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Unique amnestic syndrome after isolated left anterolateral thalamic stroke: a case report

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Abstract

Background The thalamus plays a key role in motor and sensory processes of the brain. Though thalamic stroke is among the less frequent, even a minimal lesion can result in a serious impairment and long-lasting disability. After thalamic stroke, cases of dysexecutive syndrome and so-called diencephalic amnesia have been observed, yet the precise characterisation or categorisation of such amnestic syndrome is not available. Pure amnesia can be the one and only indication of a thalamic lesion. We present a unique case of a patient after left anterolateral thalamic stroke suffering from isolated severe amnesia and disorientation. We conducted extensive neuropsychological testing of the patient's memory and discovered contrasting results in different aspects of memory.

Case presentation A 75-year-old woman was admitted for neurological rehabilitation two weeks after being diagnosed with acute left anterolateral thalamic ischemic stroke. The initial symptoms with which she presented in the emergency room were acute confusion, disorientation and memory impairment; apart from that, the patient did not have any other neurologic signs. As observed on the MRI, the lesion was restricted mostly to the left anterior nucleus without affecting the mammillothalamic tract. The neuropsychological testing revealed anterograde episodic memory loss with preserved visual recognition and auditive short term memory; inability to freely recall semantic information, spatial and time disorientation, apathy and significantly reduced intrinsic and phasic attention, and immediate and delayed prose recall deficiency.

Conclusion The knowledge that memory impairment and disorientation can be the only symptoms of the stroke is a crucial piece of information, which is needed to be able to make a decision about proper treatment. Abstention from immediate intervention often leads to irreparable memory deficit for the rest of the patient's life. The results of neuropsychological testing show the essential role of AN in creating episodic memory, in the working memory network and indicate its role as a critical interface between short-term and long-term memory. A theory explaining such a profound impairment of working and anterograde episodic memory has not yet been formulated.

Keywords Unilateral thalamic infarction, Diencephalic amnesia, Amnestic syndrome, Apathy, Working memory, Episodic memory

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Background

Due to the complex anatomy of the thalamus, thalamic strokes can manifest with a broad range of symptoms from motor and sensory deficits, aphasia, neglect or ataxia to memory impairment, executive dysfunctions, and pain. Patients often present with a range of symptoms corresponding to the interruption of reciprocal cerebral cortical-thalamic connections [1]. In rare cases with small lesions, they can suffer from isolated memory loss or executive function impairment. The mediodorsal thalamic nucleus (MD), the anterior thalamic nucleus (AN) and the mammillothalamic tract (MTT) are three key structures discussed the most in the context of diencephalic amnesia [2, 3]. The fact that the MTT, together with other nuclei, are often damaged simultaneously can lead to further symptoms like anomia, word finding difficulties, perseveration or ideomotor apraxia [4, 5].

We present a unique case of a patient with pure thalamic amnesia accompanied by partial impairment of executive functions without any other neurological deficits after an acute unilateral, left anterolateral thalamic ischemic stroke sparing the MTT.

To the best of our knowledge, there have been only three similar cases with an isolated symptom of diencephalic amnesia after a unilateral AN lesion published so far. One patient presented with a severe deficit of long-term verbal and visuo-spatial memory after a right polar and anterior thalamic infarct with partial damage of the MTT [6]. The second case describes a patient with amnesia after a unilateral left anterior thalamic infarct including the left AN, MTT and internal medullary lamina [7]. The third case described a patient with selective verbal memory disturbance with anterograde and partial retrograde amnesia after a left AN infarction [8]. Other published reports described patients who, besides amnesia, also suffered from aphasia, apraxia, mild hemiparesis or hypesthesia in the acute phase [4, 5, 9].

Case presentation

In October 2023, a 75-year-old, right-handed woman from the German-speaking part of Switzerland was admitted to our inpatient Centre for Neurological Rehabilitation two weeks after being diagnosed with an acute left anterolateral thalamic ischemic stroke.

