Recent Developments in the Management of Renal Osteodystrophy

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Evidence has accumulated to indicate that certain abnormalities in mineral metabolism and several of the therapeutic interventions designed to manage them can lead to vascular and soft-tissue calcifications in patients with end-stage renal disease. These issues are particularly relevant to the management of secondary hyperparathyroidism and renal bone disease. Accordingly, current clinical management strategies are being carefully re-examined.

Phosphate retention and hyperphosphatemia are important contributors to the development of secondary hyperparathyroidism in chronic renal failure, and adequate control of these disturbances is essential for effective treatment and prevention. The widespread use of large oral doses of calcium as to manage phosphate retention may lead however, to soft-tissue and vascular calcifications, and the concurrent use of supra-physiological doses of calcitriol or other vitamin D sterols to treat secondary hyperparathyroidism can further aggravate these potentially serious complications. As such, the benefits of controlling phosphate retention and treating secondary hyperparathyroidism must be carefully weighed against the risk of worsening vascular calcification with its attendant adverse cardiovascular consequences.

New phosphate-binding agents, such as sevelamer, that do not contain calcium are now available for clinical use in the United States and Europe. This compound and others currently undergoing clinical development may make it possible to manage phosphate retention in patients with chronic renal failure without incurring the risks associated with the long-term use of large doses of calcium. New vitamin D analogs that purportedly have lesser effects than calcitriol to raise serum calcium and phosphorus levels are also available for treating secondary hyperparathyroidism due to chronic renal failure. These agents deserve consideration when treating secondary hyperparathyroidism in an effort to limit the risk of soft-tissue and vascular calcification. Calcimimetic compounds that directly activate the calcium-sensing receptor in the parathyroid glands and diminish parathyroid hormone secretion without raising serum calcium and phosphorus levels may in the future provide an additional therapeutic alternative to clinicians.