THE IMPORTANCE OF PERIODONTAL THERAPY IN PATIENTS WITH ASSOCIATED SYSTEMIC DISEASES. CASE REPORT

Mihaela Moisei[1], Mariana Păcurar[2], Simina Chelărescu[3] , Mioara Decusară[4]

[1,4] "Lower Danube", University of Galați, Faculty of Medicine and Pharmacy, Department of Dentistry
[2] "George E.Palade" University of Medicine, pharmacy, science and Technology of Târgu Mureș, Faculty of Dentistry, Department of Orthodontics
[3] „Titu Maiorescu" University of Bucharest, Faculty of Dentistry

Corresponding authors: mihaela.moisei@ugal.ro; mioara.decusara@ugal.ro

Abstract

Aim : this paper highlights the importance of a complex oral rehabilitation treatment within a multidisciplinary team for a patient with chronic marginal periodontal stage IV pathology associated with gingival overgrowths and correlative systemic pathology.

Material and method: is analyzed the case of a 45-year-old female patient who requested dental treatment for gingival inflammation with gingivorragia, gingival overgrowths, dental mobility and malpositions of incisors, on the background of general status affected by obesity, type II diabetes mellitus and hypertension. To establish the diagnosis and the complex treatment plan was performed local clinical examination (inspection, palpation and periodontal probe-PD), orthopantomography (for the assessment of alveolar bone lysis) and blood analysis.

Results: The evolution of the periodontal status was favorable following local procedures applied (guided microbial plaque therapy, closed-field periodontal curettage, laser removal of residual gingival hyperplasia) under general antibiotic protection, and radiological, the level of bone atrophy remained constant 4 years after the start of treatment. The periodontal disease stabilized with a good prognosis during the monitoring period and a positive impact on the general status as well.

Conclusions: Adequate control of periodontal disease, with correct dental hygiene and appropriate multidisciplinary treatment can have a positive impact on systemic conditions, greatly improving the patient's quality of life.

Key words: deep periodontitis, systemic diseases, multidisciplinary therapeutic algorithm

Introduction

Periodontal disease is a complex chronic inflammatory condition , multi-causal and with non-linear progression, based on an immune dysfunction underlying the altered host response to local microbial aggression[26,29]. Epidemiologic studies have demonstrated the predisposing contribution of general factors such as diabetes, cardiovascular disease, obesity and their interaction with important local factors such as microbial bacterial plaque and gingival calculus[4,8,11]. Type II diabetes mellitus, a metabolic disease with increasing global prevalence, that affects all age groups, acts at a systemic level through tissue acidosis, generalized vascular neuritis and decreased resistance to infections[5,12]. In the oral cavity, it causes hyposialia with a decrease in the amount of salivary immunoglobulin A and implicitly a decrease in local resistance to infections and degenerative damage to the terminal

periodontal circulation[13,19]. The local effects are materialized by frequently hyperplastic gingivitis with a tendency to evolve towards destructive forms of chronic marginal periodontitis, the acceleration of bone lysis and the tendency to form periodontal abscesses. [13,19]. To these is most frequently added a defective dental hygiene, determined by the fear of the occurrence of gingival bleeding during tooth brushing[18].

Cardiovascular diseases (hypertension, ischemic heart disease, stroke), with increased incidence in many countries and starting with age groups of 40 year-old patients, also interfere with periodontal pathologies[1,7,9,15]. Thus, arterial hypertension decreases the quality of the local and general immune response to the aggression of periodontal pathogens due to the impairment of dental-periodontal vasomotility, favoring the increase of bacterial aggression and pathogeny. At the same time, long-term antihypertensive medication can stimulate local cell proliferation with periodontal manifestations of gingival over-growths through hyperplasia and demineralization of the interdental septa[10,25]. The phenomenon of atherosclerosis, which is frequently associated with cardiovascular diseases, decreases the blood supply of the periodontal fileds through an embolic mechanism, leading to an increase in gingival vascular friability[24, 28].

Case report

A 45-year-old female patient requested treatment for significant gingival over-growths, gingival bleeding, tooth mobility, purulent gingival secretions, fetid halena and difficulties in mastication against a background of general status affected by obesity, type II diabetes and hypertension. The local clinical examination was performed by inspection, palpation and periodontal survey-PD- establishing the OHI oral hygiene index, the PBI papillary bleeding index, the gingival inflammation index IG, the dental mobility index Im, the value of gingival recessions and attachment loss.

The general status revealed by the anamnesis and the evaluation of the medical documents highlighted a complex pathology: NYHA class II heart failure, HTA grade III very high risk - 13 years ago, stroke in the right carotid territory- 3 years ago, diabetes mellitus type II - 10 years ago, obesity, dyslipidemia and systemic atheromatosis.

