Thermal injuries (pediatrics)

Fever

Etiology of fever

Infectious etiology:

- bacterial
- non-bacterial
  - mold
  - parasites (including import - malaria, leishmaniasis)
  - viruses (EBV, CMV)

Non-infectious etiology:

- **metabolic-endocrine disorders**
  - ion imbalance
  - diabetes insipidus
  - hyperthyroidism
- **GIT affection**
  - Crohn's Disease
  - hepatitis
- **hematological diseases**
  - leukemia
  - lymphomas
  - primary immunodeficiency
- **neurological diseases**
  - CNS infection
  - intoxication
  - trauma
  - CNS bleeding
- **rheumatological diseases**
  - M. Still
  - SLE
- **oncological diseases**
  - neuroblastoma
- **rare units**
  - Kawasaki disease
  - Stevens–Johnson syndrome
  - Caffey–Silvermann syndrome
  - hereditary periodic fever syndromes: mevalonic aciduria syndrome, Hyper IgD syndrome

Therapy of hyperpyretic condition

- **i.v. crystalloids** over ice
- **hydrocortisone** 30–50 mg/kg for dosi
- **drugs with an α-lytic effect**: chlorpromazine 0.5–1 mg/kg for i.v.

Hyperthermia

Etiology

- **inflammations**
  - infection
  - systemic inflammations
- **overheating**
  - heat cramps
  - heat exhaustion
  - heat stroke
- **CNS insult**
  - bleeding
  - infection
  - familial dysautonomia
  - spinal cord lesions
  - status epilepticus
- **metabolic cause**
  - hyperthyroidism
  - malignant hyperthermia

    - malnourished
- **intoxication**
    - salicylates
    - anticholinergics
    - phenothiazines
    - antihistamines
    - tricyclic antidepressants
- **burns**
- **congenital anhidrosis**

Heat cramps

Laboratory

- hyponatremia, hypochloremia, reduced waste of Na in the urine

Therapy

- α-blockers, i.v. line
- **1/1 FR** or **1/1 Ringer**
- **ev. anticonvulsants** in normal doses

Heat exhaustion

- collapse from heat when staying in a hot, humid, unventilated environment

Clinically

- impaired consciousness, confusion, vertigo, headache
- sweating, hypotension, hyperventilation, tachycardia
- TT may not be increased

Laboratory

- hyperosmolar dehydration = hemoconcentration
- hypernatremia, increased urea and creatinine

Heat stroke

- after staying in the sun
- the skin is usually dry, burnt

Clinically

- disorders of consciousness, convulsions, meningismus
- vomiting, hypotension, possibly diarrhea
- absent sweating, hyperpyrexia
- oligoanuria

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Laboratory

- hyperosmolar dehydration, RAL → MAC
- elevation of muscle enzymes in rhabdomyolysis
- risk of developing DIC

- Patients with cystic fibrosis, scleroderma, ichthyosis have a high risk.

Therapy

- **cooling** of the patient: remove clothing, ice in the groin, supraclavicular fossa
- open airways/oxygen therapy
- volume **1/1 FR** or **1/1 Ringer** 10–20 ml/kg i.v. through the ice
- **acidosis therapy**
    - 0.1 x BE x kg b.w. – only for severe disorders with pH<7.15; or HCO_3<8 mmol/l
    - we correct to pH 7.2; respectively HCO_3 15 mmol/l

- **anticonvulsants**: for convulsions in normal doses
- **DIC therapy**:
  - thrombocytic influx, fresh frozen plasma, AT III, fibrinogen, low dose heparin
- **therapy ASL / tubular necrosis**

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Malignant hyperthermia

- genetic disorder (mutation of the α-1S subunit of the T-tubule calcium channel of the muscle cell or mutation of the ryanodine receptor, which controls the release of calcium from the sarcoplasmic reticulum) characterized by a rapid increase in body temperature (by 1 °C/5 minutes) due to uncontrolled muscle metabolism, which is triggered by anesthetics, succinylcholine, atropine, scopolamine, phenothiazine drugs, ketamine, but also by non-depolarizing relaxants (except pancuronium) and stress.

