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CASE REPORT

BRAINSTEM STROKE FROM RARE CASE OF FOVILLE SYNDROME

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Abstract

Introduction: Foville syndrome is a rare neurological syndrome resulting from infarction in the perforating branches of the basilar artery, involving the nuclei of cranial nerves VI and VII and the corticospinal tract. It is clinically characterized by ipsilateral horizontal gaze palsy, ipsilateral peripheral facial paralysis, and contralateral hemiparesis. Its rarity often poses diagnostic challenges in clinical practice.

Case Presentation: A 54-year-old woman presented with sudden diplopia and right-sided weakness for 16 hours. She had a history of controlled hypertension and diabetes mellitus. Neurological examination revealed left horizontal gaze palsy, left peripheral facial weakness, and right hemiparesis (muscle strength 2/5). Brain CT showed pontine infarction with severe basilar artery stenosis. The patient was treated with dual antiplatelet therapy, high-intensity statin, and supportive therapy including eye patching and physiotherapy. She was discharged after 8 days with moderate disability (mRS 3) and showed improvement to mild disability at 3-month follow-up.

Discussion: Foville syndrome results from lesions in the pontine tegmentum involving the abducens nucleus, facial nerve fibers, and corticospinal tract. It is commonly caused by ischemic infarction due to atherosclerotic basilar artery disease. Clinical findings in this case align with the classic triad. Management depends on onset time; thrombolysis was not indicated due to delayed presentation. Dual antiplatelet therapy and risk factor control remain the mainstay treatment.

Conclusions: We report a rare case of Foville syndrome that arises as a result of an infarction stroke affecting the pons.

Keywords: basilar artery stenosis, brainstem stroke, Foville syndrome, pontine infarction

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Introduction

The nervous system plays a vital role as the primary command center that regulates all physiological functions and coordinates the organs within the human body.[1] Through the transmission of complex electrochemical signals, this system is responsible for integrating sensory perception, motor responses, and autonomic functions that are crucial for survival. Its existence ensures that every biological command can be executed with

precision by target tissues throughout the body.[2] However, despite its central role, the functional integrity of the nervous system is highly vulnerable to various pathological disorders that can lead to a drastic decline in quality of life. One of the most significant manifestations of neurological disorders requiring in-depth clinical attention is stroke. This condition is characterized by a disruption in blood flow to brain tissue, which triggers rapid cellular damage and disrupts the chain of commands from the central nervous system to the entire body.[3]

Clinically, a stroke is defined as an acute neurological disorder caused by an interruption in blood flow to the brain, resulting either from a blood vessel occlusion or a vascular rupture. Based on its pathophysiology, stroke is classified into two main categories: ischemic stroke, which accounts for the majority of cases through thromboembolic mechanisms, and hemorrhagic stroke, which involves intracerebral or subarachnoid hemorrhage.[3] The urgency of stroke management is driven by its steadily rising global prevalence, with the latest data indicating that stroke remains the leading cause of long-term disability and the second leading cause of death worldwide. Stroke is also one of the most common neurological disorders, with 800,000 cases worldwide, and it causes the deaths of at least 5 million stroke patients globally.[4] The risk factors underlying this condition are multifactorial, ranging from non-modifiable variables such as age and genetics to modifiable risk factors such as hypertension, diabetes mellitus, dyslipidemia, and a sedentary lifestyle.[5]

Although most stroke manifestations involve the anterior circulation, affecting general motor and cognitive functions, there is a spectrum of vascular lesions that occur in more specific and rarely encountered anatomical locations, one of which is Foville's syndrome. Foville's syndrome is a variant of inferior pontine syndrome resulting from an infarction in the perforating branches of the basilar artery. Pathophysiologically, this condition involves the nuclei of cranial nerves VI (abducens) and VII (facial), as well as the corticospinal tract traversing the pons. The resulting clinical effects are highly characteristic, consisting of a combination of ipsilateral facial muscle paralysis, eye movement disorders (horizontal gaze palsy), and contralateral hemiparesis of the limbs. Discussions regarding Foville syndrome are of critical importance in current medical literature because its rarity often poses diagnostic challenges. A thorough understanding of this syndrome is necessary so that clinicians can accurately localize the lesion and provide appropriate interventions to minimize permanent neurological deficits in patients.[6] Furthermore, this case report aims to present the clinical presentation and therapeutic management strategies for a patient diagnosed with Foville Syndrome due to acute pontine ischemic stroke. It is hoped that this report will enhance clinical awareness and improve the accuracy of neuroanatomical lesion localization in this rare variant of brainstem stroke.

Case Presentation

A 54 year old female with sudden onset of diplopia and right side weakness with a long history of well controlled blood pressure and diabetes. She complained of double vision for 16 hours before hospital admission. The patient has no history of similar previous events. She took candesartan 16 mg and metformin 500 mg routinely. She has no history of head injury, diabetes, or malignancy. During neurological examination, the patient was cooperative and fully conscious. There was abnormality of eye movement to the temporal in the left eye and slight peripheral facial weakness in the left side. The patient had right side weakness with muscle strength 2 from 5 medical council research scale. Further brain CT examination showed pons infarction and severe basilar artery stenosis. The patient was treated with loading dose double anti platelet therapy and also given high intensity statin (atorvastatin 40 mg), folic acid, and B12 vitamin injection. Digital subtraction angiography and the possibility of stenting for the stenosis of the basilar artery was suggested, but the patient refused the suggestion. Intermittent eye patching and physical therapy was also given. The patient was hospitalized for 8 days, and was discharged from hospital with modified Rankin scale 3 (moderate disability). During 3 month follow up, the patient had mild disability, and continue the dual anti platelet therapy (clopidogrel 75 mg and aspirin 80 mg daily), atorvastatin 20 mg, candesartan 16 mg, metformin 500 twice daily, and folic acid.