The patient was monitored every 3 months by the specialist in diabetology and every 6 months by the specialist in cardiology. The specialists' recommendations were:

- low fats, carbohydrates, sweets diet

- avoid physical effort, stress, exposure to extreme temperatures

- support medication for cardiovascular diseases with antiplatelet agent (Aspenter) , beta blocker (Concor and Preductal), antihypertensive (Triplixam), coronary artery dilator (Nitromint) and statins (Sortis). For diabetes the patient is treated with one dose of insulin daily, Byetta once a week and for immune support Procor Forte a complex of

Coenzyme Q10, magnesium, selenium and zinc.

Complementary examinations also included laboratory analysis: hemoleukogram ($11x10^9$/L for leucocytes), glycosylated hemoglobin (7,6%), ESR, C-reactive protein CPR.

Following the clinical examination of the oral cavity, a poor state of oral hygiene and important changes in the periodontal soft tissues were detected (fig.1):

• marked gingival inflammation with changes: in color -bright red-, in volume - gingival overgrowth with the involvement of the papillae but also of the free gingival margin, being more marked at sextant level II and V and texture changes - fibrotic appearance and edema;

• inflammatory gingival recessions predominantly on the oral surfaces;

• purulent secretions in the gingival sulcus;

• important dental migrations with the appearance of dental spaces and diastemas;

• significant deposits of calculus and bacterial plaque;

• affecting the aesthetic, masticatory and phonatory functions.

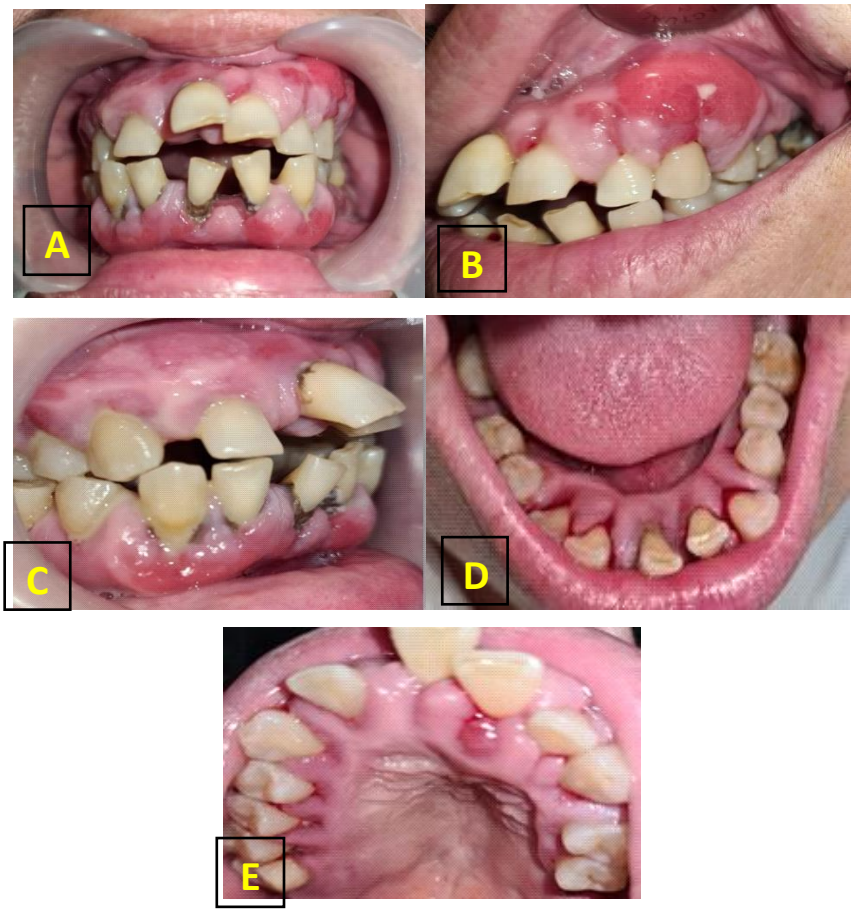

Fig. 1 Initial intraoral view: A- Occlusal frontal view, B-Left lateral view, C- Right lateral view, D-Mandibular dental arch, E- Maxillary dental arch

The quantification, by specific indexes, of the changes in the superficial periodontium showed the following results: Loe and Silness plaque index- 2, papillary bleeding index PBI 3, gingival inflammation index IG- 3.

