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Original Article

YAG laser combined with mechanical debridement enhances pain relief and inflammation control in the management of periodontitis with peri-implantitis

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Abstract: Objective: To evaluate the efficacy of Er: YAG laser combined with mechanical debridement in treating periodontitis with peri-implantitis. Methods: A retrospective analysis was conducted on 292 patients treated between 2018 and 2024. The patients were divided into an observation group (n=139, Er: YAG laser + mechanical debridement) and a control group (n=153, mechanical debridement only). Outcome measures included Visual Analog Scale (VAS) scores, periodontal probing depth (PD), clinical attachment level (CAL), bleeding index (BI), gingival index (GI), and TNF-α/IL-6 levels. Clinical efficacy and risk factors for treatment failure were analyzed using logistic regression. Results: The observation group exhibited a significantly higher total efficacy rate than the control group (89.21% vs. 64.71%, P<0.001). Additionally, VAS scores, PD, CAL, BI, GI, and TNF-α/IL-6 levels were significantly lower at 1 week, 2 weeks, and 1 month post-treatment in the observation group (all P<0.05). Multivariate logistic regression identified age (OR=1.12, 95% CI: 1.07-1.18, P<0.001), smoking (OR=6.21, 95% CI: 2.96-13.82, P<0.001), and diabetes (OR=5.74, 95% CI: 2.71-12.94, P<0.001) as independent risk factors for treatment failure, while postprandial gargling (OR=0.28, P=0.002) was identified as a protective factor. Conclusion: Er: YAG laser combined with mechanical debridement significantly reduced pain and inflammation, improving peri-implant health. Smoking, diabetes, and age increased treatment failure risk, while postprandial gargling was protective, underscoring the importance of personalized treatment strategies.

Keywords: Er: YAG laser, mechanical debridement, periodontitis, peri-implantitis, pain, inflammation

Introduction

Periodontitis is a common chronic inflammatory disease primarily caused by bacteria in dental plaque. It leads to bleeding gums, formation of periodontal pockets, and alveolar bone absorption, which may result in tooth mobility and eventual tooth loss in severe cases [1, 2]. Peri-implantitis (PI) is a special form of periodontitis that occurs around dental implants and is manifested by soft tissue inflammation, bone tissue absorption, and even implant loosening [3]. With the widespread application of dental implant technology, the incidence of PI has gradually increased, making it a major concern in dentistry [4]. The coexistence of periodontitis with peri-implantitis not only seriously compromises oral health and quality of life of patients, but may also contribute to the occurrence of systemic diseases such as cardiovascular disease and diabetes [5, 6]. Therefore, effective management of periodontitis with concomitant peri-implantitis is of great clinical significance.

Current treatment strategies for this condition mainly include mechanical debridement, drug therapy, and surgical intervention [7]. Mechanical debridement involves subgingival curettage and root planing to remove plaque and calculus, thereby reducing bacterial load and promoting periodontal healing [8]. However, clinical studies have shown that mechanical debridement alone often yields limited success

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in managing PI, and adjuvant antibiotic therapy has been recommended. However, widespread use of antibiotics may induce antibiotic resistance in the subgingival microbiota, underscoring the need for judicious dosing and frequency [9]. In recent years, erbium-doped yttrium aluminum garnet (Er: YAG) laser therapy has emerged as a novel treatment for periodontitis associated with peri-implantitis [10, 11]. Shiba et al. demonstrated that Er: YAG laser combined with implantoplasty and free gingival grafting effectively reduced inflammation-related bacteria and improved peri-implant tissue health [12]. Similarly, Lu et al. reported that Er: YAG laser treatment was more effective than conventional mechanical debridement in reducing probing depth and gingival recession [11]. However, research on Er: YAG laser combined with mechanical debridement in the treatment of periodontitis with PI remains limited, and its clinical efficacy and mechanisms require further investigation.

This study aimed to evaluate the effectiveness of Er: YAG laser combined with mechanical debridement in alleviating pain and inflammation in patients with periodontitis and PI. The findings are expected to contribute to optimizing treatment strategies, enhancing therapeutic outcomes, and improving patient quality of life, while also providing theoretical and practical guidance for the clinical application of ER: YAG laser.

Patients and methods

General information

This retrospective study analyzed clinical data from patients diagnosed with periodontitis and peri-implantitis who received treatment at Norinco General Hospital, 3201 Hospital and the 987 Hospital of the Joint Service Support Force of PLA between September 2018 and June 2024. A total of 292 patients were enrolled. Among them, 139 patients receiving Er: YAG laser combined with mechanical debridement were assigned to the observation group, while 153 patients receiving mechanical debridement alone constituted the control group.

