PATHOLOGICAL TYPES OF SCARS AND THEIR IMPACT ON TREATMENT

Dr. Tarek Ahmed Said

Professor of Plastic Surgery
Cairo University
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Pathological Types of Scars



Atrophic Scars



Normotrophic Scars



Hypertrophic Scars & Keloids

Mixed Scars



Factors affecting scar development

Inflammation and Oedema

Minimal ↓ TGF Beta 1 and 2 Insufficient Collagen Synthesis **Atrophic Scar**

Adequate Adequate TGF Beta 1 and 2 Active fibroblasts Slightly ↑ sufficient Collagen Synthesis **Normotrophic Scar**

Extensive ↑ TGF Beta 1 and 2 ↑ Active fibroblasts **Excessive collagen synthesis** Hypertrophic scar

Nonhomogenous Uneven conc. TGF Beta 1 and 2 uneven conc. Active fibroblasts collagen synthesis in Islets Mixed scar





Factors affecting scar development

Inflammation & Oedema

Minimal Atrophic

Animal bites
Acne scars
Facial Wounds
Children
Certain locations
Loss of integrity of dermis
Post-surgery scars
Deficiency of O2, vitamin C

Moderate Normotrophic

Extensive Hypertrophic & Keloid

Irritant introduction or contact
Chronic inflammation
UVR
Genetic (Keloids)
Crossing skin lines
Certain locations









Normotrophic Scars

Inflammatory stage

Debridement Oedema Control (alpha chemotrypsin) **NSAIDs Antibiotics**

Proliferarion Stage

Contractubex Compression silicone sheeting (areas famous for hypertrophic scarring) Enzymotherapy Cryodustruction

Maturation Stage

Compression hydration Dermaroller Vit C applications (Ascorbic acid) Microdermabrasion Peeling Enzymotherapy Cryodestruction PDL





Enzymotherapy

- Proteolytic enzymes dilute necrotic mass
- Hyaluronidase depolymerizes hyaluronic acid.

Occlusive Hydration

- Maximal hydration of the scar formation zone
- Reduction of transepidermal water loss
- Acceleration of cell migration
- Increase of fibroblast proliferation and synthetic activity

Cryotherapy

- Evens overhanging scar edges
- Reduces interstitial edema

Early Microdermabrasion

- Evens and improves the scar surface
- Clears away the epidermal (lipidic) skin barrier of the scar area along with epidermis, which enables medicinal products to penetrate freely into the skin.
- Scar bottom is elevated by the vacuum to the surrounding skin level, counteracting myofibroblasts

Better avoided in Fitzpatrick skin phototypes 4-6



Medium & Deep Chemical Peel

TCA 30-50% sessions

LASER

- NON Ablative PDL for erythematous scars eliminates vascular component
- Fractional CO2 Laser for non-erythematous scars

Collagen Induction Therapy (CIT) Dermaroller

Creates minute channels stimulating islets of collagen synthesis



Atrophic Scars



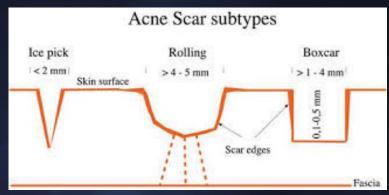








Types of Atrophic Scars











Management of Atrophic Scars

Scar considered fresh during 1st year, golden time of treatment is first 7-9 months

Aims

- To Smoothen scar demarcation line
- To even overhanging scar edges
- Transdermal induction of collagen synthesis
- To elevate Scar bottom

Tools of management in Fresh atrophic scars

?? Enzymotherapy (Proteolytic Serrapeptase, Nattokinase and Hyalorinase) / Topical creams Occlusive Hydration (Till wound bottom reaches skin level)

Cryotherapy

Microdermabrasion (NOT FOR ICEPICK ATROPHIC SCARS)

Dermaroller

Peeling

Laser Correction

NO CONTRACTUBEX



Management of Old Atrophic Scars

Tools of management

Dermaroller

Laser Correction of erythematous scars / Laser or Peeling for nonvascularised Scars

Subcision (Nokor needle)

Excision / Punch excision

Hydration

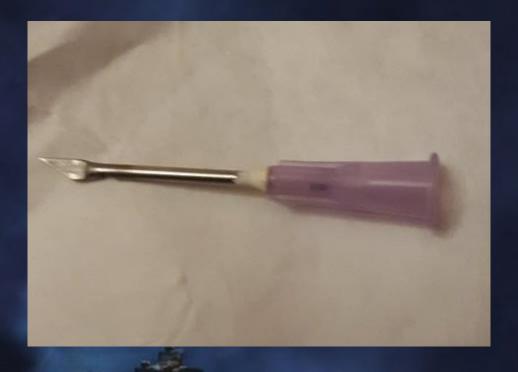
Microdermabrasion

Autofibroblasts

Autologous Fat Transfer / Fillers [only after subcision]



