CASE REPORT

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Degenerative alterations of the cementum–periodontal ligament complex and early tooth loss in a young patient with periodontal disease

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Abstract

Premature exfoliation of primary or permanent teeth in children or adolescents is extremely rare and it can be a manifestation of an underlying systemic disease. This study aims to present the histological aspects associated with early tooth loss in a case of periodontal disease developed without local inflammation and with minimal periodontal pockets and attachment loss. The maxillary left second premolar was extracted together with a gingival collar attached to the root surface. The histological analysis recorded the resorption of the cementum in multiple areas of the entire root surface with the connective tissue of the desmodontium invading the lacunae defects. The connective tissue rich in cells occupied the periodontal ligamentar space and the resorptive areas. No inflammation was obvious in the periodontal ligament connective tissue. This report may warn clinicians about the possibility of the association of cemental abnormalities with early tooth loss.

Keywords: cementum, root resorption, periodontal disease.

Introduction

Periodontal disease expression involves complex interactions of the subgingival biofilm with the host immune-inflammatory response that subsequently alter periodontal tissue homeostasis [1] leading to local inflammation and loss of the supporting tissues. It is assumed that periodontitis debuts in genetically or environmentally predisposed individuals, who are infected with virulent infectious agents and reveal persistent gingival inflammation and distinct immune response [2].

Periodontitis occurs in otherwise healthy individuals and initial mechanical treatment addressed to microbial etiologic factor succeeds in stopping the evolution of the disease. In these circumstances, in well-maintained patients, the tooth loss is a rare event in the evolution of periodontal disease [3–5].

Premature exfoliation of primary or permanent teeth in children or adolescents is extremely rare and it can be a manifestation of an underlying systemic disease [6]. Systemic diseases associated with periodontitis with or without premature exfoliation of primary teeth may include Papillon–Lefèvre syndrome, hypophosphatasia, leukocyte adhesion deficiency, neutropenia, Chédiak–Higashi syndrome and Langerhans' cell histiocytosis [7]. More recently, an association between hypophosphatemic rickets and aggressive periodontitis has been reported [8]. The aggressive evolution of the disease in systemic circumstances may be due to alterations in the immune system that interfere with the resistance to bacterial infections [9] or to disturbances of cementum formation resulting in a weakened periodontal ligament attachment to bacterial aggression [10]. Irrespective of the underlying mechanisms driving to bacterial-induced destruction in periodontitis associated with systemic diseases, the affected patients exhibit the classical signs associated with periodontitis. The clinical picture of periodontal disease evolving without local inflammation and attachment and bone loss has been seldom reported in the literature [11].

This study aims to present the histological aspects associated with early tooth loss in a case of periodontal disease developed without clinical inflammatory signs and with minimal periodontal pockets and attachment loss.

Patient and Methods

A 17-year-old adolescent addressed to the Periodontology Department of "Iuliu Haţieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania, because

excessive mobility, along with extreme pain with mastication on the maxillary left second premolar.

History

The patient's medical history revealed a previous diagnostic of a mosaic form of Turner syndrome, which was later on invalidated by a karyotype analysis. Her dental history revealed unremarkable events associated with her primary dentition. However, the mobility of permanent teeth was noticed immediately after tooth eruption. The patient ate a well-balanced diet. The family history revealed one aunt with the same dental-periodontal disease pattern.

Extra-oral examination

Extra-oral examination revealed spoon-shape nails (koilonychia) on both fingers and toes with no evidence of hyperkeratosis of the hands and feet. The patient did not have any other extra-oral clinical abnormalities besides her dysplastic nails and no other symptoms that would suggest neutrophil/leukocyte dysfunction.

Intra-oral examination

Full-mouth periodontal examination recorded the following parameters: probing depth, gingival recession, clinical attachment level, tooth mobility and furcation involvement. Grade 3 tooth mobility [12] was noted for all the teeth, in the presence of normal probing depth and attachment level in almost all probed sites. The maxillary left second premolar exhibited grade 4 mobility and was extremely painful. The periodontal parameters recorded for the tooth 2.5 are revealed by the Table 1.

Table 1 – ***The periodontal chart for the maxillary left second premolar***

Parameter	MB	B	DB	DL	L	ML	Mob
PD	3	3	5	5	3	3	4
Rec	0	0	0	0	0	0	
Att	0	1	3	3	0	0	

The recorded values are probing depth (PD), gingival recession (Rec), attachment level (Att) and mobility (Mob), respectively. PD, Rec and Att were recorded at six sites per tooth: mesio-buccal (MB), buccal (B), disto-buccal (DB), mesio-lingual (ML), lingual (L), and disto-lingual (DL).

