Clinical Reviews in Bone and Mineral Metabolism, vol. 5, no. 1, 1, 2007

1534-8644/07/5:1/$30.00 ISSN 1559-0119 (Online)

Introduction

The kidneys play a pivotal role in the regulation of calcium and phosphorus metabolism, as well as vitamin D metabolism. Derangements in kidney function, therefore, lead to disturbances involving bone and mineral metabolism. This is the scope of renal osteodystrophy (ROD).

For the past several decades, our understanding of ROD has made significant progress. Many advances have been made as far as deciphering the various pathophysiologic mechanisms involved with the different classes of ROD. This was made possible by the molecular cloning and identification of various receptors and ion channels, e.g., calcium sensing resceptor (CaSR) and the epithelial calcium channel (ECaC).

The increasing incidence of extraskeletal calcifications, e.g., calciphylaxis, has also received much attention, as of late. The use of advanced imaging studies such as electron-beam computed tomography (EBCT) to identify evidence of vascular calcifications in the early stages has also increased our understanding of the intricacies of this disease process. Furthermore, it has highlighted the link between abnormal calcium and phosphorus metabolism and increasing cardiovascular morbidity and mortality in patients with renal failure.

With bone biopsy being the gold standard for the diagnosis of ROD, attempts have been made to identify other noninvasive methods, including biochemical markers of bone formation and bone resorption, e.g., osteocalcin, tartrate resistant acid phosphatase (TRAP), and collagen degradation products (CDP). Even the time-honored test for parathyroid hormone (PTH) has met some controversy.

The progression in the development of medical treatment options, from aluminum- and calcium-containing phosphate binders to noncalcium based phosphate binders and from vitamin D analogs to calcimimetic agents, is evidence of how much our understanding of the different mechanisms of ROD has evolved.

Despite all of these advances, however, much research and studies need to be done to further our knowledge regarding ROD.

The purpose of this issue is to provide a state of the art overview of the clinical aspects of ROD. It is our goal to inform internists and family practitioners, specialists such as nephrologists and endocrinologists as well as other health-care practitioners with particular interest in diseases involving bone and mineral metabolism about the new diagnostic and therapeutic options available, when dealing with patients with ROD.

I wish to express my sincerest gratitude and appreciation to all of those who generously shared their expertise in ROD. It is my fervent hope that this issue will somehow play a role in improving the quality of care rendered to patients affected by ROD.

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