



Radicular Cyst

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Introduction

Radicular cysts are the most common cystic lesions affecting the jaws. They are most commonly found at the apices of the involved teeth, however they may also be found on the lateral aspects of the roots in relation to lateral accessory root canals. Quite often a radicular cyst remains behind in the jaws after removal of the offending tooth and this is referred to as a residual cyst (1-3).

Epidemiology

Radicular cysts are the most common of all jaw cysts and comprise about 52% to 68% of all the cysts affecting the human jaws (2). Actual prevalence of cysts is only about 15% of all apical periodontitis lesions (2,3). Their prevalence is highest among patients in their third decade of life, and higher among men than women (3).

Pathogenesis

Radicular cysts arise from the epithelial residues in the periodontal ligament as a result of inflammation. They generally result due to pulpal infection following dental caries. Bacteria from the gingival sulci or periodontal pockets have been suggested to reach the root canals of these teeth through severed periodontal blood vessels (4). Pulpal infection can also occur through exposed dentinal tubules at the cervical root surface, due to gaps in the cemental coating (5). Microbes have also been claimed to 'seed' in the necrotic pulp via the blood circulation (anachoresis) (6). 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. 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 defenses consisting of several classes of cells, intercellular messengers, antibodies, and effector molecules. The microbial factors and host defense forces encounter, clash with, and destroy much of the periapical tissue, resulting in the formation of various categories of apical

periodontitis lesions (5). Periapical cysts are a direct sequel to chronic apical periodontitis, but not every chronic lesion develops into a cyst. 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 (2).

Pathogenesis of true cysts: The periapical true cyst may be defined as a chronic inflammatory lesion at the periapex that contains an epithelium-lined, closed pathological cavity. The pathogenesis of true cysts has been described by various authors (5,7). An apical cyst is a direct sequel to apical granuloma, although a granuloma need not always develop into a cyst. Due to still unknown reasons, only a small fraction (<10%) of the periapical lesions advance into true radicular cysts (8). The pathogenesis of the true cyst has been described in three phases (2, 3, 9). During the first phase, the dormant cell rests of Malassez begin to proliferate as a direct effect of inflammation (2, 3), probably under the influence of bacterial antigens (2, 3, 10) epidermal growth factors (10, 11), 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 (2, 3).

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

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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 (3). However, the fact that an apical pocket cyst with lumen open to the necrotic root canal can become larger suggests against osmotic pressure as a potential factor in the development of radicular cysts. Further, there is increasing evidence in support of a molecular mechanism for cyst expansion. 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 (10, 12).

Pathogenesis of pocket cysts: 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 microluminal space becomes enclosed in a stratified squamous epithelium that grows and forms an epithelial collar around the root tip. 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 pouch-like 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 (3). It has been pointed out that from the pathogenic, structural, tissue dynamic, host-benefit and protection stand points, the epithelium-lined pouch-like extension of the root canal space of such lesions has much in common with a marginal periodontal pocket (9).

Site of Presentation

These cysts can occur in the periapical area of any teeth, at any age but are seldom seen associated with the primary dentition (2, 3). Anatomically, the apical cysts

occur in all tooth-bearing sites of the jaws but are more frequent in maxillary than mandibular teeth. In the maxilla, the anterior region appears to be more prone to cyst development whereas in the mandible the radicular cysts occur more frequently in the premolar region (3, 8).

Clinical Presentation

Most of the radicular cysts are symptomless and are discovered when periapical radiographs are taken of teeth with non-vital pulps. Patient often complains of slowly enlarging swellings. At first the enlargement is bony hard but as the cyst increases in size, the covering bone becomes very thin despite subperiosteal bone deposition and the swelling then exhibits 'springiness'. Only when the cyst has completely eroded the bone, there will be fluctuation. In the maxilla there may be buccal or palatal enlargement whereas in the mandible it is usually labial or buccal and only rarely lingual (3). Pain and infection are other clinical features of some radicular cysts. It is often said that radicular cysts are painless unless infected. Some patients with these lesions, however, complain of pain although no evidence of infection is found clinically and no evidence of acute inflammation is seen histologically after the cyst has been removed. Likewise, some patients have clinically infected and histologically inflamed cysts which are not painful (8, 13, 14). 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 (3). Quite often, more than one radicular cyst may be found in a patient. (Fig-1)

Radiological Features

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 (3, 13, 14). (Fig-2)

Pathological Features (Fig-3)

Gross Features: 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 (3).

Microscopic Features: Almost all radicular cysts are lined wholly or in part by nonkeratinized stratified squamous epithelium. 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



Fig 1. Radicular Cyst Showing Swelling in Relation to Periapical Region

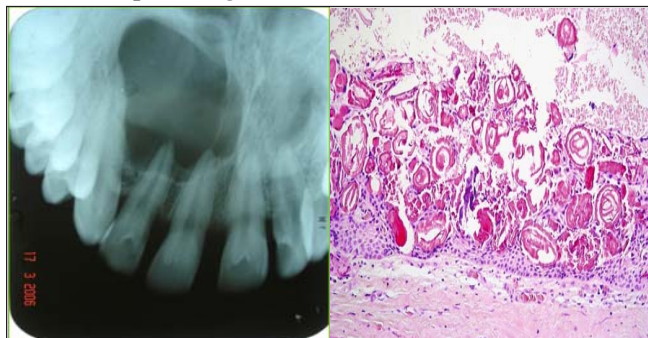


Fig 2 & 3. Radiographic Presentation of Radicular Cyst & Histological Showing Non Keratinized Stratified Squamous Epithelial Lining of Varying Thickness Containing Rushton Bodies

arcading with an intense associated inflammatory process or be quiescent and fairly regular with a certain degree of differentiation. The inflammatory cell infiltrate in the proliferating epithelial linings consists predominantly of polymorphonuclear leucocytes whereas the adjacent fibrous capsule is infiltrated mainly by chronic inflammatory cells (3). In approximately 10 per cent of periapical cysts, hyaline bodies (often referred to as Rushton's hyaline bodies) are found in the epithelial linings. Such bodies within the epithelial lining are characterized by a glassy pink (hyalinized) appearance. The origin of such bodies is believed to be related to previous hemorrhage within the inflamed cyst wall (3, 15, 16).

Treatment and Prognosis

The treatment of radicular cyst, as a disease of root canal infection, consists of eradicating microbes or substantially reducing the microbial load from the root canal and preventing re-infection by orthograde root filling. The treatment has a remarkably high degree of success. Nevertheless, endodontic treatment can fail. Most failures occur when treatment procedures, mostly of a technical nature, have not reached a satisfactory standard for the control and elimination of infection. Even when the highest standards and the most careful procedures are followed, failures still occur. This is because there are root canal regions that cannot be cleaned and obturated with existing equipments, materials,

and techniques, and thus, infection can persist. In very rare cases, there are also factors located within the inflamed periapical tissue that can interfere with post-treatment healing of the lesion (5). Lesions that fail to resolve with such therapy may be successfully managed by extraction of the associated nonvital teeth and curettage of the epithelium in the apical zone. Alternatively, a root canal filling may be performed in association with an apicoectomy to permit direct curettage of the cystic lesion (3, 15, 16).

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