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Dilaceration of the mandibular permanent incisor teeth: two case reports

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Abstract

This report involves two cases of extreme dilaceration of the permanent mandibular incisor teeth. A history of trauma sustained to the primary dentition was elicited in each case. Although a relatively rare condition, several reports have been made regarding dilacerated maxillary incisor teeth. Very few case reports could be found involving dilacerated mandibular teeth.

Introduction

The term "dilaceration" is applied to teeth which have the long axis of either the whole or part of the root formed at an angle to the crown. These deformities to the permanent tooth may be the result of trauma to the primary tooth during the development of its permanent successor.[1] Andreasen[2] conducted epidemiologic studies to investigate the effect of traumatic injuries to primary teeth on their permanent successors. His study consisted of 207 permanent teeth with developmental disturbances secondary to injuries in the primary dentition. He reported a prevalence of dilaceration injuries of 25 percent (51/207), with the vast majority of cases involving maxillary central incisors. The trauma usually occurred at two years of age with a range from less than one year to five years. The acute deviation of the crown portion was usually lingual among maxillary incisors while the opposite deviation was found in the mandible. Exceptions to this rule were noted when the trauma occurred with less than one-fourth of the crown formed at the time of injury. Andreasen found that half of the dilacerated permanent teeth were impacted. Intrusive luxation and exarticulation of the primary teeth constituted the main form of traumatic injury sustained in the primary dentition which resulted in dilaceration of the succedaneous tooth.

A study by Stewart[3] addressed the question of the percentage of dilacerated teeth which were in fact caused by trauma to their primary predecessors. He found no immediate discernible cause for the dilaceration in 72.5 percent (29/40) of the unerupted dilacerated maxillary incisor teeth. It is postulated that in a majority of these cases previous traumatic episodes are not the causative factor for the dilaceration: more likely, it is of a developmental nature in which the malformation is brought about through ectopic development of the tooth germ.

A few case reports (Kolokithas[4] and Edmondson[1]) have recently been published dealing with the dilacerated incisor tooth. These have all been maxillary incisor teeth and were unerupted upon presentation, in contrast to the present case reports.

Case Report 1

A.S., a well-developed, well-nourished six-year-old caucasian male was referred to our clinic by a local general practitioner for treatment of "abnormal permanent mandibular incisors." His parents reported a traumatic episode when the patient was nine months old, in which he fell down a full flight of stairs avulsing his maxillary primary central incisors. They could remember no damage to other oral structures as a result of this incident and reported no other traumatic episodes to date.

His past medical history was unremarkable. All teeth and supporting structures were asymptomatic at presentation. An intraoral examination revealed an Angle's class I age-appropriate mixed dentition without crowding or other occlusal disharmonies. The intraoral soft tissues were without swelling or redness with the exception of inflammation to the marginal gingiva around the mandibular right central incisor. A pocket depth of five millimeters was probed and purulent exudate could be easily expressed. A draining fistulus tract was present on the alveolar mucosa adjacent to this same tooth.

The mandibular permanent left lateral incisor and the mandibular permanent right central incisor were

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Figure 2. Microprocessor Operated Recording Equipment (MORE).

treatment phase increases. Third, that a greater frequency of fear/distress behaviors would be observed in the second appointment due to the age and relative lack of dental experience of the children.

The behavior of child, dentist, and assistant is coded independently in real time from the videotape. The equipment used for the coding is called MORE, i.e., Microprocessor Operated Recording Equipment (Figure 2). This system involves a microprocessor unit with a small keyboard, a recorder, and a computer interface device. When the coder identifies an event, he presses a series of keys. Events are timed in seconds from the first keystroke of an event to the first keystroke of the next event. Following the coding, data are transferred to audio tape for storage and transmission to the host computer. Such systems, first developed and tested by Sackett and colleagues in the early 1970s, have proved invaluable in sequential analyses of observational data.[4]

Interjudge agreement was established by directly comparing coding results of different researchers on the same child. Pearson r for major dimensions ranged from .85 to .94. Percent agreement averaged .89. Though an attempt was made to completely operationalize each behavior, it was found that the context of the behavior often must be considered when coding. For example, the dentist might say, "Sit back in the chair now." The context of the situation and voice tone will determine if this will be coded as dentist direction or use of coercion.

To this point in the project, seventy-two videotapes have been analyzed, representing two sequential appointments for thirty-six children.

Results and Discussion

In order to test the first hypothesis, the restorative appointment was divided into six distinct phases of treatment: chair placement, oral examination, injection, rubber dam, cavity preparation, and placement of filling materials. Results of analyses of variance (Table 1) indicate that the lowest percent frequency and duration of fear/distress-related child behaviors occurred during the oral examination, followed by the visual examination. The next significant increment in

Table 1. Analyses of variance for child behaviors between treatment phases and appointments.

