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ORIGINAL ARTICLE

Herpes zoster in HIV infection with osteonecrosis of the jaw and tooth exfoliation

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BACKGROUND: Herpes zoster (HZ) infection of the trigeminal nerve is associated with complications such as postherpetic neuralgia, facial scarring, loss of hearing ability and conjunctivitis. Until 2005, postherpetic alveolar necrosis and spontaneous tooth exfoliation have been described in 20 cases unrelated to HIV infection.
OBJECTIVE: The aim of this study was to describe HIV infection in patients (two women, two men, average age 30 years) who suffered from HZ attacks to their trigeminal nerves.
MAIN OUTCOME MEASURES: None of the patients had received antiherpetic medications or antiretroviral therapy. HIV infection was only diagnosed after the development of HZ. Facial scarring with depigmentation and hyperesthesia (postherpetic neuralgia) was diagnosed in all four patients. Oral findings consisted of spontaneous loss of both maxillary or mandibular teeth. Osteonecrosis of varying extent was also found. Treatment consisted of extractions of teeth and administration of antibiotics and analgesics. Healing of alveolar wounds was unremarkable.
CONCLUSION: Complications affecting the alveolar bone and teeth seem to be rare in HIV-infected patients.
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Keywords: HIV infection; herpes zoster; osteonecrosis; jaw; tooth exfoliation

Introduction

Varicella-zoster virus (VZV) is the cause of varicella (chickenpox) and herpes zoster (HZ). HZ occurs after reactivation of latent VZV in sensory ganglia. The virus spreads along the nerves to the associated dermatome causing vesicular eruptions. HZ of the trigeminal nerve is associated with painful vesicles of the skin and oral mucosa of the affected branch of the nerve. Usually, oral vesicles appear after skin manifestations. Oral vesicles rupture and coalesce presenting as large mucosal erosions (Van Wyk *et al*, 1992). Prodromal pain may occur in the distribution of the trigeminal nerve some days before vesicular eruptions. This pain may mimick toothache or pulpitis (Verbin *et al*, 1968; Gregory *et al*, 1975; Goon and Jacobsen, 1988). HZ usually is a self-limiting disease; however, complications, particularly in the immunocompromised individual, including cutaneous dissemination, prolonged atypical skin lesions, ocular complications and CNS involvement (Mintz and Anavi, 1992) have been described. According to Dechaume *et al* (1955), Gonnet was the first in 1922 to describe alveolar bone necrosis and tooth loss in association with HZ infection. Recently, Mendieta *et al* (2005) reported a case of trigeminal HZ infection associated with osteonecrosis and tooth exfoliation in a 63-year-old woman. The authors reviewed the literature from 1955 to 1999 – a period during which 20 cases of this type of complication of HZ infection have been described (Dechaume *et al*, 1955; Delaire and Billet, 1959; Hall *et al*, 1974; Chemitz, 1976; Vickery and Midda, 1976; Cooper, 1977; Delbrouck-Poot and Reginster, 1979; Dielert, 1979; Schwartz and Kvorning, 1982; Wright *et al*, 1983; Mostofi *et al*, 1987; Consolaro and Oliveira, 1990; Mckenzie and Gobetti, 1990; Toshitaka *et al*, 1990; Peñarrocha *et al*, 1992; Eury *et al*, 1993; Tidwell *et al*, 1999). Although underlying systemic diseases were recorded (chronic hepatitis, anemia, leukemia, reticulum cell sarcoma, Hodgkin's lymphoma, histiocytic lymphoma, brain tumor), in none of these patients was HIV infection as a possible associated factor recorded. However. VZV infection resulting in HZ is a well-recognized cause of morbidity in HIV-infected individuals (Glesby *et al*, 1995) and has been considered as an early manifestation of HIV infection (Friedman-Kien *et al*, 1986; Colebunders *et al*, 1988; Van de Perre *et al*, 1988). Few reports on HZ infection and oral complications including osteonecrosis

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and tooth exfoliation have been published (Schwartz *et al*, 1989; Chindia, 1997; Srisuwan, 1999). In Thailand, Srisuwan (1999) was probably the first to report on a patient with AIDS-related complex, who developed HZ infection of the trigeminal nerve with osteonecrosis of the alveolar bone and exfoliation of teeth.

