International Journal of Allied Medical Sciences and Clinical Research (IJAMSCR)

ISSN:2347-6567

IJAMSCR |Volume 10 | Issue 4 | Oct - Dec - 2022
www.ijamscr.com

Research article *Medical research*

A Study Comparing the Effect of Nerve Flossing and Conventional Therapy with Only Conventional Therapy in Treating Sciatica

M. Raja Srinivas[1*], G. Kalyan [2], J. Hari Kishore Babu[3], J. Bhaskar Rao[4], T. Sunil Kumar[5]

[1]Department of Physiotherapy, Nanded Physiotherapy College & Research Institute, Nanded, Maharashtra, India.
[2]Department of Physiotherapy, Tirumala College of Physiotherapy, Nizamabad, Telangana, India
[3]Department of Physiotherapy, Navodaya College of Physiotherapy, Raichur, Karnataka, India
[4]Department of Physiotherapy, Anji Reddy College of Physiotherapy, Piduguralla, Andhra Pradesh, India .[5]Department of Physiotherapy, Nanded Physiotherapy College & Research Institute, Nanded, Maharashtra, India.

Corresponding Author: M. Raja Srinivas

ABSTRACT

Low back pain (SCIATICA) is a very common health problem world-wide and a major cause of disability affecting performance at work. Sciatica along with back pain accounts for more medical care and social cost which affects quality of life in most of the patients. To compare the effect of nerve flossing and conventional therapy with only conventional therapy in sciatica. A RCT was conducted on 30 subjects of age group 35 – 50 years, fulfilling the inclusion criteria. Pain intensity was measured by VAS and sciatica involvement assessed by SLR test. T test was used to comparison between them. The mean of VAS of pre-treatment for group A was 7.73 and for group B was 7.6 with P value 0.7203 which was considered as not significant. The mean of VAS of post treatment for group A was 7.26 and for group B was 2.26 with P value <0.0001 which was considered as extremely significant. The mean of ROM of pre-treatment for group A was 49.6 and for group B was 48 with P value 0.6709 which was considered as not significant. The mean of ROM of post treatment for group A was 50.8 and for group B was 61 with P value <0.0195 which was considered as extremely significant. Nerve flossing technique can be utilized with other modalities in the treatment of subacute sciatic patients due to low back ache for the relief of pain and sensory symptoms like tingling and numbness, restoration of spinal mobility and to minimize functional disability.

Keywords: VAS, ROM, Nerve flossing, Sciatica

INTRODUCTION

Low back pain (LBP) is experienced in 60%–80% of adults at some point in their lifetime. Andersson[1] estimated the annual worldwide LBP incidence in adults to be 15% and the point prevalence to be 30%. Papageorgiou et al.[2] stated that at least 50% of adults would have experienced an LBP episode. Some studies have demonstrated that LBP is one of the most common cause of visits to a physician[3] and that men and women are equally affected by LBP.[4]

The literature shows that 30% of adolescents worldwide experience at least one LBP episode.[4] Various studies found that LBP is a very common problem among adolescents, with an incidence that is the highest in the third decade of life.[5] Some authors proposed that LBP in young adults and children may occur because of growth spurts and increased physical activity.[6] In contrast, Fairbank et al.[7] revealed that students with back pain were more likely to be sports avoiders than their counterparts who were involved in sports. Young adults who experienced LBP at the age of 14 years had an increased incidence 25 years later compared with those who did not experience LBP at age 14 years.[8] Therefore, preventing and avoiding LBP during early adolescence can prevent LBP progression, and thus, can decrease the associated morbidities.