Inclusion and exclusion criteria

Inclusion criteria: (1) clinically diagnosed periodontitis with PI, characterized by a probing depth (PD) ≥4 mm, gingival bleeding or suppuration, gingival swelling, and bone loss depth <25% of implant length; (2) age ≥18 years; (3) implant time ≥6 months; (4) no implant loosening; (5) no history of antibiotics or immunosuppressant use within 1 month before treatment. Exclusion criteria: (1) patients with immune system disorders or coagulation dysfunction; (2) presence of severe organic diseases; (3) psychiatric disorders that impair treatment compliance; (4) severe systemic diseases, such as cardiovascular diseases, or hepatic/renal insufficiency; (5) pregnancy or lactation; (6) incomplete clinical data or lost follow-up. This study was approved by the Medical Ethics Committee of the 987 Hospital of the Joint Service Support Force of the Chinese People's Liberation Army.

Therapeutic method

Control group: Patients received mechanical debridement using an ultrasonic cleaning device (*EMS Piezon 250, EMS, Switzerland*) to remove supragingival calculus and plaque, followed by subgingival plaque and calculus removal with a plastic scraper (*Hu-Friedy, USA*). The periodontal pockets were rinsed alternately with 3% hydrogen peroxide (*Shanghai Chemical Reagent Co., Ltd., China*) and saline (*Baxter International Inc., USA*). Subgingival curettage and root planing were performed under local anesthesia with articaine (*Septodont, France*) using an STA electronic anesthesia syringe (*STA System, Milestone Scientific, USA*). Inflammatory granulation tissue around the implants was debrided using a titanium brush (*Straumann, Switzerland*), with care taken to avoid implant surface damage. A full oral examination was conducted using a Florida periodontal electronic probe (*Florida Probe System, Florida Probe Corporation, USA*) and plastic probe (*Hu-Friedy, USA*) to record baseline clinical data. Hemostasis was achieved following the procedure, and iodoglycerin (*Shanghai Pharmaceutical Co., Ltd., China*) was applied locally.

Observation group: In addition to the above protocol, patients in the observation group received Er: YAG laser treatment (*LightWalker, Fotona, Slovenia*). Laser parameters were set to SP mode with a frequency of 30 Hz, pulse energy of 20 mJ, power output of 0.60 W, water setting at level 8, and air setting at level 4. The laser fiber was inserted vertically into the peri-

odontal and peri-implant pockets, positioned 1 mm from the pocket base. Each site - buccal, central, and lingual, was irradiated uniformly for at least 60 seconds. Post-treatment care, including hemostasis and application of iodoglycerin (*Shanghai Pharmaceutical Co., Ltd., China*), was identical to that in the control group.

Clinical data collection

Clinical data were collected from patient records before treatment and at 1 week, 2 weeks, and 1 month following treatment. Collected data included demographic information (age, gender, body mass index [BMI]), disease characteristics (disease duration, tooth position, implant site), and relevant clinical history (postprandial gargling habits, smoking history, alcohol consumption, and diabetes history). All measurements followed standardized protocols, with periodontal PD and CAL recorded using Florida electronic and plastic periodontal probes, and gingival crevicular fluid samples collected using sterile filter paper for subsequent inflammatory marker analysis.

Observational indicators

Primary outcomes: Clinical efficacy was assessed at 1 month post-treatment and categorized into four levels: healed (complete resolution of clinical symptoms with no signs of inflammation), markedly effective (significant symptom improvement, minimal inflammation), effective (moderate symptom improvement), or ineffective (no improvement or worsening of condition). The total effective rate = (healed cases + markedly effective cases + effective cases)/total cases * 100%. Pain was measured using the Visual Analog Scale (VAS, 0-10, 0=no pain, 10=severe pain) at baseline, and at 1 day, 1 week, 2 weeks, and 1 month after treatment.

Secondary outcomes: Probing Depth (PD): Measured in millimeters and averaged over six sites per tooth or implant using an electronic probe. Clinical Attachment Level (CAL): Measured as the distance in millimeters from the cementoenamel junction or implant platform to the gingival margin. Bleeding Index (BI): Scored on a 0-5 scale (0=no bleeding, 5=spontaneous bleeding) 30 seconds after probing. Gingival Index (GI): Scored on a 0-3 scale (0=healthy, 3=severe inflammation with ulceration).

Inflammatory markers: Levels of tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) in gingival crevicular fluid were measured using enzyme-linked immunosorbent assay (ELISA, mlbio, China) and quantified with a microplate reader (iMark, Bio-Rad, USA) at baseline, 1 week, 2 weeks, and 1 month after treatment.

