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Research Article

Common Peroneal Neuropathy in the Patients of Hemiparaesis

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ABSTRACT

Background and Objectives: Stroke is one of the leading causes of mortality and disability. Functional impairment in stroke patients is primarily because of brain lesion that causes loss of foot dorsiflexion and eversion. The attained foot position leads to the continuous stretch, nerve traction or compression of common peroneal nerve resulting in neuropathy.

The main objective was to study whether the continuous everted position of the paralyzed foot in hemiplegic patients in the initial first week causes injury to common peroneal nerve at the fibular head.

Method: Case control study. The control of the study was the non-paralyzed limb of the same patient. All the stroke patients within 7 days of the onset underwent nerve conduction studies on affected and normal limb.

Results: The study showed the prolongation of distal motor latency in cases as compared to control and in those patients with power of grade 3 and lesser.

Conclusion: The electrophysiological changes in the common peroneal nerve starts in the initial 7 days of the stroke development especially causing prolongation of latency. Rehabilitation of stroke patients, physiotherapist should focus on taking care of foot drop which occurs commonly due to peroneal neuropathy by providing them orthotics.

Keywords: Common peroneal nerve; Electrophysiology; Hemiparesis; Neuropathy; Rehabilitation; Stroke

INTRODUCTION

The World Health Organization (WHO) defined Stroke as "A clinical syndrome consisting of rapidly developing clinical signs of focal (or global) disturbance of cerebral function, lasting more than 24 hours or leading to death, with no apparent cause other than a vascular origin "[1]. In 2013, American Stroke Association (ASA) / American Heart Association (AHA) gave modified definition of stroke and it included the presence of objective evidence of permanent brain, spinal cord or retinal cell death due to a vascular cause based upon pathological or imaging evidence with or without the presence of clinical symptoms [2].

Worldwide, stroke is the significant cause of mortality and morbidity. It is fifth in order among all the causes of deaths, behind the diseases of the heart, cancers, chronic respiratory diseases, and injuries/accidents [3]. In 2013, there were 6.5 million deaths due to stroke all over the world. Stroke is the most common cause of disability among young adults. It has been estimated that one third of all survived stroke patients are functionally dependent after one year of stroke with motor function deficits. After 3 months, approximately 20% of stroke survivors require institutional care whereas 15% to 30% are permanently disabled making it a significant cause of functional impairment [4].

In India, there is double burden of both communicable and non-communicable diseases. Stroke is one of the leading causes of mortality and disability in India. The estimated adjusted prevalence rate of stroke ranges between 84-262/100,000 population in rural and 334-424/100,000 population in urban areas [5]. In India incidence of stroke is high in younger people i.e. below 50 years of age and also in population with low socio-economic status. Many individuals survive with stroke, but they have significant disturbance in mobility after stroke.

One of the important causes of morbidity and disability after stroke is the physiological changes that occur after stroke. There is increase in the muscle tone i.e. spasticity which is present in

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approx 90% of cases. In lower limb, spasticity is observed in the pelvic retractors, hip adductors, internal rotators, knee and hip extensors, plantar flexors of ankle, investors and toe flexors. Functional impairment in stroke patients is primarily because of brain lesion that causes a loss of foot dorsiflexion and eversion [6]. But in addition to this, the attained foot position leads to the continuous stretch, nerve traction or compression of common peroneal nerve, resulting in peripheral neuropathy i.e. common peroneal neuropathy which can aggravate the dysfunction. Foot drop is commonly a lower motor neuron disease. Some of the common cause of compression of common peroneal nerve are wearing high heel boot /shoe, plaster casts, stocking or from sitting with the leg crossed for a prolonged period of time.

Hence while rehabilitating the patients of stroke, emphasis should not be given only to strengthening exercises and physiotherapy to affected muscles but should also focus on taking care of foot drop which occurs commonly due to peroneal neuropathy by providing them with Ankle Foot Orthotics and this will provide them a fast recovery and improvement in their gait

Earlier studies like [7] studied peroneal nerve involvement symptoms in 38 patients between 12-73 days after the first stroke. The development of foot drop on affected side was observed. Significant impairment in amplitudes and distal latencies of motor and sensory nerves in affected side compared to the healthy side was reported. Another study by [8] showed that 25% of the patients with stroke suffer from spasticity within first 6 weeks of the event and is often seen in hip adductors, internal rotators, hip and knee extensors, ankle plantar flexors, investors and toe flexors. Spasticity in ankle plantar flexors of the stroke patients was observed to be 66% and results in restriction of volitional movement. Thus the limb is positioned with equinovarus foot.

