ΘΑΛΑΜΙΚΑ ΕΜΦΡΑΚΤΑ ΚΑΙ ΟΦΘΑΛΜΟΚΙΝΗΣΗ: ΣΕΙΡΑ ΠΕΡΙΣΤΑΤΙΚΩΝ

Ελένη Καρανταλή[1, 2], Δημήτριος Ντάντος[2], Αγγελική Πρεβεζιάνου[2], Πέτρος Αγγελόπουλος[2], Δημήτριος Κάζης[2]

[1] Νευρολογική κλινική, Γενικό Νοσοκομείο Σερρών, Σέρρες
[2] Γ΄ Πανεπιστημιακή Νευρολογική κλινική, Αριστοτέλειο Πανεπιστήμιο Θεσσαλονίκης, Γενικό Νοσοκομείο Θεσσαλονίκης «Γ. Παπανικολάου»

Περίληψη

Ο θάλαμος αποτελεί τον τελικό σταθμό των αισθητικών οδών πριν την φλοιική τους προβολή. Ένα θαλαμικό έμφρακτο μπορεί να εμφανισθεί με πληθώρα συμπτωμάτων, η οποία εν μέρει μπορεί να δικαιολογηθεί από την πολυπλοκότητα της θαλαμικής αγγείωσης και των νευρωνικών κυκλωμάτων. Παρουσιάζουμε τέσσερις περιπτώσεις θαλαμικών εμφράκτων με κυρίαρχη τη διαταραχή της οφθαλμοκίνησης. Η διενέργεια νευροαπεικόνισης είναι απαραίτητη για τη διάγνωση και εντοπιστική ενός οξέος θαλαμικού εμφράκτου, καθώς συχνά η κλινική εξέταση μπορεί να είναι παραπλανητική.

Λέξεις ευρετηρίου: ΑΕΕ, θάλαμος, διαταραχές οφθαλμοκίνησης, διπλωπία, MRI

THALAMIC INFARCT AND THE EYES: A CASE SERIES

Eleni Karantali[1, 2], Dimitrios Ntantos[2], Angeliki Prevezianou[2], Petros Angelopoulos[2], Dimitrios Kazis[2]

[1] Neurological Department, General Hospital of Serres, Serres
[2] Third Neurological Department, Aristotle University of Thessaloniki, General Hospital of Thessaloniki, «G. Papanikolaou», Thessaloniki

Abstract

The behavioral and psychological symptoms in dementia (BPSD) are a huge problem for patients with dementia and for their caregivers. They affect the rate of cognitive decline of the patient, lead to early institutionalization, increase the cost and become a crucial burden for the caregivers. Unfortunately, the pharmacological treatment so far either is not effective or has many adverse effects. Furthermore, the pharmacological treatment for many BPSD is not specialized such as in apathy. Hence, the non-pharmacological interventions should be a first-line solution. The Neuropsychiatric Inventory (NPI) has been used for many studies of neuropsychiatric symptoms in neurodegenerative disorders for the past 25 years. It has been translated into approximately 40 languages and has been used in approximately 350 clinical trials. There are four groups of non pharmacological interventions: a) Cognitive, b) Sensory, c) Behavioral and d) Other kind of interventions. In this review all cognitive and sensory interventions are presented. In conclusion we need more well designed studies with all kind of interventions.

Key words: stroke; thalamus; oculomotor disturbances; diplopia; MRI

Introduction

Thalamus is the most significant part of the diencephalon and the final relay of somatic and special sensory pathways before their cortical projection. The complexity of neuronal circuits and vascular supply makes thalamic lesions the "Proteus" of clinical localization. Oculomotor disturbances are usually indicative of a brainstem lesion. Rarely does a thalamic lesion underlies. We present four cases with prominent eye movement impairment.

Case reports

Patient One: A 35-year-old patient presented with abrupt onset of double vision, slurred speech, and altered mental status. Neurological examina-

tion revealed vertical gaze palsy, skew deviation with hypotropia of the right eye, mild dysarthria, and imbalance. Personal medical and family history was unremarkable. MRI depicted an acute ischemic lesion of the right thalamus (Figure 1a). After a few days, the symptoms improved. However, after a thorough workup, the stroke was considered cardioembolic due to patent foramen ovale (PFO). PFO was characterized by a large left-to-right shunt and the coexistence of an atrial septal aneurysm. We recommended a PFO closure, but the patient refused further intervention.

