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Subacute Polyneuropathy after Initiation of Peritoneal Dialysis, Improved Following Kidney Transplantation

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Introduction

Uremic neuropathy is the most common neurologic complication of uremia, and it affects more than 60% of patients with end-stage renal disease (ESRD) who undergo dialysis.[1] This type of neuropathy symmetrically involves the distal extremities, especially the lower limbs. On electrophysiological assessment, an axonal pattern is dominant. The clinical course and the intensity of uremic neuropathy vary from one individual to another. Coexisting chronic conditions such as diabetes mellitus can aggravate the symptoms,[2] while dialysis can stop or delay progression of chronic uremic neuropathy.[3] Complete improvement occurs after kidney transplantation.[4]

Recently, acute and subacute neuropathies in ESRD patients have also been reported.[5-8] These conditions have a demyelinating feature and develop after the initiation of continuous ambulatory peritoneal dialysis (CAPD). The pathogenesis of these nonchronic forms of neuropathy remains unclear. We report 2 cases of subacute uremic polyneuropathy that improved following kidney transplantation.

Case Report

Case 1. A 54-year-old man presented with progressive weakness of the extremities of 5 months' duration. He had been diagnosed with ESRD for 13 years and with diabetes mellitus for 25 years. He had been receiving peritoneal dialysis for 6 months, and his current symptoms had appeared after 3 to 4 weeks. Within 2 months, he had lost the ability to walk. Results of a neurologic examination of the cranial nerves were normal. Atrophy of the calf and thigh muscles, loss of hair in legs, and pseudomotor changes in the skin of the calf were apparent. Muscle strength of the wrist extensors and flexors, the elbow extensors, and shoulder abductors was 4/5. Muscle strength of the hip extensors and flexors, knee flexors, and knee and ankle extensors was 3/5, 2/5, and 1/5, respectively. Tendon reflexes were diminished. Pain and heat senses in the distal lower extremities were disrupted up to the knees, and the proprioceptive sense in the feet was disturbed. Electrophysiological assessment showed demyelinating peripheral neuropathy. CAPD was continued, and 2 courses of intravenous immunoglobulin therapy (20 mg daily) were initiated but had no positive effect. Subsequently, plasma exchange was attempted at 2 liters daily for 5 consecutive days and then weekly for 2 months. Considerable improvement was achieved, and the patient was able to do fine handwork and walk with the help of a cane. The patient underwent kidney transplantation, and immunomodulatory treatment was discontinued.

Neurologic symptoms and signs persisted but began to improve gradually after 4 months. By the first posttransplant year, the patient was able to do fine movements with his hands and walk on his own. However, diminished reflexes and mild muscular atrophy of the lower extremities persisted.

Case 2. A 55-year-old man presented in whom ESRD had been diagnosed following admission for acute pulmonary edema 6 months earlier. He had a 5-year history of diabetes mellitus and a 2-year history of uncontrolled hypertension.

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Following a few hemodialysis sessions, peritoneal dialysis was begun. Within 2 months, weakness of the extremities developed and previous tingling and numbness in the distal extremities became aggravated. He was not able to walk on his own after 1 month. Results of a physical examination of the 12 cranial nerves were normal. Atrophy was present in the interosseous muscles of the fingers and the muscles of forearms, thighs, calves, and feet. Muscle strength was about 4/5 in the upper and 3/5 in the low extremities. Tendon reflexes were completely diminished. Pain, heat, and positional senses in the lower extremities were abnormal. Without further treatment, the patient underwent kidney transplantation.

On electrophysiological assessment, peripheral neuropathy with a predominant demyelination pattern was detected. On follow-up, movement power in the extremities returned, and sensory disorders were alleviated. He was able to walk on his own after 6 months. The ability to do fine movements, however, remained mildly impaired. Atrophy of the calf and interosseous muscles of the hand persisted. On 1-year follow-up, no recurrence or aggravation of symptoms was seen.

