Extensive dental caries in unerupted permanent teeth of a disabled child with phenytoin-induced gingival overgrowth

Jumpei Murakami DDS, PhD1, Ichijiro Morisaki DDS, PhD2, Tomoko T. Tanaka DDS3, Shigehisa Akiyama DDS, PhD4, Atsuo Amano DDS, PhD5 and Clive S. Friedman6

1Assistant Professor; 2Professor and Chair; 3Clinical Instructor; 4Associate Professor Division of Special Care Dentistry, Osaka University Dental Hospital. 5Professor, Department of Oral Frontier Biology, Osaka University Graduate School of Dentistry, 1-8 Yamadaoka, Suita-Osaka 565-0871 Japan. 6Clinical Associate Professor, University of Western Ontario, 389 Hyde Park Road, London, Ontario, Canada

Abstract

The permanent teeth of a 9-year-old female with severe motor dysfunctions and intellectual disabilities had not yet fully erupted into the oral cavity due to being covered with overgrown gingival tissue, as a side effect of taking phenytoin. Dental caries was diagnosed in those teeth by a radiographic examination. These findings indicate that early dental examinations and preventive measures are useful for patients taking phenytoin.

Key words: Unerupted teeth, caries, gingival overgrowth

Introduction

About half of the patients taking phenytoin (PHT) are thought to develop gingival overgrowth as one of its side effects (Dahlof et al., 1991). PHT-induced gingival overgrowth generally occurs first in the anterior interdental papillae, as early as one month after the initiation of PHT therapy. This may cause subsequent deterioration in oral functions such as breathing, speech, mastication, swallowing, tooth eruption, and aesthetics (Meraw and Sheridan, 1998). However, clinical manifestations vary in severity depending on the dose and duration of PHT therapy, as well as the gender and age of the patient. The effects of PHT therapy on dental caries susceptibility in epileptic patients has not been well elucidated, although it was reported that PHT treatment increased plaque deposition and dental caries in rats infected with Streptococcus sobrinus (Morisaki et al., 1992). It has also been shown that PHT reduced dentine apposition and increased the rate of caries progression in rats (Larmas and Tjäderhane 1992).

We report the oral findings and dental treatment of a 9-year-old female, who had delayed eruption and subgingival caries in the permanent first molars and incisors, associated with PHT-induced gingival overgrowth.

Case Report

A 9-year-old female with cerebral palsy, severe motor dysfunction, intellectual disability and epilepsy was brought to the Division of Special Care Dentistry at Osaka University Dental Hospital for a dental examination, with a complaint of gingival swelling.

The patient was a first born child, delivered after 39 weeks of gestation without complications. However, she was diagnosed with hypoxia three days later. The first epileptic seizure occurred at three months of age, after which she received a variety of anti-epileptic medications, including most recently, PHT, at a dose of 170mg per day. At the first visit to our clinic, the patient weighed 20kg (1.6 SD below average for Japanese girls of the same age, as provided by the Ministry of Education, Culture, Sports, Science and Technology of Japan) and was 110cm in height (3.6 SD below average). She was unable to speak or communicate with others, and spent most of the time in bed with full support for activities of daily living.

The initial visit to our clinic was the first dental examination, including radiography, that the patient had received at a dental hospital. Previously, her dental condition had been checked annually by a general dentist at a special school, and her parents thought that she had no primary or permanent teeth, until a few permanent teeth erupted.
There were no obvious oral symptoms, such as pain, bleeding, or halitosis, reported, however, generalised and severe gingival overgrowth was noted in both the maxilla and mandible (Figure 1A). The patient had an anterior open bite. The gingivae were pink in colour, and had a firm and thick texture without acute inflammatory signs. The gingivae covering the maxillary central and lateral incisors was swollen with trapped fluid, and the tissue had the appearance of a balloon. The incisal edges of teeth numbers 12, 23, 31, and 41 were visible, and a few remaining roots of primary teeth, destroyed because of severe dental caries, were observed in the oral cavity (Figures 1B, C). A panoramic dental radiograph revealed that all permanent teeth were present and growing in a normal fashion, however, tooth eruption was considerably delayed as compared to an average dentition at the same age. Carious lesions were noted on the occlusal surfaces of all first permanent molars and the incisal edges of the mandibular central incisors (Figure 2).

