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Case Report

Nephrotic Syndrome and Cerebral Venous Sinus Thrombosis: A Case Report and Clinical Reminder

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ABSTRACT

Nephrotic syndrome is associated with a hypercoagulable state and increased risk of thromboembolic events, though cerebral venous sinus thrombosis (CVST) is rare. We report a 42-year-old female with features of nephrotic syndrome who presented with generalized seizures and altered sensorium. Magnetic resonance imaging and venography revealed extensive CVST with venous infarction. Laboratory evaluation showed significant proteinuria, hypoalbuminemia, and hyperhomocysteinemia, with negative autoimmune workup. The patient was managed with low molecular weight heparin and corticosteroids, followed by oral anticoagulation, resulting in favorable clinical recovery. This case underscores the importance of early recognition of neurological manifestations in nephrotic syndrome and prompt initiation of appropriate therapy to improve outcomes.

Keywords*: Case report, Cerebral infarction, Cerebral venous thrombosis, Magnetic resonance imaging, Nephrotic Syndrome.*

INTRODUCTION

Nephrotic syndrome is a well-recognized hypercoagulable state characterized by proteinuria, hypoalbuminemia, hyperlipidemia and edema. Thrombotic complications represent major contributors to morbidity and mortality affecting 27% adults with nephrotic syndrome [1]. While DVT, RVT and pulmonary embolism (PE) are the most reported thrombotic complications, CVST is very rare but potentially devastating thrombotic complication. Most common presenting symptoms are headache, nausea/ vomiting and altered mental status [2]. Despite its severity, CVST in nephrotic syndrome remains underrecognized.

Case Report/Case Presentation

A 42-year-old Caucasian female presented with complaints of generalized edema, worsening bilateral pedal edema, facial puffiness and abdominal distension for the past month. She did not have significant history of any medical illness including diabetes, hypertension or clotting disorders. No history of any major or minor surgeries. No family history of clotting disorders, and no history of any addictions or medication use such as contraceptives. On examination, vitals were normal (Pulse: 86 bpm, BP: 118/76 mmHg, Temperature: afebrile, SpO2: 99% on room Air) and general examination was revealed facial puffiness, abdominal distension, bilateral 3+ pitting pedal edema up to knees, otherwise unremarkable.

All routine investigations with complete blood count (CBC), comprehensive metabolic panel (CMP), thyroid profile, lipid profile, urine routine and microscopy were done, which revealed normal CBC, Creatinine:0.76mg/dL (normal range:0.5-1.2mg/dl), total cholesterol: 252.4 mg% (normal <200mg %), LDLcholesterol:178.8mg% (normal <100mg%), TSH:5.88 microIU/mL (normal range: 0.27-4.2 microIU/L), random blood sugar: 72.7mg/dL (normal <140 mg/dl). Urinalysis revealed frothy appearance and 4+ albumin. Urine protein-creatinine ratio (U-PCR) was elevated at 2103 mg/g

(normal range: 0-200mg/g).

Radiological imaging with abdominal ultrasound showed a hemangioma in segment three of the right lobe with prominent IVC and normal bilateral kidney size. Chest radiograph and ECG were unremarkable. Transthoracic echocardiogram revealed mildly reduced ejection fraction of 45%. Nephrology was consulted and patient was treated with Prednisolone 30mg, Torsemide- Spironolactone 10/25 mg, Losartan 25mg, Atorvastatin 40mg. Renal biopsy was planned in the next follow-up. Patient reported improvement in her symptoms but before the renal biopsy was done patient developed generalized tonic-clonic seizures that sustained for three minutes with, frothing from mouth and altered sensorium.

