



# CATÓLICA

## FACULDADE DE MEDICINA DENTÁRIA

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VISEU

### IMPACTO DO TRATAMENTO PERIODONTAL NUM PACIENTE COM EPISÓDIO DE ENFARTE AGUDO DO MIOCÁRDIO - CASO CLÍNICO

Dissertação apresentada à Universidade Católica Portuguesa  
para obtenção do grau de Mestre em Medicina Dentária

Por:  
Erica Alexandra Macedo Pessoa

Viseu, 2020





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Orientadora: Prof. Doutora Vanessa Silva  
Coorientadora: Prof. Doutora Patrícia N. Tannure

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*“Que todos os nossos esforços estejam sempre no desafio  
às impossibilidades. Todas as grandes conquistas  
humanas vieram daquilo que parecia impossível.”*

*(Charles Chaplin)*

## **DEDICATÓRIA**

*A minha mãe Arizete.*

*À melhor cirurgiã dentista que tive a honra de ser filha, que no dia a dia, do exercício da sua profissão me ensinou que o mais importante, é sempre o cuidado com o paciente, aliado a busca incansável de conhecimento. Uma mulher, além do seu tempo, mãe carinhosa e com atitudes concretas de amor. Amor que se desdobra em tudo ofertar aos filhos e a família. Tudo que sou, tudo que tenho, vem do seu exemplo e dos seus ensinamentos.*

*Ao meu pai, José.*

*SUCESSO, SUCESSO MINHA FILHA.*

*Suas palavras ressoam aos meus ouvidos, em cada passo, em cada projeto. Obrigado por todo incentivo, por toda disponibilidade em sempre ajudar.*

*Ao meu irmão Wellington*

*O que seria de mim sem meu irmão, aquele que sempre cuidou de mim, aquele que em todas as fases da minha vida do meu lado estava. Lembranças do ensino fundamental até o trote de calouros, lá estava ele sendo meu veterano. Como agradeço a Deus por ter você como irmão, amigo, colega de trabalho. Obrigado por SEMPRE me fazer seguir.*

*A minha irmã Patrycya*

*Deus sabia que eu precisava de uma irmã, alguém pra compartilhar o dia a dia, as aventuras e sonhos. Obrigado por me encourajar, mesmo quando tens medo. Obrigado por ser presença de Deus na minha vida. Sigamos para próximas aventuras e sonhos.*

*A meus sobrinhos Mariana, Stephanie e Victor*

*Obrigado por todo carinho e amor, contem sempre comigo.*

## **AGRADECIMENTOS**

*Cabe a mim, nesse momento agradecer a tantas pessoas que direta ou indiretamente me ajudaram no desenvolvimento e conclusão dessa etapa da minha vida.*

*À Deus, que tudo providenciou para que durante todo o período do curso, eu pudesse participar e concluir o mesmo.*

*A minha orientadora, Prof. Dra. Vanessa Silva por sua dedicação e empenho na realização deste trabalho e acima de tudo, obrigado por todo carinho e amizade neste tempo.*

*A minha coorientadora, Prof. Dra. Patrícia N. Tannure, mais uma conquista, mais um título com a sua orientação, toda minha admiração, eis um exemplo a seguir, obrigado por tudo.*

*Aos meus colegas de turma, que juntos transformamos desafios e dificuldades em amizades e boas gargalhadas.*

*A todos os docentes e funcionários da UCP, a participação e a presença de cada um foi fundamental nesse período tão especial na minha vida.*



## **RESUMO**

A correlação entre a doença cardiovascular (DCV) e a doença periodontal (DP) ocorre devido à influência de uma doença na patogénese da outra. As causas e efeitos ainda não estão totalmente estabelecidos. A doença periodontal é causada por bactérias do biofilme que podem agravar as doenças cardiovasculares, como também aumentar a probabilidade de uma futura doença. Está descrito que as trocas hormonais, que acontecem em diferentes estágios da vida, podem influenciar o sistema de condução elétrico do coração, as funções endoteliais e microvasculares, podendo originar o desenvolvimento de doenças cardiovasculares. O relato do caso clínico exposto descreve a eficácia do tratamento periodontal num paciente que sofreu um enfarte agudo do miocárdio, que após alta hospitalar e novo tratamento periodontal obteve uma redução significativa da profundidade das bolsas periodontais e da contagem bacteriana total.

