Bilateral Paramedian Thalamic Infarction Presenting as Status Epilepticus: A Case Report and Review of the Literatures

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Abstract

Purpose: Occlusion of the artery of Percheron (AOP), a rare vascular variant of basilar artery branch, is presumed to cause bilateral paramedian thalamic infarction. We present a case of acute AOP infarction with status epilepticus.

Case Report: A 65-year-old woman had past history of hypertension, type 2 diabetes mellitus, and major depressive disorder. She was found to have altered mental status on awakening. She developed tonic convulsion and progressed to status epilepticus later. The brain magnetic resonance imaging (MRI) showed acute bilateral paramedian thalamic and interpeduncular mesencephalic infarction. The electroencephalography (EEG) showed continuous epileptiform discharges. After receiving antiplatelet and anticonvulsant agents, she regained her level of consciousness and has completely recovered to previous baseline.

Conclusions: To our knowledge, this is the first case of AOP infarction presenting status epilepticus. Early recognition and treatment of seizure may reverse altered mental status in those patients.

Key Words: artery of Percheron, thalamic infarction, status epilepticus

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INTRODUCTION

Paramedian thalamus is usually supplied from each side of thalamoperforating arteries. In 1973, a French neurologist Gerard Percheron identified a rare single basilar artery branching variant, which supplies bilateral sides of paramedian thalamus. This rare variant artery was later named as the "artery of Percheron" (AOP) [(1)]. Occulsion of AOP is presumed the main cause of bilateral paramedian thalamic infarction[(2)], which may combine with or without ischemic interpeduncular mesencephalic infarction[(3)]. Typical clinical features of bilateral paramedian thalamic infarction include the triad of altered mental status on the spectrum from difficult

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arousal to coma, vertical gaze paresis, and memory defects[3]. Convulsive movement is an unusual presentation of occlusion of AOP[4]. Here, we report a patient with bilateral paramedian thalamic and interpeduncular mesencephalic infarction presenting status epilepticus.

CASE REPORT

A 65-year-old woman had acute onset of altered mental status on wakening. She has past history of hypertension, type 2 diabetes mellitus, and major depressive disorder. One day before her admission, her husband noticed that she complained of bilateral periorbital dull pain. He also mentioned that she had bizarre behavior, and could not remember any things happened earlier in the morning. When she was brought to our emergency department in the morning, she had fever up to 39 degrees Celsius and was in a stupor status (Glasgow Coma Scale, GCS E2V2M4). In the afternoon she developed intermittent paroxysmal tonic convulsion in her left extremities, which was abolished after intravenous loading with valproic acid 800 mg. The laboratory panel showed leukocytosis with 21,550 μL in white blood cell count and 93.5% neutrophil. The cerebrospinal fluid study showed white blood cell count 8/μL with 92% neutrophil and 8% lymphocyte, glucose 86 mg/dL, and total protein 25 mg/dL. On the next day, both sides of her extremities developed persistent tonic convulsion. The finding of the brain magnetic resonance imaging (MRI) revealed acute bilateral paramedian thalamic infarction and patent bilateral posterior cerebral arteries on magnetic resonance arteriography (MRA). (Figure 1) The electroencephalography (EEG) showed continuous generalized epileptiform discharges and polyspike complexes with emphasis on F8, F4, and F7 leads (Figure 2). Then we gave her intravenous anticonvulsants with levetiracetam 1,000 mg q12 h and phenytoin 500 mg as loading dose as well as phenytoin 100 mg q8 h as maintenance dose due to frequent jerky eye movements to both sides. After combination therapy of those three

