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Multispectral LED-Array Photobiomodulation in the Treatment of Charcot Neuroarthropathy with Osteomyelitis: A Case Report

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Case Report

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evaluated in this study as part of a collaborative research partnership with BEONE Technologies. Clinical care, patient follow-up, wound management, data collection, statistical analyses, interpretation of results, and manuscript preparation were conducted independently by the academic investigators. BEONE Technologies had no role in data analysis, interpretation of findings, or the decision to submit the manuscript for publication.

Abstract

Background: Charcot neuroarthropathy complicated by diabetic foot ulceration and osteomyelitis is associated with a high risk of lower-limb amputation and remains therapeutically challenging. Evidence on photobiomodulation (PBM) as an adjunctive approach in this highly complex clinical setting is scarce, particularly regarding simultaneous wound, neurological, vascular, and radiographic outcomes.

Case presentation: A 60-year-old Brazilian man of mixed-race ethnicity with a 25-year history of type 2 diabetes mellitus, diabetic retinopathy, peripheral arterial disease, severe sensorimotor neuropathy, and a 12-month active right-foot ulcer presented with progressive deformity, edema, erythema, increased local temperature, and a plantar hallux fistula with sanguinopurulent drainage. Wound culture was positive for methicillin-resistant *Staphylococcus aureus*. Clinical, laboratory, microbiological, vascular, neurological, and radiographic findings supported a diagnosis of complex diabetic foot syndrome associated with advanced Charcot neuroarthropathy, chronic osteomyelitis, and peripheral arterial occlusive disease. Given the severity of the condition and poor limb prognosis, lower-limb amputation was initially recommended. The patient received standardized dual-wavelength LED-array PBM as an adjunct to conventional wound care. After completion of the treatment protocol, ulcer area reduction exceeded 90%, local inflammatory signs regressed, neuropathic pain decreased, functional status improved, and the ankle-brachial index increased from 0.76 to 1.10. Follow-up radiographs demonstrated reduced active bone destruction, increased sclerosis, coalescence of bone fragments, reactive new bone formation, and partial consolidation, consistent with progression toward a more stable phase of Charcot neuroarthropathy. No treatment-related adverse events were observed.

Conclusion: This case documents a clinically noteworthy multidimensional improvement in a patient with severe diabetic foot disease and an initially unfavorable limb prognosis. The observed wound, neurological, vascular, and radiographic changes during adjunctive PBM supports further investigation of standardized PBM within multidisciplinary limb-preservation strategies for complex diabetic foot presentations.

INTRODUCTION

Diabetic foot syndrome remains a major cause of hospitalization, lower-limb amputation, functional disability, and reduced quality of life among individuals with type 2 diabetes mellitus (T2DM). Its clinical complexity is largely driven by the coexistence of peripheral neuropathy, peripheral arterial disease (PAD), recurrent infection, and impaired tissue repair, which together contribute to delayed wound healing, ulcer recurrence, and poor limb-related outcomes (1–3). Among its most severe musculoskeletal complications, diabetic Charcot neuroarthropathy (DCN) is characterized by progressive osteoarticular destruction, foot deformity, and mechanical instability, creating abnormal pressure points that further compromise soft-tissue integrity. When associated with osteomyelitis, diagnosis and treatment become particularly challenging because inflammatory, infectious, and neuroarthropathic changes may overlap clinically and radiographically (2, 4).

Photobiomodulation (PBM) has emerged as a potential adjunctive strategy for diabetic foot ulcers (DFU) because of its anti-inflammatory, pro-angiogenic, biostimulatory, and possible neuroprotective effects, with experimental and clinical evidence suggesting benefits for tissue repair and bone remodeling (5–8). However, evidence regarding PBM in highly complex presentations combining DFU, DCN, osteomyelitis, peripheral arterial disease, and severe neuropathy remains limited. This case is unique because it documents the simultaneous clinical, neurovascular, and radiographic evolution of a patient with a limb-threatening diabetic foot condition and an initially unfavorable prognosis, including a recommendation for lower-limb amputation, during treatment with a standardized multispectral light-emitting diode (LED) array PBM protocol as an adjunct to conventional care.

