

Clinical reports

Psoas compartment block for the treatment of lower-limb spasticity caused by spinal cord injury: report of a case

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Introduction

Muscle spasticity is a common disorder after spinal cord injury. The spasticity often can be a major obstacle to rehabilitation, cause discomfort, interfere with existing functions, or result in pain and additional complications [1,2]. For such patients, management of muscle spasticity is crucial. We describe a successful use of the psoas compartment block using electric stimulation [3–5]. For spasticity of the lower limb caused by spinal cord injury.

Case report

A 32-year-old man with a severe cervical spinal cord injury from a motorcycle accident 6 years earlier was admitted to our hospital for treatment of bilateral muscle spasticity of the lower limbs. His spasticity had been progressive in spite of rehabilitation and medical therapy (dantrolene sodium 150mg per day, baclofen 15mg per day). Cervical bone X-ray and CT findings revealed a fifth cervical vertebral fracture and a compression of the spinal cord (Figs. 1 and 2). The patient had complete paraplegia below the level of the sixth cervical spinal cord segment and an increase in the Achilles reflex, the Babinski, reflex, and muscle spasms. Uncontrolled flexor and extensor movements of the lower extremities (particularly, severe flexion and adduction spasticity of both thighs and bilateral extension spasticity of the lower legs) compromised both his abil-

ity to sit in a wheelchair and his nursing care. The bilateral range of motion of the lower limbs was limited (adduction of the hip, 10°; internal rotation of the hip, 15°; extension of the knee, 30°). The patient complained of a dull, painful sensation and sleep disturbance during episodes of severe spasticity of the lower limbs. He also had a neurogenic bladder and orthostatic hypotension.

After the patient was sedated with intravenous midazolam (2mg), we tested whether each peripheral nerve block using 2ml of 2% lidocaine relieved the spasm completely. The correct needle positions of the bilateral femoral, obturator, and sciatic nerves were searched using a commercial insulated needle (Pole, TOP Tokyo, Japan) with nerve stimulation. After we confirmed that the injection of drug stopped the contraction of the affected muscle associated with nerve stimulation and relieved the spontaneous spasm, we injected 2ml of 7% phenol solution each for the bilateral femoral, obturator, and sciatic nerves. For the next 3 days, the spasm reoccurred and the patient desired additional blocks. We repeated a similar block using a neurolytic agent two times. For the next 3 weeks, the spasticity completely ceased. The patient was discharged. Six months later, however, he came to our hospital again because of recurrence of the spasticity. Although we tried again to achieve peripheral nerve block with electrical stimulation, we could not evoke twitches because of nerve degeneration from previous blocks.

We decided to perform a bilateral psoas compartment block to decrease spasticity. After informed consent had been obtained from the patient, he was sedated with intravenous midazolam (3mg) in the operating room. In the lateral position, a commercial insulated needle (Pole, TOP Tokyo, Japan) connected to a nerve stimulator was inserted in the paralumbar sulcus at the level of the fourth lumbar vertebra. The depth and direction of the needle were manipulated to elicit maximal contractions of the upper segments of the muscles

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Fig. 1. X-ray of cervical spine of patient

of the lower limb (iliopsoas muscle, adductor muscle group, quadriceps femoris muscle, sartorius muscle) with electrical stimuli. After maximal contractions had been obtained with the lowest stimulus current (<0.1 mA), 10 ml of 7% phenol solution was injected through the needle in each side of the compartment. There was marked reduction in flexion spasticity of the thigh and bilateral extension spasticity of the lower limbs, and then the bilateral range of motion of the hips and knees was improved. The patient was discharged, and no recurrent or new abnormality of sensation or muscle function occurred during a 4-year follow-up period.

Discussion

Spasticity is a condition of excessive reflex activity associated with involuntary movements and clonus, which may be accompanied by increased muscle tone [1]. Spasticity develops in most patients with spinal cord injury and may be a troublesome and discomforting symptom or a major obstacle to rehabilitation, often interfering with patient positioning and causing the formation of decubital ulcers [1,2]. The basic program for the management of spasticity consists of prevention, removal of nociceptive stimuli, and establishment of a daily program of prolonged stretching. In combination with these measures, a variety of medications (barbiturates, benzodiazepines, baclofen, dantrolene sodium,

