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International Journal of Pharmaceutical and Biological Science Archive

Volume 4 Issue 1; January-February 2016; Page No.06-12

Index Copernicus Value 2016: 65.90

Neurophysiologic Evaluation of Masticatory function in stroke patients with Needle Electromyography of Masseter muscle

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ARTICLE INFO	ABSTRACT									
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Received 01 Jan.2016 Accepted 20 Feb.2016

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Assistant Professor, Department of Neurology, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences, Sawangi (Meghe), Wardha **Background:** Facial paresis in hemiparetic stroke leads to a major impairment in masticatory function in these patients. Along with paresis of facial musculature, there is impairment in power of muscles of mastication supplied by trigeminal nerve

Aims and Objectives: to evaluate masticatory function in patients following a stroke with hemiparesis; and to compare masticatory function in patients following an ischemic stroke with those following a hemorrhagic stroke.

Material and Methods: This prospective study was conducted over a period of one year from August 2014 to July 2015. Cases of acute stroke, with hemiparesis and upper motor were included. Patients whose CT Brain shows an infarct were included in group A. Patients whose CT Brain suggestive of a hemorrhage were included in group B. Age and sex matched controls were included in Group C. The assessment of masticatory function was done by needle electromyography of Masseter muscle on the symptomatic side. Follow up of all the patients was done at the end of three months.

Results: During the study period, 200 patients with acute stroke were screened. After applying the predefined inclusion and exclusion criteria, total of 60 patients were selected as cases. Out of these, 30 cases had acute ischemic stroke. Remaining 30 cases had acute hemorrhagic stroke. Age and sex matched 30 controls were included. Among the patients with Ischemic stroke the patient age was [Mean (SD)] 48.33 (17.3) years. Out of 30 ischemic stroke patients, 21 were male and 9 were female. Hypertension was present in 8 patients, Diabetes was present in 8 patients, Metabolic Syndrome was seen in 7 patients, Smoking was present in 7 patients and 7 patients were obese. Both ischemic and hemorrhagic stroke patients had significantly lower amplitudes and higher area of motor unit action potential (MUAP) of masseter muscle compared to controls. They did not differ significantly between groups A and B. there was significant improvement in these parameters at 3 months follow up.

Conclusions: Masseter muscle MUAP analysis is an important parameter for assessment of masticatory function in stroke patients. Furthermore, there is no significant difference in the masticatory dysfunction between patients with ischemic and hemorrhagic strokes.

Keywords: MUAP

INTRODUCTION

Hemiparesis is the most frequent neurological deficit after stroke [1]. Stroke-related physical disability can diminish quality of daily living, place care burden on families. and increase need for long-term institutionalization.[2] Stroke is also a leading cause of functional impairments, with 20% of survivors requiring institutional care after 3 months and 15% to 30% being permanently disabled. Stroke is a life-changing event that affects not only stroke patients themselves but their family members and caregivers as well. Utility analyses show that a major stroke is viewed by more than half of those at risk as being worse than death.[3] impairments, including hemiparesis, Motor incoordination, and spasticity, are the most common deficits after stroke. Most patients recover at least some of their lost motor function over time, though the degree of this recovery is variable.[4] Upper motor neuron facial paresis in hemiparetic stroke leads to a major impairment in masticatory function in these patients. Along with paresis of facial musculature, there is impairment in power of muscles of mastication supplied by trigeminal nerve. Also, stroke leads to increase in muscle tone and impairment in coordination in muscles of mastication, facial muscles and tongue. This contributes to not only impairment in oral health related quality of life but also to malnutrition. The social and psychological effects of eating and drinking difficulties are often overlooked. The collection of food around the lips during meals is an example of a little discussed but embarrassing and unsightly problem commonly experienced after a stroke. In acute stroke patients, malnutrition has a reported prevalence of 20% and is associated with dysphagia and chewing difficulty. [5] Recent functional animal studies have reported that the motor control of masticatory muscle function is bilaterally guided by both hemispheres, which may fundamentally differ from the cortical control of limb muscle function. [6] This observation can have important implication s in post-stroke rehabilitation. Only few studies have previously addressed the issue of masticatory function in stroke patients. Another important fact is that the prognosis and recovery are different in ischemic and hemorrhagic stroke. So it is worthwhile to compare masticatory function and bite force in these two group of patients, which may guide us in post stroke rehabilitation.

Considering these facts, we intend to study masticatory function following stroke with hemiparesis and compare these parameters in patients with an ischemic and hemorrhagic stroke. The aims and objectives of this study were to evaluate masticatory function in patients following a stroke with hemiparesis; and to compare masticatory function in patients following an ischemic stroke with those following a hemorrhagic stroke.

