BURNS AND SOFT TISSUE INFECTIONS.

BURNS

An injury caused by exposure to heat or flame from any source.

Wounds are caused by exposure to:

1. Excessive heat
2. Chemicals
3. Fire/steam
4. Radiation
5. Electricity.
6. Scalds from hot/boiling liquids.
7. Friction from body and another surface.

- Results in 10-20 thousand deaths annually
- Survival is best at ages 15 - 45yrs
- Children, elderly, and diabetics have poor survival rates
- Survival is best for burns that cover less than 20% of TBA (total body area)

TYPES OF BURNS

a)Thermal

exposure to flame or a hot object

b)Chemical

exposure to acid, alkali or organic substances

c)Electrical

result from the conversion of electrical energy into heat. Extent of injury depends on the type of current, the pathway of flow, local tissue resistance, and duration of contact

d)Radiation

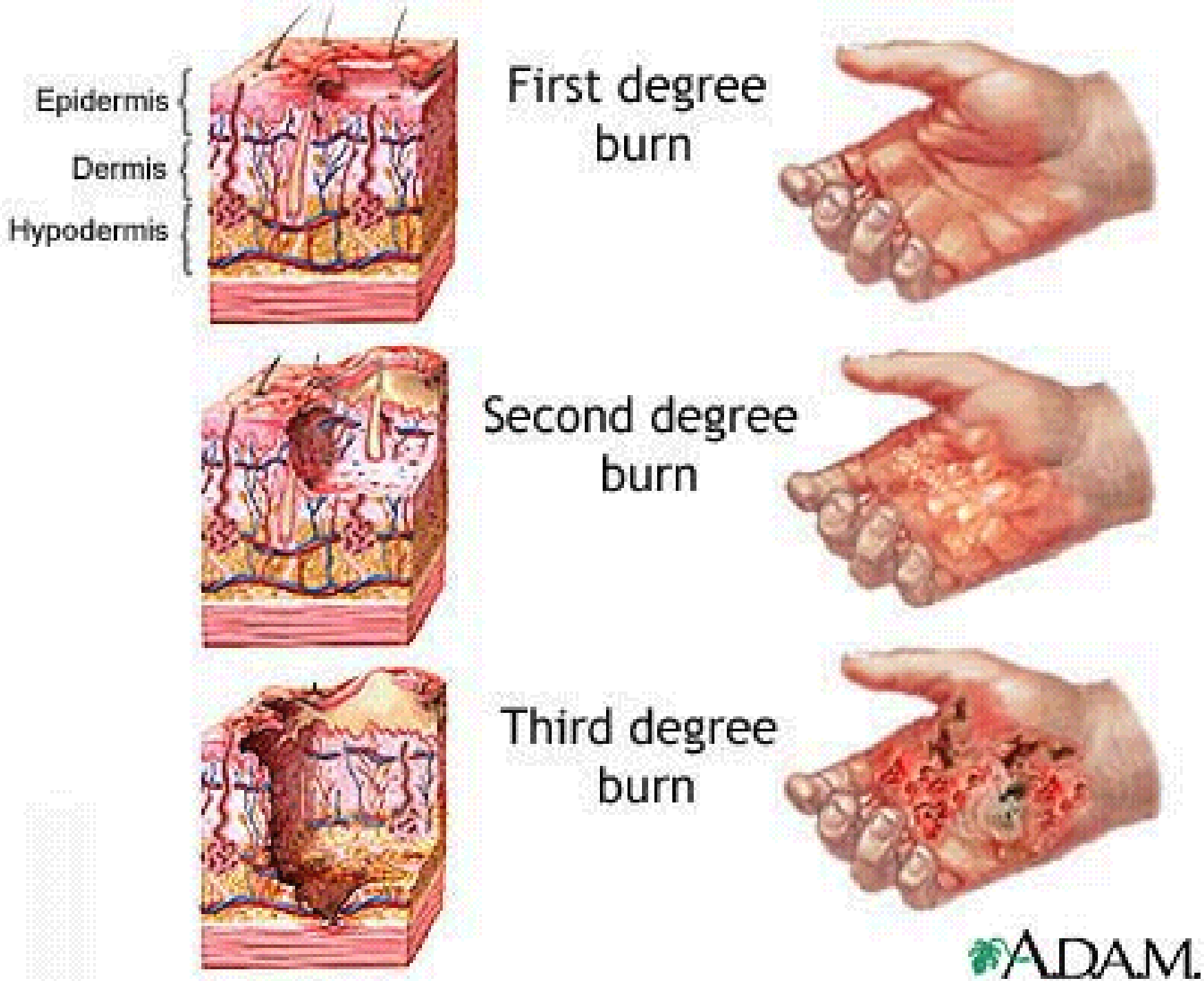
result from radiant energy being transferred to the body resulting in production of cellular toxins.

