



PTH abnormalities in chronic kidney disease

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Uremic condition creates an oxidative stress environment which affects different organs of the body including skeletal system. Uremic toxins have inhibitory effects on osteoblasts and osteoclasts functions (1,2). A complex interaction among gut, kidney, bones and parathyroid glands involve in bone metabolism and calcium-phosphorous hemostasis. Parathyroid hormone (PTH), vitamin D and its receptors, fibroblast growth factor-23 (FGF23), and calcium-sensing receptors are different arms of this metabolic pathway (3). With considering the critical role of kidneys in excretion of calcium and phosphate, thus abnormalities in bone turnover are inevitable in chronic kidney diseases (CKD) (4).

Hyperphosphatemia, hypocalcemia, deficiency of 1, 25(OH) vitamin D, skeletal resistance to vitamin D, and reduced expression of calcium sensing receptors all lead to secondary hyperparathyroidism in CKD patients (5). Low serum 25(OH) D level, which is a common finding (80% of cases) in CKD (6) - is an important factor in stimulating PTH secretion by parathyroid glands.

Despite few data available about prevalence of low serum PTH levels in CKD, some new reports suggest occurrence of over suppression of PTH glands and hypoparathyroidism in CKD populations (7-9). A new study in a large population of adults with different stages of CKD reported low serum PTH in 18% of cases with CKD stage 5, with higher frequency in peritoneal dialysis patients (31.3%) (7). High doses of vitamin D analogs and calcium-based phosphate binders may induce over suppression of PTH release.

An extended study by Akizawa et al (8) involving 8188 hemodialysis and 1207 peritoneal dialysis patients treated in 65 hospitals or clinics in Japan reported absolute (intact PTH levels <60 pg/mL) and relative (intact PTH levels 60-160 pg/mL) hypoparathyroidism in 31% and 33.4% of hemodialysis and 31.3% and 31.4% of peritoneal dialysis patients respectively. Naseri recently (9) found hypoparathyroidism as a predominant PTH abnormality (58.7%) in children and young adults on dialysis. Thus, more attention to this aspect of parathyroid hormone abnormality is required.

■ Implication for health policy/practice/research/ medical education

Conservative treatments for improving altered calcium-phosphorous hemostasis in dialysis patients and also dialysis modalities may inverse PTH abnormalities from hyper to hypoparathyroidism, an abnormality which needs completely different management.

■ **Keywords:** Fibroblast growth factor-23, Chronic kidney disease, Parathyroid hormone

Author's contribution

MN is the single author of the paper.

Conflicts of interest

The author declares no competing interest.

Ethical considerations

The author of this manuscript declares that she has followed the ethical requirements for this communication. Also, ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the author.

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