**Palavras-chave:** doença cardiovascular, doença periodontal, análise microbiológica.



## **ABSTRACT**

The correlation between cardiovascular disease and periodontal disease is based on one disease influencing the pathogenesis of the other; the causes and effects are yet to be established. Periodontal disease is caused by biofilm bacteria that may worsen cardiovascular diseases and increase the likelihood of future disease. Hormonal changes, which occur at different stages of life, have been described as potential influencers of the heart's electrical conduction system and endothelial and microvascular functions, thus meaning they could lead to the development of cardiovascular diseases. This clinical case report describes the effectiveness of periodontal treatment on a patient who suffered an acute myocardial infarction and, after hospital discharge and another periodontal treatment, showed a significant reduction in the depth of periodontal pockets and total bacteria count.

**Keywords:** cardiovascular disease, periodontal disease, microbiological analysis.



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## **LISTA DE SIGLAS E ABREVIATURAS**

DCV     Doença Cardiovascular

DP     Doença Periodontal

DM     Diabetes Mellitus

DCNT     Doença Crónica não Transmissível

EAM     Enfarto Agudo Miocardio

HTA     Hipertensão arterial

## **LIST OF ABBREVIATION AND ACRONYMS**

AMI     Acute myocardial infarction

CVD     Cardiovascular disease

DM     Diabetes Mellitus

HT     Hypertension

NCCD Non-communicable chronic disease

PD     Periodontal disease



## **I. ENQUADRAMENTO TEÓRICO**



## I. ENQUADRAMENTO TEÓRICO

As doenças cardiovasculares (DCV) são responsáveis por 17,9 milhões de mortes a nível mundial, sendo a terceira causa de morte no mundo.<sup>1</sup> Na Europa, a taxa de mortalidade tem vindo a aumentar nos últimos 25 anos devido ao stress, sedentarismo e ao aumento da esperança média de vida.<sup>2</sup> De todas as doenças cardiovasculares, o enfarte agudo do miocárdio (EAM), o acidente vascular cerebral (AVC) e a hipertensão (HTA) são as que estão associadas a uma maior taxa de mortalidade. Não há uma causa única para as cardiopatias, mas um conjunto fatores de risco que favorecem o desenvolvimento desta condição, como a diabetes mellitus, o sedentarismo, a hereditariedade e o tabagismo.<sup>3</sup>

A periodontite é uma doença crónica não transmissível (DCNT), classificada como a sexta doença mais comum na população.<sup>4</sup> Devido à alta prevalência e incidência é considerada um problema de saúde pública, interferindo diretamente na qualidade de vida da população, pois nos estágios mais avançados, se não tratada, leva a perda dentária. Estudos demonstram uma relação entre a doença periodontal (DP) e as DCV e como alterações do periodonto podem estimular ou agravar as doenças cardiovasculares.<sup>5, 6, 7</sup> Por sua vez, o tratamento periodontal pode melhorar os níveis de lípidos,<sup>8,9</sup> inflamação<sup>10</sup> e função endotelial nos pacientes. Por outro lado, pode evitar a destruição do ligamento periodontal, do osso alveolar, a migração apical do epitélio e formação de bolsas periodontais,<sup>11</sup> além de diminuir o sangramento gengival, a profundidade das bolsas periodontais e a destruição dos tecidos de sustentação dos elementos dentários.

