Critical Care Paediatrics Certification Review

Session four
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Objectives-Critical Haematology/Oncology/Immunology

- Review basic hematology, oncology and immunology disorders seen in pediatric critical care.
- Review common complications and medical emergencies in critically ill hematology oncology patients.
- Review nursing care of hematology oncology patients in CCU.
Competencies

The critical care pediatrics nurse: interprets data related to the immunologic and hematologic systems including:

- client’s health history (e.g., immune status, blood dyscrasias, oncological disease, post transplant, infection and infectious contacts); 10.1a
- physical assessment (e.g. petechiae, pallor, purpura, bleeding); 10.1b

Competencies

- recognizes actual and potential life-threatening alterations in function including:
  - immunological (e.g., burns, transplants, graft vs. host, severe combined immune deficiency syndrome [SCIDS], 22-Q deletion syndrome [DiGeorge syndrome], asplenia, systemic inflammatory response syndrome [SIRS], multi-organ dysfunction syndrome [MODS]); 10.2a
  - hematological (e.g., leukemia, sickle cell disease, disseminated intravascular coagulopathy [DIC], neutropenia, heparin-induced thrombocytopenia, thrombosis) 10.2b
  - oncological emergencies (e.g., acute tumour lysis syndrome, hemorrhagic cystitis, myocardial dysfunction, sepsis, superior vena cava syndrome) 10.2c
Competencies

- implements nursing interventions to optimize immunological function including:
  - minimizing the risk of and preventing nosocomial infections (e.g., aseptic techniques, infection control practices, nutrition, hygiene); 10.3a
  - administering pharmacological agents (e.g., antibiotics, intravenous immunoglobulin [IVIG], immunizations, anti-rejection medications, granulocyte colony-stimulating factor [G-CSF], antiretrovirals); 10.3b
  - promoting immunocompetence (e.g., breastmilk, micronutrients, adequate sleep/rest). 10.3c
  - minimizing blood loss (e.g., blood conservation techniques); 10.4b

Heamatological complications in critical care

- Nausea and vomiting
- Mucositis/Typhlitis
- Fever and neutropenia
- Anemia
- Mediastinal Mass
- DIC
- Hyperleukocytosis
- Anaphlaxis
- SCD
Anti-Neoplastic Induced Nausea and Vomiting

Mechanisms by which chemotherapy may cause N/V:

1) Direct stimulation of the emetic (vomiting) centre (medulla oblongata) through the chemoreceptor trigger zone
2) Stimulation of peripheral nerve endings in the gastrointestinal tract
3) Cortical stimulation by psychological factors (memory/anxiety)

Types of Nausea/Vomiting

- Acute
  ◦ Begins 1-2 hours post chemo
  ◦ Peaks within 4 -10 hours
  ◦ Resolves within 12 -24 hours
- Delayed
  ◦ Begins 1-5 days following chemo
- Anticipatory
  ◦ Starts prior to chemo, conditioned reflex
Complications of nausea/vomiting

- Dehydration
- Electrolyte imbalance
- Weight loss
- Weakness
- Esophageal tears
- Aspiration
- Decreased quality of life

Treatment

- Ensure patient/family understands expectations re: nausea/Vomiting
- Give antiemetics around the clock after last chemo dose-prevention
- Understand triggers and sensitivities
- Be alert for medication side effects
- Ensure adequate hydration
- Comfort cares
Mucositis

- Sores or ulcerations in the mouth and back of throat
- Related to low white cell count
- Should clear in a few days with good mouth care (unless nutritional status is compromised)
- Primarily inflammatory in nature

Mucositis

- **Etiology**
  - Chemo kills quickly replicating cells but can’t differentiate between cancer cells and others (GI epithelial, hair follicles, etc)
  - May be due to direct cellular cytotoxicity, increased susceptibility to microorganisms d/t neutropenia, trauma or both
Signs and Symptoms

- Mucosal “burning” sensation
  - 7 –10 days post chemo

- Erosion/ulceration
  - 10-15 days post chemo

- Resolution
  - 14 – 21 days post chemo

Treatment of mucositis

- Sodium Bicarbonate mouthwash
- Nystatin mouthwash
- Lidocaine viscous
  - If swallowed, impairs gag reflex
  - Administer q 2-4 hours and prior to eating
- Acyclovir IV
  - If herpetic lesions
- Analgesics
  - Acetaminophen, codeine, morphine
Mucositis

- Risk for ulcerations if orally intubated
- Comfort Measures
  ◦ Frequent, gentle mouthcare using a soft toothbrush, Q-tips or sponge sticks
  ◦ Rinse mouth frequently
  ◦ Topical steroids (be careful as this may lead to increased risk of fungal infections)
  ◦ See dentist pre treatment

Typhlitis

- Necrotizing enterocolitis – inflammation and necrosis of the cecum (the cecum is always involved d/t its distendability and diminished vascularization)

**Signs and Symptoms**
- Watery or bloody diarrhea, fever, nausea/vomiting, localized RLQ abdominal pain and/or shock (secondary to sepsis or bowel perforation)
- On examination: absent bowel sounds, direct and rebound tenderness and a palpable mass
Typhlitis

Etiology

◦ Poorly understood – but thought to be a combination of injury (from the chemo) and bacterial/viral invasion (especially from CMV, pseudomonas and candida) (nutrition)
◦ The bowel wall becomes inflamed → decreased blood flow → necrosis, hemorrhage and possible bowel perforation

Typhlitis

• Management
  ◦ Mainly symptom management
  ◦ Gut rest
  ◦ NG to low gomco
  ◦ Fluid replacement
  ◦ Antibiotics
Fever & Neutropenia

Why?
- More intensive chemo protocols
- Decrease neutrophil counts
- Disrupt skin/mucosal barriers
- Altered bacterial flora
- Fever (oral or equivalent)
  - Single spike = 38.5
  - Persistent low grade = 38.0