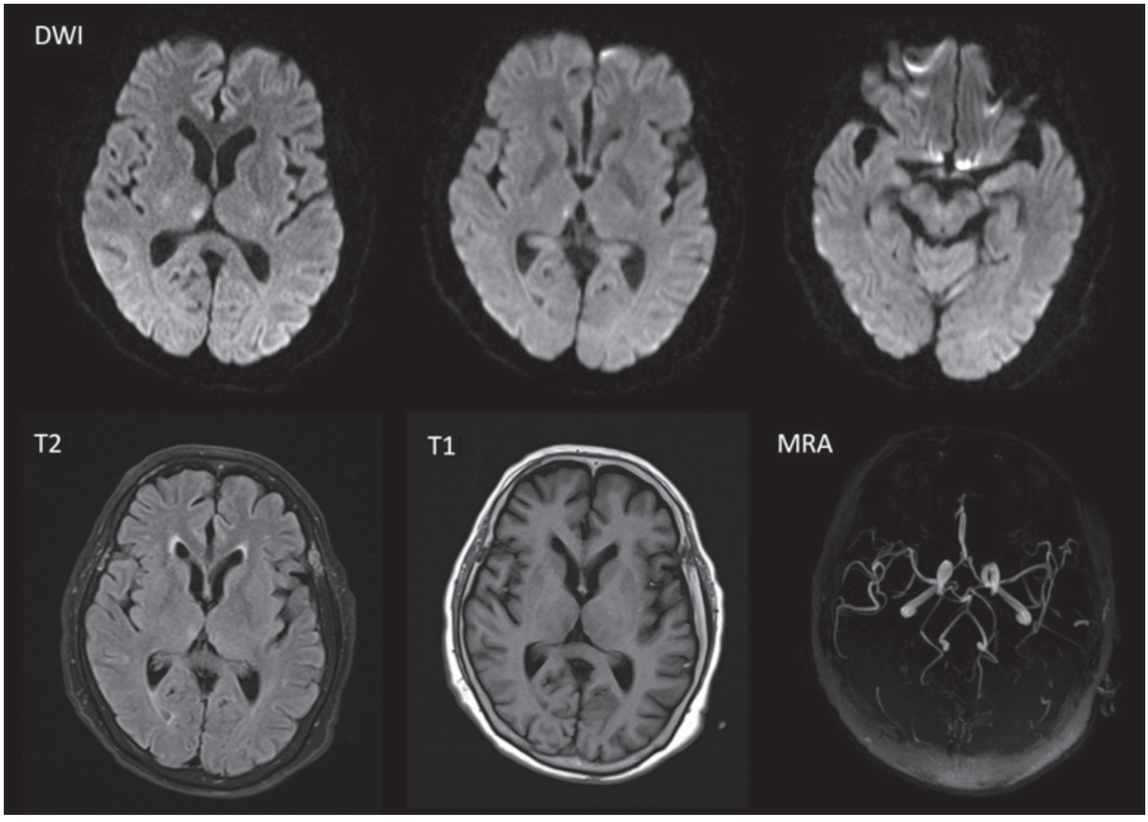

Figure 1. The brain MRI revealed recent bilateral paramedian thalamic and interpeduncular midbrain infarction.

