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Editorial

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Mário dos Anjos Neto Filho
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DECOMPRESSION OF DENTIGEROUS CYST IN MAXILLA

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ABSTRACT

Dentigerous cysts are considered benign bone diseases associated with odontogenic etiology factors. In most cases, it is seen there is a big highlight in individuals of the male gender, among their first ten years of life. The differential diagnosis of this disease is in the greater amount of time, harder to be reached with common everyday radiography, what makes it way more difficult to obtain the final diagnosis in the dental clinics in the patient's first appointment. Thus, this paper aim to show a Case Report in which the multidisciplinary planning and action were very important to the diagnosis and treatment of the dentigerous cyst located in the maxilla. This way, the limitation of the radiographic images, can many times bring you to many interpretations of the differential diagnosis, that is why exams such as the cone bean, which is a computerized light bean, are so important. In conclusion, even though the dentigerous cysts are a benign disease, sometimes the place and extension of the case, may cause great damage to the patient, that, if treated in the incorrect way may cause deformity that are very difficult to solve. The diagnosis methods like the bean cone and the conservative approach through decompression were extremely important in this case treatment.

KEYWORDS: Dentigerous Cyst, decompression, Cone Bean tomography.

1. INTRODUCTION

The dentigerous cysts are originated from residual epithelial remnants that in order stimuli undergo chemical, traumatic and inflammatory, lead to the follicle degeneration, resulting in a cyst. Considered a benign condition, has no symptoms, and their development is through the accumulation of fluid located between the remaining enamel has formed below the tooth crown, having the cementum enamel junction as a point of union. Is associated with delayed or impacted teeth erupting maxillary[1,2,3].

These tissues formation are considered the second most common odontogenic cyst, with 24% of all true cysts of the oral cavity. Observed more frequently in Caucasian men, in the first and second decade of life, is located predominantly in the jaw. According to the distribution in the arcades is in the lower third molars, canines, second premolars and upper third molars[4,5].

The information obtained during anamnesis, clinical examination and radiographic studies are insufficient to establish the diagnosis, because some diseases have similar radiographic features. Among the possible hypotheses can be found: ameloblastoma, epidermoid carcinoma, mucoepidermoid carcinoma, inflammatory periapical cyst, odontogenic keratocyst, aneurysmal bone cyst, one hyperplastic follicle and odontogenic fibroma[4].

The radiographic characteristics presented are: radiolucency, unilocular and bounded image; may be involving an included element, resembling others pathologies. Thus, to reveal the presence of dentigerous cyst is of critical importance as additional clinical maneuvers aspiration, incisional biopsy and histopathological analysis[2].

The cone beam computed tomography, known as "cone bean", used in dentistry, has been considered an important tool to aid the diagnosis of various diseases. This consists of an additional examination of better quality in relation to the conventional imaging methods for presenting location of the cyst in three planes in space, being more true to the anatomy of the patient's maxillomandibular complex. The three-dimensionality and the millimetric precision of this technique allow tomographic image slices acquired up to 0.12 mm thick. Therefore, details were provided for a more accurate diagnosis and treatment planning or surgery to be performed[6].

The literature describes several approaches of dentigerous cysts, but the most cited are enucleation and marsupialization[7]. These treatments were carried out according to the size and the surgical risks of each lesion[8]. The decompression can also be a form used to heal

this.

This article aims to present a case report where the multidisciplinary planning, involving the most advanced imaging techniques, conservative surgical procedures and orthodontic planning were essential for the treatment of dentigerous cyst in the maxilla of a child of nine years old.

2. CASE REPORT

The patient KHJP, male, nine, leucodermic, complained of a volumetric increase in the maxilla and nasal wing on the left side of the face. When extraoral examination, there was a facial asymmetry without the presence of infectious and nociceptive signals (Figure 1).

Figure 1. Extra oral photo showing facial asymmetry.

Examining the intraoral limits was noted the lack of development on the side of the arch in question, and the presence of deciduous teeth 61 and 62. However, will be ob-normality in the color of the mucosa. By palpation could be observed swelling in the background region of the vestibule in the upper left hemi-arch (Figure 2 and 3).

Figure 2. Initial photo. Frontal plane.

The panoramic X-ray analysis revealed a radiolucent image with involvement of germs of permanent teeth as a central incisor, lateral incisor and canine, all with ectopic location (Figure 4).

Figura 3. Initial photo. Oclusal plane.

Given this condition, and knowing the best quality and the advantages of cone beam CT image, the patient was sent to conduct such additional examination (Figure 5 and 6).

Figure 4. Initial panoramic radiograph.

A needle puncture was performed with a positive result for a brownish color liquid, reinforcing the suspicion of a cyst (Figure 7). The material obtained by puncture was sent for histopathological examination confirmed the diagnosis of dentigerous cyst.

Figure 5. Initial CT.

The incisional biopsy was performed under local infil-

trative anesthesia, which allowed the extraction of deciduous teeth 61 and 62 duos.

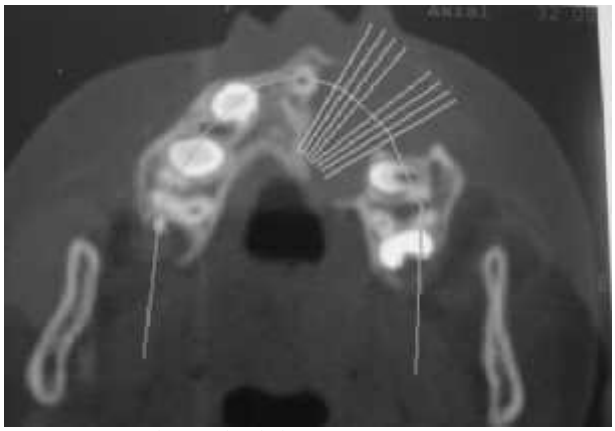

Figure 6. CT section, occlusal view.

Then, an opening in the vestibular region with the purpose of serving as a gateway for the installation of a drain which favored the therapeutic choice tica maid called decompression (Figure 8). The objective of this surgical approach was to regress the size of the lesion and preserve the included permanent teeth.

Figure 7. Needle aspiration.

The communication between the oral cavity and cystic cavity was maintained by drain installed, which allowed the application of dressings of chlorhexidine digluconate 0.12% realized in the form of daily irrigation done using disposable syringes.

Figure 8. Photo after extraction of teeth 61 and 62, and drain installation.

This procedure was performed in order to allow decompression of the cystic cavity and also to keep it away from any food residue.

Figure 9. Installing the apparatus for traction of enclosed tooth.

After 4 months, he was referred for orthodontic treatment, the orthodontic appliance installation of the element 21, which was included (Figure 9).

Figure 10. Orthodontic activation.

The orthodontic movement is underway and the panoramic radiographic examination performed 2 years after the onset of decompression of the cyst, it can be observed regression of bone defect in the area previously affected by the cyst (Figure 10 and 11).

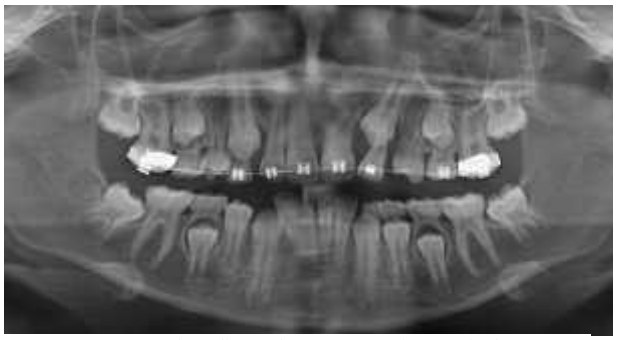

Figure 11. Panoramic radiograph, two years after surgical treatment.

3. DISCUSSION

Although they were considered benign pathologies, the dentigerous cysts can cause consequences to patients. Sapphire (2009)[8] reported that dentigerous cysts with

large extensions, may have potential for expansion leading to impairment of cortical bone, which can lead to paresthesia of the inferior alveolar nerve, when present in the mandibular region.

In this study the presence of facial asymmetry and dental impaction was observed. Thus one can agree with Costa *et al.* (2011)[2] who stated that the development of dentigerous cyst has serious clinical complications such as tooth impaction, ectopic eruption, facial asymmetry, displacement and root resorption of teeth. According Vaz (2010)[9] such cysts have a slow growth in great proportions and asymptomatic, and capable of producing facial edema due to expansion of cortical.

Tortorice (2008)[10] considers the cyst as a common, asymptomatic lesion and can be discovered on routine radiographic examination. However, several diseases can have similar radiographic characteristics[4]. Accordingly, one can agree with Costa (2011)[2] which considers the clinical maneuvers such as aspiration and incisional biopsy, added histopathological examination are essential for diagnosis of dentigerous cyst. Another important and guiding further examination of dental treatment would be the cone beam CT due to its three-dimensionality and millimeter accuracy[6].

In this case report we observed that some information covered will meet the publications Grossmann (2007)[11] and De Avila (2009)[12] who reported that the manifestation of this cyst occurs in the first decades of life and male individuals. However, there are differences in the sites involved by this injury. While Grossmann (2007)[11] claims to be the site of the mandible higher prevalence, our case report showed the dentigerous cyst with involvement of hemimaxila.

With regard to the groups of teeth more involved in the presence of dentigerous cyst, Sapphire *et al.* (2009)[8] reported that third molars, maxillary canines and premolars are presenting this higher prevalence. These authors also observed the development occurring in the first decade of life. Hyonoto (2003)[13] attributes this higher prevalence found in third molars, is due to the fact they present greater possibility of asymptomatic bone retention and behavior.

Neville (2009)[14], Grossmann (2007)[11] and Sapphire (2009)[8], in addition to agreeing with the group of individuals of the same age and the same gender involved also agree and affirm that the dentigerous cyst has a greater predilection by Caucasians. Recently Carvalho *et al.* (2011)[15] in retrospective study, evaluated 192 case report in a period of 18 years, obtained with results the males showed two times more affected than females (2:1), with predominance in Caucasians (56.7%) in the second decade of life (42.4%).

Sun (2009)[16] stated that the radiograph becomes an arbitrary method of diagnosis, where any radiolucent area pericoronal 5 mm in diameter could be considered suggestive of cyst formation and should therefore be subjected to microscopic examination.

To Piassi (2003)[17], clinical examination should be supplemented with radiographic and histopathologic examinations cos initially periapical radiograph to see if the tooth has involvement with the disease, however, according to the author, occlusal and panoramic radiographs are needed to assist diagnosis. Since Smith (1995)[18] said occlusal radiography reveals the degree of bone growth and justifies the increase in volume of the region, while the panoramic shows the extent of radiolucent lesion involves the periapical region.

Costa (2011)[2] reported that is extremely important to monitor the mixed dentition and also research into the causes of a possible delayed eruption chronological conducted through periodic examinations, because in these cases it is very likely to be the cause of this retention of origin cystic.

Radiographs are not sufficient to demonstrate the extent of the injury and impairment method. Therefore, Bontrager (2008)[19] suggests that the image of CT shows no distortion, making it possible to measure distances, displacements, diameters and thicknesses using interactive computer graphics. Garib (2007)[6] reported big advantages to using the method of imaging by computed tomography namely: multiplanar and 3D reconstructions, and reconstructions of two-dimensional radiographs; good sharpness; low contrast between hard and soft tissue and good accuracy.

Computed tomography, in this report, can show not only the location of the canine included, but also the relationship with adjacent structures. As a way of further examination Bastos (2011)[20] and Costa (2011)[2] by means of a positive aspiration, with yellowish liquid and an incisional biopsy, sent for histopathological arrived at the diagnosis of dentigerous cyst. Both cases were reported in patients in the first decade of life, with the apex forming, with great extension of the lesion and the involvement of various elements as in this study, then selecting marsupialization as a treatment, preserving the tooth and the permanent expected regression of the cyst.

