Leg Weakness in a Patient with Lumbar Stenosis and Adrenal Insufficiency

Kyoung-Tae Kim, M.D.,1 Suk-Won Ahn, M.D.,2 Jeong-Taik Kwon, M.D.,3 Young-Baeg Kim, M.D.4
Department of Neurosurgery,1 Kyungpook National University Hospital, Daegu, Korea
Departments of Neurology,2 Neurosurgery,3 College of Medicine, Chung-Ang University, Seoul, Korea

Lumbar spinal stenosis (LSS) is a common spinal disease in the elderly. The cardinal symptom of LSS is neurogenic claudication, but not all patients present with such typical symptom. The clinical symptoms are often confused with symptoms of peripheral neuropathy, musculo-skeletal disease and other medical conditions in elderly patients. In particular, LSS presenting with rapid progression of leg weakness must be distinguished from other combined diseases. We report a case of rapid progressive leg weakness in a patient with LSS and iatrogenic adrenal insufficiency that was induced by obscure health supplement.

Key Words: Leg weakness • Lumbar spinal stenosis • Adrenal insufficiency

INTRODUCTION

Lumbar spinal stenosis (LSS), first described by Verbiest in 19545,6,7, is widely diagnosed disease entity. Katz et al.8 reported that the more than 30,000 surgical procedures were performed in United States in 1994 to treat LSS. The rate of diagnosis is growing rapidly because of improvements in diagnostic imaging tools and surgical techniques, and the aging of the population9. The cardinal symptom of LSS is neurogenic claudication, defined as pain in the buttocks and legs, and numbness or cramping of one or both legs induced by walking, which is relieved when sitting and bending forward10. However, not all patients present with typical symptoms and clinical symptoms in elderly patients are often confused with symptoms of peripheral neuropathy, musculo-skeletal disease and other medical conditions11. The decision to employ surgery must be made with caution in elderly patients with rapidly progressing leg weakness, because this condition rarely occurs in the absence of other combined conditions, and the incidence of combined disease in the elderly patients is high12.13. We report a case of rapid progressive leg weakness in a patient with the LSS and iatrogenic adrenal insufficiency (AI).

CASE REPORT

Preoperative evaluation and operation

A 66-year-old male presented with rapidly progressing leg weakness and voiding difficulty, which had developed one month ago. He complained of chronic lower back pain and radiating pain in both legs, which had developed one year previously. Medical history revealed 10 years of treatment for hypertension and diabetes mellitus (DM). On physical examination, the patient appeared very tired and had moderate bilateral calf muscle atrophy. Neurological examination showed a leg motor power of grade IV/III+(right/left), with knee flexion and ankle dorsi-/ plantar-flexion power of grade IV/III. Both knee and ankle reflexes were diminished without any long-tract signs. The patient's serum sodium level was 131 mEq/L (normal range : 135-146 mEq/L) and other laboratory findings were normal.

Magnetic resonance imaging (MRI) showed severe LSS at L4-5 (Fig. 1). Electromyography (EMG) and nerve conduction...
velocity (NCV) showed bilateral L4, L5, S1 radiculopathy with mild DM neuropathy.

We concluded that the patient's main lesion was LSS and decided to proceed with a transforaminal lumbar inter-body fusion (TLIF) via a left side approach. TLIF was performed successfully and postoperative CT showed well decompression of LSS and properly located pedicle screws (Fig. 2).

**Postoperative clinical course**

At postoperative day 1 (POD #1), partial improvement was seen in radiating leg pain (visual analogue scale score decreased from 7 to 3), and leg weakness (grade IV/III increased to grade IV IV), but the patient complained of general weakness with mild confusion. At POD #3, the patient could walk with maximal walker assistance, but could not void himself without Foley catheterization. At POD #7, he had a high fever with diarrhea and severe confusion, and was diagnosed with pseudomembranous colitis from a colonoscopy. At POD #12, the patient complained of general weakness with mild fever, and still had difficulty voiding without Foley catheterization. He had cystitis and the result of uro-dynamic study suggested a neurogenic bladder. The patient still could not walk without walker assistance. At POD #21, cystitis and colitis improved, but leg weakness (grade IV IV) and voiding condition did not continue. At POD #27, general condition and leg weakness did not improve, and hypokalemia remained. Also, untreated blood pressure and sugar levels were lower limits of normal range. Because of the patient's odd medical conditions, we reviewed past medical history again and revealed that the patient had taken a health supplement for two years, and had stopped two months ago. We analyzed the health supplement and the result was surprising. The main component of the health supplement was glucocorticoid. The supplement had been purchased over the internet from foreign country. Serum cortisol level (0.52 μg/dL, normal range : 4.3-22.4), serum adrenocorticotropic hormone (ACTH) level (9.4 pg/mL, normal range : 10-60) and rapid ACTH stimulation test (moderate response) provided a diagnosis of severe secondary AI, which was treated immediately with glucocorticoid. General condition improved dramatically, with gradual improvement of leg weakness. Three days after medication, the patient could walk with a crutch, and void himself without Foley catheterization. Ten days after medication, he could walk independently and void normally. Twelve days after medication, the patient was discharged in good health. One year after operation, he can walk and void normally, but muscle atrophy is not recovered.

**DISCUSSION**

LSS is a common spinal disease in the elderly and its symp-
definite leg motor weakness. During four weeks after TLIF operation, the patient could not walk without walker assistance and void without Foley catheterization, even if the leg radiating pain and weakness partially improved (leg motor grade IV/III increased to grade IV/IV). We hypothesize that in this case, leg motor weakness and voiding difficulty, which were primarily affected by LSS, were more influenced by AI than other organs. Postoperative hypocortisolism might have delayed the recovery of the cauda equina syndrome at the operated lumbar lesion.

CONCLUSION

This case highlights two basic medical principles. One is the importance of obtaining the detailed medical history. If we had performed a detailed history taking, including the use of health supplements, and AI had been properly treated with glucocorticoid before surgery or during surgery, we could have more safely performed the lumbar operation, and the patient might have followed rapid recovery without complications. The other principle is that decisions to proceed with surgery in elderly patients with rapid progressive leg weakness must be made with caution, because rapid progression of LSS is uncommon without special circumstance, and the incidence of combined disease in the elderly is relatively high.

References