Fluid and electrolyte homeostasis

Fluid and electrolyte homeostasis is maintained by feedback mechanisms, hormones, and many organ systems, and is necessary for the body's normal physiologic functions.

Normal ranges	
Laboratory test	*Reference range*
Sodium	*135–145 mmol/L*
Potassium	*3.4–5.0 mmol/L*
Calcium	*Total: 2.12–2.60 mmol/L*
	Ionized: 1.19–1.37 mmol/L
Phosphate	*0.80–1.44 mmol/L*
Magnesium	*0.7–1.00 mmol/L*

Sodium and water

Edema

Edema is defined as a clinically detectable increase in interstitial fluid volume.
It develops when excess sodium is retained either as a primary defect in renal sodium excretion or as a response to a decrease in the effective circulating volume despite an already expanded or normal ECF volume.
Edema occurs in patients with decreased myocardial contractility, nephrotic syndrome or cirrhosis.

Types of Edema

Peripheral edema	It usually affects the legs, feet, and ankles, but it can also happen in the arms. It could be a sign of cardiovascular or renal conditions. It is called as "pedal edema" when it is limited to feet and lower parts of legs.
Lymphedema	Lymphedema may be the result of cancer treatments like surgery and radiation.
Pulmonary edema	Pulmonary edema may be due to cardiogenic causes as in heart failure or cardiogenic causes such as lung infection, acute respiratory distress syndrome (ARDS), coagulation, or even toxicity resulted from contact with ammonia, chlorine, or other toxins and chemicals.
Cerebral edema	Cerebral edema may happen due to trauma, stroke or even cancer.
Others	Other types such as macular edema.

Treatment

- ***Sodium restriction*** and correction of underlying disease state are important.
- ***Diuretics*** are the primary therapy for edema. ***Loop diuretics*** are the most potent, followed by ***thiazide diuretics*** and then ***potassium-sparing diuretics***.

Hypernatremia

Hypernatremia results from either water loss (e.g., ***diabetes insipidus***) or hypotonic fluids, or less commonly from hypertonic fluid administration or sodium ingestion.

Hypernatremia:
Serum sodium >145 mEq/L [>145 mmol/L])

Symptoms are primarily caused by decreased neuronal cell volume and can include weakness, lethargy, restlessness, irritability and confusion. Symptoms of a more rapidly developing hypernatremia include twitching, seizures, coma and death.

Causes of Hypernatremia

Loss of hypotonic fluid	Loss of water in excess of electrolytes.
	Renal hypotonic fluid loss results from: • Diuretic drugs (loop and thiazide diuretics) • Osmotic diuresis (hyperglycemia, mannitol, urea due to high-protein enteral feeding) • Postobstructive diuresis • Diuretic phase of acute tubular necrosis
	Nonrenal hypotonic fluid loss can result from: • GI: Vomiting, diarrhea, lactulose, cathartics, nasogastric suction, gastrointestinal fluid drains, and fistulas • Cutaneous: Sweating (extreme sports), burn injuries
Pure-water deficits	Water intake less than insensible losses may result from any of the following: • Lack of access to water • Psychologic and neurologic diseases • Loss of water through the respiratory tract
	Vasopressin (AVP) deficiency (diabetes insipidus) ▪ Some medications may induce nephrogenic diabetes insipidus.
Hypertonic sodium gain	• Administration of hypertonic electrolyte solutions ○ sodium bicarbonate solutions, hypertonic alimentation solutions, normal saline with or without potassium supplements • Sodium ingestion as in NaCl tablets, seawater ingestion • Sodium modeling in hemodialysis
	Water shifts into muscle cells during extreme exercise or seizures because of increased intracellular osmoles).

Diagnosis

The first step is to estimate the volume status (intravascular volume) of the hypernatremic patient. The associated volume contraction may be mirrored in a low urine Na+ (usually < 10 mEq/L).

The following lab studies are used to determine the etiology of hypernatremia:

Serum electrolytes	Na^+, K^+, Ca^{2+}
Glucose level	
Kidney function	Urea, Creatinine
Urine electrolytes	Na^+, K^+
Urine and plasma osmolality	Measurement of the urine osmolality will allow differentiation of the following: • Nonrenal causes with appropriately high urine osmolality - Isolated hypodipsia, increased insensible losses • Renal water loss indicated by inappropriately low urine osmolality
Urine output	24-hour urine volume If the patient has polyuria without hypernatremia and will be evaluated for diabetes insipidus.
Plasma arginine vasopressin (AVP) level	if indicated. It is useful to distinguish between central and nephrogenic diabetes insipidus.

