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A Rare Case of Nephrotic Syndrome Complicating to Cerebrovascular Accident

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This is a rare case of membranous nephropathy variant of nephrotic syndrome presenting with hemiplegia which turns out to be an arterial thrombosis. This patient developed arterial thrombosis-induced stroke in nephrotic syndrome which is rare as the majority of stroke in nephrotic syndrome is of venous origin.

Introduction

Cases have been reported that present chiefly as proteinuria later developing stroke or stroke further evaluated as proteinuria and nephrotic syndrome. Out of all types of nephrotic syndrome membranous nephropathy accounts for 40 % of patients developing ischemic stroke. Nephrotic syndrome complicating with ischemic stroke is very rare and chiefly presents as arterial thrombosis. Thromboembolic phenomena in nephrotic patients are postulated to be a result of the urinary loss of antithrombotic factors by affected kidneys and increased production of prothrombotic factors by the liver.

A 19 years old male who is a college student, presented with complaints of weakness in the right upper and lower limbs, swelling of both legs facial puffiness and reduced urine output for the 7 days. The weakness started after day 2 of swelling of the legs and puffiness of the face. There are no sensory disturbances. He has been evaluated for bilateral pitting pedal oedema 5 years ago but the treatment was not continuous and records were not available in general examination there was pitting pedaloedemaa more on the right side extending up to the knee.

Central nervous system examination revealed reduced power of 4/5 in both right upper and lower Limbs the plantar on the right side was extensor and the deep reflexes were brisk on the right side. The sensory system, cerebellar and autonomic functions were intact. The spine and cranium were normal.

Investigations

His complete hemogram was normal, liver and renal parameters were within normal limits, and viral markers were non-reactive. His urine albumin showed 3+; protein to creatinine ratio was 2.2:1. 24 hrs urine protein 6 gms/ day. His lipid profile was above the normal limits, where cholesterol – 285mg/dl, triglycerides – 249mg/dl, HDL – 55mg/dl, and LDL- 96mg/dl. USG abdomen revealed cortical Echoes- altered; echocardiogram was normal. Venous Doppler of both lower Limbs was done and there was no evidence of any DVT. MRI brain with MRA showed acute infarct in the left gangliocapsular region and centrum semiovale region, MRV was normal. A renal biopsy of the native kidney showed membranous nephropathy.

Management

The patient is under a modified PONTICELLI regimen right now at the fourth month of cyclophosphamide after 2 months of Steroids with one month of cyclophosphamide in between. The patient has now improved with anticoagulants, statins, physiotherapy and regular follow-up.

