



Diffuse Vascular Calcification in a Patient with End-Stage Renal Disease

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Vascular calcification, including arterial medial calcification and calcification of intimal plaque, is commonly found in patient with chronic kidney disease (CKD) and associated with cardiovascular mortality and morbidity. Vascular smooth muscle cell in arterial medial layer would transform into chondrocyte or osteoblast-like cell by multiple promoters such as dysregulated calcium and phosphate homeostasis, abnormal bone remodeling, hyperglycemia, dyslipidemia, oxidative stress and chronic inflammation in patients with CKD. Here, we presented a 73-year-old man diagnosed with end-stage renal disease under hemodialysis whose plain radiographs demonstrated diffuse vascular calcification.

Key words: vascular calcification, chronic kidney disease, hemodialysis, peripheral artery disease

Case Report

A 73-year-old man with history of diabetes, hypertension, and end-stage renal disease (ESRD) under hemodialysis for 12 years presented with left hip pain after a fall. He also had painful cramping, numbness, and weakness in both legs for over three months. Plain radiography of the pelvis revealed left femoral neck fracture (Fig. 1). Conspicuously, diffuse vascular calcification involving abdominal aorta as well as splenic, renal, common iliac, internal and external iliac, and femoral arteries was noted on pelvic and abdominal radiographs (Fig. 1 & 2). Serum concentrations of calcium (2.19 mmol/L) and phosphate (4.3 mg/dL) were normal, but the concentration of

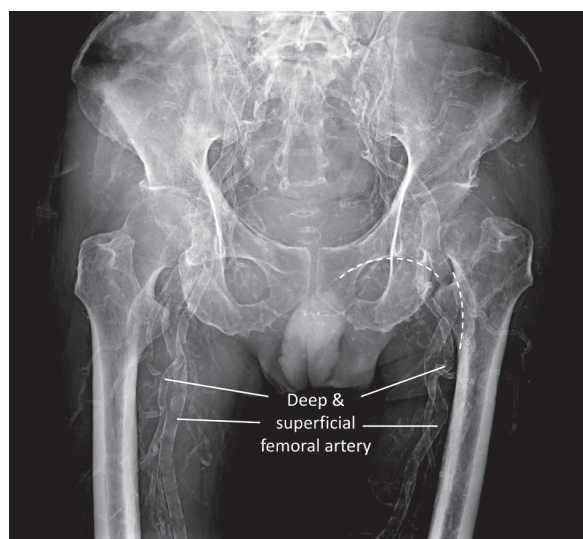


Fig. 1 Pelvic plain radiograph showing disruption of Shenton's line on the left hip (dotted line), indicating left femoral neck fracture. Note the diffuse noticeable vascular calcification involving internal and external iliac as well as superficial and deep femoral arteries.

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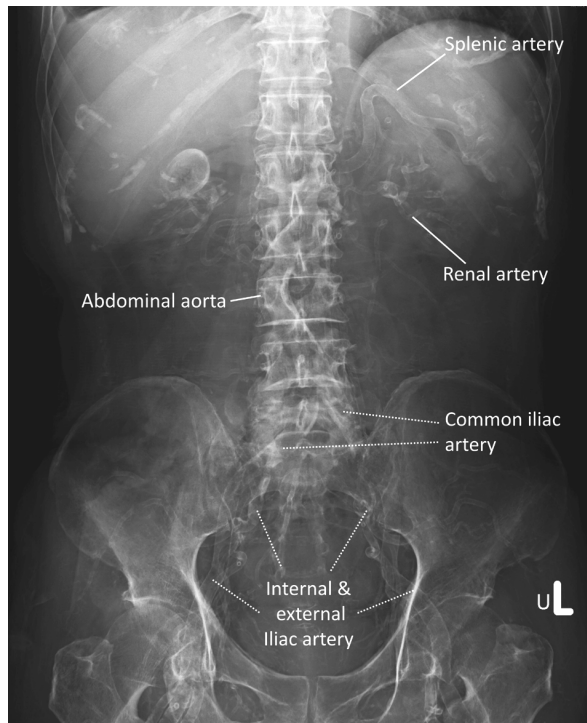


Fig. 2 Abdominal plain radiograph revealing diffuse noticeable vascular calcification involving the abdominal aorta, splenic, bilateral renal, mesenteric, common iliac as well as external and internal iliac arteries.

serum intact parathyroid hormone was marginally high (365.1 pg/mL).

Vascular calcification, a common phenomenon in patients diagnosed with chronic kidney disease (CKD), is associated with cardiovascular mortality and morbidity.¹ Two types of vascular calcification occurred in patient with CKD, including arterial medial calcification (Mönckeberg's sclerosis) as well as calcification of intimal plaque (atherosclerotic calcification) with the former being more common, especially for those with ESRD under hemodialysis.^{1,2} The mechanism of vascular calcification is an active, multifactorial and complicated process that involves vascular smooth muscle cells (VSMC), which are the main component of arterial medial layer. An imbalance between calcification promoter and inhibitor induces the transformation of VSMC into chondrocytes or osteoblast-like cells and their deposition in vascular wall.^{2,3} Multiple calcification promoters, including dysregulated

calcium and phosphate homeostasis, abnormal bone remodeling, hyperglycemia, dyslipidemia, oxidative stress, and chronic inflammation can be found in patients with CKD.² Once occurred, vascular calcification may persist even after restoration of renal function.⁴ In our patient, advanced age, diabetes, ESRD under long-term hemodialysis, and calcium-based treatment as well as possible hyperparathyroidism, all contributed to vascular calcification. For patients with advanced kidney disease, lateral abdominal plain radiography for vascular calcification and echocardiography for valvular calcification were recommended by Kidney Disease Improving Global Outcomes 2017 Guideline owing to its association with an elevated cardiovascular risk.⁵ Not surprisingly, peripheral arterial disease of the lower limbs was later diagnosed in this patient and may contribute to his long-standing lower extremity discomfort. Due to multiple comorbidities, he received conservative treatment for femoral neck fracture. Unfortunately, he succumbed to acute myocardial infarction one year later.

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