Fluoid balance in ckd

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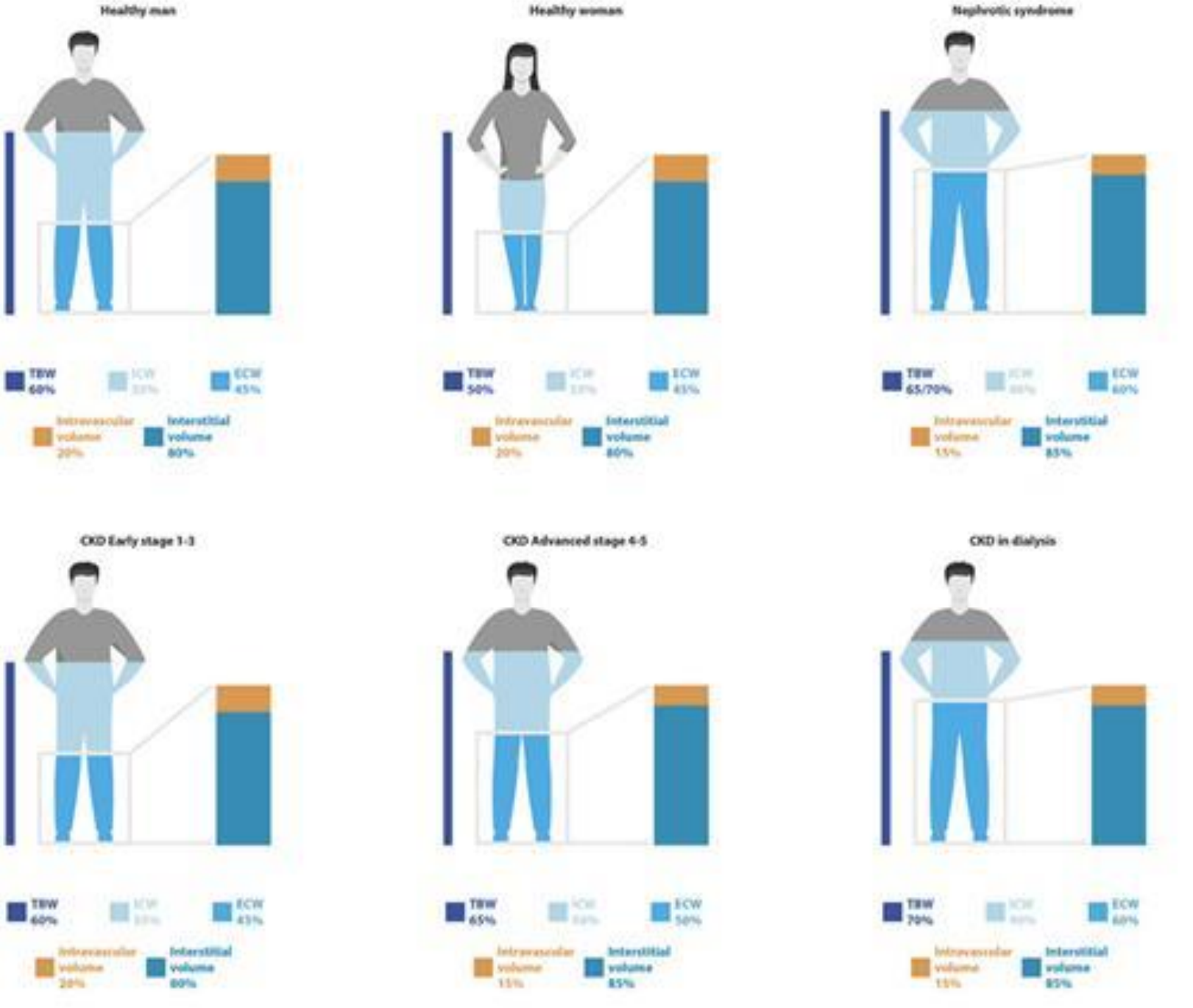
Healthy man
TBW 60%
ECW 45%
Intravascular volume 20%
Interstitial volume 80%
Healthy woman
TBW 50%
ECW 45%
Intravascular volume 20%
Interstitial volume 80%
Nephrotic syndrome
TBW 65/70%
ECW 60%
Intravascular volume 15%
Interstitial volume 85%
CKD Early stage 1-3
TBW 60%
ECW 45%
Intravascular volume 20%
Interstitial volume 80%
CKD Advanced stage 4-5
TBW 65%
ECW 50%
Intravascular volume 15%
Interstitial volume 85%
CKD in dialysis
TBW 70%
ECW 60%
Intravascular volume 15%
Interstitial volume 85%