Both the studies were carried after 15 days of stroke in the affected limb and showed changes in the peroneal nerve based on the hypothesis that the continuous everted position of the paralyzed foot which is seen in the patients of hemiplegia/ hemiparesis after acute stroke in the initial neuronal shock stage and also the spasticity of the muscles, results in the compression and continuous stretching of common peroneal nerve at the fibular head at knee and causes peroneal neuropathy resulting in foot drop, which can aggravate the dysfunction.

Our extensive search of the literature did not reveal any such research which has studied the peroneal nerve in the initial 7 days of the stroke. Therefore this study was planned with the aim

- To study whether the continuous everted position of the paralyzed foot in hemiplegic patients in the initial first week after acute stroke causes injury to common peroneal nerve at the fibular head.
- To study the motor conduction properties – distal motor latency (DML), compound muscle action potential (CMAP) and nerve conduction velocity (NCV) in common peroneal nerve (CPN) in the patients after first time stroke

MATERIALS AND METHODS

Study design: Case control study. The control of the study was the non-paralyzed limb of the same patient.

Study setting: After getting the approval of the institutional ethical committee, the study was carried out among the stroke patients admitted in the department of medicine in a rural tertiary care hospital at Mahatma Gandhi Institute of Medical sciences, Sewagram situated in central India.

Study period: The study was carried out for one and half year from Jan 2016 to June 2017.

Study population: All the stroke patients (haemorhagic or ischaemic) within 7 days of the onset of stroke, was shifted to Electrophysiology study room for performing nerve conduction studies. The calculated sample size was 100 patients. We included 108 patients in the study.Nerve conduction study was performed on affected limb and also on normal limb which was the control of the study.

Inclusion criteria: All new stroke patients (haemorhagic or ischaemic) who presented within seven days of the onset and who were willing to give consent (or consent obtained from the relatives) were included in the study.

Exclusion criteria

- Who has recurrent or repeat stroke.
- History of having diabetes mellitus or taking oral hypoglycaemic drugs or on insulin or detected to be diabetic on admission.
- Alcoholism (Alcohol abuse and/ or dependence)
- Having Megaloblastic anaemia (Mean Corpuscular Volume >90) or vitamin B12 deficiency.
- Patients on drugs such as anticonvulsants or antitubercular.
- Patients having chronic diseases such as chronic kidney disease, malignancy, cirrhosis of liver.
- Patients having thyroid disorders.
- Patients who are critically ill and cannot be shifted to electrophysiology laboratory for the Nerve Conduction Studies.
- Patient who are not willing to give consent to participate in the study.

METHODOLOGY

After taking the consent from the patient or their relatives and collecting the baseline data like the age, body mass index, hemogram, blood pressure, blood sugar levels and lipid profile, nerve conduction study was performed on common peroneal nerve in both legs of stroke patients. The test was performed with the patients in supine position on RMS-EMGII machine at the room temperature of 27 degree celsius.

Surface recordings were obtained from extensor digitorum brevis and stimulation was given at ankle, 2 cm distal to the fibular neck, at the neck of fibula and 5-8 cm above the fibular neck for peroneal nerve conduction study as shown in Figure 1 and 2, motor latency and compound muscle action potential was measured and nerve conduction velocity was calculated.

Results of the paralysed leg were compared to the normal leg which was the control of the study.

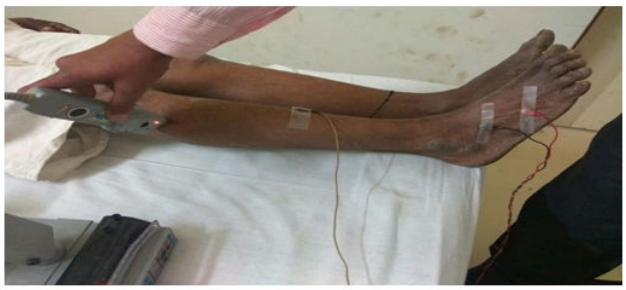


Figure 1: Surface recording of common peroneal nerve stimulating at the ankle.

