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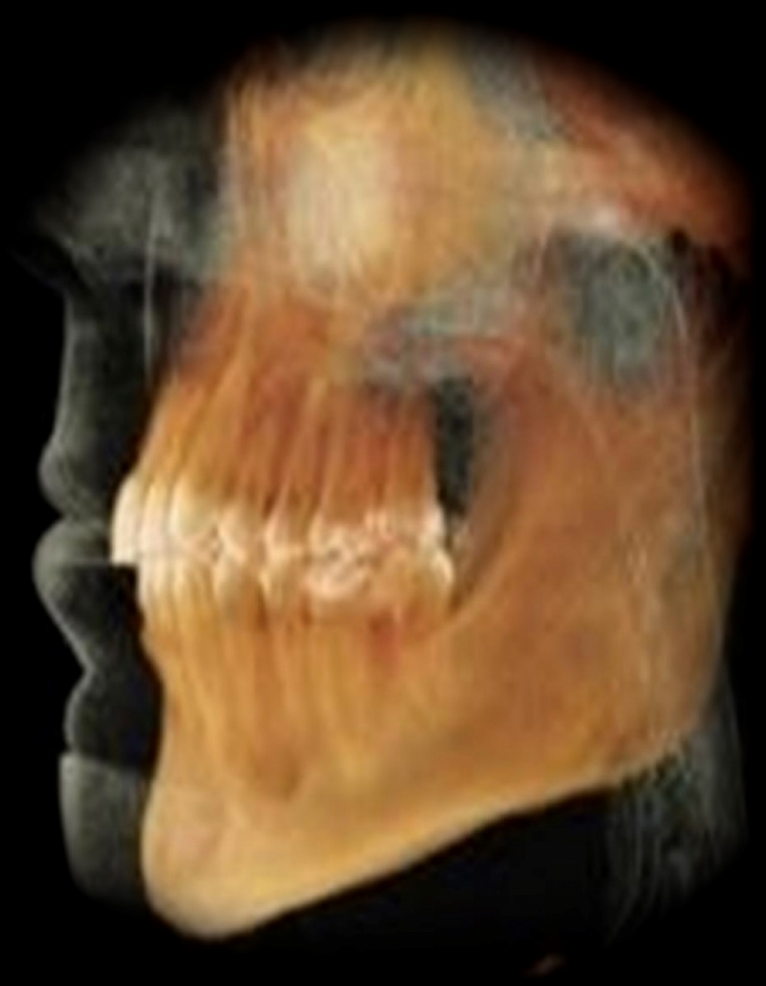

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Summary

CASE REPORT

LITERATURE REVIEW

GIANT SIALOLITHS: LITERATURE REVIEW AND REPORT OF ONE CASE WITH MOUTH FLOOR PERFORATION

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ABSTRACT

Sialolithiasis is the most common non-neoplasic disease of the salivary glands, characterizes by obstruction of the salivary secretion due to appearance of sialoliths. The symptoms are swelling, pain, and inflammation of the affected gland. Ussualy measure less than 10mm those beyond 15 mm are considered giants and rare. It is believed that sialoliths grow 1–1.5 mm per year on average, which would mean 10 years of growth for the sialolith to become giant. Sialolithiasis occurs 80% to 95% in the submandibular gland, 5% to 20% in the parotid gland, and 1% to 2% in the sublingual and smaller glands.The treatment consists in excision of the sialolith being necessary or not the gland´s resection. Were found in the literature 90 cases of giant sialoliths and of these only 24 cases pierced the mouth floor. After the analysis of 90 cases in the literature, the authors concluded that giant sialoliths affected more the fifth to seventh decade of life unlike the normal sialoliths, which affect the third to sixth decade of life.

KEYWORDS: Giant sialolith, megalith, large salivary stone.

1. INTRODUCTION

Sialolithiasis is the most common non neoplasic disease of the salivary glands, characterized by obstruction of salivary secretion due to the appearance ofcalculus, also called sialoliths[1–6]. It is estimated to affect 12 in 1.000 of the adult population[7]. The sialoliths usually measure less than 10 mm, those beyond 15 mm are considered giants and rare[2,5,8,12]. Perforation of the mouth floor by a giant sialolith is extremely rare (24 cases/ Figure 1). It occurs more often during the third to sixth decade of life, thus considered rare in children[13]. The classic symptoms are swelling, pain during meal, infection/inflammation of the affected gland, secondary to obstruction of the salivary ducts, but it is commonly asymptomatic. This disease affects twice as many men than women[14].

Several authors have reported that sialolithiasis occurs in 80% to 95% in the submandibular gland, 5% to 20% in the parotid gland, and 1% to 2% in the sublingual and smaller glands[3,7,13-15]. One-quarter of the symptomatic cases that affect the submandibular gland promote hypofunction or nonfunction of the gland[15]. According to Emir *et al.* (2010) [14] and Yildirim (2004)[15] 70% to 80% of all cases are solitary sialoliths, 3% are multiples bilateral/unilateral, and three or more sialoliths match with 5% of all cases.

Often, sialoliths are radiographic findings. The location and size of sialoliths are important factors in deciding which surgical procedure will be performed, espe-

cially in giant sialoliths[8].

The treatment consists in excision of sialolith, being necessary or not, the gland´s resection, and duct reconstruction, depending on the degree of involvement of the same [13]. The purpose of this paper is to present a literature review on giant sialoliths (Table 1).

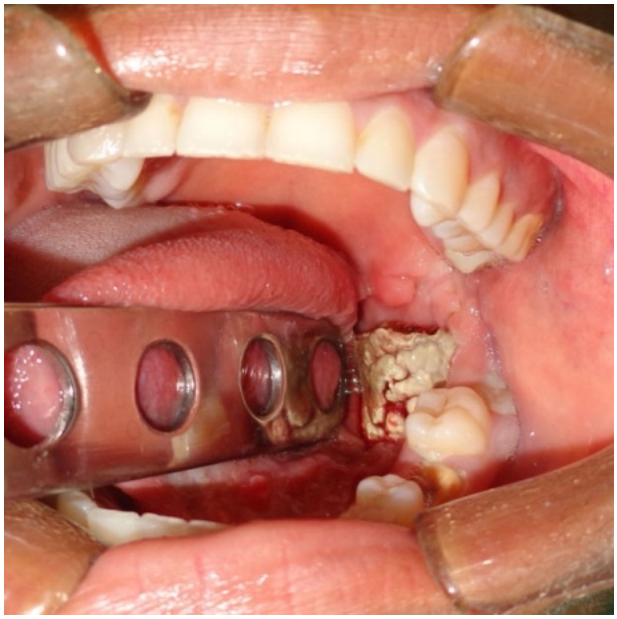

Figure 1. Intraoral view showing a giant sialolith in the left region that drilled the mouth floor.

