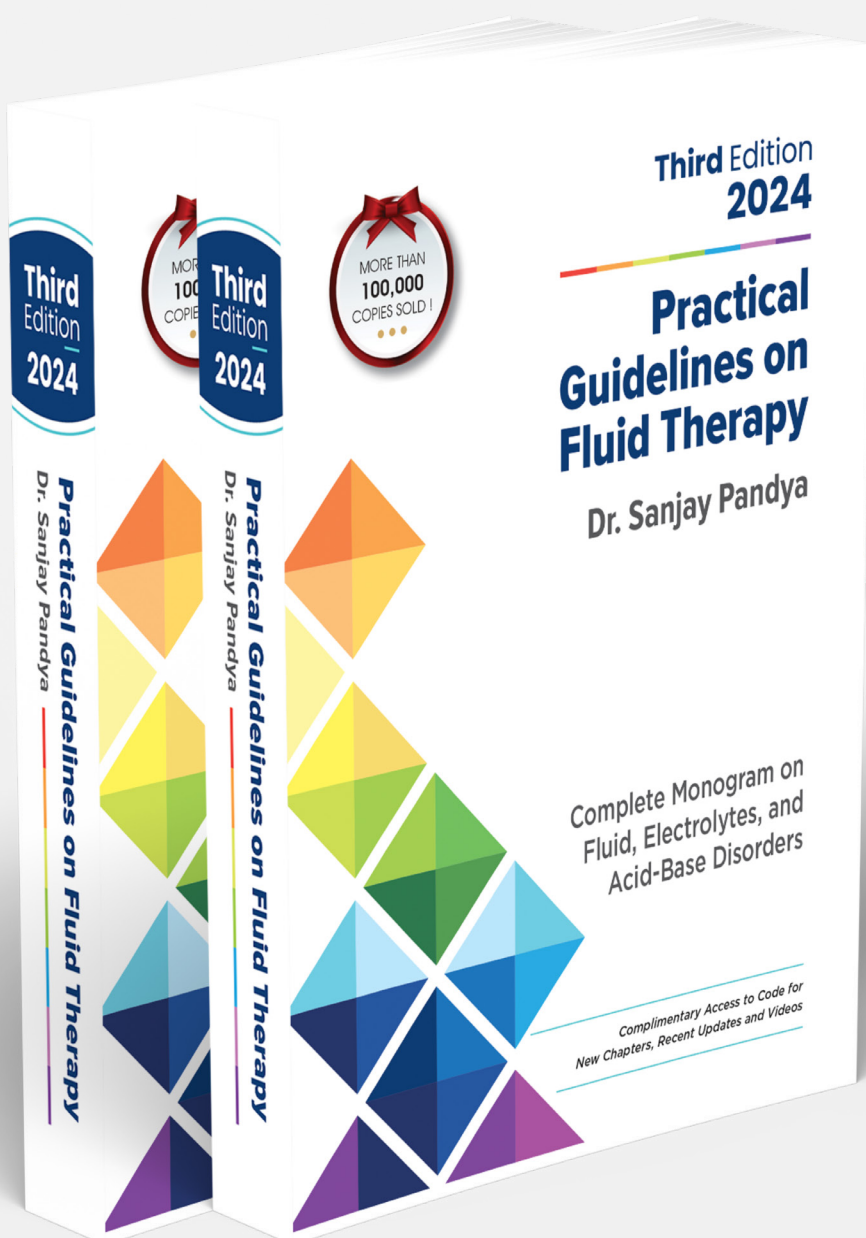


Chapter 27:

Hyperphosphatemia



To get a copy of the book, visit: www.fluidtherapy.org

Table of Contents

Part 1 Physiology

Overview of total body fluid distribution, water balance, and electrolyte compartments.

Chapter 1

Part 2 Basics of Intravenous Fluids and Solutions

Introduction to crystalloids and colloids, their composition, clinical use, precautions, and contraindications.

Chapter 2-5

Part 3 Fluid Replacement Strategies

Principles of fluid therapy, including maintenance, resuscitation, and special considerations for the elderly.

