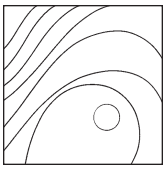
A New Approach for the Treatment of Lateral Periodontal Cysts with an 810-nm Diode Laser



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The aim of this study was to test whether the combination of diode laser therapy and surgical treatment for a lateral periodontal cyst (LPC) would result in greater clinical improvement compared with surgery alone. A total of 18 patients with LPCs were assessed for eligibility for this study. At baseline, each patient was randomly allocated to one of two regimens: diode laser plus surgery (test group) or traditional surgical treatment alone (control group). Healing parameters were assessed at 7 to 21 days to monitor short-term complications, and periodontal parameters were assessed at 3, 6, and 12 months to evaluate long-term healing. The test group demonstrated highly significant differences in both the short-term and long-term parameters compared with the control group. This study showed that diode laser treatment results in a shorter wound-healing period and could be considered valuable for the surgical treatment of LPCs. Int J Periodontics Restorative Dent 2017;37:e120–e129. doi: 10.11607/prd.2981

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A lateral periodontal cyst (LPC) is a rare but well-recognized type of epithelial developmental odontogenic cyst and has a prevalence of 1.5% among cysts of the jaw.[1] LPCs are defined as radiolucent lesions that grow along the lateral surface of an erupted vital tooth in which an inflammatory etiology has been excluded based on clinical and histologic features.[2] It has been hypothesized that LPCs arise from the reduced enamel epithelium or the epithelial rests of Malassez in the periodontal ligament.[3]

The conventional treatment approach for LPCs is surgical excision using a surgical blade and scalpel. This approach has been used for many years due to its ease, and studies have demonstrated its success in treating LPCs.[2] However, pain, bacterial infections, and hemostatic problems can negatively influence the surgical wound, particularly in patients with hemorrhagic disorders.

Laser therapy can aid in repairing the mucosa, controlling pain, and accelerating wound healing. Laser devices possess bactericidal and detoxifying effects. Through low mechanical stress on the root surfaces,[4] they have been used to successfully remove the granulated tissue and plaque present in periodontal pockets.[5,6]

Several studies have indicated that a wavelength of 810 nm is the

most effective for the treatment of oral soft tissue. The most commonly used devices in oral surgery include carbon dioxide (CO2), neodymium-doped yttrium, aluminum and garnet (Nd:YAG), and erbium-doped YAG (Er:YAG) lasers.[7,8] However, diode lasers have only recently been studied.[9]

Diode lasers are produced by the stimulation of gallium, arsenide, and phosphate with or without aluminum or indium at a wavelength of 800 to 1,064 nm.[10] The interactions of the different wavelengths on targets can have photothermal, photochemical, and photomechanical effects.[11] Diode lasers are primarily soft-tissue instruments that can be used for procedures such as crown lengthening, gingivectomies, gingivoplasties, and frenectomies.[12]

Diode lasers are considered effective and suitable for treating a variety of inflammatory and infectious oral conditions due to its high absorption by water and hemoglobin. They have shown promising results in treating oral lesions and performing periodontal surgery and tissue alterations. They might also improve the rate of tissue healing and enhance regeneration and repair.[13]

The biologically beneficial effects induced by diode lasers indicate that they are a good option for the treatment of oral diseases. Their use during LPC surgery could be a therapeutically useful and conservative approach.

Therefore, the aim of this study was to test whether the use of a diode laser as an adjunct to traditional surgery for LPCs would result in greater clinical improvement than would surgical treatment alone. The null hypothesis was that there would be no differences in the results in terms of anti-inflammatory effects and clinical periodontal parameters between traditional surgery alone and traditional surgery plus laser treatment after a 12-month follow-up.

Table 1 Characteristics of the Study Groups at Baseline

Parameters	Control group (n = 9)	Test group (n = 9)	*P*
Age (y)	47.5 ± 5.5	45.9 ± 5.8	.430
Sex			
Men (n [%])	5 (55.5)	3 (33.3)	
Women (n [%])	4 (45.5)	6 (66.7)	.422
PD (mm)	3.2 ± 0.5	3.3 ± 0.2	.148
CAL (mm)	3.7 ± 0.8	3.9 ± 0.3	.357
GR (mm)	0.5 ± 0.2	0.6 ± 0.4	.378
PI	0.9 ± 0.5	0.8 ± 0.3	.498
BOP (%)	11.1 ± 1.5	12.6 ± 1.9	.415
FMPI	1.1 ± 0.6	1.0 ± 0.7	.667
FMBOP (%)	57.2 ± 1.6	58.2 ± 1.3	.062
Maximum mouth opening (mm)	39.6 ± 1.2	40.1 ± 1.1	.229
Segment distribution of cyst			
Anterior (maxilla/mandible)	3 (2/1)	1 (1/0)	.022
Premolar (maxilla/mandible)	5 (3/2)	5 (2/3)	–
Posterior (maxilla/mandible)	1 (1/0)	3 (2/1)	.316
Mean size of cyst (mm)	11.1	12.3	.746
0.3–1 mm (n)	5	5	–
1.1–1.8 mm (n)	3	2	.422
1.9–2.7 mm (n)	1	2	.422