However, to prevent LBP, the associated modifiable and non-modifiable risk factors must be identified. Previous studies have demonstrated that high body mass index (BMI) is associated with an increased LBP incidence.[9] In addition, Webb et al.[9] revealed that hereditary plays a vital role in LBP occurrence and that a positive family history has a strong correlation with LBP incidence.[10] Risk factors for LBP are not limited to physical factors; psychosocial factors such as stress, anxiety, depression, and monotony are also potential risk factors for LBP.[11] These risk factors can result in the progression from an acute LBP episode to a chronic problem. Low back pain (LBP) is described as a very common condition that tends to affect about 70% of the population at some point in time with varying degrees of symptoms severity.[12] Low back-related leg pain or sciatica is one of the commonest variations of LBP.[13] Sciatica is known by a range of terms in the literature, such as lumbosacral radicular syndrome, radiculopathy, nerve root pain and nerve root entrapment or irritation. There is controversy in clinical and research circles about the use of sciatica as a term (as it is not thought to be representative of the nature of leg pain which is due to lumbosacral nerve root involvement) and it is strongly suggested that it should be replaced by the term nerve root pain or radiculopathy which is much more accurate and explanatory of the presenting condition.[14,15] Of all LBP presentations, sciatica is readily recognized in most cases in clinical practice. Although definitions of sciatica used in epidemiologic surveys vary, sciatic pain is generally defined as pain radiating to the leg, normally below the knee and into the foot and toes. It tends to approximate the dermatomal distribution of the nerve root affected (most often L5, S1) and is often associated with numbness or pins and needles in the same distribution.[16] Further clinical findings of neurologic deficit such as muscle weakness and reflex changes may also be present. As with LBP, sciatica is a symptom rather than a specific diagnosis, but lumbar disc herniation and lumbar canal or foraminal stenosis are typical pathologies that may cause sciatic pain. There are also some rare reasons for sciatica such as tumours, cysts or other extraspinal reasons. In most cases, the main cause of symptoms is believed to be inflammatory changes resulting from irritation or compression of the affected nerve root by its surrounding tissues.[17] Although it had been generally believed that the majority of patients with sciatica have a very favourable outcome and natural resolution of symptoms, literature indicates that they have a more persistent and severe type of pain than LBP patients, a less favourable outcome, consume more health resources and have more prolonged disability and absence from work.[18,19] Individual factors that influence the incidence of lumbar pain and sciatica have been studied in detail. To date, few studies have been able to note factors that increase risk of sciatica. Sex, age, anthropomorphic, and body posture have not been shown to correlate with incidence of disease. However, age does affect the incidence of lumbar disc degeneration. Strength and aerobic capacity improve resilience to, but not incidence of, sciatica.[20] Recent articles support the association of manual labour with an increased incidence of low back pain and sciatica. Among patients hospitalized with sciatica, the risk was greatest for bluecollar workers and motor vehicle drivers, and lowest for professional groups.[21] Among bluecollar workers, the type of work and history of back accidents increased the future risk of sciatica.[22] For example, among construction workers, concrete reinforcement workers had an increased risk of sciatic pain. Also, the incidence of sciatica increased with age depending on the number of previous back accidents. A study comparing machine operators and carpenters with office workers showed an adjusted risk ratio of sciatica of 1.4 and 1.5, respectively.[23] Previous low back pain increased the risk fourfold. Recent studies support the role of heredity in the incidence of sciatica. A study by Simmons[24] notes that in a population of patients undergoing surgery for lumbar degenerative disc disease, 45% had a positive family history, whereas 25% of the control group had a positive family history of lumbar disc disease. The family rates of spinal surgery were 18.5% and 4.5%, respectively. Studies of low back pain in children indicate a doubling of the risk of reported back pain in children of parents with back pain, although other studies showed less impressive increases. A study by Richardson[25] points to the role of heredity in sciatica. A questionnaire regarding lumbar disc disease incidence was completed by immediate relatives of patients with surgically proven lumbar disc herniations. Twenty-eight percent of respondents (versus 2% of controls) met the criteria for lumbar discogenic pain. While none of the controls had prior disc surgery, 12% of the study patients had prior lumbar disc surgery. A history of heavy lifting also correlated with the incidence of discogenic pain. Lumbar disc herniation is a multi-factorial problem and its aetiology is still an enigma. Patients with lumbar disc herniation are commonly seen in day-to-day clinical practice and a majority of these patients respond to non-operative methods of management and rarely require any form of surgical intervention.[26] The disease burden has been seldom documented in literature in India. According to certain studies, the prevalence of lumbar disc herniation has been estimated as 1%-3%. The clinical symptomatology has been largely restricted to middle age group, among people aged between 30-50 years.[27] Although the reason for symptomatic improvement remains elusive, Haro et al.[28] proposed a local inflammatory process in the epidural space which possibly stimulates host macrophages to resorb the displaced disc tissue, this has been contemplated as a probable reason. It has been suggested that the probability of symptoms resolving with conservative treatment decreases progressively with time.[29] In many studies prolonged morbidity has been regarded as a negative predictor.[30] However some have contradicted this impression.[31] However, it is potentially dangerous to carry out conservative treatment in all patients with herniated discs, especially because many reports indicate that patients with long standing pre-operative symptoms have fewer chances of obtaining satisfactory results from surgery than those whose symptoms are of short duration.[32] It is an important distinction to know that most cases of sciatica result from an inflammatory condition leading to an irritation of the sciatic nerve. Conversely, direct compression of the nerve leads to more severe motor dysfunction which is often not seen, and if present, would warrant a more meticulous and expeditious workup.[33-35] Any condition that may structurally impact or compress the sciatic nerve may cause sciatica symptoms. The most common cause of sciatica is a herniated or bulging lumbar intervertebral disc. In the elderly

