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Pediatric Fluid and Electrolyte Balance: Critical Care Case Studies

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The care of the critically ill infant or child often is complicated further by disruptions in fluid or electrolyte balance. Prompt recognition of these disruptions is essential to the care of these patients. This article uses a case study approach to provide an overview of the principles of fluid and electrolyte balance in the critically ill infant and child.

Fluid homeostasis

Body fluids are composed of water and solutes. These solutes are electrically charged electrolytes (eg, Na^+, K^+, Cl^-) and nonelectrolytes (eg, glucose, urea). Fluid and electrolyte homeostasis occurs when fluid and electrolyte balance is maintained within narrow limits, despite a wide variation in dietary intake, metabolic rate, and kidney function.

Water is one of the most significant components of the human body, and accounts for approximately 50% to 80% of total body weight. Total body water (TBW) varies from one individual to another. The percentage of TBW varies with age, gender, skeletal muscle mass, and fat content. In the average adult, water accounts for approximately 50% to 60% of total weight. The body weight of children who are less than 1 year of age has a significantly higher percentage of body water; premature infants and neonates have the highest percentages. At birth, TBW is 70% to 75% of body weight. This decreases dramatically in the first year of life. At puberty, more changes occur. Because of the lower water content of adipose tissue, TBW as a percentage of body weight is less in women than it is in men.

TBW is distributed in two compartments: intracellular fluid (ICF) and extracellular fluid (ECF). In addition to the changes in the percentage of TBW as body weight, infants and young children have higher percentages of ECF as compared with adults. More than half of the newborn infant's body weight is ECF. This changes rapidly over the first 6 to 8 weeks of life. By 3 years of age, body fluid components more closely resemble those of the adult, with an ECF of approximately 20% to 23% and an ICF of 40% to 50% [1].

Fluid compartments

ICF is made up of all of the fluid contained within the membranes of the cells; it is the largest fluid compartment in the body. ECF is not one isolated fluid compartment. It is composed of interstitial fluid, plasma, and transcellular water. Adequate ECF volume—in particular intravascular volume—is essential for normal functioning of the cardiovascular system. In contrast to older children and adults, premature infants and newborns have a significantly higher percentage of body water making up their total body weight, and significantly more of this water is found within the ECF. This is one of the reasons why infants exhibit signs of cardiovascular compromise when dehydrated faster than older children or adults.

Selectively permeable membranes separate each fluid compartment. These membranes permit the movement of water and certain solutes from one compartment to another. The movement of fluids and

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Table 1
Key physiologic concepts

Physiologic concept	Description
Osmosis	Osmosis is the movement of H_20 across a semipermeable membrane from an area of lower solute concentration to one of higher solute concentration.
Diffusion	Diffusion is the movement of particles through a solution or gas from an area of higher concentration to one of lower concentration. The greater the concentration gradient, the faster the rate of diffusion.
Filtration	Filtration is the movement of H_20 and solute from an area of increased hydrostatic pressure to an area of low hydrostatic pressure. It plays an important role in moving fluids out of the arterial end of the capillaries.
Active transport	Diffusion cannot occur in the absence of a concentration gradient or favorable electrical gradient. Energy is required to move particles against a concentration gradient. This happens through the process of active transport. The Na–K pump is one example of active transport. Active transport plays a vital role in maintaining the unique composition of the ECF and ICF.

electrolytes is dependent upon osmolality and a functioning renal system. Fluids move constantly from one body compartment to another, and then remain in specific compartments until an inequality in concentration of electrolytes develops and movement occurs. Movement of fluids and electrolytes occurs through osmosis, diffusion, active transport, and filtration (Table 1).

Fluid imbalances

Typically, fluid volume deficit is defined as a negative body fluid or water balance. When volume depletion occurs in the extracellular space, circulatory collapse can result. Fluid volume deficit is a common problem in critically ill infants and children, and occurs as the result of excess loss of fluids and electrolytes (diarrhea or vomiting), shifts of fluids and electrolytes into nonaccessible third spaces (burns, post abdominal surgery), and decreased intake of fluid and electrolytes (impaired thirst mechanism, dysphagia, prolonged NPO status). Gastrointestinal water loss from diarrheal disease usually is the most common cause of excess fluid volume loss in infants and children.

In the critically ill child, fluid volume loss through "third spacing" is a common cause of fluid volume loss. Third spacing occurs when fluids and electrolytes are found in a space other than the usual spaces of the ICF and ECF ("third space") [2]. Third spacing develops in ascites, pancreatitis, burns, peritonitis, sepsis, and intestinal obstruction.