Intravenous fluid therapy

- Intravenous (IV) fluid therapy is a crucial component of critical care of hospitalized patients.
- Fluid replacement strategies, including the **type** and **dose** of fluid administered, are a crucial part of the hospitalized patient care, requiring a high level of clinical experience
- various complications including metabolic acidosis, electrolyte imbalances, acute kidney injury (AKI) or progression of chronic kidney disease (CKD), volume overload or dehydration, lung injury, imbalance between pro-inflammatory and anti-inflammatory signals, defects in tissue perfusion and tissue damage.

4 stages of IV fluid therapy

- 1-the resuscitation phase, aiming to restore tissue perfusion in patients with hemodynamic instability and/or large total body volume loss
- 2-the replacement phase, aiming to restore physiological fluid and electrolyte balance in patients with either deficit or ongoing losses
- 3-the routine maintenance phase, aiming to maintain physiological fluid and electrolyte balance in patients unable to use the enteral route for fluid intake
- 4-the redistribution phase, aiming to establish the physiological balance between intravascular and extravascular fluids especially in patients with poor intravascular fluid retention.

Crystalloid fluids

- Crystalloid fluids can be classified into non-balanced fluids such as 0.9% normal saline or balanced fluids such as Ringer's lactate or Plasma-Lyte.

Normal saline

- Normal saline (0.9% NaCl; i.e. 154 mmol/L Na and 154 mmol/L Cl, osmolarity 308 mOsm/L) is the most prescribed IV fluid therapy
- As a consequence, administration of a large volume of normal saline has been linked to hyperchloremic metabolic acidosis, renal vasoconstriction and increased sensitivity to aldosterone [both of which lead to decline in estimated glomerular filtration rate (eGFR)], secretion of pro-inflammatory cytokines, and disruption of physiological coagulation pathways

Ringer's lactate

- Ringer's lactate has been linked to hyperglycemia through the conversion of lactate into glucose via gluconeogenesis,
- lactic acidosis in patients with chronic liver disease secondary to its inability to convert lactate into bicarbonate,
- cerebral edema for its hypotonic nature, and
- chelation of calcium with citrate when administered with blood products and certain antibiotics such as ceftriaxone

Ringer's lactate

- Lactated Ringer's is a sterile solution composed of water, sodium chloride (salt), sodium lactate, potassium chloride, and calcium chloride.1 It's often used in place of saline solution (water and 0.9% sodium chloride) because it is less likely to cause fluid to build up in the body.
- Lactated Ringer's is often recommended over saline solution for fluid resuscitation.
- Normal saline dilates blood vessels, raises blood potassium levels, and can increase the risk of metabolic acidosis.
- The lactate in lactated Ringer's solution reduces acidity as it is converted into bicarbonate, a base element that helps regulate the body's pH balance and avoid acidosis
- There is no outright contraindication for using lactated Ringer's solution, but it should not be given to someone with severe liver dysfunction. Careful consideration should also be made for people with heart or kidney disease
- Lactated Ringer's solution doesn't mix well with certain drugs intended for intravenous use

dextrose

- Glucose or dextrose (usually 5%–10%) solutions are sodium-free and do not contribute to hypervolemia.
- They contain a small number of calories (200 to 400 kcal/L for glucose and 170–340 kcal/L for dextrose) and in the presence of a conserved insulin response, will promote potassium entry into cells.
- they facilitate hyponatremia in patients with deficient water excretion mechanisms and may contain 3,4-dideoxyglucosone-3-ene (3,4-DGE), a glucose-degradation product which is cytotoxic to leukocytes and kidney cells.

Colloid fluids

- Colloid fluids have now been rarely utilized in clinical practice including the care for critically ill patients, while the most commonly utilized colloid solutions include **gelatin**, **dextrane**, **albumin** and **hydroxyethyl starch (HES).**
- The use of semisynthetic colloid solutions has been associated with higher risk for AKI, need for renal replacement therapy or mortality among critically ill patients, which may be attributable to endothelial damage induced by colloid solutions which also explains the need for equal or higher amount of required resuscitation fluids compared with crystalloid solutions.
- Another major adverse effect of colloid solutions is the high risk for anaphylaxis as shown by a large-scale multicenter study conducted over approximately 20 000 patients demonstrating anaphylaxis rates of 0.35% for gelatin, 0.10% for albumin and 0.06% for HES.

