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CASE REPORT

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Popliteal arterial thrombosis in nephrotic syndrome: a case report

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ABSTRACT

Thrombosis is a frequent cause of morbidity and mortality in patients with nephrotic syndrome (NS). Though venous thromboses are common in NS, arterial thromboses are relatively rare. Commonly involved arteries include coronary, iliac, femoral, renal, cerebral, pulmonary, mesenteric, and axillary arteries, and the aorta. Arterial thromboses are associated with poor prognosis; treatment options are limited and patients may not always be amenable to treatment. We present the case of a 39-year-old female with NS who presented with thigh pain and was found to have sub-acute popliteal artery thrombosis.

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1. Introduction

Nephrotic syndrome is associated with significant and potentially serious thromboembolic complications.[1,2] Arterial thrombotic complications are relatively rare but more serious than venous ones. Thromboembolic complications in nephrotic syndrome are multifactorial.[1,2] Steroid therapy which is commonly used in treatment of nephrotic syndrome can itself cause hypercoagulability and provoke thrombosis.[1] Awareness of the condition and assessment of the risk factors are required to allow appropriate prophylactic measures to be taken while taking care of these patients.

2. Case presentation

A 39-year-old female with past medical history of deep vein thrombosis (DVT), chronic kidney disease stage-3 with biopsy-proven focal segmental glomerulosclerosis (FSGS), and tobacco abuse, presented to the emergency department (ED) with complaint of five-day pain in left medial thigh and popliteal region limiting her ambulation and sleep. Thirteen days before presenting to the ED, she was started on high dose prednisone (60 mg daily). A week after starting prednisone, she developed the above-mentioned symptoms along with pain and numbness in her left great toe and second toe. There was no history of chest pain, shortness of breath, palpitations or trauma to the leg.

She had a history of two episodes of unprovoked DVT and received prophylactic anticoagulation during her last two pregnancies. Hypercoagulable workup performed at outside hospital during that time was negative. There was no history of miscarriage or bleeding disorder. She did not have a history of atrial fibrillation or other heart disease. There was no family history of clotting disorders. She smoked half pack of cigarettes a day.

On physical examination, she was in no acute distress. Her blood pressure was 147/77 mm Hg, pulse 68/minute, temperature 37.3°C, respiratory rate 19/minute and oxygen saturation 100% at room air. Lungs were clear to auscultation. Cardiac examination revealed normal rate and rhythm with normal first and second heart sounds with no murmur. Femoral pulses were palpable bilaterally. Left dorsalis pedis and posterior tibial artery pulse were absent. There was tenderness to palpation in left medial distal thigh and popliteal region; however, no calf tenderness or cords were noted. There was no skin breakdown, rash, pedal edema, pallor on leg elevation or dependent rubor. Strength in bilateral lower extremities was normal.

A venous ultrasound (US) of the left lower extremity (LLE) revealed a superficial muscular branch thrombosis in the popliteal region but no evidence of DVT. Arterial US duplex of LLE revealed sub-acute thrombosis of left popliteal artery and tibio-peroneal artery. Ankle-brachial index was 1 on the right and 0.57 on the left. Electrocardiogram revealed no arrhythmias. Transthoracic echocardiogram revealed normal systolic and diastolic function with no valvular abnormalities.

Pertinent labs included creatinine 1.6 mg/dL, BUN 26 mg/dL, total urine protein/creatinine ratio of 1.8,

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serum albumin 3.1 g/dL and normal C3 and C4. Complete blood count revealed WBC of $10.7 \times 10^3/\mu L$, hemoglobin of 14.4 g/dL and platelets of $127 \times 10^3/\mu L$. Coagulation profile was normal with normal INR and aPTT. Hypercoagulability workup was negative with normal fibrinogen level, protein C, protein S activity and antithrombin III activity. Factor V Leiden mutation (R506Q), prothrombin G20210A mutation and methylene-tetrahydrofolate reductase (MTHFR) C677 T gene mutation were negative. Lupus anticoagulant, anti-phospholipid antibodies and anti-nuclear antibody (ANA) were negative.

She was started on tissue plasminogen activator (tPA) at 1 mg/hr along with continuation of prednisone and analgesia. There was some improvement of her symptom but little improvement in the occlusion of the arteries. She was started on anticoagulation with heparin bridged with warfarin. Her symptoms gradually improved and she was discharged home on warfarin and prednisone. She did not have recurrence of thrombosis in the following six months.

3. Discussion

Nephrotic syndrome is characterized by nephrotic range proteinuria (>3.5 g/24 h), hypoalbuminemia, hyperlipidemia (hypertriglyceridemia and hypercholesterolemia), lipiduria and edema.[3] It is associated with potentially significant and serious arterial and venous thromboembolic complications.[1,2,4] Venous thromboembolism is thought to be related to the degree of proteinuria and hypoalbuminemia while arterial thromboembolism is related to the presence of traditional risk factors for atherosclerosis.[2] Thrombosis most frequently occurs in the earlier months of diagnoses though it may happen at any time during the course of NS.[1]

Thrombosis in NS is multifactorial, and has been attributed to hypercoagulability and abnormalities in platelet function, coagulation factors and fibrinolytic factors.[1,5] Hypercoagulable state occurs due to changes in the blood level and function of prothrombotic and antithrombotic factors.[1] There is decreased level of factors IX, XI, XII and antithrombin III due to urinary loss.[5] Similarly, there is increased level of fibrinogen, factors II, V, VII, VIII, and X due to increased synthesis in the liver in response to hypoalbuminemia.[5] Hypoalbuminemia in NS causes intravascular dehydration. Diuretics used to decrease the edema due to NS may lead to hemoconcentration and increased risk of thrombosis.[5] Similarly, steroids used in the management of NS, as in our patient, can contribute to thrombosis by altering the level of coagulation factors, thereby aggravating the hypercoagulability.[1] Hypertension, hyperlipidemia, diabetes mellitus, tobacco, and obesity are additional risk factors.[1] Our patient had arterial thrombosis most likely due to steroid administration and NS as most other causes of thrombosis had been ruled out. She had hyperlipidemia and tobacco smoking as additional risk factors.

Clinical features of arterial thrombosis depend on the site of involvement. Any arteries may be involved but those most commonly involved include renal, aortic, femoral, cerebral, mesenteric, coronary, iliac, subclavian, and other peripheral arteries in descending order.[6] Since arterial thrombosis due to NS has increased morbidity and mortality, timely diagnosis and management is warranted.[1] Thrombectomy, anticoagulation, and immunosuppressive therapy along with symptomatic management are the available treatment options.[1,7–9] Some studies suggest prophylactic anticoagulation in patients with NS with additional risks for thrombosis like steroid therapy or very low plasma albumin.[7] Additional studies need to be done regarding the role of prophylactic anticoagulation in high risk patients with multiple risk factors when they are started on steroid therapy for NS.

4. Conclusion

Thromboembolic complications, particularly arterial thrombosis, are relatively uncommon in NS. The exact pathophysiology still remains unclear and the treatment options are limited. More studies are required regarding the role of prophylactic anticoagulation when these patients are started on steroid therapy, especially when they have additional risk factors of hypercoagulability.

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Disclosure statement

No potential conflict of interest was reported by the authors.

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