Patient Two: A 27-year-old patient presented with acute onset of dizziness and diplopia. Neurological examination revealed skew deviation with a hypotropia of the right eye. Despite being an occasional smoker, patient's personal medical and family history was unremarkable. MRI revealed a right thalamic infarct (Figure 1b). After a thorough workup, the stroke was considered cryptogenic, despite the presence of a small PFO. We referred our patient to an expert Cardiologist for further monitoring.

Patient Three: A 47-year-old patient presented with acute vertigo, diplopia, and transient disturbance of consciousness, which lasted two hours. Neurological examination revealed downbeat nystagmus and mild paresis of right eye abduction. Personal medical and family history was unremarkable. MRI depicted an acute bilateral thalamic infarct, implying Percheron's artery occlusion (Figure 1c). The symptoms resolved during the second day of his hospitalization. Further investigation revealed PFO characterized by a large shunt and septal aneurysm. The patient was referred for PFO closure.

Patient Four: A 30-year-old patient presented with acute onset diplopia and transient disturbance of consciousness starting ten days ago. The symptoms began after surgical removal of peritoneal carcinomatosis due to a reproductive system's malignancy. Personal medical and family history was otherwise unremarkable. Neurological examination revealed mild right eyelid ptosis, horizontal and downbeat nystagmus, complete paralysis of the right eye abduction with esotropia, and vertical gaze palsy. MRI revealed an acute bilateral thalamic infarct, implying Percheron's artery occlusion (Figure 1d). After a thorough workup, the stroke was accredited to the underlying malignancy.

Discussion

Thalamus lies deep in the cerebral hemispheres' white matter, occupying the diencephalon's fourth-fifths. The arterial supply of the thalamus is characterized by high complexity. Four arteries are the major contributors of thalamic blood supply: 1) tuberothalamic artery, 2) paramedian artery, which occasionally originates from one side or a common pedicle (artery of Percheron), 3) thalamogeniculate arteries, and 4) posterior choroidal arteries, arising from both anterior and posterior circulation [1].

The functional complexity of the thalamic nuclei and the variations of the thalamic arterial supply can induce multiple clinical syndromes [1]. Typically, a thalamic infarct presents with a pure sensory deficit or a combination of altered level of consciousness, somatosensory findings, cognitive and behavioral symptoms. Rarely, oculomotor disturbances are the main clinical presentation.

