Role of Cone Beam Computed Tomography in Leading to the Finding of Actinomycosis Mimicking Periodontitis

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Abstract
A gram-positive anaerobic bacteria, Actinomyces, is frequently found in an extra-radicular infection. This report aims to present a case of persistent radicular cyst associated with extra-radicular Actinomyces infection in a periodontitis patient. This report highlights an extensive alveolar bone destruction and the advantages of cone beam computed tomography (CBCT) imaging by the provision of a superior information to the details of the bony architecture. A 63-year-old female presented with an endo-treated tooth of 37 with a deep probing pocket depth (PPD) in the midbuccal area with furcation involvement and mobility. CBCT images demonstrated a well-defined bone loss region involving the mesial root of tooth 37 extending to the apical 38 that cannot be detected from the periapical radiograph. The histopathological examination revealed a cystic lesion lined in small parts by non-keratinizing stratified squamous epithelium and club-shaped filaments that formed a radiating rosette pattern. While CBCT imaging helps to a great extent in determining the level of bone destruction, a histopathological examination is mandatory in establishing the diagnosis.

Keywords: Actinomyces, Actinomycosis, Cone Beam Computed Tomography, Extra-radicular infection, Periodontitis.

Introduction
Actinomyces is a facultative, anaerobic, rod-shaped Gram-positive bacteria that belongs to the Actinobacteria class. These bacteria are ubiquitous human flora typically found in the oropharynx, gastrointestinal and genitourinary tract.1 In the vicinity of oral cavity, a diverse population of Actinomyces species exist in saliva, dental plaque, dental caries, periodontal pocket and gingival crevice of individuals with healthy and diseased periodontium. These bacteria, though present in abundance, do not result in an infection provided they are retained on the mucosal surfaces. Several infections have been associated with Actinomyces species including cervicofacial actinomycosis and periapical actinomycosis, among others, of which the categorisation is based on the specific anatomic sites.1 In cervicofacial actinomycosis, the infection frequently resulted from an oromaxillofacial trauma, dental manipulation or dental caries. Bacterial invasion occurs when the mucosal integrity is disrupted and the infection is characterized by a draining fistula tract that contains characteristic purulent discharge of sulphur granules.

In recent years, cone beam computed tomography (CBCT) has emerged in periodontology field, due to its high accuracy for periodontal bone defect, especially in the intrabony and furcation defects.2,3 Unlike conventional radiographs, all structures that are not in the plane of interest can be removed from the image, thus resulting in a clearer visualisation of the respective slices.2,3 CBCT help clinicians in assisting the clinical diagnosis, prognosis, and treatment planning, particularly in complex cases.6–11


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Although *Actinomyces* infection is a known cause of persistent periapical lesions following a root canal treatment, it rarely causes an extensive periradicular alveolar bone destruction. Herein, we report a case of persistent radicular cyst associated with an extra-radicular *Actinomyces* infection in a periodontitis patient. This report highlights an extensive alveolar bone destruction and the advantages of cone beam computed tomography (CBCT) imaging in the provision of superior information regarding the details of the bony architecture.

**Case Report**

A 63-year-old female patient was referred to the Department of Periodontics for the management of her periodontal condition. She reported a history of gingival bleeding from the lower left posterior region for the past two years that was prompted by tooth brushing and chewing. Medical history revealed that she had squamous cell carcinoma of tongue that was successfully treated by partial glossectomy in 2014. She was otherwise healthy. She is a non-smoker and has never consumed alcohol.

Upon clinical examination, all but lower left first molar was present. Further history revealed that the tooth 36 was extracted due to a failure in an endodontic treatment. The lower left second molar (tooth 37) was identified as the site of complaint. Vitality test confirmed that the tooth was non-vital. There was a deep probing pocket depth (PPD) of 9 mm on the midbuccal area with a furcation involvement grade 2 and a grade 2 mobility. The surrounding gingiva was erythematous and oedematous with loss of normal gingival stippling and scalloping. An examination on the other teeth revealed a localized deep PPD of 5 to 7 mm noted on the molar teeth in all sextants. The full mouth periodontal charting is presented in Fig. 1. Her oral hygiene status was relatively good.