Incidence

- 1:14,000 in children

Clinically

- after a few minutes, the body temperature rises rapidly by up to several degrees → hot skin, tachycardia, ventricular conduction disorders, tachypnea, hypotension, cyanosis, profuse sweating, cardiac arrest

Laboratory

- high pCO_2 (despite hyperventilation), severe MAC
- hyperkalemia, myoglobinuria, transient hypercalcemia
- ARF
- 70% of patients have a high resting creatine kinase level

Pathogenesis

- this is a "disorder of calcium binding in muscles" - under normal circumstances, during muscle contraction, the released calcium returns to the sarcoplasmic reticulum, but in malignant hyperthermia, "muscle contraction persists" either by increased release or reduced by calcium feedback in the plasma reticulum
- aerobic and anaerobic cell metabolism is significantly increased → there is an "increased production of CO_2", "heat and lactate"
- relatives often have a history of complications during anesthesia, muscle diseases, ptosis, strabismus, joint hypermobility, scoliosis, kyphosis, high fevers during infectious diseases, after exercise, myoglobinuria after muscle exercise

Therapy

- **monitoring**
  - TT, Ekg, arterial BP, CVP, hourly diuresis
  - as needed: Astrup, ions, CK, creatinine, glycemia, lactate, platelets, coagulation, LDH, transaminases, myoglobin in urine
- **remedial measures**
  - immediately stop the administration of inducing substances
  - hyperventilation with pure O_2
  - external cooling of the patient to TT 38 °C
  - 10% Ca-gluconicum
  - dantrolene 2 mg/kg i.v. á 5 min up to max. 10 mg/kg
  - furosemide 1–2 mg/kg
  - MAC correction: $NaHCO_3$
  - for tachyarrhythmias: β-blockers, dexamethasone
  - hyperkalemia therapy
- **prevention**: dantrolene 4 mg/kg p.o. 24 hours before performance in 4 partial doses

Hypothermia

Etiology

- **intoxication**
  - barbiturates
  - phenothiazines
  - alcohol
- **cold**
- **drowning**
- **skin lesions**
  - burns

    - erythroderma
- **sepsis** (prediction of higher mortality)
- **cardiac insufficiency**
- **metabolic causes**
    - hypoglycemia
    - M. Addison
    - hypothyroidism
    - uremia
    - cirrhosis
    - malnourished
- **CNS insult**
    - brain lesions
    - spinal cord lesions
    - drug induced coma
- anorexia nervosa
- familial dysautonomia

Clinically

- tachycardia + increased Cl → bradycardia
- prolongation of QT interval on ECG → atrial fibrillation → ventricular fibrillation at TT <30 °C
- tachypnea → hypopnea
- disorientation, disappearance of pupillary reflexes
- increased diuresis → oligoanuria + azotemia → acute tubular necrosis, MAC
- risk of DIC

Therapy

- **mild hypothermia** = TT 32-35 °C
    - heating by approx. 1 °C/1 hour can be achieved with warm blankets, radiant heat
    - suitable for i.v. volum – liquids with a temperature of 37–40 °C
- **severe hypothermia** = TT <32 °C
    - warming the patient → heated infusion solutions 37–40 °C
    - UPV heated and humidified O_2
    - nasogastric lavage of stomach and colon FR with T 38 °C
    - possible extracorporeal blood heating
- **acidosis therapy**
    - 0.1 x BE x kg b.w. → only in severe disorders with pH <7.15; respectively HCO_3 <8 mmol/l
    - we correct to pH 7.2; respectively HCO_3 15 mmol/l
- **DIC therapy**
    - thrombocytic influx, fresh frozen plasma, AT III, fibrinogen, low dose heparin
- **ASL therapy/tubular necrosis**

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- **monitoring**: ECG, BP, diuresis, Astrup, mineralogram, glycemia, ev. CVP

Burn injury

Etiology

- hot steam/liquid
- contact with a hot body
- flame
- electricity
- chemicals
- low temperatures
- ionizing radiation

Classification of burns according to the depth of the burn

1. **I.degree**
    - involvement of the epidermis, painful erythema on the skin
2. **II.degree**
    - involvement of epidermis + dermis
        - **IIa** - blisters + pain + capillary return
        - **IIb** - ruptured blisters with a deep red base + no pain, no capillary return
3. **III degree**
    - involvement of epidermis + dermis + subcutaneous tissue, skin is white, stiffer, pain at first
4. **IV. degree**
    - damage to muscles, tendons, bones

- tissues are dark and insensitive

Classification of burns according to extent and localization

- the range is given in % or cm^2 according to the Lund–Bowder table
- as a guideline: **Patient's palm area = 1% of body surface**

Definition of severe burn

- **according to scope**
  - children <2 years with a range >5%
  - children 2–15 years with a range >10%
  - children >15 years with a range >20%
- **by localization**
  - serious is any burn of the face, neck, hands, feet, surface of joints, genitals, perineum, regardless of extent/location
- **other criteria**
  - burns caused by electric current and chemicals
  - if inhalation of CO = smoke is suspected/confirmed