Statistical methods

Data were analyzed using SPSS 25.0 statistical software (IBM Corporation, USA) and R version 4.3.3 (R Foundation for Statistical Computing, Austria). Data normality was assessed using the Shapiro-Wilk test to determine appropriate statistical tests. Normally distributed continuous data were expressed as mean ± standard deviation (SD), while non-normally distributed variables were expressed as median (interquartile range, IQR). Group comparisons of continuous variables were performed using independent t-tests for normally distributed data or Mann-Whitney U tests for non-normally distributed data. Repeated measures analysis of variance (ANOVA) was used to assess changes over time, between-group differences, and time-treatment interactions, with Bonferroni post-hoc tests applied for multiple comparisons. Tukey's honest significant difference (HSD) test was used for sensitivity analysis where appropriate. Categorical data were analyzed using χ^2 tests. Multivariate logistic regression was employed to identify independent predictors of clinical efficacy. Additionally, a nomogram was constructed based on significant variables, and its performance was assessed using receiver operating characteristic (ROC) curves and calibration curves, constructed with the rms and qroc packages in R 4.3.3. All statistical analyses were reviewed by a statistician to ensure methodological rigor. Statistical significance was set at $P<0.05$ for all analyses.

Results

Baseline patient data

No significant differences were found between the observation group (n=139) and control

Table 1. Comparison of baseline data between the two patient groups

Groups	Control group (n=153)	Observation group (n=139)	t/χ^2	*P*
Age	45.18±8.15	45.32±7.81	0.143	0.886
Gender			0.208	0.648
male	84	80		
female	69	59		
BMI	23.99±1.69	23.92±1.27	-0.39	0.697
Course of disease	4.41±1.44	4.37±1.56	0.255	0.799
Tooth position			0.541	0.910
Second premolar	26	23		
First molar	50	51		
Second molar	56	47		
Other	21	18		
Planting site			0.243	0.622
Maxillary	66	56		
Lower jaw	87	83		
Postprandial gargle			0.001	0.969
Yes	69	63		
No	84	76		
History of smoking			0.318	0.573
Yes	72	70		
No	81	69		
History of alcohol consumption				
Yes	61	43	0.186	0.667
No	92	87		
History of diabetes			0.239	0.625
Yes	65	63		
No	88	76		

Note: BMI, Body Mass Index.

Table 2. Comparison of clinical efficacy between the two patient groups

Groups	Case	Healed	Markedly effective	Effective	Ineffective	Total effective rate
Control group	153	8 (5.23)	41 (26.80)	50 (32.68)	54 (35.29)	99 (64.71)
Observation group	139	20 (14.39)	63 (45.32)	41 (29.50)	15 (10.79)	124 (89.21)
χ^2						24.231
P						<0.001

group (n=153) in baseline characteristics, including age, gender, BMI, disease duration, tooth position, implant site, postprandial gargling habits, smoking status, alcohol consumption, and diabetes (all P>0.05; **Table 1**).

Clinical efficacy

The observation group achieved a significantly higher total effective rate than the control group (89.21% vs. 64.71%, P<0.001). Notably, the complete recovery rate was also higher in the observation group (14.39% vs. 5.23%). Clinical and radiographic assessments further confirmed superior outcomes in the observation group (**Table 2**; **Figure 1**).

Pain assessment

VAS scores at baseline and 1-day post-treatment showed no significant differences between groups (P>0.05). However, from 1 week to 1 month post-treatment, the observation group exhibited significantly lower VAS scores

