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Case Report

Clinical management of an atypical dental invagination

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ABSTRACT

Dental invagination (DI) is a tooth malformation that usually affects permanent teeth. Its precise etiology is still controversial and represents a clinical challenge as it can favor the development of carious lesion or periodontal inflammation. This paper presents a case of a 23-year-old Caucasian male, where an atypical buccal DI could not be completely diagnosed in the dens invaginatus category. Furthermore, other differential diagnoses could not be confirmed. The dental malformation was seen on a permanent maxillary first incisor and was associated with periodontal inflammation and attachment loss. Successful clinical management of this case consisted of surgical restorative treatment and regular follow-up, accompanied by thorough oral hygiene procedures.

Key words: Attachment loss, dental invagination, malformation

INTRODUCTION

Dental invaginations (DIs) are developmental malformations affecting different tooth structures. The described form in literature is dens invaginatus (DeI). Scientific literature has reported different plausible etiologies such as failure of growth of internal enamel, the distortion of enamel organ, trauma from deciduous teeth impaction, and genetic predisposition.[1] DeI prevalence in adults has been estimated between 0.3% and 10% of included teeth (0.25%–26.1% of examined individuals).[2] DeI involves enamel with varying levels of involvement into the pulp, thus affecting at different degrees in the underlying dentine. In advanced forms, pulpal integrity could be compromised with a possible periodontal/pulpal communication.[3] Moreover, DeI is a risk factor for bacterial plaque accumulation, thus contributing to dental caries and/or periodontal inflammation initiation. Treatment options for DeI are linked to its stage and extent, and often require a multidisciplinary approach to restore pulpal and periodontal health in DeI associated teeth.[4] DeI has been observed on the palatal side of lateral or central incisor but not on the buccal side of anterior maxillary teeth.

In this report, an atypical DI on the buccal side of the upper left central incisor will be presented. Even though clinical and radiographic features[5] did not correspond to typical DeI condition, no other possible

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diagnosis than DI could be made. Clinical management of this DI case will be discussed.

CASE REPORT

A 23-year-old healthy Caucasian male consulted for a dull discomfort/pain felt on the buccal gingival margin of the upper left central incisor (2.1). The patient explained that he was having food impaction under the gingival margin and that he noticed that the gingiva was inflamed locally on the buccal side. Oral hygiene was satisfactory as the patient was a dental student.

Clinical examination [Figure 1] revealed a concavity on the buccal collar region, associated with a localized gingival inflammation. The concavity involved enamel and extended to root cementum through the cementoenamel junction. The surface of this concavity was sound and hard when probing with a dental probe, ruling out a decay cavity. Furthermore, clinical features did not resemble to tooth loss but rather a dental morphological anomaly. Other differential diagnoses such as acid erosion, occlusion-initiated abfraction, or mechanical abrasion were ruled out due to the absence of any corresponding etiological factors. In addition, no history of dental trauma had been reported by the patient or his parents. The patient did receive an orthodontic treatment, and occlusal stability has been checked.

Hence, periodontal probing showed a localized pocket (4 mm) on the buccal site of the 21. Probing depth ranged from 1 to 2 mm on all other sites of the anterior maxillary sector. Clinical examination failed to reveal any periodontal disease-associated clinical signs on other sites than the buccal site of 21.

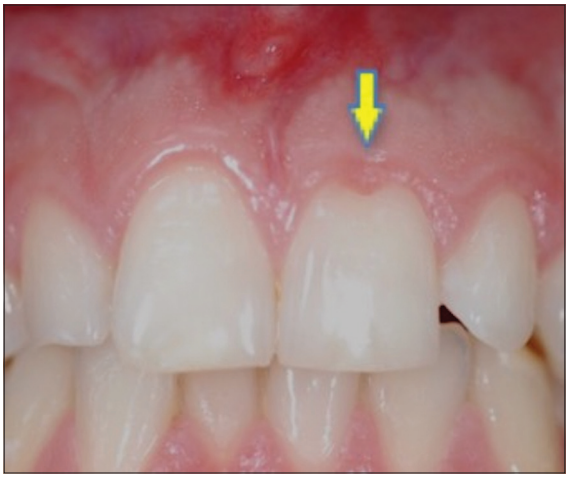

Figure 1: Clinical situation at first consultation

Pulp vitality was confirmed by cold tests. Intraoral periapical radiography (not presented) failed to show any signs of dental/periodontal pathology.

