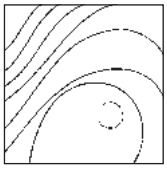
Long-Term Onlay Graft Volume Maintenance in Aggressive Periodontitis Patients



Fernando Verdugo, DDS[1]/Alon Frydman, DDS[2]/Ana Castillo, PhD[3]
José Pontón, PhD[4]/Hessam Nowzari, PhD[5]

Treatment planning in the esthetic zone has classically presented some of the greatest challenges to the practitioner. The purpose of this article is to describe a staged, multidisciplinary approach and follow-up to a case of aggressive periodontitis. Microbial sampling for suspected periodontopathogens was taken before and after treatment. Qualitative polymerase chain reaction analysis was done to detect the presence of cytomegalovirus and Epstein-Barr virus type 1 6 years after active periodontal therapy. A control computed tomography scan taken 5.5 years postaugmentation showed stable bone levels and excellent volume maintenance of the transplanted block graft. (Int J Periodontics Restorative Dent 2011;31:e71–e79.)

[1]Private Practice, Altadena, California; Consultant Periodontist, VA Hospital, Greater Los Angeles Healthcare System, Los Angeles, California, USA; Formerly, Assistant Professor of Clinical Dentistry, Advanced Periodontics, University of Southern California School of Dentistry, Los Angeles, California, USA.
[2]Assistant Professor, Advanced Periodontics, University of Southern California School of Dentistry, Los Angeles, California, USA.
[3]Professor in Microbiology, School of Medicine, University of Granada, Granada, Spain.
[4]Professor in Microbiology, School of Medicine, University of Basque Country, Leioa, Spain.
[5]Professor of Clinical Dentistry and Director, Advanced Periodontics, University of Southern California School of Dentistry, Los Angeles, California, USA.

Correspondence to: Fernando Verdugo, 2028 North Lake Avenue, Altadena, CA, 91001; fax: 626-797-0523; email: fverdugo@perio.org.

Aggressive periodontitis has multiple variations in its origin and presentation. Onset can be found at different life stages with a variable rate of progression. Treating patients with aggressive periodontitis involves multiple tiers of therapy designed to work together to eliminate the disease or, at least, halt the loss of attachment. With the damage done by the disease, a need arises to restore the dentition keeping both function and esthetics in mind.

The diagnosis of aggressive periodontitis is described as a rapid onset and loss of attachment of the periodontal apparatus in an otherwise healthy patient. The localized form generally presents with a circumpubertal onset, affecting first molars and incisors, while atypical patterns also have been described. Other factors, such as microbial sampling results, give additional information on the specific putative bacteria responsible for the progression of the disease. Often, the bacteria involved are strains of *Aggregatibacter actinomycetemcomitans* (formerly, *Actinobacillus actinomycetemcomitans*).[1]

Table 1 **Maxillary probing depths (mm) at baseline, reevaluation, and 5 years**

	Tooth no.*					
	13	**12**	**11**	**21**	**22**	**23**
Baseline						
Facial	**4**,2,**4**	**5**,**5**,**8**	**8**,**12**,**7**	**5**,3,**5**	**4**,3,**4**	**5**,3,**4**
Palatal	**4**,3,**4**	**5**,3,**5**	**6**,**5**,**6**	**5**,3,**5**	**4**,3,**4**	**4**,3,**4**
Reevaluation						
Facial	3,2,3	3,3,3	3,**10**,**4**	3,2,3	3,2,3	3,2,3
Palatal	3,2,3	3,2,3	3,3,3	3,2,3	3,2,3	3,2,3
5 years						
Facial	3,2,3	3,2,3	3,2,3	3,2,3	3,2,3	3,2,3
Palatal	3,2,3	3,2,3	3,2,3	3,2,3	3,2,3	3,2,3

Bold numbers denote bleeding on probing.
*FDI tooth-numbering system.