Fluid volume overload is the actual excess of total body fluid or a relative excess in one or more fluid compartments. It occurs as the result of increased sodium concentration and water volume because of retention or excessive intake; decreased renal excretion of water and sodium; or decreased mobilization of fluid within the intracellular space. The major causes of excess fluid volume in critically ill infants and children are cardiorespiratory dysfunction, renal dysfunction, and inappropriate secretion of antidiuretic hormone [1].

Case study #1

Jamal is a 5-kg boy who was admitted to the pediatric ICU (PICU) with respiratory syncytial virus (RSV) bronchiolitis. His mother reports a 2- to 3-day history of fever, increased work of breathing, and decreased oral intake. He is lethargic but arousable, tachycardic, and in moderate respiratory distress. While conducting an initial assessment, you note that his anterior fontanel is sunken, his mucous membranes are dry, and his peripheral pulses are diminished. Intravenous (IV) fluids are infusing by way of a 24-gauge peripheral IV at a rate of 20 mL/h.

Vital signs: temperature, 39°C, heart rate (HR), 188, respiratory rate (RR), 64; blood pressure (BP), 75/42 mm Hg.

Diagnostic studies: Na, 137; K, 3.8; Cl, 100; serum urea nitrogen, 24; creatinine, 1.3.

How severe is Jamal's fluid volume deficit?

The severity of a child's fluid volume deficit is determined on the basis of clinical manifestations, diagnostic studies, and weight loss. Jamal's clinical picture is consistent with a moderate to severe fluid volume deficit (Table 2).

Table 2
Severity of fluid volume deficit

Clinical manifestations	Mild fluid deficit	Moderate fluid deficit	Severe fluid deficit
Mental Status			
Infants and young children	Thirsty, alert, restless	Thirsty, restless or lethargic but irritable to touch	Lethargic, somnolent
Older children and adults	Thirsty, alert, restless	Thirsty, alert	Usually conscious, apprehensive
Radial pulse	Normal rate and strength	Rapid and weak	Rapid, feeble, sometimes impalpable
Heart rate	Normal or mild tachycardia	Tachycardia	Severe tachycardia that may progress to bradycardia
Respirations	Normal	Normal to rapid	Deep and rapid
Fontanel & eyes	Normal	Slightly depressed	Severely sunken
Systolic blood pressure	Normal	Orthostatic hypotension	Severe hypotension
Skin elasticity	Pinch retracts immediately	Pinch retracts slowly	Pinch retracts very slowly (>3 sec)
Tears	Present	Present or absent	Absent
Mucous membranes	Moist	Dry	Very dry
Urine output	Normal	Oliguria	Oliguria or anuria
Body weight loss (%)	3–5	6–9	≥10
Estimated fluid deficit (mL/kg)	30–50	60–90	≥100

Data from Friedman AL. Nephrology: fluids and electrolytes. In: Behrman RE, Kliegman RM, editors. Nelson essentials of pediatrics. 4th Edition. Philadelphia: WB Saunders; 2002. p. 680; and AACN Pediatric Critical Care Pocket Reference Card © 1998.

What will Jamal's initial management include?

Initial management of the critically ill infant or child who has a fluid volume deficit focuses on expansion of the ECF volume to treat or prevent hypovolemic shock. Typically, 10 to 20 mL/kg of an isotonic solution is administered by way of an IV line. Infants or children who have a severe fluid volume deficit may require as much as 60 mL/kg [1]. Peripheral perfusion, heart rate, blood pressure, and urine output are monitored continuously to determine the infant or child's response to therapy.

Following initial fluid resuscitation, ongoing management is directed toward definitive replacement of water and electrolytes. In isotonatremic or hyponatremic dehydration (Box 1), further fluid losses generally are replaced over a period of 24 hours to prevent overexpansion of the ECF [3]. The need

Box 1. Sodium imbalances and dehydration

Hyponatremic dehydration

Na^+ is less than 135 mEq/L
Fluid shifts from ECF to ICF
Earlier signs of cardiovascular collapse

Isonatremic dehydration

Na^+ is 135 to 145 mEq/L
No fluid shifts

Hypernatremic dehydration

Na^+ is greater than 145 mEq/L
Fluid shifts from ICF to ECF
''Masking'' of symptoms

Box 2. Maintenance fluid calculations

Hourly method

1 to 10 kg: 4 mL/kg/h
11 to 20 kg: 40 mL/h + 2 mL/kg/h (for kg 11–20)
More than 20 kg: 60 mL/h + 1 mL/h (for every kg >20)

Daily method

1 to 10 kg: 100 mL/kg/d
11 to 20 kg: 1000 mL/d + 50 mL/kg/d (for kg 11–20)
More than 20 kg: 1500 mL/d + 20 mL/kg/d (for every kg >20)