The patient was initially brought to A&E by her daughter because of acute confusion, disorientation and memory impairment. The cerebral CT, lumbar puncture and blood tests were without pathological findings; hence, no diagnosis was made and the patient was kept in for monitoring only. The next day, an MRI revealed an acute ischemic stroke in part of the left anterolateral thalamus. The NIHSS score at this moment was 2/24: the patient was not orientated and did not know her age. The aetiology of the stroke remained so far unclear despite the extensive examination (unremarkable long-term ECG and echocardiography).

Apart from long-term mild arterial hypertension and dyslipidemia, the patient had no other cardiovascular risk factors. It is crucial to stress that before stroke, despite the white matter T2 hyperintense lesions corresponding with Fazekas 3 in the MRI, the patient was completely independent in basic and complex activities of daily life; cooking, driving, and banking posed no problems for the patient. No memory problems had been observed by the family prior to the stroke. (The information about the pre-stroke life of our patient was obtained from her daughter, who has extensive professional experience as a medical doctor and who was in daily contact with her mother.)

On admission to neurological rehabilitation, a detailed neurological examination was carried out in the presence of the patient's daughter and husband, which revealed severe anterograde memory impairment and minimal retrograde memory deficits for recent events (previous months) combined with anosognosia. The patient did not know the purpose of her hospitalisation and that she had had a stroke two weeks before. She did not have any significant impairment of her retrograde memory, as she was only not certain where she celebrated her last birthday but could describe very well all her previous working and free-time activities.

She recognised her daughter and husband but could not say where she lived and what her home looked like exactly. She was not oriented to time, place or situation. The patient showed flattened affect, lack of motivation for any activity and partial anosognosia, expressing a wish to be discharged. The absence of anterograde long-term memory and blunted emotional reactions were among the other most prominent symptoms observed by the family. No other neurological or neurocognitive deficits such as aphasia or apraxia or hemineglect were present.

The routine blood tests did not show malnutrition, vitamin deficiency or any other abnormalities; specifically, TSH and Vitamin B1 were in normal range.

During the hospitalisation, the patient experienced a short moment of nausea with vomiting immediately after breakfast. A prompt clinical examination did not reveal any abnormalities. After twenty minutes, the patient felt well again, yet did not remember that she had experienced nausea and had had to vomit.

In the third week of the intensive interdisciplinary neurological rehabilitation, the patient got lost in the clinic. After being found, she stated again she did not understand the purpose of her hospitalisation and wished to be discharged. The patient's request was granted, and she was taken home by her family.

One month after the clinical manifestation of the stroke, a follow-up MRI was conducted (Figs. 1 and 2), showing a gliosis zone in the sense of late subacute to chronic nodular infarctions. According to the neuroradiologist, the lesion in the left anterior thalamus measured up to 6 mm, in the left lateral thalamus the lesion was up to 4 mm with hyperintense signal in the ADC (apparent diffusion coefficient) map with no evidence of involvement of the interthalamic adhesion. The MRI showed further normal representation of the *corpora mammillaria* without the mammillothalamic tract being affected in its course and normal hippocampal formation on both sides; advanced confluent supratentorial periventricular to subcortical medullary gliosis of microangiopathic aspect (Fazekas 3); finally, age-appropriate width of the cerebrospinal fluid spaces.

Neuropsychological assessment: in the initial screening (MOCA), the patient with 13 years of education reached 20/30 points. She displayed severe difficulties in working memory, delayed recall and, partially, in orientation (city).

Quantitative testing (Table 1) confirmed the deficits in verbal memory. Specifically, the working memory, overall learning ability, and delayed recall of learned words were well below the average. Notably, the patient confabulated additional words during both recalls. However, testing verbal short-term memory using 5 numbers (WAIS-IV) yielded an average score.