Bokhari *et al.* no estudo com 317 pacientes com DCV relataram que após tratamento periodontal os pacientes apresentaram além de uma melhoria no quadro clínico, uma redução significativa dos níveis de proteína C reativa, fibrinogénio e de glóbulos brancos.<sup>12</sup> Sabe-se que o impacto da doença periodontal nas DCV é biologicamente aceitável, na medida em que a disbiose da microbiota oral circulante pode induzir direta ou indiretamente uma inflamação sistémica.<sup>13</sup>

Genco *et al.*, em 2010, relataram que pacientes com DP possuem maior risco de desenvolver DCV quando comparados a pacientes saudáveis e que o tratamento periodontal reduz a incidência de AVC quando associado as intervenções de saúde oral.<sup>14</sup> No entanto ainda não existem evidências suficientes para apoiar ou refutar o potencial benefício do tratamento periodontal na prevenção ou atraso da DCV.<sup>15</sup> Diante da relevância do assunto, esse caso clínico descreve a importância do tratamento periodontal num paciente com DCV e DP que sofreu um enfarte agudo miocárdio (EAM). O objetivo da exposição deste caso é constatar o impacto do tratamento periodontal na saúde de um paciente com doença cardiovascular, através de exames microbiológicos.



## **II. ARTIGO CIENTÍFICO**



# IMPACT OF PERIODONTAL TREATMENT ON A PATIENT WITH AN EPISODE OF ACUTE MYOCARDIAL INFARCTION - A CLINICAL CASE

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## Abstract

The correlation between cardiovascular disease and periodontal disease is based on one disease influencing the pathogenesis of the other; the causes and effects are yet to be established. Periodontal disease is caused by biofilm bacteria that may worsen cardiovascular diseases and increase the likelihood of future disease. Hormonal changes, which occur at different stages of life, have been described as potential influencers of the heart's electrical conduction system and endothelial and microvascular functions, thus meaning they could lead to the development of cardiovascular diseases. This clinical case report describes the effectiveness of periodontal treatment on a patient who suffered an acute myocardial infarction and, after hospital discharge and another periodontal treatment, showed a significant reduction in the depth of periodontal pockets and total bacteria count.

## Keywords

cardiovascular disease, periodontal disease, microbiological analysis.



### **III. INTRODUCTION**



## I. INTRODUCTION

Worldwide, cardiovascular diseases (CVDs) are responsible for 17.9 million deaths and are the third cause of death.<sup>(1)</sup> In Europe, their death rate has been increasing in the past 25 years due to stress, sedentarism, and the increased average life expectancy.<sup>(2)</sup> The CVDs with the greatest death rates are acute myocardial infarction (AMI), stroke, and hypertension (HT). There is no single cause for CVDs, but some risk factors promote their development, including diabetes mellitus, sedentarism, heredity, and smoking.<sup>(3)</sup>

Periodontitis is a non-communicable chronic disease (NCD) categorized as the sixth most common disease in the world.<sup>(4)</sup> Due to its high prevalence and incidence, it represents a public health problem. Periodontitis influences the population's quality of life directly since, in more advanced stages, if untreated, it can lead to tooth loss.

Studies have shown an association between periodontal disease (PD) and CVDs and how changes in the periodontium may boost or worsen CVDs.<sup>(5, 6, 7)</sup> On the other hand, periodontal treatment may increase the patient's lipid levels,<sup>(8,9)</sup> inflammation,<sup>(10)</sup> and endothelial function. It may also prevent destruction of the periodontal ligament and the alveolar bone, apical migration of the epithelium, and formation of periodontal pockets,<sup>(11)</sup> as well as reduce gingival bleeding, periodontal pockets' depth, and destruction of tooth-supporting tissues.

Bokhari *et al.* conducted a study with 317 patients with CVDs and reported that, after periodontal treatment, patients showed improvement of their condition and a significant reduction in the levels of C-reactive protein, fibrinogen, and white blood cells.<sup>(12)</sup> The impact of PD on CVDs is considered biologically logical, as the dysbiosis of the circulating oral microbiota may induce systemic inflammation either directly or indirectly.<sup>(13)</sup> Genco *et al.*, in 2010, reported that patients with PD had a higher risk of developing CVDs compared to healthy patients and that periodontal treatment reduced stroke incidence when associated with oral-health interventions.<sup>(14)</sup> However, the existing evidence is not yet sufficient to either support or reject the potential benefits of periodontal treatment in the prevention or delay of CVDs.<sup>(15)</sup>

Considering the relevance of this subject, this clinical case describes the importance of periodontal treatment on a patient with CVD and PD who suffered an AMI. This case report aims to determine the impact of periodontal treatment on the health status of a patient with CVD, based on microbiological analyses.



