

Amnesic syndrome with a unilateral thalamic lesion: a case report

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Summary. A 40-year-old man suffered an attack of transient global amnesia. The anterograde amnesia receded after 2 days, but a deficit remained in the retrieval stage of memory, which impaired the recall of names and of the information necessary to give details of particular events. Computed tomography revealed ischaemic infarction in the left anterolateral thalamus.

Key words: Transient global amnesia – Specific memory deficit – Thalamic aphasia – Thalamic infarction

Zusammenfassung. Bei einem 40jährigen Mann trat eine amnestische Episode auf. Die anterograde Amnesie dauerte nur 2 Tage, aber es blieb eine spezifische Gedächtnisstörung, die das Wiederfinden von verbalem Material, besonders von Namen, betraf, darüber hinaus aber auch den Zugang zu jenen spezifischen Informationen und Details behinderte, die der einzelnen Erinnerung ihren individuellen Charakter geben. Das CT zeigte einen ischämischen Infarkt im linken anterolateralen Thalamus.

Language disturbances have quite frequently been reported as a sequel to unilateral left-sided thalamic ischaemia [4, 6, 7, 10] and amnesic syndromes are well known in association with bilateral thalamic infarction [18, 22]. We observed a patient with an amnesic syndrome who had unilateral infarction of the left thalamus.

Case report

Clinical course

A 40-year-old judge, who had been treated regularly, with cortisone for years because of asthma, attended a professional conference away from home. Having drunk one litre of wine the evening before, he awoke one morning with a heavy head and feeling drowsy. For the remaining 2 days of

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the conference he was only able to understand the most simple juridical matters. His behaviour was nevertheless unremarkable, except that he fell asleep several times during the lectures.

After returning home, he told his wife that he felt tired and soon went to bed. The next day he asked repeatedly where he had been during the last few days and thought he could recall that he had been in Tunisia, where the family had indeed spent several holidays years before. During the following weekend he talked very little, but kept to his daily routine and even looked through some documents to prepare for a session. He remembered day-to-day events, but had much difficulty in recalling names. He had forgotten the names of all his neighbours and even of his son. He also had difficulty finding words for juridical and other specific matters.

He was worried about his memory deficit, repeatedly asked for the names he had forgotten and brooded over what had happened at the conference and whether he had acted normally there. When he was brought to the hospital 5 days after the start of the illness, he thought that he had already been there and that he had had the medical examinations the day before.

On admission he was seen to be an adipose man with a reddish face and striae distensae on the abdomen. Neurological examination revealed slight weakness of the right corner of the mouth, as well as slight diminution of fine movements, diadochokinesis and stereognosis of the right hand. Tendon reflexes were slightly brisker on the right. There were no disturbances of vision or ocular movements. Laboratory findings including immunological examinations of the serum were normal with the exception of a slight polycythaemia of $5.7 \cdot 10^{12}/l$ red blood cells and a haematocrit of 54%.

The patient was alert and oriented to time, place and situation. His mental and emotional reactions were slow and lacked vitality and his facial expression was one of somewhat helpless strained searching. In conversations the train of his thoughts was appropriate and logical, but stereotyped. He understood spoken and written language but could not understand long and complicated sentences. Speech and writing were correct too and the patient knew the names of everyday objects, but he had severe difficulty with the recall of proper names and other specific terms. The degree of difficulty in finding words fluctuated; sometimes he spontaneously recalled a word which before he had sought in vain, but minutes later he forgot it again. Prompting was usually helpful and when given several possibilities to choose from, he recognized the right word without hesitation.

The memory deficit was not restricted to word finding. The patient had only a fragmentary recall of what had happened at the conference and his memory for events before this period was defective too. On the subject of his long-standing asthma, he could only recall the name of one drug, but could neither give a history of his illness nor describe any symptoms. Asked about his professional activity (he was concerned with civil jurisdiction) he stated that he had to handle "divorces, building concerns and other more complicated matters", which he could neither name nor describe. His description of persons whose names he had forgotten was fragmentary and superficial.

The memory deficit was essentially the same for recent and remote events, though clearing up a little for things that had happened long ago. He could name none of his present office colleagues, but several of his school teachers. However, when asked about his final school examinations, he could recall no details except their date.

The patient stayed in hospital for 2 weeks. During this time the general slowness and dullness receded and there was a slight improvement in the memory deficit. He was then seen at monthly controls. After 4 months he resumed his professional activity, but delayed many tasks because he still felt it difficult to understand complex matters and to conduct a juridical hearing. The memory deficit was now more clearly confined to verbal material, impairing on the one hand the recall of names of even old acquaintances, and on the other the ability to remember long sentences.

