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A Young Female Presented With Massive Pulmonary Embolism With Inferior Vena Cava Thrombus as a Complication of Nephrotic Syndrome: A Case Report

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Case report

Keywords: Nephrotic syndrome, Thromboembolism, Inferior vena cava thrombosis, Pulmonary embolism

Posted Date: August 17th, 2020

DOI: https://doi.org/10.21203/rs.3.rs-58762/v1

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Abstract

Nephrotic syndrome (NS); the first described in 1827 as the presence of proteinuria of ≥ 3.5 g/24 hours, albuminemia < 3.0 g, peripheral edema, hyperlipidemia, lipiduria, and increased thrombotic risk which has an incidence of three new cases per 100 000 each year in adults. Nephrotic syndrome had complicated by the induction of a hypercoagulable state with both various venous and arterial thromboembolic events. The pathophysiology of hypercoagulability in the nephrotic syndrome is imbalances of prothrombotic and antithrombotic factors, as well as impaired thrombolytic activity occurs. Here we present, a 19 years old female came to the emergency department with a complaint of chest pain and shortness of breath for three days. The patient quickly diagnosed pulmonary embolism and inferior vena cava thrombosis as a complication of nephrotic syndrome, allowing prompt initiation of anticoagulant therapy. After two weeks of admission, the patient's resolved dramatically, and his laboratory results returned to almost normal, and the patient discharged with Oral Prednisolone, Coumadin, Atorvastatin, and Ramipril. We aim to determine which is the likely cause of pulmonary embolism in patients with the nephrotic syndrome.

Introduction

Nephrotic syndrome (NS), first described in 1827 as the presence of proteinuria of ≥3.5 g/24 hours, albuminemia <3.0 g, peripheral edema, hyperlipidemia, lipiduria, and increased thrombotic risk (1,2). Nephrotic syndrome has an incidence of three new cases per 100 000 each year in adults (3). Thromboembolism, including pulmonary embolism, deep venous thrombosis (DVT), renal vein thrombosis, and inferior vena cava thrombosis, had reported as a life-threatening complication of nephrotic syndrome patients(4). Enhanced platelet aggregation and renal losses of anticoagulant proteins included AT□III thought to carry the excessive thrombotic risk in patients with nephrotic syndrome (4,5). Here, we present a case of an unusual combination of multiple venous thromboses as the presentation of nephrotic syndrome.

Case Report

A 19 years old female, previously healthy, nonsmoker, non-diabetic came to the emergency department with a complaint of chest pain and shortness of breath for three days, and she never had a similar situation before. On examination, the patient looked ill, anxious, tachyapneic, and edematous. On vital signs, her respiratory rate was 23 breath/minute, blood pressure 110/70 mm/hg, pulse 112 bpm, and oxygen saturation was 90%. Head and neck examination were unremarkable. Tachycardia was the only prominent sign of cardiovascular findings. The respiratory evaluation revealed diminished breath sounds in both lungs. Grade 2+ lower limb edema noted during the examination and the other systems were unremarkable. Laboratory investigations, complete blood count, and serum electrolytes revealed normal range. Regarding the liver function test, AST was 35U/L, ALT was 16U/L, total protein was 4g/dl, Albumin was 1.9g/dl. While total cholesterol was 450mg/dl, and lactate dehydrogenase was 639 U/l. HBV, HCV, HIV, RF, and tumor screening were all in the normal range or negative. The D-dimer (>4µg /ml) and 24-hour

urinary protein excretion (10 g) were positive. As there had no obvious predisposing factor for PE, tests for the etiology of the thrombophilic state had ordered.

Chest x-ray and electrocardiograms were unremarkable. Abdominal Ultrasonography detected mild ascites. Computed tomography (CT) angiography of the chest and abdomen was performed and revealed Thrombus at both rights and left pulmonary arteries and also inferior vena cava (figure 1-A & B). The patient admitted due to nephrotic syndrome complicated with pulmonary embolism and inferior vena cava thrombosis. The patient started with heparin, Ramipril, Methylprednisolone, atorvastatin, and two days later, added a Coumadin tablet. After two weeks of Hospitalization, the patient symptoms resolved dramatically, lower limb edema decreased, proteinuria was not detected, Albumin returned to the normal range, and the patient discharged with Oral Prednisolone, Coumadin, Atorvastatin and Ramipril. Close follow up was recommended to the patient. Six months follow up for the patient was not showing any complaints and no thrombus on the chest CT angiography (figure-2).

Discussion

Patients with nephrotic syndrome carry a high risk of both venous and arterial thrombosis(4). Pulmonary embolism, DVT, renal vein thrombosis, and inferior vena cava thrombosis had reported as life-threatening complications of nephrotic syndrome (4,5).

The etiology of NS divided into primary that included focal segmental glomerulosclerosis (FSGS), membranous nephropathy (MN) and minimal change disease (MCD), and secondary to systemic diseases which included diabetes mellitus, systemic lupus erythematosus, multiple myeloma, amyloidosis, and infections (6). The pathophysiology of hypercoagulability in the nephrotic syndrome is due to imbalances of prothrombotic and antithrombotic factors, impaired thrombolytic activity and other important contributing factors such as intravascular volume depletion, the use of diuretics, immobilization, and procoagulant diatheses (such as protein C and protein S deficiencies) (7).

Hull RPet al reported DVT at the lower limbs as the most common complication of nephrotic syndrome, but also thrombosis at renal and pulmonary vessels was noted (6). Our case first presented with pulmonary embolism as a complication of nephrotic syndrome with no previous history of NS that can occur at the presentation, but it's uncommon.

