Multiple Apical Radiolucencies and External Cervical Resorption Associated with Varicella Zoster Virus: A Case Report

Kreena Patel, BDS(Hons), MJDF RCS, Elia Schirru, DDS, Sadia Niazi, BDS, MSc, PhD, Philip Mitchell, BDS, MSc, MRD, and Francesco Mannocci, MD, DDS, PhD, FHEA

Abstract

Varicella zoster virus (VZV) is responsible for the primary infection chickenpox. After the initial infection, it remains latent but can reactivate, resulting in shingles (herpes zoster). Previous reports have implicated VZV in the pathogenesis of apical periodontitis, but the involvement of the virus has not been investigated fully. The present case describes a patient who suffered from a severe episode of shingles and subsequently developed periapical radiolucencies of all the teeth in the affected nerve distribution. Molecular and culture techniques showed the presence of VZV DNA in the root canal system in the absence of bacteria. This confirms that VZV can cause localized pulp necrosis and apical periodontitis. The lesions healed after endodontic treatment, implying chemomechanical debridement using sodium hypochlorite irrigation and a calcium hydroxide interim dressing may be effective against the virus. *(J Endod 2016;42:978–983)*

Key Words

Apical periodontitis, external cervical resorption, herpes zoster, shingles, sodium hypochlorite, varicella zoster, virus

From the Department of Endodontology, Kings College Dental Institute at Guy's King's and St Thomas' Hospital, London, United Kingdom.

Address requests for reprints to Prof Francesco Mannocci, Department of Endodontology, Kings College Dental Institute, Floor 25–Tower Wing, Guy's Hospital, Great Maze Pond, London SE1 9RT, UK. E-mail address: francesco.mannocci@kcl.ac.uk

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Apical periodontitis is a localized immune-modulated inflammatory disease caused by an infection of the dental pulp. Numerous studies have revealed the essential role of bacteria in the etiology of the disease (1–4). Microorganisms normally enter the pulp via caries, clinical procedures, or cracks (5, 6). Bacteria have frequently been isolated from teeth with necrotic pulps but clinically intact crowns (4, 7–9). It was also hypothesized that bacteria within the blood circulation could enter and infect necrotic pulps (anachoresis). However, this has been shown to be highly unlikely (10).

Several studies have suggested that other microorganisms are associated with the pathogenesis of apical periodontitis, including fungi and viruses (11–15). Among the latter, human cytomegalovirus (HCMV), Epstein-Barr virus (EBV), and varicella-zoster virus (VZV) have been most commonly isolated (14, 16, 17). These viruses belong to the family of Herpesviridae. A common feature within the family is a single double-stranded DNA molecule enclosed in a viral envelope. Eight human herpes viruses have previously been identified: herpes simplex virus 1 and 2; VZV; EBV; HCMV; and human herpes virus 6, 7, and 8. There appears to be a higher occurrence of HCMV, EBV, and VZV in symptomatic cases and larger lesions (18, 19) and a higher prevalence in human immunodeficiency virus (HIV)-infected patients (20).

VZV is responsible for the primary infection chickenpox. The virions enter from the skin or T-lymphocyte viremia and travel in a retrograde manner to the sensory nerve ganglia (21). After the initial infection, the virus remains latent in the long-lived, nondividing perineural satellite cells of the sensory ganglia (22, 23). In 20% of cases, the virus can reactivate, either spontaneously or as a result of an impaired host immune defense, resulting in shingles (herpes zoster). The virus begins to replicate and reaches the skin by anterograde nerve transport (21). Prodromal symptoms include tingling, itching, and pain in the affected dermatome. This is followed by a maculopapular rash in the region, which evolves into vesicles and pustules.

The trigeminal nerve is affected in only 13% of patients (24). The clinical diagnosis of VZV infection is sufficient most of the time although polymerase chain reaction (PCR) analysis or immunofluorescence is sometimes required. Complications include Bell palsy, ocular involvement, hearing impairment, Ramsay Hunt syndrome, and vasculopathy (25). Reported sequelae of dental relevance include devitalization of teeth (26, 27), postherpetic neuralgia, osteonecrosis, dental resorption (internal and external), and tooth exfoliation (28–30). However, these are case reports or case series, and the involvement of VZV has not been fully investigated.

This case report describes a patient who suffered from a severe episode of trigeminal herpes zoster and subsequently developed periapical radiolucencies of all the teeth in the affected nerve distribution area and external cervical resorption of #27. Molecular and culture techniques showed the presence of VZV in the root canal systems in the absence of bacteria.

Examination

A 52-year-old Asian man was referred to Guy's Dental Hospital, London, UK, in August 2014. The patient presented with periapical radiolucencies associated with #25, #26, #27, #28, #29, and #30. There was no history of trauma, metabolic bone disease, or orthodontic treatment.

The patient was medically fit and healthy at the initial consultation. In 1987, he suffered from an episode of herpes zoster affecting the right trigeminal nerve branch V3 (lower right quadrant). He was hospitalized for 10 days and experienced severe pain and vesicles localized to this distribution. He subsequently suffered from postherpetic neuralgia and reported mild anesthesia in this area.

Clinical examination revealed a minimally restored and well-maintained dentition (Fig. 1). Tooth #30 had been root treated by his dentist in 2004. Teeth #25 to 29 were sound, unrestored, and asymptomatic. The teeth in the lower right quadrant were not tender to percussion or palpation and had no mobility or pathological probing associated. None of the teeth in this quadrant responded to pulp vitality testing using electronic pulp testing and cold testing. Radiographic examination confirmed periapical radiolucencies associated with #25 to 30. Tooth #27 also had external cervical resorption (ECR) (Fig. 2*A–I*). A cone-beam computed tomographic (CBCT) scan confirmed that the resorption on #27 communicated with the root canal space and extended circumferentially and apically down the root surface (Fig. 2).

