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Review

Demodicosis: Update and Treatment With Laser and Other Light Sources

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ABSTRACT

Demodex folliculorum and *Demodex brevis* are common commensal mites of the human pilosebaceous unit that, under certain conditions, may proliferate excessively and cause demodicosis, generating a broad range of dermatological and/or ocular signs. Diagnosis is usually clinical but can be confirmed by quantifying mites using techniques such as standardized skin surface biopsy. The therapeutic approach to demodicosis includes topical and systemic treatments and, more recently, the use of laser treatments and other light sources. This review explores the pathogenesis, different clinical presentations, and therapeutic alternatives proposed for demodicosis, with special emphasis on therapies using light sources.

Introduction

Demodicosis is a skin disease caused by excessive proliferation of mites of the genus *Demodex*, which includes the species *Demodex folliculorum* and *Demodex brevis*.[1–3] These mites, first described in 1841,[4] are common commensals on human skin, colonizing the pilosebaceous unit and feeding on sebum by using lipase enzymes for digestion. *D. folliculorum* is mainly located in the follicular infundibulum, whereas *D. brevis* is found in deeper areas, such as the sebaceous glands and their ducts.[3] Both species can mainly be found on the face, paranasal area, eyelashes, or eyebrows, although *D. brevis* may also colonize other areas.[2] *Demodex* colonization is common in the population, with rates ranging from 20% to 80% and reaching 100% in older individuals, although these rates may vary greatly depending on the detection method.[3] In children and adolescents, *Demodex* colonization is significantly lower, since sebaceous follicles are colonized during late childhood and adolescence through transmission by direct contact.[2] Although these mites are saprophytes and generally do not cause clinical manifestations, under certain conditions they can become pathogenic, causing a variety of cutaneous and ocular clinical signs.

This review analyzes the known pathogenic mechanisms, the different clinical presentations, and the treatments proposed for demodicosis, including laser and intense pulsed light therapies.

Materials and methods

We conducted a literature search was in the PubMed database. The search period ranged from January 2000 through September 2025. The following search formula was used: “Demodicosis [Title/Abstract] OR Demodicidosis [Title/Abstract]”. The inclusion criteria were clinical studies or review articles evaluating pharmacological treatments or light-source-based treatments for demodicosis, or diseases related to *Demodex*, provided they analyzed the reduction in the mite population after the intervention. The exclusion criteria were basic or preclinical research, studies not performed in humans, articles not written in English or Spanish, and publications whose full text was not available.

The certainty of the evidence was assessed according to the Grading of Recommendations Assessment, Development and Evaluation (GRADE) system guidelines, using the online GRADEpro tool.[5] The quality of evidence was classified as very low, low, moderate, or high.

Pathogenesis

Demodex infestation in the pilosebaceous unit is usually asymptomatic in most individuals. However, certain factors may trigger excessive proliferation and transform this colonization into a pathological condition, altering the balance between the skin microenvironment, the cutaneous flora, and the human immune system.[2] These factors include damage to the immune system, infections such as HIV, prolonged use of corticosteroids, and damage to the skin microbiome. In addition, a higher prevalence of diseases caused by *Demodex* has been observed in patients with obesity, kidney failure, diabetes mellitus,[2] use of bio-

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logic drugs for atopic dermatitis, such as dupilumab,[6] and HLA-Cw2 and HLA-Cw4 haplotypes.[7]

The pathogenesis of demodicosis involves several mechanisms, such as blockage of follicles and sebaceous ducts by the mite itself and its role as a vector for transmitting bacteria such as *Bacillus oleronius*, which intensifies the inflammatory response. Some studies have suggested that *Demodex* can evade the immune response by inhibiting neutrophil phagocytic activity. In addition, hyperstimulation of humoral and innate immunity may be triggered in response to increased mite density per follicular unit or to mite penetration into the dermis, culminating in perifollicular inflammation, destruction of the follicular epithelium, reactive hyperkeratosis, and alteration of the skin barrier. Granulomatous reactions may also occur in response to mite particles. In cases of massive infestation, dead mites release chitinous exoskeletons and bacterial antigens, which may increase TLR-2 expression, triggering an inflammatory cascade and a more intense immune response.[1,2]

Clinical signs

The clinical signs of demodicosis are varied and depend on mite density, the host immune response, and other factors. It usually presents in individuals >40 years, with periorificial facial involvement and asymmetric distribution, with minimal associated symptoms, such as pruritus, or no symptoms.[4]

It can be associated with prolonged previous use of topical corticosteroid therapy and may worsen with topical calcineurin inhibitors, such as pimecrolimus or tacrolimus, as well as with occlusive makeup.

There is controversy surrounding the diagnostic boundaries of demodicosis. Some authors propose integrating certain forms of rosacea, such as erythematotelangiectatic and papulopustular rosacea, and demodicosis as different phenotypes or stages of the same entity, in which *Demodex* plays an essential role and shared inflammatory mechanisms are present.[8–10] In this review, however, we present the classification proposed by Chen et al., which distinguishes the following clinical forms[4] (Table 1):

- *Pityriasis folliculorum or spinulate demodicosis* (Fig. 1). This form predominantly affects women and presents as mild diffuse facial erythema, pruritus, burning sensation, fine follicular scales, and follicular plugs. It may be associated with poor hygiene and excessive application of cosmetics.[1]
- *Rosacea-like demodicidosis or Demodex folliculitis* (Figs. 2 and 3). This form is characterized by erythema, scaling, and papulopustules similar to rosacea.[3] It may be subclassified as papulopustular, nodulocystic, or conglobate. As mentioned above, papulopustular rosacea and rosacea-like demodicosis share many clinical features,[8] such as a higher density of *Demodex* compared with healthy skin. Nevertheless, demodicosis is usually associated with follicular scaling, more superficial involvement, occasionally sudden onset and rapid progression, and absence of symptoms such as flushing and photosensitivity.
- *Demodicidosis gravis*. Similar to severe granulomatous rosacea, with formation of granulomas containing phagocytosed *Demodex* remnants and caseous necrosis.[1]
- *Ocular demodicosis*. At the ocular level, it has different forms of presentation, most notably chronic blepharitis, chalazion, or, less frequently, keratoconjunctivitis.[4,11]

Other primary forms of demodicosis include perioral demodicosis, scalp demodicosis – mostly in older individuals with advanced alopecia – crusted demodicosis, and auricular demodicosis, affecting the external auditory canal or the external tympanic membrane.[4]

When the increase in the number of *Demodex* mites is associated with systemic diseases, such as immunosuppression[12] due to HIV or transplantation, chronic kidney disease, and so forth; previous treatments, such as EGFR inhibitors or phototherapy; skin tumors, such as melanocytic nevi, basal cell carcinoma, or mycosis fungoides; or other dermatoses, such as acne, papulopustular rosacea, seborrheic dermatitis, perioral dermatitis, and androgenetic alopecia,[13] it is classified as secondary demodicosis.[4,14]