Treatment

The goals of management in hypernatremia are as follows:

1. Recognition of the symptoms, when present
2. Identification of the underlying causes
3. Correction of volume disturbances
4. Correction of hypertonicity

- Treatment of hypovolemic hypernatremia is started with ***0.9% saline***. After hemodynamic stability is restored and intravascular volume is replaced, replace free-water deficit with ***5% dextrose*** or ***0.45% saline*** solution.
- ***Central diabetes insipidus*** is treated with *intranasal* or buccal desmopressin.
- ***Nephrogenic diabetes insipidus*** is treated by decreasing ECF volume with a ***thiazide diuretic*** and dietary sodium restriction (2000 mg/day).
- Sodium overload is treated with ***loop diuretics*** and ***5% dextrose***.

Hyponatremia

Hyponatremia results from an excess of extracellular water relative to sodium because of impaired water excretion.

Hyponatremia
- Serum sodium: <135 mEq/L or <135 mmol/L
- Mild: 130-134 mmol/L
- Moderate: 125-129 mmol/L
- Profound: < 125 mmol/L

Hyponatremia can be as a result of non-osmotic release of ***arginine vasopressin*** (AVP), commonly known as ***antidiuretic hormone***, which is caused by:

- Hypovolemia
- Decreased effective circulating volume as seen in patients with congestive heart failure (CHF)
- Nephrosis
- Cirrhosis
- Syndrome of inappropriate antidiuretic hormone (SIADH)

Classification of hyponatremia:

Depending on serum osmolality, hyponatremia is classified as:

I. Isotonic hyponatremia
II. Hypertonic hyponatremia
III. Hypotonic hyponatremia: it is the most common form of hyponatremia. It can be further classified as:

- *Hypovolemic hypotonic hyponatremia* is associated with a loss of ECF volume and sodium, with the loss of more sodium than water. It is relatively common in patients taking ***thiazide diuretics***.
- *Euvolemic hypotonic hyponatremia* is associated with normal or slightly decreased ECF sodium content and increased total body water (TBW) and ECF volume. It is most commonly the result of ***SIADH release***.
- *Hypervolemic hypotonic hyponatremia* is associated with an increase in ECF volume in conditions with impaired renal sodium and water excretion, such as ***cirrhosis***, ***heart failure*** and ***nephrotic syndrome***.

Clinical Presentation

- Most patients with hyponatremia are ***asymptomatic***. Presence and severity of symptoms are related to the magnitude and rapidity of onset of hyponatremia.
- Symptoms progress from nausea and malaise to headache and lethargy and, eventually, to seizures, coma, and death if hyponatremia is severe or develops rapidly.
- Patients with hypovolemic hyponatremia present with decreased skin turgor, orthostatic hypotension, tachycardia, and dry mucous membranes.

Diagnosis

In addition to history and the physical examination, there are three essential laboratory tests in the evaluation of patients with hyponatremia that help to establish the primary underlying etiologic mechanism:

Urine osmolality	Urine osmolality helps differentiate between conditions associated with impaired free-water excretion and primary polydipsia. High urine osmolality indicates impaired ability of the kidneys to dilute the urine.
Serum osmolality	Serum osmolality readily differentiates between true hyponatremia and pseudohyponatremia. Pseudohyponatremia may be secondary to hyperlipidemia or hyperproteinemia, or may be hypertonic hyponatremia associated with elevated glucose, mannitol, glycine (posturologic or postgynecologic procedure), sucrose, or maltose (contained in IgG formulations).
Urinary sodium concentration	Urinary sodium concentration helps differentiate between hyponatremia secondary to hypovolemia and syndrome of inappropriate antidiuretic hormone secretion (SIADH).