- Neutropenia
  - Absolute neutrophil count (ANC) = <0.5 x 10^9/L

Causes
- Bacteria (endogenous flora)
  - E. coli, K. pneumoniae, Coagulase negative staph, Staph aureus, hemolytic strep (alpha)
- Viruses (e.g HSV-1)
- Fungus (Candida, aspergillus)
- Parasites
- Most infections are endogenous flora
Fever & Neutropenia-nursing care

- Initial assessment/treatment:
  - Cultures:
    - Blood from CVL/PICC (all lumens), peripheral stab, art. line
    - Urine for urinalysis
    - BAL (if intubated)
    - Swab any suspicious site (lesion, IV site, etc.)
  - CXR
  - **STAT** administration of broad spectrum antibiotics

- **WASH YOUR HANDS**
- **HAVE OTHERS WASH THEIR HANDS**
- Follow isolation practices- do not cut corners
- With multi agent usage the length of time a patient stays neutropinic is lengthened therefore are high risk
- Antibiotics – must be on time!
  - Start with broad spectrum antibiotics
  - Adjust antibiotic coverage once cultures report sensitivity
  - Monitor antibiotic levels
Fever & Neutropenia-nursing care

- Frequent assessment and accurate I & Os,
- IV fluids (1.5 x maintenance)
- Stop all antineoplastics (chemo) and cotrimoxazole
- G-CSF
- Repeat cultures if pt. remains febrile >24 hours
- Repeat cultures if pt. re-spikes
- Daily CBC with Diff
- Comfort
Persistent fever

- Mucositis
- Perianal cellulitis
- Antibiotic Induced Colitis
- Pulmonary Infiltrates
- Line Infection
- Resistance organisms
- Viruses
- Fungus

Central line care

- Various devises ports, Hickman's, silastic caths
- Alternating antibiotic schedule
- Complications-infections, bleeding, thrombus and line damage
- *Safe handling of blood post chemo therapy
- Line removal for blocking, resistance to treatment or septic shock or for some positive cultures:
Fever & Neutropenia-nursing care

- Be highly observant for signs of septic shock
- Fluctuations in core temperature (high or low)
- Heart rate
- BP hypotension a late sign (cool shock)
- Peripheral perfusion more of an early sign
- Respiratory distress
- Neurological irritability (utilize parents)
- Excellent mouth and perianal care

Septic Shock

- Sepsis and Neutropenia
  - Can happen in a matter of hours from the first fever spike
  - May not exhibit the “typical” signs and symptoms due to immunosuppression
  - May only present hypotensive, with or without fever
- Nursing Management
  - Ventilatory and end organ support
  - Fluid resuscitation (IV fluids, Albumin, PRBC)
  - Inotropes/Vasopressors
  - Treat underlying infection
  - 15% of sepsis seen in the CCU is in children whose primary diagnosis is oncological in nature
Anemia

- Pre treatment r/t bone marrow embarrassment or related to treatment i.e. radiation/bone marrow suppression
- Risks: esophageal varices in patients with abdo or liver tumor, G.I bleeding, vomiting invasive devises, typhlitis
- Tolerate significant low hg in absence of hypoxia
- Individual transfusion parameters
- Consider fatigue and reserve when planning cares

Mediastinal Mass

- Risk of respiratory embarrassment, cardiac tampoande, myocardiopathy or myocarditis, cord compression, infiltrates pneumothorax, effusions
- Children develop symptoms rapidly r/t compressibility of airway
- Superior vena cava syndrome/ Superior mediastinal syndrome with tracheal compression
## Mediastinal mass

- Congestion to head, headache, respiratory distress, altered LOC if oxygenation or CBF compromised
- Place in position of comfort-high risk airway cares/have a plan!
- May have secondary swelling post irradiation
- At risk for tumor lysis syndrome
- Sedation risk at biopsy
- Treatment can complicate diagnosis

## Disseminated Intravascular Coagulopathy (DIC)
- Simultaneous bleeding and clotting
- Abnormal uncontrolled activation of coagulation system
- Micro-emboli form in capillaries and cause organ dysfunction and failure as well as poor tissue perfusion
DIC

- Etiology
  - External circulatory devices (ECMO, bypass)
  - Infection
  - Shock, septic shock, cardiogenic, hypovolemic anaphylactic
  - Trauma, head trauma, crush injury, burns, large surgical interventions
  - Malignancies (neuroblastoma)
  - Transfusion reaction
  - Thermal injury
  - Near drowning
  - Embolisms
  - Obstetrical complication

DIC

- Clinical Presentation
  - May have laboratory evidence in absence of clinical signs
  - Decreased perfusion, pallor, mottling, circulatory failure decreased or absent pulses, cyanosis or necrosis of extremities
  - Bruising, oozing from puncture sites
  - Petechiae, ecchymosis, purpura, epistaxis
  - Hematuria, melena, hematemesis, IC bleed
  - Shock and hemorrhage
DIC

- Laboratory Findings
  - Platelets decreased
  - PTT increased
  - PT increased
  - Fibrinogen decreased
  - Fibrin split products increased
  - Anemia, fragment red cells and burr cells

DIC

- Treatment
  - Goal is to correct primary problem and reduce damaging effects of DIC
  - Supportive treatment-blood products to control bleeding FFP and platelets
  - Cryoprecipitate, PRBS to expand and increase O2 carrying capacity
  - Exchange transfusion
  - Can give Heparin to decrease mirco clots and perfuse organ (with evidence of necrosis) with plasma
  - Fluids to maintain perfusion
  - Support of organ function (i.e CRRT)
DIC

• Nursing Care
  ◦ Reduce risk of hemorrhage, minimize puncture, observe for overt and subtle signs of bleeding i.e. G.I. intracranial, PE, kidney failure
  ◦ Control overt bleeding, pressure dressings, do not remove lines
  ◦ Maximize oxygenation two person suction, maintain peep

DIC

• Nursing Care (cont.)
  ◦ Art line, CV line, multiple access sites
  ◦ Neuromuscular blocked-associated cares
  ◦ Skin integrity
  ◦ Protection from injury
  ◦ Adequate analgesia, sedation
  ◦ Support for family