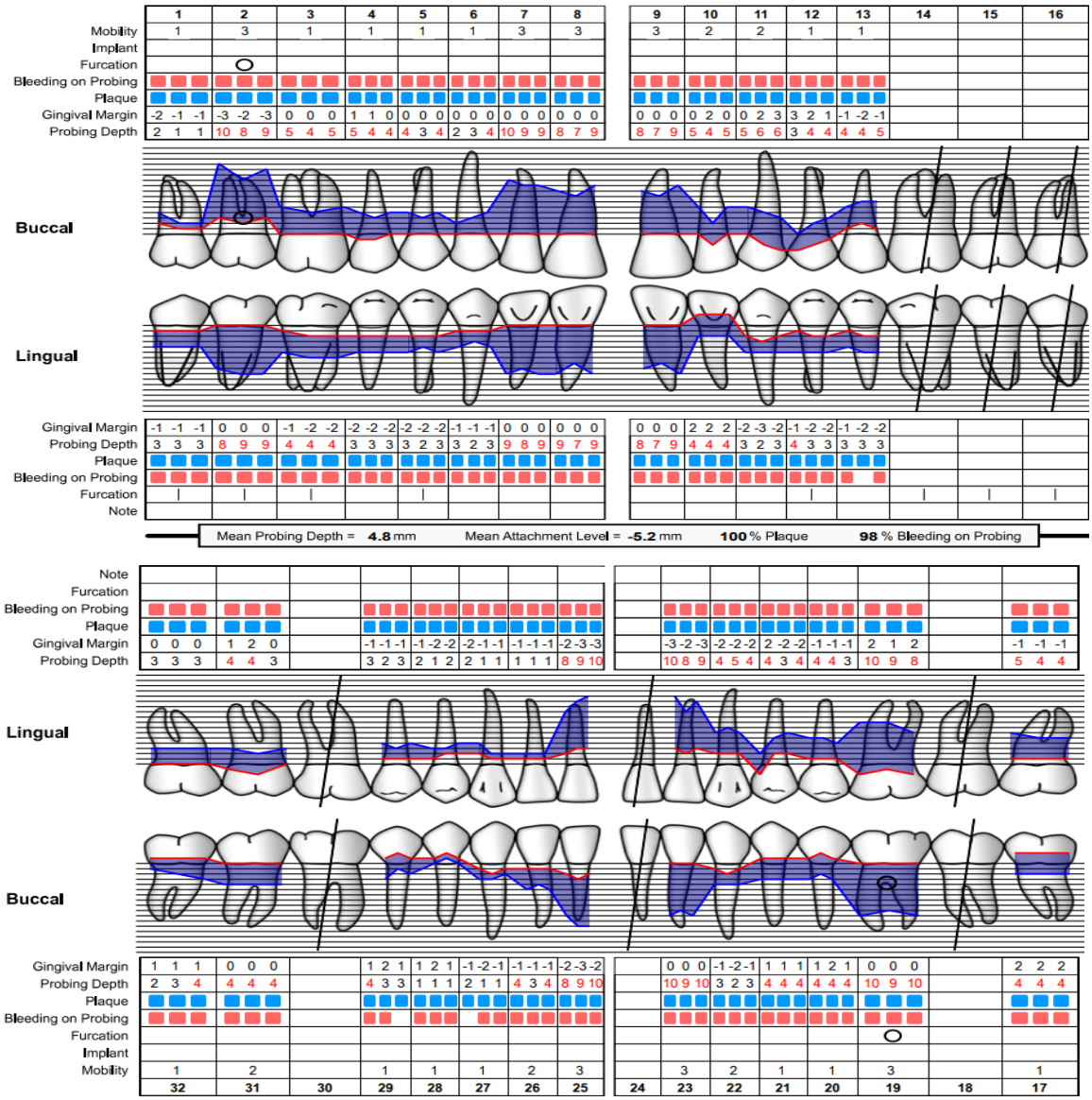

Fig.2 Initial periodontogram

The recorded values, at the probing depth, were between 5 and 10 mm and in the multi-radicular teeth, III grade furcation defect was detected at sites 1.7 and 3.6. (figure 2). They were correlated with the images provided by the orthopantomogram where, a generalized bone lysis was visualized with a predominantly horizontal profile and a

variable respectively medium and deep depth, but also total in teeth 1.6 and 3.6. Dental mobility testing revealed values of I and II at sextants 1, 3, 4, 6, and values of III at sextants 2 and 5, in accordance with the degree of bone destruction visualized on the MRI. (figure 3)