Diagnosis

The diagnosis of demodicosis is established by identifying an abnormally high density of *Demodex* mites (Dd; >5 mites/cm^2), together with compatible clinical findings and a favorable response to treatment, with subsequent eradication of the mites.[15]

The diagnostic methods most widely used to quantify the presence of *Demodex* are standardized skin surface biopsy (SSSB) and direct microscopic examination (DME).[16] SSSB involves the use of cyanoacrylate glue on a glass slide that is adhered to lesional skin, whereas DME is performed using a comedone extractor and examining the extracted material under the microscope.[1,2] Because most mites are located deep within the hair follicles, SSSB can be performed consecutively on occasions to reach greater depth, thereby allowing a larger number of mites to be obtained.[2] In that case, the cutoff is still considered to be Dd >5/cm^2 in the first biopsy, but Dd >10/cm^2 in the second.[17] However, in routine practice, diagnosis is primarily clinical, and biopsies and histological studies are usually not necessary.

In addition, dermoscopy may be useful, as *Demodex* tails can be observed, visible as whitish filaments measuring 1–3 mm that protrude from the follicle, as well as follicular openings infiltrated with yellowish amorphous material, indicating the presence of mites.[15] The use of ex vivo dermoscopy has also been described to facilitate visualization.[18]

On the other hand, advances in diagnostic techniques, such as *Demodex* DNA analysis by next-generation sequencing (NGS) or confocal microscopy, are being evaluated with promising results.[19–21]

Differential diagnosis of demodicosis includes conditions such as papulopustular rosacea, erythematotelangiectatic rosacea, cutaneous lupus erythematosus, seborrheic dermatitis, folliculitis, perioral dermatitis, contact dermatitis, and acne. Occasionally, demodicosis may present as a localized facial plaque that can be confused with actinic damage, actinic keratoses, among others. In many of the entities included in the differential diagnosis, abnormally high *Demodex* densities have been demonstrated – papulopustular and erythematotelangiectatic rosacea, seborrheic dermatitis, periorificial dermatitis, acne, blepharitis, and Meibomian gland dysfunction.[7–9,12,20,24–26] Nevertheless, a high *Demodex* density is not by itself diagnostic of demodicosis and should therefore be interpreted together with the clinical findings, dermoscopy, and response to treatment in order to guide the diagnosis appropriately.

Treatment

Treatment of demodicosis is mainly based on reducing the density of *Demodex* mites on the skin and improving the associated inflammatory symptoms. Favorable responses have been reported with both various pharmacological treatments and light sources.

Pharmacological treatment

Pharmacological treatments for demodicosis are aimed at reducing the *Demodex* mite population and decreasing associated skin symptoms.[4,14,22]

Topical treatments (Table 2)

These include salicylic acid, selenium, metronidazole, benzyl benzoate, crotamiton, lindane, sulfide, sulfur, tea tree oil, permethrin, retinoids, and topical ivermectin.[1,7,21–29] A meta-analysis of 5 clinical studies (n = 180)[27] reported a mean decrease of 70.01 mites/cm^2 and an 80% reduction in the number of *Demodex*-positive patients (≥5 mites/cm^2) after 16 weeks of treatment with 1% topical ivermectin,

Table 1
Clinical forms of demodicosis according to the classification by Chen et al.[4]

Classification	Complementary nomenclature	Clinical description
Primary demodicosis		
Pityriasis folliculorum	Spinulate demodicosis	Predominantly affects women and presents with mild diffuse facial erythema, pruritus, burning sensation, fine follicular scales, and follicular plugs. It may be associated with poor hygiene and excessive use of occlusive cosmetics.
Rosacea-like demodicosis	Papulopustular demodicosis, nodulocystic demodicosis, conglobate demodicosis	Characterized by erythema, scaling, and rosacea-like papulopustules, and may present as nodulocystic forms. No flushing.
Demodicidosis gravis	Granulomatous demodicosis	Similar to severe granulomatous rosacea, with formation of granulomas containing phagocytosed *Demodex* remnants and caseous necrosis.
Ocular demodicosis		Presents as chronic blepharitis, chalazion, or keratoconjunctivitis.
Other primary demodicoses	Perioral demodicosis, scalp demodicosis, auricular demodicosis	
Secondary demodicosis		Associated with systemic diseases – immunosuppression, HIV, transplantation, chronic kidney disease – treatments – EGFR inhibitors, phototherapy – or pre-existing dermatoses – acne, rosacea, seborrheic dermatitis.

EGFR, epidermal growth factor receptor.