Similarly to the present case, Peces R et al (8) reported 42 years old male presented with multiple venous thromboses with pulmonary artery embolism as a complication of nephrotic syndrome. In our case, the presence of pulmonary embolism and inferior vena cava thrombosis as complications of nephrotic syndrome followed by the persistence of adequate renal blood flow to prevent renal infarction.

Conclusion

The nephrotic syndrome is a risk factor for venous thromboembolism due to increased renal losses of anticoagulant proteins included Anti-thrombin III and increased production of pro-thrombotic factors by

the liver. The possible occurrence of PE in a young person with nephrotic syndrome should not be despised. Early diagnosis and management of nephrotic syndrome may prevent the occurrence of venous thromboembolism(VTE).

Declarations

Consent to participate:

Written consent had obtained from the patients.

Competing of interest and Funding:

The authors declare that they have no Competingof interest and funding source.

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Figures

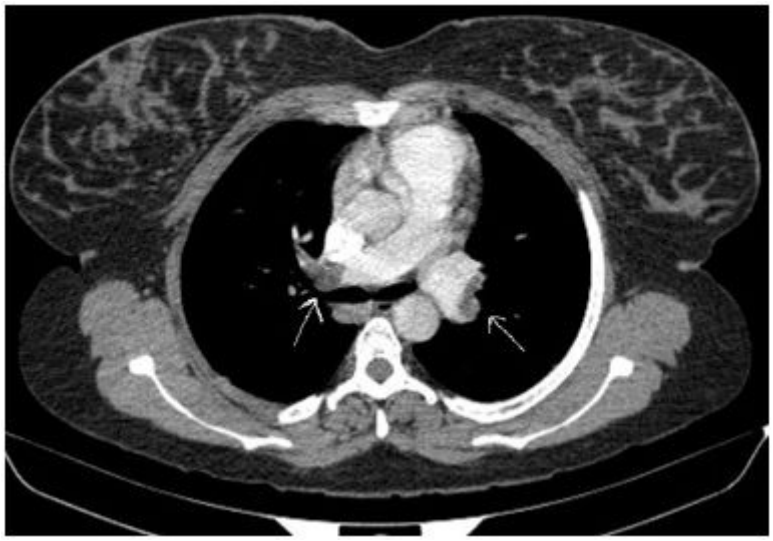

Figure 1

Computed tomography pulmonary angiography revealed Intraluminal filling defects representing emboli in the right and also left pulmonary arteries (PE).

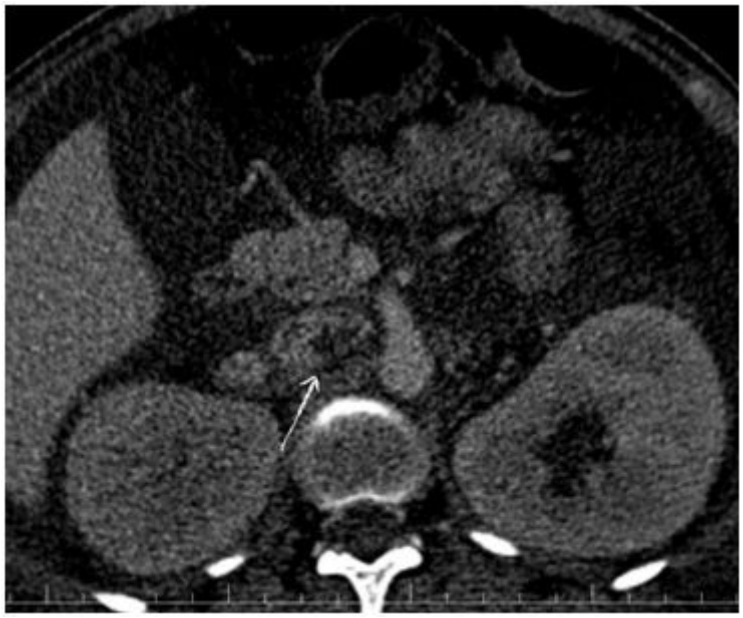

Figure 2

An abdominal CT scan showed thrombus in the inferior vena cava.

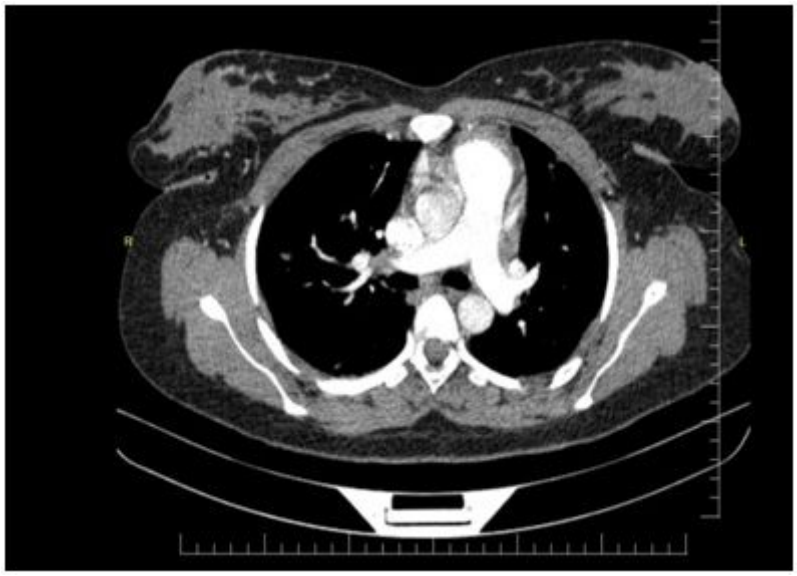

Figure 3

Computed tomography pulmonary angiography revealed normal pulmonary vessels with no thrombus or emboli.