Similarly, context dependent memory testing (WMS-IV) revealed severe impairment in immediate and delayed recall of learned short stories (14 and 25 words long). We used the German translation of the fourth edition of the Wechsler Memory Scale from 2009 for patients between 65 and 90 years. In the first context dependent memory task, the patient is presented with three simple sentences. The sentences are read out loud for 10–15 s and mention 14 items. The patient should immediately recall as many items as possible. Our patient recalled only three items; after being presented with the sentences for the second time, she immediately recalled seven items. The second task regarding prose recall considers 25 items being mentioned in three sentences, being read aloud for 30–45 s. Our patient could not recall a single item. Additionally, she confabulated new stories not remotely related to the original.

Visual memory was evaluated through remembering previously drawn figures (CERAD). The patient had no problems with copying the figures, yet the recall of these figures was moderately impaired.

Performance in attention was determined through standardised computerised reaction to stimuli. The patient showed significantly slower-than-average

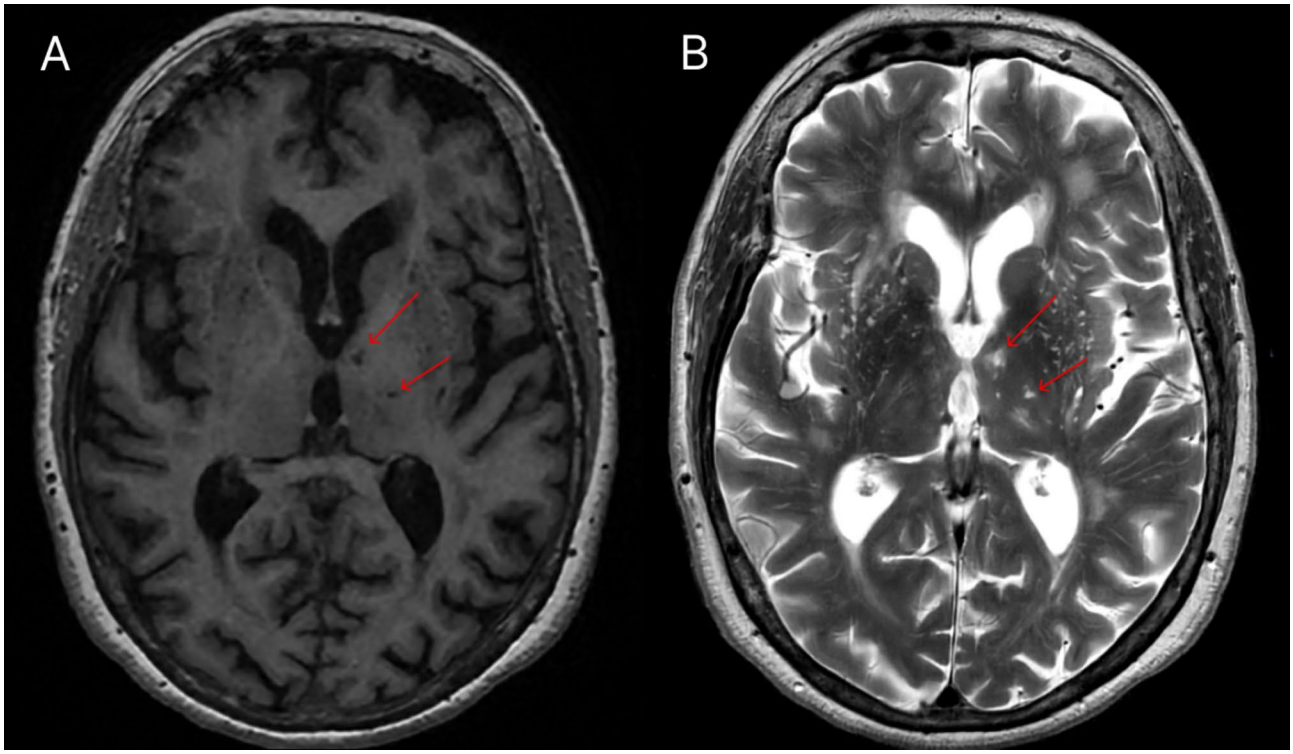

Fig. 1 The patient's MRI conducted in October 2023, one month after the stroke. The T1 weighted imaging (**A**) and T2 weighted imaging (**B**) show the lesion in the left anterior thalamus measured up to 6 mm and in the left lateral thalamus measured up to 4 mm, with no evidence of involvement of the interthalamic adhesion. Normal representation of the corpora mammillaria without the mammillothalamic tract being affected in its course and normal hippocampal formation on both sides (not shown)