Table 3
Electrolyte imbalances

Electrolyte imbalance	Causes	Clinical manifestations	ECG findings	Management
Hyponatremia $Na^+ < 135$ mEq/L	Vomiting/diarrhea Nasogastric suction ↓ Na^+ intake Fever Excessive diaphoresis ↑ water intake Burns & wounds Renal disease SIADH DKA Malnutrition	Irritability Seizures Lethargy Disorientation Cerebral edema Coma Respiratory failure Muscle cramps Nausea/vomiting Vary with alterations in fluid status	N/A	Treat underlying cause Frequent neurologic assessments Fluid replacement ±Hypertonic saline Monitor Na^+ levels
Hypernatremia $Na^+ > 145$ mEq/L	↑ Na^+ intake renal disease ↑ insensible water loss Diabetes insipidus	Irritability/agitation High-pitched cry Seizures Flushed skin Lethargy/confusion Seizures Coma Muscle weakness Muscle twitching Intense thirst Vary with alterations in fluid status	N/A	Treat underlying cause Frequent neurological assessments Strict I&O Slow correction of fluid deficit Monitor Na levels
Hypokalemia $K^+ < 3.5$ mEq/L	↓ K^+ intake Starvation Malabsorption syndromes Gastrointestinal losses Diuresis Nephritis Alkalosis	Muscle weakness, cramping, stiffness, paralysis, hyporeflexia Hypotension Lethargy Irritability Tetany Nausea/vomiting Abdominal distention Paralytic ileus Irregular, weak pulse	Flattened, inverted T waves Presence of U waves PVCs	Treat underlying cause Monitor ECG Frequent neuromuscular assessments K^+ replacement Monitor acid–base status

Hyperkalemia $K^+ > 5.5$ mEq/L	↑ K^+ intake Renal disease/failure Adrenal insufficiency Metabolic acidosis Severe dehydration Burns Crushing injuries Hemolysis	Muscle weakness Ascending paralysis Hyperreflexia Confusion Apnea N/V Diarrhea ↓ cardiac function	Tall, peaked T waves Widened QRS Prolonged PR interval Ventricular arrhythmias Asystole Cardiac arrest	Treat underlying cause Monitor ECG Administer IV fluids D/C K^+ containing fluids/meds IV calcium administration Insulin + glucose Albuterol Na bicarbonate Kayexalate Dialysis Monitor serum K^+ levels Evaluate acid–base status
Hypocalcemia $Ca^+ < 8$mg/dL iCa < 1.15	↓ dietary Ca Vitamin D deficiency Renal insufficiency Diuretics Hypoparathyroidism Alkalosis ↑ serum protein	NM irritability Tingling sensation Chvostek's sign Trousseau's sign Tetany Muscle cramps Lethargy Seizures Hypotension	Prolonged QT interval	Treat underlying cause Monitor ECG IV calcium supplements Monitor Ca & Mg levels
Hypercalcemia $Ca^+ > 10.5$ mg/dL iCa > 1.34	Acidosis Prolonged immobilization Kidney disease Hyperparathyroidism Excessive administration	Lethargy Stupor Coma Seizures Anorexia N/V Constipation NM hypotonicity	Shortened QT interval Bradycardia Cardiac arrest	Treat underlying cause Monitor ECG IV fluids Loop diuretics
Hypomagnesemia $Mg^+ < 1.4$ mEq/L	↓ intake (NPO) Malabsorption syndromes ↑ renal excretion	NM excitability Tetany Confusion Dizziness Headache Seizures Coma Respiratory depression Tachycardia	PVCs Ventricular arrhythmias	Treat underlying cause IV Mg replacement Monitor ECG Neuromuscular assessments

(continued on next page)

Table 3 (*continued*)

Electrolyte imbalance	Causes	Clinical manifestations	ECG findings	Management
Hypermagnesemia $Mg^{+} < 1.4$ mEq/L	Chronic renal disease ↓ GFR/↓ excretion ECF deficit ↑ administration of Mg containing drugs	Lethargy Muscle weakness Seizures ↓ swallow ↓ gag Tachycardia Hypotension	Prolonged PR interval Prolonged QRS Prolonged QT AV block	Treat underlying cause Monitor ECG Administer calcium IV hydration Dialysis
Hypophosphatemia $PO_4 < 3$ mg/dL	Limited intake Shift of PO_4 from ECF to ICF ↓ GI tract absorption ↑ renal excretion	Irritability Disorientation Tremors Seizures Hemolytic anemia ↓ myocardial function Potential respiratory failure Coma	Premature ectopic beats	Treat underlying cause Slow PO_4 replacement Monitor for other electrolyte imbalances
Hyperphosphatemia $PO_4 > 4.5$ mEq/L	Chronic renal failure Rapid cell catabolism Excessive intake Neoplastic disease Hypoparathyroidism	Tachycardia Hyperreflexia Abdominal cramps Nausea Diarrhea Muscle tetany	N/A	Treat underlying cause Monitor PO_4 and Ca Dietary restrictions Antacid administration Hydration Correction of hypocalcemia Dialysis