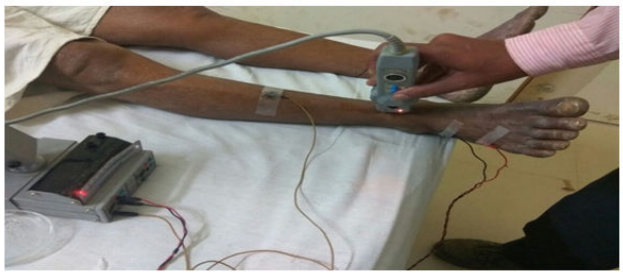
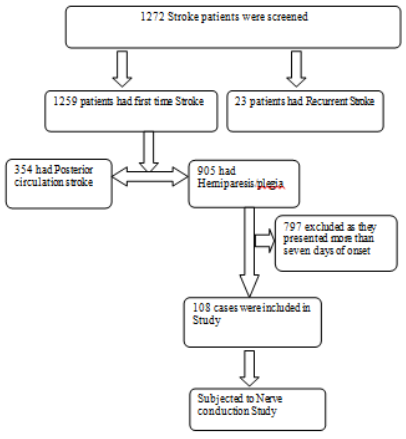


Figure 2: Surface recording of common peroneal nerve stimulating at a point 2-3 cm distal to fibular neck.

The distal motor latency is the time in milliseconds from the stimulus to the first negative deflection of CMAP as shown in graph 1. The distal motor latency is the measure of conduction in the fastest conducting motor fibers. It also includes neuromuscular transmission time and the propagation time along the muscle membrane which constitute the residual latency.



Graph 1: Electrophysiological graph of common peroneal nerve.

Distal motor latency–L=onset to start of amplitude showing distal motor latency.

Compund muscle action potential–b=Peak to peak amplitude.

Motor nerve conduction velocity is calculated by measuring the distance in millimeter between two points of stimulation, which is then divided by the latency difference in millisecond.

The amplitude of compound muscle action potential is measured from base line to the negative peak (base to peak) or between negative and positive peaks (peak to peak). The amplitude of CMAP in our case was measured from negative to positive a peak that is peak to peak as shown in graph 1. The amplitude correlates with the number of nerve fibers. The unit of measurement of CMAP is microvolt.

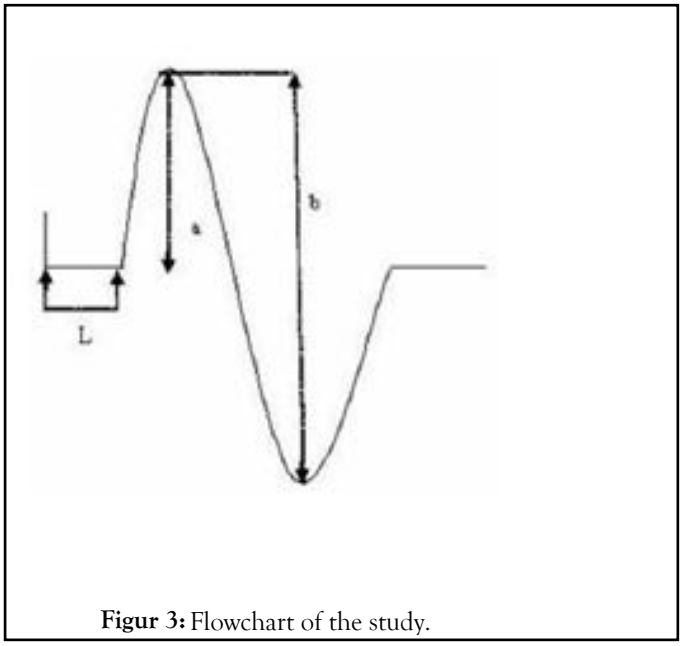
Motor nerve conduction velocity is calculated by measuring the distance in millimeter between two points of stimulation, which is then divided by the latency difference in millisecond.

Statistical analysis

Data entry was done in the Microsoft excel sheet.Statistical analysis was done by using descriptive and inferential statistics using chi square test and z-test for difference between two means and software used in the analysis were SPSS 24.0 version and Graph Pad Prism 6.0 version and $p<0.05$ is considered as level of significance.

