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AN UNCOMMON POSSIBILITY: ATYPICAL VARIANT OF GUILLAIN BARRE SYNDROME IN CHRONIC KIDNEY DISEASE PATIENT .

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ABSTRACT Patient with Chronic Kidney Disease (CKD) are frequently affected with neurological complications. These complications can potentially affect the central and peripheral nervous system . Common neurological complications in Chronic Kidney Disease include stroke, cognitive dysfunction, encephalopathy, peripheral and autonomic neuropathies(1) .These conditions have significant impact on patient's morbidity. Acute Inflammatory Demylinating Polyneuropathy (AIDP) is caused by dysregulated immune response following an infectious or non-infectious event .Here we present a case of atypical GBS in CKD patient ,which is very rare .

KEYWORDS :

INTRODUCTION

Atypical GBS ,a variant of AIDP is a rare condition to be seen in CKD patients .Common manifestations of AIDP includes progressive weakness that usually begins in the feet before involving all four limbs. At presentation 60% of patients have weakness in all 4 limbs. weakness plateaus at 2 weeks after onset in 50 % of patient and by 4 weeks in over 90% of patients, areflexia, sensory disturbances and opthalmoparesis and in severe cases diaphragmatic weakness and bulbar dysfunction requiring mechanical ventilation (1,2).The atypical presentation group of GBS was characterized by localized or regional involvement of the motor and sensory axons of the peripheral nerves and the autonomic nervous system ,atypical finding such as preserved or exaggerated reflexes.

Case Presentation

A 63 years old male with a background history of type 2 diabetes mellitus for 20 years ,hypertension for 15 years ,CKD detected 2 years back ,when he was evaluated for decreased urine output ,hesitancy , generalized weakness.His creatinine at that time was 2 mg/dl .Subsequently ,in July 2023 he was diagnosed to have BEP (Benign Prostatic Enlargement)and phimosis ,he underwent circumcision ,TURP .In September 2023,he had decreased urine output along with anorexia ,weakness, vomiting for which he was admitted in nearby hospital in Dimapur and was started on twice weekly maintainence Hemodialysis. As patient was becoming weak day by day and was bed bound ,he was bought to our hospital for further management .On examination ,he was drowsy , arousable on verbal commands with history of acute onset progressive weakness in all 4 limbs with difficulty in speaking and swallowing for 4 days .No history of loss of consciousness, seizure, bowel incontinence ,extra ocular movement – full ,Bilateral lower motor neuron type facial weakness seen , dysarthia was present . Motor system examination showed muscle power decreased in B/L upper and lower limbs . Deep tendon reflexes – B/L ankle ,knee ,bisceps ,supinator, triceps – well preserved ,plantar reflex – B/L normal flexor .Sensory system examination revealed no abnormality ,no sensory level . In view of progressive quadriparesis (Lower limb>>upper limb)with LMN bulbar weakness with retained reflexes differential diagnosis of atypical GBS verses brainstem pathology was made and Nerve Conduction Study(NCV), MRI Brain and CSF study done .MRI brain showed white matter hypodensities , NCV showed sensory motor neuropathy axonal – polyneuropathy . CSF study showed total leucocyte count – 5, neutrophil -80 %, lymphocyte - 20 % , glucose -83 ,protein -102.8 g/dl . Neurology expert opinion taken and a diagnosis of atypical GBS was made for normal leucocyte with raised protein (albumino-cytological dissociation)and IVIg was started for a duration of 5 days at the rate of 20 grams/day .Patient weakness improved gradually ,he was able to take orally ,speech became better .Patient was from remote place in Dimapur with no Hemodialysis access nearby ,hence he was initiated on CAPD .Patient tolerated CAPD exchanges well and was discharged with advice for continuing CAPD -2 exchanges /day with fluid strength (1.5 % - 1.5 %) ,dwell volume – 2 litres , dwell time -4 hours ,with good residual urine output, speech therapy and physiotherapy to continue at home .

DISCUSSION

Peripheral neuropathy (PN) is one of the most common neurological complication of CKD and is also particularly prevalent in adults with diabetes. One of the features of large-fiber peripheral polyneuropathy, which occurs through pain, sensory loss, and muscle weakness, especially expressed in foot complications, is decreased tactile sensation [1].Motor weakness with preservation of reflexes is a characteristic feature of the acute motor axonal neuropathy (AMAN) subtype of GBS . Extent of renal impaired function was found to play a key role in the progression of neuropathy, with the glomerular filtration rate falls below 12 mL/min reported concomitant clinically significant neuropathy [2]. 60–90% of dialysis patients of whom have long been considered to have advanced CKD suffer from PN [3, 4]. Recently, the presence of PN in early CKD has been demonstrated in both whole population and CKD population studies [5□8]. Signs and symptoms of uremic neuropathy include distal sensory loss to pin prick vibration as well as reduced or absent tendon reflexes in the lower limbs .Autonomic neuropathy often accompanies peripheral neuropathy but presents with distinct cardiovascular features that are clearly identifiable . As such ,similar symptoms with an acute onset or rapid progression may indicate a different causes such as GBS or vasculitic neuropathy or mimic disorders such as myelinosis and polyradiculopathies requiring referral for specialist neurological diagnosis .

Our case 63 years old male ,diagnosed ESRD presented with progressive ascending lower limb weakness, dysarthria ,decreased swallowing reflex ,preserved tendon reflexes ,flexor plantar response .MRI brain showing diffuse white matter changes and NCS showing sensory ,motor neuropathy (axonal and demyelinating polyneuropathy). CSF showed albumin-cytological dissociation and a diagnosis of atypical GBS was made .patient received IVIg for 5 days, he responded to the treatment ,dysarthria,weakness and swallowing reflex improved.

CONCLUSION

AMAN variant of GBS in CKD is a rare presentation .No case report has been seen till date .Though a rarer presentation in ESRD patients , however it should always be kept in mind while examining patients with acute onset quadriparesis /weakness with atypical features .

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