Physical Therapy Combined with Corticosteroid Intervention for Systemic Lupus Erythematosus with Central Nervous System Involvement: A Case Report

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Abstract. [Purpose] Systemic lupus erythematosus (SLE) is a chronic, immune-mediated disease, affecting 0.1% of the general population. To date, few studies have investigated the efficacy of physical therapy for SLE patients with CNS involvement. The aim of this study was to report whether the combined use of corticosteroids and physical therapy, consisting of reflex inhibition and functional training, was beneficial to functional recovery. [Subjects and Methods] A 22-year-old male SLE patient with CNS involvement requested physical therapy due to strong spasticity of the trunk and limbs in a bedridden state. Corticosteroid intervention and physical therapy were undertaken for 16 days. [Result] After 16 days of the interventions, the patient demonstrated stabilized and alleviated neurological symptoms and an improved functional level. [Conclusion] The present case indicates that physical therapy combined with corticosteroids might be a possible treatment and rehabilitation method to effectively recover motor function for SLE patients who have strong spasticity due to CNS involvement.

Key words: Corticosteroids, Physical therapy, Systemic lupus erythematosus

INTRODUCTION

Systemic lupus erythematosus (SLE) is associated with significant cerebrovascular and neuropsychiatric diseases for which multiple pathogeneses have been proposed. Involvement of the central nervous system (CNS) is one of the major causes of mortality and morbidity in SLE patients. Although the underlying mechanisms of the CNS involvement are a less understood aspect of the disease, the common etiology is the involvement of the cerebral microvasculature that causes deficits in motor functions shown in the other diseases involving the CNS. Cerebrovascular diseases in SLE have been estimated to occur in 5 to 19.6% of SLE patients. SLE patients with CNS involvement can demonstrate stroke-like symptoms such as severe hemiparesis, spasticity, loss of soft tissue elasticity, etc. Corticosteroid intervention has been considered the first-line treatment for neuropsychiatric SLE. Previous studies reported that a corticosteroid treatment improved neurological symptoms by normalization of regional cerebral blood flows for SLE patients with neuropsychiatric manifestations. However, no studies have been reported that corticosteroid intervention led to reduced spasticity, improved balance and ADL for SLE patients with CNS involvement. These facts indicate that although corticosteroid intervention could be a major approach to promote relief of symptoms in SLE patients, intervention has been limited to active and directive intervention to improve the motor function of patients. Motor functions, such as reduced spasticity, balance, gait, and ADL, in SLE are important because improvement of motor functions in patients leads to a better quality of life.

Physical therapies have been a major treatment used to improve motor functions for patients with CNS problems. A main concept for such physical therapy methods is developing inhibition of abnormal motor synergies to decrease spasticity and improve motor control abilities in such patients. These signify that physical therapy treatments might play an important role in improving decreased motor functions for SLE patients with CNS involvement. However, based on our literature searches of PubMed® and Google Scholar®, surprisingly, no studies have been reported about physical therapy applied to SLE patients with CNS involvement.

In our study, we present a patient who was diagnosed as having SLE with CNS involvement. We applied physical therapy as well as corticosteroid therapy to the patient and...
investigated the clinical pathway. We conclude that these interventions can be effective treatments for functional improvement of the patient.

CASE REPORT

Patient
A 22-year-old man with SLE was admitted to our physical therapy department, and he signed an informed consent statement for this study. The patient presented with utterance of meaningless words, jaundice, and lower limb weakness. He had been diagnosed with discoid lupus erythematosus, febrile neutrophilic dermatosis, schizophrenia, and bipolar affective disorder five months prior to presentation. He reported a four-day history of epigastric pain; four days before, there had been no other systemic features of infection.

He was diagnosed with SLE with CNS involvement and pericardial effusion. A radionucleide regional cerebral blood flow (rCBF) study with 900 MBq technetium-99 m ethyl cysteinate dimer (TECD) by single-position emission computed tomography (SPECT) revealed multifocal decreased perfusion to the anterior cingulate, left parietal, left occipital, right lateral temporal, and anterior pole of the left temporal cortices. Ultrasound revealed that the liver, spleen, pancreas, and gall bladder were normal in size, contour, echogenicity, wall thickness, and calcification. A hematological examination indicated that his hemoglobin, hematocrit, red blood cells, white blood cells, and platelets were normal. However, in a liver function test, his AST, ALT, ALP, and bilirubin levels were markedly elevated. He was also positive for anti-dsDN, anti-histone, and anti-Sm antibodies. Laboratory tests for venereal diseases and lupus anticoagulant tests were negative. Electromyography showed no abnormal spontaneous activity, and his motor unit potential was of normal amplitude and duration. He was treated with high-dose intravenous steroids (methylprednisolone; 1 g/day), which resulted in complete resolution of the SLE. His neuropsychiatric symptoms were stationary.

