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Case report

Improvement of sleep architecture in the follow up of a patient with bilateral paramedian thalamic stroke

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ABSTRACT

Normal sleep architecture and arousal require an intact thalamus. Thalamic vascular lesions, particularly in the paramedian region may cause arousal disturbances and hypersomnolence. Although hypersomnolence is one of the main characteristics of acute bilateral paramedian thalamic infarcts, there are only scarce reports in literature concerning polysomnographic follow-up of these patients. The few reported cases in literature show that sleep stages do not significantly change from the acute to chronic phase.

We present a case report of a patient with a bilateral paramedian thalamic infarct in which a polysomnographic evaluation of sleep was performed four days and five months after stroke. In the acute phase, polysomnography showed an impairment of phase 2 NREM and absence of phase 3 and 4 NREM with absent sleep spindles. After the acute stroke phase, hypersomnolence improved and sleep spindles reappeared as well as phase 3 and 4 of NREM sleep.

Our patient clear clinical and polysomnographic improvement makes us suppose that in this case the initial impairment could have been essentially due to a functional transitory impairment of the thalamocortical and corticothalamic connections.

This case report is peculiar because it discloses a marked improvement of sleep architecture which to the best of our knowledge has not been clearly described before.

1. Introduction

Normal sleep architecture and arousal require an intact thalamus. Thalamic vascular lesions, particularly in the paramedian region may cause arousal disturbances and hypersomnolence. Although hypersomnolence is a main characteristic of acute bilateral paramedian thalamic infarcts, there are only a few cases of acute bilateral thalamic stroke with neurophysiologic evaluation of sleep described in literature [1–6] and only scarce reports of their neurophysiologic follow-up [1,2]. In the few cases reported in literature, sleep stages did not significantly change from the acute to chronic phase.

2. Case report

A 39-year-old man with a history of hypertension, smoking and moderate alcohol intake was admitted after sudden conscience impairment preceded by an abrupt headache. Neurological examination, on the day of admission, showed impaired consciousness with a Glasgow Coma Scale Score of 7. Later, increased somnolence, palsy of upward gaze and of the left III nerve, right ataxic hemiparesis and sensory loss were noticed. Brain Magnetic Resonance Imaging disclosed a paramedian bilateral thalamic infarct with mesencephalic and left capsular extension (Fig. 1). Cerebral angiography showed a stenosis of the medial and upper third of the basilar artery with occlusion of the left posterior cerebral artery. Coagulation, autoimmunity and serological studies, a 24-h Holter monitoring and a transesophageal echocardiogram were unremarkable. Months afterwards, a transcranial Doppler showed normal velocities in the basilar artery and posterior cerebral arteries. During hospital stay, the patient was hypersomnolent. He spent an average of 18 h/day sleeping. This sleep pattern was different from the patient's usual pattern. He used to sleep from 11 pm to 6 or 7 am. A polysomnography was performed four days after stroke onset and registered data from 10 pm to 8 am (total sleep time of 8 h and 41 min). Sleep stages were classified according to criteria of Rechtschaffen and Kales [7]. Polysomnography showed abnormal sleep architecture with increased stage 1 and reduced stage 2 of sleep. Neither slow-wave sleep nor sleep spindles were registered and a decrease of K complexes was disclosed (Table 1). After hospital discharge, a progressive diminution of somnolence was noticed, with no need for therapy. Five months after stroke onset, sleep

