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A Rare Presentation of Pulmonary Embolism Secondary to Nephrotic Syndrome

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Abstract: Nephrotic syndrome is a renal disorder characterized by excessive protein loss in urine, leading to hypoalbuminemia, hyperlipidemia, and edema. A significant yet rare complication of nephrotic syndrome is thromboembolism, particularly pulmonary embolism (PE). The hypercoagulable state associated with nephrotic syndrome results from multiple factors, including loss of anticoagulant proteins, increased procoagulant activity, and enhanced platelet aggregation. While venous thromboembolism (VTE) is a known risk, PE as the initial presentation of nephrotic syndrome is extremely uncommon. This case report describes a young male who developed PE as the first manifestation of nephrotic syndrome due to membranous nephropathy. The case underscores the importance of considering nephrotic syndrome in patients presenting with unprovoked PE. Early diagnosis through renal biopsy and appropriate management with anticoagulation and immunosuppressive therapy are crucial in preventing life-threatening complications.

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I. INTRODUCTION

Nephrotic syndrome is a disorder characterized by excessive urinary protein loss (proteinuria), reduced albumin levels (hypoalbuminemia), high lipid levels (hyperlipidemia), and generalized swelling (edema). This condition arises due to increased permeability of the glomerular filtration barrier, often resulting from underlying glomerular diseases such as membranous nephropathy, minimal change disease, or focal segmental glomerulosclerosis.

One of the serious yet underrecognized complications of nephrotic syndrome is thromboembolism, particularly pulmonary embolism (PE), which can be fatal. The hypercoagulability observed in nephrotic syndrome is attributed to several pathophysiological mechanisms, including:

- Loss of natural anticoagulants (e.g., antithrombin III) through the urine.
- Increased production of procoagulant factors by the liver.
- Enhanced platelet activity and increased blood viscosity.

While VTE is a known complication, its presentation as the first indication of nephrotic syndrome is exceptionally rare. This case highlights an unusual presentation of PE as the initial manifestation of nephrotic syndrome secondary to membranous nephropathy.

II. CASE REPORT

A 25-year-old male was admitted to the emergency department with acute shortness of breath lasting for one day. Additionally, he reported bilateral lower limb swelling and facial puffiness persisting over the past six months.

➢ *Medical History*

- No history of diabetes, hypertension, smoking, recent travel, surgery, or prolonged immobilization.
- No prior history of similar complaints or chronic medical conditions.

➢ *Clinical Examination*

- General Findings: Bilateral lower limb edema (Figure 1).
- Vital Signs:
- Heart Rate: 140 beats per minute (tachycardia).
- Blood Pressure: 120/70 mmHg.
- Respiratory Rate: 32 breaths per minute (tachypnea).
- Respiratory Examination: Decreased breath sounds over the right lung field.

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➢ *Investigations*
- Blood Tests:
- D-dimer: Elevated at 753, suggestive of thromboembolic activity.
- Imaging Studies:
- Chest X-ray: Revealed right-sided pleural effusion (Figure 2).
- 2D Echocardiography: Demonstrated a thrombus at the bifurcation of the pulmonary artery with severe pulmonary arterial hypertension (PAH).
- Urine Analysis:
- Significant proteinuria detected.
- Renal Biopsy:
- Confirmed membranous nephropathy with positive PLA2R (phospholipase A2 receptor) antibodies, establishing a diagnosis of nephrotic syndrome.

III. MANAGEMENT

The patient was hospitalized and managed with a combination of anticoagulation, diuretics, antihypertensives, and immunosuppressive therapy.

A. Acute Management

➢ *Anticoagulation Therapy:*
- Subcutaneous heparin (5000 IU, three times daily) to prevent further thromboembolism.

➢ *Antihypertensive and Diuretic Therapy:*
- Enalapril (2.5 mg once daily) for blood pressure control and proteinuria reduction.
- Intravenous furosemide (Lasix 20-10-0) for fluid management to alleviate edema and pulmonary congestion.

➢ *Immunosuppressive Therapy:*
- Pontesilli regimen initiated for nephrotic syndrome management.
- Intravenous methylprednisolone (500 mg for three days), followed by oral corticosteroids to modulate immune activity.

B. Hospital Course and Discharge Plan

The patient responded well to treatment, with notable improvement in dyspnea and edema. He was discharged on the following medications:
- Antihypertensives and Diuretics:
- MetXL (25 mg, 1-2-1) – Beta-blocker for blood pressure and heart rate regulation.
- Dytor (30 mg, 1-0-0) – Diuretic for continued edema management.
- Immunosuppressive Therapy:
- Ayukabam (5 mg, 0-1-0) – To maintain remission of nephrotic syndrome.
- Cardace (5 mg, 1-0-0) – ACE inhibitor to minimize proteinuria and support renal function.

The patient was advised to follow up regularly with nephrology and cardiology specialists to monitor renal function, proteinuria, and cardiovascular status.

IV. DISCUSSION

Pulmonary embolism is an uncommon but serious complication of nephrotic syndrome, arising from a hypercoagulable state induced by multiple mechanisms:

➢ *Pathophysiology*
- Increased liver synthesis of fibrinogen and coagulation factors.
- Urinary loss of antithrombin III and protein S, reducing anticoagulant capacity.
- Enhanced platelet activation and blood hyperviscosity.

➢ *Risk Factors*
- Among nephrotic syndromes, membranous nephropathy poses the highest risk for thromboembolism.
- Hypoalbuminemia (<2.0 g/dL) is strongly correlated with VTE incidence in nephrotic patients.

➢ *Unique Aspects of This Case*
- PE as the initial manifestation of nephrotic syndrome is exceedingly rare.
- PLA2R-positive membranous nephropathy was confirmed via renal biopsy.
- Highlights the need for early nephrotic syndrome evaluation in unprovoked VTE cases.

V. CONCLUSION

Pulmonary embolism as an initial presentation of nephrotic syndrome is rare yet potentially life-threatening. This case underscores the necessity of considering nephrotic syndrome in patients presenting with unexplained VTE, especially PE. Membranous nephropathy with positive PLA2R antibodies carries a significant thromboembolic risk, necessitating prompt diagnosis and treatment. Early intervention with anticoagulation and immunosuppressive therapy is essential for favorable outcomes.

➢ *Clinical Implications*
- Nephrotic syndrome should be considered in patients with unexplained thromboembolic events.
- Timely renal biopsy aids in accurate diagnosis and treatment planning.
- Lifelong anticoagulation may be required in cases with recurrent thromboembolism.

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