Recent studies have suggested a coinfection with viruses from the herpesviridae family. Epstein-Barr virus type 1 (EBV-1), cytomegalovirus (HCMV), and other herpesviruses have been found in deep periodontal pockets of patients with aggressive periodontitis.[2–4] Herpesvirus-infected periodontitis lesions seem to harbor a wide variety of periodontopathic bacteria, such as *Porphyromonas gingivalis, Dialister pneumosintes, Prevotella intermedia, Parvimonas micra* (formerly, *Peptostreptococcus micros), Tannerella forsythia*, enteric rods, *Fusobacterium spp*, *Campylobacter rectus, Treponema denticola, Aggregatibacter actinomycetemcomitans*, and even *Candida albicans*.[3–5]

Periodontal herpesvirus-active infections may impair local defenses, favoring the subgingival overgrowth of these periodontopathic bacteria. The generalized form affects people under 30 years of age, although there is no age limit. Patients with this form present with generalized interproximal attachment loss affecting at least three permanent teeth other than the first molars and incisors. Moreover, the disease is associated with the presence of *A actinomycetemcomitans* and *P gingivalis*.[6]

Treatment to restore function and esthetics often requires bone augmentation procedures as well as implants. With these treatments, long-term predictability and maintenance become an important factor. There seems to be a higher risk for bacterial recolonization and bone loss than in healthy and even nonaggressive periodontitis patients.[7–10]

The following case shows a successful multidisciplinary approach in treating a patient diagnosed with localized aggressive periodontitis in whom the combination of different specialties (periodontics, endodontics, orthodontics, and prosthodontics) was required. Moreover, bone augmentation and implants were needed to restore the maxillary anterior ridge collapse and dentition.

Table 2 **Mandibular probing depths (mm) at baseline, reevaluation, and 5 years**

	Tooth no.*					
	43	**42**	**41**	**31**	**32**	**33**
Baseline						
Facial	**5**,3,**5**	**6**,**4**,**6**	**7**,**7**,**8**	**6**,**6**,**5**	**5**,**5**,**4**	**4**,3,**4**
Lingual	**4**,3,**4**	**6**,**5**,**6**	**7**,**5**,**7**	**6**,**5**,**6**	**5**,**5**,**4**	**4**,**4**,**4**
Reevaluation						
Facial	**4**,3,3	3,2,3	3,2,3	3,2,3	3,2,3	3,2,3
Lingual	3,2,3	3,2,3	3,2,3	3,2,3	3,2,3	3,2,3
5 years						
Facial	3,2,3	3,2,3	3,2,3	3,2,3	3,2,3	3,2,3
Lingual	3,2,3	3,2,3	3,2,3	3,2,3	3,2,3	3,2,3

Bold numbers denote bleeding on probing.
*FDI tooth-numbering system.

Clinical case description and results

A nonsmoking, 38-year-old man presented to the Advanced Periodontology Program at the University of Southern California, School of Dentistry, Los Angeles, California, in 2000 with the chief complaint of swelling and mobility of one of his maxillary anterior teeth. The patient's medical history was unremarkable. Plaque control was fair to good (Plaque Index [PI], 35%; 10 of 28 teeth presented mild presence of plaque). Clinical examination revealed deep pockets and suppuration from the buccal aspect of the right central incisor, with negative percussion, Class II mobility, and positive vitality for all anterior teeth. No restorations or history of trauma were evident. The patient had severe attachment loss on the mesial aspect of the maxillary right lateral incisor with 8-mm pocket depths (Table 1). The mandibular anterior teeth presented pocket depths ranging from 4 to 8 mm (Table 2). Localized moderate attachment loss was also present interproximally at the mandibular and maxillary right first and second molars. Subsequent microbial analysis aided in the diagnosis of localized aggressive periodontitis (Table 3).

The initial therapy consisted of oral hygiene instructions, scaling and root planing, and subgingival irrigation with povidone-iodine 10% (Betadine, Purdue Frederick) for 5 minutes.[11] Antimicrobial therapy was instituted thereafter (amoxicillin 500 mg + metronidazole 250 mg three times a day for 8 days).

Reevaluation was carried out at 4 weeks, revealing reduced inflammation and persistent deep pockets (10 mm for the maxillary right central incisor, Tables 1 and 2); there was no suppuration, but bleeding on probing and significant gingival recession was noted for the maxillary right incisors (Figs 1 to 3). All mandibular anterior teeth presented significant pocket depth reductions, with probings ranging from 3 to 4 mm (Fig 1). Localized 6-mm pockets at interproximal areas of the maxillary right first and second

Table 3 Microbiota (%) present at baseline and 5 years

	Baseline	5 years
A actinomycetemcomitans	0.0	0.0
P gingivalis	3.0	0.0
P intermedia	14.7	0.0
T forsythia	6.9	0.0
Campylobacter spp	2.9	1.1
Eubacterium spp	8.8	1.4
Fusobacterium spp	3.9	2.1
P micra	43.1	0.0
Enteric gram-negative rods	0.0	0.0
Yeast	0.0	0.0
D pneumosintes	3.0	0.0

