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Case Report *Volume 5 Issue 1*

A Case of Cerebral Venous Thrombosis in Nephrotic Syndrome and Challenges in Management

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Abstract

Cerebral venous thrombosis (CVT) is a rare type of disorder caused by a blood clot in the cerebral venous sinuses or cortical veins, disrupting normal blood flow from the brain. Symptoms can include severe headaches, seizures, and focal neurological deficits. Risk factors for CVT include hormonal changes, genetic conditions, infections, and head trauma. Diagnosis is primarily made through MRI and MR venography, which identify thrombi and associated brain damage. Treatment typically involves anticoagulation therapy to dissolve clots and prevent recurrence. Early detection and management are crucial for improving outcomes and reducing the risk of severe complications. CVT can be a rare complication in Nephrotic syndrome without any other procoagulant state and can be fatal if not recognized and treated promptly.

Keywords: Cerebral Venous Thrombosis; Procoagulant; Nephrotic Syndrome; Long-Term Therapy; Optimal Patient

Abbreviations

CVT: Cerebral venous thrombosis; LMWH: Low Molecular Weight Heparin; DOACs: Direct Oral Anticoagulants.

Case Report

We present a case of a 67-year-old man with a history of nephrotic syndrome, who arrived at the emergency room complaining of sudden lightheadedness and generalized weakness. Until then, he had been asymptomatic and worked as a watchman in a factory [1,2]. Following his usual routine of showering after work, he experienced dizziness, fatigue, numbness, and weakness in his left upper limb.

The patient has been managing nephrotic syndrome with oral prednisolone for the past six months and has a history of varicose veins. He was also treated for cellulitis six months ago. Upon arrival at our ER, his vital signs were stable with afebrile temperature, blood pressure of 100/80 mmHg, respiratory rate of 24 breaths per minute, pulse rate of 98 bpm, and oxygen saturation of 97%. His EKG showed normal sinus rhythm, and clinical examination revealed him to be conscious, oriented, and alert with intact cognitive functions (GCS - E4V5M6) and full power in all limbs. He promptly sought medical attention at a nearby hospital, where a CT scan revealed an intraparenchymal bleed. He was subsequently transferred to our facility for further evaluation and management [3].

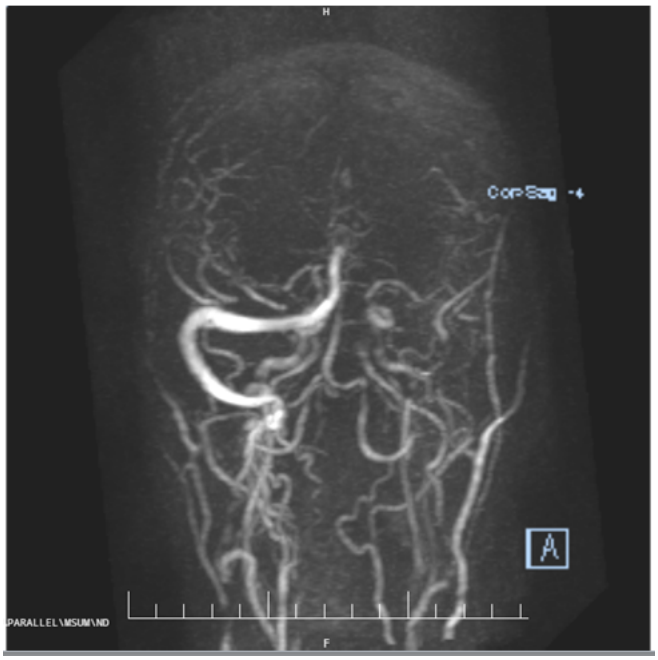

Figure 1: MRV showing occluded Venous sinuses.

