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Case Report

Radial Artery Mediacalcosis in Patient with End-Stage Kidney Disease: An Incidental Intraoperative Discovered During a Radiocephalic Native Arteriovenous Fistula (AVF) Creation

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Abstract

Medial arterial calcification (MAC) is a systemic vascular disorder due to disseminated of calcium phosphate within the medial layer, responsible for an increase in arterial stiffness and frequently associated with diabetes mellitus (DM), chronic kidney disease (CKD), and aging. We describe the case of a 70-year-old, man, diabetic, hypertensive patient with chronic kidney disease in which a Medial arterial calcification (MAC) was incidental discovered during a radio cephalic native arteriovenous fistula (AVF) creation for hemodialysis (HD) access during. The AVF was successfully performed and is used for hemodialysis sessions after a 60-day delay. The AVF remains currently permeable with good maturation parameters.

Introduction

Medial artery calcification (MAC), previously known as Monckeberg's sclerosis [1], is a systemic vascular disorder distinct from atherosclerosis due to disseminated and progressive precipitation of calcium phosphate within the medial layer [2,3], similar to that of developing bone [4-6], responsible for an increase in arterial stiffness [7] and frequently [8], associated with diabetes mellitus (DM) [9,10], chronic kidney disease (CKD) [11,12], and aging [13]. MAC is frequently an incidental finding and it's true prevalence is unknown. Some studies report a prevalence of 17% to 42% in type 2 DM (T2DM) patients [9-10] and 27% to 40% in patients with advanced CKD [11,12].

Case Presentation

We describe the case of a 70-year-old man patient, with a 30-year history of diabetes under insulin therapy, a 8-year history of hypertension under antihypertensive triple therapy, a 6-month history of End Stage Renal Disease (ESRD), which started hemodialysis sessions. He came to our department for the creation of a arteriovenous fistula. He's current preoperative biological parameters including lipidic assessment, phosphocalcic assessment, hematologic assessment, blood ionogram, hepatic assessment were normal. Uremia (18.5 mmol/l), creatinine (530.97 µmol/l), uric acid (613.09 mmol/l), blood sugar (11.44 mmol/l), were high. Glycated hemoglobin (5.2%) was normal. Elsewhere, eccentric hypertrophy and abnormal relaxation of

the left ventricle with ejection fraction at 71%, benign prostatic hypertrophy followed in urology were noted. Parathyroid hormone (PTH) and vitamin D levels, Klotho and fibroblast growth factor 23 (FGF23) were not measured. Standard radiograph of the homolateral upper limb, was not performed. After setting up the patient, preparing the surgical field using antiseptic and draping, a Radio cephalic Fistula according to Brescia-Cimino procedure [14] was performed. In fact, a vertical incision is made in the wrist midway between the radial artery and the cephalic vein. The cephalic vein was dissected, mobilized, and secured using vessel loops. Any small tributaries are being ligated and divided. The radial artery was dissected, mobilized, and secured using vessel loops. The radial artery presented an appearance of a yellowish, it was hard, rigid, incompressible, not very smooth appearance with a decrease in the intensity of the arterial pulse (Figure 1). An anterolateral arteriotomy was made. Fine calcareous granulations were noted escaping from the radial arterial wall which was indurated with a diffuse dissemination of fine granulations without focal presence of calcareous plaque of atherosclerosis. The artery was flushed with heparinized saline. An end-to-side anastomosis was made by ligating and dividing the distal cephalic vein and spatulating the end to match the size of the arteriotomy. The arteriovenous anastomosis was then performed using a running nonabsorbable monofilament suture (Figure 2). No samples were taken for histopathological study. Digital palpation was used to confirm the thrill through the fistula, as well as the distal radial artery signal. Hemostasis was achieved, subcutaneous tissues was approximated, and skin was closed using nonabsorbable sutures. Hemodialysis sessions began on the fistula, two months after its creation. The AVF remains currently permeable with good maturation parameters.

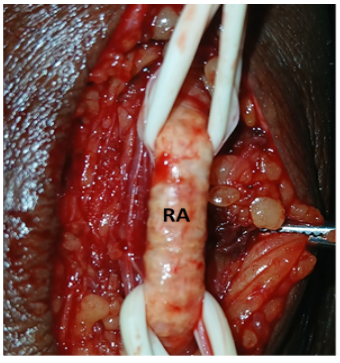

Figure 1: Intraoperative macroscopic appearance of the Radial Artery (RA) related to the Medial Artery Calcification (MAC) objectified by a yellowish smooth-less appearance.