e)Friction burn

results from excessive and repetitive action between the body and a surface

BURN WOUND ASSESSMENT

- Classified according to depth of injury and extent of body surface area involved
- Burn wounds are differentiated depending on the level of dermis and subcutaneous tissue involved;
  1. superficial (first-degree)
  2. deep (second-degree)
  3. full thickness (third and fourth degree)


Epidermis
Dermis
Hypodermis
First degree burn
Second degree burn
Third degree burn
A.D.A.M.

SUPERFICIAL BURNS (FIRST DEGREE)

- Epidermal tissue only affected
- Erythema, blanching on pressure, mild swelling
  no vesicles or blister initially
- Not serious unless large areas involved
  i.e. sunburn

DEEP BURNS (SECOND DEGREE)

- Involves the epidermis and deep layer of the dermis
- Fluid-filled vesicles –red, shiny, wet, severe pain
- Hospitalization required if over 25% of body surface involved i.e. tar burn, flame

FULL THICKNESS BURNS (THIRD/FOURTH DEGREE)

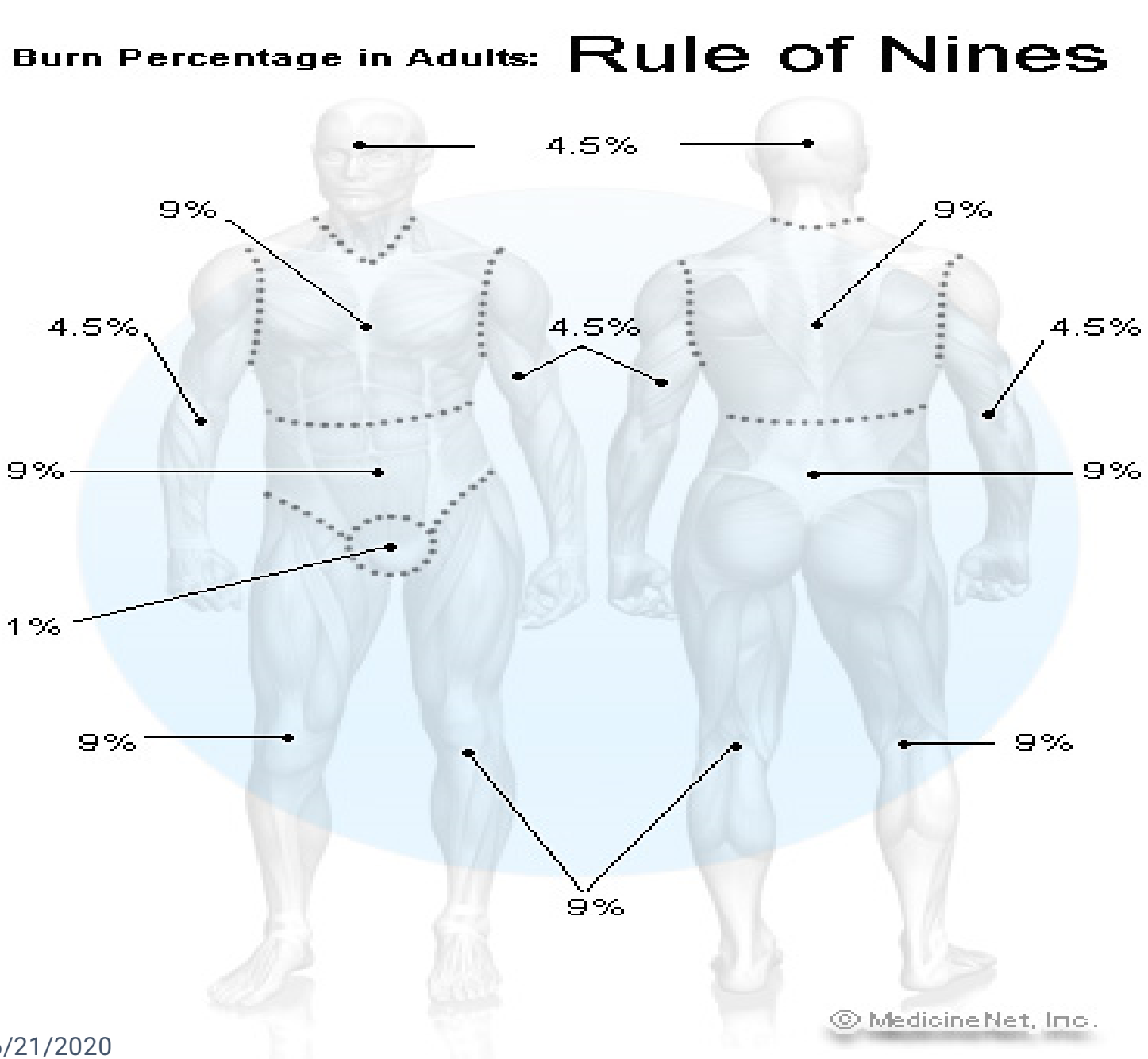
- Destruction of all skin layers
- Requires immediate hospitalization
- Dry, waxy white, leathery, or hard skin, no pain
- Exposure to flames, electricity or chemicals can cause 3rd degree burns

TOTAL BODY SURFACE AREA (TBSA)

- Superficial burns are not involved in the calculation
- Lund and Browder Chart is the most accurate because it adjusts for age
- Rule of nines divides the body – adequate for initial assessment for adult burns

RULE OF NINES

- Head & Neck = 9%
- Each upper extremity (Arms) = 9%
- Each lower extremity (Legs) = 18%
- Anterior trunk= 18%
- Posterior trunk = 18%
- Genitalia (perineum) = 1%


Burn Percentage in Adults: Rule of Nines
4.5%
9%
9%
4.5%
4.5%
4.5%
9%
9%
1%
9%
9%
9%
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Assessment of burns in children.

VASCULAR CHANGES RESULTING FROM BURN INJURIES

- Circulatory disruption occurs at the burn site immediately after a burn injury
- Blood flow decreases or cease due to occluded blood vessels
- Damaged macrophages within the tissues release chemicals that cause constriction of vessel
- Blood vessel thrombosis may occur causing necrosis
  - **Macrophage:** A type of white blood that ingests (takes in) foreign material. Macrophages are key players in the immune response to foreign invaders such as infectious microorganisms.

