Case Report

Gingival Tissue Proliferation into the Tooth Following Iatrogenic Miniscrew Insertion: A Newer Underreported Complication

Abstract

Miniscrew implant (MSI) has been reported to be an excellent, supportive orthodontic anchorage tool. The placement of MSI is not free from complication and foremost of which is penetration of MSI into the vital tooth structures. This case report is probably the first to report a newer underreported complication. Interestingly, a small bit of gingival tissue was found deep inside the dentin of the tooth, surviving and proliferating in the MSI-damaged zone. This case report is probably the first to give direct histological evidence in humans that a MSI can cause implantation phenomenon which has the potential to give rise to numerous pathologies including pulpal, periodontal, and cystic transformation. Through this report, it is emphasized that clinicians should have a structured, formal MSI placement training to avoid such instances and also underlines the need to develop protocols to be followed in the instance of an inadvertent MSI penetration.

Keywords: Dental pulp, gingival tissue proliferation, implantation cyst, miniscrew, trauma

Introduction

Temporary anchorage devices such as miniscrew implant (MSI) have gained prominence as a reliable source of anchorage. Better patient compliance, relatively low cost, and ease of placement and removal have been cited as the added advantages of the MSI. However, the technology comes with certain drawback that includes the possibilities of damage to vital tooth structures. In the available pertinent literature, there are evidences of periapical pathologies that have been attributed to the placement of MSI. Previous studies have delineated the effect of the MSI on periodontal structures, cementum, and even pulp. However, such studies have concentrated on the type and quantum of healing of hard and soft tissues associated with the teeth. In addition, literature reported in oral and maxillofacial surgery, where fixation screws are used to fix fracture reduction plates, have been shown to cause root damage of varying intensities.

All such studies have not identified the possibilities of surface epithelial ingrowth (EIg) or epithelial implantation (EIp) along the site of damage though the possibility of such EIg has been postulated and shown along the MSI-tissue interface.

When a surface epithelium is forced into the underlying tissues, including hard tissues, there is a possibility of the displaced tissue to survive, proliferate, and give rise to a pathological entity called implantation cysts. There are isolated case reports in literature, where oral implantation cysts have been associated with previous trauma or surgery. However, the possibility of self-drilling MSI driving the epithelium into the underlying tissues has not been postulated till date. The current article highlights a case of inadvertent root damage caused by MSI which resulted in the phenomenon. The possibility of such a clinical scenario and its consequences has been discussed in the current article. The precautions and strategies to prevent such an occurrence have been suggested.

Case Report

A 22-year-old otherwise healthy female voluntarily participated in an ongoing trial, which evaluated the rate of healing of the damaged root surface after prolonged MSI contact (Dentos, Daegu, Korea). This trial was an extension of a previously published study from the institution. This study was approved by the institutional review board, and written informed consent was obtained in the instance of an inadvertent MSI penetration.

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obtained from the patient. Patients warranting extraction of first premolars were included in the study. No exceptions were made in the treatment protocols to suit the study requirements. As per the schedule, MSIs were placed and left in prolonged contact with the root surface of the first premolar for a period of either 4 or 6 or 8 weeks as per randomization. After this period, MSIs were removed, and root was allowed to heal for 4 weeks, after which tooth was extracted atraumatically.

A self-drilling MSI was placed in the patient to establish a root contact and to assess repair of damaged surface after prolonged MSI contact as per the protocol mentioned in previously published literature.[13] However, for this particular patient, the routine confirmatory Intraoral periapical (IOPA) radiograph revealed that the MSI has inadvertently penetrated through the midportion of the root instead of shearing the proximal root surface [Figure 1a], in spite of taking best precautionary measures. Sticking to the standard operating procedure, MSI was immediately retrieved and reinserted into the proximal root surface. Subsequently, the repeated IOPA revealed proper positioning of the MSI as per the requirement of the study design [Figure 1b]. The MSI was left in situ for 4 weeks after which it was extracted uneventfully.

Slow decalcification of the tooth was performed using commercial decalcification agent, Osteomoll® (Merck Millipore, Germany); tissue was processed and stained with Harris hematoxylin and eosin. The slides were subjected to microscopic examination.

