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Pathogenesis of Bone Fragility in Chronic Kidney Disease

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Bone fracture is a major outcome of CKD-MBD. High fracture rate has been reported in CKD patients. One possible cause could be the decrease of bone volume caused by high concentration of parathyroid hormone (PTH), however, clear relationship between bone fragility and PTH concentration in CKD has not been fully elucidated.

We examined the changes in bone strength and quality in CKD model rats either with or without hyperparathyroidism. Despite comparable bone mineral density, value of femoral storage modulus in CKD rats assessed by dynamic mechanical analysis was lower than control rats. We further analyzed bone chemical composition by Raman spectroscopy and found that mineral matrix ratio, ratio of physical crosslinks, and apatite crystallinity were reduced, whereas pentosidine to matrix ratio increased in CKD rats. Oral adsorbent treatment ameliorated such abnormal changes. These results suggest that the accumulation of uremic toxins facilitate deteriorating mechanical properties through changes of material properties independent of changes in bone turnover and micro structural changes in CKD.