FLUID SHIFT

- Occurs after initial vasoconstriction, then dilation
- Blood vessels dilate and leak fluid into the interstitial space
- Known as third spacing or capillary leak syndrome
- Causes decreased blood volume and blood pressure
- Occurs within the first 12 hours after the burn and can continue to up to 36 hours

FLUID IMBALANCES

- Occur as a result of fluid shift and cell damage
- Hypovolemia
- Metabolic acidosis
- Hyperkalemia
- Hyponatremia
- Hemoconcentration (elevated blood osmolarity, hematocrit/hemoglobin) due to dehydration

FLUID REMOBILIZATION

- Occurs after 24 hours
- Capillary leak stops
- See diuretic stage where edema fluid shifts from the interstitial spaces into the vascular space
- Blood volume increases leading to increased renal blood flow and diuresis
- Body weight returns to normal
- See Hypokalemia

CURLING'S ULCER

- Acute ulcerative gastro- duodenal disease
- Occur within 24 hours after burns
- Due to reduced GIT blood flow and mucosal damage
- Treat clients with H2 blockers, mucoprotectants, and early enteral nutrition
- Watch for sudden drop in hemoglobin

PHASES OF BURN INJURIES

- Emergent (24 - 48 hrs.)
- Acute
- Rehabilitative

1.EMERGENT PHASE (24-48HRS)

Immediate problem is fluid loss, edema, reduced blood flow (fluid and electrolyte shifts)

Goals in management;

1. Secure airway
2. Support circulation by fluid replacement
3. Keep the client comfortable with analgesics
4. Prevent infection through wound care
5. Maintain body temperature
6. Provide emotional support

SKIN ASSESSMENT

- Assess the skin to determine the size and depth of burn injury
- The size of the injury is first estimated in comparison to the total body surface area (TBSA). For example, a burn that involves 40% of the TBSA is a 40% burn
- Use the rule of nines for clients whose weights are in normal proportion to their heights

IV FLUID THERAPY

- Infusion of IV fluids is needed to maintain sufficient blood volume for normal circulation.
- Clients with burns involving 15% to 20% of the TBSA require IV fluids
- Purpose is to prevent shock by maintaining adequate circulating blood fluid volume

Parklands formula applies:

1. 4mls/kg TBSA in 24hrs in addition to maintenance fluids.
2. Half of fluid given over the 1st 8hrs then 2rd half given over 16hrs.
3. Ringer's lactate is recommended in the 1st 24hrs and
   D5LR often used for children <20kgs.
4. Colloid/albumin after 24hrs to improve oncotic pressure.

2.ACUTE PHASE OF BURN INJURY

- Lasts until wound closure is complete
- Care is directed toward continued assessment and maintenance of the cardiovascular and respiratory system
- Pneumonia is a concern which can result in respiratory failure requiring mechanical ventilation
- Infection (Topical antibiotics – Silvadene)
- Tetanus toxoid
- Weigh daily without dressings or splints and compare to pre-burn weight
- A 2% loss of body weight indicates a mild deficit
- A 10% or greater weight loss requires modification of calorie intake
- Monitor for signs of infection

Local and systemic symptoms of infection.

- Pus from burn site
- High fevers.
- Conversion of a partial-thickness injury to a full-thickness injury
- Ulceration of health skin at the burn site
- Erythematous, nodular lesions in uninvolved skin
- GI dysfunction diarrhea, vomiting
- Metabolic acidosis

DRESSING THE BURN WOUND

- After burn wounds are cleaned and debrided, topical antibiotics are reapplied to prevent infection
- Standard wound dressings are multiple layers of gauze applied over the topical agents on the burn wound

3.REHABILITATIVE PHASE OF BURN INJURY

- Started at the time of admission
- Technically begins with wound closure and ends when the client returns to the highest possible level of functioning
- Provide psychosocial support
- Assess home environment, financial resources, medical equipment, prosthetic rehabilitation
- Health education should include symptoms of infection, drug regimens, follow up appointments, comfort measures to reduce pruritus.

DIET

- Initially NIL BY MOUTH
- Begin oral fluids after bowel sounds return
- Do not give ice chips or free water; leads to electrolyte imbalance
- High protein, high calorie

General management

- Prevent complications (contractures)
- Assess respiratory function
- Tetanus booster
- Antibiotics
- Analgesics
- No aspirin
- Strict surgical asepsis
- Turn q2h to prevent contractures
- Emotional support

SOFT TISSUE INFECTIONS

- S.T.I are diseases that involve the skin and underlying subcutaneous tissue, fascia, or muscle.
- May be localized to a small area or involve a large portion of the body.
- Affect any part of the body, though the lower extremities, the perineum, and the abdominal wall are the most common sites.
- Some S.T.I.s are harmless if treated promptly and adequately; others can be life-threatening even when appropriately treated.