Abbreviations: AV, atrioventricular; D/C, discontinue; DKA, diabetic ketoacidosis; GFR, glomerular filtration rate; GI, gastrointestinal; iCa, ionized calcium; I&O, intake & output; NM, neuromuscular; N/V, ; SIADH, syndrome of inappropriate antidiuretic hormone.

for sodium replacement should be considered in hyponatremic dehydration. In hypernatremic dehydration, these losses may be administered more slowly, over a period of 48 to 72 hours. It is important that replacement fluids not be administered too rapidly in hypernatremic dehydration as this may lead to neurologic complications related to rapid changes in serum sodium levels. Replacement fluids are administered in addition to maintenance fluids (Box 2). Potassium chloride may be added to any maintenance fluids once Jamal is producing adequate urine output. The need to replace any ongoing fluid losses must be assessed as well.

What other factors may be contributing to Jamal's fluid loss?

Factors, such as hyperthermia, hyperventilation, increased metabolic rate/activity, and overhead warmers/phototherapy, can increase insensible fluid losses. Hypothermia or sedation may lead to a decrease in insensible fluid losses. It is important to consider potential causes of increased or decreased insensible fluid losses when assessing fluid balance in the critically ill infant or child.

Why do infants, such as Jamal, have an increased risk for developing a fluid volume deficit?

During early infancy, most body water is found in the ECF. This contributes to greater and more rapid fluid loss. If an infant has decreased intake or has excessive fluid losses, a fluid volume deficit or dehydration may develop rapidly. The resulting decrease in intravascular volume leads to a decrease in circulating blood volume and inadequate systemic perfusion. Other factors that place infants at increased risk for fluid volume deficit include an increased metabolic rate, relatively greater body surface area, immature renal function, and increased fluid requirements [4].

Electrolyte imbalances

See Table 3 for a summary of electrolyte imbalances in the critically ill infant and child.

Sodium balance

Sodium (Na^+) is the major cation of the extracellular compartment. It regulates the voltage of action potentials in skeletal muscles, nerves, and the myocardium. Sodium plays a role in the maintenance of acid–base balance, and maintenance of fluid balance in the ECF through maintenance of the osmotic pressure (osmolality). Consequently, imbalances in water and sodium often occur together and are equated with alterations in serum osmolality. Extracellular sodium concentration normally is 135 to 145 mEq/L. The major factors that influence sodium excretion are glomerular filtration rate and aldosterone. Alterations in the sodium levels in the body often are the result of clinical conditions that involve fluid volume excess or deficit.

Hyponatremia is defined as a serum sodium concentration of less than 135 mEq/L. Typically, it occurs as a secondary manifestation of another disease state. In the critically ill infant or child, hyponatremia may occur as the result of excess water retention in the ECF, sodium loss from the ECF, or a combination of the two [3]. Hyponatremia may occur in conjunction with hypovolemia, euvolemia, or hypervolemia.

A decrease in serum sodium results in a shift in water from the ECF to the ICF. This shift in fluids leads to a generalized cellular swelling or edema. Within the brain, where there is limited capacity for expansion, the development of cerebral edema can have catastrophic consequences (eg, cerebral herniation and death). Severity of clinical symptoms directly correlates with the severity and rapidity of onset of the sodium deficit [5]. Serum sodium levels of less than 120 mEq/L are associated with seizures and coma. Children who develop hyponatremia over several days to several weeks may be asymptomatic or may develop mild clinical manifestations.

Hyponatremia can occur in conjunction with hypervolemia, euvolemia, or hypovolemia. Water intoxication, nephrotic syndrome, cardiac failure, renal failure, and the syndrome of inappropriate antidiuretic hormone (SIADH) are causes of hypervolemic hyponatremia. Hyponatremia in conjunction with hypovolemia may occur with renal (eg, osmotic diuresis, renal tubular acidosis) or extrarenal (eg, vomiting, diarrhea, burns) losses [3]. Other potential causes include excessive use of diuretics, osmotic diuresis, and adrenal insufficiency.

Case study #2

Marisa is a 16-year-old girl who was admitted to the PICU following a spinal fusion procedure.

Vital signs: temperature, 37.2°C; HR, 82; RR, 18; BP 110/68 mm Hg; spO_2, 97%.

Diagnostic studies: Na, 125 mEq/L; K, 3.5 mEq/L; Cl, 99; serum urea nitrogen, 10; creatinine, 0.3, serum osmolarity, 258.

On postoperative day 2, her urine output is noted to decrease from 1.5 mL/kg/h to 0.2 mL/kg/h.

What clinical condition might you suspect is occurring and how is this contributing to Marisa's hyponatremia?

