

Pseudoabducens palsy mimicking myasthenia gravis

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DESCRIPTION

A 62-year-old right-handed Caucasian man with hypertension, diabetes and alcoholic fatty liver presented with sudden onset double vision. Physical examination on admission was inward and downward deviation of the left eye, obvious on forward gaze inspection. On extraocular muscle function testing, there was limited left eye abduction and vertical gaze. The right eye failed to abduct fully and the angle of deviation varied with direction of gaze. Otherwise the rest of the examination was normal. National Institutes of Health Stroke Scale (NIHSS) on was 1, thus tissue plasminogen activator was not indicated. He received aspirin 81 mg once. During hospitalisation, he experienced new symptoms including dysarthria and worsening of diplopia, with significant fluctuating course. NIHSS ranged from 0 to 4. Myasthenia gravis was suspected due to fluctuations of his symptoms. He showed improvement of the dysarthria and diplopia with the Tensilon test. MRI of the brain showed restricted diffusion in the left thalamus with corresponding hypointensity in ADC sequence, indicating acute/subacute infarct (figure 1). CT of the thorax was negative for malignancy/thymoma. Myasthenia gravis autoantibodies were negative. The patient was discharged asymptomatic on daily aspirin and statin.

This patient presented with pseudoabducens palsy. It is a deficit of ocular abduction which is not due to dysfunction of the abducens nerve, but caused by increased convergence activity. The neurological pathways for convergence are not discrete nerve tracts. Instead, a network of fibres that

originates from the temporal-parietal-occipital junction and descends through the medial thalamus to the level of the rostral midbrain where they synapse with the medial rectus subnuclei of the oculomotor nucleus. Damage of these fibres is thought to lead to decrease inhibition and therefore increase neuronal activity, causing increased convergence and an esodeviation.^{1 2} Characteristic tonic inward and downward deviation of the eyes known as 'Peering at the tip of the nose' have been found in thalamic infarcts³ and may be caused by damage to these descending convergence fibres at the level of the thalamus.

This case highlights how pseudoabducens palsy may mimic myasthenia gravis. Stroke should be considered first in patients with acute diplopia unless demonstrated otherwise.

Learning points

- ▶ Atypical manifestations of pseudoabducens palsy include intermittent ophthalmoplegia that responded to Tensilon test.
- ▶ Early recognition of the symptoms and physical manifestations of pseudoabducens palsy is important as the process and neurological symptoms can be reversible.
- ▶ Stroke should be considered first in patients with acute diplopia unless demonstrated otherwise.

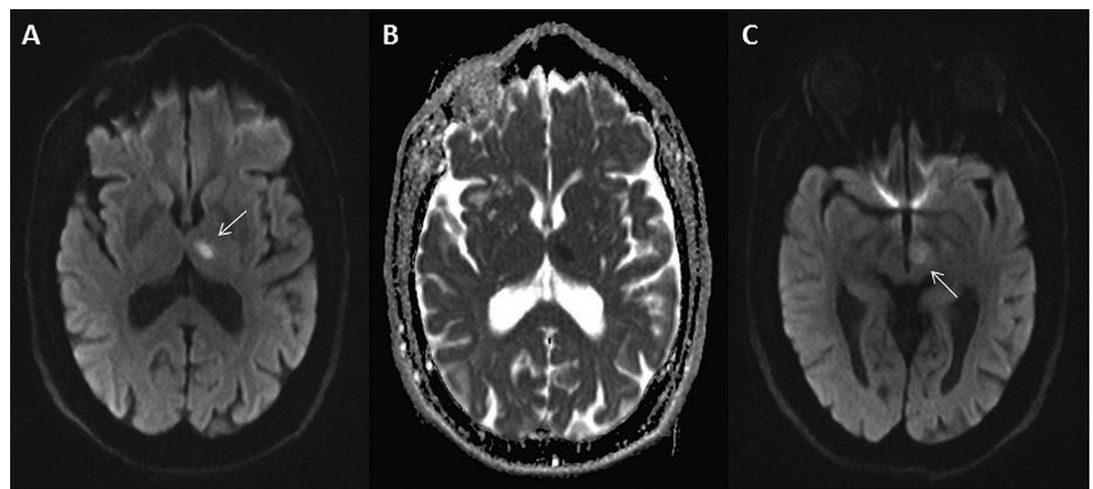


Figure 1 MRI of the brain after 6 h of symptoms onset. (A, arrow) Diffusion-weighted imaging (DWI) axial MRI shows restriction in the left medial thalamus. (B) ADC map axial MRI shows correlation with DWI sequences. There is also DWI restriction (C, arrow) in the left rostral midbrain suggesting an acute stroke. This territory is supplied by the posterior thalamo-subthalamic paramedian artery, a branch off the tip of the basilar artery.



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