Mean ± SD.
PD = probing depth; CAL = clinical attachment level; GR = gingival recession; PI = Plaque Index; BOP = bleeding on probing; FMPI = full-mouth PI; FMBOP = full-mouth BOP.

Materials and methods

Subjects

Of the total number of patients with cysts who were clinically diagnosed with LPC, 18 were assessed for eligibility between July 2001 and February 2015. Their last follow-ups occurred in July 2015 at the Department of Biomedical, Odontostomatological Sciences and of Morphological and Functional Images, University of Messina, Italy (Table 1).

The inclusion criteria were as follows: *(1)* the presence of at least 10 teeth with a probing depth (PD) and clinical attachment level (CAL) between 2 and 4 mm and with the other sides unaffected by LPCs; *(2)* a minimum of five natural teeth in each studied quadrant; and *(3)* a plaque

Table 2 Clinical Parameters of the Study Groups at Baseline and at 3, 6, and 12 Months

	PD (mm)		CAL (mm)		BOP		GR (mm)	
Follow-ups	Control (n = 9)	Test (n = 9)	Control (n = 9)	Test (n = 9)	Control (n = 9)	Test (n = 9)	Control (n = 9)	Test (n = 9)
Baseline	3.2 ± 0.5	3.3 ± 0.2	3.7 ± 0.8	3.9 ± 0.3	11.1 ± 1.5	12.6 ± 1.9	0.5 ± 0.2	0.6 ± 0.4
3 mo	3.4 ± 0.2	3.2 ± 0.3	3.9 ± 0.3	3.6 ± 0.2	12.8 ± 1.3	10.2 ± 0.2*	0.5 ± 0.1	0.4 ± 0.3
6 mo	3.2 ± 0.3	2.7 ± 0.4*	3.6 ± 0.2	3.2 ± 0.4	15.6 ± 0.2*	14.4 ± 1.2**	0.4 ± 0.3	0.5 ± 0.2
12 mo	3.3 ± 0.2	2.1 ± 0.3**	3.9 ± 0.1	2.5 ± 0.2*	16.7 ± 1.5	11.8 ± 1.6**	0.6 ± 0.2	0.4 ± 0.2

*$P < .05$; **$P < .01$.
Mean ± SD.
PD = probing depth; CAL = clinical attachment level; BOP = bleeding on probing; GR = gingival recession.

Table 3 Clinical Parameters Specific to the LPC Sites in the Study Groups at Baseline and at 3, 6, and 12 Months

	PD (mm)		CAL (mm)		BOP		GR (mm)	
Follow-ups	Control (n = 9)	Test (n = 9)	Control (n = 9)	Test (n = 9)	Control (n = 9)	Test (n = 9)	Control (n = 9)	Test (n = 9)
Baseline	4.1 ± 0.6	3.8 ± 0.2	4.3 ± 0.7	4.0 ± 0.4	11.8 ± 1.4	13.1 ± 1.7	0.2 ± 0.2	0.2 ± 0.4
3 mo	3.7 ± 0.3	3.9 ± 0.3	3.9 ± 0.5	4.9 ± 0.3	12.5 ± 1.2	9.9 ± 0.3*	0.2 ± 0.2	0.1 ± 0.3
6 mo	3.4 ± 0.2	2.9 ± 0.5*	3.4 ± 0.1	3.0 ± 0.3	15.1 ± 0.3*	13.9 ± 1.5**	0.3 ± 0.2	0.1 ± 0.3
12 mo	3.7 ± 0.3	2.5 ± 0.3*	3.8 ± 0.2	2.7 ± 0.3*	16.5 ± 1.3	11.6 ± 1.4**	0.1 ± 0.3	0.2 ± 0.1

*$P < .05$; **$P < .01$.
Mean ± SD.
PD = probing depth; CAL = clinical attachment level; BOP = bleeding on probing; GR = gingival recession.

