

Total Recovery after Bilateral Paramedian Thalamic Infarct

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Key Words

Paramedian thalamic infarct · Neuropsychological recovery · MRI · Cerebral blood flow

Abstract

Bilateral paramedian thalamic infarcts are characterised initially by the association of acute vigilance disorders and vertical gaze palsy, followed by persisting dementia with severe mnemonic disturbance, global aspontaneity and apathy. We describe a patient with a dramatic neuropsychological recovery, confirmed by testing examination and completed by a cerebral metabolism study. The pathophysiology of this type of cognitive deficit is discussed.

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Introduction

When the paramedian thalamopeduncular arteries arise from a common trunk [1], arterial occlusion may result in the rare bilateral paramedian thalamomesencephalic infarcts (BPTI), which give rise to vigilance disorders, cognitive deficit and vertical gaze palsy [2–8]. The onset is acute, with deep stupor or coma, evolving into a

lethargic state with hypersomnia. Follow-up of these patients is characterised by a persistent apathy, motor and verbal aspontaneity, anterograde and retrograde amnesia [2, 3, 8]. Vertical voluntary saccades, smooth pursuit and vestibulo-ocular movements are often impaired. We present a case of BPTI characterised by a particularly favourable outcome.

Case Report

A 53-year-old right-handed tradesman treated with coumadin for a severe atherosclerotic disease with lower limb arteriopathy suddenly lost consciousness. On admission to the emergency department, he was only reacting to painful stimuli. He emerged 2 days later in a somnolent state that gradually improved, but he tended to sleep if not stimulated. He did not speak spontaneously, but was able to answer simple questions. A total vertical gaze palsy was observed. Neurological examination was otherwise negative. Brain MRI demonstrated a bilateral paramedian thalamic and mesencephalic infarction, extending from the ventral part of the dorsal nucleus to the rostral medial midbrain (fig. 1). The left lesion appeared to be somewhat larger than the right one.

For the next 3 months, he still remained drowsy and tended to sleep up to 18 h per day. Attention was poor and ideation was slow. There was a reduction of verbal and motor spontaneity, but no perseverations. He was disoriented to time and place, and had a marked memory loss for recent and past events with no confabulations. He could not report basic biographical data nor recall 3 words after 5 min. His language comprehension and expression were normal.

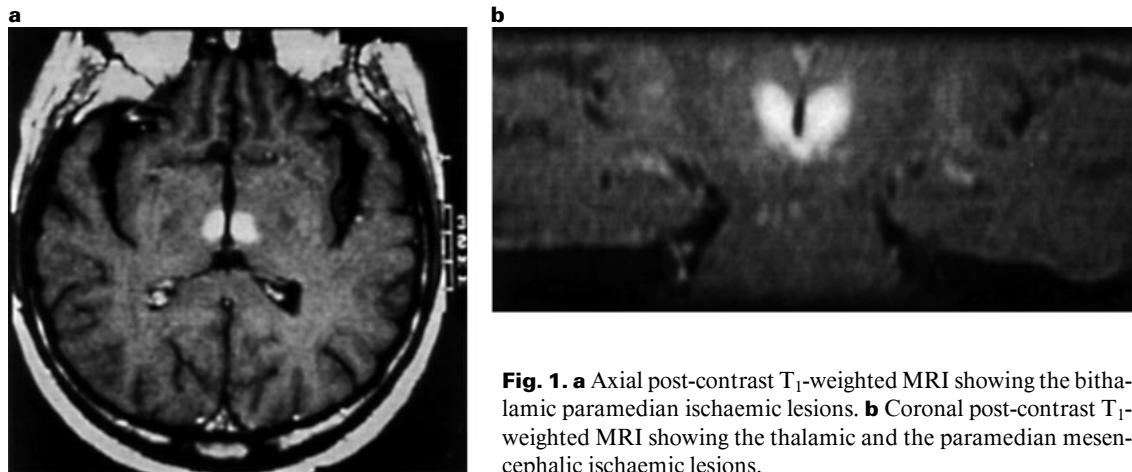


Fig. 1. **a** Axial post-contrast T₁-weighted MRI showing the bithalamic paramedian ischaemic lesions. **b** Coronal post-contrast T₁-weighted MRI showing the thalamic and the paramedian mesencephalic ischaemic lesions.

Oculocephalic eye movements improved in upward then in downward gaze, but pursuit and saccadic movements were absent. One month after onset, the Wechsler Adult Intelligence Scale [9] yielded a verbal IQ of 84 and a performance IQ of 73. The global IQ score was 77. The Signoret Memory Scale [10] showed an equal impairment on verbal and visual memory. The verbal subscore was 13/72 (mean controls 55.7, SD 5.9). The visual subscore was 19/72 (mean controls 53.2, SD 6.7).

On follow-up examination 1 year after onset, there was a dramatic improvement in his behaviour and cognitive functions. His level of consciousness was normal. The patient was oriented to time and space, well-informed on current events, and his domestical abilities were similar to his premorbid ones. There was no marked change in his personality. He only complained of blurring downward vision. His evaluated sleeping time was about 12–14 h per day, which was more than the premorbid need (around 6–8 h per day). Spontaneous speech and comprehension were normal. Clearly, he had no dementia according to the criteria of the DSM-IV [11]. Verbal and performance IQ were both 100, and the score of the Signoret Memory Scale was 110.5 out of 144 (verbal subscore 50, visual subscore 60.5). The Grober-Buschke Memory Scale [12] was normal. The most meaningful performance in this test, i.e. the delayed free recall score (at first trial) was 14 (mean controls 12.2, SD 2.7). The total delayed recall (free and cued) was 16 (mean controls 15.7, SD 0.8). The Wisconsin Card Sorting Test (WCST) [13] was normal: the patient completed 6 categories and made no perseverative errors. Forward and backward digit spans were normal.