SIADH develops as the result of excessive levels of circulating antidiuretic hormone (ADH). It is one of the most common causes of hyponatremia in children in a hospital setting [6]. SIADH associated with spinal surgeries has been reported in the literature [7]. Increased levels of circulating ADH result in the reabsorption of water that normally would be excreted in urine by the kidneys, and the development of fluid volume overload. This, in turn, leads to a dilutional hyponatremia and a decreased serum osmolality. The decreased osmolality causes a shift of fluid from the ECF to the ICF which can lead to the development of cerebral edema.

What causes the clinical manifestations that are associated with hyponatremia?

The clinical manifestations of hyponatremia are related to the cause of the hyponatremia and the rapidity of onset. Most of the symptoms occur as the result of the intracellular shift of water. Typically, the severe clinical manifestations of hyponatremia are not seen until the serum sodium decreases to levels of 120 to 125 mEq/L. At levels of less than 120 mEq/L, seizures, coma, and permanent neurologic damage may occur. Children often are at a higher risk for developing neurologic symptoms than are their adult counterparts.

What will Marisa's initial management include?

The first goal of management is to identify and treat/control the underlying cause of the hyponatremia. In Marisa's case, SIADH has been identified as the cause. In the child who has hypervolemia, management may include fluid restriction, administration of loop diuretics, and close monitoring of serum sodium levels. If serum sodium levels are increased too rapidly, cellular dehydration and neurologic damage may result. A general rule of thumb is that the serum sodium should increase no faster than 0.5 to 1.0 mEq/L/h. Other monitoring includes strict intake & output (I & O), urine specific gravity, serum electrolytes and serum osmolality (4–6 h), and daily weights.

In some cases, hypertonic saline may be considered to elevate serum sodium levels to 120 to 125 mEq/L. In the child who has hypovolemia, fluid and sodium losses must be replaced. This may or may not be done with a hypertonic saline solution.

Hypernatremia is defined as an excess of sodium in the ECF. It exists when serum sodium levels exceed 145 mEq/L. Hypernatremia may occur as the result of a pure sodium excess (eg, administration of large amounts of sodium bicarbonate) or as the result of a water deficit. Conditions that may lead to hypernatremic fluid deficit in the critically ill infant or child include diabetes insipidus, diabetes mellitus, increased insensible water loss, diarrhea, and dehydration.

The body normally responds to an increase in serum sodium with the release of ADH and stimulation of the thirst mechanism in an attempt to retain water and decrease serum sodium; however, this compensatory mechanism may not be sufficient to prevent the serum sodium from continuing to increase in the critically ill infant and child. Additionally, those patients who are unable to produce or respond to ADH are at an increased risk for the development of hypernatremia. Patients who are at risk for developing hypernatremia should be identified early on in their admission and monitored for clinical symptoms. Hypernatremia initially causes a generalized shrinking of cells as fluid moves from the ICF to the ECF. This may lead to subarachnoid, intradural, or subdural hemorrhages. Permanent central nervous system dysfunction can result when serum sodium concentrations reach levels greater than 160 mEq/L [8].

Case study #3

Hannah is a full-term, 1-month-old girl who was found pulseless and apneic after being put down for a nap. Cardiopulmonary resuscitation was initiated by the family and she was resuscitated and transported to the Emergency Department by Emergency Medical Services. Her grandmother reports that she has been irritable and "colicky" with decreased oral intake for the past 7 to 10 days. Administration of approximately 1/2 teaspoon of baking soda each day was the home remedy that was used to treat her symptoms. She was transferred to the PICU from the Emergency Department.

Hannah's physical examination upon admission to the ICU is as follows: intubated and mechanically ventilated, unresponsive, tachycardic, slightly hypotensive, sunken anterior fontanel, and "doughy" skin. She received a normal saline bolus of 10 mL/kg while

in the Emergency Department; a second bolus is now infusing.

Diagnostic studies: Na, 165; K, 4.2; Cl, 125; CO_2, 14; serum urea nitrogen, 14; glucose, 167.

What will Hannah's initial management strategies include?

Hannah has a significant hypernatremia and fluid volume deficit. The cause of the hypernatremia was the administration of baking soda (sodium bicarbonate) over a period of approximately 1 week. Any patient who has hypernatremia needs to be monitored for seizure activity. Following initial resuscitation and stabilization, an electroencephalogram reveals that Hannah is having frequent subclinical seizure activity. Appropriate antiepileptic therapy is initiated.

