



β -TCP (R.T.R.) and aggressive periodontitis

Djamila BOUZIANE - Professor, University of Oran, Algeria

Khadidja L. MAKRELOUF - Professor, University of Oran, Algeria

Mohamed BOUZIANE - Professor, University of Oran, Algeria

We have not found in the international literature any study regarding the use of β -TCP in the treatment of infrabony pockets in young patients with aggressive periodontitis, which are a clinical entity that very rapidly results in the destruction of alveolar bone with tooth morbidity as a consequence. For this reason the objective of this study is to evaluate and analyse the action of β -TCP (R.T.R.) on infrabony lesions in young patients suffering from aggressive periodontitis.

Introduction

Aggressive periodontitis, a clinical case frequent in the Maghreb, represents the most destructive form of periodontal diseases which results in bone lysis, dental mobility and then loss of teeth. Their appearance starts early in life causing aesthetic and functional damage.

These diseases are characterised by a anaerobic Gram negative flora.

There are two classes:

- The form localised in the incisors and the first molars, as shown clinically in Figure 1 and with X-ray in Figure 2.



Fig. 1: Clinical aspect



Fig. 2: Notice the terminal lysis at the level of 21

- The form generalised to the entire dentition (Fig. 3 & 4).

The size of the bone lesions requires a well targeted therapeutic strategy.

The objective of our work is to choose sites at the level of the upper or lower incisors and first molars of patients suffering from aggressive periodontitis with pockets deeper than 5 mm, infrabony defects that allow the insertion of β -TCP (R.T.R.). We will test the influence and impact of this substitute material on bone behaviour.

Materials & methods

We perform on each patient a clinical, radiological and bacteriological examination followed by a surgical treatment consisting in flap surgery combined with R.T.R. grafting material (syringe). The clinical evaluation consisted in measuring the depth of the pockets.

The X-ray examination allowed the evaluation of the bone level of our patients thanks to the retroalveolar - panoramic and radiovisiography images. The bacteriological evaluation consisted in subgingival specimens and fresh bacterial profile study, Gram staining, culture and PCR.

The therapeutic schedule consisted in:

- Initial therapy (scaling and root planing)
- A systemic anti-infectious therapy with amoxicillin 1 g/d and metronidazole 600 to 800 mg/d over 10 days.
- A surgical therapy combining flap surgery and insertion of R.T.R.
- The surgical sequence is the following:
 - Anaesthesia
 - Incisions
 - Careful debridement
 - Elimination of granulation tissue
 - Scaling and root planing
 - In situ placement of R.T.R. using the syringe presentation
 - Sutures
 - Placement of surgical pack
 - A maintenance phase with clinical and radiography re-assessments over four years



Fig. 3: Inflammatory aspect



Fig. 4: Terminal lysis at 21 and 11

Case series

4 patients were chosen for whom five sites were treated:

- 2 upper sites (16-21)
 - 3 lower sites (36-46-46)
- The first patient presented a generalised aggressive periodontitis with a severe intrabony defect at 21 associated with an extrusion and at the level of 16 a terminal bone lysis with a pocket depth of 9 mm.
 - The second patient suffered from generalised aggressive periodontitis with class 3 bifurcation involvement at the level of 46 as well as mesial and distal pocket depth of 7 to 5 mm.
 - The third patient, 17 years old, with localised aggressive periodontitis and mesial infrabony lesions with a pocket depth of 8 mm.
 - The fourth patient, 16 years old, with a localised aggressive periodontitis, presented 5 mm pockets distally at the level of 46.

Case Report no.1

A 19-year-old woman presented with severe generalised aggressive periodontitis.

On a clinical level, this patient presented an inflammatory condition of the maxillary and mandibular gum with migration of 21 (*Fig. 5*).

On a radiographic level, the panoramic X-ray of the maxillary showed infrabony lesions (*Fig. 6*) with deep lysis at the level of 21 and terminal lysis at the level of 16 (*Fig. 7*).

It was decided to treat 21 and 16 given the situation of these severe lesions.

The therapeutic schedule adopted is the following at the level of 21.

