despread use of both LMWHs and unfractionated heparins. In addition, their use in patients at high cardiovascular risk must be carefully evaluated.

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P. Fraile, P. García-Cosmes, T. García and J. M. Tabernero

Servicio de Nefrología. Hospital Universitario de Salamanca.

Correspondence: *Pilar Fraile Gómez. pilarfg9@hotmail.com. Hospital Universitario de Salamanca. Paseo de San Vicente, 58. 37007 Salamanca. España.*

Delayed presentation of a femoral pseudoaneurysm after venous hemodialysis catheter insertion

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To the editor: The use of central venous catheters for temporary vascular access in hemodialysis may occasionally result in arterial puncture.[1] Nevertheless, the frequency of clinically significant arterial damage after femoral catheterization in hemodialysis is low. Such damage may give rise to thrombosis, bleeding, pseudoaneurysms or arteriovenous fistulas.[2-4] We describe a case of delayed presentation of a right femoral arterial pseudoaneurysm following failed venous catheterization for hemodialysis. To our knowledge, only one similar case has been reported to date.[2]

A 41-year-old male was admitted with advanced chronic renal failure of indeterminate origin. The personal history included poorly controlled arterial hypertension and dyslipidemia. The start of renal replacement therapy was required. Ultrasound was used to identify the anatomical relationship between the femoral artery and vein. In both extremities the vein was located posterior to the artery, except over a short trajectory in the region of the inguinal fold, where it was found to be positioned slightly medial. Arterial puncture was observed on one attempt, as a result of which compression was applied and the left femoral vein was finally catheterized. Dialysis without anticoagulation was started in the first two sessions. After 48 hours, and following normal inguinal exploration findings, low molecular weight heparin was resumed as antithrombotic prophylaxis, with doses adjusted to renal function. A few days later during a week-end, the patient participated in a race despite indications to avoid such activities. Twenty days after catheterization he developed sudden right inguinal pain. Inguinal exploration revealed a hard and pulsatile mass with a slight murmur and intense pain in response to palpation. The peripheral pulses were preserved. Right femoral artery Doppler ultrasound confirmed the presence of a 17-mm right pseudoaneurysm. Initial treatment included strict bed rest with an inguinal compressive bandage. However, one week later the pseudoaneurysm was seen to have increased to 21 mm in size, with persistence of the pain. Aneurysm intracavitary thrombin injection under ultrasound guidance was thus performed (100 IU). This resulted in thrombosis of the pseudoaneurysm, without evidence of recurrences on occasion of the posterior ultrasound controls.

An arterial pseudoaneurysm is a pulsatile hematoma resulting from traumatic dissection of the arterial wall, creating a communication between the vascular lumen and the surrounding tissue, with the extravasation of arterial blood. The use of anticoagulants, poorly controlled arterial hypertension, vasculopathy (arteriosclerotic or of an infiltrative nature), and even the technique and arterial trajectory used for puncture can give rise to such pseudoaneurysms.[5-7] The clinical suspicion is established 6-48 hours after arterial puncture, with the identification of a painful, pulsatile mass in the inguinal zone.[7] In our patient, the administered low molecular weight heparin facilitated the delayed presentation of the complication, which was triggered by walking. Doppler ultrasound is the diagnostic technique of choice, and is moreover able to evaluate the evolution of the size of the lesion. Although surgical repair may prove necessary in cases where there is a risk of severe bleeding or limb ischemia, conservative management is initially recommended. Strict bed rest, the suspension of anticoagulation, and compression applied manually in the form of an inguinal bandage or guided by ultrasound over the aneurysmal neck can resolve over 75% of all cases.[5,7] The intra-aneurysmal injection of procoagulating substances such as thrombin represents a treatment option allowing immediate resolution without the need to suspend anticoagulation – though it is not without side effects (generally of an anaphylactic nature).[6]

It is difficult to prevent complications of this kind, considering the technique employed and the antithrombotic indications of our patients. However, compression and prolonged repose after iatrogenic arterial puncture, and the identification and early management of the complications are critical considerations for avoiding traumatic lesions with significant clinical repercussions.

