Muscle Afferent Block for the Treatment of Upper Limb Spasticity: A Report of Two Cases

Yukimasa IGAWA,* Taro OGAWA,* Katsunori IKOMA*

Muscle Afferent Block を行った 2 例

居川 幸正* 小川 太郎* 生駒 一憲*

Abstract: Two cases of upper limb spasticity were treated with Muscle afferent block (MAB). The potential of this mode of treatment as an alternative to botulinum toxin A (BTX-A) was evaluated, and the effectiveness of the MAB in each case was determined. The MAB was an intramuscular injection containing 0.5% lidocaine and pure ethanol at a ratio of 10:1 (v/v). The target muscles of the two patients, both suffering from a chronic hemiplegia after a stroke, were injected with MAB once every 3 days, and a total of 10 injections were administered. The severity of spasticity was evaluated according to the Modified Ashworth Scale Score and the passive range of motion at the affected joint. The spasticity in the injected muscle was immediately alleviated, and the patient experienced relief with regard to the problems caused by the severe spasticity. The effect of the MAB treatment persisted for approximately 2 months. The simple technical procedure involved in the MAB treatment and its immediate effects are illustrated in this case report. We concluded that MAB may be a useful alternative to BTX-A, or a neuromuscular block, for the management of upper limb spasticity. (Jpn J Rehabil Med 2005; 42: 869-874)

Key words: spasticity (痙攣), case report (症例報告), neuromuscular blockade (神経筋遮断剤), muscle afferent block (MAB療法)

Introduction

Spasticity is a characteristic component of the upper motor neuron syndrome that complicates the rehabilitation of many stroke patients. For patients who are ambulatory, walking with the elbow flexed not only impairs balance, but for some is also cosmetically unacceptable. Spasticity in the hands and wrists is especially disruptive because it can interfere with dressing, washing, and other activities of daily living. The management of spasticity remains a major challenge in rehabilitation medicine. Many studies have demonstrated the efficacy of local injections of botulinum toxin A (BTX-A) into upper limb for reducing spasticity. But among the problems of using it are an unwanted weakness, the
possibility of antibody development, and its high cost. There has also been considerable discussion of neurolytic treatment with phenol or alcohol. However these methods have limitations, especially when used in the upper limb where they often affect sensation. Muscle afferent block (MAB) was first reported by Walshe in 1924. He noted that an intramuscular injection of diluted procaine reduced the muscle rigidity of a patient with post encephalitic parkinsonism. The details of his report stated that a transient reduction of muscle tone was observed after an intramuscular treatment with local anesthetic. However, the effect was too brief for the procedure to be used in clinical settings. Kaji et al re-established the method by using an injection of 0.5% lidocaine and pure ethanol in a volume ratio of 10:1. This has been used successfully for treating focal dystonia. In 1999 the same group used an MAB for the treatment of spasticity of the upper limb, and reported its effect. Few investigations or case studies about the practical use of MAB have been presented since then. In the present report we discuss a series of 2 patients with an upper limb spasticity who were treated with MAB.

Method

The patients from our departments who gave their consent to undergo MAB for upper limb spasticity were recruited from consecutive populations of patients being treated for a neurologic impairment. The patients were evaluated by rehabilitation and kinesiological examination before MAB. The target muscles, which were the muscles most actively contributing to the abnormal limb position, were selected by a clinical determination of those groups. The material and procedure for MAB have been previously described. Briefly, the belly of the target muscle was detected by using electromyographic recording, passive movement of the appropriate joints, and palpation guided by superficial anatomy (Fig. 1). Lidocaine (0.5%) was injected first into the belly of the target muscle. Pure ethanol was then introduced into the muscle at the same location as the lidocaine by operating a bipolar connector. After the ethanol injection, lidocaine was again injected to diffuse the solution.