Treatment

- Severe symptoms: 100 mL of 3% NaCl infused intravenously over 10 minutes × 3 as needed
- Mild to moderate symptoms, in patients at low risk for herniation: 3% NaCl infused at 0.5–2 mL/kg/h
- Treatment is associated with a risk of ***osmotic demyelination syndrome*** *(central pontine myelinolysis)*. The rate of administration of NaCl (isotonic or hypertonic) should be adjusted to avoid exceeding a rise in serum sodium greater than 12 mEq/L (12 mmol/L) per day.
- SIADH is treated with 3% saline plus a loop diuretic. In some cases, administration of either ***sodium chloride*** or ***urea*** tablets with a loop diuretic or of ***demeclocycline*** can be required.
- Vasopressin receptor antagonists such as ***vaptans*** (arginine vasopressin receptor or AVP antagonists) (e.g., *i.v.* conivaptan and *oral* tolvaptan) can be used to treat SIADH as well as other causes of euvolemic and hypervolemic hypotonic hyponatremia that has been nonresponsive to other therapeutic interventions in patients with heart failure, cirrhosis, and SIADH.
- Lithium, demeclocycline, and vaptans are not recommended for patients with moderate or profound hyponatremia
- Hypovolemic hypotonic hyponatremia is treated with ***0.9% saline***.

- Hypervolemic hypotonic hyponatremia is treated with ***3% saline*** and prompt initiation of fluid restriction. ***Loop diuretic*** therapy is required to facilitate urinary excretion of free water.
- Treatment of asymptomatic hypervolemic hypotonic hyponatremia involves correction of the underlying cause and ***restriction of water intake*** to less than 1000 to 1200 mL/day. Dietary intake of sodium chloride should be restricted to 1000 to 2000 mg/day.

Potassium homeostasis

Hypokalemia

- It results from a total body potassium deficit or shifting of serum potassium into the intracellular compartment.
- Many drugs can cause hypokalemia and it is most commonly seen with use of ***loop*** and ***thiazide diuretics***.
- Other causes of hypokalemia include ***diarrhea***, ***vomiting*** and ***hypomagnesemia***.

- Hypokalemia: serum potassium level of less than 3.5 mEq/L (3.5 mmol/L).
- Moderate hypokalemia: 2.5-3.0 mEq/L,
- Severe hypokalemia: less than 2.5 mEq/L.

Mechanism of Drug-Induced Hypokalemia

1. Transcellular Shift *(tocolytic agents, theophylline, caffeine, insulin overdose)*
2. Enhanced Renal Excretion *(diuretics, high-dose penicillins, mineralocorticoids, aminoglycosides, amphotericin B, cisplatin)*
3. Enhanced Fecal Elimination *(laxatives, sorbitol)*

Clinical Presentation

- Signs and symptoms are nonspecific and variable and depend on the degree of hypokalemia and rapidity of onset. ***Mild hypokalemia*** is often asymptomatic.
- ***Moderate hypokalemia*** is associated with muscle weakness, cramping, malaise and myalgias.
- ***Cardiovascular*** manifestations, cardiac arrhythmias and ECG changes may be noticed in ***severe hypokalemia***.

Treatment

- To correct mild deficits, patients receiving chronic loop or thiazide diuretics generally need 40 to 100 mEq (40–100 mmol) of potassium.
- Whenever possible, ***potassium supplementation*** (potassium chloride) should be administered ***by mouth***.
- ***Potassium IV*** administration is used in case of severe hypokalemia, signs and symptoms of hypokalemia, or inability to tolerate oral therapy. IV supplementation is more dangerous than oral therapy due to the potential for hyperkalemia, phlebitis and pain at the infusion site.
- IV Potassium should be administered in saline because dextrose can stimulate insulin secretion and worsen intracellular shifting of potassium.
- In patients with mild renal insufficiency, the combination of an ***ACE inhibitor***, a ***potassium-sparing diuretic***, and a ***potassium supplement*** can very easily result in life-threatening hyperkalemia.

Hyperkalemia

Hyperkalemia:
Serum potassium >5 mEq/L [>5 mmol/L]

Hyperkalemia develops when potassium intake exceeds excretion or when the transcellular distribution of potassium is disturbed.
Primary causes of true hyperkalemia include:

- Increased potassium intake
- Decreased potassium excretion
- Tubular unresponsiveness to aldosterone
- Redistribution of potassium to the extracellular space.

Clinical Presentation

Hyperkalemia is frequently asymptomatic; patients might complain of heart palpitations or skipped heartbeats. ECG changes may be recognized.

