Very rare case report of multiple sclerosis in a case of chronic kidney disease

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Multiple sclerosis is a neurological disease, which affects young adults. Its course is unpredictable and runs over decades. It is considered as disease of unknown etiology in which there is demyelination of the brain and spinal cord.

There is one case report of membranous GN in a case of progressive multiple sclerosis in the extant world literature 24. We report here perhaps the first case in the world of the occurrence of multiple sclerosis in a known case of chronic kidney disease with the presentation of acute optic neuritis, having a characteristics picture on MRI of multiple sclerosis in brain, abnormal VEP, oligoclonal bands in CSF and a prior episode of acute optic neuritis 3 years back. In our opinion, this case opens the flood gates for research and work to investigate whether the oxidative stress and the smouldering proinflammatory state in CKD can precipitate multiple sclerosis in a genetically predisposed host in a given environment. Multiple Sclerosis is often associated with a neurogenic bladder, but renal function is generally considered to be normal.

Key Words: *Multiple Sclerosis, Chronic Kidney Disease, methylprednisolone*

INTRODUCTION

Multiple Sclerosis (MS) is a rare demyelinating disorder of the central nervous system. It is a complex human autoimmune- type disease with predominantly unknown etiology [1,3]. The prevalence of MS disease ranges from 30 to 100 cases per 100000 individuals [4]. MS, like other presumed autoimmune diseases, is more common in females and often first manifests clinical symptoms during young adulthood [5]. MS affects young adults; its course is unpredictable and runs over decades. The dymelination seen in MS provides a permitting condition for axonal degeneration, which seems to be causative of permanent neurologic deficit [6].

The prevalence of renal disease (unspecified) in multiple sclerosis ranged from 0% to 0.78% [7]. However the occurrence of multiple sclerosis in CKD has not been reported in literature except for one case of membranous glomerulonephritis in a case of progressive multiple sclerosis [8]. Here, we report a very rare and first case of multiple sclerosis in a case of chronic kidney disease.

CASE REPORT

A 58-year-old male, known case of hypertension since 5 years and on regular treatment, admitted with complaints of sudden painless loss of vision since 3 days in left eye with blind right eye since 3 years. Patient had history of right eye acute optic neuritis with total loss of vision 3 years back. He had no addiction of tobacco or alcohol. The past medical history was insignificant.

3 years back, he had acute optic neuritis of right eye with sudden painless complete loss of vision. He went to the hospital on the 5th day where he was investigated and found to have normal MRI and normal fundus examination but his kidney function tests were deranged (Serum creatinine 3.5, Urea 90). There was no signs of uremia and both kidneys show bilateral shrunken kidneys on ultrasound (USG) and output was adequate. He was treated but his vision did not recovered. There was no history of NSAIDS or any other drug abuse. No signs of uremia were present. Patient had no immediate complain except loss of vision.

On examination, he was conscious, oriented, afebrile with pulse of 100/min, blood pressure of 200/110 mm of Hg, respiratory rate was 18/min and SOP2 was 98% on room air. Systemic examination was unremarkable. He was euvolemic without pericardial rub. Routine labs were send. Electro Cardio Gram (ECG) showed sinus tachycardia and x-ray chest was normal. Laboratory investigations revealed haemoglobin of 7.8 gm%, WBC of 10,000/mm3, platelet count of 180,000/mm3. Serum electrolytes and liver function were normal. Kidney functions were deranged with urea of 140 and serum creatinine was 7.5 mg/dl but with adequate urine output. Serum calcium was low and serum phosphorus was normal. His ANCA profile as well as Sr. ANA, dsDNA were negative. Iron studies showed chronic iron deficiency. Ultrasound of abdomen showed bilateral shrunken kidneys with loss of CMD suggestive of chronic kidney disease. Fundus examination was normal except for hypertensive retinopathy and optic atrophy in right eye.

We suspected of optic neuritis and did MRI Brain which showed white matter hypodensity in periventricular area, tempo-parietal cortices and cerebellum suggestive of multiple sclerosis. His VEP (Visual Evoked Potentials) were abnormal.

His CSF was sent which showed acellular and CSF electrophoresis showed oligoclonal bands. Hence, we started him on I.V. methyl-prednisolone 1 gram for 3 days which improved his vision to almost complete recovery with normal vision in the right eye.

He was under regular follow up and did not complain any visual problems. Here, we report case of optic neuritis due to multiple sclerosis in a case of chronic kidney disease.

DISCUSSION

Multiple Sclerosis, a chronic inflammatory and neurodegenerative disease of the central nervous system, characterized by recurrent relapses of central nervous system inflammation ranging from mild to severely disabling. Multiple sclerosis, also known as disseminated sclerosis or encephalomyelitis disseminate. The insulating covers of nerve cells in the brain and spinal cord damaged in an inflammatory disease. This damage disrupts the ability of parts of the nervous system to communicate and resulting in a wide range of signs and symptoms [8,9].

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It was first described by Jean-Martin Charcot, a French researcher, in 1868 [10], and affects more women than men [11]. Its incidence if found to be 5.68/100.000 per year [12]. MS is believed to be an autoimmune disorder that damages Central Nervous System (CNS) myelin and axons. Typically, MS begins as a relapsing-remitting disease and evolves over time to a chronic secondary progressive condition that can lead to severe disability and even death [13].

