Esotropia and vertical gaze palsy in a patient with an acute thalamic infarct

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Introduction

The thalamus is a grey matter structure situated in the dorsal part of the diencephalon. It has an important role in sensory perception, control of consciousness and alertness, as well as being a station for ascending and descending fibres to and from the cortex. Consequently, thalamic lesions may manifest with a wide variety of presentations.

The thalamus is primarily supplied by the posterior circulation. Approximately 20% of ischaemic strokes involve structures supplied by the vertebrobasilar (posterior) circulation. Many cases are missed or misdiagnosed because of the atypical clinical presentations and the lower sensitivity of neuroimaging for the posterior fossa, and thus patients miss out on reperfusion treatment. Acute onset cranial nerve involvement, particularly in the setting of cardiovascular risk factors, should prompt investigations to rule out potential stroke.

This case highlights an unusual presentation of an isolated thalamic infarct and demonstrates that lesions near the diencephalic–mesencephalic junction may present with complex neuro-ophthalmological syndromes.

Case report

A 62-year-old right-handed woman with a background of hypertension was brought to the accident and emergency department having been noted to have woken up drowsy and leaning to her left side. She had last been seen well 3 hours earlier.

On arrival at the emergency department, she was drowsy but rousable with a Glasgow coma scale of 14 (eyes opened to verbal command – 3, orientated – 5 and obeyed commands – 6). Neurological examination demonstrated vertical gaze palsy, limited left eye abduction with an associated left-sided weakness of the lower face only, with preservation of the muscles of the upper face implying an upper motor neuron facial palsy. Examination also showed a left upper limb weakness (shoulder abduction MRC grade 3, shoulder adduction MRC grade 4, elbow flexion MRC grade 4, elbow extension MRC grade 3, wrist extension MRC grade 3, finger flexors MRC grade 3, finger abduction MRC grade 3). Of note, vertical and horizontal vestibulo-ocular reflexes were preserved, indicating that the gaze palsy was of supranuclear origin. Pupillary responses were present and symmetrical.

Computed tomography of the head, including an angiogram of the intracranial and extracranial vasculature, did not reveal any abnormalities. In view of the clinical features, a cerebrovascular event was suspected and given the degree of neurological deficit (National Institutes of Health Stroke scale score of 6), she was thrombolysed with alteplase 3.5 hours from the time she was last known to be well. Magnetic resonance imaging of the brain showed a right medial thalamic infarct (Figure 1) with no associated midbrain involvement. Holter monitor and a transthoracic echocardiogram were unremarkable. The patient was started on antiplatelet and lipid-lowering therapy for secondary prevention, and she was discharged with no residual neurology.

Discussion

Ophthalmoplegia in the context of ischaemic stroke is typically thought to localise to the brainstem. The role of the thalamus in the control of vertical gaze is a contentious issue in the neurological literature. Historically, it had been felt that patients presenting with vertical gaze palsy as a result of paramedian thalamic infarcts have associated dorsal midbrain involvement (vertical gaze centre) that could not be detected on standard neuroimaging (Babu et al, 2016). However, it has been suggested that isolated medial thalamic pathology could lead to vertical gaze restriction, and this may be attributed to interruption of supranuclear fibres as they pass through the medial thalamus on their way to the control centre in the midbrain, a hypothesis that is reinforced by this case (Guberman and Stuss, 1983; Clark and Albers, 1995).

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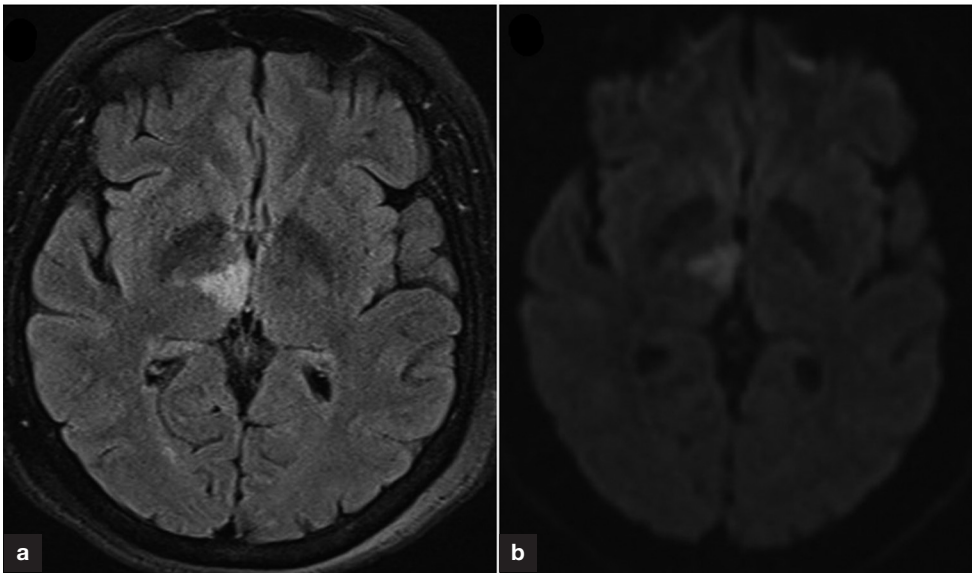