index < 1. The exclusion criteria were as follows: *(1)* a history of systemic diseases (diabetes mellitus, cancer, HIV, metabolic or endocrine diseases); *(2)* pregnancy or lactation; *(3)* chronic high-dose steroid use; *(4)* previous or current radiation or immunosuppressive therapy; *(5)* a heavy smoking habit (> 5 cigarettes/day); *(6)* orthodontic treatment; *(7)* class II or III tooth mobility; *(8)* antibiotic medication use during the 2 months preceding the study; *(9)* extensive caries lesions near the LPC site; and *(10)* heavy contamination by spirochetes or fungal pathogens on the tongue and oral mucosa that were clinically evaluated by visual inspection. All clinical parameters were measured in all quadrants of the mouth. All patients provided written informed consent, and approval from the human subjects ethics board was obtained by the university's institutional ethics committee in accordance with the terms of the World Medical Association's 1975 Declaration of Helsinki, as revised in 2000.

A baseline examination was performed by a calibrated examiner blinded to the treatment protocol who obtained a complete medical history and standard clinical periodontal parameters from each patient. This process included recording the probing depth (PD), clinical attachment loss (CAL), bleeding on probing (BOP), and gingival recession (GR), which were the primary parameters used (Tables 2 and 3). Measurements were obtained at six sites per tooth. BOP was assessed during the PD assay by evaluating the presence or absence of bleeding for more than 30 seconds after challenging the pocket with a periodontal probe (UNC 15, Hu-Friedy).

After admission to the study, the patients were given oral hygiene instructions with appropriate motivation and underwent full-mouth supragingival prophylaxis by ultrasound and/or hand instrumentation.

Intraexaminer calibration and reproducibility were guaranteed during the calibration sessions at baseline by obtaining duplicate

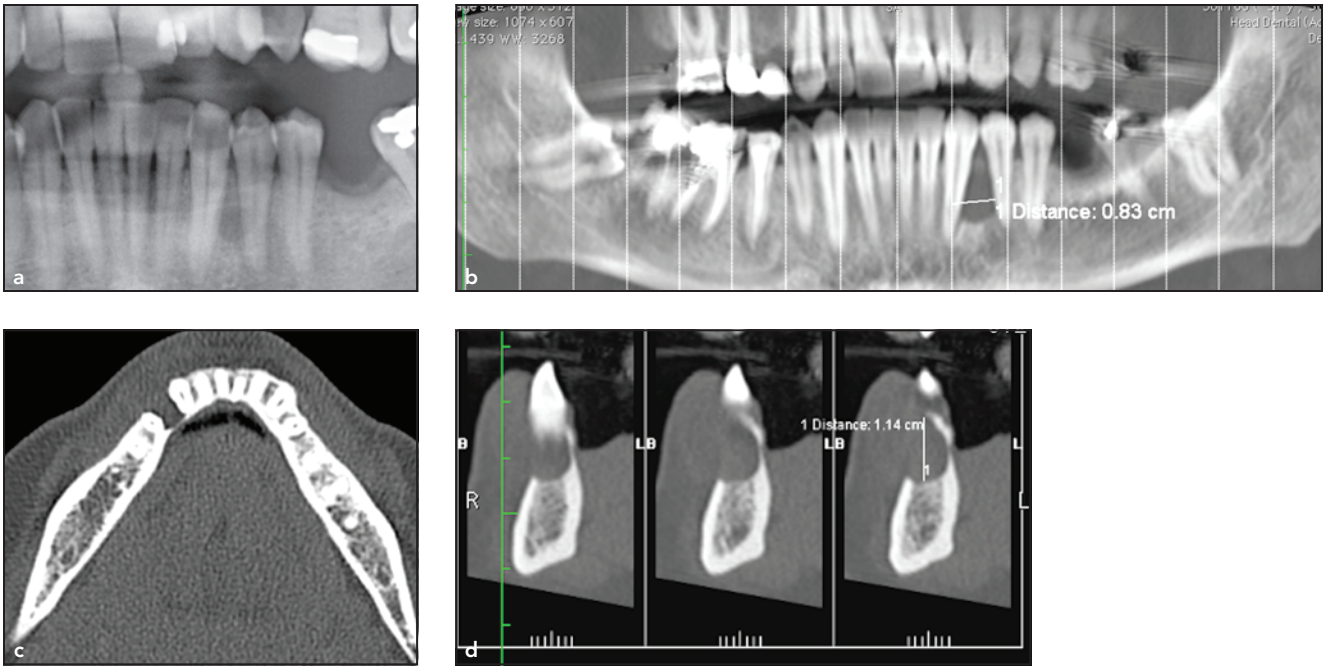

Fig 1 (a) *Preoperative radiographic image of an LPC in the test group. A radiographic examination revealed well-defined, ovoid, unilocular radiolucency with thin hyperostotic borders (arrows).* (b, c, d) *A computed tomography scan with frontal, sagittal, and transverse sections revealed an LPC that measured 11 × 8 mm and extended from an inter-radicular location into the lingual alveolar bone between the mandibular left canine and the first premolar.*

measurements of the clinical parameters from randomly selected patients. Intraexaminer agreement was verified by calculating Cohen's kappa coefficient, which ranged from 0.806 to 0.812; this coefficient predicted a good degree of reliability. The kappa coefficients were calculated for the measurements obtained at each examination.

