National Kidney Foundation®

Facts About Secondary Hyperparathyroidism and Vitamin D Deficiency

- 37 million adults in the United States are estimated to have chronic kidney disease (CKD) and millions of others are at increased risk.[1]
- As CKD gets worse, the kidneys are not able to activate vitamin D. This is an important problem because the body needs the active form of vitamin D to absorb calcium.
- Vitamin D deficiency is more common in patients with CKD than in the general population.[2,3]
- As kidney disease gets worse and blood vitamin D levels decrease, the body makes more and more parathyroid hormone (PTH), causing secondary hyperparathyroidism (SHPT).
- Most patients with CKD have SHPT.[4]
- SHPT begins as early as CKD stage 3 when eGFR (a measure of kidney function) falls below 60.[3]
- When SHPT is present there are low blood levels of vitamin D and calcium and high blood levels of PTH and phosphate.
- Patients with SHPT have high bone turnover which means that the cells that remove bone *(osteoclasts)* work faster than the cells that build new bone *(osteoblasts)*. This leads to weak and brittle bones.
- Left untreated, SHPT can lead to bone disease, increased risk for fractures, calcium buildup in soft tissue, poor health, and even death.[5]
- By the time patients are ready for dialysis, most have enlarged parathyroid glands and high blood levels of PTH.[6]
- Parathyroid glands are found in the neck behind the thyroid glands.
- When the PTH level stays above 800 for more than 6 months, even with medical treatment, surgical removal of the parathyroid gland (parathyroidectomy) may be necessary.
- Vitamin D deficiency should be prevented or treated with vitamin D supplementation to lower the chances of having SHPT. [7,8]

---

[1]Centers for Disease Control and Prevention. Chronic Kidney Disease in the United States, 2019. https://www.cdc.gov/kidneydisease/publications-resources/2019-national-facts.html. Published March 11 2019. Accessed June 27 2019.

[2]Gonzalez EA, Sachdeva A, Oliver DA, Martin KJ. Vitamin D insufficiency and deficiency in chronic kidney disease. A single center observational study. *Am J Nephrol*. 2004;24:503–510.

[3]Levin A, Bakris GL, Molitch M, et al. Prevalence of abnormal serum vitamin D, PTH, calcium, and phosphorus in patients with chronic kidney disease: results of the study to evaluate early kidney disease. *Kidney Int.* 2007; 71:31–38.

[4]Cunningham J, Locatelli F, Rodriguez M. Secondary hyperparathyroidism: pathogenesis, disease progression, and therapeutic options. *Clin J Am Soc Nephrol*. 2011;6:913-921.

[5]Nigwekar SU, Tamez H, Thadhani RI. Vitamin D and chronic kidney disease-mineral bone disease (CKD-MBD). *Bonekey Rep.* 2014 Feb;3:498.

[6]Tentori F, Zepel L, Fuller DS, et al. The DOPPS Practice Monitor for US dialysis care: PTH levels and management of mineral and bone disorder in US hemodialysis patients. *Am J Kidney Dis.* 2015;66:53653-9.

[7]Kidney Disease: Improving Global Outcomes (KDIGO) CKD-MBD Update Work Group. KDIGO 2017 Clinical Practice Guideline Update for the Diagnosis, Evaluation, Prevention, and Treatment of Chronic Kidney Disease–Mineral and Bone Disorder (CKD-MBD). *Kidney Int Suppl.* 2017; 7: 1–59.

[8]Isakova T, Nickolas TL, Denburg M, et al. KDOQI US Commentary on the 2017 KDIGO Clinical Practice Guideline Update for the Diagnosis, Evaluation, Prevention, and Treatment of Chronic Kidney Disease-Mineral and Bone Disorder (CKD-MBD). *Am J Kidney Dis.* 2017;70:737-751.