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A. E. Sirvent[1], R. Enríquez[1], D. Martínez[2] and A. Reyes[1]
[1]Sección de Nefrología. [2]Servicio de Cirugía Vascular. Hospital General de Elche. España.
Correspondence: *Ana Esther Sirvent. emma_@wanadoo.es. Servicio de Nefrología Camí de L'Almazara, s/n. 03202 Alicante. España.*

HIV-associated nephropathy without decline of renal function

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To the editor: Collapsing focal glomerulonephritis (CFG) is found in 2-10% of all HIV-infected patients.[1] It is the most common form of renal disease in HIV patients, appearing in over 60% of the renal biopsies made.[2] The presence of proteinuria and/or impaired renal function are associated with increased patient morbidity-mortality.[3] The management of nephropathy associated to HIV infection (NAHIV) has not been established, and most patients require renal replacement treatment a few months after the appearance of nephrotic syndrome.[4]

A 41-year-old black male with type 1 HIV infection not subjected to antiretroviral therapy was admitted with generalized edema and proteinuria in the nephrotic range. Upon admission, the blood pressure was 140/90 mmHg, and edema with fovea was seen to ankle level. The laboratory tests showed normocytic and normochromic anemia with an erythrocyte sedimentation rate of 157 mm in the first hour, normal kidney function (plasma creatinine 1.1 mg/dl and plasma clearance calculated by the MDRD equation 78.41 ml/min), proteinuria 5.83 g/day without Bence-Jones proteinuria, plasma albumin 1.6 g /dl with polyclonal band in gamma region 7.7 g/dl (IgG 9860 mg/dl, IgA 151 mg/dl, IgM 643 mg/dl), and a CD4+ count of 314 cells/mm^3. HBV, HCV and herpes group serology proved negative. A myelogram revealed reactive plasmacytosis, while a bone cylinder specimen showed intense polyclonal lymphoplasmacytosis. Renal ultrasound showed symmetrical kidney enlargement, with preserved corticomedullary differentiation but with a diffuse increase in echogenicity. The Doppler study proved normal. The kidney biopsy revealed collapsing glomerulopathy with preserved tubules and an interstitial lymphocytic and polyclonal infiltrate. Antiretroviral treatment was started with efavirenz, stavudine and lamivudine, together with furosemide and enalapril. At discharge the blood pressure was 130/80, with proteinuria 300 mg/day. The patient posteriorly returned to his country of origin and reappeared 14 months later, without any reported opportunistic processes or nephrotic manifestations. While in his country, the patient continued treatment with enalapril and started nevirapine, zidovudine as lamivudine as antiretroviral therapy. The patient was found to have normal blood pressure, with no edemas, and showed normocytic and normochromic anemia, with normal kidney function (plasma creatinine 0.98 ml/dl), proteinuria 3 g/day and plasma albumin 2.6 g/dl. The CD4+ count was 350 cells/mm^3.

Collapsing focal glomerulonephritis (CFG) is found in 2-10% of all HIV-infected patients,[1] and is the most common form of kidney involvement in black HIV-infected individuals.[2,6,7] CFG is characterized by glomerular collapse and severe tubulointerstitial alterations. The underlying pathogenesis appears to be related to viral infection – HIV infection being the most common example. NAHIV is characterized by proteinuria in the nephrotic range, with rapid deterioration of renal function. In this context, proteinuria and increased plasma creatinine are regarded as indicative of a poor prognosis.[3] At present there is no effective treatment for NAHIV, and most patients require renal replacement therapy on a chronic basis.[4] Some studies suggest that treatment with antiproteinuric agents and highly active antiretroviral therapy (HAART) can delay the progression of renal failure[8] and even reduce the incidence of NAHIV[5] – emphasis being placed on the importance of an early biopsy in these patients.[5] In our case it can be affirmed that combined HAART and angiotensin-converting enzyme inhibitor (ACEI) treatment avoided the deterioration of renal function, reducing proteinuria and resolving the nephrotic syndrome, in a black patient with NAHIV.

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