The aim of this study was to describe HIV infection in patients who suffered from HZ attacks to their trigeminal nerves.

Case reports

Case 1

In June 2002, a 30-year-old woman was examined in the Oral Diagnosis Section, Faculty of Dentistry, Chiang Mai University. She had pain on the upper left lateral incisor and canine for a period of 3 months. Intraoral examination revealed an excessive loosening of the upper left incisor and canine with intensive gingival inflammation and spontaneous bleeding. Severe mobility of the upper left first and second premolar was also found. The left maxillary alveolar labial plate from the lateral incisor to the mesial wall of the upper first molar was partly exposed (Figure 1a). The vestibular oral mucosa of the alveolar process was erythematous along the alveolar bone. The oral mucosa of the left and right cheek, the dorsum of the tongue and the sublingual space was covered with white patches representative of oral pseudomembranous candidiasis. Radiographic examination showed alveolar bone loss with widening of the periodontal space from the upper left lateral incisor to the second premolar. No periodontal marginal bone resorption of the involved teeth was present.

On extraoral examination, marked, deep furrow-like scars on the left side of the face including the forehead, eye, ear, and upper lip were observed (Figure 1b). These facial regions were stiff with hyperesthesia. The patient had a history of severe attack of HZ in the previous 6 months without seeking any treatment. The process started with severe pain and vesicles on the left side of the face involving maxillary and ophthalmic divisions of the trigeminal nerve. Three months later, pain on the left upper maxillary teeth developed. Her left eye was almost blind and there was hearing loss of the left ear. A generalized papular pruritic eruption on the skin, prominent on hands, arms and legs, was also found. The patient's hemoglobin level was 12 g%, hematocrit 37.3%, white blood cell count 4.640 cells mm^{-3}.

A review of the social history revealed that her husband was HIV positive for 7 years. The patient tested HIV positive. The diagnosis of osteonecrosis of the left maxilla, secondary to infection by HZV associated with HIV infection was established.

The following treatment was carried out: the upper left lateral incisor, canine, first and second premolars were extracted together with a sequestrum of alveolar bone measuring approximately 1.5×3.0 cm in diameter (data not shown). The medications given orally were Brufen 1200 mg day^{-1}, amoxycillin 2 g day^{-1} and metronidazole 600 mg day^{-1} for 7 days. Nystatin suppository 1:100 000 IU and ketoconazole orally 400 mg day^{-1} were prescribed for treating *Candida* infection. After 1 week, intraoral wound healing was satisfactory.