Approach to critically ill patient

Resuscitation phase	**1. Resuscitation with IV crystalloid solutions (i.e. 20 ml/kg in first hour followed by IV 500 ml fluid boluses as required)** • No difference among various crystalloid solutions **2. Monitor and adjust the rate of volume replacement in accordance with:** • Mean arterial blood pressure and heart rate • Urine output • Serum lactate level **3. Use of inotrope agents for target MAP value (65 mm Hg)**
Replacement phase	**1. Individualized choices in accordance with:** • Serum electrolytes and acid-base status • Volume status • Plasma glucose
Maintenance phase	**1. Calculate the need for daily fluid requirement in patient unable to tolerate oral intake** **2. Replace with IV crystalloid solutions** • Most common choice: 2 liters of (0.45% NaCl + 5% dextrose) with addition of 20 mEq/L KCl ampoules

Important considerations

Normal saline (0.9% NaCl) →
- Hyperchloremic metabolic acidosis
- Renal vasoconstriction and decline in eGFR
- Impaired coagulation
- Upregulation of pro-inflammatory pathways

Ringer's Lactate →
- Avoid in patients with chronic liver disease
- Hyperglycemia
- Intravascular crystallization when used along with blood products
- Allergic reactions

Hartmann's solution →
- Intravascular crystallization when used along with blood products
- Lactic acidosis

Plasmalyte →
- Metabolic alkalosis
- False positive galactomannan antigen test result

Special scenarios

5% dextrose → preferred in
- Hypoglycemia
- Hypernatremia
- Hyperkalemia

Hypertonic saline (3% NaCl) → preferred in
- Hyponatremia
- Cerebral edema

Contrast-induced nephropathy

- Contrast-induced nephropathy (CIN) may develop in patients with CKD that receive IV or intra-arterial iodinated contrast agents for imaging or therapeutic interventions.
- CIN may be prevented by administering 1 mL/kg/h hydration normal saline for 6–12 h both pre- and post-procedure unless the patient is already hypervolemic or receiving renal replacement therapy
- For urgent procedures, a higher dose (3 mL/kg/h) may be infused 1 h prior to and 6 h post-procedure.
- Additional preventive measures include the use of low or iso-osmolality contrast material at the lowest possible dose and withdrawal of certain medications including metformin that may trigger adverse events
- A large-scale meta-analysis study demonstrated no clinically significant beneficial effects of N-acetyl cysteine or sodium bicarbonate therapy compared with isotonic saline. However, in CKD patients, short (1 h), low volume (250 mL) 1.4% sodium bicarbonate hydration before contrast-enhanced computed tomography was non-inferior to peri-procedural saline hydration with respect to renal safety and may result in healthcare savings

CKD fluid monitoring

- Patients with CKD are prone to fluid and electrolyte imbalances and need to be monitored closely.
- CKD is associated with increased risk for AKI also during or following surgery.
- Intraoperatively, usage of diuretics is discouraged due to their association with AKI development unless fluid overload is severe.
- By contrast, fluid restriction during abdominal surgery was not associated with AKI development in a meta-analysis.
- Type of fluid is at least as important as the amount of fluid.
- Hyperchloremia is associated with worsened eGFR at baseline. A meta-analysis of 21 studies with 6253 patients confirmed that perioperative fluids containing high chloride concentrations increased the risk of hyperchloremia, metabolic acidosis and AKI.
- The colloid HES has been associated with AKI, and the Critical Care Nephrology Working Group of the European Society of Intensive Care Medicine recommended avoiding high-molecular-weight HES preparations and the US Food and Drug Administration added a **black box warning** to the prescribing information recommending avoiding HES solutions in critically ill adult patients, including those with sepsis and in patients with pre-existing renal dysfunction, and discontinuing HES at the first sign of renal injury.

Flouid balance in heart failure

HF fluid balance

- Patients who were treated with intravenous fluids had higher rates of subsequent critical care admission (5.7% vs. 3.8%; $p < 0.0001$), intubation (1.4% vs. 1.0%; $p = 0.0012$), renal replacement therapy (0.6% vs. 0.3%; $p < 0.0001$), and hospital death (3.3% vs. 1.8%; $p < 0.0001$) compared with those who received **only diuretics**.
- Many patients who are hospitalized with HF and receive diuretics also receive intravenous fluids during their early inpatient care, and the proportion varies among hospitals. Such practice is associated with worse outcomes and warrants further investigation

