

Surgical Management of an Inflammatory Cyst with Platelet Rich Fibrin (PRF)

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Abstract

Case Report

Radicular cysts are most common inflammatory jaw cysts found at the apices of teeth with infected and necrotic pulps. It involves the apex of carious tooth mostly. It is a true cyst, since the lesion consists of pathologic cavity lined by epithelium and is often fluid filled. Radicular cyst which develops subsequent to extraction is termed as residual cyst. Cyst can be managed surgically, or non-surgically. Many surgeons have different views on its management, however choice of treatment depends on site and size of cyst. The present case is a characteristic radicular cyst which was successfully managed with root canal therapy (RCT) along with surgical enucleation and placement of platelet rich fibrin (PRF), and apicectomy of the affected tooth.

Keywords: Odontogenic cyst, radicular cyst, RCT, PRF, enucleation.

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INTRODUCTION

A cyst is a closed sac-like structure within a tissue that contains a liquid, gaseous, or semisolid substance and is lined by epithelium. Radicular cysts are fall under the category of odontogenic cysts, as they are lined by nonkeratinized stratified squamous epithelium, which is derived from odontogenic epithelium [1, 4]. Radicular cyst is of inflammatory origin and is believed to be formed by proliferation of epithelial cell rests of Malassez in the area of apical periodontitis of a tooth having an infected necrotic pulp [2].

Radicular cysts are rare in the primary dentition, but are the most common (52%-68%) cystic lesions affecting the permanent dentition. But its actual prevalence is only about 15% among the apical periodontitis lesions [5, 7]. According to Bhaskar and Shear radicular cyst is most common among patients in their third decade of life and is more common in men than women. The maxillary anterior region of and the mandibular premolar region are more frequently involved [2].

Clinically, radicular cysts are associated with teeth with extensive or deep dentinal caries, have undergone previous restorative treatment, or might have sustained trauma or may have apparent root canal treatments failures. Most radicular cysts develop slowly. Patients do not have painful symptoms, unless

subsequent infection of the cyst causes swelling and pain. The lesions are often only detected during routine radiographic examination. If the cyst grows large, symptoms such as swelling, light sensitivity, dental mobility and displacement can be observed. The affected tooth does not respond to thermal and electrical tests. Large cyst can expand the cortical and cause teeth displacement. There may be discolouration of the affected teeth. At first, the enlargement is bony hard; but as the cyst increases in size, the bony covering becomes very thin and the swelling then exhibits springiness and becomes fluctuant when the cyst has completely eroded the bone. In the maxilla there may be buccal or palatal enlargement whereas in the mandible it is usually labial or buccal and only rarely lingual. In the maxilla there may be buccal or palatal enlargement whereas in the mandible it is usually labial or buccal and only rarely lingual [7, 8].

Radicular cysts arise from the epithelial residues in the periodontal ligament as a result of inflammation. Bacteria from the gingival sulci or periodontal pockets have been suggested to reach the root canals of these teeth through severed periodontal blood vessels. Pulpal infection can also occur through exposed dentinal tubules at the cervical root surface, due to gaps in the cemental coating. Microbes have also been claimed to 'seed' in the necrotic pulp via the blood circulation (anachoresis). Initially, the tooth pulp becomes infected and necrotic by an autogenous oral microflora. The endodontic environment provides a

selective habitat for the establishment of a mixed, predominantly anaerobic, flora. A sine qua non for the diagnosis of a radicular cyst is the related presence of a tooth with a non-vital pulp. Occasionally, a sinus may lead from the cyst cavity to the oral mucosa [1, 2, 7].

Collectively, this habitat-adapted polymicrobial community residing in the root canal has several biological and pathogenic properties, such as antigenicity, mitogenic activity, chemotaxis, enzymatic histolysis, and activation of host cells. The microbial invaders in the root canal can advance, or their products can egress, into the periapex. In response, the host mounts an array of defences consisting of several classes of cells, intercellular messengers, antibodies, and effector molecules. The microbial factors and host defence forces encounter, clash with, and destroy much of the periapical tissue, resulting in the formation of various categories of apical periodontitis lesions.

There are two distinct categories of periapical cysts, namely, those containing cavities completely enclosed in epithelial lining, and those containing epithelium-lined cavities that are open to the root canals. The latter was originally described as 'bay cysts' and has been newly designated as 'periapical pocket cysts'. More than half of the cystic lesions are true apical cysts, and the remainder are apical pocket cysts.

