

**Case Report**

# **Rehabilitation of Lower Anterior Teeth with Implant Therapy in Localized Aggressive Periodontitis**

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## **Abstract**

Periodontitis is the most common, destructive, inflammatory diseases of supporting tissues of the teeth in humans, with profound effects on general health. Aggressive periodontitis (AgP) comprises a rare, rapidly progressing form of periodontitis, characterized by severe destruction of periodontal attachment apparatus and tooth loss at an early age. Considering the psychological problems which these patients have during the early stages of their life, the chief aim of the treatment is to achieve functional, esthetic, and phonetic rehabilitation. Dental implant is a widely used treatment option that provides functional and esthetic resolution. Implant placement in patients with a history of AP might be considered a viable option to restore oral function; however, the risk for implant failure is significantly higher. This report presents rehabilitation of lower anterior teeth with implant therapy in a 24-year-old systemically healthy male with a history of localized AP, who had previously received complete periodontal therapy for periodontal stabilization.

**Key words:** Aggressive periodontitis, dental implants, periodontal diseases, periodontitis.

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## **Introduction**

**W**ith every periodontal entity having a distinct progressive pattern and the different bacteria associated, it becomes absolutely necessary to treat and control periodontal disease, regardless of its progression pattern and subtype before implant therapy is initiated to improve implant longevity, in the pursuit of excellence in implant dentistry. A higher incidence of peri-implantitis and a lower implant survival rate being reported in patients susceptible to periodontitis,<sup>1,2</sup> it is important to mention that the pre-existing ecologic conditions of the oral cavity influence biofilm formation on implants. Success in implant dentistry relies on the initial osseointegration and long-term stability.<sup>3</sup>

Patient systemic factors and susceptibility to periodontal diseases, implant macro and micro design, and periodontal pathogenic bacteria, among others, have all been shown to play a role in achieving long-term implant stability.<sup>4</sup>

## **Case report**

A 24-year-old healthy male was a known case of localized aggressive periodontitis (Figures 1 and 2), who had received periodontal therapy and extractions with severely drifted lower central incisors 4 months previously. He received scaling, root planing and soft tissue curettage in conjunction with systemic antibiotics. His medical history was not significant and he had no parafunctional habits.



**Figure 1.** Preoperative (before periodontal therapy for treatment of aggressive periodontitis) clinical photograph, front view.



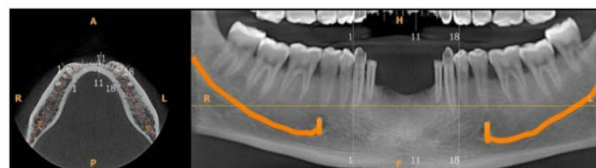
**Figure 2.** Preoperative (before periodontal therapy for treatment of aggressive periodontitis) clinical photograph, lingual view.

Intraoral examination revealed anterior open bite along with pathologic migration of maxillary and mandibular anterior teeth (Figure 3). Maxillary arch showed largely spaced, flared anterior teeth, whereas in mandibular arch a four-unit bridge spacing in anterior region was present with extracted central incisors. A CBCT examination of the mandible was carried out to assess the existing bone morphology (Figure 4). After informed consent, prosthetic rehabilitation was planned with a four-unit bridge on two implants for the lower anterior teeth.

Surgery was performed under local anesthesia, consisting of bilateral mental nerve block. The patient was given antibiotic prophylaxis for 48 hours (amoxicillin 500 mg given orally 1 hour preoperatively and every 6 hours postoperatively). Following midcrestal incision, full-thickness flaps were reflected and implant sites were prepared bilaterally (Figure 5). Two titanium plasma-sprayed coated implants, 13 mm in length and 3.3 mm in



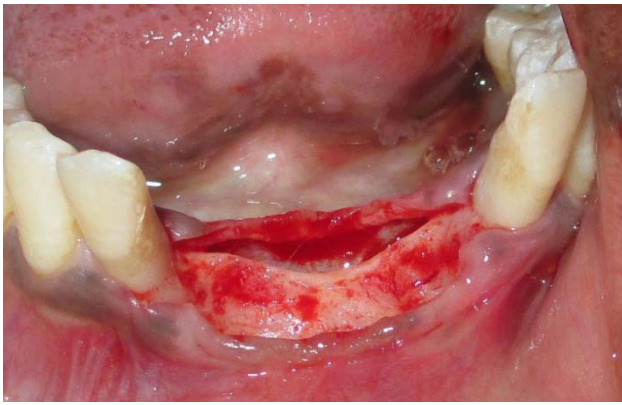
**Figure 3.** Postoperative (after periodontal therapy for treatment of aggressive periodontitis) clinical photograph, front view.