At examination after 7 months, the patient still complained of difficulties in remembering names and in quickly understanding complex verbal information. His reasoning and behaviour at the examination now appeared normal, except that he failed to recall some medical terms relating to his asthma.

Neuropsychological examination

Neuropsychologic examinations were performed 10 days after admission to the hospital and 4 and 7 months after demission. Table 1 shows the methods applied and the results.

Table 1. Results of neuropsychological examinations

Object of examination	Method	Result		
		1	2	3
<i>Psychophysiological measurement</i>				
Concentration and vigilance	Critical flicker fusion	40.7	40.7	40.8
Attention over a longer period	Motor reaction to repeated visual stimuli	119/3	121/2	121/2
Psychomotoric velocity and capacity of simultaneous reaction	Right-left oriented motor reaction to complex visual and acoustic stimuli	15/160	15/173	15/180
<i>Psychometric testing</i>				
Verbal memory	Memorizing of 10-20 items after a 5-min period of distraction [1]	1/12	3/12	8/12
Memory for associated verbal items		0/20	6/20	12/20
Memory for unassociated verbal items		0/20	2/20	5/20
Numeric memory		3/10	4/10	9/10
Visual memory		8/10	9/10	10/10
Formal intelligence	Progressive matrices [16]	122	123	122
Aphasia	Modified token test [14]	0	0	0
<i>Self-regard</i>				
Depression	Personality inventory [8]	0	0	0
<i>Projective test</i>				
Psycho-organic syndrome	Rorschach inkblot method [15]	1	0	0
				I.Q.
				Points on aphasia scale
				Points on depression scale
				Grade of psycho-organic syndrome

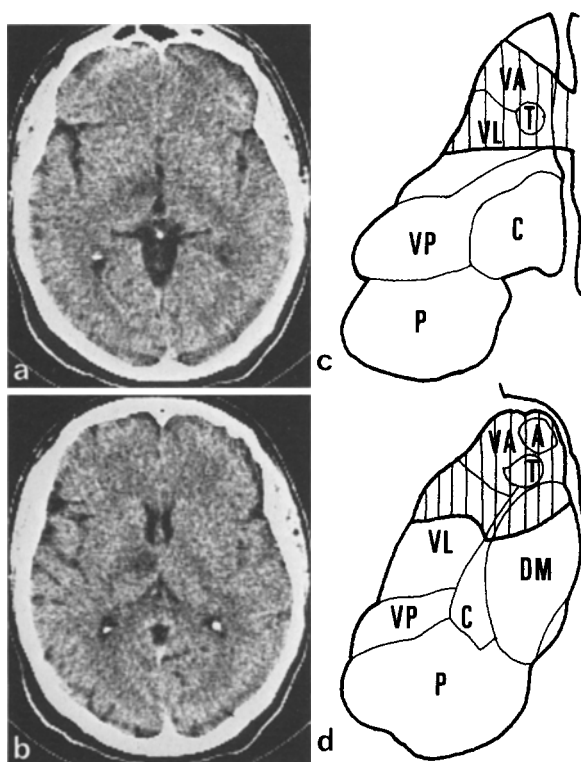


Fig. 1a-d. CT 5 days after ictus. **a, b** The lesion is visible in two consecutive 8-mm cuts. **c, d** Diagram of the lesion; *A* = anterior nucleus, *C* = centre median and intralaminar nuclei, *DM* = dorsomedial nucleus, *P* = pulvinar, *T* = mamillothalamic tract, *VA* = ventroanterior nucleus, *VL* = ventrolateral nucleus, *VP* = ventroposterior nucleus

Concentration, vigilance and formal intelligence were normal in all examinations; the patient was not markedly depressed and only the first examination gave evidence of a slight psycho-organic syndrome. In the absence of any aphasic disturbance, the memory tasks revealed a marked reduction in verbal memory capacity. Furthermore, there was a moderate reduction of psychomotor speed and of the capacity to react to multiple simultaneous stimuli. This condition did not improve.

Over the course of the follow-up, his score on the verbal memory tasks improved. The change was statistically significant for associative verbal memory ($\chi^2 = 12$; $df = 2$; $P < 0.01$) as well as for general verbal memory ($\chi^2 = 6.5$; $df = 2$; $P < 0.05$). However, the latter capacity remained on a subnormal level and there was only insignificant improvement in the tasks for unassociated verbal items. Numerical memory was less markedly impaired and returned to normal, but the improvement was not statistically significant.

Neuroradiological examinations

CT examination on the day of admission to the hospital revealed a hypodense area in the left anterior thalamus (Fig. 1). Four-vessel angiography, performed 4 days later, showed an occlusion of the left central middle cerebral artery branch and hyperaemia in the anterior thalamus (Fig. 2).

