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SKIN BURN

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ABSTRACT

Skin burns are significant public health concern , affecting millions worldwide .This review aims to provide and update on the current understanding of skin burns, including introduction, classification, pathophysiology, factors affecting, methods, approaches, prevention, cause, and management strategies. We discuss recent advance in wound management pharmacological intervention and surgical options highlighting the need for disciplinary care in severe burns, the cells and blood vessels are often injured and blood supply to the wound is disturbed. Burn wound are one of the main causes of damage skin. The review also explores emerging therapies, such as stem cell therapy.

Keywords: Skin Burns, Pathophysiology, Approaches, Causes, Treatment, Prevention, Management.

I. INTRODUCTION

Skin involved epidermis, dermis and hypodermis layers and skin is the largest organ of the body. A skin burn is an injury to the organic tissue primarily caused by heat or due to radiation, radioactivity, electricity, friction or contact with chemicals. The skin is the prone to damage by microorganism and thermal, mechanical and chemical factors. An important cause of skin damage is burn wounds. Burn wound lead to various local and systemic pathophysiological process in body .The study of physiological and molecular basis of the cutaneous wound healing could leead to more therapeutic possibilities .In this review, we discuss about pathophysiological factors affecting and some therapeutic approaches.

Fig 1: Sikn Burn

II. CLASSIFICATION

1. First degree [superficial] burn:

First degree burn affect only the outer layer of akin the epidermis .The burn site is painful, red , dry and has no blisters

For example mild sunburn

2. Second degree [partial thickness] burn:

Second degree burns involve the epidermis and part of the lower layer of the skin, the dermis. The site looks blistered, red.

3. Third degree [Full thickness] burn:

Third degree burns destroy the epidermis and dermis. They may go into the innermost layer of skin the subcutaneous tissue. The burn site may look white.

4. Fourth degree

Fourth degree burns go through both layers of the skin and underlying tissue as well as deeper tissue ,possibly involving muscle and bone. Fourth degree burns no feeling because the nerve ending is destroyed.

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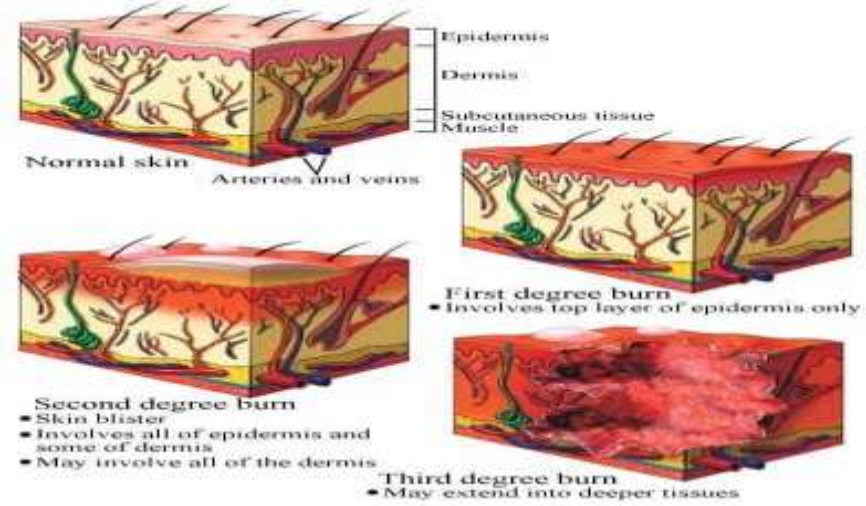

Fig 2: Classification

PATHOPHYSIOLOGY OF BURNS:

From various models of burns wound evaluation there is most commonly used is a Jacksons model. By using Jackson model 3 concentric area are detected or can be detected which can detects change in the blood flow of wounds.

