Artery of Percheron infarction

An 85 year-old woman with normal cognitive function and ability to walk unaided was admitted to hospital in May 2011 because of sudden onset of stupor and a fall at home. She had a history of hypertension, diabetes mellitus, and atrial fibrillation, and was taking aspirin. On physical examination, she was somnolent with Glasgow Coma Scale score of 8/15 (eye 1/4, motor 6/6, verbal response 1/5). Power was grade 3/5 in all four limbs. Plantar reflexes were down going and tendon jerks were not brisk. There was no vertical gaze palsy. Respiratory, cardiovascular, and abdominal examination was unremarkable. The admission random blood glucose level was 7.2 mmol/L. Complete blood picture, routine biochemistry, thyroid function tests, vitamin B12, and folate levels were all within normal limits, and her chest X-ray was unremarkable. The initial computed tomography (CT) showed no obvious brain lesion (Fig 1a). When repeated 3 days later, hypodensities were evident over the medial aspects of both thalami (Fig 1b). Magnetic resonance imaging (MRI) [Fig 2] and magnetic resonance angiography (MRA) [Fig 3] of the brain showed T2-weighted hyperintense signals over both thalami and the rostral midbrain, but there was no obvious stenosis of major intracranial vessels (including the basilar artery). The MRI was suggestive of an acute infarct in the territory of artery of Percheron. Echocardiography showed no obvious valvular lesion or intra-cardiac thrombus. After 2 months of rehabilitation, the patient still had

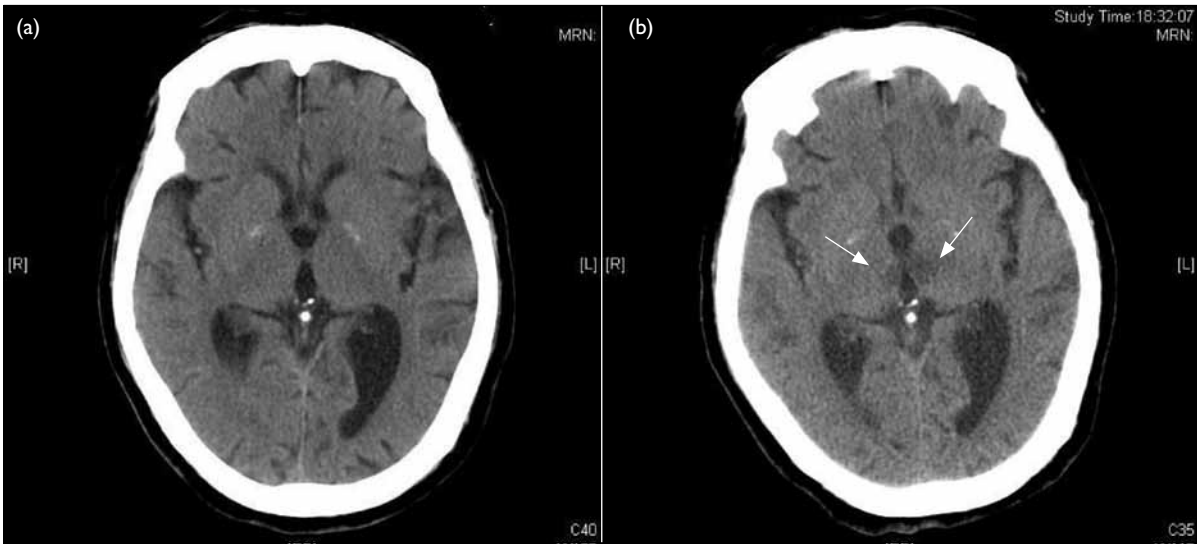

FIG 1. (a) The initial normal computed tomographic brain upon admission, and (b) on day 3, it shows hypodensities over the medial aspects of both thalami (arrows)

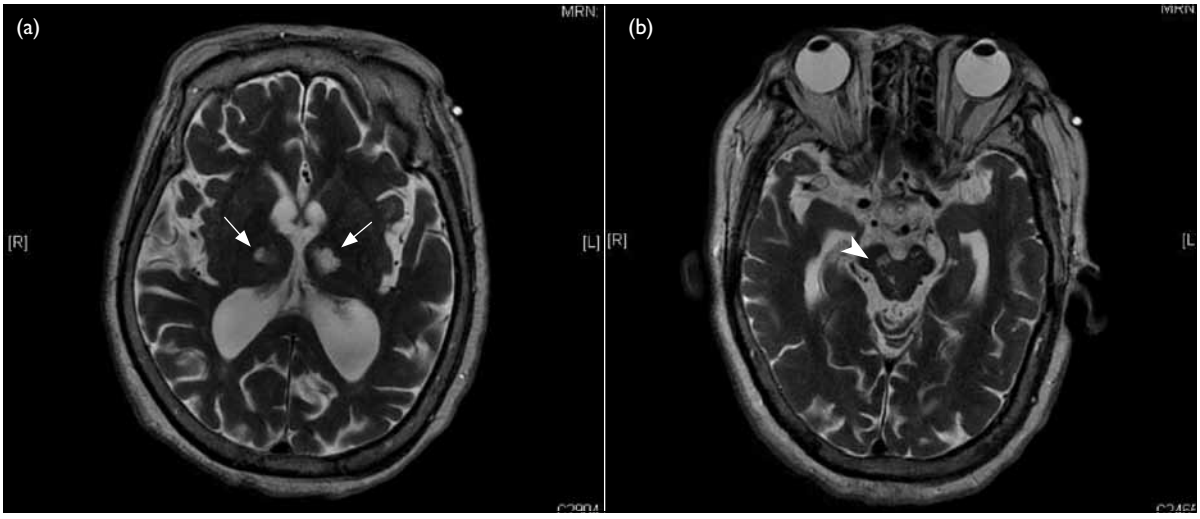

FIG 2. T2-weighted axial brain magnetic resonance images showing (a) bilateral hyperintense signals over the medial part of each thalamus (arrows), and (b) hyperintense signals over the right crus cerebri (arrowhead) at the level of the midbrain

poor memory and could only walk with considerable assistance. She also had dysphagia, for which nasogastric tube feeding was deemed necessary.

