Pathophysiology of Hypovolemia and Dehydration

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by James S. Kennedy, MD, CCS

This year ICD-9-CM expanded code 276.5, Volume depletion, and created the more specific codes 276.51, Dehydration, and 276.52, Hypovolemia, at the request of the American Academy of Pediatrics.[1] As treatments for these conditions differ and accurate coding must reflect illness severity and mortality risk for newly implemented reimbursement methodologies, coding professionals should review the pathophysiology and complications of hypovolemia and dehydration.

Dehydration ≠ Volume Depletion

Water comprises approximately 60 percent of body weight in adults, 65 percent in children, and 70 percent in infants. Intravascular space contains 25 percent of total body water, with the rest dispersed to extravascular tissue.

Dehydration is the depletion of more than 3 percent of total body water. It's usually caused by hypernatremia and hyperosmolality, unless there are excessive salt losses from renal (diuretics, diabetic ketoacidosis, interstitial kidney disease), gastrointestinal (gastroenteritis), or sweat gland (cystic fibrosis) disorders.

Hypovolemia is defined as impaired intravascular volume from which organ and tissue perfusion is reduced. It is caused by the contraction of extracellular fluid from net loss of body fluid, including diarrhea and vomiting; renal or cerebral salt wasting; extravasation of plasma into tissues from infection, burns, or trauma ("third space loss"); or dislocation of blood volume into capacitance vessels due to septic shock or loss of muscle tone.[2]

Due to higher metabolic rates and surface-to-body ratios, children are more prone to fluid loss, dehydration, and hypovolemia than adults. Tissue hypoperfusion from oliguria, lactic acidosis, or fluid-resistant hypotension can cause shock, which is almost universally fatal if not addressed early.

Clinical Manifestations and Diagnosis

Dehydration manifests itself differently in pediatrics and adults. In children, dehydration is classified as:

- **Mild** (3 to 5 percent): normal or increased pulse, decreased urine output, thirsty, normal physical examination. Patients are not hypovolemic.
- **Moderate** (7 to 10 percent): tachycardia, little or no urine output, irritable and lethargic, sunken eyes and fontanel, decreased tears, dry mucous membranes, mild tenting of the skin, delayed capillary refill, cool and pale. Hypovolemia is present from the oliguria.
- **Severe** (10 to 15 percent): rapid and weak pulse, decreased blood pressure, no urine output, very sunken eyes and fontanel, no tears, parched mucous membranes, tenting of the skin, very delayed capillary refill, cold and mottled. Shock is likely present.[3]

Adults can lose 7 to 10 percent of their water volume and still maintain normal vital signs. Symptoms vary according to the volume and rate of fluid loss, the nature of the loss (water alone, combined sodium and water, or blood), and vascular tone. Skin findings and mucous membrane moistness are valuable in the pediatric assessment; however, they add little to an adult exam. A10-mm Hg fall in systolic blood pressure when a patient is brought from a supine to standing position is generally the most accepted sign that an adult is dehydrated or hypovolemic. Cold clammy extremities, delirium, oliguria, and volume-resistant hypotension indicate shock.

Laboratory studies help diagnose dehydration or hypovolemia and give clues to their causes. In mild dehydration, the urine specific gravity is usually more than 1.020, whereas it is above 1.030 in hypovolemia. The serum sodium (Na) concentration may be high, normal, or low, depending on the cause. A disproportionate rise in the blood urea nitrogen (BUN) to creatinine

ratio of over 25 to 1 indicates hypovolemia. A usual finding is a BUN over 30 with a relatively normal creatinine. A rise of the serum creatinine by 0.5 mg/dl or more over baseline indicates profound hypovolemia and dehydration leading to acute renal failure.[4] Metabolic acidosis (serum pH < 7.30, HCO3 < 20 meq/l) usually suggests shock.

Underlying Causes

Dehydration and hypovolemia are commonly caused by:

- Renal losses: hormonal deficit, pituitary diabetes insipidus, aldosterone insufficiency, Addison's disease, hyporeninemic hypoaldosteronism, fenal tubular acidosis, Bartter's syndrome, nephrogenic diabetes insipidus, diuretic abuse, postobstructive diuresis, osmotic diuresis, late-stage or end-stage chronic kidney disease, interstitial nephritis
- Extrarenal losses: hemorrhage (acute blood loss), sweating, burns, heat stroke, diarrhea, vomiting, nasogastric tube drainage, gastrointestinal fistulas, cystic fibrosis, fever
- Decreased oral intake: pharyngitis (especially in children), encephalopathies, dementia, delirium, coma, NPO status

Laboratory studies may determine the etiology. The baseline hematocrit may be high in simple dehydration due to hemoconcentration but low due to hemorrhage due to gastrointestinal bleeding or trauma. If serum proteins or albumin are low, a chronic disease or malnutrition may be present. Low serum sodium values suggest renal or gastrointestinal causes, whereas high values indicate otherwise. Low serum potassium values suggest diuretic use or gastrointestinal fluid loss, whereas high serum potassium suggests diabetic ketoacidosis, Addison's disease, or shock as a contributing factor. The attending physician should be queried when the clinical significance of abnormal laboratory studies has not been clearly documented.