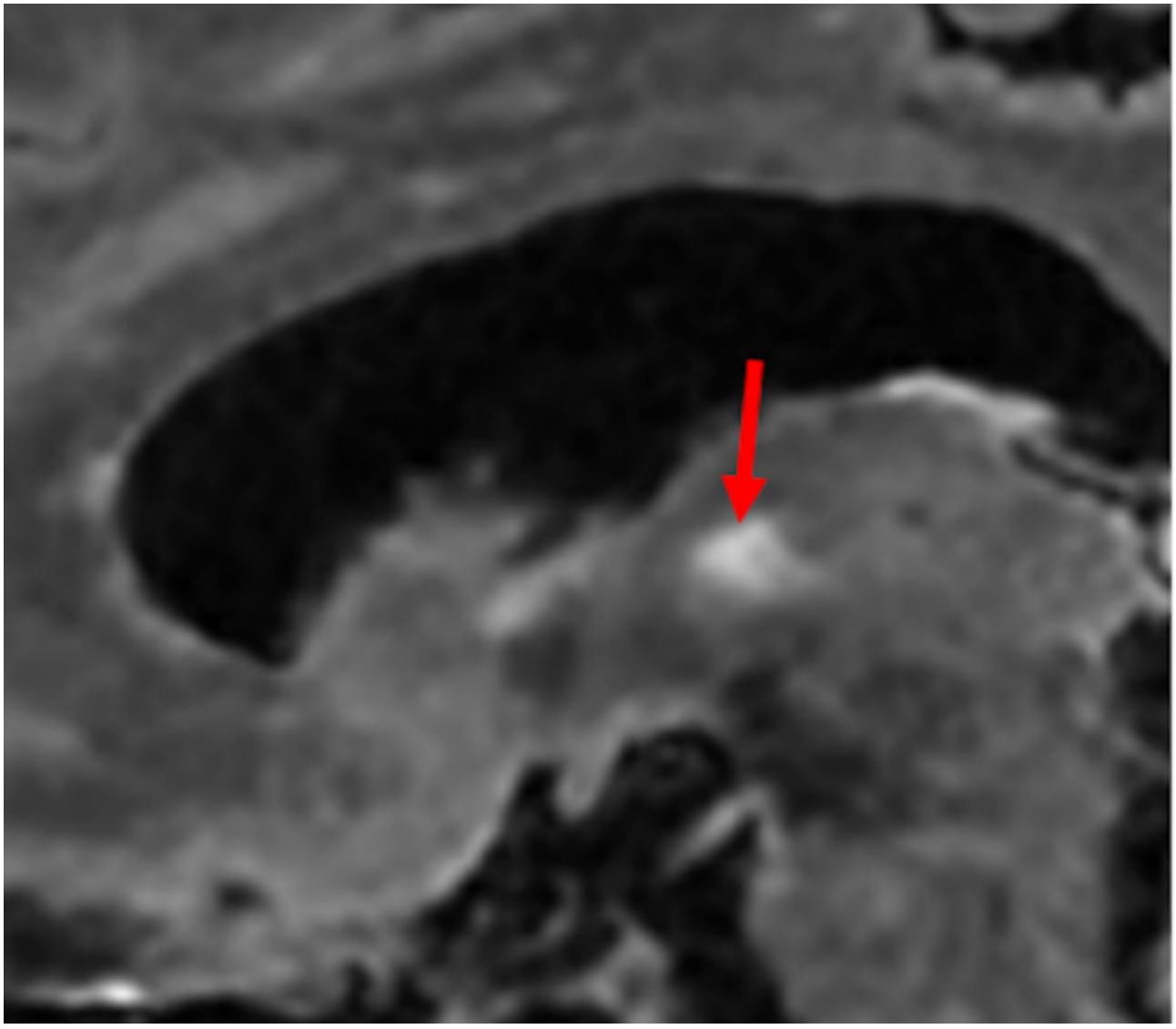

Fig. 2 The patient's MRI conducted in October 2023, one month after the stroke. Detail of sagittal section shows the lesion in the left anterior thalamus

reaction times to simple visual stimuli. The basal alertness (TAP) showed moderate deficits in intrinsic activation and severe deficits in phasic activation.

Naming, writing, and reading were normal. Semantic word fluency showed a severely below-average result. In contrast, the result of the phonetic word fluency test was within the average range. During the semantic word fluency task, the patient confabulated other words and proceeded to recall tasks and behaviours associated with that word, thereby not adhering to the rules of the task (i.e. recalling only animals).

Regarding working memory, we found contrasting results: while recalling numbers backwards (WAIS-IV), the patient presented a severe deficit; however, the result in recalling number sequences was within the average range.

Furthermore, during all neuropsychological tests, especially tests of executive functions, we observed a significantly reduced verbal working memory, in some cases significantly-impaired verbal cognitive fluency, flattening of affect, perseveration tendencies, as well as difficulties in inhibition and impulse control.

Finally, the Frontal Behavioral Inventory (FBI) was filled out together with the daughter. At thirty-four points, the FBI indicates frontal lobe dysfunction.

We contacted the patient and the family three and six months after the stroke. Although the patient refused to undergo neuropsychological testing, we managed to obtain extensive information about her daily life. The family observed almost full recovery of the retrograde memory, contrasted with no improvement of the anterograde episodic memory impairment. The patient lost her