K. Dryden-Palmer
Prevention of bleeding

- Close monitoring of CBC and coagulation
- Transfuse usually with multiple products
- Be aware of policy and procedure and methods
- Correct filter, rate etc.
- Pre medication, irradiated product
- Be alert for blood transfusion reactions—may be delayed
- No ibuprofen or aspirin

Prevention of bleeding

- Observe all potential sites for bleeding including rectum, vagina, nares etc.
- Minimize invasive procedures if possible
- Discuss risks and benefits of risky treatments i.e. physio, line removal, ETT changes etc.
- Soft tooth brush, gentle hygiene cares
- Support limbs when positioning, pad rails if moving
- Adequately sedate
Hyperleukocytosis

- Peripheral WBC count greater than 1,000,000/mm³
- Increases blood viscosity, blast cell aggregates and thrombi formation in microcirculation
- Presentation SOB, papilledema, irritability and CNS symptoms
- Treat with exchange transfusion, leukopheresis, chemo

Anaphylaxis

- Agents-L’Asparginase, contrast, interleukin-2, amphotericin-B, antibiotics
- Local and systemic reactions
- Treat as with other anaphylactic reactions
- Benadryl, epinephrine, supportive
Sickle Cell Anemia

Hemoglobin defect  HbS instead of HbA

- Sickle cell trait  30-40% of HbS but 60-70% HbA dominates therefore usually asymptomatic
- Sickle cell anemia  80-100% HbS with remainder being Hbf and HbA2

Occurs in approximately 1:625 birth

Occurs in persons of African, Mediterranean, Middle Eastern and Indian ancestry and persons from the Caribbean and parts of Central and South America

Can be identified with neonatal blood screening or when Hbf levels drop (6 months) and they become symptomatic

Blood – Sickle Cells

Cells ‘sickle’ under conditions of:
- Hypoxia
- Dehydration
- Stress
- Hypothermia
- Poor Perfusion
- Acidosis

Brian Kavanagh 2003
Pathophysiology

- Sickling occurs when the round shape of the RBC’s becomes crescent shaped in response to deoxygenation
- Sickle hemoglobin is less soluble than normal hemoglobin no O2 carrying capacity
- Sickle hemoglobin is more viscous and crystallizes
- Sickled cells are more rigid, fragile and rapidly destroyed —> hemolysis
- Sickled cells lose ability to flow through tiny capillary beds —> vaso-occlusion/infarction —> hypoxia and necrosis

Complications of SCD

**Vaso-occlusive Crisis**
- intravascular sickling with occlusion of the small vessels, tissue ischemia and/or infarction of the occluded organ

**Aplastic Crisis**
- sickling followed by hemolysis and potentially life-threatening anemia

**Splenic Sequestration Crisis**
- engorgement of splenic sinuses with blood progressing to hypovolemic shock
Syndrome of Inappropriate antidiuretic hormone

- Inappropriate antidiuretic hormone (ADH) secretion continued secretion or action of the hormone despite normal or increased plasma volume resulting in impaired water excretion
- Excess production of ADH (tumors, ventilation) that stimulate the posterior pituitary gland
- Kidneys retain H2O
- Leads to hyponatremia and hypo-osmolality

SIADH-causes

- Neurological
  - meningitis, encephalitis, head injury, damage to hypothalamus or pituitary gland tumour
- Lung disease-ventilation
- Post operative
- Altered thyroid or parathyroid function
- HIV
- Hereditary causes
SIADH or Diabetes Insipidus

**SIADH**
- Inappropriate ADH secretion
- Serum hypo-osmolarity
- Serum sodium < 130
- Decreased urine output
- Increased urine sodium

Careful correction
Diuresis
3% Na if symptomatic

**DI**
- Neurogenic or nephrogenic
- Low ADH synesthesia or secretion from pituitary
- Increasing serum sodium & osmolality
- Decreased urine SG < 1.005

Treat with DDAVP

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**SIADH**
- Oncology risks are CNS tumor and some chemotherapeutic agents-vincristine, cyclophosamide
- Serum sodium less than 130meq
- Untreated leads to seizures, coma and death
- Treatment, remove causative factor, restrict fluids (30-70%), DDAVP, hypertonic saline for clinical symptoms of hyponatremia
Tumor Lysis Syndrome

- An oncology emergency
- Occurs as a result of rapid tumor cell break down
- As cells are killed or die they release their intracellular contents into circulation
- Causing elevated levels of uric acid, potassium and phosphate

Tumor Lysis Syndrome

- At Risk Populations
  - Initial induction of chemotherapy
  - Rapidly growing tumors yet untreated
  - Burkett's lymphoma T-cell leukemia and lymphomas
  - Bulky lymphomas with high WBC counts
Electrolyte disturbances

- Increased K+
- Decreased Calcium
- Increased BUN
- Increased Cr
- Increased lactate
- Increased Phosphorous
- Decreased Magnesium
- Increased uric acid
Hyperkalemia

- K+ > 5
- Causes:
  - decreased excretion (renal failure)
  - Metabolic acidosis (shift of K+ from intracellular)
  - Multiple blood transfusions
  - RBC hemolysis - cell breakdown

Signs & symptoms of hyperkalemia

- Bradycardia, Hypotension
- Tall peaked T wave
- Prolonged PR, flat p-wave
- Complete Heart Block
- Ventricular Fibrillation (K+ > 7 mEq/L high risk)
- Muscle cramps, twitching, abdominal cramps, diarrhea, ileus
Treatment of hyperkalemia

- Remove K+ from lines
- NaHCO3 (moves K+ into cells): 1-2 mEq/Kg
- K+ losing diuretics – Lasix
- Calcium Chloride 10%: 0.1 mmol/Kg IV
- Resin exchange in gut (Kayexalate enema): 1g/kg/dose pr q2-q6h prn (max 50 mg)
- Insulin & Glucose for cellular uptake of K+:
  - D50W 2ml/Kg & Insulin 0.1 U/Kg/hr IV