The follow-up CT 2 months later showed clearer demarcation and diminution of the thalamic lesion, but additional atrophy in the left central cortex (Fig. 3), the latter probably being a sequel of the middle cerebral artery branch occlusion.

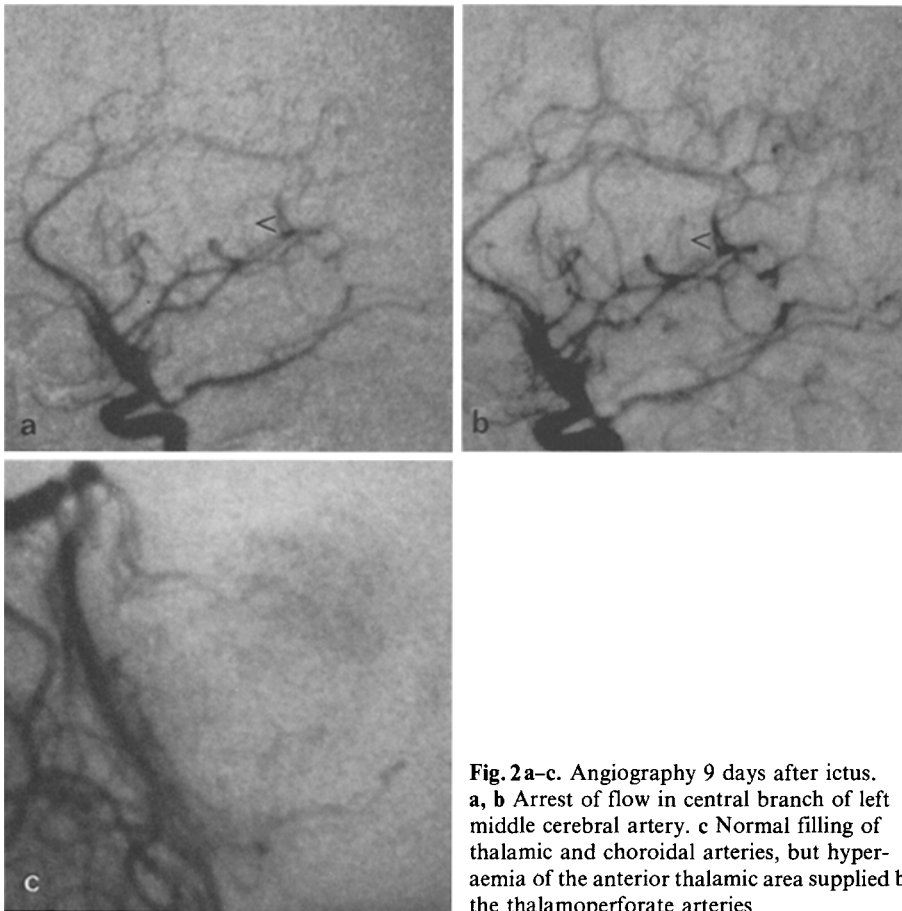


Fig. 2a-c. Angiography 9 days after ictus.
a, b Arrest of flow in central branch of left middle cerebral artery. **c** Normal filling of thalamic and choroidal arteries, but hyperaemia of the anterior thalamic area supplied by the thalamoperforate arteries

Discussion

Difficulty in word finding, sometimes in association with semantic paraphasia, is the dominant feature of the language disorder observed with ischaemic lesions of the left thalamus [4, 6, 7, 10]. Site and extension of the ischaemia in the cases reported have been evaluated by CT examination and as far as can be judged there appears to be no unequivocal relationship between the occurrence and quality of the language deficit and the lesion's exact localisation within the thalamus. After stereotactic surgery to the thalamus, language disturbances have been observed only with lesions of the left ventrolateral thalamus and pulvinar [13, 17]. Clinically, they manifest as difficulties in word finding; psychological testing reveals additional diminution of the verbal IQ and of verbal Short-term memory. If there is a common pathogenetic mechanism underlying the disorders of naming, verbal short-term memory and verbal knowledge, it should be a defect in the retrieval rather than in the storage of verbal information. This interpretation is supported by the results of stereotactic stimulation in either the storage or the retrieval stage of verbal recall tasks [12].

