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CASE REPORT

A Case of an Accelerated Uremic Neuropathy

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Abstract

We present a 62-year-old man, with a prior history of diabetes mellitus, atherosclerotic heart disease, and chronic renal failure requiring peritoneal dialysis, who developed accelerated uremic sensorimotor polyneuropathy. Our patient significantly improved after effective hemodialysis. Although renal transplantation is a curable therapy for uremic neuropathy, effective dialysis is still an important treatment for the group of patients who cannot undergo renal transplantation.

Keywords: ESRD, accelerated uremic polyneuropathy, electromyography, dialysis, kidney

INTRODUCTION

Uremic polyneuropathy is one of the most frequent neurological manifestations of end-stage renal disease (ESRD).[1] It develops at a glomerular filtration rate of under 12 mL/min.[2] Approximately 60% of ESRD patients are affected by uremic polyneuropathy.[1] The condition may progress slowly or rapidly (as a rare form). Here, we describe a 62-year-old man who developed accelerated uremic neuropathy.

CASE REPORT

A 62-year-old man with a prior history of diabetes mellitus for 3 years, atherosclerotic heart disease for 1 year, and chronic renal failure requiring peritoneal dialysis for 3 months was admitted to our clinic for the treatment of fungal peritonitis. Upon physical examination, 190/110 mmHg arterial tension, three-positive pretibial edema, and cloudy bag were revealed. Also he complained of weakness of the bilateral lower limbs that increased in the last 2 months. After neurologic examination, there were bilateral lower limb weakness (score 1/5), reduction of deep tendon reflexes, and bilateral sock-shaped hypoesthesia. Laboratory examinations showed hemoglobin 11 g/dL, white cells 8360/mm^3, blood urea nitrogen 58 mg/dL, creatinine 8 mg/dL, albumin 2.5 g/dL, sedimentation rate 111 mm/h, calcium 8.3 g/dL, phosphorus 4.9 g/dL, intact parathyroid hormone 169 pg/mL, and potassium 4.9 mEq/L. Also his creatinine phosphokinase level was in a normal range. Because the culture test was positive for fungal peritonitis, the peritoneal catheter was removed and hemodialysis was started. The patient underwent intensive daily dialysis and ultrafiltration during the first one week of the hemodialysis and then three days per week regular dialysis was continued. Also antifungal therapy was administered. To evaluate the neurological dysfunction, cranial computed tomography scan was performed. His tomography was normal. Also no pathology was found in his cranial and thoracic spinal magnetic resonance imaging tests. Electromyographic study showed diffused sensorimotor neuropathy. In addition to dialysis, the patient was administered physical therapy and gabapentin (150 mg/day). After 8 weeks of hemodialysis treatment, the patient had good recovery of bilateral lower limbs (score 4/5). Because of the improvement of his motor functions after regular dialysis, we did not perform invasive nerve biopsy. In this respect, the patient was diagnosed with accelerated uremic neuropathy, which regressed after regular dialysis with other conservative treatments.

DISCUSSION

Chronic kidney disease is a rapidly growing global health problem. The most important cause is diabetes mellitus.[1] Neuropathy is a frequent complication of ESRD.[3] Several reports that studied the pathophysiology of neurological disease in chronic kidney disease

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have tended to focus on one or more of the retained toxins that are responsible for developing the neurological dysfunction.[3]

Although the exact pathogenesis of uremic neuropathy is unknown, the most common abnormality is axonal degeneration and secondary demyelination in peripheral nerves.[4] Characteristics signs are paresthesias, pain, and sensory loss and in advanced disease reflexes are reduced. The distal lower extremities are frequently affected.[1]

This condition generally has insidious onset and progression over months. In addition to the slow progress, rapidly progressive motor neuropathy has been described in a small number of diabetic ESRD patients.[5,6]

Here we describe a 62-year-old man who developed rapidly uremic neuropathy. In his history, lower limbs weakness was started 3 months prior his admission. When we examined his past medical history, we found that he had refractory peritonitis caused by several microorganisms for 3 months and he was treated with different antibiotics. Also during that period, there was an increase in his edema levels because of ultrafiltration failure. On his admission when we switched the therapy to hemodialysis, after 8 weeks his symptoms were significantly regressed and in his neurological examination lower limb weakness score was 4/5.

Although diabetes mellitus has important effects on the nervous system, the clinical recovery after regular dialysis suggests that the neuropathy was related to ESRD and insufficient dialysis treatment. Moreover, in differential diagnosis, we suspected the presence of uremic myopathy but in this case there were no proximal myopathy symptoms; the intact parathyroid hormone, alkaline phosphatase, and creatinine phosphokinase levels were in the normal range for ESRD patients; and importantly, there was no myopathic pattern in electromyogram. Given this clinical and laboratory findings, we excluded myopathy.[7]

There are contradictory reports in the literature about the effects of dialysis on uremic neuropathy. Although early studies report that mild neuropathy can be recovered completely with adequate dialysis,[8] more recent studies have determined that complete recovery is uncommon by dialysis in uremic neuropathy.[9]

According to some studies, the middle molecules such as parathyroid hormone and B_2 microglobulin may lead to the development and progression of uremic neuropathy.[1] Several reports demonstrated that middle molecules were better dialyzed by the peritoneum than by the cellophane membranes, so patients treated with peritoneal dialysis had lower rates of neuropathy than those treated with hemodialysis,[10] whereas in another study no significant difference was found.[11] Here in this case, we did not perform the peritoneal equilibrium test because the patient had refractory peritonitis, but with both clinical and laboratory findings we suspected dialysis inadequacy. Also for the presence of the fungal peritonitis we switched to hemodialysis.

In conclusion, this case showed that, although there are contradictory reports in the literature about the effects of dialysis for uremic neuropathy especially for severe forms like our patient, we postulate that unequivocally, effective dialysis is still an important treatment for this group who cannot undergo renal transplantation.

***Declaration of interest*:** The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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