

ZEBRA HUNT

Zebra XIX. Part 2

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DIFFERENTIAL DIAGNOSIS

In last month's *Journal*, a panoramic radiograph (Fig. 1) taken in October 1996 of a 40-yr-old male was presented along with his signs and symptoms. He had been referred after an episodic toothache that progressed to a constant dull throbbing pain. At the evaluation appointment, tooth #18 and tooth #19 were both sensitive to percussion. In addition, the patient was sensitive to apical palpation in the area of tooth #18. There was no response to cold thermal testing with CO₂ snow or to electric pulp testing for tooth #18 to tooth #24. Periodontal evaluation of the mandibular left quadrant showed generalized moderate to severe probing depths with severe bone loss and class II mobility. Tooth #23 and tooth #24 had extensive gingival recession and class III mobility. Tooth #24 was discolored. There was an intact occlusal alloy in tooth #18 and a composite buildup in tooth #19. There were no radiographic or clinically detectable caries in this quadrant. A panoramic radiograph showed that the mandibular left quadrant had moderate-severe horizontal bone loss of ~40% in comparison with the mandibular right quadrant (Fig. 1). There were apical radiolucencies on tooth #18 to tooth #24. A diagnosis for tooth #18 of a necrotic pulp with acute apical periodontitis was made and root canal therapy initiated. In addition, tooth #19 was treatment planned for endodontic retreatment due to coronal leakage and acute apical periodontitis. Tooth #20 to tooth #24 were diagnosed

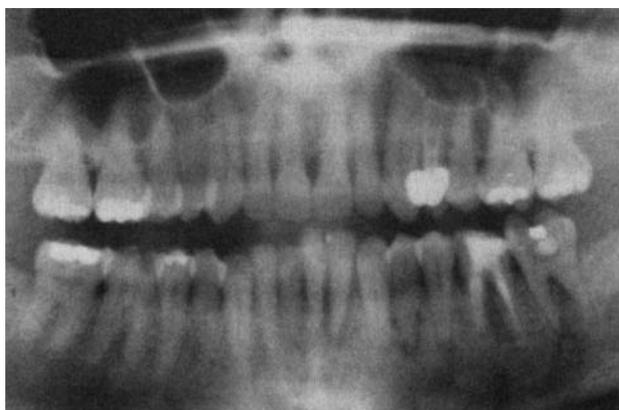


Fig 1. Panoramic radiograph taken before treatment. Tooth #19 had previous endodontic treatment with a composite coronal restoration.

as teeth with pulpal necrosis and chronic apical periodontitis. Tooth #23 and tooth #24 had a questionable periodontal prognosis, and the patient was referred for periodontal evaluation. A periodontal diagnosis was made of generalized mild periodontitis in the maxillary arch and mandibular right quadrant, with localized severe adult periodontitis in the mandibular left quadrant. Tooth #23 and tooth #24 had such poor prognosis that they were recommended for extraction. Tooth #20 to tooth #21 had a guarded prognosis. Except for tooth #30 with a carious pulp exposure, the teeth in the opposite quadrant responded normally. Tooth #31 responded to the electric pulp tester, but had moderate bone loss with a class II furcation defect that was being treated by a periodontist.

The patient gave an interesting history of discomfort on the left side of his face going back 2 yr two years earlier to December 20, 1994 when he first had spontaneous sharp pain. On December 21, 1994, the patient went to his dentist complaining of sharp, nontemperature-stimulated constant pain, and he had a "small cavity filled" in tooth #19. The pain went away with the use of local anesthetic, but returned after the appointment with increased intensity. On December 22, 1994, a red patch appeared on his left cheek. On December 23, he went back to his dentist, and root canal therapy was started on tooth #19. The patient was referred to an endodontist to complete the root canal treatment. At this time the red patch on his cheek was weeping fluid. On December 24, the patient began to experience severe pain and vesicles began to form on his cheek. He also complained of facial swelling in the area of his left eye and his hearing with his left ear was diminished. On December 25, the vesicles had burst and were weeping from the mandibular lip midline extending up to his left ear. There were no vesicles on his upper lip or up to his left eye. He did have some intraoral vesicles. Upon presentation to his dentist, he was referred to an emergency room for treatment. The patient was diagnosed with herpes zoster and was started on acyclovir and pain medication for 10 days. On December 26, the vesicles began to scab over (Fig. 2). It was 3 to 4 wk before the scabs were gone. At that time, the patient complained of a tingling sensation in his mandibular left lip and poor hearing with his left ear. The root canal treatment was completed 3 months later. The patient has residual scarring from the herpes zoster infection from his mandibular left lip midline to his left ear (Fig. 3).

