

Matching rosacea therapy to dominant features: A phenotype-led approach

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Rosacea, a chronic inflammatory skin condition, presents a significant challenge for clinicians due to its varied manifestations. Patients often exhibit a complex relationship of erythema, telangiectasias, papules, and pustules, making a one-size-fits-all treatment strategy largely ineffective. The prevailing clinical dilemma centers on how to move beyond a symptomatic approach to a more precise, phenotype-driven therapeutic selection.

A deeper understanding of rosacea's diverse clinical presentations is essential for tailoring interventions. This approach aims to match specific therapies to the most prominent and bothersome features, thereby improving efficacy and patient satisfaction.

KEY TAKEAWAYS

- **The Pivot:** Rosacea treatment should move from a general symptomatic approach to one that specifically targets the patient's dominant clinical phenotype.
- **The Data:** No specific numeric data is available for this general topic.
- **The Action:** Clinicians should thoroughly assess each rosacea patient to identify their primary phenotypic expression before initiating therapy.

Rosacea is a common, chronic inflammatory dermatosis primarily affecting the central face. Its pathogenesis is complex and multifactorial, involving neurovascular dysregulation, innate immune system dysfunction, and environmental triggers. The condition is characterized by transient or persistent erythema, telangiectasias, papules, pustules, and in some cases, phymatous changes. This heterogeneity in presentation means that a single therapeutic agent rarely addresses all aspects of the disease effectively, necessitating a more precise approach to management.

Historically, rosacea was classified into four subtypes: erythematotelangiectatic rosacea (ETR), papulopustular rosacea (PPR), phymatous rosacea, and ocular rosacea. But this classification system, while useful for initial diagnosis, often failed to guide precise treatment selection because patients frequently present with overlapping features or evolve from one subtype to another. The shift towards a phenotype-led approach acknowledges this clinical reality, focusing instead on the most prominent and bothersome signs and symptoms that drive a patient's presentation. This allows for a more targeted and potentially more effective therapeutic strategy.

Understanding the Phenotypes

The phenotype-led approach categorizes rosacea based on its dominant clinical features rather than rigid subtypes. This means identifying whether a patient's primary concern is persistent facial erythema, inflammatory papules and pustules, prominent telangiectasias, or phymatous changes. Each of these dominant features has distinct underlying

pathophysiological mechanisms, which in turn dictate the most appropriate therapeutic interventions. For instance, neurovascular dysregulation is central to persistent erythema, while an exaggerated innate immune response and microbial factors contribute significantly to papulopustular lesions.

Persistent facial erythema, often the earliest and most common sign of rosacea, is characterized by flushing and redness that can become permanent. This phenotype is largely driven by vasodilation and increased vascular reactivity. Patients with this presentation frequently report triggers such as hot beverages, spicy foods, alcohol, sun exposure, and emotional stress, all of which exacerbate vasodilation. The erythema can significantly impact quality of life, leading to self-consciousness and social anxiety. Effective management focuses on reducing vascular reactivity and vasoconstriction.

Papulopustular rosacea, on the other hand, mimics acne vulgaris but lacks comedones. It presents with inflammatory papules and pustules, often on an erythematous background. The pathogenesis here involves dysregulation of the innate immune system, particularly the toll-like receptor 2 (TLR2) pathway, leading to increased expression of cathelicidin, an antimicrobial peptide with pro-inflammatory properties. Demodex mites, while commensal, are also implicated, with higher densities found in affected skin. Treatment for this phenotype targets inflammation and, in some cases, microbial load.

Telangiectasias, visible small blood vessels, are a common feature of rosacea, particularly in the erythematotelangiectatic form. These are a consequence of chronic vasodilation and vascular damage. While not directly inflammatory, they contribute significantly to the cosmetic burden of the disease. Standard medical therapies often have limited impact on established telangiectasias, requiring different modalities for their resolution.

Phymatous changes, most commonly rhinophyma, involve thickening of the skin due to sebaceous gland hyperplasia and fibrosis. This severe form of rosacea predominantly affects men and can lead to significant disfigurement. Its pathogenesis is less understood but involves chronic inflammation and tissue remodeling. Early intervention is essential to prevent progression of disfigurement, though established phymatous changes often require procedural intervention.

Targeting Specific Features

For patients whose dominant feature is persistent facial erythema, topical vasoconstrictors are a primary therapeutic option. These agents work by directly constricting cutaneous blood vessels, thereby reducing redness. Alpha-adrenergic agonists, for example, provide a temporary reduction in erythema. But their effect is transient, and some patients may experience rebound erythema upon discontinuation. Long-term management of erythema also involves strict avoidance of known triggers and the use of broad-spectrum sunscreens, as UV radiation is a significant exacerbating factor. Systemic therapies are generally reserved for more severe cases or those unresponsive to topical treatments, though their role in pure erythema is limited compared to inflammatory phenotypes.