Fig. 1 Muscle afferent block to the forearm muscles in case 1. Lidocaine and ethanol were injected into the flexor digitorum superficialis, the flexor digitorum profundus, the flexor carpi ulnaris, and the flexor carpi radialis muscles. The volume of ethanol was 1 ml per injection for each muscle.
over the muscle and to wash the tube. The total amount of lidocaine was adjusted according to the amount of ethanol which was injected, in a volume ratio of 10 ml for each 1 ml of ethanol. Usually an injection was repeated every 3 days for a total of 10 times. To evaluate the treatment, the Modified Ashworth Scale Score (MASS) and passive range of motion (PROM) at the junction targeted for improvement were assessed before and after consecutive treatments with the MAB. The MASS and PROM have been widely used for evaluation of the effectiveness of BTX-A.9 The starting point for the duration of the effect was set as the day that a series of treatments with MAB were completed. When the MASS and PROM indicated that the targeted junction was restored to its original condition we considered that the duration of the effect was finished. After the last evaluation of a patient during admission, the patient was discharged and then had monthly visits to assess the persistence of the effectiveness of the treatment.

Case Descriptions

Case 1

A 75-year-old man with a history of left intracerebral hemorrhage and chronic right hemiplegia was referred to us for an evaluation of his spasticity. He was medicated for essential hypertension, renal dysfunction, and neurogenic bladder. His right arm was almost non-functional and had a severe sensory disturbance. He was unable to walk, dress, and perform any other activity of daily living except for sitting on a wheel chair. His Barthel index score for activities of daily living was 10 points. A physical examination revealed the presence of pressure sores on the ulnar side of the fourth finger, and on the radial side of the fifth finger, of his right hand. Progressive spasticity of the finger flexors contributed to the pressure on the skin lesions between the two fingers (Fig. 2), and could have led to an enlarged ulceration and infection. On admission, the MASS for muscle tone in his fourth finger was 3, and the PROM of the proximal interphalangeal joint for extension of the fourth finger was less than 10 degrees. His general condition was good except for the pressure sores.

Immediately after his admission, and in consulta-

tion with a dermatologist, all of the pressure sores were treated with ointment. However, the severe spasticity of his fingers made it difficult to get access to the skin lesions and to administer an effective treatment. A reduction of the spasticity of the fingers was essential so that these skin sores could be treated. To reduce the finger spasticity we selected the flexor digitorum superficialis, the flexor digitorum profundus, the flexor carpi radialis, and the flexor carpi ulnaris muscles, as target muscles for MAB. The selection of the muscles which were chosen for injection was based on many of the reported studies with BTX-A.6 The patient received MAB to each target muscle every third day for a total of 10 times (Fig. 1). Subsequently his finger spasticity was reduced, the MASS improved from 3 to 2, and the increase in the PROM was 80 degrees in the last analysis (Fig. 2). The expression

Fig. 2  Increase in PROM: \( \angle (\beta - \alpha) = 80 \) degrees. The PROM of the joints for case 1 are shown before (upper) and after (lower) MAB. The proximal interphalangeal joint of the fourth finger was almost immovable before MAB. As the muscle tone was reduced the skin lesions (arrow) were released from mechanical pressure between the two fingers.
of the first effect with MAB for his finger spasticity was seen after a single injection, but it disappeared within a few days. Repeated injections extended the duration of the effect. The course of the treatment with MAB did not produce any observable adverse effects except for a focal muscle hardening around the injected site which disappeared within 2 weeks of discontinuing the injections. The ointment was applied daily. As the muscle tone reduced it became easier to get access to the skin lesions and to treat them (Fig. 2). One week after the last MAB all of the pressure sores were epithelized. The patient was consequently discharged after this resolution of his skin problem. Two weeks after his discharge, at the first follow up visit, the condition of his finger spasticity and pressure sores was good. Six weeks after the last evaluation during his admission, he was readmitted to our hospital to have an operation for a vesical fistula. We confirmed that the MASS and PROM of the proximal interphalangeal joint for extension of his fourth finger were restored to an original condition. The duration of the effect with MAB in this case was thought to be 6 weeks in all. The comprehensive assessment of the risk factors, which involved his pressure sores, including the finger spasticity and the nutritional status, was essential to prevent the recurrence of the pressure sores. The condition of the patient was assessed every month during the outpatient follow-up visits after the last discharge.