As described previously, the patient presented to our clinic in October 1996 for evaluation and treatment of pain and swelling in the mandibular left quadrant. Root canal treatment was attempted



FIG 2. Photograph of the patient with his initial herpes zoster infection taken with a Polaroid camera at home in December 1994.

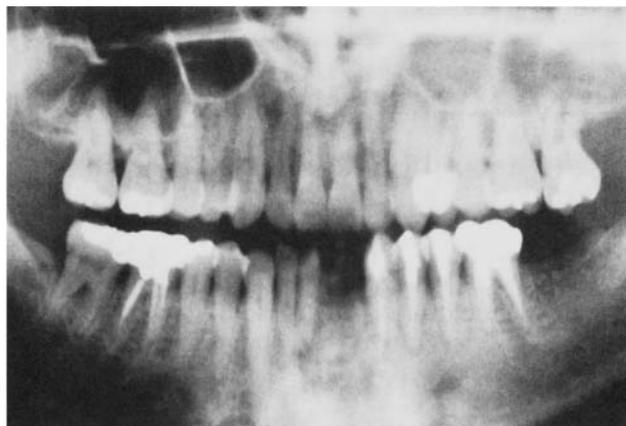


FIG 4. Panoramic radiograph taken at the 10-month posttreatment evaluation appointment. The mandibular left quadrant has moderate-severe horizontal bone loss of ~40% in comparison with the mandibular right quadrant. Tooth #31 has moderate bone loss with a class II furcation defect that is being treated by a periodontist. Tooth #31 responded to the electric pulp tester.

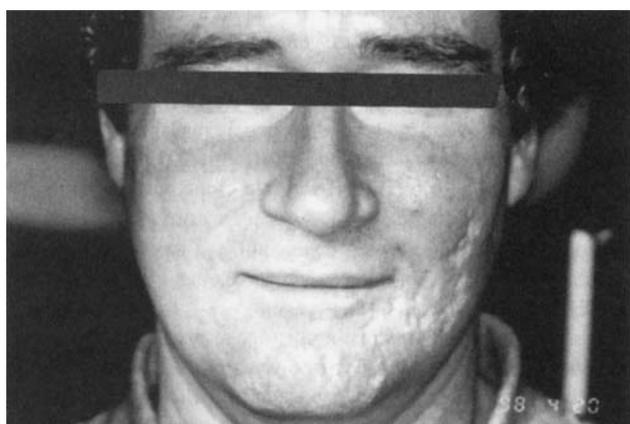


FIG 3. Photograph of patient in April 1998 at follow-up appointment. Notice postherpetic scarring.

on tooth #18, but a vertical root fracture was found and the tooth was extracted. Tooth #19 was retreated endodontically. Root canal therapy was also completed on tooth #20 to tooth #22. All had necrotic pulps. Tooth #20 to tooth #22 were later splinted together for periodontal stabilization. Tooth #23 and tooth #24 were extracted due to severe periodontal disease. A 10-month follow-up appointment showed radiographic evidence of periapical healing of the remaining asymptomatic teeth in his mandibular left quadrant (Fig. 4). Tooth #31 responded to the electric pulp tester, but had moderate bone loss with a class II furcation defect that is being treated by a periodontist. An interim mandibular removable partial denture was used by the patient while the restorative treatment plan was being completed. A panoramic radiograph taken at a follow-up appointment shows healing associated with the endodontically treated teeth in the mandibular left quadrant (Fig. 4).

Last month several possibilities were suggested for your differential diagnosis of the patient's condition associated with his mandibular left quadrant.

1. Trigeminal neuralgia occurs among a group of patients with chronic facial pain with no identifiable dental cause. However, because facial neuralgias mimic pain of dental origin, dentists are often asked to rule out odontogenic origin. Trigeminal neuralgia is usually idiopathic, but patients may have vascular malformations or neoplasms that impinge on the nerve. Trigeminal neuralgia usually affects patients over

age 40. The pain is extreme and the "attack" is often initiated by stimulation of a trigger point. The duration of the pain is usually for <60 s and is followed by a refractory period. The patient in this report did not have a trigger point and his pain was relatively constant (1, 2).

