DOI: 10.7860/JCDR/2018/35763.12167

Conjugate Gaze Palsy-The Sole Presentation of Acute Stroke

NEERA CHAUDHRY[1], KAMAKSHI DHAMIJA[2], SANTOSH P KUMAR[3], ASHISH KUMAR DUGGAL[4], GEETA ANJUM KHWAJA[5]

Keywords: Foville syndrome, Paramedian pontine reticular formation, Pontine tegmentum

A 45-year-old male, a known case of Type 2 diabetes mellitus and primary hypertension for last four years presented to the Department of Neurology, GIPMER, New Delhi with sudden onset symptoms two weeks prior to admission in the form of imbalance while walking with a tendency to sway towards his left side. Along with this, he was unable to look towards his left side. There was history of left sided facial weakness in form of drooling of liquids from left angle of mouth and deviation of angle of mouth towards right side while smiling. He had no history of double vision, vertiginous sensation, change in speech, tremulousness of extremities or any other motor and sensory deficit.

On examination, he had a blood pressure of 140/90 mmHg and pulse rate of 84 per minute. His general physical examination was normal. The neurological examination revealed left sided gaze palsy with deficient left eye abduction which did no correct on doll's eye manoeuvre. There was left lower motor neuron seventh nerve palsy. He had inability to turn the right eye medially [Table/Fig-1]. Pupils were bilaterally normal in size and equal in reaction and there was no abduction nystagmus. Motor and sensory examination was unremarkable. He had no evidence of any limb tremor or dysdiadochokinesia however on tandem gait he swayed to the left side.

Given the sudden onset and the presence of risk factors, it was clearly a vascular event and anatomically the lesion was localised to the level of inferior pontine tegmentum, left side. The structures implicated are left sixth nerve nucleus with left Paramedian Pontine Reticular Formation (PPRF) and left seventh nerve fascicle and probably the left medial longitudinal fasciculus along with some pontocerebellar fibres. Therefore, the patient was diagnosed as Foville's syndrome [1].

His routine haematological investigations were normal. The blood sugar was raised and his fasting blood sugar was 210 mg/dL and HbA1c was 6.8%. Carotid Doppler was done which revealed 30-50% stenosis in left internal carotid artery. His echocardiography was normal. Magnetic resonance imaging of brain showed infarct in the left inferior pontine tegmentum. There was evidence of T2/FLAIR hyperintensity in the region of left inferior pontine tegmentum [Table/Fig-2].

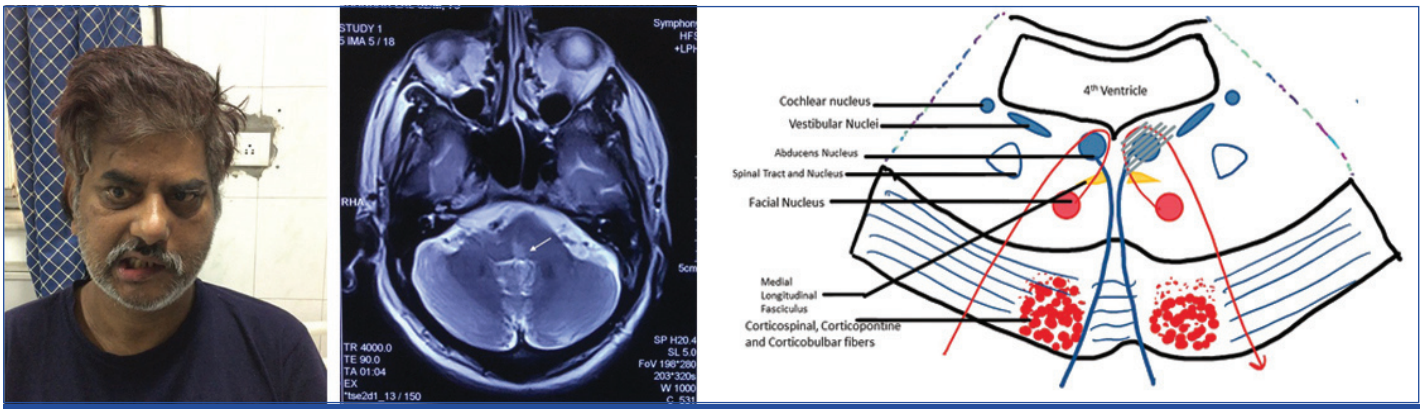
The patient was started on antiplatelets and statins. He was continued on oral hypoglycaemic agents and antihypertensives. Patient was advised good glycaemic control with dietary modifications and proper compliance with medications.

The case highlights the salient anatomical localisation for the patient's symptoms. The lower motor neuron type facial palsy localised the lesion to lower pons and the gaze palsy occurred due to the involvement of pontine tegmentum where the pontine gaze centres are located [Table/Fig-3] [2]. The syndrome is named after its first description in 1858 by Foville A [2]. The causes of this syndrome are vascular events including ischaemic and haemorrhagic strokes [3], granulomas [4] and cerebellar tumours [5].

Patients consent: The consent was procured from the patient for publishing the image.

REFERENCES

[1] Mutch JR. Foville's syndrome-record of a case. Br J Ophthalmol. 1939;23(4):225-38.



[Table/Fig-1]: Image of patient showing inability to gaze towards left and left lower motor neuron facial palsy.
[Table/Fig-2]: MRI brain image depicting an area of T2 hyperintensity in region of left inferior dorsal pons consistent with the clinical picture of patient.
[Table/Fig-3]: Figure depicting anatomical localisation of the lesion affecting left inferior dorsal pons involving paramedian pontine reticular formation, abducent nucleus, left seventh nerve fascicle.

[2] Foville A. Note on a certain paralysis of certain muscles of the eye, and its relation with some points of the anatomy and physiology of the angular protuberance. Bull Soc Anat Paris. 1858;33:393-414.
[3] Cheng HC, Yen MY, Wang AG. Foville's syndrome with ipsilateralinter nuclear ophthalmoplegia due to spontaneous pontine hemorrhage. Taiwan Journal of Ophthalmology. 2013;3:75-77.
[4] Bedi HK, Devpura JC, Bomb BS. Clinical tuberculoma of pons presenting as Foville's syndrome. J Indian Med Assoc.1973;61:184-85.
[5] Levene LJ. Benign cerebellar tumour presenting a modified Foville syndrome. J Am Geriatr Soc.1958;6:346-51.

PARTICULARS OF CONTRIBUTORS:
1. Professor, Department of Neurology, GB Pant Institute of Postgraduate Medical Education and Research, Delhi, India.
2. Senior Resident, Department of Neurology, GB Pant Institute of Postgraduate Medical Education and Research, Delhi, India.
3. Senior Resident, Department of Neurology, GB Pant Institute of Postgraduate Medical Education and Research, Delhi, India.
4. Assistant Professor, Department of Neurology, GB Pant Institute of Postgraduate Medical Education and Research, Delhi, India.
5. Director Professor, Department of Neurology, GB Pant Institute of Postgraduate Medical Education and Research, Delhi, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Ashish Kumar Duggal,
Assistant Professor, Department of Neurology, GB Pant Institute of Postgraduate Medical Education and Research,
Delhi-110002, India.
E-mail: ashishduggal2005@rediffmail.com

FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Jan 31, 2018**
Date of Peer Review: **Mar 30, 2018**
Date of Acceptance: **Jun 15, 2018**
Date of Publishing: **Oct 01, 2018**