Burns and thermal injury
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1. Burns
    a. Burn injuries account for 500, 000 medical visits annually (1)
        i. 40,000 require hospitalization
    b. The depth and extent of injury depend on the mechanism of burn and duration of exposure to the heat source (2).
        i. Scald burns are associated with a significant inflammatory response
        ii. Flame burns are associated with an increased risk of inhalation injury which is an independent risk factor for mortality
        iii. Chemical burns may have ongoing tissue damage even after removal of the inciting agent
            1. Hydrofluoric acid is associated with significant systemic toxicity
        iv. Electrical burns are associated with underlying myonecrosis and require close monitoring for cardiac abnormalities, compartment syndrome, and rhabdomyolysis
        v. Outcomes following burn injury are directly related to:
            1. Burn depth,
            2. % total body surface area (TBSA) involvement
            3. Age
            4. Proper care in a specialized burn center
    c. Depth of burn (2)
        i. First-degree burns are characterized by erythema, absence of blistering, and pain.
            1. These burns are not included in calculation of burn size
        ii. Superficial partial-thickness burns involve the entire epidermis
            1. Form blisters and are erythematous, painful, and blanch with pressure
            2. Typically heal within 2 weeks without intervention
        iii. Deep partial thickness burns behave like third-degree burns.
            1. Blister with a mottled pink and white appearance due to partially damaged blood vessels.
            2. Do not blanch and are less painful due to nerve injury.
            3. Controversy exists about whether all of these burns require surgical debridement and grafting or whether healing can occur without surgical intervention
        iv. Full-thickness, or third-degree, burns involve all skin layers
            1. May be a variety of colors and do not blanch with pressure.
            2. Insensate from superficial nerve injury.
            3. Require definitive surgical management.
    d. %TBSA
        i. The calculated %TBSA is an independent risk factor for length of stay and mortality (3)
        ii. The “rule of nines” is a rough estimation of adult body surface area
            1. May overestimate burn size in children
            2. Lund-Browder diagrams improve the accuracy of the % TBSA for children
    e. Initial Management
        i. Airway
            1. Rapid securing of the airway particularly if smoke inhalation is suspected.
            2. Clinical findings such as carbonaceous sputum, hypoxia, deep facial burns, or a hoarse voice, mandate early endotracheal intubation (4)
        ii. Resuscitation
            1. First line therapy is crystalloid infusion.
                a. Fluid administration is guided by the Parkland or modified Brooke formulas TBSA with ½ given in the first 8 hours and the rest over the next 16 hours
                b. Parkland formula - 4 mL/kg per %TBSA
                c. Modified Brooke formula - 2 mL/kg per %TBSA
            2. Lactated Ringers is usually used
            3. Resuscitation should be titrated to urine output (0.5 ml/kg/hour in adults and 1 ml/kg/hour in children) and hemodynamic parameters.
            4. Recent data suggests that lower volume resuscitation may be beneficial in prevent complications such as abdominal compartment syndrome (5)
            5. Use of colloids after the first 24 hours was previously recommended and is still a matter of some debate
                a. A reduction in complications and mortality has not been demonstrated prospective human trials and should be reserved for patients unresponsive to resuscitation with crystalloid

iii. Current American Burn Association (ABA) guidelines recommend the transfer of patients to a specialized burn center for:
    i. Patients at extremes of age
    ii. Large burns
    iii. Burns involving critical anatomy (face and hands)

f. ICU Management
  i. Traditional management strategies as for any critically ill patients
  ii. Special attention is given to
    1. Aggressive control of hyperglycemia. Particular care should be taken to avoid hypoglycemia (4)
    2. The early initiation of enteral nutrition in burn patients is widely accepted (4)
      a. A multicenter study looking at feeding within 24 vs. 48 hours of admission found that the group fed within 24 h had a shorter average ICU stay and lower rates of wound infection compared to those with delayed feeding (6)