CASE PRESENTATION

This case report describes a clinically noteworthy finding observed in a patient enrolled in an ongoing clinical trial evaluating PBM as an adjunctive therapy for the healing of DFU. The trial is registered in the Brazilian Registry of Clinical Trials (Registro Brasileiro de Ensaios Clínicos, ReBEC) under registration number RBR-7n3y2gq, registered on February 21, 2025. The clinical trial was designed to investigate DFU healing and was not specifically focused on osteomyelitis or DCN. The present case was selected for reporting because of the severity and complexity of the patient's baseline clinical condition and the noteworthy clinical and radiographic evolution observed during follow-up.

A 60-year-old Brazilian man of mixed-race ethnicity with a 25-year history of T2DM, treated with oral glucose-lowering agents, presented with a chronic right-foot ulcer and progressive foot deformity. His medical history was notable for diabetic retinopathy, PAD, and chronic alcohol consumption. The ulcer had remained active for approximately 12 months and was accompanied by progressive edema, erythema, and increased local temperature. The patient also reported burning pain, paresthesia, tingling, frequent muscle cramps, and distal weakness.

Before enrollment in the clinical trial, the patient had been under regular follow-up by vascular surgery and orthopedic specialists and was receiving conventional wound care, including regular dressings and debridement as clinically indicated. Despite ongoing multidisciplinary management, the ulcer remained active, with persistent tissue breakdown and progressive foot deformity at the time of enrollment.

At presentation, physical examination revealed marked deformity of the right foot and a plantar hallux fistula with continuous sanguinopurulent, malodorous drainage. An additional dorsal foot ulcer contained extensive necrotic tissue and had an initial area of 5.1 cm^2 (Fig. 1). The posterior tibial and dorsalis pedis pulses were not palpable, and the ankle–brachial index (ABI) was 0.76.

Neurological examination demonstrated severe diabetic peripheral neuropathy. Protective plantar sensation was completely absent on 10-g Semmes–Weinstein monofilament testing, while tactile and vibratory perception were markedly reduced. Achilles tendon reflexes were bilaterally diminished. These findings were consistent with advanced sensorimotor diabetic neuropathy with loss of protective sensation and substantial biomechanical instability.

The chronological sequence of the patient's clinical course, diagnostic assessments, therapeutic interventions, and outcomes is summarized in Table 1.

Table 1
Timeline of the patient's clinical course, assessments, interventions, and outcomes

Time point	Clinical event, assessment, or intervention
Approximately 25 years before enrollment	Diagnosis of type 2 diabetes mellitus (T2DM), followed by the subsequent development of diabetic retinopathy and PAD.
Approximately 12 months before enrollment	Persistent right-foot ulcer associated with progressive foot deformity, edema, erythema, and increased local temperature despite ongoing conventional wound care.
Baseline/enrollment	Marked right-foot deformity; plantar hallux fistula with sanguinopurulent drainage; dorsal necrotic ulcer measuring 5.1 cm^2; ABI, 0.76; HbA1c, 8.6%; CRP, 69.9 mg/L; Hb, 7.8 g/dL; MRSA-positive wound culture; and severe sensorimotor neuropathy.
Baseline diagnostic assessment	Lower-limb duplex Doppler ultrasonography demonstrated predominantly infrapopliteal PAD with reduced distal blood flow. Radiographs showed extensive osteoarticular destruction compatible with advanced DCN, and the overall findings supported concomitant chronic osteomyelitis.
Baseline therapeutic decision	The overall clinical condition was associated with an unfavorable limb prognosis, and lower-limb amputation was recommended by the vascular surgery team.
Weeks 1–2	PBM sessions 1–6 administered as an adjunct to conventional wound care.
Weeks 3–4	PBM sessions 7–12; progressive granulation tissue formation and re-epithelialization, with reduction in local inflammatory sign.
Weeks 5–6	PBM sessions 13–18; continued wound improvement, with a greater than 90% reduction in ulcer area by completion of treatment.
End of treatment	ABI increased to 1.10; neuropathic pain decreased; functional status improved; and follow-up radiographs demonstrated reduced signs of active bone destruction, increased osseous sclerosis, coalescence of bone fragments, remodeling, and partial consolidation.

DIAGNOSTIC ASSESSMENT

Laboratory investigations demonstrated poor glycemic control, with glycated hemoglobin (HbA1c) of 8.6%, marked systemic inflammation with a C-reactive protein concentration of 69.9 mg/L, and severe anemia, with a hemoglobin concentration of 7.8 g/dL and hematocrit of 22.6%. Wound-bed culture yielded methicillin-resistant *Staphylococcus aureus* (MRSA).