Treatment

- Treatment depends on the desired rapidity and degree of lowering. ***Dialysis*** is the most rapid way to lower serum potassium concentration.
- ***Calcium*** administration rapidly reverses ECG manifestations and arrhythmias, but it does not lower serum potassium concentrations. Calcium is short acting and therefore must be repeated if signs or symptoms recur.
- Rapid correction of hyperkalemia requires administration of drugs that ***shift potassium intracellularly*** (e.g., insulin and dextrose, sodium bicarbonate, or salbutamol).
- ***Potassium binders*** such as *sodium polystyrene sulfonate, patiromer sorbitex calcium* and *sodium zirconium cyclosilicate* are cation-exchange resins which as suitable for asymptomatic patients with mild to moderate hyperkalemia. The sorbitol component promotes excretion of exchanged potassium by ***inducing diarrhea***. The oral route is better tolerated and more effective than the rectal route.
- ***Salbutamol*** is an beta-adrenergic agonist that has an additive effect with insulin and glucose, which may in turn help shift potassium into the intracellular space.
- Loop diuretics markedly enhance renal potassium excretion and thus lower serum levels.
- The use of ***sodium bicarbonate*** can be considered in treatment of hyperkalemia even in the absence of metabolic acidosis.
- Magnesium sulfate is used for hyperkalemic patients with cardiac arrhythmias from digitalis toxicity.

Calcium homeostasis

ECF calcium is moderately bound to plasma proteins (40%), primarily albumin. Ionized or free calcium is the physiologically active form.

Hypocalcemia

- Hypocalcemia results from altered effects of parathyroid hormone and vitamin D on the bone, gut, and kidney. Primary causes are postoperative ***hypoparathyroidism*** and ***vitamin D deficiency***.

Hypocalcemia:
Total serum calcium <8.5 mg/dL [<2.13 mmol/L]

- ***Hypomagnesemia*** can be associated with severe symptomatic hypocalcemia that is unresponsive to calcium replacement therapy. Calcium normalization is dependent on magnesium replacement.

Clinical Presentation

- Clinical manifestations are variable and depend on the onset of hypocalcemia. ***Tetany*** is the hallmark sign of acute hypocalcemia, which manifests as paresthesias around the mouth and in the extremities; muscle spasms and cramps; carpopedal spasms; and, rarely, laryngospasm and bronchospasm.
- Cardiovascular manifestations result in ECG changes characterized by a prolonged QT interval and symptoms of decreased myocardial contractility often associated with CHF.

Treatment

- Acute symptomatic hypocalcemia requires ***IV*** administration of soluble ***calcium*** salts (e.g., calcium chloride, calcium gluconate). Calcium gluconate is preferred over calcium chloride for peripheral administration because it is less irritating to veins. After acute hypocalcemia is corrected, the underlying cause and other electrolyte problems should be corrected.
- Magnesium supplementation is indicated for hypomagnesemia.
- Oral calcium supplementation is indicated for chronic hypocalcemia due to hypoparathyroidism and vitamin D deficiency. If serum calcium does not normalize, a vitamin D preparation is added.
- Recombinant human parathyroid hormone may be required in addition to calcium and vitamin D supplementation for hypocalcemia.

Hypercalcemia

Cancer and hyperparathyroidism are the most common causes of hypercalcemia. Primary mechanisms include:

- Increased bone resorption
- Increased GI absorption
- Increased tubular reabsorption by the kidneys.

Hypercalcemia:
Total serum calcium >10.5 mg/dL [>2.62 mmol/L]

Clinical Presentation

- Clinical presentation depends on the degree of hypercalcemia and rate of onset. Mild to moderate hypercalcemia can be asymptomatic. Electrocardiographic (ECG) changes may be recognized in severe cases.
- ***Hypercalcemia of malignancy*** develops quickly and is associated with anorexia, nausea and vomiting, constipation, polyuria, polydipsia, and nocturia.
- ***Hypercalcemic crisis*** is characterized by acute elevation of serum calcium to greater than 15 mg/dL (>3.75 mmol/L), acute renal insufficiency, and obtundation. Untreated hypercalcemic crisis can progress to oliguric renal failure, coma, and life-threatening ventricular arrhythmias.
- ***Chronic hypercalcemia*** (i.e., hyperparathyroidism) is associated with metastatic calcification, nephrolithiasis and chronic renal insufficiency.