Table 1 Neuropsychological Profile of the patient

Domain		Extremely low (< 2%)	Very low (2–5%)	Low average (6–15%)	Average (16–84%)	High average (> 84%)
Attention	intrinisc		x			
	phasic	x				
Language					x	
Verbal memory	immediate recall		x			
	delayed recall		x			
	recognition	x				
Context dependent memory	immediate recall		x			
	delayed recall		x			
	recognition	x				
Verbal working memory	backward	x				
	sequential				x	
Visual memory				x		
Visual perception						x
Word fluency	semantic	x				
	phonematic				x	
Screenings	MMSE		25/30			
	MoCa			20/30		
	FBI			34		

Test Batteries. TAP: Test of Attentional Performance; CERAD: Consortium to Establish a Registry for Alzheimer's Disease; WMS-IV: The Wechsler Memory Scale; WAIS-IV: The Wechsler Adult Intelligence Scale (4th Edition); MMSE: Mini-Mental-State Examination; MoCa: Montreal Cognitive Assessment; FBI: Frontal-Behaviour Inventory

independence and was not able to carry out basic daily activities, such as driving a car or cooking, which used to be her typical daily activities before the stroke. After the stroke, the patient was unable to collect the necessary ingredients for cooking or find an alternative to unavailable ingredients and was unable to add them in the correct order. The patient still needed prompting to initiate and continue some routine activities because of reduced motivation and lack of spontaneous initiative. Only a slight improvement of mood and apathy was observed after the citalopram therapy was initiated.

