised bone loss.

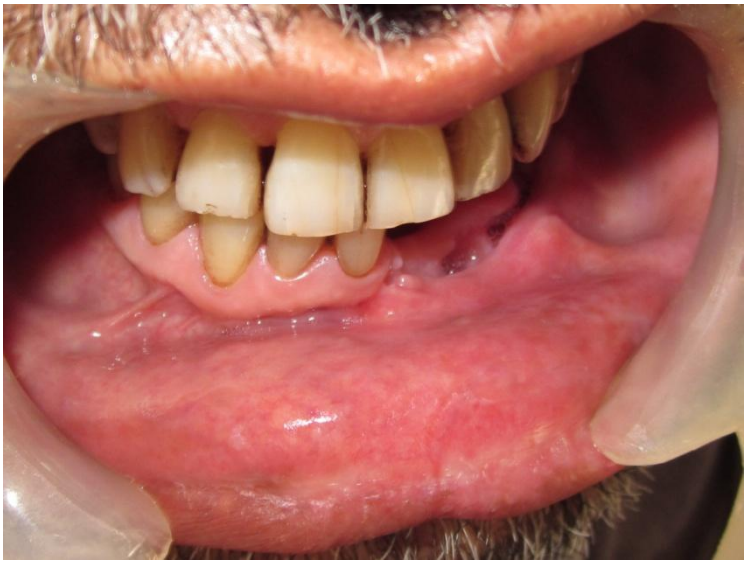

Figure 6: Exfoliated teeth and alveolar bone loss in affected area

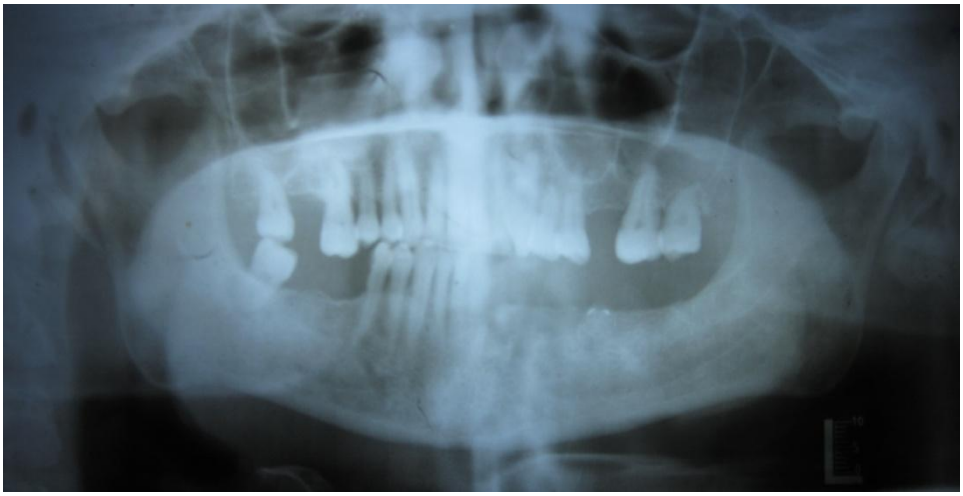

Figure 7: OPG view showing exfoliated teeth on mandibular left side and associated bone loss

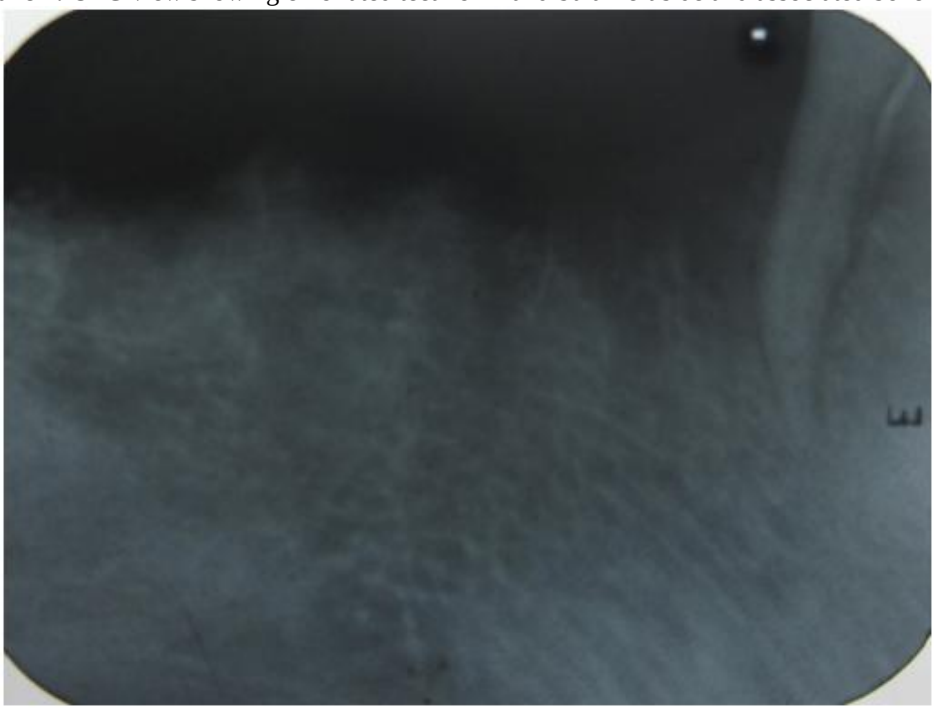

Figure 8: IOPA shows exfoliated teeth on mandibular left side and associated bone loss