## **IV. CASE REPORT**



#### **IV. CASE REPORT**

A 53-year-old male patient, J.Z., visited the Clinic of the Masters in Dentistry at Veiga de Almeida University with a chief complaint of gingival bleeding and tooth sensitivity. During anamnesis, he reported having high blood pressure and having been submitted to heart surgery for myocardial revascularization some years ago, being at the time under routine medical follow-up. He was advised to seek an oral-health assessment to check his periodontal condition. No alterations worth recording were observed in the extraoral examination. In the intraoral examination, the results of the DMFT (decayed, missing, and filled permanent teeth) index were as follows: D-0, M-2, F-11, T-2; total DMFT: 13. His oral cavity was photographed, as shown in Figures 1, 2, 3, 4, and 5.



**Figure 1** - Frontal view.



**Figure 2.** Right-side view.



**Figure 3.** Left-side view.



**Figure 4.** Upper occlusal



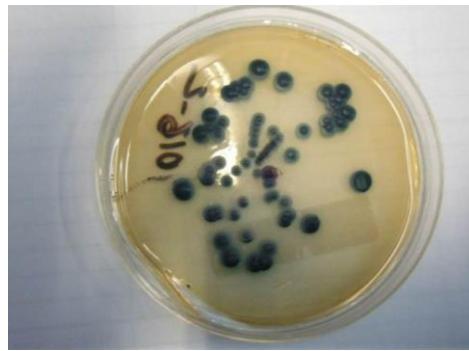
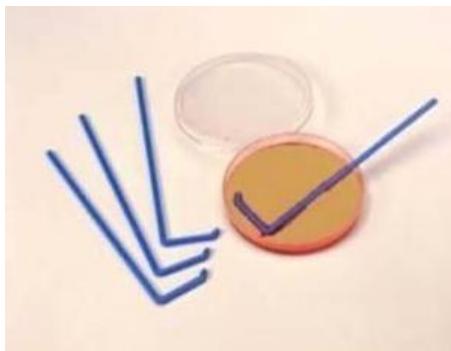
**Figure 5.** Lower occlusal view.

Periodontal probing revealed poor clinical attachment, no tooth mobility, and periodontal pockets at 4 mm and 7 mm in 7 teeth (14 distal, 26 distal, 42 mesial, 43 mesial, 46 distal, 31 mesial, and 34 distal). The patient wore a removable partial denture in his lower arch and had some tooth rotation. Mycological and bacterial analyses were conducted in every periodontal pocket, using proper instruments for crevicular fluid withdrawal (Figure 6).



**Figure 6.** Instruments used for the collection: periodontal probe, absorbent paper cone, gauze or cotton roll, scissors, dental tweezers, intraoral mirror, and Drigalski spatula to spread the cone on the culture medium.

Subgingival biofilm specimens were collected from each tooth after relative isolation, using two size-45 sterile paper cones (Dentsply, USA) – one for the bacterial analysis and the other for the mycological analysis – that were inserted in each periodontal pocket and left in place for 30 seconds. The tongue was subjected to the same assessment, with the cone scraped on its surface three times. The mycological analysis was specific for *Candida albicans* yeast growth. After collection, the material was placed on Sabouraud culture media with chloramphenicol and CHROMagar Candida (DIFCO, Detroit, Michigan, USA), and spread on the Petri plate using the Drigalski spatula with the rolling technique (Figure 7).



