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Pharmacological Insights of Wounds and Updated Review

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Abstract: *In chronic wounds, the carefully controlled, multi-phase biological process of wound healing—which includes hemostasis, inflammation, proliferation, and remodeling—becomes dysregulated. Pharmacological and bioengineering developments have advanced quickly in recent years. These include biologics and tailored growth factor therapies, antimicrobial/antibiofilm dressings, cell-derived therapies (PRP, MSCs, exosomes), and smart/nanotherapeutic delivery systems. In addition to highlighting prospective translational pathways, present limitations (safety, delivery, cost, and antimicrobial stewardship), and research objectives, this review synthesizes mechanistic knowledge and clinical evidence from 2020 to 2025.*

Keywords: Wound, Burn Wound, Treatment, Causes, Management, Incision, Safety

I. INTRODUCTION

Overview Chronic wounds, such as diabetic foot ulcers and venous/pressure ulcers, do not advance and present a significant clinical and financial burden. In contrast, acute wounds often go through a series of coordinated stages before closing. Pro-healing biologics, repurposed small compounds, and combination biomaterials that influence inflammation, induce angiogenesis, and restore extracellular matrix (ECM) dynamics are examples of pharmacological methods that have expanded beyond simple antimicrobials. These growing therapeutic classes are summed up in recent thorough evaluations, which also highlight the translational drive toward combination and delivery-focused options.[1]

2. Approaches (search plan):

Our review of the literature (2020–2025) focused on high-impact original studies, meta-analyses, and systematic and narrative reviews of pharmacological wound treatments. PubMed/PMC, Frontiers, MDPI, and review articles found through focused searches for "wound healing pharmacology," "growth factors," "antimicrobial dressings," and "platelet-rich plasma/exosomes" were among the important databases and sources.[2]

3. A basic overview of biology (pharmacological targets)

Coordination of cell migration and proliferation (fibroblasts, keratinocytes), angiogenesis, ECM deposition and remodeling, and the timely resolution of inflammation are all necessary for wound healing. Because they regulate cell recruitment, proliferation, angiogenesis, matrix production, and downstream signaling, key molecular regulators such as PDGF, TGF-β, VEGF, FGF, EGF, and downstream signaling are excellent targets for pharmaceuticals. Chronic wounds frequently fail due to dysregulated inflammation, biofilms, hypoxia, and poor angiogenesis.[3]

4. Principal pharmacological techniques

4.1 Gene-based delivery and growth factors :

Growth factors (EGF, PDGF, FGF, and VEGF) either topically or intralesionally have been shown to directly promote angiogenesis and cell division. Benefits in certain situations (such as diabetic ulcers) are supported by clinical evidence;

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nevertheless, variable results are linked to low stability, fast proteolysis in the wound environment, problems with dosage and delivery, and expense. To enhance targeted, prolonged exposure, controlled-release scaffolds (collagen matrices, hydrogels) and gene therapy are being explored. EGF, PDGF, and FGF frequently rate well for healing time and ulcer area reduction, according to recent meta-analyses and trials; nevertheless, for reproducible efficacy, their distribution must be adjusted.[4]

4.2 Stewardship, antimicrobials, and antibiofilm tactics:

Two of the main obstacles to healing are infection and biofilms. Silver, iodine, and other antimicrobials are used in modern dressings; new formulations strive for low cytotoxicity and broad efficacy. To stop resistance and maintain the equilibrium of the microbiome, antimicrobial stewardship and antibiofilm strategies (enzymes, photodynamic treatment, antimicrobial peptides, and quorum-sensing inhibitors) are being stressed more and more. Iodine and silver-containing dressings are supported by clinical evaluations in certain situations; nevertheless, selection must balance the danger of resistance and cytotoxicity against efficacy.[5]

4.3 Exosomes, platelet-rich plasma (PRP), and cell treatments:

In order to reduce inflammation, promote angiogenesis, and attract resident progenitor cells, autologous PRP, mesenchymal stem/stromal cells (MSCs), and cell-derived exosomes all work through paracrine signaling. Heterogeneity in preparation, dosage, and outcome measurements restricts conclusive recommendations, despite systematic reviews and meta-analyses demonstrating encouraging efficacy signals for PRP and MSCs in speeding closure in some wound types. Because they are easier to store and standardize than entire cells, exosome preparations—including PRP-derived exosomes—are a hot topic in preclinical and early clinical research. Biomaterials, multipurpose dressings, and nanotherapeutics Growth factors, antimicrobials, and nucleic acids can be delivered in a regulated manner thanks to nanoparticles, responsive hydrogels, and composite biomaterials. Multifunctional dressings combine mechanical protection, exudate control, moisture balance, and controlled drug release; some also include sensors or electrical/photonic stimulation.[6]