Lower-limb duplex Doppler ultrasonography demonstrated peripheral arterial occlusive disease, predominantly affecting the infrapopliteal segment, with reduced distal blood flow.

Plain radiographs of the right foot obtained in anteroposterior and oblique projections demonstrated extensive midfoot and hindfoot osteoarticular disruption, including multiple bone fragments, severe deformity, subchondral sclerosis, extensive osseous remodeling, collapse of the tarsometatarsal and intertarsal joints, loss of anatomical alignment, focal bone resorption, and reactive new bone formation. No acute fracture was identified (Fig. 2). Taken together, the clinical and radiographic findings were compatible with advanced DCN with an active fragmentation pattern.

Diagnostic interpretation was challenging because active DCN and osteomyelitis may share clinical and radiographic features, including local warmth, edema, erythema, bone fragmentation, cortical disruption, osteolysis, and reactive bone formation, particularly in the setting of chronic ulceration and infection.

The patient had substantial financial constraints and received care through the Brazilian Unified Health System (Sistema Único de Saúde, SUS). Transportation costs, expenses associated with repeated clinical visits and wound-care supplies represented additional challenges to longitudinal follow-up and access to specialized care.

The ulcer was classified as University of Texas Grade III, Stage D, indicating a deep wound with bone involvement in the presence of infection and ischemia. According to the PEDIS/IWGDF classification, the patient exhibited impaired perfusion, extensive tissue involvement, infection, and loss of protective sensation. The SINBAD score was also consistent with a high-risk DFU (Fig. 1).

The overall diagnostic assessment supported a complex diabetic foot syndrome characterized by advanced DCN, chronic infected ulceration with bone involvement consistent with chronic osteomyelitis, severe sensorimotor neuropathy, and peripheral arterial occlusive disease.

The main differential diagnoses included isolated DCN, chronic osteomyelitis without neuropathic arthropathy, advanced degenerative osteoarthropathy, deep soft-tissue infection, and ischemic tissue loss secondary to peripheral arterial disease. However, the coexistence of long-standing T2DM, severe sensorimotor neuropathy, chronic infected ulceration with bone involvement, MRSA infection, impaired distal perfusion, and extensive osteoarticular destruction favored the diagnosis of complex diabetic foot syndrome involving DCN, chronic osteomyelitis, and peripheral arterial occlusive disease.

The coexistence of chronic infection, severe neurovascular impairment, extensive osteoarticular destruction, and a high-risk DFU classification indicated an unfavorable limb prognosis. In view of the anticipated high risk of treatment failure and major limb loss, lower-limb amputation was recommended by the vascular surgery team.

THERAPEUTIC INTERVENTION

As an adjunct to conventional wound care, PBM was initiated using a customized LED-array device developed for the clinical trial registered as RBR-7n3y2gq (ISIS®, BEONE Technologies, Brazil). The device features a closed, dome-shaped configuration that allows the affected limb to be positioned within the irradiation field, thereby providing reproducible positioning and homogeneous light delivery to the ulcer bed and surrounding tissues.

The PBM protocol combined red and blue light. Red light was delivered at a wavelength of 635 nm, with an irradiance of 4 mW/cm² for 30 minutes, corresponding to an energy density of 7.2 J/cm². Blue light was delivered at a wavelength of 415 nm, with an irradiance of 2 mW/cm² for 20 minutes, corresponding to an energy density of 2.4 J/cm². Treatment was administered three times per week for 6 weeks, for a total of 18 sessions. Conventional wound care was continued throughout the intervention period, including wound dressings and debridement when clinically indicated.

The planned PBM protocol was maintained throughout the 18-session treatment period without modification.

FOLLOW-UP AND OUTCOMES

Clinical assessments were performed at baseline and after 6, 12, and 18 PBM sessions. At each assessment, the ulcer was evaluated for wound area, wound-bed characteristics, granulation tissue, exudate, re-epithelialization, and clinical signs of infection. Standardized photographs were obtained, and wound area was quantified by digital planimetry using ImageJ software.