**Figure 7.** Drigalski spatula (spreading) and culture of fungi colonies.

The specimens for the mycological analysis were sent within 24 hours to the Mycology Laboratory of the Evandro Chagas Research Institute (IPEC/FIOCRUZ/Rio de Janeiro), and were incubated at 37°C for 48 hours and then at room temperature for five additional days. The colonies were transferred to agar to obtain pure cultures and were subsequently identified through biochemical reactions (API 20C Aux system, bioMérieux Vitek, inc., Hazelwood, Mo., USA). The analysis was positive for *Candida albicans* yeasts in the tongue and the periodontal pockets of teeth 26 and 42.

The bacterial analysis was performed in the microbiology laboratory of the Guarulhos University, using the checkerboard DNA-DNA hybridization molecular identification technique, which prepares the DNA probes based on a list of 40 bacterial strains (Table 1).

**Table 1.** Identification and count of bacterial strains for the preparation of DNA probes.

Blue complex						Orange complex (cont.)			
<i>Actinomyces gerencseriae</i>		23860 <sup>a</sup>	<i>Fusobacterium</i>	<i>nucleatum</i>	ssp	25586 <sup>a</sup>			
<i>Actinomyces israelii</i>		12102 <sup>a</sup>	<i>nucleatum</i>				<i>nucleatum</i>	ssp	10953 <sup>a</sup>
<i>Actinomyces naeslundii</i> 1		12104 <sup>a</sup>	<i>polymorphum</i>				<i>vincentii</i>		49256 <sup>a</sup>
<i>Actinomyces naeslundii</i> 2		43146 <sup>a</sup>	<i>Fusobacterium periodonticum</i>						33693 <sup>a</sup>
Purple complex						<i>Parvimonas micra</i>			33270 <sup>a</sup>
<i>Actinomyces odontolyticus</i>		17929 <sup>a</sup>	<i>Prevotella intermedia</i>						25611 <sup>a</sup>
<i>Veillonella parvula</i>		10790 <sup>a</sup>	<i>Prevotella nigrescens</i>						33563 <sup>a</sup>
Yellow complex						<i>Streptococcus constellatus</i>			27823 <sup>a</sup>
<i>Streptococcus gordonii</i>		10558 <sup>a</sup>	Red complex						
<i>Streptococcus intermedius</i>		27335 <sup>a</sup>	<i>Tannerella forsythia</i>						43037 <sup>a</sup>
<i>Streptococcus mitis</i>		49456 <sup>a</sup>	<i>Porphyromonas gingivalis</i>						33277 <sup>a</sup>
<i>Streptococcus oralis</i>		35037 <sup>a</sup>	<i>Treponema denticola</i>						B1 <sup>b</sup>
<i>Streptococcus sanguinis</i>		10556 <sup>a</sup>	Other species						
Green complex						<i>Eubacterium saburreum</i>			33271 <sup>a</sup>
<i>Aggregatibacter actinomycetemcomitans</i>		43718 <sup>a</sup>	<i>Gemella morbillorum</i>						27824 <sup>a</sup>
a + b		29523 <sup>a</sup>	<i>Leptotrichia buccalis</i>						14201 <sup>a</sup>
<i>Capnocytophaga gingivalis</i>		33624 <sup>a</sup>	<i>Neisseria mucosa</i>						19696 <sup>a</sup>
<i>Capnocytophaga ochracea</i>		33596 <sup>a</sup>	<i>Prevotella melaninogenica</i>						25845 <sup>a</sup>
<i>Capnocytophaga sputigena</i>		33612 <sup>a</sup>	<i>Propionibacterium acnes I + II</i>						11827 <sup>a</sup>
<i>Eikenella corrodens</i>		23834 <sup>a</sup>							
Orange complex						<i>Streptococcus anginosus</i>			33397 <sup>a</sup>
<i>Campylobacter gracilis</i>		33236 <sup>a</sup>	<i>Treponema socranskii</i>						S1 <sup>b</sup>
<i>Campylobacter rectus</i>		33238 <sup>a</sup>							
<i>Campylobacter showae</i>		51146 <sup>a</sup>							
<i>Eubacterium nodatum</i>		33099 <sup>a</sup>							

