



International Scar Classification in 2019

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There has been a wide variety of therapies proposed for the treatment of scars, most of them with a lack of firm randomized controlled clinical trials to support their efficacy, and among other deficiencies, there has been often a lack of appropriate labeling or classification of scars to allow optimal evaluation of existing literature. There is a real benefit to a consistent classification of different types of scars so that different clinicians use a consistent vocabulary allowing a systematic evaluation of treatments and outcomes. One problem has always been the changes in scars over time so that improvements may not necessarily be due to the treatment intervention but simply scar maturation.

Scar classifications have previously been published including notably the 2002 publication in *PRS* which gained widespread acceptance because it represented the consensus of 12 experts from an international group incorporating Europe, North America, and Australia. In subsequent publications, this classification has been found to sound and not needing further modification. We will use this classification again in this updated textbook.

Scarring is the inevitable consequence of tissue injury as opposed to tissue regeneration which is the true restoration of the normal architecture of the skin. True tissue regeneration after injury occurs only in the fetus during the first two trimesters and in amphibians who can even regenerate amputated limbs. In the optimal outcome of a thin flat thin linear scar, the sequence of tissue repair after injury is tightly regulated. After initial platelet aggregation, provisional matrix is deposited, followed by influx of inflammatory cells and subsequent cell proliferation including fibroplasia and angiogenesis. Wound healing overlap, and are followed by cellular apoptosis with resolution of inflammation. Permanent matrix deposition (collagen) begins within 3 days. Maximal collagen deposition occurs in the first few weeks with a combination of type 1 and type 3, followed by many months of collagen breakdown and synthesis with increasing type 1 collagen with increased organization and scar strength. These phases of inflammation, cell proliferation and collagen remodeling result in a fine line scar in the scenario of an incision (“normal” scar), and a wider flat scar in the scenario of an injury over a broader area.

9.1 Immature Scar

Even a normal scar goes through a period when it is immature, meaning it is pink, often with a healing ridge (edema plus collagen synthesis). Collagen accumulation typically peaks about 3 weeks after surgery and then goes through about 6 months of remodeling with steadily increasing collagen organization, conversion

from a mix of type 1 and type 3 collagen to almost entirely type 1 collagen with increased cross-linking, and continued increase in tensile strength. The gains in scar strength are due to improvement in collagen organization and cross-linking rather than an increase in collagen. A useful analogy is to compare the difference between raw wool from a sheared sheep, versus the fine knit of a woolen coat. The amount of wool is the same, but the woven wool is far stronger. However, although the strength of a scar is maximal in approximately 6 months, complete scar maturation as measured by resolution of erythema typically takes a year or even longer. In a human volunteer study conducted by Renovo, superficial scars took longer than a year for the erythema to fully resolve in one-third of patients. In scars in thicker skin with more depth, or in a group of patients with more active scarring process, it probably takes even longer. In my own clinical practice with approximately 20,000 patients of all ages and ethnic backgrounds, my experience mirrors the study by Renovo (■ Table 9.1).

The resolution of erythema is a useful marker of scar maturity (■ Fig. 9.1). In all scars, erythema eventually resolves, but in a few percent of patients, I have seen it take longer than 2 years and in a rare hypertrophic scar as long as 10 years. In a study we conducted on burn patients, we found that the average scar elasticity (a measure of collagen remodeling), as measured by a device that applied a constant force to the skin and measured how much it stretched, continued to increase over 5 years (Bartell et al). Although the patients were not followed longitudinally, we simply correlated elasticity to the age of the scar, and it can be inferred that it can take a long time indeed for a scar to reach full maturity.

In an immature scar histologically, there are an increased number of inflammatory cells. After 2 weeks or so, the predominant inflammatory cells are macrophages with scattered lymphocytes and an occasional mast cell. There are increased numbers of fibroblasts including myofibroblasts, increased numbers of blood vessels, and for a period of up to a few months epidermal hyperplasia. The visual appearance is a scar that is erythematous and slightly raised due to increased fluid in the tissues and increased collagen as well as increased cellularity.

