Infarction of Bilateral Paramedian Mesencephalic: Diencephalic Junctions

Yu-Hua Lai, Jiann-Chyun Lin

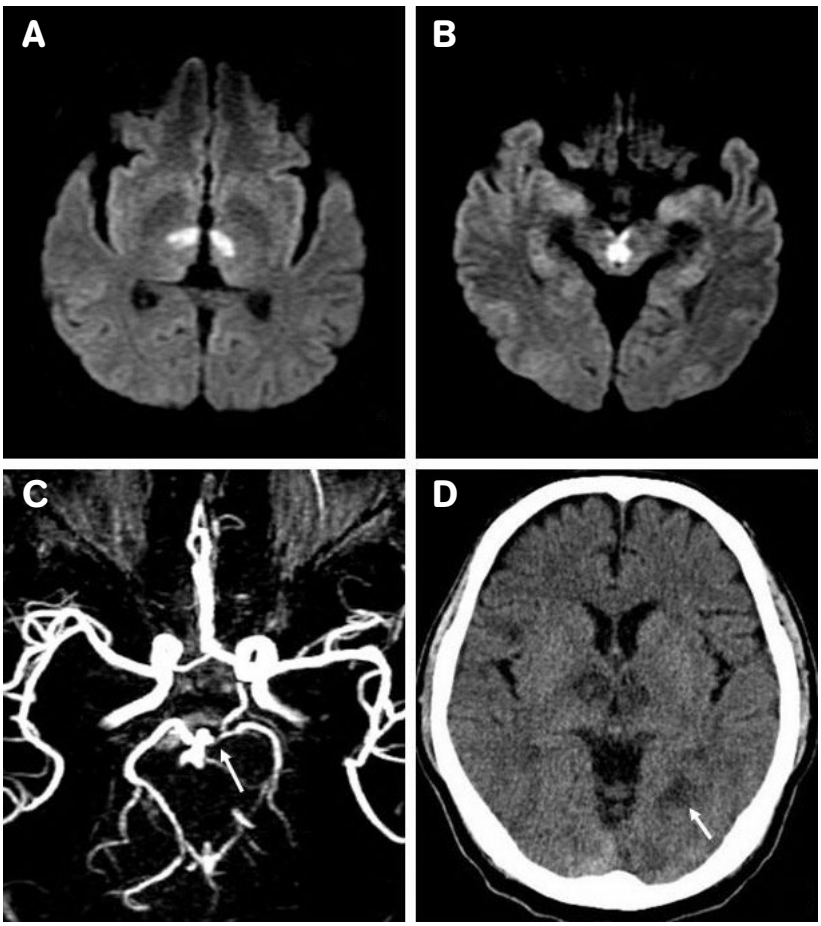

Figure 1. MR diffusion-weighted image at 6 hours of symptom onset denoted the restriction of water diffusion over bilateral paramedian thalami (A) and rostral midbrain (B). MR angiography (C) demonstrated a focal stenosis over the first segment of left posterior cerebral artery (white arrow). Brain computed tomography on three days later (D) showed hypodensity lesions at bilateral thalami as previously noted and a new-developed, poor-demarcated hypodensity lesion at left occipital lobe (white arrow).

From the Department of Neurology, Tri-Service General Hospital, Taipei, Taiwan.
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Correspondence to: Jiann-Chyun Lin, MD. Department of Neurology, Tri-Service General Hospital, No. 325, Section 2, Cheng-Kung Road, Neihu 114, Taipei, Taiwan, R.O.C.
E-mail: neurojcl@ndmctsgh.edu.tw

A 57-year-old male was brought to the Emergency Department because of acute alteration of consciousness. He had hypertension and diabetes mellitus with regular control for years. On admission, he was comatose without verbal output. His bilateral pupils measured 5mm and did not constrict to light. Eyeballs were fixed in neutral position and passive head movement failed to elicit any eye movement. The corneal and gag reflexes were normal. Noxious stimuli produced bilateral limb withdrawal reflexes symmetrically. No brisk tendon reflex or extensor plantar reflex was detected. His complete blood count, blood biochemistry and coagulation function tests were within normal limits. Electrocardiogram demonstrated normal sinus heart rate without arrhythmia. Initial brain computed tomography (CT) scan showed no remarkable finding. The emergent magnetic resonance (MR) imaging denoted acute infarctions over bilateral paramedian thalami and rostral midbrain (Fig. A, B). MR angiography presented a focal stenosis over the first segment of left posterior cerebral artery (Fig. C). The antiplatelet agent was prescribed for stroke prevention. Three days later, he received a follow-up brain CT scan because of a drop in the Glasgow coma scale of 2 points. The image revealed a newly developed hypodensity lesion at left occipital lobe, compatible with territory of left posterior cerebral artery (Fig. D). Low-molecular-weight heparin therapy was started to prevent recurrent ischemic event and there was no further neurologic deterioration. The patient remained comatose four months later.

The artery supplying bilateral paramedian mesencephalic-diencephalic junctions mainly origins from the first segment of posterior cerebral artery (P1 segment) in each side, separately. However, in some of anatomic variants, these perforating branches may arise from the ipsilateral P1 segment or a common trunk, called “artery of Pecheron” [(1)], especially if another side of P1 segment is absent or hypoplasia. Therefore, once occlusion of ipsilateral P1 segment or its perforating branches happen, the ischemic area may involve bilateral but usually asymmetric paramedian thalami and rostral midbrain. The patient presented herein had infarctions over the similar area with complete patency of the basilar artery and a focal stenosis over left P1 segment. He had a recurrent infarction over left posterior cerebral artery three days after admission. It implied that his bilateral paramedian thalami and midbrain are supplied via the same origin, left P1 segment. The clinical manifestations of the occlusion of artery of Pecheron are complex and wide-ranged [(2)], including bilateral oculomotor palsy without light reflex, pseudo-abducens palsy, confusion, hypersomnolence, abnormal circadian wake-sleep cycle, and even coma. Moreover, the “top of the basilar” syndrome [(3)] may present with similar clinical manifestation, except the occlusion of the basilar artery and infarctions in rostral brainstem and bilateral posterior cerebral artery territory on brain image.

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