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## TOPIC: VASCULAR CALCIFICATION IN CKD

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In 1985 I co-authored 2 papers assessing metastatic calcification in Chronic Kidney Disease (CKD). In CAPD patients we showed that despite control of hyperparathyroidism (paired bone biopsy data) small vessel calcification developed in 20%, large vessel calcification in 24% and soft tissue calcification in 22% of patients (Cassidy et al. *QJM* 1985, **54**: 29). In a detailed analysis of 62 patients with chronic kidney disease who underwent parathyroidectomy; despite improvement in hyperparathyroid bone disease (biochemical and bone biopsy evidence) small peripheral arterial vessel calcification developed or progressed in 56 % of patients. Non-visceral soft tissue calcification however regressed in 60 % of patients (De Francisco et al. *QJM* 1985, **55**: 289). In 1999 our group assessed the usefulness of quantitative heel ultrasound (QUS) in determining bone mineral density in chronic haemodialysis patients, comparing this to conventional DXA scanning. Eighty-eight patients were studied and though measurements between the two methods correlated significantly sensitivities and specificities of QUS were not sufficiently good for QUS to be used as an alternative to DXA (Taal et al. *NDT*, 1999, **14**, 1917). We also reported on the risk factors associated with reduced bone mineral density in this group of patients (Taal et al. *NDT*, 1999, **14**, 1922). In 2002 our group compared intact and whole molecule PTH assays in patients with histologically confirmed post-renal transplant osteodystrophy. The whole molecule PTH assay was unable to discriminate between the two patient populations and provided very little additional clinical information to that obtained from the intact PTH assays (Godber et al. *Ann Clin Biochem* 2002, **39**,314). We continued to follow up the cohort of haemodialysis patients previously studied for risk factors related to reduced BMD for a mean period of 3.5 years. During this time 43% of the patients died. We were able to show for the first time that reduced total hip bone mass was an independent risk factor in all cause mortality in haemodialysis patients and studied (Taal et al. *Kidney International* 2003, 63, 1116). We have been following up all our transplant recipients with serial DXA scans over the past 5 years and have accumulated a large data base. In a recently published cross sectional analysis we showed that only 17% of male transplant recipients had normal BMD. We speculate that elevated rates of bone resorption driven by hyperparathyroidism is the most important contributing factor to this process (Roe et al. *Osteoporosis International*. 2004, **16**, 142). We are currently following 120 patients longitudinally (half diabetic) between the ages of 20 and 65 years with CKD stage 3 and 4 to determine progression of coronary artery calcification (CAC) using planar CT. We are correlating this with demographics, femoral neck bone mineral density and serological markers of bone metabolism. Preliminary results in 67 patients (39 males) show CAC in 18% (26% in males, 7% in females). The prevalence comparing patients with and without diabetes was 33% compared to 16%. This preliminary data set demonstrates that 1 in 5 patients with CKD 3 and 4 have CAC. Male sex, older age and obesity rather than degree of kidney disease correlated with coronary artery calcification. Patients with diabetes also had an increased prevalence. This study continues.