

Radicular cyst

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ABSTRACT

The main aim of this review was to give a detailed view of the etiology, pathogenesis, clinical features, diagnosis, differential diagnosis, and treatment options for the radicular cyst. Pulpal necrosis occurs as a consequence of caries affecting the tooth along with periapical inflammatory response will result in radicular cyst. This review gives a detailed view of the cyst. The radicular cyst comes under classification of inflammatory cysts. Radicular cysts show similar radiographical characteristics in relation with pre-existing chronic periapical periodontitis, making it difficult to diagnose. This arises due to inflammation from the epithelial residues found along the periodontal ligament mostly following the pulpal necrosis. The radicular cyst spontaneously heals after extraction or root canal treatment. Various different literatures have debated about the treatment of the radicular cyst. Thus, in our review, we have detailed about the various aspects of radicular cyst.

KEY WORDS: Cyst, Periapical, Pulp, Radicular

INTRODUCTION

The maxillofacial skeleton is a complex region where the most frequent tissue-degrading lesions such as odontogenic cysts occur. Based on the etiology, these cysts are classified into inflammatory and developmental types. The dentigerous cyst, primordial cyst, gingival cyst, and eruption cyst come under developmental type, whereas the inflammatory cysts include lateral periodontal cysts and radicular cysts.^[1] According to the World Health Organization, the cysts of the jaw of inflammatory origin have one type, the radicular cyst, whereas Main and Craig believed that there are two other variants of inflammatory cysts, paradental cyst and inflammatory collateral cyst.^[2] The radicular cyst arises due to inflammation from the epithelial residues found along the periodontal ligament mostly following the pulpal necrosis.^[3-6] Of all the cysts affecting the jaw, about 52–68% are the radicular cysts.^[3] About 42–44% of all the apical lesions were found to be the radicular cysts.^[4] They have been found to be more in male population than the female population, and

also highest incidence among the patients in the third decade of life. Anatomically, the radicular cysts occur in all tooth-bearing sockets of the jaws but have been found to be more common in the maxillary region than the mandibular region.^[3,4]

ETIOLOGY

The chemical, physical, or bacterial injuries result in pulpal necrosis which is later followed by stimulation of epithelial cell rests of Malassez, which are present in periodontal ligament region. There may or may not be the presence of infection in the cyst.^[7] Actinomyces organisms have been found most commonly from the cyst whenever the infection is present. About 75% Gram-positive and 24% Gram-negative microorganisms are mainly found in the root canal, among which Streptococci are predominant and also other Gram-positive organisms such as Staphylococci, Cornebacterium, yeast, and others are found in the canal.^[8] The Gram-negative organisms which were also found are Spirochetes, Bacteroides, Neisseria, Pseudomonas, Fusobacterium, and others. Obligate anaerobes were found in the periapical lesions such as radicular cysts. Along with obligate anaerobes, hemolytic and non-hemolytic streptococci were also found in long-standing cases of periapical pathology.

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Culture of root canal materials was done using the following medias: ^[9,10]

- Trypticase soy broth with 0.1% Agar (tryptone soya agar)
- Glucose ascites broth
- Brain heart infusion broth with 0.1% Agar.

PATHOGENESIS^[11]

Radicular cysts are formed in the endodontically involved teeth in areas of apical periodontitis. Therefore, the interaction between the osteoblasts, osteoclasts, and osteocytes, as well as the RANK-RANKL/OPG system, results in the immunity-inflammatory process showing resorption of periapical alveolar bone,^[12,13] before a radicular cyst can be formed. It is suggested that during apical periodontitis, the growth factors and inflammatory cytokines released can stimulate the epithelial cell rests of Malassez, which may form a cyst by proliferation.^[10-12] Apparently, some of the epithelial cells in the islands or nests of epithelial cell rests of Malassez must maintain the regenerative potential of stem cells because mature epithelial cells are not able to undergo intensive proliferation.^[12] Similar to the basal cells of most epithelium, the basal cells of epithelial cell rests of Malassez may also behave like stem cells. The inflammatory cytokine and growth factors are the appropriate signals which stimulate the basal cells to proliferate, thereby forming a cyst. Although several theories, nutritional deficiency,^[13] abscess formation,^[14] and merging of proliferated epithelial islands,^[15] have been proposed as possible scenarios of cyst formation, the exact mechanism of cyst formation is still not clear.

Conveniently, the pathogenesis of radicular cyst is divided into three phases.^[16] They are as follows:

- Phase of initiation
- Phase of cyst formation
- Phase of cyst enlargement.

PHASE OF INITIATION

The epithelial lining of these cysts is generally found to be derived from the epithelial cell rests of Malassez in periodontal ligaments. In certain cases, the epithelial lining may be derived from the oral epithelium from fistulous tract; when periapical lesion communicates with sinus wall, the respiratory epithelium of maxillary sinus; oral epithelium proliferating apically from periodontal pocket.^[17]

Various reasons have been said to stimulate the epithelial cells, but the exact mechanism is not clear. The dead pulp having some products or the inflammation in periapical granuloma may initiate the process as well as evoking an inflammatory reaction. The activation of cell rests of Malassez may

be because of the evident local changes in supporting connective tissue.