4.5 Repurposed small compounds and systemic medications:

Through immunomodulation, better perfusion, or increased cellular metabolism, a number of repurposed medications (such as statins, several anti-inflammatory drugs, metformin, or modulators of nitric oxide pathways) exhibit preclinical pro-healing benefits. Limited clinical translation necessitates targeted research to validate safety and benefits in particular populations. Candidates and mechanisms are compiled in reviews of "pro-healing" medications. 4.6 Smart/sensing bandages and electrotherapy Prototypes for electrostimulation, photo biomodulation, and "smart" dressings that deliver stimuli (heat, light, or mild electric fields) or monitor wound condition are progressing to early clinical trials. These technologies, which include remotely monitorable gadgets and inexpensive electrotherapy bandages, can destroy biofilms, improve angiogenesis, and control inflammation. Although there aren't many large randomized trials, early results are promising.

5. Clinical evidence:

what is known and what is unknown Growth-promoting factors: Effective in certain situations (certain diabetic ulcers, for example), but inconsistent overall outcomes because of delivery and stability problems. Antimicrobial dressings: Silver and iodine dressings have been shown to help reduce infection and colonization in specific wounds; selection should be based on stewardship principles. PRP, MSCs, and exosomes: Although trial heterogeneity and varied manufacturing/preparation standards are significant confounders, meta-analyses demonstrate encouraging outcomes; standardized techniques are required. Biomaterials plus bioactives combined: Multifunctional dressings and controlled-release scaffolds exhibit better preclinical results and increase local bioavailability; their transition to standard clinical use is quickenin.[7]

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Classification:

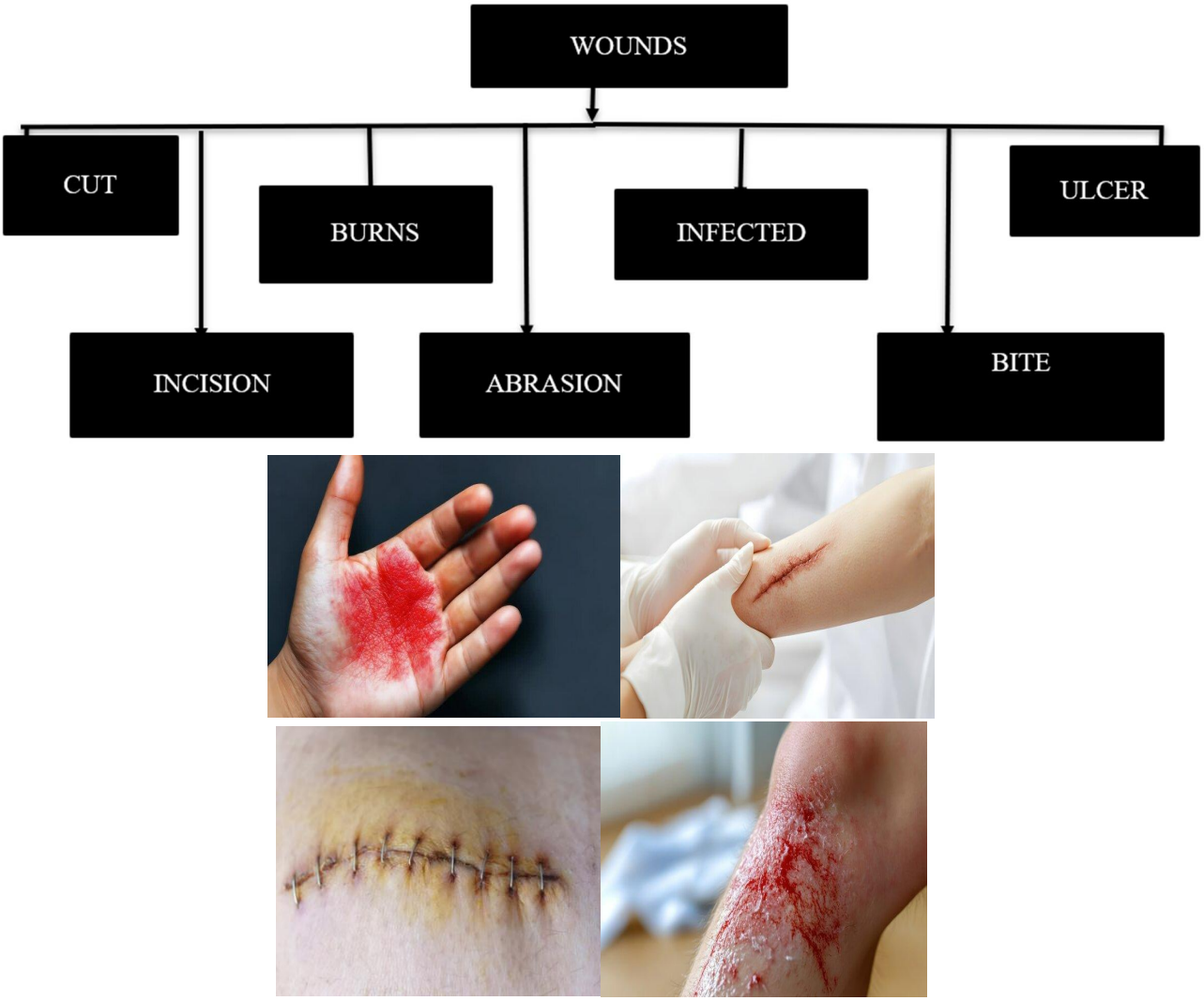

Fig No. 1 Types Of Wound

Cut Injuries:

1. Bleeding: From minor to severe, bleeding can occur from cut wounds.
2. Pain: Cut wounds may hurt, particularly if there is damage to the nerve endings.
3. Open wound: Infection risk may be elevated by cut wounds, which are open wounds.
4. Swelling and redness: Inflammation may cause the afflicted area to swell and turn red.

How to Treat Cut Wounds:

1. halt the bleeding: on halt the bleeding, apply pressure on the wound.
2. Clean the wound: To get rid of any bacteria or dirt, rinse the wound with clean water.
3. Apply antibiotic ointment: To aid in preventing infection, apply antibiotic ointment to the wound.[8]

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Fig.no. 2 Cut Wound

4. Dress the wound: To prevent more harm and infection, cover the wound with a bandage or dressing.
5. Get medical help: Get medical help if the wound is deep, big, or isn't stopping the bleeding.[9]

Cut Wound Complications:

1. Infection: If cut wounds are not adequately cleaned and cared for, they may get infected.
2. Scarring: Cut wounds may leave scars, particularly if they are large or deep. 3. Nerve damage: Numbness or tingling may result from cut wounds that harm nerve endings.

Reasons:

1. Trauma: Trauma, such as being in an automobile accident or trapped beneath a large object, can result in crush wounds.
2. Industrial Accidents: Crush injuries can happen in manufacturing or construction environments.
3. Natural calamities: Landslides and earthquakes are examples of natural calamities that might result in crush wounds.[10]

Bite Injury: Bite Injury An injury brought on by an animal or human's teeth is known as a bite wound. Bite injuries may include:

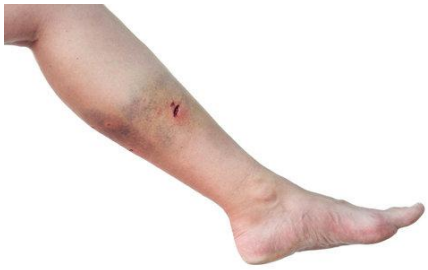

Fig No. 3 Bite Wound

Types:

1.Animal Bites: Dog, cat, or other animal bites.
2. Human Bites: Other people's bites. Qualities:

Issues:

1. Infection: Redness, swelling, and pus can result from infected bite wounds.
2. Rabies: If an animal is diseased, rabies can be spread through animal bites.
3. Tetanus: The risk of contracting tetanus can be elevated by bite wounds.[11]

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Issues:
Infection:
If abrasions are not properly cared for, they may get infected.
1.Scarring: Abrasions, particularly deep or infected ones, may leave scars.[12]
2. Avoidance:
1. Protective Gear: When engaging in activities, wear protective gear, such as elbow or knee pads.
2. Adequate Footwear: To avoid slips, use appropriate footwear. Wound from Incision An incision wound is a kind of wound that results from the skin being cut by a sharp instrument, like a knife or scalpel.[13]
Qualities:
1. Clean Cut: The borders of incision wounds are usually straight and clean.
2. Depth: The force and angle of the cut can affect how deep an incision wound is.[14]
3. Bleeding Depending on the location and extent of the incision, wounds may bleed heavily.

