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Pathologic findings of acute kidney injury caused by primary hyperparathyroidism

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Case Study: Primary hyperparathyroidism (PHPT) is the most common cause of hypercalcemia and sometime result in acute renal injury. The manifestations of PHPT include hypercalciuria, nephrolithiasis, nephrocalcinosis, renal insufficiency, renal tubular dysfunction, and simple renal cysts. The pathophysiology of renal manifestations in PHPT is explained complexly by increased the filtered load of calcium in the glomerulus, a calcium renal leak, increased excretion of urinary oxalate, uric acid, sodium, and phosphate, et al. Notwithstanding these pathophysiologies, the pathologic findings and changes of kidney have not been well known because renal biopsy is not required for the diagnosis of renal manifestations in PHPT. We report renal pathologic findings leading to acute renal injury in PHPT.
A 50-year-old woman presented with numbness of fingers, toes, lip and bone pain and fatigue. She did not have any medical conditions including hypertension, diabetes and drug history. Laboratory findings showed to be normal except increased serum calcium 13.4 mg/dL (normal range; 7.8~10.2 mg/dL), serum creatinine 2.1 mg/dL (0.4~1.5 mg/dL) and PTH 2419.8 pg/mL (15~68.3 pg/mL). There was no abnormality in the abdominal ultrasound and bone scan. Bone densitometry showed osteoporosis (T-score L2-L3: -2.6, femur neck: -2.5). Thyroid ultrasound and Tc-99m thyroid scan revealed parathyroid adenoma with parathyroid hyperfunction. To exclude other causes for renal insufficiency, renal biopsy was performed. The pathology of kidney showed that calcium deposits in tubules in medulla and dilatation of tubules with flatting lining epithelium in deep cortical area. These changes seem to precede acute renal injury and renal stone formation.