Randomization

Patients were randomly allocated to one of the two regimens: laser + surgery (test group, n = 9) or traditional surgical treatment only (control group, n = 9). Each patient was assigned a number from 1 to 18. A clinician other than the surgeon placed the clinical data for each patient into an envelope with the corresponding number. All envelopes were sealed by an examiner who was not involved in the diagnosis or treatment of the patients. All patients were placed on a list, and the number 1 (surgery + laser) or 0 (surgery) was randomly attributed to each patient using a statistical algorithm. Shortly before each surgical session, one clinician entered the operating theater, opened the envelope bearing the assigned number by which the patient would subsequently be identified, and informed the surgeon, who performed one of the two types of treatment. The clinician immediately left the operating theater. The patients and examiners were not informed of the type of treatment, thereby avoiding bias in the evaluation of the experimental data.

Surgical technique

Each patient underwent a clinical examination. The degree of tooth mobility was measured, revealing mild horizontal mobility of the affected tooth. Orthopantomography, periapical radiographs of the site, and computed tomography (CT) were performed on each patient. The radiographic examination revealed a unilocular, well-circumscribed, radiolucent area adjacent to the affected roots. Clinically, a small, vesicle-like lesion was visible on the gingiva overlying the superior lesion (Fig 1), which confirmed the clinical diagnosis of LPC. Articaine was injected for local anesthesia, and the full-thickness flap was elevated.

In each patient in the control group, following the application of

local anesthetics, the same mucoperiosteal flap was detached from the alveolar process and the cystic lesion was excised after raising the thin cortical layer that covered the lesion. Finally, the flaps, without tension, were repositioned at their presurgical levels using 4-0 interrupted interproximal sutures (Monocryl, Ethicon), which were left in situ for 14 days without surgical packing.

Laser protocol

In the test group, articaine was injected, the full-thickness flap was elevated, and the cyst capsule was detached from the bone and root by treatment with a 980-nm diode laser (Wiser Laser Doctor Smile, Lambda) operating at an 810-nm wavelength (1 W of output power, continuous wave of 66.7 J/cm^2) that was equipped with a 0.6-mm optical fiber in continuous-wave mode (Figs 2a to 2d). The beam was transmitted by a plane-wave optical fiber, and irradiation was administered by placing the end of the optical fiber tip directly on the line between the root and lesion, which facilitated root decontamination and complete LPC excision (Fig 2c). The fiber end was assessed at each irradiation session to check for a carbonized tip (hot tip), which was required to generate sufficient thermal energy to cause tissue vaporization at the incision line. Protective eyewear was worn by the patients and the surgeon to prevent injury from laser wavelength exposure.

Finally, the tension-free flaps were repositioned at their presurgical levels using 4-0 interrupted interproximal sutures (Monocryl, Ethicon), which were left in situ for 14 days without surgical packing (Fig 2e). During this period, the patients suspended home hygiene procedures in the surgical area to minimize mechanical trauma to the tissues and only used a chlorhexidine mouthrinse (Corsodyl 200 mg/100 ml, GlaxoSmithKline).

Histologic analysis

LPC specimens obtained from patients and healthy subjects were fixed with 10% neutral buffered formalin for 24 hours. The sagittal sections (5-µm thick) prepared from the specimens were stained with hematoxylin-eosin. The histologic diagnostic criteria were the presence of serine residue, a squamous nonkeratinized or cubic epithelium composed of 1 to 3 cell layers, and epithelial plaques composed of fusiform clear cells.

Second phase of treatment and follow-up

At 7, 14, and 21 days, patients were recalled to assess all clinical parameters. None of the teeth affected by LPCs required any endodontic treatment or periapical surgery.