Tooth #31 had been restored with a small occlusal composite restoration. Although the tooth did not have a periapical radiolucency present on the initial radiographs or CBCT scan, it became symptomatic and developed a large radiolucency in the following few months (Fig. 2).

The following provisional diagnoses were made:

1. Pulp necrosis and asymptomatic apical periodontitis were reached for #25, #26, #28, and #29.
2. Pulp necrosis and symptomatic apical periodontitis were reached for #31.
3. Previously treated and asymptomatic apical periodontitis was reached for #30.
4. Pulp necrosis, asymptomatic apical periodontitis, and external cervical resorption were reached for #27.

The patient had routine blood tests on November 2010, which revealed a slight leukopenia of 3.3×10^9 cells/L (normal range, $4.0–11 \times 10^9$) with normal cellular morphology. Routine blood tests run in May and October 2015 revealed the same result. Particularly, lymphopenia 1.1×10^9 cells/L (normal range, $1.2–3.3 \times 10^9$) with a low subset of CD4 257/μL (normal range, 300×1400/μL), CD3 575/μL (normal range, 700–2000/μL), and natural killer lymphocytes 66/μL (normal range, 90–600/μL). An HIV test gave a negative result, and the immunoglobulin G for VZV was positive. The persistent moderate chronic leukopenia indicates that these levels are likely to be normal for the patient.

Treatment

Nonsurgical endodontic treatment was performed on teeth #25, 26, 28, 29, 30, and 31. The teeth were all necrotic and when accessed under microscope magnification had an unusual odorless, black, pigmented substance in the pulp chamber and canals.

Chemomechanical debridement of the canals was completed using a combination of hand and rotary instruments while irrigating with 1% sodium hypochlorite and 17% EDTA. The endodontic treatment was performed over 2 visits using an interim calcium hydroxide dressing placed using a spiral filler. The teeth were obturated with gutta-percha and a zinc oxide eugenol–based sealer using a warm vertical condensation technique.

The extension and position of the resorptive lesion on #27 was not amenable to treatment and the tooth was extracted.

Sampling

During the endodontic treatment of #31, samples of the pulp chamber and canal contents were taken. All sampling was undertaken under strict aseptic conditions. The tooth was isolated using a rubber dam, and the field was cleaned with 30% (vol/vol) hydrogen peroxide and decontaminated with 2% sodium hypochlorite followed by sodium thiosulfate. After decontamination, the isolated tooth and surrounding dam were swabbed to check for contamination. The access cavity was initially prepared with a sterile round bur without water cooling and using sterile saline. This bur was replaced, and only saline irrigation was used when approaching the pulp chamber. On gaining access to the pulp, sterile files and paper points were inserted into the pulp chamber and contents obtained for testing. This was repeated with the distal root canal up to the apical foramina. Surgical sterile gloves were used and replaced regularly throughout the procedure. For bacterial sampling, all the tissues removed were transferred into 1 mL Tris-EDTA buffer (1.0 mol/L Tris-HCl containing 0.1 mol/L EDTA, pH = 8.0 prepared in ultra high quality water) (31). For viral sampling, they were placed into universal transport medium. After the extraction of #27, the root canal was sampled using the same technique. Both samples from teeth

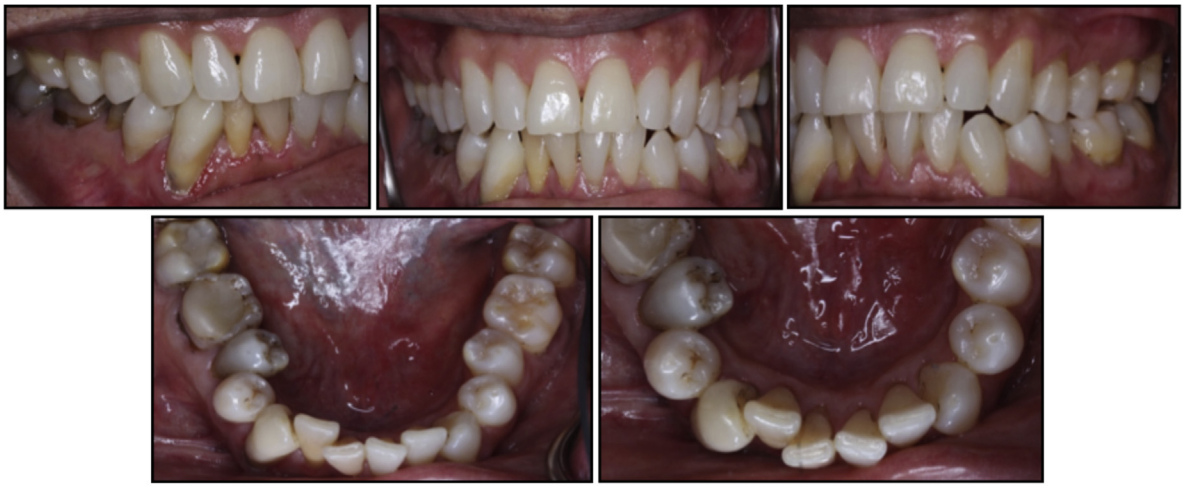

Figure 1. Preoperative photographs. Teeth #25 to 29 were unrestored; tooth #30 was root treated in 2004 by his dentist and restored with a large composite restoration; and tooth #31 had a small occlusal composite restoration present.