For selection of surgical treatment of a dentigerous cyst should previously examine the size of the lesion; communication with adjacent anatomical structures; patient age; location and possibility of access; the stage of root formation; whether there is sufficient interdental space and the degree of potential eruption, in order to preserve the permanent dentition. Much of the time, in adults, is held and enucleation in children and adolescents marsupialization[2,3,20]. Sapphire (2010)[8] stated that children have greater bone regeneration capacity than adults and teeth with open apices has great potential outbreak, suggesting in these cases, as treatment marsupialization. Blaya (2010)[7] emphasizes that enucleation is the treatment of choice when there is no risk of damag-

ing anatomical structures such as dental apex, maxillary sinus and inferior alveolar nerve. Already the marsupialization is performed in order to maintain the germ of the permanent tooth, promoting eruption, being useful for displaced teeth. Santos (2009)[21] reports that the decompression is the creation of a bony with the installation of a drain, seeking a reduction in the internal pressure of the cyst, this procedure requires patient cooperation because this will have to irrigate the bone cavity daily.

As a similarity in the treatment of a case of Blaya (2010)[7] plan there is the use of orthodontic traction in search of an ectopic tooth eruption involved in a major injury.

4. CONCLUSION

Through the study presented herein, we conclude that the dentigerous cysts despite being a benign pathology, depending on its location and extent, can cause great harm to the patient and, if handled wrongly, leading to deformities difficult to resolve. It is noteworthy that the diagnostic methods such as CT and cone beam conservative approach by decompression were important in the treatment of this case.

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SURGICALLY ASSISTED RAPID MAXILLARY EXPANSION: REPORT OF A CLINICAL CASE

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ABSTRACT

The posterior crossbite resulting from a deficiency in maxillary growth, when present in individuals with advanced bone maturity, can be corrected appropriately and provide stable and satisfactory results, by means of a maxillary expansion coupled to the osteotomy of the bone structure which resist to expansive forces. This study presents a case report of an adult patient with transverse discrepancy and unilateral crossbite, which was treated by surgically assisted rapid maxillary expansion, with satisfactory outcomes with crossbite correction with preservation and stability after 3 years.

KEYWORDS: Crossbite, Rapid maxillary expansion, Maxillary Osteotomy.

1. INTRODUCTION

The esthetic necessity has led a large number of adult patients seeking for orthodontic treatment in dental offices. Approximately 8 to18% of the patients seeking professional complaining of dentofacial deformity have a transverse maxillary deficiency[8,14].

This growth deficiency creates a discrepancy between the apical bone bases, when faced with reduced transverse dimension greater than observed important clinical features such as: ogival palate, narrow maxillary dental arch, crowded, rotated and proclined teeth, asymmetry of the hemi-arches and wide buccal corridor[5,6,10,19].

The clinical feature commonly found in these transversal disharmonies is the posterior crossbite that can be classified as: dental and/or skeletal functional posterior crossbite, that can be uni or bilateral. The correct diagnosis of the malocclusion is crucial for a successful treatment[5,18].

Rapid maxillary expansion (RME) recommended by ANGEL[1] (1860) and used in 1961 by HASS[10] is indicated for the treatment of this transverse deformity in the growth stage, when there is low resistance in the maxillary bone, producing thus more stable results[16]. However, when faced with a maturation of the maxillary suture, the use of orthodontic-orthopedic appliances lose their effectiveness using a rapid maxillary expansion associated with osteotomy of the bony structures, making it necessary to resist to expansive forces[21]. In this case, it was possible to successfully obtain the disjunction of the suture of the maxillary processes, without the expense of supporting structures involved, a procedure known as Surgically Assisted Rapid Maxillary Expansion (SARME)[4,7,22].

Both the RME and SARME using the expander deappliance may be the tooth-borne (Hyrax) or tooth-tissue-borne (Haas) both with screw activator.

The objective of this study is to present a case report of an adult patient who presented with posterior crossbite, which was subjected to an associate orthodontic treatment there is a surgically assisted rapid maxillary expansion (SARME).

2. CASE REPORT

The Patient F.D.S. male, 23 years old, leucoderm, sought to orthodontic treatment with the complaint of discontent facial aesthetics and dental, as well as dissatisfaction with the smile. When extraoral clinical examination revealed that the patient had a facial asymmetry within normal limits, profile gently convex, dolichofa-

cial standard and passive lip seal (Figure 1).

On physical exam was diagnosed Class II malocclusion right subdivision with absence of the right lateral incisor, high palate with transverse maxillary atresia, right posterior crossbite and moderate crowding, it was still observed that the maxillary and mandibular dental midlines were coincident with each other and the median sagittal plane (Figure 2).

Figure 1. Initial extraoral photographs.

On the analysis of the panoramic radiograph was observed that the dentoalveolar structures were normal with the absence of dental elements 12, 18 and 38, also observed suggestive endodontic treatment of tooth 21 and restorative treatment of the teeth 16, 17, 21, 26 , 27, 38, 37, 46 and 47 (Figure 3).

Figure 2. Initial intraoral photographs.

In the initial lateral radiograph was identified a divergence of the horizontal planes, featuring a dolichofacial pattern combined with a moderate convexity of the bone and soft tissue profile (Figure 4).

Figure 3. Panoramic radiograph.

Figure 4. Initial lateral cephalogram.

After clinical evaluation of the patient and gathering all the information, disclosure of a unilateral posterior crossbite, in the case of a patient with advanced bone maturity, the proposed treatment plan was an ortho-surgical interaction, starting from the previous installation of the Hyrax appliance (Figure 5).

Figure 5. Occlusal, front and side view of the Hyrax appliance.

After the osteotomy, the cycle of activation of the expansion device was initiated. The protocol established was the activation of ¾-turn on the first postoperative day, followed by ¼-turn in the morning and another ¼-turn in the afternoon until it reaches 5 mm of interdental diastema, after 18 days of activation the desired expansion resulted in the opening of the palatine suture (Figure 6).

Figure 6. Intraoral view after activation of the Hyrax appliance.

After obtaining the transverse expansion of the maxilla and overcorrection of the crossbite, evidenced by the presence of interincisive diastema (Figure 6), the Hyrax appliance remained for a period of 6 months as a retainer, and then was removed for the fixed appliance installation for aligning and leveling the teeth with 0.014", 0.016" Nitinol and 0.018" stainless steel archwires. At this stage, it began using intraoral Class II elastic on the left side and Class III on the right side, accompanied by the esthetic adjustments as remodeling of the right maxillary canine, the use of interdigitation elastics and finalization bends (Figure 7).

Figure 7. Installation of fixed appliance

After the period of 30 months from start of treatment with a satisfactory result and goal achieved, the fixed appliance was removed and installed the Hawley plate maxillary retainer and 3x3 bonded mandibular retainer (Figure 8).

Figure 8. Final treatment images: extra and intraoral.

Figure 9. Final lateral cephalogram.

Even with three years of follow-up treatment, the case remains stable, showing a harmonious profile, the presence of passive lip closure, occlusion denotes a better teeth accommodation, stable and balanced, did not change on the final conformation achieved in the maxillary arch (Figures 9 and 10).

Figure 10. Extra and intraoral photographs, 36 months after treatment.

3. DISCUSSION

The orthodontists need to know the type, size and magnitude of the posterior crossbite, because its severity

and resolution are directly related to the present change[16].

To generate an accurate diagnosis and outline the appropriate treatment plan the clinician should make use of diagnostic tools, such as dynamic model analysis[12,16], radiographic and cephalometric evaluation, to determine the transversal discrepancies[5,6,11,12,16]. The patient described here presents posterior crossbite with tooth absence on the same side. It has been speculated that the lack of this tooth has led to posterior occlusal disharmony, considering that this malocclusion is a clinical variation of normal growth of multifactorial origin. The clinical findings compatible with this disability as: high arched palate, upright posterior teeth and loss of conformation elliptical arch, beyond the wide buccal corridor, are confirmed with those described by other authors[17,19].

Early correction of this transversal deficiency advantages the largest bone elasticity and promotes a better relationship between the apical bases by means of orthopedic braces[5]. RME is a permanent and efficient technique in an attempt to compensate for the deficient maxillomandibular relationships[1,10].

However, there is controversy about the ideal age[2] to produce a favorable maxillary expansion, matched against the authors that agree that RME reach your goals when applied to patients still in the growth stage[15], as well as jeopardize the integrity of the supporting structures such as injuries the periodontium, excessive teeth inclination, resorption of cortical alveolar bone in case of a patient with advanced skeletal maturation[9.17].

Thus considering that the patient treated in this report has bone maturation and presents an absolute maxillary atresia[3], it was decided in this case to perform SARME which consists in expanding transversally the maxilla using bone fragility through osteotomies, generating more stable results[11,16].

The failures occurred on an attempt of a rapid maxillary expansion in adults can be attributed to the stiffness of the joints of the zygomatic complex in the maxilla that tends to be the point of greatest resistance to expansion, thus being one of the facts that explain the high rate of failure in the maxillary expansion in adults[13].

At the end of the treatment of this clinical case, it was noticed that the patient had no damage to the stomatognathic system and allowed stable, facial and occlusal satisfactory results[2,11].

4. CONCLUSION

After diagnosis and planning of the case, it was concluded that:

• The SARME gave the patient the correction of the posterior crossbite with alignment and leveling of the teeth, improving occlusion without causing damage to anatomical and biological structures.

• The ortho-surgical intervention may be considered part of the treatment plan to correct transverse deficiencies in adults with advanced skeletal maturation.

• This procedure allowed favorable and stable results.

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BULK-FILL RESIN-BASED COMPOSITES: MICROLEAKAGE OF CLASS II RESTORATIONS

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ABSTRACT

The aim of this study was to evaluate in vitro microleakage in class II restorations, using or not a fluid resin low shrinkage. Twenty human molars were used and each tooth cavities 2 slot. The teeth were randomly divided into 4 groups : G1E (enamel) - SDR + TPH3 (Dentsply Caulk); G1D (dentin) - SDR + TPH3 (Dentsply Caulk) and G2E - TPH3 (Dentsply Caulk) and G2D - TPH3 (Dentsply Caulk) . Subjected to thermal cycling (10.000 cycles), immersed in 0.5% basic fuchsin for 24 horas/37 ^{0}C. The analysis of the degree of infiltration done by optical microscope (magnification 15X). Data were statistically analyzed using the Student *t* test. The result showed that there were differences between the dental tissues (G1E - G2E X G1D - G2D) with greater infiltration for completion in dentin. To use the fill resin SureFil SDR no difference. The fluid resin low shrinkage (bulk fill composite) had the same results of microleakage in class II cavities when compared with the conventional technique of restoration with composite resin.

KEYWORDS: Dental infiltration, Composite Resins, Dental Marginal Adaptation.

1. INTRODUCTION

The big challenge in the making of a restoration with composite resin is the correct performance of the restorative technique respecting and knowing specific material characteristics, such as polymerization shrinkage which can cause serious problems in marginal sealing of that restoration. This microleakage characterized by the passage of bacteria, fluids, molecules and ions between the cavity wall and the restorative material, resulting in tooth sensitivity, recurrent caries and pulp damage to[1].

To control the marginal and consequent increased longevity of the restoration infiltration is essential to forming a union effective interface between tooth and restorative material[2,3]. The formation of the hybrid layer, which is the interface to dentin must be able to prevent the ingress of fluids and bacterial products that may lead to post- operative sensitivity, mismatch of the restoration margins secondary caries and consequently failure treatment[4]. The leakage is related mainly to polymerization shrinkage and stiffness of the restorative material that can lead to marginal leakage and occlusal maladjustments, until the loss of restoration and recurrent caries[5,6].

Direct resin restorations in class II preparations are a great challenge for the Dental Surgeon, for the control of polymerization shrinkage of composite resin preparations with the cervical margin in dentin exhibit more susceptibility to occurrence of leakage[1]. To lessen the effects of polymerization shrinkage, several techniques have been proposed, among them are: the insertion of the composite in increments[1,6] resin, using different protocols polymerization[7] and the association of different materials[6,7].

In an attempt to better dissipate stress, minimizing the stresses generated on the interface of the restoration, have emerged in the market various restorative materials. This is possible by modifying some physical and mechanical properties of these materials. Notable fluid resins fill only (bulk fill composite), whose employment associated with conventional composites would bring great benefits for esthetic restorations in posterior teeth. The fluid filling resins have a lower concentration of charge, great flow with excellent adaptation to the cavity (some still have the ability to self-leveling) and low modulus of elasticity, which in theory would support and better dissipate stress generated by thermal stresses and

masticatory , and thereby caused microfractures in the bond line and favoring marginal sealing[8]. Still possess the ability to fill the cavity without the need for overlapping layers, eliminating the stratified incremental technique[2].

