Etiopathogenetic Aspects of Radicular Cysts

SUMMARY
The aim of this study was to get more information on etiopathogenetic mechanism of radicular cysts and eventual effect of local factors in the evolution and prognosis of the cystic lesion. For this purpose we performed radiological, pathohistological and microbiological examination. 50 patients with diagnosis of radicular cysts were examined. The results obtained from the examined group were compared with the controls (35). Clinical investigation was based upon dental history and clinical data, supplemented with radiological, microbiological and pathohistological examination.

Residual cysts were dominantly present in males (66%), compared to females, being predominantly present in the third and forth decade of life. Cysts were more frequent in the maxilla compared to the mandible. The prevalence of residual cyst in the upper jaw was 66%. Clinical findings were not sufficient to confirm the presence of cystic disorder; radiological data offered more precise data, showing marked transparency with a sclerotic ring in 86% of the cases. Several bacteria species were found in the study group: Streptococcus viridans in 38%, Staphylococcus epidermidis in 16%, Streptococcus β hemoliticus in 14%. Pathohistological investigation revealed a dominant chronically exacerbated infiltrate (56%), compared to common chronic infiltrate. In relation to epithelium, squamous epithelium was dominant (72%), while cylindrical epithelium was very rare.

Although the obtained results provide data based evidence of the nature of radicular cysts, many opened questions are still left behind and further investigation is necessary to obtain reliable answers.

Keywords: Cyst, jaw; Cyst, radicular; Oral Surgery

Introduction

A cyst is a pathological cavity filled with fluid, semi-fluid or gaseous contents, which is not created by accumulation of pus. It is frequently lined by epithelium. Because of the severity of clinical features and many other reasons, radicular cysts are paid rather high importance, especially for complications from delayed or inadequate treatment, endangering general health and presenting potential foci. From etiopathogenetic aspect of view, it is accepted that the onset of inflammatory cysts, mostly radicular, could be traced into the root canal, which provokes initiation of proliferate processes1. Dominant role in cyst pathogenesis belongs to immune-pathological reactions. Suzuki5, as well as many others, proved that appearance and development of inflammatory cysts are conditioned by immune-pathological reactions. Petrovic6 suggests that cyst continues to grow independently and progressively after micro-cystic lesion formation, which is conditioned by different factors. Cystic growth cannot be arrested even after employing therapeutic procedures that influence its aetiology.

Experimental evidence point out periapical lesion immune components to be responsible for bone damage17. Torabinejad points that lost of bone results from the presence of complementary cascades, prostaglandin synthesis and many other neutrophil granulocytes17.

Analysis of inflammatory cell infiltrate in periapical lesions and surrounding tissue supports the importance of complex immune reactions. Literature data differ, although there are certain agreements. Apart from immune mechanisms in radicular cysts etiopathogenesis, many
other factors are involved, such as infection, trauma and allergy. The aim of this study was to determine predominant microorganisms and possible influence of local etiopathogenic factors to development and prognosis of cystic lesion, as well as obtaining data on surrounding tissue damage.

**Material and Methods**

We investigated 50 individuals with the diagnosis of radicular cysts and 35 healthy persons, representing a control group. Each of the patients was examined at Clinic for Oral Surgery, Faculty of Dentistry in Skopje. Supplemental examinations were carried out at the Institute of Microbiology and Parasitology, Institute of Pathology, and Institute of Radiology.

Data of dental histories, radiographic, microbiological and pathohistological findings in the study group were compared to those of the controls. Radiography of the patients of the study group were compared prior and after therapy (the interval being 4-6 months). Microbiological investigation included determination of the presence of aerobic and anaerobic bacteria in the cystic lesion. Pathohistological examinations were carried out on samples of the cystic wall, stained with HE, Gimza and van Gizon.

**Results**

The results are divided into 4 groups: clinical, pathohistological, microbiological and radiographic; surrounding tissue damage were noted, too.

According to sex distribution, it was found that radicular cysts were more frequent in males (66%) compared to females (34%). There was prevalence of maxillary (66%) related to mandibular cysts (34%). Cysts were predominantly noticed in the third and forth decade of life.