The hypovolemic child requires fluid replacement and a slow correction of her fluid deficit over 48 to 72 hours. Patients who have a serum sodium level of 150 to 160 mEq/L should receive replacement fluids over a 24-hour time period. Those who have a serum sodium level that is greater than 160 mEq/L should receive fluid replacement therapy over a greater period of time [8]. Hannah's fluid replacement was administered over 48 hours. The type of IV fluids administered will vary depending on the rate at which the serum sodium level is decreasing. Generally, the serum sodium level should decrease at a rate no faster than 0.5 to 1.0 mEq/L/h, because rapid correction of hypernatremia can lead to fluid shifts from the ECF to the ICF and the development of cerebral edema. Patients must be monitored for the signs and symptoms of cerebral edema throughout the course of their treatment. Typically, the hypervolemic child is managed with diuretics and restricted sodium administration.

Ongoing management includes frequent neurologic assessments, strict I/O to monitor fluid balance, and frequent monitoring of serum sodium levels. Depending on the severity of the neurologic manifestations, patients may require ongoing management of seizures and rehabilitation.

Potassium balance

Potassium is the body's primary intracellular cation. Potassium has four major functions within the body: maintenance of cells' electrical neutrality and osmolality, neuromuscular transmission of nerve impulses, skeletal and cardiac muscle contraction and electrical conductivity, and maintenance of acid–base balance [9]. Maintenance of intracellular osmolarity is accomplished through the "sodium–potassium" (active transport) pump. The normal range of serum potassium is 3.5 to 5.5 mEq/L, with a concentration of 160 mEq/L inside the cell.

Hypokalemia is defined as a serum potassium concentration of less than 3.5 mEq/L. Hypokalemia occurs as the result of a true deficit of potassium or a shift in potassium out of the ECF (intravascular space) into the ICF. A true deficit of potassium may be caused by decreased intake; however, excessive renal secretion, excessive gastrointestinal losses, or excessive sweating are more common causes of a potassium deficit. Conditions, such as alkalosis, and the excessive secretion or administration of insulin are potential causes of a shift of potassium out of the intravascular space into the ICF.

Case study #4

Malaki is a 2-month-old boy with trisomy 21, reactive airway disease, who is status post a complete atrioventricular canal repair at 2 weeks of age. He was admitted to the PICU unit 2 days ago in significant respiratory distress. An RSV titer is pending. He is in a 60% oxygen tent and has received additional treatments over the course of the day. He also is receiving his maintenance doses of digoxin and furosemide.

Malaki's oral intake has decreased significantly over the past 12 hours, although his urine output remains 2 mL/kg/h. He is lethargic and intermittently irritable. Serum chemistries reveal the following: Na, 138; K, 2.5; Cl, 97; PO_4, 4.

What factors may be contributing to Malaki's hypokalemia?

Several factors are contributing to Malaki's hypokalemia. Malaki has received albuterol and furosemide during his hospitalization. Albuterol activates the sodium–potassium pump and forces potassium to move intracellularly. Furosemide, a thiazide diuretic, causes increased excretion of potassium in the urine. This, in combination with his decreased oral intake, may be a contributing factor. As a child with a history of congenital heart disease, Malaki is at increased risk for the cardiovascular complications of hypokalemia.

What other diagnostic studies would you expect to be done?

Hypokalemia can cause several EKG changes—flattened, inverted T waves; presence of U waves; and

premature ventricular contractions (PVCs). A 12-lead EKG should be performed, and, if not already in place, the patient should be placed on an EKG monitor. Additionally, a digoxin level must be measured. Hypokalemia predisposes patients to digoxin toxicity, and it is important to monitor Malaki for this potential complication of his hypokalemia.

What will Malaki's initial management strategies include?

The underlying cause of the hypokalemia must be identified and controlled. In a case such as this, holding at least one dosage of his maintenance furosemide may be appropriate. He should be assessed for the development of a fluid volume deficit, and appropriate IV maintenance fluids should be administered. Additionally, ECG, cardiac function, and neuromuscular examination must be monitored closely. Typically, potassium supplements are administered when the serum potassium level is less than 3 mEq/L (Box 3). ECG, cardiac function, and serum potassium levels are monitored frequently until the cause of the hypokalemia has been corrected or controlled.

Hyperkalemia is defined as a serum potassium level of greater than 5.5 mEq/L [9]. Typically, hyperkalemia occurs as the result of altered renal excretion of potassium, impaired extrarenal regulation, a shift from the ICF to the ECF, or increased potassium intake. Alterations in renal excretion of potassium may result from a decrease in the glomerular filtration rate or a decrease in potassium secretion by the renal tubules [9].

Box 3. Administration of potassium supplements

Treatment of hypokalemia:

Two to 5 mEq/kg/d in divided dosages
Intermittent infusion by way of syringe pump or infusion pump

Maximum concentration

Peripheral: 0.08 mEq/mL
Central: 0.4 mEq/mL

Maximum administration rate

1 mEq/kg/h and monitor EKG for dosages greater than 0.3 mEq/kg/h

Case study #5

Andrew is a previously healthy 14-month-old boy who was admitted to the ICU with a new-onset leukemia and suspected tumor lysis syndrome (TLS). He is lethargic and irritable. Initial diagnostic studies reveal a serum potassium of 7.2 mEq/L and the following arterial blood gas results: pH 7.30 pCO2 35 pO2 91 HCO3 18. Intermittent PVC's are noted on his cardiac monitor.