9.2 Mature Scar

In undergoing the transition from an immature scar to a mature scar, the key visual marker is resolution of erythema (■ Fig. 9.2). At this point, the inflammatory cells, endothelial cells, and most of the fibroblasts have undergone apoptosis, and the epithelium looks completely normal as compared to the adjacent unwounded

Table 9.1 Scar Classification

Scar type	Description
Immature scar	Pink slightly raised
	Sometimes itchy
	Firm but not hard
	Begins soon after injury, months to resolve
	Peaks at a few weeks after injury
Mature flat scar	Flat scar without erythema, stable
	No symptoms
Hypertrophic linear scar	Ropy (elevated) and pink or red
	Evolves from immature scar within several weeks
	Progressive enlargement for months before slow decrease in activity
	Often itchy or slightly sore to touch
	Resolution results in a persistently elevated scar that is no longer pink
Hypertrophic wide scar	Elevated, pink or red
	Arises from widespread injury such as a burn
	Frequently with severe pruritis and can be tender
Minor keloid	Very stiff with limitation of mobility across joint surface
	Round or elevated, extends beyond scar
	Most often at site of pierced earring or surgical incision
	Strong genetic component which is different than hypertrophic scars
Major keloid	Simple surgical excision with very high rate of recurrence
	Elevated, large often irregular in shape (i.e., butterfly appearance)
	Frequently seen in multiple locations on person
	Initial injury can be very minor
	Often symptoms of pain and pruritis are debilitating
	Treatment options are limited



Fig. 9.1 Immature scar



Fig. 9.2 Mature scar

skin. What is left is a band of collagen fibers that are clearly demarcated from the surrounding dermis histologically lacking the completely ordered collagen organization characteristic of normal skin. The collagen fibers go in multiple directions giving the scar stiffer biomechanical properties and are of variable width depend-

ing on the genetics of the patient and the underlying tension placed on the healing immature scar. There is no longer any increased fluid in the tissues (edema) and so the scar is flat. The color of the scar ranges from white (absence of melanocytes) to hyperpigmented (often characteristic of ethnicities with increased melanocytes

in the basal layer of the epidermis such as Asian, South Asian, Middle Eastern, Mediterranean, and Latin American or patients with brown skin).

9.3 Atrophic Scar

In some cases, a scar will become depressed or thinned as it transitions from an immature scar (■ Fig. 9.3). This can occur when collagen synthesis is depressed and inflammation is less than usual. Examples of atrophic scars are the stretch marks or striae that can be a complication of systemic steroid excess either from exogenous steroids or Cushing's disease or in some scars after radiation therapy.



■ Fig. 9.3 Atrophic mature scar



■ Fig. 9.4 Linear hypertrophic scar

9.4 Linear Hypertrophic Scar

In many cases, scars fail to transition normally from immature to mature with resolution of inflammation and an equilibrium of collagen synthesis and breakdown. Collagen continues to accumulate, and the scar widens and becomes elevated or ropy in appearance and the erythema fails to resolve. This active process of scar growth can continue for many months but eventually slowly resolves, with resolution of erythema that can take years. The collagen accumulation stabilizes but with a residual scar that is elevated and wider than a normal mature scar (■ Fig. 9.4). During the period of active collagen accumulation and erythema, the scar can be itchy and/or painful. The residual scar is less elastic (stiffer) than normal skin and, if it crosses a joint, can limit motion. A key feature of hypertrophic scar (versus keloid) is that the scar tissue remains within the confines of the original scar (although it may be widened). Hypertrophic scars are more common in patients of color, particularly East Asians, and the susceptibility to hypertrophic scars is often inherited. Prolonged inflammation for any reason (delays in epithelization, blocked sebaceous glands, ingrown hairs, and tension) contributes significantly to the incidence of hypertrophic scars. Frequently portions of a scar will be hypertrophic in hair-bearing areas, while the adjacent scars evolve into normal mature scars.

9.5 Widespread Hypertrophic Scar

In many cases, most notably from burns, the initial injury is not linear but covers a larger area (■ Fig. 9.5). In general, when epithelization (complete epithelial resurfacing) is not completed within 2 weeks, the risk of



hypertrophic scar increases, particularly in children and in adults under the age of 40. In a hypertrophic scar, the period of collagen accumulation is prolonged up to 6–12 months resulting in a scar that is elevated and thickened and is very stiff. Erythema will be prolonged and the scars are often intensely pruritic or even painful.