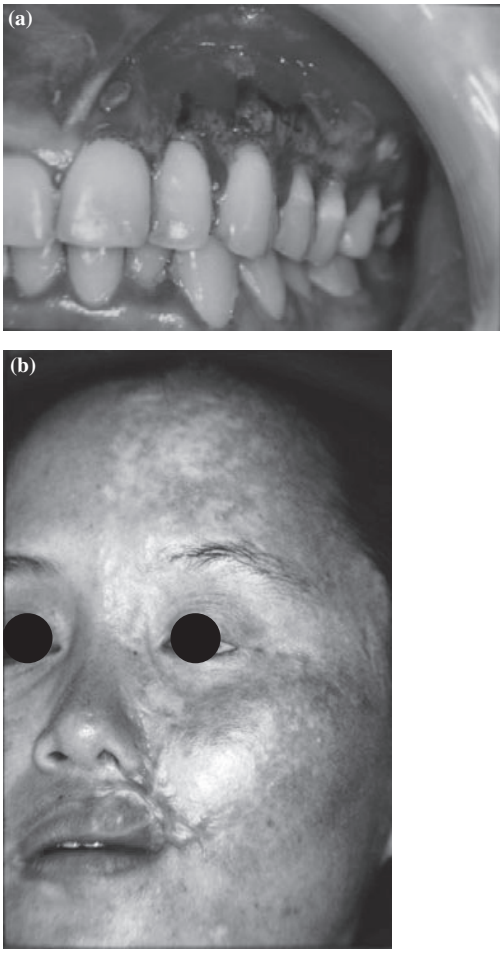

Figure 1 Case 1. (a) Extensive ulceration of the gingiva and adjacent oral mucosa of the left maxillary quadrant. The alveolar bone is partly exposed. Some whitish changes in the vestibule indicate the presence of pseudomembranous candidiasis. (b) Extensive scar formation on the left side of the face and lip showing previous involvement of the first and second branch of the trigeminal nerve. Scarring is particularly severe in the area of the nasolabial fold and left upper lip

Case 2

In February 1998, a 31-year-old Thai man consulted the Oral Diagnosis Section, Faculty of Dentistry, Chiang Mai University, with the complaint of extensive ulceration and scarring with hyperaesthesia of the right side of the face, which had been present for 1 month.

Extraoral examination revealed anesthetic patchy scars with focal hyperesthesia of the right facial skin including the forehead, ear, cheek, angle of the mouth and chin (Figure 2a). A typical neuralgic burning and sensation of itching was also present. His hearing acuity had diminished. All three divisions of the right trigeminal nerve exhibited severe involvement. The attack of HZV began 1 month prior to consultation with severe pain and coalescing vesicles followed by crusting. No antiviral medication was taken during the attack.

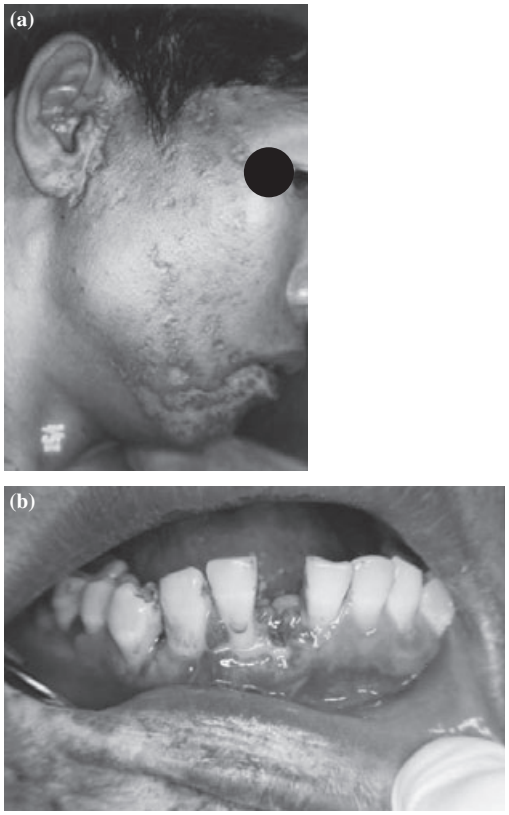

Figure 2 Case 2. (a) Severe scarring of skin of the right side of the face involving all three branches of the trigeminal nerve. (b) Non-healing alveolar socket of the right mandibular central incisor, which exfoliated spontaneously

Intraoral examination revealed generalized gingivitis with moderate calculus formation especially on the lower right side. The lower right central incisor had just exfoliated spontaneously, painlessly leaving a non-healing socket (Figure 2b). The alveolar bone was exposed with severe mobility of teeth involving the apical third of the labial side of the lower left central incisor and lingual side of the lower right first molar (data not shown). Social history revealed that the patient had been infected with HIV 6 years previously. Unfortunately, the patient was lost to follow-up.