Discussion and conclusions

Pure thalamic infarctions are relatively rare and their prevalence among ischemic strokes varies in different studies from 1.1 to 4.4% [10–13]. Due to complex reciprocal thalamo-cortical connections, thalamic infarcts can mimic practically any cortical lesions [1]. Memory difficulties and various cognitive deficits, often combined with other symptoms, appear mostly after lesions affecting AN, MTT, MD and intralaminar nuclei [2, 3]. Such symptoms rarely appear solitarily; in one of the largest studies published so far, only 10% of all pure thalamic strokes (6 patients out of 2450 with any cerebral ischemic stroke in the registry) suffered from ischemia in polar artery territory supplying anterior nuclear group [13]. Small artery diseases with the most frequent risk factors, like hypertension, hypercholesterolemia and diabetes mellitus, were the main identified causes of thalamic infarction [10, 13].

The initial symptoms of an anterior thalamic stroke (disorientation, confusion and memory loss) may lead to a vast range of differential diagnosis such as transient global amnesia, epileptic amnesia, acute encephalitis, subdural haematoma, intoxication, B1 deficiency, abnormal electrolyte concentrations or hypoglycaemia. A strategic ischemic stroke is however often not taken into consideration, probably based on the absence of other neurological symptoms. Compared with the anterior thalamus and anterior hippocampus, strokes in other locations (e.g. occipital lobe, other parts of thalamus etc.) are associated with a less severe amnesia, lasting a significantly shorter time; besides that, they are always accompanied by more common signs of a stroke, such as hemianopsia, hemiparesis, sensory loss or apraxia [10, 12, 14]. Acute memory loss without any other classic neurological symptoms of a stroke hence means a complicated diagnosis and significant time loss if MRI is not conducted. The abstention from possible acute treatment may have serious consequences for a patient's morbidity. The resulting severe amnestic syndrome after AN infarction, compared to other neurological deficits after thalamic stroke, seems to be the most long-term persistent impairment, often without regression [14]. This was also confirmed in 2022 in the first case-control prospective study with 37 patients. Although memory impairments accompanied by other neurological symptoms sometimes occur after paramedian or inferolateral thalamic strokes, the patient with an anterior thalamic stroke

showed severe verbal deficits that almost did not recover during the 24-month follow-up [15].

The fact that lesions in the thalamus can cause severe memory deficits has been known for decades and has led to the term *diencephalic amnesia* [16]. This term has been used very freely for any memory impairment after thalamic lesions without defined symptomatology.

Our patient showed severe impairment of verbal memory in a very peculiar way; she could immediately and correctly repeat five words and five numbers (immediate serial recall), but when she was asked to reproduce them immediately in the reversed order, she was unable to do so. In a direct contradiction with that, however, difficulties in performing simple calculations were not observed. The patient performed correctly the test with a series of seven subtractions starting at 100.

Other tests also indicated a problem in processing new verbal information, for which mental manipulation and further conversion is needed. The patient repeated correctly a maximum of five numbers in a sequence but was not able to repeat a three-number sequence in reverse order, sometimes struggling to reproduce even two numbers backwards. When the patient was given ten words to be read and remembered, she could immediately recall only two of them. When the wordlist was presented for the second and third time, she recalled four words. Ten minutes later, the patient was able to freely recall only one word. On the other hand, when she was shown a list with the original ten words and ten new words added, she easily recognized eight of the original words and was able to identify the other ten as new. It corresponded also to our daily experience during the hospitalisation. The patient always clearly recognised whether she had already met a member of staff (doctor, nurse, or therapist), but never remembered who they were, their name, or the circumstances under which they had met. It is also important to stress that such selective impairment was not a problem of the speech production system. In span tasks, the patient did not show any language comprehension problems or inability to articulate words and there were no difficulties when it came to writing either. In the short version of the Boston Naming Test (ability to name objects ordered in a series), the patient showed no significant problems with naming presented objects.

The most crucial impairment resulting from the stroke was the total absence of anterograde episodic memory. The patient could not remember what she had been doing five minutes ago. The significantly reduced intrinsic and phasic alertness cannot explain the impairment of the anterograde episodic memory, nor the severe problems with semantic memory.

