

CHRONIC KIDNEY DISEASE

Fatma Mohamed Samir¹, Osama El-Minshawey², Sahar Hossam El-Hini³, Mahmoud Ragab Mohamed⁴

Article History: Received: 12.12.2022 Revised:	29.01.2023 Accepted: 15.03.2023
--	---

Abstract

Chronic kidney disease (CKD): Kidney damage for \geq 3 months, as defined by structural or functional abnormalities of the kidney, with or without decreased GFR *or* GFR <60 mL/min/1.73m2 for \geq 3 months, with or without kidney damage

^{1,2,3,4}Internal Medicine Department, Faculty of Medicine, Minia University

DOI: 10.31838/ecb/2023.12.s3.066

phosphorus, and vitamin D. (Levin A. et al;2007) rHPT is associated with increased cardiovascular morbidity and mortality (De Boer IH, et al;2002) and has a significant economic burden on the US health care system (Lee A.et al;2013)

1. Introduction

Renal hyperparathyroidism (rHPT) is a common complication of CKD characterized by derangements in the homeostasis of calcium,

Stage	Description	GFR (ml/min/1.73 m ²)
1	Kidney damage with normal or ↑ GFR	> 90
2	Kidney damage with mild ↓ in GFR	60 <mark>-</mark> 89
3	Moderate ↓ in GFR	30-59
4	Severe ↓ in GFR	15-29
5	Kidney failure	< 15 (or dialysis)

 Medical management of r HPT > 6 monthes with hypercalcaemia and hyperphosphatemia .
 PTH >800 pg/ml.

3) Progressive extra-skeletal calcifications or calciphylaxis with documented elevated PTH levels and refractory hyperphosphatemia.
4) Osteoporosis (T-score >2.5 SD below mean) and pathological bone fractures.

2. References

- Block GA, Klassen PS, Lazarus JM, Ofsthun N, Lowrie EG, Chertow GM. Mineral metabolism, mortality, and morbidity in maintenance hemodialysis. J Am Soc Nephrol. 2004 Aug;15(8):2208–18. [PubMed]
- De Boer IH, Gorodetskaya I, Young B, Hsu CY, Chertow GM. The severity of secondary hyperparathyroidism in chronic renal insufficiency is GFR-dependent, racedependent, and associated with cardiovascular disease. J Am Soc Nephrol. 2002;13(11):2762–9. [PubMed]
- Fellner SK, Lang RM, Neumann A, Bushinsky DA, Borow KM. Parathyroid hormone and myocardial performance in dialysis patients. Am J Kidney Dis. 1991 Sep;18(3):320–5. [PubMed]
- Ganesh SK, Stack AG, Levin NW, Hulbert-Shearon T, Port FK. Association of elevated serum PO(4), Ca x PO(4) product, and

Am J Kidney Dis 2002; 39:S1

rHPT is classically broken into 2 types on the basis of the patient's serum calcium level. Secondary hyperparathyroidism (2° HPT) is the elevation of parathyroid hormone (PTH) in response to hypocalcaemia induced by phosphate retention and reduced calcitriol synthesis as a consequence of reduced renal function.(Martin KJ.et al:2007) In 2° HPT, all the parathyroid glands become enlarged owing to parathyroid hyperplasia. Because 2° HPT is a compensatory mechanism of the parathyroid glands, it commonly resolves with normalization of calcium and phosphorus homeostasis (eg, renal transplantation). Tertiary hyperparathyroidism (3° HPT) is seen when a patient with longstanding 2° HPT develops autonomous PTH secretion, often associated with hypercalcaemia. This is observed in up to 30% of patients with ESRD, who then undergo renal transplant. (Kerby J.et al;1998) 3° HPT is classically thought to have come from parathyroid hyperplasia, but some studies have suggested that up to 20% of patients may have single or double adenomas(Martin KJ.et al;2007).

Improving medical management with vitamin D analogs, phosphate binders, and calcimimetic drugs has expanded the treatment options for patients with rHPT, but parathyroidectomy remains necessary for many patients(Tominaga Y 2012).

Indications for parathyroidectomy in renal hyperparathyroidism(K/DOQI guidelines 2009):

parathyroid hormone with cardiac mortality risk in chronic hemodialysis patients. J Am Soc Nephrol. 2001 Oct;12(10):2131–8. [PubMed]

- Kalantar-Zadeh K, Kuwae N, Regidor DL, et al. Survival predictability of time-varying indicators of bone disease in maintenance hemodialysis patients. Kidney Int. 2006 Aug;70(4):771–80. [PubMed]
- Kerby J, Rue LW, Blair H, Hudson S, Sellers MT, Diethelm AG. Operative treatment of tertiary hyperparathyroidism: a single-center experience. Ann Surg. 1998 Jun;227(6):878– 86. [PMC free article] [PubMed]
- Lee A, Belozeroff V, Song X, Diakun D, Goodman W. Costs of treatment and clinical events for secondary hyperparathyroidism. Am J Pharm Benefits. 2013;5(2):e24–e35.
- 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 Jan;71(1):31–8. [PubMed]
- Martin KJ, Gonzalez EA. Metabolic bone disease in chronic kidney disease. J Am Soc Nephrol. 2007 Mar;18(3):875–85. DOI: http://dx.doi.org/10.1681/ASN.2006070771. [PubMed]
- National Kidney Foundation. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Am J Kidney Dis. 2002;39(2 suppl 1):S1– S266. [PubMed