Pathohistological investigation comprised inflammatory infiltrate, epithelium and granulation tissue examination. The cystic lesion was mostly lined by stratified squamous epithelium, with collagen tissue beneath (Fig. 1). Desquamation was registered in epithelial layer, with granulation tissue underlying it; cholesterol crystals and haemosiderin pigment could also be noticed (Fig. 2). Figure 3 shows numerous cholesterol crystals and inflammatory infiltrate revealing chronic traits and intra and intracellular haemosiderin pigment. Chronic exacerbation infiltrate prevailed (56.0%), but without statistical significance. Squamous epithelium was registered in 72% of the specimens (Tab. 1), which was statistically significant. Cylindrical epithelium was found in 9 cases (18%). As to granulation tissue, it was present in 43 cases (86%).

Figure 1. Pathohistological section of radicular cyst: (1) stratified squamous epithelium; (2) collagen tissue (HE; x 40)

Figure 2. Pathohistological section of a radicular cyst wall: (1) granulation tissue; (2) cholesterol crystals; (3) haemosiderin pigment enlargement (HE; x 40)

Figure 3. Pathohistological section of a radicular cyst wall: (1) cholesterol crystals; (2) haemosiderin pigment, lose and in macrophages; (3) inflammatory infiltrate (HE; x 200)
Microbiological investigation showed presence of different bacteria. Colonies of *Staphilococcus aureus* (Fig. 4), and *Streptococcus alpha haemoliticus* (Fig. 5) prevailed. Negative optohin test revealed presence of *Streptococcus viridans*. The incidence of different bacteria in the radicular cyst content is presented in table 2. The obtained results confirmed the presence of normal oral flora in the cystic content and that infection penetrates through the root canal up to the periapical space.

Radiographic findings are shown in figures 6 and 7. Cystic lesions were oval and clearly bordered from its surrounding structures by sclerotic ring. Statistical analysis of x-ray findings showed predominance (86%) of well defined bordered marginal radiolucency with a sclerotic ring. Based on the X-ray findings at 6 month interval postoperatively, complete healing was found in 70% of the cases.

Results related to the surrounding structures damage, obtained from X-rays analysis and intraoperative findings, pointed out the consecutive damage of the maxillary sinus (8%), mandibular canal (2%) and incisive canal (2%).

**Table 1. Distribution of cell type in epithelial linings**

<table>
<thead>
<tr>
<th>Epithelial lining</th>
<th>N</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ø</td>
<td>4</td>
<td>8.0</td>
</tr>
<tr>
<td>Squamous epithelium</td>
<td>36</td>
<td>72.0</td>
</tr>
<tr>
<td>Cylindrical epithelium</td>
<td>9</td>
<td>18.0</td>
</tr>
<tr>
<td>Other</td>
<td>1</td>
<td>2.0</td>
</tr>
</tbody>
</table>

**Table 2. Microbiological findings in radicular cysts**

<table>
<thead>
<tr>
<th>Microbiological finding</th>
<th>N</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ø</td>
<td>7</td>
<td>14.0</td>
</tr>
<tr>
<td>Streptococcus β haemoliticus</td>
<td>6</td>
<td>12.0</td>
</tr>
<tr>
<td>Staphylococcus epidermidis</td>
<td>7</td>
<td>14.0</td>
</tr>
<tr>
<td>Streptococcus pneumonia</td>
<td>4</td>
<td>8.0</td>
</tr>
<tr>
<td>Staphylococcus aureus</td>
<td>6</td>
<td>12.0</td>
</tr>
<tr>
<td>Streptococcus viridans</td>
<td>17</td>
<td>34.0</td>
</tr>
<tr>
<td>Streptococcus viridans and Streptococcus β haemoliticus</td>
<td>2</td>
<td>4.0</td>
</tr>
<tr>
<td>Staphylococcus epidermidis and Streptococcus pneumonie</td>
<td>1</td>
<td>2.0</td>
</tr>
</tbody>
</table>
Discussion

Dilemmas are still present relating possible etiologic and pathogenic mechanisms responsible for the genesis and development of cystic formations. Available literature reveals multiple potential etiologic factors like microbiologic, allergic, immunologic, etc. Majority of authors came to the conclusion that all of these factors participate in their own unique and distinctive way in expression and development of cysts, regardless of their nature.