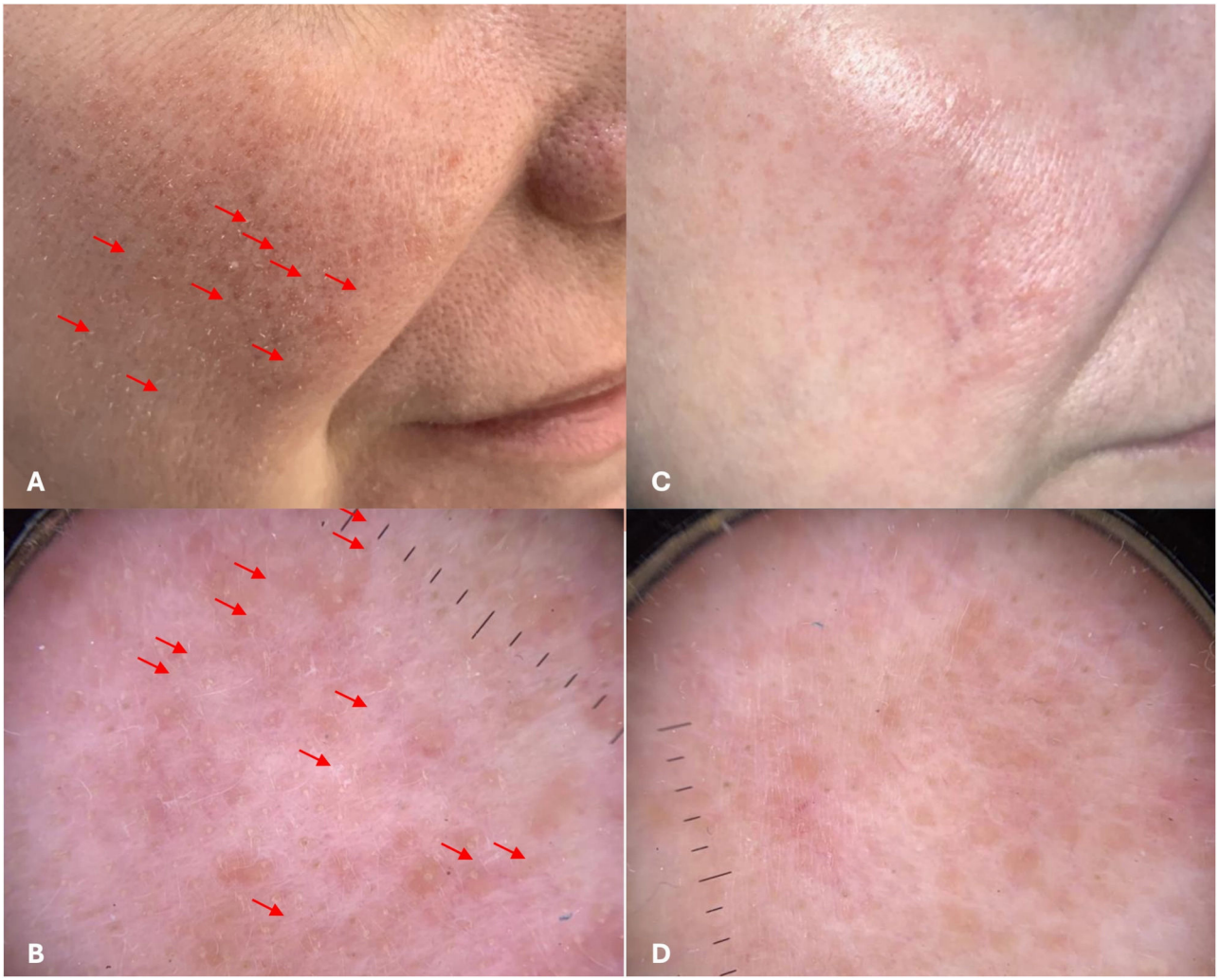

Fig. 1. Spinulate demodicosis. (A and B) Papules with fine scaling, with *Demodex* tails visible on dermoscopy in follicular openings. (C and D) Therapeutic response after 1% ivermectin cream for 6 weeks and one PDL laser session – parameters: 10 mm, 0.5 ms, 6–7 J/cm^2.

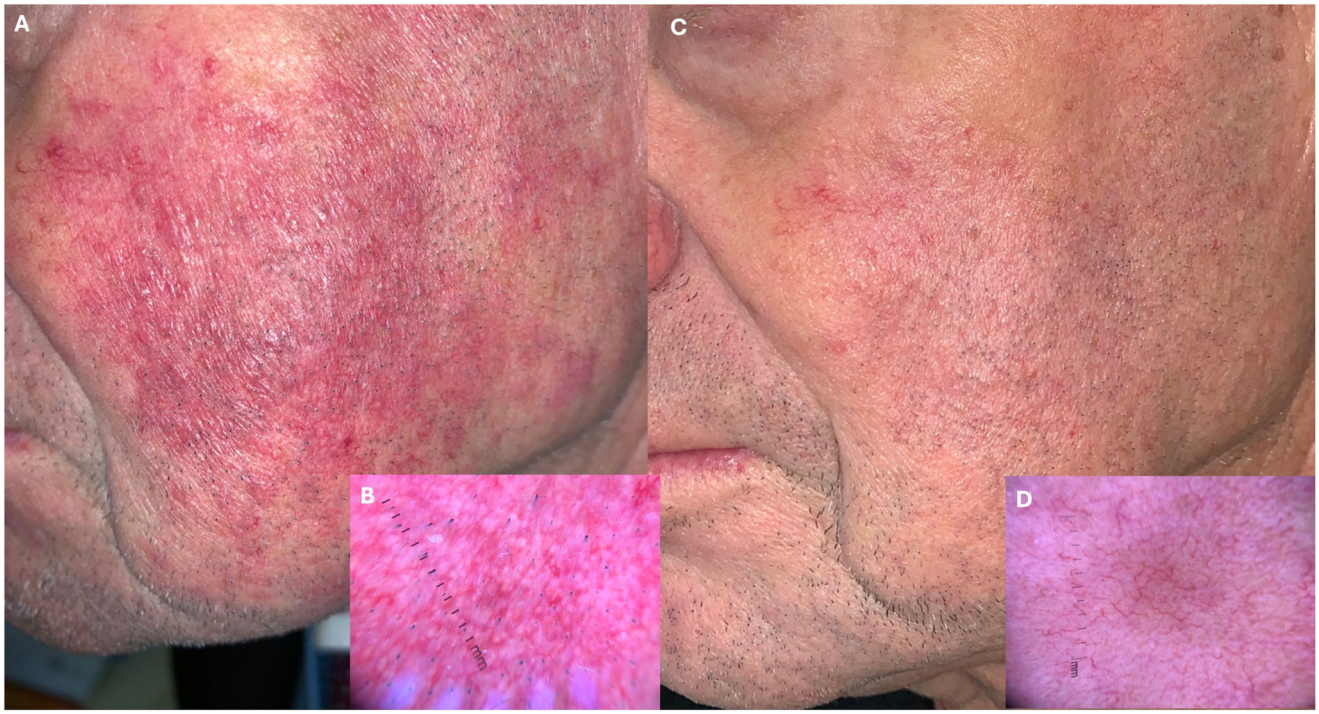

Fig. 2. Papulopustular rosacea-like demodicosis. (A and B) Papulopustular rosacea-like demodicosis on the cheeks. Dermoscopy shows *Demodex* tails in follicular openings with the presence of an isolated pustule. (C and D) Therapeutic response after 1% ivermectin cream for 6 weeks and one PDL laser session – parameters: 10 mm, 0.5 ms, 6–7 J/cm^2.

Fig. 3. Pustulocystic rosacea-like demodicosis. (A and B) Pustulocystic rosacea-like demodicosis on the cheeks and forehead. (B) Therapeutic response after isotretinoin 5 mg/day for 2 months and one PDL laser session – parameters: 10 mm, 0.5 ms, 6–7 J/cm^2.

Table 2
Topical treatment for demodicosis.

