A Study on Various Sites of Supranuclear Facial Nerve Lesion

Abstract

Facial nerve paralysis is a common clinical problem that involves the paralysis of any structures innervated by the facial nerve. It is mainly motor, controls the muscles of facial expression having some sensory fibres controlling salivation and function in conveyance of taste sensation from anterior 2/3rd of the tongue. We can divide facial nerve lesion into three categories supranuclear, nuclear and infranuclear. The supranuclear facial nerve lesion can be due to any cause like haemorrhage and infarct. Key words-facial, nerve, infarct, motor, taste.

Introduction

The facial nerve is the seventh cranial nerve and, most commonly undergoing paresis. Supranuclear facial nerve paralysis occurs due to lesion of cerebral motor cortex, basal ganglia, internal capsule, thalamus, corona radiata, cerebellum, pons. The lesion can be of various types like haemorrhage, infarction, tumour, etc. Among these basal ganglia involvement is 20-25% in all cerebrovascular accidents. Facial nerve paralysis causes noticeable disfiguration and emotional distress (Figure 1).

Riview of literatures

Hines et al. [1] found that supranuclear facial nerve lesion occurs due to damage of cell bodies of cerebral motor cortex or their axons that project through the internal capsule to motor nuclei of facial nerve. Voluntary control of lower facial muscles are lost, but that of upper forehead muscles are spared.

Motor cortex: Ugur et al. [2] in his study found that the anterior cerebral artery supplies medial 1/3rd and middle cerebral artery supply lateral 2/3rd of motor cortex i.e. precentral gyrus.

Friebberg [3] demonstrated motor homunculus in precentral gyrus where neurons controlling head is in lateral 2/3rd of the precentral gyrus.

Internal capsule: Luis & Julien [4] found that the motor homunculus in internal capsule is arranged as head and eye in anterior limb.

Hashmi [5] found in his study that the superior surface of internal capsule is supplied by middle cerebral artery and inferior surface by the anterior cerebral artery.

Thalamus: Marrison [6] found that ventrolateral and ventral anterior nucleus of the thalamus are the relay station of the fibres coming from Globus and cerebellum to motor cortex that controls the motor movements.

Basal ganglia: In his book Vishram Singh (2010) mentioned that Fibres from Globus pallidus relay in the thalamus through the internal capsule in its anterior limb, Globus pallidus in turn receives fibres from putamen.

Cerebellum: Standring [7] in his book mentioned that motor homunculus in the cerebellum is situated on superior surface of anterior lobe and corresponding vermis.

Aims and objectives

a. To determine the anatomical sites.
b. To evaluate remedial measures.

Materials and Methods

This study on supranuclear facial nerve palsy was conducted in patients from the department of medicine from 1st July 2013 to 1st July 2014 in Silchar medical college and hospital, Silchar, Assam. A total 49 cases were taken irrespective of age and sex.
having facial nerve palsy in the department of medicine, Silchar medical college and hospital, Silchar, Assam.

Materials

a. Wisp of cotton
b. Tuning fork (256)
c. Digital camera
d. CT Scan
e. Sphygmomanometer

Results and Observations

a. Agewise distribution of different types of supranuclear facial nerve palsy

49 cases from age group of 01-100 years, having supranuclear facial nerve palsy were taken from the department of medicine. Among these 18 cases were found to be in the age group of 51-60 years, 14 cases between 61-70 years, 7 cases between 71-80 years of age group, 3 cases between 81-90 years of age group. 4 cases between 41-50 years.

b. Sexwise distribution of different types of supranuclear facial nerve palsy

Out of 49 cases, 41 cases were male and 8 cases were female.

c. Distribution of supra nuclear facial nerve palsy, according to the etiology

Out of 49 cases, 40 cases were found to be due to haemorrhage, 6 cases due to infarct, 2 cases due to pathological growth, 1 case due to degenerative cause.

d. Distribution of facial nerve palsy, according to the site of lesion

Out of 49 cases, 25 cases were due to basal ganglia involvement, 12 cases were found to involve internal capsule with corona radiate, 6 cases cerebral cortex, 4 cases thalamus, 2 cases cerebellum.

Discussion

Fisher CM (1965), Tapia JF (1983), Torch RM (1990), Lauran H (1995) had described that facial nerve palsy can be of supranuclear variety, i.e. lesion can be above the pons like in the motor cortex, thalamus, basal ganglia, internal capsule and cerebellum. From the present study it has also been found that supranuclear facial nerve palsy can occur in various sites like motor cortex, basal ganglia, thalamus, cerebellum, internal capsule.

Nelson RF (1980), Fisher CM (1982), Nabutaka K (1986), Torch RM (1990) have mentioned that intranuclear haemorrhage and in fact are the main causes of supranuclear facial nerve palsy. It correlates with the present study.

Fisher CM (1965), Tapia JF (1983), Lauran H (1995), Torch RM (1990) found that most common site of supranuclear facial nerve palsy is lentiform nucleus which correlates with the present study [8,9].

Most common age group affected in supranuclear facial nerve palsy is between 50-70 years and sex is male, which correlates with findings of Baidya et al. [10].

Conclusion

From the study it is revealed that the most common site of the facial nerve lesion is supranuclear. With the help of CT Scan of brain most of the supranuclear varieties of the facial nerve lesion is found to be due to cerebrovascular accident as a result of haemorrhage or infarct. Among these haemorrhagic is most common and among location lentiform nucleus is most common. Most common age group affected is between 51-60 years and males are mostly affected (Figure 2-4).
Citation: Deka D (2016) A Study on Various Sites of Supranuclear Facial Nerve Lesion. J Neurol Stroke 4(1): 00118. DOI: 10.15406/jnsk.2016.04.00118

Figure 4: Pontine haemorrhage.

References

1. Hines S, Patrick J, Lynch MS, WilliamB (1999) Cranial nerve-facial nerve, clinical correlation-upper motor neuron lesion. Yale University School of Medicine, USA, pp. 13.

2. Ugur HC, Kahalogullari G, Coscarella E, Unlu A, Tokdemir I, et al. (2005) Arterial vascularisation of primary motor cortex. Surg Neurol 64(Suppl 2): 48-52.

3. Frebberg L (2009) Discovering biological psychology. Wadsworth Publishing, USA, Chapter 8, pp. 242.

4. Luise C, Julien B (2001) Stroke syndromes. (2nd edn) Cambridge University Press, New York, USA, p. 23.

5. Hashmi KA (2012) Blood supply of internal capsule and applied aspect. Neuros.

6. Marrison J (1976-1977) The thalamus in the motor system. Applied Neurophysiol 39(3-4): 222-238.

7. Stadning S (2008) cerebellum, diencephalon, basal ganglia, cerebral hemisphere: grays anatomy. (40th edn), Elsevier limited, Edinburgh, USA, pp. 297-335.

8. Fisher CM, Curry HB (1965) Pure motor hemiplegia of vascular origin. Arch Neurol 13: 30-44.

9. Fisher CM (1982) Lacunar stroke and infarct. American academy and neurology 32(8): 871.

10. Baidya OP, Choudhury S, Devi KG (2013) Clinico-epidemiological study of acute ischemic stroke in a tertiary hospital of northeastern state of India. International Journal of Biomedical and Advance Research 4(9): 661-665.