

Severe vascular calcification and gangrene in a haemodialysis patient

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Accepted 27 January 2014

DESCRIPTION

A 34-year-old man presented with type 1 diabetes mellitus (DM), who was on insulin treatment for the past 30 years, and had poor glycaemic control due to non-compliance with glycated haemoglobin averaging 13%. DM was complicated with peripheral vascular disease (PVD), peripheral sensory neuropathy (PSN), diabetic retinopathy and nephropathy leading to end-stage renal disease with haemodialysis vintage of 2 years. He presented with excruciating pain and progressive discolouration involving several digits over 3 months (figure 1). Apart from a history of multiple fractures, there was no fever, arthralgia or rash. Physical examination revealed dry gangrene of multiple digits while peripheral pulses remained intact.

Workup was negative for vasculitis. CT angiogram was negative for occlusive disease. Intact parathyroid hormone was 290 (15–65 ng/L) with normal serum calcium and phosphate. X-rays of the extremities demonstrate dense calcification of the vessels (black arrows) figure 2. These findings are secondary to vascular calcification (VC) of the media and internal elastic lamina, also known as Monckeberg sclerosis, common in chronic kidney disease and DM.^{1 2} The mechanism of VC is not known but the process of calcium accumulation predates dialysis initiation. Apoptosis of vascular smooth muscle cells (VSMC) is triggered in dialysis patients which results in loss of intrinsic defence mechanisms of VSMC's that result in VC.³ VC is similar to osteogenesis in bone: an active cell-



Figure 1 Dry gangrene of multiple digits.



Figure 2 Severe vascular calcification with distal cut-off.



To cite: Althaf MM,
Abdelsalam MS, Nadri Q.
BMJ Case Rep Published
online: [please include Day
Month Year] doi:10.1136/
bcr-2013-203258

mediated process rather than passive precipitation of calcium and phosphorus.⁴ Bone-mineral disorder is not associated with VC.⁵ Use of calcium-based phosphate binders and vitamin D supplementation are not associated with VC. DM and ageing are strong predictors of VC. Our patient was not exposed to vitamin K antagonists; its long-term use is associated with increased VC and vertebral fractures in haemodialysis patients.^{6,7}

Learning points

- ▶ Vascular calcification (VC) is common in dialysis patients. This process is not secondary to passive calcium-phosphate precipitation but more active processes where vascular smooth muscle cells undergo apoptosis, losing intrinsic defense mechanisms as well as osteoblast-like cell transformation.^{3,4}
- ▶ Levels of serum calcium, phosphate, calcium-phosphate product, alkaline phosphatase as well as intact parathyroid hormone are not associated with the presence of VC.⁵
- ▶ Use of calcium-based phosphate binders and vitamin D supplementation are not associated with VC. However, diabetes and ageing are strong predictors of VC in dialysis patients.⁵

While on pain management, he is worked up for possible amputation. PVD, PSN and insulin use are independent risk factors for limb amputation.⁸

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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