RESULTS

The flowchart of the study is shown in the figure 3 depicting that we screened 1272 stroke patients admitted in the rural tertiary care hospital. Out of those 1259 were having first time stroke and 23 patients were having recurrent stroke. Out of 1259, 354 had posterior circulation stroke and 905 patients had hemiparesis or hemiplegia. 797 stroke patients were excluded from the study as they presented for more than 7 days of duration and 108 were included in study. These 108 stroke patients were subjected to nerve conduction study.



Figur 3: Flowchart of the study.

The baseline characteristics are same for both the cases and cont rol as the hemiplegic/hemiparetic limb was taken as the cases and the sound limb of the same patient was taken as control as shown in Table 1.

Table 1: Baseline characteristics of the study population.

Serial no	Characters	Mean	Standard deviation (SD)
1	Age (years)	61.32	13.23
2	Systolic blood pressure (mm of hg)	156.48	23.73
3	Diastolic blood pressure (mm of hg)	92.68	10.73
4	Random blood sugar (mg/dl)	118.86	19.43
5	Total cholesterol (mg/dl)	165.88	36.50
6	High density lipoprotein (mg/dl)	51.09	10.78
7	Triglycerides (mg/dl)	110.60	46.57
8	Very low density lipoproteins(mg /dl)	21.73	9.30

Mean age of study patients was 61.32 years with SD ± 13.23, 77 were males and 31 were females, mean systolic blood pressure was 156.48 mm Hg with SD ± 23.73 and mean diastolic blood pressure was 92.68 mm Hg with SD ± 10.73. Mean blood sugar of patients were 118 .86 mg/dl with SD ± 19.43. Mean triglyceride level was 165.88 mg/dl with SD ± 26.5, mean high density lipoprotein level was 51.09 mg/dl with SD ± 10.78, and mean low density lipoprotein level was 110.60 mg/dl with SD ± 46.57 and mean very low densitylipoprotein level was 21.73 mg/dl with SD ± 9.3.

The mean DML in test group was 4.39 mm with SD ± 1.27 mm and in control group was 3.98 mm with SD±0.97 mm. This shows the prolongation of distal motor latency in test group as compared to control group and the prolongation was statistically significant ($p<0.05$) as shown in Table 2.

The mean CMAP in test group was 4.65 mv with SD ± 3.11 mv and in control group was 4.80 mv with SD ± 2.93 mv. This shows no difference in CMAP between the two groups ($p>0.05$) as shown in Table 2.

The mean NCV in test group was 46.16 m/sec with SD ± 8.29 m/sec and in control group was 47.19 m/sec with SD ± s9.43. This shows no difference in conduction velocity between the two groups ($p>0.05$) as shown in Table 2.

Table 2: Comparison of DML, CMAPs and NCV in the cases and controls.

Distal motor latency in cases and controls

Group	N	Mean	Standard deviation	z-value	p-value
Test Group	108	4.39	1.27	2.67	0.008
Control Group	108	3.98	0.97		

Compound Muscle action potential in cases and controls

Group	N	Mean	Standard deviation	z-value	p-value
Test Group	108	4.65	3.11	0.35	0.72
Control Group	108	4.80	2.93		

Nerve conduction velocity in cases and controls

Group	N	Mean	Standard deviation	z-value	p-value
Test Group	108	46.16	8.29	0.85	0.38
Control Group	108	47.19	9.43		

DML-distal motor latency

CMAPs-compound muscle action potentials

NCV-Nerve conduction velocity

DISCUSSION

The purpose of our study was to investigate whether the continuous everted position of foot and spasticity of ankle flexors and /or weakness of ankle dorsiflexors cause electrophysiological changes in the nerve conduction study of common peroneal nerve. The study showed that there is prolongation in distal motor latency on the affected side of hemiparesis or hemiplegia, whereas compound muscle action potential and nerve conduction velocity was approximately same as that of normal limb.

