Pathophysiology & Complications Of Burn
DEFINITION

Burns are wounds produced by various kinds of agents that cause cutaneous injury and destruction of underlying tissue.
TYPES OF BURNS

• Thermal injury
  – Scald—spillage of hot liquids
  – Flame burns
  – Flash burns due to exposure of natural gas, alcohol, or combustible liquids
    – Contact burns—contact with hot metals/objects/materials

• Electrical injury

• Chemical burns—acid/alkali

• Cold injury—frost bite

• Ionizing radiation

• Sun burns
Classification of Burns

1 Degree 4 (first, second, third, fourth)

2 Thickness 2 (partial, full thickness)

3 Percentage 3 (mild, moderate severe)
I. DEPENDING ON DEGREE

a. First degree:

- Epidermis is red and painful, No blisters
- Heals rapidly in 5-7 days
- By epithelialization without scarring.
b. Second degree:

- Red, painful, with blisters,
- Heals in 14-21 days.
- Superficial burn heals, causing pigmentation.
- Deep burn heals, causing scarring, and pigmentation.
• **Third degree:**
  • Charred, painless and insensitive,
  • Thrombosis of superficial vessels.
  • It requires grafting.

**-Eschar**

Charred, denatured, insensitive, contracted full thickness burn.

• These wound must heal by reepithelialisation from wound edge.
• **Fourth degree:**

  Involves the underlying tissues—muscles, bones.
II. DEPENDING ON THICKNESS OF SKIN INVOLVED

• a. **Partial thickness burns:**
  It is either first or second degree burn which is red and painful, often with blisters.

• b. **Full thickness burns:**
  It is third degree burns which is charred, insensitive, deep involving all layers of the skin.
DEPENDING ON THE PERCENTAGE OF BURNS

**Mild:**

- Partial thickness burns < 15% in adult or <10% in children.
- Full thickness burns less than 2%.
- Can be treated on outpatient basis.

**Moderate:**

- Second degree of 15-25% burns (10-20% in children).
- Third degree between 2-10% burns.
- Burns which are not involving eyes, ears, face, hand, feet, perineum.
**Major (severe):**

- Second degree burns more than 25% in adults, in children more than 20%.
- All third degree burns of 10% or more.
- Burns involving eyes, ears, feet, hands, perineum.
- All inhalation and electrical burns.
- Burns with fractures or major mechanical trauma.
JACKSON`S THERMAL WOUND THEORY

- Zone of coagulation
  Centre area of wound, where all tissues are damaged

- Zone of stasis
  Surrounds the coagulation area, some tissues are damaged

- Zone of hyperaemia
  Unburned area surrounds the stasis but it is red due to inflammation
ZONE OF COAGULATION (BURNED ZONE)

- The necrotic area of burn where cells have been disrupted
- This tissue is irreversibly damaged at the time of injury
ZONE OF STASIS

- The area immediately surrounding the necrotic zone.
- Decreased tissue perfusion.
- Can either survive (with good resuscitation) or go on to coagulative necrosis.
- Associated with vascular damage and vessel leakage
- Thromboxane A2, a potent vasoconstrictor is the main mediator
- Treatment aims to **spare** this zone to prevent its turnover to coagulative necrosis zone
ZONE OF HYPERMEIA

-is characterized by vasodilation from inflammation surrounding the burn wound

-contains the clearly viable tissue from which the healing process begins

-not at risk for further necrosis.
PATHOPHYSIOLOGY OF BURNS

Diagram showing energy expenditure over time with labeled phases:
- Ebb Phase
- Flow Phase
EBB PHASE (1ST 24 HOURS AFTER INURY)

• Occurs usually in the first 24 hours
• It’s initial period of hypofunction manifests as:
  (a) Hypotension
  (b) Low cardiac output
  (c) Metabolic acidosis
  (d) Hypoventilation
  (e) Hyperglycemia
  (f) Low oxygen consumption
  (g) Inability to thermoregulate

Responds to fluid resuscitation
The flow phase, Resuscitation

• Follows ebb phase and is characterized by gradual increases in:
  (a) Cardiac output
  (b) Heart rate
  (c) Oxygen consumption
  (d) Supranormal increases of temperature
HYPERMETABOLIC STATE

- Hypermetabolic hyperdynamic response peaks in 10-14 days after the injury after which condition slowly recedes to normal as the burn wounds heal naturally or surgically closed by applying skin grafting
SYSTEMIC RESPONSE TO BURN

- Metabolic
- Cardiac
- Renal
- Blood
- Immunologic
- Lungs
- GIT
- Edema
- Infections
Inflammatory mediators have a systemic response in >30% TBSA Burn.