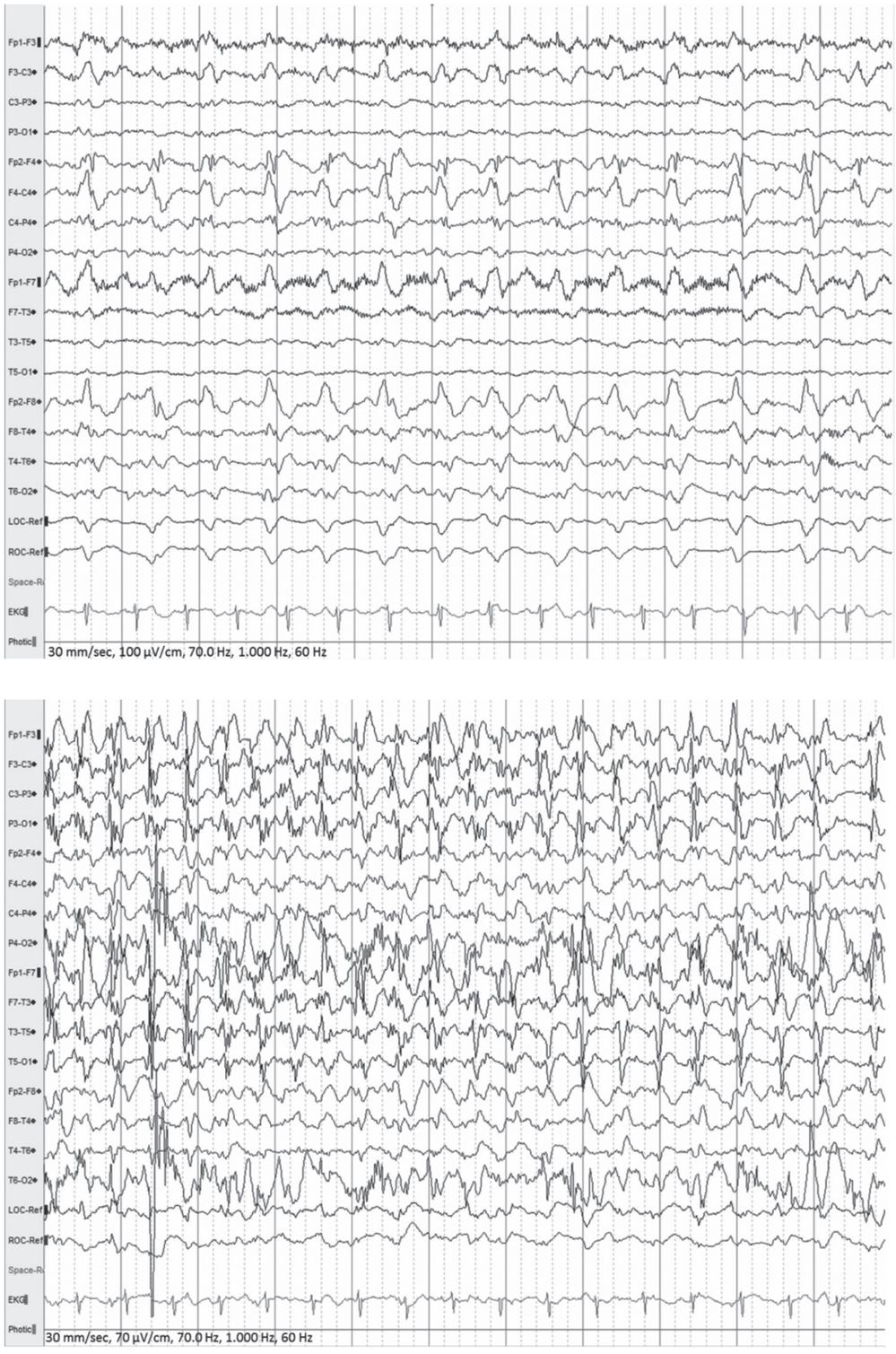

Figure 2. The EEG showed continuous polyspike complexes on F8, F4, and F7 and then progressed to generalized polyspike complexes discharges.

anticonvulsants for two days, the patient regained her level of consciousness (GCS E4V4M6) finally. She could follow simple order, and the follow-up EEG showed markedly reduced epileptic activity in comparison to the previous one. After eight days of intensive care, she became oriented and had almost completely recovered to previous status. We shifted her anticonvulsants to oral form with levetiracetam 1,000 mg q12 h and valproic acid 400 mg BID. The patient was discharged after taking rehabilitation program. She has received regular follow-up in our outpatient clinic.

DISCUSSION

Seizures in bilateral medial thalamic infarction are rarely reported in the literature (Table 1)[2,4,5-11]. Most of the documented EEG findings in these cases revealed no epileptiform discharges. Jasper, Droogleever-Fortuyn, and Penfield postulated that neurons in the brainstem reticular formation (RF) and basal diencephalon functioned as the pacemaker of seizure, and neurons in thalamus has also been supposed to be a relay center of both ascending and descending route of brainstem RF in the centrencephalic system model[12,13]. Animal studies showed electrical stimulation of mesencephalic RF can induce convulsion in limbs[14]. The electrical stimulation induced limb convulsion was abolished by creating caudal lesions in pontine tegmentum[14]. It means that tonic activity was attenuated by blocking the descending route. On the other hand, thalamus has been supposed to have backward and forward connections with cerebral cortices in the centrencephalic system model[13,15]. EEG was also a reflection of summation of neuronal activity of thalamic and cortical neurons in the thalamo–corticothalamic network[16]. Thus, we considered that activation of descending route of RF should explain the tonic convulsion in most cases and the activation of both descending and ascending routes of RF may explain tonic convulsion and ictal EEG finding of the polyspike complexes in our case.