Looking for Complications

Dehydration and hypovolemia increase morbidity and mortality if not prevented or rapidly addressed. Orthostatic hypotension can lead to dizziness, giddiness, and, if severe enough, outright syncope. Outright hypovolemia leads to lactic acidosis and hypotension resistant to fluid administration, consistent with shock. As the renal glomerular filtration rate reduces, acute renal insufficiency and possibly failure (defined as a reduction of the GFR by 50 percent or an increase in the serum creatinine by more than 0.5 mg/dl over baseline) ensues.[5] Reduced coronary blood flow may accelerate angina pectoris to occur at rest or even result in a myocardial infarction. Once shock is established, the metabolic acidosis progresses until heart failure or hyperkalemia leads to cardiac arrest and death.

Coding Issues

Coders have a critical role in identifying dehydration and hypovolemia as part of their efforts to promote data quality. Working with the medical staff, five elements of the patient's presentation must be thoroughly identified prior to code assignment and sequencing:

- **M**anifestation: syncope, lethargy, dizziness, anuria, oliguria, coma
- **U**nderlying cause (refer to the list of underlying causes above)
- **S**everity: dehydration alone or with hypovolemia or shock
- **I**nstigating cause: automobile accident, pharyngitis, patient restraints, urinary tract infection, sepsis, drug overdose, or poisoning
- **C**omplication: angina at rest or progressive angina, transient ischemic attack, acute renal failure, acute myocardial infarction, anoxic encephalopathy, systemic inflammatory response syndrome due to noninfectious or infectious causes

The correct code sequencing can be difficult to determine since dehydration or hypovolemia is often the result of another condition, as well as a severe management problem. Sequencing depends on the circumstances of the admission and the judgment of the physician. It is important to follow the definition of "principal diagnosis" as defined in ICD-9-CM.

ICD-9-CM guidelines have no specific rules regarding the sequencing of dehydration and hypovolemia should they be present on admission. *Coding Clinic* addresses acute renal failure due to dehydration, instructing coding professionals to code acute renal failure first followed by dehydration.[6,7] In the 1988 second quarter issue, *Coding Clinic* emphasizes that the circumstances of admission and the judgment of the attending physician determine if dehydration or hypovolemia should be sequenced first since the underlying cause may not be what necessitated the actual admission to the hospital. In the examples

given, dehydration is listed as the principal diagnosis if the underlying cause is not addressed or could have been treated as an outpatient. In another example, the underlying cause (adrenal metatasis) is sequenced first since dehydration is integral to this condition. Physician discussion in the history and physical, progress notes, and discharge summary of the importance of dehydration and its relationship to the underlying cause in occasioning the hospitalization is crucial in sequencing these conditions correctly.

As discussed, dehydration is the result of an underlying cause; but as *Coding Clinic* illustrates, the underlying cause may not be what necessitated the actual admission to the hospital. If the underlying cause could have been managed in the outpatient setting, the dehydration may be listed as the principal diagnosis when it necessitated the inpatient admission.

With the announcement of the proposed Medicare Consolidated Severity-Adjusted DRGs, coders should examine the All Payer Refined DRG definitions manual currently available at www.aprdrgassign.com to review the coding changes. Both dehydration and hypovolemia are relatively low weighted as secondary diagnoses in this methodology, except in the elderly, neonatal, and obstetrical patient. Acute renal failure, shock, and other underlying causes and complications receive appropriate higher weights. Mortality risk algorithms available from the Delta Group (www.thedeltagroup.com), 3M (www.3mhis.com), Healthgrades (www.healthgrades.com), or other quality measurement organizations such as the Pennsylvania Healthcare Cost Containment Council (www.phc4.org) are worthy of inquiry as well.

The new dehydration and hypovolemia codes are welcome additions to ICD-9-CM, especially in pediatric patients. Coders have a critical responsibility to partner with attending physicians for complete documentation and coding of all associated conditions.

Notes

1. Schraffenberger, Lou Ann. “ICD-9-CM Changes for Fiscal Year 2006.” *Journal of AHIMA* 76, no. 8 (2005): 70–76.
2. Holliday, M.A., et al. “Acute Hospital-induced Hyponatremia in Children: A Physiologic Approach.” *Journal of Pediatrics* 145, no. 5 (2004): 584–87.
3. Behrman, Richard, ed. *Nelson Textbook of Pediatrics*, 17th ed. Philadelphia, PA: Elsevier, 2006, p. 247.
4. Singri N., S. Ahya, and M. Levin. “Acute Renal Failure.” *Journal of the American Medical Association* 289, no. 3 (2003): 747–51.
5. Ibid.
6. American Hospital Association. *Coding Clinic*, first quarter (2003): 22.
7. American Hospital Association. *Coding Clinic*, third quarter (2002): 21, 22

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