In the mid-1990s, the modern therapeutic era began for Multiple Sclerosis (MS). While the initial disease modifying therapies had some adverse effects, such as hepatotoxicity [14], they have been relatively well tolerated and issues related to co-morbidity either increasing the risks of recognized adverse effects or being themselves adverse effects – have not been a significant concern. The prevalence of renal failure in multiple sclerosis ranged from 0% to 0.78%, with the highest estimate being from one population-based Taiwanese study [15]. Chronic kidney failure has become a global epidemic [16].

The etiology of the decreased in GFR is likely multifactorial and may represent the combined effects of chronic neurogenic bladder (incomplete emptying, frequent urinary tract infections and pyelonephritis, and nephrolithiasis), as well as increased exposure to other nephrotoxic drugs (Non-Steroidal Anti-Inflammatory Drugs [NSAID], antibiotics, and certain contrast dyes), and the marked tendency for disabled patients to remain chronically dehydrated to avoid frequent micturation. MS derived its term from the multiple scarred areas in the brain termed plaques. The acute lesion in MS is characterized by perivenular cuffing and tissue infiltration by mononuclear cells predominantly T lymphocytes and macrophages. As the lesion evolves, demyelination occurs, with macrophages and microglial cells scavenging the myelin debris. Proliferation of astrocytes lead to scar formation.

Lesions of MS typically occur at different times and in different CNS locations (i.e., disseminated in time and space). Multiple sclerosis takes several forms, with new symptoms in either isolated attacks (relapsing forms) or building up over time (progressive forms). Between attacks, symptoms may disappear completely; or permanent neurological problems often occur, especially as the disease advances [17]. The most common initial symptoms include weakness in one or more limbs, blurring of vision secondary to optic neuritis, sensory disturbances, diplopia and ataxia [18]. Disability and severity is measured by the Expanded Disability Status Scale (EDSS) [19].

As the disease progresses and disability increases, it can become difficult to differentiate the symptoms and signs of MS from those of other potentially emerging neurodegenerative conditions. The literature contains many reports of the concurrence of MS and other neurologic conditions [20]. Moreover, although MS is an Upper Motor Neuron (UMN) disease, Lower Motor Neuron (LMN) findings including muscle atrophy, especially in hand muscles have been reported [21]. Theoretically, MS-related muscle atrophy could be due to disuse, associated peripheral neuropathy, involvement of brainstem or spinal gray matter structures, or involvement of the ventral roots at their exit zone. It has an insidious onset and course, without a major contribution to the patient's complaints and dysfunction. Therefore, it is wise to be suspicious about this finding, especially when it is a source of dysfunction or is associated with a significant change in disease progression [22,23].

Renal side effects are uncommon. There exists one case report describing a case of membranous glomerulonephritis and thrombocytopaenia in a patient with progressive multiple sclerosis. Swanton and colleagues, in 2007, and Montalban and colleagues, in 2010, have made important contribution on the diagnosis of MS proposing simpler criteria allowing easer MRI evidence for Dissemination In Space (DIS) and Dissemination In Time (DIT) to be used in patients who present with Clinically Isolated Syndrome (CIS), respectively. CSF abnormalities consist of mononuclear cell pleocytosis, an elevation in the level of total Ig, and the presence of oligoclonal Ig. Evoked response testing may detect slowed or abnormal conduction in visual, auditory, somatosensory, or motor pathways. On MRI, periventricular lesions may be found on T2 weighted MRI brain scans. However, no clinical sign or diagnostic test finding is unique for MS.

The use of MRI has had a major impact on allowing the early and more precise diagnosis of the disease. Management of MS may be divided into two categories consisting of a) treatment designed to arrest the disease process and b) symptomatic therapy. Immunosuppressive drugs remain the cornerstone of therapy in the first modality of treatment, although their efficacy is limited and their chronic use entails considerable risk. Mild acute exacerbations that do not produce functional decline may not require treatment. When functional ability is affected, the standard intervention is intravenous injection of high-dose corticosteroids.

Intravenous methyl-prednisolone has been show to shorten the duration of acute exacerbations. The mechanism of action of corticosteroids used for acute relapses is not completely clear, but may involve the following actions: Prevention of inflammatory cytokine activation, Inhibition of T-cell activation, prevention of immune cells from entering the CNS and Increased death of activated immune cells. Corticosteroids hasten functional recovery after relapses. Therefore, it is important to established the diagnosis and management in patients with ocular damage which leads to multiple sclerosis.

CONCLUSION

We report here a very rare and perhaps first case of multiple sclerosis in literature so far that presented with acute optic neuritis in left eye that rapidly improved with IV Methylprednisolone for 3 days. The patient had a characteristics picture of white matter lesions in the cerebral cortex, cerebellum, periventricular lesion and optic atrophy in right eye, with oligoclonal bands in CSF and abnormal VEP in a diagnosed case of chronic kidney disease with bilateral shrunken kidneys and severely deranged renal functions.

The occurrence of multiple sclerosis in the setting of chronic kidney disease has not been mentioned anywhere in literature. There has been a single case report of membranous glomerulonephritis with thrombocytopenia in a case of progressive multiple sclerosis.

The occurrence of acute renal injury due to neurogenic bladder subsequent to statsis of urine and infections as well as to dehydration in a bedridden patient of multiple sclerosis is understood. Also, the affection of kidneys due to other associated or complicating infections or drug insult and subsequent renal injury or renal failure in a case of multiple sclerosis is also not known.

However, here we report a case of established CKD and the occurrence of multiple sclerosis later which may perhaps be the first case report in literature in India and world.

This case, in our opinion opens the flood gates for research and investigation as to whether the oxidative stress and proinflammatory states in CKD serves to precipitate the dreaded condition of multiple sclerosis in a genetically predisposed host in a given environment.

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