Editorial

Rosacea: Wonderings of a Clinician

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Dermatologists love variety. Rare conditions abound in our specialty. It is exciting to diagnose a patient's disease when only a few cases have been previously reported. However, common problems often are equally as challenging because subtle variations may suggest insights to causation or new therapeutic approaches. Rosacea is a common malady that has escaped pathophysiologic explanation. Part of the confusion lies in the need for precise definitions and clear subset classification. In spite of the guidance of a national expert committee[1] and the subsequent publication of 3 recent reviews,[2-4] much work is needed to discover why the redness occurs and by what mechanisms successful therapeutic interventions work.

I wish to emphasize that the common link of all patients with rosacea is central facial erythema of the convex surfaces.[2] Flushing, telangiectasias, papules, and pustules are variable signs but are not disease defining.

Most hypotheses of the cause of this erythema involve postulations that rosacea is a result of vascular lability. Recurrent extrusion of inflammatory mediators leads to edema of the relatively static portion of the mid face, resulting in matrix and lymphatic damage, which leads to disease exacerbation and progression.[5] This is a reasonable working theory for the explanation of events in the erythematotelangiectatic (ETR) subset of patients who experience recurrent and prolonged flushing. However, how do other patients who do not exhibit vascular lability develop the persistent centrofacial erythema that defines rosacea?

The expert rosacea committee divided patient presentations into 4 subsets: ETR, papulopustular rosacea, phymatous rosacea, and ocular rosacea. I propose that those patients with phymas express only a secondary finding and do not define a subset. I suggest that this subset be replaced by the designation of glandular rosacea, whose male members are particularly predisposed to develop phymas. We all recognize patients who present with central facial erythema whose skin is greasy and sebaceous and who have had lifelong acne, often with cystic lesions. Many of these patients are men with easily recognizable acne scars or women with a predilection for lower face inflammatory lesions. Flushing problems usually are minimal or absent, and sun damage often is not apparent in the patient with glandular rosacea.

I propose that patients with glandular rosacea develop their erythema as a result of recurrent follicular-based inflammation, which leads to lymphatic damage and is exacerbated by subsequent episodes of inflammation, edema, and stagnation of inflammatory mediators. These patients benefit most remarkably to anti-acne preparations; the patients with ETR only express irritation and vascular exacerbation with such interventions.

What about the patients with papular pustular rosacea? Individual patients may express lesser degrees of vascular lability or acne proneness than the pathophysiologically distinct ETR and glandular rosacea subsets. Both processes may be at work in contributing to matrix and lymphatic damage. Alternatively, the matrix and lymphatic damage may be related to chronic solar damage with a particularly inflammatory or particularly aggressive elastic tissue–damaging reaction pattern. I propose that lymphatic failure resulting in subsequent edema and inflammation is the final common pathway leading to central facial erythema.

Regarding flushing, the predilection for the vessels of the face or upper chest to respond to stimuli

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for vasodilation is clearly demonstrated in patients with ETR rosacea but also can be seen in carcinoid, drug-induced flushing, or simple embarrassment. Clearly, these vessels respond to vasodilatory stimuli in a profoundly exaggerated fashion compared with other skin sites. I propose that the cause is a receptor that is either functionally or qualitatively different on these vessels. If investigators could solve the riddle of why our patients flush in a prolonged pattern on their faces to a universally vasodilatory stimuli, such as heat, specific blockers may be synthesized that would provide benefit to not only rosacea patients but also menopausal women and self-conscious patients alike.

Certainly, these suppositions are only the musings of a concerned physician and may prove to be folly after proper investigation. I do hope that my postulations stimulate debate; thoughtful comment; or, at best, serious investigation. It is only with such endeavors that progress will result in the understanding of rosacea and in the development of effective therapeutic alternatives.

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