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Iwo-Amah RS
Rivers State University Teaching Hospital, Port Harcourt, Nigeria

Abbey M
Rivers State University Teaching Hospital, Port Harcourt, Nigeria

Ikiroma SE
Rivers State University Teaching Hospital, Port Harcourt, Nigeria

Ela G
Rivers State University Teaching Hospital, Port Harcourt, Nigeria

Corresponding Author:
Iwo-Amah RS
Rivers State University Teaching Hospital, Port Harcourt, Nigeria

Wound Healing in Obstetrics and Gynecology

Iwo-Amah RS, Abbey M, Ikiroma SE and Ela G

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Abstract
Why some wounds in 0bstetrics and Gynecology heal poorly, is because the different biological and nutritional requirements are suboptimal, or some disease conditions make wound healing difficult. Awareness of this, will lead to conscious efforts by the doctors and health care providers to aid wound healing.

Keywords: Poor wound healing, obstetrics and gynecology, biological factors

Introduction
Wound healing can present a significant challenge and burden on health care systems. Maintaining skin integrity and possessing a robust wound healing capacity are key pre-requisites for healthy survival. Poor wound healing can lead to high expenses and increased morbidity [1, 4, 5, 6].
Wound is a disruption of the integrity of skin, mucosal surfaces or organ tissue [2].
In Obstetrics and Gynecology, more than 80% of wounds encountered are due to surgical incisions made at the time of surgery. Other wounds are due to Lacerations, bruises, abrasions, road traffic accidents, domestic accidents, gunshot wounds, thermal burns, bacterial infections like Syphilitic ulcers etc [7-8].
The wound healing process is a cascade of synchronized events aimed at restoring skin integrity. At the time of insult, multiple cellular and extracellular pathways are activated in a tightly regulated and coordinated fashion, with the aim of restoring tissue integrity [2, 3, 5, 6].
The aim of this article is to highlight why some wounds heal poorly, the basic steps involved in the wound healing process and factors that significantly affect wound healing.

Discussion
Wound healing is a complex biological process which results in the restoration of tissue integrity, as well as preserving all other skin functions [1]. The skin is the largest organ of the human body and its key function is to protect water rich internal organs from the dry external environment. It is also a protective barrier against germs, the UV radiation and other elements [3, 5-7].
Wound healing in Obstetrics and Gynecology is similar to other parts of the human body, except that wounds in the upper part of the body heal faster than those in the lower extremities. This is because of the rich blood supply in these areas which quickly delivers oxygen and nutrients for the healing process, unlike the lower extremities which often have poorer circulation and are prone to factors like gravity and pressure that can slow healing.
There are 3 broad approaches to wound closure [7, 8]

- Primary closure or healing by primary intension. Good apposition of the wound edges and absence of foreign body will aid primary closure.
- Healing by Secondary intension, which allows the wound to heal without surgical intervention. A wound that was sutured surgically, may not heal by primary closure. In the presence of superficial dehiscence, the wound can be allowed to heal by secondary intension.
- Healing by Tertiary intension, in which wounds are surgically closed after a period of secondary healing (wound resuturing)

Phases of wound healing

There are 4 overlapping, but distinct phases of wound healing.

These are Haemostasis, Inflammation, Proliferation and Tissue Remodeling [1, 3, 8].

Haemostasis

Begins immediately after an injury or disruption of skin integrity.

The blood vessels constrict, platelets are activated by contact with exposed collagen and release their granules. This results in further platelet activation and aggregation. `

In conjunction with activation of the co-agulation cascade, it results in deposition of a provisional fibrin matrix within the wound. Also a large number of cytokines, including Transformation Growth Factor B(TGF-B) and platelet derived Growth factor are secreted to promote chemotaxis of neutrophils and macrophages, leading to commencement of the inflammatory phase.

Inflammation phase

The key aim of this phase is to prevent infection. The mechanical barrier, which was frontline against invading organisms is no longer intact [9, 11, 12].

Neutrophils, the first responders, are highly motile cells, which infiltrate the wound within an hour of insult and migrate in sustained levels for the first 48 hours. This is mediated through various chemical signaling mechanism like angiotensin 11, TGF-B.