Drug	Mechanism of action	Dose	Dosage regimen	Level of evidence
Metronidazole[25]	Reduces the number of *Demodex* mites. Anti-inflammatory properties through inhibition of ROS release in neutrophils.	0.75%–2% gel or cream	Apply 1–2 times daily for 3–270 days	Low
Ivermectin[27,28]	Antiparasitic; reduces the number of *Demodex* mites and inflammation.	1% cream	Apply once daily for 8–12 weeks	Moderate
Permethrin[25]	Insecticide; interferes with sodium channels in parasites.	1%–5% cream or lotion	Apply once daily for 3–12 weeks	Low
Crotamiton[26]	Antipruritic and anti-inflammatory. Reduces the *Demodex* population.	10% cream or lotion	Apply once daily for 3–45 days	Low
Tea tree oil[32,29]	Anti-inflammatory, antiparasitic, antibacterial, antifungal, and antiviral. Anti-inflammatory and re-epithelializing properties.	50% tea tree oil solution	Apply once weekly for 4–6 weeks	Moderate
Benzyl benzoate[23]	Neurotoxic to mites.	10%–25% cream or lotion	Apply twice daily for 45 days*	Low

ROS, reactive oxygen species.
* Used in combination with crotamiton.

with good tolerability. Another meta-analysis highlighted ivermectin, both topical and systemic, the combination of topical ivermectin and metronidazole, and tea tree oil as the treatments with the best combination of efficacy and favorable safety profile.[30]

In the case of *Demodex* blepharitis, in addition to tea tree oil, which seems to be the most effective treatment in a meta-analysis published in 2020,[31,32] lotilaner ophthalmic solution (0.25%) is an ectoparasiticide that represents an effective therapeutic alternative with less local irritation.[33] However, there are no standardized clinical practice guidelines on the therapeutic regimen or dosage.[14] More recently, a multicenter retrospective study of 2157 patients treated with topical ivermectin in a 1% compounded ointment, applied once daily for 2 months, showed a significant reduction in the degree of cylindrical sleeves, or collarettes, conjunctival erythema, and Ocular Surface Disease Index (OSDI) score, with good tolerability, after a mean follow-up of 26 months.[28]

Systemic treatments (Table 3)

In severe cases or in immunocompromised patients, the use of oral metronidazole or ivermectin may be considered.[1,12,22] However, evidence on the efficacy of oral metronidazole in demodicosis is limited and heterogeneous. Some studies have shown significant clinical improvement, possibly due to an anti-inflammatory effect, without a parallel reduction in the *Demodex* population, whereas others describe a rapid and sustained response with regimens of 250 mg 3 times daily for 2 weeks.[34,35] Recently, in a prospective study including 40 patients with facial demodicosis, after administration of oral ivermectin (200 μg/kg/week) for 8 weeks, 75% of patients achieved clinical and acarological remission, with no relevant adverse effects.[36] A randomized clinical trial including 120 patients showed superior *Demodex* clearance with combined ivermectin and metronidazole compared with ivermectin monotherapy.[35]

Furthermore, isotretinoin may be used as an adjunct to reduce sebum production and, consequently, *Demodex* density.[37] In a rare case of ivermectin-refractory demodicosis, complete clinical resolution and mite eradication were observed with combined treatment using low-dose isotretinoin (0.1 mg/kg 4 times weekly) and 1% permethrin cream.[38]

Laser and other light sources (Table 4)

Various light sources, including vascular lasers, IPL, photodynamic therapy, and photobiomodulation, have been evaluated in facial and ocular demodicosis. Their role appears to be due both to a possible thermal acaricidal effect and to anti-inflammatory mechanisms and improvement of glandular function.

Facial demodicosis

Pulsed dye laser (PDL) (Figs. 1–3). The pulsed dye laser (PDL) emits a beam of light with a wavelength of 585–595 nm, which can be absorbed by oxyhemoglobin, which is why it is widely used for the treatment of erythematotelangiectatic rosacea.

In a study of 31 patients diagnosed with rosacea, *Demodex* density (Dd) decreased significantly after a single PDL treatment session (585 nm in vascular mode; fluence, 4–6 J/cm^2; spot size, 5 mm; pulse duration, 0.45 ms; frequency, 1–2 Hz; maximum overlap, 30%): baseline Dd was 13.0 and post-treatment Dd was 6.0 (P = .002). The study did not evaluate whether the decrease in Dd was associated with clinical improvement.[39]

Yellow laser (577-nm pro-yellow laser). The 577-nm yellow laser can be used for the treatment of certain vascular skin lesions and malformations. A retrospective study (n = 34) showed that treatment with a 577-nm laser in scanner mode – parameters of 20 J/cm^2, 42–46 ms, 1.5 s, and 80% coverage – significantly reduced *Demodex* density (baseline Dd, 18.1 ± 10.7/cm^2; post-treatment Dd, 10.2 ± 7.9/cm^2 after 4 weeks; P = .001). Both clinical improvement, measured using the VAS, and the decrease in *Demodex* density were significant. However, no direct correlation was observed between mite reduction and clinical improvement.[40]

Nd:YAG laser. The 1064-nm Nd:YAG laser is mainly used for the treatment of deep vascular lesions and pigmented lesions. In one reported case, a patient with erythematotelangiectatic rosacea experienced an 11.6% to 28.1% reduction in mite density after a single Nd:YAG laser treatment session, with a fluence of 300 J/cm^2, spot size of 1.5 mm, pulse duration of 12 ms, and frequency of 1 Hz. This was associated with moderate clinical improvement in erythema 1 month after the session.[41]

Table 3
Systemic treatment for demodicosis.

Drug	Mechanism of action	Dose	Dosage regimen	Level of evidence
Metronidazole[35]	Anti-inflammatory and antiparasitic; reduces ROS.	250 mg orally	3 times daily for 1–8 weeks	Very low
Ivermectin[36]	Reduces the number of *Demodex* mites and inflammation.	200–250 μg/kg orally	Two doses, 1–2 weeks apart	Moderate
Isotretinoin[37]	Decreases sebum production, reducing mite proliferation.	0.1 mg/kg, low dose	20 mg/day	Low

ROS, reactive oxygen species.

Table 4
Treatment with laser and other light sources for demodicosis.