population, lumbar spinal stenosis may cause these symptoms as well. Spondylolisthesis or a relative misalignment of one vertebra relative to another may also result in sciatic symptoms. Additionally, lumbar or pelvic muscular spasm and/or inflammation may impinge a lumbar or sacral nerve root causing sciatic symptoms. A spinal or paraspinal mass including malignancy, epidural hematoma, or epidural abscess may also cause a mass-like effect and sciatica symptoms.[36,37] Sciatica symptoms occur when there is pathology anywhere along this course of the nerve. This pathology can be any of the conditions listed in the differential diagnosis.[38] Sciatic neuralgia is defined as 'pain in the distribution of the sciatic nerve due to pathology of the nerve itself'.[39] Radicular pain is defined as 'pain perceived as arising in a limb or the trunk caused by ectopic activation of nociceptive afferent fibres in a spinal nerve or its roots or other neuropathic mechanisms'. According to these definitions, sciatic neuralgia is clearly a form of radicular pain, and is described as a disease of the peripheral nervous system.[39] The term 'sciatica' may cause confusion as it has been used to describe any pain, including referred, felt in the leg along the distribution of the sciatic nerve. Indeed, the term has been described as 'an anachronism and should be abandoned'.[39] The ancient Greeks were familiar with sciatic neuralgia and used the term 'sciatica', to describe pain or 'ischias' felt around the hip or thigh. Hippocrates himself referred to 'ischiatic' pain affecting men between 40 and 60 yr. He observed that young men described pain that lasted about 40 days before resolving spontaneously. He also noted that pain radiating to the foot was a good prognostic sign, whereas localized hip pain was less likely to resolve.[40] The Italian anatomist Domenico Cotugno (1736– 1822) wrote the first book on sciatica in 1764 and for many years it was known as Cotugno's disease.[41] By the 19th century, sciatica was thought to be due to a variety of rheumatic conditions causing inflammation of the sciatic nerve. However, early frustrations with difficulties in identifying a cause of and treating sciatica were expressed by Fuller in his book Rheumatism, Rheumatic Gout and Sciatica (1852). He stated 'the history of sciatica is, it must be confessed, the record of pathological ignorance and therapeutic failure'.[42] The intervertebral disc was first implicated as a causative factor in sciatica in the early 20th century. Schmorl94 and Andrae (1929)[43] described posterior disc protrusions seen at post-mortem studies, but did not link these with sciatic pain and concluded they were probably asymptomatic in life. In an early surgical management of sciatica, the neurosurgeon Eslberg (1931) described removal of cartilaginous 'tumours' from the spinal canal, with subsequent improvement of symptoms. He considered the possibility that these 'tumours' could in fact be prolapsed disc material. The concept of prolapsed disc material causing pain was later revisited by Mixter and Barr who reviewed the pathology of all excised chondromas of the spine held in the Harvard Medical School pathology museum, comparing them with normal disc material. They concluded that sciatica and neurological sequelae were due to protrusion of normal disc material. The presence of pain was initially ascribed to pressure on nerve roots. This idea was challenged by Kelly,[44] who felt that pressure on a nerve would lead to loss of function rather than pain; therefore, pain must arise by a different mechanism. Around the same time, Lindahl and Rexed[45] found evidence of an inflammatory response on lumbar nerve roots at laminectomy leading to the theory that prolapse of an intervertebral disc may provoke an inflammatory reaction in lumbar nerve roots, causing the sciatic type pain. Because of human movements, various types of mechanical stresses are putted on nerves, and the nerves can withstand these stresses. When the nerve subjected to compressive, tensile or shear forces that exceed its capacity, the circulation within the nerve and axoplasmic flow are obstructed and this leads to ischemia and impaired function.[46] Disc herniations, and stenosis of the spinal canal are the main causes of compressive stress that will hinder the flow of the blood to the nerve root.[47] Compressions of the nerve root can lead to both motor and sensory dysfunction.[48] Furthermore, compressions of the nerve root causes some changes in nerve microvascular circulation and re- lease of some inflammatory mediators leading to pain. Thus, adhesions are formed among the nerve root and the injured disc as a result of inflammation leading to entrapment of nerve root sliding. In addition, intraneural oedema, neural conduction block, and mechanical sensitization are associated with nerve root compression.[49] Various Physical therapy interventions as exercise, manual therapy, and electrotherapy have been used for treatment of lumbosacral radiculopathy.[50] One of the interventions used for treatment of lumbosacral radiculopathy is the neural mobilization technique which gained considerable attention among physical therapists. It aims to mobilize the peripheral neural tissue and the structures surrounding them thus influencing the mechanical properties of peripheral nerves.[51] Physical therapists used these techniques for management of different neural tissue compression disorders and other dis- orders that might include neuropathic pain to restore the mechanical function of impaired neural tissue.[52] The proposed effects and underlying mechanisms of neural mobilization technique associated with clinical improvements were based on theory rather than research evidence and remain unclear.[53] There are many theories that have been postulated, including enhance circulation within the nerve, axoplasmic flow, viscoelasticity of the- neural connective tissue, dispersion of intraneural oedema,[54] reduction of dorsal horn and supraspinal sensitization and promote nerve excursion.[55] Hoffmann reflex (H-reflex) is considered the electrical analogue of the monosynaptic stretch reflex. H-reflex serves as a reliable estimate of spinal level motoneuron pool activity and accurate investigation of nerve root activity. H-reflex is used for assessment of the peripheral nervous system in relation to conduction of the peripheral nerve and compression of the S1 nerve root. Assessment of the S1 nerve root function is the primary clinical application of the H- reflex such as radiculopathy.[56] According to Efstathiou et al 2015, and Ellis et al 2008 on their systematic review on the effect of neural mobilization, a definite conclusion about the effectiveness of neural mobilization on patients with radiculopathy can't be reached because of the lack of well-designed randomized controlled trial that could investigate the effect of neural mobilization in radiculopathy.[57,58]