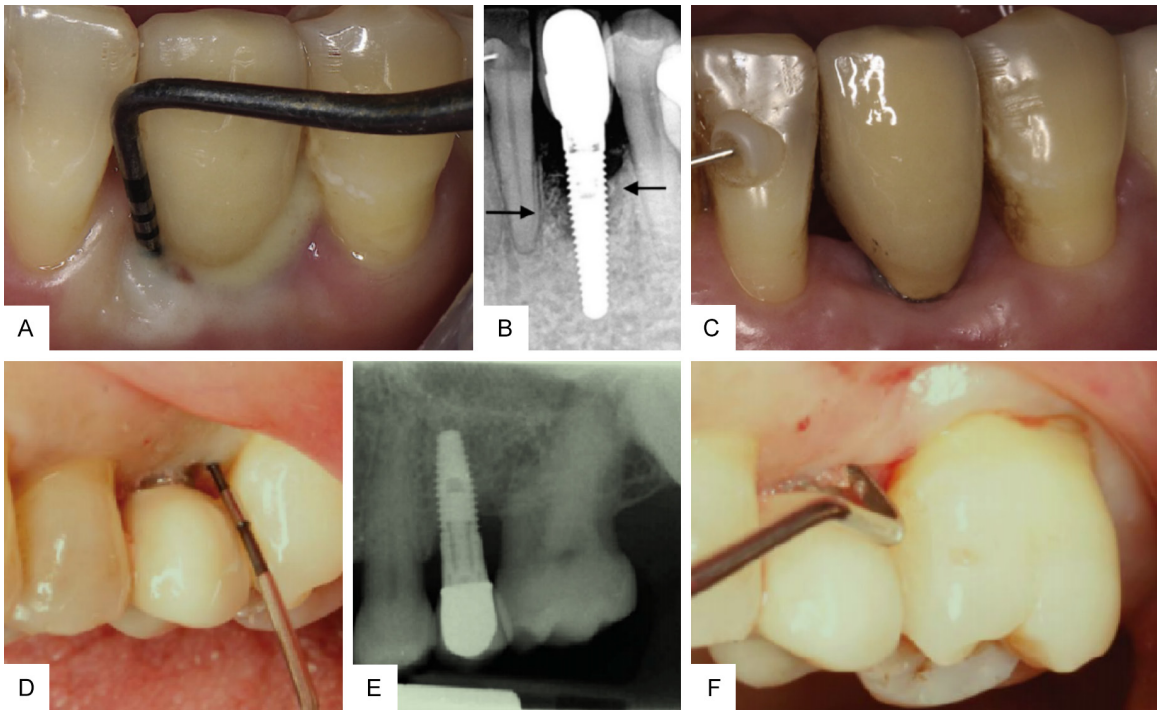

Figure 1. Clinical and radiographic images of a typical case before and after treatment. A. Preoperative clinical view showing peri-implantitis associated with chronic periodontitis, with a peri-implant pocket depth of 13 mm, accompanied by bleeding and exudate; B. Preoperative radiograph revealing a severe circumferential bone defect around the implant (indicated by the arrow); C. Postoperative clinical view at 1 month showing improved peri-implant soft tissue condition, with the pocket depth reduced to approximately 3 mm and no bleeding on probing; D. Preoperative clinical view of early peri-implantitis associated with periodontitis, with a peri-implant pocket depth of 15 mm; E. Preoperative radiograph displaying a bone defect around the implant; F. Postoperative clinical view at 1 month demonstrating improved peri-implant soft tissue recovery, with the pocket depth decreased to 4.5 mm and minimal bleeding on probing.

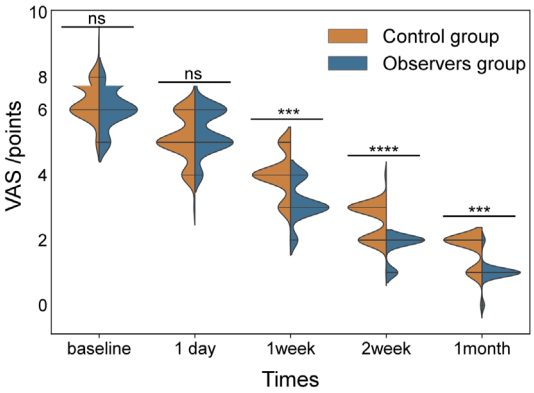

Figure 2. Comparison of VAS scores between the two groups before and at 1 day, 1 week, 2 weeks, and 1 month after surgery. Note: VAS, Visual Analogue Scale; ns P>0.05, ***P<0.001.

than the control group (all P<0.001), with a notable reduction observed at 1 month (**Figure 2**).

Dental status and peri-implant health

No significant pre-treatment differences were observed between groups in PD, CAL, BI, or GI (all P>0.05). Post-treatment, the observation group showed significantly lower PD (e.g., reduced by ~2 mm at 1 month) and CAL (e.g., reduced by ~1.5 mm at 1 month) at 1 week, 2 weeks, and 1 month, and lower BI and GI at 2 weeks and 1 month compared to the control group (all P<0.001; **Table 3**).

Inflammatory factors

Baseline levels of TNF-α and IL-6 in gingival crevicular fluid were comparable between groups (P>0.05). At 1 week, 2 weeks, and 1 month post-treatment, the observation group demonstrated significant reductions in TNF-α (e.g., ~20% reduction at 1 month) and IL-6 levels (e.g., ~25% reduction at 1 month) com-

Table 3. Comparison of PD, CAL, BI and GI levels between the two groups before and after treatment

Groups		Control group	Observation group	t/X^2	*P*
case		153	139		
PD/mm	Preoperative	5.84±0.90	5.70±0.88	-1.248	0.213
	Postoperative 1 Week	5.23±0.71	4.50±0.60	-9.454	<0.001
	Postoperative 2 Weeks	4.37±0.57	3.90±0.53	-7.322	<0.001
	Postoperative 1 month	3.29±0.42	3.06±0.36	-5.15	<0.001
CAL/mm	Preoperative	3.26±0.47	3.34±0.49	1.438	0.152
	Postoperative 1 Week	2.70±0.41	2.32±0.39	-7.994	<0.001
	Postoperative 2 Weeks	1.92±0.34	1.54±0.29	-10.38	<0.001
	Postoperative 1 month	1.32±0.21	1.15±0.18	-7.537	<0.001
BI	Preoperative	4.00 [3.00, 4.00]	4.00 [3.00, 4.00]	-0.453	0.614
	Postoperative 1 Week	3.00 [2.00, 3.00]	3.00 [2.00, 3.00]	-0.483	0.56
	Postoperative	2.00 [2.00, 2.00]	2.00 [1.00, 2.00]	-5.967	<0.001
	Postoperative 1 month	2.00 [1.00,2.00]	1.00 [1.00, 1.00]	-8.635	<0.001
GI	Preoperative	2.00 [2.00, 2.00]	2.00 [2.00, 2.00]	0.11	0.875
	Postoperative 1 Week	2.00 [2.00, 2.00]	2.00 [2.00, 2.00]	-1.038	0.139
	Postoperative 2 Weeks	2.00 [1.00, 2.00]	1.00 [1.00, 1.00]	-7.425	<0.001
	Postoperative 1 month	1.00 [1.00, 1.00]	1.00 [1.00, 1.00]	-2.435	<0.001