Case 3

In June 1995, a 29-year-old Thai man presented to the Oral Diagnosis Section, Faculty of Dentistry, Chiang Mai University, with the complaint of a painful lesion on the right buccal mucosa. Intraoral examination revealed poor oral hygiene with generalized gingivitis. The gingival area of the mandibular right second premolar was red and swollen. There was excessive loosening of the lower right first premolar and second molar. The second premolar had exfoliated spontaneously without pain leaving a non-healing wound with a necrotic alveolar socket. The retained root of the lower right first molar was covered by inflamed gingiva (Figure 3a). The buccal plate from the lower right first premolar to the second molar was exposed and necrotic.

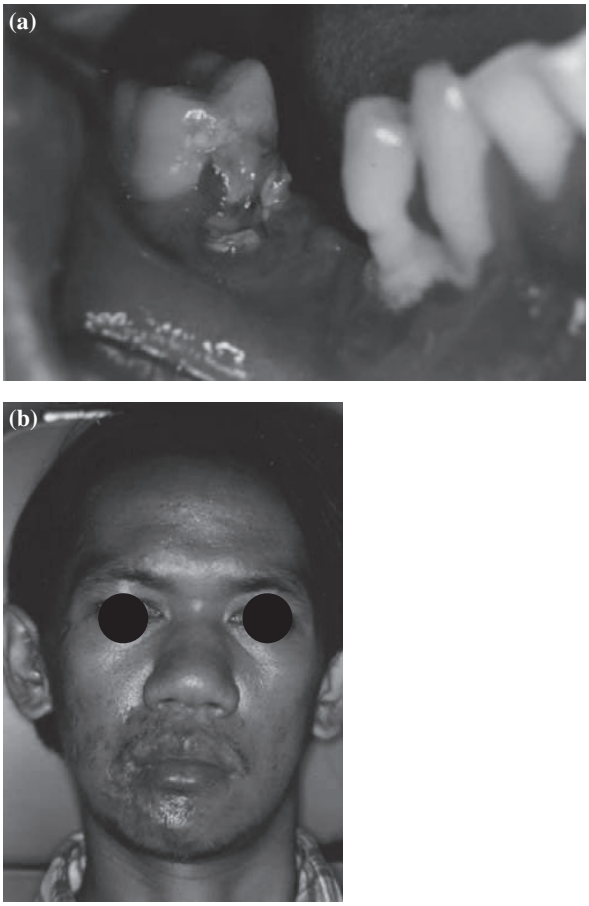

Figure 3 Case 3. (a) Area of the alveolar crest of the right mandible. The second mandibular premolar has exfoliated spontaneously. A retained root of the first mandibular molar is seen. (b) Scar formation, which is most extensive in the area of the mandibular branch of the trigeminal nerve but also involves the areas supplied by the first and second branch of the trigeminal nerve

Extraoral examination demonstrated an anesthetic patchy scar with hyperesthesia limited to the right side of the face including the lower lip, chin, cheek up to the eye, eyebrow, forehead and concha of the right ear and the anterior wall of the external meatus (Figure 3b).

Medical history revealed that 1 month prior to consultation, the patient had a severe vesicular eruption starting from the lower lip and chin and then extending to the face, eye, forehead, and ear. All three divisions of the right trigeminal nerve were affected. Typical neuralgic burning and hyperesthetic sensation on the lower right side of the tongue and oral mucosa associated with pain in the ear and conjunctivitis were experienced. The patient sought treatment of the ocular complication from the ophthalmologist at the Faculty of Medicine, Chiang Mai University. Serology for HIV was positive.

Panoramic radiography showed widening of the periodontal ligament of the lower right first premolar and second molar with moderate bone loss. There was a retained root of the first molar. The outline of the socket of the exfoliated first premolar was clearly visible.

Treatment consisted of oral amoxycillin 2 g day^{-1}, paracetamol 500–1000 mg for controlling infection and pain. Extraction of the lower right first premolar, and first and second molar was planned for the next day. The patient did not return for treatment.

