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Case Report

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ARTERIAL THROMBOSIS AND NEPHROTIC SYNDROME: ABOUT A CASE

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INTRODUCTION

Hypercoagulability, resulting in thromboembolic events, can be a life-threatening complication of nephrotic syndrome. Many factors expose to hypercoaguability: the leakage of coagulation inhibitors in the urine, imbalance in the fibrinolytic system, hyperlipidemia, as well as genetic predisposition. Arterial thrombosis is less frequent than venous thrombosis, however it poses a diagnostic and therapeutic problem, and continues to compromise the vital and functional prognosis.[1] We report the observation of arterial thrombosis of the lower limb in a patient with nephrotic syndrome.

OBSERVATION

Mr EB, 39 years old, has a nephrotic syndrome since 1998. Kidney biopsy finds minimal glomerular lesions. There were corticosensitives. The patient has been lost to follow-up since 2007. In 2018, he was hospitalized for an acute right leg ischemia. On admission, the clinical examination objectified: a cyanotic limb, cold with an abolition of the right popliteal and pedals pulse, without any notion of trauma. The biological kidney test: proteinuria at 12 g / d, hypoproteidemia at 43 g / l; with a deep hypoalbuminemia at 17g / l, renal function was normal. In the hemogram, we noted: anemia at 10.9 g / dl, hyperleukocytosis at 13590 / µl, with thrombocytosis at 704000/µl. The hemostasis testing find: a Prothrombin test and activated partial thromboplastin time: normal, a factor VIII: high (180%), a factor XII: low (30%) and factors IX and XI were normal. The Thrombophilia testing: hyperfibrinogemia at 6.6 g / l, with no other significant anomaly. Doppler ultrasound of the two lower limbs objectified: a complete thrombosis of the right popliteal artery with multiple atherosclerotic plaques of the arteries of the two lower limbs without stenosis. The computed tomography scan revealed an occlusion of the right femoral artery at the femoro-popliteal junction (Figure 1). The patient benefited urgently from an amputation of the middle 1/3 of the right leg, after the failure of an arterial thrombectomy with the Fogarty catheter. The treatment given was a curative anticoagulation based on low molecular weight heparin overlapped by AVK, and an antibiotic. The evolution was marked by rapid healing of the stump, without resorting to necrosectomy gestures (Figure 2). We realized a second renal biopsy showed lesions of segmental and focal hyalinosis, treated by oral corticosteroid therapy. We noted a partial remission of the nephrotic syndrome (proteinuria 24h: 3.86 g / d in the 21st month). We conclude to a probable cortioresistance. Rituximab was then introduced as an alternative, at a dose of 375 mg / m2 / week for 4 weeks with a favorable development.

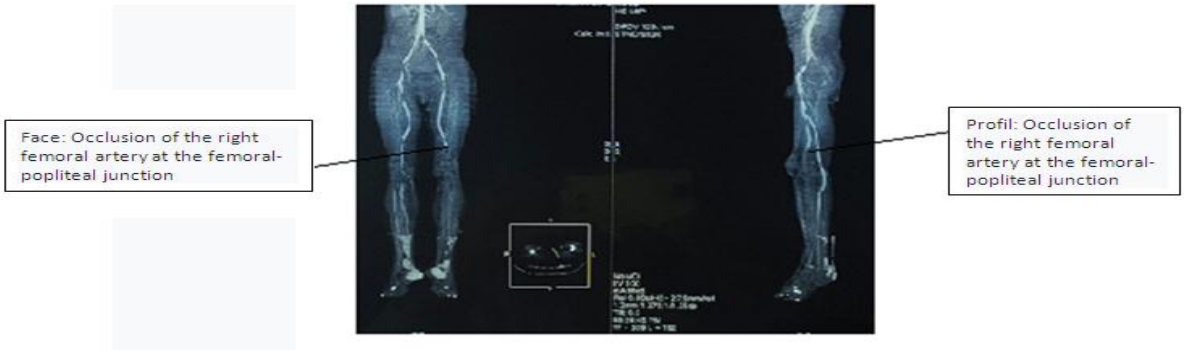

Figure 1: Occlusion of the right femoral artery at the femoral-popliteal junction.

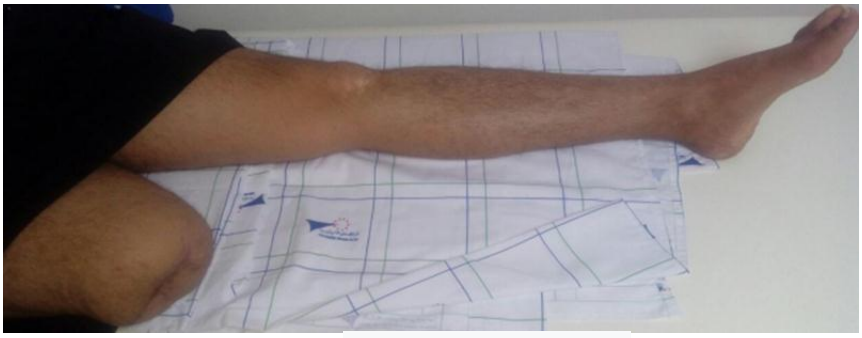

Figure 2: Patient after amputation.