This work aimed to evaluate laboratory after thermal cycling test, the influence of using fluid resin filler in combination with composite on the marginal sealing in preparation class II ending in dentin and enamel resin.

2. MATERIAL AND METHODS

It is laboratory in vitro assay developed intentionally defined and standardized sample of human molar teeth extracted and properly donated by Bank of teeth on the advice and approval of the Research Ethics Committee number 301 297 of 07/06/2013.

Exactly 20 freshly extracted human molars, teeth donated by Bank of the State University of Maringa, were used in this research; EMU with no cracks or fractures of enamel, cleaned and stored in a solution of 10% formalin for 72 hours, passing if so, the subsequent storage in saline solution of 0.9% sodium chloride.

The teeth were rinsed thoroughly with water and cleaned with pumice and rubber cup to then be held Class II mesio - occlusal- distal tooth in each, ending on cervical dentin and enamel type standardized cavities. The molars were suitably adapted (with wear through diamond drills) and fixed to the dental mannequin (Jon) with wax, the location of the upper 2nd molar removed therefrom.

The class II preparations MOD (mesial-occlusal-distal) were standardized following a cavity geometry by using 4137 diamond bur (KG Sorensen) in high speed refrigerated air-water spray and presenting Finally, the following dimensions: occluded pulp with 03 mm bucco-palatal with 03 mm and axial depending on the cervical end of the preparation.

The occlusal-cervical dimension was standardized using as reference the cementoenamel line, ie , the distal preparation was located 01 mm below this line (ending in enamel) and preparation mesial to 01 mm beyond this line (ending in cervical dentin) . After cavity preparation was adapted Unimatrix metallic matrix (TDV Dental Ltda.) and plastic wedges.

All cavities were conditioned with a solution of 37% phosphoric acid gel (Dentsply) for 30 seconds , washed with water jet for over 30s and dried with a gentle stream of air leaving the moistened surface. After using disposable brush (Microbrush), the XP - Bond adhesive system (Dentsply) was applied in two consecutive layers and light cured through the curing unit Optilight Max (Gnatus) for 20s.

The samples were divided into 02 groups of 10 teeth each, according to the restorative material used in the preparation:

Group 01: Resin fluid fill SureFil SDR (Dentsply) + TPH3 Composite resin (Dentsply);

Group 02: Composite resin monohybrid TPH3 (Dentsply).

In Group 01 to fill fluid resin SureFil SDR (Dentsply) was used, with the aid of a syringe Centrix, based only on the pulp, and cervical axial setting of the preparations reference to a thickness of 01 mm in wall pulp walls. The restoration was completed using composite TPH3 (Dentsply) the other remaining 02 mm in two increments resin.

The Group 02 has been inserted only TPH3 composite (Dentsply) in three increments.

For all groups, after polymerization of the last increment of resin composite, the finish of the margins of the restoration was accomplished: first with a scalpel blade paragraph 150C (Swann Morton), to remove any excess material - especially in the region cervical and then polished with Sof - Lex (3M) discs sequential particle size. These procedures was made to promote the smooth and free of restorations excesses.

After finishing and polishing of 20 preparations, the specimens were stored in distilled water at 37 º C for 07 days until being subjected to thermal cycling with thermal cycler machine (New Ethics , Model 521 -E Ethics Equipment Scientific S / A, São Paulo, SP, Brazil). The cycling consisted of 10,000 cycles of 30 seconds each, with an interval of 3 seconds in temperatures of 5 ºC and 55 ºC. The number of cycles corresponds to aging the samples at 12 months[9].

Upon completion of thermocycling the teeth were covered with two layers of nail polish white (Risqué) except for 01 mm below and above the interface between the tooth/ restoration. This procedure was intended to prevent the penetration of dye into unwanted areas now covered by enamel, as the apical foramen.

Following the teeth were immersed in 0.5 % basic fuchsin for 2 h to show potential leaks in the tooth -resin interface.

Properly stained teeth were sectioned in ISOMET 1000 Precision Cutting Saw machine (Buehler Lake Bluff, USA) in a mesio - distal direction at a speed of 300 rpm under constant refrigeration and each sample was separated through an identification code with pen black overhead projector (RCF code for Group 01 and Group 02 for RC code) to facilitate their subsequent measurement.

The microleakage at the interface restoration / enamel and restoration / dentin was assessed qualitatively. The rate of dye penetration was assigned, adopting the approach of various scores, previously determined under Bassiouny (Figure 1):

0 - No infiltration;

1 - Infiltration of the dye to half of the gingival wall;

2 - Infiltration across gingival part reaching the axial wall;

3 - Infiltration of the axial wall and into the pulp.

Cuts greater degree of infiltration corresponding to each specimen were selected and observed by three calibrated examiners. These examiners did not participate in earlier stages of this research, nor knew the code for the identification of the specimens (Figure 1).

Figure 1. stained and sectioned samples.

The analyzes were performed with the aid of an optical measuring microscope (Toolmaker's Microscope Mitutoyo, Japan) using a 15x objective.

The composition of the resins used was summarized in the Table 1.

Table 1. Composition of the resins used.

TPH3	Surefil SDR
Glass aluminum silicate, barium fluoride	Glass silanized barium aluminum borosilicate
Glass strontium aluminum silicate fluorine	Glass silanized fluoride barium aluminum borosilicate
Resin modified urethane dimethacrylate	BisGMA dimethacrylate
Ethoxylated bisphenol A dimethacrylate (EBPADMA)	Silica
triethylene glycol dimethacrylate (TEGDMA)	EDAB
Camphorquinone (CQ) as photoinitiator	other excipients
Butyl hydroxy toluene	
UV stabilizers	
Titanium dioxide	
Iron oxide pigments	

The samples were analyzed individually by each examiner and, in case of disagreement between the examiners, they gathered for a new evaluation to obtain agreement on a common outcome. Data were statistically analyzed using the Student *t* test with significance level of 5 %.

3. RESULTS

The results of agreement of the three examined were recorded in a table for later statistical analysis (Table 2).

Table 2. Values measured under an optical microscope after evaluation by examiners.

		Sample - Results in agreement									
		01	**02**	**03**	**04**	**05**	**06**	**07**	**08**	**09**	**10**
Group 01 (SDR+THP 3)	Enamel (G1E)	2	0	1	1	1	1	1	1	0	3
	Dentin (G1D)	2	2	2	2	2	2	2	2	2	2
Grupo 02 (THP3)	Enamel (G2E)	0	0	1	2	1	3	1	2	1	0
	Dentin (G2D)	2	2	2	3	2	3	1	2	0	0

Through the Student t test with significance level of 5 %, the same structures (enamel X dentin enamel and dentin) were compared and no significant difference between Groups 01 and 02 ($p > 0.05$). When comparing different structures, ie, X enamel dentine was no significant difference between groups ($p < 0.05$) (Table 3).

Table 3. Statistical analysis of the results.

P value	Compared Statistical	Significance Structures
0,5	SDR+TPH3 Enamel (G1E) X TPH3 Enamel (G2E)	No difference
0,196911	SDR+THP3 Dentin (G1D) X TPH3 Dentin (G2D)	No difference
0,000788	Enamel (G1E+G2E) X Dentin (G1D+G2D)	With difference

4. DISCUSSION

Conducting experiments involving restorative materials and techniques described in the literature are very for its relevance and clinical applicability.

Class II termination involving dentin type cavities has been studied by numerous authors[1,4,8,10,11,12].

The class II type wells have cervical margin in dentin, have a determining factor for longevity of same, the occurrence of infiltration by marginal leakage. In an attempt to minimize problems inherent restorations have appeared in numerous market restorative materials with physical and mechanical properties seeking to better dissipate stress, thereby causing a lower leakage[8].

With the release of fluid composites with low shrinkage stress (bulk fill composite), the posterior teeth in dentistry has totally changed, allowing the cavity fill-

ing in a single layer and with greater ease, which makes the procedure much simpler, practical and fast[13].

According to Sadeghi and Lynch in 2009[14], fluid resin is used as the first increase in gingival and axial wall for class II restorations decrease microleakage. This result was obtained using only conventional fluid resins, different from the present study that evaluated the fluid resin low shrinkage Surefil SDR as a basis for liner.

Similarly, Reddy *et al*., 2013[3], compared class II restorations using flowable composite in different thicknesses. Fifty molars underwent type and class II preparations were randomly divided into five groups according to the restoration performed: Group I, P60 (no resin flow), group II, ultrathin coating resin flow (0.5 -1mm) with coverage of composite resin and group III, thin coating (1-1.5) with composite resin coverage and group IV, ultrathin coating (0.5-1 mm) and group V, thin coating (1-1.5). The teeth were then thermally cycled for 1500 cycles (between 5 and 60 °C) and immersed in a dye for 24 hours. Leakage was measured as the degree of dye penetration. Concluded that the application of resin flow, even in ultrathin layer improved marginal sealing, reducing microleakage after thermocycling. This result is obtained by evaluating the different thicknesses of the fluid resin, which was not used in this study, where low shrinkage fluid resin remained the same results with a conventional technique composite. This difference was probably due to the smaller number of cycles of thermal cycling and the materials used.

According to Frankenberg *et al*., 2011[13], the flowing resin low shrinkage (bulk fill composite) designed to be used as basis for posterior restorations had the following advantages: single increment of up to 4 mm, without stratification (several layers), 60 % less polymerization shrinkage and 30-50 % reduction in procedure time compared with conventional composites, self-leveling consistency for optimum adaptation to the cavity (using conventional resins as base flow is no longer necessary) and compatible with current adhesive systems (resins and adhesives based on methacrylate). During curing, the technology employed in these resins allows the polymerization reaction occurs more slowly because of the presence of a modulating agent polymerization, leading to tension reduction without reducing the rate of polymerization. This results in a high rate of monomer conversion, in addition to reducing the possibility of postoperative sensitivity suggesting a better tooth-restoration interface in class II restorations in different thicknesses even when the same resin used in this study (SDR)[15].

Although not evaluated in this study, Fleming *et al*., 2012[5], concluded that the use of a flowable resin low shrinkage as a basis for class II restorations significantly reduced the deflection of the cusps of teeth premolars compared with restorations class type II using only conventional composite resin by the technique of gradual increase, important for postoperative sensitivity and dental cracks factor.

The fluid composite resins Low shrinkage stress (bulk fill composite) have four distinct characteristics: 1 - low polymerization shrinkage (decreasing the chances of leakage), 2 - polymerisation capacity of at least 4 mm (due to the material is translucent highly favorable and the transmission of light), 3 - fluid consistency (to permit easy drainage and cavity adjustment) and 4 - possess excellent physical properties such as good resistance to compression and wear[2].

However, Campos *et al*., 2002[16] evaluated through in vitro study the degree of marginal leakage with the use or not of flowable composite in class II cavities with gingival margins in dentin and concluded that the presence of resin used in the wall flow neck was unable to completely prevent the leakage nor soften it, however this category resin was not assessed in this study. Fleming *et al.*, 2012[5] rated the degree of marginal leakage with the use or absence of fluid resin low shrinkage (bulk fill flowable) class II premolars type cavities and concluded that there was no significant difference in microleakage cervical, results corroborate the present study .

According Poggio *et al*., 2013[17], the composite resin restorations class II with margins below the cementoenamel junction, ie, dentin, evaluated with different techniques and restorative materials not completely eliminate microleakage.

The unanimous in the scientific literature that there is a variation between the substrate (enamel and dentin). The dentin to be more tissue rich in organic matrix promotes the adhesion smaller independent direct restorations system adhesive or restorative technique[18]. The results of this experiment are corroborated with studies in the literature showing that no material completely eliminates leakage in both enamel and dentin[8,16,17] compared the two structures, there is a greater infiltration dentin than to enamel in both groups due to structural and morphological diversity of these structures already known.

5. CONCLUSION

The use of flowable composite resin low shrinkage stress (bulk fill composite) obtained similar results of microleakage in class II cavities when compared with the conventional technique of restoration with composite resin.

The leakage was higher in dentin than in enamel.