Case 4

A Thai woman aged 31 years came to the Oral Diagnosis Section, Faculty of Dentistry, Chiang Mai University, in May 1995 with the complaint of extreme mobility of her lower left teeth.

Intraoral examination revealed severe loosening of the lower left central incisor, canine, second premolar, first, second and third molars. The lower left lateral incisor and first premolar had exfoliated spontaneously because of total loss of the bony socket 2 days prior to admission leaving raw and necrotic bone walls. The left mandibular alveolar labial and lingual plate from the canine to the mesial wall of the lower third molar were exposed and necrotic. There was no gingival tissue surrounding these teeth (Figure 4a). All lower left teeth showed a positive electrometric pulp response. Lymphadenopathy on the left-hand side was also present. The oral hygiene was poor with generalized gingivitis.

Extraoral examination demonstrated deep postherpetic scarring limited to the left mental region including vermilion border, lower lip, chin, and lower cheek (Figure 4b). The patient gave a history of HZ attack 1 month prior to admission without receiving any treatment. Dental pain and teeth mobility started some days after the initial attack.

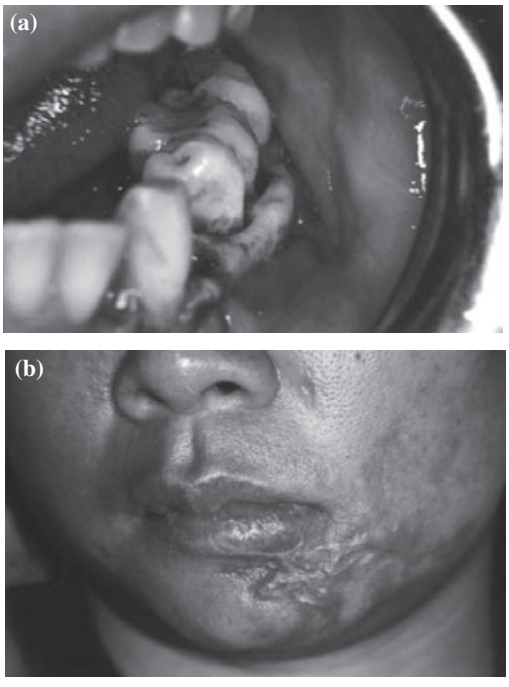

Figure 4 Case 4. (a) The lower left lateral incisor and first premolar are lost spontaneously. The alveolar bone is exposed from the area of the second premolar to the second molar. (b) Severe scarring of the left mental area including the lower lip

Radiographs demonstrated extensive marginal bone loss of the lower left canine, second premolar, first and second molars, while the third molar and central incisor were less severely affected. The outlines of the socket of the exfoliated lower left lateral incisor and first premolar were clearly visible. The contralateral teeth were not affected. A review of the social history revealed that the patient was married and worked as a bar girl. Serology for HIV was positive.

Treatment started with extraction of the lower left canine, second premolar, first and second molars together with the conservative removal of a large piece of necrotic bone. Amoxycillin 2 g day^{-1} and Brufen 800 mg three times a day given orally were prescribed for 1 week. Healing was uneventful.

Discussion

Several complications after HZ of the trigeminal nerve including postherpetic neuralgia, HZ ophthalmicus, aseptic meningitis, facial nerve palsy, myelitis, encephalitis and radiculitis (Friedman-Kien *et al*, 1986) have been described. In this study, the patients, two women and two men of an average age of 30 years, presented with late stage complications of HZ infection. While HZ of the trigeminal nerve is generally considered a complication of advanced age, the present four patients are comparatively young. All of them were HIV-infected and had no previous attack of HZ. In addition, none of the patients had received any antiviral treatment for VZV infection or any type of antiretroviral therapy. Consequently, the postherpetic scarring of facial skin was severe in all four cases with postherpetic neuralgia. In addition, all patients had oral complications with tooth exfoliation or sequestrum formation. Of interest was that the time interval between the onset of HZ and osteonecrosis with tooth exfoliation was about 1 month or even longer. Mintz and Anavi (1992) recorded a range being from immediate appearance to 42 days with a mean of 21.2 days. In the present cases both maxillary and mandibular teeth were involved. In the cases reviewed by Mintz and Anavi (1992) the clinical presentation of osteonecrosis and tooth loss was always unilateral affecting a single quadrant. Maxillary and mandibular teeth were equally affected. As in all similar cases, severe pain in the affected area was observed and reported by all four patients. Healing was uneventful after administration of antibiotics and extraction of involved teeth as well as removal of necrotic bone.