Tumor Lysis Syndrome

- Complications (cont.)
  - Hyperphosphatemia:
  - Malignant cells can have up to 4 times increased phosphate
  - Calcium phosphate precipitation in the renal tubules, which can lead to acute kidney injury
  - Symptoms
    - Renal: decreased urine output → anuria, ↑ BUN, creatinine, calcium phosphate crystals in the urine
Hypocalcemia

- Calcium < 0.8mmol/L
- Causes:
  - Excessive loss of Ca++ e.g. diarrhea, diuretics, hypoparathyroidism, chronic renal failure, binding
  - alkalosis
  - inadequate intake
  - malabsorption from GI tract
  - large quantities of citrate blood transfusions
Signs & symptoms of hypocalcemia

- tingling around mouth and fingers
- tetany
- Neuromuscular irritability: muscle tremors, cramps, tetany, seizures
- Chvostek’s sign: tap cheek above mandible.....lip quivers on the side of the tapping
- Trousseau’s sign: BP cuff to the arm and inflate it until a carpopedal spasm occurs. If none is present in 3 min = negative result
- cardiac arrhythmias e.g. prolonged QT
- low blood pressure, cardiac arrest
- affects cardiac contractility so important for maintaining good cardiac function

Treatment of hypocalcemia

- Administer Ca bolus SLOW IV push
- Administer Ca infusion – central line only
- Monitor electrolytes closely
Tumor Lysis Syndrome

- Complications (cont.)
  - Hyperuricemia:
    - precipitation of uric acid in the renal tubules and renal vasoconstriction, impaired autoregulation, decreased renal flow, oxidation, and inflammation, resulting in acute kidney injury
  - Symptoms
    - GI: N/V, diarrhea, anorexia
    - Renal: edema, oliguria, anuria, hematuria, ↑ BUN and creatinine, lethargy, renal failure

What can we do about uric acid?

- Increase Elimination. Hydration - Diuresis
- Alkalization of urine
- Conversion of Uric Acid-rasbiricase
- Dialysis-invasive
  - note: clears chemotherapy and other helpful substances
Tumor - Source of DNA

- Purines
  - Hypoxanthine
  - Xanthine
  - Uric Acid
    - Xanthine Oxidase
    - Uric Acid Oxidase [Rasburicase]

  + + soluble

Allopurinol

Tumor Lysis Syndrome

- Treatment
  - **PREVENTION**
    - Hydration – to aid the kidneys excrete the excess K⁺, phosphorous and uric acid (1.5 – 2 x maintenance)
    - Alkalization – with sodium bicarb infusion to maintain urine pH 7.0 – 7.5
    - Diuresis – may be indicated for low urine output, but not in hypovolemic patients
    - Uric acid reduction – allopurinol/rasburicase
    - Foley insitu – monitor urine output carefully
    - CVL insitu – keep well hydrated 5 – 8 mmHg
    - For severe hyperkalemia-calcium gluconate, NaHCO₃, glucose/insulin
Tumor Lysis Syndrome

- **Nursing Role**
  - Monitor for irregular pulse and hypotension
  - Assess for signs and symptoms of cardiac arrhythmias
  - Monitor lab values and communicate these results as required
  - Assess for signs of tetany (Trousseau’s sign, Chvostek’s sign)

Promote comfort for
**Heamotology/oncology patients**
- Position for comfort (mediastinal mass, edema)
- Utilize tools familiar to patient for pain evaluation
- Utilize background infusions + break through
- Remember procedural pain-emla
- May develop tolerance as long term use of opioids
- Hydro morph and other high risk drugs used with these patients
- Treat for side effect of medications
Promote comfort

- Accurate ins and outs and hydration
- Environmental temperature
- Relaxation, guided imagery, music therapy, massage and other techniques can still be utilized in CCU
- Parental or family assistance
- Sensitivity to scalp, port site etc

Promote adaptation

- Orientation to CCU environment
- Orientate to specific roles
- Tend to be highly involved in child’s management and treatment decisions
- Very tuned in to infection control
- Utilize patient and family in problem solving
- Anticipatory grieving
Promote adaptation

- Try to follow the child’s care routines from the ward as much as possible
- Ward support team will continue to follow family while on unit
- Introduce family to CCU support personnel as well

Case Study

Seth, 14 years old, has non-Hodgkin’s lymphoma, Stage III, which presents with a mediastinal mass. Following the initiation of his first round of chemotherapy, he is admitted with tumour lysis syndrome and hypodynamic septic shock.
Which one of the following sets of manifestations would indicate inadequate tissue perfusion?

a) Cool extremities; Capillary refill of 4 sec; 1+ pulses
b) Warm extremities; Capillary refill of 4 sec; 2+ pulses
c) Cool extremities; Capillary refill of 1 sec; 1+ pulses
d) Warm extremities; Capillary refill of 1 sec; 2+ pulses
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The most common electrolyte disturbance in TLS is:

a) Hypokalemia, hypermagnsmina, increased uric acid
b) Hyperkalemia, hypocalcemia
c) Hypokalemia, hypercalcemia,
d) Hypophoshatemia, elevated BUN, CR
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b) **Hyperkalemia, hypocalcemia**
c) Hypokalemia, hypercalcemia,
d) Hypophoshatemia, elevated BUN, CR

Two hours into his first chemo dose Seth is restless and sleepy. How should the PICU nurse help him to rest?

a) Reposition him on his flat on left side—his preferred sleeping position
b) Give him morphine for analgesia
c) Minimal handling, turn off the lights and don’t disturb him for 4 hours
d) Do a complete neuro assessment
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d) **Do a complete neuro assessment**

Seth develops petechiae and begins to bleed around his nasogastric tube and central line site. He is passing melena stools. Which of the following laboratory results should the critical care pediatrics nurse communicate to the physician at this time?

a) WBC with differential, INR and stools for culture and occult blood  
b) AST, ALT, GGT, bilirubin, albumin and ammonia  
c) Arterial blood gases, electrolytes, CBC and blood culture  
d) Platelet count, PT, PTT, thromboplastin fibrinogen and FSP
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Which one of the following manifestations best describes disseminate intravascular coagulation (DIC)?

a) An excessive production of thrombin and plasmin
b) A platelet count of 20 x 109/lc
c) A sudden onset of unexplainable bleeding
d) An excessive clotting process followed by bleeding
Which one of the following manifestations best describes disseminate intravascular coagulation (DIC)?

