CASE REPORT: ARTERIAL THROMBOSIS IN ADULT NEPHROTIC SYNDROME

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INTRODUCTION

Venous thromboembolic complications are frequently caused by nephrotic syndrome, while arterial thrombosis has rarely been reported and is mainly noted in pediatric population. In this article, we report the case of a 25 years-old man who presented a thrombosis of the left popliteal artery, a thrombosis of right renal artery, a thrombosis of internal iliac arteries and splenic infarction. Nephrotic syndrome was secondary to a focal segmental glomerulosclerosis. Patient underwent femoral embolectomy with anticoagulation therapy. Arterial thrombosis is rare but serious complication of nephritic syndrome. Identification of high-risk patients is necessary.

KEYWORDS: acute arterial thrombosis, nephrotic syndrome.

CASE REPORT

Mr KA, 25 years old, has a relapsed nephrotic syndrome since the age of 8 years, complicated by an intracardiac thrombus and thrombosis of the right popliteal artery leading to amputation of the right leg. He was an infrequent relapse. At the age of 21 years old, he was hospitalized in our department for relapse of nephrotic syndrome. The physical examination revealed generalized edema. Laboratory evaluation revealed nephrotic range proteinuria (24 hours urinary: protein 7 gm), hypoalbuminemia (serum albumin: 0.5 gm/dl), hypercholesterolemia (serum cholesterol: 350 mg/dl). Renal function was normal (serum creatinin: 0.6 mg/dl) and liver functions were normal. Kidney biopsy showed collapsing focal segmental glomerulosclerosis. The patient was treated by corticosteroid therapy (prednisone at a daily single dose 1mg/kg), anticoagulant therapy and diuretic. Ten days after, he was readmitted with severe pain with bluish discoloration of left lower limb and clinical features of ischemia with absence of pulsations in left popliteal and pedis arteries. Doppler study revealed absence of blood flow in territory of the popliteal artery. The patient underwent urgent thrombectomy throught the femoral artery with immediate recovery of the popliteal pulse followed by anticoagulation. Initially he was started on heparin, which was followed by oral anticoagulant. A few hours after the acute ischemia of the lower limb, he had a complaints of severs and brutal abdominal pain without nausea or vomiting or rectorrhagia. No defense or abdominal contracture. Computed tomography angiography of the abdomen demonstrated a partial thrombosis of the left renal artery, bilateral renal and splenic infarction and bilateral internal iliac arteries thrombosis. The electrocardiogram showed no atrial fibrillation. The thrombophilia assessment was negative. After 4 weeks, patient attained remission, steroids were tapered and stopped at the end of 16 weeks. Four years later, the patient relapsed once with complete remission under oral steroid therapy, renal function is still normal and he is still under anticoagulant.

computed tomography angiogram showing renal infarction
Computed Tomography Angiogram showing splenic infarction.

Computed Tomography Angiogram showing thrombosis of left renal artery.

Computed Tomography Angiogram showing bilateral internal iliac arteries thrombosis

DISCUSSION

Vascular thrombosis is a serious complication of nephrotic syndrome. Their frequency varies from 4 to 40% according to the authors. Arterial thrombosis (3 to 9%) appears to be less frequent than venous thrombosis (11 to 42%). The latter tend to affect the adult, whereas arterial thromboses are more frequent in children. The arterial thrombosis in nephrotic patient reported in the literature mainly concerned the aorta, mesenteric, femoral, axillary, brachial, renal, cerebral coronary and pulmonary arteries. Our patient had popliteal, renal and internal iliac arteries thrombosis.

Altered levels of coagulation factors and fibrinolytic system in patients with nephrotic syndrome leads to hypercoagulability. The abnormalities include increased levels of fibrinogen, factor V, Von willebrand factor, factor VII, alpha-1 macroglobulin. This is thought to be due to increased hepatic synthesis of these factors stimulated by hypoalbuminemia and decreased levels of antithrombin III, plasminogen, factor XI, factor XII, alpha one antitrypsin, protein S, and protein C due to urinary losses of these proteins.

Most of the authors noted elevated levels of serum fibrinogen and decreased level of antithrombin III. Increased blood viscosity due to hemoconcentration contributes to occurrence of thromboembolic complications, which is often aggravated by the use of diuretics. Steroids shorten prothrombin as well as accelerate partial thromboplastin times that aggravate the hypercoagulable state of nephrotic syndrome. The risk of thrombosis is related to the severity and duration of the nephrotic state and appears to be particularly increased with serum albumin concentrations ≤2.0 g/dl.

Once the diagnosis of vascular thrombosis is established, anticoagulation therapy should be started. We can use conventional or low molecular weight heparin, followed by oral anticoagulant and antiplatelet agents. Thromboembolectomy is indicated in patients with ischemic limbs due to arterial thromboembolism. Our patient was treated with conventional heparin, followed by oral anticoagulant. Warfarin is not available so we use acenocoumarol.

CONCLUSION

Arterial thrombosis is a rare but serious complication of nephrotic syndrome. Assessment of various risk factors such as the duration and severity of hypoalbuminaemia may be useful in identifying high-risk patients.

REFERENCES