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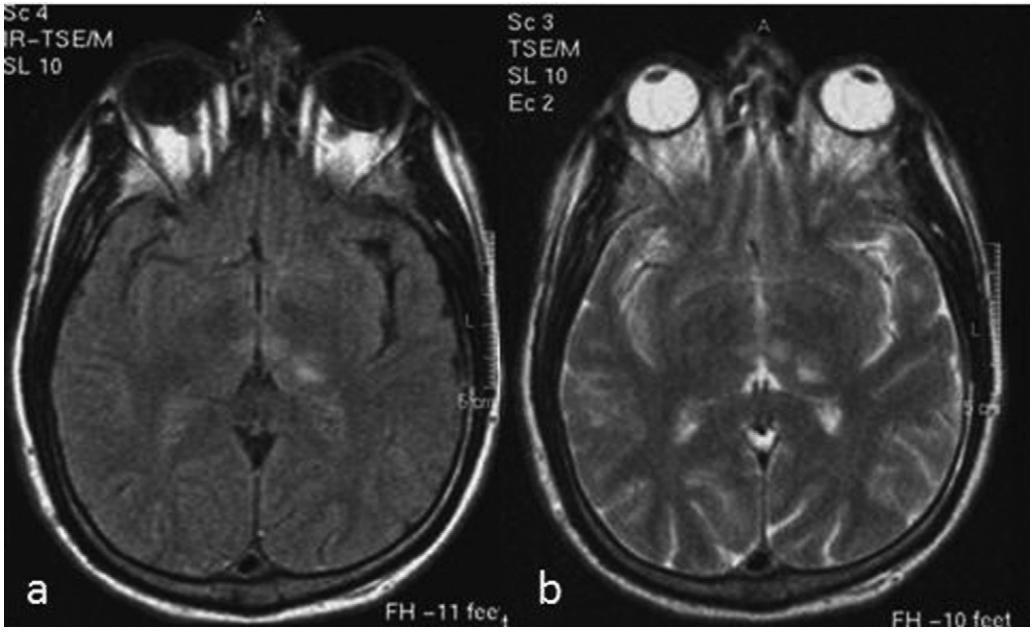

Fig. 1. MRI in T1 (a) and T2 (b) sequence disclosing acute bilateral paramedian thalamic infarct.

diaries showed an average of twelve hours of sleep. This period of sleep was distributed in two patterns. The patient could have uninterrupted sleep from 9 pm to 9 am or a sleep period distributed from 9 pm to 6 am plus 3 h after lunch. A second polysomnography was then performed and registered data from 10 pm to 7 am (total sleep time of 7 h and 20 min). It showed improved sleep architecture with stages 1–4 of NREM sleep and presence of sleep spindles (Table 1).

3. Discussion

The paramedian thalamus is assumed to have a dual role in the maintenance of wakefulness and promotion of NREM sleep [1]. The thalamus is essential in the production of spindles and slow-wave activity. These activities depend of the oscillatory activity of the reticular thalamic nucleus and thalamocortical neurons, respectively [8]. The usual changes associated with thalamic stroke are increased stage 1, decreased stage 2, variable decreases in stages 3 and 4 and unchanged REM sleep. There is also a decrease in the number of sleep spindles [1–4]. All this clinically correlates with hypersomnolence.

What is peculiar to this case report is the marked improvement in sleep architecture which happened in the time frame of 5 months in this patient that initially showed serious changes in the acute stage polysomnography (almost only stage 1 being registered). Hermann and Bassetti which also only performed overnight video polysomnographies [1,2] reported that in the patients in which follow-up was performed, the proportion of sleep stages did not significantly change from the acute to chronic phase, although sleep spindles increased. This makes us suppose that in this case, the initial impairment could have been essentially due to a functional transitory impairment of the thalamocortical and corticothalamic connections.

The reported ischemic lesion, like is mostly the case in patients with bilateral paramedian thalamic stroke, is not "purely bithalamic". There is frequently a subthalamic and mesencephalic extension [1,6]. In this case, there is also mesencephalic and left internal capsule involvement. Bassetti [1] hypothesized that phenotypical variations of paramedian thalamic strokes could be the expression of a variable involvement of other thalamic and subthalamic structures. In his series, patients with unilateral thalamic lesions with mesencephalic extension presented with more severe hypersomnia [1].

There are two main differential diagnosis in patients with bilateral paramedian thalamic stroke–occlusion of the artery of Percheron or a "top of the basilar artery" syndrome. In this case, the asymmetric distribution of the thalamic lesions with the left thalamus more affected, the non-visualization of the artery of Percheron and the normalization of velocities in the posterior circulation make a "top of the basilar artery" syndrome due to an undetected embolic source a unproven possible cause of stroke.

Table 1
Summary report of the first polysomnography performed four days after stroke and of the second polysomnography performed five months after stroke.

	First polysomnography	Second polysomnography
Total sleep time	08:41:30	07:19:30
Stage 1 (%)	80.3	17.1
Stage 2 (%)	1.1	46.2
Stage 3 (%)	0	4.6
Stage 4 (%)	0	17.6
REM	18.6	14.6
Sleep spindles	Absent	Present

4. Conclusion

This case report stands out that the changes seen in sleep architecture after a bilateral paramedian thalamic infarct although severe can be reversible.

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