Burn Wound:

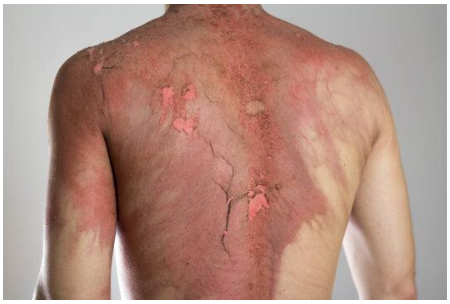

Fig No. 4 Burn Wound

Burn Injuries Burn wounds are injuries to the skin and possibly underlying tissues brought on by heat, fire, chemicals, electricity, or radiation.
Burn Wound Types:
1. First-Degree Burn: Cause pain, swelling, and redness in only the epidermis, the skin's outermost layer.
2. Second-Degree Burns: Cause blisters, redness, and swelling in both the dermis and epidermis.
3. Third-Degree Burns: These burns may appear white or burned and penetrate all layers of the skin, perhaps harming underlying tissues.
4. Fourth-Degree Burns: Inflict damage to muscles, bones, or organs beneath the skin.
Reasons:
1.Thermal Burns: Resulting from heat or fire.
2. Chemical Burns: Resulting from contact with caustic materials. When an electrical current flows through the body.[15]

Pharmacological Action :

The Pharmacology of Injuries The natural anatomical and functional continuity of tissues is disrupted by wounds. The process of their recovery is intricate and impacted by pharmacological, biochemical, and physiological elements. Understanding how medications, biological agents, and natural substances impact hemostasis, inflammation, proliferation, and remodeling—the several phases of wound healing—is the focus of wound pharmacology.[16]
1. Hemostasis Phase Objective: Create a clot and stop the bleeding. Pharmacological targets: Thrombin preparations, fibrin sealants, and tranexamic acid are examples of haemostatic drugs that hasten the development of clots. Adrenaline and epinephrine are examples of topical vasoconstrictors that lessen local bleeding.
2. Inflammatory Phase Objective: Clear germs and debris and get ready for repair.
Pharmacological targets: Anti-inflammatory drugs: corticosteroids and NSAIDs (ibuprofen, diclofenac) might lessen excessive inflammation, but they must be taken carefully because they may cause delayed recovery. Topical (silver sulfadiazine, mupirocin, iodine, honey) or systemic antibiotics are examples of antimicrobial medicines that help decrease

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microbial burden and infection. Immunomodulators, such as tacrolimus and pimecrolimus, alter the immune response in wounds that are chronic or caused by autoimmune diseases.[17]

3. Proliferative Phase Objective: Encourage re-epithelialization, angiogenesis, and granulation.

4. Remodeling (Coming of Age) Phase Objective: Restore function and strengthen tissue. Pharmacological targets: Collagen modulators are substances (such as zinc and vitamin C) that improve cross-linking. Silicone gels, onion extract, pirfenidone, and interferon-γ are anti-scarring agents. Inhibitors of matrix metalloproteinase (MMP): aid in halting excessive extracellular matrix deterioration in chronic wounds.[18]

5. Specific Wound Pharmacology Aspects Diabetic wounds can heal more quickly with the use of medications including topical insulin, metformin gels, and antioxidants. Silver-based antimicrobials, cerium nitrate, and biosynthetic burn dressings. For chronic or non-healing wounds, hyperbaric oxygen therapy and negative pressure wound therapy can be used in conjunction with other medication treatments.[19]

Factors Influencing the Healing of Wounds

1.Local elements (at the site of the wound) Oxygenation: Angiogenesis, collagen synthesis, and bacterial defence all depend on an adequate oxygen supply. Healing is delayed by hypoxia.

2Infection: Prolonged healing, tissue damage, and increased inflammation.

3.Balance of moisture: While dry wounds take longer to close, moist wounds heal more quickly (the ideal conditions for epithelial migration).

4.Supply of blood (perfusion): The supply of nutrients and oxygen is slowed by poor circulation (ischemia, peripheral vascular disease).[20]

5.Mechanical stress: Repair is hampered by excessive movement, pressure, or trauma.