2. MATERIAL AND METHODS

We conducted a review of literature, based on dental newspapers and magazines and also in specialized sites in search of scientific articles as Pubmed Central and Science Direct, about giant sialoliths from 1942 to 2014 using the key words: giant sialolith, megalith and large salivary stone. We found 90 cases of giant sialoliths with information about age, gender, affected gland, site affected, larger diameter and treatment. This information formed the basis for developing the Table 1.

Table 1. Systematic Review of Patients with Giant Sialoliths (1942-2014).

Case	Author/Year	Gender/ Age	Gland	Site	Size (mm)	Gland Status
1	Meyers/1942*	M/50	SMG	Gland	50	Preserved
2	Mustard/1945	M/52	SMG	Duct	55	-
3	Guernsey/1953*	F/65	-	Gland	33	-
4	Allen/1956	M/49	SMG	Duct	35	Preserved
5	Caar/1965	F/46	SMG	Duct	25	Preserved
6	Rust/1969	M/66	Parotid	Duct	51	-
7	Rust/1969	M/58	SMG	Gland	35	-
8	Brusati/1973	M/55	SMG	Gland	31	Preserved
9	Zakaria/1981	M/70	SMG	Gland	33	Resected
10	Deeb/1981*	M/30	SMG	Duct	16	Preserved
11	Koshal/1981	M/40	SMG	Duct	50	Preserved
12	Isacsson/1982	M/48	SMG	Duct	36	Preserved
13	Naraynsigh/1985	M/28	SMG	Gland	60	Resected
14	Frame/1986	M/50	SMG	Gland	25	Resected
15	Frame/1986	M/67	SMG	Gland	30	Resected
16	Kaltman/1987	M/53	SMG	Gland	45	Resected
17	Lakhoo/1989*	M/37	SMG	Duct	65	Resected
18	Bamgbelu/1989	M/55	SMG	Duct	35	Resected
19	Lakhoo/1989*	M/37	SMG	Duct	65	Resected
20	Asfar/1989	M/75	SMG	Gland	28	Resected
21	Asfar/1989*	M/55	SMG	Gland	38	Resected
22	Asfar/1989	M/43	SMG	Gland	20	Resected
23	Tinsley/1989*	M/48	SMG	Gland	50	Preserved
24	Hubar/1990*	M/65	-	-	52	-
25	Akin/1991	M/45	SMG	Gland	45	Resected
26	Iqbal/1992	F/48	Parotid	Duct	30	-
27	Paul/1995*	M/45	SMG	Gland	45	Preserved
28	Kesse/1998	M/64	Parotid	Gland	50	-
29	Cotter/1998*	F/63	SMG	Duct	20	Preserved
30	Bodner/1999	M/81	Parotid	Duct	25	Preserved
31	Siddiqui/2002	F/52	SMG	Duct	30	Preserved
32	Bodner/2002	M/50	SMG	Duct	50	Preserved
33	Bodner/2002	M/46	SMG	Duct	32	Preserved
34	Bodner/2002	M/25	SMG	Duct	32	Preserved
35	Bodner/2002	M/45	SMG	Duct	30	Preserved
36	Bodner/2002	M/61	Parotid	Duct	20	Preserved
37	Bodner/2002	M/61	SMG	Duct	16	Preserved
38	Gonçalves/2002	F/52	SMG	Duct	22	Preserved
39	Sutay/2003*	F/22	SMG	Gland	37	Preserved
40	Raveuthiran/2004	F/10	SMG	Duct	35	Preserved
41	Yildirim/2004	M/56	SMG	Gland	30	Resected
42	Chan/2006	M/27	SMG	Duct	35	-

43	Grazianni/2006	M/61	SMG	Duct	22	-
44	Krishnappa/2008	M/42	SMG	Duct	20	Preserved
45	Soares/2009	F/54	SMG	Duct	25	Preserved
46	Sahin/2009	M/46	SMG	Gland	26	Resected
47	Rai/2009	M/60	SMG	Duct	72	Preserved
48	Krishinan/2009*	M/41	SMG	Duct	34	Preserved
49	Krishnan/2009*	F/32	SMG	Duct	25	Preserved
50	Shetty/2010*	M/50	SMG	-	27	Preserved
51	Gungormus/2010	M/59	Sub-lingual	Gland	32	Resected
52	Wallace/2010	M/69	Parotid	Gland	23	Preserved
53	Wallace/2010	M/69	Parotid	Gland	23	Preserved
54	Wallace/2010	F/46	SMG	Duct	15	Preserved
55	Wallace/2010	M/55	SMG	Gland	15	Resected
56	Wallace/2010	M/33	SMG	Gland	25	Preserved
57	Wallace/2010	F/15	SMG	Gland	17	Preserved
58	Wallace/2010	F/50	SMG	Gland	15	Preserved
59	Cited by Wallace et al/ 2010	F/52	SMG	Duct	20	Preserved
60	Cited by Wallace et al/ 2010	M/42	SMG	Duct	56	-
61	Cited by Wallace et al/ 2010	M/55	SMG	Gland	38	Resected
62	Cited by Wallace et al /2010	M/52	SMG	Gland	45	-
63	Cited by Wallace et al/ 2010	M/34	SMG	Duct	26	-
64	Cited by Wallace et al/ 2010	M/61	SMG	Gland	22	Preserved
65	Cited by Wallace et al/ 2010	F/46	SMG	Duct	25	Preserved
66	Cited by Wallace et al/ 2010	M/70	SMG	Gland	20	Resected
67	Bofano/2010	M/48	SMG	Duct	22	Preserved
68	Emir/2010*	M/57	SMG	Gland	35	Resected
69	Gehani/2010*	M/41	SMG	-	35	Preserved
70	Gehani/2010*	F/32	SMG	Duct	25	Preserved
71	Ramesh/2010	M/55	SMG	Duct	43	Preserved
72	Cottrell/2011	M/75	SMG	Duct	28	Preserved
73	Rivera-Serrano/2011	M/69	SMG	Duct	23	Preserved
74	Oteri/2011	F/40	SMG	Duct	20	Preserved
75	Oteri/2011	F/51	SMG	Duct	15	Preserved
76	Babu/2011	M/50	SMG	Duct	62	Preserved
77	Igbal/2012*	M/55	SMG	Duct	35	Preserved
78	Rauso/2012*	M/56	SMG	Gland	56	Resected
79	Santos/2012	M/44	SMG	Duct	25	Preserved
80	Santos/2012	M/56	SMG	Duct	25	Preserved
81	Singhal/2012*	M/51	SMG	-	15	Preserved
82	Singhal/2012*	M/40	SMG	-	18	Preserved
83	Fowell/2012	M/58	SMG	Gland	41	Resected
84	Pandarakalam/2013	M/68	SMG	Gland	40	Resected
85	Gupta/2013*	M/48	SMG	Duct	28	Preserved
86	Gupta/2013	F/45	SMG	Duct	19	Preserved
87	Dalal/2013	F/40	SMG	Duct	18	Preserved
88	Singh/2013*	M/55	SMG	Duct	37	Preserved
89	Rodrigues/2014*	F/48	SMG	-	45	Preserved
90	This case*	F/25	SMG	Soft tissue	42	Preserved