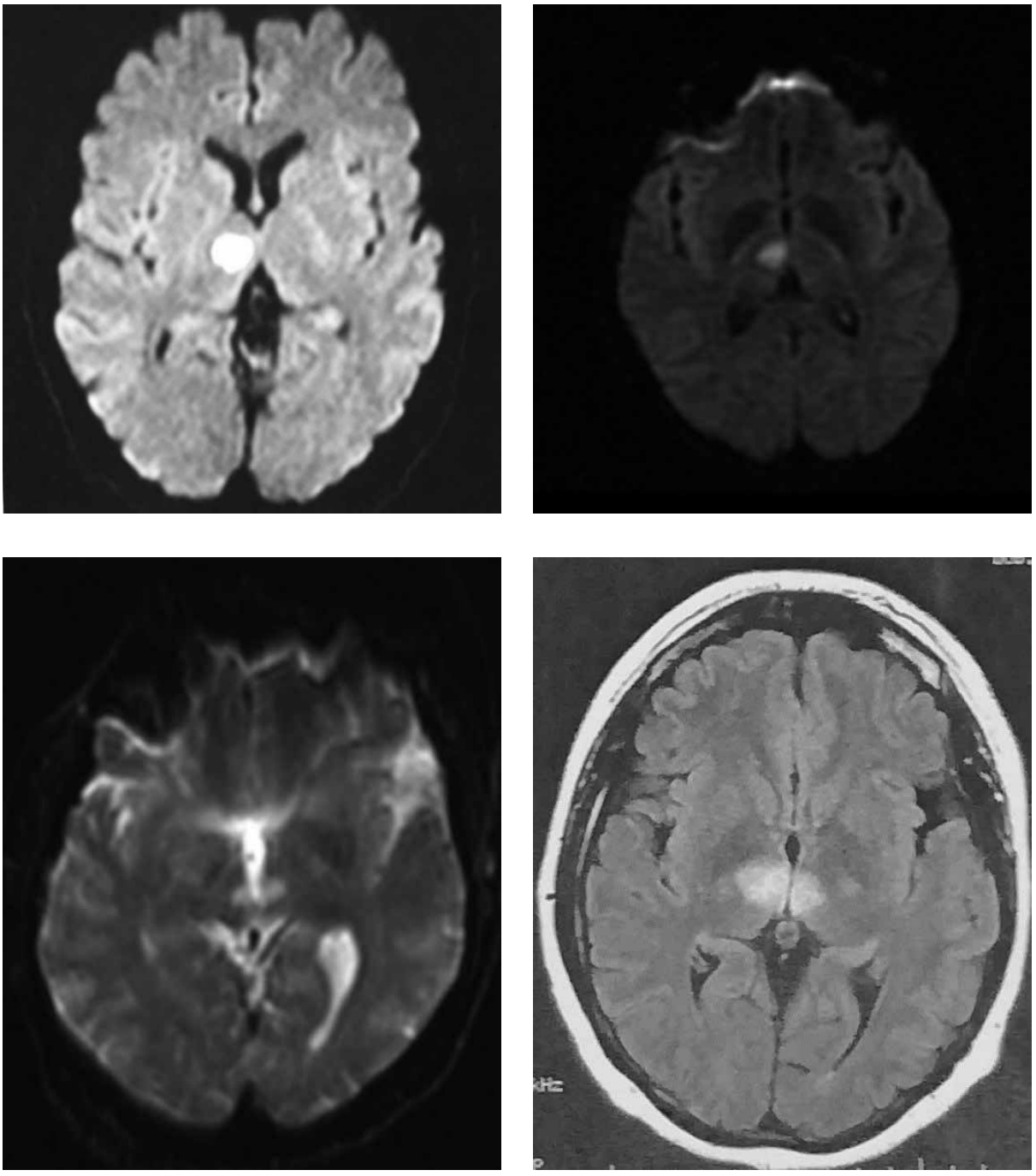
Thalamus plays a role in eye movement control, but the exact pathways are still unclear. A wide variety of oculomotor disturbances have been associated with paramedian artery occlusion [1] and have been described in the literature; skew deviation, vertical gaze palsy, loss of convergence, pseudo-abducens nerve palsy or sustained downward deviation of the eyes [2]. The intralaminar and dorsomedial nuclei of the thalamus connect with the frontal and supplementary eye fields and are supplied by the paramedian artery [1]. Uni- or bilateral inhibition of these pathways has been suggested as the underlying pathophysiologic mechanism. Nonetheless, thalamic lesions sometimes extend to the rostral mesencephalon, thereby involving the interstitial nucleus of Cajal and the rostral interstitial nucleus of the medial longitudinal fasciculus [3]. Brain MRI 1.5 Tesla used on routine parameters (slice thickness 3-5 mm) may underestimate the mesencephalic involvement of thalamic infarcts.

Our cases feature various oculomotor disturbances as the main presentation of a paramedian thalamic infarct (Figure 1, Table 1). Case one presented with vertical gaze palsy, and case two with skew deviation with hypotropia of the right eye. Both symptoms can result from disrupting the cortico-mesencephalic tract

Table 1. Overview of thalamic lesion and eye involvement per case

	Thalamic lesion	Eye involvement
Patient 1	Right	vertical gaze palsy, skew deviation with a hypotropia of the right eye
Patient 2	Right	skew deviation with a hypotropia of the right eye
Patient 3	Bilateral	downbeat nystagmus and mild paresis of right eye abduction
Patient 4	Bilateral	mild right eyelid ptosis, horizontal and downbeat nystagmus, complete paralysis of the right eye abduction with esotropia, and vertical gaze palsy

Figure 1. MRI findings
a. Patient 1, DWI acute ischemic stroke right thalamus (black arrow), **b.** Patient 2, DWI acute ischemic stroke right thalamus (white arrow), **c.** Patient 3, DWI acute bilateral thalamic infarct (black arrow), **d.** Patient 4, FLAIR acute bilateral thalamic infarct (black arrow)



within the thalamus or a rostral interstitial nucleus and interstitial nucleus of Cajal damage [1, 4]. Although vertical nystagmus is a rare finding in patients with unilateral thalamic infarcts, it has been previously described in bilateral thalamic involvement. Vertical nystagmus in cases 3-4 is hypothesized to be caused by a disturbance in both the rostral interstitial nucleus of the medial longitudinal fasciculus and the interstitial nucleus of Cajal [5]. Contralateral abduction palsy or "pseudo-abducens palsy" results from disruption of descending mesencephalic inhibitory convergence pathways [6].

Conclusion

Acute infarcts of the thalamus presenting with disturbed oculomotion can be challenging to diagnose, solely relying on clinical examination. Brain imaging is crucial for the diagnosis and localization of such lesions. Further research on the role of the thalamus in oculomotion is needed.

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