Neurovascular follow-up included measurement of the ABI, palpation of the dorsalis pedis and posterior tibial pulses, 10-g Semmes–Weinstein monofilament testing, Achilles tendon reflex assessment, vibratory perception using a 128-Hz tuning fork, and assessment using the Neuropathy Symptom Score. Right-foot radiographs were obtained at baseline and after completion of the 18-session protocol to assess changes in osteoarticular findings.

Progressive wound improvement was observed during follow-up (Fig. 1). Local erythema, edema, and other inflammatory signs decreased, accompanied by progressive granulation tissue formation and re-epithelialization. By completion of the 18-session protocol, wound area had decreased by more than 90%. Neuropathic symptoms also improved, particularly neuropathic pain. Numbness and other sensory disturbances persisted but were reported at lower intensity. The patient additionally reported improvement in functional status.

The ABI increased from 0.76 at baseline to 1.10 at the final assessment, although the dorsalis pedis and posterior tibial pulses remained nonpalpable (Fig. 3). Follow-up radiographs demonstrated persistent residual deformity and midfoot collapse but showed increased osseous sclerosis and cortical definition, fewer radiographic signs of active bone destruction, reactive new bone formation, coalescence of bone fragments, skeletal remodeling, and partial consolidation (Fig. 2). Compared with baseline, osteolysis and fragmentation were reduced, whereas evidence of remodeling and structural stabilization increased.

These findings were consistent with radiographic evolution from an active fragmentation phase toward the coalescence and reconstruction phases of DCN.

A multidimensional graphical summary of the clinical course demonstrated concurrent changes in wound area, neuropathic pain, local inflammatory signs, ABI, osteolysis, bone fragmentation, bone remodeling, and structural stability (Fig. 4).

The patient completed all 18 scheduled PBM sessions and attended the planned follow-up assessments. Treatment was well tolerated, with no treatment interruption attributable to discomfort or intolerance. No adverse events, unanticipated events, or PBM-related complications were recorded during the 6-week treatment period.

DISCUSSION AND CONCLUSIONS

This case documents a multidimensional clinical and radiographic evolution in a patient with severe diabetic foot disease, advanced DCN, chronic osteomyelitis, PAD, and an initially unfavorable limb prognosis. The concurrent changes in wound healing, neuropathic symptoms, vascular assessment, and radiographic findings are noteworthy; however, observations from a single case cannot establish causality or therapeutic efficacy.

DFU healing requires coordinated angiogenesis, regulation of inflammation, cellular proliferation, extracellular matrix deposition, and tissue remodeling. In T2DM, chronic hyperglycemia contributes to mitochondrial dysfunction, oxidative stress, persistent inflammation, endothelial dysfunction, and impaired reparative responses, thereby delaying wound healing and increasing susceptibility to infection (1, 2). PBM has been proposed to modulate several of these processes through the absorption of light by intracellular chromophores, particularly mitochondrial cytochrome c oxidase, with downstream effects on adenosine triphosphate production, cellular redox balance, and signaling pathways involved in tissue repair (3–5, 9, 10). Experimental and clinical studies have also reported modulation of pro-angiogenic pathways involving vascular endothelial growth factor (VEGF), VEGF receptor 2 (VEGFR-2), and hypoxia-inducible factor 1-alpha (HIF-1α), which may contribute to angiogenesis and microvascular function (5, 6, 11, 12). The progressive wound contraction, granulation tissue formation, re-epithelialization, and regression of local inflammatory signs observed in this patient are biologically compatible with these proposed mechanisms; however, a direct mechanistic relationship cannot be established from this case.

The increase in ABI from 0.76 at baseline to 1.10 at the final assessment is also of clinical interest. Nevertheless, this change should not be interpreted as definitive evidence of improved limb perfusion or as a direct effect of PBM. ABI measurements may be affected by arterial stiffness and medial arterial calcification in patients with diabetes, and the dorsalis pedis and posterior tibial pulses remained nonpalpable at the final assessment. Additional vascular measurements, such as toe pressure, transcutaneous oxygen pressure, or serial Doppler-derived parameters, would have provided a more comprehensive assessment of changes in lower-limb perfusion.