Under low power microscopic view, the MSI contacted root revealed two distinct areas of damage. One damage was observed in the mid-portion of the root while the other damage was associated with the proximal surface of the root [Figure 2a and b]. There was a clear, visible split in between these two areas of damage. In the deeper sections, none of the damages were observed. The damage along the proximal root surface exhibited a regular attempt to healing. Lining with surface cells, an attempt by the adjoining periodontal ligament cells to heal and seal the proximal damaged area was observed. However, the proximal damaged area was not completely filled, either by soft or hard tissue. On the contrary, the defect was fresh with no classic signs of epithelial cell lining.

The damage observed in the mid-portion of the root was clear cut with less debris and partially filled by a soft tissue. The soft-tissue proliferation exhibited fibrovascular connective tissue lined by epithelium. The epithelium was thin pseudostratified squamous type with numerous thin proliferating rete ridges in arcing fashion, architecturally resembling the classic gingival tissue. The underlying connective tissue was filled with vascular channels with red blood cells and diffusely placed chronic inflammatory cells. The proliferation was confined to the internal damage and which partially filled the defect. The split or the tract connecting these two damaged areas did not contain any connective tissue or epithelial elements but had debris in the tract [Figure 3a-c].

The pulp tissue exhibited intense inflammatory reaction. Diffuse chronic infiltration was observed all along the pulp, even at the radicular end. The end exhibited damage and large vacuolization of the odontoblast along the dentin-pulp complex, proliferating and replacing the cellular pulp with fibrous elements and with one focus resembling questionable dentin-like debris [Figure 4a-d]. The blood vessels were characteristically dilated. Consecutive serial histological sections failed to establish or associate the continuity of the surface epithelium into the defect. The deeper part of the mid-root portion damage showed shearing of the dentin at its terminal end suggesting the damage during the retrieval attempt. The damaged area showed organizing of the epithelial lining [Figure 4d]. Based on the above-mentioned information, one could infer that some dentinal debris has been pushed into the dental pulp. The damage has caused disruption of the odontoblastic layer even in remote apical region.

Figure 1: (a) First attempt of miniscrew implant insertion failed to establish proximal root contact, resulted in miniscrew implant penetration into the mid-portion of the root. Note the inadvertent penetration, (b) second attempt of miniscrew implant insertion established a proper proximal root contact. Note the lateralization

Figure 2: (a) Low power scanned version of the demineralized histopathological sections revealed two distinct areas of miniscrew implant penetration, (b) deeper sections revealed no visible miniscrew implant penetration – but remarkable changes in the pulp cavity were observed with proliferation and obstruction along the area.
Discussion

In spite of widespread usage of the MSI, many clinicians are reluctant to use MSI in their clinical practice. They were reportedly apprehensive about the possible inadvertent trauma to root and the adjoining structures.[24,25] There are reports in the literature of such inadvertent damage of the root/dentin-pulp.[9‑18,25] The healing of damage to human tooth structures and pulpal tissues was increasingly reported.[10,15] All these reports were either done by intentional MSI damage (of shorter duration) or isolated case reports (with wider time frame). The present case report is probably the first to report the possibility of an EIg or EIp phenomenon. The consecutive histological sections failed to demonstrate the “epithelial connections” from surface of the tooth (and thus probably to gingival surface). The absence of epithelium or other tissues in the tract connecting two damages, further rules out the epithelial migration from proximal surface damage to mid-portion root damage. Hence, this could be only viewed and deduced as EIp and not as an EIg phenomenon.

Half a decade ago, Henry attempted to use the gingival epithelium as a biological pulp capping material.[26] Harvested gingival epithelium was inserted into the pulp through a carefully prepared Class V cavity of teeth in young dogs. Even before that, there have been reports of cysts with epithelial lining inside the tooth. In the results of their experiment, Henry observed epithelium organizing into islands, lining the abscess (infected), and in some instances, suggestive of cyst formation as early as 96 h of insertion of the gingival tissue. This study, though failed to meet its objective, gives a clue of survival of epithelium in the dental tissues and probably forms a cyst at a later stage.[26] In a study, Petersson et al. demonstrated the EIg into the intentionally damaged teeth of dogs. The epithelium was either from surface or gingival pockets. The EIg was observed as early as 3 weeks to as late as 1-year period. However, this study failed to show organization of cyst in their samples.[27] Probably, the associated inflammatory response following the damage would probably reduce the chance of survival of the EIg in deep tissue planes. If the epithelial tissue rapidly fills the damage area and retains the connection to its external surface parent tissues, the pushed epithelium survives and proliferates. In either case, the origin of epithelium is undoubtedly gingiva.