2. Glossopharyngeal neuralgia of the ninth cranial nerve is similar to trigeminal neuralgia except in anatomical location. The pain may affect sensory areas supplied by the pharyngeal and auricular branches of the vagus nerve. Patients often point to the angle of the mandible as the site of greatest pain. Pain is often associated with jaw movement and may be difficult to differentiate from temporomandibular joint dysfunction. It differs from trigeminal neuralgia in that trigger points are not found on the external skin, except within the external auditory meatus. The patient in this report did not have a trigger point and his pain was relatively constant and not the lancinating, paroxysmal pain associated with trigeminal and glossopharyngeal neuralgias (3).
3. Atypical facial pain describes a number of conditions characterized as vague, deep, poorly localized pain in the head and neck region. Atypical facial pain is most common in women in the third through sixth decades of life. The pain tends to be continuous in nature without trigger points and nonanatomical in distribution. Patients are often irritable and fatigued because the pain keeps them awake, and many patients become depressed because of the constancy of the pain. A diagnosis of atypical facial pain is often made by exclusion of other possible causes (1, 2, 4).
4. Neuralgia-inducing cavitation osteonecrosis is also often a diagnosis made by the exclusion of other possible causes. It is believed to be a low-grade, nonsuppurative, radiographically "invisible" osteomyelitis of the jaws. Such lesions have been associated with atypical facial pain or trigeminal neuralgia. It is described as mimicking trigeminal neuralgia to the point of occasionally possessing trigger points (5).
5. Auriculotemporal syndrome develops as a result of injury to the auriculotemporal nerve after parotid abscesses, trauma, mandibular surgery, or parotidectomy. When the parasympathetic nerve fibers attempt to re-establish innervation, they become misdirected and regenerate along the sympathetic

nerve pathways. The syndrome presents with pain, flushing, and warmth in the preauricular and temporal areas. Over time sweating and flushing associated with eating steadily increase and remain constant over time. In the present case, these symptoms did not develop (3).

6. Temporomandibular joint pain and dysfunction arises from a variety of etiological factors and is primarily seen in middle-aged women. Temporomandibular joint dysfunction varies over time but is characterized by pain, altered mandibular function, and joint noises. The pain is usually localized to the periauricular area but may present as a toothache, earache, or headache. However, the pain may be associated more with the surrounding musculature and soft tissue than with the temporomandibular joint itself. Myofascial trigger point pain is characterized by circumscribed regions ("trigger points") within a muscle that produce local or referred pain on palpation. This may be the source of a constant referred pain to the extent that the patient is not aware of the myofascial trigger point (6). Myofascial trigger point pain seems to be unrelated to other temporomandibular joint disorders.
7. Benign or malignant neoplasms may produce referred pain that the patient interprets as tooth pain. Consults with physicians to rule out such possibilities for undiagnosed pain are appropriate (7).
8. Postherpetic pulp necrosis and periodontitis associated with herpes zoster. Herpes zoster (shingles) is a reactivation of a latent varicella-zoster virus in an individual who had varicella (chicken pox) years earlier. In the latent stage, the virus resides in the dorsal root spinal ganglion or the cranial nerve ganglion (8). When reactivation occurs, the virus spreads from the ganglion along sensory nerves to peripheral nerves of the sensory dermatomes. After several days of prodromal neuralgia and paresthesia in the affected dermatome, a well-delineated maculopapular rash develops followed by painful, erythematous cutaneous vesicular eruptions resembling those of varicella. Resolution usually occurs in 2 to 3 wk. Tissue injury, fibrosis, and scarring occur due to the immune response and from the direct lysis of the infected cells (9). Involvement of the various branches of the trigeminal nerve may result in unilateral oral, facial, or ocular lesions, depending on the dermatome affected (10). Osteonecrosis, tooth exfoliation, periodontitis, odontalgias, scarring, irregular short roots, missing teeth, calcified and devitalized pulps, and internal resorption are possible postherpetic dental complications (10, 11). This report presents a case of herpes zoster infection and prodromal odontalgia mimicking a toothache. In a previous episode the infection progressed to permanent facial scarring, paresthesia, periodontitis, and the devitalization of all pulps of teeth in the mandibular left quadrant.

DISCUSSION

This zebra reports the devitalization of the pulps in multiple teeth after trigeminal herpes zoster infection. Only two other cases similar to this have been reported (11, 12). The assumption of herpes zoster as the etiological agent of these sequelae seems reasonable. How the herpes zoster virus affected the dental pulps is unclear. Seltzer and Bender (13) cite studies that show injury to

odontoblasts and degeneration from systemic viral infection. Many other authors have documented trigeminal herpes zoster infection and the development of oral complications such as developmental anomalies, internal root resorption, facial scarring, osteonecrosis, and tooth exfoliation (11). The most important etiological factors for herpes zoster outbreak or reactivation are increased age or a compromised immune system. Emotional stress, especially due to recent events perceived as stressful, seems to be an important factor (13–16). This case was similar to the reports by Goon and Jacobsen (11) and Gregory et al. (12) and because it also occurred in December. Because of the holiday season, the month of December may be considered stressful. The patient in this case also lost his job 2 wk before reactivation of the virus. Because prodromal odontalgia may imitate a toothache (11, 17–20) before the development of the rash and vesicles, a proper pulpal diagnosis must be made to avoid mistreatment.

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