Legend:***= Perforation of the mouth Floor;*** SMG**= Submandibular Gland;** M**=Male;** F**=Female.**

3. DISCUSSION

There are three requirements for sialolith formation: a nest, saliva stagnation, and minerals' precipitation. Physical trauma, infection, and inflammation of the gland predispose the appearance of the sialolith by promoting the precipitation of salts[13]. According to Rauso *et al.* (2012)[16] the use of drugs predisposes a reduction of salivary flow, alteration in electrolyte concentration, an impairment of glycoprotein synthesis and a structural deterioration of the cell membranes of the salivary glands.

The sialoliths are composed of calcium phosphate and carbonate in the form of hydroxyapatite, with a lower amount of magnesium, potassium and ammonia. Submandibular calculus are composed of 82% inorganic and 18% organic material, whereas parotid calculus are composed of 49% inorganic and 51% organic material[3]. Commonly, the inorganic substances predominate in the periphery of the stone, while the center is substantially organic substances. The pathogenesis remains unclear, but it is believed that the sialolith begins with the deposition of minerals around an organic core consisting of modified salivary mucin, desquamated epithelial cells and bacteria[17]. Ledesma-Montes *et al.* (2007)[18] studied the microscopic characteristics of a giant sialolith by a scanning electron microscope and were not able to find

structures that suggested the presence of mineralized bacteria. Their hypothesis is that the bacteria may have undergone morphological changes during the mineralization process, thus losing their shape.

Sialoliths can arise in any salivary gland, the submandibular gland being the most affected (80% to 95%). There are some factors that influence this incidence: saliva from the submandibular gland is mucinous, unlike that produced by the parotid gland, which is serous, having more calcium and phosphate; the pH of saliva rich in mucin is more alkaline, the Wharton duct is longer and more tortuous than the Stensen duct, and finally, gravity acts against salivary secretion in the case of the submandibular gland[19].

It is believed that sialoliths grow 1–1.5 mm on average per year, which would mean 10 years of growth for the sialolith to become giant[20]. The ability of calculus growth to become a giant sialolith depends on two factors. The first is related to the ability of adjacent sialolith ducts to dilate, allowing almost normal salivary flow, causing an asymptomatic disease. The second relates to the inertia of the patient to notice the swelling of his face and seek care only years later[3], as in the case reported here, wherein the patient waited three years to seek care.

Salivary stones are classified as ductal (85%) or intraglandular (15%)[5]. Submandibular sialoliths are mostly found in ducts (75% to 85%)[20]. Sialograms have been reported to be 100% effective in detecting ductal and intraglandular stones[15]. Transillumination from the sialendoscope allows location and extraction of the calculus with limited dissection and gland preservation, beyond the identification of lingual and facial nerves, avoiding paresthesia[8]. Scintigraphy is able to determine how functional the gland is. Ultrasonography detects ecodenses spots in 90% of cases when the sialolith measures more than 2 mm. A computed tomography scan is also very effective in diagnosing sialoliths, however, this test is not accessible to the poor population because of high cost, therefore, panoramic, occlusal, and poster anterior radiographs are cheaper tests and assist in the diagnosis and treatment plan[15] (Figure 2).

There are three main ways to treat patients with salivary calculi: resection of the gland, removal through the oral cavity (Figure 3), and salivary endoscopy[7]. Excision of the submandibular gland is indicated in the presence of small sialoliths in the vertical portion of the Wharton duct or a giant stone inside the gland, performing the intraoral approach not being possible[13]. The resection of submandibular gland has a 0% to 8% risk of causing permanent or temporary palsy due to marginal mandibular nerve injury[20]. Damage to the lingual and hypoglossal nerves is also possible. Excision of the parotid gland is also indicated when conservative treatment fails, but there is a possibility of injuring the facial nerve[8]. However, according to Wallace *et al.* (2010)[8] histopathological studies of excised glands due to sialolithiasis showed normal architecture, moreover, patients undergoing endoscopic removal of a sialolith with preservation of the gland after one year had the return of its function. The great advantage of salivary endoscopy is the fact that it has access to sialoliths located in the hilar or glandular region without the need for removal of the gland, thus preserving the salivary flow; however, this technique has only been used for small stones[8,16].

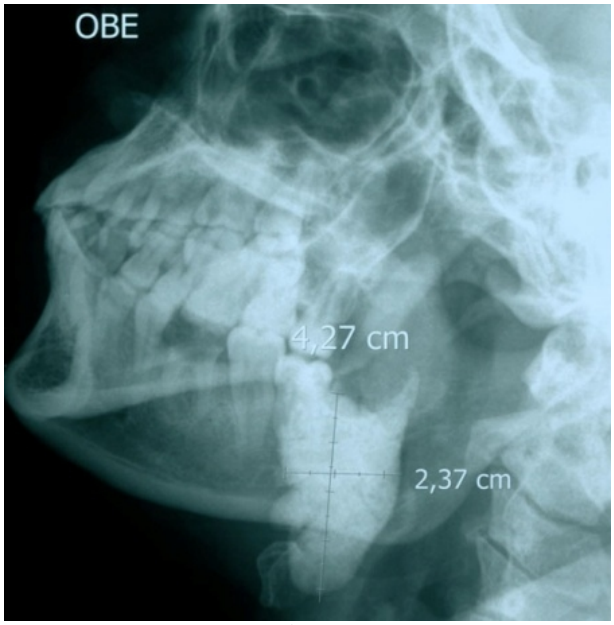

Figure 2. Oblique radiograph of jaw showing a radiopaque, conical, mineralized sialolith measuring 42 mm in its largest diameter.