Though there is no gross difference in motor latency of affected and non-affected limb but there is statistically significant difference.The prolongation in motor latency on affected side was observed in the affected limb when power grade is less than 3. It was approximately normal in limbs having power greater than grade 3. Also prolongation in latency was found when the nerve conduction study was performed after 3 days of the onset of stroke.This prolongation in motor latency of common peroneal nerve can be due to nerve traction and compression of common peroneal nerve at the neck of fibula causing demyelination or even axonopathy because of the position of the lower limb attained by the stroke patient. The other factors could be the change in skin temperature as affected limb of

stroke patient has always lower temperature as compared to non-affected limb and this change in skin temperature can alter the electrophysiological parameters.

Showed significant slowing in peroneal nerve conduction velocities in affected extremities of 27 hemiplegic patients and at the same time there is a significant difference in skin temperature between the extremities of the two sides. They supposed that a decreased diameter of nerve fibres as a result or cause of muscle atrophy could lead to decreased nerve conduction velocity [9].

Chokroverty and Medina measured motor nerve conduction velocity of common peroneal nerve in 44 hemiplegic patients and found statistically significant difference between two limbs. Also in 63 percent of patients, skin temperature was reduced [10].

A study by [8] showed that 25 percent patients with stroke suffer from spasticity within first 6 weeks of event and is often seen in hip adductors, internal rotators, hip and knee extensors, ankle plantar flexors, investors and toe flexors. Spasticity in ankle plantar flexors of stroke patients was observed to be in 66 percent and results in restriction of volitional movement, thus the limb are positioned with equinovarus foot [7].

Conducted study on 76 legs of 38 stroke patients, 12-73 days after stroke and showed statistically significant difference in motor latency and muscle action potential between paralysed and sound leg. The study concluded that permanent equinovarus position of paralysed foot might affect common peroneal nerve conduction properties at the level of fibular neck by demyelination, axonopathy or both [8].

All these studies showed changes in all the three parameters of nerve conduction study i.e prolongation of motor latency, decrease in action potential and decrease in conduction velocity on the affected side of hemiparesis. However all these studies were conducted at least 12 days or more of the onset of stroke?

In our study we conducted nerve conduction study within 7 days of the onset of stroke. Hence we found only motor latency to get prolonged whereas other two parameters compound muscle action potential and nerve velocity were approximately equal to that of normal leg. But if we would have conducted neurophysiological studies beyond 7 days, we would have found difference in compound muscle action potential and conduction velocity as showed in previous studies.

Strength

Sample size was larger in our study as compared to studies done earlier.

Limitations

The study was carried only on the subjects within 7 days of onset of the stroke. We did not include the others of more than 7 days.

REFERENCES

1. Aho K, Harmsen P, Hatano S, Marquardsen J, Smirnov VE, Strasser T. Cerebrovascular disease in the community:results of a WHO collaborative study. Bull World Health Organ. 1980;58:113-130.
2. Sacco RL, Kasner SE, Broderick JP, Caplan LR, Culebras A, Elkind MS, et al. An updated definition of stroke for the 21st century. Stroke. 2013;44(7):2064-89.
3. Heart Disease and Stroke Statistics. Update: A Report from the American Heart Association. Circulation. 2017;136(10):196.
4. Steinwachs DM, Collins-Nakai RL, Cohn LH, Garson A Jr, Wolk MJ.J The future of cardiology: utilization and costs of care. Am CollCardiol. 2000;35(5):91-98.
5. Pandian JD, Sudhan P. Stroke Epidemiology and Stroke Care Services in India. J Stroke. 2013;15(3):128-134.
6. Westhout F D, Pare L S, Linskey M E. Central Causes of Foot Drop: Rare and Underappreciated Differential Diagnoses. J Spinal Cord Med. 2007;30:62–66.
7. Tsur A. Common peroneal neuropathy in patients after first-time stroke. Isr Med Assoc J. 2007;9:866-869.
8. Aurore Thibaut, Camille Chatelle, Erik Ziegler, Marie-Aurélie Bruno, Steven Laureys, Olivia Gosseries. Spasticity after stroke: physiology, assessment and treatment.Brain Inj. 2013;27(10):1093–1105.
9. Takebe K, Narayan M G, Kukulka C, Basmajian J V. Slowing of nerve conduction velocity in hemiplegia: possible factors. Arch Phys Med Rehabil. 1975;56(7):285-289.
10. Chokroverty S, Medina J. Electrophysiological study of hemiplegia. Motor nerve conduction velocity, brachial plexus latency and electromyography. Arch Neurol. 1978;35(6):360-363.