<sup>a</sup> ATCC (American Type Culture Collection); <sup>b</sup> Forsyth Institute

The cones with collected material were immediately stored in previously-identified individual plastic tubes containing 150 µL of a TE buffer solution, 10 mM of Tris-HCL (Invitrogen Life Technologies, CA, USA), and 1 mM of EDTA (Labsynth, SP, Brazil), at a pH of 7.6; 100 µL of NaOH (Labsynth) at 0.5M were added to maintain the bacterial DNA viable. After collection, the tubes were refrigerated at -20°C. The bacteria studied with this technique were detected in 82.5% of the teeth. The bacteria most commonly found in the subgingival biofilm were *F. nucleatum* and *T. denticola*, and the least common were *A. naeslundii*, *A. odontolyticus*, and *P. acnes*. The total bacterial count was  $0.23 \pm (0.57) \times 10^5$ .

After the collection, basic periodontal treatment and further instructions on oral hygiene and plaque control were provided. The patient was diagnosed with chronic periodontitis,<sup>(16)</sup> an infectious disease that results from inflammation of the supportive tissues and progressive loss of connective-tissue attachment, also involving edema, gingival bleeding on probing, and erythema. The full-mouth protocol, which entails scaling and root planing in the whole mouth and rinsing with 2% chlorhexidine within 24 hours, and subgingival irrigation with 0.2% chlorhexidine, was followed. At home, the patient was instructed to toothbrush at least three times daily, rinse with 0.2% chlorhexidine twice a day, tongue scrap, and spray chlorhexidine onto his throat. This protocol is known as "full-mouth disinfection" and prevents reinfection caused by untreated areas.

The following week, we were informed that the patient had suffered an AMI. He was hospitalized in the Intensive Care Unit for 29 days. After his recovery, he was again subjected to periodontal probing, collection of subgingival biofilm, and new mycological and bacterial analyses in every periodontal pocket (Table 2).

**Table 2.** Variables analyzed before and after periodontal treatment.

	Before periodontal treatment	After periodontal treatment
Spontaneous gingival bleeding	present	absent
Gingival bleeding to probing	present	absent
Mean depth of periodontal pockets	5.4 mm	4.6 mm
Bacterial count	$0.23 \pm (0.57) \times 10^5$	$0.04 \pm (0.13) \times 10^5$
Bacterial frequency: The most common	<i>F. nucleatum</i> <i>T. denticola</i>	<i>P. nigrescens</i> <i>A. gerencseriae</i>
Bacterial frequency: The least common	<i>A. naeslundii</i> <i>A. odontolyticus</i> <i>P. acnes</i>	<i>F. nucleatum</i>
Microbiological analysis	82.5% affected teeth	42.5% affected teeth
Mycological analysis: <i>Candida albicans</i> yeast	present	absent

No gingival bleeding was observed, either spontaneously or after probing. The mean depth of his periodontal pockets had markedly decreased from 5.4 mm to 4.6 mm. The mycological analysis revealed no fungal species and was negative for *Candida albicans* yeast. The microbiological analysis detected 42.5% affected teeth, and the total bacterial count was:  $0.04 \pm (0.13) \times 10^5$ . Regarding bacterial frequency, the most common bacteria after treatment were *P. nigrescens* and *A. gerencseriae*, and the least common were *F. nucleatum*.



## **VI. DISCUSSION AND CONCLUSIONS**



## **VI. Discussion and Conclusions**

The oral microbiota comprises several microbial species that live in balance with the immune system; an imbalance may result in opportunistic infections.<sup>(17)</sup> The dental biofilm is considered a trigger for periodontal disease due to its bacteria and their metabolic products,<sup>(18)</sup> which can cause an imbalance and originate PD. The loss in connective-tissue attachment results from the hyperactivity of leukocytes and the presence of cytokines and metalloproteinases that arise from infectious and inflammatory responses to stimuli. Although gram-negative bacteria play a role in the pathogenesis of PDs, there is not just one etiological agent.<sup>(19)</sup> PDs are thought to result from the interaction between a complex microbiota, the subgingival environment, and the host.<sup>(20)</sup> They are associated with complex microorganisms, but some of these have not yet been identified, making it difficult to establish their role in PD's pathogenesis.<sup>(21)</sup>