A cone beam radiographic computed tomographic examination was decided to further investigate the dental morphology and the three-dimensional (3D) extent of tooth malformation.

Cone beam computed tomography

Coronal, axial, and 3D views clearly identify a buccal invagination cavity involving merely enamel structure and deepening at the cementoenamel junction and continuing at the root structure subgingivally [Figure 2]. Enamel and cementum showed radiographic continuity and thus did not resemble to root caries or external resorption lesions. Moreover, pulpal integrity was not compromised as no periodontal-endodontic communication was present. However, alveolar bony dehiscence facing the invagination was present, thus favoring plaque accumulation and progression of periodontal attachment loss. The 2.1 was the only tooth showing this malformation.

A diagnosis of an attachment loss associated with atypical buccal DI on the 2.1 was given.

Treatment

It was decided to restore the invagination area surgically. A full thickness minimal flap was raised to expose the buccal root surface of the 2.1 until the bony crest [Figure 3a]. Granulation tissue removal and gentle root planning were performed. After isolating the root from bleeding using hemostatic

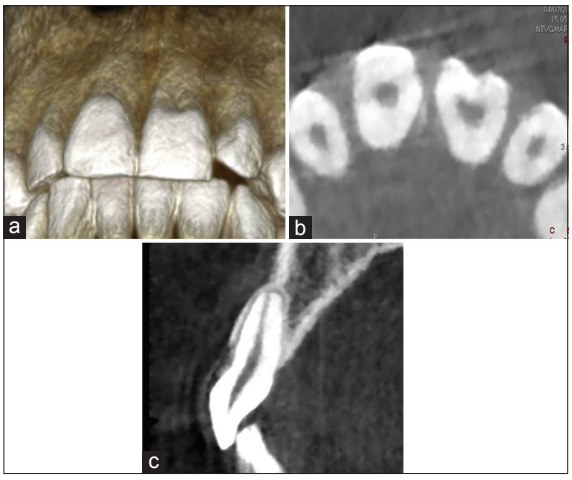

Figure 2: (a) Cone beam computed tomography of the left central incisor. Three-dimensional reconstruction. (b) Axial view showing the buccal invagination. (c) Orthogonal view of the 21, showing that invagination is limited to the cementum/dentine without pulpal communication

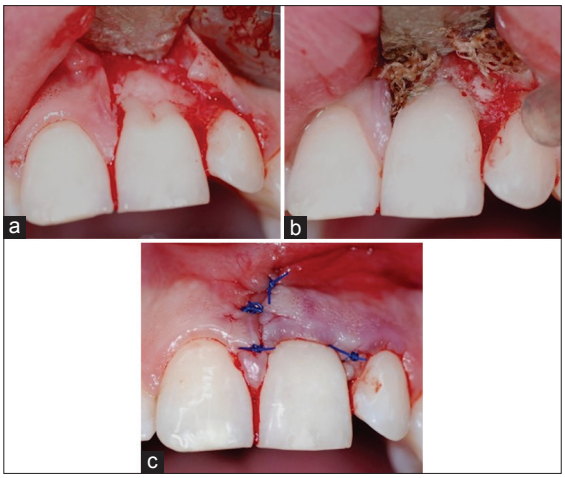

Figure 3: (a) Full thickness flap exposing the invagination. (b) After elimination of granulation tissue, a surgical gauze is used to isolate the invagination site from bleeding, then a two-stage restoration (glass ionomer apically, microhybrid composite coronally) is performed. (c) Flap sutures

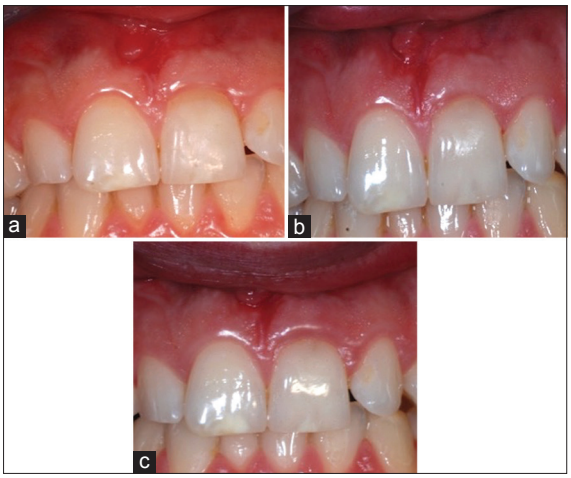

Figure 4: (a) Healing at 1 month (composite polishing was enhanced at this stage). (b) Healing at 12 months. (c) Healing at 18 months