METHODOLOGY

This Randomized control trial study was conducted in patients of low back pain (sciatica) fulfilling the criteria, referred by specialists for physiotherapy, from OPD of Tirumala College of Physiotherapy, Nizamabad, Telangana, India.
The study population was selected by simple random sampling method. The study was conducted over a period of one year.

Inclusion criteria for the study were:

- Subjects were age group of 35 –50 years.
- Both genders.
- Subjects experience neurological symptoms
- Subjects with unilateral involvement.

Subjects were excluded if:

- Subjects with systemic disease.
- Subjects with congenital deformity.
- Subjects having sciatica with vascular disorder and diabetic neuropathy.
- Subjects having sciatica due to tumour.
- Subjects with any psychosomatic and psychological disease.
- Any infection or inflammation of spine.

This study involved minimal equipment

1. Pen
2. Plinth
3. Traction Table
4. Data collection sheets

Low back pain (Sciatica) subjects were recruited from the OPD of Tirumala College of Physiotherapy, fulfilling the criteria referred by specialists for physiotherapy. After initial assessment the participants who met the inclusion and exclusion criteria were explained about the study. The procedure was explained to the participants; and were subjected to pre & Post clinical examination SLR Test was performed and noted down. Visual analogue scale was administered and responses noted down.

SLR test

SLR is also known as lasegue's test, it is one of the most common neurological tests of lower limb and is done by the examiner with the patient completely relaxed in supine position. The hip is flexed and adducted, knee extended and ankle dorsiflexed. If the patient complains of pain, then the test is positive. With the unilateral straight leg raising, the nerve roots L5, S1, S2 are normally completely stretched at 70 degrees. Pain after 70 degree is probably joint pain from lumbar area or sacroiliac joint. The subjects were included in the study if all the inclusion criteria were met and no exclusion criteria were found. 30 subjects were selected between the age group 35 to 50 years. The subjects were told all about intervention and procedural details to be followed in the study and thereafter consent was obtained. Hip Flexion Range of motion was measured using goniometer. A Visual Analog Scale was used for assessing the pain. Patients were conveniently allocated either to group A or to group B