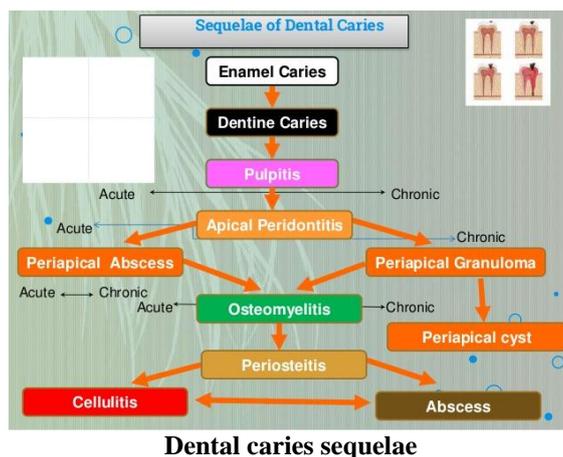
The periapical true cyst may be defined as a chronic inflammatory lesion at the periapex that contains an epithelium-lined, closed pathological cavity. An apical cyst is a direct sequel to apical granuloma, although a granuloma need not always develop into a cyst. The pathogenesis of the true cyst has been described in three phases. During the first phase, the dormant cell rests of Malassez begin to proliferate as a direct effect of inflammation, probably under the influence of bacterial antigens epidermal growth factors, cell mediators and metabolites that are released by various cells residing in the periapical lesion. During the second phase, an epithelium-lined cavity comes into existence.

There are two main theories regarding the formation of the cyst cavity. The 'nutritional deficiency theory' is based on the assumption that the central cells of the epithelial strands become removed from their source of nutrition and undergo necrosis and liquefactive degeneration, the accumulating products in

turn attract neutrophilic granulocytes into the necrotic area. Such microcavities containing degenerating epithelial cells, infiltrating mobile cells and tissue fluid coalesce to form the cyst cavity lined by stratified epithelium.

The 'abscess theory' postulates that the proliferating epithelium lines an abscess cavity formed by tissue necrosis and lysis because of the innate nature of the epithelial cells to cover exposed connective tissue surfaces. During the third phase the cyst grows, but whose exact mechanism is still unknown. It is generally believed to be by osmosis. The presence of necrotic tissue in the cyst lumen attracts neutrophilic granulocytes, which extravasate and transmigrate through the epithelial lining into the cyst cavity where they perish. The lytic products of the dying cells in the cyst lumen release a greater number of molecules. As a result, the osmotic pressure of the cyst fluid rises to a level higher than that of the tissue fluid. The latter diffuses into the cyst cavity so as to raise the intraluminal hydrostatic pressure well above the capillary pressure. The increased intracyst pressure may lead to bone resorption and expansion of the cyst. The T-lymphocytes and macrophages in the cyst wall may provide a continuous source of bone resorptive metabolites and cytokines. The presence of effector molecules such as matrix metalloproteinase-1 and -2 have also been reported in the cyst walls.

The periapical pocket cyst contains an epithelium-lined pathological cavity that is open to the root canal of the affected tooth. As mentioned previously, such lesions were originally described as 'bay cysts'. It has been postulated that biologically, a pocket cyst constitutes an extension of the infected root canal space into the periapex. The epithelial collar forms an 'epithelial attachment to the root surface so as to seal off the infected root canal and the micro-cystic lumen from the periapical milieu and the rest of the body. The presence of microorganisms at the apical foramen attracts neutrophilic granulocytes by chemotaxis into the microlumen. However, the pouchlike lumen biologically outside the body milieu acts as a 'death trap' to the externalized neutrophils. As the necrotic tissue and microbial products accumulate, the sac-like lumen enlarges to accommodate the debris, forming a voluminous diverticulum of the root canal space into the periapical area.



Radiographically most radicular cysts appear as round or pear-shaped unilocular radiolucent lesions in the periapical region. The cysts may displace adjacent teeth or cause mild root resorption. Radiographically, distinguishing between a granuloma and a cyst is impossible, although some say that if the lesion larger than 2 cm is more likely to be a cyst. It is believed if an osteolytic periapical lesion is more than 2 cm² in diameter; it may be a cystic lesion. In addition, if a well demarcated periapical osteolytic lesion is bordered by a thin rim of cortical bone, there is a strong probability that the lesion is a cyst.

