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Severity of Atherosclerosic Calcification of Femoral Bifurcation According to the P.A.R.C Score (Pheripheral Arterial Research Consortium)

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Original Research Article

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Abstract: The evaluation of calcifications of the femoral bifurcation has never been studied. Therefore, the development of endovascular surgery of this bifurcation and the impact of these calcifications on the success of procedures at this level, it becomes essential to find a way to evaluate this lesion. A CT scan using the Endosize image processing software on 83 patients treated for atherosclerotic lesions of the femoral tripod evaluated the calcifications according to the PARC (peripheral arterial research consortium) score. We found that ¾ of femoral tripods have severe calcium scores. These results explain the early failures of endovascular procedures and encourage us to plan better this technique in this region.
Keywords: Calcification, Arterial calcification, PACS, endovasculaire, Femoral arterial bifurcation.

INTRODUCTION

Arterial calcification has been recognized and described for centuries. Previous historical descriptions spoke of arterial ossification [1]. With the advances of the research on the subject, we can distinguish the medial calcification which represents an etiological group whose common point is the medial sclerosis of Monckeberg. In addition, we have intimal calcification, which is an evolutionary stage of atherosclerosis [2]. The presence of the latter in infra-inguinal situation is a challenge for endovascular procedures with placement of implants. For example, the U.S. Investigational Devices Exemption (IDE) discourages the placement of vascular implants if arterial calcification is severe [1].

On the other hand, no study defines the severity of calcification levels of the femoral arterial bifurcation. In addition, several methods for evaluating arterial calcification have been proposed for other territories. Through this work, we want to graduate the severity of atherosclerotic calcification of the femoral bifurcation with the Peripheral Arterial Research Consortium (PARC) method.

MATERIALS AND METHODS

This is a retrospective study from January 2015 to July 2017 involving 83 patients who were hospitalized in the Department of Vascular Surgery of the North Hospital of Marseille for the surgical management of lower extremity arteriopathy related to lesions of the femoral tripod. Inclusion criteria are: patients with significant lesions of the femoral tripod isolated or associated with other lesions and having angio TDM of the abdominal aorta and the lowers limbs arteries. The exclusion criteria are the absence of angio TDM of the arteries of the lower limbs of good quality allowing the extraction of a central line. In addition to the clinical data, morphological data at the femoral tripod are collected from the angio TDM analysis using the Endo Size image processing software. Images are transferred in DICOM format to a console with this software. After determining a proximal point at the level of the birth of the common femoral artery (CFA) taking as a reference the birth of the epigastric artery, and a distal point located 5cm downstream of the birth of the superficial femoral artery (SFA) automatic segmentation is performed followed by the extraction of a primary 3D central line. A secondary central line is then created that begins at the femoral bifurcation and ends 5cm downstream of the birth of the deep femoral artery (DFA).

The evaluation of the degree of calcification of the femoral tripod is made according to the calcium score of the "PARC". It is based on the circumference of the calcifications on the artery and their lengths. By combining these two criteria: length, less than half the length of the lesion or more than half, and the circumference, <180 ° or> 180 °, the calcium lesions are classified in 5 grades: Absent, focal, mild, moderate and severe.

RESULTS

Demographic and clinical characteristics (Table 1)

The mean age was 69 years old. There was a male predominance. The sex-ratio was 4:1. The average body mass index was 25.

In terms of antecedents and cardiovascular risk factors, smoking was found in 76% of patients, diabetes in 27% of patients, hypertension in 61% of patients, dyslipidemia in 21% of patients and cannabis use in 3% of patients. In 9% of patients, no cardiovascular risk factors were found.

Two patients (3%) were renally impaired at the dialysis stage. Clinically, 27% of patients were at stage 3 of the Rutherford classification. The systolic pressure index averaged 0.41 (Table 1).