The molecular identification of microorganisms in periodontal disease is achieved through checkerboard DNA-DNA hybridization and *in-situ* hybridization techniques used to understand the composition and the role of the periodontal biofilm, as well as identify periodontal pathogens such as *A. actinomycetemcomitans*, *P. gingivalis*, and *T. denticola*. These bacteria are not limited to deep periodontal pockets and may be found in high numbers in the supragingival biofilm, requiring a more effective plaque control.<sup>(22)</sup>

The cardiologist should understand the need to refer the cardiac patient to a routine periodontal assessment so as to reduce the number of bacteria and change the oral microbiota. CVD is the most common systemic condition in patients with PD, and the inflammatory process is a factor in common. According to studies conducted by the Third National Health and Nutrition Examination Survey, patients with PD have a four-times higher incidence of AMI than healthy patients.<sup>(23)</sup> It has been suggested that PD may have an etiological modulating effect and become a risk factor for some systemic diseases. Therefore, risk factors should be eliminated or kept under control, as they may negatively affect treatment.

Bokhari *et al.* reported that both the prevalence and incidence of CVDs increased in the presence of PD and that patients with PD were more susceptible to the generation of atherosclerosis due to higher levels of inflammatory mediators.

The use of hybridization techniques is a great asset for PD since the success of periodontal therapy depends on identifying the microorganisms involved in the etiopathogenesis of the disease.<sup>(24)</sup> Periodontal treatment may help in the prevention of CVDs and PD.<sup>(25)</sup> Studies have shown the effectiveness of periodontal treatment in reducing the response of inflammatory markers in the acute stage and improving the endothelial function.<sup>(26)</sup> The reduction of anaerobic gram-negative microorganisms in the subgingival plaque has been described as the most important action of periodontal treatment.

The positive impact of periodontal treatment on the patient becomes evident with a marked reduction of the mean periodontal pockets' depth, lack of clinical signs of PD, and a significant reduction of the number of bacteria and microorganisms. Therefore, it is important to identify the pathogens before and after treatment, through microbiological and mycological analysis, to establish prevention and intervention strategies for PD and to minimize its impact on the development of CVDs.



## **VII. REFERENCES**



#### **IV. REFERENCES**

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## **VIII. Attachment**



## Submission Confirmation

1 mensagem

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Para: Erica Alexandra Macedo Pessoa <[dra.ericamacedo@gmail.com](mailto:dra.ericamacedo@gmail.com)>

Author: Erica

Title: Impact of periodontal treatment on a patient with an episode of acute myocardial infaction - a clinical case.

Revista Portuguesa de Estomatologia, Medicina Dentária e Cirurgia Maxilofacial

Dear Erica

Realizou com sucesso a submissão online do seu trabalho denominado "Impact of periodontal treatment on a patient with an episode of acute myocardial infaction - a clinical case.", que se encontra atualmente em consideração para publicação na Revista Portuguesa de Estomatologia, Medicina Dentária e Cirurgia Maxilofacial. No entanto, se o seu trabalho não cumprir as exigências da revista, será notificado via e-mail das alterações específicas que deverá introduzir no manuscrito para que possa ser considerado para publicação.

Your manuscript entitled "Impact of periodontal treatment on a patient with an episode of acute myocardial infaction - a clinical case." has been successfully submitted online and is presently being given full consideration for publication in the Revista Portuguesa de Estomatologia, Medicina Dentária e Cirurgia Maxilofacial. Should your manuscript not comply with the Journal's requirements, however, the Journal's administrator will notify you via email that you need to make specific changes to your manuscript before it can be considered for publication in the Revista Portuguesa de Estomatologia, Medicina Dentária e Cirurgia Maxilofacial.

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