Due to the poor symptomatology and, frequently, only incidental diagnosis of cystic lesions in the upper and lower jaw, we made an effort to systemize the frequency of jaw cysts related to age, sex, and localization. Like many others, we provided evidence that radicular cysts are more frequently present in the upper than in the lower jaw; however, this finding disagrees with the results obtained in the Myoung’s study.

It seems that the ongoing processes inside the cystic lesion can be classified as exacerbating chronic infiltrate. According to the acquired data from the literature we consider that epithelium is transformed into a cystic capsule constituted from infiltrative granulation tissue with a mixture of chronic inflammatory cells. The infiltrate does not have the same intensity along the whole cystic capsule. Cholesterol crystals and haemosiderin are frequently found in the cystic lumen, and Rast’s bodies in 10% of the cases. This is in agreement with findings of Gordeeff and Teronen, but is not with those of Yamamoto.

Our results show that the majority of cysts are coated with the stratified squamous cell epithelium; its morphology depends on the degree of inflammation in the connective tissue layer surrounding it. In the presence of inflammation, epithelium expresses typical proliferations toward connective tissue, which gives the impression of being like arcades. Inflammation alters epithelial morphology of the cysts concerning its viscosity and density. Severe inflammation or infection can cause partial or total epithelial necrosis, going up to the total extinction. In the absence of inflammation, epithelium tends to decrease its size even without the presence of sub-basal proliferations, but with over expressed hyalinization, which is manifested through the appearance of hyaline bodies. Similar findings described Shear and Sokolović, but not Redman. Petrović states that fibrous coating of the radicular cysts, in majority of cases, consists of granulation tissue affluenty infiltrated with characteristic chronic inflammatory cells.

Numerous studies underline the connection between microbiological findings and the occurrence of inflammatory cysts. In reference to this, Ricucci et al. point out the fact that bacterial presence inside the cyst is similar to the findings from the necrotic tissue of the root canals and periapical lesions. In reference to the microorganism participation in etiologic and pathogenic events in the inflammatory cysts, Hrvacanin confirms the presence of Alpha haemolytic streptococcus, Streptococcus pneumoniae, and Staphylococcus epidermidis in cystic lesions.

Our results suggest that accurate diagnosis cannot be determined only on the basis of clinical findings. Higher accuracy can be achieved if we add X-rays to the clinical examination. Consequently, more precise information about the presence of the cystic lesion and its type can be provided.

The majority of radiographic changes inside the surgical field postoperatively, including spear like or trabecular collection, can be detected between the first and fourth month after the cyst removal, and complete bone healing is verified after 4 months postoperatively. Therefore, it is recommended that optimal time for the radiographic control and early detection of residual lesions is 4 months after surgery.

Jaw cysts are frequently asymptomatic; consequently, they can cause damage of the surrounding anatomic and morphologic structures, such as maxillary sinus, mandibular canal, incisive canal and adjacent teeth. Massive maxillary cysts can expand toward the maxillary sinus and, after complete resorption of the cystic wall, they can fill up the sinus. As far as the mandibular canal is concerned, during the expansion of the odontogenic cysts the neurovascular bundle is relatively well protected by the compact structure of the lower jaw, and also by the specific cystic growth pattern in this region.

We might presume that, under the influence of numerous chronic inflammatory stimulations, epithelial cells become to change their condition of metabolic inactivity increasing their own activity. As a consequence, the volume of cytoplasm increases mutually with accumulation of neutral lipids. Inflammation is especially influential during the mitotic cell division, and subsequently, the lesion becomes larger. Concentration discrepancies between oxygen and carbon dioxide in tissues, as well as the decreased pH values, are substantial for cystic formation. From this standpoint, contemporary interpretations about the role of microorganisms, as well as the aggressive bone resorption factors, undoubtedly confirm the key role of inflammation as the strongest initiator and inducer radicular cysts genesis.
References


Correspondence and request for offprints to:
Dr. Oliver Dimitrovski
Jurij Gagarin 23/2-4
1000 Skopje
Macedonia
e-mail: dr.oliverdimitrovski@mail.net.mk