g. Complications
  i. Compartment syndromes are classically complications of "over-resuscitation"
    1. Limb compartments
      a. Severe pain (worse with movement), numbness, cool extremity, tight feeling compartments
      b. Compartment pressure >30 mmHg
      c. Escharotomies are first line
        i. Performed laterally and medially throughout entire limb
      d. Fasciotomies may be needed if pressure does not drop after escharotomies
    2. Chest Compartment Syndrome
      a. Increased peak inspiratory pressure (PIP) due to circumferential trunk burns
      b. Escharotomies through mid-axillary line, horizontally across chest/abdominal junction
    3. Abdominal Compartment Syndrome
      a. Pressure in peritoneal cavity > 30 mmHg
      b. Signs include increased PIP, decreased urine output, hemodynamic instability
      c. Treatment
        i. Escharotomy first
        ii. Possible placement of peritoneal catheter to drain fluid
        iii. Abdominal decompression
    4. Acute Respiratory Distress Syndrome (ARDS)
      a. Increased risk and severity if over-resuscitation or with smoke inhalation
      b. Treatment largely supportive
        i. High-frequency percussive mode of ventilation (HFPV) has been used to prevent the development of ARDS and to support patients who develop ARDS following burns and smoke inhalation (4).
    5. Sepsis and MODS
      a. Sepsis is an independent risk factor of mortality following thermal injury (2)
      b. Breakdown of barrier function following burn injury provides the largest entrance point for infection.
      c. Burn wound infection is the most common, followed by pneumonia, bacteremia, and urinary tract infections

h. Wound care (2)
  i. Initial care
    1. All burn wounds should be washed with tepid water following removal of the heat source. Ice or iced water increases tissue damage and should be avoided
    2. Chemical burns must be copiously irrigated for a minimum of 15 minutes.
    3. Initially wounds should be covered with clean, dry material or nonadherent gauze.
      a. The use of wet dressings should be avoided
  ii. ICU care
    1. Cleansing and debridement of the wound is typically done with mild soap and water or chlorhexadine/normal saline washes.
    2. Debridement of all blisters larger than 0.5 cm to minimize bacterial colonization or infection.
    3. Bacterial colonization of burn wounds does not require systemic antibiotics but should be managed with early debridement and/or excision, together with appropriate topical and/or biologic dressings.

4. Topical antimicrobials agent intended to minimize colonization, not sterilize wounds
    a. Commonly utilized topical agents include silver sulfadiazine (Silvadene), mafenide acetate (Sulfamylon), and silver nitrate.

i. Surgical Management (2)

i. Deep burns are managed with surgical excision and placement of xenograft, allograft, autograft, or cultured skin substitutes

ii. Current recommendations are for early excision within the first 1 to 7 days

iii. The appropriate timing for burn wound excision and grafting involves a number of important factors including the age of the patient, extent and depth of burn, comorbidities, hospital resources, and physician preference.

iv. Staged approaches may be preferable

2. Smoke inhalation injury (7)
    a. Pathophysiology
        i. Smoke particles settle in distal bronchioles
        ii. Mucosal cells die and slough causing distal atelectasis
        iii. Increase risk for pneumonia
    b. Diagnosis
        i. History of being in a smoke-filled enclosed space
        ii. Bronchoscopy – gold standard diagnostic test
            1. Soot beneath the glottis
            2. Airway edema, erythema, ulceration
        iii. Nondiagnostic clinical tests
            1. Early chest x-ray
            2. Early blood gases
        iv. Nondiagnostic clinical findings
            1. Soot in sputum or saliva or singed facial hair
    c. Treatment
        i. Supportive pulmonary management
        ii. Aggressive respiratory therapy
            1. Nonstandard methods of ventilation such as volumetric diffusive respiratory (VDR) and airway pressure release ventilation (APRV) modes may be beneficial
        iii. Adjunctive therapies such as aggressive pulmonary toilet, nitric oxide, nebulized heparin, N-acetylcysteine, and/or bronchodilators should be considered

