Pathologic Change and Treatment of Renal Osteodystrophy

Pauling Chu

Abstract

Renal osteodystrophy is a common complication in patients with chronic renal failure. According to difference in bone turnover rates, it is classified into high turnover renal osteodystrophy (hyperparathyroid bone disease), and low turnover renal osteodystrophy (including mixed uremic osteodystrophy, aluminum-induced osteomalacia and adynamic bone disease). Prevention and treatment of hyperparathyroid bone disease includes dietary phosphate control, the administration of phosphate binder, supplementation with calcium, and administration of calcitriol. To prevent aluminum-related osteomalacia, dialysate aluminum content should be kept under a safe level and use of aluminum-containing phosphate binder should be avoided. To prevent adynamic bone disease, the administration of high concentration of dialysate calcium and the overzealous use of oral calcium and calcitriol supplement should be avoided. In patients with hypoparathyroidism, low calcium dialysate could be tried in order to stimulate the parathyroid gland.

Recently, some new drugs have been approved for use in these patients, such as aluminum-free and calcium-free phosphate binder sevelamer hydrochloride, and non-calcemic vitamin D derivative zemplar. Some new drugs are still being evaluated in clinical trials, such as non-calcemic vitamin D derivative 22-oxacalcitriol and calcemimetics R-568. These new drugs may allow clinicians more selections to therapies that have fewer side effects in clinical practice.

Key words: uremia, renal osteodystrophy, secondary hyperparathyroidism, aluminum-induced osteomalacia, adynamic bone disease.

Associate Professor, Director of the Hemodialysis Center, Division of Nephrology, Tri-service General Hospital

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Correspondence : Pauling Chu, MD, PhD, Division of Nephrology, Department of Internal Medicine Tri-Service General Hospital, National Defense Medical Center 325 Section 2, Cheng-Kung Road, Neihu 114 Taipei, Taiwan

Telphone : (02) 87927213 E-mail: pchu@seed.net.tw