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# A case of Cerebral venous thrombosis in a patient with Acute post infectious glomerulonephritis

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**Abstract**

Cerebral venous thrombosis is generally seen in a setup of Nephrotic syndrome. However, a division of Nephritic syndrome - acute glomerulonephritis is not reported to increase the risk for the same. Focal neurological manifestations, motor weakness and hemiparesis are seen in half the patients. And Seizures are seen in more than 40% patients. Volume depletion along with Nephritic state, which is not usually associative with hypercoagulable state, is responsible for development of Cerebral venous thrombosis.

**Keywords:** Cerebral, Nephritic Syndrome, Biopsy

## Introduction

Cerebral venous thrombosis is a relatively uncommon neurological disorder, with thrombus formation in dural sinus or cerebral veins. It occurs in about 5 in 1 million of population.[1] Cerebral venous thrombosis is generally seen in a setup of Nephrotic syndrome. However, a division of Nephritic syndrome - acute glomerulonephritis is not reported to increase the risk for the same.[2] Here we present a case of a young girl with Cerebral venous thrombosis with acute post infectious glomerulonephritis.

## Case

A 14 year old girl was admitted in the General medicine department, who presented with vomiting, decreased & high colored urine. On past history, she had fever with cough 10 days back, for which treatment was taken. On general examination, the girl appeared edematous and her Blood pressure was 150/105mmHg.

On laboratory examination, her urine revealed dysmorphic RBC and moreover her urine protein was 3+, Haemoglobin was normal, ESR was raised, reduced total protein and serum albumin. C3 and C4 was 45 and 25 mg/dl respectively. Her ASO titer was 290 IU/dl. USG whole abdomen with pelvis, did not reveal any significant abnormalities.

Initially, she was treated with Diuretics and anti-hypertensive drugs, after which her Blood pressure and urine protein returned to normal limits. After two days, she was started on Inj METHYL PREDNISOLONE 750mg/day for 4 days followed by, oral PREDNISOLONE 1mg/kg.

On day 7, she developed Generalized tonic clinic seizures, with altered sensorium & Left hemi paresis. CT brain was done, which showed theombosed cortical veins and Ct venogram showed delta sign, right transverse sinus thrombosis and internal jugular vein thrombosis. Renal biopsy was done, showing glomerulus with increased endocapillary thickness and neutrophil infiltration. Immunofluorescence showing strong diffuse granular coarse deposits of IgG and C3c. Biopsy was suggestive of APIGN.

We continued steroid treatment, hoping to restore renal function. The girl was discharged with oral PREDNISOLONE 40mg per day along with Anti epileptics.

**Discussion**

Cerebral venous thrombosis is a rare condition which occurs due to the clotting of blood in cerebral veins and dural venous sinus.[3] This is more common in women than in men, and seen in about 3 to 4 cases per 1 Lakh post partum cases, and more commonly now seen in young women of reproductive age.[4]

There are two main mechanisms understood behind CVT. Firstly, venous and capillary pressure rises due to thrombus formation, leading to reduced perfusion.[5] This leads to ischemia and intracellular edema. This accountable increase in pressure along with disturbance of blood brain barrier, finally leads to hemorrhage of parenchyma.[6] Secondly, partial thrombus resolving or the thrombus itself stands as a barrier to CSF absorption, which raises the Intracranial pressure and leading to parenchymal damage and haemorrhage.[7]

Focal neurological manifestations, motor weakness and hemiparesis are seen in half the patients. And Seizures are seen in more than 40% patients.[8]

Nephrotic syndrome has direct association with venous thrombosis due to increasing the coagulant factors V, VIII, X,[9] and decrease in the anti thrombotic factors such as protein C, protein S and anti thrombin III.[10] This hypercoagulable state is in direct association with the etiology of Cerebral venous thrombosis. However, Acute post infectious glomerulonephritis does not have a well known association. Our patient's reports clearly showed hypo albuminemia and elevated cholesterol and volume depletion,[11] along with these, high dose corticosteroids could have been a precipitating factor for CVT.[12] Also the patient could have had already existing thrombophilia, aggregating her condition.[13]

**Conclusion**

Hereby, we conclude that volume depletion along with Nephritic state, which is not usually associative with hypercoagulable state, is responsible for development of Cerebral venous thrombosis. It is very significant to keep this in mind, because under diagnosis or if left untreated, potentially leads to tremendous complications.

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