3. Carbon monoxide (CO) exposure (7)
    a. Byproduct of incomplete combustion
    b. Binds hemoglobin with 200 times the affinity of oxygen
    c. Leads to left-ward shift of the oxygen hemoglobin dissociated curve and impaired release of oxygen at the cellular and tissue level
    d. Diagnosis of CO poisoning
        i. History of exposure
        ii. Nondiagnostic studies
            1. PaO_2
            2. Oximeter (difference in oxy- and deoxyhemoglobin)
            3. Patient color ("cherry red")
        iii. Diagnostic
            1. Carboxyhemoglobin levels
                a. <10% is normal
                b. >40% is severe intoxication
    e. Treatment
        i. Remove source
        ii. 100% oxygen until CO levels are <10%
        iii. Hyperbaric referral

4. Electrical Injuries (7)

a. Electrical injuries mandate tailored evaluation given the propensity for compartment syndrome, cardiac dysrhythmias, muscle necrosis, and multiorgan system involvement

    b. Extent of injury may not be apparent
        i. Damage occurs deep within tissues
        ii. Damage frequently progresses

iii. Electricity contracts muscles, so watch for associated injuries

c. Cardiac arrhythmias may occur
   i. Telemetry is indicated

d. Rhabdomyolysis can occur
   i. Myoglobinuria needs to be treated
      1. Increase fluids to induce high volume urine output
      2. ?Alkalinize urine
      3. ?Mannitol or other diuretics
      4. Check for compartment syndrome

e. Compartment syndromes are common

5. Hypothermia

a. Is the unintentional lowering of body temperature to < 35^0C

b. 4 phases (8)
   i. Mild hypothermia (32–35°C)
      1. Physiological responses to reserve heat (e.g. shivering, vasoconstriction, cold diuresis)
      2. Characterized by mild states of confusion, hepatic dysfunction and hyperglycemia
   ii. Moderate hypothermia (28–32°C)
      1. Violent shivering
      2. Severe confusion with disorientation, delirium and memory loss.
   iii. Severe hypothermia (20–28°C) and profound hypothermia (less than 20°C)
      1. Cardiac dysrhythmias, shallow breathing and a progressive decline in consciousness leading to death

c. Effects of hypothermia:
   i. Vasoconstriction
   ii. Shivering to increase basal metabolic rate and heat production
   iii. Cardiac changes (9)
      1. Initial tachycardia followed by bradycardia
      2. ECG changes – initially PR, QRS, and QTc prolongation and T-wave changes. "J wave" or Osborne wave is characteristic
      3. Atrial fibrillation below 32°C
      4. Refractory VF below 28°C
   iv. Global neurological changes (including decreased peripheral nerve conduction)
      1. Typically coma below 32°C
   v. Hemodilution from increased vascular permeability
   vi. Coagulopathy due to inhibition of the clotting cascade
   vii. Renal dysfunction from tubular dysfunction, renal artery vasoconstriction, inhibition of ADH
      1. Cold diuresis due to inability to concentrate urine as well as relative central hypervolemia form peripheral vasoconstriction
   viii. Hyperglycemia from catecholamine-induced glyconeogenesis and reduced insulin release

d. Management
   i. ABG may be needed at pulse-oximetry notoriously will not "pickup" or be inaccurate
   ii. Patient in cardiac arrest
      1. CPR should be initiated for any patient who is pulseless or has a nonperfusing rhythm while rewarming is initiated
      2. Avoidance of any cardiac irritation with lines, etc. if possible.
      3. V Fib will often be refractory to defibrillation at temperatures <32°C
   iii. Hypotension is treated first with administration of IV fluid
   iv. Evaluation for associated conditions, such as trauma, is essential
   v. Rewarming
      1. Passive rewarming
         a. Used in mild hypothermia for young, relatively healthy patients with intact shivering
         b. Expose patient and apply warm dry blankets
         c. Rewarms at 0.2–0.85°C/hour (10)
      2. Active external rewarming

a. Used for patients with mild to moderate hypothermia or for individuals with significant comorbidities
b. Expose patient and apply forced air rewarming and/or resistive heating methods, such as electric blankets
c. Rewarms at 0.9–3.3°C/hour (10)

