

Arterial Calcifications and Bone Histomorphometry in End-Stage Renal Disease

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Abstract:

Arterial calcification (AC) is a common complication of end-stage renal disease (ESRD). The mechanisms responsible are complex, including disturbances of mineral metabolism and active expression of various mineral-regulating proteins. An inverse relationship between AC and bone density has been documented in uremic patients. In the study presented here, which included 58 patients with ESRD on hemodialysis (HD), bone-histomorphometry characteristics were compared with the AC scores (0 to 4) determined according to the number of arterial sites with calcifications. Patients with AC scores of 0 (no calcifications), or 1 or 2 (mild calcifications) had similar serum parathyroid hormone levels and bone histomorphometry, with larger osteoclast resorption, higher osteoclast numbers, and larger osteoblastic and double tetracycline-labeled surfaces. In contrast, patients with high AC scores (3 and 4) were characterized by lower serum parathyroid hormone, low osteoclast numbers and osteoblastic surfaces, smaller or absent double tetracycline-labeled surfaces, and high percentages of aluminum-stained surfaces. According to multivariate analysis, AC score was positively associated with age ($P < 0.0001$), daily dose of calcium-containing phosphate binders ($P = 0.009$), and bone aluminum-stained surfaces ($P = 0.0037$), and an inverse correlation was observed with osteoblastic surfaces ($P = 0.001$). A high AC score is associated with bone histomorphometry suggestive of low bone activity and adynamic bone disease. These findings suggest that therapeutic interventions associated with excessive lowering of parathyroid activity (parathyroidectomy, excessive calcium or aluminum load) favor lower bone turnover and adynamic bone disease, which could influence the development and progression of AC.