Group A (n=15) Control Group

- Traction
- TENS

Group B (n=15) Experimental Group

- Traction
- TENS
- Sciatic nerve flossing

Before starting the intervention all the patients were checked for range of motion of SLR at the hip with the help of standard goniometer and pain with the help of Visual Analogue Scale. The control group (Group A) participated in a standard rehabilitation program or conventional physical therapy treatment for the disease, traction for 10 min (intermittent) with 1/3 of bodyweight with the patient in supine and hip and knee flexed to 90º. This was followed by High TENS for 15 min. The experimental group (Group B) participated in a standard rehabilitation program supplemented with neural mobilization program for sciatic nerve, traction for 10 min (intermittent) with 1/3 of body weight with the patient in supine and hip and knee flexed to 90º. This was followed by High TENS for 10 min, 3 times in a week. The nerve flossing technique was performed actively for 10 min with 30 sec hold in each position, 3 times in a week. The participant sitting on a chair, bent the knee backwards under the chair and lowered the head at the same time and held the position. Then the participant straightened out the leg on the side in which he experienced sciatic pain and at the same time extended the neck. The participant lifted the leg out and up in front until he began to experience pain and hold the position and did not push beyond that point. After this procedure all the patients were checked for range of motion of SLR at the hip with the help of standard goniometer and pain with the help of Visual Analogue Scale.

Data analysis

Descriptive statics was used to find out the frequency, percentage, mean and standard deviation for demographic data and variables studied. Data were tabulated using Microsoft office excel and analysed by Statistical Package for Social Sciences (SPSS) version t test was used. Probability values less than 0.05 were considered statistically significant and probability values less than 0.001 were considered highly significant.

Sample size estimation

Towards estimation of sample size for this cross-sectional study, the following guidelines were used: $n = Za^2pq/E^2$

n = Number of sample size,

Za = 1.96 at 95% confidence level

p = 70% Percentage of prevalence (80 % of power)

q = 30 % (100 – p)

E = 20 % error of p with 95% Confidence level & 80% power with reference to 70% sample size obtained was 30.

n = $(1.96)^2$ (70 % of 80 % of power) 30% [100 - (70 % of 80 % of power)] 20 % of (70 % of 80 % of power)

n = 3.84 x 70 x (100- 70) / 196 n = 3.84 x 70 x 30 / 196 n = 8067.36 / 196 n = 30

RESULTS

In **group A** the mean of VAS of pre-treatment was 7.73 and of post treatment was 7.26 with P value 0.04 which was considered as significant and the mean of ROM of pre-treatment was 49.6 and of post treatment was 50.8 with P value 0.0121 which was considered as significant. In **group B** the mean of VAS of pre-treatment was 7.6 and of post treatment was 2.26 with P value <0.0001 which was considered as extremely significant and the mean of ROM of pre-treatment was 48 and of post treatment was 61 with P value <0.0001 which was considered as extremely significant (Tables 1-3 and Figures 1-4). The mean of VAS of post treatment for group A was 7.26 and for group B was 2.26 with P value <0.0001 which was considered as extremely significant and the mean of ROM of post treatment for group A was 50.8 and for group B was 61 with P value <0.0195 which was considered as extremely significant.

Table 1: Comparison of pre and post-test VAS and ROM scores of Group A

Group	Pre-test score	Post-test score	P value	Level of Significance
Group A VAS	7.73	7.26	0.04	Significant
Group A ROM	49.6	50.8	0.01	Significant

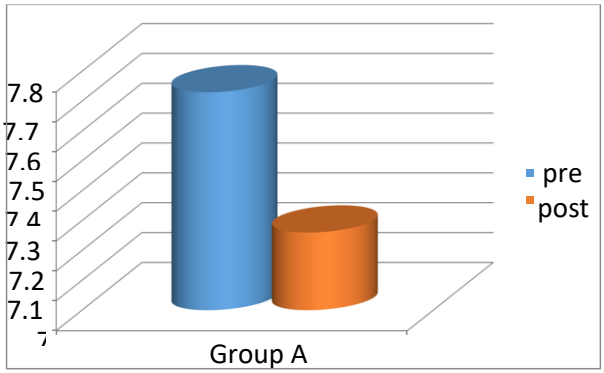

Figure 1: Comparison of pre and post-test VAS scores of Group A

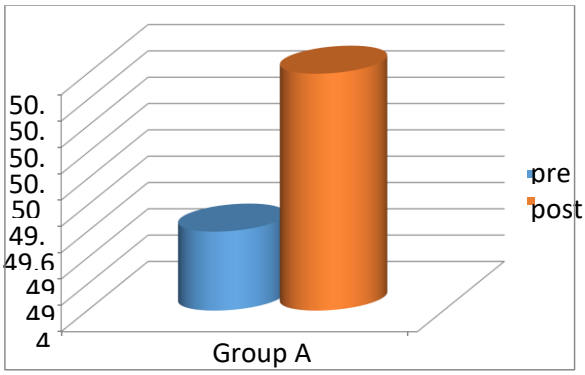

Figure 2: Comparison of pre and post range of Motion of group A

Table 2: Comparison of pre and post VAS and ROM scores of group B

Group	pre	post	P value	Level of significance
Group B VAS	7.6	2.26	<0.0001	Extremely significant

