Two cases of bilateral thalamic infarction are discussed. Both patients presented similarly with decreased consciousness and visual disturbance within a five month period of a stroke rotation. Magnetic resonance imaging (MRI) revealed bilateral paramedian thalamic infarctions. Magnetic resonance (MR) angiography showed an intact posterior circulation with the tip of the basilar artery and both posterior cerebral arteries patent, indicating a possible complete occlusion of the artery of Percheron.

Bilateral thalamic infarction is rare, accounting for only 0.6% of all cerebral infarctions, and results from a combination of predisposing factors and anatomic variations.1 The pattern of bilateral thalamic stroke is usually seen after paramedian artery infarct of the so called artery of Percheron (AOP); this is a rare anatomical variant of thalamic blood supply arising from the posterior cerebral artery, providing bilateral arterial supply to the paramedian thalami and the rostral midbrain. AOP occlusion accounts for 4-18% of all thalamic strokes and 0.1-2% of all ischaemic strokes.2,3

A CT head showed no acute abnormalities. He was commenced on high dose aspirin for a possible transient ischaemic attack (TIA). On the ward he was noted to be very sleepy, although easily rousable, and not engaging with therapy. He was orientated in person, time and place but would fall asleep mid-sentence. His increased somnolence continued and an MRI confirmed a bilateral thalamic infarct. When somnolence improved after a few days, a vertical gaze disturbance and intermittent diplopia was noted and later confirmed via orthoptics. An MRI angiogram showed an intact posterior circulation with the tip of the basilar artery clearly visible.

The prevalence of the AOP is unknown; this is a rare anatomical variant. Although infarcts restricted to the thalami were reported for the first time more than 100 years ago by Dejerine and Roussy, they remain an uncommon presentation of stroke and account for only 11% of all vertebrobasilar infarcts.4 Bilateral thalamic infarction is rare, accounting for only 0.6% of all cerebral infarctions.5 The differential diagnosis also includes the “top of the basilar artery” syndrome. In this latter entity, infarctions tend to involve also the territories supplied by the superior cerebellar and PCAs.6 We propose that when bilateral medial thalamic infarcts are found, an occlusion of the AOP should be considered as the main diagnosis.

The thalamus is involved in several functions of the body including: regulation of sleep and wakefulness, motor control, receiving auditory, somatosensory and visual sensory signals, and relaying sensory signals to the cerebral cortex. Thalamo-cortico-thalamic circuits are involved in consciousness, arousal, level of awareness, and activity. The four main symptoms found in the literature for patients with thalamic infarcts are vertical gaze palsy (65%), memory impairment (58%), confusion (53%), and comas (42%).8 In approximately 50% of cases, patients will present with reduced alertness, memory impairment, vertical gaze palsy and psychological disturbance.9 Patients may take days to weeks to recover from a thalamic infarct and seem to be in a sleep-like state. In our cases, somnolence cleared in our patients, vertical gaze palsy became apparent.

MRI including diffusion-weighted sequences (DW-MRI) has demonstrated high sensitivity for acute brainstem and isolated thalamic infarctions beginning 12 hours after ischaemic attack.9 However, there are reported cases where even MRI was normal.10 In our cases, the initial CT head scan without contrast, done on the day of admission, was normal, and MRI brain showed bilateral paramedian infarction. MRA, in case 1 showed normal flow of the vertebral arteries and the AOP could not be visualised due to complete occlusion. Performing conventional angiography may not be indicated, because lack of visualization of the artery does not exclude its presence.

Depending on clinical/anatomic variations, patients can present with different symptom complexes as a part of paramedian thalamic syndrome. Decision about thrombolysis will be difficult and will partly be affected by the initial image finding. Whether these patients should have urgent MRI so they can be treated within the time limit is a question worth considering. It has been suggested that the time window for treatment for basilar occlusion may be longer than for other stroke types, and although treatment within 4.5 hours is desirable, it may be reasonable to consider treatment (intravenous or endovascular) up to 24 hours from onset.11

Although previous case reports have noted poor prognosis, our observation as seen in the above cases show a dramatic improvement with discharge back to baseline.

Learning points:

- Increased awareness about early recognition of thalamic strokes including the differential diagnosis between sleepiness and actual coma.
- Artery of Percheron infarction should be considered in the above clinical scenario
- Appropriate imaging aid in early diagnosis which might affect the management of stroke
- Artery of Percheron occlusion may be commoner than previous thought due to improvements in brain imaging techniques


Fig 1: MRI head showing bilateral paramedian thalamic infarcts

Fig 2: MRI Angiography showed an intact posterior circulation with the tip of the basilar artery and both posterior cerebral arteries patent, indicating a possible complete occlusion of the artery of Percheron.

Fig 3: MRI head showing bilateral paramedian thalamic infarcts

Fig 4: MRI head showing bilateral paramedian thalamic infarcts

Fig 5: The four variations of the vascularisation of the thalamus and the midbrain described by Percheron, Types being the AOP I (Thalami), IIa (Midbrain), III (Posterior Cerebral Artery) and type IV showing the AOP II. Thalami. D, Medulla. PCA: Posterior Cerebral Artery. M. Midbrain arterioles.