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INFLAMMATORY ODONTOGENIC CYSTS: A BRIEF LITERATURE REVIEW

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ABSTRACT

Inflammatory odontogenic cysts are benign osteolytic asymptomatic lesions, but that, depending on the size, they can destroy the surrounding bone and let it infected. They are classified into periapical or lateral radicular cyst, residual cyst and paradental cyst, which need a source of infection to proliferate. The current study mainly aims to deepen the knowledge regarding the types of inflammatory odontogenic cysts, describing its characteristics and main aspects and highlighting the importance of the differential diagnosis for the treatment of these lesions. The methodology adopted consists in a literature review. It can be concluded that early detection and accurate diagnosis are essential for the proper treatment of the odontogenic inflammatory cysts. By avoiding the persistence of an asymptomatic lesion such as a residual cyst, the structure and vitality of the teeth are conserved and the integrity of anatomical structures is preserved, in other words, it restores the patient's oral health.

KEYWORDS: Odontogenic inflammatory cysts, accurate diagnosis, appropriate treatment. Oral health.

1. INTRODUCTION

Odontogenic cysts are the most common osteolytic lesions (90% to 97% of reported cysts) in the oral region. Its growth is slow, from remnants of odontogenic epithelium of Malassez. Thus, its histogenesis is related with debris that are trapped within the bone, enamel or gingival tissue; they are usually intraosseous location. Although benign, can become destructive, because they are frequent incidence and represent a major cause of bone destruction in the jaw and mandible[1,2].

In 1992, the World Health Organization (WHO) classified odontogenic cysts according to their development or with inflammatory cysts. Are cysts of development: the gingival cyst of childhood, the primordial cyst, the dentigerous cyst (follicular cyst), the eruption cyst, lateral periodontal cyst, calcifying odontogenic cyst, glandular odontogenic cyst and the gingival cyst of adults. In these cysts, the active factor in cyst formation is unknown, i.e., the infection is not stimulating the proliferation of odontogenic epithelium[4,5]. The inflammatory cysts are lesions that originate from the infection of root canals from caries or occurrence of a trauma that generated pulp changes. The appearance of these cysts occurs from the pre-existence of a periapical granuloma or by induction of epithelial rests of Malassez[5,6].

The inflammatory cysts can be classified as: inflammatory periapical cyst (apical radicular cyst and lateral periodontal cyst or apical), residual cyst and cyst paradental. These cysts require a source of infection for the remaining epithelial root sheath of Hertwig (or epithelial remnants of Malassez), are stimulated and begin the spread of infection. Some of these cysts, such as periapical cysts, inflammatory and lateral root cyst, depend on endodontic infection, while paradental require periodontal cyst or pericoronal infection[3,4,7]; the odontogenic inflammatory cysts constitute about 70.1% to 85% of maxillary cysts[1,2].

Due to the high incidence of inflammatory odontogenic cyst in the maxilla, the aim of this study is to deepen knowledge about the different types of odontogenic inflammatory cysts, describing its characteristics and main aspects and highlighting the importance of differential diagnosis for the treatment of these lesions.

2. MATERIAL AND METHODS

The following literature databases were searched: General Science Index, Medline, Pubmed, EBSCO host and CAPES Periodicals. Studies were selected if they scope were directly related to semi-adjustable articulators. Studies published from 1980 to 2013 were included according to the author's analysis. The keywords of this study were utilized to the consult the databases.

3. LITERATURE REVIEW

A cyst is defined as a pathological cavity lined with epithelium and odontogenic or non-odontogenic origin, showing fluid or semi-solid contents inside[1,6].

The most accepted theory for the formation of cysts would be combining its beginnings to the proliferation of epithelial remains under the influence of inflammatory cytokines and growth that are released by various cells in the apical lesion factors, leading to the formation of islets without blood vessels, which degenerate the central region of the lesion. These islets, being away from the adjacent connective tissue, release enzymes which degenerate their own cell protoplasm, liquefying the dead cells, forming a cavity searched by stratified squamous epithelium[5,8,9].

Thus, odontogenic cysts are formed from this mechanism, being classified as "developmental cysts" or "inflammatory cysts." The odontogenic inflammatory cysts require one infection to the epithelial remnants of Malassez proliferate and are the subject of this brief review of the literature.

DEFINITIONS

Inflammatory periapical cyst is truly inflammatory odontogenic cyst coated by epithelium that is adhered to the periapical region of the tooth with pulp necrosis[4,8.] The lateral radicular cyst is an example of inflammatory odontogenic cyst that is attached to the lateral surface of the root of a tooth erupted and necrosis caused by infection of the pulp chamber of the tooth[3,10].

There are two distinct categories or types of radicular cysts: those with cystic cavity completely filled by epithelial lining (true cysts) and those whose epithelium lining the cystic cavity is interrupted by the root apex, which penetrates into the lumen (cyst bay)[9,11]. More than half of the apical cystic lesions are true cysts, because they consist in a pathological cavity coated by epithelium originated from the epithelial rests of Malassez and, often, filled with liquid[6,12].

According to Neville *et al.* (2004)[7], the radicular cyst is not removed after tooth extraction via alveolar curettage procedure, the degree of inflammation can continue. Thus, the absence of periapical curettage tissue during surgical removal of a tooth can lead to the formation of an inflammatory cyst called residual cyst. The residual cyst is a type of inflammatory odontogenic cystic lesion, caused an inflammatory periapical cyst that persists retained within the bone after extraction of the affected tooth involved, or arises after incomplete removal of a cyst original, motivating the persistence of a radicular cyst. Thus, residual cysts occur in sites healed extraction[13,14,15].

The paradental cyst is an uncommon inflammatory odontogenic cyst located adhered to the cementoenamel junction of a vital tooth with partial irruption, which extends along the root surface, and is associated with pericoronitis, i.e., gingival inflammation, leading to hyperplasia and consequent cystic formation[3,4,16].

ORIGIN

The inflammatory periapical cysts originates from the epithelium of periapical granulomas, which commonly is derived from the remaining epithelial sheath of Hertwig, but may also be related to the crevicular epithelium lining the sinus or the epithelial lining of the fistulae. It is also admitted that the cyst may be caused by the proliferation of the epithelium, which seeks to take the abscess cavity in periapical granuloma, in view of the worsening of the lesion. However, this method of training is not well founded[3,7].

The lateral root cyst is similar to the inflammatory periapical cysts and extends along the side portion of the root. Also stems, commonly, the epithelial rests of Malassez or a preexisting dental granuloma. The source of the inflammation can be a periodontal disease or pulp necrosis extending through a side foramen. Toxins exit of the foramen and infect the tissue of the periodontal ligament. The inflammatory response induces proliferation of epithelial rests of Malassez or the remnants of Hertwig's epithelial sheath and the formation of a cystic lesion[3,7,10].

The residual cyst has the same origin of the inflammatory periapical cyst, i.e., stems, after extraction of the involved tooth without curettage, by proliferation of epithelial remnants of the sheath of Hertwig stimulated by endodontic infection[3,15].

The paradental cyst seems to originate in crevicular epithelium, the reduced epithelium of the enamel organ or remnants of Hertwig's epithelial root sheath in the periodontium; epithelial remnants of Malassez. Although its exact origin is not yet understood, it is believed that an inflammatory process, such as periodontitis or pericoronitis, stimulate their development and observations of scholars suggest that the formation of the enamel projection in the root bifurcation is related to the pathogenesis of cyst paradenta[4,16,17].

ETIOLOGY

The inflammatory periapical cysts are caused by odontogenic infection of low virulence and long duration, based root canal after pulp necrosis that extends to the periapical tissues and stimulates proliferation remnants of Hertwig's epithelial sheath, or epithelial remnants of Malassez contained ligament periodontal. The infection is constituted especially by anaerobic bacteria in the periapical region, which stimulate and activate the mechanisms of innate and acquired defense, allowing vascular and cellular events encourage the development

of dental granuloma and radicular cysts[4]. The lateral radicular cyst can develop from a dental granuloma. Etiologic factor an odontogenic infection of low virulence and long duration, side of the affected tooth root canal caused by an accessory canal after the pulp necrosis. This odontogenic infection appears to be similar to that observed in inflammatory periapical cyst, which is composed mainly of anaerobic bacteria[1,2,3].

The residual cyst is a common odontogenic lesion on the part of the tooth support areas of the jaws. Is closely related to the periapical or radicular cyst, so the etiology is common between the two entities, these being distinguished only by the association or not, the root of a tooth. This is periapical or radicular etiology of infectious inflammatory cyst, infection of low virulence and long, located in the root canal and the residual cyst generated from the extraction of the non-vital tooth without curettage[3,4,18].

The paradental cyst is caused by infection of the pericoronal tissues, a tooth irruption or due a tooth partially erupted. This infection triggers an inflammatory reaction of these tissues, pericoronitis, which stimulates the proliferation of the reduced enamel epithelium subsequent cystic degeneration[3].

CLINICAL CHARACTERISTICS

The inflammatory periapical cyst is the most common odontogenic cyst. The preferred location of this cyst is the anterior maxilla. There is a slight predilection for males. There are no age group with the highest incidence of inflammatory periapical cyst[12,19,20]. Its evolution is slow because months are needed to observe clinical manifestations. Most of these cysts grow slowly and does not reach full size. Patients with inflammatory periapical cysts have no symptoms. The inflammatory periapical cyst is painless, unless an acute inflammatory exacerbation occurs, since this cyst is inflammatory. The affected tooth has become normal, not sensitive to percussion, mobility or with sudden extrusion. If the cyst reaches a medium or large size (20-35 mm in diameter), or in case of worsening signs and symptoms of acute inflammation, such as toothache, swelling, increased tooth mobility, sudden extrusion, light sensitivity arise It is possible to mobility and displacement of adjacent dental elements occur. The intensity of these clinical manifestations is directly proportional to the degree of intensification. The tooth of origin does not respond to vitality tests. The pulp by cold, heat and pulp test are negative, i.e., reveal pulp necrosis[3,7].

The majority of radicular cysts (60%) are found in the maxilla[3], most frequently affecting the alveolar bone of the upper jaw, especially around incisors and canines, and having a higher prevalence in women[2,10,21]. This type of cyst usually represents an asymptomatic lesion and the displacement of adjacent teeth is possibly the first clinical manifestation of the presence of the cyst, whose growth is slow but aggressive, and may take sufficient size to produce destruction of the outer cortical bone and a swollen hard and painless[19,23,24]. Lesions that persist or increase in size are probably secondarily infected[24]. The occurrence of radicular cysts was noted that the third and fourth decades of life have highlighted the prevalence[20,22], and that are not commonly associated with primary dentition[19,20].

The residual cyst is rarely found in children. Occurs at any age, being diagnosed mainly between 40 and 60 years, more commonly in male patients (62.5%) and in both jaws, suggesting to be more common in segments of the mandible[4,14,21]. It is the third or fourth most common cystic lesion in the jaws[15], representing approximately 10% of all odontogenic cysts[25]. Although residual cysts are usually asymptomatic and is an incidental radiographic finding in edentulous areas, occasionally, can cause expansion of the affected and jaw pain, if a secondary infection is present. Often these cysts occur within the bone, being very rare in extraosseous region, but have observed and therefore should be included in the differential diagnosis of peripheral lesions of the mandible[2,4]. Typically, these cysts have limited growth potential, although further expansion can be seen, causing displacement of teeth. According to Strickland *et al.* (2013)[13], in the mandible, the mandibular canal may be displaced inferiorly. Buccal and/ or lingual expansion can occur. The displacement of the top floor of the maxillary sinus may occur when these cysts in the jaw.

The paradental cyst is an inflammatory odontogenic cyst considered rare, since its prevalence ranges from 0.9% to 5.6% of odontogenic inflammatory cysts. There seems to be an imbalance of its occurrence in relation to gender, as is more common in men than in women and in the third decade of life. Occur near the cervical margin of the lateral surface of the root, due an inflammatory process of the periodontal pocket, and is usually associated with the buccal and distal of molars erupted, where there is associated with pericoronitis history. More than half of the reported cases are associated with lower third molars, occurring at a later age than those that relate to the first or second molars and, more rarely, with anterior teeth and globulomaxillary region. The evolution of paradental cyst is slow, because the proliferation of epithelial remnants and cystic formation occur in long time. It is often painless. In some cases, discomfort, halitosis, swelling, acute pain, pain during occlusion, delayed eruption, suppuration and trismus may be present. The paradental cysts associated with the buccal surface and distobuccal third party erupted or partially erupted molars is the most frequent clinical form and usually associated with inflammatory periodontal procedures such as pericoronitis. In this location, except for cases of acute infection, clinical signs are scarce. The

pain may be associated with pericoronitis only semi-enclosed bone. Usually does not cause bone expansion. Absence of bone expansion and not being palpable lesion, its consistency cannot be perceived. The tooth related paradental cyst has pulp vitality[3,12.25].