Neither calculus nor bleeding on probing was observed. The plaque index [13] was 28%.

The radiography revealed approximately 3 mm bone loss on the distal aspect of the tooth 2.5, a widened periodontal space and the loss of lamina dura (Figure 1).

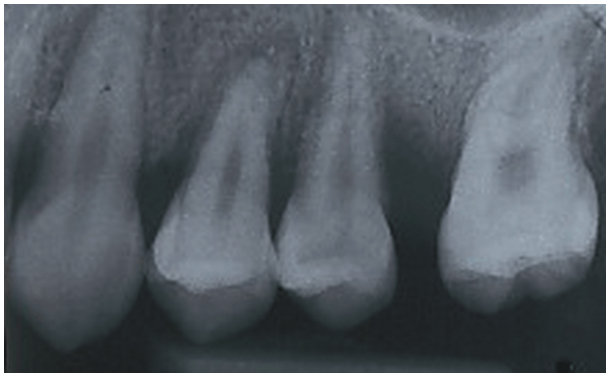

Figure 1 – ***The radiography of the tooth 2.5 shows distal bone loss, and a widened periodontal space.***

Excepting a profound carious lesion at the level of the right mandibular second molar, the other teeth were carious-free.

Treatment

The patient asked only that her painful tooth be removed and the additional investigations were postponed. She agreed that the histological analysis of the extracted tooth be performed.

The extraction of the maxillary left second premolar was realized under local anesthesia using articaine hydrochloride (4%) plus adrenaline (1:100 000) (Septanest®, Septodont, France). Before extraction, using a #15 surgical blade (Swann–Morton, Sheffield, England), a perpendicular incision to the gingival surface was realized apically to the junctional epithelium in order to detach the attached soft tissue together with the extracted tooth. The extraction was realized using a forceps. The alveolus healed uneventfully.

Histological analysis

The tooth underwent routine histological procedures. Immediately after extraction, the tooth was placed in 10% neutral buffered formalin and fixed for 48 hours. The sample was decalcified in 10% nitric acid for 12 days and than dehydrated in progressive concentrations of ethanol (80% alcohol two times for 30 minutes; 95% alcohol two times for 30 minutes; and 100% alcohol two times for 30 minutes). The sample was embedded in paraffin. Serial sections, 5 μm thick, in bucco-lingual direction were prepared using a Microtom Gm BIT HN 310 (Germany) and were stained with Hematoxylin and Eosin. The sections were fixed with DPX medium.

The sections were examined by one author (CMM) by light microscopy (Leica DM 750, Germany) and were photographed with Leica ICC 50 HD (Germany) camera connected to the microscope.

Results

The partial resorption of the cementum in multiple areas of the entire root surface with the connective tissue of the desmodontium invading the lacunae defects were recorded (Figure 2).

The resorption of the cementum extended into the dentine (Figure 3) suggesting the aspect of resorbed lacunae onto the root surface. New synthesized cementum lining resorptive areas could not be observed.

The connective tissue rich in cells suggesting an increased secretory activity of the periodontal fibers occupied the periodontal ligamentar space and the resorptive areas. Collagen fibers did not properly insert into cemental resorptive areas (Figure 4). Ligament fibers run parallel with cemental surface (Figure 4).

An intense inflammatory infiltrate was noticed in the gingival lamina propria (Figure 5).

There were found disorganized dentinal structural areas with an irregular distribution of the dentinal tubules alternating with dentin with normal structure (Figure 6). Disorganized dentinal areas resembled tertiary dentin and had no obvious relationship with cemental resorptions.

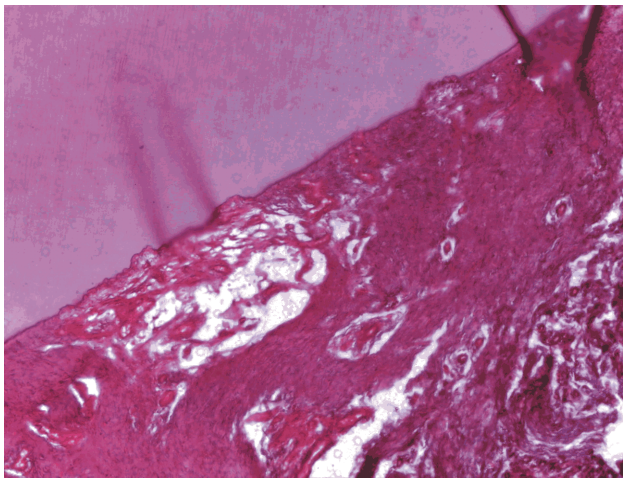

Figure 2 – ***Histological analysis of the tooth–periodontal complex: multiple areas of root resorption were recorded (ob. 10×).***