diuretics

- Different classes of diuretics are used in patients with chronic HF, although loop diuretics (**furosemide,** bumetanide and torasemide) are the most widely prescribed.
- those responding poorly to a loop diuretic alone, the combination with a **thiazide** (or thiazide-like) diuretic can be very potent
- Mineralocorticoid receptor antagonists (MRAs) are, of course, also diuretics. Two large trials, have shown that adding **spironolactone or eplerenone** to standard treatment in symptomatic patients with reduced LVEF (either chronically or after a recent myocardial infarction) produces morbidity and mortality **benefit**s.
- In the most severe cases of HF, renal dysfunction and diuretic resistance often occur, and limit the available therapeutic resources to decrease congestion. While **ultrafiltration** is an invasive solution usually reserved for patients with severe acute HF, **peritoneal dialysis** (PD) is a home-based, intermittent, therapeutic option in which the removal of the excess fluid takes place using the peritoneum as a filter.

Table. Length of stay by volume of IV fluid administration

Fluid Type and Volume	% (N= 195652)	Length of Stay (days) Median (IQR)	P-Value*
No fluid administered[¶]	75.2	4 (3, 6)	
Normal Saline Solution			
200-2000ml	17.5	5 (3, 7)	<0.0001
> 2000ml	1.9	5 (3, 8)	<0.0001
Half Normal Saline Solution			
200-2000ml	1.9	5 (3, 7)	<0.0001
> 2000ml	0.3	5 (3, 8)	<0.0001
Lactated Ringer's			
200-2000ml	0.2	5 (3, 8)	<0.0001
> 2000ml	0.0	6 (5, 8)	<0.0001
5% Dextrose			
200-2000ml	5.8	5 (3, 8)	<0.0001
> 2000ml	0.2	6 (3, 8)	<0.0001

[¶]Reference Group

*The Wilcoxon rank sum/Kruskal-Wallis test was used to compare the length of stay associated with each fluid therapy regimen versus the reference group not receiving any fluids

- Patients given both therapies, the study found, were more likely to suffer adverse consequences, such as higher rates of critical care admission, intubation, dialysis, and even death, compared to those given diuretics alone.

Electrolite and fluid balance

Sodium balance

- Sodium excretion by the kidneys is a function of GFR. Serum sodium levels in renal dysfunction is usually related to fluid shift, and those patients are at risk of both hypo- and hypernatremia.
- In ascertaining the cause and management of sodium disturbance an assessment of volaemic status is essential.
- Hypontraemia (<135 mmol/l) is a common electrolyte abnormality in hospital patients, ranging from 5–30%. **Osmotic demyelination syndrome (ODS)**
- Hypernatremia is defined as serum sodium (sNa) concentration exceeding 145 mmol/L and reflects serum hyperosmolality, which is an occasionally encountered electrolyte disorder in hospitalized patients, especially in elderly and critically ill patients
- prevalence of 0.2% to 1.0%, this prevalence is about 10 times higher at 2% to 6% in critically ill patients, whereas it is about 10% in intensive care units. mortality of hypernatremia varies from 10% to 75% **cerebral edema**

Potassium balance

- Potassium is the dominant intracellular cation with 98% of total potassium located intracellularly. Kidneys are responsible for potassium homeostasis in response to increased serum K^+, aldosterone, distal renal tubular sodium delivery and tubular fluid flow they excrete 98% of daily potassium intake.
- CKD and end-stage renal disease inevitably lead to potassium derangements and increased risk of adverse cardiovascular events and mortality.

Magnesium

- Magnesium is actively excreted by the kidney and renal dysfunction will lead to raised serum.
- Normal range is **1.3 to 2.1 mEq/L (0.65 to 1.05 mmol/L)**.
- Clinical manifestations of hypomagnesemia include anorexia, nausea, vomiting, lethargy, weakness, personality change, tetany (eg, positive Trousseau or Chvostek sign or spontaneous carpopedal spasm, hyperreflexia), tremor, and muscle fasciculations
- Symptoms of hypermagnesemia include nausea and flushing with serum levels above 2 mmol/l progressing to weakness and hypotension with rising serum levels.
- Serum magnesium levels above 8 mmol/l are associated with respiratory weakness, AV block and cardiac arrest.

Calcium and phosphate

- Calcium and phosphate homeostasis involves the kidneys, gut, bone and parathyroid glands. The electrolyte changes in kidney disease lead to secondary hyperparathyroidism and mineral and bone disease.
- An increase in phosphate, low calcium and vitamin D deficiency is thought to be the trigger for increase in parathyroid hormone.

Have a good day