They are:

1-Histamine
2-PGS
3-Thromboxane
4-Kinins
5-Serotonin
6-Catecholamines
7-Oxygen free radicals
8-CRF (corticotropin releasing factor)
9-Platelet aggregation factor
10-Angiotensin 2, Vasopressin
HISTAMINE

• Responsible for increased microvascular permeability seen immediately after burn.
• Released from mast cells in thermal-injured skin
• Its actions are only transient
PROSTAGLANDINS (PGS)

• Potent vasoactive

• Released from burned tissue and inflammatory cells

• Prostaglandin E2 (PGE2) and leukotrienes LB4 and LD4 directly and indirectly increase microvascular permeability

• PGE2 is a potent vasodilator, which, when coupled with the increased microvascular permeability amplifies edema formation
THROMBOXANE

- Thromboxane A2 (TXA2), thromboxane B2 (TXB2) produced locally in the burn wound by platelets
- TXA2 is a potent vasoconstrictor
- Decrease blood flow (ischemia) under the burn
- Cause the conversion of a partial-thickness wound to a deeper full-thickness wound
- Topically applied ibuprofen (synthesis of prostaglandins and thromboxanes) decreases both local edema without altering systemic production
• Bradykinin is a local mediator of inflammation that increases venular permeability
SEROTONIN

• Smooth-muscle constrictor of large blood vessels
• Antiserotonin agents such as ketanserin have been found to decrease peripheral vascular resistance after burn injury
CATECHOLAMINES

• Cause vasoconstriction
• Reduced capillary pressure may limit edema and induce interstitial fluid to reabsorb from nonburned skin, skeletal muscle, and visceral organs in nonresuscitated burn shock
• Via B-agonist activity, may also partially inhibit increased capillary permeability induced by histamine and bradykinin
• It has beneficial effect to reduce edema
OXYGEN RADICALS

• Superoxide anion (O2-), hydrogen peroxide (H2O2), and hydroxyl ion (OH-) from activated neutrophils

• The hydroxyl ion (OH-) is the most potent and damaging of the three

• Play an important inflammatory role in all types of shock

• High doses of antioxidant ascorbic acid *(vitamin-C)* have been found to be efficacious in reducing fluid needs in burn

• (10 – 20 g per day) of vitc
PLATELET AGGREGATION FACTOR

• increase capillary permeability
ANGIOTENSIN II AND VASOPRESSIN

- Participate in the normal regulation of extracellular fluid volume by controlling sodium balance and osmolality through renal function and thirst
- Both are potent vasoconstrictors of terminal arterioles
- Angiotensin II responsible for the selective gut and mucosal ischemia, which cause translocation of endotoxins and bacteria and the development of sepsis and even multi-organ failure
- Vasopressin, along with catecholamines responsible for increased system vascular resistance and left heart afterload, which can occur in resuscitated burn shock
CORTICOTROPHIN- RELEASING FACTOR (CRF)

- Reduce protein extravasation and edema in burn
- CRF may be is a powerful natural inhibitory mediator of the acute inflammatory response of the skin to thermal injury
2- CARDIAC

• Cardiac output decreases due to:
  1) Decreased preload induced by fluid shifts
  2) Increased systemic vascular resistance caused by both hypovolemia and systemic catecholamine release

• Cardiac output normal within 12-18 hours, with successful resuscitation

• After 24 hours, it may increase up to 2 ½ times the normal and remain elevated until several months after the burn is closed
3-RENAL

- Renal blood flow and GFR decrease soon after injury due to hypovolemia, decreased cardiac output, and elevated systemic vascular resistance.
- Oliguria and antidiuresis develops during 1st 12-24 hours.
- Followed by a usually modest diuresis as the capillary leaks seal, plasma volume normalizes, and cardiac output increases after successful resuscitation and coinciding with onset of the postburn hypermetabolic state, and hyperdynamic circulation.
BLOOD