An apathy syndrome, which is associated with an impairment of medial frontal cortex function, was consistently observed in other patients after an AN lesion [4, 5, 9, 17]. This may indicate that, together with memory, the AN also plays an essential role in the networks responsible for processes underlying behaviour directed towards or away from environmental stimuli. In the case of our patient, we observed the absolute absence of motivation for physical, cognitive, or emotional activity.

It has been widely discussed whether lesions in the AN can lead to memory deficits. Some reports even proposed that it is not the damage to the thalamic nuclei but rather the disruptions of MTT only that cause anterograde memory impairment [16]. Most of the recent studies however show evidence that the damage or disconnection of the AN leads to profound learning and memory impairments in rodents and to so-called *recollective memory impairments* in humans [18]. In some cases, for instance, the bilateral damage to the MTT impairs recollection, but not familiarity in the recognition process [19]. Other studies presented patients with combined damage of the AN and the MTT or other nuclei [20].

In their review from 2006, Carrera and Bogousslavsky tried to describe the neuropsychological impairments that the patients with AN lesion have [5]. This review was mostly based on the Ghika-Schmid and Bogousslavsky prospective study describing 12 patients with, as they called it, *an acute behavioural syndrome* [9]. All patients showed anterograde memory impairment with delayed recall deficit and better performance for recognition. Eight patients with left-sided infarcts had delay in verbal recall; four patients with right-sided infarcts demonstrated delay in visuospatial recall. Besides other minor symptoms, patients demonstrated acute perseverative behaviour; when the disorientation to time and place wore off in some patients, chronological confusion in episodic and biographic events due to improper recall of their temporal sequence was observed. All patients also had word-finding difficulties; phonematic and semantic fluency were not examined separately.

The same pattern of impairment was confirmed in the study of six patients with isolated infarction of the left AN and MTT from Japan. The neuropsychological assessment revealed verbal memory impairment, language disturbances dominated by anomia, word-finding difficulty and apathy. In the results of the neuropsychological tests, all six patients scored very similarly to our patient. They scored significantly low in the test of verbal memory and delayed recall, as well as initial fluency and spontaneous speech [4].

We decided to test both delayed and immediate recall since in some densely amnesic patients with severe anterograde amnesia the tests may produce contradictory results, i.e. good immediate, yet poor delayed prose recall alongside the preserved short-term memory for a maximum of 5 items [21]. We tested the immediate recall using The Wechsler Memory Scale. As mentioned above,

the second logic memory task regarding prose recall considers 25 items being mentioned in three sentences; the task is similar to the 24 items test Baddeley & Hitch used to illustrate their episodic-buffer theory [21, 22]. Our patient could not recall a single item and, after a few invitations to name the items, she slipped into verbose confabulation. After 20 min, the patient was again not able to recall any of the items. This immediate recall deficit is the unique symptom that differs our patient with AN lesion from all other patients with lesions in other parts of the Papez circuit [21]. The lesion in AN seems to interfere with the episodic buffer functioning. It however remains unclear whether AN is the sought-after anatomic correlate of the episodic buffer or merely one of many subcortical structures participating in the mnemonic processing and further research is required [23].

Confabulations are also one of the diagnostic hallmarks of Korsakoff's syndrome together with anterograde and retrograde amnesia, apathy and executive dysfunction. Patients with Korsakoff's syndrome in the chronic stage show rather provoked confabulation or "confabulations on demand", when they are asked to recall past events [24]. As we mentioned above, the same was observed in our patient, i.e. only provoked confabulations. It shows probably another important role of AN in composing and retrieving memories in the right temporal order, as the temporal context confusion is being discussed also in the context of Korsakoff's syndrome [25]. The presence of confabulation is in contrast with the hippocampal amnesia. In the case of bilateral damage of hippocampi, profound amnesia without confabulation usually occurs [26].

