Three Cases of Reflex Sympathetic Dystrophy in the Lower Extremity Treated with Lumbar Sympathetic Ganglion Block

Kazumichi YAMADA, Kazuo USHIJIMA, Masafumi TASHIRO, Kumi MATSUYAMA, Yasuyuki KAKIUCHI and Hisayoshi MIYAZAKI

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Reflex sympathetic dystrophy (RSD) is defined as follows: continuous pain in a portion of an extremity after trauma which may include fracture but does not involve a major nerve, associated with sympathetic hyperactivity[1]. This is a case report of three patients suggestive of RSD in the lower extremity, treated with lumbar sympathetic ganglion block (LSGB).

Report of the Cases

Case 1: A man of sixteen years, weighing 60 kg, had a light traffic accident merely causing his left knee to be sprained. Since then, he had suffered from a continuous pain of the left knee. The arthroscopy of the left knee joint, performed approximately one month after the accident under subarachnoidal block using 0.3% dibucaine, showed no significant pathogenesis of the pain. Following this arthroscopic examination, the pain gradually spread over his left leg from the knee.

On his first visit to our clinic about five months after the traffic accident, the following findings were observed in the left leg: a sustained, intractable and burning pain; hyperesthesia in the inner side of the calf; hyperpathia with a trigger zone in the knee; clammy skin with atrophy, loss of hair, and coldness; muscular atrophy; stiffness of the knee joint; and dystrophy of the femur, tibia and fibula detected by roentgenogram. The pain had caused him a gait disturbance due to inability to put the foot on the ground. The plantar core temperature in the left was 4.3°C lower than that in the right. The right and left circumferences of the legs were 38.5 and 35.0 cm at 10 cm above the patella, and 34.5 and 31.0 cm at 10 cm below the patella, respectively. The dystrophic change in the left leg is demonstrated in figure 1.

With informed consent, the left LSGB was performed six times at an interval of one week under radiographic guidance. Intramuscular atropine (0.5 mg) and hydroxyzine (50 mg) were given as premedication 30 min prior to the block. Figure 2 shows the relationship of position between the needle and the vertebrae in the left LSGB. In confirming that no blood was aspirated, a 10 ml of 1% lidocaine was given slowly to block the second lumbar sympathetic ganglion. The bilateral plantar core temperatures were measured continuously (TERUMO coretemp CTM-205^{TM}) before and throughout the block as an indicator of the effect of LSGB.

The almost complete relief of pain in the left leg was obtained immediately after each LSGB. This relief of pain continued for two to three days in the early stage; in the later LSGB, the pain relief lasted longer. After the

Department of Anesthesiology, Kumamoto National Hospital, Kumamoto, Japan

Address reprint requests to Dr. Yamada: Department of Anesthesiology, Kumamoto National Hospital, 1-5 Ninomaru, Kumamoto, 860 Japan

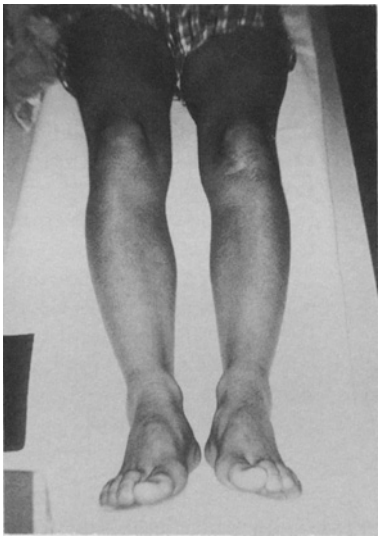

Fig. 1. The legs of the case 1. The apparent dystrophy of the left leg is shown.

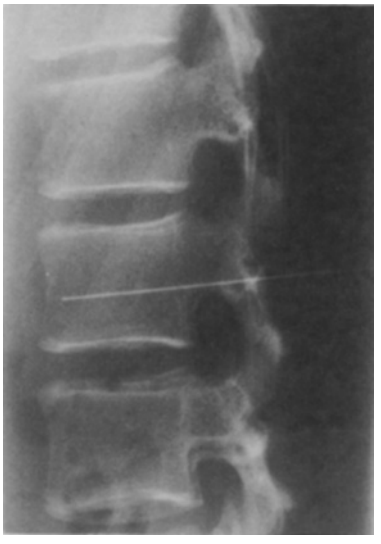

Fig. 2. Representative roentgenogram in the case 1, showing the relationship of the position between the needle and the vertebrae in the left sympathetic ganglion block. The needle is positioned through just under the transverse process of the second lumbar (L_2) vertebra, and the tip is nearby the front edge of the L_2 vertebral body, where the sympathetic ganglion lies.

third LSGB, the patient could walk using a stick putting the left foot on the ground. Following the last LSGB, there had been a lot of improvement in each of the symptoms, i.e., the disappearance of hyperesthesia, the increase in skin temperature, the enlargement of mobility of the knee joint, and the restoration from muscular atrophy.

