Pediatric Resuscitation (simulation infant)

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We have no relevant financial relationships to disclose.
Objectives

- Recognize severe pediatric diseases and consider a prioritized differential diagnosis
- Recognize impending cardiorespiratory failure and develop an approach to emergent management and support
- Identify pediatric patients that emergently require life-saving interventions and initiate care using the PALS algorithms
Case #1
Diagnosis:
Cardiogenic shock secondary to critical coarctation of the aorta
Objectives

• Discuss differential diagnosis in infant presenting with respiratory distress and shock
• Recognize presentation of ductal-dependent cardiac lesion
• Describe immediate management
Differential Diagnosis

<table>
<thead>
<tr>
<th>Pulmonary</th>
<th>Infectious</th>
<th>Cardiac</th>
<th>Hematologic</th>
<th>Metabolic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe bronchiolitis</td>
<td>Sepsis</td>
<td>Congenital heart defects*</td>
<td>Severe anemia</td>
<td>Hypoglycemia</td>
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<tr>
<td>Pneumonia</td>
<td>Meningitis</td>
<td></td>
<td>Hemoglobin dyscrasias</td>
<td>Inborn error of metabolism</td>
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<tr>
<td>Large pneumothorax</td>
<td></td>
<td></td>
<td>Hypovolemia</td>
<td>Hyper-ammonemia</td>
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Critical Coarctation of the Aorta

- 6-10% of all patients with CHD
- Discrete narrowing of the aorta
- Before closure, PDA serves to widen
Symptoms

- Classic triad: poor feeding, dyspnea, poor weight gain
- Hypoperfusion/circulatory shock when PDA closes
- Disparity in pulsation and BPs in arms and legs
- Systolic ejection murmur often heard
- Echo is diagnostic
Goals: improve cardiac performance, augment peripheral perfusion, improving oxygenation

ABCs!!

Need to open duct! → prostaglandin E₁

Surgical repair
Diagnosis:
Respiratory distress secondary to DKA
Objectives

• Discuss pathophysiology of DKA
• Explore management of DKA
• Identify life threatening complications of DKA
Pathophysiology

Absolute insulin deficiency

or

Stress, infection or insufficient insulin intake

Counterregulatory Hormones

↑Glucagon
↑Cortisol
↑Catecholamines
↑Growth Hormone

↑Lipolysis
↓Glucose utilization
↑Proteolysis
↓Protein Synthesis

↑FFA to liver

↑Ketogenesis
↓Alkali Reserve
Ketoacidosis
Lactic Acidosis

↑Gluconeogenic substrates

↑Glucogenesis

Hyperglycemia

Glucosuria (osmotic diuresis)
Loss of water and electrolytes

Dehydration

Impaired Renal Function

Decreased fluid intake
Hyperosmolarity
• **Definition:**
  - Blood glucose >200
  - pH<7.3
  - HCO3 <15

• **Symptoms:**
  - Polyuria, polydipsia, wt loss N/V, abdominal pain
  - Kussmaul respirations, altered mental status, dehydration
Management

- ABCDs!
- Fluids
- Electrolyte replacement
- Insulin
- Bicarb

- Community hospital vs tertiary care children’s hospital?
Cerebral edema

• Pathogenesis of initiation/progression unclear
  – <1% patients
  – Decrease blood flow and reperfusion injury?