The pathogenesis of osteonecrosis and tooth exfoliation associated with HZ infection is still not clear. Several hypotheses have been discussed including:

1. Local vasculitis caused by direct extension of the neural inflammatory process to adjacent blood vessels (Wright *et al*, 1983).
2. Generalized infection of terminal nerves supplying the periostium and periodontium causing avascular necrosis (Muto *et al*, 1990). Avascular necrosis of the maxilla, however, appears to be unlikely because of its rich vascular supply.

3 Denervation of bone, which also seems unlikely to cause necrosis of bone.
4 Systemic viral infection of odontoblasts causing degenerative tissue changes resulting in pulp necrosis.

Mintz and Anavi (1992) argued that preexisting pulpal or periodontal inflammatory changes may contribute to tooth exfoliation and bone necrosis.

In the present cases, HZ attack was the first serious complication in the course of HIV disease. The patients went undiagnosed as to their HIV status until they consulted the University Hospital because of their HZ infections. Generally, HZ infection in Thai HIV-infected patients is not rare. Sivayathorn *et al* (1995) reported 16.1% of HZ infections among 248 patients with skin lesions. Wananakul and Thisyakorn (1999) observed 4.4% of HZ infections, Supanaranond *et al* (2001) found 10.9% and Wiwanitkit (2004) found 9.17%. In none of these studies detailed information on oral complications such as facial scarring, tooth loss or osteonecrosis of the jaws was given. A study by Panda *et al* (1994) from India revealed that HZ infection in HIV-infected patients emerged as the most specific early HIV-related illness with a positive predictive value of 100%. Patients belonging to the age group of 12–45 years were particularly affected. One study by Jing and Ismail (1999) of 145 cases with mucocutaneous manifestations of HIV infection from Malaysia revealed HZ infection in only 3.4%. Oral manifestations were not reported. Similarly, a study from Brazil (de Gonzaga *et al*, 2002) of 30 patients with HZ showed no specific oral involvement of VZV infection.

Generally, the incidence of HZ infection is associated with underlying malignancy, particularly lymphoproliferative diseases, radiation therapy, systemic corticosteroids, and cytotoxic chemotherapy. In view of the worldwide epidemic of HIV infection and AIDS, the number of cases described with complications of HZ infection including osteonecrosis of alveolar bone and exfoliation of teeth is very small. This may be due to underreporting. Moreover, as with the present four cases, a large number of HIV-infected individuals go unnoticed until they develop serious complications related to their immune status.

Treatment of HZ infection is based on prompt initiation of antiviral therapy and aggressive analgesic treatment (Beutner, 1996). Generally, HZ infection may be treated using antiherpetic drugs including aciclovir, famciclovir, valaciclovir, and sorivudine. These therapies do not only reduce virus replication but also have a pain benefit. In addition to antiviral therapy and analgesics, steroids have been advocated for the treatment of acute HZ. One study (Wood *et al*, 1994) showed no pain benefit for systemic steroids with or without aciclovir.

References

Beutner KR (1996). Treatment of varicella zoster virus infections. *Infect Dis* **9:** 426–428.

Chemitz JE (1976). Herpes zoster in Hodgkin's disease: unusual oral sequelae. *J Dent Child* **43:** 181–186.

