Diagnosis and Management of Skin Resurfacing–Related Complications

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KEYWORDS

• Chemical peel • Laser resurfacing • Dermabrasion • Laser • Broadband light • Infection • Scar • Skin conditioning

Skin resurfacing, especially minimally invasive skin resurfacing, has gained increasing popularity among physicians and patients because of its rapid recovery, low risks for complications, and gratifying results. Skin resurfacing is a field of rapid evolution, with many new technologies that have developed over the years, and has been used extensively by physicians in a variety of specialties to achieve cosmetic enhancement. Thorough understanding of the anatomy and pathophysiology of skin, proper patient evaluation, appropriate selection of procedures based on different skin types, and underscoring potential complications of each procedure, however, are essential to achieve optimal results and to minimize complications related to skin resurfacing.

Skin serves as a physiologic barrier to prevent fluid loss and to protect the human body from exposure to trauma, ultraviolet radiation, infections, and toxins. Other major functions of the skin include sensory perception, immune recognition, and thermoregulation. The skin is composed of two layers, epidermis and dermis, that overlie the subcutaneous fat (**Fig. 1**). The epidermis is approximately 50 μm in thickness and consists of three major resident cells: keratinocytes, Langerhans' cells, and melanocytes. The melanocytes are capable of producing and transferring melanin. The dermis contains vascular structures and nerves endings in a collagen- and elastin-containing matrix, which provides circulation and nutritional and structural support for the epidermis (see **Fig. 1**). Fibroblasts, macrophages, and dendritic cells are the main resident cells in the dermis. Fibroblasts produce the collagen, elastin, and glycosaminoglycans that constitute the dermal matrix. Epidermal adnexal structures include eccrine glands, apocrine glands, sebaceous glands, and hair follicles. It is these adnexal structures that play an important role in skin reepithelialization when the overlying epidermis is removed by traumatic abrasions, dermabrasion, chemical peeling, or ablative laser skin resurfacing.

COMMON COMPLICATIONS FROM MOST TYPES OF SKIN RESURFACING MODALITIES

Skin resurfacing is accomplished by controlled skin injury to remodel the epidermis or dermis, results in smoothing of surface irregularities, and stimulates new collagen synthesis to achieve an enhanced cosmetic appearance. Many modalities have been used for resurfacing, such as chemical peels, photodynamic therapy (PDT), microdermabrasion, dermabrasion, ablative and nonablative lasers, and fractional lasers. Most resurfacing-related complications are associated with the depth of the wound created rather than the type of modality used. To achieve tightening of the skin, the depth of the wound must approach

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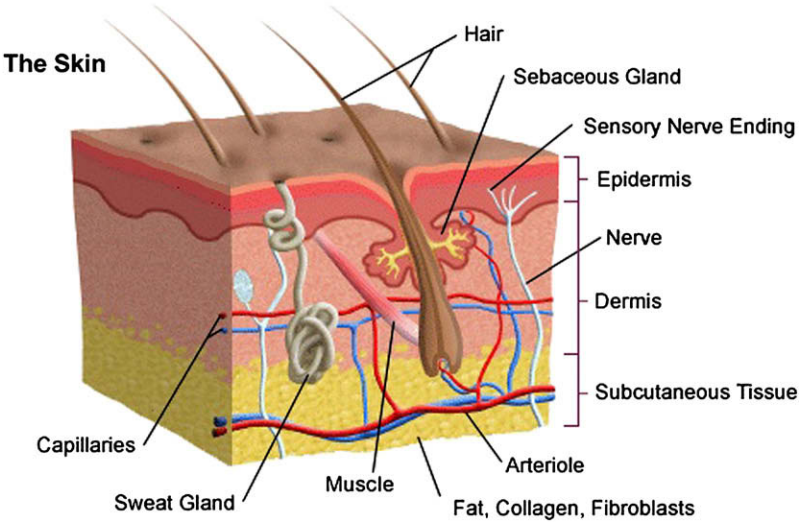

Fig. 1. Skin anatomy diagram showing the epidermis, dermis, and subcutaneous fat. Note that many of the adnexal structures are located in the dermis.

the papillary dermis. To achieve leveling of the skin surface, however, the wound depth must approach the upper reticular dermis. Thus, skin laxity is addressed with tightening levels of skin resurfacing, whereas certain acne scars and deeper rhytides require a leveling depth of wounding. As the depth of wounding exceeds the upper reticular dermis, however, the risks for permanent pigmentary changes, textual changes, and scarring increase significantly. It is essential, therefore, to control the depth of the wounding to obtain the maximal desired clinical effects with minimal complications. Other complications of resurfacing include contact dermatitis, milia formation, infection, acne and rosacea flares, postinflammatory pigmentary changes, prolonged erythema, and so forth (**Box 1**).

Acute complications occur within the first few days up to 1 month after the procedure. Long-term complications become apparent after the initial 2 weeks of healing from skin resurfacing.

Contact Dermatitis

Contact dermatitis, usually irritant in nature, is seen commonly after skin resurfacing as a result of impaired epidermal barrier function.[1–3] It rarely is a true type IV delayed hypersensitivity reaction because patch testing fails to reveal the allergens in most cases. Patients usually complain of burning and itching sensation of their skin along with increased redness. Eczematous eruptions also may occur (**Fig. 2**). Contact dermatitis may occur during the first 4 weeks after resurfacing. The deepithelialized state of the skin after resurfacing renders increased susceptibility to topical irritants, such as fragrances, propylene glycol, or lanolin and allergens in cleansers, moisturizes, and topical ointments, during the reepithelialization process. It is important to have patients avoid self-prescribed, topical, so-called herbal regimens or topical antibiotics, such as Neosporin (Johnson & Johnson) or bacitracin during the healing process. Once patients present with symptoms of contact dermatitis, immediate discontinuation of probable offending agents, cool compresses,

Box 1
Acute and long-term complications associated with skin resurfacing procedures

Acute complications
Contact dermatitis
Bacteria, fungal, and viral infections
Erythema
Pseudohypopigmentation
Delayed wound healing
Transient postinflammatory hyper- or hypopigmentation
Acne and rosacea flare
Milia
Long-term complications
Prolonged erythema
Pigmentary changes
Textural changes
Scarring (hypertrophic and atrophic)
Keloid formation