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Materials and Methods:

This study was conducted at Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences, Sawangi (Meghe), Wardha, Maharashtra, India. It is a tertiary care Hospital which treats patients from Central India. This study was conducted over a period of one year from August 2014 to July 2015. Data collection was done in first 9 months and analysis was performed over next 3 months. The design of this research was prospective observational study. Cases of acute stroke. both ischemic and hemorrhagic with hemiparesis and upper motor neuron facial paresis were included in the study. Stroke was defined as per the WHO (World Health Organization) definition, 'a syndrome of rapidly developing clinical signs of focal or global neurological disturbance lasting for more than 24 hours.' Only CT brain proved cases of acute stroke (ischemic and hemorrhagic) were included in the study. Age, sex and dental status matched healthy controls, were enrolled after explaining all the risks and complications of procedure and obtaining an informed consent. As per the sample size calculations, we included 30 patients with an ischemic stroke (Group A), 30 patients with hemorrhagic stroke (Group B) and 30 age, sex and dental status matched controls (Group C).

Inclusion Criteria:

• Patients with an acute onset hemiparesis with upper motor neuron facial paresis lasting for more than 24 hours

• Patients whose CT Brain shows an infarct were included in group A.

• Patients whose CT Brain suggestive of a hemorrhage were included in group B.

• Only class 1 dentulous patients were included.

Exclusion criteria:

• Patients with impaired comprehension due to higher mental dysfunction or aphasia.

- Edentulous patients.
- Posterior circulation stroke.
- Difficult follow up or consent not available.

Patients fulfilling inclusion criteria attending the Neurology OPD and the indoor patients of Department of Neurology of Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences, Sawangi (Meghe), Wardha, Maharashtra, India were included in the study. Written informed consent to participate in study was from the the patient or the guardian/relatives, prior to enrollment. All patients underwent detailed history taking, clinical evaluation. Patient's disability status was assessed by National

Institute of Health Stroke Scale (NIHSS). The investigations of the patient population included Routine biochemical studies. [Hemoglobin, complete blood count, kidney function test, liver function test]. Computed Tomography (CT) of Brain, following which Patients were divided according to type of stroke, i.e., ischemic stroke (Group A) and hemorrhagic stroke (Group B). Patients in all groups underwent assessment masticatory functions. of The assessment of masticatory function was done by needle electromyography of Masseter muscle the on symptomatic side.

Follow up of all the patients was done at the end of three months. A complete clinical evaluation was done

at the end of the three months. Needle electromyography of the symptomatic side Masseter muscle was done at the time of clinical follow up.

Statistical Analysis: A t-test for continuous normally distributed variables was used. For categorical data chi-square statistics was used and Fischer-exact test was used for small numbers. For variables which were not normally distributed Wilcoxon's Mann-Whitney U-test was used. P value of 0.05 or less was considered statistically significant. Cox proportional hazard regression model for analysis of multiple predictor variability was used. The study protocol is shown in Image 1

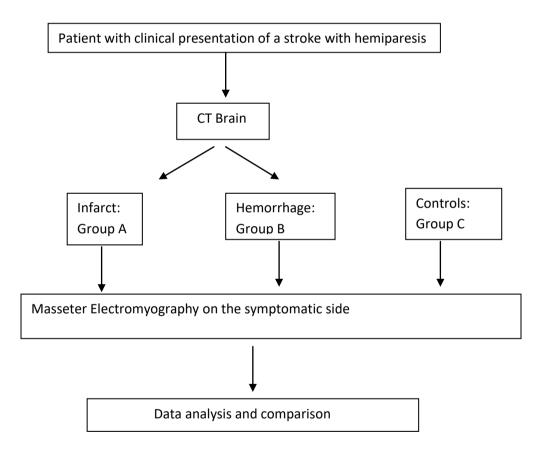


Image 1: Study Protocol:

Results

This study was conducted at department of Neurology, Jawaharlal Nehru Medical College, Datta Meghe Institute of medical Sciences, Sawangi (Meghe), Wardha, Maharashtra, India. During the study period, 200 patients with acute stroke were screened. After applying the predefined inclusion and exclusion criteria, total of 60 patients were selected as cases. Out of these, 30 cases had acute ischemic stroke. They were included in Group A. The remaining 30 cases had acute hemorrhagic stroke. These patients were included in Group B. In addition, age and sex matched 30 controls were included. These patients were included in Group C.

The baseline characteristics comparison between ischemic stroke patients and controls is shown in table 1.