Chindia ML (1997). HIV-associated fulminating herpes zoster infection with alveolar necrosis and tooth exfoliation: a case report. *Dent Update* **24:** 126–128.

Colebunders R, Mann JM, Francis H *et al* (1988). Herpes zoster in African patients: a clinical predictor of human deficiency virus infection. *J Infect Dis* **157:** 314–318.

Consolaro A, Oliveira DT (1990). Oral and cutaneous manifestations of herpes zoster: a case report. *Rev Odontoestomatol USP* **4:** 349–352 (in Portuguese).

Cooper JC (1977). Tooth exfoliation and osteonecrosis of the jaw following herpes zoster. *Br Dent J* **143:** 297–300.

Dechaume M, Descrozailles C, Garlopeau F, Robert J (1955). Localized mandibular necrosis during herpes zoster infection. *Rev Stomatol* **56:** 516–521 (in French).

Delaire J, Billet J (1959). New observations of alveolar necrosis during a zona of the mandibular nerve. *Rev Stomatol* **60:** 550–553 (in French).

Delbrouck-Poot F, Reginster JP (1979). Trigeminal herpes zoster and maxillary necrosis. *Dermatologica* **158:** 210–213.

Dielert E (1979). Clinical features of herpes zoster and pathogenesis of cranial nerves involvement. *Schweiz Mschr Zahnheilk* **89:** 135–145 (in German).

Eury J, Gilain L, Peynegre R (1993). Oral manifestations of zona. A case report. *Ann Otolaryngol Chir Cervicofac* **110:** 170–172 (in French).

Friedman-Kien AE, Lafleur FL, Gendler E *et al* (1986). Herpes zoster: a possible early clinical sign for development of acquired immunodeficiency syndrome in high-risk individuals. *J Am Acad Dermatol* **14:** 1023–1028.

Glesby MJ, Moore RD, Chaisson RE (1995). Clinical spectrum of herpes zoster in adults infected with human immunodeficiency virus. *Clin Infect Dis* **21:** 370–375.

de Gonzaga HFS, Jorge MA, de Gonzaga LHS *et al* (2002). Systemic and oral alterations in Brazilian patients with cutaneous herpes zoster. *Braz Dent J* **13:** 49–52.

Goon WWY, Jacobsen PL (1988). Prodromal odontalgia and multiple devitalised teeth caused by a herpes zoster infection of the trigeminal nerve: report of a case. *J Am Dent Assoc* **116:** 500–504.

Gregory WB Jr, Brooks LE, Penich EC (1975). Herpes zoster associated with pulpless teeth. *J Endod* **1:** 32–35.

Hall HD, Jacobs JS, O'Malley JP (1974). Necrosis of maxilla in a patient with herpes zoster. *Oral Surg Oral Med Oral Pathol* **37:** 657–662.

Jing W, Ismail R (1999). Mucocutaneous manifestations of HIV infection: a retrospective analysis of 145 cases in a Chinese population in Malaysia. *Int J Dermatol* **38:** 457–463.

Mckenzie CD, Gobetti JP (1990). Diagnosis and treatment of orofacial herpes zoster: report of a case. *J Am Dent Assoc* **120:** 679–681.

Mendieta C, Miranda J, Brunet LI, Gargallo J, Berini L (2005). Alveolar bone necrosis and tooth exfoliation following herpes zoster infection: a review of the literature and case report. *J Periodontol* **76:** 148–153.

Mintz SM, Anavi Y (1992). Maxillary osteomyelitis and spontaneous tooth exfoliation after herpes zoster. *Oral Surg Oral Med Oral Pathol* **73:** 664–666.

Mostofi R, Marchmont-Robinson H, Freije S (1987). Spontaneous tooth exfoliation and osteonecrosis following a herpes zoster infection of the fifth cranial nerve. *J Oral Maxillofac Surg* **45:** 264–266.