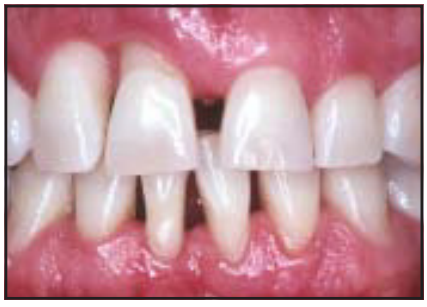


Fig 1 *Frontal view after initial therapy.*

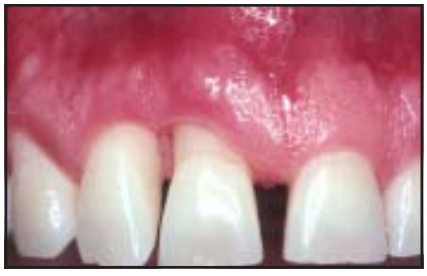


Fig 2 *Significant papillary loss was noted after nonsurgical therapy.*

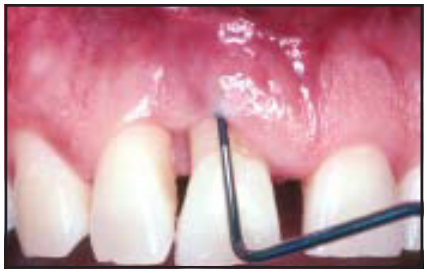


Fig 3 *At reevaluation, a 10-mm pocket depth was still present at the maxillary right central incisor.*

molars, the distal aspect of the maxillary right second molar, and the distal aspect of the mandibular right second molar were still present. Plaque control at this point was deemed very good (PI, 14%). Osseous surgery was performed for posterior areas on the maxillary right and mandibular left sextants. Open flap debridement was done for the maxillary right incisor area, revealing no visible calculus and a facial 12-mm dehiscence on the central incisor and a 7-mm dehiscence on the lateral incisor.

To correct the gingival discrepancy and address the patient's esthetic concerns, orthodontic extrusion[12,13] of the maxillary right incisors was performed, with the decision to extract the central incisor at the end of the extrusion (Fig 4). Before the initiation of orthodontic treatment, root canal therapy was done for the central incisor. The duration of orthodontic treatment lasted 6 months (8 weeks of active extrusion and 4 months of stabilization). Approximately 2 mm of enamoplasty was accomplished for the incisal edge of the lateral incisor to accommodate for the 3-mm extrusion. Alveolar

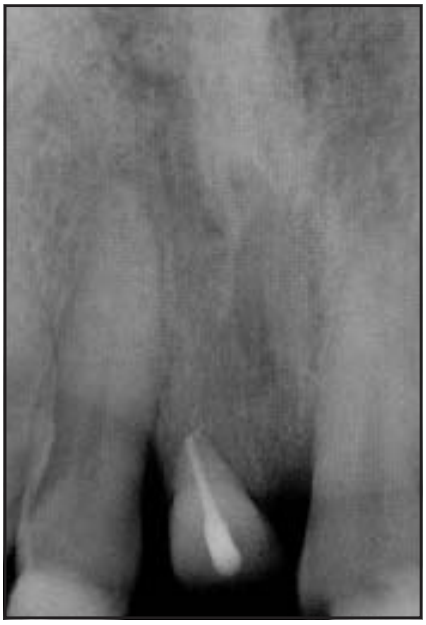
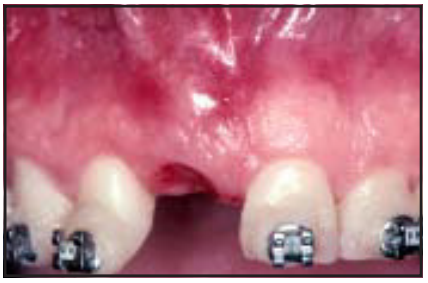


Fig 4 (left) *Periapical radiograph and* (below) *clinical photograph at the completion of orthodontic extrusion.*



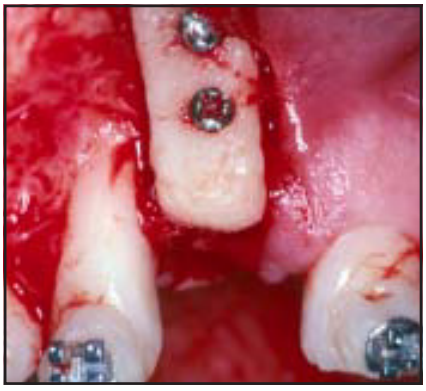


Fig 5 *Autogenous symphysis block for ridge augmentation at the maxillary right central incisor site.*