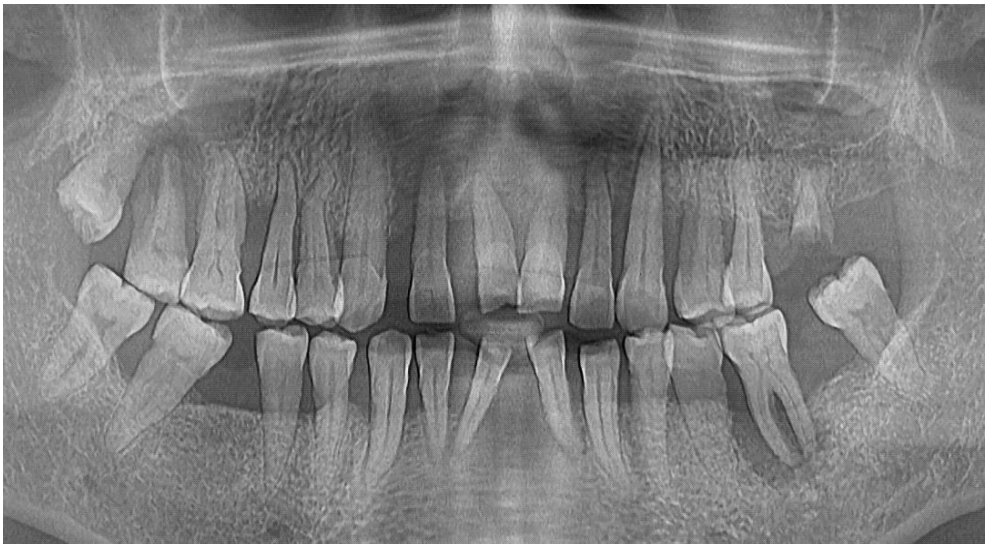

Fig. 3 Orthopantomogram - initial status

Due to the general status of the patient with a high risk of bacteremia, it was decided that the clinical investigation of the deep periodontium, by periodontal survey, should be performed after the institution of antibiotic prophylaxis and the correction of the gingival-dental brushing technique. The written consent of the attending physicians was also obtained, for the specific periodontal treatment, under safe conditions, for the patient. The general protective medication consisted of the administration of an antibiotic for 10 days, Clindamycin 600 mg.x 2/day, simultaneously with an antifungal, Diflucan 150 mg one tablet/daily. At the same time, a rigorous self-control protocol of microbial dental plaque was established with therapeutic oral hygiene products, such as a toothpaste with the addition of 0.12% chlorhexidine and non-alcoholic mouthwash with 0.2% digluconate of chlorhexidine twice a day after brushing, for 14 days.

The periodontal diagnosis was of chronic generalized periodontitis stage IV grade C, in active stage revealed by the presence of generalized purulent secretions.

The therapeutic algorithm included the following stages:

1. health education for optimal oral hygiene and increase patient awareness of the correlation between general and local pathology of the oral cavity

2. professional sanitization by Guided Biofilm Therapy and completed by manual instrumentation

3. extraction of irrecoverable teeth -1.7,1.1, 2.1, 3.6, 4.1

4. closed-field periodontal curettage assisted by diode laser therapy with a 960nm wavelength

5. gingivectomy and gingivoplasty with laser assisted for site 2.3

6. dental immobilizations intra and extracoronal by composite techniques with glass fiber strips

7. temporary adjunct prosthesis

8. monitoring at predetermined intervals, depending on the local therapeutic results and the evolution of the systemic pathology

All the procedures aimed at instrumenting the periodontal tissues and tooth extractions were carried out under antibiotic and antifungal protection, therapy approved by the attending physicians of systemic pathology. Professional cleaning initially consisted of applying a subgingival antiseptic solution and supragingival scaling with piezoelectric equipment followed by professional brushing. After 6 days, when the purulent sulcular secretion remitted, piezoelectric and manual subgingival scaling was performed, completed by the airflow and perioflow procedure. After correcting the acute periodontal inflammatory phenomenon, the unrecoverable teeth were extracted, the post-extraction wounds being treated with local hemostatic, followed by suturing. Closed-field periodontal curettage interventions were scheduled for the remaining teeth, choosing the option of minimally invasive instrumentation with the diode laser of 960nm wavelength (Figure 4). Gingivectomy with gingivoplasty for site 2.2-2.3 was also performed with laser assistance, thus reducing the risk of significant postoperative bleeding and healing time. All remaining teeth were immobilized by adhesive techniques that included fiberglass strips. (Figure 5). Adjunct prosthesis was performed for the frontal maxillary and mandibular edentulous areas. (Figure 6) The patient attended all monitoring sessions and had a favorable local status with optimal maintenance of oral hygiene and periodontal clinical and radiological parametres. After the completion of the periodontal treatment, 6 month later, the number of leukocytes decreased to a value of $8x10^9$, which corresponds to the physiological parameters.

