Case Report

Adaptive and Pathological Reshuffling in the Venous Wall in Special Hemodynamic Conditions-Acquired Arteriovenous Fistula

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ABSTRACT: Hemodialysis and peritoneal dialysis are the two possible choices in chronic renal disease in the uremic stage. Native arteriovenous fistula is susceptible to complications, some posing vital risk and requiring prompt treatment. We present the case of a patient undergoing hemodialysis on native AVF. An aneurism developed progressively on the arterialized cephalic vein. The AVF was abolished by closing the arteriotomy with a venous patch and excising the aneurismal venous segment. The pathology exam of the surgical specimen showed unequal vessel wall thickness due to hyperplasia of the media and subintimal space, with calcifications within the hyperplastic media.

KEYWORDS: *arteriovenous fistula, aneurysm, hemodialysis*

Introduction

Chronic renal disease in the uremic stage requires renal function support. The choice between the two alternative methods of renal function substitution, hemodialysis and peritoneal dialysis, is made based on etiopathogenic mechanism of the disease, comorbidities, age, sociocognitive status of the patient and his personal preference.

Hemodialysis is accomplished either through temporary angioaccess-central venous catheter (CVC), or permanently, through native or prosthetic arteriovenous fistula (AVF) or long-life type CVC. Native AVF is the optimal method for end stage renal disease patients in the uremic stage enrolled in a hemodialysis program. Although optimal, this method is still susceptible to complications, some being extreme emergencies, posing vital risk and requiring prompt treatment.

AVF failure can occur within three months from the procedure-early failure. Late failure is defined as AVF inefficiency after three months [1]. Access failure can be proved by physical exam or by imaging tests like duplex ultrasound or angiography [2,3].

Early AVF failure is frequently a consequence of a mistake in vessel selection for anastomosis or even technical fault. The late failures are mainly caused by thrombosis. This complication is due to venous stenosis, hypercoagulability, hypotension, arterial sclerosis or fistula compression.

The most severe complication is the rupture of the anastomosis or vessels with severe hemorrhage.

Case presentation

We present the case of a patient with a history of teen rheumatic fever, complicated with rheumatic endocarditis and acute glomerulonephritis. The patient later developed valvular lesions, both mitral disease, with predominant regurgitation, and aortic dysfunction. The acute glomerulonephritis became chronic later evolving into chronic renal failure in the uremic stage, complicated by grade III arterial hypertension with high cardiovascular risk, secondary anemia, systemic atheromatosis, and disruption of the calcium and phosphate metabolism, with bone hyperphosphatemia. In addition, the patient has a long history of tobacco use, with Gold stage 2 COPD resulting in a predominantly obstructive ventilator dysfunction, with 40% reduction of FEV1, and secondary tricuspid valve grade 2 regurgitation.

The patient has a BMI=25.9kg/m^2, is pale, and presents with heart murmurs. Ultrasound cardiac exam shows an ejection fraction of the left ventricle of 55%, thickened mitral valves with limited opening (maximal AV gradient of 46mmHg), mitral regurgitation grade 4, aortic regurgitation grade 2, and tricuspid regurgitation grade 2.

The preferred method of renal function substitution in this case was hemodialysis, so a radio-cephalic AVF was performed at level I on the left forearm. During the 9-year history of dialysis on native AVF an aneurism developed progressively on the arterialized cephalic vein, further complicated by thrombophlebitis, while the primary angioaccess was maintained. Tegument necrosis ensued, with denudation of the dilated vessel, and severe bleeding with arterial-type flow, requiring emergency surgery. The AVF was abolished by closing the arteriotomy with a venous patch and excising the aneurismal venous segment, completed by removal of the superjacent affected skin. Subsequently, the patient had a favorable outcome and continued hemodialysis initially on central venous catheter and later by AVF on the right forearm.

The pathology exam of the surgical specimen was performed on routinely formalin fixed paraffin embedded tissue, in the Pathology Department of The Emergency County Hospital of Craiova and showed unequal vessel wall thickness due to hyperplasia of the media and intimal space, with calcifications within the hyperplastic media. (Masson trichrome staining and HE coloration) (Fig.1 and Fig.2).

The patient gave a written informed consent for publication of these data.