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Case Study

Lee, an 11-year-old girl who weighs 50 kg, is admitted to the pediatric intensive care unit from the oncology ward. She is diagnosed with a relapse of acute lymphoblastic leukemia (ALL) and on day 26 of her induction therapy. Her blood work indicated neutropenia and thrombocytopenia, along with chemotherapy-induced diabetes. Vital signs on admission are HR 140/min, BP 105/45 (65), RR 28/min, T 38.3 C, CVP 4, O2 sat is 96% in room air. Her colour is flushed and her pulses weak. She is edematous.
Case study

Which of the following conditions best explains Lee’s presentation?

a) Diabetic ketoacidosis
b) Early septic shock
c) Respiratory distress
d) Disseminated intravascular coagulation

Case study

Which of the following conditions best explains Lee’s presentation?

a) Diabetic ketoacidosis
b) **Early septic shock**
c) Respiratory distress
d) Disseminated intravascular coagulation
Fluids and electrolyte disturbances in Critical Illness

Competencies 9.1

The critical care pediatrics nurse;

interprets data related to the endocrine/metabolic system including:

client’s health history (e.g., endocrine/metabolic status, familial/genetic disorders, consanguinity);

physical assessment (e.g., hydration status, respiratory patterns, neurological assessment, vital signs, weight changes and body mass index [BMI]);

laboratory reports (e.g., serum and urine glucose, osmolality, electrolytes, pH and acid base balance, bilirubin, ammonia, lipase); and
Competencies 9.2

The critical care nurse recognizes actual or potential life-threatening alterations in endocrine/metabolic function including regulation of:

- antidiuretic hormone (e.g., diabetes insipidus [DI], syndrome of inappropriate antidiuretic hormone [SIADH]);
- metabolism (e.g., diabetic ketoacidosis [DKA], hypoglycaemia, inborn errors of metabolism);

Competencies 9.3

The critical care nurse implements nursing interventions to optimize endocrine/metabolic function including:

- administering pharmacological agents (e.g., insulin, steroids, dextrose, antidiuretic hormone, electrolyte replacement, hypertonic saline);
- managing parenteral nutrition (e.g., considerations for compatibility, electrolyte imbalances);
- managing fluid status and acid base balance; and
Regulation of fluid & electrolytes

- Fluid & electrolyte balance is regulated by the kidneys
- Kidneys receive 20 - 25 % of cardiac output (same as the brain)
- Kidneys continuously adjust extracellular fluid volume, solute concentration & pH
- Blood flow & subsequent rate of fluid removal is autoregulated - afferent and efferent arterioles constrict or dilates in response to BP

Regulation of fluids & electrolytes cont’d

- Kidneys regulate serum electrolytes, phosphate, calcium & magnesium
- Kidneys responsible for regulation of pH & HC03 in response to acid base imbalances (compensation)
- Multiple sources of insensible loss e.g. fever, tachypnea, large body surface area
Urinary output

- Small changes in urine output may indicate a significant compromise in renal perfusion or output
- Monitor urine output closely in the critically ill child
- Normal urinary output 1-2 ml/Kg/hr
- Notify MD if < 1ml/hr X 2 consecutive hours
- Monitor for elevated BUN & Creatinine

Calculation of maintenance fluid

- Restrict to 60-80% maintenance for intubated & ventilated patients - retain fluid from positive pressure ventilation
- 100% maintenance
  - < 10 kg 4 ml/kg/hr
  - 10-20 kg 40 ml + 2 ml/kg/hr for each kg > 10 kg
  - >20 kg 60 ml + 1 ml/kg/hr for each kg > 20 kg
Dehydration

<table>
<thead>
<tr>
<th>TYPE</th>
<th>Na</th>
<th>Osmolarity</th>
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<tbody>
<tr>
<td>Hypotonic</td>
<td>Na &lt;130 mmol/L</td>
<td>280-300</td>
</tr>
<tr>
<td>Isotonic</td>
<td>Na 130 – 150 mmol/L</td>
<td>High</td>
</tr>
<tr>
<td>Hypertonic</td>
<td>Na &gt;150 mmol/L</td>
<td>Low</td>
</tr>
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Etiology

- Total output of fluids & electrolytes exceeds intake as a result of
  - Inadequate intake of fluid e.g. NPO, decreased intake, inadequate IV therapy
  - Excessive loss of fluid e.g. vomiting, diarrhea, diuretic therapy, diabetes insipidus, inadequate replacement of GI losses
- Produces deficiency in both fluids & electrolytes
Physiologic consequences

- Can result in depletion of intravascular volume & changes in serum Na
- Decreased systemic perfusion with significant loss of intravascular volume
- Acute changes in serum sodium cause free water shift in & out of intravascular space
  - Acute decrease – water moves from intravascular to interstitial causing cerebral edema
  - Acute increase – water moves from interstitial to intravascular causing hemorrhage

Treatment of dehydration

1. Restoration & maintenance of intravascular volume & systemic perfusion
   - Ensure large bore IV access
   - Normal saline or Ringers Lactate over 20-40 min
   - Plasma or blood or albumen (10-20 ml/Kg) over 1 hr

2. Correction of fluid & electrolyte disturbance
   - Avoid rapid correction
   - Do not over-correct
   - Use isotonic solutions
   - Monitor fluid balance & electrolytes closely
Hypernatremia

- Na+ > 145 mEq/L
- Results from:
  - Dehydration
  - Excessive osmotic diuretics
  - Over-use of Na containing fluids
  - Lack of ADH (diabetes insipidus)
- May cause some cardiac depression

Signs & symptoms of hypernatremia

- Thirst
- Dry mucous membranes
- Oliguria
- Fever
- Tachycardia, hypotension
- Agitation
- High pitched cry, seizures, coma, death, cerebral clots
Treatment of hypertonic dehydration