Group B ROM	48	61	<0.0001	Extremely significant

Table 3: Comparison between post VAS and ROM scores of Group A and Group B

Group	Group A	Group B	P value	Level of significance
Post score VAS	7.26	2.26	<0.0001	Extremely
Post score ROM	50.8	61	0.01	Significant

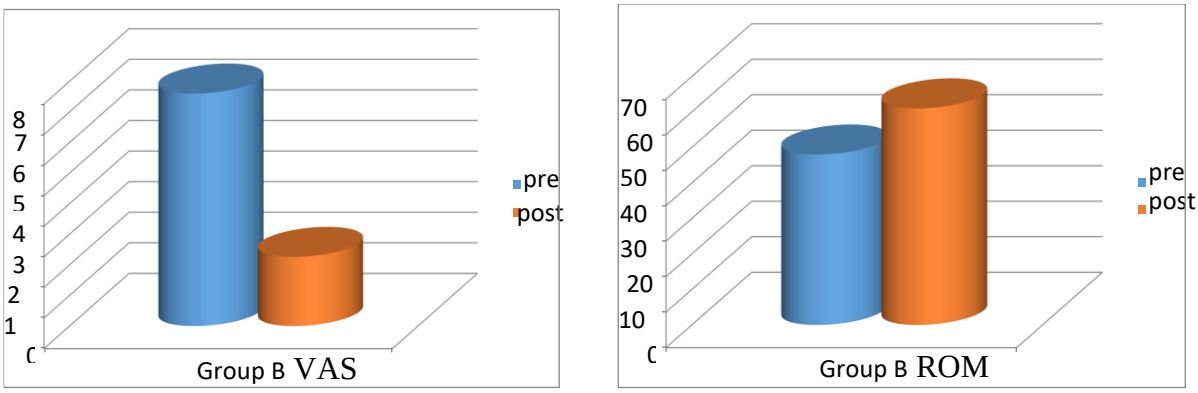

Figure 3: Comparison of pre and post VAS and ROM scores of group B

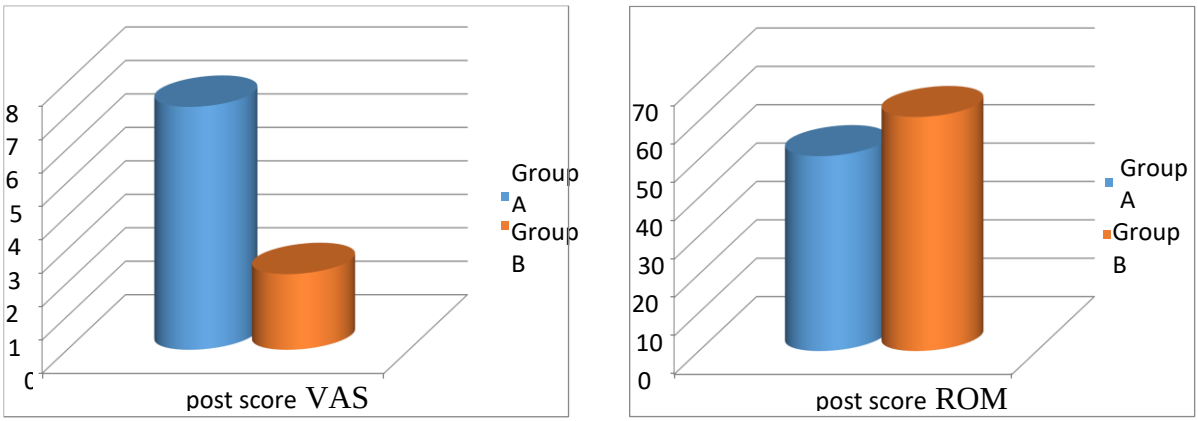

Figure 4: Comparison between post VAS and ROM scores of Group A and Group B

Table 3. Distribtion of *Acinetobacter* isolates among various wards (n=93)