Nokor needle







Pathological Scars









Pathological Scars

Feature	Hypertrophic Scars	Keloids
Incidence	Frequent	Rare
Time of Development	Soon after injury	May take months
Genetic Predisposition	None	Present
Race	None	Related to skin Colour
Sex	None	Females
Age	Children	10-30 Years
Extension	Within wound	Outgrows wound area
Natural History	Subsides with time	Rarely subsides
Sites	Areas of Motion Scars crossing joints or skin lines	Areas of little motion Sternum, shoulders, ear lobules
Aetiology	Tension ??	Unknown
Content	↓ Calcium	↑ Calcium
	↓ Magnesium	↑ Magnesium
	↓ Mucinous ground substance	↑ Mucinous ground substance
	↑ Fibroblasts	↓ Fibroblasts
	↑ Foreign body reaction	No Foreign body reaction
Fast Blue collagen stain	Blue	Reddish
Response to treatment	Good	Poor



Management of Hypertrophic Scars & Keloids

Aims:

Reduction of Scar Mass +/- Induction of new proper collagen synthesis Improving texture and elasticity of scar

Treatment options are similar but Hypertrophic scars have better prognosis

A solitary tool of management seldom works



Management of Hypertrophic Scars & Keloids

Non Invasive

Occlusive dressings / Compression therapy

Intralesional corticosteroid injections (decreases TGF Beta 1 and Inflammatory mediators)

Intralesional Immunomodulators (Interferon) injections: Inhibits type I,II,IV Collagen synthesis Intralesional Calcium Channel Blockers (Verapamil): ↓ collagen synthesis & ↑ collagenase production Intralesional Antineoplastic Agents 5FU, Belomycin, Tamoxifen & Doxorubicin

Topical Immunosuppressant Tacrolimus ointment: Inhibits TNF alpha

Topical Retinoic Acid (Retin-A): Inhibits DNA synthesis in fibroblasts and lymphocytes (Not FDA approved)

Topical Imiquimod 5% cream "Aldara" (Immune response modifier): Decreases collagen synthesis (Not FDA approved)

Botulinum toxin

Minimally Invasive

Cryosurgery for small lesions (affects microvasculature causing cell damage via intracellular crystals, leading to anoxia)

Radiation therapy

Light therapy (IPL)

Laser therapy (Non Ablative PDL & Fractional)

Surgical Excision



Management of Hypertrophic Scars & Keloids

First-line treatment

Cryotherapy, May cause hypopigmentation, pain

Intralesional corticosteroids., May cause discomfort, skin atrophy, telangiectasia

Silicone sheeting

Pressure dressing for six to 12 months

Second-line and alternative treatment

Surgical excision, If alone, can be followed by more aggressive growth in up to 84% of cases

Combined cryotherapy and intralesional corticosteroid injection

"Triple keloid therapy" (surgery, corticosteroids, and silicone sheeting)

Pulsed dye laser

Intralesion Verapamil and silicone sheeting

Intralesional Fluorouracil, Effective; May cause hyperpigmentation, wound ulceration

Bleomycin tattooing, Effective; May cause pulmonary fibrosis, cutaneous reactions

Postsurgical intralesional interferon, Expensive; May cause pruritus, altered pigmentation, pain

Radiation therapy alone or Postsurgical, May cause cancer, hyperpigmentation, paresthesias

Onion extract topical gels (e.g., Contractubex, Mederma), Limited effect alone



Compression

- Mechanically holds growth
- Compresses scar vasculature leading to anoxia

Dermabrasion

- Microdermabrasion and rotation dermabrasion have little value
- Might stimulate collagen synthesis
- Sandabrasion may have a role

Radiation

- Effect disputable
- Risk of malignancy
- Age and site restrictions







Promising therapies that aim at decreasing collagen synthesis Antiangiogenic factors, including vascular endothelial growth factor (VEGF) inhibitors **Phototherapy (photodynamic therapy [PDT] UVA-1** therapy, narrowband **UVB** therapy) **Transforming growth factor (TGF)**—beta3 **Tumor necrosis factor (TNF)-alpha inhibitors (etanercept)** Recombinant human interleukin (rhlL-10)





Dr. Tarek Said Kasr El-Aini Faculty of Medicine, Cairo University drtareksaid.com



