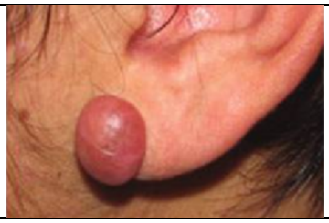
Characteristics	Atrophic scar	Hypertrophic scars	Keloids
			
Incidence	Common	Common	Seldom, increased incidence with skin pigmentation
Propagation	Only in the area of initial injury	Only in the area of initial injury	Progression over the boundaries of the initial injury
Appearance	< 6 months after injury	< 6 months after injury	> 6 months after injury
Distinguishing features	• depressed, sunken in, pitted appearance •Scarring does not extend beyond boundary of original wound • ice pick, boxcar and rolling scars	• Appear as red raised scar tissue • Scarring does not extend beyond boundary of original wound • Nodular structures containing a-SMA-producing myofibroblasts • Promote scar contractures • **Can regress with time**	•Often appear as shiny rounded protuberances, color ranges from pink to purple • **Scarring extends beyond boundaries of original wound** • Rarely nodular, no a-SMA producing myofibroblasts • Do not promote scar contractures • **Do not regress with time**
Regression	Little regression	Often spontaneous	No regression
Previous Trauma	Yes	Yes	Yes, but also minimal trauma like bites of insects or scratches possible
Localization	Whole integument	Whole integument	Whole integument, often at the neck, earlobes and sternum
Genetic predisposition	Unknown	Unknown	Estimated
Histology	• Generalized cutaneous atrophy resulting in loss of cutaneous cells in the epidermis • Collagen Type IV expression reduced • Keratinocytes in scarring epidermis were more proliferative than in normal skin	• α-actin positive Myofibroblasts • collagen in wave like patterns parallel to the epidermis	• Reduced apoptosis • Increased angiogenesis • Thick collagen parallel to the epidermis with formed knots • Reduced central cell count
TGF-ß1 involvement	⇑ TGF-ß1 was drastically elevated in APS, suggesting that the aberrant TGF-β1 signaling is an underlying modulator of all these pathological processes [7]	⇑ TGF-ß1 expression in HTS tissue and HTS fibroblasts [8] ⇑ TBRI and TßRII expression in HTS fibroblasts[9] ⇑ Smad2 nuclear localization in HTS fibroblasts[10] ⇑ TGF-ß1 serum levels in burn patients that develop HTS[11]	⇑ TGF-ß1 and TGF-ß2 expression in keloid fibroblasts[12,13] ⇑ TBRI and TßRII expression in keloid fibroblasts[14,15] ⇑ TGF-ß/Smad3 signalling in keloid fibroblasts[14,15] • Genetic association of TGF-B1 and Smad4 variants in etiology of keloid disease [16]