OPEN ACCESS Freely available online

ISSN: 2572-9462
Journal of Thrombosis and Circulation: Open Access

Opinion Article

Significant Causes of Thromboembolic Events in Nephrotic Patients

Jane Wale*

Department of nuclear cardiology, Loma Linda University, Loma Linda, USA

DESCRIPTION

The urine loss of antithrombotic factors by the damaged kidneys and the increased production of prothrombotic factors by the liver are hypothesised to be the causes of thromboembolic events in nephrotic patients. Significant and potentially dangerous thromboembolic consequences are linked to nephrotic syndrome. Although more severe than venous ones, arterial thrombotic problems are very uncommon. Nephrotic syndrome's thromboembolic consequences are multifaceted. Steroid therapy, which is frequently used to treat nephrotic syndrome, has the potential to increase thrombosis risk and hypercoagulability. It is necessary to be aware of the condition and evaluate the risk factors in order to take the proper preventative actions while caring for these individuals. Nephrotic range proteinuria (> 3.5 g/24 h), hypoalbuminemia, hyperlipidemia (hypertriglyceridemia and hypercholesterolemia), lipiduria, and edoema are the hallmarks of nephrotic syndrome. It has venous and arterial thromboembolic consequences that could be quite dangerous and significant. Whereas arterial thromboembolism is assumed to be associated to the existence of conventional risk factors for atherosclerosis, venous thromboembolism is expected to be related to the degree of proteinuria and hypoalbuminemia. Although it can happen at any point over the course of NS, thrombosis most typically happens in the first few months after diagnosis. Thrombosis in NS is complex and has been linked to changes in platelet function, coagulation factors, and fibrinolytic factors as well as hypercoagulability. Changes in the blood level and function of prothrombotic and antithrombotic agents result in the hypercoagulable state. Due to urine loss, the levels of factors IX, XI, XII, and antithrombin III have reduced. Similar to fibrinogen, factors II, V, VII, VIII, and X are also present at higher levels due to enhanced production in the liver in response to hypoalbuminemia. Intravascular dehydration is a result of hypoalbuminemia in NS. The use of diuretics to reduce the edoema brought on by NS can raise the risk of thrombosis and hemoconcentration. steroids used to treat NS can exacerbate hypercoagulability by changing the level of coagulation factors, which can lead to thrombosis. Other risk factors include obesity, cigarette use, hypertension, and hyperlipidemia. As most other potential causes of thrombosis had been ruled out, our patient's arterial thrombosis was most likely caused by steroid use and NS. Depending on the area involved, arterial thrombosis has different clinical characteristics. Any artery may be affected, but the ones that are most frequently affected are, in descending order, the renal, aortic, femoral, cerebral, mesenteric, coronary, iliac, subclavian, and other peripheral arteries. The need for prompt identification and treatment arises from the increased morbidity and mortality caused by arterial thrombosis brought on by NS. The possible treatments include thrombectomy, anticoagulation, immunosuppressive therapy, and symptomatic care. In patients with NS who have extra risks for thrombosis, such as steroid medication or very low plasma albumin, several studies recommend prophylactic anticoagulation. The role of prophylactic anticoagulation in high risk patients with various risk factors when they begin receiving steroid therapy for NS requires further research. With NS, arterial thrombosis and other thromboembolic consequences are relatively uncommon. There are few therapy options available, and the precise aetiology is yet unknown. In particular, when these patients have additional risk factors for hypercoagulability, more research is needed to determine the function of preventive anticoagulation.

Correspondence to: Jane Wale, Department of nuclear cardiology, Loma Linda University, Loma Linda, USA, E-mail: Janewale@gamil.com

Received: 22-Feb-2023, Manuscript No. JTCOA-23-20310; **Editor assigned:** 24-Feb-2023, PreQC No. JTCOA-23-20310 (PQ); **Reviewed:** 10-Mar-2023, QC No. JTCOA-23-20310; **Revised:** 17-Mar-2023, Manuscript No. JTCOA-23-20310 (R); **Published:** 27-Mar-2023, DOI: 10.35248/2572-9462.23.9.215

Citation: Wale J (2023) Significant Causes of Thromboembolic Events in Nephrotic Patients. J Thrombo Cir.9:215.