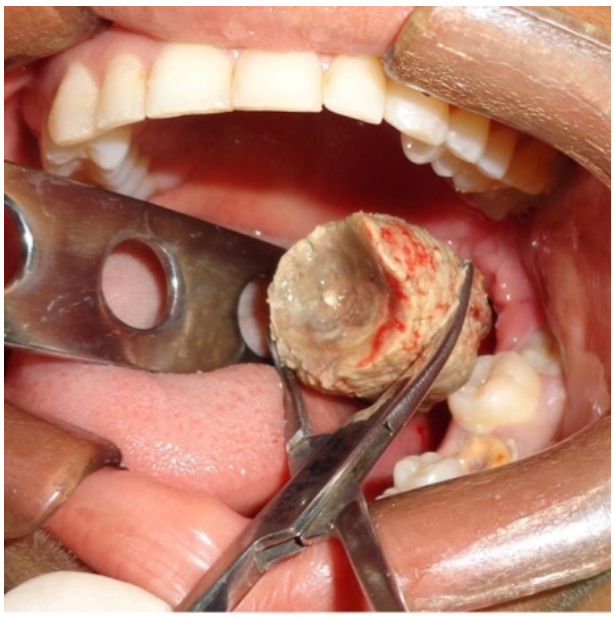

Figure 3. Removal of the first piece of sialolith.

Giant sialoliths were found in 90 cases (Table 1). We concluded that giant sialoliths affected more the fifth to seventh decade of life, representing a total of 77% of all

cases, unlike the normal sialoliths, which affect the third to sixth decade of life[19]; men were affected in 76% of cases. This means they were 3.2 times more affected compared with women; the submandibular gland was affected in 88% of cases being followed by parotid (7%) and sublingual (1%); the duct (55%) was more affected than the gland (38%); the gland was preserved in 61% of cases. In the literature, there were only 24 cases (26%) drilled the mouth floor.

4. CONCLUSION

Giant sialoliths represent a major challenge to oral surgeons in the choice of surgical approach to prevent excision of the gland and likelihood of hypoesthesia, dry mouth, or salivary fistulae. The surgical approach for removal of sialoliths should be minimized to prevent gland morbidity, preferring the intraoral approach. The purpose of this treatment is to restore normal salivary flow. Salivary obstruction for long periods can cause fibrosis and atrophy of the affected gland. Giant sialoliths should be removed even when asymptomatic to prevent complications.

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PARACOCCIDIOIDOMYCOSIS: A CASE REPORT

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ABSTRACT

Paracoccidioidomycosis (PCM) is an infectious disease with acute to chronic evolution; it is native of the Americas and is caused by *Paracoccidioides brasiliensis*. Mucocutaneous integument is a relatively common clinical form of PCM, characterized by lesions on oral mucosa, gums, tongue, soft palate and lip, nasal, pharyngeal and laryngeal mucosa. Stomatological injuries are exulcerated and ulcerated, with uneven contours and edges, granulomatous surface, yellow background, interspersed with hemorrhagic spots which give them a mulberry aspect. This work aims to report the case of a patient who attended to the oral cancer prevention campaign with ulcerations and painful symptoms on the floor of his mouth, buccal mucosa and lip; he was referred to evaluation in the Oral Diagnosis Discipline of Universidade Paranaense and had a diagnosis as Paracoccidioidomycosis.

KEYWORDS: Paracoccidioidomycosis, fungal infectious, diagnosis.

1. INTRODUCTION

The Paracoccidiodomycosis (PCM) also known as Brazilian blastomycosis, South American blastomycosis or Lutz's disease, was first observed in Brazil in 1908, by Adolpho Lutz, who noted the injuries found in the mouths of patients[1]. It is an infectious disease with acute chronic evolution, autochthonous in the Americas. It is caused by *P. brasiliensis*, fungus shows thermal dimorphism and the mycelial form at room temperature, found in soil of endemic areas dwells as Brazil, Ecuador, Colombia and Venezuela. Under temperature about 35-37° C takes the yeast form and corresponds in shape to the parasitic host tissue[1,2,3,4,5].

Many evidences indicates that the main source of infection is inhalation with subsequent development focus and primary pulmonary complex and may subsequently be disseminated to various organs and systems, causing minor injuries that often occur in the mucous membranes, lymph nodes, skin and adrenals. In dependence on factors related to the volume of the inoculum, the agent virulence and ability to host immune response, lung complex can be eliminated, develop into progressive disease state or equilibrium host agent, the so called quiescent focus with viable fungi[2,5].

In Brazil there are some endemic spots such as São Paulo, Minas Gerais and Rio de Janeiro States (Brazil). As these locations there are no Compulsory Notification, the official data on the epidemiological profile are restricted, hindering the detailed characterization of the current situation of the country related to this pathology[2,3,6].

The most common clinical manifestation is the occurrence of chronic disease in male patients, in the proportion of 10 men for a woman, between 30 and 50 years of age, almost always smokers and/ or chronic drinkers, of hygienic conditions, nutritional and socioeconomic precarious, being that the low immunity favors the advancement of disease. The difference in incidence by gender is attributed to hormonal factors. The *Paracoccidioides brasiliensis* have β-oestradiol receptors on their cytoplasmic membrane, that prevents the transformation of filamentous phase in yeast shape. It is consensual that in women, endocrinologically mature and with adequate levels of estrogens, there would be difficulty of transformation of infective conidia in yeast shape, due to protective effect of female hormones[1,2].

In Paraná State (Brazil), the PCM is an endemic dis-

ease among the population of the countryside, being important from the public health point of view, since it affects individuals in their phase of their productive activity. It is most often found in individuals linked to agricultural activity, but can also occur in children of both genders and adolescents[2,4,6]. In these rural workers, chewing leafy vegetables habits can be found, using stems and twigs to picking his teeth and presents the dirty hands of land, as well as cases where the anal toilet is made with vegetable agents[6].

Most often infection by fungus, called Pbmycosis-infection, are asymptomatic. In humans it is known the acute/ sub acute, chronic/ sequel or residual forms. In the chronic form, the oral mucosa is affected quite often and in most cases, the oligosymptomatic form, which slows the diagnosis of the infection. Some patients may experience symptoms such as drooling, bleeding at the injury site, tooth mobility, pain, burning, and also diffuse swelling lip[4]. One of the chronic clinical forms of the multifocal type, relatively frequent of PCM is the mucocutaneous integument, characterized by oral lesions, gum, tongue, soft palate and lip mucosal, nasal, pharyngeal and laryngeal. Although it is less frequent, the infection can invade the bone tissue of the mouth, causing complications such as perforation of the hard palate when the fungus is installed in the jaw[2,4].