Table-1: Demographic and Clinical Characteristics

N=83	
Age (mean)	69 years (44-95)
sex	H: 82%
BMI (mean)	24,6
Diabetes	27 % (n = 19)
Systemic arterial hypertension	61% (n = 43)
Dyslypidemia	21% (n = 17)
Smoking	76% (n = 63)
Cannabis	3% (n = 2)
Dialysis	3 % (n = 2)
ABI (mean)	0,41
Rutherford	
0-2	35 % (n = 28)
3-4	43 % (n = 33)
5-6	22 % (n = 20)

Assessment severity of calcifications of the femoral bifurcation

Grade 1 (Figure 1), which represents a circumference of calcification less than 180 ° and a calcification length less than 50% of the total length of the artery, was 10% (n = 7). Grade 2 (Figure 2) which means a circumference less than 180 ° and a length greater than 50% was 14.5% (n = 10). Grade 3 (Figure 3) with circumference greater than 180 ° and length less than 50% was 18.5% (n = 13). Finally, the grade 4 (Figure 4) characterized by a circumference greater than 180 ° and a length greater than 50% was 57% (n = 40).

DISCUSSION

There are two categories of arterial calcification: intimal calcification associated with atherosclerosis and medial calcification [1].

Arterial calcification was considered as a passive biological activity by the accumulation of excess calcium and phosphorus ions in the vessels. Now, it is proven that this is a complex mechanism that associates differentiation of macrophages and smooth muscle cells of the arterial wall into osteoclastoid cells [1-3]. Histologically, calcified plaques are presented as an osteomorphic, chondromorphic or amorphous structure [4]. This characteristic suggests a bone or osteoid nature of arterial calcifications [1, 3, 4].