Note: PD, Probing Depth; CAL, Clinical Attachment Level; BI, Bleeding Index; GI, Gingival Index.

Table 4. Comparison of inflammatory cytokine levels between the two groups before and at after treatment

Groups		Control group	Observation group	t	P
case		153	139		
TNF-α/pg·mL-1	Preoperative	155.22±19.42	150.67±21.26	-1.901	0.058
	1 Week	135.24±15.95	118.00±13.62	-9.961	<0.001
	2 Weeks	96.57±10.58	88.84±10.22	-6.343	<0.001
	1 month	64.98±7.31	58.71±7.48	-7.233	<0.001
IL-6/pg·mL-1	Preoperative	482.30±55.59	485.90±49.29	0.586	0.558
	1 Week	361.59±41.73	308.17±39.61	-11.22	<0.001
	2 Weeks	260.45±33.08	238.45±28.94	-6.06	<0.001
	1 month	150.11±19.70	133.18±15.05	-8.292	<0.001

Note: TNF-α, Tumor Necrosis Factor-alpha; IL-6, Interleukin-6.

pared to the control group (all P<0.001; **Table 4**).

Baseline characteristics by treatment outcome

Patients with effective outcomes (n=223) were younger (43.78±7.75 vs. 49.99±6.81 years), had lower baseline TNF-α levels (150.54±19.97 vs. 161.19±19.79 pg/mL), and lower rates of smoking (52.91% vs. 72.46%) and diabetes (36.32% vs. 68.12%) compared to those with ineffective outcomes (n=69; all P<0.001; **Table 5**).

Prognostic analysis

We first assigned values to the collected parameters (**Table 6**). Univariate logistic regression identified age, smoking, diabetes, baseline TNF-α, and lack of postprandial gargling as significant predictors of poor clinical efficacy (all P<0.05; **Table 7**). Multivariate analysis further confirmed smoking (OR=6.21, 95% CI: 2.96-13.82, P<0.001) and diabetes (OR=5.74, 95% CI: 2.71-12.94, P<0.001) as the strongest independent risk factors, increasing risk of treatment failure by over fivefold. Age (OR=

Table 5. Comparison of clinical data between patients with different curative effects

Group	Effective group (n=223)	Ineffective group (n=69)	t/χ^2	*P*
Age	43.78±7.75	49.99±6.81	6.392	<0.001
Gender			0.044	0.834
Male	126 (56.50)	38 (55.07)		
Female	97 (43.50)	31 (44.93)		
BMI	23.89±1.50	24.18±1.49	1.380	0.170
Course of disease	4.57±1.49	4.34±1.50	1.135	0.246
Tooth position			1.201	0.753
Second premolar	39 (17.49)	10 (14.49)		
First molar	79 (35.43)	22 (31.88)		
Second molar	75 (33.63)	28 (40.58)		
Other	30 (13.45)	9 (13.04)		
Planting site			1.357	0.244
Maxillary	89 (39.91)	33 (47.83)		
Lower jaw	134 (60.09)	36 (52.17)		
Postprandial gargle			22.643	<0.001
Yes	118 (52.91)	14 (20.29)		
No	105 (47.09)	55 (79.71)		
History of smoking			20.544	<0.001
Yes	92 (41.26)	50 (72.46)		
No	131 (58.74)	19 (27.54)		
History of alcohol consumption			0.584	0.445
Yes	89 (39.91)	24 (34.78)		
No	134 (60.09)	45 (65.22)		
History of diabetes			21.635	<0.001
Yes	81 (36.32)	47 (68.12)		
No	142 (63.68)	22 (31.88)		
Baseline inflammatory marker level				
TNF-α	150.54±19.97	161.19±19.79	3.902	<0.001
IL-6	492.66±53.02	481.34±52.33	1.566	0.119

Note: BMI, Body Mass Index; TNF-α, Tumor Necrosis Factor-alpha; IL-6, Interleukin-6.

1.12, 95% CI: 1.07-1.18, P<0.001) was also a significant risk factor, while postprandial gargling was protective (OR=0.28, 95% CI: 0.12-0.61, P=0.002) (**Table 8**).