Table 1: Comparison of Baseline Characteristics between Ischemic stroke patients and controls

Character	Group A (Ischemic Stroke) [n=30]	Group C (Control) [n=30]	P value				
Age [Mean (SD)]	48.33 (17.3)	46.23 (15.32)	0.13				
Male Sex	21	22	0.774				
Female Sex	9	8	0.774				
Hypertension	8	4	0.197				
Diabetes	8	4	0.197				
Metabolic Syndrome	7	3	0.166				
Smoking	7	2	0.071				
Obesity	7	2	0.071				
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The baseline characteristics comparison between hemorrhagic stroke patients and controls is shown in table 2.

Table 2: Comparison of Baseline Characteristics between hemorrhagic stroke patients and controls

Character	Group B (Hemorrhagic Stroke) [n=30]	Group C (Control) [n=30]	P value
Age [Mean (SD)]	45.92 (16.24)	46.23 (15.32)	0.723
Male Sex	20	22	0.573
Female Sex	10	8	0.573
Hypertension	9	4	0.231
Diabetes	6	4	0.488
Metabolic Syndrome	2	3	0.640
Smoking	2	2	1.000
Obesity	4	2	0.389

Comparison of Masseter MUAP amplitude and area between patients with ischemic stroke and controls at presentation is shown in table 3.

Table 3: Comparison of Masseter MUAP variables between ischemic stroke patients and controls at presentation

Character	Group A (Ischemic Stroke)	Group C (Control) [n=30]	P value (t
	[n=30]		value)
Masseter MUAP amplitude at	572.233 (228.055)	1246.133(157.266)	p <0.001
presentation [Mean (SD)	microVolts	microVolts	(t=-13.324)
Masseter MUAP area at presentation	35.6 (2.554)	28.767 (1.654)	P<0.001
[Mean (SD)]			(t= 12.299)

Comparison of Masseter MUAP amplitude and area between patients with ischemic stroke and controls at follow up is shown in table 4.

Table 4: Comparison of Masseter MUAP variables between ischemic stroke patients and controls at follow up

Character	Group A (Ischemic Stroke)	Group C (Control)	P value (t
	[n=30]	[n=30]	value)
Masseter MUAP amplitude at follow up	982.000 (150.959)	1295.500 (152.046)	P<0.001
[Mean (SD)			(t=-8.014)
Masseter MUAP area at follow up [Mean	30.867 (2.897)	28.767 (1.654)	P=0.001
(SD)]			(t=3.447)

Comparison of Masseter MUAP amplitude and area between patients with hemorrhagic stroke and controls at presentation is shown in table 5.

Table 5: Comparison of Masseter MUAP variables between hemorrhagic stroke patients and controls at presentation

Character	Group B (Hemorrhagic	Group C (Control) [n=30]	P value (t			
	Stroke) [n=30]		value)			
Masseter MUAP amplitude at	590.467 (305.122)	1246.133(157.266)	p <0.001			
presentation [Mean (SD)		microVolts	(t=-10.462)			
Masseter MUAP area at presentation	35.800 (3.872)	28.767 (1.654)	P<0.001			
[Mean (SD)]			(t= 9.149))			

Comparison of Masseter MUAP amplitude and area between patients with hemorrhagic stroke and controls at follow up is shown in table 6.

Table 0. Comparison of Masseler MOAF variables between nemormagic scroke patients and controls at follow up					
Character	Group B (Hemorrhagic Stroke)	Group C (Control)	P value (t		
	[n=30]	[n=30]	value)		
Masseter MUAP amplitude at follow up	1032.667 (204.724)	1295.500 (152.046)	P<0.001		
[Mean (SD)			(t=-5.645)		
Masseter MUAP area at follow up [Mean	30.867 (2.080)	28.767 (1.654)	P<.001		
(SD)]			(t=4.328)		

Table 6: Comparison of Masseter MUAP variables between hemorrhagic stroke patients and controls at follow up

Comparison of Masseter MUAP amplitude and area between patients with ischemic stroke and hemorrhagic stroke at presentation is shown in table 7.

Table 7: Comparison of Masseter MUAP variables between ischemic stroke patients and hemorrhagic stroke patients at presentation

Character	Group A (Is	nemic Group B (Hemorrhagic	P value (t
	Stroke) [n=30]	Stroke) [n=30]	value)
Masseter MUAP amplitude at	572.233 (22	3.055) 590.467 (305.122)	P=0.794
presentation [Mean (SD)	microVolts		(t=-0.262)
Masseter MUAP area at presentation	35.6 (2.554)	35.800 (3.872)	P=0.814
[Mean (SD)]			(t=-0.236)

Comparison of Masseter MUAP amplitude and area between patients with ischemic stroke and hemorrhagic stroke at follow up is shown in table 8.