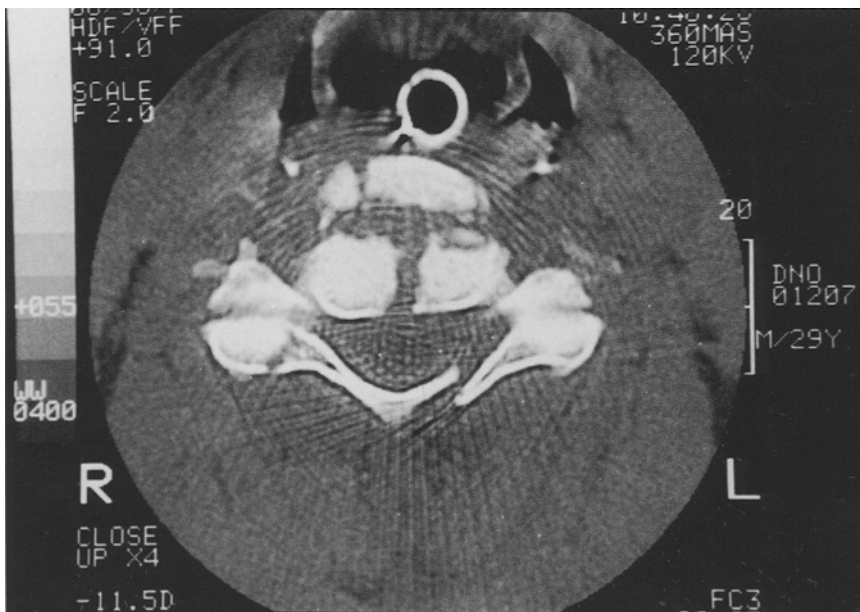


Fig. 2. CT scan of cervical spine of patient

nicotine, phenothiazine, etc.) have used to modulate spasticity [1,6]. When a satisfactory result cannot be obtained by conservative treatment, various neural blocks with neurolytic agents and spinal cord stimulation may be needed for patients with severe spasticity.

Motor-point, peripheral, or paravertebral nerve blocks by phenol have been used to reduce spasticity selectively. The epidural or intrathecal approach has also been used [1,2,5,7–10]. Musculocutaneous nerve block has been used to relieve excessive elbow flexion. Sciatic and femoral nerve block can effectively relieve spasticity of the lower limbs. Therefore, reduction of such profound skeletal muscle spasm by neurolytic block has been helpful in these patients with spasticity resulting from spinal cord injury [7].

In the present case, when the patient visited our hospital for the first time, the effect of rehabilitation and medication was insufficient, and the spasm could be so violent as to catapult the patient out of his wheelchair. We decided on the procedure of nerve block with neurolytic agents and then performed peripheral nerve blocks.

On the patient's second admission to our hospital, we attempted the psoas compartment block with 7% phenol solution. Because we suspected that potential re-iterative blocks by a neurolytic agent could cause nerve degeneration and in fact found a decrease in responsiveness to electrical stimulation, we blocked the more proximal area, the psoas compartment.

The first documented psoas compartment block was reported by Chayen et al. [1]. This was a technique to block the nerves formed from the lumbar plexus (lateral cutaneous nerve, femoral nerve, genitofemoral nerve, and obturator nerve) and some of the sacral plexus. It is useful both for reducing pain and for reducing muscle tone in the region formed from these plexuses [2,12]. Most branches of the lumbar plexus and some of the sacral plexus supplying the thigh are found close to each other in the region of the fourth lumbar vertebra in what we call the psoas compartment.

In fact, we could have chosen a paravertebral nerve block at the L2 and L3 levels, which control the motor function of the troublesome reflex in this case. However, the L2 and L3 nerve roots are very thin anatomically, and thus it is hard to achieve an accurate needle

position and successful motor nerve block, despite the use of electrical stimulation. In addition, because some residual motor functions are needed for this patient's normal activity, we did not select a subarachnoid block. With a subarachnoid block, it may prove difficult to control the extent of the blocked area.

Phenol generally exerts two actions on nerves. First, it has a short-term effect similar to that of local anesthetics, which is directly proportional to the thickness of the nerve fibers. Second, it has a long-term effect related to protein denaturation. This leads to Wallerian degeneration of the axons. The long-term effects of phenol depend on the latter [9,10]. The duration of the block with phenol is usually 4 to 6 months, but in certain patients the blocking effect can last a year or more [7].

In conclusion, the psoas compartment block with a neurolytic agent using electric stimulation may be useful for long-lasting therapy for spasticity caused by spinal cord injury.

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