The gross specimens may be spherical or ovoid intact cystic masses, but often they are irregular and collapsed. The walls vary from extremely thin to a thickness of about 5 mm. The inner surface may be smooth or corrugated. Yellow mural nodules of cholesterol may project into the cavity. The fluid contents are usually brown resulting from the breakdown of blood and when cholesterol crystals are present, they impart a shimmering gold or straw colour.

Radicular cysts are a pathologic cavity partially or completely lined by non-keratinized stratified squamous epithelium in an area of apical periodontitis of a tooth with infected necrotic pulp. These linings may be, discontinuous in part and range in thickness from one to 50 cell layers. The majority are between six and 20 cell layers thick. The epithelial linings may be proliferating and show arcading with an intense associated inflammatory process or be quiescent and fairly regular with a certain degree of differentiation arcading with an intense associated

inflammatory process or be quiescent and fairly regular with a certain degree of differentiation. In active infection, the epithelial lining of the cysts is thick and irregular, and infiltrated with inflammatory cells. In inactive infection, the epithelial lining is thin and regular, and has minimal infiltration of inflammatory cells [3, 7, 8].

Endodontic diagnosis is dependent on clinical and radiographic examinations. Periapical radiographs, orthopantomograms and cone beam computed tomography (CBCT) images represent important radiographic investigations in the detection and diagnosis of inflammatory, dysplastic and neoplastic periapical diseases.

CASE REPORT

A 25 years old patient visited the dept. of oral and maxillofacial surgery with chief complaint of swelling on the lower lip region since 1.5 months. There was no associated pain. However, discolouration of 33, 32, 31, 41, and 42 were present.

On extraoral examination, there was no significant facial asymmetry or deformity noted. On intraoral examination, localized ovoid swelling was noted in the mandibular anterior vestibular region from 33 to 42 region which was soft, nontender, non-erythematous, compressible, smooth with absence of local rise in temperature. The mentioned teeth were non-tender on percussion. There was no mobility of those teeth and neither was there any pus discharge.



Fig-1: Preoperative intraoral view of the lesion

The orthopantomogram revealed an ovoid well-defined unilocular radiolucency with sclerotic border involving periapical region from distal side of 33 to mesial side of 42 upto 6 mm above the lower border of the anterior mandible. The size of the radiolucency

was 21 X 12 mm. There was radiopacity noted in the root canals of 33, 32, 31, 41, and 42 suggestive of previous obturation. There was no evidence of any root resorption of any of the involved teeth.

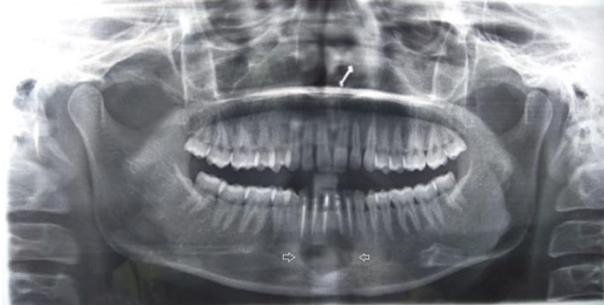


Fig-2: Orthopantomogram of the patient preoperative

The lesion was surgically enucleated in toto under local anaesthesia (bilateral mental nerve block along with lingual nerve block). The lesion was approached via a vestibular incision from distal side of 33 to distal side of 43, and a four cornered mucoperiosteal flap was reflected. The thin labial cortical bone was trephined and the cyst was enucleated in toto.

The bony cavity was thoroughly rinsed with normal saline. The teeth 33, 32, 31, 41 and 42 were retrogradely restored with Glass Ionomer Cement. Then about 20 ml of blood was withdrawn and platelet- rich fibrin (PRF) was prepared. The freshly prepared PRF was packed inside the bony cavity, and the mucoperiosteal flap was returned to its original position and primarily closed by sling suturing technique using 3-0 polyglactin suture material.