Reduction in neuropathic pain was another clinically relevant finding. Diabetic peripheral neuropathy is characterized by axonal degeneration, neural microvascular dysfunction, mitochondrial injury, and chronic neuroinflammation (7, 13). Experimental and clinical evidence suggests that PBM may influence peripheral nerve function through modulation of neurotrophic signaling, mitochondrial activity, inflammatory mediators, endoneural microcirculation, and neuronal excitability (8, 9, 14, 15). The reduction in neuropathic pain observed in this patient is consistent with previous reports describing analgesic and functional effects of PBM in peripheral neuropathies. However, numbness and other sensory deficits persisted, although at lower intensity, underscoring the chronic and only partially reversible nature of advanced diabetic sensorimotor neuropathy.

The radiographic evolution represents a particularly noteworthy aspect of this case. Despite persistent residual deformity and midfoot collapse, serial radiographs demonstrated increased osseous sclerosis and cortical definition, coalescence of bone fragments, reactive new bone formation, reduced evidence of active bone destruction, and partial consolidation. These findings were compatible with progression from an active fragmentation phase toward the coalescence and reconstruction phases of the Eichenholtz classification. DCN is characterized by dysregulated bone turnover arising from the interaction of neuropathy, persistent inflammation, autonomic dysfunction, increased osteoclastic activity, altered vascular regulation, and repetitive mechanical stress (16, 17). Experimental studies suggest that PBM may influence bone metabolism through effects on osteoblast activity, collagen synthesis, osteogenic signaling, angiogenesis, and bone repair (18). Nevertheless, progression from fragmentation toward coalescence is also part of the natural history of DCN. Therefore, the radiographic changes observed in this patient cannot be attributed specifically to PBM. Their temporal association with the intervention provides a hypothesis for further investigation using serial quantitative imaging and biomarkers of bone turnover.

The clinical course of the infection was also noteworthy because the baseline wound culture was positive for MRSA. Blue light has demonstrated antimicrobial activity in experimental models through photoexcitation of endogenous bacterial chromophores, including porphyrins, resulting in the generation of reactive oxygen species and subsequent microbial damage (19, 20). No clinical evidence of ongoing soft-tissue infection was observed during follow-up. However, serial cultures and quantitative assessments of microbial burden were not performed; therefore, an antimicrobial effect of blue-light PBM cannot be inferred from this case.

A major strength of this report is the longitudinal and multidimensional assessment of an unusually complex diabetic foot presentation. Wound characteristics, neurological findings, vascular parameters, standardized photographic planimetry, and serial radiographic changes were assessed concurrently throughout treatment. Furthermore, the patient presented with several established indicators of poor limb outcome, including long-standing T2DM, severe sensorimotor neuropathy, PAD, chronic osteomyelitis, MRSA-positive ulceration, severe anemia, poor glycemic control, systemic inflammation, extensive osteoarticular destruction, and an initial recommendation for lower-limb amputation. The

observation of substantial wound improvement and clinical stabilization in this context makes the clinical course particularly noteworthy.

Several limitations must also be acknowledged. As a single case report, this study cannot establish efficacy or causality. PBM was administered as an adjunct rather than as monotherapy, and concomitant wound care, debridement, infection management, offloading, metabolic management, specialist follow-up, and the natural evolution of DCN may have contributed to the observed outcome. In addition, microbial burden was not serially quantified, advanced vascular measurements were not performed, and osteoarticular evolution was evaluated primarily by conventional radiography. These limitations prevent determination of the relative contribution of PBM to the observed clinical and radiographic changes.

Taken together, the temporal convergence of substantial wound reduction, regression of inflammatory signs, decreased neuropathic pain, functional improvement, changes in ABI, and radiographic progression toward greater osteoarticular stability suggests that the clinical course merits further investigation. The biological plausibility provided by experimental and clinical evidence regarding PBM effects on tissue repair, inflammation, neural function, angiogenesis, and bone metabolism supports investigation of these observations in appropriately designed studies. However, the present findings should be regarded as hypothesis-generating rather than evidence of a therapeutic effect.