In the present case too, the self-drilling MSI has probably pushed the superficial epithelium, along with connective tissue into the root where they continued to survive, probably deriving nourishment from the inflammatory exudate during earlier phase and transudate in latter phase. The survived tissue components further multiplied and established angiogenesis. The presence of gingival architecture and abundant, vascular channels supports the possibility of this condition as EIp. If only surface epithelium is pushed into the root, they probably would not have survived successfully.

Alternatively, the surface epithelium along with the connective tissue, in the presence of inflammatory mediators (in response to trauma), probably has “crept” into the defect by growing along the edges into the root and this phenomenon is called EIg. If the condition had been an EIg, the continuity or the ingrowth would have been seen in at least one of the serial sections. In addition, the proliferation was seen only in the damaged area caused by first MSI penetration site and not in the second MSI penetration. Probably, the clinician used the
same puncture site, which by the second time was devoid of epithelium. The second damage site was mostly superficial which showed only connective tissue elements. Based on the above features, we had empirically concluded that the condition is an ELp rather than an ELg. The difference is clinically obscure as both entities can lead to organization of the epithelial proliferation. This at a later stage could lead to cystogenesis if conditions were suitable.

In addition, the intense damage of dentin at the apical region, the presence of intense inflammation, increased fibrotic nature, damaged odontoblastic layer, and dentinal debris, one could infer the significant pulp damal damage though the patient was clinically asymptomatic. Later, this condition could lead to periapical pation and probably an endodontic cyst, as reported in German dental literature and cited by Henry[26] though pertinent orthodontic and maxillofacial surgical literature have no reports of such “endodontic cysts” or ELg or ELp. To the best of our knowledge, this could be the first report of such ELp associated with MSI. With newer technical procedures to accelerate tooth movement using propel and other corticotomies could theoretically drive the gingival epithelium causing a ELg or ELp at a later stage.

The easier way to prevent ELg or ELp would be to avert “tissue rolling” while placing the MSI. In case, a rolling of tissue is felt while inserting MSI, copious irrigation, and cleaning of MSI with saline is essential before the next attempt. Alternatively, the best way would be to incise the region of MSI insertion and place it directly on the bone rather than on any soft tissue.

The current case highlights the mechanism of splitting of teeth by multiple MSI insertion through crack propagation and linking of the multiple damages. Literature has reports of teeth associated with unintentional MSI damage to split.[16,28]

The present case demonstrates that MSI is not free from complications, especially one that could compromise the vitality of teeth or even possibly cause a gingival tissue proliferation inside the teeth or even bone. Although the clinician was trained, calibrated, and supervised by experienced orthodontists, such unique instance has been encountered. The clinician, especially orthodontists, needs to undergo special education, training, and probably frequent clinical updating before they place MSI in their clinical practice. The following simple precautionary procedures would avoid or avert such instances.

Recently, there has been a report of an interesting episode associated with MSI.[29] A well-experienced orthodontist placed a MSI between the apices of maxillary central incisors for correction of the mal-aligned incisors. During the course of treatment, right maxillary central incisors became sensitive and started losing its natural color. As the MSI was very close to its root apex, the MSI was removed. Within 2 weeks, the acute symptoms resolved. The authors claimed that the presence of the MSI in root apex would have probably hindered normal pulpal blood flow that caused a reversible, ischemic response in the pulp. However, this report is based on radiology, and the pulpal events are largely speculative. This case report reflects the following facts:

- Even experienced dental specialists have adverse episodes of MSI placement
- Failure rate of MSI was relatively high even in the hands of a well-trained, experienced specialty dentist
- This case report adds to our current report to highlight the need for training before the placement of MSI.

**Conclusion**

This case report presents a newer iatrogenic complication induced by unintentional trauma following MSI placement to tooth, leading to subsequent gingival proliferation, into the tooth. Such inadvertent instances may happen in clinical scenarios and often go unreported. Proper protocol and evidence of practice need to be evolved. Exclusive clinical training for MSI placement shall be made mandatory as part of curriculum.

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**Conflicts of interest**

There are no conflicts of interest.

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