Aetiology of Soft Tissue Infection.

- Soft tissue infection commonly results from inoculation of bacteria through a defect in the epidermal layer of the skin, such as may occur with injury, pre-existing skin disease, or vascular compromise.

- Less commonly, soft tissue infection may be a consequence of extension from a subjacent site of infection (e.g., osteomyelitis) or of haematogenous spread from a distant site (e.g., diverticulitis or C. septicum infection in patients with colonic carcinoma).

- It may also occur de novo in healthy patients with normal-

SIGNS AND SYMPTOMS.

Subtle or nonspecific indicators:-

-pain,
-localized tenderness, and
-oedema without fever.

Obvious features:-

-necrosis,
-blistering, and
-crepitus associated with systemic toxicity.

Classifications.

- Nonnecrotizing STI- involve one or both of the superficial layers of the skin (epidermis and dermis) and the subcutaneous tissue, and they usually respond to antibiotic therapy alone.

- Necrotizing STI - involve not only the skin, the subcutaneous tissue, and the superficial fascia but also the deep fascia and muscle, and they must be treated with urgent surgical debridement.

- At times, it is difficult to distinguish between these two categories of infection, especially when obvious clinical signs of necrotizing soft

Pathogenesis of STIs

- Soft tissue infections generally induce localized inflammatory changes in the involved tissues, regardless of the species of bacteria involved.

- As the infection progresses, tissue necrosis occurs as a result of:-

(1) Direct cellular injury from bacterial toxins.
(2) Significant inflammatory oedema within a closed tissue compartment.
(3) Thrombosis of nutrient blood vessels.
(4) Tissue ischemia.

Superficial infections.

- Pyoderma, Impetigo, Folliculitis, Furuncles and carbuncles, Infections .

  developing in damaged skin, Animal bites Human bites,

  Cellulitis, Nonnecrotizing, Necrotizing.

- **Deep necrotizing cutaneous infections.**

- Necrotizing fasciitis, Myonecrosis, Gas gangrene, Metastatic

  gas gangrene

Environmental Factors That Disrupt Skin and Alter Normal Barrier Function.

- Cuts, lacerations, or contusions, Injections from contaminated needles
-Animal, human, or insect bites.
-Burns.
-Skin diseases (atopic dermatitis, tinea pedis,eczema, scabies
-Decubitus, venous stasis, or ischemic ulcers.
- Contaminated surgical incisions.

Signs and Symptoms

- Take history and do physical examination.
- Patients have pain, tenderness, and erythema of recent onset.
- Ask environmental factors that have disrupted normal skin barrier,

  any host factors that increase their susceptibility to infection.
- Ask about clinical scenarios associated with unusual pathogens, such

  as an animal bite (Pasteurella multocida), a human bite (Eikenella

  corrodens), chronic skin disease (Staphylococcus aureus), saltwater

  exposure (Vibrio vulnificus), and freshwater exposure (Aeromonas

  hydrophila).

Investigations

- Needle aspirate pus c/s
- Blood full haemogram and ESR, c/s
- X –rays
- C T scans
- MRI
- Ultra sounds

General Management of Non-necrotizing & Necrotizing Soft Tissue Infection.

- Components of treatment of necrotizing soft tissue infection are;-

(1) Resuscitation & correction of fluid & electrolytes.

(2) Physiologic support.

(3) Broad-spectrum antimicrobial therapy.

(4) Urgent and thorough debridement of necrotic tissue.

(5) Supportive care .

Antibiotics for Adult Patients with Necrotizing Soft Tissue Infections.

- **Single agents**

-Ampicillin-sulbactam.