• The red-cell mass decreases due to direct losses
• Immediate, 1-2 hours after, and delayed, 2-7 days postburn, hemolysis occurs due to damaged cells and increased fragility
• Anemia within 4-7 days is common
• Anemia persists until wound healing occur
• Early mild thrombocytopenia followed by thrombocytosis (2-4x normal) and elevated fibrinogen, factor V and factor VIII levels commonly by end of the 1st week
• Persistent thrombocytopenia is associated with poor prognosis -- suspect sepsis
• Mechanical barrier to infection is impaired because of skin destruction
• Immunoglobulin levels decreased as part of general leak and leukocyte chemotaxis, phagocytosis, and cytotoxic activity impaired
• The reticuloendothelial system's depressed bacterial clearance is due to decreases in opsonic (phagocyte) function
• These changes, together with a non-perfused, bacterially-colonized eschar overlying a wound full of proteinaceous fluid, put the patient in a significant risk for infection
EDEMA

• Injured tissue Increases permeability of entire vascular tree with loss of water, electrolytes and proteins from the vascular compartment, severe hemoconcentration occurs.
• Protein leakage causing hypoproteinemia, increase osmotic pressure in the interstitial space.
• Decreased cell membrane potential cause inward shift of Na\(^+\) and H\(_2\)O cellular swelling.
• In the injured skin, effect maximal 30 min after the burn but capillary integrity not restored until 8-12 hours after, usually resolved by 3-5 days.
• In non-injured tissues, only mild and transient leaks even for burns >40% BSA.
GIT

- Mucosal atrophy decreased absorption & increased intestinal permeability duto changes in gut blood flow

- Increased bacterial translocation and Septicemia

- Occurs within 12 hours of injury
  - Acute gastric dilatation which occurs in 2-4 days.
  - Paralytic ileus.
  - Curling’s ulcer (stress ulcer).
  - Acute a calculous cholecystitis, acute pancreatitis
  - Abdominal Compartment syndrome
INFECTIONS

- Streptococci (Beta haemolytic—most common)
- Pseudomonas
- Staphylococci
- Other gram-negative organisms
- Candida albicans
• Burn size greater than 40% TBSA, 75% of all deaths are due to infection

• **Causes:**

• Burn wound represents a susceptible site for opportunistic colonization by organisms

• Age, immunosuppressed status, extent of injury, and depth of burn in combination with microbial factors such as type and number of organisms, enzyme and toxin production and motility
• Aggressive early debridement of devitalized and infected tissue plus catheter related infections are the cornerstone of management of infections.

• Once an infection is disseminated hematogenously and becomes established in a burn patient, it is very difficult to eradicate, even with large does of broad-spectrum antimicrobial therapy

• Time-related changes in the predominant flora of the burn wound converts bacterial growth from gram-positive to gram-negative

• Treatment with two or more agents is becoming necessary in the management of these gram-negative invasive infections
SUMMARY OF PATHOPHYSIOLOGY

- Vascular permeability and edema
- Altered hemodynamics
- Immunosuppression
- Severe burn
- Hypermobolism
- Decreased renal blood flow
- Increased gut mucosal permeability
Complications of Burns

- Burn Shock
- Pulmonary complications due to inhalation injury
- Acute Renal Failure
- Infections and Sepsis
- Curling’s ulcer in large burns over 30% usually after 9th day
- Extensive and disabling scarring
- Psychological trauma
- Cancer called Marjolin’s ulcer, may take 21 years to develop
SEPSIS

- **Bacteremia**: presence of bacteria in the bloodstream without clinical manifestations
- **Sepsis**: bacteremia + clinical manifestations (fever, tachycardia, tachypnea)
- **Septic shock**: sepsis + refractory hypotension
SEPSIS

At least 3 of the following parameters:
- Temperature > 38.5 or < 36.5 °C
- tachycardia > 90 bpm in adults
- tachypnea > 30 bpm in adults
- WBC > 12000 or < 4000 in adults
- Refractory hypotension: SBP < 90 mmHg, MAP < 70, or

AND

Pathologic tissue source identified: > 105 bacteria on quantitative wound tissue biopsy or microbial invasion on biopsy.

- a SBP decrease > 40 mmHg in
- Thrombocytopenia: platelet count < 100,000/μ
- Hyperglycemia: plasma glucose > 110 mg/dl
- Enteral feeding intolerance (as diarrhea > 2500 ml/day for adults or > 400 ml/day in children)
CAUSES OF DEATH

- Hypovolaemia (refractory and uncontrolled) and shock
- Renal failure
- Pulmonary oedema and ARDS
- Septicaemia
- Multiorgan failure
- Acute airway block in head and neck burns