Treatment

- Management of asymptomatic, mild to moderate hypercalcemia begins with attention to the underlying condition and correction of fluid and electrolyte abnormalities.
- Hypercalcemic crisis and acute symptomatic hypercalcemia are medical emergencies requiring immediate treatment. Rehydration with ***normal saline*** followed by ***loop diuretics*** can be used in patients with normal to moderately impaired renal function. If saline hydration is contraindicated, treatment with ***calcitonin*** is to be initiated.
- ***Bisphosphonates*** (e.g. zoledronic acid and others) are indicated for hypercalcemia of malignancy.
- ***Denosumab*** is approved for treatment of osteoporosis. It is useful in hypercalcemia of malignancy in patients with a suboptimal response to bisphosphonates.
- ***Cinacalcet*** has been used for treatment of hypercalcemia secondary to parathyroid carcinoma.

- A ***corticosteroid*** (e.g. prednisolone or equivalent agent) is usually effective in reducing GI calcium absorption when hypercalcemia results from multiple myeloma, leukemia, lymphoma, sarcoidosis and hypervitaminoses A and D.
- **Phosphate** inhibits calcium absorption and promotes calcium deposition.

Magnesium homeostasis

Hypomagnesemia:
Serum magnesium <1.4 mEq/L
[<0.70 mmol/L]

Hypomagnesemia

Hypomagnesemia is caused by:

- Disorders of the intestinal tract or kidneys as these conditions may interfere with intestinal absorption or increase renal excretion of magnesium.
- Drugs (e.g., aminoglycosides, amphotericin B, cyclosporine, diuretics, digitalis, and cisplatin)
- It is commonly associated with alcoholism.

Clinical Presentation

- Although typically asymptomatic, the dominant organ systems involved are the neuromuscular and cardiovascular systems. Symptoms include heart palpitations, tetany, twitching, and generalized convulsions.
- ***Ventricular arrhythmias*** are the most important and potentially life-threatening cardiovascular effect.
- ***ECG changes***.
- Many ***electrolyte disturbances*** occur with hypomagnesemia, including hypokalemia and hypocalcemia.

Treatment

- The severity of magnesium depletion and the presence of symptoms dictate the route of magnesium supplementation. ***Intramuscular magnesium*** is painful and should be reserved for patients with severe hypomagnesemia and limited venous access.
- ***IV bolus*** injection is associated with flushing, sweating, and a sensation of warmth. IV magnesium is administered if serum concentrations are less than 1 mEq/L (<0.5 mmol/L) or if signs and symptoms are present regardless of serum concentration.
- ***Oral magnesium*** supplementation is appropriate when the serum magnesium concentration is greater than 1 mEq/L (0.5 mmol/L). Sustained release products are preferred due to improved patient compliance and less GI side effects (e.g., diarrhea).
- Magnesium ***dose*** should be ***reduced*** in case of *renal insufficiency*.
- ***Potassium-sparing diuretics*** (amiloride, spironolactone and triamterene) are used to avoid the loss of potassium in urine.

Hypermagnesemia

Hypermagnesemia:
Serum magnesium >2 mEq/L [>1 mmol/L]

Causes include:

- Magnesium concentrations steadily increase as the GFR decreases below 30 mL/min/1.73 m^2 (0.29 mL/s/m^2) and is generally associated with advanced CKD.
- Magnesium-containing antacids in patients with renal insufficiency
- Enteral or parenteral nutrition in patients with multiorgan system failure
- Magnesium for treatment of eclampsia
- Lithium therapy
- Hypothyroidism
- Addison disease

Clinical Presentation

- Symptoms are rare when the serum magnesium concentration is less than 4 mEq/L (<2 mmol/L).
- The sequence of neuromuscular signs as serum magnesium increases from 5 to 12 mEq/L (2.5–6 mmol/L) is sedation, hypotonia, hyporeflexia, somnolence, coma, muscular paralysis, and, ultimately, respiratory depression.
- The sequence of cardiovascular signs as serum magnesium increases from 3 to 15 mEq/L (1.5–7.5 mmol/L) is hypotension, cutaneous vasodilation, QT-interval prolongation, bradycardia, primary heart block, nodal rhythms, bundle branch block, QRS- and then PR-interval prolongation, complete heart block, and asystole.