On presentation to ER, patient was hemodynamically stable (Heart rate of 78 bpm, BP 140/80 mm of Hg, respiratory rate of 18/min, O2 saturation of 98% on room air). Patient was drowsy on examination with Glasgow coma scale (GCS) of 13 (E3V4M6). Neurological examination revealed bilateral round and reactive pupil with normal eye movements, decreased tone in all extremities, deep tendon reflexes of +1 and extensor plantar reflex in right lower limb and flexor response in the left lower limb. There were no signs of meningeal irritation. Fundus examination revealed bilateral papilledema. The patient was admitted to ICU and MRI brain showed acute venous non- hemorrhagic infarct involving the left frontal region and prominent cortical vascular channels. Following which MR venogram was performed which demonstrated of acute cerebral venous sinus thrombosis in the bilateral transverse sinus, left sigmoid sinus, straight sinus and superior sagittal sinus (shown in fig. 1, 2).

Initial labs revealed hypoalbuminemia 2.5 gm/dl (normal range: 3.5-5.5gm/dl), hyperlipidemia LDL:108.2mg/dL (normal range:<100mg/dl) with hyperhomocystinemia 20.98 micromoles/L (normal: <15 micromoles/L), U-PCR 2153 mg/g, urine microalbumin-creatinine ratio (U-ACR) 2545mg/g (>300mg/g suggestive of macroalbuminuria). Auto-immune workup with Anti-nuclear antibody, Anti-cardiolipin antibody, Anti-phospholipid antibody, Anti-Beta-2-glycoprotein antibody, Lupus anti-anticoagulant was negative. Complement 3 (C3) level was 1.6 (Norma:0.9-1.8).

Patient was treated with Low molecular weight heparin (LMWH) and IV corticosteroid in the ICU. Patient's clinical status steadily improved with normal neurological examination on third day of treatment. In view of active treatment of CVST with anticoagulation, a renal biopsy was deferred for later time. On day-5 patient remained neurologically stable and discharged on oral Prednisone and Apixaban therapy. Patient was followed up at 1 month and remained stable, currently pending Renal biopsy to confirm the etiology of Nephrotic syndrome.

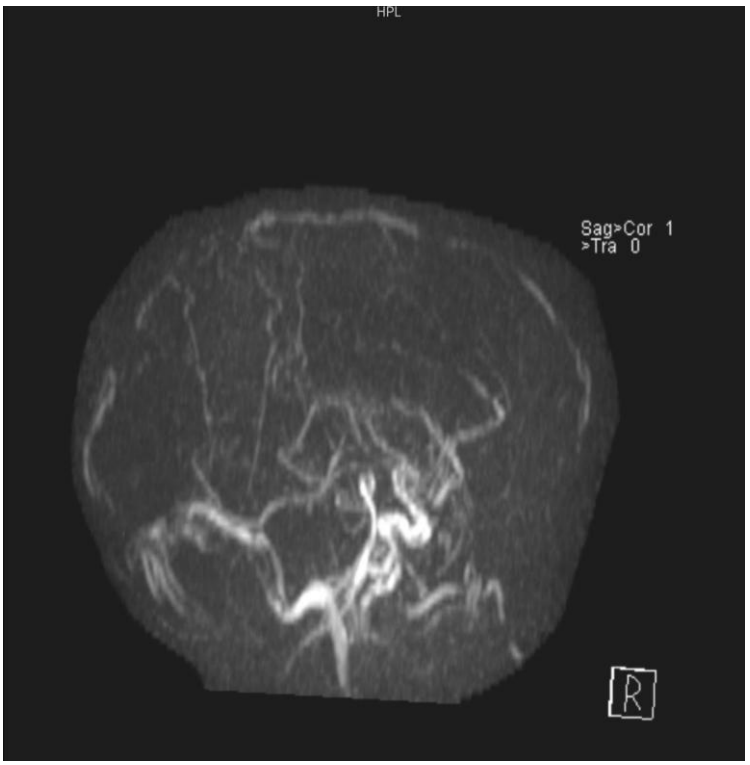

Figure 1: Oblique coronal view of MR Venogram showing absence of superior sagittal sinus

