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# Arterial Thrombosis Associated with a Case of Nephrotic Syndrome: A Case Study

El Ouazzani, M[1*], Bouchoual, M[1], Dadi, K[1], Anibar, S[1], Jabrane, M[1], Arrayhani, M[1]

[1]Department of Nephrology, SOUSS MASSA University Hospital, Faculty of Medicine and Pharmacy, Ibn Zohr University, Agadir, Morocco

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***Corresponding author:** El Ouazzani, M
Department of Nephrology, SOUSS MASSA University Hospital, Faculty of Medicine and Pharmacy, Ibn Zohr University, Agadir, Morocco

**Abstract** **Case Report**

Thromboembilic complications are common in nephrotic syndrome. Only a few cases of arterial thrombosis have been reported in the literature, mainly in adults. Their management is not codified; it depends on the location and hypercoagulable state. We report the observation of an acute ischemia of the lower limb occurring after a nephrotic syndrome in a 67-year-old diabetic patient admitted for edemas of the lower limbs evolving since three weeks before his admission.

**Keywords:** Arterial thrombosis, ischemia, nephrotic syndrome, kidney biopsy.



## INTRODUCTION

The nephrotic syndrome (NS) presents a complex scenario marked by a heightened risk of thromboembolic events, primarily affecting peripheral venous sites, with arterial thromboses being a seldom-documented occurrence [5]. This phenomenon can be ascribed to a multifaceted interplay of factors, encompassing hypercoagulability, hypovolemia, hemoconcentration induced by substantial relapses, administration of diuretics, corticosteroids, periods of immobilization, and a deficiency in antithrombin III. As an illustrative example, this article describes the observation of diffuse arterial thrombosis incidentally discovered during the assessment of a 67-year-old diabetic patient admitted for lower limb edema with signs of acute ischemia, revealing an incidental finding of nephrotic syndrome.

## OBSERVATION

This concerns a 67-year-old patient, known to have diabetes for three years and and currently under insulin therapy, admitted to the emergency department for ischemia in the left leg with sudden-onset gangrene and blistering on the right calf. This presentation was associated with lower limb edema and facial swelling evolving over the past three weeks.

Upon admission, the clinical examination identified a conscious patient with a normal body temperature of 37 °C, exhibiting hypotension at 90/50 mmHg and maintaining a regular heart rate of 76 beats per minute. Respiratory parameters were stable, and an active urinary sediment was noted on the dipstick. Examination of the limb revealed a bilateral, soft-edged edema with pitting, accompanied by cyanosis and coolness in the right lower limb. Notably, the pulses in the popliteal, tibial, and dorsalis pedis on the left side were absent.

The laboratory analysis conducted in the emergency setting revealed an inflammatory syndrome characterized by an elevated CRP level of 50 mg/L. Additionally, a profound and complex nephrotic syndrome was observed, manifested by significant proteinuria at 15.09 g/day, hypoalbuminemia at 19 g/L, and hypoproteinemia at 37 g/L. Concomitantly, a moderate functional acute renal injury was evident, as indicated by a creatinine level of 15 g/L, alongside microscopic hematuria.

Moreover, the remaining components of the biological assessment, encompassing infectious (complete blood count, chest X-ray, cytobacteriological examination of urine, abdominal ultrasound, COVID-19 PCR, and procalcitonin), serological (HIV, hepatitis B, hepatitis C, and syphilis), and immunological parameters

(antinuclear antibodies, anti-DNA antibodies, complement C3, C4, and anti-PLA2R antibodies), exhibited no abnormalities.

Subsequently, the patient underwent an urgent lower limb angiogram, revealing complete occlusion of the left iliac artery, the right superficial femoral artery (Figure 1), and the right trifurcation (Figure 2).