Chapter 6-9

Part 4 Parenteral Additives

Composition, clinical applications, and precautions for various parenteral additives.

Chapter 10-14

Part 5 Hemodynamic Monitoring

Principles and techniques for assessing fluid status and cardiac output, using basic and advanced methods.

Chapter 15-19

Part 6 Electrolyte Disorders

Causes, presentation, diagnosis, and management of various electrolyte imbalances.

Chapter 20-29

Part 7 Acid-Base Disorders

Concepts, interpretation, and management of metabolic and respiratory acid-base disorders.

Chapter 30-33

Part 8 Fluid Therapy in Medical Disorders

Guidelines for fluid management in conditions like GI diseases, liver disorders, respiratory issues, and diabetic emergencies.

Chapter 34-41

Part 9 Fluid Therapy in Surgical Disorders

Fluid management before, during, and after surgery, including TURP syndrome and burns.

Chapter 42-47

Part 10 Fluid Therapy in Pediatrics

Special considerations for fluid management in children and neonates, including resuscitation, maintenance, and oral rehydration.

Chapter 48-50

Part 11 Fluid Therapy in Obstetrics

Fluid management strategies for pregnancy, cesarean delivery, preeclampsia, and labor-related hyponatremia.

Chapter 51-54

Part 12 Parenteral Nutrition

Principles, indications, and administration of parenteral nutrition, with disease-specific guidelines and complication management.

Chapter 55-57

27 | Hyperphosphatemia

Etiology	321	Measure serum creatinine.....	322
Decreased renal phosphate excretion	321	Further investigations	322
Transcellular shift from ICF to ECF..	321	Management.....	323
Acute massive phosphate load	321	Treatment of underlying etiology ..	323
Spurious or pseudohypophosphatemia	322	Acute hyperphosphatemia.....	323
Clinical Features.....	322	Chronic hyperphosphatemia	323
Diagnosis	322	Dietary phosphate restriction...	324
History and physical examination..	322	Oral phosphate binders.....	324
		Renal replacement therapies ..	325

Hyperphosphatemia is defined as serum phosphate concentration greater than 4.5 mg/dL (1.45 mmol/L) in adults.

Renal excretion of phosphate is very effective, and therefore the person with normal renal function is unlikely to develop hyperphosphatemia because of increased phosphate intake. Therefore, Hyperphosphatemia is rare in the general population but commonly occurs in later stages of chronic kidney disease (CKD) with significantly impaired kidney function [1].

ETIOLOGY

Based on the mechanism of its development, the causes of hyperphosphatemia can be classified into four groups [1].

Decreased renal phosphate excretion

- Impaired renal phosphate excretion: Acute kidney injury (AKI) or chronic kidney disease with significant renal impairment are the most common cause of hyperphosphatemia [2].
- Increased renal tubular reabsorption: Hypoparathyroidism, vitamin D toxicity, acromegaly or thyrotoxicosis.

Transcellular shift

Transcellular shift from intracellular fluid (ICF) to extracellular fluid (ECF)

- Extensive cell destruction: Rhabdomyolysis, tumor lysis syndrome, or massive hemolysis.

- Severe acidosis: Severe lactic acidosis, diabetic acidosis and respiratory acidosis.
- Lack of insulin: Diabetic ketoacidosis (before treatment).

Acute massive phosphate load

Administration of phosphate-containing laxatives or enemas and IV or oral phosphate administration.

Want to read more?

