Physical therapy in a patient with bilateral obturator nerve paralysis after surgery. A case report

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Summary

Obturator nerve injury can result from surgical procedures. Bilateral obturator nerve injury developed in our patient as a result of edema in the obturator fossa after a debulking operation. In the postoperative period, neuromuscular electrical stimulation, electromyographic (EMG) biofeedback, exercise and a home treatment program were used as the physical therapy approach. The patient became symptom-free after the physiotherapy program.

Key words: Obturator nerve paralysis; Physical therapy; Surgery.

Introduction

Obturator nerve injury in obstetrics, gynecology and gynecologic oncology is thought to be infrequent. The reported consequences of obturator nerve injury vary in severity and management options have not been well described. Articles concerning physical therapy approaches in obturator nerve paralysis patients during the postoperative period are rare. Gynecologic oncology patients undergoing debulking, radical or sidewall operations in the obturator fossa are at increased risk of obturator nerve injury [1]. The purpose of this case report is to describe the physical therapy evaluation and treatment program of a patient with bilateral obturator nerve paralysis due to surgery.

Case Report

The patient was a 48-year-old woman who had bilateral obturator nerve paralysis because of a debulking operation-related complication. The postoperative pathologic diagnosis of the patient was ovarian fibroma. One month after the operation, the patient was first seen in our unit due to loss of hip adductor control. In the assessment; ranges of motion of the lower extremities were normal and no muscle tightness was seen. Lovett's manual muscle test method was used for assessment of the muscle strength on a scale of 0 to 5 in which 5 shows normal muscle strength where the patient can perform the activity of the muscle against gravity and hold against resistance given at the end of the range; and 0 shows absence of muscle contraction. In the electrodiagnostic evaluation EMG, faradic excitability and strength-duration tests were applied to the m. adductor longus. EMG results showed axonal degeneration. Faradic excitability and strength-duration tests supported the results of the EMG. Postural faults were evaluated with anterior, posterior and lateral posture analysis while in the standing position but there was no deficit. Observational gait analysis was then performed. The patient could not cross her legs due to loss of adductor control and had an abducted gait. Pain and sensorial assessments were performed for discrimination of light touch and temperature which the patient did not complain of. The patient's assessment showed that both hip adductors were weak. Weakness was more obvious on the right side. The patient was treated with superficial heat in ithe form of infrared for 20 minutes before neuromuscular electrical stimulation with faradic current three times a week. During electrical stimulation, in order to localize the current to the muscles, a small active electrode was applied to the motor point of the muscle, the circuit being completed with the large size dispersive electrode sited at a proximal area. The muscles were stimulated for a total of 20 minutes with a rest period of five minutes between two 10 minutes periods. After electrical stimulation, bilateral lower extremity patterns of proprioceptive neuromuscular facilitation (PNF) techniques were applied. However, one month later, PNF exercises were excluded from the treatment program because the patient could not tolerate them due to degenerative changes and the resultant pain in the lumbar region. Instead, active and active-assistive exercises were initiated. The patient repeated these exercises at home once a day. After 40 treatment sessions, neuromuscular electrical stimulation was cancelled because faradic excitability test values changed from hyperexcitability to hypoexcitability. In addition, a strength-duration curve test inclined towards the left in comparison with the first evaluation. These results showed regeneration in the nerve and she was becoming able to actively contract her muscles. Reeducation treatment was applied by EMG biofeedback, and active-assistive and active exercises were continued. The EMG biofeedback treatment was applied for 20 minutes per session on each side, and a 5-minute rest period was allowed between two 10-minute treatment periods. EMG biofeedback data were derived from motor unit action potentials of the hip adductor muscles during the active movement. EMG biofeedback treatment was applied for only four sessions because of the patient's preference. After therapy muscle strength was increased, especially on the right side; the patient no longer had problems with controlling her gait. She was discharged in accordance with the functional and electrodiagnostic assessment results. The follow-up evaluations showed no pathological findings and the patient recovered completely at the end of two years.

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Discussion

Obturator nerve injury in the form of neuropraxia, axonotmesis, and neurotmesis causes morbidity in the form of pain, sensory loss to the medial thigh, and inconstant motor loss to the adductor muscle group. Recovery in a patient with neuropraxia is usually expected to occur within six weeks.

A more severe injury which exhibits Wallerian degeneration is termed axonotmesis. In this case, neural elements distal to injury site degenerate, although the supporting structures of epineurium, perineurium, and endoneurium remain undisturbed. In these cases, functional recovery often occurs within six months to one year [1]. These residual changes are readily amenable to physiotherapy. Therapy should be directed at correcting the condition, and surgical repair should be performed if nerve division is noted during surgery (neurotmesis).

Neuromuscular electrical stimulation is used as an adjunct to physical therapy to augment or maintain local muscle strength, range of motion, motor control and coordination. The basis of the facilitation and re-education treatment programs, in healthy individuals as well as in orthopedically or neurologically involved patients, is the bombardment of the central nervous system with sensory information [2].

She was the first patient with obturator nerve paralysis referred to us. To our knowledge this report describes the first isolated postoperative obturator nerve paralysis and the physiotherapy interventions. We are aware of the cases who were given physical therapy after obturator nerve paralysis was recognized intraoperatively; one patient had no neurological deficit at the time of the most recent examination with physical therapy within a month [1], and the other patient's adductor muscle weakness, which had been unchanged for more than one year despite physical therapy, improved only after nerve grafting [3]. However, the physical therapy interventions which were applied were not described in these cases. In our case, after 44 treatment sessions within four months, voluntary bilateral hip adduction and leg crossing movements while walking were improved. At the end of the physiotherapy program, it was observed that the symptoms had disappeared completely.

Conclusion

There is always a risk of obturator nerve injury in debulking, side-wall or radical operations because of the energy modalities that are used. In order to decrease the complication of risks considerably, operation knives and clips should be used. When obturator nerve paralysis occurs in a debulking operation, postoperative physiotherapy leads to satisfactory results and it seems that the recovery period becomes shortened.

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