- **child <2 years, any burn is "severe"**
- **severe burn requires i.v. line + hospitalization**

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Inhalation of smoke (CO)

- if there is evidence of inhalation (fire in a closed space, burnt nose and nasal cavities, burns on the face, charred lips, bloody secretion from the DC, edema of the posterior pharynx), we **intubate**
- we serve **100% humidified and tempered O_2** (accelerates the breakdown of CO)
- necessary **carboxyhemoglobin examination** because pulse oximetry is unreliable (pulse oximeter ignores pathological Hb molecules and only detects oxyHb → false "good" saturation values)

Pathophysiology of burn trauma

- increased vascular permeability → edema → hypoxia
- hypermetabolism → increased demands on the need for O_2 → hypoxia
- microcirculation failure → vasodilation x vasoconstriction → hypoxia
- hypercoagulability → microthrombosis → hypoxia
- **hypoxia → cell damage → MOF** (= multiple organs failure)

Timing of burn injury

- **security:**
  - central venous catheter, arterial catheter, urinary catheter, nasogastric tube
  - EKG
  - SaO_2
  - CVP
  - TT
  - diuresis
  - indoor environment
  - according to the status of intubation/UPV

1. **Emergency period = burn shock**
   - dominated by hypovolemia and increased sympathetic activity (α receptors) = *low flow phase*
   - peripheral vasoconstriction → hypertension (this is why adrenaline, noradrenaline **are contraindicated)**
   - occurrence of local edema → ischemia on the limbs
   - suffocation → necessary releasing incisions up to the fascia
   - generalized edema → leakage of fluids into the third space
2. **Acute period**
   - dominated by high sympathetic activity (β receptors) = *flow phase*
   - catabolism dominates → often hyperglycemia without response to insulin
   - perfect and adequate parenteral nutrition required
   - increased risk of infection
3. **Reconstruction period and rehabilitation**
   - plastic-surgical solution

- **care methods**:
  - open x semi-open x closed
  - necrectomy x transplantation

Burn Injury Therapy

Minor burns

- outpatient therapy
- under sterile cooling conditions, dexpanthenol
- perforate the blisters and use them as a temporary biological cover
- prevent the substrate from drying out - greasy tulle, poultices with furantoin, chloramine

Severe burns

1. **Pre-hospital phase**
   - prevent further thermal action:
     - extinguish/remove clothing, prevent further exposure to electric current/chemicals
   - instant **cooling with cold water**
     - performed within 30 minutes of the thermal injury reduces the extent of the burn
     - we cool hands, feet, face
     - NOT the entire limb – the cooled area must not be >5% of the body surface, otherwise there is a risk of deepening the burn shock
   - **analgosedation i.v**.: pethidine 0.5 mg/kg + ketamine 1 mg/kg + midazolam 0.2-0.3 mg/kg
   - **volume expansion**: 1/1 Hartmann sol. 20-40 ml/kg
2. **Hospital care**
   - fluids/electrolytes
     - **fluid requirement during the first 24 hours**: we calculate 2-3 ml/kg/% of burned body surface + physiological need (e.g. according to the Holiday–Segar formula)
     - strategy: we give 50% of fluids in the first 8 hours and **crystalloids only**
     - another 50% fluids in the next 16 hours, colloid/crystalloid ratio = 1 : 2
     - **Na requirement in the first 24 hours** in mmol:
       - 0.5–0.75 mmol/kg/% of burned body surface + physiological need for Na (3 mmol/kg/24 h)
     - **fluid requirements during the second 24 hours**
       - we calculate 1.5 ml/kg/% of burned body surface + physiological need
     - from the 2nd day, we can give fluids evenly throughout the day

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- **dopamine + dobutamine**: in unstable circulation
- consistent **continuous analgosedation**: e.g. fentanyl 5–10 µg/kg/hour
- **α-blockers** (droperidol, chlorpromazine): in extreme peripheral vasoconstriction
- α-blockers are contraindicated without appropriate infusion therapy
- **methylprednisolone** 30 mg/kg in a single dose as soon as possible after the injury
- **prophylaxis of stress ulcers**: H2 blockers, sucralfate
- **tetanus prophylaxis**: tetanus toxoid
- **ATB prophylaxis**
- **complementary therapy**:
  - 10% Calcium gluconicum 1 ml/kg/24 hours 6 hours i.v.
  - 10% Magnesium sulfuricum 0.5 ml/kg/24 hours every 6 hours i.v.

Links

Related Articles

Source

- HAVRÁNEK, Jiří: *Thermal injuries*.