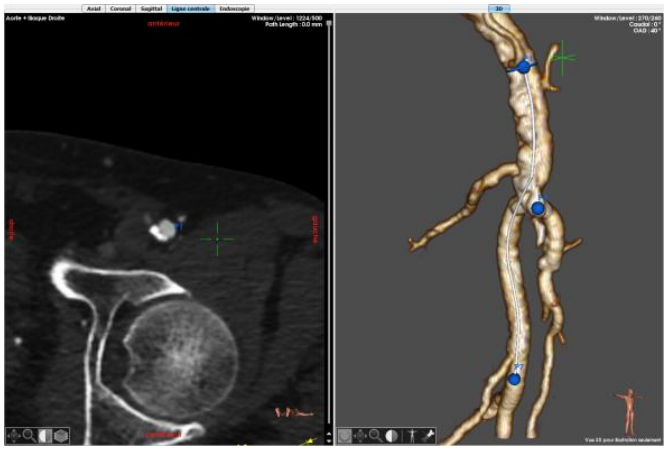

Fig-1: Grade 1 calcium score

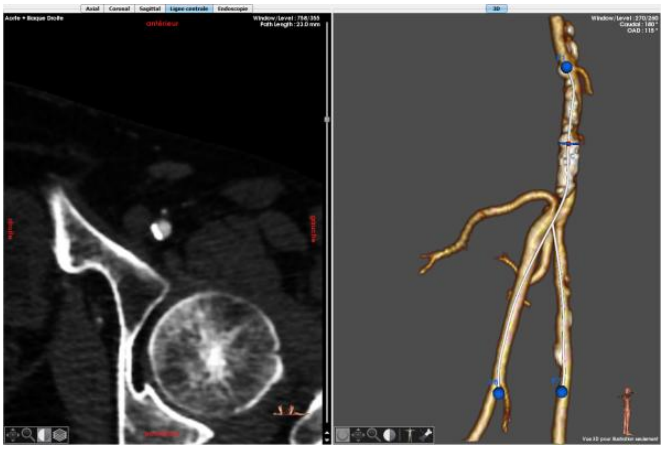

Fig-2: Grade 2 calcium score

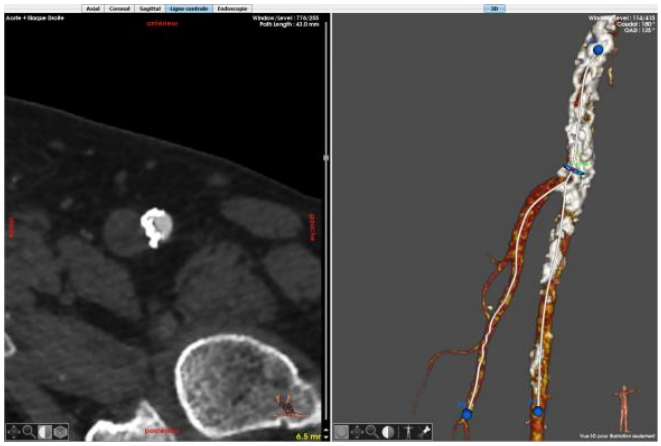

Fig-3: Grade 3 calcium score

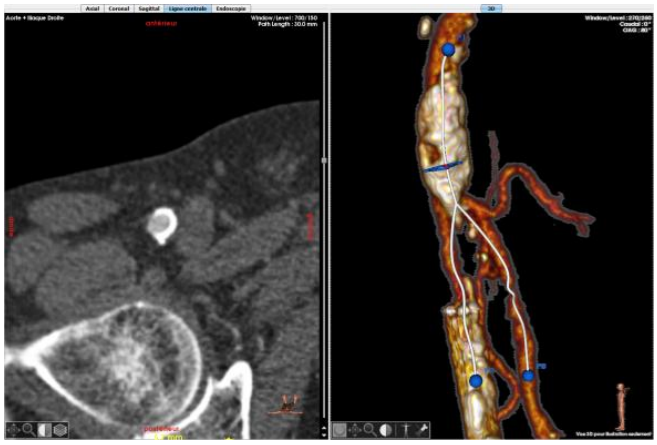

Fig-4: Grade 4 calcium score

Several theories have been suggested to explain intimal or atherosclerotic calcification. The latter is the type Vb of the Stary classification of atherosclerotic lesions [5].

At present, there are five theories: Lipid theory, hemodynamic theory, fibrin encrustation theory, non-specific mesenchymal theory, and wound response theory [5]. But all these theories are integrated and explained. Thus, histologically, arterial calcification results from the induction of osteogenic differentiation of some vascular cell populations under the influence of some inflammatory factors such as lipoproteins and cytokines [4]. Thus, the increase of concentrations of calcium and phosphorus ions in the serum, in concert with the oxidative stress caused locally by hydrogen peroxide, favors the differentiation of vascular smooth muscle cells in the arterial wall into an osteogenic phenotype [1]. These transformations are associated with a significant decrease in the endogenous inhibitors of calcification of vascular smooth muscle cells (matrix Gla protein, calcium binding protein and fetuin-A) and

the facilitation of a migration of these smooth muscle cells from the media to the intima [6]. A potential genetic role in medial calcification has been evoked. The latter is thought to be the result of a mutation of genes that regulate the production of phosphates in the extracellular matrix of vascular smooth muscle cells [7].

The prevalence of this intimal calcification increases with age and men are more affected than women [5]. Dyslipidemia is considered a very important risk factor as well as hypercholesterolemia [1, 5]. Arterial hypertension causes traumatic lesions on the arterial endothelium; starting point of calcified plaque formation [5]. In diabetics, the association dyslipidemia-HTA promotes the installation of atherosclerosis and its complications [5]. Other risk factors are associated with intimal calcification, namely: obesity, haemostatic factors, homocystinuria, progeria, Werner's syndrome and Chlamydia Pneumoniae [5].

Medial calcification represents a group of pathologies with different etiologies whose final consequence is the calcification of the arterial media [1]. The medial sclerosis of Monckeberg is the most common variant. This medial calcification is observed in the large elastic arteries (ascending aorta), the visceral middle (renal) arteries, and the small arteries with a diameter of at least 0.5mm (coronary, temporal, uterine, ovarian, parathyroid, and internal thoracic, glandular ...) [8, 9]. The formation of medial calcification is progressive and evolves in four stages ranging from the presence of calcium granules in the media to osteoform calcification [2]. This evolution is characterized by a deposit of calcium and phosphorus in the vascular smooth muscle cell matrix of the media, under the influence of some regulators such as circulating calcium inhibitors, matrix-Gla protein, inorganic pyrophosphate (PPi), fetuin A, and osteoprotegerin [2, 10-13]. Medial calcification is more common in men than in women (3: 2), in type 2 diabetics (17% and 41.5% in those taking oral antidiabetics) [1, 14], in chronic renal failure (27%) [1, 2].