Physical therapy
After admission for three days, he requested physical therapy due to marked spasticity that was uncontrollable by corticosteroid intervention. His spasticity was similar to a “bilateral stroke-like” symptom presented in his trunk and all four limbs. When admitted to physical therapy on the day of the request, his spasticity level was modified Ashworth scale (MAS) grade 3 in both lower limbs and grade 2 in both upper limbs. He had no active range of motion activities in any limb. We could not evaluate his balance ability and ADL level at the time of referral. His outcome measurements were evaluated at the time of referral and at 5 days, 7 days, 12 days, and 16 days later (Fig. 1).

Our first goal of the therapeutic treatment for the patient was spasticity relief, since he had increased muscle tone that deteriorated motor function (Fig. 1). We used the reflex inhibition pattern (based on the Bobath concept) to reduce the spasticity level(9). The reflex inhibition pattern could be described as particular movement patterns of proximal joints that affect the tone of the trunk and limbs. The applied reflex inhibition techniques to the patient consisted of passive and active ROM exercises in all four limbs and the trunk in a normal sitting posture while the hip and shoulder joints were flexed at 90 degrees. These techniques were applied for 1 hour a day (5 days/week). After five days of treatment, his spasticity decreased to an MAS grade of 2 in the lower limbs and 1+ in the upper limbs. Of note, his ankle spasticity also decreased. These outcomes suggest that spastic muscle groups were successfully inhibited by the applied physical therapy techniques.

After seven days of reflex inhibition pattern treatment, his spasticity was no longer apparent. We evaluated his balance ability with the Berg balance score (BBS), and his BBS at this time was 15, indicating that the level of walking with assistance was not achieved(9) (Fig. 1). He could maintain a sitting and standing posture with minimal assistance from a physical therapist. For these reasons, we decided to focus the physical therapy intervention on improvement of balance beginning with the intervention on the 8th day of the physical therapy session (Fig. 1). He was instructed to reach as far as possible in all cardinal directions in a sitting posture in an unsupported chair and carry out active range of motion activities. He was also asked to maintain a normal standing position on his own base of support. These techniques were applied for 1 hour a day (5 days/week). After a week, he could perform the sitting unsupported and partially perform the sitting to standing, standing to sitting, and transfers items of the BBS. Twelve days after the request for physical therapy, his BBS score reached 33 points, indicating the level of walking with assistance(9) (Fig. 1). We then set independent walking as his new goal and started gait training on the 13th day of the physical therapy intervention (Fig. 1).

After sixteen days, that is, on the last day of his physical therapy sessions, he could walk without a cane. His BBS score also reached 47 points, indicating the level of independent walking(9) (Fig. 1). The patient was discharged from physical therapy and the hospital.

DISCUSSION

In this study, corticosteroid and physical therapy interventions were applied to an SLE patient with CNS involvement. At the time of referral, he showed no active motion and a high level of spasticity in all four limbs. After 16 days of treatment, the patient demonstrated stabilized and alleviated neurological symptoms and an improved functional level. Thus, the current results indicate that the corticosteroid and physical therapy interventions applied to the pres-
ent patient effectively recovered his neurological symptoms and motor functions.

Our first clinical goal was to reduce the patient’s spasticity level because his high level of spasticity inhibited normal movement patterns. Although the mechanisms underlying spasticity have not been fully elucidated, prior works suggest that reflex inhibition and decreased reciprocal inhibition of motor neurons may make an important contribution. The reflex and reciprocal inhibition techniques used in this study might allow breakdown of the abnormal spinal stretch reflex mechanism and return of appropriate muscle activity.

Another important reason to reduce the spasticity was that it is a starting point to facilitate functional activities. This was the reason why balance and gait training were provided to the patient at the time when spasticity was no longer apparent. The results from the present case show that functional training seems to be successful in recovering balance and gait abilities in a fast and effective manner, consistent with other studies. Ultimately, he was discharged from the hospital with the ability to walk independently after sixteen days of physical therapy sessions.