- Careful correction of Na is essential
- Rapid rehydration & use of hypotonic solutions may cause cerebral swelling
- Do not lower Na more than 5-8 mmol/L/day
- Na should not fall faster than 0.5 mmol/hr
- Start off with 0.9 NaCl isotonic solution

Hyponatremia

- Serious risk – can lead to seizures, coma & death
- Rapid depletion results in fluid shifting & cerebral edema
- Causes:
  - Excessive water administration
  - Use of D5W
  - Water intoxication
  - Na+ depletion
  - SIADH (too much ADH)
Treatment of hyponatremic dehydration

- Do not increase Na+ by more than 10 mmol/day
- Correct quickly if symptomatic (seizures) - as fast as you can
- Do not increase Na+ more than 0.5 mmol/hr (so 1 mmol q2H)
- Risk of correcting too quickly - seizures, coning
- 1/2 correction in 24 hrs then the other 1/2 the next 24 hrs
- Others feel only use 3% NaCl if they are symptomatic

Hypokalemia

- K+ < 3.5 mmol/L
- Causes:
  - Alkalosis (H+ retention and K+ excretion in distal tubules)
  - Diuretic therapy without K+ replacement
  - Endocrine dysfunction (increased ACTH, thyroid storm)
  - Gastric losses (gastric surgery, NG to low Gomco)
Hypermagnesemia

- Causes:
  - Excess IV administration
  - Renal failure
  - TPN
  - Overuse of antacids

- Signs & symptoms
  - Depressed deep tendon reflexes
  - Hypotension
  - Cardiac dysrrhythmias
  - Hypoventilation

Calcium

- Ionized Ca++: 0.85-1.20 mmol/L
- Total Ca++:
  - Child 2.25-2.74 mmol/L
  - Adult 2.12 - 2.62 mmol/L

- Teeth, Bones
- Nerve cells: transmission of nerve impulses
- Part of clotting cascade
- Cardiac contractility
- Vit. D promotes absorption of CA+ from small intestine
Hypercalcemia

- Calcium > 1.25 mmol/L
- Common Causes:
  - excessive administration
  - multiple fractures,
  - excessive intake of Vitamin D,
  - prolonged immobilization,
  - increased renal absorption.

Signs & symptoms of hypercalcemia

- bradycardia
- cardiac arrhythmias e.g. short QT, prolonged PR, heart block
- muscle fatigue, weakness
- renal calculi
- Lethargy
- decreased deep tendon reflexes
- abdominal pain
- constipation
Treatment of hypercalcemia

- Diuretics - block Ca+ absorption from the kidneys & promotes excretion,
- Push fluids & hydrate to promote diuresis
- Renal replacement therapy

Diabetic Ketoacidosis
Types of Diabetes

- **Type 1**
  - Formerly known as Type I, IDDM, or Juvenile-onset diabetes.
  - Absolute insulin deficiency r/t islet cell destruction or dysfunction
    - Immune Mediated
    - Idiopathic

- **Type 2**
  - Formerly known as Type II, NIDDM, or Adult-onset diabetes.
  - Also includes MODY.
  - Insulin resistance with relative insulin deficiency

- **Gestational Diabetes**
  - Occurs in pregnancy and is usually self-resolving after delivery.
  - May predispose woman to Type 2 diabetes in future.

- **Other specific Types**
  - Include endocrinopathies, steroid or CF induced diabetes.

Etiology

- Autoimmune Type 1 DM occurs in genetically susceptible individuals.
- Diagnosis often follows a viral illness
- Dietary antigens and environmental triggers have also been suggested
- Some studies show significant seasonal and geographic distribution
- Bovine serum albumin (BSA) has been identified as a possible DM trigger in retrospective studies
  - Not concrete finding and is still under investigation
Epidemiology Cont’d

- Second most common chronic disease in childhood
- DKA is the leading cause of hospitalization, morbidity, and death in children with DM

DKA - Signs & Symptoms

- 3 P’s
  - Polyuria, Polydipsia, Polyphagia
- Weight loss
- Abdominal pain, vomiting
- Kaussmaul breathing
- Mental status changes → Coma
- Death if untreated
DKA - Cause

- DKA is always caused by insulin deficiency, either relative or absolute
- Approximately 20% of cases are new diagnoses of Diabetes
- Cause in diagnosed patients:
  - Failure to take insulin, omission
  - Insulin pump failure
  - Acute stress from trauma, febrile/viral illness (most common), or severe psychological stress
    - Increases counter-regulatory hormones: glucagon, epinephrine, cortisol, growth hormone

DKA - Diagnosis

- Serum glucose >11mmol/L
- Ketonuria
- pH <7.3
- Serum bicarbonate <18mmol/L
- Hyperosmolarity >300
- Elevated BUN, wbc's
- Mental status changes
- Electrolyte imbalances
Clinical Presentation

- Very ill
- Decreased LOC
- Dehydration 10%-15% higher in infants
- Electrolyte disturbances
- Arrhythmias
- Shock
- Cardiovascular collapse

What Happens?

- Serum osmolarity climbs with increased extra cellular glucose content
- Glucose supercedes renal threshold and glucose is ‘spilled’ into urine
- Osmotic diuresis occurs
- Dehydration compounded by inability to compensate
- Increased hemoconcentration
- Increased osmolarity and increased BUN
Treatment of DKA

- Priority is gentle correction of fluids and osmolarity
- Prevention of cerebral edema
- 31% of DKA deaths and 20% overall diabetic mortality in children

Treatment

- Ensure adequate ABC’s
- Correct fluid deficits
- Interrupt ketone and ketoacid production cycle
- Correct acidosis
- Correct electrolyte disturbances
- Investigate underlying issues, i.e. infection
Cerebral Edema

- Etiology not fully understood
- In DKA, H2O moves out of ICF to hyperosmolar ECF
- As dehydration is corrected and serum osmolarity drops, H2O moves rapidly back from the ECF to the ICF causing intracellular edema
- Thought to be a function of protective mechanism of cells protecting against total dehydration