Muto T, Tsuchiya H, Sato K, Kanazawa M (1990). Tooth exfoliation and necrosis of the mandible – a rare complication following trigeminal herpes zoster – report of a case. *J Oral Maxillofac Surg* **48:** 1000–1003.

Panda S, Sarkar S, Mandal BK *et al* (1994). Epidemic of herpes zoster following HIV epidemic in Manipur, India. *Infection* **28:** 167–173.

Peñarrocha M, Bagan JV, Sanchís JM (1992). Herpes zoster and maxillary osteonecrosis. *Rev Actual Odontoestomatol Esp* **418:** 49–55 (in Spanish).

Schwartz O, Kvorning A (1982). Tooth exfoliation, osteonecrosis of the jaw and neuralgia following herpes zoster of the trigeminal nerve. *Int J Oral Surg* **11:** 364–371.

Schwartz O, Pindborg JJ, Svenningsen A (1989). Tooth exfoliation and necrosis of the alveolar bone following trigeminal herpes zoster in HIV-infected patient. *Tandlaegebladet* **93:** 623–627.

Sivayathorn A, Srihra B, Leesanguankul W (1995). Prevalence of skin disease in patients infected with human immunodeficiency virus in Bangkok, Thailand. *Ann Acad Med Singapore* **24:** 528–533.

Srisuwan S (1999). Epidemiology and HIV-associated oral lesions in Chiang Mai province. *CM Dent J* **20:** 45–50.

Supanaranond W, Desakorn V, Sitakalin C *et al* (2001). Cutaneous manifestations in HIV-positive patients. *Southeast Asian J Trop Med Public Health* **32:** 171–176.

Tidwell E, Hutson B, Burkhart N, Gutmann JL, Ellis CD (1999). Herpes zoster of the trigeminal nerve third branch: a case report and review of the literature. *Int Endod J* **32:** 61–66.

Toshitaka M, Tsuchiya H, Sato K, Kanazawa M (1990). Tooth exfoliation and necrosis of the mandible – a rare complication following trigeminal herpes zoster: report of a case. *J Oral Maxillofac Surg* **48:** 1000–1003.

Van de Perre P, Bakkers E. Batungwanayo J *et al* (1988). Herpes zoster in African patients: an early manifestation of HIV infection. *Scand J Infect Dis* **20:** 277–282.

Van Wyk CW, Dreyer WP, Esquep A, Suarez M (1992). Varicella-zoster virus infection. In: Prabhu SR, Wilson DF, Daftary KD, Johnson W, eds. *Oral diseases in the tropics*. Oxford University Press: New York, pp. 285–286.

Verbin RS, Heinman HS, Stiff RH (1968). Localized odontologia occurring during herpes zoster of the maxillary division of the fifth cranial nerve. *Oral Surg Oral Med Oral Pathol* **26:** 441–445.

Vickery IM, Midda M (1976). Dental complications of cytotoxic therapy in Hodgkin's disease. *Br J Oral Surg* **13:** 282–288.

Wananakul S, Thisyakorn U (1999). Mucocutaneous manifestation of HIV infection in 91 children born to HIV-seropositive women. *Pediatr Dermatol* **16:** 359–363.

Wiwanitkit V (2004). Prevalence of dermatological disorders in Thai HIV-infected patients correlated with different CD4 lymphocyte count statuses: a note on 120 cases. *Int Dermatol* **43:** 265–268.

Wood MJ, Johnson RW, McKendrick MW, Taylor J, Mandal BK, Crooks J (1994). A randomized trial of acyclovir for 7 days or 21 days with and without prednisolone for treatment of acute herpes zoster. *N Engl J Med* **330:** 896–900.

Wright We, Davis ML, Geffen DB *et al* (1983). Alveolar bone necrosis and tooth loss: a rare complication associated with herpes zoster infection of the fifth cranial nerve. *Oral Surg Oral Med Oral Pathol* **56:** 39–46.