The stomatological lesions show up exulcerated and ulcerated contours and uneven edges, with granulomatous surface, yellowish background, interspersed with hemorrhagic spots that give moriform aspect. These are painful spontaneously during chewing, damaging the oral hygiene and effectively contributing to the depletion of the nutritional status of the patient. The healing of lesions causes varying degrees of microstomy as sequel of PCM[2,4].

Knowledge of Paracoccidiodomycosis presents great stomatological interest, since until recently believed to be the oropharynx the fungus gateway due to the numerous expressions found there. For this reason, in most cases, the first signs and symptoms of the disease will lead the patient to the dentist or otorhinolaryngologist. The gateway theory through injuries involving the mucosa is currently discarded. However, it is possible that in some cases, there is penetration of the mucosa by fungus or even by oral ulcerations. However, animal experiments do not support this hypothesis, either by the difficulty of obtaining widespread disease, inoculated through the mucosa or the appearance of lesions on snout and anorectal region of guinea pigs inoculated via intracardiac[4,7].

The regions of the mouth and neck are important areas of manifestation of the disease, since the oral mucosa provides substrate to saprophytic life of the fungus in soil only rich in protein, in places where climatic variations are minimal. In such environments, the molds grow in mycelial phase, producing conidia that survive for several months, enabling the dispersion in the area[2].

The PCM for is in endemic disease in many regions of Brazil, which means be belittled prevalent requires that your diagnosis is early, minimally interventional, easy and available in basic health units, so that appropriate therapy will help to avoid death early and prevent serious sequelae[5]. Pathology is required for definitive diagnosis and differential. The identification of the etiologic agent can be obtained by viewing the yeast phase of the fungus in material collected by scraping the lesions, sputum or biopsy (which can be subjected to several histological staining techniques, including the hematoxylin-eosin (HE) Periodic Acid Schiff (PAS) and the silver impregnation (Gomori-Grocott)) or fungal culture, all considered the gold standard methods; or in hematology, serology, antigen detection, molecular biology, functional and image[2,7,8].

The biopsies of extensive PCM oral lesions, ulcerative and painful are uncommon in routine dentistry, which leads many cases of ringworm be diagnosed late; leading to serious damage to the patient. Because periodontal and lip regions are the most affected in chronic oral forms of PCM, the use of cytology (diagnostic method not traumatic, fast and effective implementation of simple and low operating costs) can greatly help in the diagnosis of these lesions[6].

Given the importance of identification and correct interpretation of signs and symptoms that may manifest in the oral cavity of patients with clinical picture of PCM and the need for early diagnosis for effective treatment with fewer sequels, this paper describes the case a patient with PCM, diagnosed in the dental clinic at the Paranaense University - UNIPAR.

2. CASE REPORT

Patient A.A.L., feoderm, 49, appeared in the mouth cancer prevention campaign by the movement "Blue August" at the Medical Specialties Center (MSC) of Umuarama municipality in partnership with the dentistry course of the Paranaense University (UNIPAR), reporting appearance wound and white spots on lips and inside the mouth, with fever and severe pain two days after nine dental elements extraction. The patient was referred for oral Diagnostic discipline at the UNIPAR. In anamnesis being a smoker and drinker, was rural worker in cotton, coffee and soybeans for about 25 years; today, it works as a construction helper. Physical examination revealed painful exulcerations, contours and uneven edges, with granulomatous surface of yellow background and mulberry appearance (Figure 1) having a differential diagnosis Paracoccidiodomycosis or carcinoma.

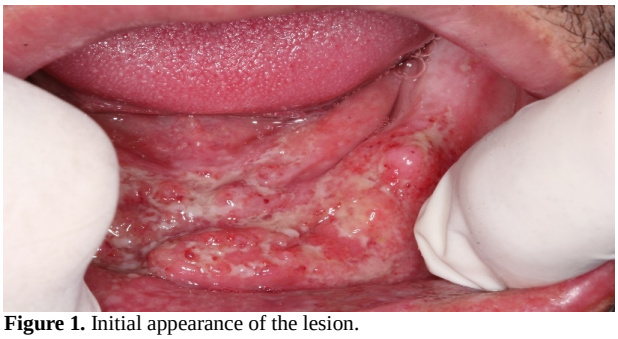

Figure 1. Initial appearance of the lesion.

Panoramic radiograph was performed (Figure 2) to assess bone condition due to recent extractions and discard possible bone involvement, and incisional biopsy to establish the diagnosis, that through the anatomopathological examination was confirmed as Paracoccidiodomycosis. The patient was referred to infectious disease and is under treatment (Itraconazole 100 mg) and follow-up of 7 months with total regression of symptoms (Figure 3).

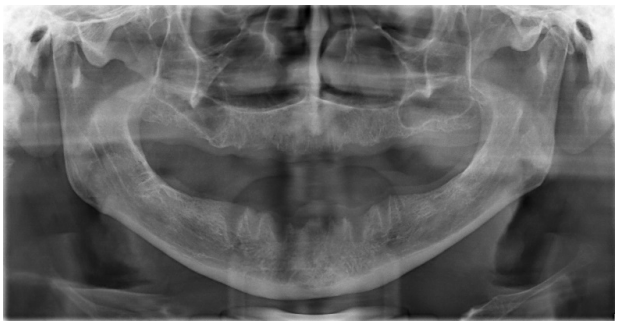

Figure 2. Panoramic radiograph showing the condition after bone extractions.

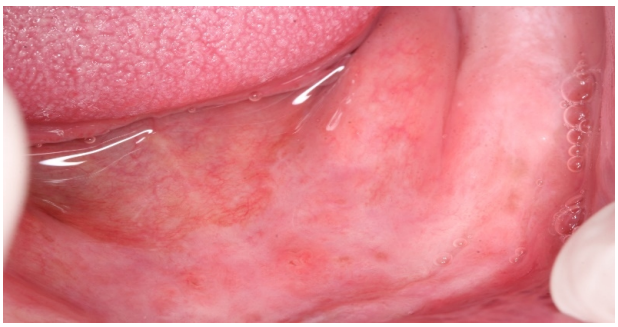

Figure 3. Aspecto da região acometida após 2 meses de tratamento.

