

Dysexecutive syndrome following anterior thalamic ischemia in the dominant hemisphere

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Abstract

In the article, we describe a rare case: a 52-year-old male patient with an isolated lesion of the anterior thalamic nuclei in the dominant hemisphere due to an ischemic stroke. The patient's syndrome of disordered verbal fluency, working memory, learning and executive functions is seen as a part of a lesion in the dorsolateral complex circuit. During the following 6 months, we observed significant improvement in visual learning, recent memory and categorisation of subjects, while verbal learning and visuospatial planning remained insufficient. In this context, the preservation of thalamic structures in different types of cognitive deterioration is discussed.

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1. Introduction

Organisation structures of the frontal lobe are divided into the five complex circuits. For the cognitive and behavioural functions, three anterior systems are important—the dorsolateral, lateroorbitofrontal and the anterior cingular [2] (Table 1).

Dysfunction of the dorsolateral circuit leads to insufficient spatial and construction planning, solving problems, disorder of verbal fluency, learning strategy disabilities and impairment of memory recall; however, recognition remains intact in most cases. Lateroorbitofrontal circuit impairment is responsible for personality changes, infantile or asocial behaviour, for decreased self-control and emotional control, affective changes, for grasp automatisms of the mouth and hand and for echopraxia and echolalia.

Disorder of the mediofrontal circuit affects (the system of) attention, causes disinhibition of instinctive behav-

iour—in more serious cases, leading to apathy, distress, loss of bladder control and akinetic mutism. The anterior thalamus is a central relay station designed to integrate different impulses from various parts of frontal circuits. However, isolated impairment of these systems is rarely diagnosed. A review of 37 case reports of isolated ischemic stroke in dominant thalamus (1981–1999) found only 26 anterior thalamic lesions, only four of them in the dominant hemisphere [5].

2. Case report

We treated a 52-year-old man, a right-hander and cigar-smoker. At the age of 45, he started having brief spells of dizziness, without nausea or loss of consciousness. Blood pressure was low then. At 46, he suffered a collapse; from then on, he developed sporadic short mild attacks of disordered speech but did not let that worry him. With regard to a history of strong snoring of several years before the accident, he was examined by MEZAM IV for the sleep apnoea syndrome (SAS), but it was not confirmed.

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Table 1

Circuit	Microanatomy	Behavioural changes
Dorsolateral–prefrontal	BA 9(10)–DL cap. ncl. Caudati–DM pallidum–ncl. Ventralis anterior a ncl. Dorsomedialis thalami–BA 9(10)	Working memory disorder: strategy of learning and retrieval, with normal recognition planning and constructive disorder conceptualisation and categorisation, disorder of verbal fluency
Orbitofrontal	BA 10–VM caput ncl. Caudati–D pallidum–ncl. ventralis anterior a ncl. Dorsomedialis thalami–BA 10	Personal changes (infantilise, asocial behaviour, lost of self-control and mood control, obsession, onomatolalia, fumbling)
Mediofrontal	BA 24–striatum ventrale (septum verum, ncl. Accumbens, tuberculum olfactum . . .)–anterior pallidum-paramedial ncl. Dorsomedialis thalami–BA24	Akinetic mutismus, urine incontinency, apathy, depression, disorders of attention, emotional changes, deliberation of primitive instincts (hypersexualism, bulimia . . .)

BA—Brodmann area, DL—dorsolateral, DM—dorsomedial, VM—ventromedial, D—dorsal, ncl—nucleus.

At the age of 52, he fell ill with cold. He became somnolent and edgy but went to work. One morning, he suddenly lapsed into a global memory disorder, with worsened pronunciation and tongue movement disorder although without any comprehension problems. For these symptoms, he was admitted to the stroke unit.

3. Examination

On admission, a mild prefrontal syndrome was found, paresis of the soft palate on the right with a corresponding displacement of the uvula and swallowing and speech impairment. There was a mild central paresis of the right upper extremity with pyramidal and cerebellar signs. No signs of apraxia were observed. Motor symptoms improved to normal status within 1 week.

