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Effect of Surface Spinal Stimulation on Spasticity in SCI: A Single Case Study

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ABSTRACT

Spasticity forms one of the major complications after a spinal cord injury. Various treatment approaches are used for its reduction. Surface spinal stimulation has been used in this case and its effect pre- and post-treatment have been compared. A 27-year-old male suffered a spinal cord injury of the level C4-C5 due to a bus accident, in which the bus had overturned. The patient is a follow-up case of C4-C5 discoligamentous injury with quadriparesis with bowel and bladder involvement. The patient developed spasticity in his upper and lower limbs owing to the spinal cord injury. He has been taking physiotherapy treatment for the same. Due to the spasticity, he is suffering from problems in maintaining a comfortable posture and complains of spasms. When assessed with modified Ashworth scale for spasticity, the patient scored 4 for both left and right dorsiflexors of the lower limb. The patient was given surface spinal stimulation for reduction in spasticity and at the end of 15 days, the spasticity rating was 1 and 2 for left and right limbs respectively. Surface spinal stimulation has proved to be an effective tool for reducing spasticity after spinal cord injury. It helped the patient in being more comfortable and he was able to participate in his exercises more actively. The patient was given a session for 45 minutes each day for five days a week for fifteen days and MAS and functional outcomes of spinal cord injury measure, adductor tone rating scale, spasm frequency were recorded on day 0, day 8, and day 15. The treatment session consisted of application of two pad electrodes (5 × 9 cm) at paravertebral region at the level of T10-L2 vertebrae. There was significant reduction in the score of MAS in the individual and, therefore, it can be said that spasticity was reduced.

Keywords: *Spinal cord injury, spasticity, modified Ashworth scale (MAS), surface spinal stimulation*

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1. BACKGROUND

More than two decades ago, spinal cord injury (SCI) meant being limited to a wheelchair for life and being dependent on others. The treatment choices were limited to a few and the provision of care for an individual with spinal cord injury was difficult and caused frustration. With recent advances in neurosciences, new options are being laid out that are helping improve the lifestyle of these individuals. New advances are promising and researchers are looking at various ways that can enable the patient to be able to lead a normal life.

Spinal cord injury claims a huge number of healthy individuals every year. It is not limited to a specific area, but affects the population globally. According to a survey done in 2004 by the Rick Hansen Institute, it is clear that the population of people living with SCI is steadily increasing around the world [1]. According to Indian statistics, about 20% of all spinal injuries result in a neurological deficit in the form of paraplegia in the thoraco-lumbar spine injuries, or quadriplegia in the cervical spine injuries [2].

Spinal cord injury is the term used for an injury caused to the cord by means of a traumatic incident. The level at which the

spinal cord and nerve roots are damaged, cause a variety of symptoms to arise ranging from pain to paralysis to incontinence [3, 4]. The most common causes of spinal cord injury are road traffic accidents and falls from height [5, 6]. Injuries at the level of the cervical spine usually result in full or partial tetraplegia (quadriplegia), and those at or below the thoracic spinal levels result in paraplegia [2].

The injury progresses in a primary and secondary fashion. The initial mechanical trauma is usually caused by traction and compression forces. The fractured and displaced bone fragments, disc material, and ligaments cause further injury to the neural elements affecting both the central and peripheral nervous systems. The damage can be extensive affecting blood vessels, disruption of axons, and neural-cell membranes. Within a very short span of time, microhaemorrhage occurs in the central grey matter and in due course of time it extends radially and axially. As a consequence, the spinal cord swells to occupy the entire diameter of the spinal canal at the level of the injury causing secondary ischemia. These set of events cause a disruption in the autoregulation of the blood flow leading to spinal neurogenic shock.

A vicious cycle is formed and ischemia is caused. Due to ischemia, toxic chemicals are released from the disrupted neural membranes and the subsequent electrolyte shifts trigger a secondary injury that greatly multiplies the initial mechanical damage by harming or killing neighboring cells. Thus the injury causes hypoperfusion in grey matter [7] and extends to the surrounding white matter. This hypoperfusion reduces or completely blocks propagation of action potentials along axons, leading to the development of spinal shock.

The injury is not the end in itself as it has a set of events that follow in the form of secondary complications which develop easily after a spinal cord injury; some of the commonest are pressure ulcers, contractures and spasticity.