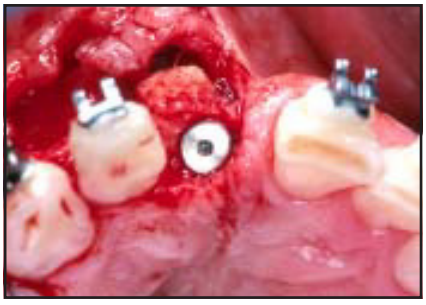


Fig 6 *Occlusal view of implant placement 4 months after ridge augmentation.*

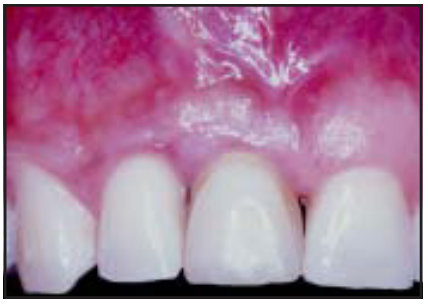
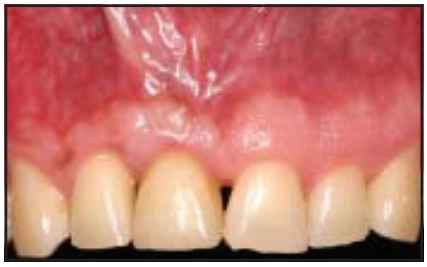


Fig 7 *Frontal view of interproximal area of the maxillary right incisors 4 months after delivery of the definitive screw-retained restoration at the central incisor site.*

bone mapping showed a deficient buccolingual width at the central incisor. Therefore, an autogenous block graft from the symphysis was performed (Fig 5). A single implant was placed 4 months later (Fig 6).

At second-stage implant uncovery, soft tissue augmentation for the facial aspect of the maxillary right incisors was accomplished using a pedicle of connective tissue from the palate. The implant was loaded with a screw-retained provisional restoration at 5 months, and a definitive restoration was delivered 3 months thereafter (Fig 7). A nightguard was delivered to prevent further incisal wear of the anterior teeth.

The patient was placed on a regular supportive periodontal therapy program every 3 months for the first 2 years and every 6 months thereafter. He was monitored clinically (Fig 8) and radiographically (Figs 9 to 12). Periapical radiographs and a control cone beam computed tomography scan (NewTom, QR sr 1) were taken 5.5 years from the time of ridge augmentation to evaluate the long-term outcome (Figs 9 and 10).[14] Microbial sampling was repeated at 5 years using paper



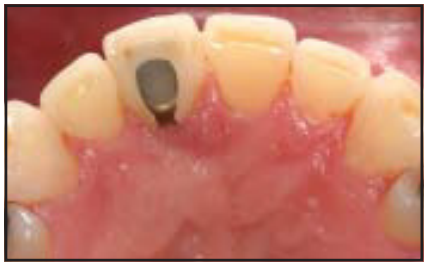


Fig 8 (left) *Frontal view and (right) palatal view of the maxillary anterior teeth at 5.5 years.*

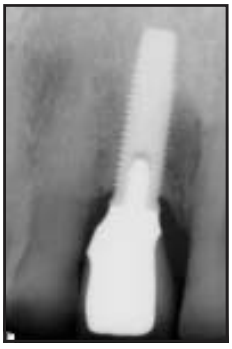


Fig 9 (left) *Periapical radiograph 5 years and 2 months after implant placement.*