Figure 3 shows the changes in plantar core temperatures induced by the left LSGB in the case 1. Before LSGB, the temperature in the left side was 3.3 ± 0.8°C (mean ± SD) lower than that in the right. The left plantar core temperature reached to the maximum approximately 15 min after the administration of lidocaine; it rose significantly to 34.4 ± 1.6°C from 27.4 ± 1.7°C before the block as compared using a one-way analysis of variance followed by the Bonferroni t-test ($P < 0.05$). There was no significant thermal change in the right side.

Case 2: A woman fifty-one years old, 48 kg in weight, had an operation of bilateral vein stripping for the varices in the lower extremities under epidural anesthesia using 2% lidocaine without any complications. Since the early postoperative days, she had complained of pain in the entire right leg. Oral analgesics and suppositories given for the pain were not so effective. She visited our clinic about eight months after the operation.

The pain was continuous and burning in the entire right leg being out of accord with the operative scar, and aggravated by movement such as walking and relieved by immobilization. A definite trigger zone was not recognized. For the associated symptoms with pain, there were hyperesthesia (not only in the right leg but also in the whole body), clammy skin with coldness and hyperhidrosis, edema, dystrophy of the nails, and rigidity of the knee joint. Though the operation was performed on both the legs, these findings were limited to the right. Muscular atrophy was not seen. The plantar core temperature in the right was 2.9°C lower than that in the left.

The right LSGB was weekly carried out nine times by a similar method to that in the case 1. The right plantar core temperature rose significantly by 6.0 ± 0.9°C during the

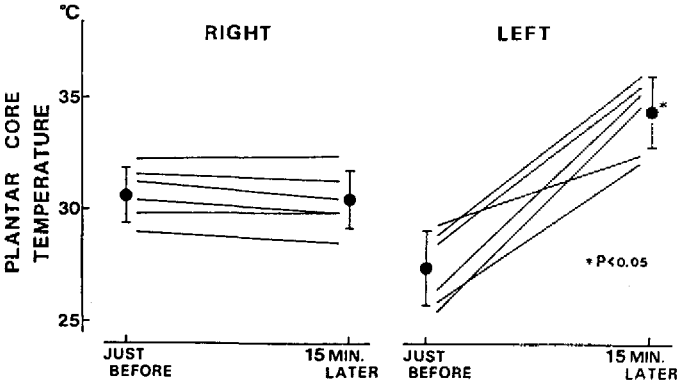

Fig. 3. Changes in the plantar core temperatures induced by the left lumbar sympathetic ganglion block (LSGB) in the case 1. The mean values with standard deviations are also shown. The temperature in the left side rose significantly to 34.4 ± 1.6°C (mean ± SD) 15 min after LSGB from 27.4 ± 1.7°C just before LSGB ($P < 0.05$). No significant thermal change was observed in the right side. Before LSGB, the temperature in the left was 3.3 ± 0.8°C lower than in the right.

LSGB ($P < 0.05$). The early LSGBs relieved the pain completely for three to four days. Moreover, the last LSGB brought her not only the lasting relief of the pain but also the remarkable improvement of the associated symptoms. Normally the right leg became warmer rather than the left.

Case 3: A sixty-year-old man, weighing 55 kg, had a pain limited in the right ankle. However, there could not be found out any significant traumas that might induce the pain. He underwent many kinds of both invasive and noninvasive examinations such as magnetic resonance imaging, bone scintigraphy, myelography, femoral angiography, etc., all of which showed no pathological changes. The pain increased gradually in degree and range.

He visited us ten months after the onset of the pain. The following findings were observed then around the right ankle: a burning and continuous pain exacerbated by movement or cutaneous stimulation causing a gait disturbance, hyperesthesia, hyperalgesia with a trigger zone in the big toe and instep, edema with cyanosis in the entire foot including the toes, deformity of the nails, atrophy of the calf and the thigh, and stiffness of the ankle. These were strongly suggestive of RSD, although he had no history of trauma and the higher plantar core temperature in the right by 2.0°C rather than that in the left in contrast to the other cases.

As the mode of the onset of pain was not typical, a continuous lumbar epidural block was initially attempted for one week. However, the effect of the epidural block was insufficient. The right LSGB was then performed four times similarly to the case 1. Even four times of the LSGB failed to lessen the pain. No significant increase in the plantar core temperature was detected during the LSGB. The patient has been treated with an α_1-blocker (bunazosin), prostaglandin E_1, an antidepressant (amitriptyline), an analgesic (indomethacin), a kampo medicine (Keishibukuryou-gan), and physical therapy. These have improved slightly the mobility of the ankle and the ability in walking. Still the pain and most of the associated symptoms continue with little changes for eight months since the last LSGB.

In all cases, there was no evidence of any systemic disorders that might result in neuropathy such as diabetes mellitus, and the laboratory examinations showed the normal values. Physical therapy begun simultaneously with LSGB has been proceeding. No significant complications occurred with the LSGB except in the case 2, in which a transient hyperventilation syndrome was observed once after the sixth LSGB, recovered with a sedative (5 mg of diazepam).