WARD	Male	%	Female	%	Total	%
ICU	32	50	14	48	46	49
Surgery ward	12	19	6	21	18	19
Medicine ward	13	20	2	7	15	16
PICU	1	2	3	10	4	4
Orthopaedics ward	5	8	0	0	5	5
Paediatric ward	1	2	1	3	2	2
Gynaecology ward	0	0	3	10	3	3
Total	64	100	29	100	93	100

DISCUSSION

The result of this study shows that neural mobilization technique is effective in increasing range of motion at hip and decreasing pain thus reducing the symptoms of sciatica. The mean value of ROM of group B where neural mobilization was given shows more significant increase as compared to group A. The mean of VAS and ROM at hip joint of group B was extremely significant as compared to group A. Group B include nerve flossing, TENS, Traction showed better result as compared to group A that include Traction and TENS. Decrease in pain and increase in ROM at hip joint was due to neural "flossing" effect, because it restores normal mobility and length relationship, blood flow and axonal transport dynamics in compromised neural tissue. Neural mobilization is very effective in breaking up the adhesions and bringing about mobility. The conventional treatment effectively reduces pain and increases ROM at the joint but is unable to eliminate the root cause of the problem. Nerve flossing also causes proximal sliding of lumbar nerve roots with neck and knee flexion and causes distal sliding of lumbar nerve roots with neck and knee extension and also improve the actual excursion of the sciatic nerve, it also reduces Oedema also decrease adhesions and reducing symptoms. TENS was used to relief pain. In the gate control theory, stimulation of mechanoreceptors within the joint capsule and surrounding tissues causes an inhibition of pain at the spinal cord. It could also be directly associated with the immobilization reduction in the neurogenic inflammation. TENS produce analgesic effect by activation of cutaneous afferent fibers at the site of application. Traction is used as spinal decompression therapy. During spinal decompression therapy a negative pressure is created in disc because of this disc material that has been protruded or herniated can be assisted back within the normal confines of the disc and permit healing to occur. Pressure is released off of inflamed nerve root allowing the inflammation to subside.

CONCLUSION

Nerve flossing technique can be utilized with other modalities in the treatment of subacute sciatic patients due to low back ache for the relief of pain and sensory symptoms like tingling and numbness, restoration of spinal mobility and to minimize functional disability.

REFERENCES

1. Howard A, O'Donoghue M, Feeney A, Sleator RD. *Acinetobacter baumannii*: an emerging opportunistic pathogen. Virulence. 2012 May 1;3(3):243-50. doi: 10.4161/viru.19700.
2. Lahiri KK, Mani NS, Purai SS. *Acinetobacter* spp as Nosocomial Pathogen: Clinical Significance and Antimicrobial Sensitivity. Med J Armed Forces India. 2004 Jan;60(1):7-10. doi: 10.1016/S0377-1237(04)80148-5. Epub 2011 Jul 21. PMID: 27407568; PMCID: PMC4923491.
3. Sikora A, Zahra F. Nosocomial Infections. [Updated 2022 Sep 23]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK559312/.
4. Khanna A, Khanna M, Aggarwal A. Serratia marcescens- a rare opportunistic nosocomial pathogen and measures to limit its spread in hospitalized patients. J ClinDiagn Res. 2013 Feb;7(2):243-6. doi: 10.7860/JCDR/2013/5010.2737.
5. Manchanda V, Sanchaita S, Singh N. Multidrug resistant *Acinetobacter*. J Glob Infect Dis. 2010 Sep;2(3):291-304. doi: 10.4103/0974-777X.68538.
6. George M. Eliopoulos, Lisa L. Maragakis, Trish M. Perl, *Acinetobacter* baumannii: Epidemiology, Antimicrobial Resistance, and Treatment Options, Clinical Infectious Diseases, Volume 46, Issue 8, 15 April 2008, Pages 1254–1263, https://doi.org/10.1086/529198.
7. Young LS, Sabel AL, Price CS et al. Epidemiologic, clinical and economic evaluation of an outbreak of multidrug resistant *Acinetobacter baumannii* infections in a surgical intensive unit. Infect Control Hosp Epidemiol. 2007; 28(11): 1247-54.
8. VanLooveren M, Goossens H. ARPAC steering Group. Antimicrobial resistance of *Acinetobacter* species in Europe. Clin Microbial Infect. 2004; 10(8): 684-704.
9. Callie Camp, Owatha L. Tatum, A Review of *Acinetobacter* baumannii as a Highly Successful Pathogen in Times of War, Laboratory Medicine, Volume 41, Issue 11, November 2010, Pages 649–657, https://doi.org/10.1309/LM90IJNDDDWRI3RE.
10. Al Bshabshe, A., Joseph, M. R., Al Hussein, A., Haimour, W., & Hamid, M. E. (2016). Multidrug resistance *Acinetobacter* species at the intensive care unit, Aseer Central Hospital, Saudi Arabia: A one year analysis. Asian Pacific journal of tropical medicine, 9(9), 903-908.
11. Raina D, Sharma N, Mahawal B et al. Speciation and antibiotic resistance pattern of *Acinetobacter* spp in a tertiary care hospital in Uttarakhand. IntJourn of Med Research & Health Sci. 2016; 5(4): 89-96.
12. Malathy, K. (2015). Identification, Speciation, Antibiogram and Molecular Characterization of *Acinetobacter* Isolated from Various Clinical Samples received in Microbiology Laboratory, Thanjavur Medical College and Hospital (Doctoral dissertation, Thanjavur Medical College, Thanjavur).
13. Rajkumari, S., Pradhan, S., Sharma, D., & Jha, B. (2020). Prevalence and Antibiogram of *Acinetobacter* Species Isolated from Various Clinical Samples in a Tertiary Care Hospital. Journal of College of Medical Sciences-Nepal, 16(1), 26-32.