Fig-3: Mucoperiosteal flap reflection



Fig-4: The cyst within the bone



Fig-5: The cyst was enucleated out of the bone in toto followed by smoothing of the surrounding bone

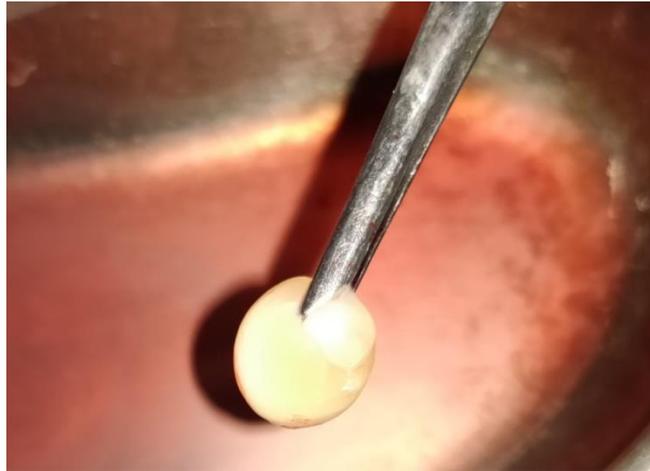


Fig-6: The freshly prepared platelet-rich fibrin



Fig-7: Interrupted sling suturing done to close the surgical wound

Endodontic treatment of the mentioned teeth was completed successfully thereafter. Histopathological report confirmed the diagnosis as radicular cyst of anterior mandible. The patient was recalled several times for follow-up and underwent uneventful healing of the surgical wound.

DISCUSSION

Periapical cyst is most frequently found and was classified as inflammatory. Pulpal necrosis leading to periapical inflammation appears to be the most common etiology of periapical cyst. Radicular cysts, with an incidence of 0.5-3.3% of the total number in both primary and permanent dentition. Occur more commonly between third and fifth decades, more common in males than in females, and more frequently

found in the anterior maxilla than other parts of oral cavity [1, 2, 4].

The treatment options for radicular cyst can be conventional nonsurgical root canal therapy when lesion is localized or surgical treatment like enucleation, marsupialization or decompression when lesion is large. The choice of treatment may be determined by some factors such as extension of the lesion, relation with noble structures, evolution, origin, clinical characteristics of the lesion, cooperation and systemic condition of the patient. Treatment However in large lesions the endodontic treatment alone is not efficient and it should be associated to a decompression or a marsupialization or even to enucleation.

Post-surgically, the formation of blood clots initiates the healing and regeneration of hard and soft tissues. Wound healing is an organized process involving leukocyte and platelet activity. For this process to work effectively, the platelets play a vital role. Growth factors present in blood platelets are important to guide the regenerative cells to the healing area.

One of the latest achievements in dentistry is the use of platelet-rich fibrin (PRF) for the improvement of reparation and regeneration of the soft and hard tissues after different surgical procedures. Platelet-rich fibrin is concentrated platelets in a small volume of plasma. During platelet degranulation, many biologic active substances are released, which participate in the primary haemostasis and help the following reparation and regeneration of the soft and hard tissue. The proteins derived from platelets include platelet-derived growth factor (PDGF), transforming growth factor (TGF- β), vascular endothelial growth factor (VEGF), and epidermal growth factor (EGF). Plasma contains certain natural growth factors in the name of insulin-like growth factor (IGF) and hepatocyte growth factor (HGF).⁷ The ability of PRGF in accelerating soft and hard tissue healing has stimulated the research of its clinical applications in various areas of oral and maxillofacial surgery, which includes healing of extraction sockets, including impacted tooth, implantology, cleft lip and palate, ulcer management, and bisphosphonate-related osteonecrosis of the jaw. The two most important of these growth factors are platelet-derived growth factor or PDGF and transforming growth factor- β or TGF- β . Numerous studies, including some dental research have shown that PDGF, TGF- β , and IGF-I are found in PRF, and because of their impact on wound healing, the use of these factors has led to promising results.

PRF is efficacious clinically and radiologically in treatment of intrabony defects after enucleation of various periapical lesions, where complete bone regeneration is seen within 6th months postoperatively. The use of PRF in intraoral bony defects after cystic enucleation promotes faster regeneration because of gradual release of growth factors lodged in the fibrin matrix [9, 10].

CONCLUSION

Treatment of the cyst is still under discussion. Various treatment option has been suggested depending on the size and location of cyst. Uneventful and enhanced wound and bone healing is desirable and critical in ascertaining quality of life after surgery.

Accelerating the healing process makes the treated area to be less sensitive to external factors (mechanical, bacterial and chemical), at the same time influencing the aesthetic result and post-operation patient comfort. PRF speeds up the healing process, as

shown in our case report in which PRF is effective in the healing of the bone defect resulting from cyst enucleation in a faster rate, both clinically and radiologically.

This case presents successful surgical treatment of large radicular cyst of anterior mandible along with endodontic treatment.

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