Predictive model performance for clinical efficacy

A nomogram was developed based on independent predictors to predict clinical efficacy. Age was the most influential factor, followed by postprandial gargling, smoking, and diabetes history. The predictive model demonstrated a strong discriminatory ability, with an area under the receiver operating characteristic (ROC) curve (AUC) of 0.847, indicating good predictive accuracy. Calibration analysis showed adequate agreement between predicted and observed outcomes (Sum of squared errors =36.35, P=0.850), confirming the model's reliability (**Figure 3**).

Discussion

In recent years, significant progress has been made in exploring therapeutic strategies for periodontitis with peri-implantitis [13, 14]. Although conventional mechanical debridement can effectively remove plaque and calculus from parts of the periodontal pocket, it is often insufficient to remove completely the bacterial biofilm around dental implants due to the complex surface morphology of implant structures, leading to persistent or recurrent inflammation

Table 6. Assignment table

Variable	Assignment
Age	≥45 years =1, <45 years =0
Gender	Male =1, Female =0
BMI	≥23.5=1, <23.5=0
Course of Disease	≥4 years =1, <4 years =0
Dental Position	Second Premolar =0, First Molar =1, Second Molar =2, Other =3
Implant Site	Maxilla =0, Mandible =1
Postprandial Gargling	Yes =1, No =0
History of Smoking	Yes =1, No =0
History of Alcohol Consumption	Yes =1, No =0
History of Diabetes	Yes =1, No =0
Baseline TNF-α	≥155 pg/mL =1, <155 pg/mL =0
Baseline IL-6	≥485 pg/mL =1, <485 pg/mL =0
Clinical Efficacy	Effective Group =0, Ineffective Group =1

Note: The cutoff value is used as the classification standard for all measurement data. BMI, Body Mass Index; TNF-α, Tumor Necrosis Factor-alpha; IL-6, Interleukin-6.

Table 7. Univariate Logistic regression analysis of factors associated with clinical efficacy

Variable	β	S.E.	P	OR	95% CI
Age	0.109	0.021	<0.001	1.115	1.073-1.164
Gender	-0.058	0.277	0.834	0.944	0.549-1.631
BMI	0.129	0.094	0.170	1.138	0.948-1.373
Course of disease	0.103	0.093	0.267	1.108	0.925-1.331
Dental position	0.107	0.150	0.473	1.113	0.831-1.496
Planting site	0.322	0.277	0.245	1.380	0.800-2.378
Postprandial gargle	-1.485	0.328	<0.001	0.227	0.115-0.420
History of smoking	1.321	0.302	<0.001	3.747	2.104-6.907
History of alcohol consumption	-0.219	0.287	0.445	0.803	0.452-1.400
History of diabetes	1.320	0.293	<0.001	3.745	2.130-6.757
Base line TNF-α level	0.016	0.007	0.022	1.016	1.002-1.029
Baseline IL-6 level	0.004	0.003	0.119	1.004	0.999-1.010

Note: BMI, Body Mass Index; TNF-α, Tumor Necrosis Factor-alpha; IL-6, Interleukin-6; OR, Odds Ratio; CI, Confidence Interval; S.E., Standard Error.

Table 8. Multivariate Logistic analysis of independent factors for treatment efficacy

Variable	β	S.E.	P	OR	95% CI
Age	0.110	0.025	<0.001	1.116	1.065-1.176
Postprandial gargle	-1.269	0.406	0.002	0.281	0.123-0.609
History of smoking	1.826	0.391	<0.001	6.206	2.959-13.82
History of diabetes	1.747	0.396	<0.001	5.739	2.711-12.937
Baseline TNF-α	0.016	0.009	0.065	1.016	0.999-1.034

Note: OR, Odds Ratio; CI, Confidence Interval; S.E., Standard Error.

[15]. To address these challenges, the application of laser technology in the treatment of periodontitis has gained increasing attention [16]. Er: YAG laser has emerged as a promising therapeutic approach due to its precise ablation capabilities and strong bactericidal effects. It can effectively eliminate subgingival deposits and inactivate the bacteria on implant surface

