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Case Report

Bilateral Blepharospasm Due to Unilateral Thalamomesencephalic Lesion

Afrantou T, Natsis KS*, Angelou A and Grigoriadis N

Second Department of Neurology, AHEPA University Hospital, Greece

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***Corresponding author**

Natsis KS, Department of Neurology, AHEPA University Hospital, Thessaloniki, Greece, Tel: +302313303247;
Email: kostna1980@gmail.com

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Abstract

Blepharospasm has been rarely reported as a consequence of structural lesions of the thalamus. We present a patient with bilateral blepharospasm due to a non-vascular thalamomesencephalic lesion of the non-dominant hemisphere. A disruption among internuclear pathways linking the non-dominant cerebral hemisphere, the basal ganglia, thalamus and the brainstem, is a possible pathogenic mechanism.

Introduction

Eyelid motor abnormalities including ptosis, blepharospasm and apraxia of eyelid opening, are known to occur sometimes as a consequence of stroke usually associated with large nondominant hemispheric infarctions. Blepharospasm has been rarely reported as a consequence of structural lesions involving the thalamus, upper brainstem and basal ganglia [1,2]. In these cases the lesions are observed to the non dominant cerebral hemisphere and sometimes are bilateral. The important role of basal ganglia and thalamus in the control of eyelid movements has been recently shown by functional brain imaging studies in patients with essential blepharospasm.

Case Report

A 63 year old, hypertensive, right-handed woman presented to our department with bilateral blepharospasm and blinking that started 3 months ago. She reported photophobia. Although most of the time she was able to open her eyes, a reflex blepharospasm could be produced by stretching the lids. On neurological examination there was no evidence of pyramidal signs or extrapyramidal disorder. At the time of admission she had been already treated with botulinum toxin so the severity of her symptom had become milder. The brain MRI revealed a lesion to the right thalamus which was isointense on TW1, hyperintense on TW2 and FLAIR, without enhancement on TW1 images (Figure 1). The involved, affected areas were centromedian nucleus of thalamus, lateral thalamic nuclei and red nucleus while the lesion seemed to extend to the upper brainstem. Since there were no typical signs of ischaemic infarction we performed a second MRI 3 months later which showed the very same findings; the absence of hypointesity on T1 images was indicative of a space-occupying lesion.

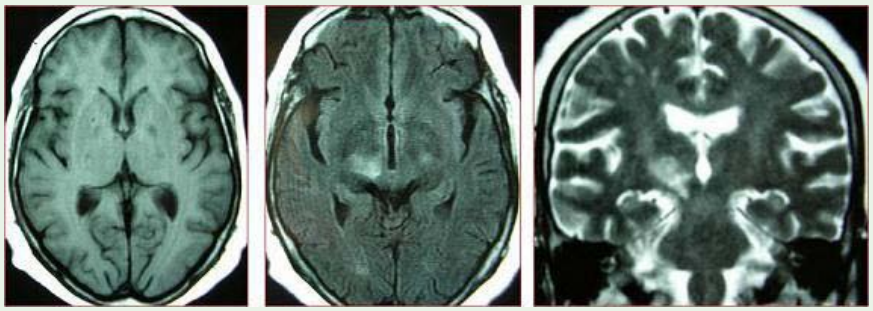

Figure 1: a) T1 MRI showing no obvious thalamic lesion b) High signal lesion in axial FLAIR images on the right diencephalic – upper brainstem area c) Coronal T2 image showing the same high signal thalamic lesion.

Discussion

Symptomatic dystonia can be the result of various metabolic, degenerative diseases, the consumption of certain medications or exposure to toxic agents. Symptomatic dystonias after focal structural lesions provide information for the study of aetiopathogenesis in idiopathic dystonias [3]. Blepharospasm is rarely reported after structural damage and it may reflect a disruption

among internuclear pathways linking the nondominant cerebral hemisphere, the basal ganglia and thalamic nuclei and the brainstem [4]. We observed, in our case, an isolated eyelid movement disorder, without any evidence of hemiparesis, ataxia or hemisensory loss, which was not transient because of the non ischaemic origin of the lesion, emphasizing the role of specific areas of thalamus in the pathophysiology of eyelid motor disorders. To our knowledge this is a very rare case of a patient suffering from blepharospasm due to a non vascular unilateral thalamomesencephalic lesion.

References

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