Cerebral Edema Causes

- IV fluid administration or improper administration
- Insulin
- Idiogenic osmoles (protective?)
Risk factors—Cerebral Edema

Higher BUN—worse dehydration?
Lower initial PaCO2 levels—worse acidosis
Initial sodium levels
Use of bicarbonate—paradoxical cerebral acidosis (CO2 crossing bld-brain barrier during correction of serum acidosis)

DKA and Cerebral Edema

Correct Fluids

- Resuscitation only with vascular compromise
- N/S 7-10 ml/kg over first hour
- Then decrease to 3.5-5 mls/kg
- Cautious rehydration over 36-48 hours
- Measures of dehydration may not be accurate (ICF vs ECF)
- Aim to decrease effective osmolarity 1-2 mmol/hr

Acidosis

- Should see improvement with hydration
- Need to turn off gluconeogenesis with insulin
- Maintain adequate oxygenation
- Use of bicarb remains controversial
- Tolerate a PH to 7
- Risks of hypokalemia, CNS acidosis, hypernatremia, alkalosis
- Avoid bolus doses
- 1-2 meq/kg over 1 hour
Glucose correction

- Necessary to correct acidosis
- Continuous low dose insulin infusion
- NEVER BOLUS
- 0.1 units /kg/hr
- run separately from maintenance fluid
- Dropping serum glucose not the priority
- Drop infusion by ½ when sugar reaches 300mg/dl and add dextrose to IV fluids

Glucose correction

- Blood glucose < 15mmol/l or falling greater than 5 mmol/hr and acidosis improving change to D5N/S or D10N/S if glucose < 10
- If acidosis not improving check delivery or ? sepsis
Electrolyte imbalances

- Hyperkalemia-relative hypokalemia (3.5-5.5 meq/L)
- K+ added to all fluids (exception for K+>5.5, ECG changes indicating hyperkalemia or renal failure)
- Insulin will shift K+ back into cells
- Correction of acidosis will cause cells to exchange K+ for Hydrogen
- May give potassium phosphate to treat phosphate deficit

Electrolyte corrections

- Hyperchloremia-r/t sodium content of resuscitation fluids and retention of chloride in ECF (95-103 meq/L)
- Hyponatremia present with low NA r/t urinary losses, may drop further as glucose falls drawing in more ECF and diluting NA (136-140)
- Hypophosphatemia-related to O2 utilization and hypoxia (3.0-4.5)
- Monitor for hypocalcaemia and hypomagnesaemia related to phosphate and bicarb dosing
Neurological concerns

- Any alteration in LOC or clinical findings indicating increasing ICP
- Remember the timeline
- Troubleshooting
  - Neurological deterioration * Sick kids protocol*
    - Mannitol 5cc/kg over 20 mins
    - If Na decreasing 2-4ml/kg 3%NaCl over 10-20 min
    - Decrease insulin to 0.04-0.05U/kg/hr
- Hypoglycemia/relative hypoglycemia
  - 0.5-2ml/kg 25% glucose

DKA – Initial Monitoring

- BG q1h
- Serum osmolarity, BUN, Cr, ABG, lytes q1h
- Hourly neuro assessment
- I&O q1h
- Art line. CVP line, Foley, lots of access
- May or may not be intubated
Diabetic ketoacidosis

- **Ongoing Management**
  - **Acidosis improving**
    - Blood glucose < 15 mmol/l OR falls > 5mmol/hr
    - Change IV to D5/NS (D10/NS if glucose < 10mmol/l)
    - May need to decrease insulin
  - **Acidosis not improving**
    - Check insulin delivery system, consider spesis

Nursing Interventions

*Metabolic Acidosis*

- Monitor respiratory status
- Ensure adequate IV access and rehydration
- Monitor ABG’s
- Acidosis, related to dehydration and lipid metabolism, should correct with insulin and hydration.
- If respiratory effort is normal, the child should ‘blow off’ CO₂, if not able – prepare to intubate
Nursing Interventions
CNS Dysfunction

- Monitor neurological status q1h using appropriate tool (GCS)
- The most acute complication of DKA is cerebral edema
- CE presents most commonly 4-10 hours after the initiation of treatment

Nursing Interventions
Cardiovascular Status

- Monitor ECG for signs of rhythm changes
- Monitor perfusion status
- Electrolyte imbalances present in DKA may cause arrhythmias
- DKA patient may progress to decompensated shock
- Child may need CVP, Art line, Foley for monitoring
Case

- George, who is 9 years old and weighs 24 kgs, has not been feeling well for three weeks. He has been getting up twice each night to urinate and take a drink of water. His mother thinks that he has lost some weight. His mother became really alarmed yesterday when he began to vomit, was very weak and drowsy. She became so worried when he could not keep even water in his stomach, that she brought him to the emergency room.

Case study cont.

- George is restless and irritable. HR 138, B/P 83/54, resp 36, temp 37.1 t, pulses are decreased mildly, skin is flushed and dry.
- An insulin infusion has been started. Over the next 2 hours,
George's level of consciousness deteriorates (GCS form 15 to 12). What is the most concerning cause of this change?

a) Metabolic acidosis
b) Cerebral edema
c) Hypoglycemia
d) Fatigue

An insulin infusion has been started. Over the next 2 hours, George's level of consciousness deteriorates (GCS form 15 to 12).

What is the most concerning cause of this change?

- Metabolic acidosis
- **Cerebral edema**
- Hypoglycemia
- Fatigue
The physician decides to insert a radial arterial line. George expresses concerns regarding the procedure. Which one of the following is the most common fear associated with the developmental level of a 9 year-old?

a) Separation anxiety
b) Pain *
c) Loss of control
d) Loss of body image
What might you expect for Georges lab results?

a) Elevated glucose; elevated osmolality; decreased pH
b) Elevated glucose; decreased osmolality; increased pH
c) Decreased glucose; elevated osmolality; decreased pH
d) Decreased glucose; decreased osmolality; increased pH

What might you expect for Georges lab results?

a) Elevated glucose; elevated osmolality; decreased pH
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Case continued

- George GCS has dropped and he is increasing confused. You have taken him to CT scan and returned

Cerebral edema has been diagnosed. What are the main goals of medical management at this stage?

a) Diuresis with lasix
b) Hyperventilation
c) Give glucose
d) Mannitol
Cerebral edema is suspected. What are the main goals of medical management at this stage?