Discussion

The common trigger of RSD is a minor and nonspecific trauma, such as a light traffic accident and the following arthroscopic examination as in case 1, and as an operation of vein stripping in case 2. On

the other hand, case 3 had no history of trauma suggesting that various kinds of diseases other than trauma might also induce RSD-like symptoms. The possible ones are thrombophlebitis, vasospastic arterial disease, postfrostbite syndrome, postphlebetic edema, postthrombotic painful edema, and so on[2].

The onset of RSD is usually weeks after the injury, while in some instances it is immediate following the trauma. Although the exact incidence of RSD caused by surgical procedures is unknown, we should notice and treat it in the early stage. Early sympathetic nerve block may be useful. Furthermore, if early, even more local anesthesia in the trigger zone or in the painful region is often effective to relieve or to prevent worsening the symptoms[3]. In our cases 1, 2 and 3, the intervals between the onset of pain and the first LSGB were about 5, 8 and 10 months, respectively. These differences might partly influence the effects of LSGB, i.e., the poorest in case 3.

The mechanism of RSD is regarded as follows. The noxious stimuli from the injured region are initially sent to the spinal cord through the sensory afferent neurons, then the stimuli from the same level of spinal cord are reflexly returned to the injured region through the efferent sympathetic fibers. In consequence, the impulses are continuously sent to the sensory center[4]. Some theories to explain this mechanism have been presented; 1) failure of the gate control system in the dorsal horn[5], 2) formation of the vicious cycle of reflexes through interneurons in the lateral and ventral horns[2], 3) artificial synapse connecting the efferent sympathetic and the afferent sensory fibers[6-8], and 4) sensitization of the wide-dynamic-range neurons[9,10]. The effects of sympathetic nerve block like LSGB on RSD may be brought by cutting the efferent sympathetic stimuli concerning the mechanism. Therefore, sympathetic block can be employed not only to treat a pain but also to diagnose it as RSD or the related pain. Early regional anesthesia may block the afferent sensory stimuli and prevent forming the vicious cycle or the artificial synapse.

As a supplementary therapy, several medicines like α-blockers, Ca-blockers, antidepressants and anticonvulsants have been used[11]. Intravenous sympathetic blockade with guanethidine or reserpine is occasionally useful[12]. Physical therapy is also important and effective to improve the peripheral circulation and to avoid the stiffness of joints. However, it may aggravate the pain by movement if the control of pain is insufficient.

Our case 3 showed the most obvious findings of vasomotor dysfunction, i.e., edema, cyanosis, etc. In such a case, local metabolism might be impaired and some substances to induce pain might be produced. This may result in the further exacerbation of the vicious cycle of pain[2], and the activation of interneurons may soon get permanent, lowering the effects of sympathetic or sensory nerve block.

The hyperventilation syndrome observed in case 2 suggests that psychiatric factors may partly play a significant role in RSD[13].

In summary, three patients suggestive of RSD in the lower extremity were treated with LSGB. In two cases, LSGB was very effective and showed the marked increase in the plantar core temperature. It is important to detect and treat RSD in the early stage.

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References

1. The International Association for the Study of Pain Subcommittee on Taxonomy: Classification of chronic pain: Descriptions of chronic pain syndromes and definitions of pain terms. Pain 26 (suppl. 3):S29–S30, 1986
2. Bonica JJ: Causalgia and other reflex sympathetic dystrophies, The Management of Pain. Philadelphia, Lea & Febiger, 1953, pp. 913–987
3. Hyodo M: Reflex sympathetic dystrophy: Definition and diagnostic criteria. Pain Clinic 10:568–574, 1989
4. Roberts WJ: A hypothesis on the physiological basis for causalgia and related pains. pain 24:297–311, 1986
5. Melzack R, Wall PD: Pain mechanism: A

new theory. Science 150:971–979, 1965
6. Livingstone WK: Pain mechanisms, A Physiologic Interpretation of Causalgia and Its Related States. New York, Plenum Press, 1943, pp. 83–127
7. Barnes R: The role of sympathectomy in the treatment of causalgia. J Bone Joint Surg 35:172–180, 1953
8. Sternschein MJ: Causalgia. Arch Phys Med Rehabil 56:58, 1975
9. Roberts WJ, Foglesong ME: Spinal recordings suggest that wide-dynamic-range neurons mediate sympathetically maintained pain. Pain 34:289–304, 1988
10. Roberts WJ, Foglesong ME: Identification of afferents contributing to sympathetically evoked activity in wide-dynamic-range neurons. Pain 34:305–314, 1988
11. Miyazaki T, Tokuda H: Reflex sympathetic dystrophy: A point of view and change of its treatment. Pain Clinic 10:575–580, 1989
12. Hannington-Kiff JG: Intravenous sympathetic blockade with guanethidine. Lancet 1:1019–1020, 1974
13. Wiesenfeld-Hallin Z, Hallin RG: Possible role of sympathetic activity in abnormal behavior of rats induced by lesion of the sciatic nerve. J Auton Nerv Syst 7:385–390, 1983