**Figure 4.** CBCT examination of the mandible to assess existing bone morphology.

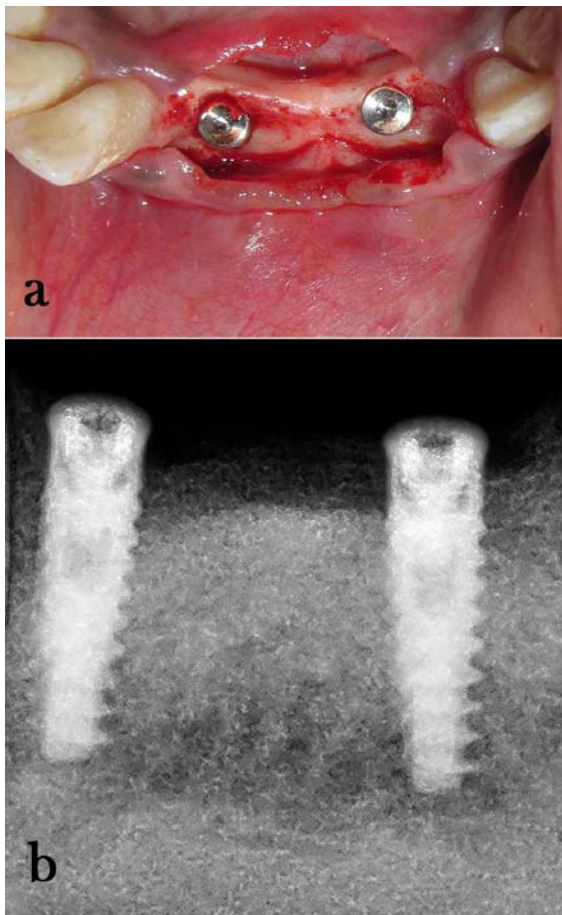
diameter, were placed in the anterior region of the mandible (Figures 6.a and 6.b). Flaps were approximated using 4-0 absorbable (vicryl) interrupted sutures (Figure 7). Analgesic (ibuprofen, 400-mg tablets every 4–6 h) along with antimicrobial rinse (0.2% chlorhexidine gluconate twice a day for 4 weeks) were prescribed and the patient was recalled after 7 days for follow-up.

At 3-month postoperative interval, the implant sites were reopened with crestal incisions and gingiva formers were screwed. Healing was uneventful with both implants being well osseointegrated and phase 2 surgical uncovering was accomplished. The

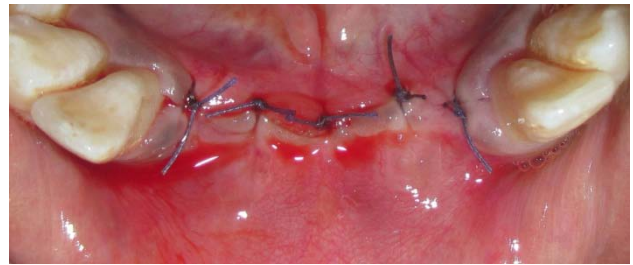


**Figure 5.** After full-thickness flap reflection at proposed implant sites in the anterior region of the mandible.

gingiva formers were replaced with abutments 3 weeks later (Figure 8) and a four-unit porcelain-fused-to-metal (PFM) prosthesis was inserted (Figures 9.a and 9.b). The patient was kept on regular maintenance and follow-up every 4 months



**Figure 6.a:** Two implants, 13 mm in length and 3.3 mm in diameter, placed in the anterior region of the mandible. **Figure 6.b.** IOPA radiograph showing well aligned implants in the anterior region of the mandible, immediately after insertion.