• Potential risk factors not clearly defined
  – No association between serum glucose and rate of change during treatment

• Warning signs?
Cerebral edema management

• Reduce rate of fluid administration by 1/3
• Mannitol vs hypertonic saline
  – Mannitol: 0.5-1g/kg IV over 15-20 min, can repeat
  – *Hypertonic saline: 5-10ml/kg over 30 minutes
• Elevate head of bed
• Intubation may be necessary but tricky!
Diagnosis:
Hemorrhagic shock secondary to iron ingestion
Objectives

- Recognize an initial presentation of iron ingestion as GI bleed and resultant hemorrhagic shock
- Manage acute iron toxicity
Iron Toxicity

• Elemental Iron
  – Children’s chewable: 20 mg/tablet
  – Adult MVI: 50 mg/tablet
  – Prenatal MVI: 100 mg/tablet

• Risk factors:
  – Young age (unintentional) or Adolescent (intentional)
  – Birth of a new sibling

• 40 mg/kg = seek medical care
• 60 mg/kg = severe toxicity
Clinical Manifestations

• Phase 1 (0-6 hr):
  – Reduction-oxidation rxn $\rightarrow$ free radicals $\rightarrow$ oxidative damage in GI tract
  – N/V/D, GI bleed

• Phase 2 (6-24 hr):
  – Latent period

• Phase 3 (12-24 hr):
  – HD instability, shock, coagulopathy, metabolic acidosis

• Phase 4 (24-48 hr):
  – Hepatotoxicity

• Phase 5 (3-6 weeks):
  – Obstructive complaints $\rightarrow$ GI strictures/fistulas
Diagnosis

• Acutely it is a CLINICAL diagnosis!
  – HIGH suspicion in pediatric GI bleed

• Serum iron level:
  – Peak at 4-6 hr
  – Cutoff for clinical concern is 300
    • After this iron ingested is free iron

• TIBC: NOT helpful

• Other labs: NOT diagnostic

• KUB: NOT diagnostic
### Treatment

**YES**

- Whole bowel irrigation
  - Neutral solution (Go-lytely)
    - 9 mo-6 yr: 500 ml/hr
    - 6-12 yr: 1000 ml/hr
    - 13+ yr: 2000 ml/hr

- IV Chelation
  - Deferoxamine chelates ferric iron to ferrioxamine → excreted in urine
  - Who?
    - Serum Fe >500
    - Significant clinical manifestations
    - Metabolic acidosis

**NO**

- Gastric lavage:
  - No evidence for routine use
    - Case report of success

- Oral Chelation
- Charcoal
- Hemodialysis
Diagnosis:
Bradycardia secondary to hypoxia
• Describe causes of bradycardia
• Review the PALS algorithm for bradycardia
• Perform effective bag valve mask and CPR
Differential diagnosis

• Respiratory
• Respiratory
• Respiratory
• H’s and T’s
Pediatric Bradycardia
With a Pulse and Poor Perfusion

1. Identify and treat underlying cause
   - Maintain patent airway; assist breathing as necessary
   - Oxygen
   - Cardiac monitor to identify rhythm; monitor blood pressure and oximetry
   - IO/IV access
   - 12-Lead ECG if available; don’t delay therapy

2. Cardiopulmonary compromise continues?
   - Yes
   - CPR if HR <60/min with poor perfusion despite oxygenation and ventilation

3. CPR if HR <60/min with poor perfusion despite oxygenation and ventilation
   - Yes
   - Cardiopulmonary Compromise
     - Hypotension
     - Acutely altered mental status
     - Signs of shock
   - No
     - Bradycardia persists?
       - Yes
         - Epinephrine
         - Atropine for increased vagal tone or primary AV block
         - Consider transthoracic pacing/transvenous pacing
         - Treat underlying causes
       - No
         - Support ABCs
         - Give oxygen
         - Observe
         - Consider expert consultation

4. If pulseless arrest develops, go to Cardiac Arrest Algorithm

5. Doses/Details
   - Epinephrine IO/IV Dose:
     0.01 mg/kg (0.1 mL/kg of 1:10 000 concentration)
     Repeat every 3-5 minutes.
     If IO/IV access not available but endotracheal (ET) tube in place, may give ET dose:
     0.1 mg/kg (0.1 mL/kg of 1:1000).
   - Atropine IO/IV Dose:
     0.02 mg/kg. May repeat once.
     Minimum dose 0.1 mg and maximum single dose 0.5 mg.
References

Contact Information

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