3.1. Selected laboratory examination

MRI of the brain revealed a 1.5×1.9 cm lesion in the dominant anterior thalamus, of low signal on the T1WI, hyperintensive on T2WI. Gd-DTPA administration brought no enhancement. This finding was significant for diagnose of the ischemic stroke in the anterior thalamic nuclei: the ncl. mediodorsalis (NMD), ncl. ventralis anterior (NVA) and internal medullary lamina (main ncl. anterior) (Fig. 1).

Ultrasonography of the extracranial vessels demonstrated hypoplasia of the vertebral artery on the left.

Echocardiographic examination detected foramen ovale patens and mild mitral regurgitation.

Three weeks after admission, all-night poly-MEZAM monitoring showed mild SAS, but, 5 months later, control examination failed to confirm this disorder.

4. Neuropsychological assessment

Mini-Mental state examination was normal (29/30).

4.1. Speech

Comprehension and speech repetition were undisturbed. There were no paraphasias, no problems with prosody, but spontaneous speech lacked fluency. At first, the patient's speech was brief, monosyllabic, but correct. In spite of improvement, the Verbal Fluency Test was never normal during the following 5 months (score 23; normal control M 41.5, S.D. 12.3).

4.2. Attention

The activity rate (tapping), attention and short-term memory (simple reaction time, continuous performance

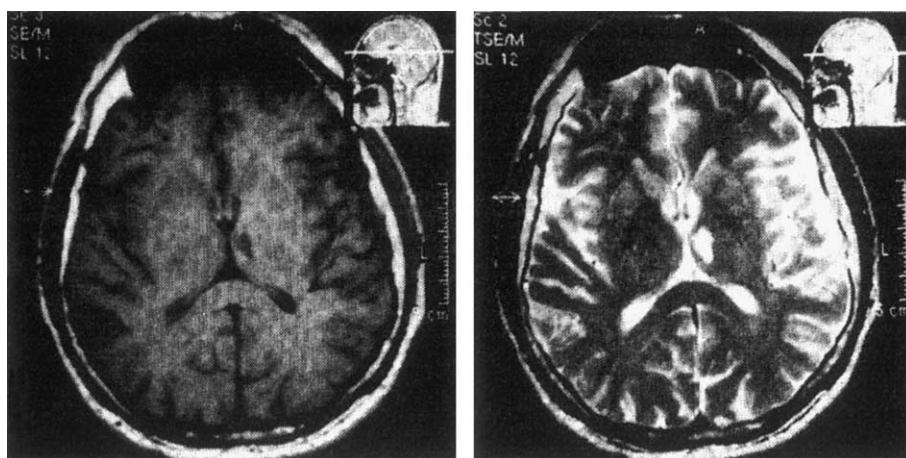


Fig. 1. T1 axial slice, TSE mode. T2 axial slice, TSE mode.

test, high-speed memory scanning, digit span) were all above average.

4.3. Learning and memory

In this field—in imprinting especially, there was evidence of strong deterioration, particularly pronounced in tests of verbal [California verbal learning test (CVLT)] as well as visual quality (Rey visual design learning test). In the former, we found problems with recognition, in the

latter, the main feature was recall impairment. Learning in the pair-association test was significantly impaired as well.

A significant although mild improvement of verbal recall (two to three points in every CVLT set) appeared after 6 months' training. However, long-term recall showed no change. In the visual modality, the tested parameters improved to normal in all cases, including long-term recall (Table 2).