Spasticity is present in up to 80% patients with spinal cord injury [8]. The increase in spasticity may be due to neural sprouting or changes in the sensitivity of neural receptors. Spasticity can be elicited with many stimuli but stretch and touch are the most common [9].

Many tests are used to quantify spasticity but the two most commonly used are the Tardieu scale [10] and the Modified Ashworth scale [11, 12]. The neurophysiology of spasticity is complex and not fully understood. It can have many features but the two key features are the abnormal and velocity-dependent increase in resistance to stretch [13]. The main implications of spasticity are that it predisposes patients to pain, contractures and pressure ulcers, and makes movement and hygiene difficult [8, 9, 14]. For some patients, spasticity limits function and quality of life [15].

Spasticity is known to be one of the symptoms resulting from injury to the upper motor neurons within the central nervous system (CNS) [16, 17]. The most commonly cited definition for spasticity is that published by Lance in 1980 [18]: Spasticity is a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motoneuron syndrome. Decq has recently suggested the use of a modified definition, whereby "spasticity, in general, is defined as a symptom of the upper motor neuron syndrome characterized by an exaggeration of the stretch reflex secondary to hyperexcitability of spinal reflexes" [19].

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Spasticity is primarily managed with pharmacological agents [20]. Two main categories of drugs are used, of which some act predominantly within the central nervous system (e.g., baclofen, diazepam, gabapentin, clonodine, tizanidine) and the others act peripherally, either within the muscle or at the neuromuscular junction (e.g., dantrolene sodium and botulin toxin). Although medical intervention may seem to be a good intervention option but the effect of the drug is short lived and the dosage has to be repeated. Long-term use of these drugs has its own adverse effects. Some include withdrawal symptoms like hallucinations, dizziness, nausea, psychosis and mania. Others are tachycardia, autonomic dysreflexia, tremors and hyperpyrexia [21].

Botulin toxin has several adverse effects which may include double vision, allergic reactions, fatigue, and muscle paralysis [22]. Apart from the pharmacological interventions, there are a set of known physiotherapy modalities which promise to produce relief from spasticity, but there is not much evidence if the effects of these modalities is long lasting enough. These physiotherapy interventions include hydrotherapy, static stretching, heat and cold therapies, electrical stimulation and TENS, therapeutic exercises, passive movements, weight bearing in standing and vibration [20, 23].

Therefore, the physiotherapy interventions become a long process. Another upcoming treatment approach for spasticity after spinal cord injury is surface spinal electrical stimulation. It is a non-invasive technique and involves the application of electric currents superficially at the skin surface. Surface spinal stimulation (SSS) is the process of stimulating the spinal cord with various electrical currents in order to produce stimulation and reduction in spasticity at the corresponding level of body segment [24]. A study done by Wang and others [25] has concluded that it is an effective way of treatment and can become a potentially

important treatment method. Several other studies conclude the same.

2. CASE REPORT

A young male aged 27 years, resident of Ghittorni, New Delhi, met with an accident on 2nd February, 2009, while traveling in a bus. His present chief complaint was inability to walk and severe tightness in his both legs and tightness in arms. After the accident, he was taken to the nearest hospital at Sitapur from where he was referred to CSMU, Lucknow. He was unable to move any of his limbs after the accident. The CT scan and MRI revealed spinal cord injury at C4 C5 which was compressive in nature. The patient was diagnosed with C4-C5 discoligamentous injury with quadriparesis and bladder and bowel involvement. The patient was kept in the paraplegia ward in the same hospital for 50 days and he received sessions of massage daily for 7–8 h. When no improvement was seen, he was referred to AIIMS, New Delhi. He was then shifted to the Indian Spinal Injuries Centre (ISIC), New Delhi, on 5th January, 2010, where his treatment was begun at the rehabilitation unit. At the rehabilitation unit of ISIC, the patient received passive stretching for calves and hamstrings and quadriceps; also he was given stretching for thoraco-lumbar fascia. He did weight bearing on standing frame with the help of knee brace and lumbar corset. This was done in front of a full-length mirror so that he could see himself and try and do conscious correction of posture. For upper limb, the patient did weight bearing on both hands in sitting and passive stretching for hands' lumbricals and wrist 7 was done along with stretching of biceps, triceps and muscles of the forearm. To avoid deterioration in muscle status, the patient was doing passive cycling and was given passive exercises for upper and lower limbs. To improve sitting balance, he was receiving balance training in sitting on a physioball. Apart from this, he was doing exercises for improving functional status, which included, turning sides on the bed, sitting up from lying, and training for wheelchair transfers.