Treatment	Indication	Parameters	Treatment regimen	No. of studies (No. of patients)	Clinical response	Response in *Demodex* population
Intense pulsed light (IPL)[47–50]	Ocular demodicosis/Meibomian gland dysfunction/blepharitis	500–1200 nm, 9–20 J/cm^2	1–4 sessions, every 2–4 wk	6 (329)	Significant symptomatic improvement – decrease in erythema, eyelid pruritus, and ocular symptom VAS score.	Mean decrease in Dd of 28.2 mites/cm^2 (95%CI, −53.6 to −2.8); eradication RR, 1.41 (1.18–1.69).
Pulsed dye laser (PDL)[39]	Facial rosacea/rosacea-like demodicosis	Fluence, 4–6 J/cm^2; frequency, 1–2 Hz; spot size, 5 mm; pulse duration, 0.45 ms	1 session	1 (31)	–	Decrease in median Dd: 54% at 3 weeks.
Yellow laser (577 nm)[40]	Facial rosacea/rosacea-like demodicosis	Fluence, 20 J/cm^2; pulse duration, 42–46 ms; 80% coverage	1 session	1 (34)	Mean clinical improvement VAS: 6.3/10.	Mean decrease in Dd of 44%.
Nd:YAG laser[41]	Facial telangiectasias/facial erythema	Fluence, 300 J/cm^2; spot size, 1.5 mm; pulse duration, 12 ms; frequency, 1 Hz	1 session	1 (1)	Decrease of 33% in clinical erythema and 67% in telangiectasia grade.	Decrease of 12%–28% in Dd.
KTP 532-nm laser	Facial demodicosis	Fluence, 8–10 J/cm^2; spot size, 10–12 mm; pulse duration, 10 ms	1 session	0 (0)	–	–
Photodynamic therapy (ALA-PDT)[42,43]	Papulopustular rosacea/rosacea-like demodicosis	Topical 5% ALA; red LED light, 630–635 nm; fluence, 37–50 J/cm^2	A total of 3–5 sessions. Frequency: every 2–4 wk	2 (80)	Reduction in inflammatory lesions of 70%–80%; improvement in erythema up to 58%; good/excellent overall responses in 78%.	Significant decrease in Dd (70%; $P < .001$).
Photobiomodulation[51]	Meibomian gland dysfunction	Continuous red LED, 630 nm, 100 mW/cm^2, 15 min	4 sessions every 2 wk	1 (24)	Significant improvement in OSDI (56%) and Meibomian gland function.	No significant changes.

RR, relative risk; Dd, *Demodex* density; OSDI, Ocular Surface Disease Index; VAS, Visual Analog Scale.
Studies of combined light sources are not included in the table.

KTP 532-nm laser. In our experience, the KTP 532-nm laser – parameters: 10 mm, 10 ms, 7–10 J/cm^2 – has proven effective in reducing *Demodex* density, with associated clinical improvement (Fig. 4).

Other light sources. Photodynamic therapy (PDT) has been used in the context of papulopustular rosacea and rosacea-like demodicosis. In a single-center comparative study, treatment with ALA-PDT (3–5 sessions) was noninferior to oral minocycline 100 mg for 8 weeks in reducing papulopustules and improving quality of life, although it was less effective in controlling erythema (35% vs 67%). *Demodex* density and relapse rates were similar in both groups after 24 weeks of follow-up.[42] Other open-label studies have confirmed sustained clinical improvement at 12–24 weeks with ALA-PDT, associated with mild and transient adverse effects, including pain, erythema, and postinflammatory hyperpigmentation.[43]

The use of phototherapy is not recommended as a therapeutic tool for the treatment of demodicosis. Whereas some studies have failed to find significant differences in *Demodex* density or in the prevalence of facial demodicosis in patients treated with narrowband UVB (NB-UVB) or UVA-1,[44] a cross-sectional study of 45 patients treated with phototherapy (PUVA or NB-UVB) observed a significantly higher prevalence of demodicosis in patients treated with phototherapy (28.9%) than in con-

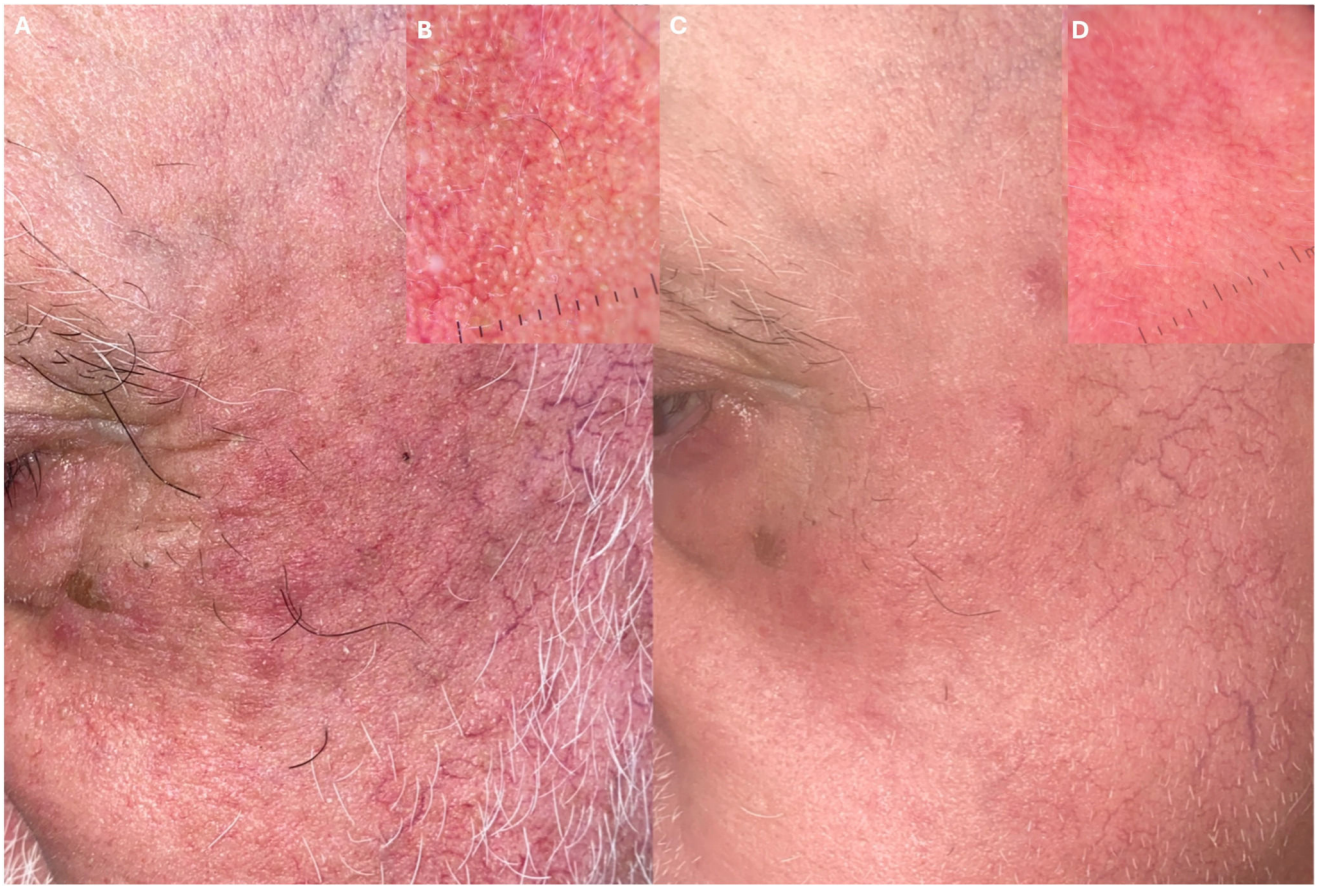

Fig. 4. Plaque demodicosis. (A and B) Asymmetric plaque demodicosis with fine scaling and underlying telangiectasias. Dermoscopy shows *Demodex* tails in follicular openings. (C and D) Therapeutic response after 1 session of KTP 532-nm laser – parameters: 10 mm, 10 ms, 7–10 J/cm^2.

trols (7%) (P = .01), being especially high among those treated with PUVA (58.3%). The authors attributed this increase to the immunosuppressive effects of UV radiation and to induced sebaceous hyperplasia.[45]

We did not find any studies evaluating the response to intense pulsed light (IPL) or photobiomodulation in facial demodicosis.