Treatment

- ***IV calcium*** (e.g., calcium gluconate) is indicated to antagonize the neuromuscular and cardiovascular effects of magnesium. Doses should be repeated as often as hourly in life-threatening situations.
- Forced diuresis with ***0.45% NaCl*** and ***loop diuretics*** can promote magnesium elimination in patients with normal renal function or stage 1, 2, or 3 CKD. In dialysis patients, dialysate should be changed to a magnesium-free dialysate.
- ***Glucose*** and ***insulin*** may help promote magnesium entry into cells. Glucose should be administered with insulin to prevent hypoglycemia.

Phosphorus homeostasis

Hyperphosphatemia

Hyperphosphatemia:
Serum phosphorus >4.5 mg/dL [>1.45 mmol/L]

Most commonly caused by decreased phosphorus excretion, secondary to decreased glomerular filtration rate (GFR).

- Intracellular phosphate release can occur with rhabdomyolysis, hemolysis, and ***tumor lysis syndrome***, a complication of chemotherapy administered to treat acute leukemia and lymphoma.

Clinical Presentation

- Acute symptoms include gastrointestinal disturbances, lethargy, obstruction of the urinary tract, and, rarely, seizures.
- Calcium phosphate crystals are likely to form when the product of the serum calcium and phosphate concentrations exceeds 50 to 60 mg/dL (4–4.8 mmol/L).
- The major effect is related to the development of hypocalcemia and damage resulting from calcium phosphate precipitation into soft tissues, intrarenal calcification, nephrolithiasis or obstructive uropathy.

Treatment

- The most effective way to treat non-emergent hyperphosphatemia is to decrease phosphate absorption from the GI tract with phosphate binders such as ***aluminum salts*** and ***calcium carbonate***.
- Phosphate binders bind to dietary phosphate in the gastrointestinal tract. The phosphate is then eliminated in the feces, thus limiting intestinal absorption.
- Other non-calcium-non-aluminum phosphate binders
    - Sevelamer
    - Lanthanum carbonate
    - Sucroferric oxyhydroxide
    - Ferric citrate
- Severe symptomatic hyperphosphatemia manifesting as hypocalcemia and tetany is treated by the IV administration of ***calcium salts***.
- Diuretics lower phosphate serum levels by enhancing renal excretion.
    - Loop diuretics
    - Carbonic anhydrase inhibitors (i.e. acetazolamide)

Hypophosphatemia

Hypophosphatemia results from decreased GI absorption, reduced tubular reabsorption or extracellular to intracellular redistribution. It is associated with:

- Parenteral nutrition with inadequate phosphate supplementation
- chronic alcoholism
- Chronic ingestion of antacids
- Diabetic ketoacidosis
- Prolonged hyperventilation

Hypophosphatemia:
Serum phosphorus <2 mg/dL [<0.65 mmol/L]

Clinical Presentation

Severe hypophosphatemia (serum phosphorus <1 mg/dL [<0.32 mmol/L]) has diverse clinical manifestations that affect many organ systems, including the following:	
Neurologic manifestations	Progressive syndrome of irritability, apprehension, weakness, numbness, paresthesias, dysarthria, confusion, obtundation, seizures, and coma.
Skeletal muscle dysfunction	Myalgia, bone pain, weakness, and potentially fatal rhabdomyolysis.
Respiratory	Respiratory muscle weakness and diaphragmatic contractile dysfunction resulting in acute respiratory failure.
Others	Congestive cardiomyopathy, arrhythmias, hemolysis, and increased risk of infection can also occur.
Chronic hypophosphatemia can cause osteopenia and osteomalacia because of enhanced osteoclastic resorption of bone.	

Treatment

- Asymptomatic patients or those who exhibit mild to moderate hypophosphatemia can be treated with ***oral phosphorus*** supplementation (potassium phosphate in cap or liquid form).
- Severe or symptomatic hypophosphatemia should be treated with ***IV phosphorus*** replacement (potassium phosphates or sodium phosphates).
- Patients with renal dysfunction or receiving IV phosphorus should be **monitored** frequently for their serum phosphorus and calcium levels.
- ***Phosphorus*** is added routinely to hyperalimentation solutions to prevent hypophosphatemia.
- Vitamin D enhances intestinal and renal absorption of phosphate.
- Burosumab fully human monoclonal IgG1 antibody that binds fibroblast growth factor 23 (FGF23). It is indicated for X-linked hypophosphatemia (XLH) in adults and children aged 6 months or older.