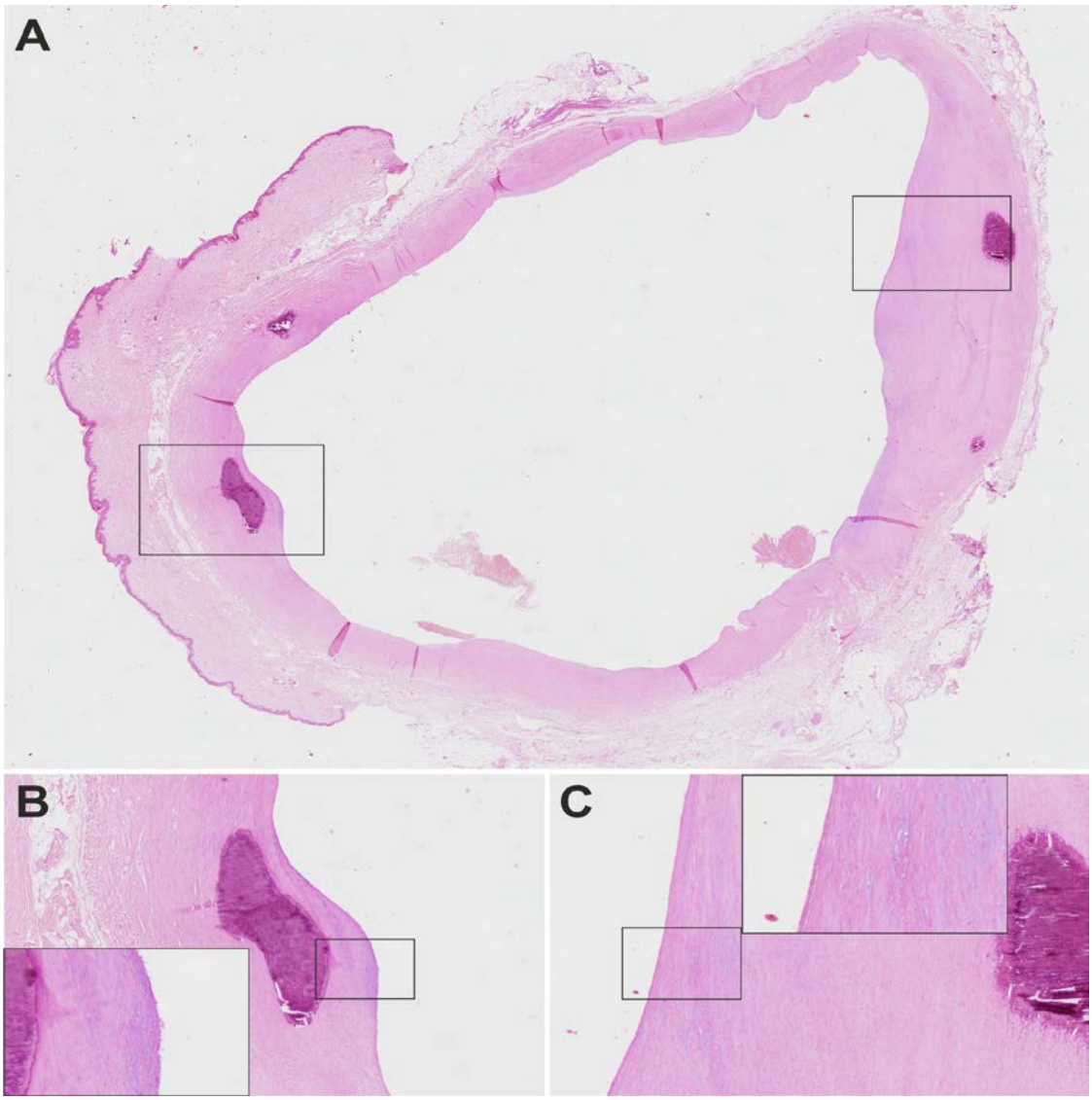

Fig.1. Arterialized venous wall with variable thickening of the media (collagen fibers, elastic fibers and muscular fibers), (A) whole slide scan, HE staining, 40x; (B-C) Mature atheroma plaques with lipid deposition in and beneath the intima, with foamy macrophages, and with calcifications in the upper and deeper media. Insets represent the enlarged respective images

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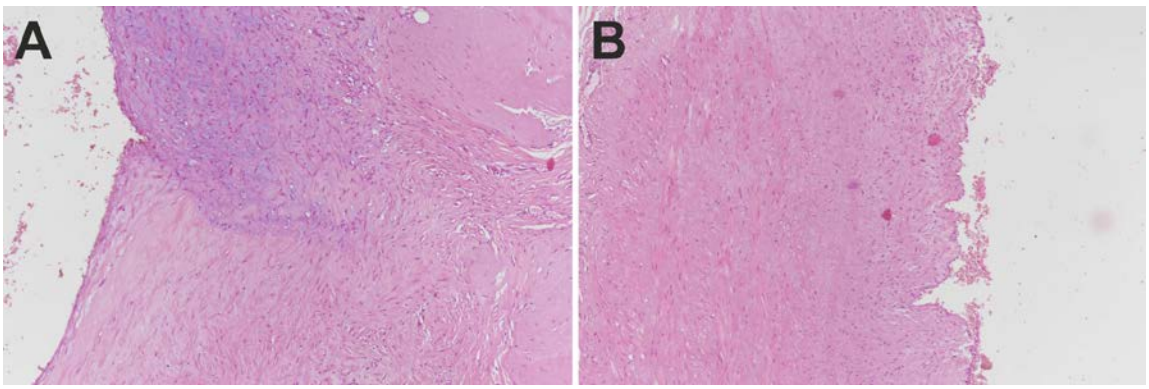

