CASE REPORT

Eyelid apraxia associated with bilateral paramedian thalamic infarct

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Abstract

We report a case of bilateral thalamic infarct associated with bilateral eyelid apraxia. This extremely rare condition provides us with an opportunity to map clinical and neuroimaging findings to help better understand brain function.

Keywords: eyelid apraxia, top of the basilar syndrome, thalamic infarct, intra-arterial thrombolysis, elderly

Case report

A 63-year-old right-handed woman with a history of paroxysmal atrial fibrillation was brought into the Accident and Emergency department after being found unconscious at home. The time of collapse was thought to be between 6:15 AM (when her partner noted over the phone that she appeared to be confused) and 7:15 AM (when he arrived at her home). On arrival she had a reduced conscious level with Glasgow Coma Scale of 8. She also had a right third nerve palsy, right internuclear ophthalmoplegia, upper motor neurone seventh nerve palsy, bilateral arm weakness (left weaker than right) and mild left-leg weakness. Figure 1 shows an emergency arteriogram (digital subtraction arteriogram, DSA) demonstrating an embolus at the terminal end of the basilar artery with clot extending into the right posterior cerebral artery. Both vertebral arteries and proximal basilar artery appear small and there is a prominent trigeminal artery on the right side which fills the distal basilar artery (Figure 1). Intra-arterial thrombolysis was administered approximately 6 h after initial collapse and recanalisation of the basilar artery was achieved [1]. MRI scan (T2W sequence) showed bilateral paramedian thalamic infarct (Appendix 1, available on the journal website http://www.ageing.oxfordjournals.org/).

The patient’s motor weakness resolved within 24–48 h. However, the patient continued to have poor episodic memory, a non-fluent dysphasia with perseveration of single words and phrases, a vertical gaze palsy, and an inability to voluntarily open her eyes (eyelid apraxia) (Appendix 2, available on the journal website http://www.ageing.oxfordjournals.org/). These impairments complicated the patient’s rehabilitation and led to recurrent falls. Therefore, warfarin could not be commenced despite the cause of the stroke being cardio-embolic in origin.

The patient was unable to open her eyes voluntarily and persistently held her own eyes open with her fingers. When her eyes were opened for her she showed paradoxical contraction of the bilateral orbicularis oculi muscles. There was no evidence of a geste antagoniste, persistent excessive muscle contraction or a trigger to eye closure to suggest blepharospasm. Lundy’s Loops glasses assisted eyelid opening and the patient was more mobile and walked more safely when wearing these. The patient made a gradual recovery and was discharged home after 3 months. The eyelid apraxia continued to improve 6 months after her stroke.

Discussion

Bilateral paramedian infarcts are usually present with reduced level of consciousness and memory problems. They can also lead to thalamic dementia, akinetic mutism and vertical gaze palsy and have a variable prognosis [2]. Some of these abnormalities were noted in our patient. Thalamic infarct has previously been reported to be associated with bilateral ptosis. It is thought to be due to the presence of reciprocal connections between the ventro-lateral nucleus of the thalamus and the precentral cortex of the facial area, and/or diffuse cortical projections to the frontal cortex (Brodmann area 6) from the ventro-anterior thalamus, collectively and vaguely termed as supranuclear pathways. Other possibilities include involvement of the interstitial nucleus of the medial longitudinal fasciculus and the central
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Figure 1. Catheter arteriography (DSA) showing clot (embolus) at the top of the basilar artery and persistent foetal trigeminal artery providing main supply to basilar artery.

caudal subnucleus of the 3rd nucleus [3, 4]. However, in this case, the eyelid features were of apraxia rather than bilateral ptosis as described above. Apraxia in eyelid opening has previously been reported following hemispheric stroke, extrapyramidal disorders and motor neurone disease [5, 6]. However the precise neuroanatomical basis for this disorder is poorly understood and is likely to be a distributed network. Involvement of parts of the frontal cortex and rostral brain stem have been implicated in previous reports [5]. A case of eyelid apraxia during deep brain stimulation to the subthalamic nuclei has recently been reported—a suggested mechanism being excitation of trigeminothalamic fibres with consequent inhibition of the caudate [7]. To our knowledge this is the first reported case of bilateral eyelid apraxia following bilateral paramedian thalamic infarct. This case suggests that paramedian thalamic nuclei may have a role in the pathogenesis of this rare but disabling condition.

Key points

- Eyelid apraxia is a rare condition and has previously been reported in hemispheric stroke, extrapyramidal disorders and motor neurone disease.
- We report a case of eyelid apraxia following a bilateral thalamic infarct following intra-arterial thrombolysis of an embolus at the top of the basilar artery.
- This case suggests that paramedian thalamic nuclei may have a role in the pathogenesis of this rare but disabling condition.

Conflicts of interest

None.

Supplementary data

Supplementary data for this article are available online at http://ageing.oxfordjournals.org.

References


Received 20 May 2007; accepted in revised form 29 October 2007