Case 2

A 64-year-old man with a chronic right hemiplegia was admitted to our hospital for spasticity control. Except for ascending and descending stairs he was almost completely independent for his daily living activities. His Barthel index score was 85 points on admission. He complained that severe elbow spasticity always disturbed his walking, produced a slow gait, and caused an unstable posture when standing. The MASS for his right elbow was 3, and the PROM for extension was restricted to 60 degrees. A gait analysis revealed that during walking his right arm was pressed onto his anterior chest due to a severe spasticity at the right elbow (Fig. 3).

This patient received treatment with MAB for his right biceps and brachioradialis muscles every third day, for a total of 10 injections. The amount of ethanol was 1 ml for each muscle, per injection. The treatment reduced his right elbow spasticity, the MASS changed from 3 to 2, and the PROM for extension was improved to 120 degrees. After a series of treatments with MAB the increase in the PROM was 60 degrees. The severe spasticity of the

Fig. 3 The change in the right arm position of the patient in case 2 is shown before (left) and after (right) MAB. His right elbow spasticity was controlled, and his chest was not compressed by his right arm, after MAB.
patient’s right elbow was successfully controlled with the MAB, and side effects were not observed. He was comfortable during walking and without compression of his chest by his right arm. After discontinuing the MAB, his elbow spasticity gradually became worse. Two months after the treatment with MAB, we confirmed that his right elbow had returned to the original condition observed in an outpatient visit. The duration of the effect in this case was considered to be 8 weeks. The patient believed that he required an additional MAB treatment several months after the first treatment. The second MAB treatment also produced results identical to the first treatment, and the patient was pleased because the spasticity of his right elbow was under control.

Discussion

The purpose of this case report was to identify the characteristics of the MAB in each case. The mechanism of MAB has been discussed in the studies of other investigators. Lijestrand and Magnus reported that an injection of procaine into the triceps brachii of the decerebrate cat nearly abolished the rigidity of the muscle without altering its response to electrical stimulation of brachial plexus. Walsh reported that intramuscular procaine injections eliminated the rigidity while preserving the voluntary muscle power. This was later shown to be mediated by a blockade of either muscle afferents or gamma motor efferents. It has been postulated that the prompt effect of MAB is due to its action on the injected muscle via a sodium channel blockade of gamma motor fibers, and that the longer lasting effect is through alpha motor denervation after repeated treatments. We observed in case 1 that the muscle tonus was reduced immediately as the MAB injection was repeated, and the treatment resolved the difficulty of gaining access to the skin lesions. The expression of the first effect after treatment with BTX-A has been reported as not earlier than 2 to 3 days after an injection, and the peak effects were reported to be between 2 to 6 weeks post-injection. It is considered that the first effect of MAB is earlier than that of BTX-A. For this reason MAB may be a good choice if a rapid reduction of muscle tonus is required. The reported disadvantages and side effects of a neuromuscular block with phenol, or alcohol, are the complicated technique and the possibility of sensory disturbance, for example, dyseaesthesia or neuralgia. A neurolytic agent is usually injected into nerve fibers near the motor point and using neuromuscular stimulation for guidance. In contrast, MAB is injected into a muscle without neuromuscular stimulation. Among the other advantages of MAB are its simple technique and the possibility of less risk of any sensory deficit than when a conventional nerve block with phenol or ethanol is used. The reported duration of the effects of treatment with BTX-A has varied between 10 weeks and 4 months. In our case 1 and 2, the duration of the effects with MAB was approximately within 2 months. This result suggests a disadvantage in using MAB because its effect may be relatively shorter than BTX-A.

Following the present report, we recommend that additional investigation should be conducted into the management of upper limb spasticity using MAB through large prospective, double-blind and placebo-controlled studies.

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