3. DISCUSSION

Being in agreement with the standard PCM of a patient, this case occurred in an individual male, smoker and heavy drinker, who reported having worked in the countryside[1,2,3,5,6].

The patient sought treatment only because the oral manifestations of the disease. It emphasizes the need for Dental Surgeon of Paraná state and other endemic regions know the signs and symptoms of PCM (in this case were exulcerations the lip region, buccal mucosa and floor of the mouth, which is consistent with Araujo (2003)[6] and Vieira & Borsatto-Galera (2006)[1], reporting as the most affected sites in the seed coat mucocutaneous regions the oral mucosa, gums, tongue, soft palate and lip mucosal, nasal, pharyngeal and laryngeal], in order to establish the correct diagnosis and referral of patients for treatment and resolution of the case.

The PCM lesions are painful spontaneously during chewing, damaging the oral hygiene and effectively contributing to the depletion of the nutritional status of the patient. The healing of lesions causes varying degrees of microstomy as sequel of PCM[2,4]. The patient reported weight loss prior to treatment because of pain in the region and difficulty chewing and two months after starting treatment, reported the lip strain of difficulty, hooked in the lip and feeling smaller mouth (microstomy), and the appearance various spots on the chest, which signal was evident from the second day of the start of treatment.

According to Araujo *et al.* (2003)[6] and Vieira & Borsatto-Galera (2006)[1], even if less frequent infection can invade the bone tissue of the mouth, causing complications. Given the fact that the patient has carried out multiple recent tooth extraction, the realization of panoramic radiography has become necessary to evaluate the bone of the patient's condition and rule out possible involvement or bone complications[9].

On the obligation of the pathology for definitive diagnosis and differential[2,8], the patient underwent incisional biopsy, considered the most reliable method for diagnosis using as histological staining technique to HE[10].

4. CONCLUSION

Because it is up an endemic disease in Paraná, commonly manifesting their first symptoms in the oral cavity and patients seek care due to these oral manifestations, it is essential knowledge on the part of Dental Surgeons of the signs and symptoms of this disease in order to achieve a correct and rapid diagnosis, enabling the patient to early treatment instituted from referral to the infectious disease physician with a minimum of sequelae. It is also essential for the correct diagnosis, such as biopsy, even though these examinations not yet constitute a common reality in dental clinics.

Finally, there is the importance of prevention and health promotion campaigns, for allowing access to a large number of people to rapid tests in accessible days most of the population in order to identify and refer patients with early changes, untreated, might lead to other consequences.

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BACTERIAL ENDOCARDITIS IN DENTISTRY: DISEASE AND DRUG PROPHYLAXIS

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ABSTRACT

In dentistry, the gingival fissure around the teeth or tissue damage produced by surgical and/or accidental procedures can be a gateway to oral microbiota, directly into the bloodstream, which can result in the colonization of the endocardium, causing infective endocarditis. Infective endocarditis is caused by the infection of the endocardial surface, characterized as a rare disease, but which, if triggered, can cause inflammation and destruction of the endocardium, heart valves or vascular endothelium. Since the discussion about the use or non-use of antibiotic prophylaxisis is not pacified in the Dental literature, this review aims to explain about the necessity of antibiotic prophylaxis prescription to the patients considered at risk, with potential risk and low risk of infection associated with dental surgery.

KEYWORDS: Endocarditis, antibiotics, dental surgery.

1. INTRODUCTION

In dentistry, it is already established in the literature that a simple gingivitis or periodontitis may be the gateway to the infective endocarditis. Romans & App (2009)[1] shown that, with one gingival bleeding, endocarditis can be triggered by using a simple water jet for cleaning. Thus, it is possible to say that periodontitis can be chronic and infectious diseases with the potential to result secondarily severe systemic infectious diseases in predisposed individuals or under favorable conditions. Conversely, considering the development of systemic infectious diseases, periodontal disease should be considered as an important contributing factors[2].

The aims to explain about the necessity of antibiotic prophylaxis prescription to the patients considered at risk, with potential risk and low risk of infection associated with dental surgery.

2. MATHERIAL AND METODS

The development of this integrative review we chose the proposal of Ganong (1987)[3], according to the following steps: 1) identification of the research question, followed by a search of the descriptors or keywords; 2) determining the criteria for inclusion or exclusion of research in online databases; 3) categorization of studies, summarizing and organizing relevant information; 4) assessment of studies for critical analysis of the extracted data; 5) discussion and interpretation of the examination results, contextualizing theoretical knowledge and evaluating their applicability as; 6) presentation of the integrative review and synthesis of knowledge of each article reviewed briefly and systematic way.

In the present study the guiding question of the integrative review was: to review the literature to compile a study about the use or non-use of antibiotic prophylaxisis in order to prevent bacterial endocarditis.

Bases (Latin American and Caribbean Literature on Health Sciences) LILACS, SciELO (Scientific Electronic Library on Line) and PubMed (NCBI US National Library of Medicine National Center for Biotechnolin Dentistry Information) were consulted. Studies that have addressed the thematic, published from 1973 to 2011, regardless of the languages of publication were included. Lastly, following controlled for the search and also used as keywords descriptors were used: Endocarditis, antibiotics, dental surgery.

3. LITERATURE REVIEW

Horder (1909)[4], based on his studies found an association between dental health and infectious endocarditis, as the oral cavity, as well as the high digestive tract surfaces are populated by a native microbiota. So, the gingival crevice around the teeth or even micro trauma can

act as a gateway for these microorganisms of the oral microbiota into the bloodstream, resulting in bacteremia, transient or none. Table 1 lists some of the major systemic infectious diseases that may be associated with periodontal diseases.

Table 1. Correlation between periodontal diseases and secondary systemic diseases.

AFFECTED SYSTEM/ ORGAN	SYSTEMIC DISEASES CORRELATED TO INFECTIOUS PERIODONTAL DISEASES
Heart	Angina pectoris, myocardial infarction, endocarditis
Vascular System	Vascular Accident (CVA)
Central Nervous System	Meningitis, brain abscess
Lung	Pneumonia

Source: Herbet *et al.* (2006)[5].

For this reason, classically COTRAN *et al.* (2010)[6] defined that infective endocarditis is a disease caused by colonization or invasion of the heart valves (or the mural endocardium) by a microbiological agent, leading to local formation of thrombotic masses full of microorganisms, known as infective vegetation. Similarly, this colonization can occur in the aorta and aneurysm sac in other vascular beds.