There was no history of trauma, recent surgical procedures, hypertension or history to suggest dehydration. An MRI of the brain identified a venous hemorrhagic infarct in the left parietal lobe, along with dural venous sinus thrombosis affecting the superior sagittal sinus, left transverse and sigmoid sinuses. Additionally, moderate to severe atherosclerotic luminal narrowing was observed in the proximal C2 segment of the left internal carotid artery, with mild narrowing in the C5 and C6 segments of bilateral internal carotid arteries, and mild to moderate narrowing in the right posterior cerebral artery (P2 segment) and left middle cerebral artery (M1 segment).

The patient underwent a cardiology consultation that confirmed good left ventricular function on echocardiography. Abdominal sonography showed minimal perinephric fluid with no significant abnormalities in the kidney, ureter, or bladder.

Admitted to the neurology ICU, the patient was closely monitored for 24 hours and received treatment with LMWH and antiplatelet therapy. Nephrology consultation recommended continuing oral prednisolone. Histopathology of his kidney revealed focal segmental glomerulosclerosis. A comprehensive prothrombotic workup, including tests for fibrinogen, homocysteine, ANA, IgG and IgM phospholipid antibodies, protein C, and factor Leiden mutation, returned normal results. However, decreased functional activity of protein S was noted which on repeat examination became normal.

Discussion

Nephrotic syndrome, a kidney disorder marked by significant proteinuria, hypoalbuminemia, and edema, significantly increases the risk of developing CVT due to its associated hypercoagulable state. Nephrotic syndrome leads to a state of hypercoagulability, which heightens the risk of thrombotic events including CVT. This hypercoagulable state is primarily driven by the loss of anticoagulant proteins such as antithrombin III and protein C through the urine, alongside increased synthesis of procoagulant factors. Proteinuria reduces serum albumin levels, disrupting the balance between procoagulant and anticoagulant factors and favoring thrombus formation. Additionally, the increased blood viscosity and altered platelet function further contribute to the development of CVT in these patients. The following principles may be followed in managing such cases [4].

In the acute management of conditions such as thrombosis, anticoagulation therapy plays a crucial role. Initially, intravenous heparin, either unfractionated or low molecular weight like enoxaparin, is administered to manage acute thrombosis. Once the patient is stabilized, the therapy is transitioned to oral anticoagulants, such as warfarin or direct oral anticoagulants (DOACs) like rivaroxaban or dabigatran, based on patient characteristics and clinical guidelines. The duration of anticoagulation typically spans 6-12 months, depending on individual risk factors and therapeutic response. In severe cases, where life-threatening complications such as extensive cerebral venous sinus thrombosis or significant neurological deterioration occur, thrombolysis may be considered. This may involve the use of intravenous thrombolytics like rtPA, though this is less common due to bleeding risks.

For managing nephrotic syndrome, the treatment focuses on addressing proteinuria with medications like corticosteroids (prednisone), immunosuppressants (cyclophosphamide, calcineurin inhibitors), and ACE inhibitors or ARBs. In cases of severe hypoalbuminemia, intravenous albumin is administered to alleviate significant symptoms. Edema is controlled using diuretics, with careful monitoring of electrolytes and renal function to ensure appropriate dosage adjustments. Symptom management involves using anticonvulsants such as levetiracetam or valproic acid for seizure control, and analgesics or pain management strategies, including NSAIDs or acetaminophen, for headache relief. Regular imaging is recommended for follow-up to assess thrombus resolution and detect potential complications. Ongoing anticoagulation therapy should continue as prescribed, with regular INR monitoring for

warfarin or periodic assessment for DOACs. In managing nephrotic syndrome, long-term therapy is necessary to maintain remission and prevent relapse, requiring regular monitoring of proteinuria, renal function, and albumin levels. Patient education is also vital, emphasizing the importance of adherence to both anticoagulant therapy and nephrotic syndrome management, while discussing potential complications and the need for regular follow-up.

Conclusion

Managing cerebral venous thrombosis in the setting of nephrotic syndrome is complex and requires a comprehensive approach. It involves acute management of the thrombotic event, addressing the underlying nephrotic syndrome, and ongoing monitoring to prevent recurrence. Effective treatment hinges on a multidisciplinary approach, early diagnosis, and vigilant long-term management to ensure optimal patient outcomes.

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