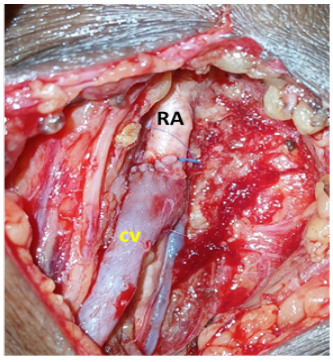

Figure 2: Intraoperative view of a radiocephalic native arteriovenous fistula creation by an end-to-side anastomosis of Cephalic Vein (CV) to Radial Artery (RA)

Discussion

MAC, long considered as an innocent bystander, is currently recognized as the silent killer of the cardiovascular system especially in diabetic patients [10], in chronic kidney disease (CKD) patients with long history of hemodialysis [11] and older patient [13]. The pathogenesis of MAC was remains long-time enigmatic [10]. It was long presumed to be occured by a passive and degenerative process of calcium/phosphate precipitation. However, currently, it is clearly considered as an active, cell-mediated, driven primarily by vascular smooth muscle cell (VSMCs) [15,16]. Damages and overburdens of VSMCs due to hostile conditions in its microenvironment cause them the lost of them essential defensive mechanisms and undergo a cell death and trans differentiation with creation of an osteo/chondrogenic to phenotype that can actively drive mineralization [15]. In patients with chronic kidney disease, medial calcification is thought to due in part to hyperphosphatemia leads to a variety of changes that affect VSMCs including calcium-phosphate deposits, promotion of an osteogenic phenotype, and apoptosis [17-20]. Vascular calcification is due to crystallization of calcium/phosphate in the form of hydroxyapatite in the extracellular matrix in both the intima (atherosclerosis) and the media (MAC) of the arterial wall. MAC is an entity distinct from atherosclerosis. In fact, intimal calcification is associated with atherosclerosis and forms in association with vascular smooth muscle cells (VSMCs), macrophages, and the necrotic lipid core. In atherosclerosis Intimal, calcification is associated with vascular smooth muscle cells (VSMCs), macrophages, and the necrotic lipid core leading to the formation of atherosclerotic plaques can lead to obstruction in blood flow and ischemia. In MAC (independent of atherosclerosis), medial calcification is associated with diabetes mellitus, CKD and aging [16]. Deposition of hydroxyapatite crystals occurs directly abutting VSMCs without inflammatory cells. Medial calcification causes vascular stiffening and decreased compliance of the vessel [15]. The decrease of the arterial compliance is associated with increase peripheral vascular resistance and a decrease vasodilatory response hence, the decrease of peripheral organ perfusion [1, 21, 22]. However, it is likely that other factors also behind MAC-due decrease perfusion, warranting further investigation [1]. The diagnosis of mediacalcosis in this clinical case was based on epidemiological arguments (long standing diabetes and chronic renal failure in hemodialysis and advanced age of the patient) but also on the intraoperative macroscopic appearance of the artery (yellowish appearance, hard, rigid, incompressible, smooth-less character, fine and disseminated calcareous granulations in arterial wall without focal presence of calcareous plaque of atherosclerosis). The realization of histological study, a standard radiograph of the homolateral upper limb and the dosage of traditional markers such as calcium, phosphate, parathyroid hormone (PTH) and vitamin D levels and new biomarkers such as Klotho and fibroblast growth factor 23 (FGF23) could allow to refine this diagnosis.

Conclusion

Medial artery calcification remains a reality in older diabetes

mellitus and chronic kidney disease. Its diagnosis is frequently an incidental finding that one must know how to think about.

Declarations

Consent for publication: The authors affirm that human research participants provided informed consent for publication.

Data Availability: The datasets used and/or analysed during the current study available from the corresponding author on reasonable request.

Competing Interests: The author declare no conflict.

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Author's contributions: All authors have approved the submitted version (and any substantially modified version that involves their contribution to the study) and have agreed to be personally accountable for their own contributions. Furthermore, all authors commit to ensuring that questions related to the accuracy or integrity of any part of the work, even those in which they were not personally involved, are appropriately investigated, resolved, and that the resolution is documented in the literature.

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