Osteomyelitis as a possible cause of rootless Turner’s tooth: a case report

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Abstract

Tooth extraction is an ordinary type of treatment that is performed routinely at dental treatment units. The procedure of dental extraction may be accomplished without any collateral complications or, on the other hand, be followed by problematic consequences. Many factors control the outcome of the extraction procedure, whether it is conducted smoothly or in a vigorous way. In the current study, we present a case of an ordinary extraction of a primary tooth revealed a more advanced infection, i.e. chronic suppurative osteomyelitis, which might have caused a cease of the root formation and hypoplasia of the crown of the succedaneous tooth.

Key words
Osteomyelitis, Extraction, Hypoplasia

1 Introduction

Osteomyelitis is an infection of bone (periosteum and bone marrow), including the harvesian system, of which many clinical varieties are known. For example, chronic recurrent multifocal osteomyelitis is an aspetic inflammatory condition whose etiology is not yet known and it affects mainly children and adolescents [1]. Osteomyelitis can be manifested as an acute episode of osseous infection (acute type) or chronic low-grade inflammation (chronic type).

The occurrence of osteomyelitis is sometimes associated with the formation of a fragment of necrotic bone that has separated from the adjacent vital bone, known as sequestrum. In the facial skeleton, the occurrence of osteomyelitis in the mandible is more common than in the maxilla, owing to the poorer blood supply of the former [2]. The etiology of osteomyelitis is varied; but form, dental abscess is the primary cause where periapical infection can spread through the marrow spaces and cause infection. Physical injury, e.g., local trauma such as fracture, penetrating wounds, surgery, and extraction can also predispose the bony tissue to osteomyelitis [2].

The infectious agents can also migrate through a hematogenous route as in the case of upper respiratory tract infection [3] and staphylococcal infection of skin.

In addition, special infections such as tuberculosis, syphilis, actinomycosis can associate with osteomyelitis [4].
Entry of microorganisms into bone marrow is followed by their proliferation in the marrow spaces, which gives rise to inflammatory reaction, tissue necrosis and suppuration. Once marrow spaces become filled with pus, suppuration tend to spread through the marrow spaces and cortical bone to involve the periosteum; the osteoblasts differentiate and necrotic bone (sequestrum), bathed in pus, separates from the surrounding vital bone.

The treatment spectrum of osteomyelitis ranges from drainage and/or removal of the cause such as non-vital tooth, and prescription of a course of antibiotics to prevent further spread and complications [5]. Surgical debridement of the sequestrum (sequestrectomy) becomes a necessity if such necrotic bone was encountered during the treatment [6].

Each case should be judged individually because of the variations in disease severity, type of the microorganisms involved and the patient’s immune status.

If osteomyelitis is left untreated it may develop into periostitis, soft issue abscess, or cellulitis raising the chances of pathological fracture [7].

2 Case report

A five-year-old female patient presented to the Department of Pediatric Dentistry, Faculty of Dentistry, complaining of pain and swelling related to a lower left tooth in the posterior region. The culprit tooth had been treated with pulpectomy several months earlier. On examination, there was a temporary filling in lower left primary second molar. Besides being tender to finger pressure, it showed a mobility of grade 3. A dentoalveolar abscess was associated with the mobile tooth, and the buccal gingival margin surrounding the tooth had a reddish discoloration (see Figure 1).

Excluding the area of complaint, all other soft tissues were normal.

Later, the lesion was diagnosed as a chronic dentoalveolar abscess due to decayed lower left second primary molar. As a requirement of emergency treatment, the tooth was planned for extraction, followed by teaching the patient the proper way of oral hygiene practice. The patient would then be appointed for restoration of other carious teeth and construction of distal shoe space maintainer.

The tooth was anesthetized, and while the tooth was being removed, a piece of bone came along with the tooth. This bony piece (see Figure 2), which seemed to be a necrotic sequestrum, was nearly of the same size of the removed tooth. After removing both structures, the socket was sutured with pressure pack applied, and the patient was dismissed and kept on...
follow-up. Meanwhile, the bony mass was sent for histopathological examination; which revealed non-vital osseous tissue with empty canaliculi traversing the whole structure. A diagnosis of chronic suppurative osteomyelitis was confirmed.