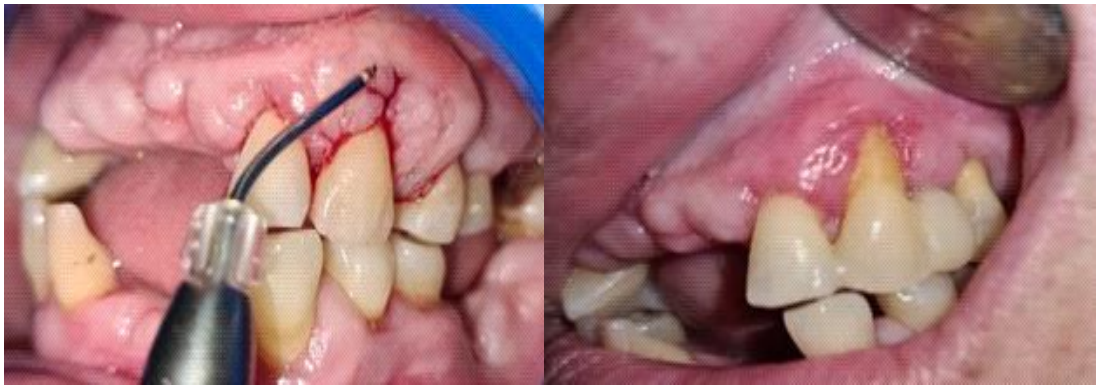

Fig. 4 Intraoral appearance laser gingivectomy treatment and healing evolution after applying the immobilization system

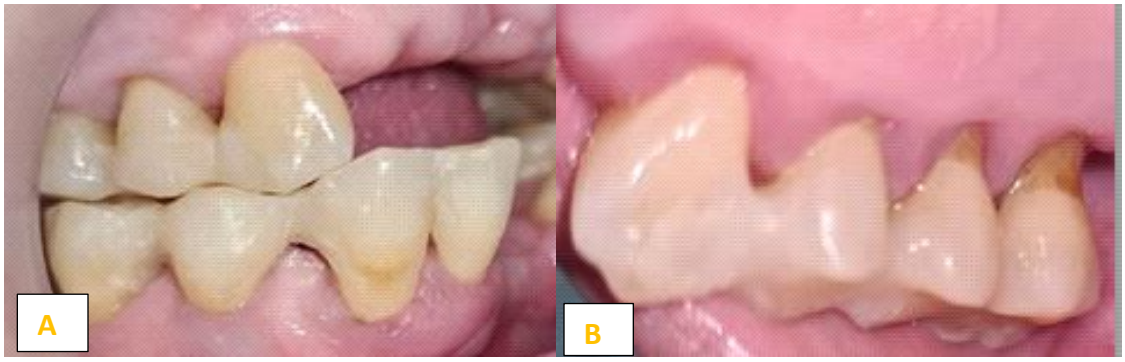

Fig 5 Intracoronal immobilization –quadrant II and IV , vestibular (a) and palatinal (b) aspect

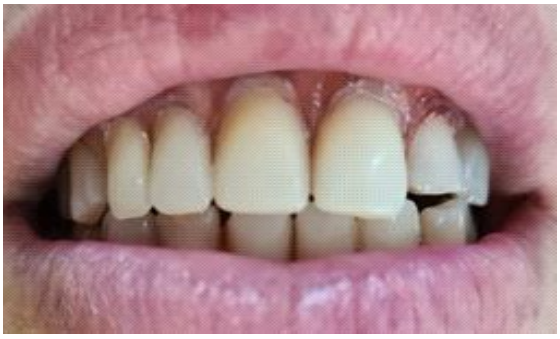

Fig.6 Normal frontal aspect with adjunct prosthesis

The patient was monitored at 3-month intervals for 2 years and later at 6 months, being under observation for four years.

Periodic evaluations recorded physiological values for oral hygiene indices, gingival inflammation and papillary bleeding, and at the periodontal survey the values were between 2 and 3 mm. (figure 7)

Fig. 7. Intraoral views after 4 years: A –Frontal view ,B – Left view, C-Right view

The radiological image taken 4 years after the initiation of the treatment reveals the maintenance of a constant bone level with the mineralization of the interdental septa (figure 8).