14. Clinical and Laboratory Standards Institute. Performance standards for antimicrobial susceptibility testing; 22nd informational supplement, CLSI document M100-S22. Wayne PA: Clinical and Laboratory Standards Institute; 2014.
15. Sarshar, M., Behzadi, P., Scribano, D., Palamara, A. T., &Ambrosi, C. (2021). *Acinetobacter* baumannii: an ancient commensal with weapons of a pathogen. Pathogens, 10(4), 387.
16. Rangel, K., Chagas, T. P. G., & De-Simone, S. G. (2021). *Acinetobacter* baumannii infections in times of COVID-19 pandemic. Pathogens, 10(8), 1006.
17. Alotaibi, T., Abuhaimed, A., Alshahrani, M., Albdelhady, A., Almubarak, Y., & Almasari, O. (2021). Prevalence of multidrug-resistant *Acinetobacter* baumannii in a critical care setting: A tertiary teaching hospital experience. SAGE Open Medicine, 9, 20503121211001144.
18. Ababneh, Q., Abulaila, S., &Jaradat, Z. (2022). Isolation of extensively drug resistant *Acinetobacter* baumannii from environmental surfaces inside intensive care units. American Journal of Infection Control, 50(2), 159-165.
19. Gupta, N., Gandham, N., Jadhav, S., & Mishra, R. N. (2015). Isolation and identification of Acinetobacter species with special reference to antibiotic resistance. Journal of natural science, biology, and medicine, 6(1), 159.
20. Mohammed, S. H., Ahmed, M. M., AbdAlameer Abd Alredaa, N., HaiderAbdAlabbas, H., Mohammad Ali, Z. D., Abed Al-Wahab, Z. Z., ... & Yahya Abid Zaid, N. (2022). Prevalence of *Acinetobacter* Species Isolated from Clinical Samples Referred to Al-Kafeel Hospital, Iraq and Their Antibiotic Susceptibility Patterns from 2017-2021. Iranian Journal of Medical Microbiology, 16(1), 76-82.
21. Albayrak, H., Bayraktar, M. T., &Zeyrek, F. Y. (2021). Antibiotic Resistance Profile of *Acinetobacter* Species Isolated from Blood Cultures of Inpatients in Harran University Hospital. Harran ÜniversitesiTıpFakültesiDergisi, 18(2), 165-169.
22. Dash, M., Padhi, S., Pattnaik, S., Mohanty, I., &Misra, P. (2013). Frequency, risk factors, and antibiogram of *Acinetobacter* species isolated from various clinical samples in a tertiary care hospital in Odisha, India. Avicenna Journal of Medicine, 3(04), 97-102.
23. https://1library.net/document/7q01nmvz-multivariate-analysis-acinetobacter-species-tertiary-care-hospital.html.
24. Gajdács, M., Burián, K., &Terhes, G. (2019). Resistance levels and epidemiology of non-fermenting gram-negative bacteria in urinary tract infections of inpatients and outpatients (RENFUTI): a 10-year epidemiological snapshot. Antibiotics, 8(3), 143.