A 57-year-old woman with chronic kidney disease (CKD) on hemodialysis for 5 years underwent subtotal parathyroidectomy for secondary hyperparathyroidism. Three years later, she presented with gradually increasing serum parathormone (above 1000 pg/ml) and phosphate levels despite treatment with high doses of phosphate binders with active vitamin D metabolites. Simultaneously, bone pain developed together with painful right shoulder and left hip mobility restriction, causing difficulties in walking. The physical examination revealed palpable nodular masses overlying the area of both joints, which occurred to be extensive soft tissue calcifications on radiograms (FIGURE). A double-phase 99mTc-MIBI parathyroid scan showed an abnormal uptake in the area where a residual parathyroid tissue had been left during the surgery, suggesting nodular hyperplasia of the gland. Calcimimetic (cinacalcet) therapy was introduced, resulting in a significant decrease of serum parathormone and phosphate levels but without any improvement in soft tissue calcifications.

Mineral and bone disorder is a clinical syndrome inextricably linked with CKD. It embraces the entire spectrum of mineral and hormonal abnormalities (with secondary hyperparathyroidism among others), various forms of renal osteodystrophy together with vascular and soft tissue calcifications and is associated with significant morbidity and mortality. Treatment is aimed to maintain serum calcium and phosphate in the normal range and serum parathormone concentration between 2 and 9 times the upper normal limit for the assay. It involves adequate dialysis therapy, low-phosphate diet, the use of the combination of phosphate binders, active vitamin D analogs, calcimimetics and – in selected cases – surgical parathyroidectomy.2,4

FIGURE Nodular massive calcifications around the right shoulder (A) and left hip (B)
REFERENCES