Wound healing may progress in the absence of neutrophils, but macrophages (derived from activated monocytes) aid phagocytosis and produce more cytokines and growth factors that promote fibroblast proliferation, angiogenesis and keratinocyte migration

Proliferative Phase

Within 2–3 days of the initial injury a sufficient number of fibroblasts migrate to the wound and herald the beginning of the proliferative phase, that lasts up to 3 weeks in a healing cutaneous wound [12, 13].

Fibroblasts play a key role in this phase, they produce disorganized collagens, high in immature Type III collagens, into the provisional matrix. Fibroblasts recruited to the wound tissue may transform to become myofibroblasts, under the influence of several cytokines, leading to increased collagen production and eventual wound contraction [12-15].

Remodeling phase

In the final remodeling phase of wound healing, granulation tissue is replaced by permanent scar. Net collagen production continues actively for 4–5 weeks, followed by replacement of Type III reticular collagens with Type I fibrillary collagen over the next year [14-16].

Zinc dependent endopeptidases known as matrix metalloproteinases, secreted by the epidermal cells, play a central role in tissue remodeling.

Wound tensile strength continues to increase with increasing collagen production — production from 3% on week 1, 20% after week 3, at three months post injury, tensile strength peaks at 80% of uninjured skin, never reaching 100% [10, 11].

This phase involves a balance between synthesis and degradation, as the collagen and other proteins deposited in the wound become increasingly well organized.

Eventually they will regain a structure similar to that seen in the unwounded tissue (replacing Type1 collagen with Type 3 collagen)

An abnormal healing response can result in raised, thick and often red scar due to excess collagen production during the healing process, called Hypertrophic scar. When the scar tissue grows beyond the original wound and does not regress over time, it is a Keloid. These are abnormal responses of the healing process.

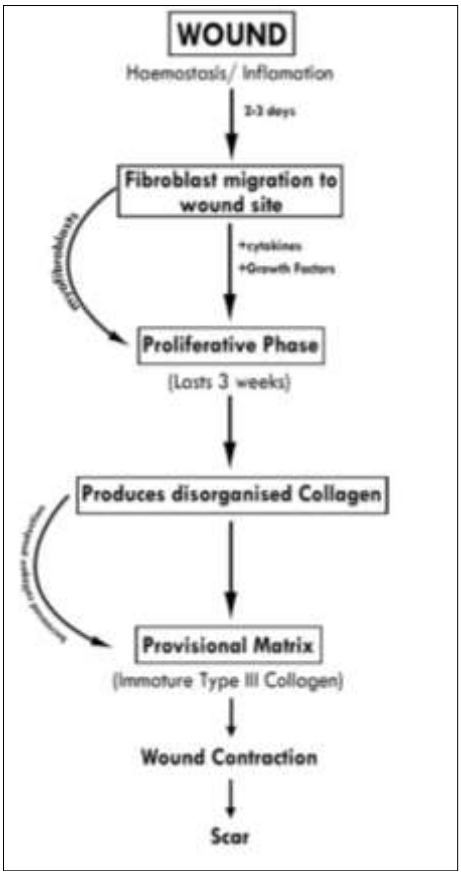

Fig 1: Wound healing

Factors that affect wound healing

Nutrition

The healing process requires macronutrients, as well as micronutrients to facilitate skin integrity restoration.

Macronutrients such as carbohydrates, fats, proteins and fluids.

Micronutrients like amino acids, vitamins, minerals.

Overall caloric requirements to synthesize proteins are estimated to be 0.9 Kcal/g and a 1 mm thick section of granulation tissue would require 10 mg of collagen, therefore small wounds may not always pose a nutritional challenge [17-20].

The nutritional deficit begins to widen with increasing wound size, especially large thermal burns.

Vitamin C

Vitamin C has been widely associated with healing. It functions as a co-substrate for hydroxylase enzymes required for collagen synthesis.

Vitamin A

Required for epithelial growth, angiogenesis and collagen synthesis.

