AN INTERESTING CASE OF CEREBRAL INFARCTION AS A COMPLICATION OF NEPHROTIC SYNDROME- A CASE REPORT

ESHWARAPPA
Department of General Medicine, COIMBATORE MEDICAL COLLEGE

Abstract: Arterial thrombosis is a rare complication in a patient with nephrotic syndrome compared with venous thrombosis. The nephrotic syndrome as a hypercoagulable state is clearly predispose to increased risk of spontaneous thrombosis and embolism. In adults, most thrombosis are venous, while in children arterial thromboses are more common. Here we report a rare case of a 43 yr-old man with cerebral arterial infarction associated with nephrotic syndrome due to membranous nephropathy.

Keyword: Nephrotic syndrome, Hypercoagulabe state, Arterial thrombosis, membranous nephropathy

INTRODUCTION:
The nephrotic syndrome is a constellation of renal and extrarenal manifestations that can be caused by systemic diseases as well as by primary insults to the kidney. The nephrotic syndrome is classically characterized by proteinuria exceeding 3.5 g/24 hr, hyperlipidemia, edema, hypoalbuminemia. Hypercoagulability is also a well-known complication of the nephrotic syndrome which leads to increased risk of spontaneous thrombosis and embolism of which venous thrombosis is most common and arterial thrombosis is rare in adults.

CASE SUMMARY:
A 43 yr old male, chronic smoker and alcoholic, not a diabetic/ hypertensive or IHD patient, admitted with H/O progressive swelling of both legs, face and abdomen of 2 months duration and H/o Weakness of left sided limbs, which was sudden in onset of 2 days duration without any history of vomiting, convulsions, loss of consciousness, head injury, palpitation, chest pain, oliguria or haematuria.

On examination, Patient’s higher mental functions were normal except Dysarthria. He had bilateral pedal edema and facial puffiness. His vital signs were normal including jugular venous pressure. On Neurological examination, Left sided upper motor neuron facial palsy and spastic hemiparesis of left sided limbs noted. Abdomen examination shows free fluid without any organomegaly. Cardiovascular and Respiratory system were normal clinically. After investigating his complete haemogram was normal, urine routine was showing Albumin-3 +, RBC-25 cells/hpf, no casts, 24hr urine protein-1.4gm.

Blood urea, Serum creatinine, Serum electrolytes, blood sugar lipid profile and ECG were within normal limits. Liver function test was showing Serum bilirubin-0.4 mg/dl; SGOT-24 IU/L, SGPT-22 IU/L, Serum proteins- Total-3.2gm%, Albumin-1.7gm%, Globulin-1.5gm/dl. Fibrinogen level was 760mg/dl. X-Ray chest PA view was showing mild pleural effusion left side with increased vascular markings (figure 1).

Ultrasonography abdomen was showing bilateral mild pleural effusion, both kidneys mild increase cortical echoes and moderate ascitis. Renal biopsy was done and histopathological examination was showing membranous nephropathy. CT brain was showing acute infarct seen in right temporal lobe-right MCA territory (figure 2).

MRI brain was showing acute infarct in the right parieto temporal region (figure 3).

figure 1

figure 2

figure 3
MRA brain was showing occlusion of the right middle cerebral artery (figure4).

**figure 4**

FINAL DIAGNOSIS:-Membranous nephropathy/ Ischemic stroke/ right middle cerebral artery infarct. Patient was treated with steroids, antiplatelets and anticoagulants.

**DISCUSSION:**

The nephrotic syndrome is characterised by proteinuria, edema, hyperlipidemia, lipiduria, hypoalbuminemia, and hypercoagulability. Common causes of nephritic syndrome are Minimal change disease, Focal segmental glomerulosclerosis, Membranous glomerulopathy, Membranoproliferative glomerulone phritis. Membranous glomerulopathy is one of the most common primary renal causes of the nephrotic syndrome, accounting for 30% to 40% of all cases in adults. The hypercoagulable state associated with the nephrotic syndrome is caused by an increased urinary loss of antithrombin III, altered activity and levels of proteins C and S, increased hepatic synthesis of fibrinogen, and increased platelet aggregation. Clearly, these conditions predispose patients to an increased risk of spontaneous thrombosis and embolism. In adults, most thromboses are venous, while in children arterial thromboses are more common. Renal vein thrombosis is present in approximately 30% of patients with the nephrotic syndrome, and the rate is highest in patients with membranous glomerulopathy. Although arterial thrombosis has rarely been reported in adults, it is a potentially serious problem in nephrotic patients. Although its accurate pathogenesis remains unclear, hypercoagulability is regarded as a major factor for thrombosis in nephrotic patients.

Our patient found to have an elevated level of fibrinogen and this may be responsible for his hypercoagulable state which led to cerebral artery infarction.

**References :**