Due to damage coagulation of protein and tissue necrosis maximum with irreversible tissue loss is found. This come under first zone which is the zone of coagulation which decreased perfusion. The ischemia is reversed because of the ischemic zone which is flow necrosis .Therefore, the main aim of burn is to increase tissue perfusion and prevent the futher damage. The outermost layer is the hyperemia of zone. Here is severe sepsis or prolonged hypoperfusion because of tissue perfusion is increased in zone of hyperemia. In a 2 phase pro inflammatory and anti-inflammatory response there is released of chemical factors from damage cell are activated .In a severe burn injury there is activation of first phase, a transcriptional activator means protein, nuclear factors KB in which tumor necrosis factors alpha [TNF -alpha] and intercellular adhesion molecule -2 [I GAM -2] to regulate the induction of several proinflammatory mediators

Neutrophils, monocytes and trigger this mediators are activated. Various cell in the wound area that are interleukins, and 6 [IL -1, IL -6] and apoptosis that TNF- alpha is responsible for the secretion of proinflammatory mediator. Hypermetabolism is increase because of thermal injuries which lead to increased production of anti-inflammatory, cytokines, reactive oxygen species [ROS],

T helper [TH] 2 lymphocytes and the secretion of 3 cytokines : IL -4 ,IL-10 and TNF- alpha are related of anti-inflammatory phase of burn injury.

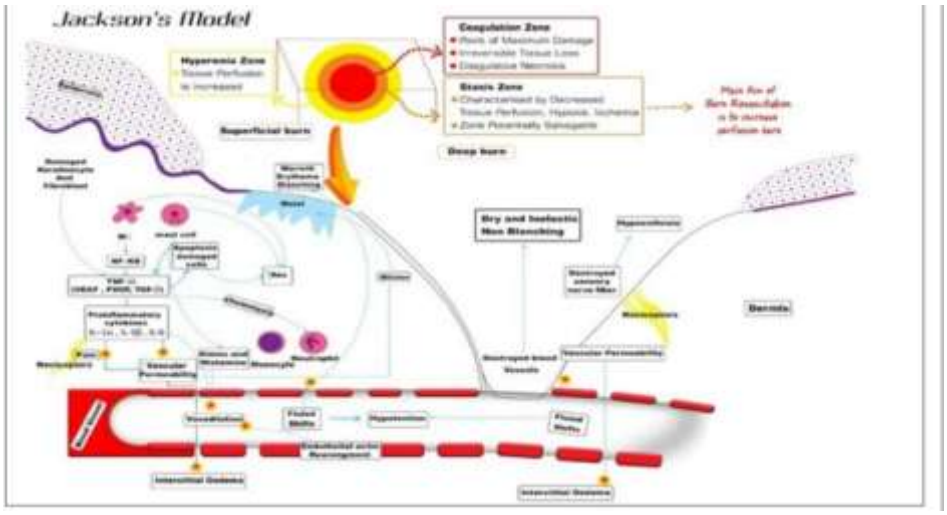

Fig 3: Jackson Model

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III. METHODS FOR SKIN BURNS

Assessment Method:

1) Rules of nines: Estimates total body surface area (TBSA) burned.
2) Lund-browder chart:Evalutes TBSA burned in pediatric patients.
3) Burn severity index (BSI):Assesses burn severity based on depth,size and location.
4) ABCDE(Airway, Breathing, circulation ,Disability ,Exposure)

Wound Management Methods:

1. Debridement: Removes dead tissue and promotes healing.
2. Dressing and wound care: Protects wound, promotes healing and prevents infection.
3. Topical Antimicrobials: Prevents infection and promotes healing .
4. Biologics and skin substituents: Enhance wound healing.

Surgical Methods:

1. Skin Drafting: Transfers healthy skin to burned areas.
2. Reconstructive surgery: Repairs damaged tissue and restores function.
3. Inflammation modulation: Corticosteroids and non-steroidal anti-inflammatory drugs (NSAIDS).

Emerging Methods:

1) Stem cell thearpy: Enhance wound healing and tissue regeneration.
2) Nanotechnology: Develops advanced wound dressings and tissue engineering scaffolds.
3) Gene Thearpy: Modulates inflammatory response and promotes wound healing.

Factor Affecting skin burns:

With various chemokines come together and leads to the wound healing due to which there is intiation at the topical response and it leads to constriction of impaired area.Due to sympathetics stimulation it leads to the formation of hypovoluemia renal failure and myocardical dysfunction.

High mortality rate leads to the progressive organ dysfunction. Nutrition aging , oxygen therapy and stress playes important role in systemic response.

Local Factor And Responses In Burn Injuries:

Local Factor:

1) Temperature: Thermal Damage
2) Duration: Length of exposure to heat
3) Depth: Penetration of burn into tissue
4) Area: Size of burned area
5) Location: Burns to face, hands hands, feet or genetila require special consideration.