Get Printed Version

Get Kindle Version

REFERENCES

1. Leaf DE, Wolf M. A physiologic-based approach to the evaluation of a patient with hyperphosphatemia. *Am J Kidney Dis.* 2013;61(2):330–6.
2. García Martín A, Varsavsky M, Cortés Berdonces M, et al. Phosphate disorders and clinical management of hypophosphatemia and hyperphosphatemia. *Endocrinol Diabetes Nutr (Engl Ed).* 2020;67(3):205–215.
3. Liamis G, Liberopoulos E, Barkas F, et al. Spurious electrolyte disorders: a diagnostic challenge for clinicians. *Am J Nephrol.* 2013;38(1):50–7.
4. Vallée M, Weinstein J, Battistella M, et al. Multidisciplinary Perspectives of Current Approaches and Clinical Gaps in the Management of Hyperphosphatemia. *Int J Nephrol Renovasc Dis.* 2021;14:301–311.
5. Ikizler TA, Burrowes JD, Byham-Gray LD, et al. KDOQI clinical practice guideline for nutrition in CKD: 2020 update. *Am J Kidney Dis.* 2020;76(3):S1–S107.
6. Ketteler M, Block GA, Evenepoel P, et al. Executive summary of the 2017 KDIGO chronic kidney disease–mineral and bone disorder (CKD-MBD) guideline update: what's changed and why it matters. *Kidney Int.* 2017;92(1):26–36.
7. Sekar A, Kaur T, Nally JV, et al. Phosphorus binders: The new and the old, and how to choose. *Cleve Clin J Med.* 2018;85(8):629–638.
8. Ketteler M, Block GA, Evenepoel P, et al. Diagnosis, evaluation, prevention, and treatment of chronic kidney disease–mineral and bone disorder: synopsis of the kidney disease: Improving Global Outcomes 2017 Clinical Practice Guideline Update. *Ann Intern Med.* 2018;168(6):422–30.
9. Evenepoel P, Selgas R, Caputo F, et al. Efficacy and safety of sevelamer hydrochloride and calcium acetate in patients on peritoneal dialysis. *Nephrol Dial Transplant.* 2009;24(1):278–285.
10. Patel L, Bernard LM, Elder GJ. Sevelamer Versus Calcium-Based Binders for Treatment of Hyperphosphatemia in CKD: A Meta-Analysis of Randomized Controlled Trials. *Clin J Am Soc Nephrol* 2016;11(2):232–44.
11. Manns B, Klarenbach S, Lee H, et al. Economic evaluation of sevelamer in patients with end-stage renal disease. *Nephrol Dial Transplant* 2007;22(10):2867–78.
12. Chan S, Au K, Francis RS, et al. Phosphate binders in patients with chronic kidney disease. *Aust Prescr.* 2017;40(1):10–14.
13. Spoendlin J, Paik JM, Tsacogianis T, et al. Cardiovascular Outcomes of Calcium-Free vs Calcium-Based Phosphate Binders in Patients 65 Years or Older With End-stage Renal Disease Requiring Hemodialysis. *JAMA Intern Med* 2019;179(6):741–749.
14. Lioufas NM, Pascoe EM, Hawley CM, et al. Systematic Review and Meta-Analyses of the Effects of Phosphate-Lowering Agents in Nondialysis CKD. *J Am Soc Nephrol.* 2022;33(1):59–76.
15. Finn WF, SPD 405–307 Lanthanum Study Group. Lanthanum carbonate versus standard therapy for the treatment of hyperphosphatemia: safety and efficacy in chronic maintenance hemodialysis patients. *Clin Nephrol* 2006;65(3):191–202.
16. Zhao L, Liu A, Xu G. Safety and effectiveness of lanthanum carbonate for hyperphosphatemia in

-
- chronic kidney disease (CKD) patients: a meta-analysis. *Ren Fail.* 2021;43(1):1378–1393.
17. Drüeke TB. Lanthanum carbonate as a first-line phosphate binder: the “cons”. *Semin Dial.* 2007;20(4):329–32.
18. Ogata H, Fukagawa M, Hirakata H, et al. Effect of Treating Hyperphosphatemia With Lanthanum Carbonate vs Calcium Carbonate on Cardiovascular Events in Patients With Chronic Kidney Disease Undergoing Hemodialysis: The LAND-MARK Randomized Clinical Trial. *JAMA.* 2021;325(19):1946–1954.



Join the Mission to Fight Kidney Diseases

Explore the world's largest multilingual website created by a global team of over 100 nephrologists.

www.KidneyEducation.com

- » Read online or download the 200-page book "Save Your Kidneys" in 40 languages—completely free.
- » This comprehensive resource offers valuable information on preventing and managing common kidney problems, tailored for kidney patients and their families.
- » It's an authentic guide, prepared by nephrologists and free from any external funding.