**Figure 7.** Flaps were approximated using 4-0 absorbable (vicryl) interrupted sutures.



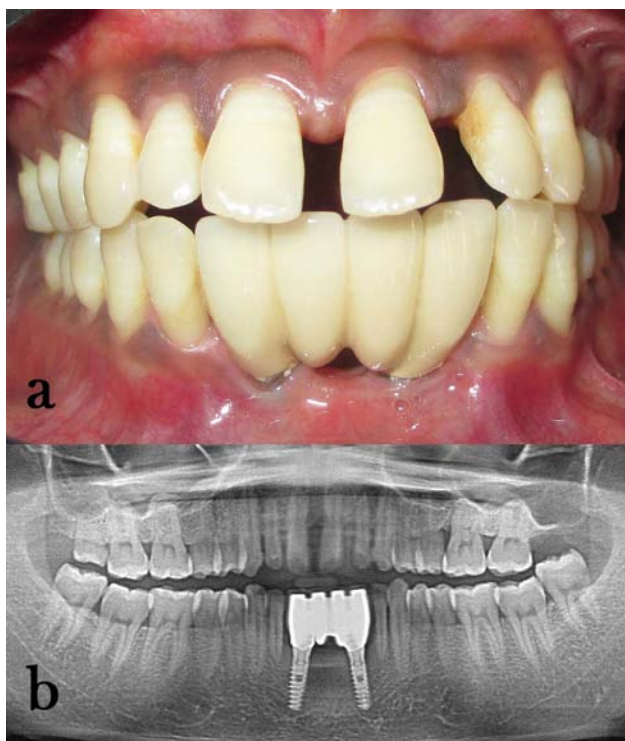
**Figure 8.** Two abutments placed 3 weeks after gingiva former placement.

(Figure 10). Both implants were functioning and immobile when tested clinically, with no evidence of peri-implant radiolucency in IOPA radiograph and CBCT at 1-year follow-up after loading (Figures 11 and 12).

**Discussion**

Baer (1971)<sup>5</sup> defined aggressive periodontitis as “a disease of the periodontium occurring in an otherwise healthy adolescent, which is characterized by a rapid loss of alveolar bone around more than one tooth of the permanent dentition.” According to American Academy of Periodontology (AAP 1999)<sup>6</sup> Classification, the term “aggressive periodontitis” was adopted as a new name for this unique disease classification, replacing the term “early-onset periodontitis” and classified the disease into localized and generalized forms:<sup>7</sup>

- Localized aggressive periodontitis (LAP):
  - ✓ Circumpubertal onset.
  - ✓ Localized first molar/incisor presentation with interproximal attachment loss on at least two permanent teeth (one of which is a first molar) and involving no more than two teeth other than first molars and incisors.



**Figure 9.a:** Four-unit porcelain-fused-to-metal (PFM) prosthesis inserted. **Figure 9.b:** OPG showing well-connected prosthesis.

- ✓ Robust serum antibody response to infecting agents.
- Generalized aggressive periodontitis (GAP):
- ✓ Usually affecting persons under 30 years of age, but patients may be older.
- ✓ Generalized interproximal attachment loss



**Figure 11.** IOPA radiograph at 1-year follow-up after loading.



**Figure 10.** Clinical photograph showing patient's normal smile before and after prosthetic insertion in the anterior region of the mandible.

affecting at least three permanent teeth other than first molars and incisors.

- ✓ Pronounced episodic nature of the destruction of attachment and alveolar bone.
- ✓ Poor serum antibody response to infecting agents.

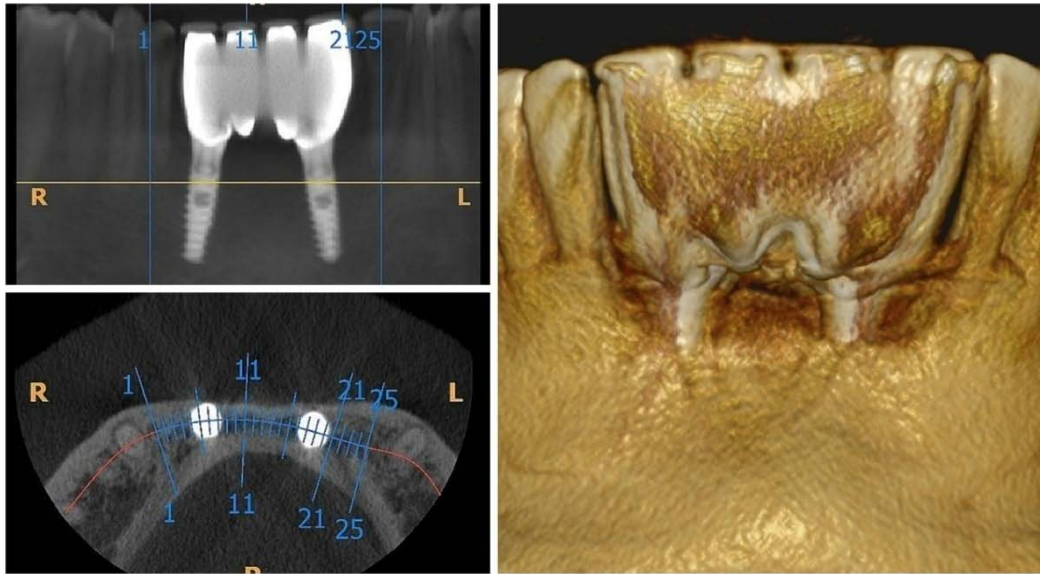
Three major characteristics were used to define the aggressive disease:<sup>7,8</sup>