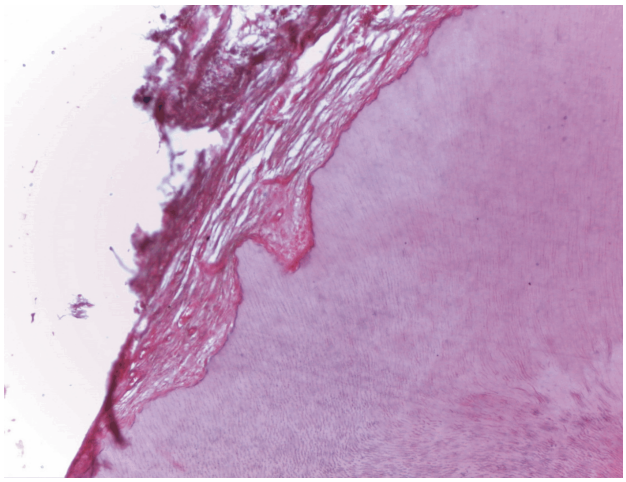

Figure 3 – ***Histological analysis of the tooth–periodontal complex: the resorption of the cementum extended into the dentine with no signs of new synthesized cementum lining resorptive areas (ob. 10×).***

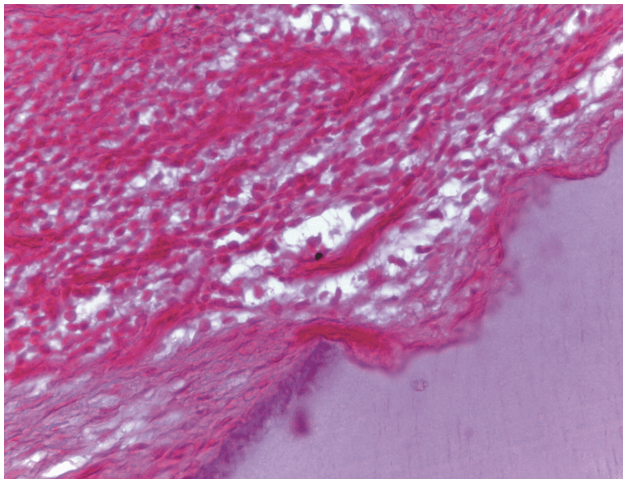

Figure 4 – ***Histological analysis of the tooth–periodontal complex: rich-cells connective tissue was present in the desmodontal space (ob. 20×).***

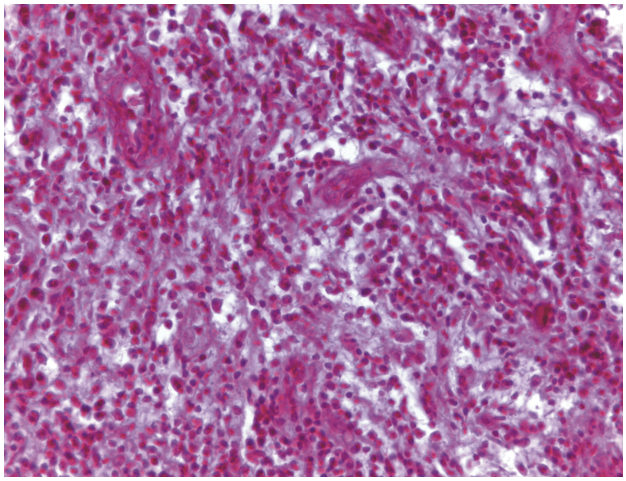

Figure 5 – ***Histological analysis of the gingival connective tissue: intense inflammatory infiltrate was observed in the gingival lamina propria (ob. 40×).***