Ocular demodicosis

Intense pulsed light (IPL). Intense pulsed light (IPL) uses a range of visible-light wavelengths that can be absorbed by different chromophores located in the skin and transformed into heat energy in the treated areas. Several mechanisms have been proposed by which IPL could target *Demodex* mites, including coagulative necrosis and reproductive suppression.[46–49]

A recent meta-analysis including 6 studies – 5 randomized clinical trials and 1 nonrandomized study – with a total of 329 patients analyzed the efficacy and safety of IPL alone or combined with glandular massage or tea tree oil. The fluences used ranged from 9 to 20 J/cm^2, with 1–4 sessions separated by intervals of 2–4 weeks. Treatment was associated with a higher *Demodex* eradication rate compared with controls (RR, 1.41; 95%CI, 1.18–1.69; P < .01) and with a significant reduction in mite density, with a pooled mean difference of −28.2 mites/cm^2 (95%CI, −53.6 to −2.8). Clinical improvement was also significant in most studies, with decreases in pruritus, eyelid erythema, and inflammatory ocular signs. No serious adverse effects were reported, and only 3% of patients reported a mild, transient burning sensation.[50]

Other light sources. A randomized trial of 24 patients assessed the efficacy of photobiomodulation – continuous red LED, 630 nm, 100 mW/cm^2, 15 min – in patients with Meibomian gland dysfunction, either alone or in combination with intense pulsed light (IPL, 580–1200 nm, 12–14 J/cm^2). Both groups showed significant symptom improvement after 4 sessions, although the reduction in *Demodex* did not reach statistical significance.[51] Another retrospective study also evaluated the combination of IPL with photobiomodulation – blue and red light using LED masks, 12 min per color, mean of 7.6 sessions – in 17 patients with *Demodex*-associated blepharitis. Significant improvement was observed in clinical signs, including reduction of cylindrical sleeves, or collarettes, saponification, conjunctival hyperemia, corneal staining, and dry-eye symptoms. *Demodex* density was not quantified.[52]

No studies were found on other light sources – lasers, phototherapy, or photodynamic therapy – used in ocular demodicosis.

Discussion

Demodicosis is a cutaneous and ocular disease caused by overpopulation of the *Demodex* mite, which induces a local inflammatory response in the pilosebaceous unit or eyelid margin. The therapeutic arsenal includes topical or systemic acaricidal treatments, drugs aimed at sebaceous control, such as isotretinoin, and, in recent years, several light sources that have shown potential both in reducing parasitic burden and in controlling associated inflammation.

The light-based therapies evaluated include vascular lasers – PDL, KTP, yellow laser, and Nd:YAG – intense pulsed light (IPL), photodynamic therapy (ALA-PDT), and photobiomodulation. Overall, the evaluated studies describe significant clinical improvement and a reduction in *Demodex* density in most cases treated with laser or IPL, usually after one or a few sessions. In addition, in our experience, KTP and PDL vascular lasers may be particularly useful, especially when combined with topical ivermectin. In many cases, a single session is sufficient to observe clear clinical improvement.

At the ocular level, IPL shows ocular eradication rates >80% in *Demodex*-associated blepharitis, with parallel improvement in Meibomian gland function and ocular symptoms. However, as occurs with other light-based therapies in dermatology, the methodological quality of the evidence is limited: retrospective studies predominate, with small sample sizes and follow-up periods shorter than 6 months, and with no data on recurrence or duration of effect.

Regarding other light sources, photodynamic therapy (ALA-PDT) and photobiomodulation appear to act mainly through anti-inflammatory mechanisms, with good clinical results but without a demonstrated acaricidal effect.

The main advantages of light sources are their safety, low rate of adverse effects, and the possibility of considering them as an alternative in patients in whom systemic treatments are contraindicated – for example, during pregnancy or breastfeeding – provided that their use is individualized. Conversely, their limitations are relevant: costly equipment, low availability, and still limited evidence.

Ultimately, light-based therapies open a promising therapeutic pathway in the comprehensive management of demodicosis, but their role should be defined as higher-quality evidence and longer follow-up become available.

Conclusions

Demodicosis is a common entity in dermatological and ophthalmological practice. It is difficult to diagnose because of clinical overlap with other similar diseases and has multiple therapeutic options.

Topical and systemic acaricidal treatments, especially ivermectin, constitute one of the main pillars of current treatment. However, light-source-based therapies – including vascular lasers, IPL, and ALA-PDT – represent safe and potentially effective alternatives, with results in some studies comparable to topical or oral pharmacotherapy.

The available evidence remains limited and heterogeneous, supported by small series and short follow-up periods. Prospective randomized trials with prolonged follow-up and joint assessment of clinical and acarological response will be necessary to define the duration of effects, recurrence rates, and the exact place that each light-based modality should occupy within the therapeutic algorithm for demodicosis.

Conflict of interest

The authors declare no conflict of interest.