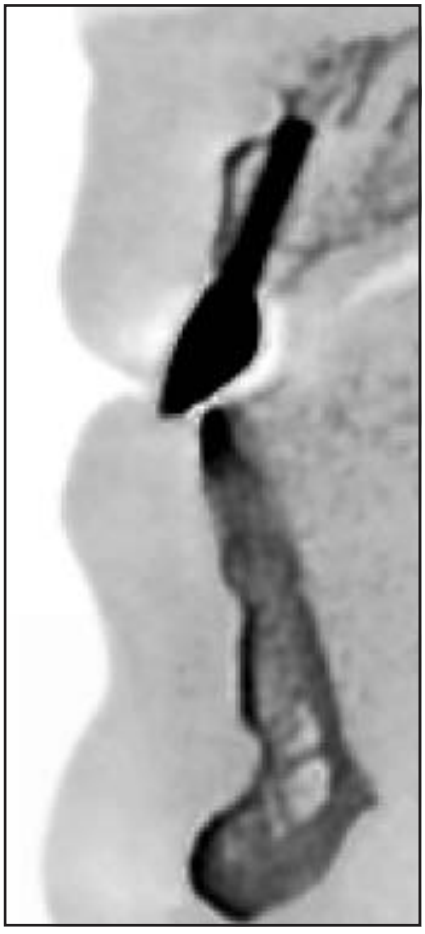


Fig 10 (right) *Cross-sectional negative image of the implant at 5.5 years.*

points (Table 3). An anaerobic culture showed no significant presence of common periodontopathogens at teeth or implant sites. Qualitative polymerase chain reaction also failed to detect HCMV or EBV-1 at the tested sites. The patient's periodontal condition was stable throughout follow-up.

Discussion

Treatment planning in the esthetic zone has classically presented some of the biggest challenges to the practitioner. When compounding variables are present, the ability to deliver an esthetic result becomes more difficult.

This case required a multiphase, multispecialty approach to achieve the most esthetic result. The first stage involved the treatment of the disease process to stabilize the patient. The second stage of treatment involved the augmentation of a scaffold for the planned restorations. This scaffold

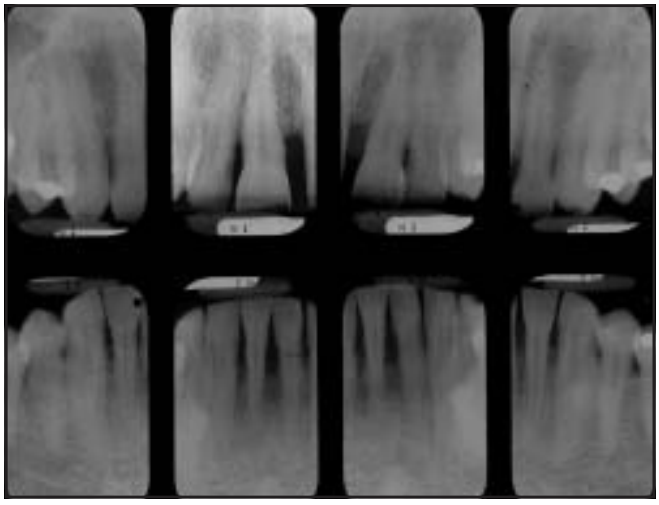


Fig 11 *Preoperative radiographs of the anterior teeth.*

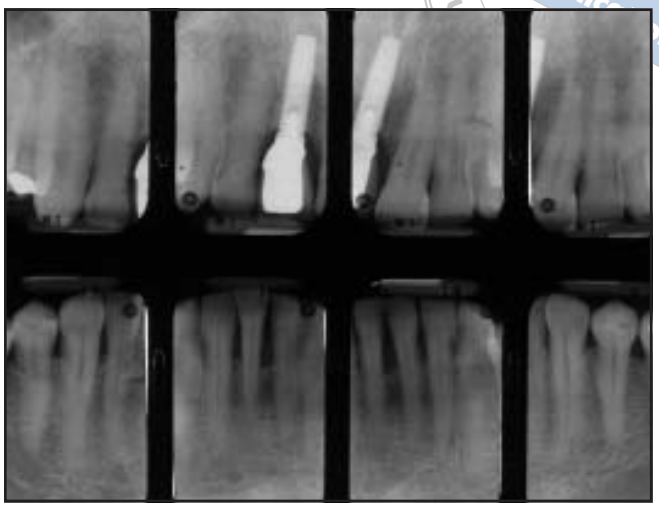


Fig 12 *Postoperative radiographs of the anterior teeth at 5 years.*

would have to support an endosseous dental implant at the maxillary right central incisor site. The combination of periodontics, endodontics, orthodontics, and prosthodontics was necessary to achieve an acceptable esthetic outcome.