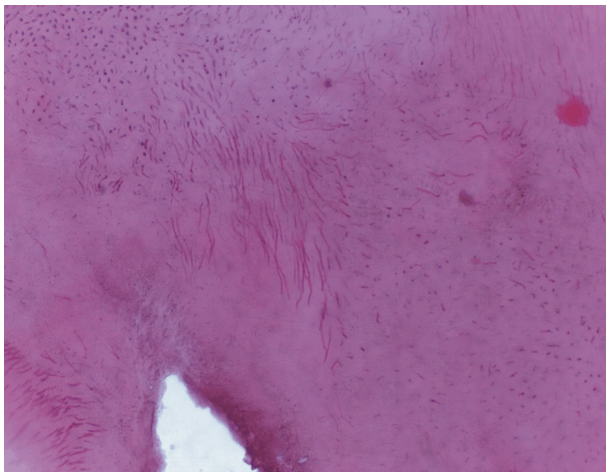

Figure 6 – ***Histological aspect of the radicular dentin: disorganized dentinal areas resembled tertiary dentin (ob. 10×).***

Discussion

This paper presents the histological aspects of the desmodontal–root interface of a tooth lost prematurely in a patient with periodontal disease developing with extreme tooth mobility in the absence of clinical inflammatory signs and with minimal attachment loss. The excessive tooth mobility mimicked aggressive periodontitis, but the lack of classical signs accompanying periodontitis, particularly the attachment loss and crestal bone level led to the assumption that the clinical picture in this case occurred due to a defect of the cementum and a consecutive detachment of the periodontal ligament from the cementum.

Defective cementum and consecutive periodontal ligament detachment were recorded by a study using a mouse model of hypophosphatemic rickets [14]. The disturbance of the cementum is thought to result in a weakened periodontal ligament attachment, which is more susceptible to infection, by periodontal pathogens [10]. Hypophosphatemic rickets is a rare genetic disorder having several forms. A defective Dentin Matrix Protein-1 reported in one form of hypophosphatemic rickets is associated with abnormal dentinogenesis [8] and cementogenesis, impairing the periodontal response to physiological mechanical loading [15, 16]. The defect

of this regulatory protein may explain the diminution of lamina dura with a widening of the periodontal ligament space and also the histological modifications of the dentin for the presented case. However, our patient did not show signs related to growth retardation characteristic for hypophosphatemic rickets. Moreover, cemental resorption recorded for the presented case was not reported as being associated with hypophosphatemic rickets [14].

Alkaline phosphatase is important in bone formation, but also in cementogenesis. In patients suffering from hypophosphatasia, which is associated with a low activity of alkaline phosphatase, the periodontal ligament is not firmly anchored by Sharpey's fibers [17]. However, no biochemical data is available at this time in order to advance in the general diagnosis for the present case.

Cementum hypoplasia has been reported on teeth affected by juvenile periodontitis and was associated with wide areas of root resorption in generalized juvenile periodontitis patients [18]. Generalized juvenile periodontitis reported by the above-mentioned study is presently considered periodontitis-associated with systemic diseases [7], which means that a systemic condition, more probably a genetic one, interfered with the normal periodontal homeostasia. As the authors further considered in the article, the disturbance of cementum formation might have been caused by hereditary systemic factors (such as hypophosphatasia) which subside with increasing age of the patient [18]. Both the present study and that of Blomlöf L *et al*. [18] described periodontal cases developed in systemic circumstances.

Cemental resorption due to periodontal herpes virus infection should not be neglected. It is well known that cytomegalovirus infection may increase the pathogenicity of periodontal microbiota by acting as new bacterial binding sites, by impairing local antibacterial immune mechanisms and by triggering a release of pro-inflammatory cytokines that have the potential to activate osteoclasts [19]. These aspects do not fit with the data of the present case, because the inflammation was minimal and so were the periodontal pockets. Advancing more in the viral hypothesis in the etiology of periodontal disease Ting M *et al.* [20] assumed that a primary cytomegalovirus infection at the time of root formation of permanent incisors and first molars could give rise to a defective periodontium, demonstrated by cemental hypoplasia in the teeth affected by localized aggressive periodontitis [18]. As excessive mobility was recorded for all the teeth in the reported case, a viral trigger in the pathogenesis of the periodontal disease is less probable.

A change in the magnitude of functional loads could exceed the adaptive capacity of the bone–periodontal ligament–tooth complex leading to root resorption [21, 22]. In the presence of a deficient structure of the periodontal/cemental entity, even normal functional loading could lead to degenerative changes like radicular resorption and might explain the histological alterations described by the present study.

Conclusions

Further laboratory analysis and genetic testing are necessary in order to identify the underlying systemic factors that triggered the described severe periodontal degeneration. This report may warn clinicians about the possibility of the association of cemental abnormalities with early tooth loss.

Acknowledgments

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