Infarction of AOP includes four distinct patterns, namely bilateral paramedian thalamic infarction either with or without ischemic interpeduncular mesencephalic involvement, and bilateral anterior thalamic infarction either with or without ischemic interpeduncular mesencephalic involvement[3]. Revealing an arising

Table 1. Case series of bilateral medial thalami with or without midbrain infarctions with convulsion presentation

Age (years)/Sex	Time	Movements	Stroke risk factors or comorbidities	Reference
51/M	Onset	Convulsive seizure	Onset hypertension	Segarra et al. 1970[5]
51/M	Onset	Clonic movements of both arm	(No mentioned)	Castaigne et al. 1987[6]
29/M	Onset	Clonic limb movements	(No mentioned)	Ropper 1988[7]
	2 weeks	Tonic-clonic right arm shaking followed by tonic extension of the right arm and leg		
50/M	Early	Tonic-clonic movements in all limbs	CAD post stenting, hypertension, hyper- cholesterolemia,	Matheus et al. 2003[2]
76/M	Onset	Clonic movements of both arms	Hypertension, hyperlipidemia, aortic regurgitation, hypertrophic cardiomyopathy	Naganuma et al. 2005[8]
70/M	Onset	Generalized clonic seizure	Atrial fibrillation	Naganuma et al. 2005[8]
0 (2 days) /M	Onset	Right-hand sided clonic seizures	MTHFR heterozygote mutation	Bain et al. 2009[9]
84/ F	Onset	Clonic movement in upper limbs	Alzheimer's disease, depression	Wells et al. 2011[10]
71/M	Onset	Clonic movements of both arms	Chronic hemodialysis,	Yamashiro et al. 2011[4]
66/M	Onset	Clonic movements of all four limbs	Atrial fibrillation, ischemic stroke	Wang et al. 2013[11]
65/F (our case)	Onset	Tonic convulsion of four limbs	Atrial fibrillation, hypertension, diabetes mellitus, depression	Current case report

CAD, Coronary artery disease; MTHFR, Methylenetetrahydrofolate reductase

solitary trunk of AOP from PCA is usually limited on MRA. The restricted visualization on MRA includes two main reasons (A) the vessel is occluded[2], and (B) the inadequate signal-to-noise ratio in modern 1.5 Tesla systems, which is however still widely used in the world. Besides, performing conventional angiography only for the purpose of revealing an occluded AOP on imaging may not be indicated. Finally, imaging of bilateral paramedian thalamic infarction despite lack of visualization of AOP on MRI or MRA does not exclude the cause of AOP infarction[2].

Clinical deficits of AOP infarction include obtundation, oculomotor and pupillary deficits, vertical gaze paresis, and memory defects[3]. These symptoms and signs should correspond to their neuroanatomical lesions. Altered mental status presents on the spectrum from difficult arousal, confusion, stupor to coma. Changed consciousness is understood as interruption of ascending reticular activating system that is localized in the mesencephalic neurons. These neurons project their fibers to both thalami and then to the cortex[17]. Oculomotor and pupillary deficits are also implicated in mesencephalic lesions. Vertical gaze palsy is explained by disruption of supranuclear inputs that traverse the thalamus on their way to the rostral interstitial medial longitudinal fasciculus[18]. It has been proved by existence of vertical gaze palsy in AOP cases without midbrain involvement[18]. Memory defect is suggested to be a result from disruption of mammillothalamic tract and anterior thalami, both of which belong to the internal connected components of Papez circuit[17].

CONCLUSION

In summary, convulsive movements were rarely seen in bilateral paramedian thalamic infarction. Our patient is the first one, who is reported to have bilateral paramedian thalamic infarction with initial presentation of status epilepticus. Although difficult arousal and coma are predicted symptoms among cases of bilateral thalamic infarction, we learned that early recognition of epileptic discharges and treatment of seizure may reverse altered mental status in AOP infarction.

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