One of the limitations of the present case study is that it is unclear whether the patient’s spasticity and motor function recovery were primarily due to the corticosteroid intervention or the physical therapy intervention. Based on previous studies, however, the corticosteroid intervention was primarily administered to stabilize and alleviate the patient’s neurological symptoms, and there was no evidence of functional recovery. Although it is hard to differentiate the effects of the corticosteroid and physical therapy interventions, we conclude that the two interventions played with each other to improve the patient’s clinical status. Specifically, the present impressive recovery in the patient’s functional abilities resulted largely from the physical therapy intervention, while the corticosteroid treatment had an effect on stabilization and alleviation of the neurological symptoms. Further studies may be needed to evaluate the possible differences between corticosteroid interventions and physical therapy interventions in the treatment of SLE patients with CNS involvement.

REFERENCES

1) Greene ER, Yonan KA, Sharrar JM, et al.: Middle cerebral artery resistivity and pulsatility indices in systemic lupus erythematosus: evidence for hyperperfusion. Lupus, 2012, 21: 380–385. [Medline] [CrossRef]
2) Unterman A, Rolte JE, Boaz M, et al.: Neuropsychiatric syndromes in systemic lupus erythematosus: a meta-analysis. Semin Arthritis Rheum, 2011, 41: 1–11. [Medline] [CrossRef]
3) Postal M, Costallat LT, Appenzeller S: Neuropsychiatric manifestations in systemic lupus erythematosus: epidemiology, pathophysiology and management. CNS Drugs, 2011, 25: 721–736. [Medline] [CrossRef]
4) Chiewthanakul P, Sawanyawisuth K, Focheran C, et al.: Clinical features and predictive factors in neuropsychiatric lupus. Asian Pac J Allergy Immunol, 2012, 30: 55–60. [Medline]
5) Ishishaka K, Age T, Fukuda K, et al.: A case of SLE presenting stroke-like symptoms. Intern Med, 2011, 50: 359–362. [Medline] [CrossRef]
6) Baca V, Lavalle C, Garcia R, et al.: Favorable response to intravenous methylprednisolone and cyclophosphamide in children with severe neuropsychiatric lupus. J Rheumatol, 1999, 26: 432–439. [Medline]
7) Cuadrado MJ, Khamashia MA, Ballestero A, et al.: Can neurologic manifestations of Hughes (antiphospholipid) syndrome be distinguished from multiple sclerosis? Analysis of 27 patients and review of the literature. Medicine (Baltimore), 2000, 79: 57–68. [Medline] [CrossRef]
8) Sanna G, Bertolaccini ML, Mathieu A: Central nervous system lupus: a clinical approach to therapy. Lupus, 2005, 12: 935–942. [Medline] [CrossRef]
9) Sun SS, Huang WS, Chen JJ, et al.: Evaluation of the effects of methylprednisolone pulse therapy in patients with systemic lupus erythematosus with brain involvement by Tc-99m HMPAO brain SPECT. Eur Radiol, 2004, 14: 1311–1315. [Medline] [CrossRef]
10) Hashidate H, Shiomi T, Sasamoto N: Effects of 6 months combined functional training on muscle strength, postural balance and gait performance in community-dwelling individuals with chronic stroke hemiplegia. J Phys Ther Sci, 2011, 23: 617–623. [Medline] [CrossRef]
11) Bobath B: The treatment of neuromuscular disorders by improving patterns of co-ordination. Physiotherapy, 1969, 55: 18–22. [Medline]
12) Cherry DB: Review of physical therapy alternatives for reducing muscle contracture. Phys Ther, 1980, 60: 877–881. [Medline] [CrossRef]
13) Berg KG, Wood-Dauphinee SL, Williams JJ, et al.: Measuring balance in the elderly: validation of an instrument. Can J Public Health, 1992, 83: S7–S11. [Medline]
14) Bhagchandani N, Schindler-Ivens S: Reciprocal inhibition post-stroke is related to reflex excitability and movement ability. Clin Neurophysiol, 2012, 123: 2229–2246. [Medline] [CrossRef]
15) Graham LA: Management of spasticity revised. Age Ageing, 2012, 41: 167–177. [Medline] [CrossRef]
16) Stevenson VL: Rehabilitation in practice: spasticity management. Clin Rehabilitation, 2010, 24: 293–304. [Medline] [CrossRef]
17) Mazzare N, Grey MJ, Sinkjaer T: Contribution of afferent feedback to the soleus muscle activity during human locomotion. J Neurophysiol, 2005, 93: 167–177. [Medline] [CrossRef]
18) Simonsen EB, Dyhre-Poulsen P: Amplitude of the human soleus H reflex during walking and running. J Physiol, 1999, 515: 929–939. [Medline] [CrossRef]
19) Stein RB, Thompson AK: Muscle reflexes in motion: how, what, and why? Exerc Sport Sci Rev, 2006, 34: 145–153. [Medline] [CrossRef]