Child Variable	% frequency % duration	Difference Between Phases F	Difference Between Phases p	Differences Between Appointments F	Differences Between Appointments p
Fear/Distress - related Behaviors (Composite Scale)	%f	2.88	.037	2.308	NS
	%d	12.272	.001	11.387	.001
Minor Child Movement	%f	5.353	.001	2.358	NS
	%d	11.111	.001	2.655	NS
Major Child Movement	%f	1.549	NS	.008	NS
	%d	1.923	NS	3.695	NS
Statement of Hurt	%f	6.108	.001	.230	NS
	%d	2.641	.051	2.710	NS
Verbal Protest	%f	1.180	NS	2.135	NS
	%d	.642	NS	4.580	.033
Whimpering	%f	2.498	NS	.413	NS
	%d	1.533	NS	1.942	NS
Loud Crying, Screaming	%f	5.065	.002	1.744	NS
	%d	2.994	.034	1.947	NS
Physical Discomfort	%f	3.927	.001	5.559	.018
	%d	5.975	.001	9.109	.003

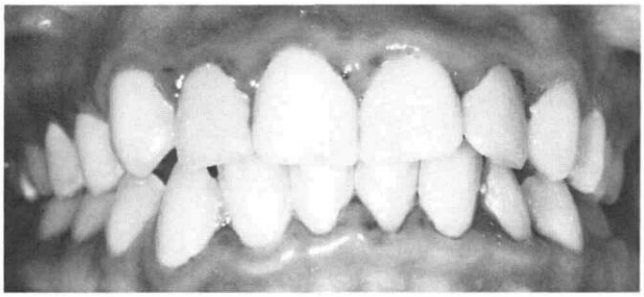

Figure 1. Initial intraoral view demonstrating gingivitis and spontaneous gingival hemorrhage.

March, 1979, with complaints of malaise and prolonged epistaxis. The patient denied any episodes of jaundice or hematuria. The family history was negative for sickle cell anemia or Fanconi's anemia. The patient had been transfused with 5 units of random donor platelets and packed cells prior to arrival at Riley Hospital.

Physical examination revealed a well-developed black female in no acute distress. Heart rate was 80, respiratory rate 18, weight 38.1 Kg. The examination was unremarkable with the exception of mucosal and petechial hemorrhage. Specifically, there was no hepatosplenomegaly or lymphadenopathy. No abnormality of the digits was noted. The patient was admitted to the hospital for evaluation. Admission laboratory data were as follows: Hemoglobin 7.6 gr.%, hematocrit 24%, white cell count 2,400/cu mm, platelets 10,000/cu mm, PT 11.5 seconds, PTT 23.0 seconds, SGOT 20, SPGT 19, and Alkaline Phosphatase 91. Antinuclear antibody was negative.

The patient was transfused with packed red cells, whole blood and five units of random donor platelets shortly after admission. A repeat hemoglobin was 9.1 gr.%. A bone aspiration and biopsy were performed. These demonstrated a marked decrease in precursors of all cell lines. Prominent mast cells and plasma cells were noted. There were no megaloblastic changes noted. Subsequent normal B_{12} and folate levels were documented. A sucrose hemolysis test proved negative. In the absence of historical evidence for marrow toxins and the fact that the physical findings were not consistent with familial aplastic anemia, the diagnosis of idiopathic aplastic anemia was made. A histocompatible donor was unavailable for marrow transplantation. The patient was discharged from the hospital to her parents five days after admission. Subsequent attempts at treatment with high dose corticosteroids and antithymocyte globulin were unsuccessful.

At the request of the hematology service, the patient was seen in the dental clinic of Riley Hospital on October 18, 1979 for evaluation of gingival hemorrhage and oral lesions. Her oral temperature was 38.9° C, hematocrit 20%, and a platelet count of 1,000 cu mm.

Oral and Radiographic Examination

Oral examination demonstrated generalized gingivitis with spontaneous gingival hemorrhage (Figure 1). Periodontal examination demonstrated no pocket formation greater than three millimeters. Multiple round, raised lesions approximately 3 mm in diameter were present on the anterior half of the tongue (Figure 2). Bilateral lesions approximately 1 cm in diameter were distal to the second permanent molars on the buccal mucosa (Figure 3). There was no history of oral trauma. Radiographic examination, including panorex, bite-wing and two maxillary anterior periapical radiographs were negative for pathology.

The patient reported severe pain during toothbrushing and had not been practicing regular oral hygiene in the past. At this time the patient was readmitted to the hospital for dental evaluation and treatment of the oral condition.

Initial Dental Treatment

The patient received two transfusions of 10 units each of platelets. The platelets were matched by major blood groups only. This brought her platelet

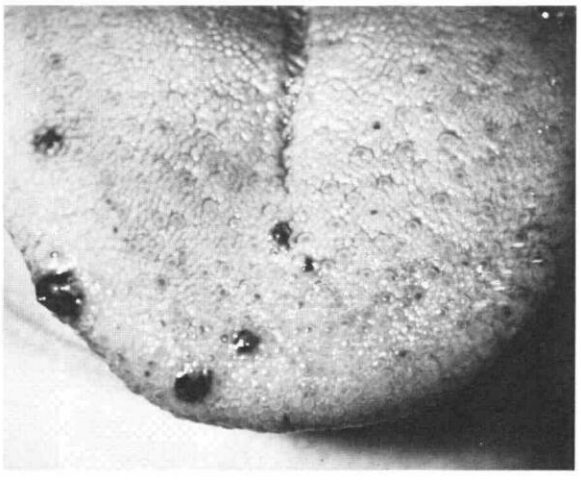

Figure 2. Multiple round, raised lesions on the anterior half of the tongue (left).

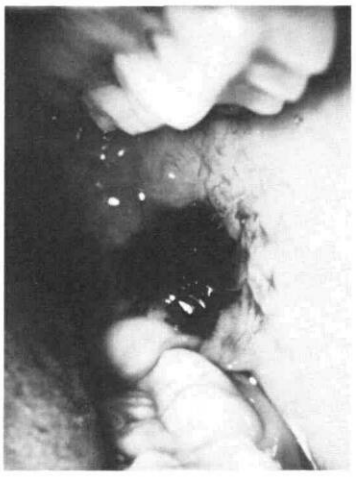

Figure 3. Intraoral view of lesion on patient's left buccal mucosa (right).