Discussion

Acute and subacute forms of uremic neuropathy in ESRD patients have been reported in few studies. These forms, which are demyelinating peripheral neuropathies, become symptomatic within a few weeks after the start of CAPD and cause severe disability in the patients within 1 to 3 months.[5-8] Ropper, in 1994, described 4 cases of subacute and acute neuropathy following CAPD. The patients developed generalized limb weakness over days or weeks, severe imbalance, diminished reflexes, and numbness within a few weeks after the initiation of CAPD. Spinal fluid protein levels were elevated, and some demyelinating features were noted on electrophysiological testing. In 1 patient, neuropathy was alleviated by more frequent peritoneal dialysis and improved after kidney transplantation. But in 2 patients with diabetes mellitus, the neuropathy progressed.[5] Toepfer and colleagues reported 3 patients with acute inflammatory demyelinating neuropathy, which developed 4 to 10 weeks following the initiation of CAPD. They also detected elevated spinal fluid protein and signs of demyelinating neuropathy. None of the patients improvement with intensified peritoneal dialysis, but immunomodulatory treatments were effective. One patient responded to hemodialysis and improved completely after receiving a kidney allograft.[6] Lui and coworkers have described 2 cases of acute neuropathy, occurring 6 to 10 weeks after peritoneal dialysis. The disease resolved after kidney transplantation in 1 and with immunomodulatory therapies in another.[8]

In all reported acute and subacute uremic neuropathy cases, the patients have had ESRD and a history of prolonged diabetes mellitus. It seems that the combination of ESRD and diabetes predisposes patients to this type of neuropathy. Acute and subacute forms of nerve involvement (eg, plexopathy and radiculopathy) are common,[9,10] but they affect the extremities asymmetrically, while uremic neuropathy has a symmetric pattern and affects both upper and lower limbs at the same time.

Another pathologic cause of neuropathy in these patients can be acute inflammatory polyneuropathy. This condition, namely Guillain-Barre syndrome, develops over a period of less than 1 month and results in severe disability. High-dose intravenous immunoglobulin therapy and plasma exchange can aid a more rapid remission of patients.[11,12] A relative improvement with immunomodulatory treatments in some patients, as was true in our second patient, suggests a role for an inflammatory process and the coincidental occurrence of subacute inflammatory neuropathy in ESRD patients. Nonetheless, evidence exists against this explanation for subacute neuropathy in ESRD patients. First, immunomodulatory treatments are not always effective in these cases, and second, resolution of disease after kidney transplantation can provide reason to reject this hypothesis.

In agreement with other studies,[5-8] our patients were involved with polyneuropathy when they received peritoneal dialysis. This indicates a causal relationship, probably due to a metabolic disturbance, between acute and subacute polyneuropathy and CAPD. However, further studies are warranted to ellucidate the pathogenesis of this disease.

References

1. Asbury AK. Uremic neuropathy. In: Dyck PJ, Thomas PK, Lambert EH, Bunge R, editors. Peripheral neuropathy. 2nd ed. Philadelphia: WB Saunders; 1984. p. 1811.
2. Tyler HR. Neurologic disorders in renal failure. Am J Med. 1968;44:734-48.
3. Nielsen VK. The peripheral nerve function in chronic renal failure. VII. Longitudinal course during terminal renal failure and regular hemodialysis. Acta Med Scand. 1974;195:155-62.
4. Bolton CF, Baltzan MA, Baltzan RB. Effects of renal transplantation on uremic neuropathy. A clinical and electrophysiologic study. N Engl J Med. 1971;284:1170-5.
5. Ropper AH. Accelerated neuropathy of renal failure. Arch Neurol. 1993;50:536-9.
6. Toepfer M, Schiffl H, Fricke H, et al. Inflammatory demyelinating neuropathy in patients with end-stage renal disease receiving continuous ambulatory peritoneal dialysis (CAPD). Perit Dial Int. 1998;18:172-6.
7. Chen J, Guest S. Inflammatory demyelinating neuropathy presenting in a temporal relationship with the initiation of peritoneal dialysis. Perit Dial Int. 1998;18:542-3.
8. Lui SL, Hui CK, Chan TM, Lo WK, Lai KN. Progressive demyelinating neuropathy after initiation of continuous ambulatory peritoneal dialysis–report of two cases. Clin Nephrol. 2001;56:407-10.
9. Bastron JA, Thomas JE. Diabetic polyradiculopathy: clinical and electromyographic findings in 105 patients. Mayo Clin Proc. 1981;56:725-32.
10. Garland H. Diabetic amyotrophy. Br Med J. 1955;(4951):1287-90.
11. Hughes RA, Rees JH. Clinical and epidemiologic features of Guillain-Barre syndrome. J Infect Dis. 1997;176 Suppl 2:S92-8.
12. Kuwabara S. Guillain-Barre syndrome: epidemiology, pathophysiology and management. Drugs. 2004;64:597-610.