

Chapter 27:

Hyperphosphatemia







27 Hyperphosphatemia

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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.

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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.
- 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.
- Liamis G, Liberopoulos E, Barkas F, et al. Spurious electrolyte disorders: a diagnostic challenge for clinicians. Am J Nephrol. 2013;38(1):50–7.
- 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.
- 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.
- 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.
- 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.
- 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.

- 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.
- 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.
- Chan S, Au K, Francis RS, et al. Phosphate binders in patients with chronic kidney disease. Aust Prescr. 2017;40(1):10–14.
- 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.
- 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.
- 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 metaanalysis. Ren Fail. 2021;43(1):1378–1393.

- Drüeke TB. Lanthanum carbonate as a firstline phosphate binder: the "cons". Semin Dial. 2007;20(4):329–32.
- 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.