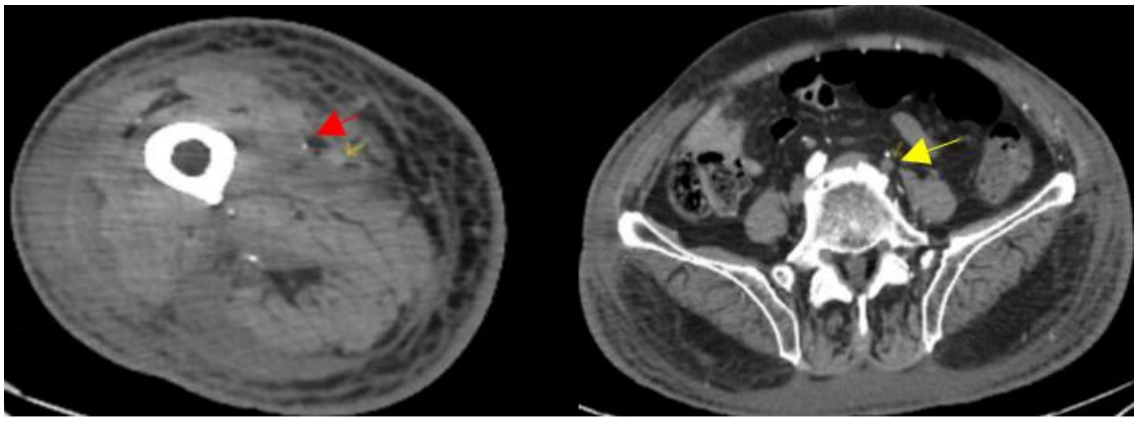

**Figure 1: Angioscanner indicates occlusion in the right superficial femoral artery and left common iliac artery**

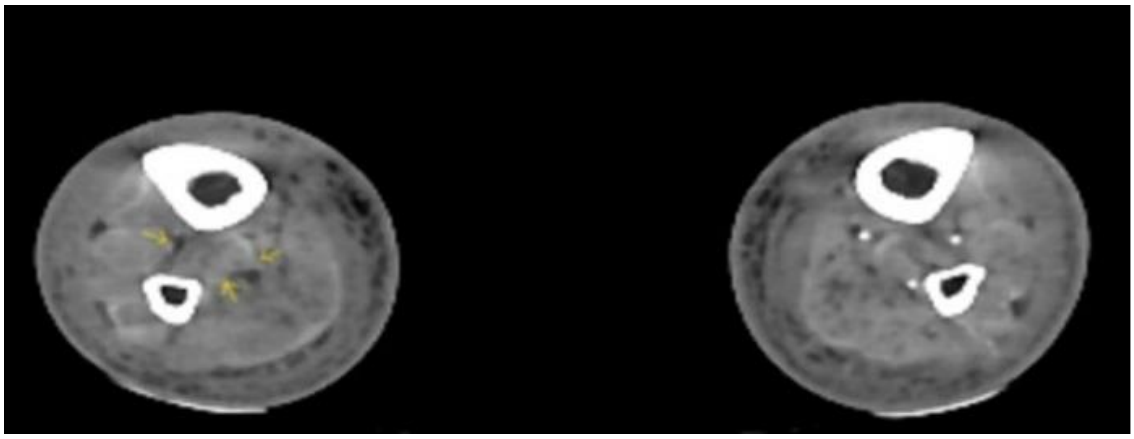

**Figure 2: Angioscanner of the lower limb reveals occlusion of the right trifurcation**

The therapeutic decision involved a partial left leg amputation. Postoperatively, the patient received prophylactic anticoagulation therapy, nephroprotective treatment, and intravenous antibiotic therapy.

After one-week post-amputation, the patient underwent a renal biopsy, which revealed AA amyloidosis associated with diabetic nephropathy features (Figure 3). The etiological workup of amyloidosis showed no specific findings. The patient was initiated on nephroprotective therapy and preventive anticoagulation with close surveillance.