References

1. Baima B, Sticherling M. Demodicidosis revisited. *Acta Derm Venereol*. 2002;82:3–6, http://dx.doi.org/10.1080/000155502753600795.
2. Paichitrojjana A. *Demodex*: the worst enemies are the ones that used to be friends. *Dermatol Rep*. 2022;14:9339, http://dx.doi.org/10.4081/dr.2022.9339.
3. Zirbs M, Mourantchanian V, Radopoulou E, et al. Demodicidosis associated with psoriasis therapy – two case reports. *Australas J Dermatol*. 2013;54:235–236, http://dx.doi.org/10.1111/ajd.12082.
4. Chen W, Plewig G. Human demodicosis: revisit and a proposed classification. *Br J Dermatol*. 2014;170:1219–1225, http://dx.doi.org/10.1111/bjd.12850.
5. *GRADEpro GDT*: *GRADEpro Guideline Development Tool [Software]*. McMaster University; 2023 [accessed 10 Oct 2024]. Available from: https://gdt.gradepro.org/app.
6. Krakowski AC, Senft SC, Heymann WR. *Demodex folliculitis* and recent dupilumab administration. *Pediatrics*. 2021;147, http://dx.doi.org/10.1542/peds.2020-029520, e2020029520.
7. Akilov OE, Mumcuoglu KY. Association between human demodicosis and HLA class I. *Clin Exp Dermatol*. 2003;28:70–73, http://dx.doi.org/10.1046/j.1365-2230.2003.01173.x.
8. Forton FMN. Rosacea, an infectious disease: why rosacea with papulopustules should be considered a demodicosis. A narrative review. *J Eur Acad Dermatol Venereol*. 2022;36:987–1002, http://dx.doi.org/10.1111/jdv.18049.
9. Forton FMN. The pathogenic role of *Demodex* mites in rosacea: a potential therapeutic target already in erythematotelangiectatic rosacea? *Dermatol Ther (Heidelb)*. 2020;10:1229–1253, http://dx.doi.org/10.1007/s13555-020-00458-9.
10. Forton FMN, De Maertelaer V. Papulopustular rosacea and rosacea-like demodicosis: two phenotypes of the same disease? *J Eur Acad Dermatol Venereol*. 2018;32:1011–1016, http://dx.doi.org/10.1111/jdv.14885.
11. Misra S, Murthy SI, Joseph J. Clinical spectrum in microbiologically proven *Demodex* blepharokeratoconjunctivitis: an observational study. *Indian J Ophthalmol*. 2024;72:1049, http://dx.doi.org/10.4103/IJO.IJO_954_23.
12. Amitay-Laish I, Solomon-Cohen E, Feuerman H, et al. Facial demodicosis in the immunosuppressed state: a retrospective case series from a tertiary referral center. *Int J Dermatol*. 2022;61:1245–1252, http://dx.doi.org/10.1111/ijd.16162.
13. Rather PA, Hassan I. Human Demodex mite: the versatile mite of dermatological importance. *Indian J Dermatol*. 2014;59:60, http://dx.doi.org/10.4103/0019-5154.123498.
14. Chudzicka-Strugała I, Gołębiewska I, Brudecki G, Elamin W, Zwoździak B. Demodicosis in different age groups and alternative treatment options – a review. *J Clin Med*. 2023;12:1649, http://dx.doi.org/10.3390/jcm12041649.
15. Segal R, Mimouni D, Feuerman H, Pagovitz O, David M. Dermoscopy as a diagnostic tool in demodicidosis. *Int J Dermatol*. 2010;49:1018–1023, http://dx.doi.org/10.1111/j.1365-4632.2010.04495.x.
16. Hsu CK, Hsu MML, Lee JYY. Demodicosis a clinicopathological study. *J Am Acad Dermatol*. 2009;60:453–462, http://dx.doi.org/10.1016/j.jaad.2008.10.058.
17. Forton FMN, De Maertelaer V. Two consecutive standardized skin surface biopsies: an improved sampling method to evaluate *Demodex* density as a diagnostic tool for rosacea and demodicosis. *Acta Derm Venereol*. 2017;97:242–248, http://dx.doi.org/10.2340/00015555-2528.
18. González HP, Santas MD, Domper LF, Agud de Dios M, Boixeda P. Ex vivo dermoscopy in demodicosis. *J Am Acad Dermatol*. 2023;88:e127–e128, http://dx.doi.org/10.1016/j.jaad.2021.07.031.
19. Kowalczyk MJ, Derebecka N, Żaba R, et al. Novel *Demodex* detection method involving non-invasive sebum collection and next-generation sequencing. *Postepy Dermatol Alergol*. 2022;39:321–326, http://dx.doi.org/10.5114/ada.2021.106028.
20. Fernandez-Nieto D, Segurado-Miravalles G, Gonzalez-Cantero A, Ortega-Quijano D, Jimenez-Cauhe J, Boixeda P. Fluorescence-advanced videodermatoscopy characterization of *demodex* mites: a comparison with reflectance confocal microscopy. *J Eur Acad Dermatol Venereol*. 2021;35:e215–e216, http://dx.doi.org/10.1111/jdv.16938.
21. Logger JGM, Peppelman M, van Erp PEJ, de Jong EMGJ, Nguyen KP, Driessen RJB. Value of reflectance confocal microscopy for the monitoring of rosacea during treatment with topical ivermectin. *J Dermatolog Treat*. 2022;33:195–203, http://dx.doi.org/10.1080/09546634.2020.1741501.
22. Jacob S, VanDaele MA, Brown JN. Treatment of *Demodex*-associated inflammatory skin conditions: a systematic review. *Dermatol Ther*. 2019;32, http://dx.doi.org/10.1111/dth.13103, e13103.
23. Forton FMN, De Maertelaer V. Treatment of rosacea and demodicosis with benzyl benzoate: effects of different doses on *Demodex* density and clinical symptoms. *J Eur Acad Dermatol Venereol*. 2020;34:365–369, http://dx.doi.org/10.1111/jdv.15938.
24. Trave I, Merlo G, Cozzani E, Parodi A. Real-life experience on effectiveness and tolerability of topical ivermectin in papulopustular rosacea and antiparasitic effect on *Demodex* mites. *Dermatol Ther*. 2019;32, http://dx.doi.org/10.1111/dth.13093, e13093.
25. Koçak M, Yağli S, Vahapoğlu G, Ekşioğlu M. Permethrin 5% cream versus metronidazole 0.75% gel for the treatment of papulopustular rosacea. A randomized double-blind placebo-controlled study. *Dermatology*. 2002;205:265–270, http://dx.doi.org/10.1159/000065849.
26. Sarac G. A comparison of the efficacy and tolerability of topical agents used in facial *Demodex* treatment. *J Cosmet Dermatol*. 2019;18:1784–1787, http://dx.doi.org/10.1111/jocd.12986.
27. Paichitrojjana A, Khuancharee K, Paichitrojjana A. Efficacy of topical ivermectin in controlling human *Demodex* infestation: evidence from systematic review and meta-analysis. *Parasite Epidemiol Control*. 2025;31, http://dx.doi.org/10.1016/j.parepi.2025.e00461, e00461.
28. Valvecchia F, Greco L, Perrone F, et al. Topical ivermectin ointment treatment of *Demodex* blepharitis: a 6-year retrospective study. *Graefes Arch Clin Exp Ophthalmol*. 2024;262:1281–1288, http://dx.doi.org/10.1007/s00417-023-06281-0.
29. Mohammadpour M, Maleki S, Khorrami-Nejad M. The effect of tea tree oil on dry eye treatment after phacoemulsification cataract surgery: a randomized clinical trial. *Eur J Ophthalmol*. 2020;30:1314–1319, http://dx.doi.org/10.1177/1120672119867642.
30. Li J, Wei E, Reisinger A, French LE, Clanner-Engelshofen BM, Reinholz M. Comparison of different anti-*Demodex* strategies: a systematic review and meta-analysis. *Dermatology*. 2023;239:12–31, http://dx.doi.org/10.1159/000526296.
31. Shah PP, Stein RL, Perry HD. Update on the management of *Demodex* blepharitis. *Cornea*. 2022;41:934, http://dx.doi.org/10.1097/ICO.0000000000002911.
32. Lam NSK, Long XX, Li X, Yang L, Griffin RC, Doery JC. Comparison of the efficacy of tea tree (*Melaleuca alternifolia*) oil with other current pharmacological management in human demodicosis: a systematic review. *Parasitology*. 2020;147:1587–1613, http://dx.doi.org/10.1017/S003118202000150X.
33. Talha M, Haris Ali M, Fatima E, Nadeem A, Ahmed A, Nashwan AJ. Efficacy and safety of lotilaner ophthalmic solution (0.25%) for the treatment of *Demodex* blepharitis: a GRADE assessed systematic review and meta-analysis of observational & experimental studies. *Am J Ophthalmol*. 2024;264:8–16, http://dx.doi.org/10.1016/j.ajo.2024.03.019.
34. Schaller M, Sander CA, Plewig G. *Demodex* abscesses: clinical and therapeutic challenges. *J Am Acad Dermatol*. 2003;49(5 suppl):S272–S274, http://dx.doi.org/10.1016/s0190-9622(03)00743-6.
35. Salem DAB, El-Shazly A, Nabih N, El-Bayoumy Y, Saleh S. Evaluation of the efficacy of oral ivermectin in comparison with ivermectin-metronidazole combined therapy in the treatment of ocular and skin lesions of *Demodex folliculorum*. *Int J Infect Dis*. 2013;17:e343–e347, http://dx.doi.org/10.1016/j.ijid.2012.11.022.
36. Paichitrojjana A, Chalermchai T. Evaluating the efficacy of oral ivermectin on clinical symptoms and *Demodex* densities in patients with demodicosis. *Drug Des Devel Ther*. 2024;18:5299–5306, http://dx.doi.org/10.2147/DDDT.S498938.