Figure 2. The bony sequestrum beside the removed tooth

After three months, the patient came again complaining of pain in another tooth. On examination of the previous extraction site i.e., of the lower left second primary molar, it seemed that it had healed uneventfully; while the lower left second premolar started eruption. By inspection, the succedaneous tooth was hypoplastic and poorly shaped (see Figure 3). To our surprise, the radiographic examination showed that the tooth was rootless (see Figure 4).

Figure 3. The clinical appearance of the yet-erupting lower left second premolar

Figure 4. The second premolar apparently had no root; only the crown can be seen
After two weeks, the patient came again and we noticed that the lower left second premolar became even more discolored (see Figure 5). We planned to restore the hypoplastic lower left second premolar with a stainless-steel crown, but unfortunately, we have lost contact with the patient since then.

![Figure 5. The increased yellowish brown discoloration of the lower left second premolar](image)

### 3 Discussion

Infection of the periapical area is often localized and does not spread beyond; thanks to the boundaries of the periapical dense tissues. However, under certain circumstances, the infectious microbes can supersede this confining barrier and spread to extra-alveolar spaces causing more devastating pathosis [8]. In this regard, osteomyelitis, which is defined as inflammation of the bone starting from the medullary cavity and rapidly involving the haversian system [9], can be among the devastating consequences of dental extractions.

Among the etiological spectrum, chronic odontogenic infection, trauma, improperly treated fractured bone in the jaw and irradiation to the mandible constitute conventional causes of osteomyelitis. Dental hypoplasia has been defined as a quantitative defect of the tooth enamel, owing to decreased number of ameloblasts, leading to visually as well as histomorphologically disturbances mostly associated with reduced thickness of enamel [10]. In this sense, dental hypoplasia is a condition which can affect a wide range of teeth in the mouth, sometimes all teeth are affected if the etiological agent is generalized e.g., in cases of infection or fluorosis. If the condition affects only one tooth, then it is referred to as Turner tooth or Turner hypoplasia [11].

In the present case, the condition started with a periapical infection around the second primary molar, which is not uncommon in her age group. What extended beyond expectation was the existence of bone infection of the mandible (osteomyelitis) that ensued upon extracting the tooth.

Padmanabhan et al., reported a case where chronic osteomyelitis was associated with a developmental defect of a lower posterior tooth (affected with odontome) in a line of mandibular fracture [12].

In our case, there was no extra-oral involvement, i.e., no sinus or scar tissue had been observed on the patient’s face. The patient also denied any history of trauma to the area of interest. Thus, we believe that the osteomyelitis in our patient had already developed in her lower left posterior region due to the low-grade infection fuelled by the second primary molar. We cannot determine whether the pain originated from the tooth or the infected bone. The chronicity of the infectious process might explain the easy and smooth removal of the circumscribed bony piece along with the tooth. Consequently, and on her next appointment, we noticed that the erupting permanent tooth was hypoplastic. Indeed, it can be argued that the cause of hypoplasia of this particular tooth was due to the periapical infection of the preceding primary second molar. However, the unusual associated feature was the absence of roots of the permanent tooth, which raises the possibility of
involvement of another more radical infection in this clinical scenario. We assume that the bony infection has destroyed the Hertwig’s epithelial root sheath, thereby hampering the root development. What remains to be answered is how the tooth erupted without a root despite some claims that root formation is critical for teeth to make their paths to the oral cavity. Recently, Wang suggested, based on previous experiments and case series observations, that for a tooth to erupt into the mouth does not necessitate the concomitant development of the root albeit it is important for later stabilization in the future dentition [13]. Unfortunately our loss of contact with the patient halted our following of the case and thus assessing the durability of this rootless tooth to withstand the mastication process and other forces which require teeth with otherwise sound crowns and fully formed roots.

References