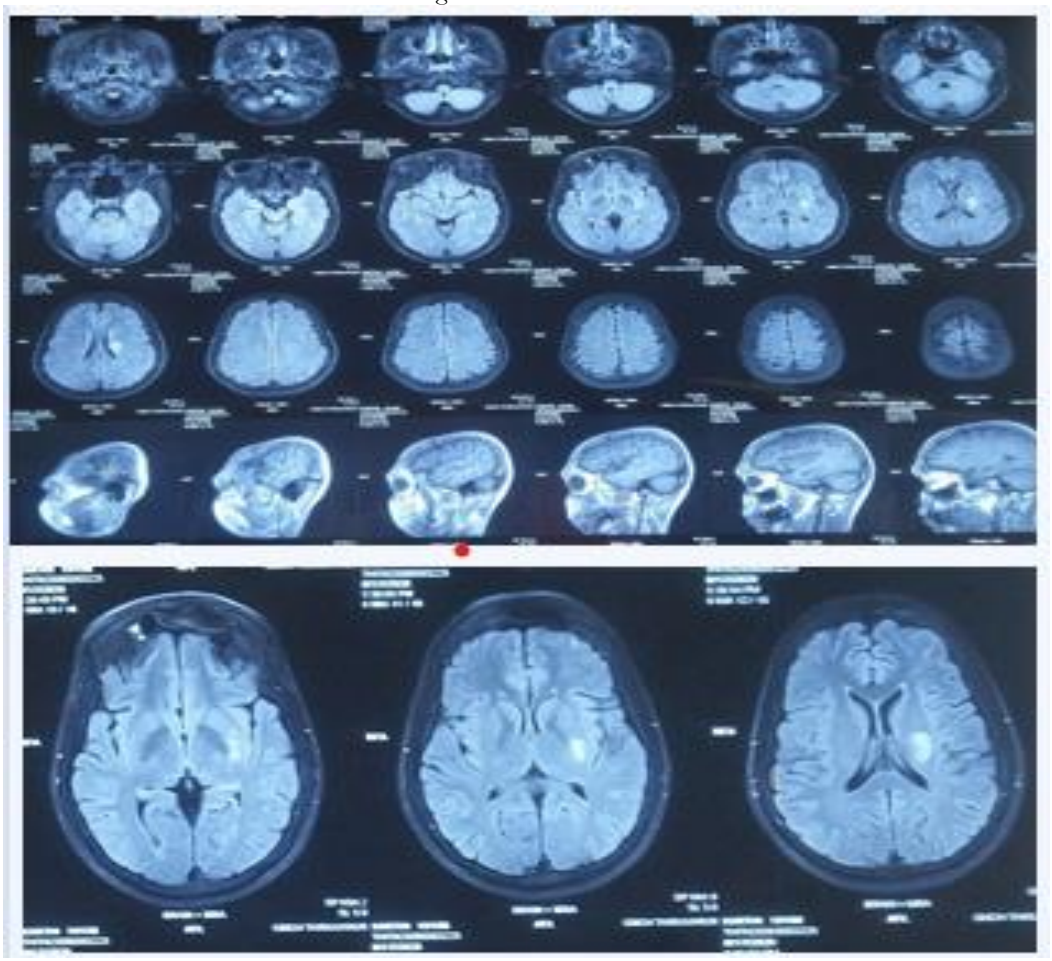
Discussion

Nephrotic Syndrome (NS) is defined by the presence of a nephrotic range of proteinuria, peripheral oedema, hypoalbuminemia, and hyperlipidemia, and has an increased risk of venous thromboembolism (VTE). It is classified as either primary or secondary. Membranous nephropathy (MN), minimal change nephropathy, and focal segmental glomerulosclerosis are the primary causes. Secondary causes include NS related to systemic disease and NS related to medication use. NS related to systemic disease includes diseases such as diabetes mellitus, systemic lupus erythematosus, multiple myeloma, and malignancy. The most common medications implicated in NS are nonsteroidal anti-inflammatory drugs.

The exact incidence and prevalence of VTE in NS remain unknown. Varying rates are reported in different studies. The variance in reported incidence and prevalence rates has been attributed to the limited data available, perhaps from underdiagnosis in this patient population.

The cause of the hypercoagulable state in patients with NS is not well-understood. In NS, damage to the glomerular membrane results in increased filtration of small proteins such as antithrombin III, plasminogen, protein C, and protein S, and this in turn leads to increased coagulability. In patients with NS, the loss of albumin and resultant hypoalbuminemia results in increased hepatic synthesis of fibrinogen which also favours the formation of thrombus. The

Figures and Illustrations



tendency of thrombus formation in the renal vein in NS may in part be due to the loss of fluid across the glomerulus. The resulting hemoconcentration in the post-glomerular circulation may promote thrombus formation in patients who are already hypercoagulable. Thromboembolism in NS can be venous or arterial; however, the incidence of arterial thrombosis is much lower compared to venous. Arterial thrombosis, though less common, can be serious and life-threatening depending on the vessels involved. Involved arterial sites include aortic, renal, femoral, mesenteric, coronary, and cerebral arteries. The most common site of arterial thrombosis is the femoral artery, occurring mainly in children with NS.

Conclusion

This case denotes the complication of nephrotic syndrome and atherosclerotic changes with high lipid profile, particularly venous thrombosis happens more commonly than arterial thrombosis. In this case, the venogram was normal. This is a reversible condition which should be identified earlier and treated.

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