37. Chakmakchi AMJ, Alatas ET, Yurekli A, Aydoğdu CT, Demir Pektas S. Therapeutic modulation of *Demodex* density via isotretinoin: insights from a prospective dermatological investigation. *J Cosmet Dermatol*. 2025;24, http://dx.doi.org/10.1111/jocd.70249, e70249.
38. Paichitrojjana A, Paichitrojjana A. Successful treatment of ivermectin refractory demodicosis with isotretinoin and permethrin cream. *JAAD Case Rep*. 2022;26:98–100, http://dx.doi.org/10.1016/j.jdcr.2022.06.017.
39. Ertaş R, Yaman O, Akkuş MR, et al. The rapid effect of pulsed dye laser on *demodex* density of facial skin. *J Cosmet Laser Ther*. 2019;21:123–126, http://dx.doi.org/10.1080/14764172.2018.1481509.
40. Temiz SA, Durmaz K, Işık B, Ataseven A, Dursun R. The effect of 577-nm pro-yellow laser on *demodex* density in patients with rosacea. *J Cosmet Dermatol*. 2022;21:242–246, http://dx.doi.org/10.1111/jocd.14085.
41. Yalici-Armagan B, Elcin G. The decrease of *Demodex* density after Nd:YAG laser application for facial telengiactasias: a case report. *Dermatol Ther*. 2020;33, http://dx.doi.org/10.1111/dth.14108, e14108.
42. Yang J, Liu X, Cao Y, et al. 5-Aminolevulinic acid photodynamic therapy versus minocycline for moderate-to-severe rosacea: a single-center, randomized, evaluator-blind controlled study. *J Am Acad Dermatol*. 2023;89:711–718, http://dx.doi.org/10.1016/j.jaad.2023.06.027.
43. Fan L, Yin R, Lan T, Hamblin MR. Photodynamic therapy for rosacea in Chinese patients. *Photodiagn Photodyn Ther*. 2018;24:82–87, http://dx.doi.org/10.1016/j.pdpdt.2018.08.005.
44. Aksoy H, Aslan Kayıran M, Dede Y, Altıntaş Kakşi S, Erdemir VA. The effect of phototherapy on *Demodex* density: a case–control study. *Int J Dermatol*. 2024;63:201–206, http://dx.doi.org/10.1111/ijd.17002.
45. Kulac M, Ciftci IH, Karaca S, Cetinkaya Z. Clinical importance of *Demodex folliculorum* in patients receiving phototherapy. *Int J Dermatol*. 2008;47:72–77, http://dx.doi.org/10.1111/j.1365-4632.2007.03336.x.
46. Fishman HA, Periman LM, Shah AA. Real-time video microscopy of in vitro *Demodex* death by intense pulsed light. *Photobiomodul Photomed Laser Surg*. 2020;38:472–476, http://dx.doi.org/10.1089/photob.2019.4737.
47. Zhang X, Song N, Gong L. Therapeutic effect of intense pulsed light on ocular demodicosis. *Curr Eye Res*. 2019;44:250–256, http://dx.doi.org/10.1080/02713683.2018.1536217.
48. Huo Y, Mo Y, Wu Y, Fang F, Jin X. Therapeutic effect of intense pulsed light with optimal pulse technology on meibomian gland dysfunction with and without ocular *Demodex* infestation. *Ann Transl Med*. 2021;9:238, http://dx.doi.org/10.21037/atm-20-1745.
49. Cheng SN, Jiang FG, Chen H, Gao H, Huang YK. Intense pulsed light therapy for patients with meibomian gland dysfunction and ocular *Demodex* infestation. *Curr Med Sci*. 2019;39:800–809, http://dx.doi.org/10.1007/s11596-019-2108-1.
50. Liu HM, Su S, Liou CM, Chuang AYC. Therapeutic effect of intense pulsed light in ocular demodicosis: a systematic review and meta-analysis. *Cont Lens Anterior Eye*. 2025;48:102477, http://dx.doi.org/10.1016/j.clae.2025.102477.
51. Chiang JCB, Dremin V, Semp DA, et al. Low-level light therapy alone versus combination therapy with intense pulsed light in the treatment of dry eye disease with meibomian gland dysfunction: a randomised paired-eye and mechanism of action trial. *Cont Lens Anterior Eye*. 2025;48:102456, http://dx.doi.org/10.1016/j.clae.2025.102456.
52. Farrant S, Giannaccare G, Lim CHL, Coco G. Intense pulsed light combined with low level blue and red light therapy for *Demodex*-associated blepharitis. *Clin Ophthalmol*. 2025;19:2575–2585, http://dx.doi.org/10.2147/OPTH.S521989.