Under the cardiovascular perspective, according to David *et al.* (2008)[7], endocarditis can originate from turbulent blood flow as a result of congenital or acquired heart disease. Turbulent blood flow can damage the integrity of the endocardium, activating the platelet adhesion and fibrin network in the cardiac tissue, resulting in a sterile vegetative formation known as non-endocardial thrombotic. Moreover, microorganisms entry into the bloodstream can result in colonization of the previously damaged endocardium, causing endocarditis. Studies of Beck *et al.* (1996)[7] corroborate the idea that periodontal disease can be correlated to cardiovascular disease. In a longitudinal study of more than 1,100 men, Herbert *et al.* (2006)[5] noted that periodontitis with great depth on probing exchanges, regardless of other factors, increases the risk of coronary heart disease.

In this respect, it seems that there is a similarity on many occasions, between etiologic factors of the disease and the microorganisms found in the oral cavity (streptococcal infection) infected dental pulp and periapical lesions. The literature has reported cases of sub acute bacterial endocarditis after dental extractions produced, since transient bacteremia often follows tooth extraction) and it almost always occurs within a few weeks to a few months after that operative procedure[9].

Etiologic agents:

Virtually all microorganisms have been implicated in the occurrence of endocarditis. Infective endocarditis is a high risk of infection, usually triggered by microorganisms (Streptococcus) of exposed defects of the buccal cavity (dental plaque formation in the heart valves). There are countless microorganisms that, when they fall into the bloodstream after trauma or manipulation of tissues, can cause infective endocarditis: bacteria, as Rickíttsias or Chlamydia, Mycoplasma or fungi. Especially susceptible areas of the cardiovascular system are those with slower blood flow or great turbulent[5]. The oral cavity is a common source of infectious microorganisms triggering of endocarditis. Streptococci are the main cause (viridans type, gram-positive) found, especially *Streptococus sanguis*. In *S. aureus* and *S. epidermidis* it has become increasingly common to the detection of *A. actinomycetem comitans*, *Hamophilus* spp., *Cardiobacterium* spp., *Eikinella corrodens*, *Kingella* spp., the genus *Capnocytophaga*, and *Neisseria* spp. For antibiotic coverage of patients at risk of infective endocarditis, are recommended bactericidal antibiotics of the family of Penicillin. Since 1983 was considered the case of use of metronidazole as an additional drug to antimicrobial prophylaxis of endocarditis[5]. However, you can observe the prevalence of some strains on the other. Infections with gram-negative microorganisms is accompanied by the release of inflammatory mediators in the bloodstream, including, systemic action of cytokines (TNF, IL-1, IL-6), growth factors and prostaglandins[5].

Gram-positive microorganisms can also cause serious heart conditions; streptococci - among those of the oral cavity, especially *S. sanguis* - trigger or aggravate the feared endocarditis. Transient bacteremia can be caused by viridans group of streptococci in the dental surgical or invasive procedures which can result in bleeding, even during routine activities. The frequency and intensity of bacteremia arising are related to the nature and magnitude of the trauma of the tissue, the density of microorganisms and the degree of inflammation or infection at the site of trauma. The species of microorganisms that enter the bloodstream depend on the endogenous microbiota that colonizes the injured site[5].

Classification of endocarditis

In the acute form, the patient does not have any heart problem although the disease is in quiet development. In the sub acute form the patient is at higher risk to get the disease. Therefore, the performance of any surgical procedure needs further care. In the production of sub acute bacterial endocarditis is a prerequisite prior damage to heart valves. The valve lesions may be congenital or caused by rheumatic fever. When there is bacterial, transient or intermittent invasion, the bacteria are eliminated by the defense mechanisms, however, in individuals who are carriers of previous heart damage, the bacteria can settle on the heart valve and cause sub acute bacterial endocarditis[9,10].

Symptoms:

The presentation of sub acute disease is very different from the acute illness. At first, the fever can be low, particularly in the elderly can be discreet or absent. In general the only signs are nonspecific - fatigue, weight loss - without much to indicate the heart. The blows are absent in 10% of patients with sub acute disease furthermore can be associated only with an existing heart abnormality[6]. Sub acute has greater interest to the dental surgeon therefore arises for introduction of microorganisms into the bloodstream during the performance of dental procedures in patients at risk. Its symptoms are vague and insidious, including fever, weakness, weight loss, dyspnea, anorexia, progressive muscular and joint pain and weakness in the face and limbs similar to stroke, so that may be months before it is diagnosed[11].

In contrast, acute endocarditis has a stormy beginning with rapid development of fever, often with peaks, chills, weakness and lassitude. There exists a higher probability of a blow with acute endocarditis due to the large size of the vegetation, and often change as they grow and vegetations fragment. The spleen presents increased with greater frequency in the acute form of the disease than in the subacute[6].

Patients with cardiac risk:

The identification of patients with differentiated risk goes through a detailed history. Questions related to heart health of the patient on the existence of prosthetic valve, if user cardio intravenous vasoactive drugs or, if you have diabetes mellitus type I or II, whether or not frequent user of alcoholic beverages, tobacco or other drugs, licit or illegal[12]. These high-risk patients can develop endocarditis after performing invasive dental procedures, with the entry of bacteria or fungi even in the circulatory system. In the case of fungi, we can mention the acute and sub acute endocarditis[2,13]. Previous studies associated with numerous reports of patients who developed infective endocarditis are the basis for the recommendation of antibiotic prophylaxis[14-20].

In this sense, strongly grounded in the literature, few clinical conditions of the patient identified as being at high risk of endocarditis or average risk of endocarditis, according discrimination below, proposed by Junior & Zanatto (2003)[12], and reviewed later by Sampaio *et al.* (2008)[21].

High risk of endocarditis

- Use of biological or mechanical valve prostheses
- Infective endocarditis history, even in the absence of heart disease

Medium risk of endocarditis

- Congenital valve defect or acquired
- Congenital heart defect, such as:
  - Aortic isthmus stenosis
  - Blood Canal (ductus botalli) open
  - Ventricular septal defect (type ostiom primum)
  - Sub aortic stenosis or supravalar
- Tetralogy of Fallot
- Palliative surgery history of congenital heart defects
- Correction of congenital heart defects incimpleta
- Hypertrophic obstructive cardiomyopathy (HOCM)
- Mitral valve prolapse (MVP) with systolic noise

Normal risk (without lift) of endocarditis

- Atrial septal defect
- Successful surgery History correction of atrial or ventricular septum (6 months without waste)
- Coronary bypass surgery history (bypass)
- Mitral valve prolapse (MVP) without systolic noise
- Physiological cardiac noise, functional or harmless
- Kawasaki disease without valvular dysfunction History
- History of rheumatic fever without valvular dysfunction
- Pacemaker Use
- Stenosis surgery History of the aortic isthmus

Management of positive cases of endocarditis in dental patients

The diagnosis of bacterial endocarditis has been made in blood cultures and viewing bacterial vegetation through the echo cardiogram[21].