Zinc
Zinc also has been documented to facilitate increasing wound strength and epithelization. There is no conclusive evidence to support supplementation of micronutrients in the non-deficient patient.

Chronic & Debilitating Illnesses
In diabetic patients, uncontrolled hyperglycemia has been shown to impair fibroblast and endothelial cell function [12, 14, 15].
Immunosuppressive disorders like Human Immune Deficiency viral infection, are associated with poor wound healing. The immune system is weakened, leading to reduced white blood cells and CD4 cells, which are crucial for fighting infections and repairing tissue. As a result, there is increased susceptibility to wound infections, slower collagen production, chronic inflammation and poor angiogenesis, thus delaying and complicating wound healing [1-3].

Smoking
Smoking has detrimental effect on wound healing, with over 400 substances found in cigarettes that may negatively impact wound healing. Nicotine specifically promotes vasoconstriction, disrupting microcirculation. Cellular migration is also impeded (chemotaxis) [21, 22].

Alcohol Abuse
Studies indicate that acute alcohol intoxication may also have detrimental effect on wound healing. It impairs immune function, reducing collagen production, causes nutritional deficiencies, slows inflammation, proliferation and remodeling phases of wound repair [23, 24].

Radiation
Ionizing radiation can damage DNA, tissues by excited subatomic particles, leading to single or double – strand breaks or cross linking of the double helix.
Radiation also creates free radicals that damage proteins and cell membranes. These abnormalities lead to slow repair and slower epithelialization, tensile strength as well as higher infection and dehiscence rates [24, 25].
Limiting the radiation field and shielding areas that are not actively radiated may minimize the deleterious impact of radiation on wound healing.

Chemo Therapy: Chemotherapy also has deleterious effect on wound healing. Chemotherapeutic drugs adversely affect and delay the inflammatory phase, leading to decrease fibrin deposition and collagen synthesis. There is also delay in wound contraction [25, 26].

Drug
Non-steroidal anti-inflammatory drugs (NSAIDS) have been shown to have depressive effect on wound healing. NSAIDS inhibit COX-1 and COX-2 and decrease prostaglandin E2 production, therefore may impede tissue repair by virtue of retarding inflammatory response. NSAIDS may have an anti proliferative effect on angiogenesis, thereby delaying healing, therefore only conservative short term use of NSAID may be beneficial for acute pain control [20, 21]

Steroids: Systemic steroids like corticosteroids generally impair wound healing by suppressing the inflammatory response, reducing fibroblast proliferation, inhibiting collagen synthesis and epithelization, increasing the risk of complications such as wound dehiscence and infection.
In contrast, low dose topical corticosteroids can sometimes accelerate healing in chronic wounds by reducing excessive inflammation, pain and fluid.

Genetic Factors
Wound healing can be affected by genetic disorders – like Genetic connective tissue disorders – Cutis Laxa(CL). This disorder is characterized by increased vascularization in the dermis, reduced collagen bundle sizes, and underdeveloped elastic fibers. These interfere with good wound healing [16-18].
Ehlers Danlos syndrome – There is generalized tissue fragility, skin hyper extensibility [27].
Hyper homocysteinemia – Can be an independent risk factor for sub optimal wound healing, causing thrombosis especially in wounds involving lower extremities.

Hypoxia
Hypoxia (low oxygen) can both stimulate early-stage wound healing by promoting cell migration and proliferation (mediated by Hypoxia-inducible factor-1 (HIF-1)), but prolonged or severe hypoxia hinders healing by impairing cell function, reducing angiogenesis, delaying fibroblast proliferation and collagen synthesis, and increasing oxidative stress [8-10].
This complex relationship means early controlled oxygen levels are beneficial, whereas chronic hypoxia is a significant factor in chronic, non-healing wounds.

Conclusion
Wound healing is a complex process requiring the coordinated actions of multiple cell types in response to a variety of differing cytokines and micro environmental conditions.
Understanding of the basic principles will help Obstetricians and Gynecologists to promote factors that will aid the healing process.
Conflict of Interest: There are no conflict of interest

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