3. Active core rewarming
    a. Used for severe hypothermia (< 30°C)
    b. Techniques include (9):
        i. Airway rewarming with arm air during intubation - 0.05–0.5°C/hour
        ii. Gastrointestinal irrigation with boluses of warm fluid via nasogastric tube - 1.5°C/hour
        iii. Peritoneal or pleural lavage
        iv. Cardiopulmonary bypass or dialysis - >9°C/hour
        v. Many of the newer devices designed for use in therapeutic hypothermia also having a warming application
4. Care must be taken with rewarming due to the development of:
    a. Arrhythmias
    b. Hypotension
    c. Cardiogenic shock or "rewarming shock" due to increased metabolic rate and vasodilatation
    d. Electrolyte abnormalities, especially hyperkalemia
    e. Profound hypoglycemia

6. Frost bite, etc.
    a. There are a number of conditions associated with unintentional hypothermia, including: (9)
        i. Frostnip
            1. Ice crystal deposition in the dermis
            2. Involves only the dermis
            3. Pale, numb, stiff tissue
            4. Treat with immersion of the affected area in warm water (37-41°C)
        ii. Frostbite
            1. Frozen skin surfaces
            2. Involves both dermis and deeper tissues
            3. Pale, numb stiff tissue
            4. Several phases (11)
                a. "pre-freeze phase," "freeze-thaw phase," "vascular stasis phase," and "late ischemic phase"
            5. Treat with immersion of the affected area in warm water (37-41°C) until tissue is warm and pliable (12)
                a. Rewarming is painful
                b. Tetanus and antibiotics for any suspected infected necrosis
                c. Early surgical debridement is contraindicated until the tissue demarcates
                    i. Exceptions: Compartment syndrome, "freeze-thaw-refreeze" injuries, or extremity trauma (11)
                d. Technetium 99 (Tc-99) triple phase scanning or MRI may be helpful in determining the extent of injury (12)
                e. Intra-arterial or intravenous thrombolysis may be considered in patients who present within 24 hours of a severe frostbite injury in whom major tissue loss is predicted (12).
        iii. Trench foot
            1. Due to immersion in cold water
            2. Tissue necrosis without freezing
            3. Pale skin with sloughing
            4. Treat with immersion of the affected area in warm water (37-41°C)
        iv. Chilblains or pernio
            1. Repeatedly partially frozen and thawed epidermis
            2. Erythematous, scaly ulcers that are painful and pruritic
            3. Treat with Calcium channel blockers

7. Hyperthermia
    a. Minor syndromes - heat edema, heat cramps, and heat exhaustion
    b. Major syndromes - heat stroke

i. Either exertional or non-exertional (very young and very old)
ii. Defined as the triad of: core temperature > 40.5°C (104.9°F), central nervous system (CNS) dysfunction, and anhidrosis

c. The prognosis is directly related to the degree of hyperthermia and its duration
d. Clinical manifestations
i. CNS dysfunction
ii. Tachycardia
iii. Tachypnea and respiratory alkalosis
iv. Metabolic abnormalities such as hypokalemia
v. Hypoglycemia with exertional heat stroke
e. Treatment (13)
i. Rapid reduction of the core temperature
1. Active cooling should stop when the core body temperature falls to 38°C–38.5°C
2. Evaporative methods
a. Constant moistening of the skin plus a fan to maximize heat loss
b. Complicated by shivering which will increase heat production
i. Treat with IV dexmedetomidine, benzodiazepines, buspirone, meperidine or warming of the hands, feet, and face ("skin counterwarming")
3. Conduction techniques include non-invasive water immersion and ice pack or cooling blanket application or newer commercially available devices (ARCTIC SUN®)
4. Invasive techniques
a. Gastric lavage
b. Peritoneal lavage
c. Cold bladder irrigation
d. Thoracic lavage
e. Devices designed for use in therapeutic hypothermia and fever management (Philips InnerCool RTx, ZOLL's Intravascular Temperature Management (IVTM™), etc.)
f. Extracorporeal methods (CRRT, ECMO) in extreme cases
f. Complications of heat stroke include renal failure, hepatic failure, DIC, rhabdomyolysis, intestinal ischemia, vascular collapse, GI bleed, and neurologic injury