Postoperative short-term complications, such as facial edema (measured from the distance between the angle of the lower jaw and the pogonion) and trismus (the maximum interincisal distance in millimeters of the opening measured before and after surgery), were recorded as the differences between preoperative (baseline) and follow-up values. Total analgesic consumption (a sign of postoperative pain) was evaluated by the patient's reported number of analgesics required (patients recorded their pain level and the time at which the analgesic was consumed). Regarding patient pain, the only analgesic allowed and prescribed was ketoprofen 80 mg (Oki, Dompé) once per day. Patients rated their pain daily for 21 days using a nine-point visual analog scale (VAS) anchored by the verbal descriptors "no pain" (0) and "very severe pain" (8). Alveolitis, ecchymosis, temporary paresthesia, and gingival necrosis episodes were recorded during the follow-up session.

The patients were also clinically and radiographically evaluated at 3, 6, and 12 months after surgery. During the follow-up sessions, support therapy was introduced that consisted of supragingival scaling, polishing, and reinforcing the oral hygiene training at home.

Statistical analysis

Statistical analysis was performed by two clinicians who were blinded to the treatment protocol. For the clinical parameter measurements, a patient-level response variable was calculated for each parameter by separately computing the mean score per patient at baseline and at the end of the study for each intervention. Numeric data are expressed as the mean and