RADIOGRAPHIC CHARACTERISTICS

The inflammatory periapical cyst reveals radiolucent image with circular or oval shape, bounded by thin radiopaque, continuous and clear line, which is reactive osteogenesis and located in the periapical region (Figure 1). The hard layer is destroyed in the image region. The occlusal radiography, bone cortical inflammatory, medium or large periapical cyst, commonly, is expanded and preserved, but can be destroyed in large cyst with rupture of the regular type, revealing their ends facing the front position. When inflammatory exacerbation suffers periapical cysts, thin radiopaque region of the limiting line shows the image is partially destroyed, as well as the adjacent cancellous bone, according to the X-rays[3].

Figure 1. Radiographic aspects of inflammatory periapical cyst. **Source:** Guttenberg, cited Moraes & Rodrigues (2011)[6].

The periapical radiograph, in a lateral radicular cyst reveals radiolucent, small, with a semilunar shape, bounded by thin radiopaque line in the cyst, which is within the alveolar bone. The part of the cyst located in the periodontal ligament merges with this normal structure, which is also radiolucent. Therefore, the picture is only half of the cyst. Cystic image is located on the lateral surface of the tooth root between the root apex and the dental neck. There is loss of lamina dura along the adjacent root and a rounded radiolucency surrounding the apex of the tooth. The root resorption is common. With the increase in radiolucency flattens out, often as the lesion approaches the adjacent tooth. It is possible a significant growth and lesions can be observed occupying a whole quadrant[3,7].

The lateral root cyst appears as a radiolucent uniloccular lesion with a round or elliptical in the apical area and with a thin edge well defined cortical bone along the side of the root portion[13]. Loss of the hard layer and a source obvious inflammation can be detected without a high index of suspicion. In the event that it is this type of cyst before surgical exploration of radiolucent areas positioned laterally, we recommend performing a full evaluation of the periodontal status and vitality of adjacent teeth[7].

Radiographically, the residual cyst reveals radiolucent image with circular, oval or elliptical, bordered by thin radiopaque line and located in the region of an extracted tooth. In case of a large cyst, the occlusal radiograph shows expansion of cortical bone that is commonly preserved. When the expanded cortical bone is found destroyed, there is a discontinuity characterized by the broken ends facing each cortical (Figure 2), suggesting benign lesion[4]. Can cause tooth displacement and root resorption and external cortical maxillary and mandibular growth may suffer. The cyst may invaginate into the maxillary antrum or moving inferiorly dental nerve canal[4].

Figure 2. Residual cyst in the anterior mandible. **Source:** Domingues & Gil (2007)[3].

According to the radiographic analysis, paradental cyst can vary in your image, depending on the overlap of anatomical structures, presence of infection, size and location of the lesion. Usually the image of the lesion is well-defined radiolucent, cortical and adjacent to the teeth and semi-included, usually located laterally (common distal) limited by a thin radiopaque line and associated with a mandibular third molar partially erupted (Figure 3). The lesion has a semi-lunar, ellipsoid or crescent shape and does not cause bone expansion. Paradental cysts were found in the root bifurcation region at the limit of the buccal amelo-cementum. In buccal location, the cysts from paradental portion show radiolucency, extending above the face of the root of the molar with a thin radiopaque line delimiting the lesion. This circular shape is projected onto the roots and periapical region, changing their shape and making diagnosis difficult. This requires a more careful analysis to view the periodontal ligament space and hard lamina in the region that were overlapped the radiolucent image. In case of paradental cyst located in the distal and mesial, the radiolucent acquires lunate shape, being outlined by radiopaque line on the edge toward the bone[3,16,25].

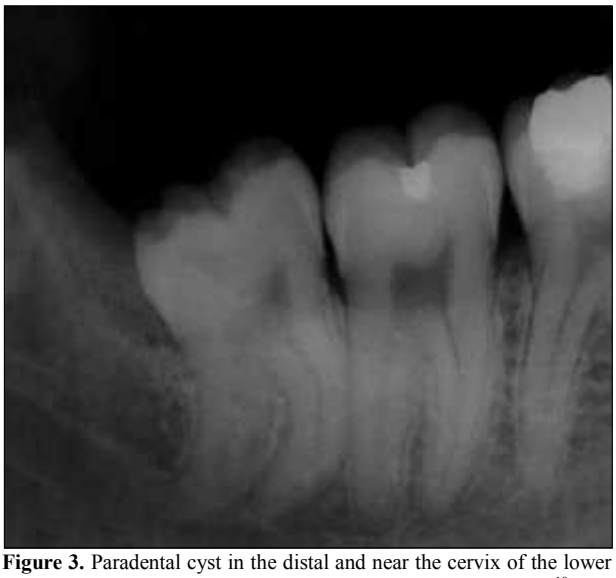

Figure 3. Paradental cyst in the distal and near the cervix of the lower third molar region partially erupted. **Source:** Castro *et al.* (2008)[18].

DIAGNOSIS

The cysts are epithelial odontogenic inflammatory lesions characterized by a pattern of slow, expansive and non-infiltrated growth. This is clear evidence of benign biological nature of these lesions, which can reach considerable size, if not diagnosed in time and treated appropriately[21].

A correct diagnosis is essential for planning the treatment of conditions that compromise the maxillomandibular complex[6]. Given that the number of cystic lesions of the jaws share similar clinical and radiographic features, the diagnosis of odontogenic cysts, usually requires detailed analysis of the clinical, radiological and histopathological findings[10].

Clinical and radiological features support the diagnosis of inflammatory periapical cyst. The radiographic image (radiolucent, with circular or ovoid shape, bounded by continuous thin and sharp line radiopaque) suggests inflammatory periapical cyst, even without bone expansion. However, if there is bone expansion and displacement of adjacent teeth, beyond the radiographic appearance described above, it is likely that the lesion is inflammatory periapical cyst. This may be confused radiographically with periapical granuloma. Radiographic features such as lesion size, continuity and sharpness of the radiopaque line and the intensity of radiolucency image can be used in differential diagnosis. The macroscopic diagnosis of periapical lesion adhered to the light, in the case of extraction, and lesion regression, after shaping and obturation of the root canal, are also effective in the definitive diagnosis of inflammatory periapical cyst[3].

Considering that lateral radicular cyst often presents no clinical manifestations, usually, is discovered by routine radiographic examination of the maxilla or mandible. The radiograph shows radiolucent image with a half-moon shape, located on the lateral surface of the root of a tooth with pulp necrosis, bordered by thin radiopaque line at the margin in contact with the alveolar bone. This cyst can be confused with other odontogenic cysts, especially with the lateral periodontal cyst and the cyst paradental. The analysis of some clinical features, such as pulp test can pinpoint the most likely clinical diagnosis of lateral periodontal cyst and the lateral radicular cyst (Figure 6). Lateral radicular cyst in the pulp of the affected tooth is necrotic; the lateral periodontal cyst, no pulp vitality of the affected tooth. The differential diagnosis can be guided by factors such as etiology, time of surgical removal of the cyst and the incidence jaws. The differential diagnosis with paradental cyst, you should use several clinical features: pulp test, the affected tooth eruption, mucous cyst of the region, adherence and the frequency jaws. The clinical diagnosis must be confirmed even without histopathological analysis, regression when the lateral radicular cyst occurs after shaping and obturation of the root canal of the affected tooth. If the lateral radicular cyst does not disappear after endodontic therapy, should be subjected to histopathological examination after its removal. Microscopically, the capsule is composed of cystic epithelium supported by connective tissue; the stratified squamous epithelial tissue is formed by several layers of cells; the tissue is infiltrated with chronic inflammatory cells, lymphocytes, and plasma cells[3,7,19].

The residual cyst commonly is detected by routine radiographic examination of the jaws, since only sometimes causes bone expansion, i.e., presents clinical manifestations that lead patients to seek care. This examination revealed a radiolucent image, circular or ovoid in area of a missing tooth. These radiographic features added to injury slow, painless and with a history of tooth extraction developments induce clinical diagnosis of residual cyst. However, this can be confused with other bone cysts, especially the primary cyst and odontogenic keratocyst tumor. The differential diagnosis between primary cyst and residual cyst is facilitated if the patient can inform the tooth extraction in image radiolucent area that, if performed, tilts the diagnosis for residual cyst. The differential diagnosis with odontogenic keratocyst, one must pay attention to bone growth, consistency and aspiration of the lesion, the incidence of these cysts in the jaw. If there is extensive bone expansion, consistent with the image size, the consistency is papyraceous or floating in the vacuum of injury is enough liquid citrus harvested, leaning diagnosis for residual cyst. However, definitive diagnosis is determined by histopathology, after removal of the cyst. The capsule is formed by cystic epithelium and underlying connective tissue. The epithelium is stratified squamous, have variable thickness

and may contain corpuscles Rushton. The connective tissue contains cells of chronic inflammation, mononuclear infiltrate and sometimes large numbers of polymorphonuclear neutrophils[3,12].

The clinical and radiographic examination is sufficient to diagnose paradental cyst. Are evidence that the patient is the third partial molar irruption, impacted, and/or mucosa with pericoronitis: bone expansion not exist; radiography reveal a radiolucent image in relation to tooth eruption (3rd impacted molar); localization especially in the root surface for distal or distobuccal and bound by thin radiopaque line in the margin in contact with the bone[3].

The definitive diagnosis of paradental cyst is obtained by adding the clinical-surgical, radiological and microscopic data. Other odontogenic cyst, as dentigerous, the radicular, the lateral periodontal, beyond of the dental follicle, odontogenic tumors such as ameloblastoma and keratocystic odontogenic tumor, and other unusual conditions such as, for example, Langerhans cell histiocytosis, can be included in the differential diagnosis[3,25]. Due to similar clinical and radiographic with other odontogenic cysts, the cysts can be confused paradental, especially with the lateral periodontal cyst and the lateral radicular cyst. Their definitive diagnosis is established by histopathology[3]. Histopathologically, the paradental cysts are invariably lined by non-keratinized stratified squamous epithelium, which appears hyperplastic, backed by a wall of granulation tissue and fibrous tissue containing intense chronic or mixed inflammatory infiltrate. Focus of hemosiderin pigment and cholesterol crystals may be present[25].

METHODS OF DIAGNOSIS OF INFLAMMATORY ODONTOGENIC CYSTS

The diagnostic tests, namely the imaginological tests, are extremely important for the correct diagnosis of odontogenic inflammatory cysts. Radiographs (digital intraoral, panoramic, periapical, occlusal and teleradiography) are often used for global assessment of the state of dentition and diagnosis of various diseases, such as periodontitis, odontogenic and non-odontogenic lesions of the maxilla and mandible, although they have anatomical and geometric limitations, relationship with the visualization and interpretation of the image obtained. For the establishment of a differential diagnosis, are available to the oral health professional methods as conventional computed tomography (CT), magnetic resonance imaging (MRI), ultrasound (US) and cone beam computed tomography (CBCT)[3,4].

Panoramic and periapical radiographs are less precision and accuracy than the cone beam computed tomography (CBCT). The CT has limited use in dentistry, because although it can provide many details in the axial plane shows the drawbacks exposure time and the high dose of radiation to which the patient is subjected. CBCT is a noninvasive method suitable for the examination of tissues stiff and not as efficient for the evaluation of soft tissue. The acquired CBCT image is composed of isotropic voxels; they are equal in all dimensions, eliminates the overlap observed in conventional radiographic images, allowing early visualization of small periapical lesions, even if they are within spongy bone, and full size on the three orthogonal planes. Thus, the cone beam technology gives the professional measuring conditions, and the thickness of the bone, the amount of cancellous and cortical bone, allowing the analysis of lesions at an early stage of development. CBCT is a versatile method of diagnosis. Facilitates differentiation and planning, streamlining the treatment and follow-up of periapical lesions[3].