Figure 1. Magnetic resonance imaging of the brain. a. Fluid attenuation inversion recovery sequence shows a medial thalamic hyperintensity. b. Diffusion-weighted image shows a corresponding restricted diffusion consistent with an acute right median thalamic infarct.

Another interesting observation in this case is the phenomenon of contralateral abduction limitation, a very rare phenomenon in this context (Gomez et al, 1988; Ghasemi et al, 2017). This is thought to represent a pseudo-abducens palsy caused by disruption of the descending inhibitory convergence pathways (which pass through the paramedian thalamus before they decussate at the level of the subthalamic region to innervate the contralateral third oculomotor nucleus) and resulting in an esotropia (eye turning inwards) as a result of tonic activation of the medial rectus mimicking an abducens palsy contralateral to the thalamic infarct (Gomez et al, 1988).

The presence of other thalamic signs (eg upgaze palsy and stupor) is a useful clue to thalamic esotropia as these signs would not be expected in a sixth nerve palsy (ie 'fellow travellers' or 'known associates'). Second, the doll's head manoeuvre (vestibulo-ocular reflexes) should not overcome a sixth nerve palsy which is nuclear or infra-nuclear and the eye would be expected to move normally on vestibulo-ocular reflex manoeuvre in the presence of supranuclear thalamic lesions. Third, occasionally in thalamic esotropia, there are brief bursts of convergence–retraction nystagmus which speak to the mechanism of thalamic esotropia (convergence spasm). Last, thalamic esotropia is a transient phenomenon and usually variable, unlike sixth nerve palsy, which is typically static.

Although the thalamus is made up of a series of nuclei, it makes more sense to perceive the thalamus as a structure with four main regions (anterior, posterior, medial and lateral). Each of these regions has its own blood supply, occlusion of which is more likely to produce a potentially recognisable neurological syndrome (Schmahmann, 2003; Saeed et al, 2017). Table 1 highlights the range of clinical presentations when any of the four thalamic regions is affected by a stroke.

Conflicts of interests

The authors declare no conflicts of interests.

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Table 1. Classification of thalamic strokes based on the thalamic region and the arterial territory involved

Arterial territory	Thalamic region involved	Main clinical features
Thalamogeniculate artery	Lateral	■ Contralateral sensory loss ■ Contralateral transient slight hemiparesis ■ Contralateral hemiataxia
Paramedian artery	Medial	■ Impaired arousal ■ Memory impairment ■ Abnormalities of eye movement (vertical gaze palsy and contralateral exaggerated convergence)
Tuberothalamic (polar) artery	Anterior	■ Anterograde memory ■ Apathy and verbal perseveration ■ Language impairment (left-sided lesions) ■ Hemineglect and impaired visuospatial processing (right-sided lesions)
Posterior choroidal artery	Posterior	■ Visual field deficit ■ Transcortical aphasia

Key points

- The thalamus acts as a relay station for fibres of different functions travelling to and from the cerebral cortex.
- The thalamus plays a vital role in the control of supranuclear gaze control.
- Esotropia (excessive convergence) may result from a thalamic stroke, and this phenomenon may mimic sixth nerve palsy.
- The presence of thalamic known associates such as drowsiness and upgaze paresis, normal eye movement on vestibulo-ocular reflexes and the transient nature of the convergence spams point towards thalamic esotropia rather than a sixth nerve palsy.
- Lesions near the diencephalic–mesencephalic junction may present with complex neuro-ophthalmological syndromes.

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