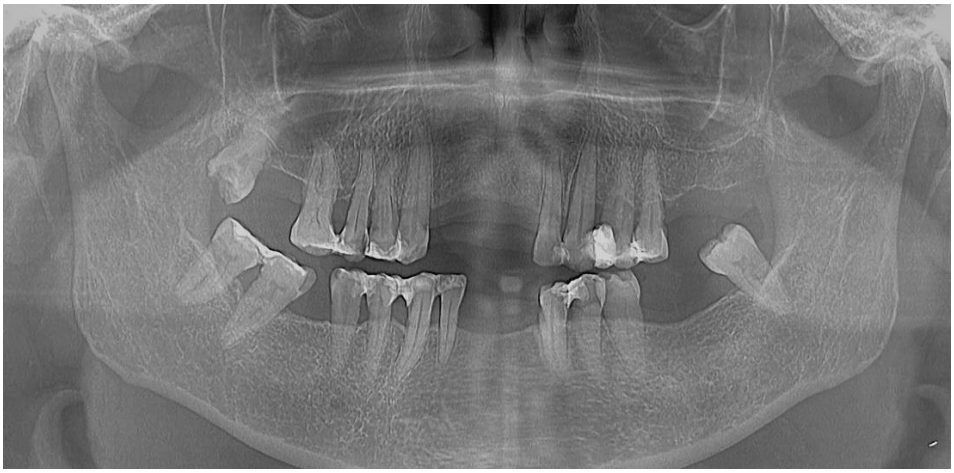

Fig.8 Orthopantomogram after 4 years

Also, the periodontogram performed 4 years after the oral rehabilitation treatment shows the stabilization of horizontal bone atrophy and the absence of vertical bone atrophy (figure 9).

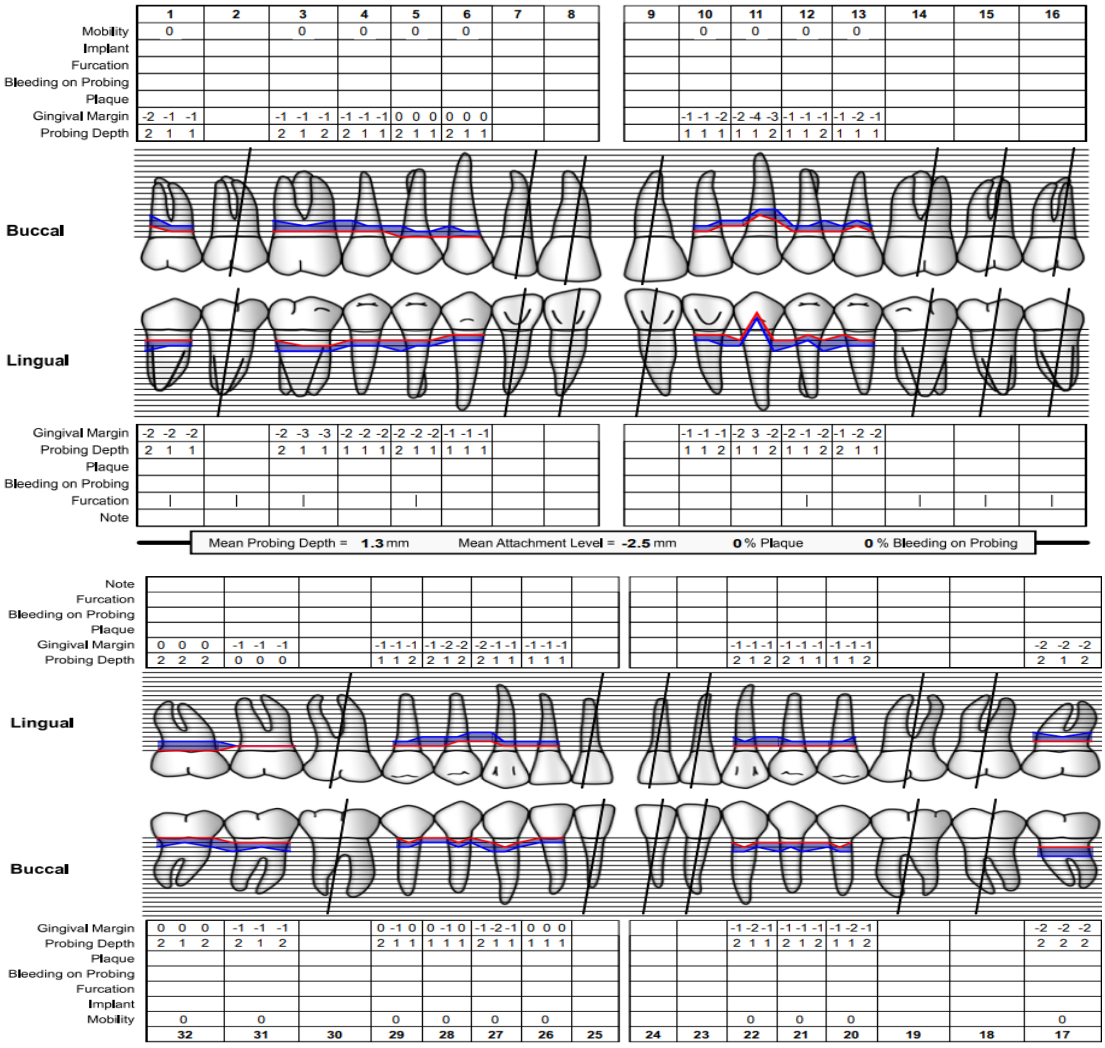

Fig.9 Final periodontogram

Discussions

The association between an important systemic pathology represented by diabetes, obesity, cardiovascular diseases and periodontal disease, in a patient with inadequate control of microbial dental plaque, led to the maintenance and aggravation of local and general pathological phenomena. A 10 year-old diabetes mellitus, hardly managed with medication, favored the onset of periodontal disease in this patient who already had a genetic predisposition on the maternal line for marginal periodontitis. The manifestations detected at the level of the superficial periodontium with bleeding, purulent secretions, ulcerations, confirmed the unfavorable repercussions of diabetes on the periodontal structures, as demonstrated by the numerous studies in the field