In addition to indicating the presence of inflammatory periapical lesion, the use of CBCT is of proven efficacy[11,27,29] for the diagnosis differentiation between inflammatory granuloma and periapical cysts. It is a precise technique preoperative diagnostic periapical inflammatory lesion, as images with fewer shades of gray (negative values), with a darker area homogeneous; with a lower density of the surrounding area, so areas less dense fluid into a cavity containing indicate cystic lesions. Areas with high number of shades of gray (positive values) with similar density to surrounding tissue, but with a sharper radiolucency suggests the presence of granulomas[6].

TREATMENT

All odontogenic cysts, with the exception of inflammatory periapical cyst and lateral radicular cyst, should be treated with surgical intervention. The periapical cystic lesions are usually treated by conservative endodontic treatment (periapical curettage) or surgical treatment (enucleation, marsupialization and decompression). In lateral radicular and inflammatory periapical cysts, surgery is indicated only if the lesions do not regress after removal of odontogenic infection intra-canal the affected tooth. In case of residual cyst, surgery is the only option[1,3].

According with Petterson *et al.* (2000)[28] the cysts of the jaws can be treated surgically by one of the following basic techniques: enucleation, marsupialization combination of the two procedures in steps or enucleation with curettage.

Research conducted by Silva *et al.* (2002)[29], to assist the dental professional in choosing the appropriate surgical treatment for different types of cysts shows that in cysts of the jaws, the treatment is purely surgical nature, therapeutic enucleation, marsupialization of and can be performed decompression.

The current recommended treatment for inflammatory periapical cyst consists of shaping and obturation of the root canal without immediate surgical intervention. However, extensive lesions in restorable teeth have been treated successfully by conservative endodontic treatment when accompanied by biopsy and marsupialization or fenestration decompression. Radiographs of control, every four months, must be made, before they had total repair of tissues. Surgical removal (enucleation cystic) is indicated when the periapical lesion does not regress or increased in size during the observation period. Surgery should not be limited to the removal of the cyst; It will hold the tooth engaged apicoectomy with or without back-filling. Still, when the tooth involved an extensive prosthetic crown, or a root pin, especially when removing the crown or pin may cause fracture of the remaining tooth structure, it is advisable to perform the paraendodontic surgery[3].

The lateral radicular cyst, initially, it should be treated by endodontic therapy, ie, shaping and obturation of the root canal of the affected tooth so as to eliminate the infection within that channel. If, after the treatment, the cysts do not regress, it will be removed by surgery. All inflammatory focus in the area of a lateral radicular cyst should be eliminated. The material should be sent for biopsy and strict monitoring of at least one to two years should be performed[3,7,26].

Treatment of residual cyst is usually surgical removal by different techniques, depending on the size and location of the lesion. Cysts to small and medium sized easy access and without compromising patient health, in general, the first option is cystic enucleation. The residual cyst is large and there is a possibility of bone fracture during their enucleation, it is advisable marsupialization of the lesion, it is introducing a drain for draining the cystic fluid. Without the pressure of the cyst on the bone, its size reduction occurs by peripheral bone formation to injury and there is no risk of bone fracture and the residual cyst can be removed[3,4].

Treatment of paradental cyst when associated with third molars is the excision of the lesion, together with the removal of the tooth. If paradental cyst affects the first or second molars, the treatment consists of enucleation of the lesion with preserving the tooth involved. But, the affected tooth is usually extracted along with the cyst, as is often impacted in the second molar[3,25].

Regarding to the treatment of cysts and tumors, Neville *et al.* (1998)[7] argue that all cysts and odontogenic and non-odontogenic tumors can mimic the appearance of a residual periapical cyst thus suggest that all these cysts and tumors should be surgically excised for complete evaluation of ignition source.

However, any surgical procedure should be thoroughly evaluated and planned. Each surgical technique has its correct indication. The professional must properly assess the type of cyst, its shape and location, the degree of expansion and involvement with the underlying structures, ie, the size of the lesion, the contents of the cystic cavity and the general condition of the patient. We must always consider the extent and anatomical relations of the cystic process, thus guiding the surgical technique and the precautions to be taken. The success of each case will depend on a treatment plan, beyond the expertise of the surgical technique and orofacial anatomy[6,15,27].

Among the objectives of surgical treatment in addition to immediate or delayed removal of the cyst, whenever possible, one should look to the structure and vitality of the teeth, as well as preserve the integrity of anatomical structures: nasal cavity, maxillary sinus, neurovascular bundles and maxillary continuity. To minimize the consequences on the region affected by the cyst, reconstructive techniques of the bone defect should be considered[3].

PROGNOSIS

Some inflammatory periapical cysts are reversible only with endodontic therapy. The prognosis is also good, when the inflammatory periapical cyst is removed by surgery because of periapical tissue repair occurs. The reason for the regression of some inflammatory periapical cysts after modeling and root canal filling without periapical surgery is unknown[4]. The prognosis of lateral radicular cyst is good, because this cyst has no tendency to relapse. Regression of the lesion after endodontic therapy may occur. This suggests that the lesion was not granuloma and cyst (regresses cavity granuloma, cysts not). It is common bone repair cystic region after surgical removal of this type of cyst[3].

The residual cyst has no tendency to relapse, so it has a good prognosis. After excision, bone healing usually occurs in the cyst region. Exceptionally, the epithelium can give rise to squamous cell carcinoma, malignant tumor[3].

Authors showed the fact that incomplete enucleation epithelium can develop a residual cyst after months to years after treatment. If the original radicular cyst, residual cyst or remain untreated, their continued growth can cause significant destruction and weakening of the maxilla or mandible. In root and residual cysts treated fairly, in general, there is bone repair[19,20].

Whatever the case of paradental cyst recurrence is not common. This type of cyst has an excellent prognosis; it has no tendency to relapse when the lesion is completely removed. After surgical removal, is common bone repair occurs in cystic region[3,27]. The prognosis of all inflammatory cysts are good. These cysts usually do not recur after appropriate treatment. Fibrous scarring may occur, especially when both cortical are broken. Rare cases of developing squamous cell carcinoma have been reported in periapical cysts. The squamous cellular carcinoma can

be occasionally originate from the epithelial lining of radicular cyst or other odontogenic cysts. Thus, even in the absence of symptoms, treatment is required for all intra-osseous changes persistent[7,19,25].

4. DISCUSSION

As already stated above, the odontogenic inflammatories cysts are lesions that depend on a focus of endodontic, periodontal or pericoronal infection of low virulence and long term, to proliferate[3,4,7]. They grow slowly from the remaining epithelial Malassez or a pre-existing periapical granulomas. The source of inflammation may be a necrotic pulp or periodontal disease[5,6,10]. They often occur within the bone, being very rare in extraosseous region, where he was observed and therefore must be included in the differential diagnosis of peripheral lesions of the mandible[2,4]. Although benign, depending on size, odontogenic inflammatories cysts can become destructive, because they are frequent incidence and represent a major cause of bone destruction in the jaw and mandible[2,3].

Clinically, odontogenic inflammatories cysts are asymptomatic, being incidental radiographic findings. Appear more often in the maxilla[2,12,22], but residual cysts were found in both jaws, most commonly in the jaw segments[15,18,23]. Show a pattern of slow, expansive and non-infiltrated growth, which clearly shows the biological nature of these benign lesions[3,7,23]. If the cyst reaches a size 20-35 mm in diameter, or in case of exacerbation, signs and symptoms appear, such as destruction of the outer cortical bone, toothache, hard, painless swelling, increased tooth mobility, sudden extrusion, light sensitivity, being possible mobility and displacement of adjacent dental elements also occur[3,22,24]. Lesions that persist or increase in size are probably secondarily infected[26]. Regarding gender, there is predilection for males[12,19,20] but in the case of radicular cyst, there was a higher prevalence in women[10,2,23]. Regarding age, no age group with the highest incidence of inflammatory periapical cyst[19,20,22]. The occurrence of radicular cysts, it was observed that the third and fourth decades of life have highlighted the prevalence[14,24,25] and radicular cysts and residual cysts are rarely found in children[20,25].

The odontogenic inflammatories cysts are diagnosed by imaging. Radiographs (digital intraoral, panoramic, periapical, occlusal and teleradiography) are often used for global assessment of the state of dentition and diagnosis of various diseases, but present anatomical and geometric limitations with respect to visualization and interpretation of the image obtained. The establishment of a differential diagnosis can be obtained with conventional CT, MRI, US and CBCT, a technique preoperative accurate and versatile diagnosis, which facilitates the differentiation and planning, streamlining the treatment and follow-up of periapical lesions[4,6]. The CBCT, besides indicating the presence of apical periodontitis, has proven efficacy[11] diagnosis for differentiating granuloma and periapical cysts

The periapical cystic lesions are usually treated by conservative endodontic treatment (periapical curettage) or surgical treatment (enucleation, marsupialization or fenestration and decompression)[3,29]. Silva *et al.* (2002)[29] emphasized that the cysts of the jaws, the treatment is purely surgical nature, therapeutic enucleation, the marsupialization and decompression can be performed. To Domingues & Gil (2007)[3] all odontogenic cysts, with the exception of inflammatory periapical cyst and lateral radicular cyst, should be treated with surgical intervention. In lateral root and inflammatory periapical cysts, surgery is indicated only if the lesions do not regress after removal of intracanal odontogenic infection of the affected tooth[3,7,28]. In the case of residual cyst or paradental cyst surgery (cystic enucleation or marsupialization) is the only option[1,4,27]. Whereas all cysts and odontogenic and non-odontogenic tumors can mimic the appearance of a residual periapical cyst, Neville *et al.* (1998)[7] suggest that all these cysts and tumors should be surgically excised for complete evaluation of ignition source. Given that every surgical procedure should be carefully evaluated and planned, each surgical technique has its correct indication. The professional need properly evaluate the cyst according to: its type, shape, location, degree of expansion and involvement with the underlying structure. Thus, the size of the lesion, the contents of the cystic cavity and the general condition of the patient, leading to immediate or delayed removal of the cyst, and conservation of the structure and vitality of teeth as well as the preservation of the integrity of anatomical structures in the treatment of odontogenic inflammatories cysts should be considered[6,16,28].

5. CONCLUSION

The inflammatories odontogenic cysts are interosseous lesions that affect the regions of the maxilla and mandible. Although asymptomatic and benign, due to its continuous increase, these lesions can become destructive, because they affect and infect the adjacent bone and thus should be treated appropriately. In this sense, it is crucial for diagnosis and treatment planning usually requires a detailed analysis of the clinical, radiological and histopathological examinations.

In dentistry, early detection and accurate diagnosis of the affected by inflammatory, neoplastic or cystic lesions, odontogenic fabric are of paramount importance for successful treatment. Therefore, the dentist must have knowledge of the biological and histological behavior of odontogenic cysts and their frequency to ensure early detection, accurate diagnosis and proper treatment.

The treatment of lateral inflammatory periapical cysts or imply conventional endodontics or alveolar curettage, post-extraction does not listen when regression of post endodontic lesion or whether it is a paradental cyst. With this, we avoid the permanence of an odontogenic lesion, which may become a residual cyst, which may present expansive and destructive potential, if not properly removed surgically.

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ORAL MANIFESTATIONS OF SYSTEMIC DISEASES INFECTIOUS IN CHILDREN

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ABSTRACT

Every day is more common the presence of children in the dental office. For this reason, it is extremely important that the dentist is equipped to perform the treatment in dentistry and who has knowledge of systemic health, since children do not always manage to express themselves about what they feel. Based on this premise, this study aimed to review the literature on oral manifestations of systemic diseases that may signal to dental professionals about the need for referral of patients for medical care.

KEYWORDS: Pediatric dentistry, infection, disease.

1. INTRODUCTION

The oral manifestations are very common and may be the first signs and symptoms in diseases or systemic changes arising from some therapies. These oral lesions may indicate the start or progression of a disease and therefore may function as an early warning system for some diseases[1]. The aim of this study was to review the literature on oral manifestations of systemic infectious diseases in childhood.

2. MATERIAL AND METHODS

This study was based on a literature review, conducted between March and May 2014. We performed a consultation to books and periodicals available on the Internet, and the University Severino Sombra library. The keywords used in the search were childhood infectious systemic diseases, Measles, Rubella, Roseola, Varicella and Scarlet as well as its synonyms. The inclusion criteria for studies were found to direct approach to the subject, containing description of the disease, manifestations and treatment. Studies reporting the use of other diseases or who had not anything to add at work were excluded. Soon after, we sought to study and understand each disease individually, and of them, their oral manifestations and treatment.