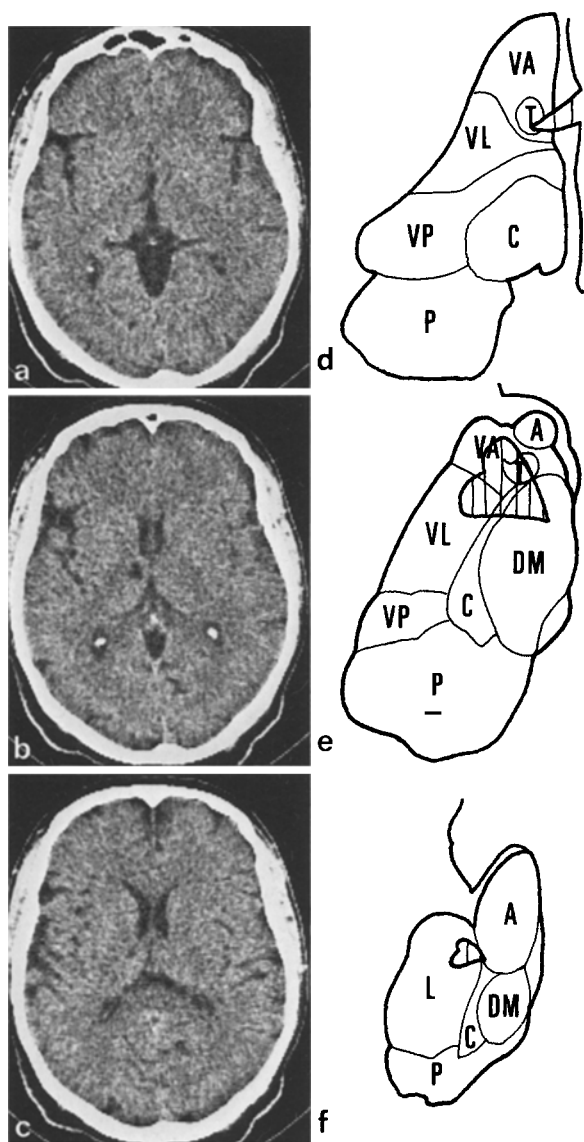


Fig. 3a-f. CT 2 months after ictus; 2-mm cuts. The lesion was visible in seven consecutive cuts. a-c show the first, fourth and seventh of them, d-f the corresponding diagrams. *L* = lateral nucleus, for other abbreviations see Fig. 1

The association of bilateral ischaemic damage to dorsomedial or intralaminar thalamic nuclei with a severe amnesic syndrome has repeatedly been reported [3, 5, 18, 22]. In most of the cases, the lesion also affected the anterior nucleus or the mamillothalamic tract, thus interrupting the "exit" of Papez's circuit. The amnesic syndrome of thalamic origin resembles that with hippocampal lesions. Its dominant feature is the inability to store new information, leading to gradual forgetting and to more or less complete anterograde amnesia. There may be

additional retrograde amnesia, reaching back for years before the illness and sparing only remote memories, thus being associated with a backward disorientation in time. Though in some cases more widespread cerebral damage could be ruled out, the amnesic syndrome is invariably associated with a disturbance of behaviour similar to the effect of frontal lobe damage.

Transient memory deficits in combination with a disorientation in time and errors in estimating the duration of time were reported after unilateral surgery to the dorsomedial thalamic nucleus [19]. There is also a well-documented case [20, 21] of a permanent amnesic syndrome after unilateral stab-wound damage to the left anterior thalamus. In this case, CT scan showed the centre of the lesion in the dorsomedial nucleus, but it extended into the ventrolateral nucleus too. The amnesia was purely anterograde and impaired verbal memory markedly more than non-verbal memory.

Our patient's initial global anterograde amnesia as well as the confabulations that were obviously due to a disorientation in time and the general slowness and reduction of vigilance could be due to the damage to the dorsomedial and anterior nuclei and to the mamillothalamic tract. The transience of these symptoms could be explained by the unilaterality of the lesion, according to the observations with amnesia of hippocampal origin, which confirm that the symptoms caused by unilateral lesions of limbic structures can be largely compensated and only bilateral lesions cause permanent memory disorders [2, 9, 11]. The residual deficit of word finding as well as the difficulties in understanding and memorizing complex verbal information could both be due to the ventrolateral thalamic damage, if one accepts a diminution of verbal short-term memory as the cause for the latter symptom. This assumption, however, does not correspond to the results of the psychological examination, which also revealed a slowing down in reactions to complex non-verbal stimuli.

Furthermore, the opposition between global anterograde amnesia and verbal retrieval defect offers no explanation for the very peculiar features which the patient's memory deficit displayed during his hospital stay. It was neither restricted to words or confined to recent memory nor could it be explained by a disorientation in time. The memory deficit could be better described as an inability to recall information that was singular and highly specific and was not necessarily needed to hold together the patient's whole network of knowledge and personal memory, but was required to give details and individuality to the single recall.

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