On examination, the vitals of the patient were found to be normal, except that the body temperature of the patient 100 °F. The fever was a result of a urinary tract infection. The patient did not have any complaint of pain of any character except for a dull mild pain in his left shoulder; the pain was greater during the day and reduced during the night. No skin abnormality was observed; the skin was healthy and intact; and the patient had old scars of an injury on his left knee. The patient was able to sit straight in the wheelchair, also he could sit with support on an inclined bed. He had developed flexion deformity in the fingers of his right hand due to spasticity. He had no tenderness and swelling was absent. He was alert and well oriented to time, place and person. No abnormality was observed in his higher mental function, cranial nerves and primary sensations. Graphesthesia could not be checked because of flexed hands. The deep

tendon reflexes were exaggerated and Babinski sign was positive. The patient had frequent flexor spasms, which were more during the day than at night. The flexor spasms were caused on sudden turning or noxious stimuli to the feet. There was no limb length discrepancy in the lower limbs of the patient.

The patient was given treatment comprising of surface spinal stimulation along with the ongoing physiotherapy treatment. The instrumentation involved was: IFC: Carrier frequency 2500 Hz, beat frequency 20 Hz. The surface electrodes were placed paravertebrally (5 cm apart, size of electrodes 4.5 × 9 cm) over the T10 to L2. The stimulus produces only a sensory stimulus and should not produce any muscle contraction [25]. The surface spinal stimulation session lasted for 45 min each for the patient for 15 days. The frequency of the session was once a day and five times a week. The patient was given stimulation on the hospital bed itself. During the 45 min period, the patient was not supposed to move in order to avoid loss of contact between the electrodes and skin surface. The recording of the variables (modified Ashworth scale (MAS), adductor tone rating (ATR), spasm frequency (SF), spinal cord independence measure (SCIM)) was done on 0 day, 8th day and the 15th day. The assessment readings for day 0 were: MAS (L) 4, MAS (R) 4, ATR 3, SF 2, SCIM 15. The treatment protocol as mentioned above was followed and readings were again noted on the 8th day. These were MAS (L) 2, MAS (R) 3, ATR 2, SF 1, SCIM 17. The same treatment protocol was continued and readings were again noted on the 15th day. These were MAS(L) 1, MAS(R) 2, ATR 2, SF 1, SCIM 22. A significant change was observed in the assessment outcomes of the patient. The patient was now able to move the wheelchair to a greater distance in lesser time as compared to earlier; this was noted on SCIM.

3. DISCUSSION

This case involves the implementation of surface spinal stimulation which was aimed at reducing spasticity of the patient. Many studies have been done so far to prove the effectiveness of spinal stimulation in spasticity in patients with stroke [25–27]. The present study deals with the problem of spasticity in SCI and effectiveness of spinal stimulation. Spasticity is conservatively managed with drugs and the conventional methods of physiotherapy that were being used for this patient at ISIC, but all these methods of physiotherapeautic approach are highly time consuming and largely depend on the therapist's ability and dedication. Also, they are a cause of strain to the therapist as they demand great physical activity and a lot of energy for correct implementation. The study was done based on the readings of the patient's functional outcomes for duration of 15 days. The treatment comprising of spinal stimulation was done and in the readings for 0, 8 and 15 days, a significant difference in the readings of all the variables was found. The

spasticity as rated on MAS showed reduction from day 0 to day 15 which was the main aim of the treatment. There was also reduction in the spasm frequency which helped the patient to be more comfortable. The adductor tone of the patient also reduced significantly. The readings of SCIM improved after the 15th day, sessions of treatment, especially that the patient was able to move in his wheelchair without any assistance for a short distance.

4. CONCLUSIONS

Each year SCI claims the independence of many individuals. More than the trauma of the spinal cord injury and the handicap that it leaves behind, it is the secondary complications that become the cause for pain and discomfort for the patients. Of all the secondary complications that develop in the patient, the most common and difficult to deal with is spasticity. The study successfully concluded that spinal stimulation produces significant difference in the functional outcomes of the patient. Therefore, it can be concluded that surface spinal stimulation is an effective method to tackle the problem of spasticity. Also, it helps the physiotherapist deliver treatment and exercises more effectively and makes the patient more comfortable.

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