literature[6,21]. At the same time, the absence of adequate self-control of microbial plaque and early periodontal treatment, played a major role in the maintenance of high glycemic values of 7.6 % of glycated hemoglobin, even under conditions of rigorous monitoring by the diabetologist. The drop of this value to 6,6%, one year after the beginning of the periodontal treatment, has proven the favorable effect on the diabetic status within the bidirectional relationship between these two pathologies[22,23,27].

Arterial hypertension, detected prior to diabetes, had, as a side effect, gingival changes such as overgrowhs. The targeted territory included, in addition to the free gingival margin and papillae, important portions of fixed gingiva. Scientific research has indicated the possibility of the appearance of these local manifestations as a side effect of the therapy with calcium antagonists, an aspect also detectable in this clinical case[3,20]. The additional installation of the NYHA class II heart failure, had, as general repercussions, the limitation of physical effort with the gradual onset of of obesity. The degenerative changes of the periodontal vasculature, already disturbed by the presence of diabetes, were, thus, amplified. Thus, in the case of the present cardiovascular pathology, the untreated periodontal disease contributed to the amplification of the inflammation of the vascular endothelium, due to the pathogeny mechanisms of the periodontal pathology microorganisms. Severe forms of marginal periodontitis, with periodic exacerbations, have been described as a risk factor for stroke, a certified situation in the given case.[2,14,16].

General antibiotic therapy was a priority in the beginning of treatment both to decrease acute periodontal inflammation and to avoid bacteremia in according to standardized protocols.[17]

Conclusions

The association between a too late treated periodontal disease and two important general conditions such as diabetes and hypertension, led to the unfavorable evolution of the entire pathology. The early treatment of these categories of patients, within a multidisciplinary team that must include a dental specialist, is essential for the stabilization and their favorable evolution, both locally and systemically.

References

1. Alessandra Blaizot, Jean-Noël Vergnes, Samer Nuwwareh, Jacques Amar and Michel Sixou Toulouse *Periodontal diseases and cardiovascular events: meta-analysis of observational stud* systematic review, International Dental Journal 2009; 59: 197-209;
2. A Lafon 1, B Pereira, T Dufour, V Rigouby, M Giroud, Y Béjot, S Tubert-Jeannin *Periodontal disease and stroke: a meta-analysis of cohort studies* Eur J Neurol 2014; Sep;21(9):1155-61
3. Balan, H., E. Popescu, and G. Angelescu., *Pathologic crossroads: cardio-vascular diseases, periodontal diseases and calcium antagonists*, Journal of medicine and life, 2011; 4(1): 2–10.