- Diuresis with lasix
- Hyperventilation
- Give glucose
- Mannitol

End case

- Questions?
Objectives

Using a case study format, participants will review the competency list for the gastrointestinal system including:

- Interpreting data related to the gastrointestinal system (7.1 a-e)
- Recognizes actual or potential life-threatening alterations to gastrointestinal function (7.2 a-f)
- Implementing nursing interventions to optimize gastrointestinal function (7.3 a-e)
Nutrition in Critical Illness

- Metabolic response to critical illness: an increase in resting energy expenditure (REE)
- Children's growth & development
- Rapid loss of fat & muscle mass
- Immunosuppression & increased susceptibility to infection
- Malnutrition
- Impaired wound healing
Enteral Feeding

- Maintains gut flora
- Minimizes bacterial overgrowth
- Reduces bacterial translocation
- Maintains gut mucosal integrity
- Prevents pancreatic & biliary flow dysfunction
- Lower cost
- Decreased length of stay

Enteral Feeding

- Nasogastric
  - Easy insertion
  - Can monitor gastric residual volume
  - Mimics physiologic feeding pattern
  - Bolus or continuous feeds
- Nasoduodenal/jejunal
  - Continuous feeds (smaller volumes)
  - Possible improved tolerance
  - More difficult to place
When to Start?

- **Early**
  - Burns – within 6 hours of admission
  - Infants – as soon as hemodynamically stable
  - Trauma – within 24 hours

- **Contraindications**
  - Absolute - NEC, bowel obstruction, ileus
  - Possible – individual evaluation
    - Persistent vomiting or diarrhea
    - Acute abdominal distension
    - Fistula
    - Upper GI bleed

Monitoring Feeding Tolerance

- Abdominal distention
- Abdominal pain or tenderness
- Emesis
- Gastric residual volumes
- Respiratory rate/effort (increasing with feeds?)
- Stool output
Parenteral Nutrition

- Hyper/hypo glycemia
- Tissue necrosis
  - May require central access
- Hyperlipidemia
- Electrolyte imbalance
- Infection
- Cholestasis
- Increased costs
Which of the following is true concerning digestion?

A. The small intestine absorbs water and electrolytes.
B. The duodenum absorbs bile salts.
C. The ileum absorbs vitamin B12.
D. The descending colon absorbs water and electrolytes.

The ileum absorbs bile salts and vitamin B12. The small intestine absorbs amino acids and proteins. The duodenum absorbs iron and water soluble vitamins. The descending colon absorbs water and electrolytes.
Which of the following is not a function of the liver?

A. Forms clotting fibers.
B. Stores glycogen and fat soluble vitamins.
C. Breaks down protein.
D. Concentrates bile.

The gallbladder concentrates bile. The liver forms clotting factors, stores glycogen and fat soluble vitamins and breaks down protein.

Which of the following is an example of a prokinetic?

A. Erythromycin.
B. Omeprazole.
C. Lansoprazole.
D. Ranitidine.

Erythromycin is a prokinetic. Omeprazole and Lansoprazole are proton pump inhibitors. Ranitidine is a histamine – 2 receptor antagonist.
Which of the following is false?

A. Esophageal varices are an example of upper GI bleeding.
B. NEC is an example of generalized GI bleeding.
C. The ligament of Treitz is the anatomical landmark that distinguishes between upper and lower GI bleeding.
D. The ligament of Treitz is the suspensory muscle of the duodenum.

NEC is an example of lower GI bleeding. Esophageal varices are an example of upper GI bleeding. The ligament of Treitz, a suspensory muscle of the duodenum, is the anatomical landmark that distinguishes between upper and lower GI bleeding.

Sarah is a 4 year old admitted to the PICU with a lower GI bleed. Which of the following manifestations is the most concerning?

A. Bright red blood per rectum.
B. Complaints of cramp like pain.
C. Rigid abdomen.
D. Heart rate of 142.

A rigid abdomen may indicate perforation which is a surgical emergency. Although a heart rate of 142 may indicate that the child is progressing to shock, a potential perforation needs to be immediately addressed. Bright red blood and cramp like pain need to be attended to but is not the highest priority.
The nurse is caring for Josh, a 12 year old child who has experienced multi-trauma. In report he is told that Josh has a positive Cullen’s sign. The nurse knows that this means which of the following?

A. Bruising to the flank.
B. Bruising around the umbilicus.
C. Pain when the abdomen is compressed slowly and then rapidly released.
D. Pain in the right lower quadrant when running or jumping.

Cullen’s sign, which is associated with retroperitoneal bleeding is bruising around the umbilicus. Bruising over the flank is Turner’s sign. Pain when the abdomen is compressed slowly and then released is Blumberg sign. Pain in the right lower quadrant when running or jumping is Markle sign.

Which of the following is the hallmark manifestation of bowel obstruction?

A. Abdominal distention.
B. Respiratory distress.
C. Abdominal pain.
D. Constipation.

Although all of the listed manifestations can indicate a bowel obstruction, abdominal distention is the hallmark sign of a bowel obstruction.
Kennedy is an infant who has just been diagnosed with NEC. The nurse would expect to see all of the following except?

A. Abdominal distention.
B. Non bilious vomiting.
C. Bloody stools.
D. Temperature instability.

Bilious vomiting, not non bilious vomiting is seen in NEC.

The nurse is caring for an infant who has a herniation of the abdominal viscera into the umbilical cord. What is this defect called?

A. Gastroschisis.
B. Omphalocele.
C. Volvulus.
D. Intussusception.

Omphalocele occurs when the abdominal viscera herniates into the umbilical cord. Gastroschisis occurs when the abdominal contents eviscerate through the abdominal wall. Volvulus is an abnormal rotation of the intestine leading to a bowel obstruction. Intussusception occurs when one part of the intestine telescopes into the other.