-Imipenem-cilastatin.

-Meropenem.

-Piperacillin-tazobactam.

-Ticarcillin-clavulanate.

- **Agents used in combination regimens**

- **Aerobic/facultative coverage**

  Ampicillin
  Cefotaxime
  Ceftazidime
  Cefuroxime
  Ciprofloxacin
  Gentamicin
  Vancomycin

- **Anaerobic coverage**

  Clindamycin
  Metronidazole

Organisms Causing Necrotizing Soft Tissue Infection.

- **Aerobes.**

-Gram-positive

Group A *Streptococcus*
Enterococcus species
Staphylococcus aureus
Group B *Streptococcus*
Bacillus species.

-Gram-negative

Escherichia coli
Pseudomonas aeruginosa.
Enterobacter cloacae
Klebsiella species
Serratia species
Acinetobacter calcoaceticus
Vibrio vulnificus

- **Anaerobes.**

- *Bacteroides* species
  Clostridium species
  Peptostreptococcus species

DIABETIC FOOT

•Epidemiology

- DM is the largest cause of neuropathy.
- Half don't know
- Foot ulcerations is most common cause of hospital admissions for Diabetics
- Expensive to treat, may lead to amputation and need for chronic institutionalized care
- After amputation 30% lose other limb in 3 years
- After amputation 2/3rds die in five years
- Type II can be worse
- 15% of diabetic will develop a foot ulcer

•Pathophysiology

- ? Vascular disease?
- Neuropathy: Sensory, Motor and autonomic

Vascular Disease

- 30 times more prevalent in diabetics

- Diabetics get arthrosclerosis obliterans or “lead pipe arteries”

- Calcification of the media

- Often increased blood flow with lack of elastic properties of the arterioles

- Not considered to be a primary cause of foot ulcers

Neuropathy

- Changes in the vasonervorum with resulting ischemia?

cause

- Increased sorbitol in feeding vessels block flow and causes nerve ischemia.
- Intraneural accumulation of advanced products of glycosylation.

- Abnormalities of all three neurologic systems contribute to

ulceration.

- *Autonomic Neuropathy:* Regulates sweating and perfusion

to the limb

- Loss of autonomic control inhibits thermoregulatory function and sweating.

- Result is dry, scaly and stiff skin that is prone to cracking and allows a portal of entry for bacteria.

- *Motor Neuropathy:* Mostly affects forefoot ulceration.

- Intrinsic muscle wasting – claw toes

- Equinous contracture

- *Sensory Neuropathy:* Loss of protective sensation

- Starts distally and migrates proximally in “stocking” distribution

- Large fiber loss – light touch and proprioception

- Small fiber loss – pain and temperature

- Usually a combination of the two

- Two mechanisms of Ulceration:

- Unacceptable stress few times-rock in shoe, glass, burn

- Acceptable or moderate stress repeatedly-Improper shoe

ware and deformity

CLASSIFICATION

Wagner's Classification

0 – Intact skin (impending ulcer).

1 – Superficial.

2 – Deep to tendon bone or ligament.

3 – Osteomyelitis.

4 – Gangrene of toes or forefoot.

5 – Gangrene of entire foot

TREATMENT

- Patient education

- Ambulation

- Shoe ware

- Skin and nail care

- Avoiding injury e.g. Hot water and F.B's.

- Medical:- Optimized glucose control-decreases by 50% chance of foot problems.

After ulcer healed

- Orthopedic shoes with accommodative (custom made insert).

- Education to prevent recurrence

Wagner 0-2

- Total contact cast
- Distributes pressure and allows patients to continue ambulation
- Principles of application: Changes, Padding, removal
- Antibiotics if infected
- Surgical if deformity present that will reulcerate
- Correct deformity
- Exostectomy

Wagner 3

- Excision of infected bone

- Wound allowed to granulate

- Grafting (skin or bone) not generally effective

Wagner 4-5

- Amputation