4. Bascones Martínez, Antonio, et al., *Periodontal disease and diabetes: review of the literature*, Med Oral Patol Oral Cir Bucal, 2011; 16 (6): e722-9;
5. Bissett SM, Pumerantz AS, Preshaw PM, *Periodontal disease and diabetes*, Journal of Diabetes Nursing, 2015; 19: 134–40;
6. Borgnakke, Wenche S., et al., *Effect of periodontal disease on diabetes: systematic review of epidemiological observational evidence*, Journal of periodontology, 2013, 84: 135-152;
7. Claudia Da Venezia, Nayib Hussein, Marcela Hernández, Johanna Contreras, Alicia Morales Macarena Valdés, Francisca Rojas, Loreto Matamala and Patricia Hernández-Ríos *Assessment of Cardiovascular Risk in Women with Periodontal Diseases* According to C-reactive Protein, Biomolecules 2021, 11(8), 1238;
8. Daniel, Rajkumar, et al., *Diabetes and periodontal disease*, Journal of pharmacy & bioallied sciences, 2012; 4 (2), S 280;
9. Fagundes NCF, Almeida APCPSC, Vilhena KFB, Magno MB, Maia LC, Lima RR *Periodontitis As A Risk Factor For Stroke: A Systematic Review And Meta-Analysis*, 2019; 15: 519—532
10. Fernandes, Marilene Issa, et al., *Effect of nifedipine on gingival enlargement and periodontal breakdown in ligature-induced periodontitis in rats*, Archives of oral biology, 2010, 55(7): 523-529;
11. FionaQ.Buia,CassioLuiz,CoutinhoAlmeidaSilvaab,BrandonHuynha,AlstonTrinha,JessicaLiua,JacobWoodwarda,HomerAsadia,DavidM.Ojcius, *Association between periodontal pathogens and systemic disease*, Biomedical Journal 2019; 42(1): 27–35
12. Graves D.T., Ding Z., Yang Y., *The impact of diabetes on periodontal diseases*, Periodontology 2000, 2019; 214-224; 2020;82(1):214-224
13. Teodor Salmen, Bianca Margareta Mihai, Ruxandra Andreea Iarca, Bianca Adriana Stan, Vlad Dima, Roxana Elena Bohiltea *Diabetes mellitus and periodontal disease*: Ro J Stomatol 2021;67(4):244-246
14. Kapellas, Kostas, et al., *Associations between periodontal disease and cardiovascular surrogate measures among Indigenous Australians*, International journal of cardiology, 2014, 173(2): 190-196;
15. Larvin, Harriet, et al., *Risk of incident cardiovascular disease in people with periodontal disease: A systematic review and meta-analysis, Clinical and experimental dental research*, 2021, 7(1): 109-122;
16. Liccardo, Daniela, et al., *Periodontal disease: a risk factor for diabetes and cardiovascular disease*, International journal of molecular sciences, 2019, 20(6): 1414;
17. Mihaela Moisei, Liliana Pasarin, Sorina Solomon, Cornelia Oanta, Diana Tatarciuc, Irina Ursarescu, Silvia Mârțu, *The Role Of Antibiotherapy In The Oral Rehabilitation Of The Periodontal Affected Patient*, Romanian Journal of Oral Rehabilitation. 2015 January/ March;6(1):107-112.
18. Mioara Decusară, Daniela Cornea, Carmen Tiutiuca, Alina Ramona Dimofte, Gabi Topor, Denisa Batir Marin, Gabriela-Violeta Iordăchiță- *Local therapy management of oral pathology in patiens with fixed orthodontic appliances during the Covid-19 pandemic*, Romanian Journal of Oral Rehabilitation, 2022, 14(2):200-206 .
19. Mirza B.A.Q., Sayed A., Izhar F., Khan A., *Bidirectional relationship between diabetes and periodontal disease: Review of Evidence*, Journal Park Medicine Association, 2010; 60, (9):766-768;
20. Naba Kumar Pattnaik1, Surya Narayan Das2, Biranchi N Biswa, *Cardiovascular Diseases and Periodontal Diseases: Review and Update,* International Journal of Scientific Study | April 2017; 5 (1): 239-244;
21. Negrato, Carlos Antonio, et al., *Periodontal disease and diabetes mellitus*, Journal of Applied Oral Science, 2013; 21:1-12;

22. Robert J. Genco, Filippo Graziani, Hatice Hasturk *Effects of periodontal disease on glycemic control, complications, and incidence of diabetes mellitus* Periodontology2000, 2020 Volume 83 (1),59-65;
23. Stanko, Peter, and Lydie Izakovicova Holla., *Bidirectional association between diabetes mellitus and inflammatory periodontal disease*, Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub, 2014;158(1): 35-8;
24. Stewart, Ralph, and Malcolm West, *Increasing evidence for an association between periodontitis and cardiovascular disease*, 2016; 549-551;
25. Sunil, Paramel Mohan, et al., Nifedipine-induced gingival enlargement: *Correlation with dose and oral hygiene*, Journal of pharmacy & bioallied sciences, 2012, 4 (2): S191;
26. Syed Ameer Hamza, Saba Asif, Zohaib Khurshid, Muhammad Sohail Zafar, Syed Akhtar Hussain Bokhari, *Emerging Role of Epigenetics in Explaining Relationship of Periodontitis and Cardiovascular Diseases*, Diseases 2021, 9(3), 48;
27. Teodor Salmen, Bianca Margareta Mihai, Ruxandra Andreea Iarca, Bianca Adriana Stan, Vlad Dima, Roxana Elena Bohiltea *Diabetes mellitus and periodontal disease*: Ro J Stomatol 2021;67(4):244-246
28. Trevisan, Maurizio, and Joan Dorn., *The relationship between periodontal disease (pd) and cardiovascular disease (cvd)*, Mediterranean Journal of Hematology and Infectious Diseases 2010;2(3) e2010030
29. Vargas Segura A. I., Ilyina A.,Segura Ceniceros E. P., Silva Belmares Y. and Méndez González L. *Etiology and microbiology of periodontal diseases: A review* , African Journal of Microbiology Research, 2015,Vol. 9(48): 2300-2306.