Vital signs: temperature, 37.7°C; pulse, 174; respiration, 32; BP 80/54 mm Hg; spO_2, 99%.

What factors are contributing to Andrew's hyperkalemia?

TLS is a group of metabolic effects that is associated with rapidly growing tumors. The metabolic abnormalities that are seen in TLS include hyperkalemia, hyperphosphatemia, hyperuricemia, and hypocalcemia (as a secondary effect of the hyperphosphatemia) [10]. The metabolic acidosis also is a contributing factor. Acidosis results in a shift of potassium from the ICF to the ECF. Although there is not a change in total body potassium content, this shift into the extracellular space also results in the clinical manifestations of hyperkalemia. These clinical manifestations are related to alterations in neuromuscular and cardiac functioning.

What will initial management strategies include?

Treatment of hyperkalemia varies depending upon the clinical presentation. If the potassium level is less than 6.5 mEq/L and no ECG changes are noted, discontinuation of fluids and medications containing potassium, along with close monitoring of serum potassium levels, may be sufficient. Administration of polystyrene sulfonate (Kayexalate) to increase potassium excretion also decreases potassium levels. Kayexalate can bind to calcium and magnesium in addition to potassium; therefore, it is important to monitor these electrolyte levels. Additionally, patients must be monitored for the clinical manifestations of hypocalcemia and hypomagnesemia. Potassium levels greater than 6.5 mEq/L or those that produce ECG changes require immediate treatment. Calcium gluconate (60–100 mg/kg) is administered to reduce the cardiac toxicity that is associated with hyperkalemia. The onset of action of calcium occurs within minutes

and the effects last for approximately 30 minutes. Additionally, IV fluids are administered to expand ECF volume and to decrease the concentration of potassium in the ECF; however, this may not be a viable option in the child who has hyperkalemia and renal failure.

Redistribution of potassium from the ECF to ICF is necessary to decrease the elevated serum potassium level. Administration of insulin (0.1 unit/kg) stimulates the sodium–potassium pump and results in increased cellular uptake of potassium. Insulin should be administered in conjunction with dextrose (0.5–1 g/kg). A decrease in serum potassium should begin within 15 minutes and lasts for approximately 60 minutes [11].

The administration of sodium bicarbonate (1–2 mEq/L) also causes an intracellular shift of serum potassium; however, it may take up to 60 minutes for the sodium bicarbonate to decrease serum potassium levels [11]. The effects will last for several hours. Acid–base status is monitored closely in children who are receiving this therapy. Children who have respiratory failure are evaluated carefully, because sodium bicarbonate increases CO_2 production and may worsen respiratory acidosis if CO_2 cannot be excreted by the lungs. The development of hypernatremia is another potential complication of this therapy.

Albuterol, a β_2 adrenergic agonist, activates the sodium–potassium pump and stimulates the pancreas to release insulin, thereby shifting potassium into the cells [9]. It is administered by way of inhalation or IV infusion. It also has been recommended that albuterol be administered in conjunction with insulin because of the additive effect of the two drugs [9,11].

Calcium balance

Calcium, in conjunction with phosphorus and magnesium, plays an important role in nerve transmission, bone composition, and regulation of enzymatic processes. Homeostasis of these three electrolytes occurs through intestinal absorption and renal excretion. Most calcium is stored in the bones and teeth, and the remainder is found in soft tissue and serum. Of the calcium that is found within the serum, approximately 50% is bound to the protein or anions and is unavailable for use by the body. The remaining 50% is ionized and is available for the essential bodily functions of cardiac function, muscular contraction, nerve impulse transmission, and clotting. The ionized calcium level is of greatest physiologic significance, and direct measurement of ionized calcium is essential during a clinically important situation in which calcium levels may play a role.

Hypocalcemia is defined as a decrease of calcium in the ECF; it exists when serum calcium levels are less than 8 mg/dL in full-term infants and older children, and when ionized calcium levels are less than 4 mg/dL or 1.15 mmol/L. There are numerous potential causes of hypocalcemia, including, but not limited to, hypoparathyroidism, hypomagnesemia, hyperphosphatemia, vitamin D deficiency, calcium deficiency, impaired renal function, malabsorption syndromes, anticonvulsant therapy, nephrotic syndrome, acute pancreatitis, and transfusion with citrate-preserved blood [1,12].