When inflammatory papules and pustules are the predominant concern, topical and oral anti-inflammatory agents are the cornerstone of therapy. Topical metronidazole, azelaic acid, and ivermectin are commonly used. Metronidazole exhibits anti-inflammatory and immunosuppressive properties, while azelaic acid reduces inflammation and has antimicrobial effects. Ivermectin, a newer option, targets Demodex mites and also possesses anti-inflammatory actions. For more widespread or severe papulopustular lesions, oral antibiotics, particularly sub-antimicrobial dose doxycycline, are effective. Doxycycline at these lower doses primarily acts through its anti-inflammatory mechanisms rather than its antimicrobial effects, avoiding the development of antibiotic resistance. Other oral options include macrolides or

isotretinoin in refractory cases, though the latter carries significant side effect risks and requires careful monitoring. For a deeper dive into specific mechanisms, clinicians might find our previous coverage on paroxetine's mechanism in rosacea insightful, even if it focuses on a different drug class.

Telangiectasias, being structural vascular changes, respond poorly to medical therapy. The most effective treatment for these visible blood vessels is light-based therapy, specifically pulsed dye lasers (PDL) or intense pulsed light (IPL). These devices target hemoglobin within the vessels, causing selective photothermolysis and subsequent vessel clearance. Multiple sessions are typically required, and patients should be counselled on realistic expectations and potential side effects such as temporary bruising or hyperpigmentation. This is a purely cosmetic intervention, but one that significantly improves patient quality of life.

Phymatous changes, particularly rhinophyma, often require surgical intervention. Early, mild cases might respond to oral isotretinoin, which can reduce sebaceous gland activity and inflammation. But for established disfigurement, surgical debulking, dermabrasion, or laser resurfacing are necessary to restore normal facial contours. These procedures aim to remove excess tissue and reshape the affected area. The decision on the specific technique depends on the extent and location of the phymatous changes. The Oxford Handbook of Medical Dermatology offers a comprehensive overview of these and other dermatological conditions, providing practical guidance for diagnosis and management.

Integrated Management and Patient Education

A comprehensive management plan for rosacea extends beyond pharmacological interventions to include trigger avoidance and appropriate skincare. Patients should be educated on identifying and minimizing exposure to their personal triggers, which can vary widely. Common triggers include sun exposure, hot or cold weather, wind, strenuous exercise, hot baths, saunas, alcoholic beverages, spicy foods, and certain cosmetic products. Using gentle, non-irritating cleansers and moisturizers is essential to maintain skin barrier function and reduce irritation. Sun protection with broad-spectrum sunscreens (SPF 30 or higher) is non-negotiable for all rosacea patients, regardless of their dominant phenotype, to prevent exacerbations and progression of vascular damage.

The psychological impact of rosacea cannot be overstated. The visible nature of the condition, particularly facial erythema and lesions, can lead to significant emotional distress, anxiety, and depression. Clinicians must address these psychosocial aspects, offering support and, when appropriate, referring patients for psychological counselling. Improving the physical symptoms often leads to an improvement in mental well-being, but sometimes direct intervention is needed. This holistic approach highlights the importance of a strong patient-clinician relationship and shared decision-making in managing a chronic condition like rosacea.

But the lack of a single, universally effective treatment means that managing rosacea often involves a trial-and-error process, adjusting therapies based on patient response and evolving symptoms. The open-label nature of many real-world treatment decisions is an obvious caveat, as formal comparative trials for every phenotype-specific strategy are scarce. The long-term efficacy and safety of some newer topical agents, while showing positive early results, still require more extensive post-marketing surveillance. The role of the microbiome and specific microbial interventions beyond *Demodex* targeting is an area of ongoing research. For instance, understanding the broader skin microbiome could offer new avenues, similar to how dietary interventions impact atopic dermatitis.

The challenge remains in identifying biomarkers that can predict response to specific therapies, moving beyond clinical observation to a more personalized medicine approach. Genetic predispositions and individual variations in immune

response likely play a role in treatment outcomes. Until such biomarkers are routinely available, careful clinical assessment and a flexible, phenotype-led treatment strategy remain the most practical and effective way to manage this complex condition.

CLINICAL IMPLICATIONS

The shift to a phenotype-led approach for rosacea is not just academic; it demands a more rigorous initial assessment from clinicians. Simply diagnosing 'rosacea' is insufficient. We must identify the dominant, most bothersome feature to guide therapy effectively, moving away from a shotgun approach that often leaves patients frustrated and undertreated.

This means a more targeted use of existing agents. Prescribing a vasoconstrictor for purely papulopustular lesions is as illogical as using an anti-inflammatory for established telangiectasias. Resources are finite, and patient adherence hinges on seeing tangible results from their prescribed regimen. A precise diagnosis leads to a precise prescription.

For patients, this approach offers the promise of more effective symptom control and improved quality of life. When therapy directly addresses their primary complaint, whether it is persistent redness or inflammatory bumps, they are more likely to adhere to treatment and report satisfaction. This also reduces the burden of polypharmacy and potential side effects from ineffective treatments.

The industry, in turn, should focus on developing agents that specifically target these distinct phenotypes, rather than broad-spectrum drugs that may dilute efficacy across multiple symptoms. Future research needs to provide clearer guidance on combination therapies and sequential treatment strategies for patients with overlapping or evolving presentations, ensuring we have the tools to match the complexity of the disease.

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