An intraoral periapical radiograph of tooth 37 with the insertion of gutta-percha through the periodontal pocket was taken and yielded an early establishment of a sinus tract at the periapical area though the tract was not clinically evident. There was an early alteration of the periradicular alveolar bone trabeculae with a reduced density. Furcation defect and horizontal bone loss involving one-third of the root length were also noted (Fig. 2). Based on the clinical presentation, two baseline diagnoses were established, the first was localized periodontitis Stage III Grade B and the second was combined periodontal-endodontic lesion, with primary periodontal and secondary endodontic involvement of 37.
A comprehensive periodontal management was initiated. The initial phase therapy consisted of full mouth scaling and root debridement of teeth with deep PPD of ≥ 5mm, including tooth 37 using a combination of hand and ultrasonic instrumentations, with copious irrigation of chlorhexidine 0.12%. Local anaesthesia was administered prior to the treatment. The root canal treatment on tooth 37 was completed in three visits, and the canals were maintained with calcium hydroxide dressings in between visits.

The first periodontal review indicated a great reduction of the deep PPD in all affected teeth. However, there was no improvement on the PPD of tooth 37. The second periodontal review visit was conducted at a three months interval, during which the patient complained of discomfort upon biting on tooth 37. Remarkably, the tooth had a progressive mobility into Grade 3 with persistently deep PPD. A periapical radiograph was repeated and a crack line was evident at the furcation area of the tooth. The density of periapical radiolucency remained the same (Fig. 3).

The patient was sent for a CBCT imaging on the same day to rule out the possibility of a root fracture. CBCT images (Fig. 4, 4A, 4C) demonstrated a well-defined bone loss region involving the mesial root of tooth 37 extending to the apical 38 with furcation involvement. The superior border of the mandible appeared resorbed with evidence of buccal cortical bone thinning and sclerotic bone formation. However, no crack line was detected from the CBCT images.

In view of unfavorable prognosis, the tooth was extracted and the underlying socket was curetted. The periapical tissue obtained was submitted for a histopathological examination. The patient also was sent for haematological investigation to exclude systemic diseases. Upon a review visit a week later, the patient came asymptptomatically as the healing was uneventful. The result of the blood test turned out to be normal.

The histopathological examination (Fig. 5) revealed a cystic lesion lined in small parts by non-keratinizing stratified squamous epithelium, therefore, confirming the diagnosis of a radicular...
cyst. The specimen consisted mainly of scattered blood pools that were diffusely infiltrated by neutrophils. Numerous microbial colonies of varying sizes were embedded within the blood pools, some of which demonstrated club-shaped filaments that formed a radiating rosette pattern. The filamentous bacteria were strongly highlighted by Grocott Methenamine Silver (GMS) and Gram stain.

**Figure 5.** Histopathology of the lesion. a) Photomicrograph showing inflamed fibrous cyst wall lined by non-keratinized stratified squamous epithelium (hematoxylin and eosin stain; original magnification x 200). b) Photomicrograph showing club-shaped filaments forming radiating rosette pattern embedded within blood pool. A diffuse neutrophilic infiltration was noted (hematoxylin and eosin stain; original magnification x 200). c) Filamentous bacteria highlighted by GMS (Grocott Methenamine Silver stain; original magnification x 200) and; d) Gram stain (Gram stain; original magnification x 200).

Based on the histopathological findings, a diagnosis of radicular cyst superimposed with *Actinomyces* extra-radicular infection was established. The patient was prescribed with Augmentin® (amoxicillin and clavulanate) with a dosage of 625 mg three times a day for ten days. She was put on a regular monitoring for the first month, followed by monthly monitoring for two months.

Three months later, the patient was called for a subsequent periodontal review. A full mouth periodontal charting showed no PPD of ≥ 5 mm. A CBCT of the previously extracted 37 region demonstrated an increased radio-density at the peripheral segment of the lingual plate in correlation with new bone (Fig. 4B and 4D). The surrounding bone appeared sclerotic, suggestive of a bony inflammatory reaction. Following positive response to the treatment, a diagnosis of a clinical gingival health on a reduced periodontium was made. She was referred to the Prosthodontic Department for rehabilitation of her missing teeth.

**Discussion**

The present study has identified periapical actinomycosis-mimicking periodontitis. Periapical (PA) actinomycosis is an uncommon finding with an incidence rate of less than 5%. The infection is frequently found in association with radicular cyst and periapical granuloma among all radiolucent PA lesions. In earlier days, most PA actinomycosis was reported as an incidental finding in biopsy specimens of persistent PA lesions following root canal treatment, resulted in the diagnosis being established retrospectively. As the disease entity becomes more acquainted, an increasing number of prospective studies have been reported mainly to evaluate the treatment outcome. Several *Actinomyces* species have been isolated in PA actinomycosis including *A. Israelii*, *A. Odontolyticus*, *A. Viscosus*, *A. Naeslundii*, *A. Meyeri* and *A. Radicidentis*. The ability of the bacteria to form a confluent aggregate enables them to escape host defense system and they are sustained in the PA region by a relatively low oxygen pressure.