In conclusion, this case documents concurrent wound, neurological, vascular, and radiographic changes in a patient with severe diabetic foot disease, DCN, chronic osteomyelitis, PAD, and an initially unfavorable limb prognosis during adjunctive LED-array PBM and multidisciplinary conventional care. These observations are hypothesis-generating and support further controlled investigation of PBM as a component of multidisciplinary limb-preservation strategies

Abbreviations

ABI – Ankle-Brachial Index

DCN – Diabetic Charcot Neuroarthropathy

DFU – Diabetic Foot Ulcers

HbA1c – Glycated Hemoglobin

LED – Light-Emitting Diode

MRSA - Methicillin-Resistant *Staphylococcus aureus*

PAD – Peripheral Arterial Disease

PBM – Photobiomodulation

T2DM – type 2 diabetes mellitus

VEGF –Vascular Endothelial Growth Factor (VEGF),

Declarations

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This case report describes a clinically noteworthy finding observed in a patient enrolled in an ongoing clinical trial evaluating photobiomodulation as an adjunctive therapy for the healing of diabetic foot ulcers. The clinical trial protocol was approved by the Human Research Ethics Committee of HUOC/PROCAPE, University of Pernambuco, Brazil (Approval No. 7.337.674), and was conducted in accordance with the ethical principles of the Declaration of Helsinki. The trial is registered in the Brazilian Registry of Clinical Trials (Registro Brasileiro de Ensaios Clínicos, ReBEC) under registration number RBR-7n3y2gq, registered on February 21, 2025. Written informed consent to participate in the clinical trial was obtained from the patient prior to enrollment.

CONSENT FOR PUBLICATION

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal upon request.

DATA AVAILABILITY STATEMENT

The data supporting the findings of this case report are available from the corresponding author upon reasonable request. Data that could compromise patient confidentiality are not publicly available.

COMPETING INTEREST

Faculty members and researchers from the University of Pernambuco (UPE) participated in the development of the photobiomodulation prototype evaluated in this study as part of a collaborative research partnership with BEONE Technologies. Clinical care, patient follow-up, wound management, data collection, statistical analyses, interpretation of results, and manuscript preparation were conducted independently by the academic investigators. BEONE Technologies had no role in data analysis, interpretation of findings, or the decision to submit the manuscript for publication.

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AUTHOR CONTRIBUTIONS

Leandro Cruz and Taciana F. Mendonca-Belmont contributed to the conceptualization, methodology, formal analysis, and drafting of the manuscript. Andreia S Silva contributed to patient care, clinical follow-up, and data collection. Sabrina P Silva and Stephanne Katrine G S Pedrosa contributed to data collection, clinical follow-up, and organization of study-related information. Bruno M Carvalho contributed to data analysis and interpretation of the results. Francisco Bandeira contributed to the conceptualization and review and editing of the manuscript. Patricia Moura contributed to the conceptualization, resources, supervision, project administration, and review and editing of the manuscript. All authors read and approved the final version of the manuscript.

DECLARATION OF GENERATIVE AI AND AI-ASSISTED TECHNOLOGIES IN THE MANUSCRIPT PREPARATION PROCESS

During the preparation of this paper the authors used ChatGPT (OpenAI) to assist with language editing and improvement of the figures. The authors take full responsibility for the content of the manuscript.

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Figures



Figure 1

Serial Clinical Evolution of a Diabetic Foot Ulcer During Photobiomodulation Therapy.

Representative clinical photographs obtained at baseline and during follow-up demonstrating progressive wound contraction, replacement of devitalized tissue by healthy granulation tissue, reduction

of inflammation and exudate, re-epithelialization, and complete wound healing following 18 PBM sessions.



Figure 2

Serial Radiographic Evolution of Charcot Neuroarthropathy and Chronic Osteomyelitis Following Photobiomodulation Therapy.

Serial radiographs obtained at baseline, and after completion of 18 sessions demonstrating progressive structural changes, including increased bone sclerosis, improved cortical definition, fragment coalescence, reactive bone formation, and partial bone remodeling, consistent with progression toward the coalescence and reconstruction stages of the Eichenholtz classification.