Case study #6

Matthew is an 11-day-old boy who is postoperative day #2 following a tetralogy of Fallot repair. His course has been complicated by issues with postoperative bleeding and seizures. He has had no seizure activity over the past 12 hours; however, he required transfusion of multiple blood products overnight and continues to have moderate amounts of bloody drainage via his chest tube.

Vital signs: temperature, 37.5°C; pulse, 180; respiration, 20; BP, 66/31(45). A recent blood gas reveals an ionized calcium of 0.92 mmol/L.

Based on the above information, what is the most likely cause of Matthew's hypocalcemia?

The hypocalcemia most likely is related to the multiple blood products that were administered overnight. Citrate, a commonly used preservative in blood products, binds with calcium, and makes it unavailable for use by the body.

Why would you be especially concerned about hypocalcemia in this patient?

He already is at increased risk for compromised cardiac function following his surgery. Adequate levels of serum calcium are necessary for optimal cardiac function. Hypocalcemia is associated with arrhythmias, such as prolonged QT interval. This patient also has been having issues with bleeding, and calcium plays a role in the activation of clotting mechanisms. Additionally, he already has had problems with seizures postoperatively and hypocalcemia is a known cause of seizure activity.

What will initial management strategies include?

Acute management of hypocalcemia involves treating the cause and administering calcium supplements as needed. Patients must be monitored for cardiac, neurologic, and neuromuscular dysfunction. Concurrent conditions (eg, hyperphosphatemia, hypomagnesemia, respiratory alkalosis) must be identified and treated as needed [1].

Acute hypocalcemia in the child who is at risk for impending cardiovascular or neurologic failure must be treated immediately through restoration of ionized calcium levels. This is accomplished through the IV administration of calcium gluconate (100 mg/kg) or calcium chloride (10–20 mg/kg). While administering calcium supplements, monitor for arrhythmias. Rapid administration of calcium salts has been associated with bradycardia and asystole [13].

Hypercalcemia occurs as the result of an excess of calcium in the ECF, and the total serum calcium level is greater than 10.5 to 11 mg/dL. Generally, symptoms are not seen until the serum calcium level is greater than 12 mg/dL. Levels greater than 15 mg/dL may be life-threatening. Hypercalcemia is not a common occurrence, but may be seen in the critically ill child. In this population, it typically is associated with hyponatremia, hyperkalemia, resolution of chronic renal failure, and prolonged immobility.

Case study #7

John is a 17-year-old boy who has acute myelogenous leukemia and received a bone marrow transplant 77 days ago. He developed acute respiratory distress syndrome following his transplant, and was intubated and mechanically ventilated in the PICU for approximately 2 months. He spent a substantial portion of this time period heavily sedated and receiving neuromuscular blockade. A tracheostomy tube was placed 5 days ago and his mechanical ventilation is being weaned aggressively. His serum calcium levels have been climbing gradually over the past 7 to 10 days, and his current level is 13.2 mEq/L. Other electrolyte levels are within normal limits. He is lethargic and complains intermittently of nausea.

What is the most likely cause of John's hypercalcemia?

Prolonged immobility is the most likely cause of the hypercalcemia. During prolonged immobility, calcium moves from the bones, teeth, and intestine into the bloodstream to compensate for hypocalcemia. Long-term complications of immobility include osteoporosis and osteomalacia; however, hypercalcemia may occur during early stages when calcium is moving out of the bones and into the serum [14]. This calcium influx into the ECF can overwhelm the calcium regulatory hormones (parathyroid hormone and vitamin D) and renal excretion mechanisms [1].

Protein and pH also affect calcium levels. Increased albumin levels result in increased serum calcium levels. A decrease in serum pH increases ionized calcium, because more calcium is removed from protein binding sites and is available for participation in chemical reactions.

What will initial management strategies include?

Patients who have hypercalcemia and those who are at risk for the development of hypercalcemia must be identified and monitored for the related clinical manifestations. Particular attention should be paid to ECG monitoring and neurologic and gastrointestinal examinations. Calcium levels of greater than 15 mg/dL may be life-threatening and must be treated immediately. The goal of treatment is to reduce the amount of calcium in the ECF. This is accomplished through administration of IV fluids and loop diuretics. Thiazide diuretics are contraindicated because they restrict calcium excretion.

In John's case, his calcium levels are less than 15 mg/dL, and he is not exhibiting significant clinical manifestations. His hypercalcemia may be managed by increasing his fluid intake, adjusting his diuretic schedule to increase calcium excretion, and monitoring serum calcium levels. It also is essential to coordinate his plan of care with occupational and physical therapy services to increase his mobility and prepare him for transfer out of the ICU setting.

Summary

This article has presented an overview of several fluid and electrolyte imbalances that may occur in the critically ill infant or child. Imbalances in magnesium and phosphorous have not been discussed in detail in this article; an overview of clinical manifestations and management can be found in Table 3.

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