Local Response:

1) Edema: Fluid accumulation in tissues.
2) Coagulation: Blood clotting and thrombosis.
3) Necrosis: Cell death and tissue damage
4) Infection: Bacterial colonization and invasion.
5) Inflammation: Burns to face, hands, feet or genitalia require special consideration.

Acute phase Response:

1) Systemic inflammatory responses syndrome.
2) Relase of acute phase proteins (e.g c-reactive protein)
3) Activation of complement system.

Healing phases:

1. Inflammatory phase (0-3 days)
2. Debridement phase (3-14 days)
3. Proliferation phase (14-21 days)
4. Remodeling phase (21+ days)

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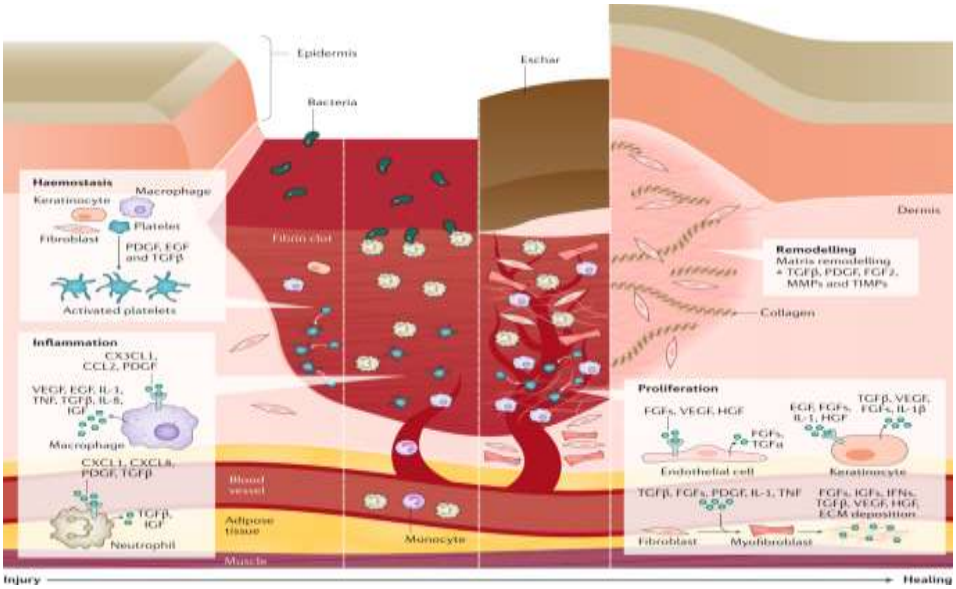

Fig 4: Healing Phases

Growth Factors involved in burns wound

Wound healing

Growth factors	Sources	Inflammatory function	Proliferation function	Tissue Remodeling function
PDGF	platelets	Affects activity of MMPS	*Promote angiogenesis *Accelerate ECM and collagen function. *Activate and proliferate fibroblasts	Involved in remodeling
EGF	keratinocytes		*stimulates epithelial cell proflieration and differentiation *Mitogenic for keratinocytes	Involved in reepithelialization
FGF	Keratinocytes Fibroblast Endothelial cells		*Involved in fibroblast chemotaxis *proliferates fibroblasts and keratinocytes.	Involved in wound contraction and repeithelization
VEGF	Keratinocytes Fibroblast Macrophages Endothelial cells		*promotes angiogenesis and lymphangiogensis *promotes endothelial cellular growth.	
HGF	Fibroblast		Mitogenic for endothelial and epithelial cells , melanocytes and keratinocytes	
TGF-beta	Fibroblast Keratinocytes Macrophages platelets	Recruits Macrophages and induces	*promotes angiogenesis *chemotactic for fibroblast *promotes fibroblast proliferation	Contribute to reepitheliazation and remodeling

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PDGF: Platelet -derived growth factor

ECM: Extracellular matrix

ECF: Epidermal growth factor

FGF: Fibroblast growth factor

VEGF: Vascular endothelial growth factor

HGF: Hepatocyte growth factor

TGF-Beta: Transforming growth factor beta

Approaches To Burn Wound Healing

First Aid:

In medical treatment first aid is an essential .Wounds cool at about 15 degree celcius for 20 miutes is current recommendation for the initial treatment of burns .The first aid technique increase the rate of reepithelialization, reduce pain, and improve the appearance and thickness of the resultant scar.