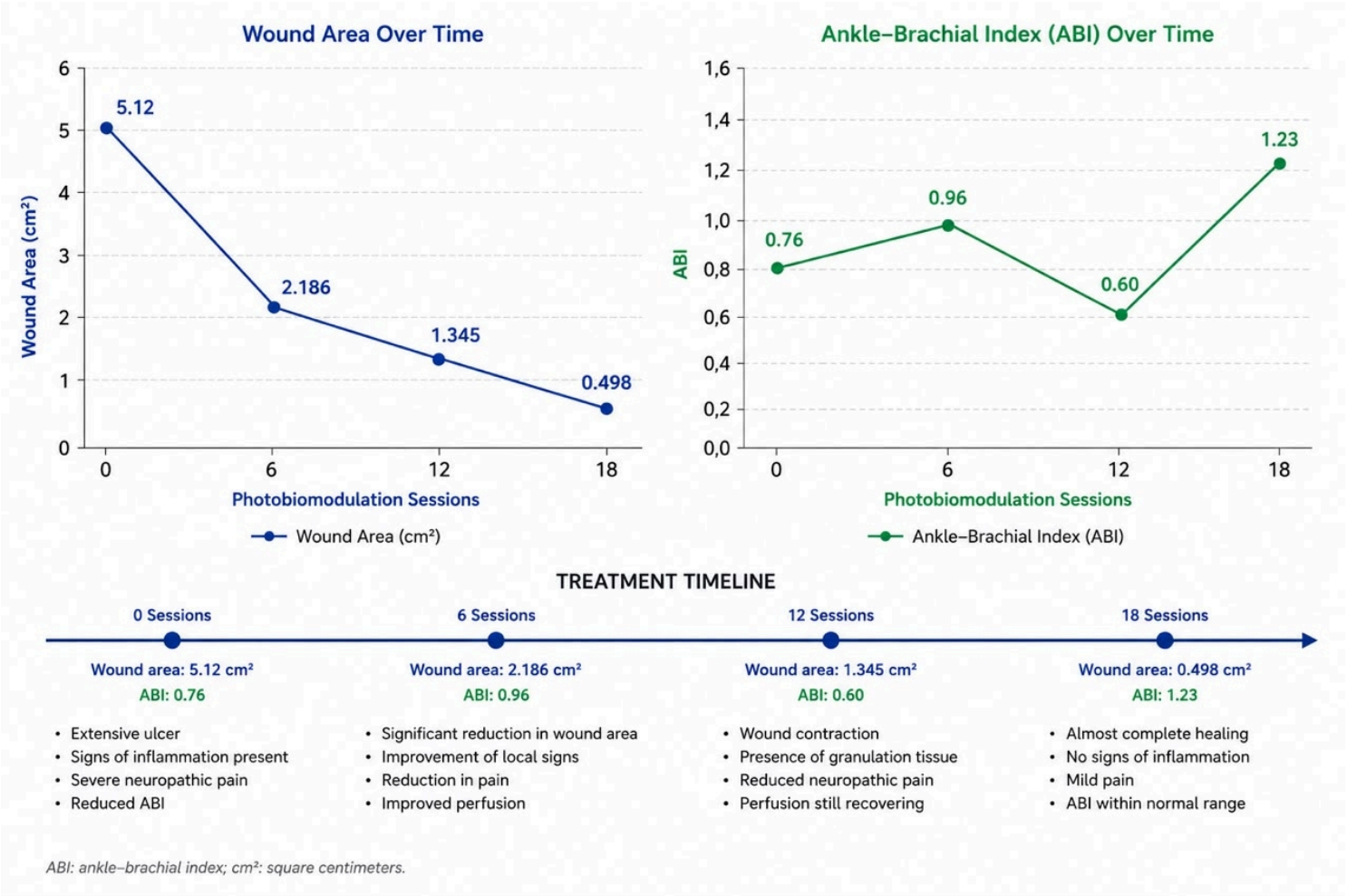
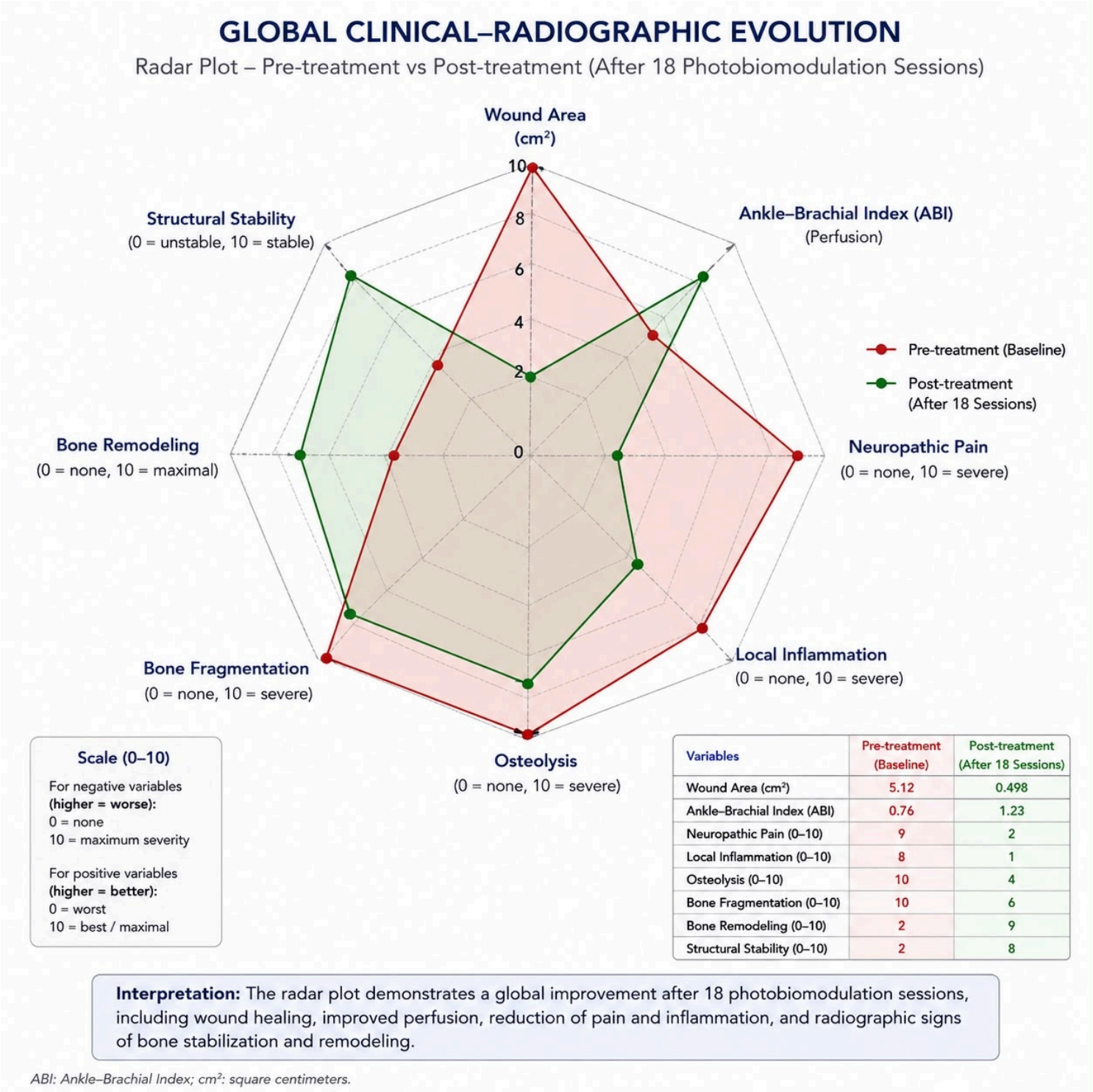