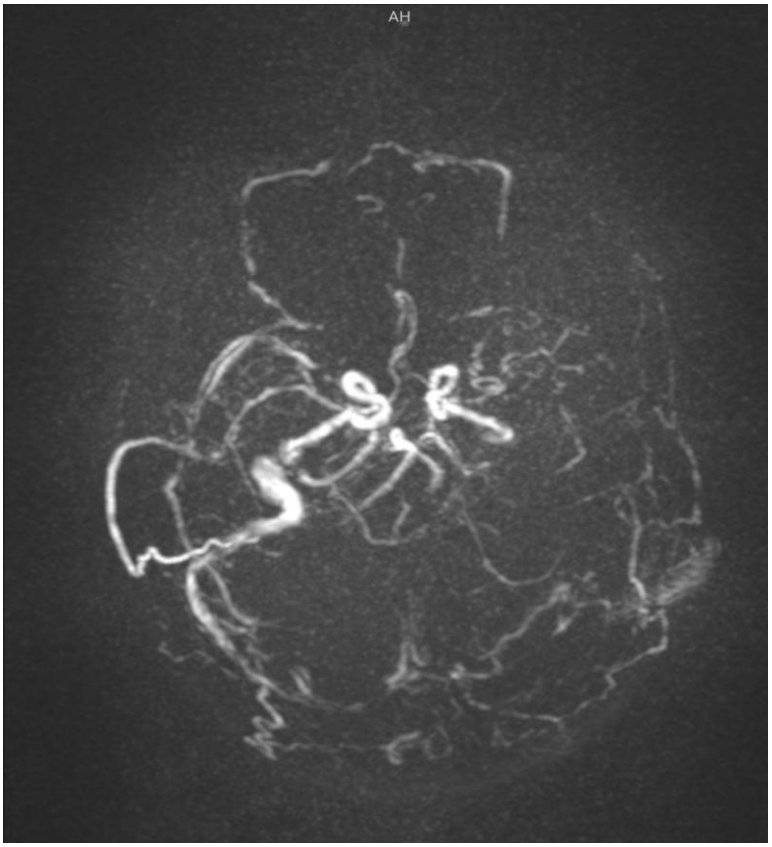

Figure 2: Axial view of MR Venogram demonstrating absence of bilateral transverse, left sigmoid and superior sagittal sinus

DISCUSSION

This case highlights extensive CVST, a rare but potential complication of Nephrotic syndrome. Neurological symptoms, especially seizures, altered sensorium, headache and vomiting in patient with nephrotic syndrome should raise suspicion of cerebral sinus thrombosis. This could be venous or arterial, although venous is more common. [3,4]

Hypercoagulability is a well-described feature of nephrotic syndrome which is manifested as thrombosis commonly of deep veins, renal veins and pulmonary veins. Pathophysiology is multifactorial including, with mechanisms such as: (a) Urinary loss of anticoagulant proteins - particularly antithrombin III, protein C, and protein S, combined with (b) hepatic overproduction of procoagulant factors (fibrinogen, factors V, VII, VIII, IX, X, and XIII) which shifts the hemostatic balance toward thrombosis. [5,6,7] Venous thromboembolism complicates up to 40% of adult cases, with the highest risk occurring within the first 6 months of diagnosis. [5,1]

Heavy proteinuria and hypoalbuminemia, as seen in our patient, significantly increase the risk of thrombosis. Patients with albumin levels less than 2.8g/dL have a 2.5 times increased risk of developing thromboembolic events. [3]
Compared with reported CVST cases in Nephrotic Syndrome, our patient shares hallmark features such as seizures, headache, and papilledema, but lacks risk factors like oral contraceptive use. Early MRI/MRV imaging was crucial in making timely diagnosis. Treatment of CVST in the setting of nephrotic syndrome includes the use of corticosteroids and anticoagulation.[3] Anticoagulation remains the mainstay of treatment and is the first line of treatment for Cerebral Venous Sinus Thrombosis. [3,4] Our Patient was initially started on Low molecular weight heparin (LMWH) and IV corticosteroids, later after stabilization switched to direct oral anticoagulant - Apixaban.
In our review of literature, there are very few reported cases of CVST in the setting of Nephrotic Syndrome in an adult.

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