3. LITERATURE REVIEW

SCARLET

According to Vranjac (2007)[2], the scarlet fever is an acute infectious disease caused by a bacterium called *Streptococcus beta hemolytic* of the group A. The scarlet comes from a reaction of hypersensitivity (allergy) to the bacterium produces substances (toxins) and can thus cause different disease in every person. It affects more children, is contagious through saliva droplets or infected secretions has incubation time of 2 to 4 days. A throat infection is a disease that appears associated with scarlet fever. As an initial oral manifestation is sore throat, white tongue and furred and with the progress of the disease to her lip and tongue tip is to look raspberry due to increased papillae. Can be early or late complications such as otitis media, sinusitis, laryngitis, meningitis, and other, being worse after curing the disease and rheumatic fever (heart valve damage), and glomerulonephritis (kidney damage that may evolve to renal failure). The diagnosis is based on clinical observation, but must be confirmed by a swab for throat swab. The treatment is penicillin; erythromycin is used in patients allergic to penicillin.

Barreto & Gonçalves (2012)[3], reported that the scarlet is an acute infectious disease caused by *Streptococcus pyogenes,* more frequent in childhood. The oral manifestations are hypertrophied tonsils,

usually covered with yellow exudate. The tongue has a white coating with red spots that match swollen tongue papillae. After a few days the tongue resembles a raspberry. Even without treatment children improve after two weeks. To avoid complications it is recommended the use of penicillin; Alternatively amoxicillin, a synthetic penicillin, is used orally. In cases of allergy, the erythromycin can be used.

Shiramizo (2012)[4], reported that the Streptococcal Pharynx (Scarlet), has a higher incidence in children aged 5 to 15 years, and is uncommon before the age of three. The scarlet is more common in winter and early spring with incubation period 2-5 days with eradication of the transmission after 24 hours of antibiotic, which is essential to prevent complications, improve symptoms and reduce transmission. The oral manifestations are sore throat, pain on deglutition, redness of the pharynx and tonsils, exudate, anterior cervical lymphadenitis, submandibular lindafenite, petechiae on the palate, uvula edema and hyperemia. Diagnosis: culture of oropharynx, collecting swab obtained from both tonsils and the posterior pharyngeal wall, as rapid test "QuickVue Strep A test", makes the detection of streptococcal antigens almost immediate. Streptococcal pharyngitis is a self-limiting disease with improved symptoms in 2-5 days without antibiotic. Recommendation: amoxicillin, orally; for allergy sufferers it is recommended to use clarithromycin, azithromycin and clindamycin. Indications: suppurative complications (abscesses) and nonsuppurative (rheumatic fever with carditis, post streptococcal glomerulonephritis, and others).

VARICELLA

Carvalho & Martin (1999)[5], have studied that varicella is caused by a herpes virus infection, varicella-zoster virus. Most cases occur in children under the age of 10, in late winter and early spring. Contagion occurs from 2 days before onset of rash, respiratory route, becoming less and less contagious. The incubation period is usually 14-16 days, but can occur from the 10th day or the 21th day. The diagnosis is usually clinical, with the appearance of vesicular lesions characteristic, on successive days, which evolve to crusts in a few days. The varicella in two situations is of great importance: when it affects children hospitalized because spreads quickly and in immunosuppressed, which is serious and sometimes fatal. After curing the virus can remain dormant, and its reactivation results in the framework of shingles. In children, the symptoms are unlike the adult, rarely leads to painful conditions, being easy to handle. In most cases of varicella and zoster in children, treatment is symptomatic. Pruritus in varicella can be alleviated by the local use of calamine or with the use of antihistamines orally. The use of antibiotics is required only in those cases where impetiginisation occur. The antibiotic of choice is the group of penicillins, such as penicillin V and Amoxycillin; the first generation of cephalosporins such as cephalexin or cefadroxil could be indicate, or an antibiotic macrolide, such as erythromycin, clarithromycin or azithromycin.

Vranjac (2003)[6] reported that, varicella (chickenpox) is a highly contagious disease caused by the varicella-zoster virus and mainly affects, under 15 years of age. Can occur throughout the year, but there are an increasing number of cases of late winter until spring. The period of the transmission is initiated two days before the appearance of blisters and goes to the phase crust. The incubation period ranges from two to three weeks, with an average of 14 to 16 days.

Castiñeiras *et al.* (2003)[7], stated that, varicella (chickenpox) is a highly communicable, acute infectious disease caused by the varicella-zoster virus. The disease is most common in children between one and ten years. The transmission of the virus occurs primarily by respiratory secretions (saliva droplets, sneezing, coughing) from an infected person or by contact with fluid from the blisters. The first lesions commonly appear on the head or neck, but as they evolve quickly arise new lesions on the trunk and limbs and also in mucous membranes (oral, genital, respiratory and conjunctival). Several antiviral drugs (acyclovir, valacyclovir, famacyclovir) are available for the specific treatment of varicella.

Neville *et al.* (2009)[8], reported the symptoms of varicella, which begins with malaise, pharyngitis, rhinitis, and then comes the characteristic itchy rash, the rash begins on the face and trunk, going to the ends. The oral and perioral manifestations are quite common and may precede skin lesions. The edge of the vermilion of the lips and palate are the places most frequently involved, followed by the buccal mucosa. The lesions begin as white-opaque vesicles 3 to 4 mm, which rupture to form ulcers 1 to 3 mm. The number and prevalence of oral lesions are related to the severity of the infection extra oral. In mild cases, oral lesions are present in approximately one third of affected individuals. Only one or two oral ulcers are evident and heal 1 through 3 days. Already in severe cases, patients have lesions which persist up to 30 for 5 to 10 days.

Kalil (2013)[9], studied that the varicella, commonly known as chickenpox, is a disease caused by the herpes zoster virus, which affects mainly children. The most common form of transmission is through contact this viral particle - present in saliva, sneezing, coughing or even talking - with inhaled or oral mucosa of the individual. Transmission may also occur through direct inoculation. The patient develops bubbles and in general, the course content and the reddish edges. These bubbles appear on the skin of the whole body, including the scalp, mouth, and other mucous leather. The diagnosis is

primarily clinical, although there is the possibility of serological confirmation. In small children, no treatment is indicated, even baths permanganate, which, barely diluted known can cause skin burns. The ideal is to make proper skin hygiene with soap and water, during the usual bath and cut your fingernails child so she does not scratch the vesicles and run the risk of infect them. Specific treatment with medication, which is acyclovir, is only indicated in adults or patients over 12 years as the complication rate of the disease is usually higher.

MUMPS

Camargo & Mello (2001)[10] showed that the mumps is a transmissible, usually benign acute infectious disease course. It is characterized by inflammation of the parotid gland, accompanied by pain and swelling. This disease can be widespread or systemic, due to the frequent involvement of the submandibular and sublingual glands, gonads, pancreas, nervous system and other organs. The mumps begins with nonspecific infection with low-grade fever, malaise, myalgia, arthralgia, and earache. After a few hours or days, is evident in the swelling of the parotid, one or both sides, with displacement of the pinna and effacement of the mandibular angle. There spontaneous pain that intensifies the opening of the mouth and even more, the intake of fatty foods. This phase lasts swollen salivary glands from 7 to 10 days. The main complications of the disease are: acute pancreatitis, orchitis, epididymitis, oophoritis, meningitis and meningoencephalitis. The virus is transmitted by direct contact with an infected person through droplets of secretion from oropharynx. The transmission period is greater than 2 days before until 2 days after onset of swelling of the parotid, although the virus may be isolated from the saliva to 6 days before to 9 days after the glandular involvement. For practical purposes, the patient can not be considered infectious 9 days after the start. The incubation period is in average 16 to 18 days, ranging from 12 to 25 days. It mainly affects children of school age, 5-15 years, without distinction of sex. Usually occurs in winter and early spring.

Campos (2010)[11] reported that the mumps, infectious parotitis, or mumps epidemic parotitis is an infectious disease whose etiologic agent is a virus of *paramyxoviriadae* family, genus *Rubulavirus* (*paramyxovirus*), and its main clinical manifestation of inflammation parotid gland (the largest of the three salivary gland pairs) gland inflammation and possibly of other major salivary glands, promoting a swelling of the face and neck, it may be unilateral or bilateral. The incubation period is 12 to 25 days. The infection may result in subclinical or asymptomatic manifestations. The main signs and symptoms are: swelling of the salivary glands, especially the parotid gland, fever, nausea, sweating, body aches, anorexia, headache, lasting 7 to 10 days, usually with spontaneous resolution. There is no specific treatment for this disease, only treat the symptoms.

The Sanofi Pasteur Group (2013)[12], pointed out that the mumps is a viral disease characterized by fever, swelling and tenderness of one or more salivary glands, usually the parotid and eventually the sublingual or submaxillary glands. Mumps virus belongs to the genus *Rubulavirus*, family *paramyxoviridae*. The mumps usually affects children. The mumps is spread from person to person by coughing and sneezing and by direct contact with saliva and secretions from the nose and throat of infected individuals. Contagion happens three days before and 4 days after onset of symptoms. Symptoms usually manifest 16 to 18 days after infection. Approximately one third of those infected are asymptomatic. There is no specific treatment for mumps. To relieve symptoms, it is recommended the use of painkillers and regular oral rinse.

RUBELLA

Kajiyama *et al.* (1982)[13], stated that rubella is also called German Measles is an infectious disease, with minimal or absent prodromal symptoms, a rash of three days' duration and generalized lymphadenopathy, particularly in retro-surricilar, suboccipital and cervical chains. The transmission occurs through nasopharyngeal secretions from infected individuals, droplets of mucus and saliva, it is also possible can be isolated from the blood and urine from feces, and the transmission can also be by air. The transmission period is approximately one week before and at least four days after the onset of the rash. Its manifestations begin on the face, in small light red spots that spread throughout the body, over time increase the diameter and become macule papules. Rubella mitigated or absent in catarrhal phenomena also occur.

Castiñeiras *et al.* (2006)[14], argued that the rubella is a respiratory immune-preventable infectious disease transmission. The infection is caused by the rubella virus which produces mild or absent manifestations. Usually has a benign evolution is more common in children and can occur in adults. Rubella occurs only once in life. The transmission period ranges from one week before until seven days after the onset of rash (red spots on the skin), being the period stains the highest risk of transmission. In 50% of cases the disease is asymptomatic. The clinical manifestations appear between 12 and 23 days after infection. The spots begin to evolve in the face and the rest of the body gradually (usually disappear in less than 24 hours), there is the swollen lymph nodes in the neck and low fever. There is no specific treatment for rubella; can be used to control symptoms of antipyretics and analgesics, with the exception of medicines

containing acetylsalicylic acid at risk of bleeding occur, because the rubella lowers the number of platelets.

Mercatelli (2013)[15], described the rubella as an infectious disease caused by togaviruses. She also explains that togaviruses when reaches pregnant women in the first three months of pregnancy can cause serious problems such as fetal death. The transmission period is relatively long, since the incubation period begins 10 days before symptoms appear and extends for 15 more days of healing. The characteristic of the disease are reddish patches that appear first on the face and behind the ears, and then spreads throughout the body. Also may have headache, discomfort when swallowing, body aches, joints and muscles, runny nose, appearance of nodes and fever. The infection occurs commonly through the airways when people aspires droplets of saliva or have contact with nasal secretions of someone who is infected with the togaviruses. No specific medication exists to rubella, seeing that it is a virus. However, the treatment is to relieve symptoms, such as antipyretics and analgesics.

ROSEOLA

Aires (2012)[16] reported that roseola is common among infants and preschool children is caused by *Herpesvirus hominis* type VI. The diseases have typical evolution: three to four days of high fever accompanied by irritability that precedes the appearance of maculopapular rash. The appearance of the eruption happens is when drastic decline of fever. There is no specific treatment for the disease, and his confirmation is used serologic testing.

Ribeiro *et al*. (2012)[1] first studied the sudden rash or roseola is a common childhood disease and has benign, self-limited evolution is caused by human herpes virus 6 and 7, is characterized by high fever a few days with the appearance of skin rashes when fever subsides. In the oral cavity appear erythematous macules and papules. The regions of the soft palate and uvula can show ulcerated with salient points rosy due to hyperplasia of lymphoid follicles in the submucosa.