Figure 3

Longitudinal Changes in Wound Healing, Perfusion, and Clinical Outcomes During Photobiomodulation Therapy.

Time-course representation of wound area reduction, ankle-brachial index (ABI), and major clinical milestones throughout treatment, demonstrating progressive tissue repair, improved lower-limb perfusion, and functional recovery.



Variables	Pre-treatment (Baseline)	Post-treatment (After 18 Sessions)
Wound Area (cm²)	5.12	0.498
Ankle–Brachial Index (ABI)	0.76	1.23
Neuropathic Pain (0–10)	9	2
Local Inflammation (0–10)	8	1
Osteolysis (0–10)	10	4
Bone Fragmentation (0–10)	10	6
Bone Remodeling (0–10)	2	9
Structural Stability (0–10)	2	8

Figure 4

Integrated Clinical, Vascular, and Radiographic Outcomes Following Photobiomodulation Therapy.

Radar plot comparing baseline and post-treatment multidimensional outcomes, integrating wound healing, local inflammation,

neuropathic pain, ankle-brachial index, bone remodeling, structural stability, osteolysis, and bone fragmentation. The post-treatment profile demonstrates a multidimensional therapeutic response extending beyond wound closure, with evidence of neurovascular recovery and progressive osteoarticular stabilization.

Supplementary Files

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- CAREchecklistEnglish2013final.pdf