VOLUME - 11, ISSUE - 02, FEBRUARY - 2022 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

General Medicine

A CASE OF FOVILLE SYNDROME

Dr. Chogle Ameya* Junior resident, Department of General Medicine, Mahatma Gandhi Medical college and research institute, Pondicherry.
*Corresponding Author

Dr. Siva Ranganathan Green Professor, Department of General Medicine, Mahatma Gandhi Medical college and research institute, Pondicherry.

Dr. Chandramouli Chandni Senior resident, Department of General Medicine, Mahatma Gandhi Medical college and research institute, Pondicherry.

ABSTRACT 60 year old diabetic and hypertensive female presented to casualty with right hemiplegia and hemianesthesia with lower motor neuron type of facial nerve palsy and loss of pain, temperature on left side of the face. She had difficulty adduction and abduction on left eye with horizontal nystagmus in the contralateral eye when abducted while vertical movements were preserved. MRI brain was done which showed acute focal non hemorrhagic infarct in the left hemi pons along with partial basilar artery thrombosis. Hence a diagnosis of foville syndrome with inter nuclear opthalmoplegia was made. Classically foville syndrome is characterised by ipsilateral sixth nerve palsy, lower motor neuron facial nerve palsy, fifth sensory nuclear palsy along with contralateral hemiparesis and hemisensory loss. The patient was started on single antiplatelet along with high intensity statin. On follow up after 3 months patients power improved.

KEYWORDS : Foville's syndrome; internuclear ophthalmoplegia; Posterior circulation stroke.

INTRODUCTION

Foville syndrome was described by French anatomist and psychiatrist Achille-Louis Francois Foville in 1858. It is characterised by ipsilateral sixth nerve palsy, lower motor neuron facial nerve palsy, fifth sensory nuclear palsy along with contralateral hemiparesis and hemisensory loss. The site of the lesion is in pons.[1] Here we report a case of foville syndrome with inter nuclear opthalmoplegia due to partial basilar artery thrombosis.

Case Presentation

A 60 year female presented to emergency department of Mahatma Gandhi medical college and research institute with the complaints of weakness of right upper and lower limb for 6 hours, which was insidious in onset and gradually progressive along with deviation of angle of mouth to right and slurring of speech. She also complained of giddiness associated with nausea and one episode of vomiting. There was no history of palpitations, seizure, loss of consciousness and involuntary micturition or defecation. She's a known diabetic and hypertensive for which she was taking medications irregularly. There was no prior history of stroke or cardiovascular vascular events. At presentation she was conscious and obeying commands, her pulse rate was 82 beats per min and blood pressure was 150/90 mmHg. Central nervous system examination revealed reduced tone and power was 0/5 in right upper and lower limb. Deep tendon reflexes were exaggerated on right side and plantar was extensor. Further examination revealed right hemianesthesia along with loss of pain, temperature and crude touch on the left half of face along with weakness of both upper and lower part of the face on the left. Bilateral pupils were reacting to light. She had difficulty in adduction and abduction on left eye while vertical movements were preserved (figure 1).

Figure 1. Difficulty in adduction of left eye.

Though right eye all extraocular movements were preserved but gaze evoked nystagmus was seen. MRI Brain was done which showed acute focal non hemorrhagic infarct in the left hemi pons along with partial basilar artery thrombosis (figure 2). Hence a diagnosis of foville syndrome was made.

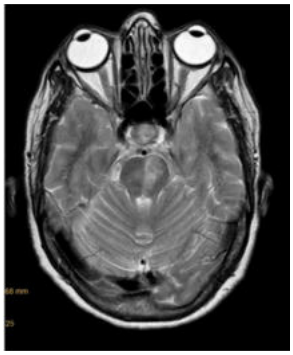

Figure 2. Mri Brain Showing Acute Focal Non Hemorrhagic Infarct In Left Hemi Pons

ECG showed normal sinus rhythm and other blood parameters were within normal limits. The patient was started on single antiplatelet along with high intensity statin. Heparin was added for deep vein thrombosis prophylaxis. Patient was given daily limb physiotherapy. In hospital blood sugars were monitored and insulin dose was adjusted accordingly. There was no worsening of sensorium or weakness. Om follow up after 3 months patient is doing well and power improved to 2/5.

DISCUSSION

Foville syndrome was first reported and described by French anatomist and psychiatrist Achille-Louis Francois Foville in 1858. Patient has crossed paralyses, ipsilateral cranial nerve deficit along with contralateral motor or sensory long tract deficits. Classically foville syndrome is characterized by ipsilateral sixth nerve palsy, lower motor neuron facial nerve palsy, fifth sensory nuclear palsy along with contralateral hemiparesis and hemisensory loss. Other rare presentations include facial hypoesthesia, peripheral deafness, ataxia, thermal hypoesthesia and Horner syndrome. Infarction of pons is the most common etiology but causes such as

granulomas, tumor and haemorrhage have also been reported.[2] In this case there was partial occlusion of basilar artery causing infraction.

The long tract signs are due to involvement of corticospinal tract and medial lemniscus. There is contralateral hemiplegia and hemisensory loss. If the descending sympathetic nerves are involved then there will ipsilateral Horner syndrome. Involvement of afferent fibres through middle cerebellar peduncle can lead to ipsilateral cerebellar ataxia.

Our patient had weakness of both upper and lower part of face on ipsilateral side suggestive of lower motor neuron type of seventh nerve palsy. Loss of pain, temperature and crude touch on ipsilateral part of the face was also present which suggests involvement of fifth sensory nuclear palsy due to involvement of sensory nucleus of trigeminal nerve. Classically there is ipsilateral sixth nerve palsy presenting as ipsilateral horizontal gaze palsy. In our case there was ipsilateral inter nuclear opthalmoplegia and ipsilateral conjugate horizontal gaze palsy along with horizontal nystagmus in the contralateral eye when abducted which is seen with the involvement of ipsilateral sixth nerve, medial longitudinal fasciculus and paramedian pontine reticular formation. Some cases reported ipsilateral sensorineural hearing loss due to eight nerve involvement.

In this patient there was partial basilar artery occlusion leading to infarction, IV tissue plasminogen activator is standard of care if there is no hemorrhage on CT and symptom onset within 4.5 hours. This patient presented outside the thrombolysis window period hence she was not thrombolysed. According to American heart association guidelines there is no clear indication for anti coagulation in management of acute stroke. Poor outcome is indicated by higher NIHSS score, lack of recanalization, symptomatic intracranial hemorrhage, history of atrial fibrillation and greater age.[3]

In conclusion, we report a rare case of foville syndrome with internuclear opthalmoplegia due to partial basilar artery occlusion. Early diagnosis and treatment leads to improvement in outcome of the patient.

REFERENCES

1. Brogna C, Fiengo L, Türe U. Achille Louis Foville's atlas of brain anatomy and the Defoville syndrome. Neurosurgery. 2012 May;70(5):1265–73; discussion 1273.
2. Cheng H-C, Yen M-Y, Wang A-G. Foville's syndrome with ipsilateral internuclear ophthalmoplegia due to spontaneous pontine hemorrhage. Taiwan J Ophthalmol. 2013 Jun 1;3(2):75–7.
3. 2021 Guideline for the Prevention of Stroke in Patients With Stroke and Transient Ischemic Attack: A Guideline From the American Heart Association/American Stroke Association | Stroke.