Fernandes (2013)[17] observed that the sudden rash (roseola) is a contagious disease caused by viruses - human herpes virus type 6 (2/3 of cases) and 7 (1/4 cases), and echovirus 16, among others. Children are more affected than adults. After incubation period of 5-15 days, appears high fever lasting about 3-4 days. Tracking the fever may arise: runny nose, cough, headache ("headaches"), hyperemic oropharynx without exudates ("red throat without white spots"), vomiting, diarrhea and enlarged lymph nodes ("swollen glands volume") cervical. As treatment, the use of antipyretics (acetaminophen/ ibuprofen) and enhance the intake of fluids during the febrile period. Provide a baths cooling, if necessary.

MEASLES

McDonald & Avery (1986)[18], stated that measles is an acute contagious viral disease, which mainly affects children and typically occurs in epidemic form. The route of transmission is the respiratory tract after transmission by direct contact or aerosol infection. After incubation period of 8 to 10 days, develop malaise, fever, cough, conjunctivitis and photophobia, and finally maculopapular skin lesions appear on the face, which spread to the trunk and extremities. Oral lesions, termed "Koplik spots" are prodromal manifestation of the disease and typically occur two to three days before the development of cutaneous lesions. It is reported that these Koplik spots occur in 95 percent of patients with measles; they develop characteristically in the oral mucosa and appear as small blue-white blotches, surrounded by bright red border, then increase the number and coalesce into small patches. As a rule, the Koplik spots disappear by the time the skin lesions appear. The only treatment for the disease is rest and support measures.

Penna *et al.* (2002)[19] reported that measles is an acute infectious disease, viral in nature (an RNA virus) which belongs to the genus Morbillivirus, family *paramyxoviridae*. It is extremely contagious and done directly from person to person through the nasopharyngeal secretions expelled by coughing, sneezing, speaking or breathing. Measles is very common in childhood. The evolution presents three well-defined periods: a) prodromal or catarrhal period: lasts 6 days; early in the disease arises fever, accompanied by productive cough, runny nose and seromucoso eye pain, conjunctivitis and photophobia. The lymph nodes are slightly increased in the cervical region and sometimes the abdominal give painful reactions in the abdomen. In the past 24 hours the period arises at the time of the premolars, in gemiana region, the signal Koplik - small white patches with erythematous halo, considered pathognomonic sign of measles; b) Exanthematous period: accentuation of all the symptoms already described, with significant depletion of the patient and the appearance of the characteristic rash occurs. The injury is exanthematous maculopapular, reddish in color, with distribution in cephalocaudal direction. c) Period of convalescence: a stain become darkened and appears fine scaling, remembering flour. Its incubation period usually lasts 10 days from the date of exposure to onset of fever, and about 14 days before rash onset. It is transmissible from 4 to 6 days before the onset of the rash until 4 days after. The diagnosis is clinical, epidemiological and laboratory. The treatment is symptomatic, with use of antipyretics, oral hydration, nutrition therapy to promote breastfeeding and proper hygiene of the eyes, skin and upper airway may be used.

According to Ribeiro *et al.* (2012)[1], measles is a systemic, acute exanthematous disease one of the most contagious of all communicable diseases. It is caused by the family *paramyxoviridae* and the genus *Morbillivirus* virus. In the oral cavity, one pathognomonic lesion of measles occur; it is known as Koplik spots. They precede skin manifestations 1-3 days. Are small bluish white spots that are formed mainly in the cheek, near the opening of the mucosa Stenon channel and are surrounded by a bright red halo. These blemishes increase in number and coalesce into plaques, generalized inflammation and swelling, ulcerations in various locations may occur (gingiva, palate and throat).

Vranjac (2013)[20], maintains that measles is a viral disease, respiratory transmission is highly contagious. Symptoms usually appear 7-18 days after exposure to a case and include: fever, runny nose, cough, conjunctivitis, and red patches all over the body. The virus can be transmitted to 5 days before and 5 days after the rashes.

4. DISCUSSION

Vranjac (2007)[2] and Barreto & Gonçalves (2012)[3], agreed that scarlet fever is an acute infectious disease caused by *Streptococcus pyogenes*. Vranjac (2007)[2], states that children are the most affected because Shiramizo (2012)[4], says the focus is on children and adolescents up to 15 years, and is uncommon before age 3 years old. Shiramizo (2012)[4], said incubation time of the disease is 2 to 5 days, since Vranjac (2007)[2], 2 to 4 days. Vranjac (2007)[2] and Shiramizo (2012)[4], agreed that one of the initial symptoms are sore throat. Vranjac (2007)[2] and Barreto & Gonçalves (2012)[3], agreed on oral manifestations of the disease are tongue with white coating with red spots, with swollen buds, leaving her with raspberry aspect. Barreto & Gonçalves (2012)[3], added that the tongue is covered with yellow exudate.

Carvalho & Martins (1999)[5]; Vranjac (2003)[6]; Castiñeiras *et al.* (2003)[7] and Kalil (2013)[9] stated that varicella is an infection caused by the varicella-zoster virus. Carvalho & Martins (1999)[5] reported that most cases occur in children under the age of 10, already Vranjac (2003)[6] said that affects younger than 15 years, also disagreeing with Castiñeiras *et al.* (2003)[7] reporting that is common among children one to ten years and Kalil (2013)[9] stated that only affects children, without specifying age. Carvalho & Martin (1999)[5] and Vranjac (2003)[6] agreed that the transmission period beginning two days before the appearance of vesicles. All the authors consulted, agree that the varicella is spread by the respiratory route, as Kalil (2013)[9] that also commented that the virus is present in saliva, sneezing, coughing or even talking, so having regard to the oral mucosa of the patient. Neville *et al* (2009)[8], affirmed that the oral manifestations, lesions begin as white-opaque vesicles that rupture and form ulcers already Kalil (2013)[9] said the injuries have clear content and red border. Carvalho & Martins (1999)[5] and Cartiñeiras *et al.* (2003)[7] reported that the varicella has no specific treatment for the disease but to the symptoms. Kalil (2013)[9] said that in small children no treatment is indicated. Castiñeiras *et al.* (2003)[7] and Kalil (2013)[9], the agreed statement of Acyclovir as treatment indication, however Carvalho & Martins (1999)[5] indicated drug such as: cephalosporins, (cephalexin or cefadroxil) and macrolides (clarithromycin or azithromycin).

Carmargo & Mello (2001)[10], Campos (2010)[11] and Sanofir Group Pasteur (2013)[12] agreed that the Mumps is an acute infectious and transmissible disease, but only Campos (2010) 11 and Sanofir Group Pasteur (2013) 12 agree between Mumps itself that has a virus as the etiological agent of Paramyxoviriadae family, genus Rubulavirus (paramyxovirus). Carmargo & Mello (2001) 10, Campos (2010) 11 and Sanofir Group Pasteur (2013) 12 agreed that such oral manifestation of the disease have, inflammation of the parotid, promoting swelling of the neck as a general manifestation everyone agreed it causes fever and malaise. Carmago & Mello (2001)[10] supplement adding that with the swelling of the parotid there is a displacement of the pinna and effacement of the mandibular angle, causing pain and mouth opening in the intake of fatty foods, also said that this phase of swollen glands lasts from 7 to 10 days. Regarding the transmission of the virus, Carmargo & Mello (2001)[10] and Sanofir Group Pasteur (2013)[12] agree that is made by droplets of secretions of infected people, and disagree with respect to the time of transmission, Camargo & Mello (2001)[10] said it was two days before until two days after the onset of edema already Sanofir group Pasteur (2013)[12] said to be three and four days earlier after onset of the disease. About the period of incubation of the disease, Carmago & Mello (2001)[10] and Campos (2010)[11] agreed be 12 to 25 days. Campos (2010)[11] and Sanofir Group Pasteur (2013)[12] agreed that mumps has no specific treatment, just to the symptoms of the disease.

Kajiyama *et al.* (1982)[13], Cartiñeiras *et al.* (2006)[14] and Mercatelli (2013)[15] agree that rubella is an infectious disease and is transmitted by nasopharyngeal secretions, but Kajiyama *et al.* (1982)[13] also stated that can be blood isolated from urine and faeces. According to the period of communicability Kajiyama *et al.* (1982)[13] said it was a week before to four days after onset of the rash of injuries, contradicting Cartiñeiras *et al.* (2006)[14], who stated that the transmission period will a week before to seven days after the appearance of the spots. Kajiyama *et al.* (1982)[13] also diverge Mercatelli (2013)[14] who claimed to be ten days prior to fifteen days after the cure. Kajiyama *et al.* (1982)[13], Cartiñeiras *et al.* (2006)[14] and

Mercatelli (2013)[15] agree that as a manifestation of the disease have red spots that will begin on the face and spreading to the body, also having increased of nodes in the neck and fever. Kajiyama *et al.* (1982)[13] also stated that mitigated or absent in Rubella catarrhal phenomena occur. Cartiñeiras *et al.* (2006)[14] said that the clinical manifestations appear between 12 and 23 days after infection, and the stains of the face and body usually disappear within 24 hours. Mercatelli (2013)[15] said that beyond these manifestations also have headache, discomfort when swallowing, body aches, joints and muscles, and runny nose. Cartiñeiras *et al.* (2006)[14] and Mercatelli (2013)[15] agreed that rubella has no specific treatment, medicines to control symptoms, such as antipyretics and analgesics may be used as well. Cartiñeiras *et al.* (2006)[13] further exalting should be avoided drugs containing acetylsalicylic the risk of bleeding, since the disease diminishes the number of platelets.

Ribeiro *et al.* (2012)[1] and Fernandes (2013)[17] agreed that roseola is a disease caused by the human herpes virus 6 and 7 already Aires (2012)[16] says only be caused by the human herpes virus 6. Aires (2012)[16] said roseola is common among infants and preschoolers, as Ribeiro, *et al.* (2012)[1] affirmed that is common in childhood, and Fernandes (2013)[17] says that it is a disease that affects more children than adults. Aires (2012)[16] and Ribeiro et al. (2012)[1] agree that the disease causes high bluegrass and when it happens its decline come the maculopapular rash (skin rash). Ribeiro *et al.* (2012)[1] said that in the oral cavity appear erythematous macules and papules, since the region of the soft palate and uvula are shown protruding ulcerated with rosy points due to hyperplasia of lymphoid follicles in the submucosa. Aires (2012)[16] and Fernandes (2013)[17] agree that there is no treatment for roseola, Fernandes (2013)[17] complements saying that you can use antipyretics and enhance the intake of fluids for symptoms, baths and cooling if required.

McDonald & Avery (1986)[18], Penna *et al*. (2002)[19] and Ribeiro *et al*. (2012)[1] agreed that measles is an acute viral infectious disease of nature, Penna *et al*. (2002)[19] and Ribeiro *et al*. (2012)[1] further stated that their virus belonging to the genus Morbillivirus, family *paramyxoviridae*. McDonald & Avery (1986)[18] and Penna *et al.* (2002)[19] agree that the transmission is via nasopharyngeal, and the disease is most common in childhood. The oral manifestations spots measles McDonald & Avery (1986)[18], Penna *et al.* (2002)[19] and Ribeiro *et al.* (2012)[1] agree is the "Koplic spots" that are pathognomonic sign of the disease are bluish white circled in red, erythematous-halo. McDonald & Avery (1986)[18] and Ribeiro *et al.* (2012)[1] agreed that Koplic stains disappear before the skin lesions appear. Penna *et al.* (2002)[19] said that lymph nodes are swollen in the neck and sometimes the abdominal give intentional reactions in the abdomen. The transmission period affirmed that will 4-6 days before the appearance of the rash until 4 days after, now, Vranjac (2013)[20] said that the virus can be transmitted to 5 days before and 5 days after the rash. McDonald & Avery (1986)[18] and Penna *et al*. (2002)[19] agree that the specific treatment for measles is existent but McDonald & Avery (1986)[18] recommends rest and support measures for treatment since Penna *et al*. (2002)[19] said that antipyretics, oral hydration, nutritional therapy and hygiene of eyes, skin and upper airways can be used.

5. CONCLUSION

Based on the literature reviewed conclude that it is essential for the Dental Surgeon knowledge about infectious systemic diseases contributing to the alert and diagnosis for early treatment thus avoiding complications and sequelae.

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