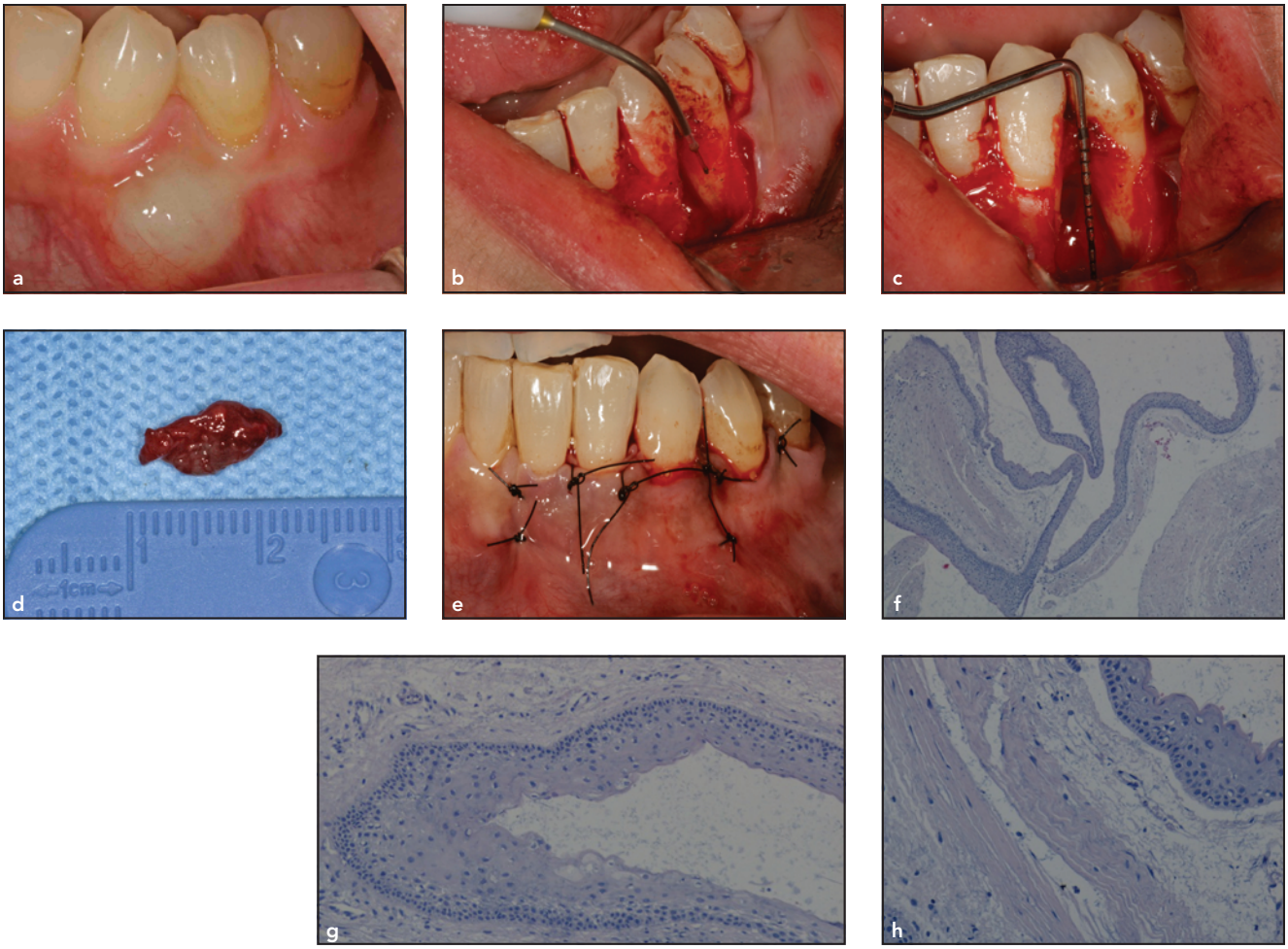

Fig 2 (a) *Clinical appearance of the LPC.* (b) *Intraoperative view of an LPC in the test group. Clinical defect measurements were taken of the bone levels.* (c) *Bone over the cyst was removed with a diode laser, and the bone defect was treated after removal of the cyst.* (d) *The lesion after surgical enucleation.* (e) *Application of single surgical sutures.* (f, g) *Photomicrographs of histologic sections of the biopsied tissue that confirmed the diagnosis of LPC. The micrographs showed that the cyst had a fibrous wall lined by a nonkeratinized, stratified squamous epithelium, which was mainly thin but had occasional tufts characterized by the formation of epithelial plaque in the lining.* (h) *A small cluster of glycogen-rich clear epithelial cells in one of the epithelial plaques.*

standard deviation (SD), and categoric variables are expressed as numbers and percentages. Most examined variables were verified by Kolmogorov-Smirnov test. Statistical comparisons between the two examined groups were performed using Student *t* test. To assess the associations between categoric variables (eg, sex, segment distribution, and size of cyst), chi-square test was performed; in the presence of expected values < 5, Fisher exact test was used. Statistical analyses were performed using SPSS software, version 11.0, for Windows. A *P* value < .05 was considered statistically significant.

Results

All enrolled patients completed the study. The mean ages of the patients were 47.5 ± 5.5 years in the control group and 45.9 ± 5.8 years in the test group, and there was a slight female predominance in the test group (Table 1).