It has now been established that PA actinomycosis does not exist independently. The source of an extra-radicular infection is from an intra-radicular infection following the detection of *Actinomyces* species in both untreated and root canal-treated teeth. In the latter, the microorganisms were found to colonise the apical foramen and the underlying PA lesions concomitantly. Although some studies have suggested that extra-radicular is an entity independent of intra-radicular infection, the evidence are somewhat weak and the probability of an intracanal *Actinomyces* being pushed into the extra-radicular space was not ruled out. In long-standing PA lesions, the existence of a draining sinus or fistula tract is not uncommon and when present, it indicates an extra-radicular infection. *Actinomyces* can be easily introduced...
into the root canal system through a leaked coronal seal and an inadequately root-filled canal, and therefore effective controls of the intraradicular infection is an important element in ensuring closure of the tracts and subsequent periradicular healing.26

In our case, there was an early formation of a sinus tract that was detected on PA radiograph. Given the complex periodontal-endodontic relationship, the presence of a sinus tract can be misdiagnosed as a suppurative apical periodontitis or periapical abscess. Many studies reported a high success rate for nonsurgical treatment of teeth with sinus tracts.26 However, there was no mention about periodontal conditions in most of the cases, and when mentioned, the periodontal condition appeared stable. The diagnosis of PA actinomycosis could be easily dismissed in the present case if biopsied tissue was not submitted for histopathological examination since the tissue submission for microscopic examination is not routinely done in periodontology practice.

Our case demonstrates superior advantages of CBCT in acquiring detailed structural and dimensional changes and the extent of alveolar bone destruction which was not visualized on PA radiograph. Although the two-dimensional intraoral PA image is commonly used as an adjunct to clinical examination in periodontal diagnosis, it demonstrates an incomplete representation of bony destruction due to hard tissue superimpositions.9 On the contrary, CBCT images demonstrate an accurate measurement of multi-planar intrabony defects.5 In the present case, this differences could be observed from the periapical and CBCT image that were taken in a same day. However, due to the difference in radiographic devices used prior to and after root canal treatment in our case, the size of the PA radiolucency could not be compared accurately.

A huge alveolar bone destruction was an interesting observation in our case. Whether or not the size of actinomycotic colonies correlated with the size of PA lesions could not be ascertained due to limited number of studies and there were inconsistencies in the findings.20,30

Notwithstanding, extensive alveolar bone destruction was unusual. It is difficult to infer herein if the inflammatory process that has already existed in the periodontium was exacerbated by PA actinomycosis, leading to an extensive alveolar bone destruction. Such extrapolation is hampered by the fact that radicular cyst can reach to a substantial size even without a histologic evidence of Actinomycotic colonies.31 In regard to tooth 36, no histologic examination was done and therefore we could not ascertain if extra-radicular Actinomyces infection had occurred from which the disease process might have instigated and perpetuated to involve tooth 37 in view of proximity.

A long term course of antibiotics has been recommended and prescribed for the patients diagnosed with actinomycosis.32,33 Individualised antimicrobial therapy depending on the initial burden of the disease, the site of infection, or the clinical and radiological response to treatment need to be considered.34 In the present case, our patient was prescribed with the combination of penicillin (amoxicillin) and a beta-lactamase inhibitor (clavulanate) for a wide coverage against penicillin-resistant aerobic and anaerobic co-pathogens.32 However, other study indicated a treatment of 1.5 g amoxicillin once daily for 10 days.20

Conclusions

A non-healing area of localized periodontitis or perio-end lesion which failed to respond following completion of periodontal therapy and endodontic treatment should raise a suspicion of extra-radicular Actinomyces infection. CBCT was found to be more accurate than periapical radiograph in assessing molar with furcation involvement. A histopathological examination should be considered in the case of unusual destruction in the oral tissues to establish the diagnosis.

Consent

The authors certify that they have obtained all appropriate patient consent form. In the form, the patient has given her consent to perform ordinary procedures, including radiograph and photograph for the purpose of education.

Declaration of Interest

The authors report no conflict of interest regarding the publication of this paper. All authors have made substantive contribution to this study and/or manuscript, and all have reviewed the final paper prior to its submission.
References