- 1) Clinically healthy with the exception of periodontitis.
- 2) Rapid attachment loss (AL) and bone breakdown.
- 3) Familial aggregation.

Other characteristics can also be used in the diagnosis of the disease:<sup>7-9</sup>

- 1) Amounts of microbial deposits inconsistent with the severity of periodontal tissue breakdown.
- 2) Elevated proportions of *Aggregatibacter actinomycetemcomitans* and *Porphyromonas gingivalis*.
- 3) Phagocyte abnormalities.
- 4) Hyper-responsive macrophage phenotype, including elevated levels of prostaglandin E2 and interleukin (IL)-1b.
- 5) Self-arresting progression of AL and bone loss.

Polymorphisms in genes regulating the expression of IL-1, IL-6, IL-10, tumor necrosis factor, E-selectins, Fc-g receptor, cluster of differentiation 14, toll-like receptors, caspase recruitment domain 15, vitamin D-receptor, lactoferrin, caldesmon, heat shock



**Figure 12. Radiographic examination; CBCT at 1-year follow-up after loading.**

protein 70, and Stac protein 23 and major histocompatibility complexes A9 and B1524 were associated with AP. As a consequence of these polymorphisms, the inflammatory profile is altered, including, but not limited to, polymorphonuclear neutrophil (PMN) transendothelial migration and signaling functions,<sup>10</sup> reduced chemotactic response, and depression in neutrophil phagocytosis and superoxide production.<sup>11</sup>

Apse et al<sup>12</sup> stated that the peri-implant sulcus behaves similar to the periodontal sulcus, and therefore, an inflammatory process similar to periodontitis occurs around implants, i.e. peri-implantitis. Mombelli et al<sup>13</sup> identified the same pathogens in peri-implant lesions as the ones that were present 6 months before in natural dentition. Implant placement in patients with a history of AP might be considered a viable option to restore oral function with survival outcomes similar to those found in both patients with chronic periodontitis and healthy patients. However, the risk ratio for implant failure in patients with AP is significantly higher when compared with healthy patients and those with chronic periodontitis.<sup>14</sup>

In AP patients often unmodifiable factors would potentially play a role in implant success. These factors include:<sup>10,11,15</sup>

- 1) Genetic polymorphisms,
- 2) alterations of the immune system (phagocyte abnormalities and hyper-responsive macrophage phenotype, altered polymorphonuclear neutrophil transendothelial migration and signaling functions, reduced chemotactic response, and depression in phagocytosis and superoxide production),

- 3) depression, stress, and loneliness,
- 4) oral hygiene,
- 5) tobacco consumption.

For an implant to be deemed successful, the first-year mean of bone loss of about 0.9–1.6 mm has been reported to be acceptable. During the subsequent years, the mean bone loss (MBL) has been reported to decrease to 0.05–0.13 mm annually.<sup>16,17</sup> Mengel et al<sup>18</sup> showed that patients with GAP exhibited MBL of 2.07 mm during the first year after implant placement.

### Conclusion

Both in AP and peri-implantitis processes, a comprehensive implant maintenance program to identify peri-implant bone loss early is highly encouraged, specifically in patients with a history of aggressive periodontal disease due to unmodifiable conditions that might play a dominant role. A good case selection, meticulous treatment plan, and good patient compliance with long-term regular follow-up confers successful placement of implants in patients with AP.

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