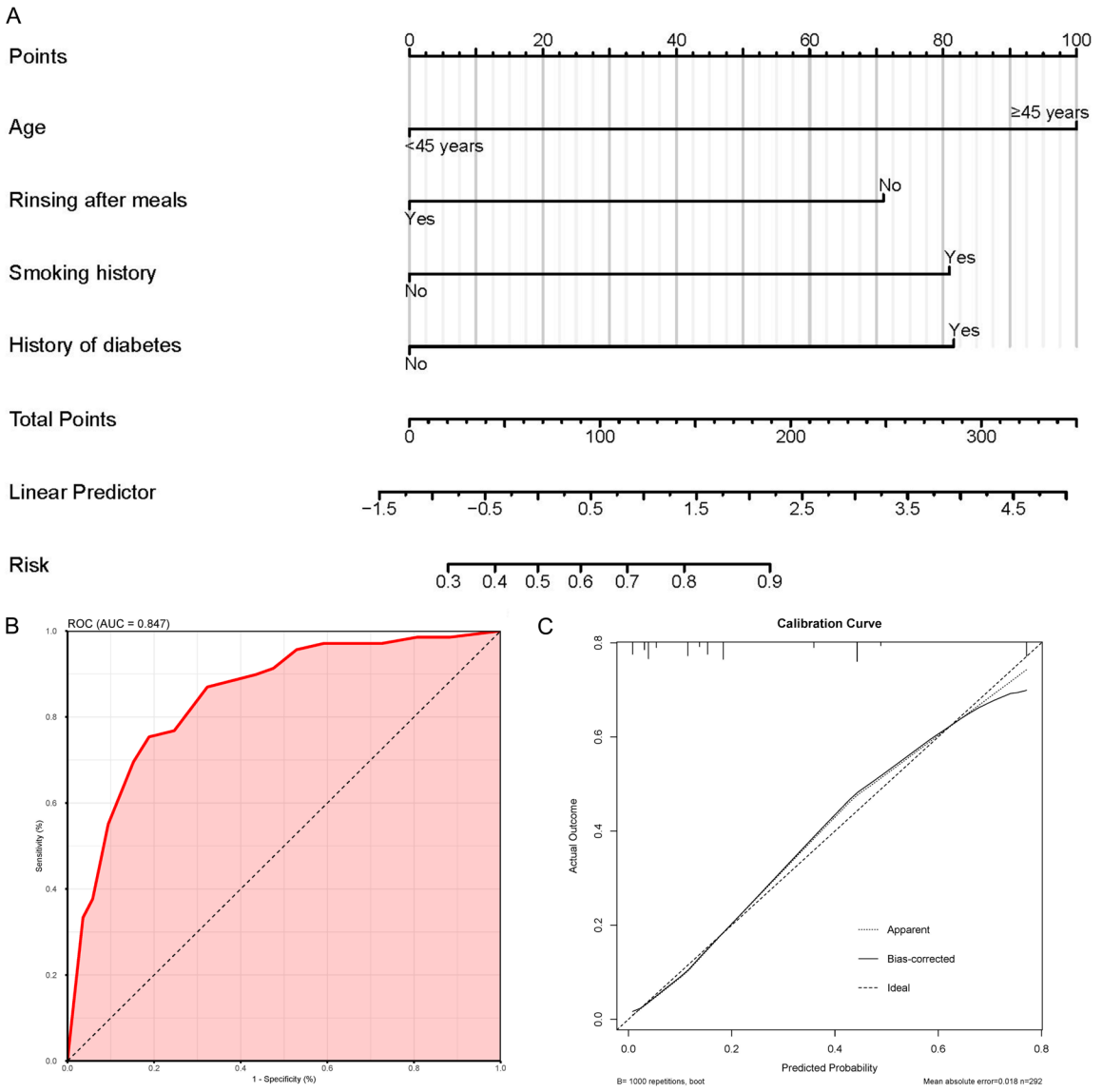

Figure 3. Nomogram, ROC Curve, and Calibration Curve for predictive efficacy. A. Nomogram illustrating the contribution of predictive factors to clinical efficacy; B. ROC curve; C. Calibration curve showing the agreement between predicted and observed outcomes (Sum of squared errors =36.35, P=0.850). The solid line represents the model's predicted value, while the dashed line indicates ideal calibration. Note: ROC, Receiver Operating Characteristic; AUC, Area Under the Curve.

simultaneously [17]. In addition, Er: YAG laser exerts biostimulatory effects that promote periodontal tissue regeneration and healing, while also alleviating pain and inflammation [18, 19]. However, studies on Er: YAG laser combined with mechanical debridement in treating periodontitis with PI remain limited, and its clinical effect and underlying mechanism still need to be further explored.

The results of this study showed that the clinical efficacy in the observation group was significantly better than that of the control group (89.21% vs. 64.71%). Er: YAG laser combined with mechanical debridement was superior to mechanical debridement alone in relieving pain and inflammation in patients with periodontitis and PI. Specifically, the VAS scores of the observation group were significantly lower at 1 week, 2 weeks, and 1 month after treatment compared to the control group, indicating more effective pain relief. In addition, improvements in periodontal and peri-implant data, including PD, CAL, BI and GI, were significantly more pro-