3. DISCUSSION

Thromboembolic complications have a considerable incidence during nephrotic syndrome. Their frequency varies according to the authors. Venous thromboses are more frequent than arterial thromboses (9% versus 42%),[1,2,3] and they are especially seen in the pediatric population.[4]

The localizations reported are ilio-femoro-leg, renal, cerebral, as well as coronary thrombosis.[5]

3.1. Physiopathology

During nephrotic syndrome, we have various abnormalities of hemostasis, which contribute to an imbalance between pro-coagulant factors and coagulation inhibitors. Hypercoagulability is due, on the one hand to the elevation of the hepatic synthesis of the various coagulation factors (fibrinogen, factor V, VII, XIII) which are frequently found during nephrotic syndrome; On the other hand, there is urinary loss of antithrombotic factors like albumin, antithrombin III, factors IX and XI, that are not always compensated by the increase in hepatic synthesis. Primary hemostasis abnormalities have also been reported: thrombocytosis with platelet hyperaggregability secondary to platelet activation. Fibrinolysis is also disrupted by to the decrease of plasminogen and the increase of fibrinolysis inhibitors. Other factors have been reported in the literature: diuretics, hypovolaemia, infection, trauma, immobilization, venipuncture and arterial puncture but also corticosteroid therapy by an increase in hepatic factor V. These various anomalies of hemostasis are summarized below (fig. 3.),[1] Corticosteroid resistance multiplies by 2.5 the risk of thrombosis.[1,2] In our observation, the thromboembolic accident occurred after 18 years of progression of the disease. However, histological lesions like membranoproliferative glomerulonephritis, Membranous Nephropathy and focal segmental glomerular sclerosis are associated to an important thrombogenic risque. Our patient had focal segmental glomerular sclerosis.

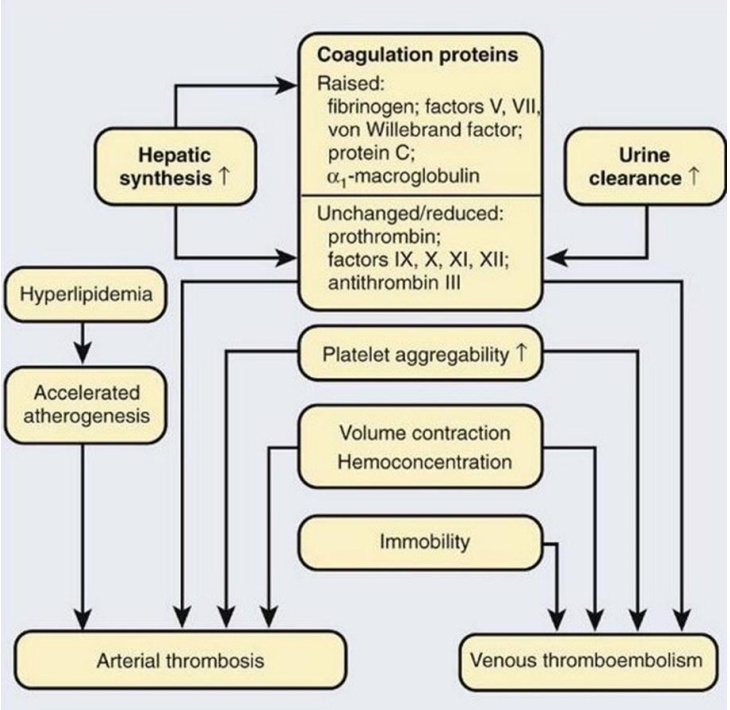

Figure 3: Coagulation abnormalities in Nephrotic Syndrome.

3.2. Therapeutic care

Our patient benefited from an amputation of the middle 1/3 of his right leg, after the failure of the revascularization gesture, followed by anticoagulation. There is no consensus regarding the treatment of thromboembolic complications during nephrotic syndrome,[2] Some teams,[11] recommend the use of local thrombolysis injections (streptokinase) after catheterizations for 5 to 8 days, with the aim of permeabilizing the distal arteries and reduce the risk of amputation.

3.3. Prevention

Some authors reserve preventive treatment in cases of ananasarc while others recommend it if we had patient with: albuminemia <20g /l, fibrinogemia > 6g / l, D-plasma dimers > 1000ng / ml, antithrombin III <70% of normal. In case of a history of thromboembolic complications, anticoagulant treatment at a preventive dose is indicated, immediately, by certain teams.[2,4,7,8,9,10] Obtaining a remission or better healing of the nephrotic syndrome is imperative in order to avoid this dreaded complication. General measures are to be adopted: regular physical activity, avoid prolonged decubitus and deep venous approaches, Correction of addictive factors.

4. CONCLUSION

Arterial thrombosis during nephrotic syndrome are rare but it is a serious complication that can threats the patient's functional and life prognosis. Their diagnosis must be early. Anticoagulants treatment have provided additional options for the treatment and prevention of thromboembolic events.

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