Fig.2. Details of the arterialized venous wall with (A) mature atheroma plaque in the intima-media, and (B) with thickening of the subendothelial space (loose connective tissue with collagen fibers, in a rich fundamental substance). HE staining, 100x

Discussion

AVF is associated with both acute and chronic complications. Acute complications are most frequently caused by surgical technique errors and inadequate topographic choices of the AV fistula placement. Chronic complications are the result of the tissue architecture rearrangement at the level of the graft depending on the circumstances concerning his comorbidities and lifestyle, and the technique of angioaccess used during dialysis sessions [4].

The most frequent late complication is stenosis, caused by both loco-regional and general factors related to coagulation status and hypotension. The literature estimates place the frequency of the juxta-anastomotic stricture at 50-60%, with 20% occurring at the level of the central vein and the remaining, down the arterialized vein [5,6]. In fact, the downstream stenosis in the arterialized vein territory may contribute to aneurysm development, a complication reported in about 3.6 to 4.2% of cases, depending on study cited. The downstream narrowing could be signaled by dialysis session events like difficult cannulation, excessive post dialysis bleeding, and high pump functioning pressure. This condition could potentially lead to AVF thrombosis or aneurism development and requires prompt treatment, either endovascular, by percutaneous transluminal angioplasty (PTA) and stenting (if residual stenosis >30%), or surgical, by proximal re-anastomosis or interposition of graft [1,3].

Other causes of aneurysmal dilatation of the arterialized vein are surgical technique errors, excessive vein dilation and mobilization, conducive to lesions of the vessel wall prone to aneurism formation, and deficient vein puncture technique in the dialysis process [7]. Another possible cause is an anastomosis flow over 1500ml/min, with consequences not only for the efficiency of the dialysis, but also for the cardiovascular system, causing the hyperkinetic syndrome, and leading to sclerosis and dilation of the vessels [8].

The possible consequences of a vein aneurysm are arterial insufficiency, with cooling and numbness of the hand, venous insufficiency with edema, skin laceration and, very important, the rupture of aneurysm with hemorrhage and vital risk. Therefore, the aneurysm must be operated on. The ssurgery, under local anesthesia, is performed with vascular control. In literature a procedure with three incisions was described [7]. Our procedure was performed with a single incision, having a better esthetic result. Another way for management of aneurysm of AVF is angioplasty and stenting, but these proved to be inefficient [9].

By definition, the aneurism is a vessel dilation of 2cm or larger. In literature AVF was depicted with maximum diameter of 5cm [10].

Normally, in the case of acquired AV fistula, vessel remodeling occurs, with dilation of both participating artery and vein. In AVF vein is modified by the thickening of the intima and media [11]. The vascular endothelium and the media of vasculature wall play an important role in this adaptive remodeling. The adaptive response to the shear stress occurs with cytoskeleton rearrangement, resulting in fragmentation of the lamina interna and fenestration, which allow for distensibility increase [12]. This process can be augmented by metalloproteinase release from endothelial cells, assisting the destruction of the internal elastic lamina. The enzymes are activated by the plasminogen-plasmin system and contribute to

the development of the focal ectasia of the vein wall at the origin of the pathogenic vicious circle. Weakening of the wall leads to dilation, which in turn, based on the Laplace law, results in increased wall stress, further promoting the dilation of the vessel. Additionally, the uremic state itself induces vascular remodeling and creates the inflammatory status.

Focal dilation and the wall tissue destruction have also iatrogenic causes pertaining to the vein puncture technique, more precisely the puncture density per unit of wall area. The ectasia generates flow turbulence, which further causes stenosis with secondary post-stenotic dilation, and reentry in the same vicious circle mechanism. In the end, this leads to compromised cardiac function due to hyperkinetic syndrome, local complications (e.g. neurological dysfunction caused by median nerve compression), and poses severe risks (e.g. aneurysm rupture with catastrophic bleeding).

Conclusion

The morpho-pathological AVF vessel wall remodeling has an adaptive role for dialysis efficiency of the vascular assembly and can be exacerbated and become detrimental, both for the loco-regional evolution, and for the systemic hemodynamic status. The control of parietal remodeling risk factors is of outmost importance for the proper maintenance of the vascular access assembly, and for the avoidance of life-threatening complications like severe bleeding from ruptured aneurysms of the arterialized veins.

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