(a)

(b)

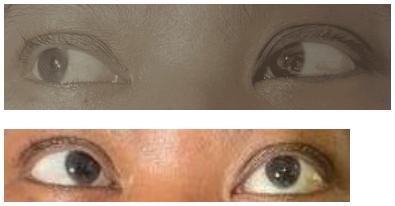

Figure 1. The palsy of left abducens nerve

(a)

(b)

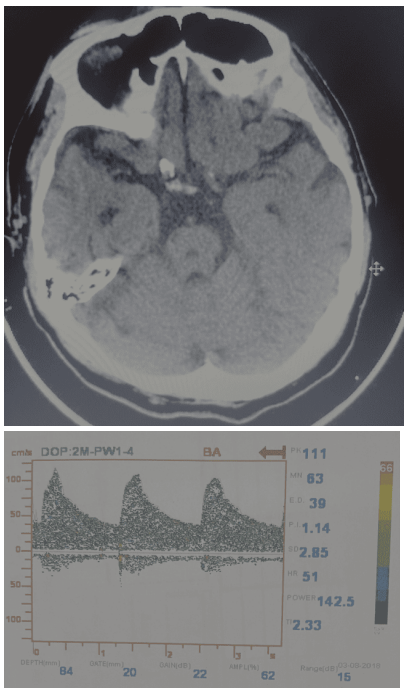

Figure 2. The pons stroke with severe basilar artery stenosis from trans cranial doppler examination

Discussion

Foville syndrome is a rare neurological syndrome caused by lesions in the inferior-medial tegmentum of the pons, and is clinically characterized by a triad of ipsilateral horizontal gaze palsy, ipsilateral peripheral facial paralysis, and contralateral hemiparesis. Pathologically, this lesion is located in the abducens nucleus or the Paramedian Pontine Reticular Formation (PPRF), the facial nerve fasciculus (CN VII), and the corticospinal tract prior to decussation. Foville syndrome is very rare and most of the available

information comes in the form of individual case reports, therefore comprehensive epidemiological information is still limited. Furthermore, this syndrome is frequently misdiagnosed with other brainstem syndromes that present in a similar manner.[6]

Foville syndrome caused by pontine infarction and other etiologies such as hemorrhage, granuloma, and pontine tumors. Atheromatous plaques in the basilar artery can block smaller paramedian pontine branches through its perforating ostium, resulting in occlusion and infarction. These infarctions may also occur due to reduced blood flow in the paramedian perforating arteries originating from the basilar artery. Hemiparesis may result from extensive infarction of the unilateral corticospinal tract, while ipsilateral abducens and facial nerve palsies may occur due to involvement of the nerve fibers and nuclei. A history of controlled hypertension and diabetes mellitus may also be risk factors contributing to the chronic atherosclerotic process in the basilar artery, which eventually triggers a pontine infarction and results in Foville syndrome.[6,7]

In our case, the patient presented with symptoms of diplopia, impaired temporal eye movement in the left eye, left-sided peripheral facial weakness, and right-sided weakness with a muscle strength of 2/5 on the Medical Research Council scale. These findings are consistent with previous reports stating that Foville syndrome is characterized by a variable clinical presentation due to the involvement of multiple structures, including involvement of the sixth cranial nerve nucleus, which causes restricted abduction and non-crossed diplopia; involvement of the facial nerve nucleus, causing ipsilateral lower motor neuron facial palsy affecting the upper and lower parts of the face, and involvement of the corticospinal tract, causing contralateral hemiparesis or hemiplegia.[8,9]

Thrombolytic therapy is typically administered to patients who present within the 4.5-hour “golden period” following symptom onset without contraindications.[6,10] In our case, thrombolytic therapy was not administered since the onset of symptoms had exceeded the golden period, which is 16 hours prior to hospital admission. As a result, the primary treatment consisted of antiplatelet therapy with aspirin and clopidogrel. It is also not recommended for patients with severe intracranial stenosis in the vascular territory of an ischemic stroke to receive angioplasty and stenting as first-line therapy. Aggressive medical management of risk factors and short-term dual antiplatelet therapy are preferred.[11] In our patient, although digital subtraction angiography and possible stenting of the basilar artery stenosis were recommended, the patient refused these procedures. Treatment was continued with dual antiplatelet therapy, a high-intensity statin (atorvastatin 40 mg), folic acid, and vitamin B12 injections. Along with pharmacological management, patients also received rehabilitation consisting of intermittent eye patching to address diplopia and physical therapy for motor weakness.

In general, the prognosis for ischemic lesions is more favorable than that for hemorrhagic lesions, and most

patients with pontine paramedian artery infarction have a good prognosis. In our case, the patient had moderate disability at discharge but improved significantly. Some factors may have contributed to this improvement, such as ischemic etiology, the adherence of the patient to dual antiplatelet therapy and statins, and the control of risk factors including hypertension and diabetes mellitus.[6]

Conclusion

We report a rare case of Foville syndrome that arises as a result of an infarction stroke affecting the pons.

Conflict of Interest

The authors declared no conflict of interest.

Acknowledgment

The authors declared no acknowledgement.

Consent

Verbal informed consent has been obtained from the patient and family.

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