HEALING OF SKIN WOUND

Here, we specifically describe the healing of skin wounds.
As it involves both epithelial regeneration & the formation of connective tissue scar, it is thus illustrative of the general principles that apply towound healing inall tissues.
Healing of skin wounds : Either

I. Healing by Primary intention. (Primaryunion)

Occurs in an uninfected clean sterile wound without tissue loss as in surgical incision approximated by surgical sutures .
The incision causes only focal disruption (loss of continuity) of epithelial BM & death of
relatively few epithelial & connective tissue cells.
As a result, epithelial regeneration predominates over fibrosis.
A small scar is formed , but there is minimal wound contraction.
When an incision is made in the skin & subcutaneous tissue , blood escapes from the cut
vessels, it clots on the wound surface & fills the gap between the wound edges , which is narrow in
sutured wound

Within 24 hours: neutrophils are seen at the incision margin, migrating toward the fibrin clot.
This is called traumatic inflammatory response .
Mean while the Basal cells atthe cut edge of the
epidermis begin to exhibit mitotic activity.

Within 24 to 48 hours , epithelial cells from both
edges have begun to migrate & proliferate along the
dermis, depositing basement membrane components as they progress .
The cells meet in the midline beneath the surface scab , yielding a thin but continuous epithelial layer . The basal cell proliferation stops
by contact inhibition .

By day 3 neutrophils have been largely replaced by macrophages , followed by angiogenesis & granulation tissue , which consists
of proliferating capillaries & fibroblasts progressively invades the incision space .
Collagen fibers being layed down by the fibroblasts are now evident atthe incision margins,
but these are vertically oriented & do not bridge
the incision .
Epithelial cell proliferation continues, yielding a thickened epidermal covering layer.

By day 5, angiogenesis reaches its peak as granulation
tissue fills the incisional space & collagen fibrils
become more abundant & begin to bridge the incision.
The epidermis recovers its normal thickness as
differentiation of surface cells yields a mature epidermal
architecture with surface keratinization.

During thesecond week:
There is continued collagen accumulation & fibroblasts
proliferation. TheWBC infiltrate, edema, & the vascularity
are substantially diminished. The long process of "blanching“
(pallor) begins, accomplished by: collagen deposition within
the incisional scar& the regression of vascular channels.

By the end of the first month the scar comprises a cellular
connective tissue, devoid of inflammatory cells & covered by
anessentially normal epidermis.
Hair follicles & sebaceous glands which are destroyed in
the line of incision are permanently lost.

II. Healing by secondary intention (Secondary union)

Secondary union differsfrom the primary in several
aspects

(I) A larger clotor scab rich in fibrin and fibronectin
forms at the surface of the wound

(2)Inflammation is more intense because large
tissue defects have a greater volume of necrotic debris ,
exudate , and fibrin that must be removed .
Consequently large defects have a greater potential
for secondary inflammation – mediated injury .

(3)Larger defects require greater volume of granulation tissue to fill in the gaps & provide the
underlying framework for the regrowth of tissues
epithelium . A greater volume of granulation tissue
generally results in agreater mass of scar tissue .

(4) Secondary healing involves wound contraction

Within 6 weeks large skin defects may be reduced to 5%-10% of their original size largely by contraction .
This process is due to the presence of myofibroblasts, a modified fibroblasts exhibiting
many of the ultrastructural & functional features of contractile smooth muscle cells.

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