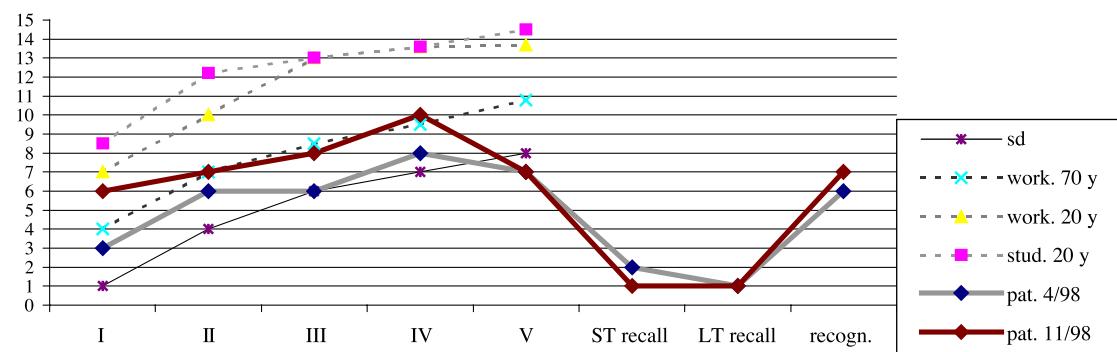
No agnosia, alexia, apraxia, agraphia, acalculia or neglect were present.

Table 2

Patient's performance trends in the verbal and visual learning tests related to age and education matched control

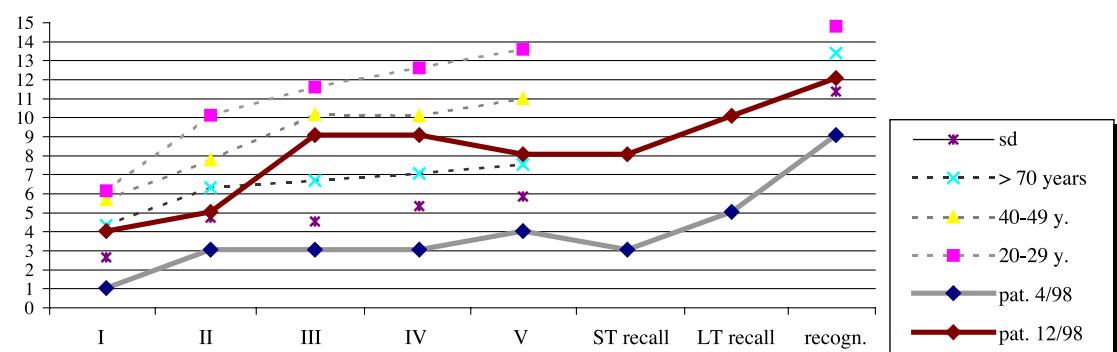
CALIFORNIA VERBAL LEARNING TEST

Trial of presentation (15 words)	I.	II.	III.	IV.	V.	ST recall	LT recall	recognition
				reproduction				
Control: workers 70 years (sd)	4(3)	7(3)	8.5(1.5)	9.5(1.5)	10.8(2.8)			
Control: workers 20 years	7	10	13	13.6	13.7			
Control: students 20 years	8.5	12.2	13	13.6	14.5			
pat. 2 months after stroke	3	6	6	8	7	2	1	6
pat. 9 months after stroke	6	7	8	10	7	1	1	7



REY VISUAL DESIGN LEARNING TEST

Trial of presentation (15 words)	I.	II.	III.	IV.	V.	ST recall	LT recall	recognition
				reproduction				
Control > 70 year (sd)	4.29 (1.7)	6.26 (1.5)	6.64 (2.1)	7 (1.7)	7.46 (1.7)			13.3(2)
Control 40–49 year	5.64	7.71	10.07	10	10.93			
Control 20–29 year	6.09	10.04	11.52	12.52	13.48			14.7
Pat. 2 months after stroke	1	3	3	3	4	3	5	9
Pat. 9 months after stroke	4	5	9	9	8	8	10	12



ST—short time, LT—long time, pat.—patient, work.—workers, stud.—students.