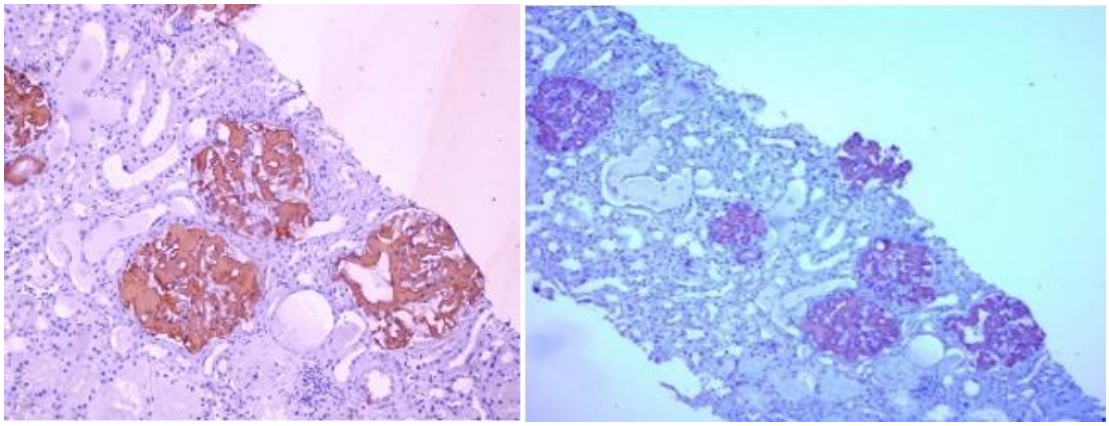

**Figure 3: Trichrome staining and immunohistochemistry demonstrate diffuse AA renal amyloidosis**

## DISCUSSION

The incidence of thromboembolic complications in nephrotic syndrome is high, occurring in approximately 1.8% to 5.3% of pediatric cases [1].

Peripheral vein thromboses are frequently observed, with an incidence of 12%. Arterial thromboses, though less common, are more prevalent in pediatric populations [2, 3], particularly in individuals aged over 12 years.

Several pathophysiological factors contribute to the occurrence of these thrombotic events. Nephrotic syndrome precipitates an imbalance between pro-coagulant factors and anticoagulant mechanisms: hepatic synthesis of fibrinogen escalates, particularly with serum albumin levels ranging between 30 and 35 g/L. Fibrinolysis is impeded by reduced plasminogen levels, heightened concentrations of tissue plasminogen activator inhibitors, and urinary excretion of α2-antiplasmin and α1-antitrypsin [6, 7]. Furthermore, there is a surge in cholesterol levels and elevated levels of factors V, VIII, and XII, alongside augmented availability of arachidonic acid.

These various modifications in hemostasis lead to a predisposition to thrombosis. Other factors have also been implicated, including diuretics, hypovolemia, infection, immobilization, venous and arterial punctures, as well as corticosteroid therapy through an increase in hepatic factor V.

There is currently no consensus on the optimal anticoagulation strategy for thromboembolic events. The choice of therapy is usually determined by the specific location and severity of hypercoagulability. Typically, initial treatment involves high-dose heparin therapy due to its reliance on antithrombin III for anticoagulant activity [4, 8].

The initial treatment involves the use of therapeutic doses of unfractionated heparin or low molecular weight heparin. Therapeutic anticoagulation is continued for a period of 5 to 7 days (with low-molecular heparin or unfractionated heparin) [9]. Prophylactic anticoagulation may be warranted in cases with a history of thromboembolism, as per recommendations from other medical teams [10].

To prevent thromboembolic events, it is essential to implement general measures such as regular physical activity, avoiding prolonged immobilization and deep vein access, correcting hypovolemia, and refraining from unnecessary infusions.

## CONCLUSION

Arterial thromboses occurring in nephrotic syndrome are infrequent yet severe complications, posing a significant threat to both the patient's functional capacity and overall survival. Prevention can be achieved through simple measures as: regular physical activity, avoiding vascular punctures, avoiding prolonged immobility, and correcting hypovolemia. The risk of thrombus expansion and subsequent pulmonary embolism is a tangible concern.

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