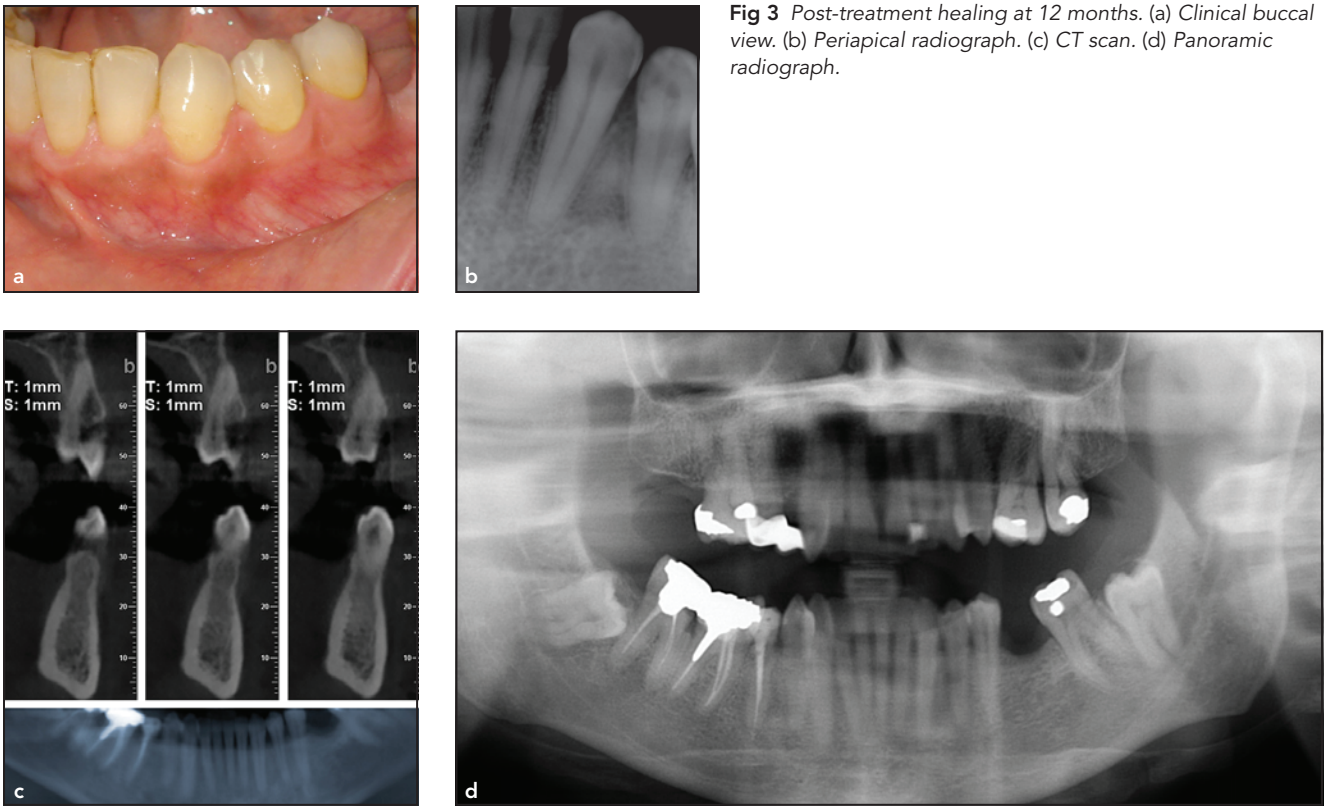

Fig 3 *Post-treatment healing at 12 months.* (a) *Clinical buccal view.* (b) *Periapical radiograph.* (c) *CT scan.* (d) *Panoramic radiograph.*

All cases were confirmed via histologic diagnoses of the biopsies performed at the end of surgery (Figs 2f to 2h). Histologic sections showed a single caries lesion with a fibrous wall lined by a nonkeratinized, stratified squamous epithelium that was primarily thin but had occasional tufts characterized by the formation of epithelial plaques in the lining. The histologic findings supported the diagnosis of LPC in all treated cases. The LPC was usually located at the mandibular premolar level or in the anterior region of the lower maxilla (Table 1).

In five patients, an inverted pear-like image was noted (maxilla), and the remaining lesions were rounded or oval (mandible). In the control group, three lesions were located in the premolar maxillary region between the premolar and canine, and one lesion was located in the mandibular incisor/canine region. In the test group, five lesions were located in the premolar region between the premolar and canine, and three lesions were located in the posterior region. The lesions were measured by CT and presented a range of 0.3 to 2.7 cm in diameter, with mean sizes of 1.1 cm in the control group and 1.2 cm in the test group. The largest lesions were found in the maxilla (Table 1).

Regarding cyst distribution in the maxilla and mandible, no significant differences were found between the right and left sides of the mouth ($P > .05$). In the maxillary region, LPCs were allocated proximal to the root in six patients and distal in three patients. In the mandible, they were mesial to the root in three patients and distal in two patients.

The short- and long-term parameters confirmed complete healing of the LPC in all enrolled patients (Fig 3). There were highly significant differences in the short-term complications at 7, 14, and 21 days for facial edema, trismus, total analgesic consumption, and pain between the test and control groups ($P < .01$), but there were no significant differences in ecchymosis, alveolitis, temporary paresthesia, or gingival necrosis between the two groups (Table 4).

Table 4 Short-Term Healing Complications of the Study Groups

Parameters	7 d		14 d		21 d	
	Control (n = 9)	Test (n = 9)	Control (n = 9)	Test (n = 9)	Control (n = 9)	Test (n = 9)
Facial edema	4.8 ± 0.2	2.9 ± 0.1*	2.8 ± 0.2	1.9 ± 0.3*	0.5 ± 0.1	0.3 ± 0.2*
Trismus (mm)	28.4 ± 0.4	36.7 ± 0.5*	31.6 ± 0.2	37.4 ± 0.3*	38.5 ± 0.1	39.2 ± 0.2*
Total analgesic consumption (n)	4.3 ± 0.2	2.2 ± 0.1*	2.4 ± 0.3	1.5 ± 0.5*	0.6 ± 0.2	0.3 ± 0.3**
Pain (value)	6.4 ± 0.1	4.5 ± 0.3*	4.2 ± 0.2	2.9 ± 0.3*	2.1 ± 0.1	0.8 ± 0.2*
Ecchymosis (n)	3	1	2	1	1	–
Alveolitis (n)	2	–	2	–	1	–
Temporary paresthesia (n)	2	1	2	–	1	–
Gingival necrosis (n)	2	–	1	–	1	–

Mean ± SD
*$P < .01$.