The association between intimal and medial calcification is frequent in the same individual as well as the extension of one to the other [2]. In addition, intimal hyperplasia with intimal calcification may be seen, especially in stage 4 [2].

Arterial calcification is clearly related to mortality. The Kaplan-Meier 5-year survival curve is less than 50% in chronic renal failure patients with atherosclerotic calcification or medial calcification, whereas it is 90% in patients without arterial calcification [3]. After adjusting for age, dialysis, gender, ethnicity, diabetes, non-dialysis kidney failure, high blood pressure, smoking, parathyroid surgery, body mass index, medial calcification and intimal increase the relative risk of mortality by 5 and 12 times compared to patients without calcification [15]. Intimal calcification is localized mainly at the aortic, coronary, cardiac valve, peripheral arteries, cerebral arteries and visceral arteries [3]. On the other hand, medial calcification is not frequent in peripheral arteries; it is mostly found in the micro-circulation (arterioles from 10 to 500 micrometers in diameter) [2].

In addition, severe arterial calcification is another factor recognized by many authors as predicting poor endovascular outcomes [1,13]. Calcifications alter the morphology and compliance of the arterial wall and thus reduce the effectiveness of angioplasty and stenting [16]. Technical successes and permeabilities are altered in cases of severe arterial calcification [17, 18] by increasing the risk of arterial dissection and recoil [16] after balloon angioplasty and by promoting underdevelopment, malposition and stents fracture [19, 20]. The importance of an accurate assessment of the degree of calcification preoperatively is a necessity. None of the "endovascular" series of the femoral bifurcation objectively quantified the degree of calcification of their treated cohorts.

Different methods of assessing the degree of arterial calcification have been proposed in the literature. Computed tomography, MRI, ultrasonography, flow velocity measurement, echocardiography, standard radiography and indirectly the measurement of the systolic pressure index. their often make it possible to diagnose renal, peripheral arterial and carotid calcifications [1]. In contrast, computed tomography, MRI and intravascular ultrasonography are the imaging techniques used to evaluate the extent of arterial calcification. The most reliable is that using endovascular ultrasound. The latter has a high sensitivity and allows locating exactly the calcium lesion within the wall of the artery [21], however its relatively high cost limits its generalization.

Other, more accessible methods have emerged, including angiographic methods [21, 22]. These methods are based on the radiopaque property of calcifications and the evaluation is done on two orthogonal angles by referring to two criteria: their extent in length and in circumference. Thus the three main calcium scores have been developed (Table 2).

Our choice was focused on PARC calcium score, which seems more adapted and reproducible to us. For this score, grades 3 and 4 are considered severe. These represent 76% of femoral bifurcations studied. These results support the hypothesis of more severe calcifications at the femoral bifurcation compared to other peripheral arterial territories. As a result, they explain, in part, the poor outcomes of endovascular femoral bifurcation surgery compared to endarterectomy [23].

Table-2: The different angiographic calcium scores

PARC (pheripheal arterial research consortium)

Absence	Focal	Legere	Moderate	Severe
No calcification	<180 ° on one side of the vessel Less than half the length of the lesion	<180 ° on one side of the vessel More than half the length of the lesion	>180° on both side of the vessel Less than half the length of the lesion	>180° on both side of the vessel More than half the length of the lesion

Table-3: PACSS (Peripheral Artery Calcification Scoring System)

Grade 0	Grade 1	Grade 2	Grade 3	Grade 4
No calcification	Unilateral calcification <5cm	Unilateral calcification > or = 5 cm	Bilateral calcification <5 cm	Bilateral calcification > or = 5cm

Sub-grades: a = intimal, b = medial and c = mixed

Table-4: DEFINITIVE Ca++

Absence	Modérée	Sévère
No calcification	On one side of the arterial wall or <1 cm in length	On both sides of the arterial wall and greater than 1 cm in length

CONCLUSION

The severity of calcifications on the femoral bifurcation was known empirically. These statistical results allow us to measure their magnitude. Therefore, a change of attitude about endovascular techniques should take place. The latter should be based on physiopathological studies comparing the different hypotheses of atherogenesis in this region and univariate and multivariate analyzes of the different variables involved.

DECLARATION OF CONFLICTING INTERESTS

The Authors declare that there is no conflict of interest.

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