Single photon emission tomography (SPECT)-hexamethyl propylene amine 99 metastable technetium (HMPAO 99m-Tc) studies were performed 6 and 12 months after the stroke. The first examination showed a relative hypoperfusion in the frontal lobes (parietal/frontal ratio = 1.14). Right parietal lobe perfusion was somewhat higher than the left one. The relative frontal hypoperfusion was confirmed by a 133 Xenon regional cerebral blood flow (CBF) measurement (left frontal lobe CBF value 37 ml/min/100 g; left hemisphere CBF 42 ml/min/100 g; mean controls 44.7 ml/min/100 g, SD 4.5). On the second SPECT examination, no significant change was observed in the frontal lobes compared to the first examination, but increased perfusion was seen in the parietal lobes, especially in the right one (+11.3%, fig. 2).

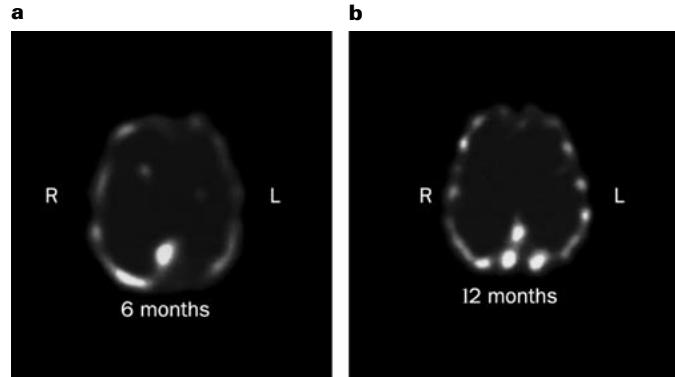


Fig. 2. SPECT-HMPAO 99m-Tc performed 6 months after the stroke showing a relative hypoperfusion in the frontal lobes (**a**) and 12 months after the stroke showing increased perfusion in the parietal lobes compared to the first examination (**b**).

Discussion

The outcome of BPTI is bad, mostly characterised by persistent hypersomnia, akinetic mutism and global impairment of intellectual performances. Patients are apathetic, presenting poor insight and slowness of thought [3, 4]. Perseverations and confabulations have been reported [3, 6, 14, 15]. Persistent impaired IQ and a low memory quotient are always observed. Our case appears to be exceptional because of the clinical cognitive recovery confirmed by the psychometric testing. One year after the stroke, memory and language examinations did not reveal any abnormality; neither did tests evaluating mental flexibility like the WCST. The only clinical abnormalities were represented by a relative hypersomnia of 10–12 h per day and the ocular-motor deficit.

The pathophysiology of memory disorders after thalamomesencephalic lesions has often been debated. Firstly, the initial attentional deficit due to the interruption of ascending noradrenergic pathways in the mesencephalic reticular formation and intralaminar nuclei do not allow good consolidating and retrieving processes that lead to formation and activation of memory traces [4, 6, 16]. Secondly, the mammillothalamic tracts, that connect mammillary bodies with the anterior nuclei of the thalamus, can be interrupted bilaterally [2, 4, 6, 16]. Lastly, lesions of the inferior part of the internal medullary lamina or the dorsomedian nucleus itself must interrupt the amygdalothalamic projections. Akinetic mutism and lethargic state are quite similar to those seen in lesions of the internal frontal lobe and the anterior cingulate cortex. This aspect of frontal lobe syndrome has been related to lesions of the intralaminar and median nuclei, and their connections with the prefrontal, orbitofrontal and anterior cingulate cortex [12, 14, 15]. The involvement of these connections in the origin of the 'frontal symptomatology' may be confirmed by the study of functional neuroimaging.

A pre-eminent bifrontal reduction of regional CBF is not usually observed in BPTI [7, 14, 17]. There is rather a

diffuse hypoperfusion of the cortex, which is matched to a similar hypometabolism on positron emission tomography, as the perfusion-metabolism coupling is preserved [17, 18]. In our case, the dramatic neuropsychological improvement is not correlated to frontal CBF evolution. The relationship between the clinical outcome and the increase in parietal CBF is not clear. Firstly, we can suppose that some connections between the thalamic nuclei respected by the infarction and the parietal cortex may have been restored after a period of oedema and neurotransmission impairment. Secondly, the smaller right infarct on MRI and the better cortical perfusion with greater improvement in the right parietal lobe suggest that some connections may have been preserved on the right side. Lastly, the role of the dorsomedian nucleus in thalamic amnesia, which is the principal structure involved in this case, has been debated [4, 16]. Von Cramon et al. [16] emphasised that its role was minor, they reported 1 case of pure BPTI without amnesia, whereas large parts of the dorsomedian nucleus were involved. Our case presenting a lesion of both dorsomedian nuclei supports this opinion.

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