Although the patient had been receiving anti-epileptic medication, a convulsion usually occurred once a day, which lasted for about two minutes. Recent medication consisted of phenytoin (170mg), clonazepam (1.3mg), sodium valproate (750mg), and nitrazepam (6mg) as anti-epileptics, along with dantrolene sodium, famotidine, sodium picosulfate, levocarnitine chloride, cyproheptadine HCL, and melanin. All nutrients were given orally three times a day in paste form and drugs in powder form were given after dissolving in water.

The parents reported that they brushed the child’s mouth with a toothbrush to remove retentive foods after each meal, three times a day, though they had no idea that teeth were present and erupting below the gingivae.

The central and lateral incisors were facilitated to erupt by excising the overgrown gingival tissues with an Er: YAG laser (120mJ, 10 pulses per second; Erwin®, Morita Co., Japan) under infiltration anaesthesia (Figure 3B). One month after the first excision, the same laser treatment was performed twice, at one month intervals, for the other incisors, canines, and premolars to complete tooth exposure (Figure 4). Dental caries in the mandibular central incisors was restored with adhesive composite resin (Figure 4). One of the severely decayed permanent molars (16) was extracted, while the less severely decayed molars (26, 36, 46) received temporary polycarboxylate cement restorations, followed by composite restoration. This resulted in a significant improvement, aesthetically and functionally, as shown in Figure 4.

Discussion

For patients with PHT-induced gingival overgrowth, dis-continuation of PHT is a simple means to stop or reduce the undesirable side effect (Dahllof et al., 1991). However, in many instances, as in this case, it is not always possible to change drugs primarily for the reason of dental side effects. PHT can be a safe and effective option for controlling epileptic seizures, and is widely used.
Figure 2: Panoramic dental radiograph taken at the first visit (9 years 8 months). Tooth eruption was delayed, however, all permanent teeth were seen. Caries lesions were detected in all four first permanent molars (16, 26, 36, 46) and primary teeth (55, 64, 65, 74, 75, and 85; arrows).

Figure 3: Intraoral view after undergoing a gingivectomy in the anterior region.
A. Numbers 12, 11, 21, and 22.
B. Numbers 32, 31, 41, and 42. Numbers 31 and 41 received adhesive composite resin restorations.

Figure 4: Intraoral view at four weeks after the surgical and restorative care.
There were three risk factors for gingival overgrowth in this patient: insufficient oral hygiene, long term treatment with PHT starting at a very young age, and multiple anticonvulsant drug therapy (Seymour et al., 2000). It is interesting to note that the present case had extensive dental caries in apparently unerupted permanent teeth, beneath the overgrown gingiva. Little information is available regarding drug-induced gingival overgrowth and dental caries in humans, however, our findings suggest that drug-induced gingival overgrowth could be a risk factor for dental caries in epileptic patients. Further, Rajavaara et al. (2003) speculated that factors related to epilepsy and epilepsy medication might increase the risk of caries. It is likely that a small amount of communication existed between the oral cavity and unerupted teeth in the present patient. Atlay and Cengiz (2002) reported that minimal communication between unerupted teeth and the oral cavity can cause decay in infraoccluded primary teeth. Most foods and medications, which may contain sugar, are given in paste form to patients with similar conditions, and only limited brushing and oral irrigation can be provided. Therefore, it is likely that food and/or medications were in prolonged contact with the tooth structures and this was a causative factor for the decay seen in this patient.

In the present case, an Er:YAG laser was successfully utilised. This device was developed for cavity preparation in dental hard tissue and has also been utilised for gingivectomy procedures (Kesler et al., 2000). Few reports have shown the efficacy of an Er:YAG laser in Special Care Dentistry, though we have found it be an alternative and less invasive surgical tool for use in patients with severe mental and/or physical disabilities.

Conclusions

The present findings strongly suggest that the following are advisable for patients taking PTH:

- Early professional intervention, including oral hygiene guidance given to inform patients, parents, or caregivers
- Careful radiographic and macroscopic dental examinations
- Treatments to prevent gingival overgrowth and its complications, such as dental caries in apparently unerupted teeth or oral dysfunction.

References


Address for correspondence:
Jumpei Murakami
Assistant Professor
Division of Special Care Dentistry
Osaka University Dental Hospital
1-8 Yamadaoka, Suita-Osaka 565-0871
Japan
Email: jumpei@dent.osaka-u.ac.jp