T0

- Etiological therapy + amoxicillin + metronidazole antibiotic therapy
- Surgical phase: flap + bone substitute: R.T.R. (*Fig. 8 & 9*)

T 1 year

- Clinical and X-ray reassessment. On the X-ray, filling of infrabony lesion with 25% bone gain. (*Fig. 10*)
- At this stage the migration treatment was performed.
- Orthodontic treatment phase due to migration of 21 (*Fig. 11*)

A stabilisation of the bone gain after initiation of orthodontic treatment was observed (*Fig. 12*)

The reassessment of the infrabony lesion at 4 years by radiovisiography showed a bone gain of 50% (*Fig. 13*). A definitive fixation was performed.



Fig. 5: Clinical inflammatory aspect

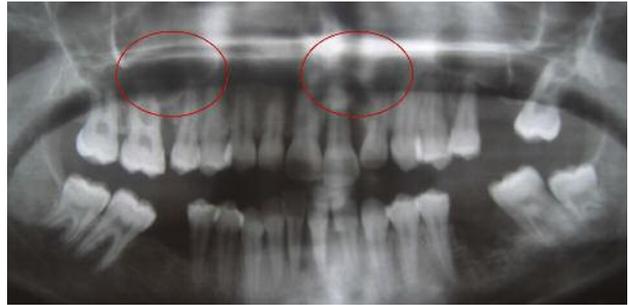


Fig. 6: X-ray :Generalised lysis at the level of the maxillary



Fig. 7: Severe infrabony defect at the level of 21



Fig. 8: Incisions - raising of a flap



Fig. 9: Insertion of bone substitute



Fig. 10: Result at 1 year



Fig. 11: DFO treatment of migration at 1 year



Fig. 12: Stabilisation of bone gain



Fig. 13: RVG 50% bone gain at 4 years



Fig. 14: Clinical aspect



Fig. 15: Terminal lysis

The study of the posterior site (16) in the same patient showed:

- On a clinical level, the probing detected pocket depths of 9 to 11 mm (Fig. 14).
- On the X-ray, a terminal bone lysis (Fig. 15).

The therapeutic schedule of the infrabony lesion of 16 is the following:

- Etiological therapy
- Surgical phase: mucoperiosteal flap associated with in situ placement of R.T.R.

The surgical treatment sequence:

- Incision and raising of the flap (Fig. 16)
- Debridement of lesion
- Elimination of granulation tissue
- Polishing and planing of root
- Insertion of R.T.R. using the syringe: the granules are mixed with a few drops of blood (Fig. 17)
- Sutures
- Surgical pack (Fig. 18)
- Placement of surgical pack



Fig. 16: Incision and raising of flap



Fig. 17: In situ placement of R.T.R.

The reassessment at 1 year shows the filling of the infrabony lesion (Fig. 19).

At 4 years it shows a 50% bone gain (Fig. 20).



Fig. 18: Surgical pack



Fig. 19: At 1 year



Fig. 20: At 4 years

Case Report no.2

A 18-year-old patient presented with severe generalised aggressive periodontitis (*Fig. 21*). The panoramic x-ray showed at the level of 46: Infrabony lesion + bifurcation involvement and depth of pockets of 7 mm (*Fig. 22*).

Clinical aspect before surgery (*Fig. 23*). Placement of R.T.R. (*Fig. 24*).

The radiography X-ray shows an infrabony lesion associated with a class 3 bifurcation involvement (*Fig. 25*). At 15 days the filling material is in place (*Fig. 26*).

At 4 years the reduction in the pocket depth is of 4 mm, we noticed the absence of bifurcation involvement (*Fig. 27*).



Fig. 21: Clinical aspect



Fig. 22: Class 3 bifurcation



Fig. 23: Clinical aspect

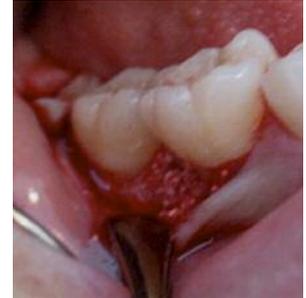


Fig. 24: Placement of R.T.R.



Fig. 25: Before treatment



Fig. 26: 15 days after the filling



Fig. 27: Result at 4 years

Case Report no.3

A 17-year-old patient presented with localised aggressive periodontitis (*Fig. 28*) with an average pocket depth of 8 mm and mesial infrabony lesion of 36 (*Fig. 29*).