Table 8: Comparison of Masseter MUAP variables between ischemic stroke patients and hemorrhagic stroke
patients at follow up

Character	Group A (Ischemic Stroke)	Group B (Hemorrhagic Stroke)	P value (t
	[n=30]	[n=30]	value)
Masseter MUAP amplitude at follow	982.000 (150.959)	1032.667 (204.724)	P=0.280
up [Mean (SD)			(t=-1.091)
Masseter MUAP area at follow up	30.867 (2.897)	30.867 (2.080)	P=1.000
[Mean (SD)]			(t=0.000)

Discussion

Mastication is an important activity of daily living. It is important not only from the point of view of nutrition but also from the point of view of wellbeing. Patients develop weakness of mastication due to motor dysfunction following stroke. This results in a poor quality of life following stroke. This was a unique study in which we assessed the masticatory function in patients with ischemic stroke and hemorrhagic stroke at presentation and at three months follow up. Various muscles are involved in the motor function of mastication. But the most important muscle of mastication is Masseter. The masticatory function has a strong correlation with the strength of masseter muscle. The best parameters to assess function of masseter muscle are the Motor Unit Action Potential (MUAP) amplitude and area as assessed by needle electromyography (EMG). Needle EMG represents the activation of the muscle after motor stimulation and hence correlates with the muscle strength [7].

Among the patients with Ischemic stroke the patient age was [Mean (SD)] 48.33 (17.3) years. Out of 30 ischemic stroke patients, 21 were male and 9 were female. Hypertension was present in 8 patients, Diabetes was present in 8 patients, Metabolic Syndrome was seen in 7 patients, Smoking was present in 7 patients and 7 patients were obese. Among the patients with hemorrhagic stroke, the patient age was 45.92 (16.24) years. Out of 30 [Mean (SD)] hemorrhagic stroke patients, 20 were male and 10 were female. Hypertension was present in 9 patients, Diabetes was present in 6 patients, Metabolic Syndrome was seen in 2 patients, Smoking was present in 2 patients and 4 patients were obese. Among the controls, the age was [Mean (SD)] 46.23(15.32) years. Out of 30 controls, 22 were male and 8 were female. Hypertension was present in 4 controls, Diabetes was present in 4 controls, Metabolic Syndrome was seen in 3 controls, Smoking was present in 2 controls and 2 controls were obese. Thus, it was noted that the controls were well matched with patients with Ischemic stroke as well as hemorrhagic stroke patients.

Advancing age, diabetes, hypertension, metabolic syndrome, smoking are all risk factors for both ischemic and hemorrhagic stroke. However, hypertension is a significant risk factor for hemorrhagic stroke. Hypertension and diabetes are important risk factors for lacunar strokes and strokes due to intracranial atherosclerotic disease (ICAD) [8]. Smoking, metabolic syndrome are risk factors for ischemic stroke due to carotid artery atherosclerotic, resulting in artery-toartery embolic strokes.

In both the ischemic stroke and hemorrhagic strokes, there was significantly low MUAP amplitude and increased area of the MUAP in masseter muscle, compared to the controls. In addition, it was also observed that there was no difference in the MUAP amplitude and area in the masseter muscle, when a direct comparison was made between the patients with ischemic stroke and hemorrhagic stroke. The MUAP amplitude denotes the summation of the maximum action potentials achieved at the motor end plate when a stimulus reaches the myoneural junction [9,10]. Electromyography can pick up the impaired activity at the motor end plate [11,12,13,14,15]. It represents the depolarization with the entry of sodium from extracellular to intracellular compartment. The lower values in our patients indicate that this process is impaired in both ischemic as well as hemorrhagic strokes. On the other hand, the area of the motor unit action potential denotes the temporal dispersion of the action potential wave at the motor end plate. Thus, the significantly wider area in patients with ischemic as well as hemorrhagic stroke patients denotes the weakening of the action potential waveform at the motor end plate during acute stroke. We also demonstrated that there was significant improvement in both ischemic as well as hemorrhagic stroke patient population in the masseter MUAP parameters at the three month follow up.

Thus, our study concludes that Masseter muscle MUAP analysis is an important parameter for assessment of masticatory function in stroke patients. Furthermore, there is no significant difference in the masticatory dysfunction between patients with ischemic and hemorrhagic strokes. The limitations of the study were smaller sample size and single center study. We suggest that these observations should be retested in a larger population with a multicenter study.

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