6.Foreign bodies: Necrotic tissue, dirt, and sutures slow healing and encourage infection.

7.Edema: Decreases oxygen transport and raises tissue pressure.[21]

Factors that are systemic Age: Due to decreased cell turnover and inadequate circulation, elderly individuals heal more slowly. Proteins in nutrition: immune system function, collagen synthesis. Vitamins (Vitamin K for coagulation, Vitamin A for epithelialization, and Vitamin C for collagen). minerals (iron for carrying oxygen, zinc for synthesising DNA). Co-occurring conditions: Angiogenesis, neuropathy, and infection risk are all impacted by diabetes mellitus. Chronic liver and renal disease results in decreased toxin clearance and protein synthesis. Reduced supply of oxygen due to anaemia.[22]

Clinical Wound Management:

The goals of wound care include promoting optimal healing, preventing infection, relieving symptoms, and restoring tissue integrity. It entails a methodical, multidisciplinary strategy.

1. First Evaluation History: comorbidities, medications, and the cause of the wound (burn, diabetic, pressure ulcer, trauma, or surgery). Examine for necrosis, discharge, infection symptoms, size, depth, and kind (acute vs. chronic). Investigations (if necessary) include Doppler studies (for vascular status), blood sugar testing (for diabetic wounds), and wound swabs for culture.[23]

2. Preparing the Wound Bed (TIME Principle):T-Tissue management: Debridement, or the removal of necrotic or devitalized tissue. I. Control infection and inflammation: Apply topical or systemic antibiotics as needed, along with antiseptics. M-Moisture balance: To promote quicker epithelialization, keep the wound damp but not soggy. E-Edge advancement: Treat non-healing edges and encourage keratinocyte migration.

3. Debridement and Cleaning Normal saline, which is non-cytotoxic, is the ideal option for wound washing. Steer clear of harsh antiseptics like hydrogen peroxide and iodine unless there is an infection. Debridement methods: Sharp or surgical → necrosis removal right away. enzymatic (papain-urea, collagenase). autolytic (dressings made of hydrogel or hydrocolloid). biological (treatment with maggots).[24]

4. Choosing a Dress A moist, protecting, and infection-free environment should be maintained via dressings. Hydrogels and hydrocolloids for dry wounds. Alginate dressings and foam are used for exudative wounds. Silver dressings, iodine

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dressings, and honey are used to treat infected wounds. Silver sulfadiazine and biosynthetic dressings for burn injuries. Hydrocolloid, foam, and negative pressure wound therapy (NPWT) are methods for treating pressure ulcers.

5. Management of Infections Topical antimicrobials: povidone-iodine (short-term), honey, and silver. Systemic antibiotics: Only in cases of osteomyelitis, sepsis, or cellulitis.

6. Analgesics for Pain Management (NSAIDs, paracetamol). local anaesthetics (like lidocaine gel) for dressing changes.

7. Advanced Medical Treatments Negative Pressure Wound Therapy (NPWT): Enhances exudate clearance and granulation. Growth factors include VEGF, EGF, and PDGF. bioengineered tissue grafts and skin substitutes. PRP, or platelet-rich plasma. For diabetic or ischemic wounds, hyperbaric oxygen therapy (HBOT) is used. treatments based on stem cells.

8. Optimization of Systems management of underlying conditions (malnutrition, vascular disease, diabetes). Iron, zinc, vitamin C, and a high-protein diet provide nutritional support. Change of lifestyle: cut back on alcohol and quit smoking

9. Surgical Procedures split-thickness and full-thickness skin transplants. flap operations (free, regional, and local flaps).[25]

II. CONCLUSION

In conclusion from topical single-agent treatments to intricate combination tactics that incorporate biologics, biomaterials, antimicrobial management, and sensing/delivery technologies, pharmacological approaches to wound healing have advanced. Growth factors, PRP/MSCs, and multifunctional dressings all exhibit promise, but overcoming delivery, standardization, and cost obstacles is necessary to achieve steady clinical benefit. For the next generation of wound therapies to become standard practice, coordinated translational research, biologic product standardization, and practical clinical trials are necessary. A cut wound is a kind of injury that happens when a sharp object, like a knife, glass, or metal edge, cuts or tears the skin. Depending on their size, depth, and location, cut wounds can range in severity from superficial to deep and may need medical attention.

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