9.6 Keloid

A keloid unlike a hypertrophic scar behaves more like a tumor in that growth can occur even years after the original injury and extend far beyond the confines of the



■ Fig. 9.5 Hypertrophic broad scar

original scar. The keloid often has a mushroom or cauliflower type of appearance. Keloids are frequently symptomatic with pain or itch. The genetics of keloids are quite complex and beyond the scope of this chapter, but many are familial and are much more common among many African tribes and to a lesser degree among Asians. The central portion of a keloid is very dense fibers with a characteristic pattern histologically and relatively acellular, while the active spreading edge has a significant inflammatory and cellular component.

9.6.1 Minor Keloid

Most keloids arise from localized injuries such as a pierced ear (the most common location of minor keloid), and although they extend beyond the margins of the original scar, their growth stabilizes and allows more options for treatment including surgical excision combined with other modalities such as steroid injections or radiation therapy (■ Fig. 9.6).

9.6.2 Major Keloid

In extreme situations in patients with a very strong genetic predisposition, keloids can extend into large plaques with a very actively growing outer edge that in some areas has a characteristic butterfly pattern (■ Fig. 9.7a) due to forces on the keloid exerted by the surrounding skin. The peripheral edge of the keloid is active, while the central portion is less active with less cell proliferation. Often the original injury can be as minor as a scratch or seemingly spontaneous (■ Fig. 9.7b) Keloid arising from small tracheotomy



■ Fig. 9.6 Minor keloid

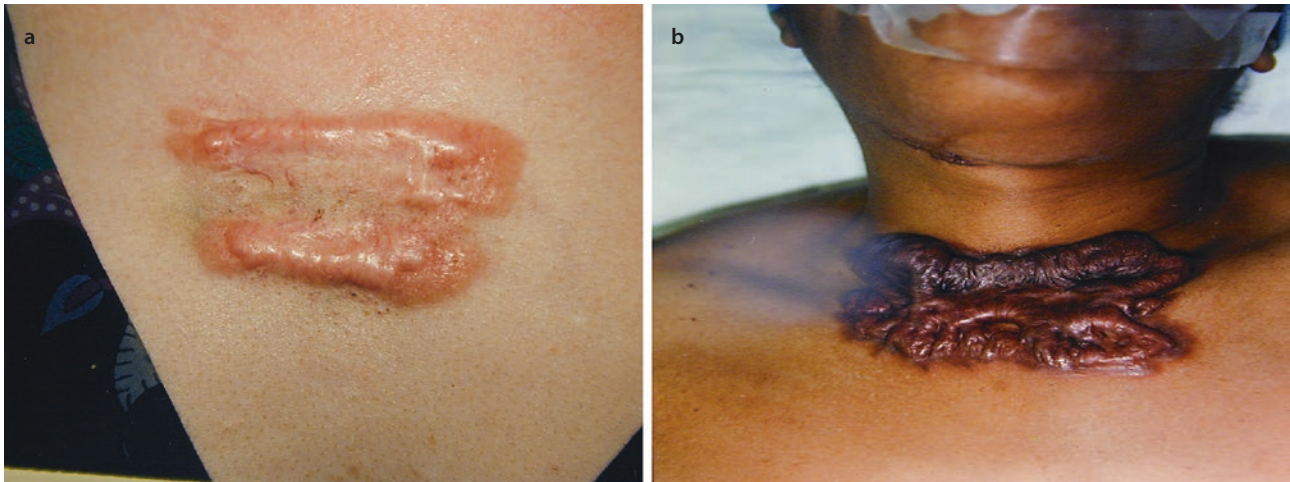


Fig. 9.7 Major keloid. **a** Chest keloid with typical butterfly pattern. **b** Major keloid

scar), and the unfortunate patients with major keloids can form them all over their body. These are extraordinarily difficult to treat and are both deforming and debilitating. Intensive research on the defining genetic characteristics and pathogenesis of keloids continues, and in a few cases genetic loci have been identified, but the genetics are complex, as well as the pathogenesis.

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