minent in the observation group at all post-treatment time points. These findings suggest that Er: YAG laser combined with mechanical debridement can more effectively improve the health status of the affected teeth and peri-implant tissue. In terms of inflammatory response, levels of TNF- α and IL-6 in gingival crevicular fluid of the observation group were significantly lower than those of the control group at 1 week, 2 weeks and 1 month after surgery, further confirming its advantage in reducing inflammatory response. Er: YAG laser has a strong bactericidal ability, which can effectively destroy the bacterial biofilm on the periodontal pocket and the implant surface, and reduce the bacterial load [20]. Mechanistically, the Er: YAG laser induces bacterial cell membrane rupture through thermal and photochemical effects, leading to bacterial death [21]. Er: YAG laser also has good tissue ablation ability, enabling the precise removal of calculus and diseased cementum in the periodontal pocket without damaging healthy tissues [22, 23]. Beyond its decontamination and debridement properties, the Er: YAG laser also exhibits biostimulatory effects that promote the regeneration and healing of periodontal tissue. Klepper et al. reported enhanced fibroblast proliferation and collagen maturation at 2 and 6 weeks after Er: YAG laser treatment in patients with chronic periodontitis [24]. At the molecular level, Lin et al. found that the Er: YAG laser at an energy density of 4.2 J/cm^2 promoted optimal cell proliferation, migration, and invasion of periodontal cells, partly by the regulation of galectin-7 expression [25].

Multivariate logistic regression analysis identified age, postprandial gargling habits, smoking, and diabetes history as independent factors affecting the clinical efficacy of treatment for periodontitis with PI. With increasing age, immune function gradually declines, impairing the host's ability to clear bacterial pathogen and control inflammation [26]. Additionally, the regenerative capacity of periodontal tissue is reduced in older individuals, which slows healing and negatively impacts treatment outcomes [27]. Maintaining good postprandial gargling habits can timely remove food residues and oral bacteria, and reduce the formation of dental plaque, thus reducing the risk of recurrence. Alhakeem et al. found that suboptimal oral hygiene behavior - such as reduced frequency of brushing - was an independent risk factor for bleeding (OR=3.20; P=0.04) [28]. Smoking compromises blood supply to periodontal tissue, impairs nutrient delivery and tissue repair, and inhibits the function of immune cells, thereby weakening host defenses [29]. In addition, smoking promotes the formation of dental plaque and bacterial colonization, increasing the incidence and severity of periodontitis and peri-implantitis [30]. Costa et al. reported in their study involving 350 individuals aged 35 years that the incidence of peri-implantitis was 18.2% in non-smokers (NS), 19.7% in former smokers (FS), and 30.5% in current smokers (CS), with a stunningly higher prevalence of periodontitis observed in the CS group (54.2%). This suggests that cumulative exposure to smoking and shorter duration of cessation are directly associated with a high risk of peri-implantitis [31]. Hyperglycemia in diabetic patients enhances the production and release of inflammatory factors, aggravating the inflammatory response in periodontal tissues. It also inhibits the proliferation and differentiation of fibroblasts and osteoblasts, impairing tissue repair and regeneration [32]. Peri-implantitis prevalence was significantly higher in patients with type 2 diabetes compared to non-diabetics (35.6% vs. 8.1%). Although microbial composition in shallow peri-implant pockets did not differ significantly, deep peri-implant pockets in diabetic patients showed elevated levels of *C. rectus, P. gingivalis, A. actinomycetemcomitans, and T. forsythia* [33]. Factors such as age, postprandial gargle habits, smoking history and diabetes influence treatment outcomes by distinct yet synergistic mechanisms. In clinical treatment, these factors should be fully considered, and targeted measures, such as strengthening oral health education, smoking cessation intervention and diabetes management, should be adopted to improve treatment effect and patient outcomes.

This study investigated the clinical effect of Er: YAG laser combined with mechanical debridement in treating periodontitis with peri-implantitis and revealed its significant advantages in relieving pain and inflammation. However, several limitations should be acknowledged. First, the relatively small study sample may have limited the generalizability of the findings. Second, the study mainly focused on the short-term effect, with insufficient observation of poten-

tial long-term effect and complications. Third, no stratified analyses were conducted based on implant surface properties or periodontitis severity, limiting the ability to fully elucidate differential treatment responses. Future studies should expand the sample size, extend the follow-up time, and deeply explore the long-term effect and safety of Er: YAG laser therapy combined with mechanical debridement. At the same time, stratified studies can be conducted according to the implant surface characteristics and periodontitis severity to optimize individualized treatment protocols. In addition, other adjuvant treatments, such as pharmacologic therapy or biomaterials, can also be combined to further improve the therapeutic effect.

Conclusion

This study confirmed that Er: YAG laser combined with mechanical debridement is superior to mechanical debridement alone in the treatment of periodontitis with peri-implantitis, significantly improving clinical outcomes and reducing pain and inflammation. Furthermore, age, smoking history, and diabetes were significant risk factors for poor prognosis, while postprandial gargling habits was a protective factor. These findings provide valuable clinical guidance and a basis for optimizing treatment strategies in this patient population.

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Disclosure of conflict of interest

None.

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