For this particular patient, the periodontal destruction requiring a multispecialty approach was localized to the maxillary anterior sextant (see Fig 1). The anticipation of ridge collapse postextraction further complicated esthetic demands and anatomical possibilities. The use of the remaining periodontal ligament surrounding the hopeless tooth (right central incisor) allowed for augmentation of the ridge vertically (Fig 5), correcting the infrabony defects and creating a new papilla (Figs 6 and 7) through orthodontic extrusion.[12,13] Despite the Class II mobility and severe attachment loss (see Fig 2), it was possible to gain vertical dimension of bone and build the papilla interproximally between the right incisors (Figs 6 and 7). The orthodontically augmented site (Fig 5) served as a scaffold for the onlay block graft to further augment the buccolingual width, allowing subsequent implant placement (Fig 9)

In a recent long-term retrospective study[8] performed on 1,060 patients who received 5,787 implants, aggressive periodontitis was identified as a risk factor for implant failure. Most of the patients with implant failure (70%) presented chronic or aggressive periodontitis. Mengel et al[9] followed two groups of 5 patients each who received dental implants: periodontally healthy and generalized aggressive periodontitis (GAgP). Microbiologically, GAgP subjects had fewer cocci and more motile rods and filaments at teeth and implants than periodontally healthy subjects. Implant survival rates were 100% in periodontally healthy subjects and 83.33% in GAgP subjects. In a previous study, Mengel and Flores-de-Jacoby[15] performed guided bone regeneration in a group of treated GAgP patients who received implants 6 to 8 months later. Patients were followed for a period of 3 years and presented increased attachment and bone loss at implants placed in the regenerated bone. They concluded that the possibility of continuous attachment loss and bone loss occurring at teeth and implants in regenerated bone cannot be ruled out in patients treated for aggressive periodontitis.

The remaining teeth of partially edentulous patients with a history of chronic or aggressive periodontitis may play a role in the bacterial composition of the peri-implant sulcus. Some authors[16] have found black pigmented gram-negative bacteria to be elevated in the peri-implant sulcus of partially, but not completely, edentulous patients. Others have shown that periodontal pathogens (*P gingivalis* and *T forsythia*) can colonize the peri-implant sulcus within 4 weeks after loading.[17]

Successful elimination of periodontal pathogens from pockets and oral mucosal surfaces has been reported with the administration of systemic antimicrobials (amoxicillin + metronidazole).[18,19] However, persistence of certain microorganisms (*A actinomycetemcomitans, T forsythia, P micros, P intermedia*) in peri-implantitis patients with full-mouth implant rehabilitations has been documented previously. Resistance to repeated antimicrobial therapy failed to stop the disease progression. The authors concluded that preoperative infection control is paramount, and the remaining teeth affected by periodontitis can pose a serious risk factor for peri-implantitis.[20]

HCMV was also positively associated with subgingival *D pneumosintes,* which was associated with disease-active periodontitis sites and alveolar bone loss.[21] Both HCMV and *D pneumosintes* affected bone loss independently. Moreover, *P gingivalis*, EBV-1, and Herpes simplex virus were significantly increased in periodontitis-active sites when compared to stable sites.[21]

Patients with a history of aggressive periodontitis may be at a greater risk for complications at some point after treatment completion. The present outcome suggests that an efficient antimicrobial therapy and maintenance program may have helped suppress existing aggressive periodontopathogens and keep a stable situation long term.

It may be plausible to speculate that different combinations or critical concentrations of specific pathogens could elicit different responses in susceptible individuals. Moreover, specific periodontal pathogens in a microbial biofilm could escape from the antimicrobial effects of systemic therapy. Prospective clinical trials are much needed to elucidate the pathogenesis and disease progression in peri-implantitis patients.

This case showed successful antimicrobial therapy in eliminating microorganisms such as *P gingivalis*, *T forsythia*, *P micra*, *P intermedia*, and *D pneumosintes* associated with localized aggressive periodontitis and alveolar bone loss. Furthermore, the absence of subgingival viruses (HCMV and EBV-1) and the presence of lamina dura (Fig 12) 5.5 years later emphasizes the stability of the outcome.[21,22]

The clinical applications of CT scanning are multiple, aiding in diagnosis and treatment planning prior to implant placement as well as assessment of regenerative therapy outcomes.[14,23–25] The CT scan evaluation showed an almost intact buccolingual width of the augmented site with a well-corticalized buccal wall (Fig 10). The volume maintenance of the block graft at 5.5 years together with the positive laboratory results (see Table 3) reinforce the stable outcome reported previously.[26]

More surveys and case reports are needed to elucidate the best method of treatment, monitoring, and maintenance in patients with aggressive periodontitis. Additionally, determination of host susceptibility to infectious complications within this patient population should be investigated and followed over the long term.

Conclusions

Autogenous osseous transplants can predictably reconstruct function and esthetics of anterior ridge defects in aggressive periodontitis patients and appear to maintain long-term stable bone volume around endosseous implants. A multidisciplinary approach may enhance the esthetic results and seems to be the ideal choice for a successful therapy outcome.

Acknowledgments

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