The therapeutic schedule is the following:

- Etiological therapy associated with medical treatment which consisted in a combination of amoxicillin and metronidazole during 10 days
- Surgical treatment: incision (*Fig. 30*), raising of the flap, placement of the bone substitute (*Fig. 31*).

The results at 1 year (*Fig. 32*) and at 4 years (*Fig. 33*) are very satisfactory.



Fig. 28: Clinical aspect



Fig. 29: Deep lysis



Fig. 30: Placement of R.T.R.



Fig. 31: Sutures



Fig. 32: At 1 year



Fig. 33: At 4 years

Case Report no.4

The 16-year-old patient presented with inflammatory gum condition (*Fig. 34*). In the X-ray, we observed prior to treatment an infrabony defect of 46 with a pocket depth of 5 mm and 25% bone loss (*Fig. 35*).

For this patient, it was decided to follow the same therapeutical protocol as previously described.

At 4 years we see the filling of the infrabony lesion with an absence of periodontal pocket and a normal aspect of the desmodontal space (*Fig. 36*).



Fig. 34: Clinical aspect



Fig. 35: Infrabony defect before treatment



Fig. 36: Bone repair

Discussion

The use of R.T.R. (β -TCP) allowed:

- A reduction in the depth of pockets and an attachment gain.
- A decrease in the dental mobility index.
- The panoramic and the visiography X-rays showed a bone gain with filling of infrabony lesions.
- Modifications of the bacterial biofilm in numerous studies (Haffajee and al.) show that certain species of the red complex (*Tannerella forsythus*, *Treponema denticola*) and of the orange complex (*Prevotella intermedia*, *Campylobacter rectus*) can evolve differently. Depending on the surgical debridement, these species can recolonise the sites in a very delayed manner due to the decrease in their

toxic potential and the modification of their tissue environment.

We thus observed in our patients a decrease in bacteria such as *Tannerella forsythus*, *Prevotella intermedia*, *Porphyromona gingivalis*, *Aggregatibacterium actinomycetemcomitans*, *Treponema denticola* at 1 year and 4 years.

The flap combined with the filling would be in favour of the restoration of the epithelial barrier at the bottom of the pocket with an almost absence of the available nutrients essential for the red and orange complex bacteria.

- The bone gain obtained would be related to the use of phosphocalcium derivative bioactive materials which increase bone formation.

Conclusion

Our work demonstrates that an advanced aggressive periodontitis with the presence of terminal lysis could be currently treated whereas about fifteen years ago tooth extraction was the only alternative.

A significant improvement of the depth of the pockets, attachment level, decrease in dental mobility, modification of subgingival bacterial biofilm and bone gain are the results obtained at 4 years.

The success of our therapy would not have been possible without fighting against the bacterial biofilm, or without the full cooperation and consent of our patients.

These diseases constitute a public health problem due to the speed and severity of their evolution with functional consequences and psychosocial repercussions related to the early loss of teeth. This technique has given excellent results in young subjects.

Authors



Prof. Djamila Bouziane: University Hospital Professor (University of Oran/Oran University Hospital) - Head of Department of Periodontology - Head of Department of Dental Surgery - President of the scientific council of the health research topic agency - President of the national dental medicine teaching committee - President of the regional speciality teaching committee (Parodontology) - President of the national speciality teaching committee (Periodontology) - Director of the oral biology laboratory - Member of the scientific council of the Oran university academy - ANDRS expert (National

Agency for the development of health research) in charge of building a national health research program - Founder and President of the Société Algérienne de Médecine Dentaire (SAMD, Algerian Dental Medicine society) - Member of the Société Algérienne d'Odontologie Pédiatrique (SAOP, Algerian Paediatric Odontology Society) - Member of the National Dental Committee - Vice-President of the International Conference of French language Deans - Member of the Académie Française de Chirurgie Dentaire (ANCD, French Academy of Dental Surgery) - Leader of numerous research projects.

Mohamed Bouziane: University Hospital Professor (University of Oran/Oran University Hospital) - Head of Department of Prosthetics.

Leila Khadidja Makrelouf: University Hospital Professor (Periodontology) (University of Oran/Oran University Hospital).

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