4.4. Executive functions

There was a significant disturbance of “frontal” functions—conceptualisation and mental operations. The patient was not able to explain a simple sentence or detect relation between terms like “corn–flour–bread” (he explained that problem by the word “food”). He was not able to distinguish between the meanings of “brother’s father” and “father’s brother.” He was aware of that deficit, noting that his “thinking did not start well.” This disturbance inclined to spontaneous improvement so that, at a follow-up visit 5 weeks later, there were no signs of disordered understanding. However, even today, half a year later, the patient still has problems to understand and memorize a solution of more complicated tasks and create an effective strategy for solving unknown tasks. In the Wisconsin’s Card Sorting Test, he achieved only five classes of sorting out of six required and was unable to reproduce the differences between particular sorting dimensions. Perseverations were not observed. On the other hand, he found the “Tower of Hanoi” task in its simplest version insurmountable even 6 months later.

4.5. Mood

The patient never suffered from depression or apathy at any time. His mood was appropriate. He fully controlled his instinctive and emotional behaviour and social status. But his performance and position in the family were still impaired. He was very motivated and practised systematically. For the “To rehabilitate his cognitive abilities,” the patient used a personal computer and special training programs. His family’s and his own assessment of the training was very positive.

5. Discussion

Although the main affected nuclei NMD and NVA thalami are both connected in the dorsolateral and orbitofrontal complex circuits, we observed especially isolated symptoms of the dorsolateral circuit (in our case) without signs of attention or personality deficiency. The main signs of damage (as noted above) are learning strategy and retrieval disabilities, while recognition remains intact. However, in our case, we found also disordered verbal recognition. On the other hand, visual recognition, which is considered a function of the nondominant hemisphere, was unaffected. In addition to impaired learning strategy, the simple defect of imprinting (characterized by rapid decrease of verbal information from recent memory) was coresponsible for the learning

disability. There were also signs of executive dysfunction. Hence, our case conforms to the definition of “prefrontal memory problems” rather than to “mediotemporal-like amnesia” according to Van der Werf et al. [5]. Such type of dominant thalamic lesion is rare, and only few cases have been documented well enough. The authors assume that candidate structures for such deficit comprise the midline nuclei, the intralaminar nuclei and mediodorsal nucleus, while the mammilo-thalamic tract remains intact. However, in our opinion, the role of the NVA in this review may be underestimated.

After 6 months, during computer-assisted training, the score in the immediate verbal memory sets improved two to three points, while imprinting remained affected all the time without change. In the category of visual memory, the performance became generally normal. These findings reflect plasticity of the sustained memory functions, which can lead to partial compensation of the cognitive defects. In particular, the ability to categorize executive function terms improved soon albeit short of full normalization. Spatial planning remained impaired permanently. Although intralaminar nuclei play a significant role in vigilance, attention and cooperation in the frontal subsystems [4], we found no attention disorder. Nevertheless, some partial transitory affection of the ascendent reticular activation system may well have been present as a cause of transient SAS. Positive air pressure was found to have no influence on cognitive functions.

The patient confirms the benefit of cognitive rehabilitation on computer. In fact, computer-assisted training with respect to isolated mental dysfunction improvement remains questionable. Admittedly, computer-assisted training of mental functions may be an effective complement to cognitive rehabilitation in some cases at least [3,1]. The authors accept that, in the field of the visual memory, and immediate verbal memory at least, this method could accelerate restitution of functions and thus improve the quality of life.

References

- [1] Friedl-Francesconi H, Binder H. Training in cognitive functions in neurologic rehabilitation of craniocerebral trauma. *J Exp Psychol* 1996;43:1–21.
- [2] Cummings JL. Frontal–subcortical circuits and human behavior. *Arch Neurol* 1993;50:873–80.
- [3] Chen SH, Thomas JD, Glueckauf RL, Bracy OL. The effectiveness of computer-assisted cognitive rehabilitation for persons with traumatic brain injury. *Brain Inj* 1997;11(3):197–209.
- [4] Groenewegen HJ, Berendse W. The specificity of the nonspecific midline and intralaminar thalamic nuclei. *TINS* 1994;17(2):52–7.
- [5] Van der Werf YD, Witter MP, Uylings HBM, Jolles J. Neuropsychology of infarctions in thalamus: a review. *Neuropsychologia* 2000;38:613–27.