Although endocarditis be related recurrently in dental patients, today many professionals know little or unaware of this form of complication, let alone how to prevent it or treat it. Nascimento *et al.* (2011)[22] showed that a large proportion of professionals knew the possibility of heart infectious complication after performing surgery, but did not declare consistent knowledge of antibiotic prophylaxis or merely stated that they mastered topics related to endocarditis.

Prevention of bacterial endocarditis

Thus, antibiotic prophylaxis would be indicated prior to invasive procedures, such as periodontal treatment, extractions, dental replantation, endodontic instrumentation, intra ligamentous injections, among others. On the other hand, in non-invasive procedures such as dental restorations, antibiotic prophylaxis may be eliminated. Currently, the antibiotic of choice is amoxicillin.

Prophylactic protocols

It is definitely accepted to be administered before the antibiotic in patients who have rheumatic fever or other known sign of any valve damage and will undergo certain dental treatments, including treatment of root canals[9].

The adult patient must receive 2g of the drug one

hour before the surgical procedure. Amoxicillin is used by being well absorbed from the gastrointestinal tract, have broad action spectrum for positive and Gram-negative bacteria but also by its favorable historic or low levels of adverse side effects, although it is a synthetic penicillin. However, there are patients allergic to amoxicillin. In these cases, it is possible to hold the antibiotic prophylaxis using: clarithromycin or azithromycin, both drugs of the macrolide class, or clindamycin group of lincosamides. If there is the need for multiple invasive procedures on the same patient, research shows that to be respected a minimum of 10 days between a procedure and another, so that there is the emergence of microorganisms resistant to the antibiotic of choice. Another strategy would be to alternate the use of the above antibiotics, in order to create an element of surprise to the microorganism and thus minimizing the chance of emergence of resistant strains[23].

Table 2. Prevention of endocarditis - antibiotic prophylaxis

Patient	Antibiotic	Adult	Child	Use before intervention
Prophylaxis standard	Amoxicillin	2 g oral	500 mg/kg oral	1h
Do not swallow pills	Ampicillin	2 g im/iv	50 mg/kg im/ iv	30 min
Allergic to penicillin	Clindamycin	600 mg oral	200 mg/kg oral	1h
	Cephalexin Cefradoxil	2 g oral	50 mg/kg oral	1h
	Azithromycin Clarithromycin	500 mg oral	15 mg/kg oral	1h
Not swallows pills and is allergic to penicillin	Clindamycin Cefalozin	600 mg iv 1g im/iv	20 mg/kg oral 25 mg/kg oral	30 min 30min

Source: Herbert *et al*. (2006)[5]; Wilson e*t al*. (2007)[23].

Necessary or unnecessary prophylaxis in practice

Table 3. Type of dental procedure and antimicrobial prophylaxis recommendation.

Dental interventions: Recommended	***Dental interventions: Non-recommended***
Anesthesia - injection intra ligamentous	Local anesthesia (except intra ligamentous)
Surgery - tooth extraction, other interventions	Prosthetic or restorative treatment, with or without the use of retraction cord
Periodontics - Survey; scaling and root planning; surgery; control sessions, maintenance/ recall; placement of release devices subgingival drugs.	Endodontics: canal treatment, intra-radicular pin placement, etc.
Implantology - Surgeries to implant placement.	Other: Isolation with rubber dam and suture removal; moldings; X-rays; adjustment of orthodontic appliances
Endodontics - Instrumentation or surgery exceeding the root apex	Deciduous teeth extraction in exfoliation-way
Procedimentos de profilaxia, quando a ocorrência de sangramento é provável	Perform prophylaxis when bleeding is provided

Source: Herbert *et al.* (2006)[5]; Sampaio *et al.* (2008)[21].

Table 3 summarizes the main dental procedure with its respective recommendation or absence of antimicrobial prophylaxis.

When a treatment with a long time is planned, it should be expected to use in this period of complementary medicine - combined with an antiseptic regime. When it opts for antimicrobial prophylaxis for use, before dental procedures, the main antibiotics used are shown in Table 2 and 4, so that the information from both tables are complementary.

Table 4. Protocol for administration of antibiotics for dental procedures.

Administration route	Medication	Child-Dosage before procedure	Adult-Dosage before procedure
Oral	Amoxicillin	50 mg/ kg	2 g
	Clindamycin	20 mg/ kg	600 mg
Allergic to Penicillin	Azithromycin	15 mg/ kg	500 mg
	Clarithromycin	15 mg/ kg	500 mg
I.M / I.V.	Ampicillin	50 mg/ kg	2 g
	Cefazolin	50 mg/ kg	1 g
	Ceftriaxone	50 mg/ kg	1 g
I.M / I.V. to Allergic to penicillin	Clindamycin	20 mg/ kg	600 mg

Source: Cavezzi Jr (2010)[24].

4. CONCLUSION

Infective endocarditis as a possible complication that should be taken seriously by Dental Surgeons, as suggested by many authors, which describes emphasizing the loss of specific functions of the cardiovascular system, with real risk of death for the infected patient.

The routine use of antibiotic prophylaxis must be performed in high-risk patients who will undergo invasive dental procedures such as handling or drilling gingival tissue of the oral mucosa. The Dental Surgeon could also consider the use of antimicrobial prophylaxis in patients defined as low-risk, in order to standardize its procedures in clinics, especially considering the existence of clinical-school, with the attendance of thousands of people annually. However, non-invasive procedures such as restorations, application of sealants and others should not use antibiotic prophylaxis, since it takes the patient's health assessment immune feasibility assumption of the patient and the negative of infectious diseases in the recent past of the patient.

Thus, the Dental Surgeon could consider two fundamental aspects to indicate antibiotic prophylaxis of infective endocarditis: 1- the identification of high-risk patients to acquire the infective endocarditis and have chance to develop into a more serious condition; 2- the identification of dental procedures with greater risk to provide the endocardium infection.

Finally, we reiterate that antimicrobial prophylaxis is still one of the most effective ways to effectively prevent infective endocarditis in patients with known risk, potential or remote.

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