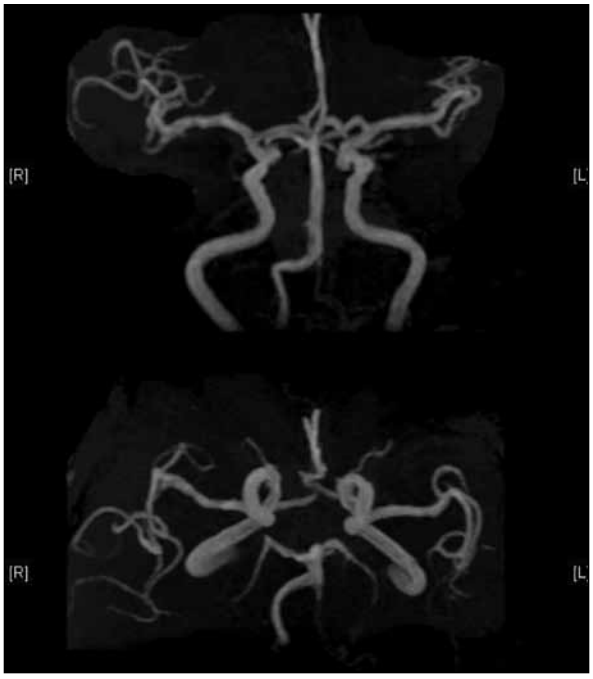

FIG 3. Magnetic resonance angiography demonstrating the patent basilar and posterior cerebral arteries; the left vertebral artery is hypoplastic

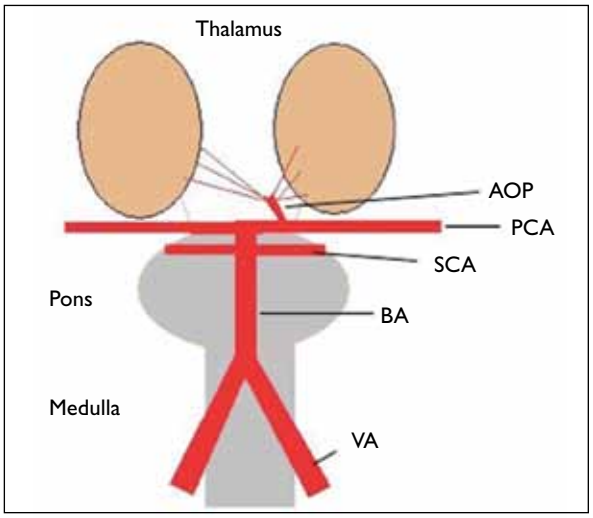

FIG 4. A schematic diagram illustrating the artery of Percheron (AOP)
PCA denotes posterior cerebral artery, SCA superior cerebellar artery, BA basilar artery, and VA vertebral artery

Discussion

The medial part of the thalamus is usually supplied by the paramedian arteries that arise from the first segment of posterior cerebral arteries on both sides. In about one third of humans, these arteries arise from a single artery known as the artery of Percheron (AOP) [Fig 4],[1] which is too small to be visualised by MRA[2] and can only be detected on angiograms.[3] As in our patient, midbrain infarct may also result from AOP occlusion, which usually also involves the peri-aqueductal grey matter.[4] Our patient presented with two out of the three typical features of this stroke syndrome, that is, altered mental status and memory impairment.[4] The altered mental status is explained by involvement of reticular activating system[2] and the disrupted connections between the thalamus and the anterior, orbitofrontal and medial prefrontal cortices.[1] The third feature of the triad is vertical gaze palsy, which is due to disruption of the cortical input that traverses the thalamus to reach the rostral interstitial medial longitudinal fasciculus.[1,4] Some patients may also have oculomotor nerve palsy and hemiplegia. Such AOP infarcts account for 0.1 to 0.3% of all ischaemic strokes,[3] and 22 to 35% of all thalamic infarcts[1] for which the commonest aetiology is embolism from the heart.[1] Early diagnosis is best made by a diffusion-weighted imaging (DWI) sequence using MRI.[5] The AOP infarct could also show up as bilateral symmetrical hyperintense signals in both thalami on fluid-attenuated inversion recovery images. Decreased conscious level, lack of focal motor or sensory deficit, and bithalamic hypodensities in CT of the brain are unusual in typical stroke syndromes. Hence the diagnosis may be delayed and the therapeutic window for thrombolytic therapy can be missed, and result in significant neurological impairment. For patients presenting with sensorial change, vertical gaze palsy should be looked for and consideration given to undertaking an MRI with DWI sequencing, in order to diagnose AOP infarction when thrombolytic therapy might still be feasible.[3]

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