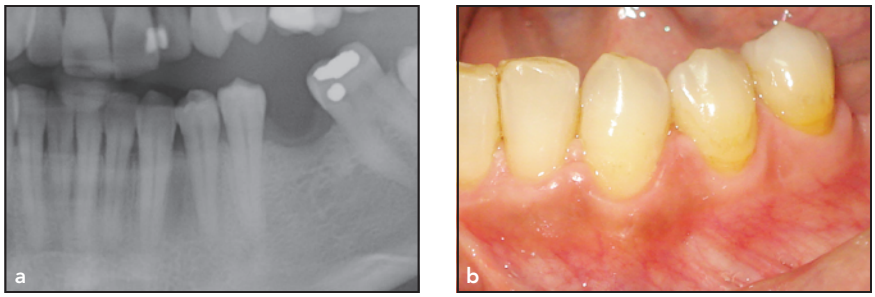

Fig 4 (a) *A postoperative radiographic image showed complete bone regrowth in the test group after 12 months.* (b) *Clinical healing of an LPC in the test group at 12 months.*

The clinical parameters assessed at baseline and after 3, 6, and 12 months are shown in Table 2. At 12 months, healing was confirmed (Fig 4). Treatment with a diode laser + surgery (test group) significantly reduced the PD and BOP values in the sites affected by LPCs at 6 and 12 months compared with the control group (Table 3). At 12 months, the PD reduction was highly significant in the test group ($P < .001$) compared with the control group (Tables 2 and 3), and the CAL gain was significant in the test group ($P < .05$) compared with the control group. Clinical and radiographic examinations 1 year after surgery indicated that only one patient in the control group presented with slightly incomplete soft tissue healing and bone fill of the initial defect.

Discussion

This study demonstrated the clinical efficacy of a comprehensive surgical treatment protocol for LPCs based on diode lasers used in combination with conventional surgical treatment. This combined approach resulted in a significant reduction in postoperative complications and an improvement in the periodontal parameters assessed after a 12-month follow-up period.

Because oral tissues consist mostly of water and diode lasers have an affinity for water and hemoglobin, this type of surgery is readily applicable for oral tissues, including the treatment of LPCs.[14] Patients in the test group who received laser treatments consistently showed rapid and persistent wound healing compared with patients in the control group.

Patients in the test group also took fewer analgesics during the postoperative period than did those in the control group. These results are in accordance with those from other studies assessing the same parameters when considering diode laser surgery for the management of oral tissues.[15,16]

Photobiostimulation of gingival tissue through a minimally invasive surgical technique using a surgical

blade or laser might be beneficial in accelerating the healing process.[17,18] In the present study, clinical healing was significantly faster in the control group (14 ± 1.6 days on average) than in the test group (21 ± 1.4 days on average). Ejiri et al[19] recently showed that laser treatment significantly increases epithelial cell proliferation and migration in association with the activation of the mitogen-activated protein kinase/extracellular signal-regulated kinase, suggesting that laser irradiation might accelerate gingival wound healing. Shimizu et al[20] demonstrated the responsiveness of periodontal ligament cells and gingival fibroblasts to low-level laser energy in vitro.

It is important to differentiate LPCs from other inflammatory cysts that occur in response to odontogenic keratocysts or other interdental lesions that could result in inappropriate management decisions, such as unnecessary endodontic therapy, periodontal procedures, tooth extraction, or aggressive surgical excision.[21–23] One possible cause of failure after LPC surgery is contamination of the site by the most aggressive periodontopathogens, such as spirochetes, which is related to the severity of the local inflammatory process.[24] Laser treatment can prevent superinfection in hard and soft tissues while ensuring the complete removal of infected tissue and the inflamed epithelium,[25] which reportedly is usually contaminated by intracellular periodontopathogens.[26] This process might reduce the risk of bacterial regrowth.[27,28]

Moreover, a diode laser was chosen in this study because it has been shown to have antibacterial activity on dentinal tubules.[29] In this context, the bactericidal activity of the diode laser, which can penetrate more than 1,000 µm into the dentin, could be used to eliminate bacteria from the dentin and thereby one cause of unsuccessful healing.[30]

The findings in the literature are not directly comparable with this study because, to the authors' knowledge, this is the first study to compare the results of surgical treatment alone with diode laser + traditional surgical treatment for LPCs. It was decided that no biomaterial graft would be used to better evaluate the characteristics and the efficacy of diode laser therapy, which probably also contributed to healing through a decortication of the bone underlying the cyst.

The use of a diode laser can raise safety concerns even with accurate setting of the irradiation parameters to reduce the possibility of iatrogenic gingival or root damage due to overheating. Another issue to take into account is the need to obtain irradiation using a noncarbonized tip. Lasers, unlike the traditional surgical protocol, have the potential advantages of bactericidal and detoxification effects, which are desirable properties for the treatment of gingival tissues and could be considered valuable for surgical treatment of LPCs.

Conclusions

This study showed that diode laser treatment results in a shorter wound healing period and could be considered valuable for surgical treatment of LPCs. This initial study is promising and demands further study to better understand the role and potential benefits of diode laser therapy in the treatment of LPCs.

Acknowledgments

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