gauze (Surgicel®, Neuchâtel, Switzerland), a resin charged ionomer (Ketac Fil plus® 3M ESPE, Neuss, Germany) was used to fill the subgingival part of the invagination (until the cement-enamel junction). For the supragingival zone, after etching with phosphoric acid (37%) and adhesive application (Scotchbond®, St. Paul, USA), a microhybrid composite (Spectrum®, Dentsply, USA) was used [Figure 3b]. Special burrs and micro discs were used for polishing, and the flap was sutured [Figure 3c].

Recall visits were scheduled at different intervals. Satisfactory periodontal healing was observed on the buccal site. Gingival inflammation and food impaction have disappeared 1 month after surgery [Figure 4a]; during this visit, composite polishing was performed again to assure an optimal contour and surface smoothness. Twelve months after intervention, the site was still periodontally healthy and the patient presented with full satisfaction of the clinical results and the esthetic outcome of the direct restoration [Figure 4b]. Periodontal probing showed the reduction of pocket depth to 2 mm at the buccal/middle site of the 2.1. The last recall visit was at 18 months with a good stability of clinical parameters [Figure 4c].

CONCLUSION

The diagnosis of DI is not obvious and can be questionable at the first glance. In fact, the buccal localization and the absence of visible enamel infold do not correspond to the definition of DeI. Typically, DeI is an accentuation of the lingual pit, in our case we thought that the observed invagination was an accentuation of the cementoenamel junction. Furthermore, sound/smooth dental structures, integrity/continuity of the groove surface, and absence of etiological conditions made us dismiss other differential diagnosis. This DI could have been caused by trauma on primary incisors, but such episode is usually known to the patients and more commonly to the parents. The latter certified that the patient had never had dental trauma during childhood.

This atypical case of DI was successfully managed by a surgical/restorative treatment. The cervical lesion had caused localized periodontal breakdown, thus generating discomfort and unaesthetic gingival inflammation. Untreated, this invagination could have led to carious radicular lesion and further alveolar bone resorption and attachment loss,[6] hence compromising the incisor long-term prognosis.

Therefore, the periodontal repair was achieved on the site, probably with some epithelial reattachment on the glass ionomer wall, which was unexpected knowing the difficulty for the epithelial junction to attach on a restorative material wall.[7,8] Multiple polishing sessions to prevent plaque accumulation, the biocompatibility of the restorative material used, and the awareness of hygiene practices by the patient could have favored clinical healing.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Thakur S, Thakur NS, Bramta M, Gupta M. Dens invagination: A review of literature and report of two cases. J Nat Sci Biol Med 2014;5:218-21.
2. Khan SA, Khan SY, Bains VK, Bains R, Loomba K. Dens invaginatus: Review, relevance, and report of 3 cases. J Dent Child (Chic) 2012;79:143-53.
3. Mupparapu M, Singer SR. A review of dens invaginatus (dens in dente) in permanent and primary teeth: Report of a case in a microdontic maxillary lateral incisor. Quintessence Int 2006;37:125-9.
4. de Sousa SM, Bramante CM. Dens invaginatus: Treatment choices. Endod Dent Traumatol 1998;14:152-8.
5. Vier-Pelisser FV, Pelisser A, Recuero LC, Só MV, Borba MG, Figueiredo JA. Use of cone beam computed tomography in the diagnosis, planning and follow up of a type III dens invaginatus case. Int Endod J 2012;45:198-208.
6. Vehkalahti M, Paunio I. Association between root caries occurrence and periodontal state. Caries Res 1994;28:301-6.
7. Saldanha DV, Gomes SC, Souza DM, Cavagni J, Oppermann RV. Periodontal response to subgingival restorations in dogs with periodontitis. Acta Odontol Latinoam 2012;25:45-52.
8. Santamaria MP, Suaid FF, Carvalho MD, Nociti FH Jr., Casati MZ, Sallum AW, *et al.* Healing patterns after subgingival placement of a resin-modified glass-ionomer restoration: A histometric study in dogs. Int J Periodontics Restorative Dent 2013;33:679-87.