PHASE OF CYST FORMATION

The cavity becomes lined by proliferating epithelium is the process of phase of cyst formation. There are two main theories regarding this process, which are as follows: ^[18]

1. The initial reaction which results in the cyst formation as suggested by the most widely accepted theory is the proliferation of epithelial rests in the periapical area involved by granuloma.^[19] By the division of cells on the periphery corresponding to the basal layer of surface, epithelium results in increasing of size of epithelial mass as the proliferation continues to occur. The cells found in the central portion of the mass fail to obtain diffident nutrition, as they become separated further and further from nutrition in comparison with basal layer, thus eventually degenerating and becoming necrotic and liquifying. Thus, a cavity filled with fluid lined by epithelium is created.
2. Another theory suggests a pre-existing cavity formed through focal necrosis and degeneration of connective tissue in periapical granuloma,^[20] results in cyst formation by proliferation of epithelium which lines the cavity. However, near the area of necrosis finding an epithelium or epithelial proliferation is not common.

PHASE OF CYST ENLARGEMENT

There is basic evidence from the experimental work that increase in the size of cyst is contributed by the osmosis. Investigators have said that the gamma globulin level in the fluids of radicular cysts is high by almost more than half to patient's own serum. The contribution of high osmotic pressure of cystic fluid on cyst walls by the hyaluronic acid and plasma protein exudate as well as products of cell breakdown causes the resorption of bone and enlargement of cyst.

CLINICAL FEATURES

The erosion of the floor of the maxillary sinus is caused by the expansion of the cyst. The expansion starts as soon as it enters the maxillary antrum and it occurs faster as there is space available for expansion inside the sinus.^[12] There is shooting pain on tapping the affected teeth which is characteristic feature of pulpal infection and is of almost importance in diagnosis. The frequency of this cyst is found to be about 52.3% of cystic jaw lesions and is most common cystic jaw lesion.^[8] Large quantity of cases are found to have a gradual decline after their predominant activity in the 4th and 5th decades of life. It is most predominant in males than females where the males show 58% and females show 42%.^[9] The white

patients have a frequency of about twice that of black patients showing a higher prevalence. It is commonly found in the maxilla in about 60% of cases.^[7] The maxillary anterior region is the most common site of occurrence even though it is found to be present in all tooth-bearing areas of the jaws. The usual offending teeth are the upper lateral incisors and dens in dente. The apices of involved teeth are the most common sites. In certain cases, it may also be found in the lateral accessory root canals.

GROSS FEATURES

Ovoid or spheroidal intact cystic masses but often collapsed and irregular are seen in the gross specimen. The thickness of the walls may vary from extremely thin to about 5 mm. The inner surface of the cavity may show corrugated yellow mural nodules of cholesterol may project into the cavity or the surface may be smooth. The fluid may be orange gold or straw color due to the presence of cholesterol crystals or also may be brown colored from the breakdown of blood.

Differential diagnosis:

- Periapical granuloma
- Traumatic bone cyst
- Periapical scar
- Periapical cemental dysplasia
- Periapical surgical defect
- Globulomaxillary cyst
- Aneurysmal bone cyst
- Mandibular infected buccal cyst
- Periapical cemento-osseous dysplasia.

TREATMENT^[21]

Endodontic Treatment

Once the causative agents are removed, the peripheral lesions including radicular cyst are eliminated. Surgical intervention is not required as the major number of radicular cysts resolve following a root canal treatment. The radicular cyst gets converted into a granuloma due to a transitory acute inflammation resulting in destruction of epithelial lining, and this occurs when a file or other root canal instrument is inserted beyond the apical foramen by 1–2 mm, thereby leading to resolutions of the cysts.

Surgical Treatment^[22]

Enucleation – the tooth is either preserved by root canal treatment with apicoectomy or can be extracted. To gain adequate access, a mucoperiosteal flap over the cyst should be raised and a window is created in the bone. From its bony wall, the cyst is separated carefully and the entire cyst should be removed intact. The edges must be smoothed off, controlling the free bleeding is done, and the cavity is there for irrigated

for the removal of debris. The sutures are placed once the mucoperiosteal flap is replaced.

Marsupialization – in this procedure, the cyst is opened similarly like enucleation, but the epithelial lining has to be sutured to mucous membrane at the margins of the opening. The main objective of the procedure is to give a self-cleaning cavity, which eventually becomes an invagination of oral tissues.^[23] The cavity is packed initially with a ribbon gauze and once the margins are healed an extension of denture or a plug is made to close the openings. By the regrowth of the surrounding tissues and the restoration of normal contour of that part, the cavity usually closes. However, there are high chances of closing of produce and reformation of the cyst.^[24] The temporary decompression of exceptionally large cyst where fracture of the jaw is the risk factor is the main outcome of this procedure. The cyst can be enucleated when adequate new bone is formed.

PROGNOSIS

The prognosis depends three main factors. They are as follows:

- The particular tooth
- Extent of bone destroyed
- Accessibility for treatment.

The expected complications are as follows:^[25]

- Carcinomas/neoplasticism changes: Epidermoid carcinoma or squamous cell carcinoma may occasionally arise from epithelial lining of radicular cyst
- Pathological jaw fracture: This may be caused when a very rare case of radicular cyst presents in the posterior region where the cyst have completely eroded the bone specially
- Secondary infection: The cyst may create further complications and may get secondarily infected.

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