One of the limitations of our case study is the absence of language testing with an established test battery. We performed only 30 min formal screening on the admission day conducted by professional logopaedist. It revealed only word-finding difficulties during free-recall in demanded semantic categories. There were no phonological problems, no paraphasia or problems with naming objects, no articulatory errors, and writing was flawless. We thus classified the impairment of the free recall of objects in demanded semantic categories as a specific memory problem. Nevertheless, more classical signs of aphasia like anomia and semantic paraphasia were occasionally observed in other patients with AN lesion [4].

To sum up, we have a patient suffering from the most severe anterograde episodic memory loss with preserved visual recognition and auditive short term memory for 5 ± 2 items, inability to freely recall semantic information, spatial and time disorientation, apathy, significantly reduced intrinsic and phasic attention, and immediate and delayed prose recall deficiency.

An almost identical case of a patient suffering from ischaemic infarction in the left anterolateral thalamus was published more than 40 years ago [27]. The authors from Vienna University Hospital described a case of a 40-year-old man with a specific memory deficit. The initial neurological examination revealed slight impairment of fine movements of the right hand with minimal facial paresis, global anterograde amnesia with confabulation, verbal retrieval defect, disorientation in time and general slowness. Detailed neuropsychological test results were not reported, but residual deficit of word finding as well as difficulties in understanding and memorising complex verbal information were still present during the examination conducted 7 months after discharge. The authors described the memory deficit as an inability to recall information that was singular, highly specific and was required to give details and individuality to the single recall.

We conclude, based on our results, together with assessing other similar cases, that the AN may be more than a simple relay station in the memory networks; only a specific part of the declarative memory was impaired, mostly working memory with anterograde episodic memory and semantic memory.

The common feature of anterograde amnesia after hippocampal and diencephalic damage led Aggleton & Brown to formulate the theory of the *"extended hippocampal system"*, comprising of the hippocampus, fornix, mammillary bodies and anterior thalamic nuclei. They stressed the importance of the hippocampal-anterior thalamic axis for episodic memory and proposed that the hippocampal system is not required for tests of item recognition that primarily tax familiarity judgements [28]. Although this statement may be true, this conclusion itself does not explain the peculiar neuropsychological test results in our and other similar cases. Unfortunately, the detailed description and categorisation of memory deficits or structural comparison to other known deficits has not been conducted and the authors themselves admitted in their final comments that the essential question as to why additional diencephalic processing is required for anterograde episodic memory and why we observe such a profound anterograde amnesia often without recovery remained unanswered.

We presented a case of a patient suffering from anterograde episodic memory loss with preserved visual recognition and auditive short term memory, inability to freely recall semantic information, spatial and time disorientation, apathy and significantly reduced intrinsic and phasic attention and immediate and delayed prose recall deficiency after a left unilateral AN lesion. These results show the essential role of the AN in creating episodic memory, in the working memory network and indicate its role as a critical interface between short-term and long-term memory.

Abbreviations

ADC	Apparent diffusion coefficient
AN	Anterior thalamic nucleus
CERAD	Consortium to Establish a Registry for Alzheimer's Disease
ECG	Electrocardiogram
FBI	Frontal Behavioral Inventory
CT	Computed tomography
MD	Mediodorsal thalamic nucleus
MRI	Magnetic resonance imaging
MTT	mammillothalamic tract
NIHSS	The National Institutes of Health Stroke Scale
TAP	Test of Attentional Performance
TSH	Thyroid stimulating hormone
WAIS-IV	Wechsler Adult Intelligence Scale– Fourth Edition
WMS	The Wechsler Memory Scale

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Conception and design of the work– JG. Neuropsychological testing and analysis– EL. Drafting of the abstract, background, discussion and conclusions– JG. Drafting of the case presentation– JG, EL. Final revision and editing of the manuscript– JG, EL, RM.

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Data availability
The authors declare that all the data are contained within the manuscript.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
Written informed consent for publication was obtained from both the patient and her husband.

Competing interests
The authors declare no competing interests.

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