8. Neuroleptic Malignant Syndrome (NMS)
a. Is an idiosyncratic reaction to neuroleptic and antidopaminergic agents
b. Characterized by muscular rigidity, altered mental status, autonomic dysfunction, and hyperthermia.
i. The hyperthermia is generally less severe and life-threatening than in MH.
c. Typically develops within a few days of initiation of therapy.
i. Has also been reported with sudden withdrawal of neuroleptic medications
d. Diagnosis is suggested by (14,15):
i. Treatment with neuroleptics within 7 days

ii. Hyperthermia $>38^0$ C

iii. Muscle rigidity
iv. Exclusion of other systemic or drug-related illnesses
v. Any 5 of the following:
1. Altered mental status
2. Tachycardia
3. Hypo- or hypertension
4. Tachypnea or hypoxia
5. Diaphoresis
6. Tremor
7. Incontinence
8. Elevated CK or myoglobinuria
9. Leukocytosis
10. Metabolic acidosis
e. Treatment includes immediate removal of the offending agent and supportive measures
i. Treatment of hyperthermia with fluid resuscitation and cooling measures

ii. Careful monitoring of airway due to development of dystonia and bulbar dysfunction
iii. Use of dopamine agonists such as bromocriptine (2.5 mg q8h), amantadine, and levodopa/carbidopa
iv. Dantrolene may be used to relax muscle, decrease fever, and sequelae of rigidity
v. Neuromuscular blockade can be used for severe cases

9. Malignant Hyperthermia (MH)
    a. Associated with certain anesthetic agents causing severe muscle contractions and life-threatening hyperthermia
    b. Classically associated with the volatile anesthetics (isoflurane, sevoflorane, etc.) and muscle relaxants
    c. Causes rapid increase in intracellular hypercalcemia in skeletal muscle due to a defective protein in the sarcoplasmic reticulum.
        i. The intracellular hypercalcemia causes marked increase in actin and myosin activity leading to uncontrolled tetany. This activates metabolic pathways that result in adenosine triphosphate (ATP) depletion, acidosis, membrane destruction, and ultimately cell death.
        ii. The surge in metabolic activity causes production of heat leading to total body rigidity and extreme hyperpyrexia
        iii. Endogenous heat production is solely responsible for hyperpyrexia
    d. Susceptibility to MH is inherited as an autosomal dominant disorder and most susceptible individuals are completely asymptomatic until exposed to triggering agents.
        i. Occur most often in children
        ii. Incidence ranges from approximately 1 in 10,000 to 1 in 50,000 individuals who are exposed.
    e. MH is characterized by:
        i. Severe hyperthermia
        ii. Muscle rigidity
        iii. Metabolic acidosis
    f. Diagnosis may be heralded by rapid increase in end-tidal CO2 due to increased metabolic rate and masseter muscle rigidity and trismus which can rapidly progress to diffuse rigidity and hyperpyrexia
    g. Sequelae include:
        i. Mixed metabolic and respiratory acidosis
        ii. Hyperkalemia, hypercalcemia, hypermagnesemia
        iii. Severe chest wall rigidity resulting in impaired ventilation
        iv. Rhabdomyolysis
        v. Acute renal failure
        vi. DIC
    h. Mortality is related to peak core temperature
    i. Treatment is immediate cessation of offending agent
        i. Dantrolene (2-3 mg/kg every 5-10 minutes) should be immediately administered. Requirement of doses up to 10 -20 mg/kg have been reported.
            1. Followed by 1 mg/kg IV every 6 hours for at least 24-48 hours
            2. Initial response should occur within minutes
        ii. Rapid systemic cooling should be initiated
        iii. Nondepolarizing neuromuscular blockade do not treat MH as the pathology resides in the muscle itself beyond the neuromuscular junction
        iv. Dysrhythmias can be treated with beta-blockers, procainamide, and lidocaine
        v. Calcium channel blockers should be avoided

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