Activation of kallikrein in human plasma damaged tissues. Research also has shown cold water inhibits the release of histamine.

Traditional And Drug Treatments:

Cooling burn wounds with water eliminates heat, preventing further burn development . This is essential to carried out within 20 minutes by immersion in water at 15 degree celcius.

Chemicals burns should be washed with plenty of water.

Fig 5:

Moist wound healing:

Wound environment is defined as the environment direct contact with its surface.

Types of moist burn dressings:

TYPE	CHARACTERISTICS
Hydrocolloids	*Dressing composed of gelatin pectin and carboxymethylcellulose. *suitable for low -to-moderate exudative burns *suitable for all burn depths
Foams	*suitable for large burns *Usable with or without silver *Good absorption of exudate,therefore can help manage and prevent hypergranulation.
Alginates	*Highly absorbent , biodegradable dressing derived from seaweed *Useful for superficial bleeding ulcers.

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Hydrogels	*High water interactive dressing *Absorb Exudate *No harmful to granulation tissue or reepithelization

Future Direction For Skin Burn:

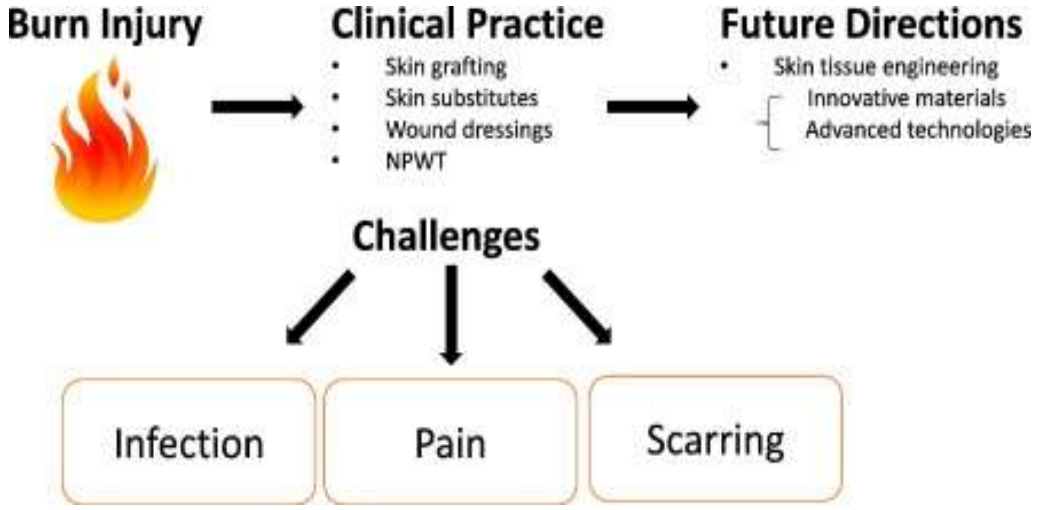

Fig 6:

- Cell based technique
- Skin bioprinting
- Artifical skin substitutes
- System biology and computational tools
- Oxandrolone

Treatment For A Burn

Fig 7:

Causes of skin burns:

< Fire

< Hot liquid or steam

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< Hot metal, glass or other objects
< Electrical currents
< Non solar radiation ,such as that from x-rays
< Sunlight or others sources of ultraviolet
< Chemicals such as strong acid ,lye paint thinner or gasoline
< Abuse

Complications of skin burns:

< infection
< Skin cancer
< Fluid loss
< Breathing problems
< Irregular heartbeat
< Scars and changes in skin color
< Pain
< Depression
< Anxiety

IV. CONCLUSION

Skin burn are a complex and multifaceted injury, requiring a comprehensive understanding of their pathophysiology ,classification, causes, complications ,future direction , treatment and management .This review highlights the advancement in burn care including wound management ,pharmacological intervention and surgical options .Despite these advancement ,skin burn remain a significant public health concern, with substantial morbidity and mortality.

It is critical that burn patients are treated as per their specific challenges and factor (e.g age, TBSA and comorbidities). Research and increased knowledge on burn pathobiology, infection, stem cells, transplantation and rehabilitation will cause improved individual care and treatment options.

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